and Colombia and resembles the first cases of CA-MRSA described in the early 1990s as occurring in indigenous people living in remote areas of Western Australia (*4*). Second, considering that the most predominant CA-MRSA clones in Latin America carry SCC*mec* IV (*1*,*5*), finding SCC*mec* V in this isolate was not expected. MRSA carrying SCC*mec* V have been well characterized as colonizers and agents of infection in animals and in humans in close contact with animals (mainly in Europe but also in other parts of the world) (*6*). These livestock-associated MRSA clones predominantly belong to ST97 (which are usually not PVL producers) and ST398. In addition, ST398 SCC*mec* V MRSA isolates from pigs in Peru have been described (*7*). Of note, methicillin-susceptible *S. aureus* t701 and MRSA t701 carrying SCC*mec* II have recently been found in China, isolated from patients during food poisoning outbreaks and from colonized pork butchers, respectively (*8*,*9*). In South America, isolation of non–PVL-producing MRSA t701 (carrying SCC*mec* IVc) and methicillin-sensitive S. *aureus* t701 from colonized inpatients has been well described (*10*). Although speculation that animal carriage might have played a role in this infection is tempting, further studies are needed to recognize the origin of this MRSA ST6-SCC*mec* V PVL producer in this area of the Amazon Basin.

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Exposure to Bat-Associated *Bartonella* spp. among Humans and Other Animals, Ghana

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To the Editor: Human contact with wildlife is a leading cause of disease spillover. Bats, in particular, host numerous zoonotic pathogens, from henipaviruses to lyssaviruses (*1*). In Ghana, the straw-colored fruit bat (*Eidolon helvum*) frequently and closely interacts with humans through roosting in urban areas and human harvesting of bushmeat. Large colonies live in Accra, the capital city, and >128,000 bats, on average, are hunted for food yearly in southern Ghana alone (*2*). Serologic evidence of human infection with novel paramyxoviruses from *E. helvum* bats (*3*) supports concerns regarding this contact. In addition, Kosoy et al. (*4*) isolated several new strains of *Bartonella* that were found in >30% of *E. helvum* bats, whereas Billeter et al. found *Bartonella* in 66% of their ectoparasites (*5*), with *Bartonella* transmissibility to other species unknown. This prevalence causes concern because many *Bartonella*

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species are zoonotic and cause substantial human disease (*6*). Previous studies of febrile patients in Thailand have shown prevalence rates of ≤25% for antibodies against zoonotic *Bartonella* species (*7*). Serologic studies have been conducted in Europe and in the United States, but few studies have examined such prevalence in Africa among patients and in the general population (*8*).

To address these concerns, we conducted a prevalence study in Ghana, West Africa, for evidence of bat-associated *Bartonella* infection in humans and other common animal species. We sampled humans who had close contact with fruit bats and also sampled domestic animals that lived around the bat colonies.

We obtained serum samples from 335 volunteers from Accra and the Volta region who had close contact with *E. helvum* bats and also sampled 70 domestic animals that lived underneath bat colonies (5 cats, 23 chickens, 7 cows, 6 dogs, 21 goats, 8 sheep) in Accra. We used 3 testing approaches: culture, PCR, and indirect immunofluorescent assays for serologic testing. We tested serum specimens for antibodies against *B. henselae*, *B. quintana*, *B. clarridgeiae*, *B. vinsonii vinsonii*, *B. elizabethae*, and *Bartonella* strain E1–105, which had been isolated from *E. helvum* bats (*6*).

All culture results for human and domestic animal samples were negative for *Bartonella* species. One human serum sample was positive for *B. clarridgeiae* by PCR, which was confirmed by repeat testing. No other human samples were consistently positive by PCR. Of 70 animal blood clots and 62 serum samples tested by using PCR, 1 serum sample from a cat tested positive for *B. henselae*. One human serum sample was positive by immunofluorescent assay for antibodies against *B. henselae* at titers of 1:128, another had reactivity to *B. henselae* at 1:64, and 1 sample was reactive at 1:32. Five human serum samples were reactive to *B. quintana* at titers of 1:32.

The absence of evidence of any human exposure to bat-associated *Bartonella* suggests that the species isolated from *E. helvum* bats never or rarely infects humans in Ghana. If *Nycteribiidae* bat flies serve as the vector for *Bartonella* transmission between bats as hypothesized, then the high host specificity of these vectors (*8*) could explain why little infection is spilling over to other species. However, no experimental studies have confirmed that bat flies are competent vectors of bat-associated *Bartonella* species or that these ectoparasites only bite bats. These facts must be confirmed because bat flies are occasionally found on other animals and whether the parasites can successfully use these animals as hosts is unknown (*9*). Although further studies are needed to clarify the dynamics of *Bartonella* species infection in *E. helvum* bats, as well as the species' zoonotic potential, the current risk of spillover of this bat-associated *Bartonella* species appears low in West Africa. This fact may be useful in directing limited public health resources.

The seroprevalence to *B. henselae* in healthy human participants in this study was <1%. The low levels of seropositivity to *B. henselae* and *B. quintana* are consistent with those found in the only other study on *Bartonella* in humans in sub-Saharan Africa: a survey of 155 subjects in the Democratic Republic of Congo showed 1% seroprevalence of *B. henselae* and <1% seroprevalence of *B. quintana* (*8*).

The results of study in the Democratic Republic of Congo and this study contrast with some studies in Asia and Europe, which show higher rates of human exposure to *Bartonella* species. For example, a study of febrile patients in Thailand found serologic evidence of exposure to *Bartonella* infection in 25% of patients (*7*).

Laudisoit et al. (*8*) were, to our knowledge, the first to report evidence of *Bartonella* infection in humans in Africa. Our study contributes to this nascent effort of understanding *Bartonella* on the continent. Because a substantial proportion of *Bartonella* prevalence studies have been done on hospital patients, our study provides a survey of the general population to help determine background infection rates and illuminate the complex risks posed by this zoonosis.

The research for this article was carried out during fieldwork in southern Ghana; at the 37 Military Hospital in Accra; at the University of Cambridge, Cambridge, UK; and at the Centers for Disease Control and Prevention, Fort Collins, Colorado, USA.

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Isolation of Zika Virus from Febrile Patient, Indonesia

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To the Editor: Arthropodborne viruses (arboviruses) cause substantial human disease worldwide and have a pronounced effect on public health throughout Asia. Zika virus, discovered in Uganda in 1947 (*1*), is a flavivirus related to the following viruses: dengue (DENV), West Nile, Japanese encephalitis, and yellow fever. Like DENV, Zika virus is transmitted by *Aedes* mosquitoes. Zika virus emerged as a public health problem in 2007, when it caused an epidemic in Micronesia (*2*). Since then, the virus has caused epidemics elsewhere in the Pacific islands (*3*) and recently emerged in South America (*4*). Zika virus has been reported to cause mild and self-limited infection that can be misdiagnosed as dengue because of similar clinical features and serologic cross-reactivity (*2*). Zika virus has not, however, been reported to cause substantial thrombocytopenia or result in the serious vascular leakage that can be fatal in DENV infection.

Until recently, most evidence for Zika virus infection in Asia, including in Indonesia (*5*), has been serologic, but recent virus strains isolated from persons in Thailand (*6*), the Philippines (*7*), and Cambodia (*8*) have begun to clarify its genomic diversity. Phylogenetically, Zika virus appears to have 2 major lineages, African and Asian (*9*).

During December 2014–April 2015, a confirmed outbreak of dengue (determined by reverse transcription PCR [RT-PCR] for DENV and nonstructural protein 1 [NS1] antigen detection; data not shown) occurred in Jambi Province, central Sumatra, Indonesia. We received samples from 103 case-patients with clinically diagnosed dengue; these samples had been negative for DENV by RT-PCR, NS1 antigen detection, or evidence of seroconversion by ELISA (data not shown). We tested the samples for other viruses using alphavirus and flavivirus RT-PCR (targeting genome positions 6533–6999 and 8993–9258, respectively). In parallel, we attempted virus isolation using Vero cells.

One sample, JMB-185, came from a 27-year-old man who sought treatment at the Jambi city hospital 2 days after illness onset with a sudden high fever, headache, elbow and knee arthralgia, myalgia, and malaise. He did not exhibit some common clinical characteristics of Zika virus infection (*10*), including maculopapular rash, conjunctivitis, or peripheral edema. Hematologic testing revealed lymphocytopenia and monocytosis; platelet count was within reference range. Results of all assays were negative for DENV, including NS1 antigen detection with NS1 Ag Rapid Test (SD Bioline, Kyong, South Korea); PanBio Dengue Early NS1 ELISA (Alere, Brisbane, Australia); PanBio Dengue Duo IgM and IgG ELISA (Alere); and Simplexa real-time RT-PCR (Focus Diagnostics, Cypress, CA, USA). The illness was self-limiting, and the patient recovered 2 days after he sought treatment without any complications.

Of the 103 DENV-negative specimens we tested, only JMB-185 was positive for flavivirus and displayed cytopathic effects when cultured in Vero cells for 10 days. A subsequent passage was performed, and supernatants from both passages were tested for flaviviruses by RT-PCR. A 265-bp amplicon was generated from JMB-185 by using flavivirus consensus primers. This consensus amplicon product had ≈85% nucleotide identity with the prototype Zika virus (strain MR 766, 1947, Uganda). An additional larger amplicon was generated (nt 9278–9808 of NS5 gene), and a phylogenetic tree was constructed based on the partial sequence of the NS5 region (530 bp) for JMB-185 (GenBank accession no. KU179098) and other Zika virus sequences, including those from Cambodia, Yap Island, Thailand, and the Philippines (Figure). Phylogenetic analysis indicated that JMB-185 belonged to the Zika virus Asian lineage and had 99.24% nucleotide identity to an isolate from a Canadian visitor to Thailand (*10*). It was also close to a Zika virus strain isolated from an Australian traveler who had visited Java (on the basis of a different NS5 region; data not shown). The original serum and passage samples were