Fatal Tick-Borne Encephalitis Virus Infections Caused by Siberian and European Subtypes, Finland, 2015

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In most locations except for Russia, tick-borne encephalitis is mainly caused by the European virus subtype. In 2015, fatal infections caused by European and Siberian tick-borne encephalitis virus subtypes in the same *Ixodes ricinus* tick focus in Finland raised concern over further spread of the Siberian subtype among widespread tick species.
been bitten by ticks while at his cottage on Kuutsalo Island. Two weeks before hospitalization, he had persistent fever. By the time he was hospitalized, tetraparesis and urinary retention had developed. Magnetic resonance images showed increased signal in cerebellar vermis, facial nerves, cortical sulci, and radicular regions (Figure, panel C). CSF analysis showed pleocytosis. Serum and CSF were negative for TBEV IgM and RNA. The patient’s condition deteriorated rapidly; tetraplegia developed, and he lost consciousness despite treatment with acyclovir, doxycycline, ceftriaxone, plasmapheresis, and immunoglobulin. One week after hospitalization, his CSF was positive for TBEV IgM but his CSF, serum, and urine were RNA negative. Hypogammaglobulinemia was observed. The patient died 4 weeks after hospitalization.

Postmortem examination showed signs of severe coronary disease, cardiac hypertrophy, atherosclerosis in the aorta, and bronchopneumonia. Examination for neuropathology showed abundant perivascular lymphocytosis continuing to brain parenchyma causing glial reactivity and neuronophagy, altogether demonstrating viral encephalitis prominent in the spinal cord, brain stem, basal ganglia, and cerebellum (Figure, panel D). The brain was positive for TBEV RNA (online Technical Appendix Figure 1, panel B). A complete genome for TBEV-Eur was sequenced from the cerebellum (online Technical Appendix Figure 2).

In September 2017, a total of 80 ticks were collected from Kuutsalo Island. One, collected at the cottage of patient 2, was positive for TBEV RNA. The virus was isolated in SK-N-SH cells, and a TBEV-Eur genome was sequenced. This virus and the virus from patient 2 had 6 nt differences, resulting in 1 aa difference (F2995Y).

For both patients, progression of TBE was rapid and aggressive; neither patient had been vaccinated. Patient 1, who was young and previously healthy and who was infected with TBEV-Sib, died of brain herniation. Patient 2, who had predisposing conditions, was infected with TBEV-Eur and died of tetraplegia and subsequent complications.

In Finland, TBEV-Eur has been found atypically in *I. persulcatus* ticks, and TBEV-Sib has been found in *I. ricinus* ticks (4,5). TBEV-infected *I. ricinus* ticks are typically found in Kotka archipelago. The detection of TBEV-Eur from patient 2 was unexpected in a known TBEV-Sib focus. The high sequence similarities between the viruses from patients and ticks confirm that the infections were acquired from Kotka archipelago. This finding suggests that TBEV-Eur and TBEV-Sib co-circulate in Kotka archipelago in *I. ricinus* ticks and raises concern for further spread of TBEV-Sib in this tick species, which is widespread in Europe. The coexistence of 2 virus subtypes and the potential emergence of more pathogenic variants requires further surveys of TBEV epidemiology and consideration of vaccination guidelines.
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We analyzed blood samples from infants born with microcephaly and their mothers in Guinea-Bissau in 2016 for pathogens associated with birth defects. No Zika virus RNA was detected, but Zika virus IgG was highly prevalent. We recommend implementing pathogen screening of infants with congenital defects in Guinea-Bissau.

In 2016, the health authorities in Guinea-Bissau reported 4 cases of Zika virus infection and 5 cases of microcephaly (J) to the World Health Organization. The Zika virus strain detected in Guinea-Bissau was the African strain (J) originally detected in Africa in 1947 and in Portuguese Guinea (now Guinea-Bissau) during 1964–1965 (2). As of March 2018, the Asian strain, which has spread throughout the Americas and Cape Verde (2) and is linked to microcephaly and other congenital abnormalities, has not been reported in Guinea-Bissau (3), and the African Zika virus strain has not been linked with microcephaly.

We report an in-depth investigation of pathogens commonly associated with birth defects in 15 infants born with microcephaly in Guinea-Bissau in 2016. Field epidemiologists identified cases of microcephaly through reports from health center personnel across the country and surveillance at Hospital Nacional Simão Mendes in Bissau, Guinea-Bissau (which has 6,000 births/y). Most cases were found in the northern and eastern regions (Gabú, Bafatá, and Oio) of Guinea-Bissau (online Technical Appendix Tables 1, 2, https://wwwnc.cdc.gov/EID/article/24/5/18-0153-Techapp1.pdf). Blood samples were collected from the mothers (median age 22 years, range 15–31 years) and infants (median age 5 months, range 1 day–9 months) and sent to Statens Serum Institut (Copenhagen, Denmark) for analysis. Three infants died before sampling, and 1 sample was lost during transport; hence, we analyzed blood samples from 11 of the 15 infants with microcephaly. For comparison, we also analyzed blood samples from 10 mothers (from Tantam Cossé, Bafatá region) of infants born without microcephaly (M.W. Rosenstierne, unpub. data). We assayed for Zika virus and TORCH pathogens (Toxoplasma

Zika Virus IgG in Infants with Microcephaly, Guinea-Bissau, 2016

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