

Q Fever

*Query Fever,
Coxiellosis,
Abattoir Fever*

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Importance

Q fever is a highly contagious zoonotic disease caused by the intracellular pathogen *Coxiella burnetii*. Although this infection was first described in the 1930s, it is still poorly understood. Many domesticated and wild animals including mammals, birds, reptiles and arthropods can carry *C. burnetii*. In most cases, the infection is asymptomatic, but abortions or stillbirths can occur in ruminants. In sheep, 5-50% of the flock may be affected. Both symptomatic and asymptomatic animals shed *C. burnetii* in large quantities at parturition. Shedding can also occur in feces, milk and urine. These organisms persist in the environment for long periods and can be spread long distances by the wind.

Human outbreaks can result from the inhalation of aerosolized organisms. More often, sporadic cases occur in people who are occupationally exposed. These cases tend to result from exposure to parturient ruminants; however, cats, dogs, rabbits and other species have also been implicated. Although Q fever is usually asymptomatic or mild in humans, a few people develop serious disease. Pneumonia or hepatitis may occur in acute cases, and chronic infections can result in endocarditis or a wide variety of other diseases.

Etiology

Q fever results from infection by *Coxiella burnetii*. This organism is an obligate intracellular pathogen and has been traditionally placed in the family Rickettsiaceae; however, recent phylogenetic studies have demonstrated that *C. burnetii* is more closely related to *Legionella*, *Francisella* and *Rickettsiella*. This organism is now classified in the family Coxiellaceae and order Legionellales in the gamma subdivision of Proteobacteria.

C. burnetii forms unusual spore-like structures that are highly resistant to environmental conditions. This organism also has two distinct antigenic phases, phase I and phase II. Phase I and II cells are morphologically identical, but differ in some biochemical characteristics including their lipopolysaccharide (LPS) composition. Organisms isolated from infected animals or humans express phase I antigens and are very infectious. Organisms expressing phase II antigens are less infectious and are recovered after the bacteria are passaged repeatedly in cell cultures or eggs. Experimentally infected animals first produce antibodies to phase II antigens and later produce antibodies to phase I antigens. A similar response occurs in humans, and is used to distinguish acute from chronic infections.

Geographic Distribution

Q fever has been found worldwide, except in New Zealand.

Transmission

C. burnetii can be transmitted by aerosols or direct contact; it is also spread by ingestion. Infections in animals can persist for several years and possibly lifelong. Organisms localize in the mammary glands, supramammary lymph nodes, uterus, placenta and fetus in animals; bacteria can be shed in milk, the placenta and reproductive discharges during subsequent pregnancies and lactations. *C. burnetii* can also be found in the feces and urine, and in the semen of bulls. Sexual transmission has been demonstrated in mice. Ticks may be important in transmission among wildlife, and can also spread infections to domesticated ruminants. In addition, *C. burnetii* has been found in lice, mites and parasitic flies.

Most human infections are associated with cattle, sheep and goats, and often occur when the animal gives birth. Cases have also been linked to other species including cats, dogs, rabbits and wild animals. One outbreak was associated with pigeon feces. Humans are usually infected via aerosols, but transmission may also occur by the ingestion of unpasteurized milk or other contaminated material. In addition, transmission has been documented in blood transfusions and can probably occur by sexual contact. Vertical (transplacental) transmission appears to be possible but rare, and tick-borne infections are thought to be uncommon or nonexistent. Persistent (dormant) infections can occur in humans; these organisms may be reactivated by immunosuppression or other factors.

C. burnetii is highly resistant to environmental conditions and is easily spread by aerosols; infectious airborne particles can travel up to 11 miles. Viable organisms can be found for up to 30 days in dried sputum, 120 days in dust, 49 days in dried urine from infected guinea pigs, and for at least 19 months in tick feces. At 4-6°C (39-43°F), organisms can survive for 42 months in milk and 12 to 16 months in wool.

Disinfection

C. burnetii is highly resistant to physical and chemical agents. Variable susceptibility has been reported for hypochlorite, formalin and phenolic disinfectants; 0.05% hypochlorite, 5% peroxide or a 1:100 solution of Lysol® may be effective. *C. burnetii* is also susceptible to glutaraldehyde, ethanol, gaseous formaldehyde, gamma irradiation or temperatures of 130°C (266°F) for 60 minutes. High temperature pasteurization destroys the organism.

Infections in Humans

Incubation Period

In humans, the incubation period for acute Q fever varies from 2 to 48 days; the typical incubation period is approximately 2 to 3 weeks. Chronic Q fever can occur from months to many years after infection.

Clinical Signs

Symptomatic infections can be acute or chronic. Many cases of acute Q fever are asymptomatic or very mild, and remain unnoticed. The symptoms of acute disease are flu-like and can include high fever, chills, a headache, fatigue, malaise, myalgia, sore throat and chest pain. The headache may be very severe. The illness is often self-limiting, and generally lasts from a week to more than three weeks. Some patients with Q fever develop atypical pneumonia. These patients usually have a nonproductive cough, with pneumonitis on X-ray. In severe cases, lobar consolidation and pneumonia may be seen; severe infections are particularly common in elderly or debilitated patients. Patients with atypical pneumonia can be ill for up to three months. Hepatitis can also occur in acute Q fever. Three forms of hepatitis may be seen. In one form, an infectious hepatitis-like form is accompanied by hepatomegaly. The clinical signs may include fever, malaise and right upper abdominal pain. Jaundice can occur, but it is uncommon. Other patients with hepatitis experience prolonged fever of unknown origin with granulomas on liver biopsy. Clinically asymptomatic hepatitis is also seen. The syndromes that accompany acute Q fever vary with the geographic region, with atypical pneumonia more common in some countries and hepatitis the predominant form in others.

Other, less common, signs can also occur in acute disease. Rashes have been reported in a few patients. Complications are uncommon but may include pericarditis and/or myocarditis, aseptic meningitis and/or

encephalitis, polyneuropathy, optic neuritis, hemolytic anemia, transient hypoplastic anemia, thyroiditis, gastroenteritis, pancreatitis, lymphadenopathy that mimics lymphoma, erythema nodosum, bone marrow necrosis, hemolytic-uremic syndrome, splenic rupture and others. A serious systemic infection was reported in an acute case of Q fever in a transplant patient.

Chronic Q fever is an uncommon condition that develops months or years after the acute syndrome. Endocarditis is the most commonly reported syndrome. It usually occurs in people who have pre-existing damage to the heart valves or are immunosuppressed. The symptoms are nonspecific and similar to subacute or acute bacterial endocarditis. Arterial embolisms occur in some patients. Other syndromes that have been reported in chronic Q fever include infections of aneurysms or vascular grafts, osteoarthritis, osteomyelitis, tenosynovitis, spondylodiscitis, paravertebral abscesses, psoas abscess and hepatitis. Rare cases of pericardial effusion, amyloidosis, pulmonary interstitial fibrosis, pseudotumor of the lung, lymphoma-like presentation, and mixed cryoglobulinemia have also been reported. *C. burnetii* has also been linked to chronic fatigue syndrome by some authors.

Approximately 98% of cases in pregnant women seem to be asymptomatic; however, *C. burnetii* has been linked to premature delivery, abortion, placentitis or lower birth weight in some women. Pregnancy complications have been reported with both acute and chronic Q fever. The consequences of congenital Q fever are unknown.

Communicability

Person to person spread is very rare. Generally, isolation is not considered necessary.

Diagnostic Tests

In humans, Q fever is usually diagnosed by serology or PCR. Serologic tests can be done as early as the second week of illness; they may include immunofluorescence, enzyme-linked immunosorbent assay (ELISA), microagglutination or complement fixation. Antibodies to the protein antigens found in phase II organisms predominate in acute Q fever; high levels of antibodies to the lipopolysaccharide of phase I organisms, combined with steady or falling titers to phase II, indicate chronic Q fever. Polymerase chain reaction (PCR) assays can detect the organism in a wide variety of samples including blood, cerebrospinal fluid, various tissue samples and milk.

Isolation of *C. burnetii* is dangerous to laboratory personnel and is rarely done. In specialized laboratories, organisms can be recovered from blood or tissue samples; bacteria are isolated in cell cultures, embryonated chicken eggs or laboratory animals including mice and guinea pigs.

C. burnetii antigens can be detected in tissues by immunoperoxidase staining, capture ELISA, an enzyme-linked immunosorbent fluorescence assay system, or

other assays. Antigen assays are particularly useful in patients with chronic Q fever.

Treatment

Antibiotics can shorten the course of acute illness and reduce the risk of complications; treatment is most effective if it is begun early. Treatment of chronic cases is more difficult and may require long-term antibiotic therapy. Surgical replacement is sometimes necessary for damaged valves and some other conditions.

Prevention

Most human cases are associated with exposure to ruminants, particularly when the animal has given birth. Whenever possible, the placenta from sheep, goats and cattle should be removed and destroyed immediately. Pens should be cleaned. In a recent outbreak, a pregnant sheep that gave birth at a market was responsible for nearly three hundred human cases. The authors recommend not displaying sheep in public places during the third trimester, and testing susceptible animals in petting zoos for *C. burnetii*. Because ingestion is a potential route of exposure, unpasteurized milk and milk products should be avoided. Manure from contaminated farms should not be spread in suburban areas and gardens. Facilities that study susceptible ruminants should use good laboratory practices, and the animals should be negative for *C. burnetii*. Biosafety level 3 is required for the manipulation of contaminated specimens and cultivation of this organism.

Effective vaccines may be available for people who are occupationally exposed. A licensed vaccine is available in Australia. In the United States, an investigational vaccine can be obtained from special laboratories such as the U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID).

People at high risk for chronic Q fever, such as those who are immunosuppressed, should consider staying away from susceptible ruminants, particularly parturient ruminants. It may be advisable to avoid all animals that have recently given birth, as cases have also resulted from exposure to cats and other species.

Morbidity and Mortality

The worldwide incidence of Q fever is unknown; however, this disease is common in some countries. The mean annual incidence in Germany ranges from 0.1-3.1 per million people, and varies with the region. In southern France, the prevalence of acute Q fever is 50 cases per 100,000 inhabitants. Symptomatic cases are rarely reported in the U.S., but this may be the result of underreporting and poor recognition of the disease. Until 1999, reporting of Q fever was optional in the U.S. Between 1948 and 1977, 1,164 infections were reported to the Centers for Disease Control and Prevention (CDC). Fewer than 30 cases were reported annually between 1978 and 1986. Twenty-one cases were reported in 2000, after reporting became mandatory. Twenty-six cases were

reported in 2001, 61 cases in 2002, 71 in 2003 and 70 in 2004.

In endemic areas, Q fever can occur sporadically or in outbreaks. Most cases are seen in people occupationally exposed to farm animals or their products: farmers, abattoir workers, researchers, laboratory personnel, dairy workers and woollsorters have an increased risk of infection. Outbreaks have also been reported when pregnant animals were displayed in public, or strong winds dispersed organisms from infected farms. In an outbreak in 1954, approximately 500 people were infected by exposure to a pregnant cow that aborted at a farmer's market. Recently, nearly 300 people were infected when a pregnant sheep gave birth at a market in Germany.

Acute Q fever is usually self-limiting, and most people recover spontaneously within a few weeks. Most acute infections are mild or subclinical. Up to 60% are thought to be asymptomatic, most of the remainder experience mild illness, while 2-5% develop severe disease and require hospitalization. The overall mortality rate is 1-2% in untreated cases and lower in those who are treated. The mortality rate in patients with atypical pneumonia is 0.5% to 1.5%.

Chronic Q fever usually occurs in people who are immunosuppressed or have predisposing conditions such as cardiac valvular disease or vascular grafts. The incidence of chronic disease is stated to be less than 1% by one source, and 5% by another. Estimates of the mortality rate in chronic Q fever vary widely, with one source suggesting a mortality rate of 1-11%, and another stating that as many as 65% may die of the disease.

Infections in Animals

Species Affected

C. burnetii can infect many species of domesticated animals and wildlife; in many species, the infection appears to be asymptomatic. Its reservoirs may be only partially known. Sheep, goats and cattle seem to be the most common domesticated animal reservoirs. Wild rodents may be important reservoirs in some areas, and cats are suspected in urban outbreaks. *C. burnetii* has also been isolated from dogs, rabbits, horses, pigs, camels, buffalo, deer, pigeons, swallows, parrots, crows, geese and other mammals and birds. Antibodies have been found in coyotes, raccoons, opossums, badgers, jackrabbits, black bears, musk ox and other species. There are also reports of *C. burnetii* in fish and snakes.

Incubation Period

The incubation period is variable; reproductive failure is usually the only symptom. Abortions generally occur late in pregnancy.

Clinical Signs

Many species are susceptible to infection, but most species seem to be infected asymptotically. Abortion,

stillbirth, retained placenta, endometritis, infertility and small or weak offspring can be seen in sheep, goats and cattle. Most abortions occur near term. Several abortions may be followed by uncomplicated recovery, particularly in small ruminants; in other cases, the disease may recur yearly. In dogs and cats, infections have been associated with stillbirths and weak offspring. Abortions and perinatal death occur after experimental (intraperitoneal) infection of pregnant mice.

With the exception of reproductive disease, animals are usually asymptomatic. Goats sometimes have a poor appetite and are depressed for 1 to 2 days before an abortion. Placental retention for 2 to 5 days and agalactia have also been reported. Clinical signs including fever, anorexia, mild coughing, rhinitis and increased respiratory rates occur in experimentally infected sheep but have not been reported in natural infections. Experimentally infected cats develop fever, lethargy and anorexia that last for several days. Experimentally infected mice may have pneumonia, hepatitis or splenomegaly, depending on the route of inoculation.

Communicability

Large numbers of organisms are found in the placenta, fetal fluids, aborted fetus, milk, urine and feces. Asymptomatic seropositive and seronegative animals, as well as symptomatic animals, may shed organisms.

Post Mortem Lesions [Click to view images](#)

Placentitis is a characteristic sign in ruminants. The placenta is typically leathery and thickened, and may contain large quantities of white-yellow, creamy exudate at the edges of the cotyledons and in the intercotyledonary areas. In some cases, the exudate may be reddish-brown and fluid. Severe vasculitis is uncommon, but thrombi and some degree of vascular inflammation may be noted. Fetal pneumonia has been seen in goats and cattle and may occur in sheep; however, the lesions in aborted fetuses are usually non-specific.

Diagnostic Tests

C. burnetii can be detected in vaginal discharges, the placenta, placental fluids and aborted fetuses (liver, lung or stomach contents), as well as milk, urine and feces. Organisms are not shed continuously in milk and colostrum. In the placenta, organisms can be identified in exudates or areas of inflammation with a modified Ziehl-Neelsen, Gimenez, Stamp, Giemsa or modified Koster stain; *C. burnetii* is an acid-fast, pleomorphic, small coccoid or filamentous organism. This organism is not usually detected by Gram stains. The presence of organisms, together with serological tests and clinical findings may be adequate for a diagnosis at the flock or herd level. Bacterial identity can be confirmed by immunohistochemistry or capture ELISA. PCR techniques are also available in some laboratories. Fresh, frozen or paraffin-embedded samples of blood, milk,

feces, vaginal exudates, placenta, fetal tissue and other tissues can be tested by PCR.

A number of serologic tests are available; the most commonly used assays include indirect immunofluorescence, ELISA and complement fixation. Serology may be more helpful in screening herds than in individual animals. Some animals do not seem to seroconvert, and others shed organisms before they develop antibodies. Animals can also remain seropositive for several years after an acute infection. Cross-reactions have been seen between some strains of *C. burnetii* and *Chlamydia* in ELISA and immunoblot assays.

C. burnetii can be isolated in cell cultures, embryonated chicken eggs or laboratory animals including mice and guinea pigs; however, isolation is dangerous to laboratory personnel and is rarely used for diagnosis.

Treatment

Little is known about the efficacy of antibiotic treatment in ruminants or other domestic animals. Prophylactic treatment is sometimes recommended to reduce the risk of abortion. Antibiotics may suppress rather than eliminate infections.

Prevention

In a *C. burnetii*-free flock, introduction of new stock should be minimized, and contact with wildlife should be prevented as much as possible. Good tick control should also be practiced. Prevention may be difficult, as this organism can also be introduced on fomites or in aerosols over long distances. In an infected flock, isolating infected pregnant animals and burning or burying the reproductive membranes and placenta can decrease transmission. The amount of *C. burnetii* in the environment can also be reduced by regular cleaning, particularly of areas where animals give birth. Cleaning can be followed by disinfection with 10% bleach. Antibiotics may be given prophylactically before animals give birth.

Vaccines are not available for domesticated ruminants in the United States but are used in some countries. Vaccines may prevent infections in calves, decrease shedding of organisms and improve fertility in infected animals. They do not eliminate shedding of the organism.

Morbidity and Mortality

Information on the prevalence of Q fever in the U.S. is limited. Surveys report infection rates that vary with the state, testing method and year the study was done. Across the U.S., reported seroprevalence rates in cattle range from 1% to 82%. The highest rates have generally been reported in California. In one area of California, 18% to 55% of sheep had antibodies to *C. burnetii*; the number of seropositive sheep varied seasonally and was highest soon after lambing. In other surveys, 82% of the individual

cows in some California dairies were seropositive, (with a herd infection rate of 98-100%) as well as 78% of the coyotes, 55% of the foxes, 53% of the brush rabbits and 22% of the deer in Northern California. Some studies suggest that the incidence of Q fever has been increasing, and this infection may currently be common throughout the U.S. A recent study found *C. burnetii* by PCR in 94% of bulk tank milk samples throughout the country, with little regional variation. Another survey reported antibodies in 92% of the dairy herds associated with U.S. veterinary schools.

C. burnetii infections may also be common in Canadian livestock. In Ontario, infections were found in 33-82% of cattle herds and 0-35% of sheep flocks. Close contact with sheep appears to increase the risk of infection in dogs.

Significant morbidity can be seen in some species. In sheep, abortions can affect 5-50% of the flock. In one California study, Q fever may have been responsible for 9% of all abortions in goats. Deaths are rare in natural infections.

Internet Resources

Centers for Disease Control and Prevention (CDC)
http://www.cdc.gov/ncidod/diseases/submenus/sub_q_fever.htm

Public Health Agency of Canada. Material Safety Data Sheets
<http://www.phac-aspc.gc.ca/msds-ftss/index.html>

Medical Microbiology
<http://www.gsbs.utmb.edu/microbook>

World Organization for Animal Health (OIE)
<http://www.oie.int>

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals
http://www.oie.int/eng/normes/mmanual/a_summry.htm

The Merck Manual
<http://www.merck.com/pubs/mmanual/>

The Merck Veterinary Manual
<http://www.merckvetmanual.com/mvm/index.jsp>

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*Link defunct as of 2007