



Short communication

Coxiella burnetii (Q fever) in *Rattus norvegicus* and *Rattus rattus* at livestock farms and urban locations in the Netherlands; could *Rattus* spp. represent reservoirs for (re)introduction?

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ABSTRACT

The Q fever outbreak in the Netherlands in 2007–2010 prompted government interventions to reduce the human incidence by reduction of Q fever shedding at dairy goat farms. Mandatory hygiene measures were taken, including the control of animal reservoirs. It has been postulated that brown rats, through their commensal nature, form an important factor in the persistent dissemination of endemic circulating *Coxiella burnetii* in nature to domestic animals, livestock and humans. Here, the occurrence of *C. burnetii* in rats captured at different types of location during the Q fever outbreak in the Netherlands, viz. urban areas, nature areas and various types of farm has been determined. This is a first step towards the elucidation of the reservoir status of rats in veterinary and human Q fever epidemiology. *C. burnetii* DNA was detected in the spleen of 4.9% of the brown rats (*Rattus norvegicus*) and 3.0% of the black rats (*Rattus rattus*). Evidence for *C. burnetii* infection was also found in liver, kidney, lung and intestinal tissue but not in heart, brain and pancreas. *C. burnetii* IgGs were detected in 15.8% of the brown rats. Positive rats were collected at goat, pig, cattle and poultry farms, and urban locations; including locations outside the designated 5 km “increased-risk” zones around bulk milk positive goat farms. The percentage of rat-positive locations was the highest for goat farms (50%) and cattle farms (14.3%). The presence of actively infected rats outside the lambing season and at multiple environmental settings including urban locations might suggest that rats are not merely a spill-over host due to infection by a contaminated environment but might represent true reservoirs, capable of independent maintenance of *C. burnetii* infection cycles and thereby contributing to spread and transmission of the pathogen. If frequent (re)introduction of *C. burnetii* to small ruminant farms can be caused by rats as maintenance reservoirs, mandatory wildlife control and lifelong vaccination of herds will be necessary.

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1. Introduction

Q fever is a zoonotic disease caused by the bacterium *Coxiella burnetii* and is considered a sporadic and

(internationally) mainly an occupational zoonosis (Raoult et al., 2005). Goats, sheep and cattle are the primary animal reservoirs for zoonotic transmission but many other domesticated and wild animals including mammals, birds, reptiles and arthropods can be carriers of the pathogen (Rodolakis, 2006). Urine, feces and birth material of infected animals are sources for *C. burnetii* contamination of the environment. The shedding of the bacteria is the highest during birth or abortion (Berri et al.,

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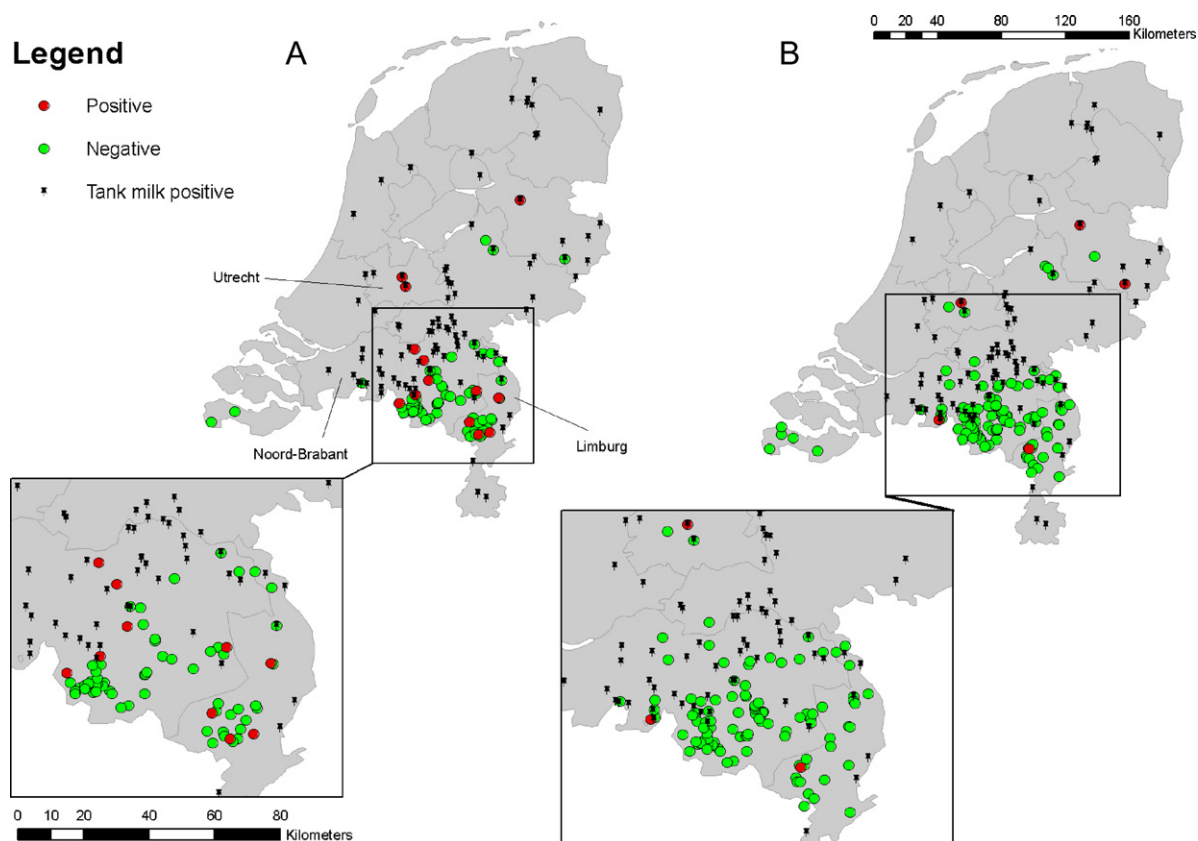


Fig. 1. Geographic distribution of sampled locations and test results for *C. burnetii* circulation in the Netherlands. (A) Results ELISA on serum of collected rats. (B) Results PCR on spleen tissue of collected rats. Green symbols indicate the absence of respectively *C. burnetii* IgG or DNA, Red symbols indicate their presence. The location of bulk milk positive goat and sheep farms, as identified during the Q fever outbreak in the Netherlands, are indicated with black pins. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

2007). The bacterium can survive for years in the environment and travel long distances in the form of aerosols (Marrie, 1990). In a community outbreak in the United Kingdom cases were identified up to 18 km from the source (Hawker et al., 1998). Experimental studies have shown that contaminated aerosols can be found within 2 km of a Q fever affected farm. In addition, the level of *C. burnetii* DNA found in aerosols obtained from within (goat) farm stables is higher than in aerosols obtained at distances of 0.5, 1.0 and 2.0 km from the affected farm (de Bruin, pers. comm.). Inhalation of aerosolized bacteria and direct contact with contaminated products (e.g., straw, animals) are considered the primary routes of infection for humans besides the gastro-intestinal route (raw dairy products) and human-to-human transmission (blood-transfusion, sexual transmission) (Angelakis and Raoult, 2010; Anonymous, 1977; Cerf and Condron, 2006; Milazzo et al., 2001).

Cats, dogs, rabbits, foxes and rodents are thought to constitute a reservoir for maintenance of infection in the domestic cycle of *C. burnetii* (Aitken, 1989). The role of wild vertebrates in the sylvatic cycle is established with a presumed role of ticks in the transmission (Babudieri, 1959). It has been postulated that brown rats (*Rattus norvegicus*), through their commensal nature, form an important factor

in the persistent dissemination of endemically circulating *C. burnetii* in nature to domestic animals and humans. The often implicated role of cats in human Q fever epidemiology could be related to a combination of high prevalence of *C. burnetii* in rats and cats' predatory behavior towards these natural reservoirs (Webster et al., 1995). Recent findings in Germany indicate waste control work, regular sightings of wild rodents and contact with pet rats (which are often bred from wild rats) as risk factors for *C. burnetii* infection in humans, even suggesting wild rats as a direct source for human infection (Brockmann et al., 2010). A small number of human Q fever patients in the Netherlands reported sighting of rodents, rabbits or reptiles. Univariate analysis of these sightings indicated an association with the Q fever occurrence in 2007 (Karagiannis et al., 2009).

The endemic and sporadic nature of this zoonosis is reflected in the annual average number of 17 human Q fever cases in the Netherlands until the end of 2006. However, since the beginning of 2007 there has been a sharp increase in the human incidence with 168, 1000 and 2356 reported cases in 2007, 2008 and 2009 respectively (Hoek et al., 2010). Epidemiological studies indicated dairy goat production, and to a lesser extent sheep farms, as main sources for the Q fever outbreak (Hoek et al., 2010; Karagiannis et al., 2009; Schimmer et al., 2010) and prompted govern-

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