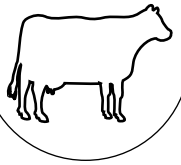

Seroprevalence of *Coxiella burnetii* in selected populations of domestic ruminants in Newfoundland

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Abstract — The seroprevalence of *Coxiella burnetii* among cattle, sheep, and goats in Newfoundland was determined by microimmunofluorescence. Seropositivity to phase II antigen increased in sheep from 3.1% in 1997 to 23.5% in 1999–2000 ($P < 0.001$). Cows (24%) had antibodies to phase I antigen; goats (15.6%) had antibodies to phase II antigen. Seroprevalence of *C. burnetii* is increasing among sheep.

Résumé — Séroprévalence de *Coxiella burnetii* dans des populations choisies de bétail de Terre-Neuve. La séroprévalence de *Coxiella burnetii* chez les bovins, les moutons et les chèvres de Terre-Neuve a été vérifiée par microinmunofluorescence. La séropositivité à l'antigène de phase II, chez le mouton, est passée de 3,1 % en 1997 à 23,5 % en 1999–2000 ($P < 0,001$). Les vaches (24 %) possèdaient des anticorps à l'antigène de phase I et les chèvres (15,6 %) à l'antigène de phase II. La séroprévalence de *Coxiella burnetti* est en augmentation chez les moutons.

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Coxiella burnetii, an obligate intracellular pathogen, is the causative agent of Q fever, a zoonosis with worldwide occurrence (1–2). *Coxiella burnetii* has been found in many wild and domestic animals (1,3–7). The most common reservoirs for infection of humans are domestic farm animals, such as cattle, goats, and sheep (8–14). *Coxiella burnetii* is shed in the urine, feces, and milk from infected animals and has a particularly high concentration in the products of conception (15). Exposure to infected parturient cats has resulted in many small outbreaks of Q fever in Nova Scotia (14).

Recently, we described an outbreak of Q fever in early 1999 among goats on a 9-farm caprine cooperative in Newfoundland (15). *Coxiella burnetii* exists mainly in the phase I form in animals and in phase II in tissue culture (13). The purpose of this paper is to provide data on the seroprevalence of *C. burnetii* in sheep, prior and subsequent to the outbreak, and in cows and goats subsequent to the 1999 outbreak in areas of the province remote from the outbreak.

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Blood samples were obtained from 293 sheep in 1997. These sheep were housed on farms scattered throughout the province. As many pastures as possible were sampled. A maximum of 10 sheep per pasture were sampled. From May 1999 through January 2000, blood samples from 34 sheep housed on farms scattered throughout the province were obtained.

Blood samples were obtained from 64 goats during 2000. These goats were all remote from the cooperative where the 1999 outbreak occurred. Blood samples were obtained from 75 cows in a province-wide convenience sampling from June 2000 through February 2001.

Antibody titers to *C. burnetii* were determined by using a microimmunofluorescence test, as previously described (3). A titer of 1:8 to either phase I or phase II antigens was considered to be positive. Proportions were compared using the chi-squared test or Fisher's exact test.

In 1997, none of the sheep tested had a significant level of phase I antibody and 3.1% had a significant level of phase II antibody; all of the latter had low titers (Table 1). In 1999–2000, 8 of the 34 sheep tested (23.5%) had a significant level of phase II antibody. The 64 goats that were tested had low rates of positivity (Table 1). In contrast, 18 of 75 (24%) cows tested had a significant level of phase I antibody, some with high titers (Table 1).

In 1964, McKeil (16) determined antibody titers to *Coxiella burnetii* in cattle in all 10 Canadian provinces by using a capillary tube agglutination test and a complement fixation test. The former was used to detect

Table 1. Number and percent of animals with an antibody titer (and the highest antibody titer) to phase I and phase II *Coxiella burnetii* antigen

		Phase I		Phase II	
Animals	No. tested	No. positive (%)	Highest titer	No. positive (%)	Highest titer
Sheep	293 (1997)	0		9 (3.1%)	1.16
Sheep	34 (2000)	0		8 (23.5%)	1:32
Goats	64	2 (3.1%)	1:8	10 (15.6%)	1:256
Cows	75	18 (24%)	1:2048	7 (9.3%)	1:64

antibodies to phase I antigen and the latter to phase II antigen. Eighty herds in Newfoundland were tested and 1 (1.3%) was positive. Seven of the 22 (31.8%) cattle in the positive herd had antibodies to phase I antigen. The highest antibody titer was 1:8. Nine of the 22 (40.9%) had antibodies to phase II antigen, as measured by the complement fixation test; 6 at a titer of 1:8, 3 at 1:16, and 1 at 1:32. None of the animals tested in Nova Scotia, New Brunswick, or Prince Edward Island were positive. In contrast, 39.6% of the herds tested in Quebec were positive. In 1984, Lang (17) noted that 33% to 82% of the herds tested in Ontario were positive compared with 0% to 10% during McKiel's survey.

Our data show a remarkable and very significant change in the seroprevalence of *C. burnetii* among sheep, from 3.1% seropositivity in 1997 to 23.5% in 1999–2000 ($P < 0.001$). About the same rate of seropositivity was seen among cows.

One hundred forty-seven goats were tested serologically as part of the workup of the outbreak of Q fever in 1999. The goats had been imported from Prince Edward Island, Maine, and Ontario. One farm had goats that were born in Newfoundland and this farm had a very low seropositivity rate. Overall 55.8% of the goats were seropositive with antibody titers ranging from 1:8 to > 1:4096. Indeed, 43% of the goats had a phase I antibody titer of 1:64, while 20% had such a titer to phase II antigen. The abortion rates were 16% to 22% per farm. Sixty-six of 179 (36.9%) farmers, farm workers, or their contacts developed Q fever (15). A previous study in late 1980s showed that 20% of 20 goat herds in Ontario (426 animals) had antibodies to *C. burnetii* (18) and cases of human Q fever followed exposure to infected goats at the 1991 Royal Ontario Winter Fair (19). In the acute form of human infection (fever, pneumonia, hepatitis), antibodies to phase II dominate, while, in other animals, phase I antibodies predominate, as they do in humans with chronic infection (endocarditis); in tissue culture, *C. burnetti* exists mainly in the phase II form (13).

In the current study, phase II antibody predominated among sheep and goats, and the reverse was true among cattle. We have previously observed that infected cats that were associated with cases of Q fever had high phase I antibody titers, while phase II antibody titers predominated among seropositive cats that were not associated with cases of Q fever (20). It is tempting to attribute a cause and effect relationship between the outbreak of Q fever on the caprine cooperative and the increasing seroprevalence of Q fever among sheep in Newfoundland; however, to conclude this, it would be necessary to show that the strains are identical. Nevertheless, we can conclude that the seroprevalence of Q fever among sheep is increasing in Newfoundland. CVJ

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