Epidemiologic investigation of our case-patients that included questioning relatives, patients, and butchers found that eating raw horse meat imported from Canada (patient 1) or Brazil (patient 2) was the most likely source of the parasites. The geographic origin of the horse meat eaten by patient 3 is unknown.

Moreover, an atypical *T. gondii* strain was isolated after mouse inoculation with horse meat from the first patient's butcher. In all 3 cases, close relatives encouraged the patients to eat raw horse meat regularly because the practice is traditionally thought to reinforce health. Human toxoplasmosis cases associated with horse meat consumption are rarely reported but are probably underestimated (*2*). In the European Union, France and Italy account for more than two thirds of all horse meat eaten, predominantly raw, thereby increasing the likelihood of infection by various parasites, including *Trichinella* spp. and *Toxoplasma* spp. (*4*). Under natural conditions, serologic prevalence of *T. gondii* parasites in horses worldwide may range from 0% to 80% (*5*). Many factors could account for this variation, including the sensitivity and specificity of the serologic test, ages of animals, geographic area and hygienic condition of farm management (*5*). The only prevalence survey of horses slaughtered for food that we are aware of was conducted in Canada and the United States and found antibodies to *T. gondii* parasites in 124 (6.9%) of 1,788 serum samples (*6*).

T. gondii tissue cysts in meat are immediately killed by reaching an internal temperature of 67°C in all parts of meat during cooking (*7*). Deep freezing (≤–12°C for at least 3 days) of meat before cooking is recommended because it reduces the risk for infection by inactivating most tissue cysts (*7*). These precautions are often not applied to horse meat because these imported carcasses are usually shipped as "fresh meat" and frequently eaten raw. Eating raw horse meat imported from non-European countries may expose consumers to high inocula of highly virulent atypical *Toxoplasma* spp. strains, which may cause life-threatening primary infection (case-patient 1) or severe congenital toxoplasmosis with atypical outcome of acquired toxoplasmosis in the mother (case-patient 3). Risk assessment for toxoplasmosis from horses slaughtered for food and imported into the European Union, as was recently done in France for ovine meat, is urgently needed (*3*).

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Israeli Spotted Fever, Tunisia

To the Editor: Mediterranean spotted fever (MSF) caused by *Rickettsia conorii* was the first rickettsiosis described in Tunisia. *R. conorii* was thought to be the only species existing in this country. However, authors reported other rickettsioses either from spotted fever group or typhus group (*1*,*2*). In Sfax, a town in southern Tunisia, physicians noted patients with severe forms of MSF and suspected the presence of other species or a virulent *R. conorii* strain. We report 2 cases of Israeli spotted fever (ISF) from Sfax that were confirmed by detection of rickettsial DNA in skin biopsy specimens.

In September 2009, two previously healthy men, 45 and 46 years of age (patients 1 and 2, respectively), were hospitalized in the infectious disease department of Hedi Chaker University Hospital (Sfax, Tunisia). They came

from suburban areas 30 km apart. The men were admitted with histories of fever of a few days' duration and cutaneous maculopapular rash. Patient 1 had fever of 38°C, chills, headache, and arthromyalgia without hemodynamic abnormalities. Patient 2 was admitted with fever of 41°C, conjunctivitis, and cardiovascular collapse; he was treated in an intensive care unit for 1 day. No inoculation eschar was found on either patient. Biological findings for the 2 patients showed a leukocyte count within normal ranges, anemia, thrombopenia, high levels of C-reactive protein, and elevated liver enzymes. Both patients had contact with dogs, but neither patient reported a tick bite. The patient with more severe illness worked in the livestock importation industry; his illness developed 5 days after his return from a 2-week trip to Libya. The patients received 200 mg doxycycline per day for 10 days and improved rapidly.

Skin biopy specimens from the rash and whole blood samples were obtained from the 2 patients. PCRs targeting outer membrane protein (*omp*) *A* and *B* genes were done by using previously described primers (*3*). A negative control (sterile water and DNA from a sterile biopsy specimen) and a positive control (*R. montanensis* DNA) were included in each test. Amplicon sequencing confirmed *R. conorii* ISF strain DNA in the 2 skin samples and in the blood sample of patient 1. For both patients, the sequence homology to *R. conorii* ISF strain DNA was 99% for *ompB* gene (833 pb) and 100% for *ompA* (596 pb) (GenBank accession nos. AF123712.1 and AY197564.1, respectively). Serologic testing performed by a microimmunofluorescence assay yielded negative results for the first blood samples. A second blood sample was tested only for patient 2 and showed immunoglobulin M titers of 64 and immunoglobulin G titers of 128.

We demonstrated human infection caused by *R. conorii* ISF strain in Tunisia. This strain has been recently suggested for classification with 3 others as a subspecies within the species *R. conorii* on the basis of multilocus sequence typing (*4*). ISF was first described in Israel where it is endemic (*5*). The disease appears to be more widely spread in the Mediterranean countries than first believed because cases from Italy and Portugal have been reported (*6*,*7*). Recently, a patient from Switzerland with confirmed ISF was suspected to be infected in Libya (*8*). One patient in our study may also have been infected during his stay in Libya. Thus, geographic distribution of ISF seems to be extended to all Mediterranean countries and not limited to Israel, Italy, and Portugal. Its distribution areas probably overlap with those of MSF because the 2 infections share the same vector, the dog tick (*Rhipicephalus sanguineus*) (*9*). Although a history of tick bite could not be documented from the recorded anamnesis data, contact with dogs was noted in our cases. Furthermore, the 2 cases were diagnosed during the same month (September), corresponding to seasonal fluctuations generally observed for MSF in our region. Although 1 of our patients reported recent travel, the second patient affirmed he had not left his locality; thus, endemicity of ISF in our region in Tunisia is possible.

De Sousa et al. reported the differences between patients infected with *R. conorii* Malish and ISF strains (*10*). The characteristic eschar at the site of the tick bite was markedly less noted in ISF. The absence of this eschar has been also described in other studies (*9*,*10*). In our report, patients were treated with a delay of 10 and 6 days and neither patient died, but 1 patient did experience severe illness. Our observations suggest that the supposed ability of the ISF *Rickettsia* sp. to cause more severe illness is not ascribed to late diagnosis but may be due to more virulent strains, as suspected by De Sousa (*10*).

Finally, PCR applied to whole blood and tissue samples was more effective in diagnosing these cases earlier than serology because antibodies appear to have slow kinetics. Physicians should be alert to the possibility of ISF in febrile patients in our region, especially because fatal outcomes of this infection have been reported (*8*).

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Catabacter hongkongensis Bacteremia with Fatal Septic Shock

To the Editor: *Catabacter hongkongensis* is a newly described anaerobic agent that is likely an intrinsic component of normal gut flora; it was first reported by Lau et al. in 2007 (*1*). We report a fatal case of infection caused by *C. hongkongensis* that was identified by 16S rRNA sequence.

A man 52 years of age was admitted to an intensive care unit in France for septic shock. He was a retired service member, smoker, and alcohol drinker. He had a history of hypertension but no previously known gastrointestinal disease. He sought treatment for acute abdominal pain and diarrhea of several hours' duration. On admission, he had persistent abdominal pain with generalized abdominal distension, arterial hypotension, and hypoxemia but was not febrile. Two sets of anaerobic and aerobic blood cultures were performed at a 1-hour interval, and empiric treatment with amoxicillin/clavulanic acid and gentamicin was started. Biochemical screening showed severe metabolic acidosis, acute renal insufficiency, and systemic inflammatory response syndrome. An abdominal radiograph revealed massive pneumoperitonitis. Laparotomy showed multiple lesions and intestinal perforation at the ascending and first part of the transverse colon, with a large amount of purulent fluid in the peritoneal cavity. A complete colectomy was performed, with rectum closure and end ileostomy. Despite fluid resuscitation and catecholamine infusions, hemodynamic instability worsened rapidly and led to the patient's death.

Microbiological analysis of abdominal fluid revealed the presence of *Pseudomonas aeruginosa*, *Escherichia coli*, and *Enterococcus* spp., but no anaerobic agent. On day 3 postincubation, 1 of the 2 anaerobic blood cultures grew a motile gram-positive bacillus, which grew only in strictly anaerobic conditions. Phenotypic analysis showed catalase production but not indole positivity or nitrate reduction. Standard phenotypic tests were performed with the rapid ID 32A and api20A strips (bioMérieux, Marcy l'Etoile, France). The numeric profiles obtained were 0002000010 and 4030121, respectively. The bacteria produced acid from arabinose, glucose, mannose, and xylose, was negative for glycerol fermentation and leucine arylamidase and positive for rhamnose fermentation. Despite these results, phenotypic tests failed to identify the isolate. Antimicrobial drug susceptibility was determined by disk diffusion method and Etest for MICs. The isolate showed susceptibility to metronidazole (MIC <0.016 μg/mL), vancomycin, and colistin (MIC <0.016 μg/mL) and resistance to penicillin (MIC 2 μg/mL), gentamicin, netilmycin, kanamycin, amikacin, and cefotaxime (MIC >32 μg/mL) according to Eucast clinical breakpoints (www.eucast.org). No other bacteria were isolated in the blood cultures.

Genetic analysis was performed by 16S rRNA gene sequencing of a 1,265-bp fragment by using DG74 and RDRO80 primers (*2*). The nucleotide sequence obtained was compared with known sequences in GenBank by multiple sequence alignment using the ClustalW program (*3*). It was 100% identical to *C. hongkongensis* (GenBank accession no. AY574991).

The first 4 case-patients with *C. hongkongensis* infection were described by Lau et al. in 2007 (*1*). Two of these patients lived in Hong Kong and the 2 others in Canada. As in our case, only 1 patient died. Since there was a high degree of phenotypic and genetic difference with other anaerobic agents, the authors proposed a new genus and species and affiliation with a new family, *Catabacteriacae.* The 2 isolates from Canada differed from the 2 others by being negative for glycerol fermentation and positive for rhamnose fermentation and leucine arylamidase, similar to our case, except for leucine arylamidase, which in our case was negative. In the previously reported cases, *C. hongkongensis* was susceptible to metronidazole, vancomycin, and kanamycin; variably susceptible to penicillin (MICs 0.5–4.0 μg/mL); and resistant to colistin and cefotaxime (*1*).