land. Furthermore, *Ct. felis* often feeds on humans.

Clinicians encountering patients with fever or rash (or both) and a history of cat contact or flea bites should consider a diagnosis of *R. felis*. Laboratory confirmation of infection is not easy, but in vitro culture of *R. felis*, and hence material for a serologic assay for the diagnosis of human *R. felis* infections, has recently been described, and serology appears to be an accurate indicator of exposure (9). As with other spotted fever group rickettsial infections, molecular diagnostics may provide a useful alternative approach to detecting and identifying *R. felis* in infected tissues. In culture, *R. felis* has been shown to be resistant to erythromycin (unlike other rickettsia), gentamicin, amoxicillin, and trimethoprim-sulfa-methoxazole. Thus, infection with this bacterium should be considered in cases of antibiotic-insensitive fever with a rash, especially in young, old, and immunosuppressed persons. The organism is sensitive to doxycycline, rifampicin, thiampenicol, and fluoroquinolones (10).

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**References**

1. Azad AF, Sacchi JB Jr, Nelson WM, Dasch GA, Schmidtman ET, Carl M. Genetic characterization and transovarial transmission of a novel typhus-like rickettsia found in cat fleas. Proc Natl Acad Sci U S A 1992;89:43–62.
2. Higgins JA, Radulovic S, Schriefer ME, Azad AF. *Rickettsia felis*: a new species of pathogenic rickettsia isolated from cat fleas. J Clin Microbiol 1996;34:671–4.
3. Bouyer DH, Stenos J, Croquet-Valdes P, Moron CG, Popov VL, Zavala-Velazquez JE, et al. *Rickettsia felis*: molecular characterization of a new member of the spotted fever group. Int J Syst Evol Microbiol 2001;51:339–47.
4. Oliveira RP, Galvao MA, Mafra CL, Chamone CB, Calic SB, Silva SU, et al. *Rickettsia felis* in *Ctenocephalides* spp. fleas, Brazil. Emerg Infect Dis 2002;8:317–9.
5. Marquez FJ, Munain MA, Perez JM, Panchon J. Presence of *Rickettsia felis* in the cat flea from southwestern Europe. Emerg Infect Dis 2002;8:89–91.
6. Schriefer ME, Sacchi JB Jr, Dumlen MG, Azad AF. Identification of a novel rickettsial infection in a patient diagnosed with murine typhus. J Clin Microbiol 1994;32:949–54.
7. Richter J, Fournier PE, Petridou J, Haussinger D, Raoul D. *Rickettsia felis* infection acquired in Europe and documented by polymerase chain reaction. Emerg Infect Dis 2002;8:207–8.
8. Zavala-Velazquez JE, Ruiz-Sosa JA, Sanchez-Elias RA, Becerra-Carmona G, Walker DH. *Rickettsia felis* rickettsiosis in Yucatan. Lancet 2000;356:1079–80.
9. La Scola B, Meconi S, Fenollar F, Rolain JM, Roux V, Raoul D. Emended description of *Rickettsia felis* (Bouyer et al. 2001), a temperature-dependent cultured bacterium. Int J Syst Evol Microbiol 2002;52:2035–41.
10. Rolain JM, Stuhl L, Maurin M, Raoul D. Evaluation of antibiotic susceptibilities of three rickettsial species including *Rickettsia felis* by a quantitative PCR DNA assay. Antimicrob Agents Chemother 2002;46:2747–51.

**Community Transmission of Extended-Spectrum β-Lactamase**

**To the Editor:** The spread of multiresistant gram-negative bacteria in the general population is a problem of paramount importance, but the responsible mechanisms are poorly understood. Several studies have focused on β-lactam resistance in *Enterobacteriaceae* isolated from stools in healthy people, but they did not specifically investigate the extended-spectrum β-lactamases (ESBL). Furthermore, none of these studies detected ESBL in the evaluated population (1,2). We performed three survey studies to determine the incidence of *Enterobacteriaceae* strains producing ESBLs in the stools of outpatients attending our hospital. The first study was performed during a 4-month period (February–May 2001), the second during a 3-month period (April–June 2002), and the third during 1 month (October 2002).

Stool samples were spread onto plates of MacConkey agar containing 2 mg/L of cefotaxime. A colony of each distinct morphotype was analyzed further. Species were identified according to conventional methods (3). The susceptibility to β-lactam antibiotics was determined by the disk-diffusion test, following recommendations of the National Committee for Clinical Laboratory Standards (4,5). The interpretative reading of the antibiotic disk was performed according to standard guidelines (4–6). The MICs of cefotaxime and ceftazidime, with and without clavulanic acid, were later determined by Etest (AB Biodisk, Solna, Sweden). Strains producing ESBL were defined as strains showing synergism between amoxicillin-clavulanic acid and cefotaxime, ceftazidime, cefepime, or aztreonam (4,5).

All strains suspected of carrying a resistance pattern compatible with
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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References
1. Britñas L, Zarazaga M, Saenz Y, Ruiz-Larrea F, Torres C. β-Lactamases in ampicillin-resistant Escherichia coli isolates from foods, humans, and healthy animals. Antimicrob Agents Chemother 2002;46:3156–63.
2. Österblad M, Hakanen A, Manninen R, Leistevuo T, Peltonen R, Meurman O, et al. A between-species comparison of antimicrobial resistance in enterobacteria in fecal flora. Antimicrob Agents Chemother 2000;44:1479–84.
3. Murray P, Baron E, Pfaffer M, Tenover F, Yokken R. Manual of clinical microbiology. 7th ed. Washington: American Society for Microbiology; 1999.
4. National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial disk susceptibility test. Document M2-A7. 7th ed. Wayne (PA): The Committee; 2000.
5. National Committee for Clinical Laboratory Standards. Supplemental tables: disk diffusion. Document M100-S10. Wayne (PA): The Committee; 2000.
6. Livermore DM. β-Lactamases in laboratory and clinical resistance. Clin Microbiol Rev 1995;8:557–84.
7. Sabaté M, Miró E, Navarro F, Vergés C, Aliaga R, Mirelis B, et al. Beta-lactamases involved in resistance to broad-spectrum cephalosporins in Escherichia coli and Klebsiella spp. clinical isolates collected between 1994 and 1996, in Barcelona (Spain). J Antimicrob Chemother 2002;49:989–97.
8. Hernández JR, Pascual A, Cantón R, Martínez-Martínez L, Grupo de Estudio de Infección Hospitalaria (GEIH). Escherichia coli y Klebsiella pneumoniae productores de betalactamasas de espectro extendido en hospitales españoles (Proyecto GEIH-BLEE 2000). Enferm Infecct Microbiol Clin 2003;21:77–82.
9. Chambers HF. The changing epidemiology of Staphylococcus aureus? Emerg Infect Dis 2001;8:178–82.
10. Garau J, Xercavins M, Rodriguez-Carballeira M, Gómez-Vera JR, Coll I, Vidal D, et al. Emergence and dissemination of quinolone-resistant Escherichia coli in the community. Antimicrob Agents Chemother 1999;43:2736–41.
11. Tomasz A. New faces of an old pathogen: emergence and spread of multidrug-resistant Streptococcus pneumoniae. Am J Med 1999;107:55S–62S.

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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,