

of the *Rickettsia* spp. we identified. As shown (2), multilocus analysis with *ompA-ompB* sequences was highly informative about the phylogenetic relationship between *Rickettsia* spp. and *R. conorii* strains (Figure, panel B).

In Sicily, *R. conorii* Malish strain has been characterized in MSF patients (4), and *R. slovaca* DNA was identified in ixodid ticks (5). However, to our knowledge, *R. slovaca* in humans in Sicily and *R. conorii* Indian tick typhus strain infection in Sicily and Europe have not been reported. The only previous report outside India and Pakistan was documented in a traveler with severe clinical manifestations in France (10). Differences were not observed between *R. conorii* Indian tick typhus strain and *R. slovaca*-infected patients. Both patients had similar clinical symptoms compatible with MSF; in both, only IgM for rickettsiae was detected at hospital admission, but IgM and IgG were detected during convalescence. Tache noire were detected in the neck and right arm of patients with *R. conorii* Indian tick typhus strain and *R. slovaca*, respectively.

These results demonstrated that new rickettsiae, such as *R. conorii* Indian tick typhus strain, of public health relevance are emerging in Europe. The widespread distribution of tick vectors in Europe and the transtadial and transovarial transmission of the pathogen in ticks might favor transmission to humans.

This research was supported by the Italian Ministry of Health, project IZSSI 08/08.

**Alessandra Torina,  
Isabel G. Fernández de Mera,  
Angelina Alongi,  
Atilio J. Mangold,  
Valeria Blanda,  
Francesco Scarlata,  
Vincenzo Di Marco,  
and José de la Fuente**

Author affiliations: Istituto Zooprofilattico Sperimentale della Sicilia, Palermo, Sicily, Italy (A. Torina, A. Alongi, V. Blanda, V. Di Marco); University of Palermo, Palermo (V. Blanda); Instituto de Investigación en Recursos Cinegéticos (IREC-CSIC-UCLM-JCCM) Ciudad Real, Spain (I.G. Fernández de Mera, J. de la Fuente); Universidad Complutense de Madrid, Madrid, Spain (I.G. Fernández de Mera); Estación Experimental Agropecuaria Rafaela, Santa Fe, Argentina (A.J. Mangold); Istituto di Patologia Infettiva e Virologia dell'Università di Palermo, Palermo (F. Scarlata); and Oklahoma State University, Stillwater, Oklahoma, USA (J. de la Fuente)

DOI: <http://dx.doi.org/10.3201/eid1806.110966>

## References

- Nicholson WL, Allen KE, McQuiston JH, Breitschwerdt EB, Little SE. The increasing recognition of rickettsial pathogens in dogs and people. *Trends Parasitol.* 2010;26:205–12. <http://dx.doi.org/10.1016/j.pt.2010.01.007>
- Zhu Y, Fournier PE, Ereemeeva M, Raoult D. Proposal to create subspecies of *Rickettsia conorii* based on multilocus sequence typing and an emended description of *Rickettsia conorii*. *BMC Microbiol.* 2005;5:11. <http://dx.doi.org/10.1186/1471-2180-5-11>
- Ciceroni L, Pinto A, Ciarrocchi S, Ciervo A. Current knowledge of rickettsial diseases in Italy. *Ann N Y Acad Sci.* 2006;1078:143–9. <http://dx.doi.org/10.1196/annals.1374.024>
- Giammanco GM, Vitale G, Mansueto S, Capra G, Caleca MP, Ammatuna P. Presence of *Rickettsia conorii* subsp. *israelensis*, the causative agent of Israeli spotted fever, in Sicily, Italy, ascertained in a retrospective study. *J Clin Microbiol.* 2005;43:6027–31. <http://dx.doi.org/10.1128/JCM.43.12.6027-6031.2005>
- Beninati T, Genchi C, Torina A, Caracappa S, Bandi C, Lo N. Rickettsiae in ixodid ticks, Sicily. *Emerg Infect Dis.* 2005;11:509–11. <http://dx.doi.org/10.3201/eid1103.040812>
- Tzianabos T, Anderson BE, McDade JE. Detection of *Rickettsia rickettsii* DNA in clinical specimens by using polymerase chain reaction technology. *J Clin Microbiol.* 1989;27:2866–8.
- Fernández de Mera IG, Zivkovic Z, Bolaños M, Carranza C, Pérez-Arellano JL, Gutiérrez C, et al. *Rickettsia massiliae* in the Canary Islands. *Emerg Infect Dis.* 2009;15:1869–70.
- Roux V, Fournier PE, Raoult D. Differentiation of spotted fever group rickettsiae by sequencing and analysis of restriction fragment length polymorphism of PCR-amplified DNA of the gene encoding the protein rOmpA. *J Clin Microbiol.* 1996;34:2058–65.
- Choi YJ, Jang WJ, Ryu JS, Lee SH, Park KH, Paik HS, et al. Spotted fever group and typhus group rickettsioses in humans, South Korea. *Emerg Infect Dis.* 2005;11:237–44. <http://dx.doi.org/10.3201/eid1102.040603>
- Parola P, Fenollar F, Badiaga S, Brouqui P, Raoult D. First documentation of *Rickettsia conorii* infection (strain Indian tick typhus) in a traveler. *Emerg Infect Dis.* 2001;7:909–10. <http://dx.doi.org/10.3201/eid0705.010527>

Address for correspondence: José de la Fuente, Instituto de Investigación en Recursos Cinegéticos IREC-CSIC-UCLM-JCCM, Ronda de Toledo s/n, 13005 Ciudad Real, Spain; email: [jose\\_delafuente@yahoo.com](mailto:jose_delafuente@yahoo.com)

## Detection of European Strain of *Echinococcus multilocularis* in North America

**To the Editor:** In 2009, an alveolar hydatid cyst, the intermediate stage of the cestode *Echinococcus multilocularis*, was detected in the liver of a dog from Quesnel, British Columbia (BC), Canada (1), 600 km west of the nearest known record of this parasite in central North America (Figure). Alveolar hydatid cysts normally occur in rodent intermediate hosts. However, humans can serve as aberrant intermediate hosts; cysts generally originate in the liver and, in about one third of cases, metastasize throughout the body (2). Detection of the larval stage of this pathogen in an unusual host in a new geographic region required application of multiple molecular epidemi-

ologic techniques to determine if this was range expansion of a native strain or introduction of a new strain of veterinary and public health concern.

Alveolar hydatid cyst material was surgically excised from the dog, frozen, and shipped to the Western College of Veterinary Medicine in Saskatoon, Saskatchewan. DNA was extracted by using the QIAGEN DNeasy Blood and Tissue Kit (QIAGEN, Inc., Valencia, CA, USA). PCR was performed by using primers for 4 mitochondrial loci: NADH dehydrogenase subunit 1 (*nad1*) and 2 (*nad2*), cytochrome b (*cob*), and cytochrome c oxidase subunit 1 (*cox1*) (3,4). Sequence of a 488-bp region of the *nad1* gene (GenBank accession no. JF751034) was 99%–100% identical to *E. multilocularis* sequences from Asia (AY389984) and Europe (AB668376). Sequence data for 91 of 112 positions at the *cox1* (partial), *cob1*, and *nad2* genes (GenBank accession nos. JF751033, JF751035, and JF751036) grouped with haplotypes from Europe (4); 2 nucleotide differences from the E4 haplotype in foxes in France and Belgium were found, and 1 additional nucleotide difference (position 663 in *cob1*) did not correspond to any of the haplotypes defined previously.

Two independent subsamples of cyst material were fixed in 70% ethanol and shipped to the University of Regina, Regina, Saskatchewan, for PCR using primers targeting microsatellite loci EmsB and NAK 1 (5,6). PCR products were sized with single-basepair resolution by using capillary electrophoresis on a DNA sequencer (Genome Lab GeXP; Beckman-Coulter, Fullerton, CA, USA). Peaks that had <15% of the amplitude of the highest peak were excluded from analysis. EmsB electrophoregrams from the 2 samples of cyst material were identical and displayed 10 peaks spanning 220–238 bp. Visually, the EmsB profile from the BC dog sample most closely matched European profile H

from foxes in west-central Europe (5). The BC dog sample was homozygous for the 198-bp allele at NAK 1, a genotype found in a variety of locations in Europe and Japan but not in North America, where the dominant genotype appears to be homozygous 192 (5).

Mitochondrial and microsatellite characterization showed that the genotype of *E. multilocularis* found in the BC dog was most similar to those described from west-central Europe. If the BC report represented a westward range expansion of a native North

American strain of *E. multilocularis*, it would most likely be the N2 strain, established in the North Central Region, which includes the 3 Canadian prairie provinces and 12 contiguous north central American states (4) (Figure). This case demonstrates the utility of molecular epidemiology for detecting incursion of foreign pathogens and tracing their origins, as well as the feasibility of using animal sentinels to detect the introduction of a disease of public health concern into a new area.

Because the dog had never left BC and cestode eggs were not de-

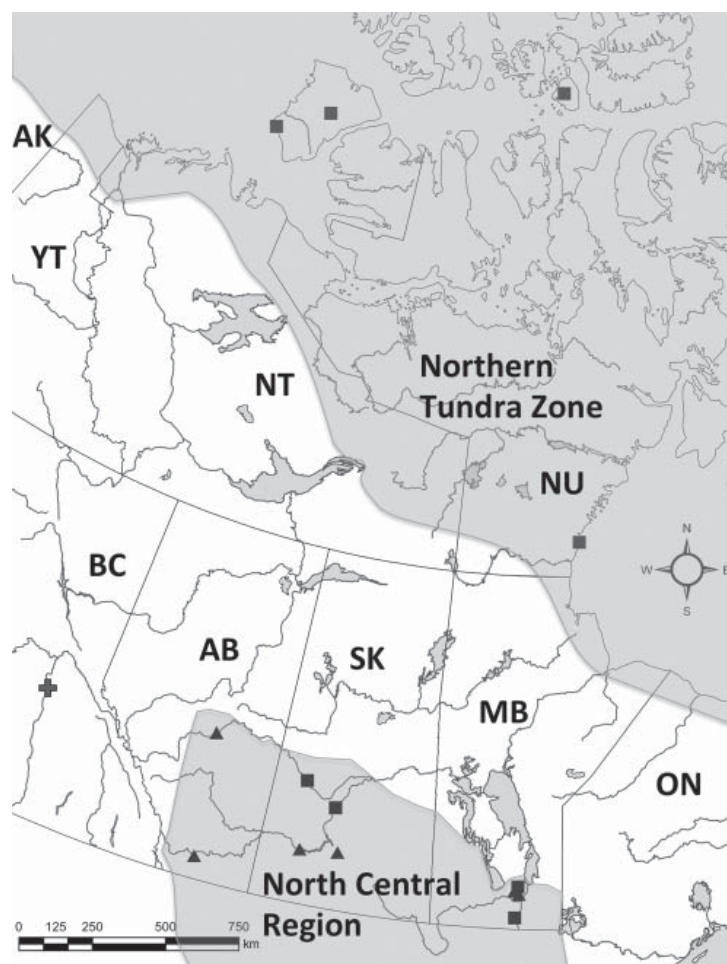


Figure. Location where European-type strain of *Echinococcus multilocularis* (plus sign) was detected in this study in British Columbia (BC) and previous reports of *E. multilocularis* parasites in 8 definitive (squares) and 6 intermediate (triangles) hosts in Canada. Gray shading indicates currently accepted distribution of *E. multilocularis* in North America. The North Central Region includes southern portions of the 3 Canadian prairie provinces (Alberta [AB], Saskatchewan [SK], and Manitoba [MB]) and 12 contiguous US states (not shown). The western portion of the Northern Tundra Zone is based roughly on the established distribution of Arctic fox in Alaska (AK), the Yukon Territory (YT), Northwest Territories (NT), Nunavut (NU), northern MB, and northern Ontario (ON).

tected from a single fecal sample examined on microscopy (1), infection most likely resulted from consumption of infective eggs in the feces of a carnivore-definitive host. This host could have been a translocated domestic dog, thought to be the mechanism of recent introduction of *E. multilocularis* parasites into Sweden (7). It is also possible that a European strain of the parasite was introduced into North America in the last century, when red fox from France and Scandinavia were introduced (8).

The possible establishment of a European strain in North American wildlife, with spillover into domestic dogs, may have implications for public health and require increased vigilance by medical and veterinary personnel in the newly endemic region. Compared with native North American strains, European strains of *E. multilocularis* appear to have greater potential to cause alveolar hydatid disease (AHD) in humans. These strains are emerging worldwide (increasing in both prevalence and distribution) as a result of changes in landscape, climate, and wildlife-human interfaces (2,9,10). In Europe, human AHD can be fatal (definite or probable cause of death in 23.5% of 119 recent cases) and has low cure rates (5% of 408 recent cases) (2). As of 2000, in Europe and Asia, the estimated cost per case of AHD was US \$100,000–\$300,000 (9). Therefore, better understanding of the distribution, genetic diversity, and pathogenicity of strains of *E. multilocularis* is needed to assess risks and mitigate costs for public and veterinary health, as well as to provide evidence for the regulation and screening of imported domestic animals and translocated wildlife.

**Emily J. Jenkins,  
Andrew S. Peregrine,  
Janet E. Hill, Christopher Somers,  
Karen Gesy, Brian Barnes,  
Bruno Gottstein,  
and Lydden Polley**

Author affiliations: University of Saskatchewan, Saskatoon, Saskatchewan, Canada (E. Jenkins, J.E. Hill, K. Gesy, L. Polley); University of Guelph, Guelph, Ontario, Canada (A. Peregrine); University of Regina, Regina, Saskatchewan, Canada (C. Somers); Westview Veterinary Hospital, Powell River, British Columbia, Canada (B. Barnes); and Universitat Bern, Bern, Switzerland (B. Gottstein)

DOI: <http://dx.doi.org/10.3201/eid1806.111420>

## References

1. Peregrine AS, Jenkins EJ, Barnes B, Johnson S, Polley L, Barker IK, et al. Alveolar hydatid disease (*Echinococcus multilocularis*) in the liver of a Canadian dog in British Columbia, a newly endemic region. *Can Vet J*. In press.
2. Kern P, Bardonnnet K, Renner E, Auer H, Pawlowski Z, Ammann RW, et al. European echinococcosis registry: human alveolar echinococcosis, Europe, 1982–2000. *Emerg Infect Dis*. 2003;9:343–9.
3. Bowles J, McManus DP. NADH dehydrogenase I gene sequences compared for species and strains of the genus *Echinococcus*. *Int J Parasitol*. 1993;23:969–72. [http://dx.doi.org/10.1016/0020-7519\(93\)90065-7](http://dx.doi.org/10.1016/0020-7519(93)90065-7)
4. Nakao M, Xiao N, Okamoto M, Yanagida T, Sako Y, Ito A. Geographic pattern of genetic variation in the fox tapeworm *Echinococcus multilocularis*. *Parasitol Int*. 2009;58:384–9. <http://dx.doi.org/10.1016/j.parint.2009.07.010>
5. Knapp J, Bart JM, Glowatzki ML, Ito A, Gerard S, Maillard S, et al. Assessment of use of microsatellite polymorphism analysis for improving spatial distribution tracking of *Echinococcus multilocularis*. *J Clin Microbiol*. 2007;45:2943–50. <http://dx.doi.org/10.1128/JCM.02107-06>
6. Nakao M, Sako Y, Ito A. Isolation of polymorphic microsatellite loci from the tapeworm *Echinococcus multilocularis*. *Infect Genet Evol*. 2003;3:159–63. [http://dx.doi.org/10.1016/S1567-1348\(03\)00070-4](http://dx.doi.org/10.1016/S1567-1348(03)00070-4)
7. Osterman Lind E, Juremalm M, Christenson D, Widgren S, Hallgren G, Agren EO, et al. First detection of *Echinococcus multilocularis* in Sweden, February to March 2011. *Euro Surveill*. 2011;16:pii=19836.
8. Kamler JF, Ballard WB. A review of native and nonnative red foxes in North America. *Wildl Soc Bull*. 2002;30:370–9.
9. Eckert J, Conraths FJ, Tackmann K. Echinococcosis: an emerging or re-emerging zoonosis? *Int J Parasitol*. 2000;30:1283–94. [http://dx.doi.org/10.1016/S0020-7519\(00\)00130-2](http://dx.doi.org/10.1016/S0020-7519(00)00130-2)
10. Jenkins EJ, Schurer JM, Gesy KM. Old problems on a new playing field: helminth zoonoses transmitted among dogs, wildlife, and people in a changing northern climate. *Vet Parasitol*. 2011;182:54–69. <http://dx.doi.org/10.1016/j.vetpar.2011.07.015>

Address for correspondence: Emily J. Jenkins, Department of Veterinary Microbiology, University of Saskatchewan, 52 Campus Dr, Saskatoon, Saskatchewan S7N 5B4, Canada; email: [emily.jenkins@usask.ca](mailto:emily.jenkins@usask.ca)

## Recognition and Diagnosis of *Cryptococcus gattii* Infections in the United States

**To the Editor:** An outbreak of *Cryptococcus gattii* cryptococcosis has been ongoing in the US Pacific Northwest (PNW) since 1999 (1–3). In contrast to *C. neoformans* infections, which typically cause meningitis in HIV-infected persons, outbreak-associated *C. gattii* infections occur primarily in persons without HIV and often cause pneumonia (1–3). Sporadic, nonoutbreak-associated *C. gattii* infections often cause meningitis and have been reported outside the PNW (1–4). The prevalence of both types of *C. gattii* infection in the United States is unknown because diagnostic practices and awareness vary among physicians.

Some reports indicate that patients with *C. gattii* infections may respond to treatment more slowly and relapse more frequently than patients with *C. neoformans* infections and, thus, may require more aggressive clinical management (5–8). Therefore, differentiation of *C. gattii* from *C. neoformans* infections may be necessary for optimal patient