References

1. Gottschalk M. Streptococcocis. In: Straw BE, Zimmerman JJ, D'Allaire S, Taylor DJ, editors. Diseases of swine. 10th ed. Ames (IA): Blackwell Publishing; 2012. p. 841–55.
2. Wertheim HF, Nghia HD, Taylor W, Schultsz C. *Streptococcus suis*: an emerging human pathogen. Clin Infect Dis. 2009;48:617–25. http://dx.doi.org/10.1086/596763
3. Nghia HD, Tu le TP, Wolbers M, Thai CQ, Hoang NV, Nga TV, et al. Risk factors of *Streptococcus suis* infection in Vietnam. A case–control study. PLoS ONE. 2011;6:e17604. http://dx.doi.org/10.1371/journal.pone.0017604
4. Brousseau R, Hill JE, Prefontaine G, Goh SH, Harel J, Hemmingsen SM. *Streptococcus suis* serotypes characterized by analysis of chaperonin 60 gene sequences. Appl Environ Microbiol. 2001;67:4828–33. http://dx.doi.org/10.1128/AEM.67.10.4828-4833.2001
5. Gottschalk M, Higgins R, Boudreau M. Use of polyvalent coagglutination reagents for serotyping of *Streptococcus suis*. J Clin Microbiol. 1993;31:2192–4.
6. Gottschalk M, Higgins R, Jacques M, Mittal KR, Henrichsen J. Description of 14 new capsular types of *Streptococcus suis*. J Clin Microbiol. 1989;27:2633–6.
7. Gottschalk M, Lacouture S, Bonifait L, Roy D, Fittipaldi N, Grenier D. Characterization of *Streptococcus suis* isolates recovered between 2008 and 2011 from diseased pigs in Quebec, Canada. Vet Microbiol. 2013;162:819–25. http://dx.doi.org/10.1016/j.vetmic.2012.10.028
8. Lopreto C, Lopardo HA, Bardi MC, Gottschalk M. Primary *Streptococcus suis* meningitis: first case in humans described in Latin America [in Spanish]. Enferm Infecc Microbiol Clin. 2005;23:110. http://dx.doi.org/10.1157/13071618
9. Nagel A, Manias V, Busquets N, Sniadowsky S, Anzardi J, Mendez Ede L. *Streptococcus suis* meningitis in an immunocompetent patient [in Spanish]. Rev Argent Microbiol. 2008;40:158–60.
10. François B, Gissot V, Ploy MC, Vignon P. Recurrent septic shock due to *Streptococcus suis*. J Clin Microbiol. 1998;36:2395.

Address for correspondence: Marcelo Gottschalk, Department of Pathology and Microbiology, University of Montreal, 3200 Sicotte, St-Hyacinthe, Québec J2S 2M2, Canada; e-mail: marcelo.gottschalk@umontreal.ca

Use of trade names is for identification only and does not imply endorsement by the Public Health Service or by the US Department of Health and Human Services.

Cutaneous Leishmaniasis Caused by *Leishmania killicki*, Algeria

To the Editor: Cutaneous leishmaniasis (CL) is a widespread and resurging vector-borne disease caused by a protozoan parasite belonging to genus *Leishmania* (*1*). After Afghanistan, Algeria is the second largest focus of CL in the world. Although CL is a serious public health problem in Algeria, few data are available from this country.

During 2004–2008, an average of ≈44,050 CL cases were reported per year, and the estimated annual incidence ranged from 123,300 to 202,600 cases. Two main forms of CL have been described for more than a century in Algeria, the zoonotic, caused by *L. major* and the sporadic, caused by *L. infantum*. Since 2004, 11 strains belonging to the *L. tropica* complex, including *L. killicki* (*2*), were identified in 1 focus in the northern part of the Sahara (*3*) and in 2 foci in the northeastern Algeria (*4*,*5*). We report here a recent outbreak of CL, including infection with *L. killicki* strains, in the Tipaza area of northern Algeria.

Patients who sought treatment at Hajout hospital in Hajout, Algeria (a community of ≈51,000 persons), from January 2010 through April 2013 with cutaneous lesions consistent with leishmaniasis, underwent clinical examination. For each patient (146 total), we collected epidemiologic data (geographic origin, traveling history, especially to other leishmaniasis-endemic areas) and clinical data (number and size of lesions and clinical forms). Informed consent was obtained from all patients or their legal guardians. A particular characteristic of the infections was the unusual duration of some episodes, one of which persisted for >4 years, which is compatible with leishmaniasis recidivans (*6*).

Microbiological data were obtained as follows. Tissue samples, obtained by scraping the internal border of skin lesions from patients, were smeared onto a glass slide, fixed with methanol, stained with Giemsa, and examined by microscopy. Slides showing *Leishmania* amastigote forms were then processed further for molecular analyses. The immersion oil used to examine each slide was wiped off the smear with tissue paper, and then the dry smear was scraped from its slide by using a sterile scalpel. DNA extraction from smear scrapings was performed with the NucleoSpin Tissue kit (Macherey-Nagel, Düren, Germany). Species identification was performed by amplifying the topoisomerase II gene, followed by DNA sequencing (*7*).

In total, 60 patients exhibited *Leishmania*-positive cutaneous lesions as determined by microscopy. The topoisomerase II gene was successfully amplified and sequenced from samples from 38 patients. *Leishmania* species were identified by comparing sequences with those of the reference strains *L. infantum* MHOM/FR/78/LEM75, *L. killicki* MHOM/TN/80/LEM163, and *L. major* MHOM/MA/81/LEM265 (*7*). *L. infantum* was identified in 36 cases and *L. killicki* in 2 cases (Figure). No *L. major* isolates were found in this series.

The low proportion of *L. killicki* strains was similar to that found recently in the Annaba focus in northeastern Algeria (*5*). However, the observation of a new focus of CL and *L. killicki* as etiologic agent may indicate a modification of the epidemiology of CL in Algeria. This focus, located far from other previously described areas where the *L. tropica* complex is endemic, may reflect geographic spread of this complex in Algeria.

The results of this study can be placed in a larger framework as well. Since 2004, strains in the *L. tropica* complex have been increasingly reported as responsible for CL

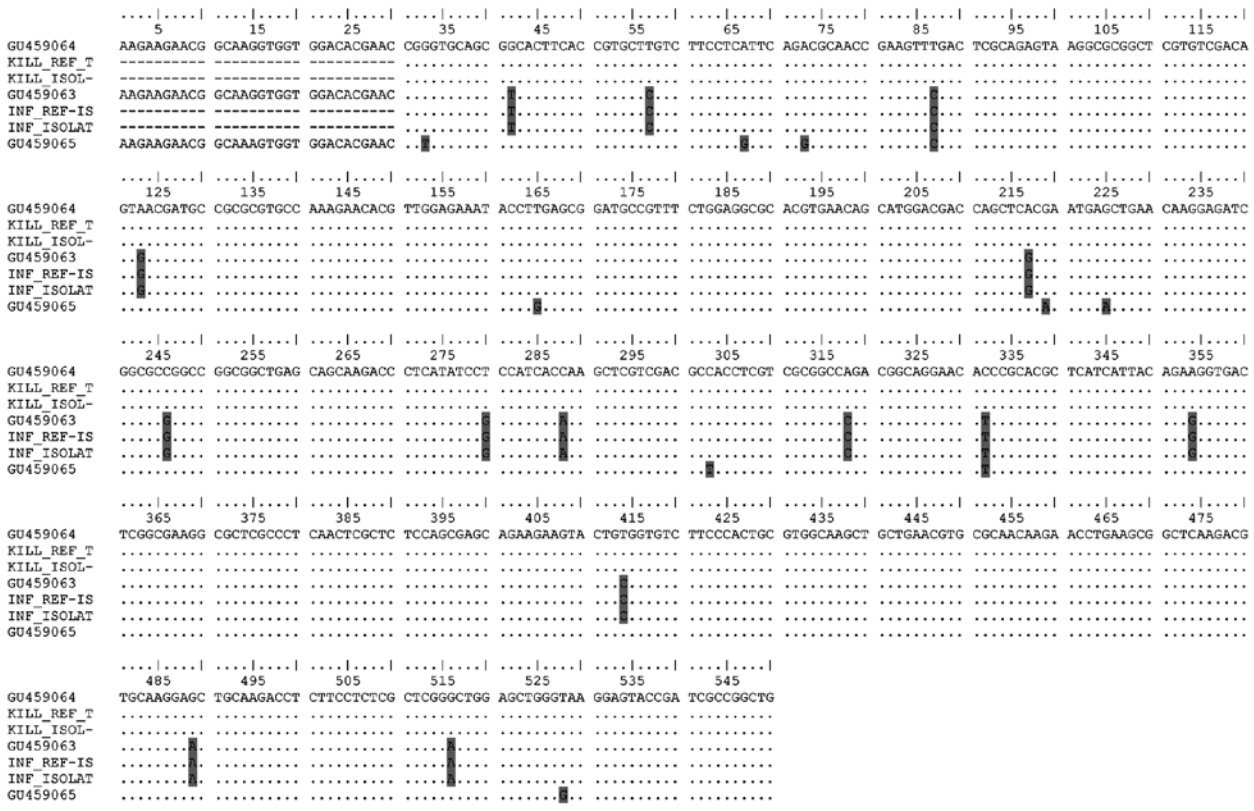

Figure. Alignment of topoisomerase II nucleotide sequences of *Leishmania killicki*, *L. infantum*, and *L. major*. Point mutations discriminating *Leishmania* species are outlined on a gray background. The references strains are GU459063: *L. infantum* MHOM/FR/78/LEM75; GU459064: *L. killicki* MHOM/TN/80/LEM163; GU459065: *L. major* MHOM/MA/81/LEM265 ; KILL_REF_T: *L. killicki* and INF_REF-IS: *L. infantum*, strains genotyped by the *Leishmania* National Reference Center, Montpellier, France. The isolates are: KILL_ISOL-: *L. killicki* (n = 2); INF_ISOLAT: *L. infantum* (n = 36).

in Mediterranean countries, in the Near East and Middle East (*2*), possibly in relation to changes in environmental conditions. Urbanization and/or climatic changes that have occurred in recent years could have played a role in the spread of the disease. The cases reported here were observed in urban areas, which suggests transmission according to an anthroponotic mode.

Each species responsible for CL has its own epidemiologic pattern. Clinicians must be aware of the specificity of leishmaniases that may be encountered in North African countries. *L. tropica* complex lesions heal spontaneously over a period of 12 months or more, a duration longer than for *L. major* infections (*8*). *L. tropica* infections are also less responsive to treatment compared to infections with other Old World *Leishmania* species. In addition, *L. tropica* may cause leishmaniasis recidivans. This type of CL, appearing often years after the initial infection showed signs of complete resolution, manifests as papules that transform slowly into a spreading granuloma resembling lupus vulgaris (*6*). *L. tropica* can also produce visceral infections on rare occasions, resulting in unexplained systemic illness, including classic symptoms of visceral leishmaniasis, in persons returning from areas where this *Leishmania* complex is endemic (*9*).

Other epidemiologic studies are required to detect additional foci, including those of the *L. tropica* complex, that may coexist with those of *L. infantum* and *L. major* in Algeria. Travelers to North Africa should also be informed about the existence of this spreading disease (*10*).

Arezki Izri, Amina Bendjaballah, Valérie Andriantsoanirina, and Rémy Durand

Author affiliations: Hôpital Avicenne–Assistance Publique-Hôpitaux de Paris, Bobigny, France (A. Izri, V. Andriantsoanirina, R. Durand); and Hôpital de Hadjout, Hadjout, Algeria (A. Bendjaballah)

DOI: http://dx.doi.org/10.3201/eid2003.131152

References

1. Alvar J, Vélez ID, Bern C, Herrero M, Desjeux P, Cano J, et al. Leishmaniasis worldwide and global estimates of its incidence. PLoS ONE. 2012;7:e35671. http://dx.doi.org/10.1371/journal.pone.0035671

2. Pratlong F, Dereure J, Ravel C, Lami P, Balard Y, Serres G, et al. Geographical distribution and epidemiological features of Old World cutaneous leishmaniasis foci, based on the isoenzyme analysis of 1048 strains. Trop Med Int Health. 2009;14:1071–85. http://dx.doi.org/10.1111/j.1365-3156.2009.02336.x
3. Harrat Z, Boubidi SC, Pratlong F, Benikhlef R, Selt B, Dedet JP, et al. Description of a dermatropic *Leishmania* close to *L. killicki* (Rioux, Lanotte & Pratlong 1986) in Algeria. Trans R Soc Trop Med Hyg. 2009;103:716–20. http://dx.doi.org/10.1016/j.trstmh.2009.04.013
4. Mihoubi I, Picot S, Hafirassou N, de Monbrison F. Cutaneous leishmaniasis caused by *Leishmania tropica* in Algeria. Trans R Soc Trop Med Hyg. 2008;102:1157–9. http://dx.doi.org/10.1016/j.trstmh.2008.06.013
5. Mansouri R, Pratlong F, Bachi F, Hamrioui B, Dedet JP. The first isoenzymatic characterizations of the *Leishmania* strains responsible for cutaneous leishmaniasis in the area of Annaba (Eastern Algeria) [cited 2014 Jan 16]. The Open Conference Proceedings Journal. 2012;3 (Suppl.2–M2):6–11. http://www.benthamscience-publisher.com/open/toprocj/articles/V003/SS0001TOPROCJ/6TOPROCJ.pdf
6. Klaus S, Frankenburg S. Cutaneous leishmaniasis in the Middle East. Clin Dermatol. 1999;17:137–41. http://dx.doi.org/10.1016/S0738-081X(99)00006-1
7. Haouas N, Garrab S, Gorcii M, Khorchani H, Chargui N, Ravel C, et al. Development of a polymerase chain reaction-restriction fragment length polymorphism assay for *Leishmania major*/*Leishmania killicki*/*Leishmania infantum* discrimination from clinical samples, application in a Tunisian focus. Diagn Microbiol Infect Dis. 2010;68:152–8. http://dx.doi.org/10.1016/j.diagmicrobio.2010.06.011
8. Morizot G, Kendjo E, Mouri O, Thellier M, Pérignon A, Foulet F, et al. Travelers with cutaneous leishmaniasis cured without systemic therapy. Clin Infect Dis. 2013;57:370–80. http://dx.doi.org/10.1093/cid/cit269
9. Magill AJ, Grogl M, Gasser RA, Sun W, Oster CN. Visceral infection caused by *Leishmania tropica* in veterans of Operation Desert Storm. N Engl J Med. 1993;328:1383–7. http://dx.doi.org/10.1056/NEJM199305133281904
10. Maubon D, Thurot-Guillou C, Ravel C, Leccia MT, Pelloux H. *Leishmania killicki* imported from Tunisian desert. Emerg Infect Dis. 2009;15:1864–5. http://dx.doi.org/10.3201/eid1511.090148

Address for correspondence: Rémy Durand, Service de Parasitologie-Mycologie, Hôpital Avicenne, 125 rue de Stalingrad 93009 Bobigny Cedex, France; email: remy.durand@avc.aphp.fr

Rift Valley Fever in Kedougou, Southeastern Senegal, 2012

To the Editor: Rift Valley fever (RVF) is an acute, febrile, viral disease caused by Rift Valley fever virus (RVFV), a phlebovirus of the family *Bunyaviridae* that is endemic to sub-Saharan Africa. RVF mortality and abortion rates among young domesticated ruminants and pregnant females are high.

In humans, clinical manifestations range from mild to severe syndromes, which can include neurologic, hemorrhagic, and hepatic features and retinitis, and which sometimes result in death (*1*). Diagnosis of RVF is challenging for clinicians because clinical manifestations are not specific (*2*). Heavy rainfall and flooding create conditions for emergence of RVF vectors (*Aedes* and *Culex* spp. mosquitoes), and dispersion of this disease into new areas is linked to migration of infected livestock, wildlife, or mosquitoes.

Since 1987, when the Diama dam was built, RVF outbreaks in Mauritania have been reported regularly (*3*). In Kedougou, southeastern Senegal, RVFV was isolated 4 times from *Ae. dalzieli* mosquitoes and once from a person with a mild case of RVF (*4*). We report results of a field investigation and laboratory findings for a human case of RVF detected by surveillance of acute febrile illnesses in Kedougou.

On October 16, 2012, a 27-year-old man (school teacher) who lived and worked in Baya village in the Kedougou region of Senegal (12°27′50″N, 12°28′6″W) visited the Kedougou military health post because of high fever, chills, headache, back pain, myalgia, and arthralgia that started on October 14. He reported regular contact with domesticated animals (cows, sheep, and goats) during farming.

A thick blood smear for the patient showed a positive result for malaria, and specific treatment was given. As part of surveillance for acute febrile illnesses, blood samples from the patient were tested for IgM against RVF, chikungunya, dengue, West Nile, yellow fever, Zika, and Crimean-Congo hemorrhagic fever viruses; and for viral RNA and virus (*5*,*6*). All test results for IgM against the 7 viruses were negative

RVFV was isolated from newborn mice that were intracerebrally inoculated with a blood sample from the patient. Viral RNA was detected by reverse transcription PCR in serum from the patient. Phylogenetic analysis of the partial nonstructural protein gene on the small RNA segment showed that the RVFV isolate was closely related to a strain that had circulated in Mauritania in 2012 (Figure).

An epidemiologic field investigation was conducted to assess the extent of RVFV circulation. During this investigation, the case-patient provided an additional blood sample. In addition, 115 contacts of the case-patient, including primary school students, friends, family members and neighbors (median age 12 years, range 6–75 years; female:male sex ratio 1.6) were also sampled and questioned to identify asymptomatic and benign cases. A total of 218 samples from patients attending the nearest health posts in Ibel and Thiokoye villages during October 2012 were also tested during surveillance of acute febrile illnesses.

All 334 samples were negative for RVFV RNA and IgM and IgG against RVFV except for samples from 3 patients, including the case-patient, which were positive for RVFV-specific IgG and malaria parasites. The 2 other patients were a 32-year-old tradesman and a 20-year-old housewife sampled during surveillance of acute febrile illnesses in Kedougou and Bandafassi, which is 30 km from Baya (online Technical Appendix Figure, wwwnc.cdc.gov/EID/article/20/3/13-1174-Techapp1.pdf). No RVFV RNA was detected