

vaccine effectiveness is 92% for persons who received 1 dose and 95% for those who received 2 doses (9). Assuming that vaccine effectiveness is lower shifts the curve (Figure) to the left and would result in a lower estimate of vaccination coverage. Second, different numbers of persons who received 1 and 2 doses complicate the identification of overall vaccine effectiveness. Third, vaccination status is unknown for some measles case-patients. The proportion of nonvaccinated persons among those case-patients might be higher than that among those known to be vaccinated, also leading to a lower estimate of vaccination coverage. Finally, nonvaccinated persons might be clustered together, and their risk for infection could be higher than that for the general population (10). This scenario would imply that the estimated vaccination coverage does not reflect the general population but instead corresponds to a clustered subpopulation among whom vaccination rates are lower. The effects of these complexities warrant further investigation. However, as the examples demonstrate, a model ignoring those effects is in good agreement with empirical data.

Our analysis suggests that the number of vaccinated measles case-patients should be closely followed through surveillance programs. A continuous decrease in the proportion of measles case-patients who had been vaccinated over the years could indicate a decrease in vaccination rates. Conversely, an increase in the proportion of measles case-patients who had been vaccinated would demonstrate the effectiveness of ongoing efforts to increase vaccination rates and could serve as a benchmark toward measles elimination.

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References

- Centers for Disease Control and Prevention. Measles cases and outbreaks [cited 2015 Feb 13]. <http://www.cdc.gov/measles/cases-outbreaks.html>
- European Centre for Disease Prevention and Control. Surveillance report, measles and rubella monitoring, February 2014 [cited 2015 Apr 8]. <http://ecdc.europa.eu/en/publications/Publications/measles-rubella-monitoring-february-2014.pdf>
- Wichmann O, Hellenbrand W, Sagebiel D, Santibanez S, Ahlemeyer G, Vogt G, et al. Large measles outbreak at a German public school, 2006. *Pediatr Infect Dis J*. 2007;26:782–6. <http://dx.doi.org/10.1097/INF.0b013e318060aca1>
- van Boven M, Kretzschmar M, Wallinga J, O'Neill PD, Wichmann O, Hahné S. Estimation of measles vaccine efficacy and critical vaccination coverage in a highly vaccinated population. *J R Soc Interface*. 2010;7:1537–44. <http://dx.doi.org/10.1098/rsif.2010.0086>
- Orenstein WA, Bernier RH, Dondero TJ, Hinman AR, Marks JS, Bart KJ, et al. Field evaluation of vaccine efficacy. *Bull World Health Organ*. 1985;63:1055–68.
- Centers for Disease Control and Prevention. Measles—California, December 2014–February 2015. *MMWR Morb Mortal Wkly Rep*. 2015;64:153–4.
- Centers for Disease Control and Prevention. Measles—United States, January 1–May 23, 2014. *MMWR Morb Mortal Wkly Rep*. 2014;63:496–9.
- Richard JL, Masserey Spicher V. Large measles epidemic in Switzerland from 2006 to 2009: consequences for the elimination of measles in Europe. *Euro Surveill*. 2009;14:19443.
- Marin M, Nguyen HQ, Langidrik JR, Edwards R, Briand K, Papania MJ, et al. Measles transmission and vaccine effectiveness during a large outbreak on a densely populated island: implications for vaccination policy. *Clin Infect Dis*. 2006;42:315–9. <http://dx.doi.org/10.1086/498902>
- Salathé M, Bonhoeffer S. The effect of opinion clustering on disease outbreaks. *J R Soc Interface*. 2008;5:1505–8. <http://dx.doi.org/10.1098/rsif.2008.0271>

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Lassa Virus in Multimammate Rats, Côte d'Ivoire, 2013

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To the Editor: Lassa fever is a zoonosis caused by Lassa virus (LASV; family Arenaviridae, genus Lassavirus). The primary reservoir of LASV is the multimammate rat (*Mastomys natalensis*), which is found throughout sub-Saharan Africa. LASV outbreaks among humans occur only in West Africa in 2 noncontiguous areas: 1 in Guinea, Liberia, and Sierra Leone; and 1 in Nigeria. Rare cases and evidence of exposure of humans have been documented in neighboring countries (i.e., Benin, Burkina Faso, Côte d'Ivoire, Ghana, Mali, and Togo) (1). LASV RNA has been detected in only 4 patients: 1 in Germany who had traveled in Burkina Faso, Côte d'Ivoire, and Ghana (2); 1 in the United Kingdom who had returned from Mali (3); and 2 in

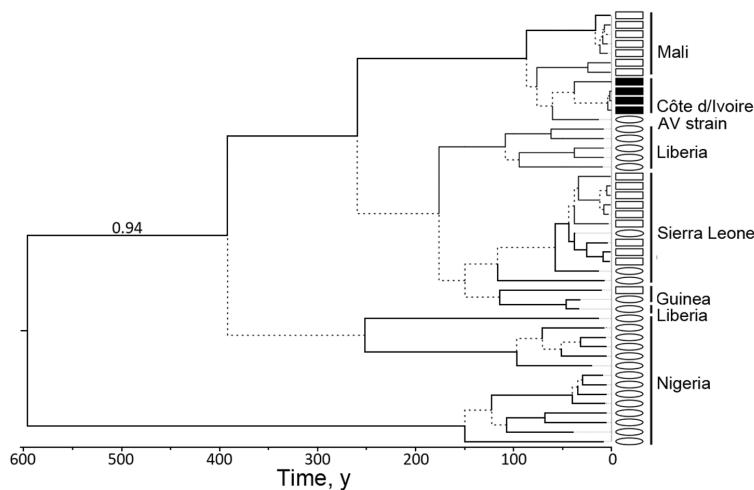


Figure. Bayesian chronogram of Lassa virus (LASV) sequences determined on the basis of a fragment of the large genomic segment. Branches receiving posterior probability values <0.95 and bootstrap values <50 (poorly supported) are dashed. LASV sequences of human origin are indicated by ovals, and those of multimammate rats are indicated by squares. Sequences reported in this study are indicated by black squares. This tree was built under the assumption of a molecular clock and is therefore rooted. The numerical value on the tree's most basal branch is the root posterior probability of this branch; it supports the notion that LASV sequences from Nigeria and other countries are not reciprocally monophyletic. GenBank accession nos. of sequences used for phylogenetic analyses are shown in online Technical Appendix Table 2 (<http://wwwnc.cdc.gov/EID/article/21/8/15-0312-Techapp.pdf>). AV strain indicates the strain from a German patient.

Ghana, for whom no viral sequence was available because detection was performed by reverse transcription PCR only (4). In the region in Mali where the patient from the United Kingdom was infected, identical LASV sequences were found in multimammate rats (5). The sequence of the strain identified from the patient in Germany, who was designated AV, is the closest known relative of the clade formed by sequences from Mali (5). However, LASV was not found in its natural host in any of the countries visited by patient AV (6,7).

For a study investigating zoonotic pathogens in rural habitats, we caught small mammals in 3 ecologic zones of Côte d'Ivoire: 1) dry bushland in northern Côte d'Ivoire, around Korhogo (2); semiarid bushland in central Côte d'Ivoire, around Bouake; and rainforest in southwestern Côte d'Ivoire, near the Taï National Park (3) (online Technical Appendix Figure, <http://wwwnc.cdc.gov/EID/article/21/8/15-0312-Techapp.pdf>). Traps were installed within and around 15 villages and enabled the capture of 27 eulipotyphlans and 254 rodents during August–October 2013. Animals were assigned at the genus level in the field on the basis of morphology. For 88% of them, assignment could later be refined to the species level by sequencing a fragment of the mitochondrial cytochrome *b* gene. A total of 14 animal species representing 8 genera were detected. All host sequences were deposited in Dryad (<http://www.datadryad.org/>; online Technical Appendix Table 1). Multimammate rats were the dominant commensals at all sampling locations, comprising 64.5% of the overall sample (online Technical Appendix Figure).

Tissue samples were collected from all animals according to standard protocols. Total nucleic acids were extracted from lung samples and tested for the presence of LASV RNA by using a real-time PCR system amplifying a 400-bp fragment of the large genomic segment (8) (online Technical

Appendix). LASV RNA was detected in 4 of 18 specimens of *M. natalensis* captured in Gbalôhò, near Korhogo (online Technical Appendix Figure). This site is much farther north in Côte d'Ivoire than previously examined sites (6). The 4 PCR-positive animals were 3 males and 1 female that were all captured indoors, 3 in the same house. PCR products were sequenced according to the Sanger method (GenBank accession nos. LN823982–LN823985). According to phylogenetic analyses performed in maximum likelihood and Bayesian frameworks (online Technical Appendix), LASV sequences identified in multimammate rats from Côte d'Ivoire formed a robust clade with sequences from the human AV strain and the LASV infecting multimammate rats in southern Mali (bootstrap 97, posterior probability 1.00; Figure). This phylogenetic placement opens up the possibility that patient AV was infected during her travel through Côte d'Ivoire, possibly in or near the city of Korhogo. Tip date calibration of Bayesian analyses showed that the most recent common ancestor of all LASV sequences from Côte d'Ivoire and Mali circulated ≈ 90 years ago (Figure; online Technical Appendix Table 2).

Further studies will be needed to investigate the geographic distribution of LASV in Côte d'Ivoire and the frequency of human infections. The current lack of diagnosed cases in the area may be caused by underreporting. Sensitization campaigns are needed to increase awareness of the risk for LASV infection among the local population and to improve detection of cases by health workers.

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References

1. Sogoba N, Feldmann H, Safronetz D. Lassa fever in West Africa: evidence for an expanded region of endemicity. *Zoonoses Public Health*. 2012;59(Suppl 2):43–7. <http://dx.doi.org/10.1111/j.1863-2378.2012.01469.x>
2. Günther S, Emmerich P, Laue T, Kuhle O, Asper M, Jung A, et al. Imported lassa fever in Germany: molecular characterization of a new lassa virus strain. *Emerg Infect Dis*. 2000;6:466–76. <http://dx.doi.org/10.3201/eid0605.000504>
3. Atkin S, Anaraki S, Gothard P, Walsh A, Brown D, Gopal R, et al. The first case of Lassa fever imported from Mali to the United Kingdom, February 2009. *Euro Surveill*. 2009;14: pii:19145.
4. Dzotsi EK, Ohene SA, Asiedu-Bekoe F, Amankwa J, Sarkodie B, Adjabeng M, et al. The first cases of Lassa fever in Ghana. *Ghana Med J*. 2012;46:166–70.
5. Safronetz D, Lopez JE, Sogoba N, Traore SF, Raffel SJ, Fischer ER, et al. Detection of Lassa virus, Mali. *Emerg Infect Dis*. 2010;16:1123–6.
6. Coulibaly-N'Golo D, Allali B, Kouassi SK, Fichet-Calvet E, Becker-Ziaja B, Rieger T, et al. Novel arenavirus sequences in *Hylomyscus* sp. and *Mus (Nannomys) setulosus* from Côte d'Ivoire: implications for evolution of arenaviruses in Africa. *PLoS ONE*. 2011;6:e20893. <http://dx.doi.org/10.1371/journal.pone.0020893>
7. Kronmann KC, Nimo-Printsil S, Guirguis F, Kronmann LC, Bonney K, Obiri-Danso K, et al. Two novel arenaviruses detected in pygmy mice, Ghana. *Emerg Infect Dis*. 2013;19:1832–5. <http://dx.doi.org/10.3201/eid1911.121491>
8. Lecompte E, Fichet-Calvet E, Daffis S, Koulemou K, Sylla O, Kourouma F, et al. *Mastomys natalensis* and Lassa fever, West Africa. *Emerg Infect Dis*. 2006;12:1971–4. <http://dx.doi.org/10.3201/eid1212.060812>

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***Rickettsia felis* Infection among Humans, Bangladesh, 2012–2013**

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To the Editor: *Rickettsia felis*, which belongs to the spotted fever group of rickettsiae, causes febrile illness in humans. The main vector of this bacterium is the cat flea (*Ctenocephalides felis*). Since publication of reports of *R. felis* as a putative pathogen of humans in the United States in 1994, *R. felis* infection in humans worldwide has been increasingly described, especially in the Americas, Europe, Africa, and eastern Asia (1,2). *R. felis* infection is common among febrile patients (≈15%) in tropical Africa (3) and among apparently healthy persons in eastern coastal provinces of China (4). However, little is known about prevalence of *R. felis* infection of humans in southern Asia, although 3 serologically diagnosed cases in Sri Lanka have been described (5) and *R. felis* has been detected in rodent fleas in Afghanistan (6). Hence, we conducted a cross-sectional study in Bangladesh to explore the presence of rickettsial pathogens among patients with fever of unknown origin.

Study participants were 150 patients at Mymensingh Medical College (MMC) hospital in Mymensingh, north-central Bangladesh, from July 2012 through January 2014, and 30 healthy control participants from the staff at the same college. Selected patients met the following criteria: 1) fever (axillary temperature >37.5°C) for >15 days that did not respond to common antimicrobial drug therapy; 2) any additional clinical features including headache, rash, lymphadenopathy, myalgia, and eschars on skin; and 3) titer according to the Weil-Felix test (antibodies against any of 3 *Proteus* antigens) of >1:80. Patients with evident cause of fever (e.g., malaria diagnosed by blood smear or immunochromatography) were excluded from the study. This research was approved by the college institutional review board, and informed consent was obtained from patients (or guardians) and healthy controls before their entry into the study.

Venous blood samples were aseptically collected from the patients, and DNA was extracted by conventional method by using proteinase K and sodium dodecyl sulfate. Nested PCR selective for the 17-kDa antigen gene was used to screen for rickettsiae according to the method described previously (7); ≈100 ng of DNA in a 50-mL reaction mixture was used. For each PCR, a negative control (water) was included and utmost care was taken to avoid contamination. Among the 150 samples tested, results were positive with a 232-bp amplified product for 69 (46%) and negative for all controls.

PCR products from 20 samples were randomly selected for sequence analysis. All nucleotide sequences from