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Fatal *Cronobacter sakazakii* Sequence Type 494 Meningitis in a Newborn, Brazil

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We describe a case of infection with *Cronobacter sakazakii* sequence type 494 causing bacteremia and meningitis in a hospitalized late premature infant in Brazil. We conducted microbiological analyses on samples of powdered infant formula from the same batch as formula ingested by the infant but could not identify the source of contamination.

In September 2017, a healthy boy was born at 35 weeks' gestation in Brazil. The newborn was fed breast milk and reconstituted powdered infant formula (PIF) while in the hospital. On postnatal day 4, he began sleeping more than usual and experienced hypoactivity, pallor, jaundice, seizures, metabolic acidosis, and finally respiratory insufficiency, necessitating mechanical ventilation and empiric treatment with cefepime and ampicillin. We obtained 2 blood cultures on postnatal day 4 that yielded *Cronobacter* spp. with resistance to cephalothin and cefoxitin, intermediate resistance to nitrofurantoin, and susceptibility to other antimicrobial drugs, including cefepime and ampicillin. A transfontanel ultrasound on postnatal day 6 showed grade 2 periintraventricular hemorrhage with hypoxic-ischemic lesions. Subsequent computed tomography and nuclear magnetic resonance (NMR) imaging revealed biparietal cerebral abscess (Figure). Culture of the cerebral abscess on postnatal day 33 yielded *Cronobacter* spp. that had the same pattern of antimicrobial drug susceptibility as that found in blood isolates. Because of the

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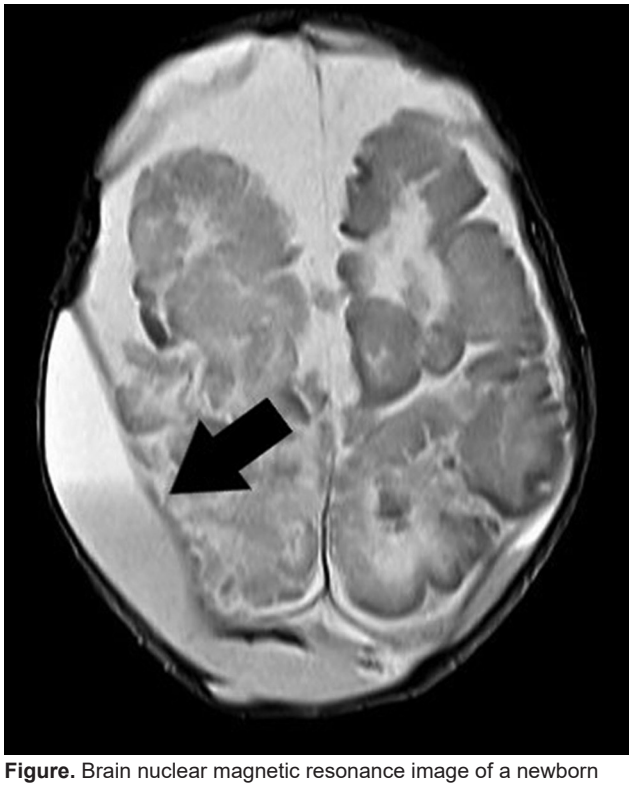

Figure. Brain nuclear magnetic resonance image of a newborn with *Cronobacter sakazakii* sequence type 494 meningitis, Brazil. Extradural collections are visible in both parietal regions. Arrow indicates the more pronounced extradural collection, measuring ≈1.8 cm, in the right parietal region.

patient's progressive clinical deterioration, we changed the antimicrobial therapy to meropenem on postnatal day 10; however, the infant failed to improve, and he died on postnatal day 46.

The mother's pregnancy was uncomplicated except for a urinary tract infection (UTI), for which she received cephalexin, in the third trimester. She experienced another UTI caused by *Enterococcus faecalis* and endometritis shortly after giving birth and underwent endometrial curettage and received ampicillin/sulbactam. We did not identify *Cronobacter* spp. in the urine or endometrial curettage material.

We were unable to analyze a sample from PIF container, the contents of which had been used. Because contaminated PIF from opened cans has been identified as the vehicle in nearly all infant *Cronobacter* infections in the past decade for which a source has been found (*1*), this lack of testing is probably the most significant limitation of this investigation. Subsequently, we sent an unopened can of the PIF from the same lot consumed by the newborn to Nacional Institute of Quality Control in Health from Oswaldo Cruz Foundation (INCQS/Fiocruz), a public laboratory in the Brazilian System of Sanitary Surveillance, where the PIF was analyzed in accordance with 2 standard procedures (*2,3*). Neither method recovered *Cronobacter.*

According to the hospital's standard procedure, all PIF was reconstituted with potable water that was heated >70°C, cooled, and used immediately. However, we could not trace the total time between the reconstitution of the PIF, the time it was maintained during cooling, and the subsequent feeding to the newborn, because the hospital does not have procedures for recording this process.

Ten newborns, 6 of whom were preterm infants, had been fed from the same lot of PIF. We followed the infants clinically during their hospital stay, and none showed signs or symptoms of *Cronobacter* infection.

We did not obtain swab specimens for surveillance. We collected environmental samples from the newborn's location after birth and from formula preparation equipment for microbiological tests in the hospital approximately 3 weeks after illness onset. However, several cleaning and disinfection procedures had been performed, limiting our chances to detect the pathogen in these samples. We collected 1 rectal swab from the newborn brother of the patient on postnatal day 4. We streaked samples onto the surface of ChromID CPS agar (bioMérieux, Rio de Janeiro, Brazil), and test results were negative for *Cronobacter*. However, this method is not specific for *Cronobacter* isolation.

Contaminated PIF and expressed breast milk have been epidemiologically linked with *Cronobacter* infections in neonates (*1,4*), and cases in Brazil have been reported in the literature (*5,6*). The magnitude of *Cronobacter* disease in Brazil is unclear, partly because it is not a compulsory notifiable disease. The most recent reported cases occurred in 2013, when *C. malonaticus* sequence types (ST) 394 and ST440 were responsible for bacteremia in 3 neonates, and the source of contamination was not identified (*5,7*).

In our study, the analyzed PIF did not show *Cronobacter* contamination. In addition, the method of PIF preparation used in the hospital (using water >70°C) would probably inactivate any *Cronobacter* present in the PIF. Because we analyzed only 1 sample, it is possible that we did not detect *Cronobacter* because contamination was not homogeneous across the lot or was below the limit of detection for our methods. We recommend the use of sterile liquid infant formulas in the hospital for patients in neonatal intensive care units unless there is no suitable alternative.

We used the *Cronobacter* MLST Database (http://pubmlst.org/cronobacter) to perform multilocus sequence typing on the 3 *Cronobacter* isolates we detected (*8*). We identified the strains as *C. sakazakii* ST494, an ST which is not in any of the recognized *C. sakazakii* clonal complex (CC) pathovars, such as *C. sakazakii* CC4, which is strongly associated with neonatal meningitis (*9*).

Cronobacter bacteria can cause severe meningitis, resulting in brain abscess formation. Virulence studies of *C. sakazakii* ST494 strains are needed to elucidate their pathogenicity and to compare with *C. sakazakii* CC4 strains.

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Introduction of Eurasian-Origin Influenza A(H8N4) Virus into North America by Migratory Birds

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We identified a Eurasian-origin influenza A(H8N4) virus in North America by sampling wild birds in western Alaska, USA. Evidence for repeated introductions of influenza A viruses into North America by migratory birds suggests that intercontinental dispersal might not be exceedingly rare and that our understanding of viral establishment is incomplete.

Research of and surveillance for influenza A viruses in wild birds inhabiting western Alaska have consistently provided support for the exchange of viruses between East Asia and North America via Beringia (*1*,*2*). Sampling of wild birds inhabiting Izembek National Wildlife Refuge (NWR) and surrounding areas in Alaska (≈55°N, 163°W) conducted during 2011–2015 has been used in recent research to identify the dispersal of influenza A(H9N2) viruses among China, South Korea, and Alaska (*3*); provide inference about the evolutionary pathways of economically important foreign-origin poultry pathogens introduced into North America (*4*); and identify sampling efficiencies for optimizing the detection of evidence for intercontinental virus exchange (*5*).

During September–October 2016, we collected 541 combined oral-pharyngeal and cloacal swab samples from hunter-harvested waterfowl (*Anseriformes* spp.) and 401 environmental fecal samples from monospecific flocks of either emperor geese (*Chen canagica*) or glaucous-winged gulls (*Larus glaucescens*) within and around Izembek NWR. Samples were deposited into viral transport media, placed in dry shippers charged with liquid nitrogen within 24 h, shipped, and stored frozen at –80°C before laboratory analysis. We screened samples for the influenza A virus matrix gene and subjected them to virus isolation; resultant isolates were genomically sequenced in accordance with previously reported methods (*5*). A total of 116 samples tested positive for the matrix gene, and 38 isolates were recovered of the following combined subtypes: H1N2, H3N2, H3N2/N6 (mixed infection), H3N8, H4N6, H5N2, H6N2, H7N3,