rural southwestern United States (*10*) and a strain of pathogenic *B. elizabethae*, a bacteria that can cause human endocarditis, in the Huayllacallán Valley in Peru (*3*).

Because most identified *Bartonella* spp. have been reported as infectious agents for humans, our results should prompt public health concern. However, our findings require further investigation about the pathogenicity of these *Bartonella* genotypes. The detection of both pathogens in intradomestic and peridomestic areas where humans are in close contact with rodents could indicate that the incidence of both diseases in humans from Echarate District might be underestimated.

This study was supported by Agencia Española para la Cooperación Internacional y el Desarrollo under Programa de Cooperación Interuniversitaria (A1/037176/11), the Spanish Ministry of Foreign Affairs and Cooperation (project Red de Investigación Colaborativa de Centros de Enfermedades Tropicales; RD06/0021/0005); and the Spanish Ministry of Health, Madrid. A.M.-A. was supported by a PhD grant from Agencia Canaria de Investigación, Innovación y Sociedad de la Información. M.A.Q.-R. was supported by a research contract from Centro de Excelencia Internacional–Plataforma Atlantica para el Control de las Enfermedades Tropicales.

Aarón Martin-Alonso, Mayday Soto, Pilar Foronda, Elsa Aguilar, Guillermo Bonnet, Rosa Pacheco, Basilio Valladares, and Maria A. Quispe-Ricalde

Author affiliations: University of La Laguna, Canary Islands, Spain (A. Martin-Alonso, P. Foronda, G. Bonnet, B. Valladares, M. A. Quispe-Ricalde); and National University of San Antonio Abad, Cusco, Peru (M. Soto, E. Aguilar, R. Pacheco)

DOI: http://dx.doi.org/10.3201/eid2006.131194

References

1. Kamani J, Morick D, Mumcuoglu Y, Harrus S. Prevalence and diversity of Bartonella species in commensal rodents and ectoparasites from Nigeria, west Africa. PLoS Negl Trop Dis. 2013;7: e2246. http://dx.doi.org/10.1371/journal.pntd.0002246
2. Gage KL, Kosoy MY. Natural history of plague: perspectives from more than a century of research. Annu Rev Entomol. 2005;50:505–28. http://dx.doi.org/10.1146/annurev.ento.50.071803.130337
3. Birtles RJ, Canales J, Ventosilla P, Alvarez E, Guerra H, Llanos-Cuentas A, et al. Survey of *Bartonella* species infecting intradomicillary animals in the Huayllacallán Valley, Ancash, Peru, a region endemic for human bartonellosis. Am J Trop Med Hyg. 1999;60: 799–805.
4. Parola P, Shpynov S, Montoya M, Lopez M, Houpikian P, Zeaiter Z, et al. First molecular evidence of new *Bartonella* spp. in fleas and a tick from Peru. Am J Trop Med Hyg. 2002;67:135–6.
5. Billeter SA, Gundi VA, Rood MP, Kosoy MY. Molecular detection and identification of *Bartonella* species in *Xenopsylla cheopis* (Siphonaptera: Pulicidae) collected from *Rattus norvegicus* in Los Angeles, California. Appl Environ Microbiol. 2011;77:7850–2. http://dx.doi.org/10.1128/AEM.06012-11
6. Hinnebusch J, Schwan TG. New method for plague surveillance using polymerase chain reaction to detect *Yersinia pestis* in fleas. J Clin Microbiol. 1993;31:1511–4.
7. Kosoy MY, Regnery R, Tzianabos T. Distribution, diversity, and host specificity of *Bartonella* in rodents from the southeastern United States. Am J Trop Med Hyg. 1997;57:578–88.
8. Chan KS, Kosoy M. Analysis of multi-strain *Bartonella* pathogens in natural host population—do they behave as species or minor genetic variants? Epidemics. 2010;2:165–72. http://dx.doi.org/10.1016/j.epidem.2010.08.002
9. Hinnebusch BJ, Gage KL, Schwan TG. Estimation of vector infectivity rates for plague by means of a standard curve-based competitive polymerase chain reaction method to quantify *Yersinia pestis* in fleas. Am J Trop Med Hyg. 1998;58:562–9.
10. Iralu J, Bai Y, Crook L, Tempest B, Simpson G, McKenzie T, et al. Rodent-associated *Bartonella* febrile illness, southwestern United States. Emerg Infect Dis. 2006;12:1081–6. http://dx.doi.org/10.3201/eid1207.040397

Address for correspondence: Pilar Foronda, Institute of Tropical Diseases and Public Health of the Canary Islands, University of La Laguna, Avda. Fco. Sanchez s/n, 38203, Tenerife, Canary Islands, Spain; email: pforonda@ull.edu.es

Buruli Ulcer Disease in Republic of the Congo

To the Editor: Buruli ulcer, which is caused by the *Mycobacterium ulcerans* bacterium, is a severe disabling necrotic disease of the skin, occurring mainly in swampy rural areas of western and central Africa. This tropical disease is neglected, despite being the third most common mycobacterial disease of humans, after tuberculosis and leprosy. The disease has become substantially more frequent over the past decade, particularly around the Gulf of Guinea, and has been detected or suspected in at least 31 countries (*1*). Clinical diagnosis of Buruli ulcer disease should be confirmed by PCR, as recommended by the World Health Organization (WHO); and case-patients should be treated with rifampin/streptomycin daily for 8 weeks (therapy available since 2004), combined, if necessary, with surgery.

Although confirmed cases of Buruli ulcer disease have been reported in all countries neighboring the Republic of the Congo (hereafter called Congo) (*2–4*), only 1 report of a confirmed case in Congo has been published (*5*) (Figure, panel A). During 2007–2012, a total of 573 clinical cases of Buruli ulcer disease were reported to WHO by the National Leprosy, Buruli Ulcer and Yaws Control Program in Congo. We report 108 cases (19% of all cases reported) that

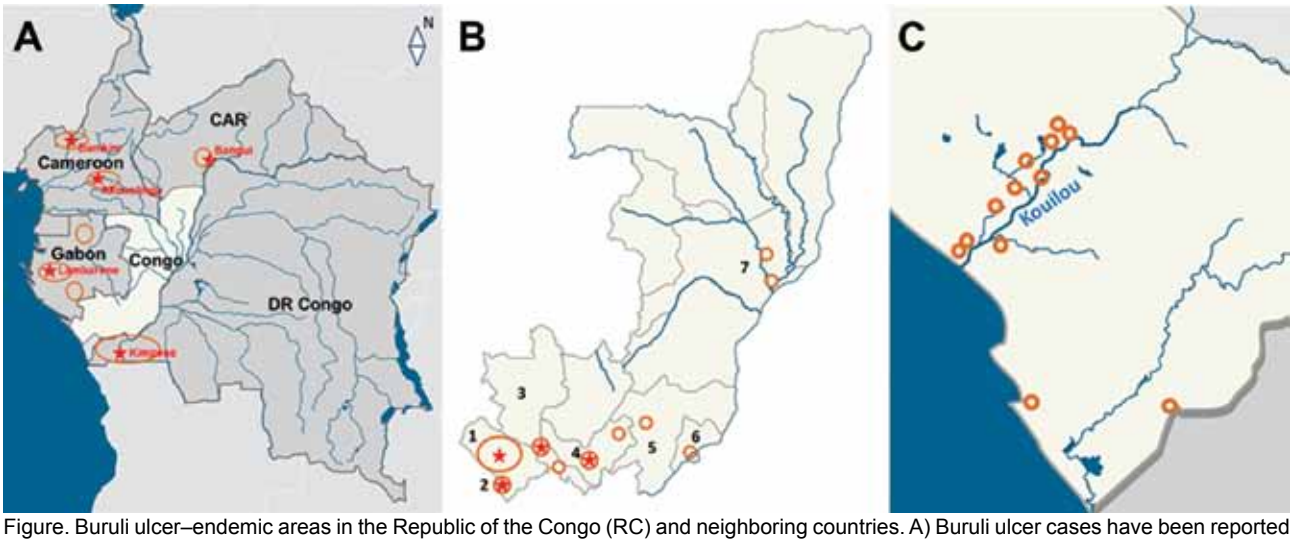

Figure. Buruli ulcer–endemic areas in the Republic of the Congo (RC) and neighboring countries. A) Buruli ulcer cases have been reported in all countries neighboring RC. CAR, Central African Republic; DR Congo, Democratic Republic of the Congo. B) RC (representing the white area in panel A). The numbers indicate the 7 departments or communes (of 12 total) where PCR-positive cases of Buruli ulcer disease were diagnosed. 1, Kouilou Department; 2 Pointe Noire Commune; 3, Niari Department; 4, Bouenza Department; 5, Pool Department; 6, Brazzaville Commune; 7, Cuvette Department. C) Kouilou Department (department 1 in panel B). Most Buruli ulcer case-patients from Kouilou Department were living close to the Kouilou River. Stars indicate locations of health centers that treat Buruli ulcer disease; circles indicate areas where persons with Buruli ulcer disease were identified. A color version of this figure is available online (wwwnc.cdc.gov/eid/article/20/6/13-1498-F1.htm)

were confirmed, in accordance with WHO recommendations, by quantitative PCR, the most sensitive and specific testing method available (*6*).

The National Leprosy, Buruli Ulcer, and Yaws Control Program, with the support of the Raoul Follereau Foundation (Paris, France), performed passive and active surveillance of Buruli ulcer in Congo during 2007–2012. Fine-needle aspirate or swab samples were obtained from patients with suspected Buruli ulcer and sent to Angers University Hospital (Angers, France) for confirmation by quantitative PCR as described (*6*,*7*). Of the 283 samples analyzed, 114 (40%) from 108 different patients were PCR positive. Of the 114 PCR-positive samples, 20 (18%) were fine-needle aspirate samples and 94 (82%) were swab samples (at least 2 swabs/lesion). The 108 case-patients included 60 (56%) female and 48 (44%) male patients; 56% of the case-patients were <15 years of age. The most common clinical form of the disease (86% of cases) was the ulcerative stage with edema or plaque. All confirmed Buruli ulcer case-patients were treated in accordance with WHO recommendations (*8*): antibiotic treatment (rifampin/streptomycin) plus surgery if necessary. All patients with nonconfirmed cases were treated according to the alternative diagnosis reached by the clinician.

Our findings show that Buruli ulcer disease affects persons in several of Congo's administrative divisions (Figure, panel B); of the 108 patients, 77 (71%) were from Kouilou Department (Figure, panel C). The village of residence was recorded for 55of these 77 patients, 46 (84%) of whom lived in 9 villages along the Kouilou River, encompassing an area of ≈50 km × 20 km: Madingo-Kayes, Kanga, Loukouala, Mfilou, Koubotchi, Mboukoumassi, Tchisseka, Magne, and Loaka villages. This disease-endemic area includes 2 lakes, Dinga and Nanga, both of which are fed by the Kouilou River. The remaining 31 (29%) confirmed patients (i.e., those not living in Kouilou Department) lived in Niari Department (9%), Bouenza Department (6.5%), Pool Department (3%), or Cuvette Department (5.5%) or in Pointe Noire Commune (2%) or Brazzaville Commune (3%) (Figure, panel B).

The distribution of Buruli ulcer cases in Congo is unusual. The Kouilou River region was most affected, but several other areas, all in southern Congo, have confirmed Buruli ulcer patients. Cuvette Department is the 1 exception; although it is in northeastern Congo, this department did have a cluster of cases. The cases in Cuvette were identified (and the infections were diagnosed and treated) during active research into Buruli ulcer during 2009–2010. (Note that there has been no survey in this region since 2010.)

Buruli ulcer is also endemic in some areas of the countries neighboring Congo. In the Democratic Republic of the Congo, the disease is highly endemic in the Bas Congo region, which shares a border with departments in southern Congo where the disease is endemic (*9*). By contrast, the small cluster of cases diagnosed in Cuvette Department in northeastern Congo seems to be isolated from other areas where the disease is known to be endemic.

M. ulcerans is known to be associated with wetlands, and the Kouilou River environment is certainly suitable for its spread (*10*). Identification of this zone as a high-risk area for Buruli ulcer disease will help the Ministry of Health improve early detection, biological confirmation, and treatment programs. In the other regions, active and continuous surveillance is necessary to establish a detailed map of the villages and areas where Buruli ulcer disease is endemic; such information would enable the implementation of targeted control activities. However, active surveillance in Congo has substantially declined since 2011. Our findings support the reactivation of such surveillance campaigns to ensure the early identification and confirmation of Buruli ulcer cases and to improve patient management.

This work was supported by the Fondation Raoul Follereau, the Institut National de la Santé et de la Recherche Médicale (INSERM, Programme INSERM Avenir); Agence Nationale de la Recherche (ANR 11 CEPL 007 [EXTRA-MU]), and Agence Nationale de Recherche sur le SIDA et les Hépatites (Programme INSERM Avenir).

Estelle Marion, Damas Obvala, Jeremie Babonneau, Marie Kempf, Kingsley B. Asiedu, and Laurent Marsollier

Author affiliations: Fondation Raoul Follereau, Pobè, Bénin (E. Marion); Centre Hospitalier Universitaire d'Angers, Angers, France (E. Marion, J. Babonneau, M. Kempf, L. Marsollier); INSERM, Angers (E. Marion, J. Babonneau, L. Marsollier); Ministère de la Santé, Brazzaville République du Congo (D. Obvala); and World Health Organization, Geneva, Switzerland (K.B. Asiedu)

DOI: http://dx.doi.org/10.3201/eid2006.131498

References

1. Walsh DS, Portaels F, Meyers WM. Buruli ulcer: advances in understanding *Mycobacterium ulcerans* infection. Dermatol Clin. 2011;29:1–8. http://dx.doi.org/10.1016/j.det.2010.09.006
2. Kibadi K, Panda M, Tamfum JJ, Fraga AG, Longatto Filho A, Anyo G, et al. New foci of Buruli ulcer, Angola and Democratic Republic of Congo. Emerg Infect Dis. 2008;14:1790–2. http://dx.doi.org/10.3201/eid1411.071649
3. Minime-Lingoupou F, Beyam N, Zandanga G, Manirakiza A, N'Domackrah A, Njuimo S, et al. Buruli ulcer, Central African Republic. Emerg Infect Dis. 2010;16:746–8. http://dx.doi.org/10.3201/eid1604.090195
4. 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
5. Kibadi K, Stragier P, Muyembe-Tamfum JJ, Pedrosa J, Portaels F. Follow-up of the first case of *Mycobacterium ulcerans* infection documented by PCR, genotyping and culture in the Republic of Congo-Brazzaville [in French]. Med Trop (Mars). 2008;68:137–43.
6. Cassisa V, Chauty A, Marion E, Ardant MF, Eyangoh S, Cottin J, et al. Use of fine-needle aspiration for diagnosis of *Mycobacterium ulcerans* infection. J Clin Microbiol. 2010;48:2263–4. http://dx.doi.org/10.1128/JCM.00558-10
7. Marion E, Eyangoh S, Yeramian E, Doannio J, Landier J, Aubry J, et al. Seasonal and regional dynamics of *M. ulcerans* transmission in environmental context: deciphering the role of water bugs as hosts and vectors. PLoS Negl Trop Dis. 2010;4:e731. http://dx.doi.org/10.1371/journal.pntd.0000731
8. World Health Organisation. Treatment of *Mycobacterium ulcerans* disease (Buruli ulcer): guidance for health workers. 2012 [cited 2013 Oct 10]. http://apps.who.int/iris/bitstream/10665/77771/1/9789241503402_eng.pdf
9. Phanzu DM, Suykerbuyk P, Imposo DB, Lukanu PN, Minuku JB, Lehman LF, et al. Effect of a control project on clinical profiles and outcomes in Buruli ulcer: a before/after study in Bas-Congo, Democratic Republic of Congo. PLoS Negl Trop Dis. 2011;5:e1402. http://dx.doi.org/10.1371/journal.pntd.0001402
10. Johnson PD, Stinear T, Small PL, Pluschke G, Merritt RW, Portaels F, et al. Buruli ulcer (*M. ulcerans* infection): new insights, new hope for disease control. PLoS Med. 2005;2:e108. http://dx.doi.org/10.1371/journal.pmed.0020108

Address for correspondence: Estelle Marion, ATOMycA, INSERM Avenir Team, U892, CHU Angers, France; email: stel.marion@yahoo.fr

Rapid Metagenomic Diagnostics for Suspected Outbreak of Severe Pneumonia

To the Editor: Recent outbreaks of severe pneumonia or acute respiratory distress syndrome (ARDS) have attracted much public interest. Given current awareness levels, clinical personnel and health officials must rapidly and adequately respond to suspected outbreaks to prevent public disturbances. We report a case that highlights the potential of next-generation sequencing (NGS) to complement conventional diagnostics in such scenarios.

On March 29, 2013, a police officer (patient 1) was admitted to the emergency department of the University Medical Centre Hamburg–Eppendorf in Hamburg, Germany, because of ARDS. The patient was given mechanical ventilation; all diagnostic test results for pathogens commonly known to cause pneumonia were negative (www.virus-genomics.org/supplementaries/EID1406.pdf). Although treatment with antimicrobial drugs was immediately initiated, the patient died 6 days later of multiple organ failure.

On April 5, a second police officer (patient 2) from the same county was admitted to the same emergency department because of ARDS. He was transferred to the intensive care unit and given mechanical ventilation. Similar to the situation for patient 1, diagnostic test results were negative, prompting the news media to suspect an outbreak of a novel or mutated virus (*1,2*). Especially because of simultaneous outbreaks of avian influenza and infections with Middle East respiratory syndrome coronavirus in other parts of the world, these reports caused serious concern among the public and health officials.

After the death of patient 1 and hospitalization of patient 2, we subjected