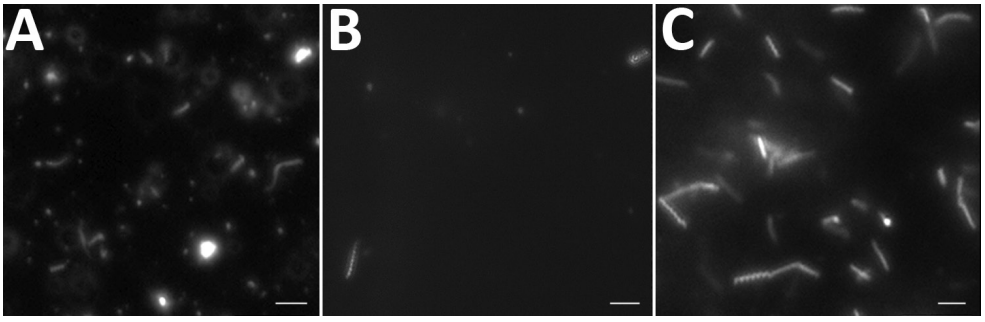

Figure. Direct examination with dark-field microscopy of specimens from a patient with agammaglobulinemia who had *Spiroplasma apis* infection, France. A) Helical and motile bacteria in blood culture. B) Elongated and coccoid bacteria in joint fluid. C) Helical and motile bacteria in culture from joint fluid in modified SP4 broth medium. Scale bar indicates 10 μm.

than honeybees. The insect stings in this patient are a likely gateway of the reported infection.

In summary, clinicians and microbiologists should be aware of fastidious organisms in atypical infections in immunocompromised patients. Our findings indicate a need for prolonged culture on specific agar on all joint fluids in patients with agammaglobulinemia and targeted molecular methods to identify *S. apis* organisms.

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References

1. Schabereiter-Gurtner C, Nehr M, Apfalter P, Makristathis A, Rotter ML, Hirschl AM. Evaluation of a protocol for molecular broad-range diagnosis of culture-negative bacterial infections in clinical routine diagnosis. J Appl Microbiol. 2008;104:1228–37. http://dx.doi.org/10.1111/j.1365-2672.2007.03648.x
2. Devulder G, Perrière G, Baty F, Flandrois JP. BIBI, a bioinformatics bacterial identification tool. J Clin Microbiol. 2003; 41:1785–7. http://dx.doi.org/10.1128/JCM.41.4.1785-1787.2003
3. Meeus I, Vercruysse V, Smagghe G. Molecular detection of *Spiroplasma apis* and *Spiroplasma melliferum* in bees. J Invertebr Pathol. 2012;109:172–4. http://dx.doi.org/10.1016/j.jip.2011.11.006
4. Blanchard A, Bébéar CM. Mycoplasmas of humans. In: Razin S, Herrmann R, editors. Molecular biology and pathogenicity of mycoplasmas. New York: Kluwer Academic/Plenum Publishers; 2002. p. 45–71.</edb>
5. Mouches C, Bové JM, Tully JG, Rose DL, McCoy RE, Carle-Junca P, et al. *Spiroplasma apis*, a new species from the honey-bee *Apis mellifera.* Ann Microbiol (Paris). 1983;134A:383–97.
6. Schwarz RS, Teixeira ÉW, Tauber JP, Birke JM, Martins MF, Fonseca I, et al. Honey bee colonies act as reservoirs for two *Spiroplasma* facultative symbionts and incur complex, multiyear infection dynamics. MicrobiologyOpen. 2014;3:341–55. http://dx.doi.org/10.1002/mbo3.172
7. Mueller NJ, Tini GM, Weber A, Gaspert A, Husmann L, Bloemberg G, et al. Hepatitis from *Spiroplasma* sp. in an immunocompromised patient. Am J Transplant. 2015;15:2511–6. http://dx.doi.org/10.1111/ajt.13254
8. Lorenz B, Schroeder J, Reischl U. First evidence of an endogenous *Spiroplasma* sp. infection in humans manifesting as unilateral cataract associated with anterior uveitis in a premature baby. Graefes Arch Clin Exp Ophthalmol. 2002;240:348–53. http://dx.doi.org/10.1007/s00417-002-0453-3
9. Aquilino A, Masiá M, López P, Galiana AJ, Tovar J, Andrés M, et al. First human systemic infection caused by *Spiroplasma.* J Clin Microbiol. 2015;53:719–21. http://dx.doi.org/10.1128/JCM.02841-14

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Mycoplasma ovipneumoniae in Wildlife Species beyond Subfamily *Caprinae*

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Elucidating the emergence of *Mycoplasma ovipneumoniae*–associated respiratory disease in ruminants requires identification of the pathogen host range. This bacterium was thought to be host restricted to subfamily *Caprinae*, but we describe its identification in healthy moose, caribou, and mule deer and diseased mule and white-tailed deer, all species in subfamily *Capreolinae*.

Mycoplasma ovipneumoniae was identified in Queensland, Australia, in 1972 as an infectious agent associated with pneumonia in domestic sheep (*Ovis aries*) (*1*). Since then, it has most frequently been identified in healthy and diseased domestic sheep, domestic goats (*Capra aegagrus hircus*), and bighorn sheep (*Ovis canadensis*). Although *M. ovipneumoniae* was identified in respiratory disease outbreaks in bighorn sheep as early as 1980 (*2*), the past decade has brought it under scrutiny because of evidence supporting its association with bighorn sheep pneumonia in western North America (*3*). Because most reports have described this bacterium in sheep and goats, and fewer in muskoxen (*Ovibos moschatus*) (*4*), some have concluded that *M. ovipneumoniae* is specific to the subfamily *Caprinae* (*5*) or has a host range limited to *Caprinae* (*6*), despite publications describing *M. ovipneumoniae* in non-*Caprinae* species, including Beira antelope (*Dorcatragus megalotis*) with respiratory disease in Qatar (*7*) and in 9 cattle (*Bos taurus*) in Colorado, USA (*8*). Unfortunately, description of the method(s) used to identify *M. ovipneumoniae* in those reports was limited to stating the use of PCR with no supporting sequence data.

In general, definitive claims of host range restrictions are absent from mycoplasma literature, because "assumptions about restricted host range of mycoplasmas, based on the host from which they were first or frequently isolated, are usually made in the context of nearly complete absence of representative sampling of the vast majority of potential vertebrate hosts" (*9*). In addition to insufficient sampling of potential hosts, the fastidious and variably culturable nature of *M. ovipneumoniae* often requires molecular techniques for identification. We used molecular techniques to analyze multiple species from the subfamily *Capreolinae* for the presence of *M. ovipneumoniae*.

During July 2017–January 2018, the US Department of Agriculture Agricultural Research Service in Pullman, WA, USA, received nasal swab samples from 230 moose (*Alces alces*) and 243 caribou (*Rangifer tarandus*) from Alaska and 5 mule deer (*Odocoileus hemionus*) from Arizona (online Technical Appendix, https://wwwnc.cdc.gov/EID/article/24/12/18-0632-Techapp1.pdf). Also received in February 2018 was an isolate of *M. ovipneumoniae* that had been cultured by Newport Laboratories (Worthington, MN, USA) from lung tissue from a white-tailed deer (*Odocoileus virginianus*) that died during a pneumonia outbreak at a captive facility in the upper Midwest region of the United States in 2016. We extracted DNA from swab samples and from the white-tailed deer isolate, performed PCR using a modified published PCR method (*10*) to amplify part of the 16S rRNA gene, and sequenced amplicons of the correct size (online Technical Appendix). Forward and reverse sequences were merged, manually inspected for errors, and trimmed to 290 bp using Sequencher 5.2.2 (Gene Codes, Ann Arbor, MI, USA) corresponding to a 103–392-bp region of the 16S rRNA gene of type strain Y98 obtained from American Type Culture Collection (ATCC; Manassas, VA, USA). Sequences were blastn queried (https://www.ncbi.nlm.nih.gov/BLAST/) and identified as *M. ovipneumoniae* at 100% coverage and ≥97% identity to Y98 (GenBank accession no. NR_025989.1). The analyzed region represents the most divergent region of the 16S rRNA gene among strains of *M. ovipneumoniae* and between *Mycoplasma* spp. of the highest percentage identity to *M. ovipneumoniae* (online Technical Appendix).

We detected *M. ovipneumoniae* in 6 moose (2.6%), including 3 from 2 captive facilities and 3 free-ranging; 5 free-ranging caribou (2.1%); and 2 of 5 mule deer, 1 of which was coughing and had nasal discharge at the time of sample collection. The identity of the lung isolate, cultured from the white-tailed deer that had died from pneumonia, was confirmed as *M. ovipneumoniae*. For sequence comparison, we generated a percent identity matrix with the *M. ovipneumoniae* sequences from the *Capreolinae* species, nasal swabs collected from 2 healthy *M. ovipneumoniae*–positive wild sheep (online Technical Appendix), Y98, and bacteria of the closest identity to Y98 and sequences generated in this study based on blastn queries (*M. dispar*, *M. hyopneumoniae*, and *M. flocculare*) (online Technical Appendix). The percent identity matrix revealed 2 groupings of *M. ovipneumoniae* and illustrates the divergence from the other *Mycoplasma* spp. of closest identity to *M. ovipneumoniae*. Sample sequences have been submitted to GenBank (online Technical Appendix).

This report describes *M. ovipneumoniae* carriage in multiple members of the subfamily *Capreolinae* (moose, caribou, and mule deer), and emergence of *M. ovipneumoniae*–associated respiratory disease in deer. These findings are of importance to epidemiologic investigations because current dogma regarding host specificity may dissuade laboratories from pursuing identification of this fastidious bacterium in hosts beyond the subfamily *Caprinae*. Improved diagnostic methods to increase detection sensitivity are warranted based on information provided in this report (online Technical Appendix). Full-length genome sequencing and phylogenetic analysis of *M. ovipneumoniae* isolates are necessary next steps in inferring evolutionary relationships and origin of this bacterium in identified host species.

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References

1. Carmichael LE, St George TD, Sullivan ND, Horsfall N. Isolation, propagation, and characterization studies of an ovine *Mycoplasma* responsible for proliferative interstitial pneumonia. Cornell Vet. 1972;62:654–79.
2. Bunch TD. Chronic sinusitis in desert bighorn sheep in northwestern Arizona. In: Douglas CL, Krausman PR, Leslie DM, editors. Desert Bighorn Council meeting 1985. Las Vegas (NV, USA): the Council; 1985. p. 1–3.
3. Besser TE, Highland MA, Baker K, Cassirer EF, Anderson NJ, Ramsey JM, et al. Causes of pneumonia epizootics among bighorn sheep, Western United States, 2008–2010. Emerg Infect Dis. 2012;18:406–14. http://dx.doi.org/10.3201/eid1803.111554
4. Handeland K, Tengs T, Kokotovic B, Vikøren T, Ayling RD, Bergsjø B, et al. *Mycoplasma ovipneumoniae*—a primary cause of severe pneumonia epizootics in the Norwegian muskox (*Ovibos moschatus*) population. PLoS One. 2014;9:e106116. http://dx.doi.org/10.1371/journal.pone.0106116
5. Cassirer EF, Manlove KR, Plowright RK, Besser TE. Evidence for strain-specific immunity to pneumonia in bighorn sheep. Journal Wildl Manag. 2017;81:133–43. http://dx.doi.org/10.1002/jwmg.21172
6. Besser TE, Cassirer EF, Potter KA, Foreyt WJ. Exposure of bighorn sheep to domestic goats colonized with *Mycoplasma ovipneumoniae* induces sub-lethal pneumonia. PLoS One. 2017;12:e0178707. http://dx.doi.org/10.1371/journal.pone.0178707
7. Gull JM, Hebel C, Deb A, Arif A, Clauss M, Hatt JM, et al. Blood values of captive beira antelope (*Dorcatragus megalotis*) prior to and during an outbreak of fibrinous pleuropneumonia syndrome (FPPS). J Zoo Wildl Med. 2014;45:735–43. http://dx.doi.org/10.1638/2013-0073.1
8. Wolfe LL, Diamond B, Spraker TR, Sirochman MA, Walsh DP, Machin CM, et al. A bighorn sheep die-off in southern Colorado involving a *Pasteurellaceae* strain that may have originated from syntopic cattle. J Wildl Dis. 2010;46:1262–8. http://dx.doi.org/10.7589/0090-3558-46.4.1262
9. Brown DR, Zacher LA, Wendland LD, Brown MB. Emerging mycoplasmoses in wildlife. In: Blanchard A, Browning GF, editors. Mycoplasmas: molecular biology pathogenicity and strategies for control. Norfolk (UK): Horizon Bioscience; 2005. p. 382–414.
10. McAuliffe L, Hatchell FM, Ayling RD, King AI, Nicholas RA. Detection of Mycoplasma ovipneumoniae in Pasteurella-vaccinated sheep flocks with respiratory disease in England. Vet Rec. 2003;153:687–8. http://dx.doi.org/10.1136/vr.153.22.687

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Locally Acquired Leptospirosis in Expedition Racer, Manitoba, Canada

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Leptospirosis is found worldwide, except in northern regions. We report a case associated with a backcountry adventure race in Manitoba, Canada. Initially, nonspecific symptomatology and diagnostic pitfalls contributed to a delay in identification. Careful attention needs to be paid to exposure to and risk for leptospirosis in northern and temperate climates.

Leptospirosis is a zoonotic disease caused by *Leptospira interrogans* (motile bacterial spirochetes). Human transmission occurs by direct contact with contaminated urine or animals (*1*). The organism has a worldwide distribution outside of polar regions and is common during the rainy season in tropical and temperate climates (*2*).