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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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 antinuclear 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 MAYV. Results of serologic tests were negative for 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′-TGGCGCTAT-GATGAAATCTGGAATGTT-3′) and VIR2052R (5′-TACGATGTT-GTCGTCGCCGATGAA-3′) (8) and quantitative MAYV real-time RT-PCR (in-house) with primers MayaroF (5′-CCTTCACACAGATCAGAC-3′), MayaroR (5′-GCCTGGAAGTA-CAAAGAA-3′), probe labeled with 6-carboxyfluorescein (FAM) and black hole quencher 1 (BHQ-1) MayaroP (5′-FAM-CATAGACATCCT-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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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 treatment of a pet dog for pharyngitis. The patient was believed to have been vaccinated against diphtheria during childhood. 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 (bioMérieux, Marcy l'Etoile, France) to evaluate the swab samples suggested that the infection was caused by either *C. ulcerans* or *C. pseudotuberculosis* (50% CI). The isolate detected from this process was sent to the *Streptococcus* and Diphtheria Reference Unit, Public Health England, Colindale, UK, and was confirmed to be a toxigenic *C. ulcerans* strain that we designated RAH1. Throat swab samples were collected from family members of the patient and were negative for *C. ulcerans*. The family dog was not tested for presence of the organism, although it is known that *C. ulcerans* infections are often of a zoonotic nature (*1,2*). After treatment, the patient made a full recovery.

Toxigenic *C. ulcerans* can produce both diphtheria-like and Shiga-like toxins (*3*); to identify the genetic basis of toxin production and other potential virulence factors in this strain, a whole genome sequencing approach was applied to the isolate. The genome was sequenced by using an Ion PGM System (Thermo Fischer Scientific, Loughborough, Leicestershire, UK) and resulting reads (2,965,044 reads, ≈90× 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 *rbp* 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