hyperproduction of the chromosomal enzymes, as well as resistant strains without synergy, were disregarded. During the first period, 15 (2.1%) of 707 outpatients were carriers of *Escherichia coli* (14 patients) or *Proteus mirabilis* (1 patient) with ESBL. This percentage increased during the second period, when 17 (3.8%) of 454 outpatients were carriers of *E. coli* with ESBL, and again in the third period, when 12 (7.5%) of 160 were carriers of *E. coli* (11 patients) or *Enterobacter cloacae* (1 patient) with ESBL. Characterization of the different ESBL isolated during the three study periods is in process. Although *Klebsiella pneumoniae* carrying ESBL has been detected in our hospital (7), as well as in other hospitals in Barcelona (8), no ESBL-producing *K. pneumoniae* strains were identified in this survey.

Although we did not disregard either the patients' previous treatment with antibiotics or previous hospitalization, these patients came to the hospital from the community carrying strains that express ESBL. Moreover, during these three periods we observed a significant increase in the frequency of ESBL carriers (from 2.1% to 7.5%; $p<0.005$). These data suggest that the community could be a reservoir for these enzymes, as occurs with other microorganisms (9–11). Many questions remain unanswered regarding the diffusion mechanisms of this resistance in the community. Confirmation of community-based transmission of ESBL would indicate a need for heightened vigilance and further studies to determine the reservoirs and vehicles for dissemination of ESBL within the community.

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Polymyxin-Resistant *Acinetobacter* spp. Isolates: What Is Next?

To the Editor: In Brazilian hospitals, *Acinetobacter* spp. has been an important etiologic agent of nosocomial infections, mainly pneumonia (1–3). In general, ampicillin/sulbactam and carbapenems remain the last therapeutic options for treatment of such infections (3,4). However, resistance rates to carbapenems have increased, reaching rates approximately 12% or higher in some Brazilian hospitals (1,3,4). Thus, more toxic agents such as polymyxins have been used as alternative therapeutic drugs against multidrug-resistant *Acinetobacter* infections (5,6). The clinical use of polymyxins has been based on antimicrobial susceptibility results and previous clinical experience. However, the National Committee for Clinical Laboratory Standards (NCCLS) documents do not currently provide interpretative criteria for the testing of polymyxins (7). In addition, the disk diffusion technique was reported to be an unreliable method for evaluating the susceptibility to polymyxins (8). Since *Acinetobacter* clinical specimens exhibiting high MICs for polymyxins (MIC, 8–32 μg/mL) were recently detected, we searched for the frequency of occurrence of *Acinetobacter* spp. strains exhibiting reduced susceptibility to polymyxin B among 100 bloodstream isolates of *Acinetobacter* spp. (8). The bacterial isolates were consecutively collected between September 1999 and December 2000 from a tertiary Brazilian hospital,

where *Acinetobacter* spp. infections have reached endemic levels and polymyxins have been frequently used. Only one isolate per patient was included in the study.

The isolates were identified to the species level using the BBL Crystal System (Becton Dickinson, Sparks, MD). The susceptibility to polymyxin B and meropenem were tested by disk diffusion and agar dilution techniques according to NCCLS recommendations (9,10). The susceptibility interpretative criteria for meropenem and polymyxin B were based on the current and former NCCLS documents, respectively (7,11). The MIC was defined as the lowest antimicrobial concentration that inhibited bacterial growth. *Pseudomonas aeruginosa* ATCC 27853, *Staphylococcus aureus* ATCC 25923, and *Escherichia coli* ATCC 25922 were used as quality control strains. Testing errors and agreements were determined by comparing the results of the disk diffusion with the standard criterion agar dilution method. Categorical agreement was obtained when the isolates were classified within the same susceptibility category. The very major and major errors were related to false susceptibility and false resistance results, respectively. To evaluate whether the polymyxin B-resistant strains isolates were epidemiologically related, these isolates were molecularly typed by pulsed-field gel electrophoresis (PFGE) as previously described (12). PFGE patterns were considered identical if they shared every band, similar if they differed from one to three bands, and distinct if they differed by four or more bands (12).

Despite the limitation of commercial systems for identifying the genus *Acinetobacter* at species level, *Acinetobacter baumannii* (80.0%) was the most commonly identified species, followed by *A. lwoffi* (4.0%). Sixteen percent of the *Acinetobacter* isolates were not identified to species level by the BBL Crystal System. Meropenem (MIC50, 1 μg/mL) and polymyxin B (MIC50, 1 μg/mL) showed similar in vitro potency. However, meropenem exhibited the highest susceptibility rate (99.0% susceptible). In contrast to previous studies, only one strain was resistant to meropenem (1,2,3,8), which indicates that the carbapenem-susceptibility rates among *Acinetobacter* spp. isolates may vary according to the period evaluated even in the same institution. By using the polymyxin B resistance breakpoint (MIC ≥4 μg/mL) presented by the former NCCLS document, which was recently validated, we found that five *Acinetobacter* spp. isolates were considered resistant to polymyxin B (MICs, 8–32 μg/mL) (8,11). All isolates were susceptible to meropenem and belonged to *A. baumannnii* (4) and *A. lwoffi* (1) species. The polymyxin B–resistant isolates were categorized as susceptible by disk diffusion (100%, very major error). The disk diffusion method is widely used in Brazil and worldwide. However, disk diffusion was confirmed to be an unreliable test for detecting *Acinetobacter* spp. isolates with reduced susceptibility to polymyxins. These results are in agreement with those previously reported (8).

Among the five polymyxin B–resistant *Acinetobacter* spp., four distinct patterns were characterized by PFGE. Two polymyxin B–resistant strains, which were isolated from different units of the São Paulo Hospital complex, shared an identical PFGE pattern. The PFGE results suggest that the polymyxin B use may have played a role in the selection of resistant strains. On the other hand, two isolates shared an identical PFGE pattern, which raises the possibility of patient-to-patient transmission of epidemic strains. Intra- and interhospital dissemination of multidrug-resistant *Acinetobacter* spp. clones has already been reported in Brazilian hospitals (13).

Our findings suggest that the polymyxin B–resistant strains have emerged because of antimicrobial selective pressure and dissemination of clonal strains. Further epidemiologic studies are necessary to correlate the emergence of polymyxin-resistant *Acinetobacter* spp. isolates to the clinical response with polymyxin B therapy. Since the emergence of polymyxin B resistance may leave no efficacious drugs for the treatment of infections caused by multidrug-resistant *Acinetobacter* spp. isolates, strict infection control measures must be adopted to avoid the emergence and spread of such isolates. The low accuracy of routine susceptibility tests, especially disk diffusion, may jeopardize rapid implementation of such measures.

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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 ($bla_{\text{TEM1-like}}$), kanamycin and neomycin (*aphA1*), streptomycin (*strA/B*), sulphametoxazol (*sul2*), tetracyclin (*tet*[A]), trimethoprim (no *dfrA1, 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 [*stx*1], intimin beta 1 [*eae* β1], and EHEC-hemolysin [E-*hly*A]) 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 multiresistance 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 heterogenicity of antimicrobial drug–resistance patterns, the increase of multidrug-resistant strains over time