



Q fever

CDNA National Guidelines for Public Health Units

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The Series of National Guidelines ('the Guidelines') have been developed by the Communicable Diseases Network Australia (CDNA) and noted by the Australian Health Protection Principal Committee (AHPPC). Their purpose is to provide nationally consistent guidance to public health units (PHUs) in responding to a notifiable disease event.

These guidelines capture the knowledge of experienced professionals and provide guidance on best practice based upon the best available evidence at the time of completion.

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Q fever

CDNA National Guidelines for Public Health Units

1. Summary

Public health priority

Sporadic cases: Routine. Action should be carried out as part of routine duties. Data entry should be completed within 5 working days.

Outbreak: High. Act as soon as possible, generally within one working day. Data entry should be commenced within 3 working days.

Case management

Q fever cases can be treated with appropriate antibiotics. All notifications should be followed up to ascertain the most likely source of infection, and to determine if there are any other linked cases.

Management of co-exposed persons

Whilst person-to-person transmission is rare, there may be individuals who have been exposed to a common source (co-exposed). Identify co-exposed individuals (e.g. those at the same workplace), and advise them of the early signs and symptoms of Q fever to aid early diagnosis and treatment.

In responses to Q fever linked to workplace/occupational settings, the local workplace health and safety regulator should be involved, as is the animal health authority (if relevant); workers should be assessed for immunity. Non-immune workers should not perform work that exposes them to Q fever risks until at least 15 days after vaccination against Q fever.

2. The disease

Infectious agents

The infectious agent is *Coxiella burnetii*, an obligate intracellular Gram negative coccobacillus. It is a highly infective and efficient pathogen, with an extremely long biological half-life.¹ The disease was first described as Q (for query) fever in 1937 by Edward Derrick in Queensland,² with the organism subsequently identified through culture almost simultaneously by Cox³ in the US and Burnet⁴ in Australia. The organism has since been found around the world (with the exception of Antarctica and possibly New Zealand).⁵

Reservoir

Cattle, sheep, and goats are the primary reservoirs for *C. burnetii*,^{6, 7} but a wide range of domestic^{8, 9} and wild animals can be infected,¹⁰ 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.¹¹

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.¹² Shedding of high numbers of organisms from infected animals occurs particularly with birth products (e.g. placental tissue and birth fluids).¹² *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.^{13, 14}

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).¹⁵ It is resistant to a variety of harsh environmental conditions, including elevated temperatures, desiccation, osmotic shock, UV light, and chemical disinfectants.¹⁶ It can survive as an infectious agent on wool at 15–20°C for 9 months and on fresh meat in cold storage for more than a month.

Mode of transmission

- Respiratory route: the most common mode of transmission to humans is via the respiratory route following inhalation of contaminated aerosols or dust,¹⁷ 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
 - *C. burnetii* can survive in dust for months to years. Windborne spread of contaminated dust can disperse the organism over several kilometres.¹⁸ 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,^{17, 19} 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 limited⁷
- Vector-borne: *C. burnetii* has been detected in numerous tick species in Australia,^{11, 20, 21} but human infections from ticks have been infrequently documented,^{19, 21, 22} possibly through tick bites or inhalation of tick excreta.
- Person-to-person transmission is very rare but can occur through:
 - Blood transfusion²³ or bone marrow transplant²⁴
 - Vertical or perinatal transmission²⁵
 - Autopsy of infected cadavers⁵
 - Sexual transmission²⁶

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.²⁷

C. burnetii has been listed as a Category B bioterrorism agent by the US Centers for Disease Control and Prevention,^{12, 28} due to its ease of production, survival in desiccation, and transmission through inhalation.

Incubation period

Typically, the incubation period is 2–3 weeks, depending on the size of the infecting dose (range 4 days to 6 weeks).⁷

Infectious period

Person-to-person spread rarely occurs. Immunity following recovery from clinical illness may be life-long,¹² 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.

Clinical presentation and outcome

Following infection with *C. burnetii*, the majority of cases (60%) will be asymptomatic/subclinical infections.¹⁹ 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.⁵ Commonly reported signs and symptoms include fever, chills, sweats, severe headache (especially behind the eyes), photophobia, weakness, anorexia, nausea, myalgia, cough, and weight loss. Patients can present

with mild hepatitis associated with *C. burnetii* infection, which is more frequently acquired in sheep and goat-breeding areas.⁶

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.

A minority of infected cases ($\leq 1\%$) may develop pericarditis, myocarditis,²⁹ or neurologic complications (e.g. meningoencephalitis, encephalomyelitis).⁵ Infection in pregnant women (symptomatic or not) can lead to abortion, premature delivery, or low birth weight.³⁰ The case fatality rate for untreated acute cases is usually less than 1%.¹²

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.

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.¹⁹ The most important factors associated with progression to endocarditis following primary Q fever infection are underlying valvular heart disease/valvular prosthesis.^{29, 31} 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%.¹²
2. Osteoarticular infections. Bone and joint *C. burnetii* infections have been reported, occurring in less than 1% of hospitalised Q fever cases.³² Osteomyelitis appears to present more frequently in children than in adults, with evidence of granulomatous bone lesions.²⁹ Reported joint infections involve multiple locations, including wrist, tibia, ankle, shoulder, and prosthetic joints (the knee and hip).²⁹
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%.²⁹ The abdominal or thoracic aorta is the most frequent site for vascular infection.

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.³³ QFS is the most common sequela following

acute infection in Australia, occurring in approximately 10–15% of patients with acute Q fever.¹⁹

Persons at increased risk of disease

- At-risk occupational groups (including contractors within the industries) are those with contact of high-risk animals or animal products,¹⁹ 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).
 - 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,^{34, 35} meatworks,³⁶ 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.
 - 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:^{29, 31}
 - Immunosuppressed persons
 - Pregnant women
 - Persons with valvular heart disease/valvular prosthesis
 - Persons with aneurysms/vascular grafts.

Disease occurrence and public health significance

Q fever is a zoonotic disease that occurs around the world. The true incidence of disease is greater than that reported because of subclinical infection, as well as limited clinical suspicion and testing. In Australia, there were around 500–800 notifications (2.5–5.0 per 100,000 population) annually in the 1990s.³⁷ During 2001–2006, 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.³⁸ 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 2016.³⁷ Q fever notification rates are relatively high in Australia compared with European countries (0.18 per 100,000 population in 2014)³⁹ and the US (0.04 per 100,000 population per year).⁴⁰

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 2011–2015.³⁷ The notification rate remains highest in south west/central west Queensland and northwest New South Wales,³⁸ 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 (Table 1). 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).^{41, 42} 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 2009.⁴³

Table 1: Summary of some Q fever outbreaks reported in Australia (up to 2015)

Outbreak setting	Year	Number of cases
Abattoir in Victoria ⁴⁴	1979	110 abattoir workers
Abattoir in NSW ^{45, 46}	1998	29 confirmed and 8 suspected cases
Goat farm in Queensland ⁴⁷	2003	5 cases
Animal saleyard in South Australia ⁴⁸	2004	25 cases exposed to infected sheep and dust
Cosmetics factory in ⁴⁹ Victoria	2006	4 cases linked to processing partially defrosted sheep placentas and fetal tissue
Abattoir in South Australia ⁵⁰	2007	5 confirmed cases and 1 possible fatal case
Veterinary hospital in NSW ⁸	2010	9 veterinary personnel and 1 cat owner linked to an infected cat undergoing a caesarean section
Farm in Victoria ⁵¹	2011	5 cases involved in calving

Outbreak setting	Year	Number of cases
Veterinary hospital in NSW ⁵²	2012	3 veterinary nurses attending to an infected dog undergoing a caesarean section
Goat dairy farm in Victoria ¹³	2012–2014	18 confirmed cases (17 employees and 1 family member)
Remote rural town in NSW ⁵³	2014–2015	14 confirmed cases (3 in high risk occupations and 11 in non-animal related occupations)
Abattoir processing feral goats in Queensland (unpublished)	2015	9 abattoir workers

3. Routine prevention activities

Vaccination⁵⁴

Q fever vaccine (Q-VAX[®]) has been available in Australia since 1989, with efficacy estimated at 83-100%.¹⁹ 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 2 (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 2) 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.⁵⁴

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](http://www.qfever.org) 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^{55, 56}

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
- Yard facilities for sheep, goats, and cattle should be situated well away from residential domestic living areas.

Safe workplace practices^{55, 56}

- 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 foetuses 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.⁷ In the Q fever outbreak settings in the Netherlands, the population risk of infection was substantially higher within five kilometres of infected dairy goat farms.⁵⁷

4. Surveillance objectives

1. To monitor trends in Q fever with respect to time, population groups, geography, and risk factors.
2. To identify a likely source of infection so that the likelihood of further cases from the same source can be minimised, such as in workplace settings.
3. To detect and guide immediate action and control measures for outbreaks to prevent further transmission.
4. To guide the planning and implementation of policy, service provision, prevention strategies, and other public and animal health interventions.

5. Data management

Within 5 working days of notification, enter cases onto the notifiable diseases database. In an outbreak setting, data should be entered within 3 working days following notification.

No evidence of reinfection has been documented, and full recovery from an acute Q fever infection usually confers life-long immunity. Chronic manifestations of Q fever following a primary infection are not considered as reinfection. As such, there should be no second notification of Q fever from the same person.

6. Communications

Public Health Units work collaboratively with healthcare providers and patients to ascertain Q fever cases, complete the case investigation, identify further cases of similar exposures, and provide information and education on Q fever prevention and control (see Section 9 for details).

In the context of responding to a Q fever outbreak or cases occurring in workplace settings, the following jurisdictional government agencies should be included for information sharing and joint investigation (see Section 12 for details):

- Workplace health and safety regulator
- Animal health authority
- Local government authority
- Health authorities of neighbouring jurisdictions, when appropriate.

7. Case definition

The case definition may have been updated since the publication of this guideline. Please check the [case definitions webpage](http://www.health.gov.au/internet/main/publishing.nsf/Content/cdna-casedefinitions.htm) on the Australian Department of Health's website (www.health.gov.au/internet/main/publishing.nsf/Content/cdna-casedefinitions.htm) for the latest version.

Reporting

Only confirmed cases should be notified.

Confirmed case

A confirmed case requires either:

1. Laboratory definitive evidence
- OR
2. Laboratory suggestive evidence AND clinical evidence.

Laboratory definitive evidence

1. Detection of *Coxiella burnetii* by nucleic acid testing
- OR
2. Seroconversion or significant increase in antibody level to Phase II antigen in paired sera tested in parallel in absence of recent Q fever vaccination
- OR
3. Detection of *C. burnetii* by culture (note this practice should be strongly discouraged except where appropriate facilities and training exist: Section 8 - culture)

Laboratory suggestive evidence

Detection of specific IgM in the absence of recent Q fever vaccination.

Clinical evidence

A clinically compatible disease.

The most recent Australian national notifiable diseases [case definition](http://www.health.gov.au/casedefinitions) for Q fever can be found at the Department of Health website (www.health.gov.au/casedefinitions).

Please note, the above Q fever case definition does not differentiate between acute and chronic Q fever, and potentially excludes some chronic Q fever cases due to exclusion of serology testing for antibodies to Phase I antigen in the definition.

8. Laboratory testing

Testing guidelines

A series of blood specimens should be requested if acute Q fever infection is suspected and should include:

1. Unclogged blood or serum for Q fever PCR (and possible culture), AND
2. Paired (acute and convalescent) serum/clotted blood specimens taken 2–3 weeks apart for serology. The collection of convalescent sera from all cases is critical, even if the patient has since recovered.

The steps for laboratory diagnosis are illustrated in Appendix 4. Further detail on tests and interpreting results is provided below, as well as on Public Health Laboratory Network (PHLN) laboratory case definitions website.

Consideration should also be given toward other zoonotic diseases based on risk exposures.

PCR testing

In acute Q fever cases, the organism may be detected in blood up to 2 weeks after illness onset. If the patient presents within this period, unclotted blood or serum should be submitted for PCR (and possible culture – see below). Whilst PCR offers a highly sensitive method for detecting both live and dead *C. burnetii*, a negative result alone does not exclude a Q fever diagnosis, and serological testing should be completed for all cases.

In chronic Q fever cases, *C. burnetii* DNA may be detected by PCR in peripheral blood mononuclear cells or in biopsy specimens from focally infected tissue (e.g. heart valves, bone, synovium). The sensitivity of PCR in serum in patients with endocarditis or vascular infection is low to modest, in the order of 23–67%.⁵⁸

PCR positive with negative serology results confirm acute Q fever, and theoretically convalescent serology is not indicated, however it is useful if serial testing is performed at intervals to screen for chronic infection.

Serology testing

Indirect immunofluorescence assay (IFA) is the reference method, but the complement fixation test (CFT) and enzyme immunoassays (EIA) are also used to support diagnoses. For the diagnosis and follow up of *C. burnetii* infection, IFA to both phase I and phase II antigens for subclasses IgM, IgG, and IgA is recommended.

It is important to note that single serology tests (EIA, CFT, or IFA) are unable to distinguish between acute, past and chronic infections, and antibody detection is highly dependent on the timing of specimen collection. Two serum/clotted blood samples should, therefore, always be collected if Q fever is suspected — one at presentation, and another 2–3 weeks later, even if the patient has since recovered. Seroconversion usually occurs within 7–15 days after exposure, and ninety per cent (90%) of cases have seroconverted by the third week after exposure.

Significant antibody titres may take 3–4 weeks from illness onset to appear in some cases. In cases not definitively confirmed by other means, a third sample is recommended, which should be collected 3–4 weeks after fever onset.

The interpretation of Q fever serology results can be challenging. There are different patterns of antibody response during the course of acute Q fever and chronic Q fever, specifically in terms of antibody subclasses to Phase I and Phase II antigens.¹⁹ For example, in acute Q fever cases, IgM to Phase II antigen tends to rise first, followed by rise of IgG and IgA to Phase II antigen as detected by IFA at various times after the onset of disease.⁵⁹ In contrast, for chronic Q fever (e.g. endocarditis), IgG, with or without IgA to Phase I antigen, is present at high titre.^{19, 60} A chart and reference tables are provided at Appendix 5 to assist in interpreting serology results. Where required, Public Health Units should seek expert guidance in interpreting these results.

Management of acute Q fever includes the measurement of serial antibody titres over time. This can aid in identifying those at risk of developing chronic Q fever, and allow early interventions. It is preferable that confirmed cases have testing undertaken at a laboratory with such capacity.

Culture

Isolation of *C. burnetii* by culture can only be performed by vaccinated staff in an appropriate physical containment level 3 (PC3) facility.⁶¹ For this reason, culture is strongly discouraged except where appropriate facilities and training exist – currently culture is only performed at the Australian Rickettsial Reference Laboratory (ARRL), Geelong, Victoria.

9. Case management

Response times

Sporadic cases: Routine response. Initial case investigation and data entry should be completed within 5 working days.

Outbreak: High priority response. The responsible Public Health Unit should act and notify the jurisdictional Communicable Disease Branch as soon as possible, generally within 1 working day. Data entry should be commenced within 3 working days.

Response procedure

Case investigation

Public Health Unit staff carry out case investigation in collaboration with the case's treating doctor and the case.

Information required from treating doctor:

Complete relevant sections of the Q fever Case Investigation Form (Appendix 3) with treating doctor to:

- Determine clinical details (including onset date, symptoms, acute or chronic presentation, hospitalisation, and outcome)
- Determine previous vaccination status
- Request repeat serology to confirm the diagnosis, if required
- Document case treatment and follow-up of those who are at higher risk of chronic Q fever (see Case treatment)
- Obtain contact details of the case, and ensure that the case has been informed of their diagnosis and an understanding that a follow up interview will occur.

Case interview and exposure investigation:

Complete relevant sections of the Q fever Case Investigation Form (Appendix 3) with the case (or carer) to:

- Explore risk factors for infection including occupational risk factors, contact with animals through farming, hunting or other activities, and environmental exposures

- Determine the possible source of infection
- Confirm vaccination history
- Provide education (see below).

If the case involves an occupational exposure, see Section 12 for additional actions.

Case treatment

Commence empiric treatment if Q fever is clinically suspected. Do not wait for laboratory results. Refer to latest edition of the *Therapeutic Guidelines: Antibiotic*.

A two week course of oral doxycycline is generally used to treat acute Q fever. Trimethoprim+sulfamethoxazole is recommended for pregnant women until 32 weeks of gestation, even if recovered, to prevent fetal and maternal complications.³⁴

After treatment of *C. burnetii* primary infection, it is recommended to screen for risk factors of chronic Q fever infection, including pre-existing valvular heart disease/valvular prosthesis, vascular aneurysms/vascular grafts, and immunosuppression.²⁹ A cardiac assessment, which may include echocardiography, is recommended to assess whether there are underlying abnormalities of the heart valves. Those who are at higher risk of chronic Q fever should be monitored serologically and clinically at 3, 6, 9, 12, 18, and 24 months after acute infection. There is a body of evidence to suggest antibiotic prophylaxis for 12 months in case of cardiac valve problems/valvular prosthesis may be of benefit.²⁹

In chronic disease (e.g. endocarditis), prolonged combination therapy (with addition of hydroxychloroquine) and cardiac surgery may be required. Expert advice from an infectious diseases physician and other specialist physicians should be sought as appropriate.

Education

The case should be advised of the nature of the infection and its mode of transmission, and of appropriate precautions necessary to prevent others from becoming infected from exposure to the same source. The case should also be advised about seeking medical care if symptoms do not resolve following completion of treatment, or new symptoms develop that may indicate a complication or a chronic Q fever infection (Appendix 1: Q fever Factsheet).

Isolation and restriction

Exclusion of infected persons is not required. Q fever is rarely transmissible from person to person.

Active case finding

See Section 12 for active case finding in special situations.

10. Environmental evaluation

C. burnetii is highly resistant to desiccation and may remain viable in dust for more than a year. It is killed by heat (>63 degrees Celsius for 30 minutes), and some disinfectants including hydrogen peroxide, sodium hypochlorite (at concentrations of

greater than 5 per cent), and 2 per cent formaldehyde. A 1:100 dilution of household bleach is also an effective solution.²⁹

Appropriate animal and environmental management plays an important role in reducing transmission of *C. burnetii* to humans. Lessons and experience drawn from Q fever outbreaks in Victoria¹³ and the Netherlands,¹⁴ which were linked to goat dairy farms, point to effective measures in reducing Q fever risk, including:

- Immediate removal of animal abortive and birth materials and safe disposal by deep burial
- Appropriate treatment of animal manure: no removal of manure from the deep litter sheds or yards for at least one month after the kidding season; composting manure or alternatively storing manure for three months prior to spreading on farm land for fertiliser
- Manure should be covered during storage and transport and must be under-ploughed immediately when spreading on farm land
- There is no vaccination available for use in animals in Australia. Where available, vaccination has been used in female animals prior to their first pregnancy⁶² to prevent them becoming a source of *C. burnetii* transmission, and human infection.

11. Management of co-exposed persons

Identification of co-exposed persons

In occupational settings and outbreaks, active case finding among identified co-exposed persons should be considered (Section 12). The aim of identifying co-exposed persons is to alert them to the possibility that they could develop disease due to a common source exposure.

Co-exposure definition

A co-exposed person is defined as anyone who may have experienced the same occupational, animal, or environmental exposures as the case, or who may have been exposed to contaminated items associated with the case (e.g. clothing/boots). Person-to-person transmission is extremely unlikely. Co-exposed persons may include people at the workplace (including those without direct contact with animals or animal products) and home.

Prophylaxis

Vaccination during the incubation period does not prevent the disease. Post-exposure antibiotic prophylaxis is not recommended.

Education

Q fever information (Appendix 1) should be provided to co-exposed persons with advice to seek medical attention should they develop symptoms. Q fever vaccination should be recommended to all non-immune workers in high-risk occupations.

Isolation and restriction

Those workers/co-exposed persons with the same exposure that do not have immunity to Q fever through natural infection or vaccination should not visit the setting, enter high risk workplaces or perform work that exposes them to Q fever risks without wearing a properly fitted particulate respirator (e.g. disposable P2 respirator).

12. Special situations

In addition to the generic case and co-exposed person follow-up requirements described above, further actions are required in the following instances:

Cases and outbreaks linked to workplace/occupational settings

Q fever case investigation may identify a plausible link with a workplace (such as an abattoir or dairy farm). Two cases or more within a three-month period in an at-risk workplace is considered a workplace outbreak.

Responses to cases occurring in workplace settings (including outbreaks) need to be carried out in collaboration between the Public Health Unit, the local workplace health and safety regulator, and the animal health authority if relevant. Unvaccinated Public Health Unit staff are not to be exposed to Q fever risks as part of the disease investigation and response.

Immediate responses include working with the employer and management to:

- Conduct active case finding in the at-risk setting, including urgent testing of workers with a current or recent clinically compatible illness. Laboratory-definitive evidence should be actively pursued for all suspected cases, including obtaining convalescent sera from ill workers with a single negative serology result (even if they have since recovered)
- Assess vaccination status of all workers (if not already known), institute a Q fever vaccination program urgently if one is not in place, and maintain written records of employee Q fever status and vaccination
- Restrict non-vaccinated workers (including not working in a high risk area for Q fever until at least 15 days after vaccination against Q fever); or (if restricting is not possible) provide other interim means of personal protective equipment such as use of a properly fitted particulate respirator (e.g. disposable P2 respirator).

The role of the workplace health and safety regulator is to investigate and identify unsafe working conditions, and to monitor and enforce compliance. This may involve a site visit and discussions with the employer. The workplace health and safety regulator may, in consultation with the health department and the animal health authority, provide information and advice to the employer.

Community clusters/Family clusters

The term 'cluster' is taken to mean the occurrence of more cases than expected in the community, where sources of infection are not apparent. For example, the year to date number of Q fever notifications from a region is over 2 standard deviations more than the previous five year mean over the same period.

The goal of community cluster detection is to further explore potential sources of infection and risk factors for Q fever in a broader community context, thereby informing public health action to interrupt transmission and prevent further cases.

Elements of the cluster response include:

- In-depth analysis of epidemiological information of cases, e.g. age-specific rates, region-specific rates, timeline and mapping of cases and possible exposure sites and sources. In small populations it may be more useful to focus on the number of cases rather than the rate
- Consider environmental and meteorological conditions, such as use of animal manure as fertiliser on farm land, wind direction, and rain patterns in recent weeks, to determine possible high risk zones. Consider defining 1 km/5 km zones around the potential or identified source
- Work collaboratively with GPs and hospital emergency departments in the local area, to be alert for Q fever cases and initiate active case finding
- If potential sources of infection are suspected, work collaboratively with relevant organisations (e.g. environmental health, animal health authority, workplace health and safety authority) to conduct risk assessment and take actions to minimise/eliminate risks
- Depending on the extent of the cluster, consider liaising with the Australian Red Cross Blood Service and other appropriate national institutes as blood donation services in cluster locations may need to be restricted
- Consider targeted vaccination programs to reduce the risk of disease in groups identified at higher risk. If a significant outbreak, additional funds may be available to support the vaccination strategy.

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14. Appendices

- Appendix 1: Q fever Factsheet
- Appendix 2: PHU Q fever Checklist
- Appendix 3: National Q fever Case Investigation Form
- Appendix 4: Q fever Laboratory Diagnosis Flowchart
- Appendix 5: Q fever Laboratory Result Interpretation

15. Jurisdiction specific issues

[Links](#) to State and Territory Public Health Legislation, the Biosecurity Act 2015 and the National Health Security Act 2007.

www.health.gov.au/internet/main/publishing.nsf/Content/cda-state-legislation-links.htm

Appendix 1: Q fever Factsheet

What is Q fever?

Q fever is an illness caused by the bacterium *Coxiella burnetii*, carried by animals such as cattle, sheep, goats, and kangaroos. Humans usually catch the infection by breathing in droplets and dust contaminated by birth fluids, faeces, or urine from infected animals. *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), which can also lead to infection and disease. Spread of infection from person-to-person is rare. Q fever can be treated with antibiotics.

Q fever is usually an acute (immediate) infection, but sometimes it can lead to a chronic (long-term) illness.

What are the symptoms?

Many infected people have no symptoms. People who do become sick often have a severe flu-like illness. Symptoms begin about 2–3 weeks after exposure to the bacteria. However this period can be as short as 4 days and as long as 6 weeks.

Typical symptoms of acute Q fever include:

- Fever and chills
- Sweats
- Severe headache (especially behind the eyes)
- Muscle pain
- Weakness and tiredness
- Weight loss.

Some patients may develop pneumonia and hepatitis during the course of acute illness. Most people make a full recovery and become immune to future Q fever infections.

Occasionally people may develop chronic infections that affect the heart (endocarditis), bone (osteomyelitis), or joints. Some people develop chronic fatigue (post-Q fever fatigue syndrome) that can last for many years. Persons at increased risk for chronic Q fever after acute infection include: immunosuppressed persons (e.g. cancer patients with chemotherapy, patients with organ transplantation), pregnant women, and persons with heart valvular abnormalities.

How is it spread?

The bacteria are found in many animals, including cattle, sheep, goats, dogs, cats, horses, pigs, rodents, camels, and kangaroos. The bacteria are also found in ticks. Infected animals usually have no symptoms, but abortion, stillbirth, and infertility may result.

Infected animals shed high numbers of bacteria in birth by-products such as the placenta and birth fluids. The bacteria can also be shed to the environment from faeces, urine, and milk of infected animals. The bacteria are highly infective and can survive in the general environment (e.g. in dust and soil) for months and years.

- Humans most commonly catch the infection by breathing in droplets and dust containing the bacteria from birth fluids, faeces, urine, or blood of infected animals, in circumstances such as:
 - Assisting animal birth
 - Animal slaughtering/skinning/meat processing
 - Herding
 - Shearing/wool processing

- Working with animal manure
- Transporting infected animals
- Veterinary/diagnostic procedures
- Infection can also occur through direct contact with infected animal tissue or fluids on broken skin - for example, through cuts with contaminated knives or needle-stick injuries when working with animals
- Consuming unpasteurised (raw) milk or milk products from infected animals may carry a risk of contracting the infection
- Tick to human transmission occurs infrequently, through tick bites, breathing in tick excreta or direct contact (e.g. removal of ticks from domestic animals, aerosol-generating activities such as shearing, or crushing ticks with bare hands)
- Person-to-person spread of infection is rare, but can occur through blood transfusion and mother-to-baby transmission.

Who is at risk?

People whose work exposes them to high risk animals, animal products, and animal excreta have high risk of developing Q fever. These high risk occupations include:

- Abattoir and meat workers
- Agriculture, livestock and dairy farmers and 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 working with veterinary specimens
- Agriculture college staff and students working with high risk animals
- Animal shooters/hunters
- Laboratory personnel working with materials containing the bacterium *Coxiella burnetii*
- Wildlife/zoo workers, animal trainers
- 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 high 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/goats, meatworks, land being fertilised by untreated animal manure)
- Visitors to at risk environment (e.g. farms, abattoirs, animal saleyards)
- People living near livestock transport routes with the potential being exposed to contaminated dust from the passing animals
- People involved in mowing which stirs up dust potentially contaminated by animal excreta, in areas where there are livestock or wild animals
- People who observe or assist animal births.

How is it prevented?

A Q fever vaccine is available to protect people against the disease. Vaccination is recommended for all people who are working in, or intend to work in, a high risk occupation (see Who is at risk?). High risk workplaces should have a vaccination program to protect their workforce.

People at risk of Q fever through non-occupational, environmental exposures (see Who is at risk?) are also recommended for vaccination.

People must be screened and tested before they are vaccinated against Q fever. Check the [Australian Q fever Register](http://www.qfever.org) (www.qfever.org) to find a doctor specially trained for Q fever vaccination services.

Apart from vaccination, people can take steps to reduce the risk of Q fever to the community, including:

- Washing the hands and arms thoroughly in soapy water after any contact with animals
- Wearing a P2 respirator (available from pharmacies and hardware stores) and gloves in handling and disposing of animal products, waste, placentas, and aborted fetuses
- Preventing animals from eating placenta, immediate removal of animal abortive and birth materials, and safe disposal by deep burial – do not use them in compost
- Personal protective equipment and contaminated clothing 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
- Appropriate treatment of animal manure: no removal of manure from the deep litter sheds or yards for at least one month after the kidding season; composting manure or alternatively storing manure for three months prior to spreading on farm land for fertiliser
- Manure should be covered during storage and transport and must be under-ploughed immediately when spreading on farming land
- Minimising dust and aerosols in slaughter and animal housing areas.

How is it diagnosed?

Your doctor can diagnose Q fever based on symptoms, clinical examination, and laboratory tests on blood samples. Two or more blood samples on separate occasions are often required to confirm a Q fever diagnosis.

How is it treated?

A two week course of oral antibiotics is generally used to treat acute Q fever. Chronic Q fever requires prolonged treatment with antibiotics.

What is the public health response?

Laboratories must notify cases of Q fever to the health department/Public Health Units.

Public Health Unit staff talk to the treating doctor and patient (or carer) to determine the possible source of infection, identify other people at risk of infection, ensure control measures are in place, and provide information and education.

If Q fever cases are linked to a workplace (e.g. an abattoir or dairy farm), the local Public Health Unit will involve the local workplace health and safety regulator and the relevant animal health authority.

The role of the workplace health and safety regulator is to investigate and identify unsafe working conditions, make recommendations to the employer, and monitor the control measures implemented.

Appendix 2: PHU Q fever Checklist

Patient ID number:

Contact the patient's doctor to:

- Ascertain patient's history
- Obtain patient's contact details and permission to contact the patient
- Confirm results of relevant pathology tests.

Contact the patient (or care giver) to:

- Confirm onset date and symptoms of the illness
- Identify likely source of infection
- Identify co-exposed persons and obtain their contact details
- Complete Q fever Disease Investigation Form
- Provide Q fever Factsheet.

Contact laboratory to:

- Check samples received and obtain any outstanding results.

Confirm case

- Assess information against the case definition and classify the case.

Contact co-exposed persons of the patient to:

- Identify other possible cases in occupational settings and outbreaks.

Other issues:

- Document case treatment and follow-up of those who are at higher risk of chronic Q fever
- Involve the local workplace health and safety regulator and the relevant animal health authority if Q fever cases occur in an occupational setting where the workplace is the presumed site of exposure
- Consider long term follow up of cases for early ascertainment of chronic Q fever and refer for management.

Appendix 3: National Q fever Case Investigation Form

Red text denotes core fields for enhanced national surveillance

NOTIFICATION:

Date notified:/...../..... Notification ID:

Interviewer name: PHU:

CASE DETAILS:

First name: Last name:

Sex: Female Male Date of birth:/...../..... Age (years):

Address:

State/Territory: Postcode:

Telephone: Mobile:

Email:

Parent/carer:

Indigenous status: Aboriginal Torres Strait Islander Aboriginal & Torres Strait Islander
 Non-Indigenous Unknown

Country of birth: Primary language:

Occupation(s) in month prior to illness:

Primary activities/duties at work (list all):

Company/employer: Name:

Address:

Contact person: Phone:

CLINICAL DETAILS:

Treating doctor: Name: Practice name:

Address:

Phone:

Date of onset of symptoms:/...../..... Date of first consultation:/...../.....

- | | | | | |
|--|---|---|--|---|
| <input type="checkbox"/> Fever | <input type="checkbox"/> Abdominal pain | <input type="checkbox"/> Headache | <input type="checkbox"/> Cough | <input type="checkbox"/> Eye pain |
| <input type="checkbox"/> Sweats | <input type="checkbox"/> Nausea | <input type="checkbox"/> Weight loss | <input type="checkbox"/> Sore throat | <input type="checkbox"/> Any heart problems |
| <input type="checkbox"/> Chills/rigors | <input type="checkbox"/> Vomiting | <input type="checkbox"/> Loss of appetite | <input type="checkbox"/> Shortness of breath | |
| <input type="checkbox"/> Fatigue/lethargy | <input type="checkbox"/> Diarrhoea | <input type="checkbox"/> Jaundice | <input type="checkbox"/> Chest pain | |
| <input type="checkbox"/> Joint/muscle pain | <input type="checkbox"/> Malaise | <input type="checkbox"/> Abnormal LTFs | <input type="checkbox"/> Pneumonia | |

Other symptoms:

Hospitalised: Yes No Unk If yes, hospital name: Days in hospital:

Underlying conditions: Immunosuppressed Valvular heart disease Other:

Pregnancy (case or case's partner): Yes No Unk If yes, gestational age (weeks):

Complications: Yes No Unk If yes, specify:

Outcome: Still ill Recovered Died If died, date of death:/...../.....

Duration of illness (days): Time off work (days):

Family member with similar illness: Yes No Unk If yes, list name, relationship, onset date:

LABORATORY CRITERIA:

Tests completed:	Specimen collection date:	Results: (for each serology test completed, list method (e.g. EIA/CFT/IFA), target antibodies & titres, if available)
<input type="checkbox"/> PCR/NAT/...../.....	<input type="checkbox"/> <i>C. burnetii</i> detected <input type="checkbox"/> Not detected
<input type="checkbox"/> Serology 1 (acute sample)/...../.....	
<input type="checkbox"/> Serology 2 (convalescent sample)/...../.....	
<input type="checkbox"/> Serology 3 (repeat, if needed)/...../.....	
<input type="checkbox"/> Culture*/...../.....	<input type="checkbox"/> <i>C. burnetii</i> isolated <input type="checkbox"/> Negative

*Culture is not considered a routine diagnostic test, and is strongly discouraged except where appropriate facilities and training exist.

History of past Q Fever infection? Yes No Unk

... If yes, describe (incl. any lab results):

VACCINATION HISTORY:

Previous screening: Yes No Unk If yes, date:/...../..... Result:

Previous vaccination: Yes No Unk If yes, date:/...../.....

If not vaccinated, reason: Did not know about vaccine/Q fever Previous infection/skin test reactive

(tick all that apply) Cost/too expensive Too young Chose not to No local providers

Other, specify:

EXPOSURE HISTORY: All questions in this section relate to the month prior to illness onset**Exposure period:**

Date:/...../..... to Date:/...../.....
(Onset of symptoms - 1 month) (Date of onset of symptoms)

Animal exposures:

If yes for any, give details: (e.g. activities, animals involved, locations, use of personal protect equipment)

Direct contact with animals: Y N U

... if yes, tick all type(s) that apply: Cattle Sheep Domestic goats Feral goats Domestic pigs

Feral pigs Kangaroos Small marsupials (e.g. bandicoots)

Cats Dogs Other, specify:

Direct contact with animal tissues or fluids (e.g. blood, bone, viscera, skin/hides, urine): Y N U

Slaughtering, skinning or meat processing: Y N U

... if yes, was this in an abattoir: Y N U

Assisted or observed an animal birth: Y N U

... if yes, direct contact with birthing materials (e.g. placenta, fluids) or newborns: Y N U

Hunting or shooting: Y N U

Shearing, wool processing or wool classing: Y N U

Contact with pelts or hides (incl. tanning): Y N U

Contact with straw or animal bedding: Y N U

Contact with animal manure/animal fertiliser: Y N U

Attended a saleyard or animal show: Y N U

Animal exposures: **If yes for any, give details: (e.g. activities, animals involved, locations, use of personal protect equipment)**

- Observing veterinary practices: Y N U _____
- Directly undertaking veterinary practices: Y N U _____
- Consumed unpasteurised milk or milk products: Y N U _____

Environmental exposures: **If yes, give details for each exposure: (e.g. activities, location, any animals present, etc.)**

- Travel within state, interstate or overseas: Y N U _____
- Lives on a farm/station or rural property: Y N U _____
- Visited a farm/station or rural property: Y N U _____
- Visited a facility that processes animal products (e.g. abattoir, factory, etc.): Y N U _____
- Exposure to dust from paddocks/animal yards: Y N U _____
- Lives/works near an abattoir/animal grazing area/saleyards: Y N U _____
- Exposure to trucks transporting livestock: Y N U _____
- Direct contact with clothes worn by someone who works with animals (e.g. laundered): Y N U _____
- Direct contact with ticks: Y N U _____
- ... if yes, bitten by ticks: Y N U _____
- Gardening in areas with, or mowing over, wildlife (e.g. kangaroos) faeces: Y N U _____

NOTIFICATION DECISIONS:

Place acquired: Within Australia, specify State/Territory: _____ Overseas, specify country: _____ Unknown

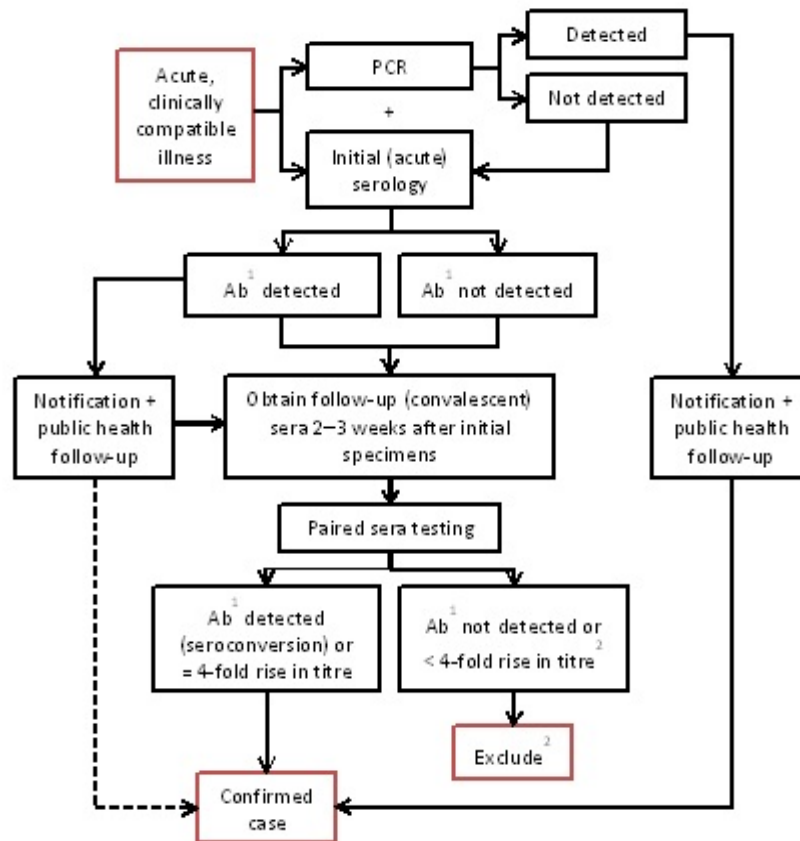
Source of infection: _____

Case classification: Confirmed acute Q fever Probable acute Q fever Chronic Q fever
 Unlikely Q fever Lab results pending

ADDITIONAL COMMENTS:

Appendix 4: Q fever Laboratory Diagnosis Flowchart

Flowchart illustrating the idealised laboratory diagnosis of acute Q fever infection

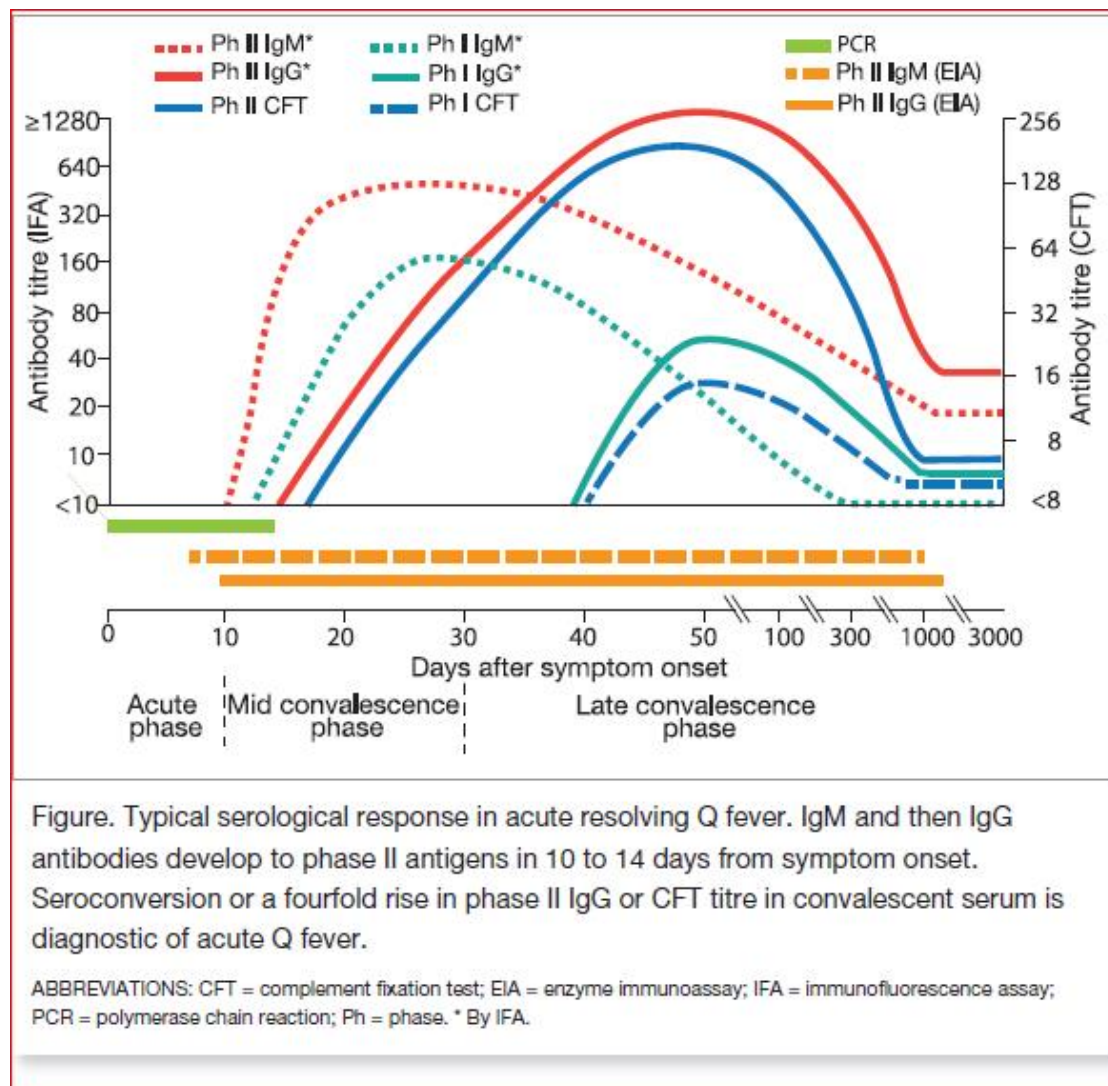


¹ Serological patterns and the detection of Q fever-specific antibody (Ab) is dependent on the time from onset of illness, assay completed (i.e. IFA, CFT, EIA) and past exposure to the organism. Refer to Section 8 and Appendix 5 for detail.

² Significant antibody titres may take 3–4 weeks from illness onset to appear in some cases. In cases not definitively confirmed by other means, a third sample is recommended, which should be collected 3–4 weeks after fever onset.

Appendix 5: Q fever Laboratory Result Interpretation

Typical serological response in acute Q fever⁵⁹



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Source: Medicine Today April 2013; 14(4): 54-59.

Serological patterns and PCR results for acute Q fever and chronic Q fever

Acute Resolving Q fever						
Symptoms onset		< 7 d	14-21 d	3 m	6 m	12 m
PCR	Serum	Positive	Negative	Negative	Negative	Negative
EIA	IgM - phase 2	± Positive	Positive	Equivocal*	Negative	Negative
	IgG - phase 2	Negative	Positive	Positive	Positive	Positive**
CFT	CFT - phase 2	< 8	64	512	256	64
	CFT - phase 1	<8	<8	16	32	16
IFA	IgM - phase 2	10-80	320	640	160	<10
	IgG - phase 2	<10	320	640	1280	640
	IgA - phase 2***	<10	<10	<10	40	20
	IgM - phase 1	<10	80	320	160	<10
	IgG - phase 1	<10	<10	320	160	160
	IgA - phase 1***	<10	<10	<10	<10	<10

* May persist for months to years and not clinically significant

** May become undetectable over a long period of time

***Not always performed - clinical relevance uncertain

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Serology Compatible with evolving Chronic Q fever						
Onset		< 7 d	14 - 21 d	3m	6m	12m
PCR	Serum/tissue	Positive	± Positive*	± Positive*		
CFT	CFT - phase 2	< 8	64	512	1028	512
	CFT - phase 1	<8	<8	512	1028	1028
IFA	IgM - phase 2	10-80	320	640	160	<10
	IgG - phase 2**	<10	320	1024	2560	10240
	IgA - phase 2	<10	<10	<10	160	20
	IgM - phase 1	<10	160	640	160	<10
	IgG - phase 1**	<10	<10	1024	2560	20480
	IgA - phase 1***	<10	<10	<10	160	160

* Poor sensitivity in serum ~ 50%

** Persisting phase 1 and to a lesser extent phase 2 IgG (CFT and IFA)

*** Elevated phase 1 IgA > 160 may be present in chronic infection

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