sensu stricto or *B. burgdorferi* sensu lato was detected (4/12 sites each), prevalence was 0.6%–2.2% and 0.7%–2.5%, respectively. *B. miyamotoi* was detected at 7/12 sites, and prevalence ranged from 0.7% to 7.5%. A previous survey of *B. burgdorferi* in nearby Santa Cruz County recreational areas reported an infection prevalence of ≈6% among adult *I. pacificus* ticks (*6*); the study did not, however, differentiate between *Borrelia* spp. and therefore may have included *B. miyamotoi* among its prevalence measures (*5*). In our study, *B. burgdorferi* was found more frequently in woodland habitats, but it was also detected in a grassland–chaparral habitat several hundred meters from the nearest woodland. We did not detect *B. bissettii*, a species recently implicated as a human pathogen in Mendocino County, California (*10*). The high level of habitat variation in northwestern California presents a varied risk for *Borrelia*-associated tick-borne disease in humans because of diverse variations in vertebrate reservoir ecology, tick abundance, and human exposure to ticks. This variation emphasizes the need to understand the local epidemiology and ecology of a disease.

In adult *I. pacificus* ticks in the San Francisco Bay area, *B. miyamotoi* is as abundant as its congener *B. burgdorferi.* Human disease caused by *B. miyamotoi* infection has not been reported in California, and transmission efficiency of *B. miyamotoi* by *I. pacificus* ticks is unknown. However, it is possible that *B. miyamotoi* infections in ticks and humans have not been accurately diagnosed. We advocate for increased scrutiny of the eco-epidemiology of *B. miyamotoi* in human, tick, and possible vertebrate host populations in northwestern California.

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Daniel J. Salkeld, Stephanie Cinkovich, and Nathan C. Nieto

Author affiliations: Stanford University, Stanford, California, USA (D.J. Salkeld); and Northern Arizona University, Flagstaff, Arizona, USA (S. Cinkovich, N.C. Nieto)

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References

1. Mun J, Eisen RJ, Eisen L, Lane RS. Detection of a *Borrelia miyamotoi* sensu lato relapsing-fever group spirochete from *Ixodes pacificus* in California. J Med Entomol. 2006;43:120–3. http://dx.doi.org/10.1603/0022-2585(2006)043[0120:DOABMS]2.0.CO;2
2. Padgett KA, Bonilla DL. Novel exposure sites for nymphal *Ixodes pacificus* within picnic areas. Ticks and Tick-Borne Diseases. 2011;2:191–5. http://dx.doi.org/10.1016/j.ttbdis.2011.07.002
3. Krause PJ, Narasimhan S, Wormser GP, Rollend L, Fikrig E, Lepore T, et al. Human *Borrelia miyamotoi* infection in the United States. N Engl J Med. 2013;368:291–3. http://dx.doi.org/10.1056/NEJMc1215469
4. Gugliotta JL, Goethert HK, Berardi VP, Telford SR III. Meningoencephalitis from *Borrelia miyamotoi* in an immunocompromised patient. N Engl J Med. 2013;368:240–5. http://dx.doi.org/10.1056/NEJMoa1209039
5. Barbour AG, Bunikis J, Travinsky B, Hoen AG, Diuk-Wasser MA, Fish D, et al. Niche partitioning of *Borrelia burgdorferi* and *Borrelia miyamotoi* in the same tick vector and mammalian reservoir species. Am J Trop Med Hyg. 2009;81:1120–31. http://dx.doi.org/10.4269/ajtmh.2009.09-0208
6. Holden K, Boothby JT, Anand S, Massung RF. Detection of *Borrelia burgdorferi, Ehrlichia chaffeensis,* and *Anaplasma phagocytophilum* in ticks (Acari: Ixodidae) from a coastal region of California. J Med Entomol. 2003;40:534–9. http://dx.doi.org/10.1603/0022-2585- 40.4.534
7. Eisen RJ, Mun J, Eisen L, Lane RS. Life stage-related differences in density of questing ticks and infection with *Borrelia burgdorferi* sensu lato within a single cohort of *Ixodes pacificus* (Acari: Ixodidae). J Med Entomol. 2004;41:768–73.
8. Bunikis J, Tsao J, Garpmo U, Berglund J, Fish D, Barbour AG. Typing of *Borrelia* relapsing fever group strains. Emerg Infect Dis. 2004;10:1661–4. http://dx.doi.org/10.3201/eid1009.040236
9. Scott MC, Rosen ME, Hamer SA, Baker E, Edwards H, Crowder C, et al. High-prevalence *Borrelia miyamotoi* infection among wild turkeys (*Meleagris gallopavo*) in Tennessee. J Med Entomol. 2010;47:1238–42. http://dx.doi.org/10.1603/ME10075
10. Girard YA, Fedorova N, Lane RS. Genetic diversity of *Borrelia burgdorferi* and detection of *B. bissettii*-like DNA in serum of north-coastal California residents. J Clin Microbiol. 2011;49:945–54. http://dx.doi.org/10.1128/JCM.01689-10

Address for correspondence: Daniel J. Salkeld, 1219 W Mountain Ave, Fort Collins, CO 80521, USA; email: dansalkeld@gmail.com

Buruli Ulcer in Liberia, 2012

To the Editor: Buruli ulcer, a necrotizing skin disease caused by *Mycobacterium ulcerans*, is highly endemic to West Africa (*1*,*2*) and is characterized by large ulcerations on the lower limbs (60% of cases) as well as on the upper limbs (30%) and other parts of the body (10%). Although the mode of transmission is unknown, most cases of Buruli ulcer occur around swampy and riverine areas; children <15 years of age are most often affected (*2*,*3*). The recommended treatment consists of a combination of daily oral rifampin and intramuscular streptomycin for 8 weeks, supplemented by wound care when appropriate (*4*). Large ulcers may require debridement and grafting to facilitate wound closure, and physiotherapy is often indicated to prevent functional limitation, particularly for lesions located over joints.

In Liberia, 2 Buruli ulcer patients were reported in 1981 in the Foya region, along the Manor River basin; 4 more patients were observed in the same area in 1984 (*5*). Since then, some patients from Liberia have received treatment for Buruli ulcer in Côte d'Ivoire (*6*), and suspicious cases have been detected in some parts of Liberia since the end of a civil war in 2004.

Recently, Buruli ulcer cases have been suspected in 3 counties of Liberia, Bong, Lofa, and Nimba; these regions share borders with the Buruli ulcer–endemic regions of Côte d'Ivoire and Guinea. During 2012, the government of Liberia, with assistance from the Medical Assistance Program International and with technical support from the World Health Organization (WHO), conducted a rapid status assessment in these 3 counties. In January 2012, a core team of national and county health personnel was trained in the recognition and assessment of Buruli ulcer. Assessment was conducted during February 18–27, 2012, by a team made up of those who had received the preassessment training and WHO Buruli ulcer consultants.

During the preassessment training, notice was given to all health facilities to record all lesions with features suggestive of Buruli ulcer. The persons identified during this period then came to the nearest health facility to be examined by the assessment team or were traced to their homes. A detailed history was collected and physical examination conducted, and swab specimens and fine-needle aspirates (2 for each lesion) were obtained for confirmation of diagnosis by classical PCR (*7*).

On the basis of the WHO case definition for Buruli ulcer (*1*), 60 of 181 persons screened were suspected to have Buruli ulcer. All cases were documented by photography and registration on a modified version of the WHO Buruli ulcer assessment form (*1*). For 21 (35%) of the 60 patients, *IS2404* PCR testing at Komfo Anokye Teaching Hospital in Ghana confirmed the clinical diagnosis; these patients received the recommended treatment for Buruli ulcer. Those with negative test results received wound care and supportive management.

A total of 21 confirmed cases occurred: 9 in Nimba County, and 6 each in Bong and Lofa Counties (Figure). Nine (35%) of the 21 patients were children ≤15 years of age; 11 patients were male and 10 female. Most (17 of 21) lesions were on the lower limbs; 3 were on the upper limbs and 1 on the thorax. Fifteen patients had ulcers, 2 edema, and 3 osteomyelitis. No lesions were classified as category I, but 11 (52.4%) were category III.

Our findings suggest that Buruli ulcer in Liberia may be more prevalent than previously thought. Although only 3 of 15 counties were assessed, results show that Buruli ulcer has not disappeared from Liberia and that the absence of regular reporting should be investigated. A long civil war and lack of familiarity with the disease by health care workers may have contributed to poor reporting.

It has been almost 3 decades since the last published report of Buruli ulcer cases in Liberia (*5*), but other studies have found the disease in countries many years after it was last reported. In Cameroon, a case search in 2001 in 2 districts where cases had last been reported 24 years earlier found 436 active and inactive cases (*8*). In southwestern Nigeria, a case search in 2006 found 14 active and inactive cases 30 years after the most recent publication (*9*). More recently, in 2012, a similar situation was reported in Gabon (*10*).

Several measures might improve Buruli ulcer control and surveillance in Liberia. First, treatment and control activities should be included in the Neglected Tropical Diseases Control Program at all levels to enhance

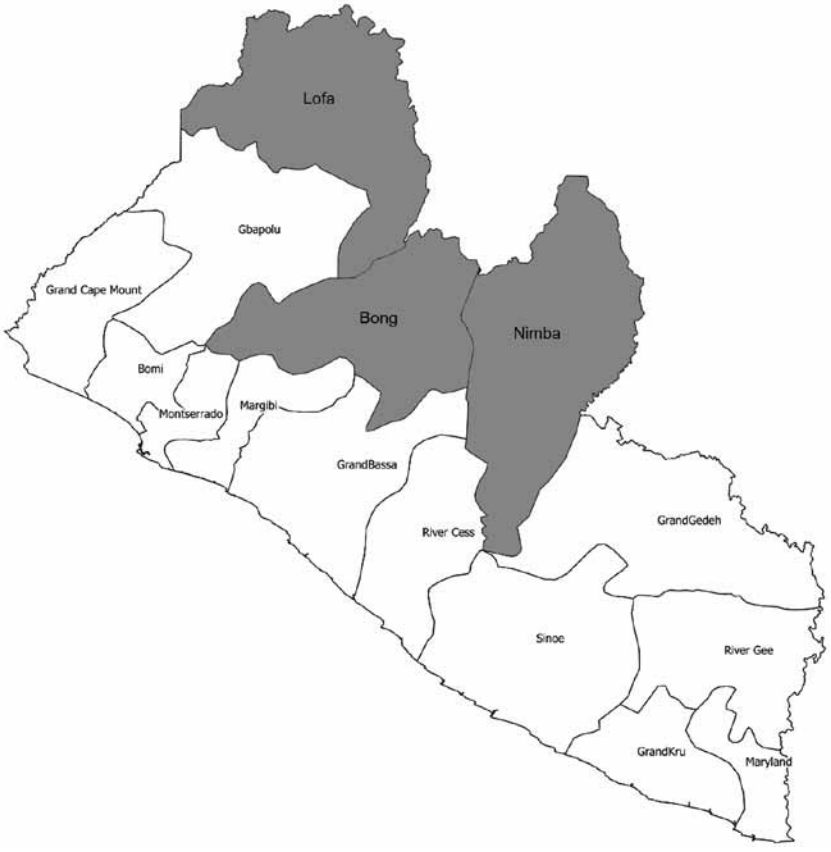

Figure. Counties in which cases of Buruli ulcer were found during 2012 (gray shading), Liberia.

surveillance. Second, health workers at all levels should be trained to recognize the disease. Third, a detailed assessment of the extent of Buruli ulcer in the 3 counties visited as well as in other counties should be prepared. Fourth, partner/donor support for Buruli ulcer activities should be enhanced. Fifth, capacity of the National Reference Laboratory to be able to perform PCR for confirmation of Buruli ulcer cases should be expanded. Last, Buruli ulcer should be incorporated into the national surveillance system to enable better data collection.

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Karsor Kollie, Yaw Ampem Amoako, Julien Ake, Tarnue Mulbah, Fasseneh Zaizay, Mohammed Abass, Linda Lehman, Albert Paintsil, Fred Sarfo, Clement Lugala, Alexandre Tiendrebeogo, Richard Phillips, and Kingsley Asiedu

Author affiliations: Neglected Tropical Diseases Control Program, Monrovia, Liberia (K. Kollie, T. Mulbah); Komfo Anokye Teaching Hospital, Kumasi, Ghana (Y.A. Amoako, F. Sarfo, R. Phillips); Medical Assistance Program International West Africa Region, Abidjan, Côte d'Ivoire (J. Ake, F. Zaizay); Agogo Presbyterian Hospital, Agogo, Ghana (M. Abass); American Leprosy Missions, Greenville, South Carolina, USA (L. Lehman); Korle Bu Teaching Hospital, Accra, Ghana (A. Paintsil); World Health Organization (WHO), Monrovia (C. Lugala); WHO, Brazzaville, Republic of Congo (A. Tiendrebeogo); and WHO, Geneva, Switzerland (K. Asiedu)

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References

1. Asiedu K, Raviglione M, Scherpbier R, editors. Buruli ulcer: *Mycobacterium ulcerans* infection. Geneva: World Health Organization; 2000. WHO/CDS/CPE/GBUI/2000.1.
2. Buruli ulcer disease. *Mycobacterium ulcerans* infection: an overview of reported cases globally. Wkly Epidemiol Rec. 2004;79:194–9.
3. Buruli ulcer: first programme review meeting for West Africa—summary report. Wkly Epidemiol Rec. 2009;84:43–8.
4. World Health Organization. Provisional guidance on the role of specific antibiotics in the management of *Mycobacterium ulcerans* disease (Buruli ulcer). Geneva: The Organization; 2004. WHO/CDS/CPE/GBUI/2004.10.
5. Monson MH, Gibson DW, Connor DH, Kappes R, Heinz HA. *Mycobacterium ulcerans* in Liberia: a clinicopathologic study of 6 patients with Buruli ulcer. Acta Tropica. 1984;41:165–172.
6. Kanga JM, Kacou DE. Epidemiological aspects of Buruli ulcer in Côte d'Ivoire: results of a national survey. Bull Soc Pathol Exot. 2001;94:46–51.
7. Phillips RO, Sarfo FS, Osei-Sarpong F, Boateng A, Tetteh I, Lartey A, et al. Sensitivity of PCR targeting *Mycobacterium ulcerans* by use of fine-needle aspirates for diagnosis of Buruli ulcer. J Clin Microbiol. 2009;47:924–6. http://dx.doi.org/10.1128/JCM.01842-08
8. Noeske J, Kuaban C, Rondini S, Sorlin P, Ciaffi L, Mbuagbaw J, et al. Buruli ulcer disease in Cameroon rediscovered. Am J Trop Med Hyg. 2004;70:520–6.
9. Chukwuekezie O, Ampadu E, Sopoh G, Dossou A, Tiendrebeogo A, Sadiq L, et al. Buruli ulcer, Nigeria. Emerg Infect Dis. 2007;13:782–3. http://dx.doi.org/10.3201/eid1305.070065
10. Ngoa UA, Adzoda GK, Louis BM, Adegnika AA, Lell B. Buruli ulcer in Gabon, 2001–2010. Emerg Infect Dis. 2012;18:1206–7. http://dx.doi.org/10.3201/eid1807.110613

Address for correspondence: Yaw Amoako, Department of Medicine, Komfo Anokye Teaching Hospital, Kumasi, Ghana; email: yamoako2002@yahoo.co.uk

Candidatus Neoehrlichia mikurensis and *Anaplasma phagocytophilum* in Urban Hedgehogs

To the Editor: *Candidatus* Neoehrlichia mikurensis is a member of the order Rickettsiales, family *Anaplasmataceae* (*1*). Manifestations of infection with these bacteria are atypical and severe and include cough, nausea, vomiting, anemia, headache, pulmonary infiltration, malaise, myalgia, arthralgia, fatigue, recurrent fever for ≤8 months, and/or death (*2–5*). *Candidatus* N. mikurensis has been detected in *Ixodes ovatus*, *I. persulcatus*, and *Haemaphysalis concinna* ticks in Asia (*1,5*).

Candidatus N. mikurensis has been identified as one of the most prevalent pathogenic agents in *I. ricinus* ticks throughout Europe (*2,3,6*). Rodents of diverse species and geographic origins have been shown to carry these bacteria, but transmission experiments have not been conducted to unambiguously identify natural vertebrate reservoirs (*1–3,5–7*). This emerging tickborne pathogen has been detected mainly in immunocompromised patients in Sweden (n = 1), Switzerland (n = 3), Germany (n = 2), and the Czech Republic (n = 2) and in immunocompetent patients in China (n = 7) (*2–5*).

Anaplasma phagocytophilum is an obligate, intracellular, tickborne bacterium of the family *Anaplasmataceae* and causes granulocytic anaplasmosis in humans and domestic animals. In Europe, *I. ricinus* ticks are its major vector, and red deer, roe deer, rodents, and European hedgehogs (*Erinaceus europaeus*) are suspected reservoir hosts (*8*).

Northern white-breasted hedgehogs (*Erinaceus roumanicus*) are urban-dwelling mammals (order Eulipotyphla, family Erinaceidae) that serve as major maintenance hosts for the 3 stages of