

Figure. Duration of pandemic (H1N1) 2009 excretion in nasal swabs from patients treated with oseltamivir. The number of days from start of oseltamivir treatment to achievement of negative results of reverse transcription–PCR (RT-PCR) is indicated for 16 patients. The 3 patients classified in the last group (>5 days) are 1 patient with a negative RT-PCR result on day 7 posttreatment and 2 patients who still had positive results on day 5 posttreatment but provided no additional sample for testing.

pandemic (H1N1) 2009 to oseltamivir. This latter point is now under study.

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Marburg Virus in Fruit Bat, Kenya

To the Editor: Lake Victoria Marburgvirus (MARV) causes severe hemorrhagic fever with a high case-fatality rate in humans. Index cases occurred in Europe during 1967 among laboratory workers who handled tissues and blood samples of nonhuman primates from Africa (1). Thereafter, MARV was reported throughout sub-Saharan Africa. Most outbreaks in humans were associated with visits to caves and mines (2–6). In Kenya, human cases of MARV infection were reported in 1980 and 1987; these occurred after visits to the Kitum Cave at Mount Elgon (7,8). MARV was detected in tissues of Egyptian fruit bats (*Rousettus aegyptiacus*) and other bat species from the Democratic Republic of Congo (DRC), Gabon, and Uganda (3–6).

We collected bats from across Kenya during June–July 2007 within the framework of the Global Disease Detection Program, which is dedicated to investigation of emerging pathogens. Collection protocols were approved by the National Museums of Kenya and by the Centers for Disease Control and Prevention (Atlanta, GA, USA). Blood, fecal and oral swab specimens, and selected tissue samples were collected from bats and stored on dry ice.

For MARV detection, total RNA was extracted from pooled or individual liver, spleen, and lung samples from 272 bats. Nested reverse transcription–PCR (RT-PCR) with primers specific for MARV nucleoprotein gene was performed as described (5). When a band of the expected size was detected after electrophoresis on an agarose gel, the RT-PCR product was sequenced. Laboratory cross-contamination was not a concern because no work with MARV had been conducted in the facility where the examination was performed.

MARV RNA was detected in pooled liver, spleen, and lung tissue of an apparently healthy, pregnant,

female *R. aegyptiacus* bat obtained at Kitum Cave in July 2007 (Figure). A faint band was obtained only after nested RT-PCR, which suggests that the RNA load was limited. Attempts at virus isolation were not performed. Phylogenetic comparisons demonstrated that the virus (KE261, GenBank accession no. GQ499199) was relatively distant from previous isolates from Kenya (Musoke and Ravn). It was similar to viruses isolated from index cases in Europe in 1967 (Popp and Ci67). This lineage also contained virus 02DRC99, which was isolated from a human in the DRC in 1999 (online Appendix Figure, www.cdc.gov/EID/content/16/2/352-appF.htm). MARV isolates obtained from bats and humans in Uganda in 2007 belong to distinct lineages (6) (online Appendix Figure). Tissues of other bats, including 75 *R. aegyptiacus* (29 pregnant females) from Kitum Cave and neighboring Makingeni Cave, were negative for MARV RNA.

Histopathologic examination of liver of the infected bat showed no lesions that could be ascribed to MARV

infection, and no MARV antigens were detected by immunohistochemical analysis. Other tissues were not examined.

Our results are similar to those reported from Gabon and the DRC, where MARV RNA was detected in tissues of 1.4% and 3.1% of *R. aegyptiacus* bats, respectively, with negative isolation attempts (3,5). A higher prevalence (5.1%) was detected in *R. aegyptiacus* bats from Uganda in 2007, where several MARV isolates were obtained from bats with high virus loads (6). In the DRC, MARV RNA was also detected in insectivorous bats, including 3.0% of *Miniopterus inflatus* and 3.6% of *Rhinolophus elocuens* (3,5). However, in Uganda, MARV RNA was detected in only 1 (0.2%) of 609 insectivorous bats (*Hipposideros* spp.) (6).

To date, bats are the only wild mammals, besides nonhuman primates, in which filoviruses have been detected. Whether bats serve as principal reservoir hosts for filoviruses is unclear. The pathogenesis and clinic manifestation of filovirus infection in

bats are unknown. Colonies of *R. aegyptiacus* bats in caves often consist of thousands of bats. The opportunity for conspecific exposure rates in such colonies is high. Therefore, bat populations should have a high seroprevalence rate for these viruses. For example, seroprevalence to lyssaviruses in some bat species that live in colonies was reported as high as 60%–70% (9). In contrast, seroprevalence of MARV-neutralizing antibodies in colonies of *R. aegyptiacus* bats in which PCR-positive bats were collected was only 12% (5) or as low as 2.4% (6). This low seroprevalence may be interpreted as a result of a limited spillover of MARV into bats from another source.

The association of human cases of MARV with visiting caves often inhabited by *R. aegyptiacus* and other bat species is obvious (3,5,6). This association was reinforced by MARV infection in tourists who visited caves in Uganda (4,10). For Kenya, our finding is consistent with reported human cases tentatively associated with visiting of Kitum Cave (7,8). We do not know if MARV has persisted in this area continuously or has reemerged sporadically. Kitum Cave and other similar caves are easily accessible and frequently visited by tourists and local persons. The likelihood of MARV spillover into humans is presently limited. However, because transmission mechanisms and sources of spillover infections are unknown, public awareness must be increased and health authorities informed about the presence of MARV.

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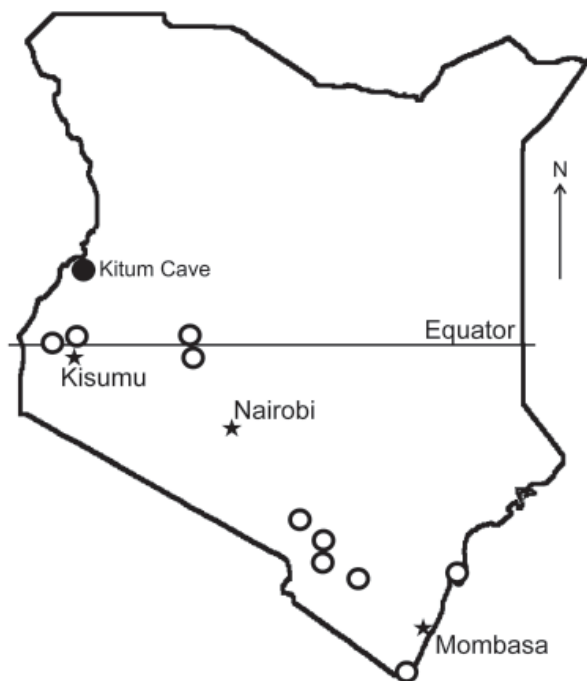


Figure. Bat collection sites (open circles) and location of Kitum Cave, Kenya, where Lake Victoria Marburgvirus was detected (solid circle).

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Human African Trypanosomiasis in Areas without Surveillance

To the Editor: Human African trypanosomiasis (HAT), sleeping sickness, is a systemic protozoan disease transmitted by the bite of a tsetse fly; untreated infection is fatal (1). Control of HAT caused by *Trypanosoma brucei gambiense*, which caused 97% of all cases reported from 1997 through 2006 (2), is based on active screening of the population at risk by mobile teams and treatment of all infected persons, with or without vector control.

The epidemiologic curve of reported new cases varies considerably; incidence peaks were high in the 1920s and 1990s but low in the 1960s and in the past decade (2000–2009) (2–4). The recent reduction of reported cases (69% decrease from 1997 through

2006) was made possible by 1) cessation of large-scale civil wars (e.g., in Angola); 2) increased commitment of donors, national control programs, the World Health Organization (WHO), and nongovernment organizations; and 3) free production and supply of antitrypanosomal drugs. In May 2007, after a WHO informal consultation on sustainable sleeping sickness control, representatives from countries to which HAT is endemic concluded that HAT elimination is possible (5). Médecins sans Frontières (MSF), an international nongovernment organization, wishes to challenge this conclusion.

Because of insufficient coverage by surveillance systems, only a fraction of HAT cases are reported. In 2004, for example, although WHO received reports of only 17,500 new cases, they estimated that the actual incidence was 50,000–70,000 cases (6). Recent MSF HAT projects in remote and politically unstable areas of the Central African Republic and the Democratic Republic of Congo are finding new information about the location and nature of some of these blind spots (areas without surveillance) (Table).

In the zones de santé (administrative districts) of Doruma, Ango, and Bili, in northeastern Democratic Republic of Congo, no HAT control activities have taken place over the past 3 decades, mainly because of extreme remoteness of these areas. In July 2007, MSF launched a HAT control program and found high (3.4%) disease prevalence and a large proportion of patients in the first stage of the disease (60%), indicating intense transmission. In March 2009, the MSF team was attacked by rebels from the Lord's Resistance Army, leading to total suspension of the project for an indefinite period. The lack of trained staff in existing health structures and the complexity of HAT management prevented emergency handover of the project to local partners.