



radiography.²² Over 60% of patients may have high liver enzyme activities, and granulomatous hepatitis has been reported in as many as 40%.²² In Australia, a chronic fatigue-like illness has been reported in as many as 10% of patients immediately following acute Q fever.²⁶ Meningoencephalitis and myocarditis are potential rare complications of acute Q fever.²² Most patients with acute Q fever recover without medical intervention; however, treatment with doxycycline may shorten the clinical course of illness.²⁷

Approximately 1 to 2% of patients acutely infected with *C burnetii* go on to develop chronic infection, although sequelae may not be manifested for decades after acute infection.^{10,28} Because some acutely infected individuals may be asymptomatic or have only mild illness, development of chronic disease may be the first time Q fever is recognized. One of the most common sequelae of chronic Q fever is endocarditis.^{22,29} Chronic granulomatous hepatitis, osteoarticular infection, pericarditis, and vascular complications have also been reported.^{22,30,31} Persons with underlying heart valve abnormalities, prosthetic valves, or immune compromise are at increased risk for Q fever endocarditis. Chronic Q fever endocarditis can be life threatening, and patients may require excision and replacement of affected valves. Treatment consists of long-term administration of doxycycline and hydroxychloroquine.³²

Q Fever in Animals

Animals typically acquire Q fever through exposure to other infected animals, either through direct contact with contaminated body fluids or aerosol exposure to infectious materials. Sheep, cattle, and goats are considered the most common livestock reservoirs for *C burnetii* on the basis of epidemiologic and laboratory evidence. Cats and dogs are also susceptible to infection and may transmit *C burnetii* to humans.^{6,33,34} Antibodies against *C burnetii* have been detected among many wildlife species, including snowshoe hares, moose, and white-tailed deer in Nova Scotia; wild Dall sheep in Alaska; and black bears in Idaho and California.³⁵⁻³⁷ Q fever among humans has been reported after contact with wild rabbits.³⁸ Rodents are susceptible to *C burnetii* infection and may play a role in the natural maintenance of Q fever among wildlife.³⁹

Coxiella burnetii has been isolated from a wide variety of tick species and may be transmitted among animals via tick bite. The original prototype strain of *C burnetii* was isolated from a *Dermacentor andersoni* tick collected from Nine Mile Creek in Montana.² Ticks are not thought to play an important role in transmission of *C burnetii* to humans, but may be essential in natural maintenance cycles.

In ruminants, Q fever occasionally results in abortion, stillbirths, and complicated deliveries. Experimentally infected sheep are pyretic, anorectic, and tachypneic, followed by delivery of stillborn or unviable lambs⁴⁰; aborted fetuses may appear thin but

Q Fever in the United States

It is difficult to describe the epidemiologic characteristics of Q fever in the United States, because it is not a notifiable disease in many states. Furthermore, the disease is generally underrecognized because of the nonspecific nature of clinical signs and the need for laboratory confirmation. Limited national surveillance data for Q fever in humans in the United States are available for the years 1948 through 1986; during this time, less than 30 states required reporting of Q fever. Between 1948 and 1977, a total of 1,168 cases of Q fever were reported to the Centers for Disease Control and Prevention (mean, 58.4 cases/yr; Fig 1).^{48,a} Most of these cases (67%) were reported from California, where the disease is endemic and there has traditionally been heightened interest in surveillance. Between 1978 and 1986, a total of 228 cases were reported nationally (mean, 28.5 cases/yr).⁴⁹

Although there have been no national seroprevalence studies in the United States, geographically limited investigations targeting specific populations and

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