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**Q Fever**


*Query Fever*  
*Coxiellosis*



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**Overview**

- Organism
- History
- Epidemiology
- Transmission
- Disease in Humans
- Disease in Animals
- Prevention and Control



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In today's presentation we will cover information regarding the organism that causes Q Fever and its epidemiology. We will also talk briefly about the history of the disease, how it is transmitted and clinical disease in humans and animals. Finally, we will address prevention and control measures for.

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**Organism**



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**The Organism**

- *Coxiella burnetii*
  - Rickettsial agent
  - Obligate intracellular parasite
  - Stable and resistant
  - Killed by pasteurization
  - Two antigenic phases
    - Phase 1: virulent
    - Phase 2: less pathogenic



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*Coxiella burnetii* is a obligate, intracellular, gram-negative, rickettsial agent. It replicates in host monocytes and macrophages. It has tremendous stability and can reach high concentrations in animal environments. Because it forms unusual spore-like structures, it is highly resistant to environmental conditions and many disinfectants. *Coxiella burnetii* can survive 7-10 days on wool at room temperature, 1 month on fresh meat in cold storage, 120 days in dust and more than 40 months in skim milk. The organism is killed by pasteurization. *Coxiella burnetii* exists in two antigenic phases. This is important in the diagnosis of Q fever. Phase I is pathogenic and found in infected animals or in nature. Phase II, is less pathogenic and is recovered only after multiple lab passages in eggs or cell cultures. Increased antibodies to phase II antigens, indicated acute cases while a rise in phase I reflects a chronic infection of Q fever.

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**History**



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### History

- 1935
  - 1st described in Queensland, Australia
  - Found in ticks in Montana
- Outbreaks
  - Among military troops
    - When present in areas with infected animals
  - Cities and towns
    - Downwind from farms
    - By roads traveled by animals




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Q “Query” fever was first reported in Brisbane, Queensland, Australia, in 1935, by Derrick, who described outbreaks of febrile illness in abattoir workers in Queensland. Burnet and his associate Freeman, successfully isolated the organism and investigated the epidemiology of the disease. Concurrently, a similar agent (initially called the “Nine Mile agent”), was isolated from ticks in Montana by Davis and Cox and was subsequently found to be the same organism as that found in Queensland. Soon after in 1938, the organism was named *Coxiella burnetii* in honor of Cox and Burnet, who had identified the organism as a new rickettsial agent. In 1944, there were outbreaks among British and American troops stationed in the Mediterranean (Italy), during World War II and during the Persian Gulf. Outbreaks have also been reported among persons residing in cities and towns downwind from sites where infected animals are kept.

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### Transmission



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### Transmission

- Aerosol
  - Parturient fluids
    - 10<sup>9</sup> bacteria per gram of placenta
  - Urine, feces, milk
  - Wind-borne
- Direct contact
- Fomites
- Ingestion
- Arthropods (ticks)



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Q fever can be transmitted via a variety of routes. Domestic ruminants represent the most frequent source of human *C. burnetii* infection. However, pets, (i.e., cats, dogs and rabbits) have also been involved as sources of urban outbreaks. Aerosolization is the primary mode of transmission in humans. Organisms can be found in airborne droplets or dust contaminated by placental tissues, birth fluids, or excreta of infected animals. Shedding of *C. burnetii* into the environment occurs mainly during parturition; over 10<sup>9</sup> bacteria per gram of placenta are released at the time of delivery. Aerosol or direct transmission can occur when infected animals are processed as meat, during necropsies or assisting deliveries. Due to the persistence of the organism in the environment, dried infective material can contaminate dust or soil, which can be carried considerable distances by wind and has been documented to travel downwind up to ½ mile or more. This has resulted in cases of patients without any evident contact with animals. Fomites (i.e., newborn animals, wool, bedding, clothing) can also be contaminated by such materials and serve as a source of the transmission. Organisms shed in urine and feces of infected animals can also serve as a source of water, dust, soil or fomite contamination. Water may be contaminated and act as a vehicle for dissemination. Shedding in the milk occurs due to infected mammary glands, but pasteurization kills this organism. *C. burnetii* has been naturally and experimentally isolated from a variety of arthropods, (mainly ticks but also cockroaches, beetles, flies, fleas, lice, mites). Over 40 tick species are naturally infected with *C. burnetii*, and transovarial (mother to offspring) and transstadial (between developmental life stages) transmission has been documented. Feces of infected arthropods can serve as a source of *C. burnetii* infection and can remain infective for at least 19 months. Animals typically acquire Q fever through exposure to other infected animals, either through direct contact with contaminated material or aerosol exposure.

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### Transmission

- Person-to-person (rare)
  - Transplacental (congenital)
  - Blood transfusions
  - Bone marrow transplants
  - Intradermal inoculation
  - Possibly sexually transmitted

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Person-to-person transmission is extremely rare, with the exception of transplacental transmission resulting in congenital infections. Transmission from blood transfusions, bone marrow transplants, intradermal inoculations have been reported. Transmission via sexual intercourse has been hypothesized. Sexual transmission of *Coxiella burnetii* has been documented in mice and guinea pigs and hypothesized for a rare number of human cases.

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### Epidemiology

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### Epidemiology

- Worldwide
  - Except New Zealand
- Reservoirs
  - Domestic animals
    - Sheep, cattle, goats
    - Dogs, cats
  - Birds
  - Reptiles
  - Wildlife



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Q fever is a zoonosis with worldwide distribution. It has been reported on all continents, except New Zealand and is endemic in areas where reservoir animals are found. The animal reservoir is large and include many wild and domestic mammals, birds and arthropods. However, the primary reservoirs are considered to be cattle, sheep, goats and ticks. Wildlife species reported as reservoirs include snowshoe hares, moose and white-tailed deer in Nova Scotia, wild Dall sheep in Alaska, and black bears in Idaho and California.

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### Epidemiology

- Occupational and environmental hazards
  - Farmers, producers
  - Veterinarians and technicians
  - Meat processors, abattoir
  - Laboratory workers

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Q fever is primarily an occupational hazard in persons in contact with domestic animals, such as cattle, sheep and goats. Persons at risk include farmers, veterinarians, abattoir workers, those in contact with dairy products, and laboratory personnel performing culture and diagnostics. The has been an increase in reports of sporadic cases in people living in urban areas after occasional contact with farm animals or after contact with infected pets, such as dogs and cats.

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### Q Fever in the U.S.: 2002

Q FEVER, Reported cases — United States and U.S. territories, 2002

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There were a total of 61 human cases of Q fever reported to the CDC in 2002; the above map shows the geographic distribution. [N=Report of disease is not required in this jurisdiction]. Although Q fever has been a nationally notifiable disease in the U.S. since 1999, epidemiologic data is limited. Much of this is due to the self-limiting nature of most cases of the disease, thereby making the disease underreported. Between 1948-1977, a total of 1,168 cases of Q fever were reported to the CDC (58.4 cases per year). Most of these cases (67%) were reported from California. Map from the Summary of Notifiable Diseases 2002, CDC website.

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**Disease in Humans**



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**Human Disease**

- Incubation: 2-5 weeks
- One organism may cause disease
- Humans are dead-end hosts
  - Usually show clinical signs of illness
- Disease
  - Asymptomatic (50%)
  - Acute
  - Chronic

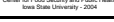


In humans, the incubation period varies from 2-40 days (mean around 20 days). As few as one organism is capable of causing disease. Humans are considered to be dead end hosts and are the only species known to develop illness regularly as a result of infection. Most cases of Q fever are asymptomatic. Only about 50% of all people infected with *C. burnetii* show signs of clinical illness. The two clinical forms of the disease are acute (less than 6 months duration) and chronic (greater than 6 months duration).

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**Acute Infection**

- Flu-like, self limiting
- Atypical pneumonia (30-50%)
  - Non-productive cough, chest pain
  - Acute respiratory distress possible
- Hepatitis
- Skin rash (10%)
- Other signs (< 1%)
  - Myocarditis, pericarditis, meningoencephalitis
- Death: 1-2%




Symptoms of acute disease can vary in severity and duration. Usually it manifests as a self-limited febrile or flu-like illness. Signs include fever (up to 104-105°F), chills, “sweats”, retrobulbar headache, fatigue, anorexia, malaise, myalgia, and chest pain. Illness typically lasts from one to three weeks. Thirty to 50% of patients with symptomatic illness will develop pneumonia. In more severe cases, a nonproductive cough with pneumonitis may develop. Radiographs of patients with pneumonia resemble those of patients with viral pneumonia etiologies. Multiple rounded opacities of both lungs on x-ray may be noted. Pleural effusion may also be seen. Additionally, many clinically ill patients will have abnormal liver enzymes and some will develop hepatitis. Jaundice is rare. Exanthema (rash) occurs in about 10% of cases. Rarely meningoencephalitis or pericarditis may occur with acute infection. Only 2% of acute infections require hospitalization and a similar percentage result in death.

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**Chronic Disease**

- 1-5% of those infected
  - Prior heart disease, pregnant women, immunocompromised
- Endocarditis
- Other
  - Osteomyelitis
  - Granulomatous hepatitis
  - Cirrhosis
- 50% relapse rate after antibiotic therapy





Chronic Q fever (infection greater than six months in duration) occurs in 1-5% of those infected and is relatively uncommon. It typically develops in persons with pre-existing cardiac valvular disease. Immunocompromised persons and pregnant women are also at great risk for the chronic form. Endocarditis is the major clinical presentation and accounts for 60-70% of all chronic Q fever cases. Infection can also affect the liver causing granulomatous hepatitis or cirrhosis. Kupffer cells are considered to be target cells for *C. burnetii*. Involvement in bone and arteries has also been reported. Patients who have had acute Q fever may also develop the chronic form as soon as 1 year or as long as 20 years after initial infection.

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**Risk to Pregnant Women**

- Most asymptomatic
- Transplacental transmission
- Reported complications
  - In-utero death
  - Premature birth
  - Low birth weight
  - Placentitis
  - Thrombocytopenia

Pregnant women who become infected by *C. burnetii* are typically asymptomatic. However, the organism can be transplacentally transmitted. Depending on the timing of infection, resulting abortions or neonatal deaths, premature births, low birth weights or placentitis may occur. The greatest risk is during the first trimester. Women contracting Q fever also are at great risk of developing chronic Q fever infection. Pregnant women with Q fever can also pose a degree of risk to medical staff in contact with infected pregnant women.

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**Prognosis**

- Overall case-fatality rate <1 - 2.4%
- 50% cases self-limiting
- Only 2% develop severe disease
- Active chronic disease
  - Usually fatal if left untreated
  - Fatality for endocarditis: 35-55%
  - 50-60% need valve replacement

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Q fever is usually a self-limiting illness and most cases resolve within 2 days to 2 weeks, but may take up to 6 weeks. Approximately 50-60% of cases are thought to be asymptomatic. The overall case-fatality rate for Q fever ranges from <1-2.4%. Complications from the acute form of disease are rare. Only 2% of persons infected with *Coxiella burnetii* develop severe disease and require hospitalization. In general, for untreated cases, the mortality rate is 1% and lower if treated. Active chronic disease is usually fatal if untreated and for chronic disease patients with endocarditis, the fatality rate can range from 45-65%. Additionally, 50-60% of these patients will need valve replacement surgery.

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**Diagnosis**

Serology (rise in titer)

- IFA, CF, ELISA, microagglutination
- DNA detection methods
  - PCR
- Isolation of organism
  - Risk to laboratory personnel
  - Rarely done

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In humans, Q fever is usually diagnosed by serology (rise in antibody titer levels), which can be done as early as the second week of illness. There are a variety of serological tests for Q fever including IFA (immunofluorescence assay), CF (complement fixation), ELISA (enzyme-linked immunosorbant assay), and microagglutination. The indirect IFA is the most dependable and widely used method. *C. burnetii* may also be identified in infected tissue by IHC(immunohistochemistry) and DNA detection methods (PCR-polymerase chain reaction). Clinical signs and patient history can also aid in diagnosis. Isolation of the organism is rarely done due to the risk *C. burnetii* poses for laboratory personnel. Q fever is considered a Biosafety Level 3 agent.

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**Treatment**

- Treatment
  - Doxycycline
  - Chronic disease - long course
    - 2-3 years of medication
- Immunity
  - Long lasting (possibly lifelong)

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The antibiotic treatment of choice is doxycycline. Antibiotic treatment is most effective when initiated within the first three days of illness. For chronic disease, treatment may be necessary for 2-3 years. Doxycycline and quinolones are contraindicated in pregnant women but long term therapy with co-trimoxazole (trimethoprim/sulfamethoxazole combination) has prevented fetal death in some cases.

Persons recovering from Q fever are thought to develop long lasting (possibly lifelong) immunity. For chronic disease, treatment may be necessary for 2-3 years.

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**Large Animal Case**

- Male dairy farmer
  - Age 46
  - Sudden onset
    - Fever, chills, cough
    - Weight loss
- Initially thought it was influenza
- Symptoms persisted for 2 weeks
- Presented to emergency room
  - Again influenza was the diagnosis




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A 46 year old male dairy farmer from Georgia reported a sudden onset of fever, chills, cough, and weight loss. A physician initially diagnosed influenza in the patient. Two weeks later the symptoms still persisted and the patient presented to the emergency room where he was again diagnosed with influenza.

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**Large Animal Case**

- Referral to infectious disease specialist
  - Tested positive for Q fever
  - Antibiotics for 5 days
  - Resolved in 2 weeks
- Epidemiology
  - No recent calvings on his farm
  - Two beef cattle herds across the road
    - 2 out of 14 tested positive for Q fever



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The emergency room doctor then referred the patient to an infectious disease specialist. The infectious disease specialist tested the patient for Q fever and he was positive. The patient took a five day course of gatifloxacin (a fluoroquinolone), and symptoms resolved within 2 weeks. Although the patient owned several dairy cows, no recent calvings had occurred at his farm. Two beef cattle herds (approximately 35 animals per herd) were pastured across the road from the patient's farm. Fourteen animals from the neighboring beef herds were then tested; two animals were found to be positive for Q fever. *MMWR* October 18, 2002/51(41);924-927.

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**Case Points**

- Naturally occurring cases occur
- Recognize the signs and seek medical attention
- Isolated incident
- What if it was more serious or a cluster of producers were ill?


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This was a naturally occurring case of Q fever in a dairy producer, but what if it was more serious than that? We need to recognize the signs of illness both in our animals and ourselves, especially when they are persistent.

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**Small Animal Case**

- 1985, Nova Scotia, Canada
  - 33 cases of Q fever
    - 25 were exposed to cat
    - 17 developed cough
    - 14 developed pneumonia
  - Most common symptoms
    - Fever, sweats, chills, fatigue, myalgia, headache
  - Cat tested positive for *C. burnetii*
    - 1:152 to phase I antigen
    - 1:1024 to phase II antigen




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In 1985, a cluster of Q fever cases occurred in Nova Scotia, Canada. Most of the affected persons had symptoms of fever, sweats, chills, fatigue, myalgia and headache. Seventeen of the patients developed a cough and 14 had pneumonia. Epidemiological investigation revealed that 25 patients were exposed to a cat that had given birth to stillborn kittens 2 weeks prior. The cat had also had vaginal bleeding three weeks prior to this delivery. The majority of human cases lived or worked in 4 buildings near the apartment where the cat lived. The cat visited the other buildings frequently. Exposure to cattle, sheep, and goats was uncommon for the human cases. The cat was tested positive for antibodies to *C. burnetii*. An antibody titer of 1:152 to phase I antigens and 1:1024 to phase II antigens was reported. All human patients recovered uneventfully. Most cases of Q fever recently reported in Nova Scotia have been associated with exposure to parturient cats.

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**Animals and Q Fever**



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**Animal Disease**

- Sheep, cattle, goats
  - Usually asymptomatic
  - Reproductive failure
    - Abortions, stillbirths
    - Retained placenta
    - Infertility
    - Weak newborns
    - Low birth weights
    - Mastitis in dairy cattle
  - Carrier state

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Domestic livestock, sheep, cattle, and goats, are the most common reservoirs of Q fever. The incubation period for animals is variable. Affected animals are usually asymptomatic; when clinical disease occurs, reproductive failure is usually the only symptom seen. This can be manifested as abortions, stillbirths, retained placentas, infertility, weak newborns and mastitis in dairy cattle. Anorexia and abortions have been reported more frequently in sheep and goats while infertility, sporadic abortion and low birth weights are seen in cattle. Lambings subsequent to *Coxiella* abortions have been found to be carried to term. However ewes can remain chronically infective and continue to shed organisms. Organisms may be shed in milk, and feces for several days after parturition.

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**Animal Disease**

- Other animal species
  - Dogs, cats, horses, pigs, camels, buffalo, pigeons, other fowl
  - Asymptomatic
  - Reproductive failure
- Laboratory Animals
  - Rats, rabbits, guinea pigs, hamsters
  - Varies from asymptomatic to fever, granulomas, or death

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Dogs, cats, rabbits, horses, pigs, camels, buffalo, rodents, pigeons, geese and other fowl may also carry *C. burnetii*. Antibodies to the organism have been found in a variety of other animal species as well. Dogs and cats may be infected by a tick bite, consumption of placentas or milk from infected ruminants or by the aerosol route. Q fever infection in parturient dogs may lead to early death of pups. Human Q fever cases were described in Nova Scotia after contact with parturient cats. Twelve patients developed a febrile illness two weeks after playing poker in a room where a cat had given birth to kittens. All the infected persons had handled the cat or its litter, and specific antibodies were demonstrated in the cat serum.

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### Diagnosis and Treatment

- Diagnosis
  - Identification of organism
    - Histology, IHC
  - Serologic tests: IFA, ELISA, CF
  - PCR
  - Isolation of organism
    - Hazardous - Biosafety level 3
- Treatment
  - Tetracycline prior to parturition

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Diagnostic testing for animals can be done through a variety of methods. *C. burnetii* can be detected in vaginal discharges, the placenta or its fluids, and aborted fetuses, as well as milk, urine and feces. Organism identification can be accomplished with Modified Ziehl-Neelson or Gimenez stains, but are usually not detected by Gram stain. Immunohistochemistry (IHC) can also confirm bacterial identity. Polymerase chain reaction (PCR) techniques are also available in some laboratories. A number of serological tests are also available (i.e., immunofluorescence (IFA), enzyme-linked immunosorbent assays (ELISA) and complement fixation (CF). The complement fixation test is done most commonly. Although isolation of the organism can be accomplished in cell cultures, embryonated chicken eggs or laboratory animals, it is dangerous to laboratory personnel, and must be completed in a biosafety-Level 3 laboratory. It is therefore rarely used. Little is known about the effectiveness of treating animals with antibiotics. Tetracycline has been given in water in the weeks preceding parturition in enzootic herds. This may help reduce shedding in birthing materials. Antimicrobial therapy may not eradicate the “carrier” state of *C. burnetii* infection, but may suppress the number of abortion.

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### Morbidity and Mortality

- Prevalence unknown
  - Endemic areas
    - 18-55% of sheep with antibodies
    - 82% of dairy cattle
- Morbidity in sheep: 5-50%

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Information on the prevalence of Q fever in animal species is limited. In endemic areas, (i.e. areas of California), it was found that 18-55% of sheep had antibodies to *C. burnetii* and up to 82% of cows in some dairies. Significant morbidity can be seen in some species. In sheep, abortion can affect 5-50% of the flock.

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### Post Mortem Lesions

- Placentitis
- Placenta
  - Leathery and thickened
  - Purulent exudate
    - Edges of cotyledons
    - Intercotyledonary areas
- Aborted fetus
  - Non-specific

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Placentitis is the most characteristic lesion in ruminants. The placenta is typically leathery and thickened. It may contain large amounts of creamy, white-yellow exudate at the edges of cotyledons and in the intercotyledonary area. Lesions in aborted fetuses are usually non-specific.

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### Prevention and Control



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### Prevention and Control

- Pasteurization
- Vaccination
  - Human and animal
  - Not available in U.S.
- Eradication not practical
  - Too many reservoirs
  - Constant exposure
  - Stability of agent in environment



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Pasteurization of milk from cows, sheep, and goats is important in stopping the spread of Q fever by contaminated milk sources. Vaccines have been developed for both animals and humans, but are not commercially available in the United States. Persons previously exposed to *C. burnetii* should not receive the vaccine because severe reactions are possible. Practical control is difficult because of environmental stability and infectivity for wild animals, arthropods, and humans. For livestock, control includes good animal husbandry, vaccination, screening, and culling.

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### Prevention and Control

- Education
  - Sources of infection
- Good husbandry
  - Disposal of birth products (incinerate)
    - Lamb indoors in separate facilities
  - Disinfection
    - 0.05% chlorine
    - 1:100 Lysol
- Isolate new animals

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Education is an important part of prevention and control of Q fever. It should focus on potential sources of infection and ways to reduce environmental contamination from infected placental membranes and aborted materials. When possible, lambing should take place indoors and in separate facilities for parturition. Placentas, aborted materials and fetal tissues should be disposed of appropriately. Birth products should be incinerated and lambing areas treated with Lysol, bleach, or hydrogen peroxide.

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### Q Fever as a Biological Weapon

- Accessibility
- Low infectious dose
- Stable in the environment
- Aerosol transmission
- WHO estimate
  - 5 kg agent released on 5 million persons
    - 125,000 ill - 150 deaths
    - Could travel downwind for over 20 km

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Because of its highly infectious nature, stability in the environment and aerosol route of transmission, *C. burnetii* can be considered a potential agent of bioterrorism. Although overall mortality associated with the disease is low, it could be considered a debilitating agent. The World Health Organization (WHO) estimated that if Q fever was aerosolized in a city of approximately 5 million people there would be 125,000 ill and 150 deaths. They estimated that the agent could travel downwind for greater than 20 km.

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