Comparison of ordered nucleic acids against sequence databases might inform the synthesis set-up at companies. This comparison could be combined with the already existing protocol for nucleic acid–synthesizing companies regarding synthesis of high-risk pathogens (6). Other measures might include separate production facilities for long and short nucleic acids. The necessity for this change was highlighted by a 16th laboratory that failed to order their primers and probes through explicit routing of a company to avoid contamination with popular PCR targets. E gene–contaminated primers and probes were received at the end of March 2020.

This report provides a warning to manufacturers of oligonucleotides and diagnostic laboratories alike to remain vigilant for contamination issues in popular RT-PCR reagents. Vigilance will help avoid delays in crucial laboratory responses now and in future outbreak events.

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Dengue Virus Type 1 Infection in Traveler Returning from Benin to France, 2019

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We investigated a case of dengue virus type 1 infection acquired in Benin. Phylogenetic analysis revealed the strain belongs to genotype V but clusters with Asian, rather than with known African, strains. Our finding suggests the introduction of Asian dengue virus in West Africa.

Dengue fever, a major public health concern throughout tropical and subtropical regions of the world, is a mosquito-borne disease caused by 4 distinct dengue virus (DENV) serotypes that share antigenic relationships (DENV-1–4).

1These authors contributed equally to this article.
Although DENV is endemic to most countries in Africa, laboratory-confirmed dengue remains rare. Active transmission of DENV on the African continent lacks consistent molecular detection and characterization. In Benin, West Africa, probable cases of dengue fever have been described as far back as 1987 (1). In 2010, the first confirmed cases of dengue fever acquired in Benin were reported (2), and DENV-3 was identified (3). Since 2010, only a few cases have been reported in Benin (4). We investigated confirmed DENV-1 infection acquired in Benin in a traveler returning to France.

In February 2019, acute fever associated with intermittent headaches developed in a 16-year-old girl after she returned to her home in Marseille from a 10-day stay in urban Benin. She sought medical care 2 days after symptom onset and was hospitalized for suspected malaria at the public hospital of Marseille. She did not take antimalarial prophylaxis and did not experience symptoms during her stay in Benin. Physical examination revealed rash on her face, torso, and limbs.

At admission, laboratory results indicated a mild increase of aspartate transaminase (74 IU/L
[reference <25 IU/L]), a moderate increase in C-reactive protein (25 mg/L [reference <5 mg/L]), and hypokalemia (potassium 3.07 mmol/L [reference 3.5–4.5 mmol/L]). The rapid diagnostic test result for malaria was negative. We detected DENV nonstructural protein 1 antigen (SD Bioline Dengue Duo Combo kit; Alere [Abbott], https://www.alere.com) and DENV RNA (5) in the patient’s serum collected at admission. Serotype-specific real-time reverse transcription PCR (5) identified DENV-1. DENV serology (ELISA; Euroimmun, https://www.euroimmun.com) showed absence of IgM and presence of IgG against DENV, suggesting the patient had been previously infected by other DENVs or serologically close flaviviruses. After 48 hours of hospitalization and symptomatic treatment (acetaminophen 2 g/d), the patient recovered without complications and was discharged. She and her mother (legal guardian) provided written consent to publish her clinical and biological data collected by the Assistance Publique Hôpitaux de Marseille during her stay.

We isolated DENV from a positive sample on C6/36 (American Type Culture Collection CRL-1660) cells. We obtained the complete viral genome sequence of this DENV-1 strain, named 2019/BJ/9943 (deposited in GenBank under accession no. MN600714), from cell culture supernatant using next-generation sequencing as previously described (6).

Phylogenetic analysis showed that the 2019/BJ/9943 strain belongs to DENV-1 genotype V (Figure). Within genotype V, American-Caribbean, Asian, and African clades are strongly associated with DENV strains detected in these specific geographic regions, suggesting a local dispersion of those viruses. The African clade of DENV-1 genotype V comprises only 3 full-genome sequences, all of which come from West Africa. Surprisingly, the 2019/BJ/9943 strain belongs to the Asian clade. The Asian clade already contains another strain from Asia to Africa. Considering the lack of molecular data on DENV-1 strains from West Africa, it is difficult to estimate when the 2019/BJ/9943 strain was introduced to the continent from Asia. However, after the 2008 financial crisis, an increased number of migrant workers from China arrived in West Africa because of a rapid development of economic exchange and a gold rush in Ghana in 2013 (8). Additional molecular data from West Africa are needed to determine the effect of these population flows on the circulation of DENV between Asia and West Africa and to further clarify the epidemiology for this virus of serious public health concern.

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We report a case of human metapneumovirus infection that spread from humans to chimpanzees and back to humans. Bronchopneumonia developed in 4 of 6 members of a chimpanzee family, and 2 subsequently died. The chimpanzees’ keeper also became ill. Sequencing showed 100% identity between virus sequences from chimpanzees and the keeper.

Apes are the closest nonhuman primate relative of humans and are therefore susceptible to many human pathogens. Chimpanzees have been kept at the Ljubljana Zoo in Slovenia since 1974. The zoo had never experienced a severe or fatal case of viral respiratory infection among the chimpanzee family, which consisted of 6 members (10, 11, 13, 15, 25, and 38 years of age). We report an outbreak of human metapneumovirus (hMPV) infection in chimpanzees and a zookeeper at this zoo.

This study was performed in accordance with the Helsinki Declaration. Written consent was obtained from the human patient and archived.

On June 19, 2013, four of the youngest chimpanzees at the zoo started showing signs of a cold (snorting, sneezing, and coughing). Veterinarians suspected a viral infection, but because of the possibility of secondary bacterial infection, the chimpanzees were given amoxicillin and clavulanic acid. The next day, clinical signs of pneumonia (apathy, dyspnea, and loss of appetite) appeared. The youngest chimpanzee died of respiratory failure on June 22; necropsy showed acute bronchopneumonia.

We tested animals for influenza A and B viruses, respiratory syncytial virus, hMPV, human coronaviruses (NL63, OC43, HKU1, and 229E), human bocavirus 1, human rhinoviruses, adenoviruses, and enteroviruses by using real-time reverse transcription PCR. Only hMPV was detected in nasal and throat swab specimens and lung tissue (1). The same day, the health of the other chimpanzees deteriorated.

The second-youngest chimpanzee that had signs of acute respiratory distress was sedated, ventilated, and given Ringer solution, bronchodilators, and intravenous antimicrobial drugs. However, it died on June 24 because of bronchopneumonia and pleural effusion, which was confirmed by necropsy.

In nasal and throat swab specimens and lung tissue, only hMPV and *Klebsiella pneumoniae* were detected. Histopathologic examination of hematoxylin and eosin–stained lung tissue samples of both chimpanzees that died showed severe bronchointerstitial pneumonia, including necrosis and sloughing of bronchial and bronchiolar epithelium; alveolar spaces filled with an exudate composed of foamy macrophages, neutrophils, and fibrin; and multifocal intraalveolar hemorrhage. In the second chimpanzee, we observed multifocal manifestations of bacilli-like bacteria. For the remaining 2 ill chimpanzees, clinical signs gradually disappeared in 6 days.

The keeper of the chimpanzees was a 31-year-old man, a nonsmoker who had a history of persistent allergic rhinoconjunctivitis and childhood asthma. He reported signs of a cold on June 21. His health deteriorated over the next 2 days; he had fever, sore