in *A. albopictus*–endemic areas of the country. The reported number of serologically proven cases probably underestimates the true extent of the diseases in Israeli travelers because underdiagnosis and underreporting are common. Both dengue and chikungunya virus infection result in viremia that may last up to 5 days, and viremic patients living in *A. albopictus*–endemic areas put the area population at risk for infection.

In summary, we report conditions in Israel suitable for autochthonous transmission of dengue and chikungunya viruses. Although no autochthonous cases have been reported in Israel, they have been reported from other countries where *A. albopictus* mosquitoes are newly endemic. In Israel and other areas where this species is newly endemic, both dengue and chikungunya virus infection should be considered in the differential diagnosis of acute febrile illnesses, even when the patients do not report recent travel to tropical areas. Enhanced surveillance may be needed to prevent epidemic spread of these diseases. Consideration must be taken to isolating suspected (viremic) dengue and chikungunya patients to prevent the establishment of autochthonous transmission.

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Possible Pet-associated Baylisascariasis in Child, Canada

To the Editor: *Baylisascaris procyonis*, a roundworm parasite of raccoons (*Procyon lotor*), increasingly is being documented as a cause of severe human disease (*1*). Approximately 130 species of wild and domesticated animals have been affected with *B. procyonis* neural larva migrans, and the parasite is increasingly recognized as a cause of human encephalitis (*2*; K. Kazacos, unpub. data). The first recognized human case was reported in 1984 in a 10-month-old child in Pennsylvania, USA (*3*). Since then, ≈30 additional cases of severe or fatal *B. procyonis* encephalitis have been reported in the United States (*4–7*; K. Kazacos, pers. comm.). To our knowledge, only 1 account of human *B. procyonis* infection has been reported in Canada (in 2009) (*8*). We report another case of human *B. procyonis* infection in Canada, indicating its probable transmission from peridomestic raccoons.

In 2008, a 14-month-old previously healthy boy in Hamilton, Ontario, Canada, sought care for fever, regression in speech for 5 days, and failure to bear weight for 2 days. His parents also noticed that he was not tracking with his eyes. Caregivers recalled a macular rash on the face and trunk that had faded over time. The child was hospitalized, and a workup for encephalitis was initiated. He was hemodynamically stable and had flaccid tone, with inability to bear weight. No visible rashes were found. A fundoscopic examination indicated no evidence of unilateral chorioretinitis. The child was unable to track objects, which suggested vision loss in both eyes. Blood cultures, urine cultures, and lumbar puncture were performed. Results of blood analyses showed the

following: lymphocytes 24% (45%–76%), monocytes 41% (3%–6%), eosinophils 32% (0%–3%), protein 34 g/L (42–74 g/L), and glucose 3.0 mmol/L (3.3–5.8 mmol/L). Magnetic resonance imaging of the brain showed diffuse white matter lesions scattered in the subcortical and deep white matter over both cerebral hemispheres and periventricular region, most prominent in the left parietal lobe and frontoparietal regions, and subtle hyperintense lesions in bilateral dentate nucleus (Figure, panel A). No meningeal enhancement was noted.

Because of eosinophilic meningoencephalitis, thorough analyses were conducted for immunologic and infectious etiologies. The family confirmed the presence of numerous raccoons in their backyard, which raised a concern for *Baylisascaris* encephalitis, and samples of cerebrospinal fluid and serum were sent to Purdue University (West Lafayette, IN, USA) for serologic testing. On the basis of clinical findings, the child was given albendazole 200 mg orally 3×/day and prednisone 25 mg orally for 4 weeks. Results of ELISA were positive for *B. procyonis* from serum (optical density = 0.744; cutoff 0.250) but negative from cerebrospinal fluid. *B. procyonis*–specific protein bands were seen on Western blotting (*9*). The parents reported that the child had no access to the backyard, but they and their dog often moved between the backyard and the house. Environmental sampling was conducted in conjunction with the public health department. Thirty samples were taken from the patient's home and yard. A sample of raccoon feces taken from a garbage bag from the porch of the house contained embryonated *B. procyonis* eggs (Figure, panel B). No eggs were identified in the dog. Nine months after the initial hospitalization, the child had substantial physical and motor delays, was legally blind in both eyes, and had epilepsy.

To our knowledge, this is the second case of *Baylisascaris* encephalitis identified in Canada. Both cases are noteworthy for profound neurologic impairment. Similar to cases reported from the United States, the case reported here highlights the dangers of peridomestic raccoons, which are becoming increasingly common in both countries. Although the classical risk factors for pica/geophagia or developmental delay were not reported by the patient's parents, he could have become infected only through ingestion of infective eggs, from an as-yet-undetermined location, object, or source. The case reported here illustrates the need for a collaborative approach in unusual cases; we included clinicians and public health and laboratory specialists in the workup of this case. We found a strong correlation between the serologic findings, the child's clinical signs, other clinical information (e.g., eosinophilia, magnetic resonance imaging findings), the age of the child, and the recovery of embryonated *B. procyonis* eggs from his environment. We postulate that he became infected by ingesting raccoon feces/infective eggs unintentionally brought into the home or through exposure in adjacent structures, such as the porch. Although no eggs were identified in the dog, several reports have documented intestinal infection of domestic dogs with *B. procyonis*, albeit at a prevalence thousands of times lower than that in raccoons (*3*; J. Yang, unpub. data). That the dog served as a vector after being exposed to raccoon feces and infective eggs is far less likely. A recent report from the Centers for Disease Control and Prevention suggests possible transmission from pet kinkajous (*10*). We speculate that more common domestic pets also might serve as possible reservoirs for and sources of infection.

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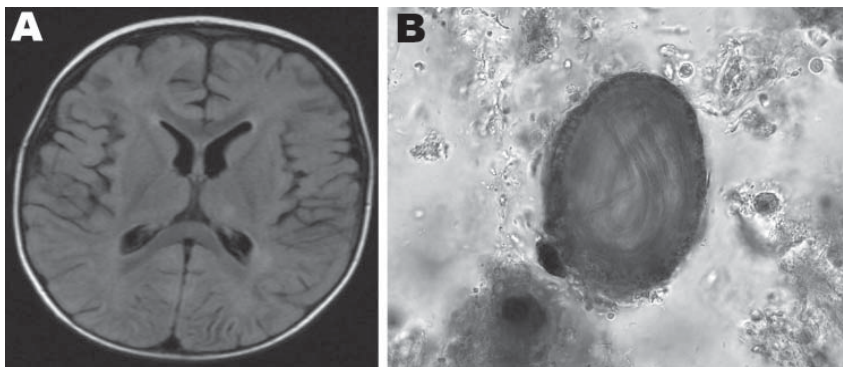
Figure. A) Magnetic resonance imaging of the brain of a 14-month-old child with baylisascariasis encephalitis. B) *Baylisascarasis procyonis* embryonated egg found in wet preparation of raccoon feces; original magnification ×100. A color version of this figure is available online (wwwnc.cdc.gov/EID/article/18/2/11-0674-F.htm).

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Zika Virus Infection, Cambodia, 2010

To the Editor: *Zika virus* (ZIKV), a member of the family *Flaviviridae,* genus *Flavivirus*, was first isolated from the blood of a sentinel rhesus monkey from the Zika Forest of Uganda in 1948 (*1*). Since that time, serologic studies and virus isolations have demonstrated that the virus has a wide geographic distribution, including eastern and western Africa; the Indian subcontinent; Southeast Asia; and most recently, Micronesia (*2–5*). The virus is transmitted primarily through the bite of infected mosquitoes and most likely is maintained in a zoonotic cycle involving nonhuman primates (*1*), although recent evidence suggests the possibility of occasional sexual transmission in humans (*4*). Few case reports have described the clinical characteristics of ZIKV infection in humans. Most reports describe a self-limiting febrile illness that could easily be mistaken for another arboviral infection, such as dengue or chikungunya fever. We report a confirmed case of ZIKV infection in Cambodia.

Since 2006, the US Naval Medical Research Unit No. 2 (NAMRU-2) has conducted surveillance for acute fever to determine causes of the infection among patients who seek health care at local clinics in Cambodia. Patients were enrolled by the health clinic physician after they gave informed consent in accordance with an institutional review board protocol approved by NAMRU-2 and the National Ethics Committee for Human Research of Cambodia. At enrollment, the physician administered a questionnaire and collected specimens (blood and throat swabs). All items were transported to the NAMRU-2 laboratory in Phnom Penh, where testing was conducted for a variety of viral, bacterial, and parasitic pathogens. In August 2010, a blood specimen was collected from a 3-year-old boy at a health clinic in Kampong Speu Province, Cambodia. The child's reported clinical symptoms included 4 days of fever and sore throat and cough and a headache for 3 days. A maculopapular rash was not observed, and the boy was not hospitalized. The clinic staff conducted a follow-up interview and reported that the patient recovered fully.

ZIKV infection was confirmed in this patient by using PCR, sequencing, and serology and through virus isolation. ELISA for chikungunya and dengue virus IgM and IgG antibodies on acute- and convalescent-phase serum was negative. A universal flavivirus real-time PCR screen that targets the nonstructural (NS) 5 gene (*6*) determined that the patient's serum was positive for flavivirus RNA, but subsequent species-specific PCR ruled out 2 other flaviviruses that are highly endemic to the region (dengue and Japanese encephalitis viruses) (*7–9*). This result was the first nondengue, non–Japanese encephalitis virus flavivirus detected after samples from ≈10,000 enrolled patients were tested. Nucleic acid sequencing of the amplicon isolated by gel purification produced a 100-bp fragment with 100% sequence identity to ZIKV (nucleotide position 8,969 of the NS5 gene of the isolate GenBank accession no. EU545988). ZIKV infection subsequently was serologically confirmed by hemagglutination-inhibition tests on paired serum samples. The patient's