The presence of cluster I in both ICUs, identical to the endemic cluster described by Saalffeld et al,[4] demonstrates the importance of interhospital dissemination. Although the environment was not the main cause of dissemination, we believe that the contamination of the hands of healthcare professionals may have contributed to the dissemination of *A. baumannii* isolates, and the failure to verify this dissemination route was a limiting factor in our study.

Our study showed that after the ICU was re-established in a new building (ie, a new ICU), the dissemination of endemic clone–producing OXA-23 was maintained even though the new ICU environment was not contaminated. This occurrence demonstrates that additional measures are required to control the dissemination of this important hospital pathogen.

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Extended-Spectrum Beta-Lactamase (ESBL)-Producing *Escherichia coli* versus *Klebsiella pneumoniae:* Does type of germ really matter?

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To the Editor—We read with great interest the recent article by Scheuerman et al[1] showing that extended-spectrum β-lactamase (ESBL)-producing *Escherichia coli* (ESBL-EC) and *Klebsiella pneumoniae* (ESBL-KP) bloodstream infections (BSIs) differ significantly in terms of mortality (33.7% vs 17.4%; $P = .016$). Because their study concerns a highly relevant and popular topic, some points should be discussed.

First, ESBL-KP–infected patients were more often hospitalized in ICU than those infected by ESBL-EC ($P < .001$), partly due to a septic shock, which may explain such a high rate of mortality (33.7%) for a bloodstream infection (BSI). Indeed, the observed mortality rate for ESBL-KP was similar to the average mortality for those with gram-negative BSIs in the ICU (35%) according to the prospective EUROBACT International cohort study.[2] Also, ICU-acquired BSIs are associated with a 40% increase in the risk of 30-day mortality.[3] Therefore, it is hard to believe that such a difference could be accounted for in any statistical adjustment, and thus, it constitutes a selection bias.

Second, the main source of BSI was urinary tract in the ESBL-EC arm ($P = .005$), while it is acknowledged that the severity of urinary tract infection is not related to the presence of bacteremia.[4] Such data underly the hypothesis that ESBL-KP infections might have been more severe than those due to ESBL-EC. For instance, multidrug-resistant BSIs complicating respiratory tract infections have been associated with an increased mortality (odds ratio [OR], 3.26; 95% confidence interval [CI], 1.29–8.22).[7]

Third, no information is provided about the respective antimicrobial regimens between ESBL-EC and ESBL-KP patients. However, it is currently argued that carbapenem alternatives are associated with a higher mortality rate than carbapanems for the treatment of ESBL BSI. In fact, the MERINO trial by Harris et al[5] was recently suspended due to an increase in mortality in the arm

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receiving piperacillin-tazobactam (12.3%) versus meropenem (3.7%).[5] Such data should have been discussed. Likewise, no information is provided on treatment duration or dose, which may have varied between the 2 groups in the present study.[1] Both factors play a role in the outcome of treatment, especially when used against multidrug-resistant organisms.[6]

Interestingly, we previously showed that BSI severity or mortality among spinal cord injury patients over 15 years was not related to the multidrug-resistant characteristics of the microorganism.[7] Although our sample size was small ($n < 30$), a closer look at the outcome between ESBL-EC ($n = 26$) and ESBL-KP ($n = 13$) did not reveal any statistical difference in terms of mortality rate (7.7% in each arm). Moreover, the mortality rates were similar for other ESBL microorganisms (*Enterobacter* spp, *Morganella* spp, and *Proteus* spp ($n = 21$)), ~9.5% ($P = .99$, data not shown).

In fact, we believe that the findings of Scheuerman et al, which showed no impact of CTX-M isolates in comparison to other ESBL genotypes, might support the idea that the type of germ does not play a major role. Indeed, mortality seems more related to patient comorbidities and severity of infection, as shown in Table 2 of the article,[1] with significant discrepancies between the 2 groups in terms of length of stay to bacteremia ($P = .017$), source of infection ($P = .005$), ICU ward admission ($P < .001$) and underlying cardiovascular disease ($P < .001$). Moreover, in a rabbit model of sepsis induced by a multidrug-resistant *Klebsiella pneumoniae*, Zhou et al[8] showed that mortality was higher for the rabbits infected by susceptible than those infected with multidrug-resistant strains.

Overall, the impact of ESBL-KP isolates on mortality rate might have been overestimated, in the light of the severity of the patient condition.

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Differences in mortality between infections due to extended-spectrum-beta-lactamase–producing *Klebsiella pneumoniae* and *Escherichia coli*

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To the Editor—We read with interest the recent article by Scheuerman et al,[1] in which they found that patients with extended-spectrum-β-lactamase (ESBL) producing *Klebsiella pneumoniae* infections had higher 30-day mortality than patients with ESBL producing *Escherichia coli* infections. We have recently published on mortality, readmissions, recurrences, and the benefit of infectious diseases consultation for patients with various multidrug resistant organism infections.[2,3] We included in our study patients with various ESBL producing *Enterobacteriaceae* infections, among them *K. pneumoniae* and *E. coli*. Given the recent findings of Scheuerman et al, we conducted a retrospective evaluation to determine the association between ESBL producing organism (*K. pneumoniae* or *E. coli*) and 30-day all-cause mortality at our institution.

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