which directly and indirectly affect the occurrence and distribution of malaria (*10*). Whether malaria will return as a major health threat likely depends on the size and fragmentation of the individual plantation areas. The required size of a plantation for the survival of the vector population is unclear, but large areas of plantation tend to offer dense vegetation and, therefore, high humidity and shade, which provide suitable environmental conditions for larval habitats, even during the dry season (*8*). Conversely, during the rainy season, conditions at the edges of fragmented forests, where human settlements are often located, become favorable for larval habitats, rendering villagers susceptible to the disease (*6*). In addition to changes in habitat and microclimate, social or political changes in the region may affect the transborder movement of malaria into Thailand with consequences for potential reemergence (*7*).

Although the association between rubber plantations and malaria is well known in Southeast Asia, the potential for reemergence should receive substantially more attention from economic, agricultural, and environmental planning bodies. Changes in land use and land cover have the potential to facilitate the transmission of disease to humans. Understanding the influence of land use change on malaria occurrence is critical for shaping future surveillance and control strategies.

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Fatal Borreliosis in Bat Caused by Relapsing Fever Spirochete, United Kingdom

To the Editor: Tick-borne relapsing fevers caused by members of the genus *Borrelia* have been encountered throughout Africa, Asia, the Americas and, rarely, in southern Europe (*1*). The *Borrelia* species associated with relapsing fevers form a monophyletic group within the genus, although not all members of this group have yet been implicated as agents of human disease. For example, a novel spirochete that is closely related to the relapsing fever agent *Borrelia turicatae* has recently been detected in *Carios kelleyi*, an argasid bat tick (*2*,*3*). We report the discovery of a spirochete causing fatal borreliosis in a bat in the United Kingdom.

The infected bat was a juvenile female *Pipistrellus* species that was found alive but on the ground near the town of Mevagissy in southwestern England in August 2008; despite rehabilitation efforts, it died a few days later. A postmortem examination showed pale skeletal muscles, anemia, excess blood-tinged pleural fluid, a healthy thymus, but enlarged cranial thoracic lymph nodes. The liver was greatly enlarged and mottled, the spleen was also large and unusually dark, and the adrenal glands were enlarged and pale with areas of hemorrhage. The kidneys were pale with a fine speckling

pattern over the cortex. Histopathologic examination of the liver showed multifocal necrosis and vacuolation of hepatocytes and infiltration by macrophages. The lungs were congested and infiltrated by inflammatory cells, and large numbers of granulocytes were found in the blood vessels. The spleen showed marked extramedullary hemopoiesis. Tissue sections stained by the Warthin-Starry technique exhibited numerous long, undulating, argophilic bacilli. These organisms were present in large numbers in the liver lesions (Figure), but were also found in the parenchyma of lung and spleen and in blood vessels.

On the basis of these observations, a diagnosis of fatal hepatitis and septicemia caused by a spirochete was made. DNA from the bat's liver was extracted and analyzed by using a PCR specific for an almost complete fragment of the 16S rRNA-encoding gene, as previously described (*4*), but with an annealing temperature of 45°C. This DNA extract was also incorporated into PCR assays targeting *glpQ* and *flaB* gene fragments (*5*). The products of these reactions were sequenced, and sequence data were assembled and analyzed by using Staden (*6*) and MEGA (*7*).

We obtained unambiguous sequence data for all 3 loci, comprising of 1,364 bp of the 16S rRNA-encoding gene (GenBank accession no. FJ868583), 1,239 bp of *flaB* and flanking regions (GenBank accession no. FJ868584), and 480 bp of *glpQ* (GenBank accession no. FJ868585). Each of these was aligned with homologous sequences available for other *Borrelia* species and used for phylogenetic analyses. Inferences made by using all loci were congruent, with the UK bat–associated spirochete lying close to, but distinct from, a cluster containing *B. recurrentis, B. duttonii,* and *B. crocidurae* (Figure; data not shown).

These 3 species are associated with relapsing fevers in Africa and Asia. The UK bat–associated spirochete bore no specific evolutionary relatedness to *B. johnsonii*, the newly characterized member of the relapsing fever group of *Borrelia* species associated with *C. kellyi* in the United States (Figure) (*3*). An *Argas vespertilionis* larval tick was found attached to the infected bat and may have been the source of its infection. PCR was not performed on the tick because it was near-replete with blood that was intensely infected with spirochetes. *A. vespertilionis*, commonly known as the short-legged bat tick, is widely distributed, parasitizing numerous bat species across Europe, southern Asia, and North Africa (*8*).

Given the close relationship between the novel spirochete we encountered and known pathogens, the reported propensity of *A. vespertilionis* to bite humans (*9*), and the wide geographic range of this tick, our findings have repercussions for public health in many parts of the Old World. Furthermore, although bats are likely the reservoir host for this organism, our study also identifies it as a pathogen, and as such its discovery has implications for the conservation of numerous threatened bat species across Europe and throughout the world.

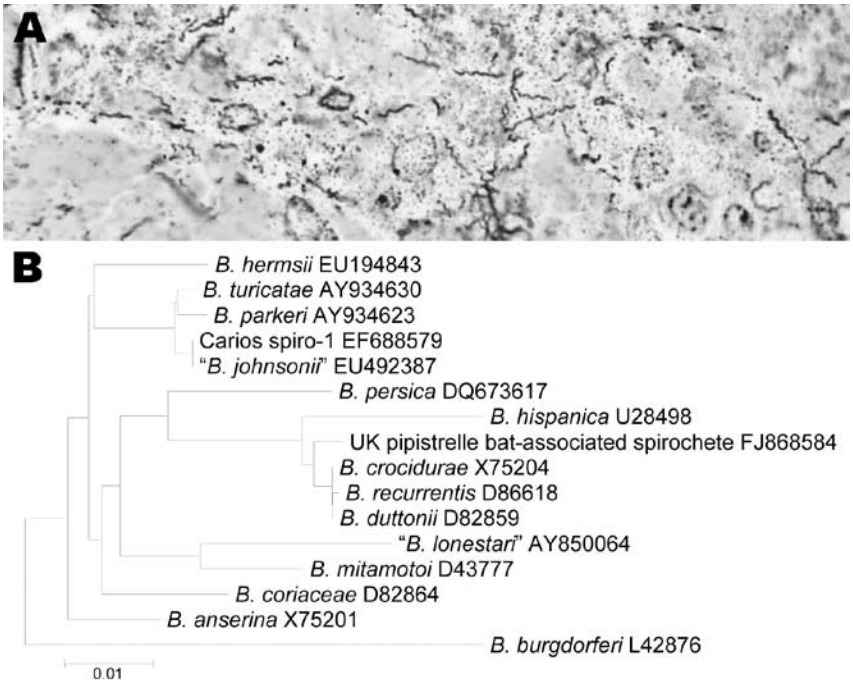

Figure. A) Warthin-Starry–stained section of bat liver showing numerous spirochetes. B) Phylogram inferred from 776-bp alignment of *flaB* fragments obtained from infected bat liver tissue and for other members of the relapsing fever group of *Borrelia* species for which sequence data were available. *B. burgdorferi* is included as an outgroup. The numbers appearing after the names of the *Borrelia* species are the relevant GenBank accession numbers. Scale bar indicates nucleotides substitutions per site.

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Past, Present, and Future of Japanese Encephalitis

To the Editor: We are writing in response to the perspective on Japanese encephalitis (JE) by Erlanger et al. (*1*). Growing awareness is encouraging, yet because JE is a largely neglected disease, information is often contradictory or not readily available. We would like to supplement the authors' review with clarification on available vaccines and actions countries are taking to evaluate and control JE.

There is room for improvement or expansion on collecting and reporting JE surveillance data. However, as vaccine availability increases, many countries are eager to determine the impact of JE and to make informed decisions on immunization programs. For example, surveillance in Indonesia from 2005 through 2006 confirmed human cases throughout the country (*2*). In Cambodia, JE surveillance commenced in 2006, and an immunization program is being planned (*2*). Regional JE laboratory networks established by the World Health Organization are also helping countries gather this information by strengthening diagnostic capacity.

Cambodia plans to introduce the live, attenuated SA 14-14-2 vaccine from China's Chengdu Institute of Biological Products. This vaccine has recently become internationally available and is increasingly replacing the inactivated, mouse brain–derived vaccine in Asia. A single dose of the SA 14-14-2 vaccine demonstrated 96% efficacy after 5 years (*3*), and the Institute's commitment to an affordable price for developing countries has broadened accessibility (*4*). The government of India introduced the SA 14-14-2 vaccine in 2006, and nearly 50 million children 1–15 years of age have been reached through vaccination campaigns and routine immunization. The vaccine also is available through public programs or private markets in China, Nepal, South Korea, Sri Lanka, and Thailand.

JE vaccine candidates in late-stage development for children include a live, attenuated chimeric virus vaccine and an inactivated, Vero cell–derived vaccine, each based on the SA 14-14-2 virus strain. Additionally, 2 inactivated, Vero–cell derived vaccines based on the Beijing-1 strain are being developed in Japan (*5*).

New vaccine development, along with progress in surveillance and immunization, offers promise for sustainable control of clinical JE. To achieve this, global partners are working together to develop a strategic plan for JE control by 2015 (*6*).

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