

although its mechanism of action against HCV and HEV is uncertain. Data are limited on the use of ribavirin in patients with chronic hepatitis E and hematologic malignancies (10). The outcome for our patient suggests that ribavirin might be useful for treating hepatitis E in such patients.

In conclusion, all patients with hepatitis of unknown origin should be tested for HEV, in particular, immunocompromised patients, because they are at risk of acquiring chronic hepatitis and having an adverse outcome. Ribavirin appears to be efficacious in treating hepatitis E and should be considered for any immunocompromised person who has viremia 3 months after acute infection.

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References

- Romanò L, Paladini S, Tagliacarne C, Canuti M, Bianchi S, Zanetti AR. Hepatitis E in Italy: a long-term prospective study. *J Hepatol*. 2011;54:34–40. <http://dx.doi.org/10.1016/j.jhep.2010.06.017>
- Martelli F, Caprioli A, Zengarini M, Marata A, Fiegna C, Di Bartolo I, et al. Detection of hepatitis E virus (HEV) in a demographic managed wild boar (*Sus scrofa scrofa*) population in Italy. *Vet Microbiol*. 2008;126:74–81. <http://dx.doi.org/10.1016/j.vetmic.2007.07.004>
- Emerson SU, Nguyen HT, Torian U, Mather K, Firth AE. An essential RNA element resides in a central region of hepatitis E virus ORF2. *J Gen Virol*. 2013;94:1468–76. <http://dx.doi.org/10.1099/vir.0.051870-0>
- Kamar N, Rostaing L, Legrand-Abravanel F, Izopet J. How should hepatitis E infection be defined in organ transplant recipients? *Am J Transplant*. 2013;13:1935–6. <http://dx.doi.org/10.1111/ajt.12253>
- Kamar N, Bendall R, Legrand-Abravanel F, Xia NS, Ijaz S, Izopet J, et al. Hepatitis E. *Lancet*. 2012;379:2477–88. [http://dx.doi.org/10.1016/S0140-6736\(11\)61849-7](http://dx.doi.org/10.1016/S0140-6736(11)61849-7)
- Pfefferle S, Frickmann H, Gabriel M, Schmitz N, Gunther S, Schmidt-Chanasit J. Fatal course of an autochthonous hepatitis E virus infection in a patient with leukemia in Germany. *Infection*. 2012;40:451–4. <http://dx.doi.org/10.1007/s15010-011-0220-7>
- le Coutre P, Meisel H, Hofmann J, Rocken C, Vuong GL, Neuburger S, et al. Reactivation of hepatitis E infection in a patient with acute lymphoblastic leukaemia after allogeneic stem cell transplantation. *Gut*. 2009;58:699–702. <http://dx.doi.org/10.1136/gut.2008.165571>
- Gerolami R, Borentain P, Raissouni F, Motte A, Solas C, Colson P. Treatment of severe acute hepatitis E by ribavirin. *J Clin Virol*. 2011;52:60–2. <http://dx.doi.org/10.1016/j.jcv.2011.06.004>
- Mallet V, Nicand E, Sultanik P, Chakvetadze C, Tessé S, Thervet E, et al. Case reports of ribavirin treatment for chronic hepatitis E. *Ann Intern Med*. 2010;153:85–9. <http://dx.doi.org/10.7326/0003-4819-153-2-201007200-00257>
- Alric L, Bonnet D, Beynes-Rauzy O, Izopet J, Kamar N. Definitive clearance of a chronic hepatitis E virus infection with ribavirin treatment. *Am J Gastroenterol*. 2011;106:1562–3. <http://dx.doi.org/10.1038/ajg.2011.158>

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Letters

Letters commenting on recent articles as well as letters reporting cases, outbreaks, or original research are welcome. Letters commenting on articles should contain no more than 300 words and 5 references; they are more likely to be published if submitted within 4 weeks of the original article's publication. Letters reporting cases, outbreaks, or original research should contain no more than 800 words and 10 references. They may have 1 Figure or Table and should not be divided into sections. All letters should contain material not previously published and include a word count.

Q Fever Surveillance in Ruminants, Thailand, 2012

To the Editor: Two cases of fatal endocarditis in Khon Kaen Province in northeastern Thailand were found to be caused by *Coxiella burnetii* (1). Although *C. burnetii* is known to be present in many countries, including in Thailand (2), human infection is more commonly associated with sheep and goats, possibly because these animals shed the organism more frequently in vaginal secretions and feces than do large ruminants (3).

Surveillance for Q fever, which is caused by *C. burnetii*, in livestock is currently based primarily on serologic or PCR testing of milk (4). However, problems in estimating prevalence include serologic assay insensitivity (5,6) or unavailability of milk from nondairy animals.

For diagnosis of Q fever, the placenta of the animal is commonly tested, but testing is usually conducted only when abortions occur, which is only likely when uninfected animals first encounter *C. burnetii*. Therefore, this approach might underestimate true organism distribution in a disease-endemic area (7). In addition, nearly all abortion storms have occurred in sheep or goats, which are rare in Thailand. Ruminant abortion is rarely reported to veterinary authorities in Thailand.

Comparison of paired colostrum and placental samples from sheep showed that *C. burnetii* was found more frequently in placental samples (8), which suggested that the placenta is a better sample than milk for surveillance purposes. Also, a placenta may be more useful because it is more likely to contaminate the farm environment. Milk is an unlikely source of Q fever in adult persons because it is seldom consumed by adults in Thailand.

The ideal surveillance strategy would include all relevant samples (serum, milk, and products of conception, both normal and abnormal). However, in practice, cost and logistical limitations dictate refinement of sampling. *C. burnetii* is frequently detected in normal ruminant placentas, but offspring are apparently not affected (9). We report that surveillance of normal placentas can provide useful surveillance data.

To test this hypothesis, in 2012 we asked local veterinarians in selected subdistricts in Thailand to contact farmers at their convenience to request that the veterinarians be alerted when a ruminant gave birth. Only grossly normal placentae from normal births of apparently healthy offspring were sampled. Cotyledonary (preferred) or intercotyledonary chorioallantoic tissue was obtained, chilled, and shipped cold to the National Institute for Animal Health (Bangkok, Thailand) for analysis. Tissue was ground, extracted, and analyzed by PCR for IS1111 of *C. burnetii* in a Light Cycler 2.0 Apparatus (Roche, Basel, Switzerland) as described (10). To minimize false-positive results, we repeated the PCR with a separate portion of tissue from the original sample. Samples were considered positive if the PCR had a cycle threshold <35 for each assay, or suspected of being positive if this occurred in 1 of 2 separate assays.

Results indicate a high frequency of *C. burnetii* infections in some provinces (Table), which roughly match locations where fatal human cases of endocarditis have occurred (Figure, Appendix, wwwnc.cdc.gov/EID/article/19/12/13-0624-F1.htm). It is common practice among the agrarian population in Thailand to consume ruminant placenta. Although this tissue is reportedly cooked before consumption, the preparation process may result in environmental contamination sufficient to expose persons who were not in close contact with the infected animal.

This study demonstrates that sampling and PCR of grossly normal ruminant placenta is a viable stand-alone approach for surveillance of *C. burnetii* that might enable the generation, at a minimal cost, of a highly detailed map showing areas where humans and animals are at risk for Q fever. The results indicate that *C. burnetii* is highly endemic in the study region. However, in light of the extreme rarity of serious complications in human infections and lack of any indication of a serious effect on animal production, these results do not indicate a need for veterinary control measures. Nonetheless, food safety practices should be addressed. It is essential that physicians monitoring patients with underlying heart valve conditions encourage such patients to seek diagnosis of any febrile illness so that appropriate treatment may be initiated to minimize risk for complications.

We report a novel approach to Q fever surveillance, which is potentially useful for countries such as Thailand, where subclinical ruminant infections are common. Our results also provide an initial indication of risk factors associated with recent cases of fatal Q fever endocarditis in Thailand. Follow-up research should include broader reservoir species surveillance, environmental surveillance, and comparison of genotypes of organisms found in ruminant placenta with those found in persons with endocarditis. These further efforts will result in clearer understanding of Q fever ecology and potential routes of human exposure.

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References

- Pachirat O, Fournier PE, Pussadhamma B, Taksinachanekij S, Lulitanond V, Baggett HC, et al. The first reported cases of Q fever endocarditis in Thailand. *Infectious Disease Reports*. 2012;4:e7. <http://dx.doi.org/10.4081/idr.2012.e7>
- Suputtamongkol Y, Rolain JM, Losuwanaruk K, Niwatayakul K, Suttinont C, Chierakul W, et al. Q fever in Thailand. *Emerg Infect Dis*. 2003;9:1186–7. <http://dx.doi.org/10.3201/eid0909.030086>
- Rodolakis A, Berri M, He'chard C, Caudron C, Souriau A, Bodier CC, et al. Comparison of *Coxiella burnetii* shedding in milk of dairy bovine, caprine, and ovine herds. *J Dairy Sci*. 2007;90:5352–60. <http://dx.doi.org/10.3168/jds.2006-815>
- López-Gatius F, Almeria S, Garcia-Ispuerto I. Serological screening for *Coxiella*

Table. PCR results for Q fever surveillance in ruminants, Thailand, 2012

Province (no. sites)	Animal	No. cases		
		Positive	Negative	Suspected
Chaiyapum (13)	Beef cattle	3	10	0
Chaiyapum (3)	Dairy cattle	1	0	2
Chaiyapum (1)	Goat	0	0	2
Chiang Mai (1)	Buffalo	0	2	2
Kalasin (1)	Goat	1	0	0
Khon Kaen (8)	Beef cattle	8	0	0
Khon Kaen (2)	Buffalo	2	0	0
Maha Sarakham (1)	Goat	2	6	0
Nakon Pathom (9)	Goat	1	9	0
Nakon Ratchasima (1)	Beef cattle	1	1	0
Nakon Ratchasima (17)	Dairy cattle	20	30	0
Nakon Ratchasima (3)	Goat	15	13	0
Prachuap Kiri Khan (9)	Dairy cattle	3	1	6
Ratchaburi (2)	Goat	1	9	0

- burnetii* infection and related reproductive performance in high producing dairy cows. *Res Vet Sci*. 2012;93:67–73. <http://dx.doi.org/10.1016/j.rvsc.2011.07.017>
5. Berri M, Souriau A, Crosby M, Rodolakis A. Shedding of *Coxiella burnetii* in ewes in two pregnancies following an episode of *Coxiella* abortion in a sheep flock. *Vet Microbiol*. 2002;85:55–60. [http://dx.doi.org/10.1016/S0378-1135\(01\)00480-1](http://dx.doi.org/10.1016/S0378-1135(01)00480-1)
 6. Böttcher J, Vossen A, Janowitz B, Alex M, Gangl A, Randt A, et al. Insights into the dynamics of endemic *Coxiella burnetii* infection in cattle by application of phase-specific ELISAs in an infected dairy herd. *Vet Microbiol*. 2011;151:291–300. <http://dx.doi.org/10.1016/j.vetmic.2011.03.007>
 7. Centers for Disease Control and Prevention. Notes from the field: Q fever outbreak associated with goat farms—Washington and Montana, 2011. *MMWR Morb Mortal Wkly Rep*. 2011;60:1393.
 8. Runge M, Binder A, Schotte U, Ganter M. Investigations concerning the prevalence of *Coxiella burnetii* and *Chlamydia abortus* in sheep in correlation with management systems and abortion rate in Lower Saxony in 2004. *Berl Munch Tierarztl Wochenschr*. 2012;125:138–43.
 9. Hansen MS, Rodolakis A, Cochonneau D, Agger JF, Christoffersen AB, Jensen TK, et al. *Coxiella burnetii* associated placental lesions and infection level in parturient cows. *Vet J*. 2011;190:e135–9. Epub 2011 Feb 2. <http://dx.doi.org/10.1016/j.tvjl.2010.12.021>
 10. Christensen DR, Hartman LJ, Loveless BM, Frye MS, Shipley MA, Bridge DL, et al. Detection of biological threat agents by real-time PCR: comparison of assay performance on the R.A.P.I.D., the Light-Cycler, and the Smart Cycler platforms. *Clin Chem*. 2006;52:141–5. <http://dx.doi.org/10.1373/clinchem.2005.052522>

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Treponemal Infection in Nonhuman Primates as Possible Reservoir for Human Yaws

To the Editor: In 2012, the World Health Organization launched plans for a second campaign to eradicate the neglected tropical disease, yaws (*1*). The first campaign, conducted during the mid-20th century, was tremendously successful in terms of treatment and reduced the number of cases by 95%. However, it failed to eradicate the disease, and when local efforts to prevent new cases proved insufficient, yaws resurged in some areas. Comments on the new yaws eradication campaign have emphasized the need for sustained support and resources. Here we draw attention to an additional concern that could impede yaws eradication efforts.

The success of any eradication campaign depends on the absence of a nonhuman reservoir. Smallpox had no known animal reservoir, and polio and dracunculiasis (guinea worm disease), which are currently the focus of the World Health Organization eradication campaigns, also have none. By contrast, compelling evidence suggests that yaws exists in wild nonhuman primate populations residing in regions where humans are also infected (Figure).

The subspecies of the bacterium *Treponema pallidum* that cause the non-sexually transmitted diseases yaws (subsp. *pertenue* infection) and endemic syphilis (subsp. *endemicum* infection) and the sexually transmitted infection syphilis (subsp. *pallidum*) are close relatives. The 3 diseases cannot be distinguished serologically. Instead, the diseases they cause are usually differentiated by clinical characteristics and geographic distribution. Whereas syphilis is a venereal disease with a worldwide distribution,

yaws primarily affects children in hot and humid areas of Africa and Asia, and endemic syphilis occurs in arid regions. Because methods available to differentiate between the *T. pallidum* subspecies were unavailable in the past, prevalence data for yaws were sometimes vague and inaccurate. Recently, molecular tests capable of distinguishing between the subspecies by using single nucleotide polymorphisms have been developed (*2,3*). These tests have enabled us to learn more about the *T. pallidum* strains that infect wild nonhuman primates.

During the 1960s, researchers reported that many baboons in West Africa were seropositive for treponemal infection (*4*). Since then, high levels of infection have been documented in other monkey species in West Africa and in great apes (*5*). Recently, we documented *T. pallidum* infection in olive baboons (*Papio anubis*) at Lake Manyara National Park in Tanzania (*6*). In West Africa, clinical signs of infection in nonhuman primates are usually mild, if present at all, consisting of small lesions around the muzzle, eyelids, and armpits (*4*). A recent survey in 2013 at Parc National du Niokolo-Koba, Senegal, revealed *T. pallidum* antibodies in Guinea baboons (*P. papio*) with no signs of infection (S. Knauf et al, unpub. data). By contrast, severe manifestations resembling tertiary-stage yaws have been reported in wild gorillas (*5*). In terms of genetic distance, studies thus far indicate that the organisms infecting baboons in West and East Africa closely resemble *T. pallidum* subsp. *pertenue*, the agent responsible for yaws in humans (*2,7*). In fact, the genome sequence of a *T. pallidum* strain collected from a baboon in Guinea indicates that it should be considered a *T. pallidum* subsp. *pertenue* strain (*8*). Infection has been confirmed by serologic tests in a variety of nonhuman primate species in the yaws belt of Africa and by PCR in baboons from East and West Africa (Figure).

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