First Shiga Toxin-Producing *Escherichia coli* Isolate from a Patient with Hemolytic Uremic Syndrome, Brazil

To the Editor: Infection by Shiga toxin (Stx)-producing *Escherichia coli* (STEC), particularly strains of serotype O157:H7, can cause sporadic cases and outbreaks of diarrhea, hemorrhagic colitis (HC), and hemolytic uremic syndrome (HUS) (1). Some other serotypes (e.g., O26:H11, O111:H8, O111:NM, and O113:H21) share a similar pathogenic potential. STEC are distributed worldwide, but most of the HC and HUS cases were reported from industrialized nations of the Northern and Southern Hemispheres (2). In South America, HUS is a major cause of acute renal failure in infants in Argentina (3) and Chile (4). However, in Brazil human STEC infections have been restricted to sporadic cases of nonbloody diarrhea (5,6). Although a high frequency of STEC strains was recently found in foods and animal reservoirs (7,8), only some of the serotypes identified in animals (8) were recognized as causes of human illness (e.g., O157:H7, O22:H16, O82:H8, and NT:H21). Moreover, there is currently no nationwide surveillance system for HUS in Brazil, and STEC-associated HUS has not been previously reported in our country.

We describe the case of an 8-month-old boy from a northeastern state in Brazil, who was admitted to the emergency room of Hospital São Paulo, São Paulo, on March 17, 2001; the boy had anemia, oliguria, and edema of lower extremities. He had an acute diarrheal prodromal illness 3 weeks before hospital admission. On the same day as admission, respiratory failure developed, and the child was transferred to the pediatric intensive-care unit of the hospital. The boy had hemolytic anemia (hemoglobin level 11.9 g/dL at admission, and 9.1 g/dL several days later), renal failure (blood urea nitrogen 43.8 mg/dL and serum creatinine 1.5 mg/dL), and thrombocytopenia (platelet count of 70,000/mm^3), leading to a diagnosis of HUS. The patient received treatment with fresh frozen plasma and needed renal support (peritoneal dialysis) for 7 days. Once renal function was reestablished, the patient's outcome was good.

Feces were collected as soon as HUS was suspected and plated onto MacConkey Sorbitol Agar (Difco, Becton Dickinson Microbiology Systems, Sparks, MD). Only sorbitol-positive colonies grew and were biochemically identified as *E. coli* by standard procedures. The *E. coli* isolates expressed Stx1, as identified by cytotoxicity and neutralization assays on Vero cells (5). Presence of *stx1* and intimin (*eae*) gene sequences was confirmed by polymerase chain reaction (9,10). The *E. coli* strain belonged to serotype O26:H11 and produced enterohemorragic *E. coli* hemolysin (enterohemolysin).

This report is the first on the isolation of an STEC strain in a HUS patient in Brazil. The serotype O26:H11 has been described as an agent of HC and HUS in other countries and was the second most frequent serotype found in STEC strains isolated from diarrheal cases in our settings (6). Moreover, expression of Stx1 and enterohemolysin and the presence of *eae* are virulent characteristics usually found in the human STEC strains isolated so far in Brazil. These findings show the importance of looking for non-O157 STEC strains besides O157:H7 in patients with HC and HUS in Brazil. Surveillance for HUS, either nationally or in sentinel population-based studies, should be performed in Brazil, and studies on the occurrence of HUS and its association with STEC infections are under investigation in our laboratory.

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Emergence of Vancomycin-Intermediate *Staphylococcus aureus* and *S. sciuri*, Greece

To the Editor: Staphylococcal isolates with reduced susceptibility to glycopeptides, such as vancomycin and teicoplanin, are a serious public health problem because staphylococci frequently show multidrug resistance, and glycopeptides are the only remaining effective drugs. Since the early reports of glycopeptide-resistant staphylococci, teicoplanin resistance has become more common than vancomycin resistance, particularly among coagulase-negative staphylococcal species (1-3). In cases of staphylococci with reduced susceptibility to vancomycin (vancomycin-intermediate staphylococci), an increasing number of strains showing heteroresistance are reported (strains that contain subpopulations of cells at frequencies $\geq 10^{-6}$ for which the vancomycin MICs are 8 μg/mL to 16 μg/mL); homogeneous resistance still appears to be rare (2,4-7). In northern Greece, resistance to teicoplanin has recently been documented in *S. haemolyticus* strains isolated from clinical infections (8). We report the first bloodstream infections in Greece associated with *S. aureus* and *S. sciuri* strains that have homogeneous intermediate-resistance to vancomycin (MIC = 8 μg/mL).

In our department, all clinically significant staphylococcal isolates are screened for reduced susceptibility to vancomycin and teicoplanin by an agar incorporation method (9), which has been routinely performed since January 1999. An inoculum of 10^4 CFU/spot from a log-phase broth culture was spread on Mueller-Hinton agar plates containing appropriate antibiotic concentrations. The strains were incubated for a full 24 hours before the MICs were read. When a reduced susceptibility to vancomycin was observed (MIC 8 to 16 μg/mL), the test was repeated for confirmation of the result and the strains were also tested by National Committee for Clinical Laboratory Standards (NCCLS) broth microdilution (9) and E-test (AB Biodisk, Solna, Sweden) with BHI agar (Oxoid, Ltd., Basingstoke, Hampshire, UK) and an inoculum density adjusted to 0.5 McFarland value. *S. aureus* ATCC 29213, which had MICs for vancomycin of 1 μg/mL and for teicoplanin of 0.5 μg/mL, was used as a control for the estimation of the MICs. Two vancomycin-intermediate staphylococcal isolates (one *S. aureus* and one *S. sciuri*) were recovered in our hospital during December 2000 and April 2001, respectively. The organisms were identified with the Vitek system (bioMerieux Vitek, La Balme les Grottes, France). Slide-coagulase test and Staph ID 32 API system (API system, bioMerieux) confirmed identification. Susceptibility to 18 antimicrobial agents was evaluated with the Vitek system according to the recommendations of the manufacturer, and carriage of the *mecA* gene was confirmed with a polymerase chain reaction (PCR) that amplifies a 449-bp product.

The first strain (*S. aureus*) was recovered from a 52-year-old man who was hospitalized after a severe traffic accident. The patient had multiple injuries, including an external laryngeal trauma, pelvic ring disruption, and various fractures of the extremities. He underwent immediate tracheotomy, and a neurosurgical operation was performed to evacuate an extracerebral hematoma. Ceftazidime, clindamycin, ciprofloxacin, metronidazole, teicoplanin, and vancomycin were periodically administered as prophylaxis. An oxacillin-resistant *S. aureus* isolate was recovered from two blood cultures 4 weeks after the patient's admission. The strain was also resistant to tobramycin, macrolides, tetracyclines, rifampicin, and fusidic acid, and had intermediate resistance to vancomycin (MIC 8 μg/mL) and teicoplanin (MIC 16 μg/mL) by all tested methods (agar dilution, broth microdilution, and E-test). The strain was susceptible to chloramphenicol, cotrimoxazole, fosfomycin, gentamicin, kanamycin, nitrofurantoin, and ofloxacin. The removal of an intravenous catheter and treatment with gentamicin and vancomycin eradicated the infection.

The second strain (*S. sciuri*) was recovered from a 35-year-old man who was an intravenous drug user. He was admitted with renal failure, electrolyte disturbances, and acute respiratory distress, which necessitated intubation and mechanical ventilation. The patient became febrile, and multiple courses of antibiotics (amikacin, cefepime, ciprofloxacin, metronidazole, and vancomycin, alone or in combinations) were administered before the *S. sciuri* strain was isolated. Seven weeks after his admission, an oxacillin-resistant *S. scuiri* strain that had cross-resistance to aminoglycosides, macrolides, quinolones, rifampicin, and tetracycline was found in subsequent blood cultures. The MIC of the strain for vancomycin was 8 μg/mL and for teicoplanin 16 μg/mL by the agar dilution method, and the result was confirmed by the E-test and the broth microdilution method. The strain was susceptible only to cotrimoxazole, fosfomycin, and nitrofurantoin. The patient improved clinically and was subsequently discharged on cotrimoxazole and vancomycin therapy.