into regions that are ill-equipped to manage imported cutaneous leishmaniasis has resulted in outbreaks in Turkey and Lebanon (*5*,*6*).

Our findings emphasize the importance of contemporaneous disease tracking to identify human populations at highest disease risk. To ameliorate the current cutaneous leishmaniasis crisis, particularly during the winter when cases start to appear, accurate disease monitoring and strategic training of persons based within refugee camps (medical staff, aid workers, volunteers, and military personnel) needs to be prioritized. Moreover, clinicians and other medical personnel residing in refugee-hosting countries must be suitably trained to diagnose cutaneous leishmaniasis because other local diseases (e.g., sarcoidosis and cutaneous tuberculosis) can have similar manifestations. Along with vector and rodent control, new cutaneous leishmaniasis outbreaks should be managed by prompt diagnosis and treatment, which are even more pertinent given that *L. tropica*–associated cutaneous leishmaniasis typically is resistant to several treatment regimens. In summary, the coexistence of sand fly populations and *Leishmania* spp. within refugee camps, together with the considerable influx of persons who already have cutaneous leishmaniasis, create a dangerous cocktail that can lead to an outbreak unprecedented in modern times.

References

1. Hotez PJ. Vaccine science diplomacy: expanding capacity to prevent emerging and neglected tropical diseases arising from Islamic State (IS)–held territories. PLoS Negl Trop Dis. 2015;9:e0003852. http://dx.doi.org/10.1371/journal.pntd.0003852
2. Jacobson RL. Leishmaniasis in an era of conflict in the Middle East. Vector Borne Zoonotic Dis. 2011;11:247–58. http://dx.doi.org/10.1089/vbz.2010.0068
3. Hayani K, Dandashli A, Weisshaar E. Cutaneous leishmaniasis in Syria: clinical features, current status and the effects of war. Acta Derm Venereol. 2015;95:62–6. http://dx.doi.org/10.2340/00015555-1988
4. Haddad N, Saliba H, Altawil A, Villinsky J, Al-Nahhas S. Cutaneous leishmaniasis in the central provinces of Hama and Edlib in Syria: vector identification and parasite typing. Parasit Vectors. 2015;8:524. http://dx.doi.org/10.1186/s13071-015-1147-0
5. Alawieh A, Musharrafieh U, Jaber A, Berry A, Ghosn N, Bizri AR. Revisiting leishmaniasis in the time of war: the Syrian conflict and the Lebanese outbreak. Int J Infect Dis. 2014;29:115–9. http://dx.doi.org/10.1016/j.ijid.2014.04.023
6. Koltas IS, Eroglu F, Alabaz D, Uzun S. The emergence of *Leishmania major* and *Leishmania donovani* in southern Turkey. Trans R Soc Trop Med Hyg. 2014;108:154–8. http://dx.doi.org/10.1093/trstmh/trt119
7. Inci R, Ozturk P, Mulayim MK, Ozyurt K, Alatas ET, Inci MF. Effect of the Syrian civil war on prevalence of cutaneous leishmaniasis in southeastern Anatolia, Turkey. Med Sci Monit. 2015;21:2100–4. http://dx.doi.org/10.12659/MSM.893977
8. Fawcett JM, Hay RJ. Cutaneous leishmaniasis and human conflict. Acta Derm Venereol. 2015;95:3–4. http://dx.doi.org/10.2340/00015555-1994
9. Ben Taleb Z, Bahelah R, Fouad FM, Coutts A, Wilcox M, Maziak W. Syria: health in a country undergoing tragic transition. Int J Public Health. 2015;60(Suppl 1):S63–72. http://dx.doi.org/10.1007/s00038-014-0586-2
10. Hyams KC, Riddle J, Trump DH, Graham JT. Endemic infectious diseases and biological warfare during the Gulf War: a decade of analysis and final concerns. Am J Trop Med Hyg. 2001;65:664–70.

Phylogeny of Zika Virus in Western Hemisphere, 2015

Robert S. Lanciotti, Amy J. Lambert, Mark Holodniy, Sonia Saavedra, Leticia del Carmen Castillo Signor

Author affiliations: Centers for Disease Control and Prevention, Fort Collins, Colorado, USA (R.S. Lanciotti, A.J. Lambert); Department of Veterans Affairs, Palo Alto, California, USA (M. Holodniy); VA Caribbean Health Care System, San Juan, Puerto Rico, USA (S. Saavedra); Laboratorio Nacional de Salud Guatemala, Villa Nueva, Guatemala (L. del Carmen Castillo Signor)

To the Editor: Zika virus belongs to the genus *Flavivirus*, family *Flaviviridae,* and is transmitted by *Aedes* spp. mosquitoes. Clinical signs and symptoms of human infection include fever, headache, malaise, maculopapular rash, and conjunctivitis.

Zika virus was first isolated in 1947 from the blood of a febrile sentinel rhesus monkey during a study of yellow fever in the Zika Forest of Uganda (*1*). During the next 20 years, Zika virus isolates were obtained primarily from East and West Africa during arbovirus surveillance studies in the absence of epidemics. During those 20 years, cases of Zika virus infection were detected sporadically; however, given the clinical similarity of Zika and dengue virus infections and the extensive cross-reactivity of Zika virus antibodies with dengue viruses, it is possible that Zika virus was associated with epidemics that were incorrectly attributed to dengue viruses. Beginning in 2007, substantial Zika virus outbreaks were reported first in Yap Island (Federated States of Micronesia), then in French Polynesia, and then in other Pacific Islands (*2*–*4*).

Genetic studies have revealed that Zika virus has evolved into 3 distinct genotypes: West African (Nigerian cluster), East African (MR766 prototype cluster), and Asian. It has been postulated that the virus originated in East Africa and then spread into both West Africa and Asia ≈50–100 years ago (*5*). In early 2015, cases of Zika virus infection were detected in Rio Grande State, northern

Figure. Phylogenetic tree of Zika virus isolates identified from Guatemala and Puerto Rico in December 2015 (indicated in boldface) compared with reference isolates obtained from GenBank. The isolates from Guatemala and Puerto Rico grouped with other Asian genotype viruses. The tree was derived by neighbor-joining methods (bootstrapped 1,000 times) using complete-genome sequences. Location, year identified, and GenBank strain identification for the viruses used in tree construction are shown. Scale bar indicates number of nucleotide substitutions per site. GenBank accession nos.: KU321639 (Brazil 2015 SPH2015), KJ776791 (French Polynesia H/PF/2013), KF383115 (Central African Republic ARB1362), KF383116 (Senegal 1968 ArD7117), KF383117 (Senegal 1997 ArD128000), KF383118 (Senegal 2001 ArD157995), KF383119 (Senegal 2001 ArD158084), KF268948 (CAR 1979 ARB13565), KF268949 (CAR 1980 ARB15076), KF268950 (CAR 1976 ARB7701), EU545988 (Yap 2007), KF993678 (Thailand 2013 PLCal_ZV), JN860885 (Cambodia 2010 FSS13025), HQ234499 (Malaysia 1966 P6-740), HQ234501 (Senegal 1984 ArD41519), HQ234500 (Nigeria 1968 IbH 30656), LC002520 (Uganda 1947 MR766), KU501215 (Puerto Rico PRVABC59), KU501216 (Guatemala 8375), and KU501217 (Guatemala 103344).
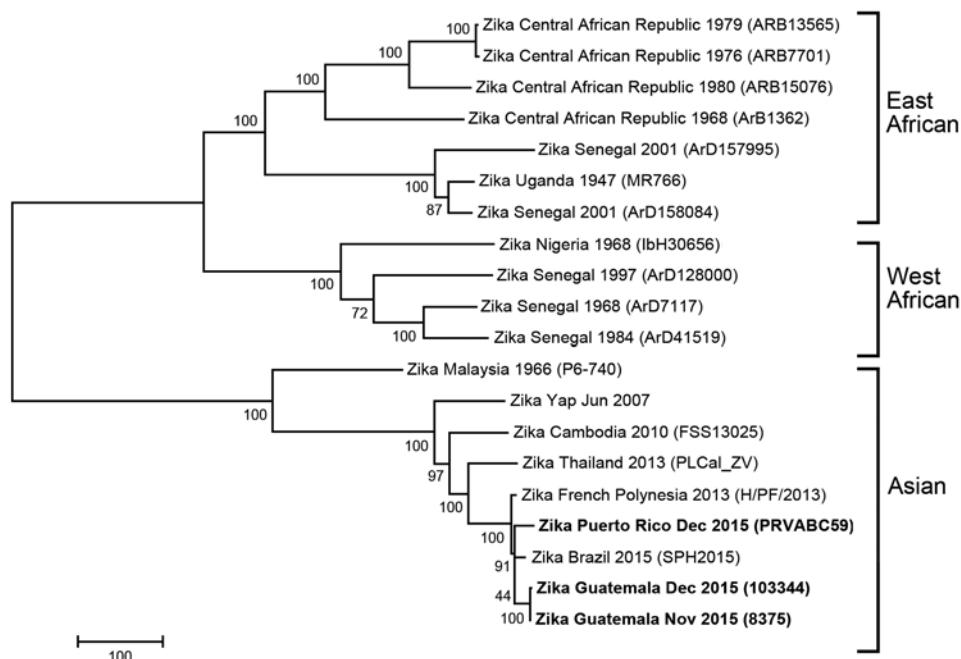
Brazil, and limited sequence analyses revealed that the virus was most closely related to a 2013 isolate from French Polynesia, within the Asian clade (*6*).

In December 2015, the Centers for Disease Control and Prevention Arbovirus Diagnostic Laboratory detected Zika virus in serum specimens collected from persons in Guatemala and Puerto Rico. The complete nucleotide sequence of the virus was derived directly from 3 of these serum specimens by using next-generation sequencing on the Ion Torrent (Thermo Fisher Scientific, Waltham, MA, USA) platform. The raw sequence reads were analyzed and assembled by using the CLC bio Genomics Workbench (CLC bio, Waltham, MA, USA) and Lasergene NextGen (DNAStar, Madison, WI, USA). The complete genome sequences were aligned by using ClustalW (http://www.megasoftware.net/) with all available full-length Zika virus sequences from GenBank representing the 3 genotypes. Nearly identical phylogenetic trees were generated by using several methods (minimum-evolution, maximum-likelihood, neighbor-joining), and a neighbor-joining tree was generated and analyzed with 1,000 replicates for bootstrap testing (Figure). GenBank accession numbers for sequences presented in this article are KU501215 (Puerto Rico PRV-ABC59), KU501216 (Guatemala 8375), and KU501217 (Guatemala 103344).

In agreement with the initial sequencing of samples from Brazil conducted by Zanluca et al. (*6*), the 3 newly sequenced Zika viruses from Guatemala and Puerto Rico are all within the Asian genotype and most closely related to strains recently isolated from Brazil (2015) and French Polynesia (2013). The tree topology confirms previous findings and indicates that Asian genotype viruses have been gradually evolving and spreading geographically throughout Asia and the Pacific Islands since at least 1966; the tree suggests that the Malaysia 1966 isolate is representative of an ancestral genotype (*7*). The percent nucleotide identity among all the Western Hemisphere Zika viruses is >99%, and as a group, these Western Hemisphere viruses are ≈89% identical (96% aa) to viruses of the East African and West African genotypes.

As reported by Musso et al. (*8*), the phylogeny and movement of Zika and chikungunya viruses are strikingly similar. Each virus is grouped into 3 genotypes of very similar geographic distribution: East Africa, West Africa, and Asia. For both viruses, it also seems that viruses from East Africa moved into Asia ≈50–100 years ago and evolved into a unique Asian genotype (*9,10*). In addition, the similarity with respect to the recent movement of these viruses from Asia into the Pacific Islands and then into the New World (*9*) is noteworthy. It seems that similar ecologic and/or human social factors might be responsible for the movement

of chikungunya and Zika viruses and into the New World at approximately the same time. Further studies might elucidate the exact mechanism of this transcontinental movement, leading to effective prevention strategies.

References

1. Dick GW, Kitchen SF, Haddow AJ. Zika virus isolations and serological specificity. Trans R Soc Trop Med Hyg. 1952;46: 509–20. http://dx.doi.org/10.1016/0035-9203(52)90042-4
2. Lanciotti RS, Kosoy OL, Laven JJ, Velez JO, Lambert AJ, Johnson AJ, et al. Genetic and serologic properties of Zika virus associated with an epidemic, Yap State, Micronesia, 2007. Emerg Infect Dis. 2008;14:1232–9. http://dx.doi.org/10.3201/eid1408.080287
3. Cao-Lormeau VM, Roche C, Teissier A, Robin E, Berry AL, Mallet HP, et al. Zika virus, French Polynesia, South Pacific, 2013. Emerg Infect Dis. 2014;20:1085–6. http://dx.doi.org/10.3201/eid2011.141380
4. Musso D, Nilles EJ, Cao-Lormeau VM. Rapid spread of emerging Zika virus in the Pacific area. Clin Microbiol Infect. 2014;20:O595–6. http://dx.doi.org/10.1111/1469-0691.12707
5. Faye O, Freire CC, Iamarino A, Faye O, de Oliveira JV, Diallo M, et al. Molecular evolution of Zika virus during its emergence in the 20th century. PLoS Negl Trop Dis. 2014;8:e2636. http://dx.doi.org/10.1371/journal.pntd.0002636
6. Zanluca C, de Melo VC, Mosimann AL, Dos Santos GI, Dos Santos CN, Luz K. First report of autochthonous transmission of Zika virus in Brazil. Mem Inst Oswaldo Cruz. 2015;110:569–72. http://dx.doi.org/10.1590/0074-02760150192
7. Haddow AD, Schuh AJ, Yasuda CY, Kasper MR, Heang V, Huy R, et al. Genetic characterization of Zika virus strains: geographic expansion of the Asian lineage. PLoS Negl Trop Dis. 2012;6:e1477. http://dx.doi.org/10.1371/journal.pntd.0001477
8. Musso D, Cao-Lormeau VM, Gubler DJ. Zika virus: following the path of dengue and chikungunya? Lancet. 2015;386:243–4. http://dx.doi.org/10.1016/S0140-6736(15)61273-9
9. Volk SM, Chen R, Tsetsarkin KA, Adams AP, Garcia TI, Sall AA, et al. Genome-scale phylogenetic analyses of chikungunya virus reveal independent emergences of recent epidemics and various evolutionary rates. J Virol. 2010;84:6497–504. http://dx.doi.org/10.1128/JVI.01603-09
10. Lanciotti RS, Lambert AJ. Phylogenetic analysis of chikungunya virus strains circulating in the Western Hemisphere. Am J Trop Med Hyg. In press 2016.

Address for correspondence: Robert S. Lanciotti, Centers for Disease Control and Prevention, 3150 Rampart Rd, Fort Collins, CO 80521, USA; email: rsl2@cdc.gov

Congenital Trypanosomiasis in Child Born in France to African Mother

Marie-Alix Lestrade-Carluer De Kyvon, Zoha Maakaroun-Vermesse, Philippe Lanotte, Gerardo Priotto, Pere Perez-Simarro, Anne-Marie Guennoc, Bertrand De Toffol, Luc Paris, Louis Bernard, Alain Goudeau, Jacques Chandenier, Guillaume Desoubeaux

Author affiliations: Centre Hospitalier Régional Universitaire de Tours, Tours, France (M.-A. Lestrade-Carluer De Kyvon, Z. Maakaroun-Vermesse, P. Lanotte, A.-M. Guennoc, B. De Toffol, L. Bernard, A. Goudeau, J. Chandenier, G. Desoubeaux); Université François-Rabelais, Tours (P. Lanotte, L. Bernard, A. Goudeau, J. Chandenier, G. Desoubeaux); World Health Organization, Geneva, Switzerland (G. Priotto, P. Perez-Simarro); Hôpitaux Universitaires La Pitié Salpêtrière–Charles Foix, Paris, France (L. Paris)

To the Editor: Sleeping sickness, or human African trypanosomiasis, is a neglected tropical parasitic infection transmitted by the tsetse fly bite. In central and western Africa, trypanosomiasis is caused by the *Trypanosoma brucei* subspecies *gambiense*. Chronic symptoms of the disease include neurologic impairment and sleep disorders (1). Infected children and adults can exhibit other nonspecific symptoms (1,2) attributable to biologic inflammatory syndrome, usually accompanied by an increase of IgM. Sleeping sickness can be fatal if left untreated (3). Although the efforts of the World Health Organization (WHO), national control programs, and nongovernmental organizations such as Médecins Sans Frontières have substantially reduced the global burden of human African trypanosomiasis, its current annual incidence is still estimated to be ≈10,000 new cases (1,4). Most cases occur during long stays in trypanosomiasis-endemic areas. Rare alternative routes of transmission are possible in nonendemic areas (e.g., through blood transfusion or organ transplantation) (5). We report the case of a 14-month-old boy infected with *Tr. brucei* through the transplacental route.

The child was referred to a pediatric care unit because of psychomotor retardation and axial hypotonia. His mother was from the Democratic Republic of the Congo (DRC) and had arrived in France 3 years earlier. The pregnancy, which had been initiated and monitored in France, was normal through delivery. Nonetheless, the newborn was placed with a foster family because his mother had vigilance disorders, aphasia, fluctuating hemiparesia and tetraparesia, convulsions associated with choreiform movements, anxiety, and severe depression. The foster family reported that the child did not smile and had been unable to grasp objects in the last 8 months.

Clinical examination showed z-scores of –2.25 SD and –1.38 SD for height and weight, respectively; multiple lymph node enlargement; and absence of tendon reflexes. Intermittent fever and dystonic movements were reported later during a subsequent hospitalization. The results from blood tests were compatible with chronic inflammatory syndrome (albumin 28 g/L; hypergammaglobulinemia with IgM 7.38 g/L and IgG 31.3 g/L; leukocytes 20.9 g/L; hemoglobin concentration 98 g/L). Cranial magnetic resonance