

levels of susceptibility because the MIC₅₀ was only 1 dilution below the current breakpoint (7,8). Aztreonam resistance could not be transferred by conjugation between IMP-1-producing (aztreonam-resistant) *P. putida* and *P. aeruginosa* (2) and is not associated with a transposon carrying blaVIM-2 (6). No evidence of extended-spectrum β-lactamases was detected in our isolates by classic synergy assays with clavulanate plus aztreonam, ceftazidime, or cefotaxime. VIM-6-producing *P. putida* isolates from Singapore (9) were more resistant to aztreonam (MIC >128 μg/mL), ceftazidime, and cefepime (MIC >256 μg/mL).

Detection of bla_{VIM-2} in *Pseudomonas* in South America was initially reported by the SENTRY Antimicrobial Surveillance Program (10) and included 1 *P. fluorescens* isolate in Chile and 3 *P. aeruginosa* isolates in Venezuela. To the best of our knowledge, our report is the first of VIM-2 in *P. putida* in Latin America. VIM-2-producing *P. putida*, which were originally restricted to East Asia and only very recently found in France, may represent an emerging pathogen or function as reservoirs for resistance because of their widespread presence in the hospital environment.

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Multidrug-resistant *Acinetobacter baumannii*, Russia

To the Editor: During the past decade, nosocomial infections due to multidrug-resistant *Acinetobacter baumannii* have been described with increasing frequency, mostly in intensive care units (ICUs), resulting in therapeutic difficulties (1). The main mechanism for resistance to extended-spectrum cephalosporins in *A. baumannii* is attributed to the overexpression of chromosome-encoded cephalosporinases or to plasmid-encoded Ambler class A, B, and D β-lactamases (2). *A. baumannii* that produce PER-1 extended-spectrum β-lactamase (ESBL) are rarely isolated outside Turkey and remain susceptible to carbapenems (3). Here we describe what we believe is the first ESBL-producing *A. baumannii* isolate resistant to carbapenems and the first characterization of a PER-1 *A. baumannii* isolate from Russia, further supporting the emergence and dissemination of PER-1 *A. baumannii* strains in eastern Europe and outside Turkey (3,4).

On April 17, 2005, a 79-year-old man was hospitalized in the cardiology ward of a private hospital in Moscow, Russia, with cardiac arrhythmia and a pulmonary infarction subsequent to a pulmonary embolism. After 1 week, he was transferred to the ICU for multiple organ failure related to a nosocomial infection caused by an *A. baumannii* strain

susceptible to several antimicrobial drugs, including imipenem (with positive lung aspiration and blood cultures). He received imipenem and amikacin at high doses. On May 5, 2005, he was transferred to the internal medicine ward of the American Hospital of Paris, Neuilly-sur-Seine, France. On the day of admission, bacterial cultures taken from sputum showed a multidrug-resistant *A. baumannii* MOS-1 strain, susceptible only to colistin and rifampin. The patient received intravenous and aerosolized colistin 3 times a day plus rifampin at 1,200 mg/d so that he could return to Russia. Rapid identification of *A. baumannii* MOS-1, increased awareness as a result of a French national alert signaling the emergence of ESBL VEB-1-producing *A. baumannii* (5), and implementation of strict barrier precautions prevented dissemination of this strain. No other multidrug-resistant *A. baumannii* isolate with a similar resistance profile has been isolated in the hospital before, during, or after this period.

A. baumannii MOS-1 was susceptible to colistin and rifampin only, and no synergy image could be observed between clavulanic acid and cefepime or ceftazidime discs on a routine antibiogram performed by the disc diffusion method. Only the use of cloxacillin-containing Mueller-Hinton agar plates (200 µg/mL) to inhibit the activity of the naturally occurring cephalosporinase (AmpC) allowed detection of a synergy image, signature of the presence of an ESBL (5). MICs for imipenem, determined by the agar dilution method (6), were >64 µg/mL. Clavulanic acid addition (2 µg/mL) decreased ticarcillin MIC from >512 to 256 µg/mL and ceftazidime MIC from 512 to 128 µg/mL but did not affect MIC of imipenem.

Genes coding for ESBLs and for class B and D carbapenemases were sought by PCR as previously described (4,5). Primers used for

detection of TEM and PER β-lactamases gave 894-bp and 825-bp PCR products, respectively (4). Sequence analysis showed identity with *bla*_{TEM-1} and *bla*_{PER-1} genes (4). Results of isoelectric focusing showed 3 isoelectric point values (5.3 for PER-1, 5.4 for TEM-1, and >8.5 for AmpC) in *A. baumannii* MOS-1 (4). A crude β-lactamase extract of that isolate had no significant imipenem hydrolysis activity, which suggests that the carbapenem resistance may have emerged through a nonenzymatic mechanism such as mutations in porins (7). *Bla*_{PER-1} gene in *A. baumannii* MOS-1 was located on a composite transposon, Tn1213, identical to that characterized by Poirel et al. (8). Attempts to demonstrate plasmids

or transfer genes encoding TEM-1 or PER-1 failed (data not shown), which suggests that the genes were chromosomally encoded. *A. baumannii* MOS-1 was not clonally related to well-characterized PER-1 *A. baumannii* strains from Turkey, France, and Belgium (4,9) (Figure), further supporting genetic heterogeneity of PER-1 *A. baumannii* isolates, even though the immediate genetic environment of *bla*_{PER-1} gene was similar (8).

The emergence and spread of ESBL-producing *A. baumannii* strains are of concern because they will increase carbapenem use, thus raising the risk for emergence of carbapenem-resistant isolates. *A. baumannii* MOS-1 is, we believe, the first description of an ESBL-producing *A.*

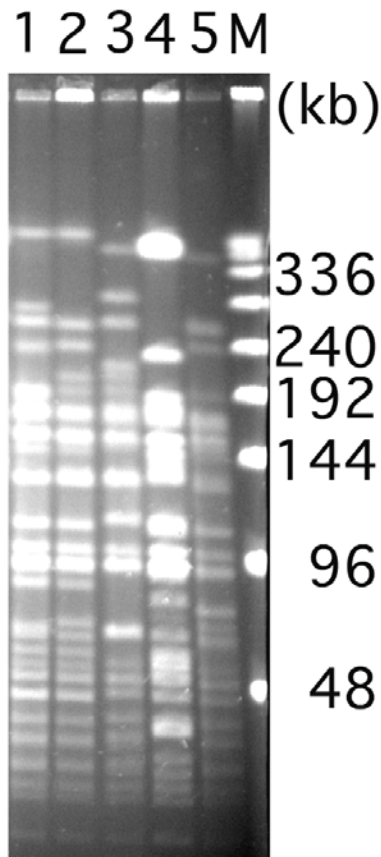


Figure. Pulsed-field gel electrophoresis of *Apal* restricted analysis of *Acinetobacter baumannii* isolates. Lane 1, *A. baumannii* AMA-1 from France (4); lanes 2 and 3, *A. baumannii* IST-1 and *A. baumannii* IST-2 from Turkey (4); lane 4, *A. baumannii* isolate from Belgium (4); lane 5, *A. baumannii* MOS-1 from Russia (current study); lane M, bacteriophage lambda molecular mass ladder. Numbers on the right side of the figure represent the sizes in kb.

baumannii isolate also being resistant to carbapenems. This resistance was likely acquired in vivo under imipenem treatment, but the susceptible strain was not available for strain comparison. This is also the first description of a PER-1 *A. baumannii* isolate from Russia, a country from which little epidemiologic data on antimicrobial drug resistance are available, except for the emergence of ESBLs of CTX-M type in *Enterobacteriaceae* (10).

This study highlights the importance of international patient transfer in the spread of antimicrobial drug resistance, thus emphasizing the need for hospitals to isolate and screen for multidrug-resistant pathogens in all patients admitted to hospitals from foreign countries. This is particularly critical when the foreign country is known for a high prevalence of multidrug-resistant bacteria or when no antimicrobial drug resistance data are available.

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Human Infection with *Rickettsia* sp. related to *R. japonica*, Thailand

To the Editor: Although rickettsioses caused by scrub typhus and typhus group rickettsiae are well recognized in Thailand, few spotted-fever group rickettsiae (SFGR), including *Rickettsia honei* TT118 and *R. felis*, have been documented to be associated with human illnesses (1,2). We report a case of human infection with an SFGR species closely related to *R. japonica* in Thailand.

In January 2005, a 36-year-old man with prolonged fever, pneumonia, and septic shock was transferred from a private hospital to Phramongkutklao Army Hospital in Bangkok. Two weeks before the onset of fever, the patient had camped at Khao Yai National Park, \approx 175 km northeast of Bangkok. The park is a popular location for tourists and the largest national park declared as a natural wildlife reserve area. The patient reported the presence of wild deer around the camping area but did not recall being bitten by an arthropod. Ten days before hospitalization, he developed flulike symptoms, fever, and sore throat. Six days later, he noted petechiae on his lower extremities, and his condition worsened. At the time of hospital admission, the patient had fever of 38.6°C, tachycardia, dyspnea, hypotension, nausea, vomiting, generalized maculopapular rash, and subconjunctival hemorrhage. Laboratory investigation showed thrombocytopenia (platelets 64,000/mm³), leukocytosis (14,000/mm³), and elevated levels of serum hepatic enzymes (aspartate aminotransferase 287 IU/L [reference 5–50 IU/L]; alanine aminotransferase 186 IU/L [reference 5–40 IU/L]). Chest radiograph showed interstitial pneumonitis. Serum antibody test results were negative for leptospira and