often given with prednisolone, would have been necessary.

In pregnant women with West African (*T. brucei gambiense*) stage II disease, either melarsoprol or eflornithine can be used, but neither is effective for East African disease. Although eflornithine can abort early pregnancies and cause disordered organogenesis (*9*), the severe encephalopathy associated with melarsoprol makes eflornithine a preferable option for single-agent treatment. However, nifurtimox–eflornithine combination therapy will soon replace single-drug regimens for stage II *T. brucei gambiense* cases (*10*).

We believed evidence was insufficient to withhold suramin therapy for this highly fatal disease. Because of the uncertainty about effects of pregnancy on the ability to clear trypanosomes, the patient will be followed up for signs of relapse. The danger of HAT should be specifically highlighted for all travelers to trypanasomiasis-endemic regions, particularly pregnant travelers because of potential harm to unborn children.

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Rickettsia africae Infection in Man after Travel to Ethiopia

To the Editor: The first human case of African tick-bite fever was described in 1992 as occurring in Zimbabwe. The causative agent was identified as a new serotype of the spotted fever group (SFG) rickettsiae and named *Rickettsia africae* (*1*). These findings confirmed observations made by Pijper in the 1930s which suggested that there were 2 different kinds of human SFG rickettsioses in sub-Saharan Africa: Mediterranean spotted fever caused by *R. conorii* and transmitted by *Rhipicephalus* species, ticks of dogs, and African tick-bite fever caused by *R. africae* and transmitted by *Amblyomma* species, ticks of cattle and wild ungulates. African tick-bite fever has subsequently been diagnosed in patients from several other sub-Saharan countries and also from the West Indies (*2*,*3*).

In a recent analysis of the spectrum of diseases among returning travelers, tick-borne spotted fever was (after malaria) the second most frequent cause of systemic febrile illness among those returning from sub-Saharan Africa. It occurred more frequently than typhoid fever and dengue fever (*4*). The following case description reports an infection with *R. africae* in a man in France who recently returned from Ethiopia.

On November 4, 2005, a 62-year-old French man sought care at the Medical Center of the Institut Pasteur in Paris for fever, along with chills, headache, neck and shoulder pain, and fatigue over the previous 4 days. At the onset of these symptoms he had noticed dark nodular lesions on his neck and his left groin followed 2 days later by a slightly painful eruption on his arms and his trunk. He had spent a month in southwest Ethiopia, north of Kelem near the Sudanese bor-

der, and returned to France on October 26, 2005. While in Ethiopia, he had assisted with a production of a documentary film about an Ethiopian tribe and had been in contact with cattle in the villages. He had not noticed any tick bites. On physical examination he had a fever of 38°C, a nodular lesion with a central dark crust on his neck, a second lesion on his left inguinal fold (Figure, panel A), and a vesicular eruption on his arms and his trunk (Figure, panel B). Leukocyte count was 3,200, including 1,869 neutrophils and 867 lymphocytes. The platelet level was 174,000/mm^3. The C-reactive protein level was 28.3 mg/L. The aspartate aminotransferase level was slightly elevated. The patient was treated with doxycycline 200 mg/day for 1 week for suspected African tick-bite fever. Follow-up showed a quick recovery from his symptoms except for fatigue that persisted for ≈1 month.

A commercial immunofluorescence assay for *R. conorii* and *R. typhi* immunoglobulin G performed both on an initial blood sample and a second sample taken 1 week later were negative. A blood sample and a biopsy specimen of the inguinal eschar were sent to the National Reference Center of Rickettsiae in Marseille, France. Although cellular culture of both specimens and molecular testing of the blood sample were negative, PCR for the sequences of citrate synthase (GenBank accession no. RAU59733, 93.1% homology) and rickettsial OmpA (GenBank accession no. RAU83436, 99.3% homology) applied on the skin biopsy detected *R. africae* and confirmed the diagnosis of African tick-bite fever.

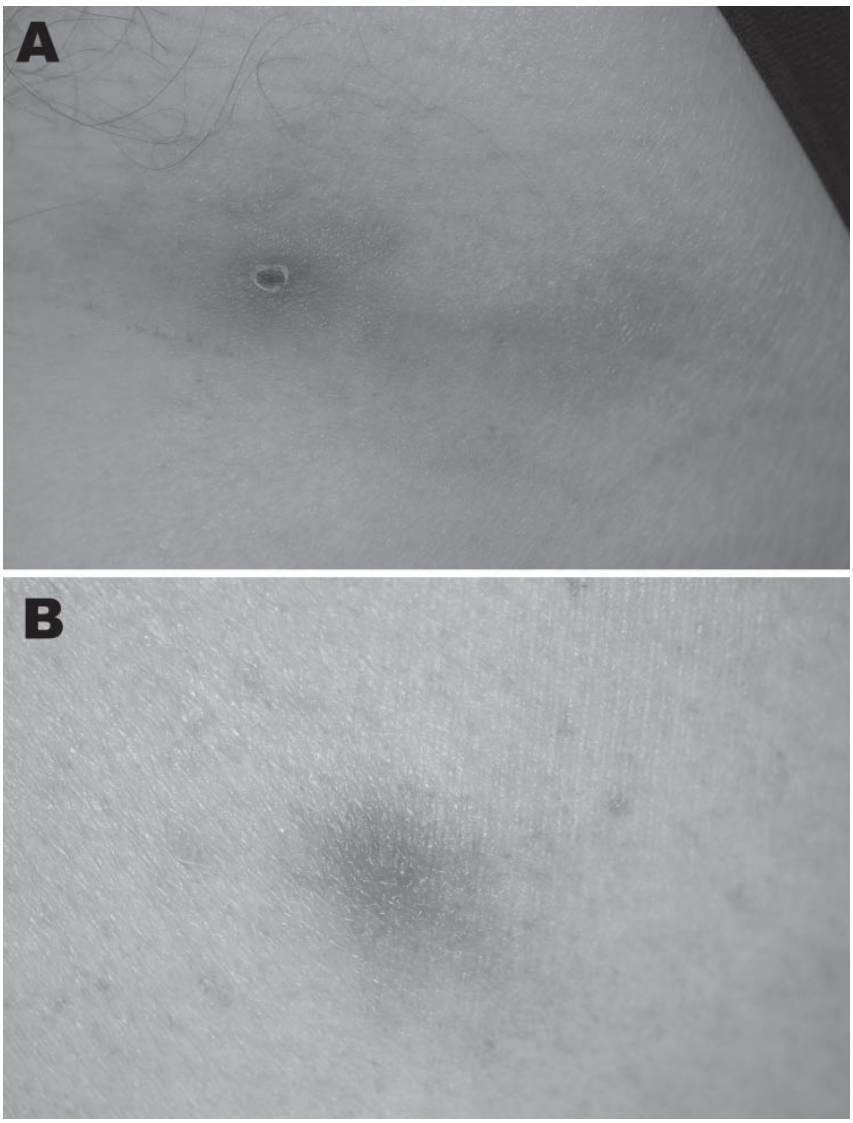

Figure. Inoculation eschar on left inguinal fold (A) and vesicular skin lesion (B) in a traveler recently returned to France from Ethiopia.

From 1969 to 1971, SFG rickettsiae were isolated from *Amblyomma* spp. ticks collected in Ethiopia. They were regarded as *R. conorii* or as closely related bacteria (*5*). Later, more specific tests using western immunoblots with monoclonal antibodies showed that these rickettsiae differed from *R. conorii* (*6*). In 1992 SFG rickettsiae isolated from *Amblyomma* ticks collected in Zimbabwe and from the blood of a patient in Zimbabwe were compared to *R. conorii*, to other pathogenic SFG rickettsiae, and to a SFG rickettsia isolated from an *Amblyomma* spp. tick in Ethiopia 20 years before. The SFG rickettsia isolates from Ethiopia were identical to isolates obtained in Zimbabwe from the *Amblyomma* ticks and the patient's blood and were different from *R. conorii* and other pathogenic SFG rickettsiae. This new serotype of SFG rickettsiae was named *R. africae* (*1*,*7*). A recent study confirmed the presence of *R. africae* in ticks collected in Ethiopia, as well as *R. aeschlimanii* (*8*). Thus, evidence of *R. africae* in Ethiopia has been known for a long time.

The geographic distribution of African tick-bite fever is related to the presence of *Amblyomma* spp. ticks, vectors and reservoirs of *R. africae*. Consequently African tick-bite fever should also be considered as a possible diagnosis in patients with febrile illness returning from countries where *R. africae* has been detected in *Amblyomma* ticks, even if a human infection has not yet been reported (*9*,*10*).

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Rickettsia massiliae in the Canary Islands

To the Editor: *Rickettsia massiliae* was recently recognized as a human tick-borne spotted fever group rickettsia (*1*). We report the finding of *R. massiliae* in *Rhipicephalus pusillus* ticks from Gran Canaria, Canary Islands, Spain. Introduction of this pathogen into the Canary Islands is thought to have resulted from translocation of the European wild rabbit *Oryctolagus cuniculus* (Linnaeus), a preferred host of *R. pusillus* ticks (www.kolonin.org/16_4.html), from the Iberian Peninsula 600 years ago (*2*).

We collected questing adult ticks in 2008 in Gran Canaria and identified 2 tick species, *Hyalomma lusitanicum* (n = 82 [46 females]) and *R. pusillus* (n = 8 [5 females]). Whole ticks were preserved in 70% ethanol and used for DNA extraction by using TriReagent (Sigma, St. Louis, MO, USA) according to the manufacturer's instructions. We identified rickettsial sequences by using PCR primers that amplify fragments of 16S rRNA, *ompB, atpA, dnaA, dnaK*, and *recA* genes (Table). Amplicons were cloned into pGEM-T (Promega, Madison, WI, USA), and 3 independent clones were sequenced from both ends for each gene marker. Sequence similarity search was performed by using BLAST (www.ncbi.nlm.nih.gov). Rickettsial DNA was detected in 2 *R. pusillus* males only; sequences were identical in both ticks. Fragments of 16S rRNA were 99% identical to the *R. massiliae* strain Mtu5 (CP000683) isolated from *R. sanguineus* ticks in southern France (*3*), and fragments of *ompB, atpA, dnaA, dnaK*, and *recA* genes were 100% identical to the *R. massiliae* strain Bar29 (AF123710, AY124739, DQ821798, DQ821828, and AY124750, respectively), previously isolated from *R. sanguineus* ticks in Catalonia, Spain (*4*) (Table).

R. massiliae was first isolated in 1992 from *R. sanguineus* ticks collected near Marseille, France (*5*). Since then, the pathogen has been identified in different *Rhipicephalus* species in France, Greece, Portugal, Switzerland, Spain, North and Central Africa, Argentina, and the United States (*6*,*7*). *R. massiliae* has been identified in southern Spain (*8*) but not in the Canary Islands. *R. pusillus* ticks are commonly found in southern Europe (Portugal, Spain, and France) and northern Africa (Tunisia and Morocco). All stages of these ticks inhabit burrows of wild rabbits and feed on them (www.kolonin.org/16_4.html).

Wild rabbits were introduced into the Canary Islands at the end of 14th century during colonization by the kingdom of Castilla. Colonists were asked to bring rabbit couples with them to provide food in the islands (*2*), a practice continued by new colonists because of their interest in hunting this rabbit species. Introduction of wild rabbits by colonists led to establishment of parasites, such as helminths, coccidia, and viruses in the Canary Islands (9). *R. pusillus*, a common ectoparasite (tick) that feeds on wild rabbits on the Iberian Peninsula, was also introduced this way. *R. massiliae* could have been introduced in the islands by infected *R. pusillus* ticks or by infected wild rabbits if this species serves as a natural reservoir host for the pathogen.

To find evidence for this hypothesis, we tested blood and liver samples of 150 wild rabbits from both Canary Islands and Andalucía (southern Spain) by using *Rickettsia*-specific PCR primers (Table). No *R. massiliae* DNA was detected in the rabbit samples tested,