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.

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Congenital Trypanosomiasis in Child Born in France to African Mother

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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

imaging showed several lesions of the white matter, mostly in the left frontotemporal lobe. The boy's cerebrospinal fluid (CSF) contained glucose at 2.5 mmol/L, protein at 0.88 g/L, and 125 leukocytes/mL (100% lymph cells). Microscopic observation of CSF and blood smear highlighted *Tr. brucei* trypomastigotes (Figure, panels A and B; Video, http://wwwnc.cdc.gov/EID/article/22/5/16-0133-V1.htm).

All the clinical and biologic findings were compatible with a diagnosis of autochthonous congenital trypanosomiasis in the meningo-encephalitic second stage. In addition to diffuse bilateral lesions of her white matter and biologic inflammatory syndrome, the mother also exhibited the same neurologic symptoms as the boy, at least since her last stay in DRC, a trypanosomiasis-endemic country that currently harbors ≈90% of new cases of African trypanosomiasis worldwide (*6*). She previously lived in Kinshasa and also reported a short visit into the bush in Angola. Until trypomastigotes were observed in her son's CSF, the mother had not received a definitive diagnosis. She was thereafter invited for clinical and laboratory examination. Lumbar puncture and blood tests were then performed: Mott cells were present in her CSF, but no parasite was detected (Figure, panel C). Diagnosis of trypanosomiasis was subsequently supported by positive serologic test results (*7*).

The 2 patients were administered 400 mg/kg eflornithine monotherapy (difluoromethylornithine) daily for 2 weeks (*8*,*9*). The clinical outcome was globally satisfactory for the mother. Her serologic tests remained positive 8 months after treatment, which is long but not unusual, and confirms that serologic testing is unreliable for posttherapeutic follow up (*4*,*7*). Magnetic resonance imaging showed a substantial decrease of her brain lesions. Her son still exhibited serious neurologic sequelae, although trypomastigotes quickly disappeared from his CSF after treatment. He still had a restricted grip, axial hypotonia, and peripheral hypertonus 2 years later. To date, he has limited contact with his environment and is not able to stand up or eat alone.

Altogether, this case is exceptional for 2 reasons: 1) the diagnosis of human African trypanosomiasis in adults and children is very unusual in countries where the disease is not endemic (<100 cases are estimated to have occurred over a 10-year period) (*10*); and 2) congenital transmission seems to be extremely rare, even in Africa (*2*). To our knowledge, 17 probable cases of congenital trypanosomiasis have been reported to date, but only 14 were sufficiently described to confirm mother-to-fetus transmission (13 were attributed to *Tr. brucei gambiense*, and 1 was attributed to *Tr. brucei rhodesiense*, the eastern African subspecies) (*2*). Because not all official guidelines mention this route of transmission, the incidence of congenital trypanosomiasis is thought to be underestimated (*4*).

The diagnosis of human trypanosomiasis should always be considered for persons who have neurologic and sleep disorders and have spent time in sub-Saharan Africa as soon as infection is suspected. Congenital trypanosomiasis is rarely studied, and incidence data are limited; however, because this is another possible route of infection, even when infection of the fetus occurs long after infection of the mother, we believe congenital transmission occurs more often than suspected.

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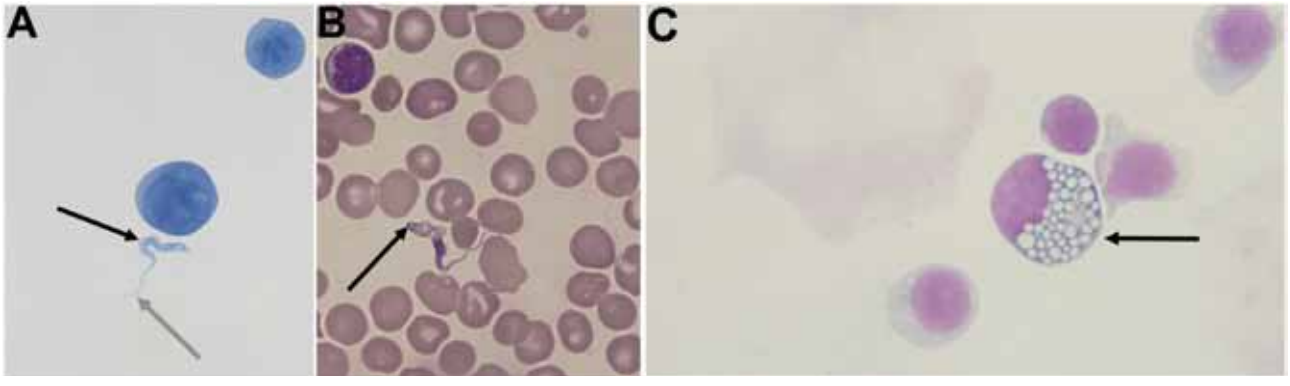

Figure. A) Cytological slide prepared from cerebrospinal fluid (CSF) from a child with congenital trypanosomiasis who was born in France to an African mother (Gram staining, original magnification ×1,000). B) Blood smear from the child (May-Grunewald Giemsa [MGG] staining, original magnification ×1,000). C) Mott cell in the mother's CSF (MGG staining, original magnification ×1,000). Trypomastigote forms of *Trypanosoma brucei* are extracellular structures, 2 × 25 μm, with a terminal flagellum (gray arrow, panel A), which is prolonged by an undulating membrane (black arrow, panel A). A central nucleus, which was difficult to visualize by Gram staining, shows that the microorganism is eukaryote. The kinetoplast (arrow, panel B) is more visible by MGG staining. It is a small organelle at the end of the cell that permits the synchronous movement of the flagellum and the undulating membrane. The kinetoplast contains circular mitochondrial DNA (*1*). The numerous mononuclear cells in CSF and blood are activated lymphocytes. The Mott cell (arrow, panel C) is a plasma cell that has spherical inclusions packed into its cytoplasm. It is often found in human trypanosomiasis (*3*). Diagnosis was subsequently confirmed by PCR. Video available online http://wwwnc.cdc.gov/eid/article/22/5/16-0133-vid1.

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Asian Genotype Zika Virus Detected in Traveler Returning to Mexico from Colombia, October 2015

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To the Editor: Zika virus is an emerging arbovirus spread by *Aedes aegypti* mosquitoes and belongs to the genus *Flavivirus* of the Spondweni serocomplex (*1*,*2*). Most often, signs and symptoms of infection are maculopapular rash, fever, arthralgia, myalgia, headache, and conjunctivitis; edema, sore throat, cough, and vomiting occur less frequently.

Zika virus is an RNA virus containing 10,794 nt, and diagnostic tests include PCRs on acute-phase serum samples to detect viral RNA (*1*). The genome contains 5′ and 3′ untranslated regions flanking a single open reading frame (ORF) that encodes a polyprotein that is cleaved into the structural proteins capsid (C), premembrane/membrane (prM), and envelope (E), and 8 non-structural proteins (NS1, NS2A, NS2B, NS3, NS4A, 2K, NS4B, and NS5) (*3*). Genetic studies in which nucleotide sequences derived from the NS5 gene were used indicated 3 Zika virus lineages: East African, West African, and Asian (*4*,*5*).

In Brazil, the first identified cases of dengue-like syndrome with subsequent Zika virus confirmation were documented in the early months of 2015 in the state of Rio Grande do Norte (*6*). Later that year, autochthonous transmission was reported in Colombia and Suriname during October–November (*5*) and Puerto Rico in December (*6*,*7*). During the same period, imported cases in the United States and Mexico were reported (*6*). By December 2015, we had already identified at least 15 autochthonous and 1 imported Zika cases in Mexico, initially detected by real-time reverse transcription PCR (RT-PCR). Here, we report on the documentation of a case of Zika virus infection in a male traveler returning to Mexico from Colombia in October 2015.

On October 21, 2015, we identified an imported case of Zika virus infection in the central state of Querétaro, Mexico. The patient, a 26-year-old man, had visited Santa Martha, Colombia, during the previous 12 days. Symptoms including fever, muscle pain, mild to moderate arthralgia, arthritis, back pain, chills, and conjunctivitis began on October 19, two days after his return to Mexico. A sample was collected at a primary healthcare clinic. Initial molecular testing for dengue virus at the