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## Multidrug-Resistant Shiga Toxin–Producing *Escherichia coli* O118:H16 in Latin America

**To the Editor:** We report the first isolation of a multiple antimicrobial drug-resistant strain of Shiga toxin-producing *Escherichia coli* (STEC) O118:H16 from cattle in Latin America. The strain was isolated during a study of fecal STEC in 205 healthy and 139 diarrheic cattle on 12 beef farms in the state of São Paulo, Brazil, in February 2000; one case of STEC was found in a 1-month-old calf with diarrhea. This bovine STEC O118:H16 strain showed resistance to eight antimicrobial substances; the following resistance (R)-genes were detected: ampicillin (*bla*<sub>TEM1-like</sub>), kanamycin and neomycin (*aphA1*), streptomycin (*strA/B*), sulphamethoxazol (*sul2*), tetracyclin (*tet[A]*), trimethoprim (no *dfpA1, A5, A7, A12, A14, or A17*), and trimethoprim/sulphamethoxazol. The STEC O118:H16 strain from Brazil was found to be similar for virulence genes (Shiga toxin 1 [*stx1*], intimin beta 1 [*eae β1*],

and EHEC-hemolysin [*E-hlyA*]) and for antimicrobial drug resistance to STEC O118:H16 strains, which were isolated in different countries of Europe (1). Beginning in 1986, STEC O118:H16 was identified as an emerging pathogen for calves and humans in Belgium and Germany (2–4). Cattle and human STEC O118:H16 isolates were similar in virulence attributes and antimicrobial drug resistance and belonged to a distinct genetic clone (1). Transmission of these pathogens from cattle to humans on farms was observed (5).

Beginning in 1996, STEC O118:H16 has become important as an emerging pathogen in humans and has been associated with bloody diarrhea and hemolytic uremic syndrome (2). Analysis of the antimicrobial resistance profiles showed that >96% of the European STEC O118:H16 strains showed resistance to one or more antimicrobial drugs in contrast to the 10% to 15% drug-resistant strains that were detected among STEC belonging to other serotypes (1,6,7). STEC O118:H16 showing multi-resistance in up to eight different antimicrobial drugs predominated among younger isolates, indicating

that drug resistance genes have accumulated over time in STEC O118:H16 strains. The frequency of antimicrobial drug resistance in STEC and Stx-negative *E. coli* in humans and animals was compared in a study by Schroeder et al. (8). Among human clinical *E. coli* isolates, antimicrobial resistance was less frequently observed in STEC than in Stx-negative strains, whereas in cattle, antibiotic-resistant strains were found at similar frequencies in both groups of *E. coli*. The relatively higher frequency of antimicrobial-resistant STEC in cattle was explained by the use of antimicrobial drugs in cattle production, whereas human infections with STEC are generally not treated with antibiotics (8). Cattle could thus be an important source of new emerging antibiotic-resistant STEC strains such as O118:H16.

The genetic basis of antimicrobial resistance in STEC O118:H16 is broad, including R-plasmids, integrons, transposons, and chromosomally inherited drug-resistance genes. Fluoroquinolone resistance has also been acquired by some STEC O118:H16 strains (1). The heterogeneity of antimicrobial drug-resistance patterns, the increase of multidrug-resistant strains over time

of isolation, and the evidence for multiple acquisition and genetic location of R-determinants indicate that strains belonging to the STEC O118:H16 clone have a propensity to acquire and accumulate R-genes. The finding that multidrug-resistant STEC O118:H16 is isolated from cattle in South America indicates the global spread of this new emerging EHEC type.

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### Correction, Vol. 9, No. 6

In “Serogroup W-135 Meningococcal Disease during the Hajj, 2000,” p. 665, author Sahar Makki was inadvertently not included. The correct author list should read as follows:

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