transmission via the balneotherapy room was the most likely route of this transmission. Wound infection by antibiotic-resistant organisms should be considered a potential risk, and their presence must be identified by microbiological surveillance, systematic screenings, and periodic cultures from various parts of the wound. Water sampling strategies could be planned on a regular basis in order to maintain healthcare workers' vigilance. Moreover, knowing microbial colonization, antimicrobial susceptibility, and trends in nosocomial infections in burn units can help healthcare workers choose the optimal empirical antibiotic treatment. Raising healthcare workers' knowledge about this subject is essential to control the risk of transmission in these departments, in association with strict infection control procedures in burn units.

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Carbapenemase-Producing Enterobacteriaceae (CPE) in the Pediatric Setting: Results from an 18-Month Survey

To the Editor—The emergence of carbapenemase-producing Enterobacteriaceae (CPE) in children is a serious matter of concern, because it severely limits treatment options. Rapid identification is crucial, both for appropriate antimicrobial therapy and for implementation of infection control measures.[1] Few epidemiological data on CPE infections in children are available.[2]

Our study aimed to evaluate the occurrence of CPE in an Italian tertiary pediatric center, the Regina Margherita Children's Hospital of Turin. Between May 1, 2012, and October 31, 2013, patients from whom CPE strains had been isolated from any clinical specimens were included and prospectively investigated. Only the first isolate from each patient was considered. Identification and antimicrobial susceptibility testing were performed using the Vitek-2 automated system (bioMérieux), and results were interpreted according to European Committee on Antimicrobial Susceptibility Testing breakpoints.[3] Colistin and tigecycline minimum inhibitory concentrations (MICs) were additionally determined by Etest (bioMérieux). Enterobacteriaceae isolates with imipenem and/or meropenem MICs of 2 mg/L or greater were tested for carbapenemase production using a combined disk assay (KPC/MBL and OXA-48 Confirm Kit, Rosco Diagnostica).[4-7]

During the study period, 15 patients (9 males) were identified as infected or colonized with CPE. The mean age (± standard deviation [SD]) was 10.6 ± 5.84 years. Eleven cases (73%) were found in the hemato-oncology unit, 2 (13.5%) were found in the intensive care unit, and 2 (13.5%) were found in the infectious diseases ward. One patient had been transferred from a pediatric hospital in Maracaibo, Venezuela, 2 months before; 1 patient had been admitted after a 3-month stay in the Italian hospital of Cairo, and 1 patient had just been discharged from an adult ward. Eleven patients had malignancies (10 had acute leukemia or lymphoma, and 1 had medulloblastoma); of these 11 patients, 8 had refractory or relapsed neoplasms, whereas 3 were not receiving therapy, and 2 had undergone allogeneic hematopoietic stem cell

transplantation (HSCT) within the previous 6 months. Among the remaining 4 patients, Rett syndrome, perinatal encephalopathy, recent heart transplantation, and congenital immunoglobulin G2 deficiency were identified as comorbidities. All patients had been treated with antibiotics effective against gram-negative bacteria for at least 20 days in the previous 3 months: 11 had received carbapenems, 7 had received quinolones, and 9 had received aminoglycosides.

CPE strains were isolated from urine specimens in 7 cases (47%), blood specimens in 4 (27%), urinary catheter tips in 2 (13%), and skin and nasal swab samples in 1 case each. Identified bacteria included *Klebsiella pneumoniae* ($n = 11$; 73%) and *Escherichia coli* ($n = 4$, 27%). Twelve isolates (80%) produced *K. pneumoniae* carbapenemases (KPCs), 2 (13%) produced oxacillinase (OXA)–48-like carbapenemases, and 1 (7%) produced metallo-β-lactamase. All strains showed resistance to amoxicillin-clavulanic acid, piperacillin-tazobactam, third- and fourth-generation cephalosporins, and trimethoprim-sulfamethoxazole. Eleven isolates were resistant (MIC > 8 mg/L), and 3 showed intermediate resistance (2 mg/L < MIC ≤ 8 mg/L) to imipenem, whereas the remaining 1 was susceptible to imipenem but resistant to meropenem (MIC > 8 mg/L). All isolates were sensitive to colistin (MIC ≤ 2 mg/L). Susceptibility rates to the other antibiotics tested were as follows: nitrofurantoin, 100% (4 of 4 isolates); fosfomycin, 67% (4 of 6); amikacin, 67% (10 of 15); gentamicin, 27% (4 of 15); ciprofloxacin, 20% (3 of 15); and tigecycline, 20% (1 of 5).

Six patients were considered to be infected with CPE. Four had bloodstream infection, with prescription of appropriate antibiotic regimen 3 days after the onset of bacteremia, when antimicrobial susceptibility testing results became available. Among the antibiotic combinations prescribed were colistin plus meropenem and tigecycline, ciprofloxacin plus meropenem, and colistin plus meropenem. Two patients died, with CPE infection being identified as a contributing factor. The remaining 2 were successfully treated with intravenous antimicrobial therapy for a mean (± SD) of 14 ± 5.66 days. The last 2 patients had symptomatic urinary tract infection: 1 patient was treated with amikacin for 7 days on the basis of antibiogram results; the second patient was treated empirically with ceftazidime and did not receive appropriate antibiotic therapy according to in vitro susceptibility testing because of clinical improvement. In both cases, complete recovery occurred within 5 days after the onset of infection, and control urine cultures were negative. None of the patients developed reinfection with CPE strains. Nine of the 15 patients included in our study were considered to be colonized with CPE. Four received appropriate antibiotic therapy on the basis of antibiogram results; however, 1 patient remained colonized for more than 1 month after the first isolate was obtained, in addition to 2 untreated subjects.

CPE infections represent a serious threat in the pediatric setting as well. In particular, bloodstream infections are associated with a high mortality rate in spite of appropriate antimicrobial treatment, as emerged from our survey. Immunosuppressed children, particularly with refractory or relapsed neoplasms or recent history of allogeneic HSCT, children recently hospitalized in adult wards or in countries where infections with CPE strains are endemic, and children with carbapenem exposure within the previous 3 months may be the optimal target pediatric population for CPE screening by rectal swab. Early recognition of CPE colonization is essential for timely implementation of control measures to reduce patient-to-patient transmission and infection-related morbidity and mortality.

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Endotoxin Overproduction of *Enterobacter cloacae* and Mortality Rate

To the Editor—We want to applaud the great work done by Arduino et al[1] in their article that established growth rates and endotoxin production in vitro in propofol using 10 clinically important microorganisms associated with outbreaks that have been implicated in extrinsic contamination of this intravenous anesthetic, as published by the Centers for Disease Control and Prevention in May and June 1990.[2] We would like to mention other studies that were reported after Arduino et al[1] to lend additional credence to their findings. According to the analysis by Arduino et al,[1] endotoxin was not detected in the gram-negative cultures at the start of the experiment, but after 24 hours, endotoxin production increased rapidly to a substantial level. *Enterobacter cloacae* was the best endotoxin producer of all of the microorganisms tested at all time points (2,412–4,820 ng/mL in 24 hours; 9,420–18,840 ng/mL in 48 hours; 7,360–14,720 ng/mL in 72 hours). Translating these results to clinical practice, 11 years later, Weist et al[3] reported outbreaks caused by multiple dose vials from 1983 to 2002, including 2 fatalities and 4 infected patients whose cases were associated with the administration of propofol contaminated by nothing more and nothing less than *E. cloacae*. Additionally, Mattner and Gastmeier[4] refer to *E. cloacae* and *Serratia marcescens* as the microbial species most commonly associated with death in the 7 reported outbreaks associated with propofol use.

We would again like to congratulate Arduino et al[1] for the practical knowledge generated by this study, which focused on specific strains that overproduce endotoxin, such as *E. cloacae*. Consequently, this species has been shown to be associated with a high mortality rate, as reported in several studies.[3,4]

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Different Compliance with Central Line Insertion Bundle between Intensivist and Nonintensivist Staff in Intensive Care Units

To the Editor—The use of central venous catheter (CVC) is increasing for monitoring hemodynamic status and providing venous access in the intensive care unit (ICU). However, as CVC use increases, complications of central line–associated bloodstream infection (CLABSI) after the insertion of CVCs increase as well and become another important cause of morbidity and mortality.[1,2] Therefore, several prevention efforts were developed to reduce the occurrence of CLABSI in the clinical setting of the ICU. "Insertion bundles" for reducing the risk of infection during the insertion of CVCs and "maintenance bundles' for minimizing the risk of infection for patients with CVCs are the 2 essential care bundles for prevention of CLABSI. CVC insertion is always performed by physicians in the ICU; however, ICU physicians may be intensivist or nonintensivist staff, and studies that compare CVC insertion bundle compliance of these 2 different types of physicians in the ICU are scarce. Therefore, this study was conducted to investigate the physician factors associated with CVC insertion compliance in the ICU.