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Risk Factors for Acquisition of Imipenem-Resistant *Acinetobacter baumannii*: a Case-Control Study

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Risk factors for the nosocomial occurrence of imipenem-resistant *Acinetobacter baumannii* (IRAB) were determined. A case-control study design was used for a comparison of two groups of *A. baumannii*-positive patients with control patients. Nosocomial IRAB was isolated from the first group of *A. baumannii*-positive patients, and imipenem-susceptible *A. baumannii* (ISAB) was isolated from the second group. The control patients were randomly selected in a 4:1 ratio from the same medical or surgical services from which the *A. baumannii*-positive patients were receiving care when the isolation of IRAB occurred. Risk factors analyzed included demographic variables, comorbid conditions, variables related to hospitalization, and the antimicrobials used. IRAB was isolated from 104 patients, and ISAB was isolated from 387 patients between January and December 2000. The risk factors for IRAB were a previous intensive care unit (ICU) stay (odds ratio [OR], 21.54; 95% confidence interval [CI], 10.73 to 43.23) and prior exposure to imipenem (OR, 9.18; 95% CI, 3.99 to 21.13) or third-generation cephalosporins (OR, 2.11; 95% CI, 1.13 to 3.95). Risk factors for ISAB were a previous ICU stay (OR, 8.05; 95% CI, 5.67 to 11.44) and exposure to third-generation cephalosporins (OR, 2.07; 95% CI, 1.47 to 2.91). Our results suggest that the nosocomial occurrence of IRAB or ISAB is strongly related to an ICU stay, and IRAB occurrence may be favored by the selection pressure of imipenem.

Acinetobacter baumannii strains are becoming increasingly important nosocomial pathogens (3), especially in intensive care units (ICUs), where outbreaks due to this microorganism have been reported (2, 5, 11). A particular concern has been the frequent multiple antimicrobial resistances exhibited (12, 13, 14, 16). Imipenem and meropenem have retained in vitro activities that are superior to those of other antimicrobials, and in many centers, they are the drug of choice for patients with infections caused by *A. baumannii* (3). Unfortunately, recent analyses of hospital outbreaks have documented the spread of imipenem-resistant strains (6, 8, 17). This emergence of imipenem-resistant *A. baumannii* (IRAB) has become a worldwide problem and a troublesome development that threatens the continued successful treatment of *Acinetobacter* species infections (1).

In our institution, the first case of IRAB bacteremia was detected in August 1997. There was only one case of IRAB bacteremia each year between 1997 and 1998; however, the number of cases increased to five in 1999 and four in 2000. Four cases were in medical wards, three were in the medical ICU, one was in a surgical ward, and three were in surgical ICUs. Since November 2000, we have performed surveillance cultures at ICU admission and isolated patients with multidrug-resistant *A. baumannii*. As cases of IRAB infection or colonization increased, we reinforced a combination of control measures, such as equipment decontamination, strict attention to hand washing, and isolation procedures. In addition, since March 2001, the use of imipenem has been restricted by requiring approval from an infectious disease specialist.

The aim of this study was to identify risk factors for the nosocomial occurrence of IRAB and to contrast those factors with those for imipenem-susceptible *A. baumannii* (ISAB). Potential risk factors of particular interest were a previous stay in an ICU and antimicrobial drug exposures. The rationale for the study was that previous studies with the same aim had involved smaller numbers of *A. baumannii* culture-positive patients, thereby limiting the ability to detect differences (6, 8). More importantly, for the present study, control group selection was refined according to improvements in epidemiologic methodology, and therefore this study was able to assess more accurately and without bias the effect of antibiotic usage on the risk of IRAB infection (9, 10, 15).

MATERIALS AND METHODS

Case definition, control definition, and study design. The microbiology laboratory database was electronically searched to identify all the clinical cultures positive for *A. baumannii* from patients admitted between January and December 2000 at Asan Medical Center in Seoul, Korea, a 2,200-bed tertiary-care teaching hospital with 159 ICU beds. A case-control study design was used, with two retrospective case-control studies conducted concurrently. Patients from whom *A. baumannii* isolates had been recovered within 48 h of admission were excluded. Isolates obtained from surveillance cultures were also excluded. Identification and susceptibility testing for *A. baumannii* were performed by using the Vitek (bioMérieux-Vitek, Hazelwood, Mo.) or the MicroScan system (Dade Behring, West Sacramento, Calif.). Resistance to imipenem was defined as an MIC of imipenem exceeding 8 μg/ml. This included moderately resistant isolates.

The first group of cases included those of patients from whom IRAB had been isolated in clinical cultures, and the second group included those from whom ISAB had been isolated. Only one isolate from each patient was included (only the first *A. baumannii*-positive specimen). Control group 1 patients were selected from the same medical or surgical services to which the *A. baumannii*-positive

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patients had belonged when IRAB was isolated. The control patients were admitted during the same time period as the *A. baumannii*-positive patients. Patients who were admitted for less than 48 h or from whom IRAB had been isolated during their hospital stay were excluded from the control group. For each *A. baumannii*-positive patient with IRAB, four control patients were randomly chosen by an independent observer on the sole basis of exclusion criteria, other variables being unknown. Control group 2 was identical to control group 1 except that it excluded patients with ISAB.

Risk factors analyzed. Data were collected from administrative, pharmaceutical, and laboratory computerized databases by means of a relational database management system (Business Objects SA; Levallois-Perret, France). The relational database is maintained by the Medical Information Team of Asan Medical Center. We reviewed and validated 10% of the data from *A. baumannii*-positive patients and 5% from control patients by examining medical charts. The positive and negative predictive values of the data were greater than 99%.

Variables explored as possible risk factors included age; gender; the presence of underlying diseases or comorbid conditions; the Charlson score (the last two obtained by using the codes of the ninth revision of the International Classification of Diseases) (7); an ICU stay(s) prior to the outcome of interest; surgery prior to the outcome of interest; number of admissions to the hospital in the prior year; and length of hospital stay prior to the outcome of interest, which we referred to as the time at risk (length of stay prior to *A. baumannii* isolation for *A. baumannii*-positive patients and the complete length of hospital stay for the controls). Prior exposures to antimicrobial drugs were also explored. Prior antibiotic exposures were defined as at least 24 h of therapy during the 14 days prior to isolation of the organism for the *A. baumannii*-positive patients and prior to discharge for the controls. The rationale behind the choice of 14 days was to avoid analyzing antibiotics that the patients had received during the initial phase of a lengthy hospitalization.

Statistical analysis. All statistical analyses were performed using SPSS version 10.0 (SPSS, Chicago, Ill.). Bivariate analyses of categorical variables were done by the chi-square test or Fisher's exact test. Continuous variables were compared by using Student's *t* test or the Mann-Whitney U test. Odds ratios (ORs) and 95% confidence intervals (CIs) were also calculated.

Variables with a P value of < 0.1 in the bivariate analyses were included in a logistic regression model for multivariate analysis. A forward selection process was used. Risk factors were checked for confounding and colinearity. Confounders were included in the multivariable models if covariate inclusion changed the coefficient of any statistically significant variable in the logistic regression model by 10% or greater. Colinearity was verified by Spearman correlation among covariates and by viewing changes in standard errors of multivariate models. All tests were two-tailed, and a P value of < 0.05 was considered significant.

RESULTS

During the study period, 104 patients with IRAB (case group 1) and 387 patients with ISAB (case group 2) were identified. A total of 416 control patients were included in control group 1. ISAB was isolated from 5 of these control patients during their hospital stay, and therefore 411 patients were included in control group 2.

IRAB was most frequently isolated from respiratory secretions (57%). Other sites of isolation included wound cultures (19%), blood (4%), urine (3%), and bile (3%). ISAB was also most frequently isolated from respiratory secretions (64%). Other sites of isolation included urine (10%), wound cultures (9%), bile (7%), and blood (2%). The medical or surgical services from which patients with IRAB and ISAB were receiving care on the date that a positive culture result was obtained included medical wards (34 and 41%, respectively), surgical wards (49 and 45%, respectively), solid organ transplantation services (13 and 12%, respectively), and leukemia and bone marrow transplantation services (4 and 2%, respectively).

Results of the bivariate analysis of risk factors for IRAB are outlined in Table 1, and those for ISAB are outlined in Table 2. The results of multivariable analyses of risk factors for both IRAB and ISAB are delineated in Table 3.

The multivariable logistic-regression analysis demonstrated that the time at risk, defined as the time from admission to positive-culture date for *A. baumannii*-positive patients, was a significant risk factor for the isolation of IRAB (OR, 1.02; 95% CI, 1.002 to 1.03). Age was also identified as a risk factor (OR, 1.03; 95% CI, 1.01 to 1.05). An ICU stay prior to the event of interest was a significant risk factor (OR, 21.54; 95% CI, 10.73 to 43.23). Patients with nosocomial occurrence of IRAB were more likely to have been exposed to the antibiotics imipenem (OR, 9.18; 95% CI, 3.99 to 21.13) or third-generation cephalosporins (OR, 2.11; 95% CI, 1.13 to 3.95) in the 14 days prior to the date of a positive culture.

The multivariate analysis for ISAB demonstrated that the time at risk was a significant risk factor (OR, 1.02; 95% CI, 1.01 to 1.03). Age (OR, 1.02; 95% CI, 1.01 to 1.03) and male gender (OR, 1.47; 95% CI, 1.03 to 2.09) were also identified as risk factors. Having had an ICU stay prior to the event of interest was a significant risk factor (OR, 8.05; 95% CI, 5.67 to 11.44). Patients with nosocomial occurrence of ISAB were more likely to have been exposed to third-generation cephalosporins (OR, 2.07; 95% CI, 1.47 to 2.91) in the 14 days prior to the date of a positive culture.

Prior exposures to vancomycin or quinolones were highly associated with the occurrence of IRAB (Table 1), and prior use of vancomycin was also associated with the occurrence of ISAB (Table 2) in bivariate analyses. However, exposure to these drugs was not significant in multivariate analyses.

DISCUSSION

The spread of antibiotic resistance is a major threat to public health. A popular method of examining the risk factors for the acquisition of antibiotic-resistant microorganisms is the case-control study. Many case-control studies have now been published; however, these have often produced conflicting results due to the lack of methodological standards. In recent publications, methodological issues specific to risk factor analyses when using the case-control study design have been described (9, 15).

The appropriateness of control selection is the primary challenge in the design of case-control studies. Control patients should be randomly selected from the same population source; for example, they should be selected among all hospitalized patients, not only patients in whom the antibiotic-susceptible organism of interest has been isolated. The selection bias introduced by using control patients with susceptible microorganisms is likely to have the strongest impact on estimating the effect of exposure to antibiotics that are active against the susceptible microorganisms (9). Indeed, the OR for imipenem exposure as a risk factor for IRAB is 6.34 when randomly selected controls are used, whereas it is 27.12 when patients with ISAB are used as controls (10). The second important point for nosocomial case-control studies that analyze antibiotic resistance is the length of hospital stay. Controlling for length of hospital stay may be accomplished either by including it as a variable in a logistic regression model or by matching during the process of control patient selection (10). In both methods, the period of time at risk should not include the time

TABLE 1. Bivariate risk factors for the occurrence of imipenem-resistant *A. baumannii*

Variable	Study group[a]		*P* value	OR (95% CI)
	Control (n = 416)	*A. baumannii*-positive (n = 104)		
Demographics				
Mean age (yr)	52.3	58.5	<0.0001	1.03 (1.01–1.05)
Male gender	250 (60.1)	72 (69.2)	0.11	1.49 (0.94–2.37)
Comorbidity				
Cardiac disease	13 (3.1)	6 (5.8)	0.24	1.90 (0.70–5.12)
Diabetes	44 (10.6)	17 (16.3)	0.14	1.65 (0.90–3.03)
Malignancy	128 (30.8)	29 (27.9)	0.65	0.87 (0.54–1.40)
Cerebrovascular accident	20 (4.8)	10 (9.6)	0.10	2.11 (0.95–4.65)
Pulmonary disease	37 (8.9)	9 (8.7)	1.00	0.97 (0.45–2.08)
Hepatic disease	36 (8.7)	11 (10.6)	0.67	1.25 (0.61–2.55)
Renal disease	20 (4.8)	6 (5.8)	0.88	1.21 (0.47–3.10)
Charlson comorbidity scale (mean)	1.25	1.45	0.18	1.11 (0.96–1.28)
Related to hospitalization				
Time at risk (days)[b]	15.0	32.3	<0.0001	1.04 (1.03–1.06)
ICU stay	67 (16.1)	91 (87.5)	<0.0001	36.46 (19.28–68.96)
Surgery	120 (28.8)	46 (44.2)	0.004	1.96 (1.26–3.04)
No. of admissions past year	0.67	0.69	0.89	1.01 (0.87–1.18)
Antibiotics				
Imipenem	14 (3.4)	47 (45.2)	<0.0001	23.68 (12.26–45.72)
Piperacillin	4 (1.0)	1 (1.0)	1.00	1.00 (0.11–9.04)
Ampicillin-sulbactam	59 (14.2)	10 (9.6)	0.29	0.64 (0.32–1.31)
Vancomycin	19 (4.6)	32 (30.8)	<0.0001	9.29 (4.99–17.28)
Cephalosporins				
First generation	55 (13.2)	11 (10.6)	0.58	0.78 (0.39–1.54)
Second generation	32 (7.7)	4 (3.8)	0.24	0.48 (0.17–1.39)
Third generation	111 (26.7)	52 (50.0)	<0.0001	2.75 (1.77–4.27)
Aminoglycosides	55 (13.2)	22 (21.2)	0.06	1.76 (1.02–3.05)
Quinolones	59 (14.2)	31 (29.8)	<0.0001	2.57 (1.56–4.25)

[a] The data are the numbers (%) of patients unless otherwise indicated.
[b] For *A. baumannii*-positive patients, time at risk prior to isolation of *A. baumannii* on clinical culture, and for controls, complete length of hospital stay.

after which the outcome of interest has occurred (9). The third important point is the adjustment for comorbid illnesses (9).

In our study, the significant risk factors for ISAB were a previous ICU stay (OR, 8.05; 95% CI, 5.67 to 11.44) and prior exposure to third-generation cephalosporins (OR, 2.07; 95% CI, 1.47 to 2.91). The identification of an ICU stay as a strong risk factor is not unexpected. A longer stay in a high-risk unit has been identified as a risk factor in previous studies (12, 14). Prior use of broad-spectrum penicillins (12) or fluoroquinolones (13) was a significant risk factor for ISAB in other studies. However, the results of these studies were not comparable because the time at risk was not appropriately considered in these studies (12, 13).

A few analyses of risk factors for the acquisition of multidrug-resistant *A. baumannii* and ISAB have been published (12, 13, 14). In these studies, the selections of control patients were appropriate. The controls were all or randomly selected patients in the same unit at the same time as the infection sources. However, the studies in which *A. baumannii*-positive and control patients were selected based on the results of clinical cultures (12, 14) may be subject to bias, since cases of colonized patients without clinical manifestation would not be included (15). Particularly in studies that were limited to certain units, colonized patients might have been misenrolled as controls because of patient-to-patient cross-transmission (12). To overcome this problem, prospective and periodic surveillance for *A. baumannii* may be required (13).

There have been few studies analyzing risk factors for the acquisition of IRAB. In our study, the significant risk factors for IRAB were a previous ICU stay (OR, 21.54; 95% CI, 10.73 to 43.23) and prior exposure to third-generation cephalosporins (OR, 2.11; 95% CI, 1.13 to 3.95) or imipenem (OR, 9.18; 95% CI, 3.99 to 21.13). The identification of an ICU stay as a strong risk factor is not unexpected. A previous study using the Therapeutic Intervention Scoring system suggested that the high workload in a high-risk unit (surgical ICU) contributed to IRAB acquisition (8). In the study of a nested case-control study within the cohort design, case selection was appropriate for the prospective and periodic surveillance for IRAB done in ICUs; however, the controls were patients with ISAB (6). The OR for carbapenem as a risk factor for IRAB in this study was 4.58, and this risk might be overestimated because of a control selection bias (6).

Possible risk factors for nosocomial acquisition of antibiotic-resistant microorganisms are not only prior exposures to antibiotics but also variables related to hospitalization. Controlling for these variables, such as length of hospital stay and ICU stay, may be accomplished either by including them as variables in a multivariate analysis or by matching during the process of control patient selection (10). We did not choose controls by matching for length of hospital stay and ICU stay to delineate the relationship to the acquisition but included them in the final multivariate analyses. Other possible risk factors are variables related to invasive devices or procedures. In our study, prior exposure to a mechanical ventilator might be important, because IRAB and ISAB were most frequently iso-

TABLE 2. Bivariate risk factors for the occurrence of imipenem-susceptible *A. baumannii*

Variable	Study group[a]		*P* value	OR (95% CI)
	Control (n = 411)	*A. baumannii*-positive (n = 387)		
Demographics				
Mean age (yr)	52.3	56.6	<0.0001	1.02 (1.01–1.03)
Male gender	246 (59.9)	275 (71.1)	0.001	1.65 (1.23–2.21)
Comorbidy				
Cardiac disease	13 (3.2)	21 (5.4)	0.16	1.76 (0.87–3.56)
Diabetes	44 (10.7)	45 (11.6)	0.76	1.10 (0.71–1.71)
Malignancy	127 (30.9)	111 (28.7)	0.54	0.90 (0.66–1.22)
Cerebrovascular accident	19 (4.6)	55 (14.2)	<0.0001	3.42 (1.99–5.88)
Pulmonary disease	35 (8.5)	28 (7.2)	0.59	0.84 (0.50–1.41)
Hepatic disease	35 (8.5)	39 (10.1)	0.52	1.20 (0.75–1.94)
Renal disease	20 (4.9)	14 (3.6)	0.49	0.73 (0.37–1.47)
Charlson comorbidity scale (mean)	1.25	1.33	0.39	1.05 (0.94–1.16)
Related to hospitalization				
Time at risk (days)[b]	14.4	26.8	<0.0001	1.03 (1.02–1.04)
ICU stay	64 (15.6)	251 (64.9)	<0.0001	10.01 (7.13–14.04)
Surgery	117 (28.5)	154 (39.8)	0.001	1.66 (1.24–2.23)
No. of admissions past year	0.67	0.61	0.55	0.97 (0.87–1.08)
Antibiotics				
Imipenem	13 (3.2)	24 (6.2)	0.06	2.02 (1.02–4.03)
Piperacillin	4 (1.0)	2 (0.5)	0.69	0.53 (0.10–2.90)
Ampicillin-sulbactam	59 (14.4)	51 (13.2)	0.70	0.91 (0.61–1.36)
Vancomycin	17 (4.1)	74 (19.1)	<0.0001	5.48 (3.17–9.47)
Cephalosporins				
First generation	54 (13.1)	64 (16.5)	0.21	1.31 (0.89–1.94)
Second generation	32 (7.8)	37 (9.6)	0.44	1.25 (0.76–2.05)
Third generation	110 (26.8)	186 (48.1)	<0.0001	2.53 (1.88–3.40)
Aminoglycosides	54 (13.1)	75 (19.4)	0.02	1.59 (1.09–2.33)
Quinolones	56 (13.6)	58 (15.0)	0.65	1.12 (0.75–1.66)

[a] The data are the numbers (%) of patients unless otherwise indicated.
[b] For *A. baumannii*-positive patients, time at risk prior to isolation of *A. baumannii* on clinical culture, and for controls, complete length of hospital stay.

lated from respiratory secretions (57 and 64%, respectively). However, we could not explore the use of mechanical ventilators as a risk factor because the computerized databases of our institution did not provide data on their use. We considered that exposure to mechanical ventilators could probably be related to an ICU stay.

Despite many intensive efforts, the nosocomial acquisition of *A. baumannii* remains problematic, especially in ICUs. Molecular epidemiologic studies have revealed the presence of heterogeneous strains in a given hospital or service where they have become both endemic and epidemic (16, 18). Although the relative importance of infection endemic to a hospital or service and epidemic infection is difficult to assess, *A. baumannii* now accounts for a substantial proportion of infections endemic to hospitals. *A. baumannii* is capable of rapidly adapting to the hospital environment, and outbreaks may result from intrinsic contamination of the medical equipment or devices used with patients for monitoring or therapy and/or from contamination of the environment (3). Contamination may also result via the airborne route as well as from contact with patients (3, 4). These factors together explain the difficulty of controlling *A. baumannii* infections endemic to and epidemic in hospitals.

In our study, not all colonized patients may have been included in the group of *A. baumannii*-positive patients because the control patients were not screened by active surveillance culture for the presence of *A. baumannii*. In addition, we were unable to assess the role of patient-to-patient transmission. Despite these limitations, our study differs from previous analyses of risk factors for IRAB in that a larger number of cases led to increased accuracy, and the control group selection process was more refined.

In conclusion, our results suggest that the nosocomial occurrence of IRAB or ISAB is strongly related to an ICU stay, and IRAB occurrence may be favored by the selection pressure of imipenem.

TABLE 3. Multivariable analysis of risk factors for the occurrence of *A. baumannii*[a]

Imipenem-resistant *A. baumannii*		Imipenem-susceptible *A. baumannii*	
Variable	OR (95% CI)	Variable	OR (95% CI)
Age	1.03 (1.01–1.05)	Age	1.02 (1.01–1.03)
		Male gender	1.47 (1.03–2.09)
Time at risk	1.02 (1.002–1.03)	Time at risk	1.02 (1.01–1.03)
ICU stay	21.54 (10.73–43.23)	ICU stay	8.05 (5.67–11.44)
Imipenem	9.18 (3.99–21.13)		
Ceph 3[b]	2.11 (1.13–3.95)	Ceph 3[b]	2.07 (1.47–2.91)

[a] Only statistically significant risk factors are shown in this table.
[b] Ceph 3, third-generation cephalosporin.

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REFERENCES

1. **Afzal-Shah, M., and D. M. Livermore.** 1998. Worldwide emergence of carbapenem-resistant *Acinetobacter* spp. J. Antimicrob. Chemother. **41:**576–577.
2. **Beck-Sagué, C. M., W. R. Jarvis, J. H. Brook, D. H. Culver, A. Potts, E. Gay, B. W. Shotts, B. Hill, R. L. Anderson, and M. P. Weinstein.** 1990. Epidemic bacteremia due to *Acinetobacter baumannii* in five intensive care units. Am. J. Epidemiol. **132:**723–733.
3. **Bergogne-Bérézin, E., and K. J. Towner.** 1996. *Acinetobacter* spp. as nosocomial pathogens: microbiological, clinical, and epidemiological features. Clin. Microbiol. Rev. **9:**148–165.
4. **Brooks, S. E., M. A. Walczak, and R. Hameed.** 2000. Are we doing enough to contain *Acinetobacter* infections? Infect. Control Hosp. Epidemiol. **21:**304.
5. **Contant, J., E. Kemeny, C. Oxley, E. Perry, and G. Garber.** 1990. Investigation of an outbreak of *Acinetobacter calcoaceticus* var. *anitratus* infections in an adult intensive care unit. Am. J. Infect. Control **18:**288–291.
6. **Corbella, X., A. Montero, M. Pujol, M. A. Domínguez, J. Ayats, M. J. Argerich, F. Garrigosa, J. Ariza, and F. Gudiol.** 2000. Emergence and rapid spread of carbapenem resistance during a large and sustained hospital outbreak of multiresistant *Acinetobacter baumannii*. J. Clin. Microbiol. **38:**4086–4095.
7. **Deyo, R. A., D. C. Cherkin, and M. A. Ciol.** 1992. Adapting a clinical comorbidity index for use with ICD-9-CM administrative databases. J. Clin. Epidemiol. **45:**613–619.
8. **Fierobe, L., J.-C. Lucet, D. Decré, C. Muller-Serieys, A. Deleuze, M.-L. Joly-Guillou, J. Mantz, and J.-M. Desmonts.** 2001. An outbreak of imipenem-resistant *Acinetobacter baumannii* in critically ill surgical patients. Infect. Control Hosp. Epidemiol. **22:**35–40.
9. **Harris, A. D., T. B. Karchmer, Y. Carmeli, and M. H. Samore.** 2001. Methodological principles of case-control studies that analyzed risk factors for antibiotic resistance: a systematic review. Clin. Infect. Dis. **32:**1055–1061.
10. **Harris, A. D., M. H. Samore, M. Lipsitch, K. S. Kaye, E. Perencevich, and Y. Carmeli.** 2002. Control-group selection importance in studies of antimicrobial resistance: examples applied to *Pseudomonas aeruginosa*, enterococci, and *Escherichia coli*. Clin. Infect. Dis. **34:**1558–1563.
11. **Kaul, R., J. A. Burt, L. Cork, H. Dedier, M. Garcia, C. Kennedy, J. Brunton, M. Krajden, and J. Conly.** 1996. Investigation of a multiyear multiple critical care unit outbreak due to relatively drug-sensitive *Acinetobacter baumannii*: risk factors and attributable mortality. J. Infect. Dis. **174:**1279–1287.
12. **Koeleman, J. G. M., G. A. Parlevliet, L. Dijkshoorn, P. H. M. Savelkoul, and C. M. J. E. Vandenbroucke-Grauls.** 1997. Nosocomial outbreak of multiresistant *Acinetobacter baumannii* on a surgical ward: epidemiology and risk factors for acquisition. J. Hosp. Infect. **37:**113–123.
13. **Lortholary, O., J.-Y. Fagon, A. B. Hoi, M. A. Slama, J. Pierre, P. Giral, R. Rosenzweig, L. Gutmann, M. Safar, and J. Acar.** 1995. Nosocomial acquisition of multiresistant *Acinetobacter baumannii*: risk factors and prognosis. Clin. Infect. Dis. **20:**790–796.
14. **Mulin, B., D. Talon, J. F. Viel, C. Vincent, R. Leprat, M. Thouverez, and Y. Michel-Briand.** 1995. Risk factors for nosocomial colonization with multiresistant *Acinetobacter baumannii*. Eur. J. Clin. Microbiol. Infect. Dis. **14:**569–576.
15. **Paterson, D. L.** 2002. Looking for risk factors for the acquisition of antibiotic resistance: a 21st-century approach. Clin. Infect. Dis. **34:**1564–1567.
16. **Struelens, M. J., E. Carlier, N. Maes, E. Serruys, W. G. V. Quint, and A. van Belkum.** 1993. Nosocomial colonisation and infection with multiresistant *Acinetobacter baumannii*: outbreak delineation using DNA macrorestriction analysis and PCR-fingerprinting. J. Hosp. Infect. **25:**15–32.
17. **Tankovic, J., P. Legrand, G. De Gatines, V. Chemineau, C. Brun-Buisson, and J. Duval.** 1994. Characterization of a hospital outbreak of imipenem-resistant *Acinetobacter baumannii* by phenotypic and genotypic methods. J. Clin. Microbiol. **32:**2677–2681.
18. **Villers, D., E. Espaze, M. Coste-Burel, F. Giauffret, E. Ninin, F. Nicolas, and H. Richet.** 1998. Nosocomial *Acinetobacter baumannii* infections: microbiological and clinical epidemiology. Ann. Intern. Med. **129:**182–189.

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ERRATUM

Risk Factors for Acquisition of Imipenem-Resistant *Acinetobacter baumannii*: a Case-Control Study

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Volume 48, no. 1, p. 224–228, 2004. Page 224, column 2, line 16: "risk of IRAB infection" should read "risk of IRAB occurrence."

Page 225, column 2, line 49: "IRAB" should read "imipenem-resistant *Pseudomonas aeruginosa*."

Page 225, column 2, line 51: "ISAB" should read "imipenem-susceptible *P. aeruginosa*."