remains to be clarified. However, our previous hypothesis that prenatal or perinatal transient infection was an unlikely mode of virus acquisition needs to be modified because PARV4 infection in newborns has recently been demonstrated (10).

Although we lacked IgM and IgG serologic data to interpret our findings, our study suggests that PARV4 genotype 3 infection might be characterized by viral persistence, reactivation, or reinfection. Additional longitudinal studies, including serologic testing for short intervals, are needed to determine the pathogenesis and potential public health role of PARV4 infection.

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Multidrug-Resistant Salmonella enterica, Democratic Republic of the Congo

To the Editor: Salmonella enterica serotype Typhi and the nontyphoid S. enterica (NTS) are leading causes of bacteremia in sub-Saharan Africa, but little information is available from central Africa (1,2). We describe an epidemic increase of S. enterica bacteremia in Kisantu in southwestern Democratic Republic of the Congo (DRC).

The Hospital of Saint Luc in Kisantu is a 274-bed referral hospital serving a community of 150,000 inhabitants. As part of an ongoing microbiological surveillance study in DRC (3), we identified pathogens grown from blood cultures (BacT/ALERT; bioMérieux, Marcy L’Etoile, France) and assessed them for antimicrobial drug susceptibility (Vitek II system; bioMérieux) (4) and serotype (Sifin, Berlin, Germany). We determined MICs for nalidixic acid, ciprofloxacin, and chloramphenicol using the Etest macromethod (bioMérieux) (6). For salmonella isolates, we defined decreased ciprofloxacin susceptibility as an isolate with an MIC >0.064 mg/L (5) and multidrug resistance (MDR) as co-resistance of the isolate to amoxicillin, chloramphenicol, and trimethoprim/sulfamethoxazole (6).

Screening for mutations causing decreased ciprofloxacin susceptibility included assessment of the quinolone resistance–determining regions of the gyrA, gyrB, and parC genes and the plasmid-mediated qnrA, qnrB, and qnrS genes (7). Multilocus variable-number tandem-repeat analysis was performed on a subset of 37 S. enterica ser. Enteritidis isolates (8).

The pathogens isolated were S. enterica ser. Typhi (n = 17, 14.4%), Enteritidis (n = 79, 67.0%), and Typhimurium (n = 22, 18.6%). The increased incidence of S. enterica bac-
teremia was caused by an increased incidence of *S. enterica* ser. Enteritidis infection from 1 and 2 isolates reported in 2008 and 2009, respectively. The rate of infection by serotypes Typhi and Typhimurium had remained constant during this period.

During September 2010–May 2011, the proportion of pathogens isolated from blood cultures increased to 53.2% (197/370), compared with 19.7% (63/319) and 25.2% (85/328) for 2008 and 2009, respectively (p<0.001). *S. enterica* isolates represented 59.9% (118/197) of pathogens, compared with 53.3% (70/131) and 30.9% (84/272) for the same months during 2008–2009 and 2009–2010, respectively (p<0.001). Of 118 *S. enterica* samples isolated, 89 (75.4%) were isolated from specimens from children <5 years old and 17 (15.3%) from children 5–10 years old. Clinical signs and symptoms were nonspecific; malaria and gastrointestinal infection were the leading diagnoses on admission. Data for in-hospital deaths (retrieved for 87 patients) revealed case-fatality rates of 23.0% (17/74) for children <5 years old, compared with 1 in 10 patients 5–10 years old. Because of the retrospective nature of the study, it was not possible to assess population incidence rates, and we had no estimates of the number of children who were referred to the hospital but died before reaching the emergency department. There was no apparent geographic clustering, but the epidemic coincided with the onset of the rainy season, which had started late and had unusually heavy rainfall.

All NTS isolates were MDR; 1 (1.3%) *S. enterica* ser. Enteritidis and 1 (4.5%) *S. enterica* ser. Typhimurium isolate had additional decreased ciprofloxacin susceptibility. Most (16/17, 94.1%) *S. enterica* ser. Typhi isolates were resistant to amoxicillin and trimethoprim/sulfamethoxazole, 4 (23.5%) and 7 (41.2%) were MDR and had decreased ciprofloxacin susceptibility, respectively. Three combinations of resistance genes encoding decreased ciprofloxacin susceptibility were found (Table). No resistance to cefotaxime was observed. Multilocus variable-number tandem-repeat analysis typing of the *S. enterica* ser. Enteritidis isolates revealed 2 major profiles (differing in 3 tandem repeats in 1 locus) and 3 minor profiles (differing from the major profiles by 1 tandem repeat at 1 and 2 loci, respectively). Considering the long sample period, we concluded that 1 clonal type had caused the infections in which *S. enterica* ser. Enteritidis was isolated.

A recent literature review of bacteremia reported aggregated data from 16 studies from eastern (Kenya, Tanzania), western (the Gambia), and southern Africa (Malawi, Mozambique) (1). *S. enterica* ser. Typhi and NTS represented 0–42% and 9%–84%, respectively, of associated pathogens. The study reported that most NTS iso-

### Table

| Serotype   | MIC, mg/L | No. isolates | Mutations               |
|------------|-----------|--------------|-------------------------|
| Enteritidis| 128       | 0.094        | 1 Asp82-Asn in gyrA + qnrB |
| Typhimurium| >256      | 0.125        | 1 Asp87-Tyr in gyrA + qnrB |
| Typhi      | >256      | 0.19–0.25    | 7 Ser63-Phe in gyrA     |

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Co-Circulation and Persistence of Genetically Distinct Saffold Viruses, Denmark

To the Editor: Cardioviruses are positive-sense, single-stranded RNA viruses of the family Picornaviridae, genus Cardiovirus. Until recently, cardioviruses were primarily known for their ability to infect rodents. In 2007, findings of a retrospective study of undiagnosed enteric illnesses in the United States were published, including results from analysis of a fecal sample from an infant girl whose symptoms were diagnosed as fever of unknown origin in 1981. The novel human cardiovirus that was isolated was designated Saffold virus (SAFV) (1). Eight genotypes of SAFV have been described (1–4), and a ninth SAFV genotype (SAFV-2) was recently isolated in Nigeria (O. Blinkova, unpub. data). Serologic studies indicate that infection with SAFV genotypes 2 and 3 generally occurs in early life (5), although the clinical significance of these findings remains unclear.

The first SAFV infection in Denmark was recorded in 2009 (6). To elucidate the molecular epidemiology of SAFV, we performed a 3-year surveillance study of SAFV in Denmark. During 2009–2011, we tested 1,393 fecal samples from 454 children. Surveillance included collection of fecal samples from children at 6, 10, and 15 months of age; additional fecal samples were collected when the children had gastroenteritis. Most of the SAFV-positive samples reported in this study were obtained from a randomized trial in the pediatric department of University Hospital of Holbaek (Holbaek) on the effect of probiotic therapy on the incidence of infection during early childhood (M. Gyhrs, unpub. data). The study was approved by the local ethics committee; Den Regionale Videnskabsetiske Komité for Region Sjælland, Denmark.

Nucleic acids were extracted from 200-μL fecal suspension (10% in phosphate-buffered saline) by using the Cobas AmpliPrep Total Nucleic Acid Isolation Kit (Roche Diagnostics, Ltd., Mannheim, Germany) on the MagnaPure LC instrument (Roche Diagnostics). We used 5 μL of extracted nucleic acid for reverse transcription PCR (RT-PCR) (total volume 25 μL) using the OneStep RT-PCR Kit (QIAGEN, Hilden, Germany). The samples were tested for SAFV by using realtime RT-PCR primer/probe, and all positive samples were genotyped by partial sequencing of the viral protein (VP) 1 gene (6). Overall, 38 (2.8%) of the clinical samples were positive for SAFV (online Technical Appendix, wwwnc.cdc.gov/EID/point_table_v_2.0_120221.pdf), all of which fell into genotype 2 (SAFV-2), which is most prevalent in Western nations. Of these samples, 31 had sequence information of sufficient length for additional analyses. All SAFV-2 sequences were submitted to GenBank (accession nos. JX048000–JX048030).

To determine the evolutionary history of strains of SAFV identified in persons in Denmark, we combined the VP1 sequences collected here with all others available on GenBank. We aligned sequences as described using MUSCLE software (7), then checked the alignments using manual calculations. We performed phylogenetic analysis using the