

# Q fever and seroprevalence of *Coxiella burnetii* in domestic ruminants

Andreana Pexara\*, Nikolaos Solomakos and Alexander Govaris

Laboratory of Hygiene of Foods of Animal Origin, Faculty of Veterinary Medicine, University of Thessaly, Greece

\*Corresponding author at: Laboratory of Hygiene of Foods of Animal Origin, Faculty of Veterinary Medicine, University of Thessaly, Greece.  
e-mail: apexara@vet.uth.gr.

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## Keywords

*Coxiella burnetii*,  
Domestic ruminants,  
Q fever,  
Seroprevalence.

## Summary

Q fever is a zoonosis caused by *Coxiella burnetii*, an obligate intracellular gram-negative bacterium. Infection by *C. burnetii* has been demonstrated in many animal species, but ruminants are the major reservoirs and the main sources of human infection. In ruminants, *C. burnetii* infection is often asymptomatic, but it has been also associated with infertility and abortions. In humans, Q fever was considered predominately an occupational hazard due to close contact with infected ruminants by means of their contaminated birth products, urine, feces or milk. Q fever has recently gained renewed attention after the large outbreak in the Netherlands in 2007-2009, indicating its importance as an emerging public health threat. The seroprevalence of *C. burnetii* in ruminants is commonly detected by various tests but no official standard technique is still available. According to surveys conducted in many countries of the five continents, a relatively high proportion of farm ruminants are found seropositive to *C. burnetii*. The only country with an apparent zero prevalence is New Zealand. The seroprevalence in goats and sheep is usually higher than cattle.

## Febbre Q e sieroprevalenza di *Coxiella burnetii* nei ruminanti domestici

### Parole chiave

*Coxiella burnetii*,  
Ruminanti domestici,  
Febbre Q,  
Sieroprevalenza.

### Riassunto

La febbre Q è una zoonosi causata dal batterio gram-negativo *Coxiella burnetii*, un patogeno intracellulare obbligato. I principali serbatoi e fonte di infezione umana sono i ruminanti ma l'infezione da *C. burnetii* è stata dimostrata in molte specie animali. Nei ruminanti è spesso asintomatica ma è stata anche associata a infertilità e aborti; nell'uomo, è stata considerata prevalentemente un rischio professionale, determinata dal contatto con prodotti infetti come placenta, urina, feci o latte. La febbre Q è tornata ad essere un'emergenza per la salute pubblica dopo la vasta epidemia avvenuta nei Paesi Bassi tra il 2007 e il 2009. Nonostante la sieroprevalenza di *C. burnetii* nei ruminanti sia comunemente rilevata dai vari test diagnostici utilizzati nei laboratori, non è ancora disponibile un metodo ufficiale per questo patogeno. Le numerose indagini condotte in vari paesi mostrano percentuali elevate di infezione nei ruminanti domestici specie nelle pecore e nella capre. L'unico paese con una prevalenza apparente zero è la Nuova Zelanda.

## Introduction

Q fever is a zoonosis with a world-wide distribution (except New Zealand) caused by *Coxiella burnetii*, an obligatory intracellular bacterium.

Q or 'query' fever was first observed in slaughterhouse workers in Brisbane, Queensland, Australia in 1933 and was initially described by Derrick as a self-limiting febrile illness of unknown etiology (Derrick 1937). At that time, the etiological agent was considered

to be a virus and all trials to isolate the pathogen by inoculating guinea pigs with blood or urine of infected patients were unsuccessful (Derrick 1937). Burnet and Freeman (Burnet and Freeman 1937) isolated an intracellular bacterium from guinea pigs that had been previously injected with blood or urine from the infected slaughterhouse workers and named it *Rickettsia burnetii*. In the same period, a laboratory-acquired fever infection occurred in the Rocky Mountain Laboratory in Hamilton, Montana,

USA (Davis and Cox 1938). As *Dermacentor andersoni* was collected near the infected guinea pigs with a febrile illness and enlarged spleens in Nine Mile creek, Montana, it was concluded that the fever was acquired by means of possible vectors (Cox 1938). The causative agent with filterable properties was characterized as the 'Nine Mile agent'. The organism was observed intravacuolarly in infected tissue cultures and could be also transmitted to humans (Cox 1938). The American and Australian research groups then demonstrated that the Australian Q fever and the Nine Mile agents were in fact isolates of the same microorganism which was classified as *Rickettsia burnetii* (Maurin and Raoult 1999). In 1948, Philip re-classified *R. burnetii* according to cultural and biochemical characteristics. To honor both Cox and Burnet, the Q fever pioneers, they re-named it as *Coxiella burnetii* (Philip 1948).

In Europe, Q fever was first reported in humans in Greece during the Second World War, when the microorganism was detected in sera of German soldiers who had febrile illness, known as the 'Balkan flu' (Caminopetros 1946). In 1945, American soldiers who returned to USA from Italy, developed an acute febrile illness accompanied by pneumonia. The cause of the epidemic was identified by serological test as *Rickettsia* of Q fever (Commission on acute respiratory diseases 1946).

Q fever is listed within the category of multiple species diseases in the World Organisation for Animal Health list (OIE 2016). Several domestic and wild animals as well as birds, reptiles and arthropods (particularly ticks) can harbour the pathogen, but cattle, goats and sheep are the main reservoirs. In most animals the infection is asymptomatic, but abortions or stillbirths may occur. The bacteria are spread to the environment by secretions of infected animals (urine, feces and milk) but predominantly via the birth products (more than  $10^9$  bacteria/g placenta) (Arricau-Bouvery and Rodolakis 2005).

In humans, the airborne pathway is the main mode of transmission. The infection is usually caused by inhalation of infectious aerosols directly from birth fluids or via inhalation of dust contaminated by dried placental material, birth fluids and excreta of infected animals (Tissot-Dupont and Raoult 2008). The bacterium can become airborne, traveling on wind currents for miles, resulting in outbreaks (Tissot-Dupont et al. 2004). Humans can also be infected by direct contact with infected animals particularly during abortion and parturition. The infection in humans by ingestion of unpasteurized milk or dairy products, has been also recorded (Tissot-Dupont and Raoult 2008). In addition to ruminants, cats and dogs are also able to shed the organism. In humans, the main characteristic of Q fever is its clinical polymorphism. Following

primary infection more than half of the patients remain asymptomatic. Q fever may manifest as acute or chronic Q fever with long-term sequelae. Acute Q fever usually develops as a non-specific febrile illness, pneumonia or hepatitis (Karakousis and Trucksis 2006). Atypical pneumonia and hepatitis are usually the most classic forms of Q fever. Hepatitis may be expressed as infectious-like hepatitis or fever of unknown origin (FUO) with characteristic hepatic granulomas on liver biopsy. Less common manifestations of acute Q fever include myocarditis, pericarditis, meningoencephalitis and skin rash (Angelakis and Raoult 2010). The chronic infection can manifest itself as endocarditis, chronic fatigue syndrome and problems related to pregnancy (Arricau-Bouvery and Rodolakis 2005).

Q fever was considered predominately an occupational hazard and close contact with ruminants appeared to be strongly associated with the disease in humans (Psaroulaki et al. 2006). Q fever has recently gained renewed attention after the largest-ever recorded outbreak which involved over 4,000 human cases in the Netherlands in 2007-2009 (Vanderburg et al. 2014). This outbreak highlighted the importance of Q fever as an emerging public health threat. Moreover, the widespread distribution of *C. burnetii* in food producing animals and its occurrence in foods of animal origin, particularly in milk, necessitates the investigation of food as a significant vehicle for the transmission of this zoonotic bacterium to humans. Unpasteurized milk is the most significant source of *C. burnetii*. There are epidemiological indications that consumption of milk and/or milk products containing *C. burnetii* has been associated with sero-conversion in humans. Moreover, unpasteurized milk and derived dairy products have been proposed by several authors as sources of human infection (Fishbein and Raoult 1992, Hatchette et al. 2001, Maltezou et al. 2004). However, the contribution of milk ingestion, mainly drinking unpasteurized milk, to Q fever infection in humans is difficult to establish. Moreover, *C. burnetii* was detected in animal products such as raw-milk cheese and butter prepared from raw milk as well as in the meat of infected animals (Capuano et al. 2012, Hirai et al. 2012, Eldin et al. 2013, Hilbert et al. 2015). The pathogen was detected even in chicken eggs from Japan and Iran (Tatsumi et al. 2006, Rahimi and Doosti 2012). In addition to the adverse effects to human health, *C. burnetii* infection in animals can result in the decrease of the livestock production with important socioeconomic effects (Perry et al. 2011). This review summarizes the current knowledge on *C. burnetii* infection in domestic ruminants with special focus on serological prevalence.

## Characteristics of the bacterium *Coxiella burnetii*

*C. burnetii* is a small pleomorphic rod (0.2-0.4 mm wide, 0.4-1.0 mm long) with a membrane similar to that of a Gram-negative bacterium (Maurin and Raoult 1999). It replicates to high numbers within a parasitophorous vacuole of eukaryotic host cells, with an estimated doubling time of 20-45 h (Mertens and Samuel 2007). *C. burnetii* was originally classified in the *Rickettsiales* order, the *Rickettsiaceae* family, and the *Rickettsiae* tribe together with the genera *Rickettsia* and *Rochalimaea*. To date, following phylogenetic investigations based on genome comparison and 16s rRNA sequence analysis, the bacterium was reclassified from the order *Rickettsiales* to *Legionellales*, and falls in the gamma group of *Proteobacteria* (Raoult et al. 2005).

The microorganism produces resistant spore-like forms (Coleman et al. 2004). This ability has been attributed to the existence of *C. burnetii* cycle variants described in *in vitro* studies: large-cell variants (LCV), small-cell variants (SCV), and small dense cells (SDC) (Coleman et al. 2004). The LCV is the larger and the metabolically active intracellular form of *C. burnetii*. It undergoes sporogenic differentiation and produces the resistant, spore-like forms. The SDC and SCV represent the small morphological variants of the bacteria likely to survive extracellularly as infectious particles, a trait that is important for persistence in the environment and transmission (ECDC 2010, Kersh et al. 2010). The environmentally stable SCV (or endospore) is the form phagocytosed by macrophages during early infection and the form associated to food-borne risk (EFSA 2010). The endospores display a tropism for reproductive organs including the mammary gland, are secreted in the milk of infected animals, both from clinical cases and asymptomatic carriers and are also excreted in the detritus of normal births and abortions as well as in the urine and faeces of infected animals. Endospores are released after mother cell lysis and since they are metabolically inactive, they remain stable in soil and dust over many years (Angelakis and Raoult 2010) and can be spread in dust or windborne aerosols for up to 11 miles (18 km) (Hawker et al. 1998).

*C. burnetii* is resistant to acids (up to pH 4.5), temperature (62°C for 30 min), UV light and pressure (up to 300,000 kPa) (Frangoulidis 2010). Furthermore, the organism can survive for more than 6 months in 10% salt solutions. *C. burnetii* is killed by exposure for 30 min to 5% H<sub>2</sub>O<sub>2</sub>, 0.5% hypochlorite, 70% ethanol, for less than 30 min to 5% chloroform or formaldehyde gas (in a 80% humidified environment). Pasteurization of milk (71.66°C for 15 s) is effective in killing *C. burnetii* (Frangoulidis 2010). Endospores can

remain viable for several months in dairy products, meat and meat products, water, aborted fetuses, manure, wool, hay, equipment, and clothing during conditions of high humidity, low temperatures, and no sunlight (EFSA 2010). For example, *C. burnetii* can survive 12 to 16 months in wool, 120 days in dust, 49 days in dried urine and 30 days in dried sputum (NABC 2010).

*C. burnetii* has two distinct antigenic phases, phase I and phase II. Phase I and II variants are morphologically identical, but differ in some biochemical characteristics including their lipopolysaccharide (LPS) composition. LPS II is of rough type in contrast to LPS I, which is phenotypically smooth and contains a noticeable amount of two sugars virenose and dihydrohydroxystreptose (Narasaki and Toman 2012). Organisms isolated from infected animals or humans express phase I antigens and are highly infectious. Organisms expressing phase II antigens are less infectious and are obtained by repeated *in ovo* or *in vivo* passages. In experimentally infected animals, antibodies to phase II antigens are initially produced, while antibodies to phase I antigens are produced in later stages. *C. burnetii* is able to survive permanently inside the macrophages, causing an infection after an acute episode (Gwida et al. 2012).

## Q fever in domestic ruminants

Domestic ruminants are the primary animal reservoirs of *C. burnetii*. Q fever is an airborne disease and inhalation of infected aerosols and dust is the main route of infection of domestic ruminants (Tissot-Dupont et al. 2004). Also, ruminants may become infected by ingestion of contaminated pastures, hay and straw (Maurin and Raoult 1999). It is likely that *C. burnetii* contaminated manure plays a role on the maintenance of infection in animal populations (EFSA 2010). The pathogen has been isolated in several tick species (Maurin and Raoult 1999). Ticks appear to play an important role in enzootic transmission cycles in domestic ruminants (Beaman and Hung 1989). A strong correlation has been reported between seropositivity and ticks' infestation in animals (Psaroulaki et al. 2006).

The presence of Q fever in animals is also related to the characteristics of certain *C. burnetii* strains, and in particular infectivity, virulence and resistance to environmental conditions (Barberio 2015). Maintenance of *C. burnetii* infection in animal populations may be also affected by other factors such as manure management (capture, storage, treatment and utilization), farm characteristics (herd/flock size, animal and herd/flock density) and farm environmental conditions (temperature and relative humidity) (EFSA 2010).

## Clinical signs of Q fever in domestic ruminants

Although *C. burnetii* infection in domestic ruminants is common, clinical disease is rather rare. The infection is generally asymptomatic; but sometimes it can induce reproductive disorders, which differs among ruminant species. Infected sheep may deliver live or dead lambs as well as large abortion waves, mainly at the end of gestation, without specific signs until abortion is imminent (Roest et al. 2013). In contrast to ewes, goats remain chronically infected and can abort twice following an infection (Berri et al. 2007). Martinov (Martinov 2007) has described also experimentally induced Q fever with respiratory manifestations in sheep. In cattle, yet the symptoms described have so far been inconsistent. Factors linked to the disease in cattle have been infertility, abortion and metritis and mastitis in many studies (Arricau-Bouvery and Rodolakis 2005, Barlow et al. 2008). Also, correlation between *C. burnetii* seropositivity and fertility and a low abortion risk has been reported (Lopez-Gatius et al. 2012, Garcia-Ispuerto et al. 2013). However, the presence of *C. burnetii* in dairy herds has been not yet clearly demonstrated to negatively affect reproductive performance. In fact, a recent study showed that seropositive shedding cows had better reproduction than non-infected cows (Garcia-Ispuerto et al. 2013).

Both symptomatic and asymptomatic infected ruminants shed *C. burnetii* in large amount in the environment. Shedding of *C. burnetii* into the environment mainly occurs during abortion and parturition; placentas of infected small ruminants can contain over  $10^9$  hamster infective doses or bacteria per gram of tissue (Fournier et al. 1998). Goats may shed the bacterium in placenta and vaginal mucus in two successive parturitions after a Q fever infection (Berri et al. 2007). At their first kidding, young goats shed more *C. burnetii* cells than adults (de Cremoux et al. 2012). A similar pattern is observed in cattle herds (Guatteo et al. 2008). In contrast, ewes are usually show one abortion and did not shed the pathogen in vaginal mucus at subsequent lambing (Berri et al. 2002). The pathogen excretion can also occur in faeces, milk and urine.

*C. burnetii* shedding can persist for a long time. In goats, shedding of *C. burnetii* in vaginal mucus, faeces, and milk lasted 1 to 5 weeks, 2 to 5 weeks, and 1 day to 6 weeks, respectively. In sheep, the shedding lasted 71 days, 8 days after lambing and 8 days in vaginal mucus, faeces, and milk, respectively. In cows, longest observed duration of excretion of *C. burnetii* in faeces and milk was 14 days and 13 months, respectively (Arricau-Bouvery and Rodolakis 2005). Goats and cows mostly shed *C. burnetii* in milk (Guatteo et al. 2006, Rodolakis et al. 2007) whereas ewes shed the pathogen mostly

in faeces (Rodolakis et al. 2007). In asymptomatic herds, apparently healthy goats and cows may shed the bacterium in milk for several months or years (Rodolakis et al. 2007). *C. burnetii* has been also isolated in bull semen but the role of males in the persistence of infection has not extensively examined (Kruszewska et al. 1997).

## Q fever diagnosis in domestic ruminants

Since *C. burnetii* infection is frequently subclinical, Q fever disease in domestic ruminants remains unclear and not properly investigated. Reproductive disorders should trigger an investigation, considering Q fever among the differential diagnoses. In many countries, diagnosis of Q fever in domestic ruminants still relies mainly on modified Ziehl-Neelsen (MZN)-stained smears of placental material from aborted fetuses, supplemented by immunohistochemistry (IHC) where appropriate, although polymerase chain reaction (PCR) is increasingly being used for disease confirmation in developed countries (Jones et al. 2010). PCR can be used to detect *C. burnetii* DNA in a wide range of samples, including placenta tissues, faeces, vaginal mucus and milk (Horigan et al. 2011). High level of specificity and sensitivity were acquired by PCR method applied with the primers consisting of repetitive transposon-like element (Kirkkan et al. 2008). Real-time PCR is now also commonly used to support a diagnosis of *C. burnetii* abortion/stillbirth in animals faeces, vaginal mucus and milk (Horigan et al. 2011).

A variety of indirect methods (serologic assays) have been used to detect *C. burnetii* antibodies in animal serum samples, including complement fixation test (CFT), enzyme linked immunosorbent assay (ELISA), microagglutination test (MA), indirect immunofluorescence assay (IFA) and indirect fluorescent antibody test (IFAT) (Roest et al. 2013). The CFT is weakly sensitive and the antigen used in this test frequently fails to detect antibodies in sheep or goats (Horigan et al. 2011). The ELISA is more sensitive than the CFT and is able to test a higher number of animals and flocks (Rodolakis 2006).

A combination of both direct and indirect methods is recommended in current protocols to detect Q fever on herd level; however, no official standard technique is still available (Roest et al. 2013).

EFSA outlined the need for harmonized schemes for the passive and active monitoring/reporting of Q fever in animals so that its prevalence/incidence could be compared over time and between countries (EFSA 2010). Recently, some proposals have been elaborated for the development of harmonized monitoring and reporting schemes for the seroprevalence of *C. burnetii* in various countries.

Given the lack of a standard technique, efforts are encouraged both for the validation of the methods and for development of reference reagents for quality control, proficiency and harmonization purposes.

### The seroprevalence of *C. burnetii* in domestic ruminants

Q fever in animals has been detected worldwide, whilst the only country with an apparent zero prevalence is New Zealand (Guatteo *et al.* 2011). Although a great number of *C. burnetii* seroprevalence studies have been conducted throughout the world, the extent of disease in productive ruminants is difficult to quantify. Data reported are mostly based on herds with high increase in abortion (Georgiou 2013). Published data revealed differences in seroprevalence in the animal population in various countries.

### Europe

Accomplished data on the serological prevalence of *C. burnetii* in domestic ruminants from published studies conducted in European countries are

summarized in Table I. In the Netherlands, according to an early (1987) survey of seroprevalence of Q fever by using ELISA showed values of 3.5% and 1% in sheep and goats, respectively (Houwers and Richardus 1987). More recently, according to results of a serological study in Dutch cattle, *C. burnetii* antibodies were present in 16% of lactating cows, but only in 1.0% of young animals (Muskens *et al.* 2011). In Switzerland, Magouras and colleagues (Magouras *et al.* 2017) reported a herd-level seroprevalence of 5.0% in sheep flocks and 11.1% in goat farms. Animal-level seroprevalence was 1.8% in sheep and 3.4% in goats.

In Germany (Southern Bavaria), seroprevalence of *C. burnetii* in cattle tested by ELISA was estimated 11.8% at animal-level and 81.0% at herd-level (Reháček *et al.* 1993). In another study conducted in Germany, 7.8% of cattle, 1.3% of sheep and 2.5% of goats were found to be infected with *C. burnetii* (Hartung 1999). In a study conducted in France, seroprevalence was found in 13/14 and 24/28 of dairy goat herds in 2006 and 2008, respectively (Dubuc-Forfait *et al.* 2009). In Denmark, serum samples taken from 164 high-risk cows with abortion problems showed a prevalence of 10% by CFT and 18% by ELISA, in 2003. In Denmark,

**Table I.** The serological prevalence of *C. burnetii* in domestic ruminants from published studies conducted in European countries. — cont'd

Country	Year of study	Animal species	Number tested		% positive		Method	Reference
			Animals (n)	Herds (n)	Animals	Herds		
Albania	1995-1997	cattle	311	-	10.9	-	ELISA	Cekani <i>et al.</i> 2008
	1995-1997	sheep	350	-	8.8	-	ELISA	Cekani <i>et al.</i> 2008
	1995-1997	goat	443	-	8.9	-	ELISA	Cekani <i>et al.</i> 2008
	1999	cattle	552	-	8.5	-	ELISA	Cekani <i>et al.</i> 2008
	1999	sheep	292	-	12.3	-	ELISA	Cekani <i>et al.</i> 2008
	1999	goat	260	-	4.2	-	ELISA	Cekani <i>et al.</i> 2008
Austria	-	sheep	-	70	-	2.86	CFT	Wagner <i>et al.</i> 2005
	-	goat	-	30	-	16.7	CFT	Wagner <i>et al.</i> 2005
Bulgaria	2002-2006	cattle	15,866	-	8.53	-	CFT	Martinov 2007
	2002-2006	sheep	8,727	-	11.59	-	CFT	Martinov 2007
	2002-2006	goat	3,928	-	13.69	-	CFT	Martinov 2007
Cyprus	-	cattle	75	-	24	-	IFA	Psaroulaki <i>et al.</i> 2006
	-	sheep	481	-	18.9	-	IFA	Psaroulaki <i>et al.</i> 2006
	-	goat	417	-	48.2	-	IFA	Psaroulaki <i>et al.</i> 2006
Denmark	2003	cattle	164	-	10	-	CFT	Christoffersen 2007
					18	-	ELISA	
	2004	cattle	80	-	35	-	ELISA	Christoffersen 2007
France	2006	cattle	266	-	25	-	ELISA	Christoffersen 2007
	2006	goat	359	14	36	92.9	ELISA	Dubuc-Forfait <i>et al.</i> 2009
	2008	goat	1,057	28	32	85.7	ELISA	Dubuc-Forfait <i>et al.</i> 2009
Germany	1991	cattle	1,095	-	12	-	ELISA	Reháček <i>et al.</i> 1993
	1998	cattle	21,191	-	7.8	-	ELISA	Hartung 1999
	1998	sheep	1,346	-	1.3	-	ELISA	Hartung 1999
	1998	goat	278	-	2.5	-	ELISA	Hartung 1999

IFA = Indirect Immunofluorescence assay; IFAT = Indirect Fluorescent Antibody Test; ELISA = Enzyme Linked Immunosorbent assay; CFT = Complement Fixation test; MAT = Microagglutination test.

continued

**Table I.** The serological prevalence of *C. burnetii* in domestic ruminants from published studies conducted in European countries.— *cont' d*

Country	Year of study	Animal species	Number tested		% positive		Method	Reference
			Animals (n)	Herds (n)	Animals	Herds		
Greece	-	sheep	554	-	10.5	-	IFA	Pape <i>et al.</i> 2009
	-	goat	61	-	6.6	-	IFA	Pape <i>et al.</i> 2009
	-	cattle	711	99	4.4	13.1	CFT	Martini <i>et al.</i> 1994
	-	cattle	-	-	14	-	IFAT	Capuano <i>et al.</i> 2004
	-	sheep	-	-	11.8	-	IFAT	Capuano <i>et al.</i> 2004
	-	goat	-	-	6.3	-	IFAT	Capuano <i>et al.</i> 2004
Italy	1999-2002	sheep	-	675	-	38	ELISA	Masala <i>et al.</i> 2004
	1999-2002	goat	-	82	-	47		Masala <i>et al.</i> 2004
	-	cattle	650	-	44.9	-	ELISA	Cabassi <i>et al.</i> 2006
	2012	sheep	2,553	111	15.5	38.7	ELISA	Rizzo <i>et al.</i> 2016
	2012	goat	3,185	206	16.2	19.5	ELISA	Rizzo <i>et al.</i> 2016
Montenegro	-	sheep	954	-	5.03	-	MAT & IFA	Lausevic 2001
	1987	sheep	3,603	191	3.5	27.2	ELISA	Houwers and Richardus 1987
Netherlands	1987	goats	594	-	1	-	ELISA	Houwers and Richardus 1987
	2008	cattle	2,936	-	16	-	ELISA	Muskens <i>et al.</i> 2011
	2008	cattle (young animal)	1,831	-	1	-	ELISA	Muskens <i>et al.</i> 2011
	2007-2008	cattle	626	46	6.7	43	ELISA	Ruiz-Fons <i>et al.</i> 2010
Spain	2007-2008	sheep	1,379	42	11.8	74	ELISA	Ruiz-Fons <i>et al.</i> 2010
	2007-2008	goat	115	11	8.7	45	ELISA	Ruiz-Fons <i>et al.</i> 2010
	2005	sheep	1,011	34	8.9	67.6	ELISA	Garcia-Perez <i>et al.</i> 2009
	2009-2010	cattle	1,306	-	12.3	-	ELISA	Astobiza <i>et al.</i> 2012
Slovakia	2000	sheep	269	-	37.22	-	ELISA	Dorko <i>et al.</i> 2010
	2009	sheep	269	-	58.42	-	ELISA	Dorko <i>et al.</i> 2010
Switzerland	-	sheep	-	100	1.8	5	ELISA	Magouras <i>et al.</i> 2015
	-	goat	-	72	3.4	11.1	ELISA	Magouras <i>et al.</i> 2015
	-	goat	98	-	79.6	-	MAT	Platt-Samoraj <i>et al.</i> 2005
Poland	2011-2012	cattle	169	-	11.83	-	ELISA	Bielawska-Drózda <i>et al.</i> 2014
	2011-2012	cattle	169	-	10.65	-	CFT	Bielawska-Drózda <i>et al.</i> 2014
UK (Ireland)	2009	cattle	5,182	273	6.2	48.4	ELISA	McCaughey <i>et al.</i> 2010
	-	sheep	1,022	58	12.3	62.1	IFA	McCaughey <i>et al.</i> 2010
	-	goat	54	7	9.3	42.9	IFA	McCaughey <i>et al.</i> 2010
UK (Great Britain)	-	goat	5791	384	0.9	10.2	ELISA	Lambton <i>et al.</i> 2016
	-	sheep	522	145	0.8	3	ELISA	Lambton <i>et al.</i> 2016

IFA = Indirect Immunofluorescence assay; IFAT = Indirect Fluorescent Antibody Test; ELISA = Enzyme Linked Immunosorbent assay; CFT = Complement Fixation test; MAT = Microagglutination test.

a seroprevalence of 35% was found in blood samples from 80 cattle in 2004, and 25% in 266 cattle in 2006 (Christoffersen 2007).

In Austria, Wagner and colleagues (Wagner *et al.* 2005) examined blood samples from 70 Styrian sheep by CFT and found that 1.5 % of the samples contained antibodies to *C. burnetii*.

In Italy (Emilia-Romagna Region), the Q fever seroprevalence in cattle was 13.1% at herd level and 4.4% at animal level (Martini *et al.* 1994). In Italy (Campania), Capuano and colleagues (Capuano *et al.* 2004) reported a seroprevalence of 11.8% in sheep, 6.3% in goats and 14% in cattle. In Sardinia, Italy, antibodies to *C. burnetii* have been detected in

38% and 47% of sheep and goat herds, respectively (Masala *et al.* 2004). In Northern Italy, 44.9% out of 650 cattle with experienced abortion were seropositive for *C. burnetii* (Cabassi *et al.* 2006). In Northwest Italy, the animal level seroprevalence was 15.5% in sheep and 16.2% in goats. The sheep-farm and goat-farm seroprevalence was 38.7% and 19.5%, respectively (Rizzo *et al.* 2016).

In Northern Ireland, 6.2% of cattle and 48.4% of tested herds were seropositive in 2009 (McCaughey *et al.* 2010). In Great Britain, estimates of animal and flock/herd seroprevalences were 0.9% and 10.2%, respectively, for sheep and 0.8% and 3%, respectively, for goats (Lambton *et al.* 2016).

In Spain (Basque region), a serosurvey conducted during the years 2007-2008 showed a prevalence of 11.8%, 8.7% and 6.7% in sheep, goats and beef cattle, respectively (Ruiz-Fons *et al.* 2010). In the same study, a *C. burnetii* prevalence of 74%, 45% and 43% for ovine, caprine and bovine herds, respectively, was also recorded (Ruiz-Fons *et al.* 2010). In Northern Spain, 8.9% of the sheep were seropositive, 67.6% of the flocks had at least one seropositive animal, but only 14.7% of them showed a seroprevalence higher than 25% (Garcia-Perez *et al.* 2009). In Northern Spain, in a study conducted by Astobiza *et al.* (2012) cows showed a statistically significantly higher seroprevalence (12.3%) than heifers (1.1 %) and calves (0%) (Astobiza *et al.* 2012).

In Albania, Cekani and colleagues (Cekani *et al.* 2008) conducted a survey on *C. burnetii* and reported that the seroprevalence detected in sheep and goats (9.8%) was higher than in cattle (7.9%). In Bulgaria, Martinov (Martinov 2007) found that the seropositivity for *C. burnetii* was 8.53% in cattle, 11.59% in sheep and 13.69% in goats. In Montenegro, Lausevic (Lausevic 2001) reported a 5.03% seroprevalence of *C. burnetii* in sheep. In Slovakia, the seropositivity in sheep was 37.22%

in 2000 and 58.42% in 2009 (Dorko *et al.* 2010). In Poland, Platt-Samoraj and colleagues (Platt-Samoraj *et al.* 2005) examined serum samples from a goat farm with animals affected with reproductive disorders and found that 79.6% of the samples had *C. burnetii* antibodies in a more recent survey 11.83% and 10.65% of cattle had *C. burnetii* antibodies when serum samples were tested by ELISA and CFT method, respectively (Bielawska-Drózda *et al.* 2014).

In Greece, Pape and colleagues (Pape *et al.* 2009) found *C. burnetii* antibodies in 10% of the animals examined. The seroprevalence was higher in sheep flocks (10.4%) compared to goats' herds (6.5%). In Cyprus, the prevalence of antibodies against *C. burnetii* was 48.2% in goats, 18.9% in sheep, and 24% in cattle (Psaroulaki *et al.* 2006).

### Asia, Africa, America and Oceania

Data on seroprevalence of *C. burnetii* in domestic ruminants collected from studies conducted in countries in Asia are presented in Table II.

In Turkey, Cetinkaya and colleagues (Cetinkaya *et al.* 2000) in 1998, found a seroprevalence

**Table II.** The serological prevalence of *C. burnetii* in domestic ruminants from published studies conducted in Asian countries. — cont'd

Country	Year of study	Animal species	Number tested		% positive		Method	Reference
			Animals (n)	Herds (n)	Animals	Herds		
Bangladesh	2009-2010	cattle	620	-	0.65	-	ELISA	Haider <i>et al.</i> 2015
	2009-2010	goats	529	-	0.76	-	ELISA	Haider <i>et al.</i> 2015
China	-	cattle	1140	19	33	84	ELISA	El-Mahallawy <i>et al.</i> 2016
	2011-2013	sheep	2112	-	14.39	-	ELISA	Yin <i>et al.</i> 2015
	2008	cattle	93	12	10.75	16.6	ELISA	Khalili and Sakhaee 2009
	2008	goats	76	9	65.78	100	ELISA	Khalili and Sakhaee 2009
	2009	sheep	85	-	29.42	-	ELISA	Sakhaee and Khalili 2010
	2010	cattle	246	19	22.3	78.9	ELISA	Azizzadeh <i>et al.</i> 2011
	2010-2011	sheep	253	-	23.7	-	ELISA	Mostafavi <i>et al.</i> 2012
	2011-2012	sheep	1100	-	19.5	-	ELISA	Asadi <i>et al.</i> 2013
Iran	2011-2012	goat	180	-	27.2	-	ELISA	Asadi <i>et al.</i> 2013
	-	sheep	255	29	36.5	89.6	ELISA	Keyvani <i>et al.</i> 2014
	-	goat	205	28	29.8	78.5	ELISA	Keyvani <i>et al.</i> 2014
	2014	cattle	120	10	0.83	10	ELISA	Edalati-Shokat <i>et al.</i> 2015
	2014	sheep	200	10	27.5	100	ELISA	Edalati-Shokat <i>et al.</i> 2015
	2014	goat	50	10	54	100	ELISA	Edalati-Shokat <i>et al.</i> 2015
	-	sheep	253	-	33.6	87.50	ELISA	Esmaili <i>et al.</i> 2014
Japan	1982-1991	cattle	562	-	46.6	-	IFAT	Htwe <i>et al.</i> 1992
	1974-1989	sheep	256	-	28.1	-	IFAT	Htwe <i>et al.</i> 1992
	1974-1989	goat	85	-	23.5	-	IFAT	Htwe <i>et al.</i> 1992
Korea	2010	cattle	1000	-	1.3	-	ELISA	Jang <i>et al.</i> 2011
	2010-2013	cattle	1,095	-	6.2	-	ELISA	Kim <i>et al.</i> 2014
	-	goat	575	-	19.1	-	ELISA	Jung <i>et al.</i> 2014

IFA = Indirect Immunofluorescence assay; IFAT = Indirect Fluorescent Antibody Test; ELISA = Enzyme Linked Immunosorbent assay; CFT = Complement Fixation test; MAT = Microagglutination test.

continued

**Table II.** The serological prevalence of *C. burnetii* in domestic ruminants from published studies conducted in Asian countries. — cont'd

Country	Year of study	Animal species	Number tested		% positive		Method	Reference
			Animals (n)	Herds (n)	Animals	Herds		
Pakistan	-	sheep	271	-	26.9	-	ELISA	Zahid et al. 2016
	-	goat	271	-	34.9	-	ELISA	Zahid et al. 2016
	1998	cattle	416	48	5.8	35.4	IFAT	Cetinkaya et al. 2000
	1998	sheep	411	47	10.5	44.7	IFAT	Cetinkaya et al. 2000
	-	sheep	184	-	38.59	-	IFA	Kalender 2001
	-	sheep	224	-	11.01	-	IFA	Kalender 2001
	-	cattle	177	-	5.6	-	ELISA	Seyitoğlu et al. 2006
	-	cattle	53	-	22.6	-	ELISA	Seyitoğlu et al. 2006
	2006-2008	cattle	92	-	26.3	-	ELISA	Ceylan et al. 2009
	2006-2008	sheep	92	-	5.4	-	ELISA	Ceylan et al. 2009
Turkey	-	sheep	465	-	21.07	-	ELISA	Karaca et al. 2009
	2001-2004	sheep	743	42	20	81	ELISA	Kennerman et al. 2010
	2002	sheep	100	-	3	-	CFT	Kilic et al. 2005
	-	cattle	200	-	20	-	ELISA	Parin and Kaya 2015
	-	sheep	200	-	29	-	ELISA	Parin and Kaya 2015
	-	goats	200	-	21	-	ELISA	Parin and Kaya 2015
	-	cattle	200	-	22	-	IFA	Parin and Kaya 2015
	-	sheep	200	-	29	-	IFA	Parin and Kaya 2015
	-	goats	200	-	23	-	IFA	Parin and Kaya 2015

IFA = Indirect Immunofluorescence assay; IFAT = Indirect Fluorescent Antibody Test; ELISA = Enzyme Linked Immunosorbent assay; CFT = Complement Fixation test; MAT = Microagglutination test.

of *C. burnetii* of 5.8% and 35.4% in cattle at herd and animal level, respectively, and a seroprevalence of 10.5% and 44.7% at flock and animal level, respectively. In the same country, Kalender (Kalender 2001) reported a *C. burnetii* seropositivity of 38.59% in aborted ewes and of 11.01% in non aborted ewes. In Western Turkey, Kilic and colleagues (Kilic et al. 2005) found a relative low seropositivity in sheep (3%). In the same country, according to a study of Seyitoğlu and colleagues (Seyitoğlu et al. 2006), the seropositivity was 5.6% in healthy cattle and 22.6% in cattle with an abortion history. In eastern Turkey, the seropositivity was found to be 16.3% in cattle and 5.4% in sheep (Ceylan et al. 2009). Two other separate studies in sheep showed a seroprevalence of *C. burnetii* of 21.07% (Karaca et al. 2009) and 20% (Kennerman et al. 2010). More recently, a seroprevalence of 12.4% was found in dairy cattle (Gazyagci et al. 2011) and of 20.0%, 29.0% and 21.0% in cattle, sheep and goats, respectively, when tested by ELISA. In the same animals the prevalence was of 22.0%, 29.0% and 23.0%, respectively when tested by IFA (Parin and Kaya 2015).

In Southeast Iran, in 2008, a significantly higher average seroprevalence (65.78%) was observed in goats than in cattle (10.75%) (Khalili and Sakhaee 2009). The seroprevalence of *C. burnetii* was 29.42% and 23.7% in sheep in two studies conducted in Southeast Iran (Sakhaee and Khalili 2010) and Northern Iran, respectively (Mostafavi

et al. 2012). In Eastern Iran, 22.3% of dairy cattle were found seropositive to *C. burnetii* according to a study conducted by Azzizadeh and colleagues (Azzizadeh et al. 2011), in 2010. In Northeastern Iran, seroprevalence of *C. burnetii* at animal level was 36.5% for sheep and 29.8% for goat populations (Keyvani et al. 2014). In Northwestern Iran, 33.6% of sheep and 87.50% of sheep herds were found positive for *C. burnetii* (Esmaeili et al. 2014). Recently, in Western Iran, antibodies to *C. burnetii* were detected in 27.5% of sheep, in 54% of goats and in 0.83% of dairy cattle (Edalati-Shokat et al. 2015). In Iran, according to a study conducted by Asadi and colleagues (Asadi et al. 2013), the seroprevalence of *C. burnetii* in sheep and goats with a history of abortion was 19.5% and 27.2%, respectively.

In Pakistan, seroprevalence was 26.9% and 34.9% for sheep and goat, respectively (Zahid et al. 2016). In Bangladesh, a low seropositivity in cattle and goats (0.65% and 0.76%, respectively) was recorded by Haider and colleagues (Haider et al. 2016).

In Japan, a survey on prevalence of *C. burnetii* antibodies in healthy ruminant farms showed values of 25.4% in cattle, 28.1% in sheep and 23.5% in goats. In this study, the seroprevalence reached values of 84.3% in bovine herds with reproductive disorders (Htwe et al. 1992). In China, *C. burnetii* seropositive cattle (33% of studied animals) were detected in 13 of the 15 surveyed provinces and in 16 of the 19 herds (84%) (El-Mahallawy et al. 2016). In the same



**Table III.** The serological prevalence of *C. burnetii* in domestic ruminants from published studies conducted in African countries.

Country	Year of study	Animal species	Number tested		% positive		Method	Reference
			Animals (n)	Herds (n)	Animals	Herds		
Cameroon	2000	cattle	13,377	146	31.3	68	ELISA	Scolamacch et al. 2010
	1999-2000	cattle	195	-	4	-	ELISA	Schelling et al. 2003
	1999-2000	sheep	142	-	11	-	ELISA	Schelling et al. 2003
	1999-2000	goat	134	-	13	-	ELISA	Schelling et al. 2003
Chad	-	goat	72	-	16.8	-	IFA	Mazyad and Hafez 2007
	-	cattle	54	-	13	-	ELISA	Nahed and Khaled 2012
	-	sheep	55	-	32.7	-	ELISA	Nahed and Khaled 2012
	-	goat	30	-	23.3	-	ELISA	Nahed and Khaled 2012
Egypt	-	sheep	89	-	22.5	-	IFA	Mazyad and Hafez 2007
	2012-2013	cattle	1,194	-	13.2	-	ELISA	Gwida et al. 2014
	2012-2013	cattle	1,194	9	13.2	100	ELISA	Gwida et al. 2014
Gambia	2012	sheep	398	-	18.5	-	ELISA	Klaasen et al. 2014
	2012	goat	490	-	24.2	-	ELISA	Klaasen et al. 2014
Japan	1982-1991	cattle	562	-	46.6	-	IFAT	Htwe et al. 1992
	1974-1989	sheep	256	-	28.1	-	IFAT	Htwe et al. 1992
	1974-1989	goat	85	-	23.5	-	IFAT	Htwe et al. 1992
Nigeria	-	cattle	306	-	59.8	-	CAT	Adesiyun et al. 1984
Transvaal	1985-1986	cattle	8,900	-	7.78	-	CFT	Gummow et al. 1987
Sudan	2010-2011	goat	460	-	24.22	-	ELISA	Hussien et al. 2012
	2011	cattle	242	-	14.8	-	ELISA	Dean et al. 2013
Togo	2011	sheep	207	-	14.4	-	ELISA	Dean et al. 2013
	2011	goat	198	-	8.3	-	ELISA	Dean et al. 2013
Zimbabwe	-	cattle	180	-	39	-	IFA	Kelly et al. 1993
	-	goat	180	-	10	-	IFA	Kelly et al. 1993

IFA = Indirect Immunofluorescence assay; IFAT = Indirect Fluorescent Antibody Test; ELISA = Enzyme Linked Immunosorbent assay; CFT = Complement Fixation test; MAT = Microagglutination test.

country, 14.39% of the examined Tibetan sheep were *C. burnetii* seropositive (Yin et al. 2015). In Korea, two separate studies in cattle showed a seroprevalence of *C. burnetii* of 1.3% (Jang et al. 2011) and 6.2% (Kim et al. 2014). In Korea, the estimated seroprevalence in native goats was 19.1% (Jung et al. 2014).

In Africa, summarized data on seroprevalence of *C. burnetii* in animals are shown in Table III. In Nigeria, an antibody prevalence of 59.8% was detected among 306 dairy cows (Adesiyun et al. 1984). In Zimbabwe, serological evidence of Q fever infection was found in 39% of cattle and in 10% of goats (Kelly et al. 1993). In South Africa's Transvaal Province, 8,900 cattle were examined for antibodies to *C. burnetii* and 7.78% were found positive (Gummow et al. 1987). In Cameroon, Q fever in cattle had a seroprevalence of 31.3% (Scolamacchia et al. 2010). In Sudan, Hussein and colleagues (Hussein et al. 2012) reported a *C. burnetii* prevalence of 24.22% in caprine serum samples from 8 states. In Togo, Dean and colleagues (Dean et al. 2013) reported a *C. burnetii* seroprevalence of 14.8%, 14.4% and 8.3% in cattle, sheep and goats, respectively. In Chad, the seroprevalence was 4%, 11% and 13% in cattle, sheep and goats, respectively (Schelling et al.

2003). In Gambia, goats had a significantly higher seroprevalence (24.2%) than sheep (18.5%) (Klaasen et al. 2014).

In a serological study conducted in Egypt, 22.5% of sheep and 16.5% of goats were found seropositive to *C. burnetii* (Mazyad and Hafez 2007). In Egypt, Nahed and Khaled (Nahed and Khaled 2012) reported a seroprevalence of 13%, 32.7%, and 23.3% in cattle, sheep and goats, respectively. More recently, in Egypt, anti-*Coxiella* antibodies were detected in 13.2% of cattle (Gwida et al. 2014).

Data on the serological prevalence of *C. burnetii* in animals from published studies conducted in countries of America and Oceania are summarized in Table IV. In the USA, goats were found to have a significantly higher average seroprevalence (41.6%) than sheep (16.5%) or cattle (3.4%) (McQuiston and Childs 2002). In California (USA), DeForce and Cone (DeForce and Cone 2006) estimated the seroprevalence of *C. burnetii* in bighorn sheep at a level of 10%. In Missouri (USA), blood samples from Boer goats were tested by ELISA and animal and herd-level seroprevalence estimates for *C. burnetii* were 1.2% and 4.2%, respectively (Baker and Pithua

**Table IV.** The serological prevalence of *C. burnetii* in domestic ruminants from published studies conducted in countries of America and Oceania.

Country	Year of study	Animal species	Number tested		% positive		Method	Reference
			Animals (n)	Herds (n)	Animals	Herds		
<b>AMERICA</b>								
Canada	1997	sheep	234	-	3.1	-	MIF	Hatchette <i>et al.</i> 2002
	1999	goat	147	-	55.8	-	IFA	Hatchette <i>et al.</i> 2001
	2000-2001	cattle	75	-	24	-	MIF	Hatchette <i>et al.</i> 2002
	2000	sheep	34	-	23.5	-	MIF	Hatchette <i>et al.</i> 2002
	2000	goat	64	-	15.6	-	MIF	Hatchette <i>et al.</i> 2002
	2010-2012	sheep	2,363	72	14.7	48.6	ELISA	Meadows <i>et al.</i> 2015
Colombia	-	cattle	357	-	25	-	CFT	Lorbacher de Ruiz 1977
	-	cattle	125	-	17	-	CFT	Lorbacher de Ruiz 1977
Ecuador	2008-2010	cattle	2,668	386	12.6	46.9	ELISA	Carbonero <i>et al.</i> 2015
Mexico	-	cattle	NA	-	28	-	ELISA	Salinas-Malendez <i>et al.</i> 2002
	-	cattle	NA	-	10	-	ELISA	Salinas-Malendez <i>et al.</i> 2002
	-	sheep	NA	-	40	-	ELISA	Salinas-Malendez <i>et al.</i> 2002
	-	goat	NA	-	35	-	ELISA	Salinas-Malendez <i>et al.</i> 2002
<b>USA</b>								
(California)	1992-1999	sheep	268	-	10	-	CFT	DeForge and Cone 2006
(Washington)	2010-2011	goat	1794	105	8	8.6	ELISA	Sondgeroth <i>et al.</i> 2013
(Missouri)	2012	goat	249	24	1.2	4.2	ELISA	Baker and Pithua 2014
(Washington, Montana, and Oregon)		goat	567	-	12	-	ELISA	Anderson <i>et al.</i> 2015
Venezuela	-	goat	315	-	60.63	-	ELISA	Oropeza <i>et al.</i> 2010
<b>OCEANIA</b>								
Australia (Victoria)	1970	cattle	1576	49	0.5	12.2	CFT	Hore and Kovesdy 1972
Australia (Western)	NA	cattle	329	-	0.61	-	ELISA	Banazis <i>et al.</i> 2010
	NA	sheep	50	-	0	-	ELISA	Banazis <i>et al.</i> 2010
New Zealand	1990-1992	cattle	2181	-	0	-	CFT	Hilbink <i>et al.</i> 1993

IFA = Indirect Immunofluorescence assay; IFAT = Indirect Fluorescent Antibody Test; ELISA = Enzyme Linked Immunosorbent assay; CFT = Complement Fixation test; MAT = Microagglutination test.

2014). In a study conducted in Washington State (USA), the results identified *C. burnetii* antibodies in 8.0% of goat serum samples, 8.6% of goat herds, and 25.8% of counties (Sondgeroth *et al.* 2013). In an epidemiological investigation of a Q fever outbreak conducted in Washington, Montana, and Oregon (USA), Anderson and colleagues (Anderson *et al.* 2015) tested 567 goats from 17 herds finding a *C. burnetii* seroprevalence of 12%.

In Canada (Newfoundland), 55.8% of the examined goats were seropositive to *C. burnetii* (Hatchette *et al.* 2001). In the same area of Canada, Hatchette and colleagues (Hatchette *et al.* 2002) found that seropositivity increased in sheep from 3.1% in 1997 to 23.5% in 1999-2000. They also observed a seroprevalence of *C. burnetii* of 24% and 15.6% in examined cows and goats, respectively. In Canada (Ontario), the seroprevalence of *C. burnetii* ranged between 33 and 82% in cattle herds and between 0 and 35% in sheep flocks (Martin and Innes 2002). Recently, also in Ontario, in a study conducted by Meadows and colleagues (Meadows *et al.* 2015), 14.7% of sheep were found infected with *C. burnetii*.

The seroprevalence was higher in dairy sheep (24.3%) than in meat sheep (10.2%). In the same study, 48.6% of farms had at least one seropositive sheep. In Australia (Queensland), in a study conducted by Cooper and colleagues (Cooper *et al.* 2011), 16.8% beef cattle were positive to *C. burnetii* antibodies.

In Mexico, the 28% of the dairy cattle, 10% of beef cattle, 40% of sheep and 35% of goats were seropositive (Salinas-Malendez *et al.* 2002). In Colombia, Lorbacher de Ruiz (Lorbacher de Ruiz 1977) found that 25% and 17% of tested dairy cows and beef cattle heifers, respectively, were seropositive. In Venezuela, a high seropositivity (60.23%) was recorded in goats belonging to herds with a history of high percentage of abortions (Oropeza *et al.* 2010). In Ecuador, the prevalence of *C. burnetii* reached 12.6% with a herd prevalence of 46.9% (Carbonero *et al.* 2015).

In Australia, extremely low prevalence values (0.67% and 0.5%) were recorded in cattle by Banazis and colleagues (Banazis *et al.* 2010) and Hore and Kovesdy (Hore and Kovesdy 1972), respectively;

conversely, no seropositive sheep were found in 2009 (Banazis *et al.* 2010). In New Zealand, in the study of Hilbink and colleagues (Hilbink *et al.* 1993), all tested animals were seronegative.

In conclusion, despite the observed differences, a relatively high proportion of domestic ruminants has *C. burnetii* antibodies worldwide. Effective control strategies are necessary to limit the impact of the zoonotic risk of Q fever. The control plan of this disease in both animals and humans should rely on control measures of preventing infection

of domestic ruminants and, in particular, sheep and goats. Vaccination of domestic ruminants is reported to be effective in preventing abortion and reducing bacterial shedding, especially after several years of administration (Roest *et al.* 2013). The implementation of good hygiene and other management practices including manure management and risk materials handling, may also reduce the environmental load and, in turn, may result in a decrease of *C. burnetii* human and animal infection (Roest *et al.* 2013).

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