In this case, the IC precaution level was critical because MERS requires airborne and contact precautions, whereas influenza requires droplet and contact precautions. Clinically, she improved, her symptoms resolved, and she was discharged on hospital day 3 without a definite diagnosis.

Epidemiologically, given her recent travel to Dubai, her ILI could have been MERS because she became ill during the MERS incubation period (1.9–14.7 days).[1–3] Alternately, because this illness occurred during peak influenza season, she could have had influenza. The incorrect operating diagnostic premise was that if the patient tested positive for influenza, she could not have MERS. Unfortunately, this does not appear to be the case. Recently, 5 MERS cases were reported from Iran, and importantly, 2 of these also tested positive for influenza.[3,4] Clearly, in a returning traveller from an endemic MERS area with an ILI, a positive or negative nasal swab for influenza does not eliminate MERS as a diagnostic possibility. MERS-CoV PCR positivity is highest in distal airways, and nasopharyngeal or proximal respiratory tract specimens may be negative.[1] Optimal specimens for MERS-CoV testing are obtained from distal airway specimens via bronchoscopy, but bronchoscopy was not indicated in this patient.

In a returned traveler presenting with an ILI from a MERS-endemic area, without a definite diagnosis we concluded that it was "better to be safe than sorry" regarding IC precaution levels. HCWs are particularly predisposed to acquiring MERS in caring for hospitalized MERS patients. MERS precautions (airborne and contact) best protects HCWs from MERS-CoV bodily secretion exposures.[5–9] If she had MERS, influenza precautions (droplet and contact) would have been inadequate.

Recently, it has become apparent that some MERS cases initially have no fever or diarrhea and may have only mild ILI symptoms early. The risk of MERS transmission to HCWs from asymptomatic or mild cases that later become severe, is unknown. Even though she did not fit the classic MERS case definition, we believe that the nasopharyngeal swabs should have been tested for MERS-CoV.[10] Because the patient was in the IDCU, originally designed for Ebola care, HCWs viewed MERS as an Ebola-like infection. Few US hospitals have had MERS patients. We believe this near-MERS experience was an instructive exercise in IC precautions for HCWs caring for patients with potential imported viral zoonotic infections. [2]

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High Endemic Rates of OXA-23-Producing Carbapenem-Resistant *Acinetobacter baumannii* Isolates Caused by the Persistence of Major Clones in Hospitals in a Brazilian City 5 Years After an Outbreak

To the Editor—Acinetobacter baumannii is a major pathogen related to several nosocomial infections, particularly ventilator-associated pneumonia. The worldwide emergence

TABLE 1. MICs of the 4 *Acinetobacter baumannii* Isolates Resistant to Polymyxin B

		MICs (mg/L)								
Clone	Hospital	POB	IMP	MEM	CAZ	FEP	SAM	CIP	AMK	TIG
B	1	≥64	16	16	≥256	≥512	32	≥64	64	2
B	2	≥64	64	16	128	32	64	64	2	1
B	3	8	64	32	64	32	32	32	16	2
E	3	≥64	64	64	≥256	≥512	32	≥64	512	0.5

NOTE. AMK, amikacin; CAZ, ceftazidime; CIP, ciprofloxacin; FEP, cefepime; IMP, imipenem; MEM, meropenem; MIC, minimum inhibitory concentration; POB, polymyxin B; SAM, ampicillin-sulbactam; TIG, tigecyclin.

of carbapenem-resistant *A. baumannii* isolates (CRAB) constitutes a real threat owing to the few available therapeutic options.[1] This resistance is most commonly mediated by carbapenemases, notably OXA-type carbapenemases but also metallo-β-lactamases.[1]

In 2007, several hospitals in Porto Alegre, the capital of the southernmost Brazilian state, presented CRAB outbreaks,[2,3] as did other Brazilian cities.[4] After these first outbreaks, most Brazilian institutions remained with endemic rates of CRAB, including most hospitals of Porto Alegre.[3] In this study, we evaluated the molecular epidemiology of CRAB in 3 tertiary care hospitals in this city, 5 years after the outbreak, in order to assess whether clonal dissemination was, at least, one of the factors responsible for maintenance of high endemic rates of these isolates.

All *Acinetobacter* spp. isolates recovered from patients admitted at 3 tertiary care hospitals of Porto Alegre from March 1 through December 31, 2011, were included in the study.

Bacterial identification was performed by the Vitek 2 system (bioMérieux). The presence of $bla_{\text{OXA-51-like}}$, $bla_{\text{OXA-23-like}}$, $bla_{\text{OXA-24-like}}$, $bla_{\text{OXA-58-like}}$, and $bla_{\text{OXA-143}}$ was evaluated by polymerase chain reaction assay using primers and a multiplex assay.[5] Minimum inhibitory concentrations (MICs) for cefepime, ciprofloxacin, ceftazidime, ampicillin/sulbactam, amikacin, polymyxin B, tigecycline, imipenem, and meropenem were determined by broth microdilution in CRAB isolates and interpreted according to Clinical and Laboratory Standards Institute guidelines[6]; an MIC less than or equal to 2 mg/L was considered susceptibility to tigecycline. *Pseudomonas aeruginosa* ATCC 27853 was included as quality control in all tests. CRAB isolates were submitted to molecular typing by *Apa*I DNA macrorestriction followed by pulsed-field gel electrophoresis.[7] Results were interpreted by means of a dendrogram constructed using the band-based Dice coefficient method and, for the purposes of this study, isolates with at least 85% match were considered clones.

A total of 122 *Acinetobacter* spp. isolates were evaluated during the study period. Of these, 115 (94.3%) were identified as *A. baumannii,* owing to the presence of $bla_{\text{OXA-51-like}}$ gene. Eighty-four *A. baumannii* isolates (73.0%) were resistant to both carbapenems tested. They were recovered from respiratory secretions (59.5%), urine (15.4%), blood (11.9%), and other sites (14.1%). Among the 84 CRAB isolates, the MIC_{50} and MIC_{90} of both imipenem and meropenem were 32 and 64 mg/L, respectively (range, 16 to 256 mg/L). The MIC_{50} and MIC_{90} of polymyxin B were 0.25 and 0.5 mg/L, respectively (range, ≤0.125 to >64 mg/L); and 1 and 2 mg/L for tigecycline, respectively (range, ≤0.03 to 4 mg/L). Forty-eight isolates (41.7%) of the 115 isolates were resistant to all antimicrobials, except to polymyxin B and tigecycline. These extensively drug-resistant isolates were present in the 3 hospitals. Resistance to polymyxin B was identified in 4 (4.8%) of the 84 CRAB isolates (Table 1).

The presence of $bla_{\text{OXA-23-like}}$ gene was detected in all CRAB isolates and no other gene was detected in any isolate. Molecular typing revealed that the isolates were clustered into 7 clones (A to G). Clones A (33.7%), B (31.2%), and C (15.0%) represented 80% of all isolates. The remaining 20% belonged to the other 4 clones at similar proportions. A, B, and D were found at the 3 hospitals evaluated. Polymyxin B–resistant strains belonged to 2 different pulsed-field gel electrophoresis clones (Table 1).

CRAB outbreaks have been observed since the early 1990s.[8] However, in Brazil the first CRAB outbreak was reported only in 2003.[2] Later on, in 2007, a wide CRAB outbreak occurred in our city, Porto Alegre. Since then, endemic rates of CRAB have been observed. Our study showed a high prevalence of OXA--23–mediated carbapenem resistance among *A. baumannii* isolates 5 years after the outbreak. Notably, when compared with the isolates investigated during the outbreak in 2007,[2,3] it was observed that the same clones remained in the city hospitals (data not shown). This fact indicates that horizontal transmission has a major role in the maintenance of these high rates of carbapenem resistance among our institutions. It also suggests that specific clones may be better adapted to the nosocomial environment, resulting in a "homogeneous" persistence of specific CRAB strains. All CRAB isolates were positive for $bla_{\text{OXA-23-like}}$ gene and negative for all the other OXA-encoding genes investigated, including that encoding OXA-143, which is highly frequent in the southeastern region of the country.[9]

The prevalence of polymyxin B resistance among CRAB strains may still be considered low, even though these few isolates presented resistance to virtually all other classes with the few exceptions of amikacin for 1 isolate and tigecycline for all.

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Our study was limited by the lack of multilocus sequence typing analysis, which would contribute to the knowledge of the molecular epidemiology of CRAB isolates. Although many distinct sequence types of CRAB, including some international clones, have been identified in Brazil,[10,11] there still are no data from this Brazilian region.

In summary, we demonstrated the persistence of a few clones responsible for endemic levels of CRAB isolates in hospitals in a Brazilian city. Notably, 3 of 7 clones remained as the major strains at least 5 years after an initial outbreak in this city. These findings challenged the effectiveness of infection control measures to control the dissemination of CRAB after an initial large outbreak.

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Primary and Secondary Literature Should Be Distinguished When Searching for Data Used in Systematic Reviews of Nosocomial Outbreaks

To the Editor—In a recently published letter the editor,[1] Zorrilla-Vaca and Vaca-Gonzalez questioned the methodology and the results of our systematic review on nosocomial outbreaks due to contaminated drugs, especially on outbreaks due to contaminated propofol.[2] In their opinion, important articles had not been included in our review because of a poor search strategy and/or insufficient bibliographic sources, resulting in an incorrect mortality rate. Herewith, we would like to respond to their questions and remarks.

The main concern of Zorrilla-Vaca and Vaca-Gonzalez addresses our omission of an article by Bennett et al[3] in 1995, which summarizes 7 nosocomial outbreaks that could be traced to contaminated propofol. Although we were well aware of this publication at the time of our review, we decided not to include it because all of these outbreaks had previously been published by the Centers for Disease Control and Prevention (CDC)[4] and this primary publication had already been included in our work, cited as reference 113. Thus, including the