To the Editor: Mulvey et al. (1) reported the emergence of ciprofloxacin resistance in *Salmonella enterica* serovar Kentucky of multilocus sequence type 198 (ST198) in Canada (1). Ciprofloxacin resistance in *S. enterica* ser. Kentucky was reported in 2011 in patients from Europe, most of whom had traveled to Africa and the Middle East. Since then, *S. enterica* ser. Kentucky ST198 with additional resistance to third-generation cephalosporins and carbapenems has been reported from France and Morocco, again associated with travel (3). Poultry has been implicated as the most likely vehicle for infection by this sequence type (2). Resistance to third-generation cephalosporins and carbapenems has not been seen in North America; however, the emergence of ciprofloxacin-resistant infections has been observed (1).

In the United States, *S. enterica* ser. Kentucky is the most common serotype isolated from chickens and the second most common found among retail chicken, but ciprofloxacin resistance has not been documented among these sources (4). We sought to determine if ciprofloxacin- or ceftriaxone-resistant *S. enterica* ser. Kentucky has emerged in humans in the United States. We examined isolates and data from the National Antimicrobial Resistance Monitoring System to document antimicrobial resistance and sequence type and to assess possible risk factors for acquiring infection.

Participating state and local public health laboratories submit every 20th nontyphoidal *Salmonella* (NTS) isolate to the Centers for Disease Control and Prevention for susceptibility testing. MICs of ≥15 antimicrobial agents were determined by using broth microdilution (Sensititer, Cleveland, OH, USA) according to the manufacturer’s instructions. Where available, Clinical and Laboratory Standards Institute performance standards were used for interpretation of MICs; otherwise, interpretations established by the National Antimicrobial Resistance Monitoring System were used (5,6).

During 2009–2012, a total of 21 (0.2%) of the 9,225 NTS isolates tested were *S. enterica* ser. Kentucky. Six (29%) were resistant to ciprofloxacin; all were susceptible to ceftriaxone (Table 5). As was observed in Canada, the 6 resistant isolates were >80% similar by pulsed-field gel electrophoresis analysis (XbaI; data not shown), and all 6 were ST198. Although a rare cause of human infection, *S. enterica* ser. Kentucky represented 23% (6/26) of all ciprofloxacin-resistant NTS detected during 2009–2012.

The median age of the 6 patients with ciprofloxacin-resistant *S. enterica* ser. Kentucky infections was 32 years (range 9 months–56 years); 5 (83%) were female. Of the 4 patients for whom information was available, 2 were hospitalized, and 1 died. Specimen sources were stool (n = 3) and urine (n = 3). Travel histories were obtained for 5 patients, and all had traveled internationally in the 7 days before specimen submission: 2 were residents of other countries (Saudi Arabia and Ethiopia), and 3 were US residents who had returned from travel to India. By comparison, only 3 of 10 patients with ciprofloxacin-susceptible infections had traveled (p = 0.02).

Resistance to ciprofloxacin in *Salmonella* is a growing concern because it limits treatment options for invasive disease. We describe ciprofloxacin-resistant *S. enterica* ser. Kentucky isolated from 6 patients in the United States. The emerging global story of *S. enterica* ser. Kentucky ST198 demonstrates the need for international integration of surveillance for antimicrobial drug resistance.

Table. Patient and isolate information for 6 cases of infection with ciprofloxacin-resistant *Salmonella enterica* serotype Kentucky sequence type 198 detected by the National Antimicrobial Resistance Monitoring System, United States, 2009–2012*.

| Isolate ID | Patient age, y/sex | Patient race | Patient travel history | Year specimen collected | Specimen type | Antimicrobial resistance† |
|------------|--------------------|--------------|------------------------|------------------------|--------------|---------------------------|
| AM41047    | <1/F               | Black        | Ethiopia               | 2009                   | Stool        | AMP, FIS, GEN, NAL, STR, TET |
| AM45820    | 54/F               | Unknown      | Unknown                | 2010                   | Urine        | AMP, COT, FIS, GEN, NAL, STR, TET |
| AM47052    | 56/F               | Asian        | India                  | 2011                   | Urine        | AMP, FIS, GEN, NAL, STR, TET |
| 2012AM-1081| 2/F                | Asian        | India                  | 2012                   | Stool        | AMP, NAL               |
| AM50773    | 37/M               | Asian        | India                  | 2012                   | Stool        | AMP, AUG, FIS, GEN, NAL, STR, TET |
| 2012AM-0353| 42/F               | White        | Saudi Arabia           | 2012                   | Urine        | AMP, FIS, FOX, KAN, NAL, STR, TET |

*ID, identification; AMP, ampicillin; AUG, amoxicillin-clavulanic acid; COT, trimethoprim-sulfamethoxazole; FIS, sulfisoxazole; FOX, cefoxitin; GEN, gentamicin; KAN, kanamycin; NAL, nalidixic acid; STR, streptomycin; TET, tetracycline.
†Resistance of isolate from infected patient to antimicrobial agents other than ciprofloxacin.
Unique Strain of Crimean–Congo Hemorrhagic Fever Virus, Mali

To the Editor: Crimean-Congo hemorrhagic fever (CCHF) is an acute viral infection that causes mild to severe hemorrhagic fever characterized by petechiae, ecchymosis, disseminated intravascular coagulation, and multi-organ failure (1). The etiologic agent, CCHF virus (CCHFV; family Bunyaviridae, genus Nairovirus), is maintained in enzootic cycles involving agricultural and wild animals and the vector, *Haemoloma* ticks (2). CCHF predominantly affects persons who have 1) substantial contact with ticks and/or agricultural animals in areas where CCHF is endemic or 2) close contact with infected persons, predominantly close relatives and health care workers. The case-fatality rate for CCHF is generally accepted as 30% (1).

CCHF has a wide geographic distribution; cases have been reported in >30 countries across Africa, southeastern Europe, the Middle East, and western Asia. In the western African countries of Nigeria, Mauritania, and Senegal, serologic evidence of CCHFV infections in humans and agricultural animals has been documented frequently (3–5); however, reports of the disease in humans have been limited to Senegal and Mauritania (6,7). In neighboring Mali, where the tick vector is known to be present, little information exists regarding the presence of CCHFV. Thus, to determine if the virus is circulating undetected in Mali, we conducted a study to determine if CCHFV is present in *Haemoloma* ticks in the country.

In November 2011 and March 2012, unfed *Haemoloma* ticks (adults and nymphs) were collected from 20 cattle at the Daral livestock market (12° 49.855′ N, 08° 05.651′ W) near the town of Kati, Mali, ≈25 km from the capital, Bamako. In the field, ticks were visually identified to genus and pooled accordingly (3–4 ticks per pool, all collected from the same animal). A total of 23 tick pools, representing 80 ticks, were manually homogenized, and RNA was extracted and tested for the presence of CCHFV RNA by using in-house assays that selected for 3 virus genes. Of the 23 tick pools tested, 1 was positive for CCHFV by all 3 assays. Phylogenetic analysis of the complete nucleocapsid protein gene (KF793333) showed that the CCHFV strain from Mali most closely resembled a strain from Mauritania (GenBank accession no. ArD39554), sharing 98% sequence identity (Figure, panel A).

Further analysis of fragments of the medium segment (pre-Gn coding region, KF793334) and large segment (polymerase coding region, KF793335) confirmed these findings, showing sequence identities of 91% and 98%, respectively, with ArD39554 (Figure, panels B, C). In a Biosafety Level 4 facility at Rocky Mountain Laboratories, Hamilton, Montana, USA, the original homogenates from the positive pool were passaged in multiple cell lines. After 3 passages, no discernible cytopathic effect was observed and, aside from the initial passage, CCHFV RNA was not detected.

Genetic identification of ticks in the CCHFV RNA–positive pool was conducted as described (8,9). Amplified sequences most closely resembled those of *H. dromedarii* (97.2%–100% sequence identity), although genetically, we cannot exclude the possibility that *H. truncatum* and *H. rufipes* were present with individual sequence identities of >97% to published sequences.

The Daral cattle market in Kati is the largest of its kind in Mali, and animals from across the country come into the market every week. Although the market provided a convenient opportunity for collecting ticks, we cannot determine where the infected