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Coxiella burnetii Infection in a Community Operating a Large-Scale Cow and Goat Dairy, Missouri, 2013

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Abstract. *Coxiella burnetii* is a zoonotic pathogen that causes Q fever in humans and is transmitted primarily from infected goats, sheep, or cows. Q fever typically presents as an acute febrile illness; however, individuals with certain predisposing conditions, including cardiac valvulopathy, are at risk for chronic Q fever, a serious manifestation that may present as endocarditis. In response to a cluster of Q fever cases detected by public health surveillance, we evaluated *C. burnetii* infection in a community that operates a large-scale cow and goat dairy. A case was defined as an individual linked to the community with a *C. burnetii* phase II IgG titer ≥ 128. Of 135 participants, 47 (35%) cases were identified. Contact with or close proximity to cows, goats, and their excreta was associated with being a case (relative risk 2.7, 95% confidence interval 1.3–5.3). Cases were also identified among individuals without cow or goat contact and could be related to windborne spread or tracking of *C. burnetii* on fomites within the community. A history of injection drug use was reported by 26/130 (20%) participants; follow-up for the presence of valvulopathy and monitoring for development of chronic Q fever may be especially important among this population.

INTRODUCTION

Coxiella burnetii is the causative agent of Q fever, an underrecognized and underreported zoonotic disease. Acute Q fever typically manifests as an acute febrile illness, sometimes characterized by pneumonia or hepatitis. Acute Q fever is most often self-limited and is effectively treated with doxycycline.[1–3] A small percentage of those infected with *C. burnetii* (< 5%) may develop chronic Q fever. Chronic Q fever is a serious and potentially fatal infection that may manifest as endocarditis, chronic vascular infection, chronic hepatitis, or chronic pulmonary infection. Chronic Q fever most often occurs in those with a predisposing condition, such as valvular heart disease, an arterial aneurysm, or a vascular graft.

Coxiella burnetii is most commonly transmitted to humans by infected ruminants (primarily goats, sheep, and cows), which can shed large quantities of organism in birth products, feces, and milk. The route of human transmission is most commonly via inhalation of contaminated aerosols or dust, and the infectious dose of *C. burnetii* may be as low as a single organism.[4] Once shed into the environment, the resilient spore-like form of *C. burnetii* can persist for years, and windborne spread of the organism can occur.[5–10] In the United States, Q fever outbreaks have most commonly been described in association with livestock farms, slaughterhouses, and research settings where livestock are housed.[11]

Case report. On June 5, 2013, a man participating in a residential substance abuse rehabilitation program in Community A, Missouri was hospitalized with 4 days of symptoms, including fever to 105°F, rigors, headache, myalgia, and syncope. The rehabilitation program was located on a large-scale cow and goat dairy, where participants routinely worked in close proximity to ruminants as part of their treatment plan. The patient worked in the kitchen of the program's dormitory, which is located < 0.5 km from a manure lagoon and approximately 2 km from the cow dairy. He routinely delivered meals to the cow- and goat-dairy workers at their worksites but reported no direct animal contact. He had no history of recent travel and had resided at the rehabilitation facility for approximately 4 months. Acute serology for Q fever sent on June 6, 2013, tested negative. Follow-up serologic testing for Q fever on June 17, 2013, confirmed acute Q fever, with phase II IgG 2048, phase I IgG < 16, phase II IgM ≥ 2048, and phase I IgM ≥ 2048.

In the 5 months after the presentation of the index case, an additional 11 individuals with confirmed or probable acute Q fever and a suspected link to Community A were identified through local public health surveillance. An unusual increase in the number of positive laboratory results from two adjacent counties led to the initial recognition of a possible outbreak by public health authorities. The 12 cases of acute Q fever within a 5-month period compared to an average of three (range 1–5) cases reported annually throughout the state of Missouri over the past 5 years. Cases identified among rehabilitation program participants generated particular concern because risk factors that predispose to chronic Q fever, such as cardiac valvulopathy and prior endocarditis, may be more prevalent among recovering injection drug users. In December 2013, an investigation was undertaken to determine the extent and epidemiology of *C. burnetii* infection among humans and animals in Community A, identify persons at risk for chronic Q fever, and provide community-specific recommendations for prevention and control.

METHODS

Setting. Community A, located in rural Missouri, is self-contained and has a population estimated around 500 persons, including 343 documented employees, of which

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276 (81%) are male. Men, women, and children of all ages reside in the community, but complete demographic information was unavailable. Community A contains crop fields as well as large-scale cow and goat dairy herds, numbering approximately 8,900 cows and 2,000 goats. The community-affiliated residential substance abuse rehabilitation program hosts approximately 120–135 male participants at any given time, and working with ruminants (defined as cows or goats in this investigation) on the dairy operation is part of the treatment plan for most participants. Many graduates of the rehabilitation program continue to reside in the community with their families.

Human investigation. To determine *C. burnetii* seroprevalence, potential risk factors for infection, and the prevalence of predisposing conditions for chronic Q fever among community members, we recruited participants from Community A from December 11–17, 2013. Participants had blood drawn for *C. burnetii* serologic testing and were interviewed using a standardized questionnaire. Recruitment methods included electronic notices on the community intranet, community e-mail announcements, a community bulletin board notice, and word-of-mouth. The questionnaire collected information on demographics, work-related activities, animal exposures, dairy product consumption, febrile illness history, and medical conditions predisposing to chronic Q fever. A recall period of June 1, 2013, through the date of interview was used for questions about exposures and febrile illness history.

Animal and environmental investigations. Cow and goat specimens were collected and tested to evaluate for *C. burnetii* shedding among the herds. Milk was collected into sterile conical vials from representative samples of the cow and goat herds. Cow milk was sampled from milking carousels and parlors as follows: two cows per rotation of the 60-stall carousel; one cow per rotation of the 20-stall carousel; and one cow per every 16 in the 16-stall parlor. Additionally, one filter from the bulk milk tank at each of the three cow milking sites was collected. In the goat dairy, bulk milk samples representing 24 goats per sample were collected from the bulk tank during each milking cycle, and one bulk milk tank filter was collected. Vaginal swabs were collected from peri-parturient cows and goats. Fecal swabs were collected from male goats.

Environmental sampling was performed to assess the presence and locations of environmental contamination with *C. burnetii*. Environmental samples were collected using convenience methodology targeting high risk areas and specimens, including birthing pens, newborn housing areas, compost piles (consisting of cow and goat manure, bedding, animal carcasses, and placentas), boot storage spaces, an animal carcass pit, a stillborn calf, and a cow placenta. Bulk samples, such as bedding and soil, were collected into sterile conical vials where possible; otherwise sterile swabs were used.[5]

Laboratory methods. Human serum specimens were screened for *C. burnetii* phase II IgG using the indirect immunofluorescence antibody assay (IFA) with a detection cutoff titer of 32. If positive at or above this cutoff, endpoint titers for IgG phase I and phase II antibodies to *C. burnetii* were determined. Animal and environmental samples were tested for the presence of *C. burnetii* nucleic acid using polymerase chain reaction (PCR) targeting the IS*1111* insertion element.[12,13]

Investigation case definitions. For analysis, we defined a case as an individual with an epidemiologic link to Community A between June 1 and December 17, 2013, and a *C. burnetii* phase II IgG titer ≥ 128 by IFA from a commercial, state public health, or Centers for Disease Control and Prevention laboratory.[1] An individual was classified as a non-case if serum screened negative by IFA for *C. burnetii* phase II IgG (i.e., titer < 32). Low-positive titers (i.e., titer ≥ 32 and < 128) were not classified as cases or non-cases.

Data analysis. Data were entered into a Microsoft Access (Microsoft Corp., Redmond, WA) database. Analyses were done using STATA, version 12 (STATACorp, College Station, TX) statistical software. Descriptive statistics are presented as proportions, medians, and ranges. Pearson's χ^2 test and Fisher's exact test were used to compare categorical data. Risk ratios and 95% confidence intervals were calculated when appropriate. All *P* values were 2-sided and were evaluated for statistical significance at the 0.05 significance level. Data from individuals with low-positive titers were excluded from risk factor analysis.

RESULTS

Human investigation. In all, 135 individuals participated in a standardized interview and *C. burnetii* serologic testing, including the 12 previously identified Q fever cases. Forty-seven (35%) of 135 met the investigation case definition. Fifty-four (40%) were classified as non-cases. Thirty-four (25%) had low-positive titers. Overall, 81 (60%) participants demonstrated *C. burnetii* seropositivity at a titer ≥ 32. The median age of cases was 38 (range 10–74) years, and 38 (81%) were male (Table 1). Of 47 cases, 24 (51%) reported a febrile illness between June 1 and December 17, 2013. Common symptoms associated with a recent febrile illness among cases were chills, fatigue, decreased appetite, myalgia, and arthralgia.

Of 47 cases, 40 (85%) reported contact with or close proximity to ruminants (contact includes direct and indirect contact, including contact with ruminant excreta or birth products). Regular, job-related ruminant contact was reported by 32 (68%) cases. Eight (17%) cases reported only casual or indirect contact such as visits to the cow or goat dairy or occasional petting of goats or cows. Five (11%) cases reported no exposure to ruminants since June 1, 2013, but reported having household members who had regular ruminant contact. Two (4%) cases

TABLE 1
Demographics of individuals tested for *Coxiella burnetii* infection, Community A, Missouri, December 2013

	Total, *N* = 135 *n* (%)	Cases, *N* = 47 *n* (%)	Non-cases, *N* = 54 *n* (%)	Low-positive titer, *N* = 34 *n* (%)
Male	94 (70)	38 (81)	31 (57)	25 (74)
Age, median (range) years	35 (9–74)	38 (10–74)	32 (9–67)	35 (18–66)
Residence				
Rehabilitation center	44 (33)	18 (38)	13 (24)	13 (38)
Other residence within community	79 (59)	26 (55)	36 (67)	17 (50)
Residence outside community	12 (9)	3 (6)	5 (9)	4 (12)

had no identified risk factors other than living and working in Community A. The risk of being a case was 2.7 times greater among those with ruminant contact compared with those without (95% confidence interval [CI]: 1.3–5.3) (Table 2). Among those not reporting ruminant contact, those with household members who had ruminant contact were at greater risk of being a case (relative risk [RR] 4.8, 95% CI: 1.1–20.7).

Having worked in the goat dairy or in the environmental department since June 1, 2013, was significantly associated with increased risk of *C. burnetii* infection compared with those who had not worked in these jobs (RR 2.0, 95% CI: 1.3–2.9, and RR 1.9, 95% CI: 1.3–2.8, respectively) (Table 2). Of the nine participants who reported working at the goat dairy, six (67%) were cases, two (22%) had low-positive titers, and one (11%) was a non-case. Questions about specific goat exposures revealed that individuals with frequent goat interactions had significantly increased risk of being a case (Table 2). Having worked in any capacity in the cow dairy since June 1, 2013, was not associated with increased risk (RR 1.0, 95% CI: 0.6–1.7). However, certain cow interactions, including

TABLE 2
Association of exposures with case status, Community A, Missouri, June 1–December 17, 2013

	Exposed			Unexposed			
Risk factor	Cases no.	Total no.	%	Cases no.	Total no.	%	Relative risk (95% CI)
Job description*							
Cow dairy	12	25	48	35	76	46	1.0 (0.6, 1.7)
Goat dairy	6	7	86	41	94	44	2.0 (1.3, 2.9)
Construction	3	9	33	44	92	48	0.7 (0.3, 1.8)
Environmental†	8	10	80	39	91	43	1.9 (1.3, 2.8)
Food services	6	8	75	41	93	44	1.7 (1.1, 2.7)
Education/management/administration	6	19	32	41	82	50	0.6 (0.3, 1.3)
Student	3	11	27	44	90	49	0.6 (0.2, 1.5)
Goat interactions‡							
Goat contact or close proximity	28	44	64	19	57	33	1.9 (1.2, 2.9)
Milking	4	4	100	43	97	44	2.3 (1.8, 2.8)
Feeding/watering	5	5	100	42	96	44	2.3 (1.8, 2.9)
Cleaning holding areas	6	6	100	41	95	43	2.3 (1.8, 2.9)
Removing/handling manure	10	11	91	37	90	41	2.2 (1.6, 3.0)
Replacing bedding	4	4	100	43	97	44	2.3 (1.8, 2.8)
Veterinary care	4	4	100	43	97	44	2.3 (1.8, 2.8)
Help with or observe a birth	4	4	100	43	97	44	2.3 (1.8, 2.8)
Direct contact with newborn kid	4	5	80	43	96	45	1.8 (1.1, 2.9)
Direct contact with dead goat	5	5	100	42	96	44	2.3 (1.8, 2.9)
Direct contact with birth products	6	6	100	41	95	43	2.3 (1.8, 2.9)
Cow interactions‡							
Cow contact or close proximity	28	50	56	19	51	37	1.5 (1.0, 2.3)
Milking	10	21	48	33	73	45	1.1 (0.6, 1.8)
Feeding/watering	9	14	64	38	86	44	1.5 (0.9, 2.3)
Cleaning holding areas	15	24	63	32	76	42	1.5 (1.0, 2.2)
Removing/handling manure	20	32	63	27	68	40	1.6 (1.1, 2.3)
Replacing bedding	11	15	73	36	85	42	1.7 (1.2, 2.6)
Veterinary care	10	11	91	37	89	42	2.2 (1.6, 3.0)
Help with or observe a birth	9	9	100	38	90	42	2.4 (1.9, 3.0)
Direct contact with newborn calf	11	16	69	36	84	43	1.6 (1.1, 2.4)
Direct contact with dead cow	12	15	80	35	85	41	1.9 (1.4, 2.8)
Direct contact with birth products	13	15	87	34	85	40	2.2 (1.6, 3.0)
Other goat and cow exposures							
Known exposure to:							
Animal that had an abortion or stillbirth§	13	17	76	34	84	40	1.9 (1.3, 2.7)
Animal that had newborn death§	15	19	79	32	82	39	2.0 (1.4, 2.9)
Animal that had weak newborn§	15	20	75	32	81	40	1.9 (1.3, 2.7)
Dispose of animal carcasses	14	21	67	33	79	42	1.6 (1.1, 2.4)
Clean animal holding areas	18	28	64	29	73	40	1.6 (1.1, 2.4)
Clean animal holding areas after birthing	7	9	78	40	92	43	1.8 (1.2, 2.7)
Contact or work with ruminant excreta	25	38	66	22	63	35	1.9 (1.3, 2.8)
Contact or close proximity with ruminants‖	40	69	58	7	32	22	2.7 (1.3, 5.3)
Household exposures							
Household member with cow or goat contact‖ (among those with no personal exposure)	5	6	45	2	19	10	4.8 (1.1, 20.7)
Work boots brought into home	12	13	92	19	41	46	2.0 (1.4, 2.9)
Work clothing laundered at home	17	25	68	20	36	56	1.2 (0.8, 1.8)
Miscellaneous exposures							
Consume raw dairy products	9	14	64	38	87	44	1.5 (0.9, 2.3)
Consume pasteurized goat cheese	23	42	55	24	56	43	1.3 (0.8, 1.9)

CI = confidence interval.
*More than one job may have been reported in the past 6 months; only jobs reported by ≥ 5 persons were analyzed.
†Includes work with animal waste, compost management, land application of compost, and spray irrigation from manure lagoons.
‡Ordinal independent variables describing frequency were dichotomized to 'never or hardly ever' and 'daily, several times per week or several times per month'.
§Answers of "don't know" or "not applicable" classified as "no" for analysis.
‖Contact includes work with ruminant excreta.

frequently handling cow manure, changing cow bedding, giving medicines or medical care to cows, helping with or observing the birth of a calf, direct contact with cow birth products, and direct contact with a dead cow were associated with being a case (Table 2). Known exposures to animals that had experienced an abortion or stillbirth, death of a newborn, or had a weak newborn were also significantly associated with increased risk (RR 1.9, 95% CI: 1.3–2.7; RR 2.0, 95% CI: 1.4– 2.9; RR 1.9, 95% CI: 1.3–2.7, respectively).

Among individuals who reported ruminant contact or had a household member with ruminant contact, 13 (24%) of 54 reported work boots were routinely brought inside the home. Compared with those who either left their work boots at the worksite or left them outside the home, bringing boots inside the home was associated with an increased risk of being a case (RR 2.0, 95% CI: 1.4–2.9). Laundering work clothing at home compared with laundering outside the home (including sending it directly from the worksite or a common bin to a commercial laundry facility) was not associated with significantly different risk (RR 1.2, 95% CI: 0.8–1.8). Raw dairy product consumption was reported by 23 (17%) of 135 participants and was not significantly associated with being a case (RR 1.5, 95% CI: 0.9–2.3).

Six (13%) of 47 cases reported ≥ 1 known risk factor for development of chronic Q fever (Table 3). A history of injection drug use (IDU) was reported by 26 (20%) of 130 respondents, including 10 (22%) of 45 responding cases. Nine individuals had phase I IgG titers ≥ 1024, meeting laboratory criteria for chronic Q fever. Diagnosis of chronic Q fever requires demonstration of organ involvement, in addition to supportive laboratory criteria.[1] Determinations of chronic Q fever status could not be made by the investigation team because clinical assessments to determine organ involvement were not performed as part of the outbreak investigation.

Animal and environmental investigations. *Coxiella burnetii* PCR results were positive for three (2%) of 132 cow milk samples, two (7%) of 28 cow vaginal swabs, five (17%) of 29 goat milk samples, eight (26%) of 31 goat vaginal swabs, and all four (100%) bulk tank milk filters (three from the cow dairy and one from the goat dairy). All five fecal swabs from male goats were negative.

Interviews with farm managers revealed no recent abortion storms or health concerns among the goat herd. Kidding typically occurred during September–October and March–April, but births also occurred at other times throughout the year. Among the cow herd, increased abortions were noted in spring and early summer 2013, and a cause other than coxiellosis was determined by a local veterinarian. Cow abortions had returned to baseline levels by the time we began our investigation, and we were not able to confirm the cause of the earlier increase. Cow births were reported to occur year-round.

Of 26 environmental samples, eight (31%) were positive for *C. burnetii* DNA. Positive samples included those taken from goat birthing pens, manure in a pit where animal carcasses are disposed, cow placenta, and the goat dairy boot locker. The highest levels of *C. burnetii* DNA were detected in the goat birthing pens and in manure from the animal carcass pit.

DISCUSSION

This investigation in response to a cluster of Q fever cases in Community A identified 47 individuals meeting the investigation case definition and a seroprevalence to *C. burnetii* of 60% among participating community members. The high seroprevalence to *C. burnetii* among Community A participants compares to an estimated seroprevalence of 3% among the general U.S. population and 13% among U.S. agricultural workers in a non-outbreak setting.[14,15] We documented active shedding of *C. burnetii* among the goat and cow dairy herds as well as environmental contamination on the farm. Contact with or close proximity to ruminants was frequent among cases and was significantly associated with being a case. Cases were also identified among individuals without ruminant contact; this finding could be related to windborne spread or tracking of *C. burnetii* on fomites from the dairies to other areas in the community. The prevalence of past injection drug use among individuals in Community A requires particular attention, and follow-up for the presence of valvulopathy and monitoring for development of chronic Q fever will be important among this population.

The highest risk jobs for *C. burnetii* infection were goat dairy and environmental work. Eighty percent of participating environmental workers were cases. Environmental work encompassed spray irrigation from manure lagoons and compost management, including land application of composted material. Land application of goat manure following kidding

TABLE 3
Risk factors for chronic Q fever, Community A, Missouri, December, 2013

	Total, $N = 135$ n (%)	Cases, $N = 47$ n (%)	Low positive titers, $N = 34$ n (%)
Risk factor for chronic Q fever			
Any risk factor*	18 (13)	6 (13)	4 (12)
Heart valve abnormality	10 (7)	5 (11)	2 (6)
Heart murmur	10 (7)	1 (2)	3 (9)
Aneurysm	1 (1)	0	0
Synthetic vascular graft†	0	0	0
Other medical history			
History of injection drug use	26/130 (20)	10/45 (22)	5 (15)
Smoking in past 6 months‡	15 (11)	5 (11)	3 (9)
Current pregnancy	1 (1)	0	0
Pregnancy in past 6 months	4 (3)	1 (2)	1 (3)
Immunosuppressive medications	4 (3)	1 (2)	2 (6)

*More than one risk factor may have been reported by an individual, but those with >1 risk factor were counted only once in the total.
†Coronary artery stents or vein grafts associated with coronary artery bypass not included.
‡Smoking was not permitted in Community A; therefore, so few persons reported having smoked in the past 6 months.

season has been associated temporally and spatially with human Q fever in the Netherlands[16]; however, proper composting for at least 90 days should decrease the burden of *C. burnetii* and reduce risk of transmission.[17] Manure lagoons rely on anaerobic digestion, and commonly encountered digestion temperatures may not be sufficient to inactivate some spore-forming organisms[18]; however, it is not known whether the spore-like form of *C. burnetii* may remain viable through this process. In-depth examination of compost and manure lagoon practices was beyond the scope of our investigation, but determining the role of these processes, if any, in *C. burnetii* transmission among environmental workers and within Community A will be important.

Most (68%) cases in this investigation reported regular occupational exposure to ruminants; however, cases were also identified among individuals with infrequent, casual, or no ruminant contact. Q fever in the absence of reported ruminant exposure is commonly observed in passive surveillance in the United States, with the majority of cases reported from 2000 to 2012 having no known exposure to goats, cows, or sheep.[19] Windborne spread of infected particulates is a recognized mode of exposure implicated in several Q fever outbreaks.[7–9,20] Abnormally dry and drought conditions in the region of Missouri where Community A is located during Summer 2013 could have been especially conducive to windborne spread of *C. burnetii* in dust.[10,21–24] Additionally, uncovered compost piles and transport of uncovered manure or bedding were reported or observed in Community A. These practices can result in aerosolization of *C. burnetii*, particularly on windy days, putting environmental workers and the larger community at risk. This concept is exemplified by previous Q fever outbreaks documented along the routes of vehicles carrying *C. burnetii*-contaminated straw, manure, and dust from farms.[25] Covered compost piles and covered transport of manure, bedding, and carcasses were recommended.[17]

Individuals residing in the same household as farm workers who had regular ruminant contact were at increased risk of being a case. Fomite transmission within the household could explain this finding. *C. burnetii* transmission via aerosols generated from contaminated clothing and other fomites has been described.[26,27] In Community A, bringing work boots worn on the farm inside of one's residence was associated with increased risk of being a case among workers and their household members. Boot or shoe contamination and related foot traffic could result in *C. burnetii* introduction into areas beyond the farm as has been proposed as a source of *C. burnetii* contamination within homes in other Q fever outbreaks.[5] Decontamination of boots at the work site or dedicated work boots left on site was recommended to reduce the spread of *C. burnetii* via foot traffic.[5,17]

A unique feature of the investigation setting was the residential substance abuse rehabilitation program. Approximately 20% of all participants and 22% of cases reported past IDU. Valvular abnormalities are a well-established risk factor for chronic Q fever endocarditis and may be more prevalent among injection drug users.[28–33] Additionally, injection drug users are at increased risk for infective endocarditis caused by organisms other than *C. burnetii*, which can result in valvular damage and increased risk for recurrent endocarditis.[34–36] Concerns about IDU history among community members included the potential for asymptomatic, undetected valvular abnormalities at a rate higher than that of a non-IDU population and the possibility for future valvular damage in the setting of IDU relapse, either of which could increase the risk for Q fever endocarditis.

Patients with risk factors for development of chronic Q fever warrant close clinical and serologic follow-up for at least 2 years after acute infection. Monitoring at 3-, 6-, 12-, 18-, and 24-month intervals is recommended to evaluate for progression to chronic Q fever.[1] Patient education is important in ensuring awareness of chronic Q fever symptoms and the ability to relay a history of Q fever to health care providers. We provided letters to participants explaining their serology test results. The community physician was given detailed guidance on clinical follow-up of Q fever patients, including the need for clinical assessments and serologic follow-up in patients meeting laboratory criteria for chronic Q fever. We are not aware of any diagnosed cases of chronic Q fever among community members.

This investigation was subject to limitations. Participants voluntarily presented during a 7-day testing campaign or were diagnosed with acute Q fever by the community physician; therefore, seroprevalence estimates are based on a convenience sample rather than a systematic, population-based assessment. Given the approximate community population, we estimate testing was performed for approximately 25–30% of the community. We are unable to compare characteristics of community members who did and did not present for testing. Additionally, timing of *C. burnetii* infection cannot be determined based on a single serologic result, and nearly all participants had only a single serum sample tested. However, seroconversion in three individuals from June to August 2013 and very high phase II IgG titers (≥ 4096) in eight community members support recent *C. burnetii* transmission within Community A. Risk factor analysis was limited by common exposures among community members and relatively small numbers, making it difficult to associate seropositivity with independent exposures. Recall bias is possible given the 6-month recall period for exposures. Responses were missing or unclear for some open-ended questions, including those about handling of work boots and clothing, which resulted in reduced numbers for analysis. We were unable to obtain human *C. burnetii* isolates; therefore, we could not evaluate genotypes of human compared with animal isolates.

Although elimination of *C. burnetii* from the herds and the farm environment might not be a realistic goal, education and changes in farm practices can help reduce human transmission. Community specific recommendations to reduce the risk of animal-to-human transmission of *C. burnetii* were provided.[17,37] Patient and health care provider education aimed at early recognition, treatment, and close follow-up of those at high risk for chronic Q fever is important to reduce morbidity and mortality.[1] Health care providers and public health professionals should be aware of the risk for Q fever among those who work with or live in close proximity to ruminants.

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