mammalian hosts (*9*). Drug resistance or sensitivity is based on sequences of M2 and NA. Substitution within residues including L26I, V27A/I, A30S, and S31N of the M2 ion channel protein was used to predict amantadine-resistant mutants, and H274Y of the NA was used to predict for oseltamivir resistance (*10*). The virus observed in 2006 isolates from Phichit was resistant to amantadine but sensitive to oseltamivir, whereas the isolate from Nakhon Phanom was sensitive to amantadine and oseltamivir, which implies that infected patients received different antiviral drugs.

According to previous World Health Organization reports, the HA sequences of most influenza (H5N1) viruses that circulated in avian species during the past 3 years are separated into 2 distinct phylogenetic clades. Clade 1 viruses that circulated in Cambodia, Thailand, and Vietnam were responsible for human infections in those countries during 2004 and 2005. Clade 2 viruses that circulated in birds in People's Republic of China and Indonesia during 2003–2004 and 2005–2006 spread westward to the Middle East, Europe, and Africa. This latter genetic group of viruses has been principally responsible for human infections during late 2005 and 2006 (*11*). The latest wave of the outbreaks in Thailand was caused by viruses closely related to those that caused outbreaks in Thailand in 2004–2005 and to viruses recently circulating in southeast People's Republic of China and other Southeast Asian countries. This finding raises concern for development of new candidate influenza (H5N1) vaccine strains. Geographic spreading, epidemiology, and genetic properties of recently circulating influenza (H5N1) viruses should be considered when developing candidate H5N1 strains of influenza vaccine.

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Kala-azar Control, Uganda

To the Editor: Much of the leishmaniasis in Africa is concentrated in East Africa. In this region, visceral leishmaniasis (kala-azar) is caused by *Leishmania donovani* and is endemic in remote parts of Somalia, Sudan, Ethiopia, Kenya, and Uganda (*1*).

In Uganda, kala-azar is transmitted by the sandfly *Phlebotomus martini,* and transmission is thought to be anthroponotic. Studies in Sudan and Kenya have detected *L. donovani* in domestic animals (*2*,*3*), but whether these play a role in Uganda is

unknown. In Uganda, the disease appears to be restricted to Pokot County, a semiarid lowland area in Nakapiripirit District (see online map in Appendix Figure, available at www.cdc.gov/EID/content/13/3/507-appG.htm). This focus is an extension of a larger focus in West Pokot District in Kenya (*4*). The area is mainly inhabited by the Pokot, a seminomadic tribe of pastoralists. Nakapiripirit is one of the most underserved districts of Uganda, plagued by tribal clashes.

Though kala-azar has been reported in East Africa since the early 1900s, it was not described in Uganda until the 1950s (*5*) and remained largely unnoticed until 1997, when Médecins Sans Frontières (MSF, Swiss Section) began to provide assistance to Amudat Health Centre in Pokot County. In 2000, MSF initiated a kala-azar control program, focusing on passive case detection and treatment.

From January 2000 to February 2006, a total of 3,645 patients suspected of having kala-azar were screened at Amudat Health Centre by using the direct agglutination test or rK39 antigen–based dipsticks (*6*); 2,088 patients with confirmed disease were treated with daily intramuscular injections of sodium stibogluconate or meglumine antimonite, 20 mg/kg bodyweight, for 30 days. Overall, 80% of the patients were <15 years of age, 75% were male, and 70% were from Kenya. From 2000 through 2005, the number of patients treated more than tripled, from 175 to 690 cases per year. Although this increase likely results, at least in part, from greater case detection due to the availability of treatment, we cannot exclude a real increase in disease because kala-azar prevalence in the area is unknown.

Information on local vector behavior and risk factors for infection or disease (e.g., malnutrition and HIV coinfection) is limited, and which potential interventions are appropriate is unclear. A pilot entomologic study in 2004 (J. Stevenson, master's thesis) demonstrated that termite mounds (Figure) are important vector breeding and resting sites and that the practice of sitting on termite mounds while guarding livestock might increase the risk for infection. In contrast, humans may be protected from kala-azar when in close proximity to livestock (i.e., because of diversion of sandflies to alternative hosts) or when lighting fires indoors (smoke acts as a repellant to most biting flies). Ownership of insecticide-treated nets, which could protect persons from sandfly bites and reduce kala-azar transmission (*7*), was low. Although most of the local population had heard of kala-azar, known locally as *termes*, and regarded it as potentially fatal, few were aware of how kala-azar is transmitted (J. Stevenson, master's thesis).

Figure. A large termite mound occupies the central area of this characteristic Pokot compound. The mound provides a resting and breeding site for the sandly vector of visceral leishmaniasis. Photographer: J.H. Kolaczinski.

MSF's treatment of kala-azar is crucial because it reduces the human reservoir and hence transmission. However, current control activities only reach the tip of the iceberg: a large, underlying pool of infected and infectious persons likely exists (*8*,*9*).

Kala-azar in Uganda will not likely be controlled unless the epidemiology of the disease is better understood and preventive activities are undertaken. This knowledge gap is being addressed by a partnership among the Malaria Consortium, MSF, the London School of Hygiene and Tropical Medicine, and the Vector Control Division of the Ugandan Ministry of Health. A case-control study to determine the local risk factors of kala-azar is almost completed and will be followed by seroprevalence studies in several Pokot villages, using a similar approach to recent work in the Baringo District, Kenya (*9*). The results will be used to formulate an integrated control strategy aimed at achieving our ultimate goal of eliminating kala-azar from Uganda.

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Chikungunya Virus Infection in Traveler to Australia

To the Editor: Chikungunya is a mosquitoborne alphavirus in the family *Togaviridae*. Recently, a chikungunya virus epidemic that affected thousands of persons occurred in islands in the southwestern Indian Ocean, including Mauritius and Reunion (*1*). An outbreak is ongoing in India, and cases are being exported to many other countries (*2–4*) The likelihood of importation of exotic infectious agents into Australia increased during events such as the March 2006 Commonwealth Games in Melbourne. Urgent diagnosis of rarely seen infections is a travel health challenge, particularly when serologic tests used for diagnosis in areas with high prevalence are not locally available. Only 1 previously diagnosed case of chikungunya virus infection has been reported in Australia; it involved importation of the virus from Indonesia to Darwin in 1989 (*5*). We report a second case of infection with this virus.

A 59-year-old man came to a hospital emergency department in North Melbourne, Australia, on March 12, 2006, 5 days after traveling from Mauritius for the Commonwealth Games. He reported a 2-day history of acute swelling and erythema of the left leg and associated malaise. Twenty-four hours earlier, severe lumbar back pain and arthralgias that involved the lower limbs had developed, with associated fevers, rigors, and headache.

The patient had a temperature of 39°C, bilateral conjunctivitis, tender and markedly swollen Achilles tendons, a swollen left ankle, and a maculopapular rash that involved the left forefoot and anterior portion of the shin. Initial blood examinations showed leukopenia (leukocyte count 1.8×10^9/L, lymphocyte count 0.2×10^9/L), mild thrombocytopenia (platelet count 105×10^9/L), and abnormal liver function test results (alanine aminotransferase 133 IU/μL, γ-glutamyl transpeptidase 141 IU/μL, bilirubin 7 mmol/L). Malaria blood films and dengue serologic results were negative. A validated, in-house, generic alphavirus reverse transcription–PCR (RT-PCR) showed positive results 24 hours after collection of blood when the patient was admitted.

The patient received supportive treatment and was discharged from the hospital 3 days after admission, at which time leukopenia and thrombocytopenia had improved. The patient had fully recovered on review 1 week after discharge.

For virus isolation, plasma and leukocyte fractions were placed onto Vero E6 cells and incubated at 37°C for 5 days. Cells were observed daily for virus-specific cytopathic effects. A virus isolate was obtained after 4 days of cell culture. A 10-mL volume of supernatant from infected cells was applied to a carbon-coated grid, stained with phototungstic acid, and examined by electron microscopy. This procedure showed virus with morphology similar to Togavirus (data not shown).

Chikungunya virus was identified by a heminested RT-PCR for the non-