few studies have addressed progression to infection among colonized patients with CPE, using the recovery from clinical sites as a marker for infection.[6–8] Interestingly, our results were similar to these studies, mainly in relation to the incidence rate of infections among colonized patients and the predominance of KPC-2-KP among the CPE isolates. On the other hand, in these studies, no data on polymyxin B resistance was reported.

In conclusion, despite a low incidence of infections by KPC-2-KP in previously colonized patients, a KPC-2-KP predominant clone presenting with a high polymyxin B resistance level has been responsible for most intensive care unit infections due to CPE isolates. Although our results should be validated by further studies, they serve as a warning to prevent the spread of polymyxin-resistant KPC-2-KP by the early detection of carriers, especially among critically ill patients.

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CPE Clearance—A Response to Kim et al.

To the Editor—We were interested to read the article by Kim et al[1] in a recent issue of this journal. Their research confirms previous studies, including our own, regarding carriage of carbapenemase-producing Enterobacteriaceae (CPE).[2] We studied patients who were released from the hospital after a CPE-positive culture. We followed up with rectal swab cultures taken retrospectively and prospectively for the study or as part of clinical follow-up. In the 97 patients with follow-up cultures, mean time to CPE negativity was 387 days. At 3 months, 78% of patients (64 of 82) had positive cultures; 65% (38 of 58) had positive cultures at 6 months, and 39% (12 of 30) had positive cultures at 1 year. Repeated hospitalization was associated with increased duration of carriage. A small minority of our patients had a positive culture after a negative one (unpublished data), and we considered these patients to be continual carriers. Similarly, Schechner et al[3] showed, in a study published in this journal, that 60% of patients (14 of 23) who had a positive follow-up screening test were screened within 3 months of the index positive culture. Thus, it is unsurprising that Kim et al found very high rates of continuing carriage during a single hospitalization.

It should be noted that both studies cited examined only KPC-type carbapenemases, which represent the major mechanism of carbapenem resistance in the United States and in our region (Israel).

Because of their findings of high rates of carriage and high rates of CRE-positive surveillance cultures after negative cultures, the authors suggest that more than 3 negative cultures are needed to consider a patient a noncarrier. However, according to their and our studies, duration of CRE carriage is expected to last throughout a given hospitalization and beyond. As such, and as is our institutional policy, given the

high pretest probability, we do not suggest retesting patients for CRE who have already tested positive during a given hospitalization. Rather, such patients are assumed to be positive throughout the duration of that hospitalization. The current study supports this policy.

Only in subsequent hospitalizations are patients retested and assumed to be negative following 3 negative cultures. This policy requires a better evidence base, but it is not in any way contradicted by the current study.

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