

40 years [4], respectively). Because IgG against LCMV can persist for years, seroprevalence would be expected to be higher for an older population as a result of more chances for exposure. Also, a serosurvey of >1,000 hospitalized persons from upstate New York in the 1970s detected no positive antibody titers (6), consistent with our findings.

Our serosurvey had a few limitations. Blood samples from an entire county cannot detect potential household- or neighborhood-scale areas of increased risk for LCMV exposure, which may be related to focal distribution of populations of LCMV-infected house mice. Serosurveys of house mice have previously shown evidence for clustering of LCMV-infected individuals (7); however, the prevalence of LCMV in house mice in Onondaga County is unknown. Additionally, because blood donors were volunteers, the population sampled did not necessarily reflect the population at risk for LCMV exposure. Despite these considerations, the low prevalence of LCMV antibodies suggests low occurrence of LCMV exposure in this population.

Although little is known about frequency of human exposure and infection, LCMV seems to be rare with a propensity for inducing severe disease. LCMV infection has been associated with high incidence of clinical disease, including a pet hamster-associated outbreak in 1973–1974 that resulted in at least 181 cases and 46 hospitalizations in 12 states (8).

LCMV-related disease is reportable in only 3 states (Wisconsin, Massachusetts, Arizona) and 1 city (New York, New York) and is considered to be widely undertested and underdiagnosed. A recent survey of health care providers in Connecticut found that LCMV diagnostic tests were not requested for all patients suspected to have LCMV infection (9); thus, missed diagnoses are possible. Additional studies are

needed to understand the incidence of LCMV-related disease and LCMV seroprevalence in the general population (10).

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References

1. Peters CJ. Lymphocytic choriomeningitis virus—an old enemy up to new tricks. *N Engl J Med*. 2006;354:2208–11. doi:10.1056/NEJMp068021
2. Barton LL, Mets MB. Congenital lymphocytic choriomeningitis virus infection: decade of rediscovery. *Clin Infect Dis*. 2001;33:370–4. doi:10.1086/321897
3. Childs JE, Glass GE, Ksiazek TG, Rossi CA, Oro JG, Leduc JW. Human–rodent contact and infection with lymphocytic choriomeningitis and Seoul viruses in an inner-city population. *Am J Trop Med Hyg*. 1991;44:117–21.
4. Park JY, Peters CJ, Rollin PE, Ksiazek TG, Katholi CR, Waites KB, et al. Age distribution of lymphocytic choriomeningitis virus serum antibody in Birmingham, Alabama: evidence of a decreased risk of infection. *Am J Trop Med Hyg*. 1997;57:37–41.
5. Centers for Disease Control and Prevention. Notes from the field: congenital lymphocytic choriomeningitis—New York. *MMWR Morb Mortal Wkly Rep*. 2011;60:16.
6. Vanzee BE, Douglas RG, Betts RF, Bauman AW, Fraser DW, Hinman AR. Lymphocytic choriomeningitis in university hospital personnel. Clinical features. *Am J Med*. 1975;58:803–9. doi:10.1016/0002-9343(75)90635-X
7. Childs JE, Peters CJ. Ecology and epidemiology of arenaviruses and their hosts. In: Salvato MS, editor. *The Arenaviridae*. New York: Plenum Press; 1993. p. 331–84.
8. Gregg MB. Recent outbreaks of lymphocytic choriomeningitis in the United States of America. *Bull World Health Organ*. 1975;52:549–53.
9. Centers for Disease Control and Prevention. Survey of lymphocytic choriomeningitis virus diagnosis and testing—Connecticut, 2005. *MMWR Morb Mortal Wkly Rep*. 2006;55:398–9.
10. Barton LL. Human infection with lymphocytic choriomeningitis virus. *Emerg Infect Dis*. 2010;16:1046–7.

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Tickborne Relapsing Fever Caused by *Borrelia persica*, Uzbekistan and Tajikistan

To the Editor: Tickborne relapsing fever (TBRF) is caused by several *Borrelia* species and transmitted by argasid soft ticks of the genus *Ornithodoros*. The disease is endemic to many parts of the world, especially Africa (*B. duttonii* and *B. crociduræ* most prevalent), and the Mediterranean basin (*B. hispanica* most prevalent). In Eurasia, TBRF is mainly caused by *B. persica* (1–3). We report a patient who returned to France with *B. persica* infection after visiting Uzbekistan and Tajikistan.

In September 2008, a 32-year-old man sought care at the Saint Louis Hospital in Paris, France, for a fifth relapsing episode of fever. Three weeks earlier (July 31–August 18), he had trekked through Uzbekistan and Tajikistan and spent 7 nights in a tent in Uzbekistan. The day before his return to France, he had fever (39.5°C) that lasted 5 days without other symptoms. After a 4-day interval, fever recurred for 1 day, then relapsed 3 other times (every 2 days with fever for 12 hours). Laboratory

investigations performed earlier by his general physician on August 19 and 20 showed lymphopenia (0.76×10^9 cells/L and 0.44×10^9 cells/L), thrombocytopenia (94×10^9 cells/L and 80×10^9 cells/L), and C-reactive protein level 300 mg/L (reference level <6 mg/L).

Physical examination when the man was afebrile found no clinical abnormalities. He did not recall tick bites, and no lesion was seen on his skin. Laboratory investigations on September 2 indicated persistent lymphopenia (1.0×10^9 cells/L), C-reactive protein 96 mg/L, and mild cholestasis (alkaline phosphatase level 125 UI/L and gamma glutamyl transferase level 92 UI/L). Thick and thin Giemsa-stained blood smears showed neither *Plasmodium* spp. nor *Borrelia* spp. However, quantitative buffy coat analysis (QBC; Becton Dickinson, Le Pont de Claix, France) showed numerous spirochetes, which prompted a reevaluation and careful analysis of the slides, during which spirochetes were infrequently seen. PCR and sequencing of the 16S rRNA gene, performed as previously described, from a whole blood sample identified these bacteria as *B. persica* (1).

According to current recommendations in France, the patient was given doxycycline, 200 mg/d, for 10 days. During the first 12 hours, he was monitored for a Jarish-Herxheimer reaction, which was not observed. By the end of therapy, inflammatory syndrome and lymphopenia had resolved.

Despite high-level sequence conservation, identification of *Borrelia* spp. by sequencing the 16S rRNA gene is reliable and useful for clinical practice (1,4–6). The sequence obtained from the patient reported here was identical (100% identity over 1,472 bp) to the *B. persica* HM161645 reference sequence available from GenBank and sampled from the Galilee region of Israel. It differed by 2 nt (99.86%

identity) from *B. persica* U42297 and another unpublished sequence (*B. persica* 11/95), each from Iran. *B. persica* 11/95 was obtained from blood from a rodent collected in Iran and examined at the Institut Pasteur of Iran. We submitted the 2 *B. persica* sequences (1 from the patient reported here and 1 from the 11/95 sample) to GenBank (accession nos. HQ610930 and HQ610931, respectively). We confirmed identification of *B. persica* from the patient reported here by sequencing of both *flaB* and intergenic spacer domains (data not shown), as described (7,8).

Although recently reviewed, the epidemiology of TBRF in the area of the former Union of Soviet Socialist Republics has not been extensively described (3). In most of these countries, the infection is mainly attributed to *B. persica* and transmitted by *Ornithodoros tholozani* ticks, which live in caves, soil, wall crevices, houses, and cow sheds. Mammalian reservoirs, if any, are not known (3). However, other agents have been reported, such as *B. latyschewii* (transmitted by *O. tartakovskyi* ticks, which inhabit rodent burrows) in Kazakhstan, Uzbekistan, and Turkmenistan and *B. caucasica* (transmitted by *O. verrucosus* ticks) in the western shore of the Caspian Sea (Armenia, Azerbaijan, and Georgia) (9,10). Other soft ticks (*O. nereensis* and *O. alactagalis*) have been described in central Asia.

To our knowledge, TBRF cases caused by *B. persica* in Uzbekistan and Tajikistan have been rarely reported. The clinical illness of the patient reported here did not differ substantially from that of patients in Israel, Iran, or Jordan, where the infection is more frequently detected. The illness appears benign without chills, headache, vomiting, arthralgia, epistaxis, or hematuria. However, lymphopenia, which resolved rapidly, has not been described for other TBRF cases.

Our report highlights that TBRF is endemic to countries of the former Union of Soviet Socialist Republics. Physicians should consider this diagnosis for febrile patient returning from this area. Efforts to prevent tick bites should be emphasized. Accurate microbiological diagnosis comprises molecular detection or quantitative buffy coat analyses, each of which enhances sensitivity. However, in most disease-endemic countries, diagnosis is based only on examination of direct blood smears, which can lead to false-negative results and underestimation of the actual extent of this infection (1).

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References

1. Sarih M, Garnier M, Boudebouch N, Bouattour A, Rihani A, Hassar M, et al. *Borrelia hispanica* relapsing fever, Morocco. Emerg Infect Dis. 2009;15:1626–9.
2. Cutler SJ, Abdissa A, Trape JF. New concepts for the old challenge of African relapsing fever borreliosis. Clin Microbiol Infect. 2009;15:400–6. doi:10.1111/j.1469-0691.2009.02819.x

3. Assous MV, Wilamowski A. Relapsing fever borreliosis in Eurasia—forgotten, but certainly not gone! *Clin Microbiol Infect*. 2009;15:407–14. doi:10.1111/j.1469-0691.2009.02767.x
4. Ras NM, Lascola B, Postic D, Cutler SJ, Rodhain F, Baranton G, et al. Phylogenesis of relapsing fever *Borrelia* spp. *Int J Syst Bacteriol*. 1996;46:859–65. doi:10.1099/00207713-46-4-859
5. Scott JC. Typing African relapsing fever spirochetes. *Emerg Infect Dis*. 2005;11:1722–9.
6. Wyplosz B, Mihaila-Amrouche L, Baixench MT, Bigel ML, Berardi-Grassias L, Fontaine C, et al. Imported tickborne relapsing fever, France. *Emerg Infect Dis*. 2005;11:1801–3.
7. Assous MV, Wilamowski A, Bercovier H, Marva E. Molecular characterization of tickborne relapsing fever *Borrelia* spp. *Emerg Infect Dis*. 2006;12:1740–3.
8. Bouattour A, Garnier M, M'Ghirbi Y, Sarih M, Gern L, Ferquel E, et al. *Borrelia crociduræ* infection of *Ornithodoros erraticus* (Lucas, 1849) ticks in Tunisia. *Vector Borne Zoonotic Dis*. 2010;10:825–30. doi:10.1089/vbz.2009.0151
9. Rodhain F. *Borrelia* et fièvres récurrentes: aspects épidémiologiques actuels. *Bull Inst Pasteur*. 1976;74:173–218.
10. Rasulov I. Ticks status in central Asia with a special emphasis on Uzbekistan. *Parasitol Res*. 2007;101(Suppl 2):S183–6. doi:10.1007/s00436-007-0691-8

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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.

Toxoplasmosis and Horse Meat, France

To the Editor: *Toxoplasma gondii* parasites are obligate intracellular apicomplexans that can infect virtually all warm-blooded animals; felids are definitive hosts. The most common sources of human infection are ingestion of tissue cysts in undercooked meat or of food or water contaminated with oocysts shed by felids and transplacental transmission. Acquired toxoplasmosis in immunocompetent humans is frequently asymptomatic but is associated with cervical or occipital lymphadenopathy in ≈10% of patients. Severe or fatal outcomes for immunocompetent patients have been attributed to the virulence of specific *T. gondii* genotypes (1). We describe 3 cases of toxoplasmosis caused by atypical strains probably acquired by from ingestion of raw horse meat imported from Canada and Brazil.

Patient 1, a 74-year-old man, was hospitalized locally (Antibes-Juan Les Pins, southern France) in March 2009 for asthenia and persistent febrile bronchitis. His medical history included severe smoking-related chronic obstructive pulmonary disease and coronary artery disease. He received broad-spectrum antimicrobial drugs and methylprednisolone. On day 23 after admission, he was transferred to our teaching hospital in Nice because of clinical deterioration and persistent fever. Disseminated toxoplasmosis was diagnosed on the basis of serologic evidence of recent primary *T. gondii* parasite infection and quantitative PCR detection of high *Toxoplasma* DNA levels in peripheral blood. Despite specific antitoxoplasma therapy with sulfadiazine and pyrimethamine, he remained febrile, his respiratory function worsened, and he died on day 27.

Patient 2, a 24-year-old pregnant woman, was hospitalized in Draguignan, France, in December

2009 for full-term delivery. Three weeks earlier, routine serologic testing showed *T. gondii* parasite infection seroconversion. The newborn's and mother's ophthalmologic examinations were unremarkable. Congenital toxoplasmosis was diagnosed on the basis immunoglobulin M in the infant's serum, positive quantitative PCR of samples from the placenta, and strain isolation after inoculation of mice with a placental preparation. Sulfadiazine and pyrimethamine were started.

We performed a retrospective epidemiologic investigation of an unusual case of toxoplasmosis that occurred in March 1991. Patient 3, a 21-year-old pregnant woman living in the Nice area, was treated with spiramycin because routine serologic testing had shown *T. gondii* parasite infection seroconversion at 22 weeks' gestation. Amniocentesis showed *T. gondii* tachyzoites in amniotic fluid by microscopic examination. At 26 weeks' gestation, the woman underwent termination of pregnancy for ultrasonography-detected fetal severe abnormalities. Fetal necropsy showed numerous cerebral, cardiac, and hepatic abscesses with *T. gondii* tachyzoites. A few days after pregnancy termination, the woman experienced cervical lymphadenopathy, which lasted 3 years. She reported having eaten raw horse meat regularly during her pregnancy.

Genetic analyses with microsatellite markers of the *Toxoplasma* spp. strains isolated from the 3 patients found 3 different atypical genotypes. Atypical strains are common in South America but unusual in France, where >95% of reported strains collected from human and animal toxoplasmosis cases belonged to the type II clonal lineage (2,3). Hence, isolation of an atypical *Toxoplasma* genotype from a patient in France strongly suggests contamination by a non-European strain, either during residence abroad or after ingestion of imported meat.