



Figure. Leptospirosis cases (N = 291) in Taiwan, 2001–2006. A) Distribution by age and sex. B) Relationship between rainfall and leptospirosis incidence. Data represent averages for each month during the 6 years.

sociated with several days of heavy rainfall from typhoons. Therefore, the typhoons may be the reason for high incidence in October–December.

The annual incidence of leptospirosis in Taiwan is relatively lower than that in other countries with tropical or subtropical climates. Our study does not conclusively document the reason for lower incidence, although it does suggest an association between amount of rainfall and incidence. An understanding of the relationship between leptospirosis incidence and rainfall is indispensable for implementing appropriate preventive measures.

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References

- Levett PN. Leptospirosis. *Clin Microbiol Rev.* 2001;14:296–326.
- Vinetz JM. Leptospirosis. *Curr Opin Infect Dis.* 2001;14:527–38.
- Bharti AR, Nally JE, Ricaldi JN, Matthias MA, Diaz MM, Lovett MA, et al. Leptospirosis: a zoonotic disease of global importance. *Lancet Infect Dis.* 2003;3:757–71.
- World Health Organization. Human leptospirosis: guidance for diagnosis surveillance and control. Geneva: The Organization; 2003. p. 109.
- Michel V, Branger C, Andre-Fontaine G. Epidemiology of leptospirosis. *Rev Cubana Med Trop.* 2002;54:7–10.
- Trejevo RT, Rigau-Perez JG, Ashford DA, McClure EM, Jarquin-González C, Amador JJ, et al. Epidemic leptospirosis associated with pulmonary hemorrhage—Nicaragua, 1995. *J Infect Dis.* 1998;178:1457–63.
- Narita M, Fujitani S, Haake DA, Paterson DL. Leptospirosis after recreational exposure to water in the Yaeyama Islands, Japan. *Am J Trop Med Hyg.* 2005;73:652–6.
- Ku TY, Lin CC. Abundance and distribution of field rodents in Taiwan. *Plant Prot Bull.* 1980;22:397–420.
- Lu KH. 2004. Strategy of field rodent control in Taiwan. In: Proceedings of seminar on management of plant health. Taiwan. p. 259–76.
- Smits HL, van der Hoorn MA, Goris MG, Gussenhoven GC, Yersin C, Sasaki DM, et al. Simple latex agglutination assay for rapid serodiagnosis of human leptospirosis. *J Clin Microbiol.* 2000;38:1272–5.

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Leishmania (*Leishmania*) *amazonensis* Infection, Suriname

To the Editor: A 17-year-old man was seen at the Dermatology Service in Paramaribo (Suriname) with a skin condition that he had had since he was 5 years of age. The condition consisted of multiple cutaneous ulcerations, nodules, and fibrotic plaques disseminated on his face, limbs, and trunk, and subcutaneous nodules on lymph-draining tracts on his hands, arms, and legs (online Appendix Figure, panel A, available from www.cdc.gov/EID/content/14/5/857-appG.htm). He had lived his entire life in an inland village, located at Brokopondo Lake (central-eastern Suriname); he had never traveled outside the country. The diagnosis of cutaneous leishmaniasis (CL, a parasitic disease caused by the protozoa *Leishmania*) was presumed. The patient received pentamidine therapy in 1997, 1998, and 2005, but without sustained clinical effect. Rapid screening tests for HIV were negative (Determine [Abbott Laboratories, Tokyo,

Japan] and Unigold [Trinity Biotech, Co. Wicklow, Ireland]). In 2006, the diagnosis of CL was confirmed with histopathology, culture, and PCR. The parasite was identified by a PCR restriction fragment length polymorphism method on the small subunit-internal transcribed spacer genes (1) and by multilocus enzyme electrophoresis at the National Reference Center of *Leishmania* (Montpellier, France).

After promising results were obtained with miltefosine in a patient with anergic diffuse cutaneous leishmaniasis (ADCL) in Venezuela (2), the patient received 150 mg/day oral miltefosine (Impavido, Zentaris, Germany) for 98 days and the lesional parasite load was quantified with quantitative nucleic acid sequence-based amplification (3). Skin biopsy specimens were collected from 1 target lesion before treatment; during treatment at day 14, day 28, day 42 (all in duplicate); and at day 70 (single biopsy).

The strain causing infection (MHOM/SR/2006/SP100) was identified as *Leishmania (Leishmania) amazonensis*, and the enzymatic profile was equal to *L. (L.) amazonensis* zymodeme MON-41. Histopathology showed large macrophages containing abundant *Leishmania* amastigotes and few lymphocytes and plasma cells without granuloma formation. A considerable clinical improvement was observed during the first 2 months of therapy. The lesions slowly decreased in size and duration. At day 70, all ulcerative lesions were re-epithelialized, without signs of infiltration or lymphangitis (online Appendix Figure, panel B). At start of treatment, parasite counts of 360,000 and 310,000 parasites per biopsy were detected; these counts decreased to 0 parasites/biopsy at day 70. Histopathologic studies at day 70 showed no *Leishmania* bodies, a dense lymphocytic and plasma cellular infiltrate, and fibrosis. Apart from mild elevation of creatine and urea during treatment, no subjective or adverse side effects were reported.

L. (L.) amazonensis causes CL and 2 very serious manifestations of CL, disseminated cutaneous leishmaniasis (DCL) and ADCL (4). Both forms are histopathologically characterized by heavily parasitized macrophages and an absence of cell-mediated immune responses in therapy-naïve patients (4). ADCL is resistant to any form of therapy, and cell-mediated immune responses never seem to occur. In contrast, the cell-mediated immune response in DCL can eventually arise upon therapy response, even in patients with previous therapy failures (4). The therapy response in DCL patients is histopathologically characterized by the appearance of a lymphocytic and plasma cellular infiltrate. The diagnosis of DCL is plausible in our patient based on the histopathologic findings before, during, and after therapy; the clinical picture (erythematous infiltrated plaques, lymphadenitis, and lymphangitis), and the favorable therapy response. He was last seen 7 months after end of therapy, at which time new lesions had not developed.

In general, *L. (L.) amazonensis* infection is rare in humans (5). In French Guiana, bordering the eastern side of Suriname, few patients ($\approx 1.9\%$) are reported to be infected with this species (5). However, the sandfly vector of *L. (L.) amazonensis*, *Lutzomyia flaviscutellata*, was detected earlier in Suriname (6), which may indicate transmission of *L. (L.) amazonensis* infection to humans by means of the bite of this sandfly in Suriname. Our patient had no history of transfusion or intravenous drug use.

Many gold diggers from the northern part of Brazil work and travel in Suriname and are familiar with CL. In the Brazilian State Pará, a region bordering Suriname in the South, the infection rate with *L. (L.) amazonensis* is high (34.8%) (7). It is thus conceivable that infected gold diggers from that area have introduced *L. (L.) amazonensis* into Suriname. Our patient used to live in a village where many Brazilian

gold diggers worked around the time that his skin lesions developed. Migration of laborers is associated with an increased risk for CL infection (8). The zymodeme MON-41 is widespread in Central America and the northern part of South America, and has been reported in Venezuela, Brazil, Panama, French Guiana, and Colombia (F. Pratlong and J.P. Dedet, Montpellier International Cryobank of *Leishmania*, pers. comm., 2007). Therefore, speculations on the exact origin of the infection need to be made cautiously.

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References

1. Rotureau B, Ravel C, Couppié P, Pratlong F, Nacher M, Dedet JP, et al. Use of PCR-restriction fragment length polymorphism analysis to identify the main New World *Leishmania* species and analyze their taxonomic properties and polymorphism by application of the assay to clinical samples. *J Clin Microbiol*. 2006;44:459–67.
2. Zerpa O, Blanco B, Kannee C, Ulrich M, Sindermann H, Engel J, et al. Treatment of diffuse cutaneous leishmaniasis with miltefosine: a case report. *Int J Dermatol*. 2006;45:751–3.

3. van der Meide WF, Schoone GJ, Faber WR, Zeegelaar JE, de Vries HJ, Ozbel Y, et al. Quantitative nucleic acid sequence-based assay as a new molecular tool for the detection and quantification of *Leishmania* parasites in skin biopsies. *J Clin Microbiol*. 2005;43:5560–6.
4. Silveira FT, Lainson R, Corbett CE. Further observations on clinical, histopathological, and immunological features of borderline disseminated cutaneous leishmaniasis caused by *Leishmania (Leishmania) amazonensis*. *Mem Inst Oswaldo Cruz*. 2005;100:525–34.
5. Rotureau B, Ravel C, Nacher M, Couppié P, Curtet I, Dedet JP, et al. Molecular epidemiology of *Leishmania (Viannia) guyanensis* in French Guiana. *J Clin Microbiol*. 2006;44:468–73.
6. Burgos A, Hudson J. Annotated list of the phlebotominae (diptera) of Suriname. *Mem Inst Oswaldo Cruz*. 1994;89:171–8.
7. Silveira FT, Lainson R, Shaw JJ, De Souza AA, Ishikawa EA, Braga RR. Cutaneous leishmaniasis due to *Leishmania (Leishmania) amazonensis* in Amazonian Brazil, and the significance of a negative Montenegro skin-test in human infections. *Trans R Soc Trop Med Hyg*. 1991;85:735–8.
8. Rotureau B, Joubert M, Clyti E, Djossou F, Carne B. Leishmaniasis among gold miners, French Guiana. *Emerg Infect Dis*. 2006;12:1169–70.

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Household Transmission of Carbapenemase- producing *Klebsiella* *pneumoniae*

To the Editor: Since its first description in 2001, carbapenemase-producing *Klebsiella pneumoniae* has become a frequent nosocomial pathogen in the eastern United States (1).

This bacterium was introduced into Israel in 2005 and is endemic now in several hospitals in the country (2). We recently documented transmission of this organism within a household, the source being a debilitated patient who returned home after a long hospitalization.

A 73-year-old man had a urologic procedure (transurethral resection of the bladder neck) in a community hospital in early October 2007. He was initially evaluated on September 23, 2007, at an outpatient clinic where a routine urine sample was obtained for culture. Carbapenemase-producing *K. pneumoniae* was cultured. Identification and susceptibility testing of the isolate were completed by using the VITEK 2 system (bioMérieux, Marcy l'Etoile, France). *K. pneumoniae* carbapenemase was confirmed by using the modified Hodge test (3). Two repeat urine cultures grew the same organism; however, a stool culture was negative for carbapenemase-producing *K. pneumoniae*.

The medical history of the patient included hypertension and carcinoma of the prostate gland that was treated with high-intensity focused ultrasound in May 2007, followed by transurethral resection of prostate in June 2007. The 2 procedures were performed in 2 different private hospitals, and each required a 24-hour hospitalization. No carbapenemase-producing *K. pneumoniae* was documented in these hospitals. Two months before detection of carbapenemase-producing *K. pneumoniae*, the patient received a 1-week course of oral amoxicillin-clavulanate for presumed urinary tract infection, although urine culture obtained on July 29, 2007 was sterile. A repeat urine culture 2 weeks later (August 13, 2007) remained sterile.

Because the circumstances of strain acquisition and patient characteristics were not typical for epidemiology of carbapenemase-producing *K. pneumoniae* (3), he was further questioned about possible contacts of

relevance. The patient disclosed that his wife, who had amyotrophic lateral sclerosis that required mechanical ventilation, had been hospitalized in a tertiary hospital in the Tel Aviv area for 9 weeks until July 19, 2007. After discharge, she has been staying at home where she was cared for by her son, sister, and nurses; the patient stated that he had limited contact with his wife (he did not participate in her care). The infection control unit of the tertiary hospital was contacted, and the name of the wife was identified in the hospital registry. Carbapenemase-producing *K. pneumoniae* was isolated from her urine on June 8, 2007.

Despite limited contact, the patient probably acquired carbapenemase-producing *K. pneumoniae* from his wife, who was a documented carrier of this organism. Because his early urine cultures (taken after his wife was discharged from hospital) were sterile, we can assume that the transmission of the organism occurred at their home. We cannot rule out that the strain was transferred by an intermediary, such as the couple's son. It is unlikely that the organism was acquired at the private hospitals from which no case of carbapenemase-producing *K. pneumoniae* was reported (in Israel reporting carbapenemase-producing *K. pneumoniae* isolates to health authorities is mandatory). Also, the patient had 2 negative urine cultures.

Carbapenemase-producing *K. pneumoniae* is a recent addition to the pool of multidrug-resistant nosocomial pathogens. Most publications on this organism have focused on issues of structural and molecular epidemiology. Little is known regarding clinical characteristics and importance of infection with this organism. Until now, the strain has been recovered only from hospitalized patients with a longer hospital stay, those given multiple antimicrobial drug courses, and those mechanically ventilated (3,4). The strain can colonize the urinary,