sanguineus (1/2) were positive in all PCR analyses (*gltA*, *ompA*, *and htrA*); 11 of these ticks were from the patient's dogs. In all properties where ticks were collected, at least 1 was PCR positive. Thus, we detected *R. parkeri* in half (4/8) of investigated households.

All the sequences generated for the *ompA* and *htrA* genes showed 100% identity to sequences from the *Rickettsia parkeri* strain Portsmouth (GenBank accession no. CP003341.1). We deposited into GenBank the sequences of the *ompA* gene (KX196265) and *htrA* gene (KX196266) from samples analyzed in this study. The *ompA* sequence we obtained for *R. parkeri* showed 98% identity with *Rickettsia* sp. strain Atlantic Rainforest (GenBank accession no. GQ855237.1).

Although *Rickettsia* sp. strain Atlantic Rainforest had previously been considered the only SFG *Rickettsia* in southern Brazil, we demonstrate here the presence of *R. parkeri* in Rio Grande do Sul in the Pampa biome. We detected *R. parkeri* infection in *A. tigrinum* ticks collected at the probable site of infection (the patient's home) of a confirmed case of human spotted fever. Considering the *A. tigrinum* tick abundance in southern Brazil and its remarkable ability to parasitize domestic and wild animals (*8*), in addition to the high *R. parkeri* infection rate observed (28%), further epidemiologic studies are needed to address the role of *A. tigrinum* ticks as vector of spotted fever in the Pampa biome. Finally, our results show that, in addition to *R. rickettsii* and *Rickettsia* sp. strain Atlantic Rainforest, *R. parkeri* occurs and might be associated with cases of spotted fever in Brazil. Additional surveys are needed to assess the infection prevalence of *R. parkeri* in *A. tigrinum* ticks in other areas of Pampa and in other regions of Brazil.

This work was supported by the Fundação de Amparo à Pesquisa do Estado do Rio Grande do Sul (FAPERGS), Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq), Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES), and INCT Entomologia Molecular.

References

1. Galvão MA, Mafra CL, Moron C, Anaya E, Walker DH. Rickettsiosis of the genus *Rickettsia* in South America. Ann N Y Acad Sci. 2003; 990:57–61. http://dx.doi.org/10.1111/j.1749-6632.2003.tb07337.x
2. Parola P, Labruna MB, Raoult D. Tick-borne rickettsioses in America: unanswered questions and emerging diseases. Curr Infect Dis Rep. 2009;11:40–50. http://dx.doi.org/10.1007/s11908-009-0007-5
3. Barros-Battesti DM, Arzua M, Bechara GH. Carrapatos de Importância Médico-Veterinária da Região Neotropical: um guia ilustrado para identificação de espécies. São Paulo (Brazil): Vox/ ICTTD-3/Butantan; 2006.
4. Aljanabi SM, Martinez I. Universal and rapid salt-extraction of high quality genomic DNA for PCR-based techniques. Nucleic Acids Res. 1997;25:4692–3. http://dx.doi.org/10.1093/nar/25.22.4692
5. Labruna MB, Whitworth T, Horta MC, Bouyer DH, McBride JW, Pinter A, et al. *Rickettsia* species infecting *Amblyomma cooperi* ticks from an area in the state of São Paulo, Brazil, where Brazilian spotted fever is endemic. J Clin Microbiol. 2004;42:90–8. http://dx.doi.org/10.1128/JCM.42.1.90-98.2004
6. Regnery RL, Spruill CL, Plikaytis BD. Genotypic identification of rickettsiae and estimation of intraspecies sequence divergence for portions of two rickettsial genes. J Bacteriol. 1991;173:1576–89.
7. Azad AF, Webb L, Carl M, Dasch GA. Detection of rickettsiae in arthropod vectors by DNA amplification using the polymerase chain reaction. Ann N Y Acad Sci. 1990;590:557–63. http://dx.doi.org/10.1111/j.1749-6632.1990.tb42266.x
8. Evans DE, Martins JR, Guglielmone AA. A review of the ticks (Acari, ixodida) of Brazil, their hosts and geographic distribution - 1. The state of Rio Grande do Sul, southern Brazil. Mem Inst Oswaldo Cruz. 2000;95:453–70. http://dx.doi.org/10.1590/S0074-02762000000400003

Address for correspondence: José Reck, Instituto de Pesquisas Veterinárias Desidério Finamor (IPVDF), Estrada do Conde, 6000, Eldorado do Sul, 92990-000, RS, Brazil; email: jose.reck@gmail.com.

Shigella flexneri with Ciprofloxacin Resistance and Reduced Azithromycin Susceptibility, Canada, 2015

Christiane Gaudreau, Pierre A. Pilon, Gilbert Cornut, Xavier Marchand-Senecal, Sadjia Bekal

Author affiliations: Centre Hospitalier de l'Université de Montréal–Hôpital Saint-Luc, Montreal, Quebec, Canada (C. Gaudreau, G. Cornut, X. Marchand-Senecal); Université de Montréal, Montreal (C. Gaudreau, P.A. Pilon, G. Cornut, X. Marchand-Senecal, S. Bekal); Centre Intégré Universitaire de Santé et de Services Sociaux du Centre-Sud-de-l'île-de-Montreal, Montreal (P.A. Pilon); Laboratoire de Santé Publique du Québec/Institut National de Santé Publique du Québec, Sainte-Anne-de-Bellevue, Quebec (C. Gaudreau, S. Bekal)

DOI: http://dx.doi.org/10.3201/eid2211.160883

To the Editor: In 2015, a locally acquired, multidrug-resistant *Shigella flexneri* infection was identified in Montreal, Quebec, Canada, in an HIV-positive man who had sex with men (MSM). In September, the 53-year-old man consulted his physician at an outpatient clinic after experiencing abdominal pain, fatigue, and diarrhea without blood in stools or fever. The week before the symptom onset, although he had not traveled, he had unprotected oral and anal sexual contact in a Montreal bathhouse with a man visiting Canada from an unknown country. The patient did not work in daycare centers or healthcare facilities, and he was not a food handler. He did not have sex during illness.

He was HIV positive and was receiving antiretroviral treatment; recent CD4 cell count was 480 × 10^6/L, and HIV viral load was <40 copies/mL. *S. flexneri* was isolated from his culture of a fecal sample, and *Neisseria gonorrhoeae,* diagnosed by PCR, was found in a throat specimen. The patient did not have a medical record of other past sexually transmitted infections.

Phenotypic identification of the *S. flexneri* was confirmed at Laboratoire de Santé Publique du Québec (*1*). Serologic identification, pulsed-field gel electrophoresis (PFGE), and antimicrobial susceptibility testing were performed as described (*1*). This *S. flexneri,* serotype 2a pulsovar 21 (a new PFGE combination pattern in the province of Quebec), was resistant to ampicillin, trimethoprim/sulfamethoxazole (TMP/SMX), nalidixic acid, ciprofloxacin, tetracycline, and chloramphenicol. The isolate was also nonsusceptible to azithromycin and amoxicillin/-clavulanic acid, and susceptible to ceftriaxone, cefixime, ertapenem, and gentamicin (Table). The *mph*(A) gene, which codes for the macrolide 2′-phosphotransferase, tested positive by PCR (*1*).

On day 10 of diarrhea, the patient was treated with ceftriaxone, 250-mg dose, intramuscularly, followed by cefixime, 800 mg/day, for 5 days; the patient's condition showed progressive improvement. Two control cultures of fecal specimens were negative 7 and 16 days, respectively, after completion of a regimen of cefixime.

Shigella spp. are transmitted from person-to-person through low inocula of the bacteria, directly or indirectly (*1,3–5*). In MSM, *Shigella* spp. are mostly transmitted sexually, with clusters documented in many countries (*1,3–5*). In Canada and the United States, *Shigella* isolates have high levels of resistance to ampicillin and TMP/SMX (*1,3–6*). In adult patients, when antimicrobial drug treatment is indicated, ciprofloxacin and azithromycin are, respectively, the agents of first and second choices for treating *Shigella* infections (*1,3–5*).

In the United States, *Shigella* spp. resistant to at least nalidixic acid and azithromycin have been found in surveillance isolates: 1/293 in 2011 (*Shigella* spp.), 1/353 in 2012 (*S. sonnei*), and 1 of 344 in 2013 (*S. flexneri*) (*6*). In Illinois and Montana, during September 2014–April 2015, 3 of 5 patients infected with multidrug-resistant *S. sonnei* (resistant to ampicillin, TMP/SMX, ciprofloxacin, and nalidixic acid and nonsusceptible to azithromycin), identified themselves as MSM, and 2 of these patients had diarrhea for >14 days (*3*).

Clinical treatment failure has been reported in patients infected with azithromycin-nonsusceptible *Shigella* isolates treated with this drug (*7,8*), including 1 of our patients (unpub. data). In a previous study, the *mph*(A) gene was acquired by 4 of 7 locally acquired *Shigella* pulse types infecting MSM. This raises concern that reduced *Shigella* susceptibility to azithromycin is developing rapidly (*1*). Azithromycin epidemiologic cutoff values for wild- and non–wild-types of *S. flexneri* and *S. sonnei* are newly reported by CLSI (*8*). In recent years, ciprofloxacin-resistant and/or azithromycin-nonsusceptible *Shigella* spp. acquired during international travel or acquired locally were reported in the United States and in our hospital center (*1,3–6*; unpub. data). *S. flexneri* that is resistant to ceftriaxone and ciprofloxacin has been reported in the United States (*9*). Infections with multidrug-resistant *Shigella* spp. may be of longer duration and have higher costs (*3*).

When evaluating patients with diarrhea, physicians should identify risk factors and request bacterial cultures of fecal specimens. Antimicrobial drug susceptibility testing of *Shigella* isolates is essential for effective antimicrobial drug treatment. Serologic identification and PFGE are essential for epidemiologic purposes for ascertaining clusters or multidrug-resistant *Shigella* isolates (*1,3–5*). Patients with *Shigella* infection should be advised about preventive practices such as frequent handwashing and precautions when handling food and water (*3*). MSM should use barriers during oral, anal, and genital sex and wash their genitals, anus, and hands before and after sex (*1,3–5*).

We suggest obtaining 2 control cultures of fecal specimens on days 2 and 3 after the patient completes

Table. Antimicrobial susceptibility of the *Shigella flexneri*,serotype 2a pulsovar 21, isolated in Montreal, Quebec, Canada, 2015*

Antimicrobial agent	Disk diffusion, mm	MIC, mg/L	Interpretation
Ampicillin	6	≥32	R
TMP/SMX	6	≥320	R
Ciprofloxacin	12	≥4 and 8	R
Nalidixic acid	6	NA	R
Ceftriaxone	33	≤0.25	S
Cefixime	26	0.25	S
Azithromycin†	6	>256	NS
Tetracycline	6	32	R
Chloramphenicol	NA	>256	R
Amoxicillin-clavulanic acid	14	16	I
Ertapenem	NA	≤0.5	S
Gentamicin	21	≤1	S

*I, intermediate; NA, not available; NS, nonsusceptible; R, resistant; S, susceptible; TMP/SMX, trimethoprim/sulfamethoxazole.
†Azithromycin epidemiologic cutoff values for wild-type (MIC ≤8 mg/L) and non–wild-type (MIC ≥16 mg/L) *Shigella flexneri* (*2*) and the susceptibility and resistance breakpoints for the other 11 antimicrobial agents were CLSI *Enterobacteriaceae* breakpoints (*2*).

antimicrobial treatment for infection with multidrug-resistant *Shigella* spp. Patients should avoid sex during symptomatic infections and wait for 2 negative stool cultures. Montreal public health officials investigated and counselled this patient as they do for every patient with *Shigella* infections. In Quebec, physicians and microbiology laboratories are notified of *Shigella* clusters and multidrug-resistant *Shigella* infections.

To our knowledge, no other ciprofloxacin-resistant and azithromycin-nonsusceptible *Shigella flexneri* isolates have been documented in the province of Quebec. No PFGE matches to *S. flexneri* serotype 2a pulsovar 21 have been identified in Canada. Multidrug-resistant *Shigella* isolates, including those with both resistance to ciprofloxacin and nonsusceptibility to azithromycin, may be underestimated and incidence may be increasing (*1,3–5*).

Acknowledgments

We thank Guy Lapierre and the personnel of bacteriology sections of Centre Hospitalier de l'Université de Montréal–Hôpital Saint-Luc and of Laboratoire de Santé Publique du Québec for their assistance.

References

1. Gaudreau C, Barkati S, Leduc JM, Pilon PA, Favreau J, Bekal S. *Shigella* spp. with reduced azithromycin susceptibility, Quebec, Canada, 2012–2013. Emerg Infect Dis. 2014;20:854–6. http://dx.doi.org/10.3201/eid2005.130966
2. CLSI. Performance standards for antimicrobial susceptibility testing; 24th informational supplement; no. M100-S. 26th ed. Wayne (PA): CLSI; 2016.
3. Centers for Disease Control and Prevention. Ciprofloxacin- and azithromycin-nonsusceptible shigellosis in the United States. CDC Health Alert Network June 4, 2015 [cited 2016 Jan 20]. http://emergency.cdc.gov/han/han00379.asp
4. Gaudreau C, Ratnayake R, Pilon PA, Gagnon S, Roger M, Lévesque S. Ciprofloxacin-resistant *Shigella sonnei* among men who have sex with men, Canada, 2010. Emerg Infect Dis. 2011;17:1747–50. http://dx.doi.org/10.3201/eid1709.102034
5. Heiman KE, Karlsson M, Grass J, Howie B, Kirkcaldy D, Mahon B, et al. *Shigella* with decreased susceptibility to azithromycin among men who have sex with men—United States, 2002–2013. MMWR Morbid Mortal Wkly Rept. 2014;63:132–3.
6. Centers for Disease Control and Prevention. National Antimicrobial Resistance Monitoring System. Enteric bacteria 2013. Human isolates final report. [cited 2016 Feb 29]. http://www.cdc.gov/narms/reports/
7. Boumghar-Bourtchai L, Mariani-Kurkdjian P, Bingen E, Filliol I, Dhalluin A, Ifrane SA, et al. Macrolide-resistant *Shigella sonnei*. Emerg Infect Dis. 2008;14:1297–9. http://dx.doi.org/10.3201/eid1408.080147
8. Hassing RJ, Melles DC, Goessens WHF, Rijnders BJA. Case of *Shigella flexneri* infection with treatment failure due to azithromycin resistance in an HIV-positive patient. Infection. 2014;42:789–90. http://dx.doi.org/10.1007/s15010-014-0594-4
9. Jue S, Hardee R, Mays E, Bowen A, Whichard J, Greene K, et al. Emergence of *Shigella flexneri* 2a resistant to ceftriaxone and ciprofloxacin—South Carolina, October 2010. MMWR Morb Mortal Wkly Rep. 2010;59:1619.

Address for correspondence: Christiane Gaudreau, Microbiologie Médicale et Infectiologie, CHUM-Hôpital Saint-Luc, 1058 Rue Saint-Denis, Montréal, Québec, Canada H2X 3J4; email: christiane.gaudreau.chum@ssss.gouv.qc.ca

HIV/Hepatitis C Virus Co-infection among Adults Beginning Antiretroviral Therapy, Malawi

Münevver Demir, Sam Phiri, Rolf Kaiser, Thom Chaweza, Florian Neuhann, Hannock Tweya, Gerd Fätkenheuer, Hans-Michael Steffen

Author affiliations: University Hospital of Cologne, Cologne, Germany (M. Demir, R. Kaiser, G. Fätkenheuer, H.-M. Steffen); Lighthouse Clinic, Lilongwe, Malawi (S. Phiri, T. Chaweza, H. Tweya); Institute of Public Health, University Hospital of Heidelberg, Heidelberg, Germany (F. Neuhann); German Centre for Infection Research, Cologne, (G. Fätkenheuer)

DOI: http://dx.doi.org/10.3201/eid2211.160892

To the Editor: Throughout the world, ≈115 million persons have hepatitis C virus (HCV) antibodies, ≈37 million are infected with HIV type 1, and an estimated 2.3 million persons are infected with both viruses (*1*). The estimated prevalence of HIV infection among adults in Malawi is 9.1% (*2*). Data concerning HCV seroprevalence in Malawi are conflicting and range from 0.0% to 18.0%, depending on the studied population and the chosen methods for HCV infection diagnosis (*3–6*). In a recent study, researchers used stored blood samples (without HCV confirmatory assays) from studies in rural and urban Malawian populations (1989–2008); an HCV seroprevalence of 6.8% was found in HIV-positive patients (*7*). In contrast, in a cohort of HIV-negative mothers (2006–2010), only 0.5% were found to be HCV positive with confirmatory HCV testing by immunoblot (*8*). These studies were not included in a 2015 metaanalysis that estimated the seroprevalence of HCV infection and HIV/HCV co-infection in Malawi to be 7.7% and 2.0%, respectively (*9*). Liver disease progresses more rapidly in HIV/HCV co-infected patients than in HCV monoinfected patients (*10*), and the highly effective second-generation direct-acting antiviral therapies are less toxic than interferon-based treatment regimens. It is crucial to gather accurate epidemiologic information on the burden of HIV/HCV co-infection to support the design and implementation of HCV treatment initiatives in resource-limited settings such as sub-Saharan Africa.