and G1P[6] along with G2P[4] and G9P[8]. Our study results are similar to those of recent studies conducted in other African countries (5–8) and confirm results of studies that found that the same genotypes circulated in western Cameroon in 2003, albeit at different percentages (4,9).

Our study provides relevant data about the genotypes of rotavirus-A from children in the Central African Republic, 25 years after the most recent study (2). These data represent baseline information that will help with monitoring for potential changes in genotype prevalence after the introduction of rotavirus-A vaccine in the Central African Republic.

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Ionela Gouandjika-Vasilache, Alexandre Manirakiza, Jean Chrysostom Gody, Virginie Banga-Mingo, Odilon Omon Kongome, Mathew D. Esona, Michael D. Bowen, and Diane Waku-Kououmou

Author affiliations: Institut Pasteur, Bangui, Central African Republic (I. Gouandjika-Vasilache, A. Manirakiza, V. Banga-Mingo); Complexe Pédiaétrique, Bangui (J. Chrysostom Gody); Village SOS Enfants, Bouar, Central African Republic (O. Omon Kongome); and US Centers for Disease Control and Prevention, Atlanta, Georgia, USA (M.D. Esona, M.D. Bowen, D. Waku-Kououmou)

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Address for correspondence: Ionela Gouandjika-Vasilache, Str Stefan cel Mare, Nr 15B, Apt 10, 600358 Bacau, Romania; email: ionela 512@yahoo.fr

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Genome Analysis of Mayaro Virus Imported to Germany from French Guiana

To the Editor: Mayaro virus (MayV), a mosquito-borne New World alphavirus of the family Togaviridae, causes a febrile arthralgia syndrome resembling dengue and chikungunya fever. The virus is maintained in a natural cycle involving nonhuman primates and Haemagogus spp. mosquitoes in tropical rainforest areas of South America (1). After an incubation time of 7–12 days following an infectious mosquito bite, rash, fever, headache, and arthralgia develop in patients, followed by restoration to their original conditions after several weeks (1).

Outbreaks of Mayaro fever have been reported from the Amazon region (1,2). There are increasing reports of travel-related infections imported from South America to Europe and the United States (3–7). We describe an acute MayV infection in a German traveler who returned from French Guiana. Full-length MayV genome amplification was performed on virus obtained from a serum sample of the patient.

In August 2013, a 44-year-old woman (bookkeeper) came to an outpatient clinic with fever (temperature ≤38.7°C), chills, a mild headache, severe fatigue, highly painful swelling of small finger joints, and pain in both feet. Symptoms appeared 2 days before when she experienced aches in her wrists and left forefoot. Four days before, the patient had returned from a 2.5-week visit to French Guiana, where she traveled with her partner and caught butterflies. She had conducted these activities during her holidays for the past 5 years, mostly in spring or autumn. In July 2013 at the end of the rainy season, she had many mosquito bites, especially on her hands, despite use of repellents and bed nets.
Physical examination showed a body temperature of 38°C, throat enanthema, generalized macular exanthema, and slightly swollen and tender interphalangeal joints of the hands and feet. Her medical history was unremarkable, and her partner was asymptomatic. Laboratory tests showed reference values for hemoglobin concentration; platelet count; and levels of liver enzymes, creatinine, and anti–nuclear and anti–citrulline peptide antibodies. C-reactive protein level was increased (24.2 mg/L; reference value <5 mg/L), and serum lactate dehydrogenase level was slightly increased (4.4 µkat/L; reference value <4.12 µkat/L). Leukopenia (2.4 G/L; reference value 4.0–10.0 G/L) was present, which intensified the next day (2.0 g/L). The leukocyte count returned to a reference value 8 days after disease onset and the patient fully recovered.

Malaria, dengue fever, and rickettsiosis were excluded by using several tests. Blood cultures obtained on day 2 after disease onset remained sterile, and a viral infection was suspected. Follow-up investigation on day 16 of illness showed an increased IgG titer (80) against chikungunya virus (by indirect immunofluorescence assay; reference value <1:20) (6) but no IgM titer. Additional tests for alphaviruses were then performed on the same sample, and indirect immunofluorescence assay showed an IgM titer of 2,560 and an IgG titer of 10,240 (reference value <20) (6) against Ross River virus. IgM (80) and IgG (10,240) titers for antibodies against Venezuelan equine encephalitis virus, Eastern equine encephalitis virus, and Oropouche virus. IgM (80) and IgG (160) titers for antibodies against Ross River virus were low.

An acute MAYV infection was strongly suspected and a stored serum sample from day 2 underwent generic reverse transcription PCR (RT-PCR) for alphaviruses with primers VIR2052F (5’-TGGCGGCTATGATGAAATCTGGAATGTT-3’) and VIR2052R (5’-TAGCATGGTT-GTCGTCCCGATGAA-3’) (8) and quantitative MAYV real-time RT-PCR (in-house) with primers MayaroF (5’-CTCTCACAGATCAAGAC-3’), MayaroR (5’-GGCTGGAGATCAAAAGA-3’), probe labeled with 6-carboxyfluorescein (FAM) and black hole quencher 1 (BHQ-1) MayaroP (5’-FAM-CATAGACATCTCTT-GATAGACTGCCACC-BHQ1 3’) by using the AgPath-ID One-Step RT-PCR Kit (Life Technologies, Carlsbad, CA, USA) according to the manufacturer’s instructions. The generic RT-PCR for alphaviruses showed a positive result, and direct sequencing of the amplicon showed a MAYV-specific sequence. The serum sample had an MAYV viral load of 1.24 × 10^7 copies/mL when in vitro–transcribed RNA from a reference plasmid was used as a quantification standard.

Attempts to isolate MAYV in cell culture were not successful. Therefore, the serum sample was used to obtain the complete MAYV genome sequence by using primers designed from multiple alignments of the MAYV genomes obtained from databases. (Primer sequences used are available on request.) The complete MAYV genome (strain BNI-1, KJ013266) was amplified from the serum sample, and phylogenetic analysis of a 2-kb genomic fragment showed that strain BNI-1 belonged to genotype D (9) and is closely related to strains circulating in Brazil (Figure, http://wwwnc.cdc.gov/EID/article/20/7/14-0043-F1.htm).

In 2 clinic-based syndromic surveillance studies in South America, 0.8%–3% of febrile episodes were caused by MAYV infection (2,10). In travelers, MAYV infections were acquired in tropical rainforest or wildlife conservation areas (7) and were sometimes associated with insect-hunting activities (5). Successful complete genome amplification of MAYV strain BNI-1 from a clinical sample might help identify regions in the MAYV genome that undergo rapid mutations caused by the isolation process in cell culture and improve phylogenetic and functional genome analysis. Moreover, the viral load in our patient was high enough for efficient transmission of MAYV to a susceptible mosquito vector (S. Becker, pers. comm.). Thus, in disease-endemic regions, patients with an acute MAYV infection should be protected from mosquito bites during the first week of disease to prevent spread of the virus.

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This letter is dedicated to the late Ursula Herrmann (1927–2014), for making this study possible.

Barbara Friedrich-Jänicke,1 Petra Emmerich,1 Dennis Tappe1, Stephan Günther, Daniel Cadar, and Jonas Schmidt-Chanasit

Author affiliations: Institute of Tropical Medicine and International Health, Berlin, Germany (B. Friedrich-Jänicke); Bernhard Nocht Institute for Tropical Medicine, Hamburg, Germany (P. Emmerich, S. Günther, D. Cadar, J. Schmidt-Chanasit); and German Centre for Infection Research, Hamburg (J. Schmidt-Chanasit)

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1 These authors contributed equally to this article.
Diphtheria-like Disease Caused by Toxigenic Corynebacterium ulcerans Strain

To the Editor: Toxigenic Corynebacterium ulcerans is an increasingly reported cause of diphtheria in the United Kingdom and is often associated with a zoonotic origin (1,2). Here, we report a case of diphtheria caused by toxigenic C. ulcerans in a woman, 51 years of age, from Scotland, UK, who was admitted to a hospital in August 2013 with a swollen, sore throat and a gray-white membrane over the pharyngeal surface. The patient had returned from a 2-week family holiday in the state of Florida, United States, before the admission and also reported recent contact with animals in Peru. She was immediately admitted to an isolation ward and treated with a combination of clindamycin, penicillin, and metronidazole.

Microscopic examination of the throat biofilm (collected by using a swab) showed gram-positive bacilli; swab samples from the exudative membrane and throat produced small, black colonies indicative of Corynebacterium spp. on Hoyle medium. Further efforts to identify the strain by using VITEK MS and VITEK2 ANC card systems to identify the strain by using VITEK MS and VITEK2 ANC card systems (bioMérieux, Marcy l’Etoile, France) were negative for C. ulcerans and resulting reads (2,965,044 reads, ≥90x coverage. Data are available on GenBank SRA: high-throughput DNA and RNA sequence read archive (http://www.ncbi.nlm.nih.gov/Traces/sra/sra.cgi?view=search_obj, accession no.: SRR1145126) and were mapped onto the published genome sequences of a Shiga-like toxin–producing clinical isolate 809, asymptomatic canine strain BR-AD22 (3), and diphtheria-like toxin–producing strain 0102 (4). Most of the previously identified virulence genes (3,4) were present in the patient isolate (Table). The tox gene, encoding diphtheria toxin, was present, which verified the diphtheria-like disease in the patient. The rhp gene, responsible for Shiga toxin–like ribosome-binding protein, was absent. However, strain RAH1 also possessed the venom serine protease gene (vsp2), which, in C. ulcerans strain 809, has been implicated in the increased virulence in humans. The tox gene was present in a prophage that showed similarities to ΦCULC809I (3) and ΦCULC0102-I (4). Genome-based phylogenetic analysis of the RAH1 strain (ClonalFrame analysis [5]) and strains 809, BR-AD22, and 0102 indicates a much wider phylogenetic diversity of C. ulcerans strains than previously appreciated (data not shown).

This case raises the issue of waning vaccine protection in older patients and suggests that toxin-mediated corynebacterial disease remains a threat to public health. The declining costs of next-generation sequencing and availability


Address for correspondence: Jonas Schmidt-Chanasit, Bernhard Nocht Institute for Tropical Medicine, World Health Organization Collaborating Centre for Arbovirus and Haemorrhagic Fever Research and Reference, Bernhard-Nocht-Strasse 74, 20359 Hamburg, Germany; email: jonassi@gmx.de

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