lineages are widespread and common in northern England. Furthermore, the considerable sequence divergence between samples in Cheshire and Northumberland is consistent with a long-standing endemicity in northern England. Given that PUUV has never been recorded in the United Kingdom (*2*,*10*), the possibility should be considered that a Tatenale-like virus could have been responsible for some of the HFRS cases that have occurred here. More studies are needed to confirm whether other common rodents in the United Kingdom are hosts for this virus and to further characterize its phyletic relationships and zoonotic potential. Cross-reactivity of the sera from Tatenale-like virus–infected individuals to antigens of other relevant hantaviruses should be determined to inform future serologic surveys and the diagnosis of human HFRS cases.

Acknowledgments

We thank Rebecca Barber, Lukasz Lukomski, Steve Price, and Ann Lowe for their assistance.

This work was funded by the Natural Environment Research Council, United Kingdom (research grant NE/L013517/2).

Ms. Thomason is a doctoral student at the School of Environment and Life Sciences at the University of Salford, Manchester, United Kingdom. Her interests are in the ecological dynamics of infectious disease in wildlife.

References

1. Pounder KC, Begon M, Sironen T, Henttonen H, Watts PC, Voutilainen L, et al. Novel hantavirus in wildlife, United Kingdom. Emerg Infect Dis. 2013;19:673–5. http://dx.doi.org/10.3201/eid1904.121057
2. Bennett E, Clement J, Sansom P, Hall I, Leach S, Medlock JM. Environmental and ecological potential for enzootic cycles of Puumala hantavirus in Great Britain. Epidemiol Infect. 2010;138:91–8. http://dx.doi.org/10.1017/S095026880999029X
3. Jameson LJ, Newton A, Coole L, Newman EN, Carroll MW, Beeching NJ, et al. Prevalence of antibodies against hantaviruses in serum and saliva of adults living or working on farms in Yorkshire, United Kingdom. Viruses. 2014;6:524–34. http://dx.doi.org/10.3390/v6020524
4. McKenna P, Clement J, Matthys P, Coyle PV, McCaughey C. Serological evidence of hantavirus disease in Northern Ireland. J Med Virol. 1994;43:33–8. http://dx.doi.org/10.1002/jmv.1890430107
5. Jameson LJ, Taori SK, Atkinson B, Levick P, Featherstone CA, van der Burgt G, et al. Pet rats as a source of hantavirus in England and Wales, 2013. Euro Surveill. 2013;18:18.
6. Jackson JA, Begon M, Birtles R, Paterson S, Friberg IM, Hall A, et al. The analysis of immunological profiles in wild animals: a case study on immunodynamics in the field vole, *Microtus agrestis.* Mol Ecol. 2011;20:893–909. http://dx.doi.org/10.1111/j.1365-294X.2010.04907.x
7. Klempa B, Fichet-Calvet E, Lecompte E, Auste B, Aniskin V, Meisel H, et al. Hantavirus in African wood mouse, Guinea. Emerg Infect Dis. 2006;12:838–40. http://dx.doi.org/10.3201/eid1205.051487
8. Ronquist F, Huelsenbeck JP. MrBayes 3: Bayesian phylogenetic inference under mixed models. Bioinformatics. 2003;19:1572–4. http://dx.doi.org/10.1093/bioinformatics/btg180
9. Nylander J. MrModeltest v2. Uppsala: Evolutionary Biology Centre, Uppsala University; 2004.
10. Pether JV, Lloyd G. The clinical spectrum of human hantavirus infection in Somerset, UK. Epidemiol Infect. 1993;111:171–5. http://dx.doi.org/10.1017/S095026880005679X

Address for correspondence: Joseph A. Jackson, University of Salford, School of Environment and Life Sciences, Peel Building, Salford M5 4WT, UK; email: J.A.Jackson@Salford.ac.uk

Measles Cases during Ebola Outbreak, West Africa, 2013–2106

Francesca Colavita,[1] Mirella Biava,[1] Concetta Castilletti, Serena Quartu, Francesco Vairo, Claudia Caglioti, Chiara Agrati, Eleonora Lalle, Licia Bordi, Simone Lanini, Michela Delli Guanti, Rossella Miccio, Giuseppe Ippolito, Maria R. Capobianchi, Antonino Di Caro; and the Lazzaro Spallanzani Institute for Research and Health Care Ebola Virus Disease Sierra Leone Study Group[2]

Author affiliations: Lazzaro Spallanzani Institute for Research and Health Care, Rome, Italy (F. Colavita, M. Biava, C. Castilletti, S. Quartu, F. Vairo, C. Caglioti, C. Agrati, E. Lalle, L. Bordi, S. Lanini, G. Ippolito, M.R. Capobianchi, A. Di Caro); Emergency Nongovernmental Organization, Milan, Italy (M. Delli Guanti, R. Miccio)

DOI: https://dx.doi.org/10.3201/eid2306.161682

The recent Ebola outbreak in West Africa caused breakdowns in public health systems, which might have caused outbreaks of vaccine-preventable diseases. We tested 80 patients admitted to an Ebola treatment center in Freetown, Sierra Leone, for measles. These patients were negative for Ebola virus. Measles virus IgM was detected in 13 (16%) of the patients.

The Ebola virus disease (EVD) outbreak in West Africa during 2013–2016 was one of the worst public health disasters in recent history; it caused >28,646 cases and 11,323

[1]These authors contributed equally to this article.

[2]Members of this group are listed at the end of this article.

deaths (*1*). Consequences of EVD include social instability, poor food reserves, breakdown of healthcare systems, and reduced vaccination coverage (*2*,*3*). Breakdown of healthcare systems and reduced vaccination coverage might have been the worst consequences because nearly all health resources were shifted to the EVD response. Disruptions of local health systems could lead to underreporting of other infectious diseases cases and a second crisis that could kill as many as persons as the original outbreak, if not more.

The 3 countries most affected by this outbreak (Sierra Leone, Guinea, and Liberia) have been a major part of the World Health Organization Expanded Programme on Immunization through vaccination campaigns for reducing childhood deaths from vaccine-preventable diseases, such as measles. Although there are no cures for EVD or measles, a potent measles vaccine is available, which can prevent spread of this disease. Use of this vaccine is crucial because measles is far more contagious (1 case-patient with measles can transmit infection to 12–18 persons) than EVD and might be the primary cause of major epidemics (*3*,*4*). These 3 countries reported nearly 93,685 cases of measles during 1994–2003 (although Sierra Leone did not report cases for 4 years), and during November 1, 2009–July 13, 2010, a total of 1,094 confirmed measles cases were reported in Sierra Leone. Plans for measles vaccination campaigns were implemented before the EVD outbreak because of an increase in susceptibility to measles in these 3 countries (*3*,*5*).

Historically, measles outbreaks have followed humanitarian crises, such as war (*6*), natural disasters (*7*), and political crises (*8*). Recent studies have shown that measles is one of the causative agents of secondary outbreaks during the EVD epidemics in West Africa (*9*), probably due to the disruption in vaccination campaigns, nonfunctional healthcare systems (including detection and reporting of measles cases), lack of specific treatment, and a sense of fear of contracting EVD with reluctance to approach health assistance (*10*).

Probable underreporting of and lack of data for measles cases during EVD outbreaks prompted us to investigate measles in Sierra Leone during the recent EVD outbreak. Although during the preparedness phase of the European Mobile Laboratory Project (http://www.emlab.eu), measles was to be included among diseases tested for differential diagnosis of EVD, we could not implement this approach in the field during the outbreak.

We performed a retrospective serologic study to partially investigate the role of measles in the EVD outbreak by testing serum samples negative for Ebola virus by reverse transcription PCR for measles virus IgM from persons suspected of having EVD. Samples were obtained at the Emergency Nongovernmental Organization Ebola Treatment Center (Goderich, Freetown, Sierra Leone). This study was approved by Ethical Committee of Sierra Leone.

We analyzed 80 patients, of whom 27 were ≥8 years of age and ≤25 years of age (median age 30 years) during December 2014–June 2015, who had fever (temperature ≥37.5°C) and diarrhea or vomiting. Only 1 patient had a history of rash. Measles virus IgM was detected by using an indirect immunofluorescence assay for 13 patients (16%), most (69%) of whom were in this age group ($p<0.001$). Although we could not determine whether measles IgM in these patients was caused by vaccination failures during the outbreak or failures of the Expanded Programme on Immunization, this age distribution might be indicative of noneffective immunization campaigns, especially a deficiency in the healthcare system in detecting and reporting measles cases.

The recent EVD outbreak caused breakdowns of healthcare systems in the affected countries, leading to possible secondary outbreaks (*2*,*3*). A higher risk for vaccine-preventable diseases, in particular measles, is often an early result in interruption in delivery of public health services. Recent studies have shown that the increase in measles cases during the EVD outbreak in 2013–2016 was caused by disruption of vaccination programs and underreporting of measles cases, which is probably related to effects of EVD on healthcare systems (*9*).

Our results for samples obtained from Ebola-negative patients showed a high number of measles infections during the outbreak in different age groups. Although few (n = 80) patients have been tested, our results provide useful insights into measles cases during other outbreaks in different age groups, adding new evidence from a study that focused on children (*9*).

Our findings indicate the need for correct and rapid differential diagnoses during such outbreaks to avoid spread of other infectious diseases. Furthermore, local public health systems should be strengthened in those countries that are now recovering from the EVD outbreak to reduce risks for other infectious diseases outbreaks.

Members of the Lazzaro Spallanzani Institute for Research and Health Care Ebola Virus Disease Sierra Leone Study Group: Antonella Vulcano, Francesca Colavita, Carolina Venditti, Paola Zaccaro, Antonio Mazzarelli, Concetta Castilletti, Angela Cannas, Serena Quartu, Sabrina Coen, Silvia Meschi, Claudia Minosse, Roberta Chiappini, Mirella Biava, Maria Beatrice Valli, Germana Grassi, and Daniele Lapa.

Acknowledgments

We thank Italian Ministry of Health (Ricerca Corrente and Ricerca Finalizzata) for supporting deployment of laboratory personnel; the Sierra Leone Ministry of Health and Sanitation, the Pharmacy Board of Sierra Leone, and the Sierra Leone National Laboratory Service for collaborating in overall laboratory activities implemented in the laboratory at the Princess Christian Maternity Hospital (Freetown, Sierra

Leone); staff (nurses, physicians, pharmacists, logisticians, administrators, cleaners, drivers, cooks, guards) at the Emergency Ebola Treatment Center (Goderich, Freetown, Sierra Leone) and the Emergency Surgical and Pediatric Hospital (Goderich) for their efforts and support; and the people of Goderich for their energy, optimism, cordiality, and positive attitude.

This study was supported by the Italian Ministry of Foreign Affairs (Directorate General for Development Cooperation). The Lazzaro Spallanzani Institute for Research and Health Care was supported by the European Union Horizon 2020 Research and Innovation Programme (EVIDENT, grant no. 666100); the European Union Innovative Medicine Initiative) Programme (projects EbolaMODRAD [grant no. 115843] and FILODIAG [grant no. 115844]); and the US Food and Drug Administration project (FDABAA-15-00121).

Dr. Colavita is research scientist at the Laboratory of Virology, Lazzaro Spallanzani Institute for Research and Health Care, Rome, Italy. Her research interests are infections with emerging viruses and risk group 3 and 4 pathogens, host immune responses, and host–pathogen interactions.

References

1. World Health Organization. Ebola Situation report, 2015 [cited 2017 Mar 29]. http://www.who.int/csr/disease/ebola/situation-reports/en/
2. United Nations Development Group. Socio-economic impact of Ebola virus disease in West African countries, 2015. New York: United Nations Development Programme [cited 2017 Mar 29]. http://reliefweb.int/sites/reliefweb.int/files/resources/ebola-west-africa.pdf
3. Takahashi S, Metcalf CJE, Ferrari MJ, Moss WJ, Truelove SA, Tatem AJ, et al. Reduced vaccination and the risk of measles and other childhood infections post-Ebola. Science. 2015;347:1240–2. http://dx.doi.org/10.1126/science.aaa3438
4. World Health Organization. Global vaccine action plan 2011–2020. 2013 [cited 2017 Mar 29]. http://www.who.int/immunization/global_vaccine_action_plan/en/
5. Truelove SA, Moss WJ, Lessler J. Mitigating measles outbreaks in West Africa post-Ebola. Expert Rev Anti Infect Ther. 2015;13:1299–301. http://dx.doi.org/10.1586/14787210.2015.1085305
6. Sharara SL, Kanj SS. War and infectious diseases: challenges of the Syrian civil war. PLoS Pathog. 2014;10:e1004438. http://dx.doi.org/10.1371/journal.ppat.1004438
7. Surmieda MR, Lopez JM, Abad-Viola G, Miranda ME, Abellanosa IP, Sadang RA, et al. Surveillance in evacuation camps after the eruption of Mt. Pinatubo, Philippines. MMWR CDC Surveill Summ. 1992;41:9–12.
8. UNICEF. Sanctions: children hard hit in Haiti [cited 2017 Mar 29]. http://www.unicef.org/sowc96/dsanctns.htm
9. Suk JE, Paez Jimenez A, Kourouma M, Derrough T, Baldé M, Honomou P, et al. Post-Ebola measles outbreak in Lola, Guinea, January–June 2015. Emerg Infect Dis. 2016;22:1106–8. http://dx.doi.org/10.3201/eid2206.151652
10. Shrivastava SR, Shrivastava PS, Jegadeesh R. Legacy of Ebola outbreak: potential risk of measles outbreak in Guinea, Sierra Leone and Liberia. J Res Med Sci. 2015;20:529–30. http://dx.doi.org/10.4103/1735-1995.163982

Address for correspondence: Antonino Di Caro, Laboratory of Virology, Lazzaro Spallanzani Institute for Research and Health Care, Via Portuense 292, 00149 Rome, Italy; email: antonino.dicaro@inmi.it

Angiostrongylus cantonensis Meningitis and Myelitis, Texas, USA

Roukaya Al Hammoud, Stacy L. Nayes, James R. Murphy, Gloria P. Heresi, Ian J. Butler, Norma Pérez

Author affiliation: McGovern Medical School, University of Texas Health Science Center at Houston, Houston, Texas, USA

DOI: https://dx.doi.org/10.3201/eid2306.161683

Infection with *Angiostrongylus cantonensis* roundworms is endemic in Southeast Asia and the Pacific Basin. *A. cantonensis* meningitis and myelitis occurred in summer 2013 in a child with no history of travel outside of Texas, USA. Angiostrongyliasis is an emerging neurotropic helminthic disease in Texas and warrants increased awareness among healthcare providers.

In summer 2013, a previously healthy Caucasian 12-month-old girl was brought for treatment to a children's hospital in Houston, Texas, USA, on the 11th day of illness (day 11), manifesting intermittent fever, lethargy, and emesis. She had been evaluated by a pediatrician on day 3 and diagnosed with presumed viral infection. She attended day care, had no history of sick contacts, and apart from dogs in the house, had no notable other exposures.

At hospital admission, physical examination showed vital signs within reference ranges, mild distress, lethargy, and irritability with no focal deficits or signs of meningeal irritation. Blood test results showed leukocytosis (17,900 cells/mm^3 with 20% eosinophils). Cerebrospinal fluid (CSF) examination showed 8 erythrocytes and 568 leukocytes/mm^3 with 26% eosinophils. Results of bacterial cultures and PCR of CSF for herpes simplex virus and enterovirus were negative. She had no serologic evidence of acute infection with West Nile virus or HIV. Magnetic resonance imaging (MRI) of the brain showed normal results. She received ceftriaxone, vancomycin, and acyclovir from days 11 through 15 with no clinical improvement.

On day 16, because the child had been exposed to dogs, she was empirically treated for presumed *Toxocara* infection with albendazole and prednisone for 5 days. Her clinical