Relevance of Digestive Tract Colonization in the Epidemiology of Nosocomial Infections Due to Multiresistant *Acinetobacter baumannii*

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Fecal colonization with multiresistant *Acinetobacter baumannii* was evaluated in 189 consecutive patients in intensive care units (ICUs) during two different 2-month periods (October–November 1993 and May–June 1994). Rectal swabs were obtained weekly from admission to discharge from the ICU. Overall, 77 patients (41%) had multiresistant *A. baumannii* fecal colonization; colonization was detected in 55 (71%) of the patients within the first week of their ICU stay. Clinical infections due to multiresistant *A. baumannii* occurred more frequently in patients with fecal colonization than in those without fecal colonization (26% vs. 5%, respectively; $P < .001$). The reinforcement of isolation measures between study periods reduced both the number of fecal carriers of multiresistant *A. baumannii* (from 52% to 31%; $P < .01$) and the number of patients with multiresistant *A. baumannii* infections (from 17% to 11%; no statistical significance). The digestive tract of ICU patients could be an important epidemiologic reservoir for multiresistant *A. baumannii* infections in hospital outbreaks. Further prospective studies should be undertaken to define the relative significance of digestive tract colonization compared with other body site colonizations.

Over the last decade, *Acinetobacter baumannii* has become a nosocomial pathogen of worldwide concern [1, 2]. It has been increasingly involved in hospital outbreaks, particularly in intensive care units (ICUs) [3, 4]. In most of these outbreaks, different inanimate objects in the hospital environment have been implicated as the principal source of infections [5, 6]. However, the genus *Acinetobacter* is known to be a normal inhabitant of human skin, and some investigators have postulated that, in the setting of an outbreak of *A. baumannii* infection, humans could be not only transient skin carriers facilitating cross-contamination but also a potential source for hospital spread [5–10].

In 1993, Timsit et al. [11] reported that the digestive tract may be colonized by *A. baumannii,* providing permanent carriage in patients in ICUs and a possible source for epidemic infections. Similar suggestions can be inferred from several studies wherein *A. baumannii* colonization in hospitalized patients was evaluated [12–15]. Nevertheless, there are scattered prospective clinical studies demonstrating that patients can be the major epidemiologic reservoir of infection during outbreaks.

At the end of 1993, a cluster of infections due to extended-spectrum β-lactamase-producing *Klebsiella pneumoniae* emerged in our hospital, prompting us to initiate a surveillance program of fecal carriage in ICU patients. Unexpectedly, a high prevalence of fecal colonization with multiresistant *A. baumannii* was found, and a prospective study was undertaken.

Patients and Methods

Setting. Hospital de Bellvitge is a 1,000-bed teaching hospital for adults in Barcelona, Spain, that provides acute medical and surgical care (excluding pediatrics, obstetrics, and burn care) and has an active organ transplantation program. It has three 12-bed ICUs with one nurse for every two patients, except for transplant patients who each have a nurse assigned exclusively to them. In 1992, a significant increase in the number of isolations of multiresistant *A. baumannii*—mainly recovered from ICU patients and susceptible only to ticarcillin, imipenem, and sulbactam—was observed. A control program for multiresistant *A. baumannii* infection was introduced.

Study design. The study took place in the ICUs. The objectives of the study were as follows: (1) to assess the relevance of multiresistant *A. baumannii* fecal colonization; (2) to identify risk factors for multiresistant *A. baumannii* fecal colonization; and (3) to evaluate the efficacy of the reinforcement of isolation precautions. Rectal swabs were obtained from patients admitted to the ICUs during the first 48 hours after admission and weekly thereafter during the study period, and a final rectal swab was taken at the time of ICU discharge. All those patients admitted

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to the ICU for >48 hours from whom at least two rectal swabs were obtained were included in the study, and their data were evaluated on the basis of a specific computer-assisted protocol.

To analyze the efficacy of the reinforcement of infection control measures, the study was performed during two different 2-month periods (1 October to 30 November 1993 and 1 May to 30 June 1994). Reinforcement of control measures included implementation of isolation precautions focusing on glove and gown removal and hand washing; special educational sessions for ICU personnel as well as continuing information on rates of colonization with multiresistant *A. baumannii* were provided. Selective digestive decontamination was not performed between study periods.

Definitions of clinical colonization and infection. Standard definitions of the Centers for Disease Control and Prevention were used [16]. Fecal samples were not considered clinical samples.

Environmental surveillance. Environmental cultures were performed over the two study periods; environmental specimens were obtained from bed rails, walls, floor, tabletops, ventilator tubes, washbasins, faucets, doorknobs, intravenous drip supports, and ventilator and monitor touch keys.

Exposure to risk factors. The simplified acute physiological score and the McCabe classification were defined according to previous reports [17, 18]. Organ transplantation was considered a risk factor when it had been performed during the present admission. Prior antibiotic therapy was defined as one or more antimicrobials administered for >48 hours from 1 week before ICU admission to the occurrence of fecal colonization.

Microbiological methods. Rectal swabs were plated on MacConkey agar supplemented with 6 μg of gentamicin/mL and 5% sheep blood agar. The plates were incubated at 37°C for 48 hours. Since all multiresistant *A. baumannii* strains isolated from clinical samples during the outbreak were resistant to gentamicin, this antibiotic was selected for screening of fecal samples. Environmental samples were obtained with a moistened swab that was incubated on brain-heart infusion broth (Oxoid, Basingstoke, Hampshire, England) and subcultured onto MacConkey agar. Gloves were immersed in brain-heart infusion broth and subcultured onto MacConkey agar plates after 24 hours of incubation.

Isolates were identified as *A. baumannii* on the basis of standard biochemical reactions and their ability to grow at 44°C and by the MicroScan system (NegCombo Type 6I plates; Baxter Laboratories, West Sacramento, CA). Antibiotic susceptibility testing was performed by a microdilution method (MicroScan system). The resistance of the strains was classified according to the criteria of the National Committee for Clinical Laboratory Standards [19].

Pulsed field gel electrophoresis (PFGE) was performed on 36 multiresistant *A. baumannii* strains recovered during the two study periods. Of the 36 strains, 27 were isolated from rectal samples (four obtained within the first 48 hours after admission); 4, from blood; and 5, from environmental surfaces. Genomic DNA was prepared in agarose plugs as previously described [20]. DNA inserts were digested with *Sma* I according to the manufacturer's specifications (New England BioLabs, Beverly, MA). Restriction fragments were separated by PFGE with use of a CHEF-DR III apparatus (Bio-Rad, Richmond, CA). The conditions for PFGE were 200 V for 20 hours; pulse times ranged from 0.5 to 15 seconds. We assigned isolates to PFGE clonal groups according to the criterion that one to three different bands constituted clonally related strains, whereas more than three different bands constituted a different clonal group.

Statistical analysis. Risk factors of patients with or without multiresistant *A. baumannii* fecal colonization were compared by means of the χ^2 test, Fisher's exact test, or Student's *t*-test when appropriate. The number of previous days in the ICU for different invasive procedures (insertion of vascular or urine catheters or nasogastric tubes or intubation) was not included as a variable in the univariate analysis because the extraordinary earliness of fecal colonization hampered its appropriate statistical evaluation. Parameters selected in the univariate analysis were evaluated by multiple logistic regression. A two-tailed *P* value of <.05 was considered statistically significant. Statistical analyses were performed with the SPSS-PC statistical package (SPSS, Chicago).

Results

One hundred twenty of the 309 patients admitted to the ICUs during the study periods were excluded (ICU hospitalization of ≤48 hours, 97 patients; only one rectal swab obtained, 23 patients). No demographic differences were observed between the two study periods for the 189 patients who were finally enrolled in the study (table 1).

Relevance of multiresistant A. baumannii fecal colonization. Multiresistant *A. baumannii* fecal colonization was found in 77 (41%) of the 189 patients; of these 77 patients, 55 (71%) were colonized during the first week; 10 (13%), during the second week; 9 (12%), during the third week; and 3 (4%), during the fourth to fifth weeks. The rectal swabs obtained from 20 patients within the first 48 hours were already positive; eight of these patients were admitted to the hospital from the community and did not have previous hospital admissions, and 12 were from other wards of the hospital (10 had a short previous hospital stay [mean, 5.5 days; range, 3–9 days], and two had a longer previous hospitalization [15 and 26 days, respectively]). The probability of remaining free of fecal colonization is shown in figure 1.

Seventy-eight clinical samples from 47 (25%) of the 189 patients were positive for multiresistant *A. baumannii* (respiratory tract, 29; blood, 14; catheter tip, 14; wound, 10; urine, 5; bone, 2; other sites, 4). Positive clinical samples were mainly recovered from those patients with multiresistant *A. baumannii* fecal colonization (38 [81%] of 47). Predictive values positive for isolation of multiresistant *A. baumannii* from clinical sam-

Table 1. Demographic variables of 189 patients in the ICUs during both study periods.

Variable	October through November 1993 (n = 84)	May through June 1994 (n = 105)	P value
Males/females	58 (69)/26 (31)	65 (62)/40 (38)	NS (.30)
Mean age (y) ± SD	53.5 ± 16.5	56.6 ± 15.7	NS (.19)
Mean SAPS ± SD	10.2 ± 2.9	10.2 ± 3.1	NS (.96)
McCabe classification*	15 (17.9)	12 (11.4)	NS (.20)
ICU stay (d) ± SD	18.8 ± 17.3	15.2 ± 15.2	NS (.13)
Diabetes mellitus	11 (13.1)	16 (15.2)	NS (.83)
Chronic pulmonary disease	9 (10.7)	13 (12.4)	NS (.89)
Polytrauma	11 (13.1)	8 (7.6)	NS (.21)
Chronic renal failure	2 (2.4)	3 (2.9)	NS (.83)
Liver cirrhosis	9 (10.7)	6 (5.7)	NS (.32)
Solid cancer	9 (10.7)	7 (6.7)	NS (.46)
Major surgery	67 (79.8)	80 (76.2)	NS (.55)
Immunosuppression	14 (16.7)	11 (10.5)	NS (.21)
Organ transplant	10 (11.9)	8 (7.6)	NS (.45)
Prior antibiotic therapy	50 (59.5)	54 (51.4)	NS (.26)

NOTE. Unless stated otherwise, data are no. (%) of patients. ICU = intensive care unit; NS = not significant; SAPS = simplified acute physiology score.

* Groups 2 and 3 [18].

ples were significantly higher for those patients with multiresistant *A. baumannii* fecal colonization than for those without fecal colonization (49% vs. 8%, respectively; $P < .001$). Multiresistant *A. baumannii* was considered responsible for a total of 33 episodes of infection in 26 (14%) of the 189 patients.

Infections included respiratory tract infections in 16 patients (tracheobronchitis, 9; pneumonia, 7), bacteremia in 11, and surgical site infections in 6 (peritonitis, 4; osteoarthritis, 1; meningitis, 1). The infection-related mortality rate was 11.5%. Most patients who had multiresistant *A. baumannii* infections were fecal carriers of multiresistant *A. baumannii* (20 [77%] of 26). Predictive values positive for a clinical episode of infection by multiresistant *A. baumannii* were also higher for patients with multiresistant *A. baumannii* fecal colonization than for those without fecal colonization (26% vs. 5%, respectively; $P = .0001$).

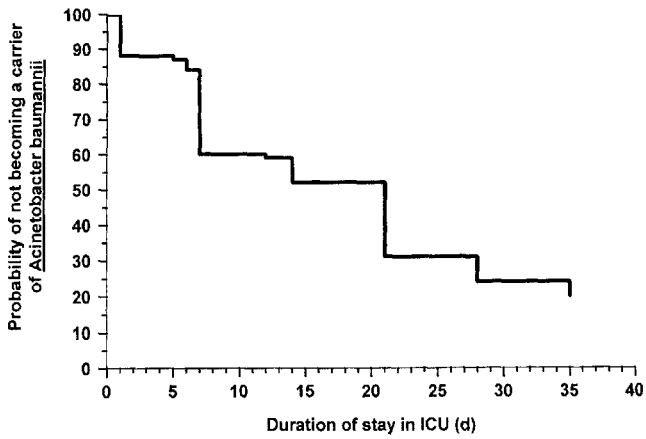

Figure 1. The probability of remaining free of fecal colonization with multiresistant *Acinetobacter baumannii*. ICU = intensive care unit.

Table 2. Antibiotic susceptibilities of 86 multiresistant *Acinetobacter baumannii* strains.

Antibiotic	Breakpoint of susceptibility (μg/mL)	Percentage of susceptible strains: October through November 1993 (n = 46)	Percentage of susceptible strains: May through June 1994 (n = 40)
Imipenem	≤4	100	100
Sulbactam	≤8	100	100
Ticarcillin	≤16	88.2	85.8
Piperacillin	≤16	0.6	1.2
Ceftazidime	≤8	3.3	10.4
Amikacin	≤16	15.2	2.5
Gentamicin	≤4	0	0
Tobramycin	≤4	0	0
Ciprofloxacin	≤1	0	0
Tetracycline	≤4	0	0
Co-trimoxazole	≤2	0	0

Over the two periods of the study, multiresistant *A. baumannii* was recovered from five (19%) of a total of 26 ICU environmental samples (monitor touch keys, 3; ventilator tubes, 1; floor, 1). Furthermore, multiresistant *A. baumannii* was also isolated from three of five latex gloves worn by staff members who had been touching patients.

The antibiotic susceptibilities of 86 multiresistant *A. baumannii* strains isolated from fecal and clinical samples (one per colonized patient: 77 from fecal carriers and nine from patients without fecal colonization from whom clinical isolates were recovered) are shown in table 2. Two different antibiotypes were found among the 86 strains; 78 strains showed resistance to piperacillin, ceftazidime, amikacin, tobramycin, gentamicin, ciprofloxacin, co-trimoxazole, and tetracycline, and eight had the same resistance profile but were susceptible to amikacin (MICs, 1 μg/mL). A single clonal type could be defined by PFGE among 36 multiresistant *A. baumannii* strains studied. The PFGE patterns for 17 multiresistant *A. baumannii* strains are shown in figure 2.

When less than three band differences were considered, four distinct subtypes could be distinguished: subtype 1, 24 isolates; subtype 2, 10; subtype 3, 1; and subtype 4, 1. There was no correlation between antibiotype and PFGE subtype. Isolates from rectal swabs and blood from four patients were analyzed, and an identical macrorestriction pattern was found.

Risk factors for multiresistant A. baumannii fecal colonization. The results of a comparison between patients with and

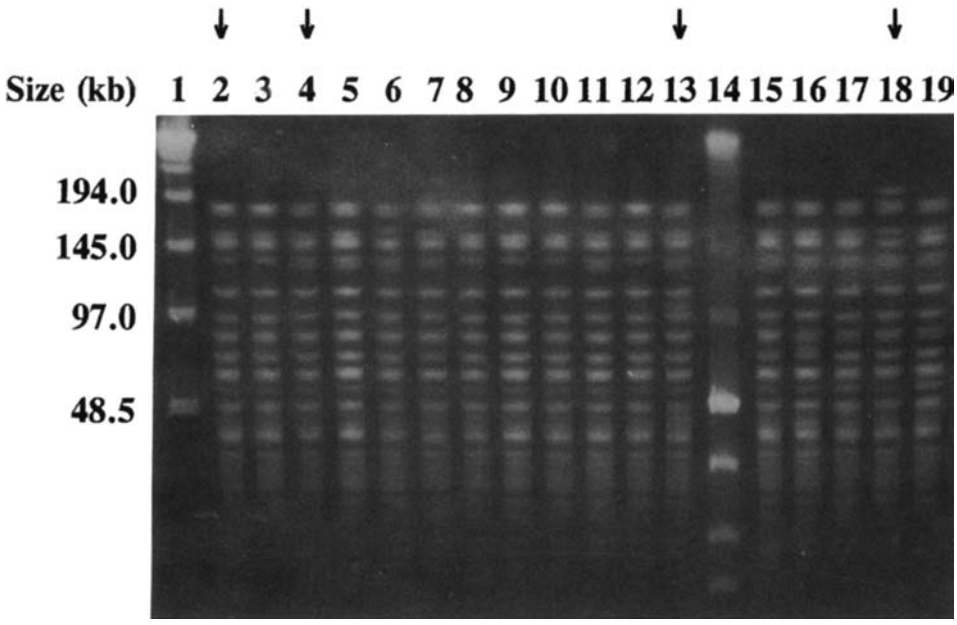

Figure 2. Pulsed field gel electrophoresis (PFGE) patterns of genomic DNA from 17 multiresistant *Acinetobacter baumannii* strains after *Sma* I digestion. *Lanes 2* and *3*, fecal and blood isolates, respectively, from patient A; *lanes 4* and *5*, fecal and blood isolates, respectively, from patient B; *lanes 6* and *7*, fecal and blood isolates, respectively, from patient C. All but two strains (*lanes 8* and *9*) had the amikacin-resistant antibiotype. *Lanes 8* and *9*, two amikacin-susceptible fecal isolates of *A. baumannii*. *Lanes 10–13*, strains recovered from rectal swabs obtained within the first 48 hours of hospital admission. *Lanes 15–19*, environmental isolates (ventilator, monitor, gloves, bed, and floor, respectively). *Lanes 1* and *14*, λ ladder and low-range PFGE marker (New England BioLabs, Beverly, MA), respectively. Arrows indicate four distinct PFGE subtypes. kb = kilobases.

without multiresistant *A. baumannii* fecal colonization are shown in table 3. Multiple logistic regression selected polytrauma (OR, 5.0; 95% CI, 1.5–16.1) as the only positively associated independent risk factor for multiresistant *A. baumannii* fecal colonization and organ transplantation as a negatively associated independent risk factor for multiresistant *A. baumannii* fecal colonization (OR, 0.1; 95% CI, 0.01–0.7).

Table 3. Risk factors for fecal colonization with multiresistant *Acinetobacter baumannii:* results of univariate and multivariate analyses.

Risk factor	Patients without fecal colonization (n = 112)	Patients with fecal colonization (n = 77)	P value
Males/females	67 (60)/45 (40)	56 (73)/21 (27)	NS (.067)
Mean age (y) ± SD	57.3 ± 14.1	52.2 ± 18.2	.041
Mean SAPS ± SD	10.2 ± 3.0	10.2 ± 2.9	NS (.94)
McCabe classification*	20 (17.9)	7 (9.1)	NS (.09)
Diabetes mellitus	15 (13.4)	12 (15.6)	NS (.67)
Chronic pulmonary disease	13 (11.6)	9 (11.7)	NS (.98)
Polytrauma†	4 (3.6)	15 (19.5)	.0004
Chronic renal failure	4 (3.6)	1 (1.3)	NS (.33)
Liver cirrhosis	13 (11.6)	2 (2.6)	.024
Solid cancer	11 (9.8)	5 (6.5)	NS (.41)
Major surgery	89 (79.5)	58 (75.3)	NS (.50)
Immunosuppression	20 (17.9)	5 (6.5)	.023
Organ transplant†	16 (14.3)	2 (2.6)	.007
Prior antibiotic therapy	56 (50.0)	48 (62.3)	NS (.09)

NOTE. Unless stated otherwise, data are no. (%) of patients. NS = not significant; SAPS = simplified acute physiology score.

* Groups 2 and 3 [18].

† Parameters selected as independent risk factors in the multivariate analysis.

Efficacy of the reinforcement of isolation precautions. The rate of fecal carriage of multiresistant *A. baumannii* was clearly reduced between the two study periods (52% to 31%; $P < .01$). In addition, the number of patients with infections due to multiresistant *A. baumannii* also decreased between the two periods (17% to 11%; not statistically significant).

Discussion

The major features of the epidemiology of nosocomial outbreaks of *A. baumannii* infection remain as yet unexplained. There is no doubt that both the surprising ability of this microorganism to acquire antimicrobial multiresistance and its high capacity for survival on most environmental surfaces are important factors for nosocomial spread. In fact, different contaminated objects in hospital environments have been implicated as the main source of infections [2, 5, 6]. Nevertheless, in the last few years, several studies have suggested that patients may play an important epidemiologic role in large and sustained outbreaks [9, 10].

The high rates of fecal carriage in ICU patients that have been reported by Timsit et al. [11] and other investigators [12–15] strongly point toward the digestive tract as the reservoir for epidemic infections, occurring in a manner similar to most nosocomial outbreaks of infections due to gram-negative bacilli. Although *A. baumannii* is not considered to be an inhabitant of the digestive tract in healthy humans [21], in severely ill hospitalized patients, the normal flora of the bowel—which provides resistance to intestinal colonization—can be modified [22]. Under these conditions, patients are predisposed to persistent colonization by exogenous pathogens causing nosocomial epidemics.

The results of our study are in agreement with this line of thinking. The probability of remaining free of multiresistant *A. baumannii* fecal colonization on the 30th day of ICU hospitalization was <25%. Although two different antibiotypes were found among our epidemic strains, they showed only slight differences regarding amikacin susceptibility. Since there was no correlation between antibiotype and PFGE subtype and since segregation of PFGE subtypes was irrelevant in the epidemiologic surveillance, a single epidemic clone could be responsible for the outbreak during the two study periods.

We did not design the study to analyze the major epidemiologic features implicated in our outbreak, but the high prevalence of digestive tract colonization in the ICU patients and the comparatively low prevalence of positive environmental cultures point toward patients as the principal epidemiologic reservoir in our hospital.

The earliness with which digestive colonization occurred should be noted. Seventy-one percent of the patients were colonized within the first week, and 25% were colonized during the first 48 hours of ICU hospitalization. Although we cannot totally rule out the possibility that some of the patients for whom the first rectal swab was already positive were previously colonized, we believe that multiresistant *A. baumannii* fecal colonization was mainly acquired during ICU hospitalizations since patients came from either the community or other hospital wards after a short previous hospital stay in which no significant epidemiologic problems had been detected. Similar experiences have been recently reported by Go et al. [9], thus reaffirming the extraordinary contagiousness of *A. baumannii* infection.

This early colonization hampers the identification of risk factors commonly associated with the acquisition of infections due to other nosocomial pathogens, such as host-dependent risk factors and the number of previous days in the ICU for different invasive procedures. Therefore, the risk factors selected in our study could correspond to several reasons and must be carefully interpreted. The positive association of polytraumatism with multiresistant *A. baumannii* fecal colonization may indicate the high number of manipulations that these patients undergo. Most patients with polytrauma were young, which could be the reason why age was selected in the univariate analysis but not in the multivariate analysis.

On the other hand, the inverse correlation of organ transplantation and colonization may be related to the fact that transplant recipients were each assigned their own nurse. Furthermore, liver cirrhosis and immunosuppression were also negatively associated in the univariate analysis; however, these parameters were closely related to organ transplantation (liver transplantations were most common).

In our outbreak, the relevance of cross-contamination via transmission by the staff's hands was supported by the positive cultures of gloves worn by staff members who had been touching patients. The role of latex gloves in this transmission has been clearly demonstrated by Patterson et al. [8], thereby stressing the importance of adequate application of isolation measures to control outbreaks. In fact, the reinforcement of these measures decreased the rates of fecal colonization in our ICU patients, although these measures were not enough to control the outbreak.

Because most multiresistant *A. baumannii* clinical colonizations or infections occurred in multiresistant *A. baumannii* fecal carriers and because digestive colonization occurred mainly before clinical isolations, a fecal surveillance program in an outbreak setting could be considered necessary for early implementation of isolation precautions for these patients.

Further studies should be undertaken to define the relative significance of digestive tract colonization compared with other body site colonizations. Both other body site colonizations and the antimicrobial multiresistance of *A. baumannii* may limit the efficacy of selective intestinal decontamination as an additional useful measure for achieving definitive control of these kinds of outbreaks.

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References

1. Bergogne-Bérézin E, Joly-Guillou ML. Hospital infection with *Acinetobacter* spp.: an increasing problem. J Hosp Infect **1991**;18(suppl A): 250–5.
2. Bergogne-Bérézin E, Joly-Guillou ML, Vieu JF. Epidemiology of nosocomial infections due to *Acinetobacter calcoaceticus*. J Hosp Infect **1987**; 10:105–13.
3. Vila J, Almela M, Jimenez de Anta MT. Laboratory investigation of hospital outbreak caused by two different multiresistant *Acinetobacter calcoaceticus* subsp. *anitratus* strains. J Clin Microbiol **1989**;27:1086–9.
4. Contant J, Kemeny E, Oxley C, Perry E, Garber G. Investigation of an outbreak of *Acinetobacter calcoaceticus* var. *anitratus* infections in an adult intensive care unit. Am J Infect Control **1990**;18:288–91.
5. Cefai C, Richards J, Gould FK, McPeake P. An outbreak of *Acinetobacter* respiratory tract infection resulting from incomplete disinfection of ventilatory equipment. J Hosp Infect **1990**;15:177–82.
6. Sherertz RJ, Sullivan ML. An outbreak of infections with *Acinetobacter calcoaceticus* in burn patients: contamination of patients' mattresses. J Infect Dis **1985**;151:252–8.
7. French GL, Casewell MW, Roncoroni AJ, Knight S, Phillips I. A hospital outbreak of antibiotic resistant *Acinetobacter anitratus:* epidemiology and control. J Hosp Infect **1980**;1:125–31.
8. Patterson JE, Vecchio J, Pantelick EL, et al. Association of contaminated gloves with transmission of *Acinetobacter calcoaceticus* var. *anitratus* in an intensive care unit. Am J Med **1991**;91:479–83.
9. Go ES, Urban C, Burns J, et al. Clinical and molecular epidemiology of acinetobacter infections sensitive only to polymyxin B and sulbactam. Lancet **1994**;344:1329–32.
10. Mulin B, Talon D, Viel JF, et al. Risk factors for nosocomial colonization with multiresistant *Acinetobacter baumannii*. Eur J Microbiol Infect Dis **1995**;14:569–76.

11. Timsit JF, Garrait V, Misset B, Goldstein FW, Renaud B, Carlet J. The digestive tract is a major site for *Acinetobacter baumannii* colonization in intensive care unit patients [letter]. J Infect Dis **1993**;168:1336–7.
12. Sakata H, Fujita K, Maruyama S, Kakehashi H, Mori Y, Yoshioka H. *Acinetobacter calcoaceticus* biovar *anitratus* septicaemia in a neonatal intensive care unit: epidemiology and control. J Hosp Infect **1989**;14: 15–22.
13. Wise KA, Tosolini FA. Epidemiological surveillance of *Acinetobacter* species. J Hosp Infect **1990**;16:319–29.
14. Lortholary O, Buu-Hoï A, Fagon JY, et al. Evaluation of swabs for the detection of colonization/infection with multiresistant *Acinetobacter baumannii* [abstract no 585]. In: Program and abstracts of the 6th European Congress of Clinical Microbiology and Infectious Diseases. Seville, Spain: European Society of Clinical Microbiology and Infectious Diseases, **1993**:168.
15. Joly-Guillou ML, Wolff M, Decré D, Calvat S, Bergogne-Bérézin E. Colonization and infection with *Acinetobacter baumannii* in ICU patients receiving selective digestive decontamination: results of case-control and molecular epidemiologic investigations [abstract no J-213]. In: Program and abstracts of the 34th Interscience Conference on Antimicrobial Agents and Chemotherapy (Orlando). Washington, DC: American Society for Microbiology, **1994**:103.
16. Garner JS, Jarvis WR, Emori TG, Horan TC, Hughes JM. CDC definitions for nosocomial infections, 1988. Am J Infect Control **1988**;16:128–40.
17. Le Gall JR, Loirat P, Alpérovitch A, et al. A simplified acute physiology score for ICU patients. Crit Care Med **1984**;12:975–7.
18. McCabe WR, Jackson GG. Gram negative bacteremia. Am J Med **1962**; 110:847–55.
19. National Committee for Clinical Laboratory Standards. Performance for antimicrobial susceptibility testing: fifth informational supplement. NCCLS document M110-55. Villanova, Pennsylvania: National Committee for Clinical Laboratory Standards, **1994**.
20. Allardet-Servent A, Bouziges N, Carles-Nurit MJ, Bourg G, Gouby A, Ramuz M. Use of low-frequency-cleavage restriction endonucleases for DNA analysis in epidemiological investigations of nosocomial bacterial infections. J Clin Microbiol **1989**;27:2057–61.
21. Grehn M, von Graevenitz A. Search of *Acinetobacter calcoaceticus* subsp. *anitratus:* enrichment of fecal samples. J Clin Microbiol **1978**;8:342–3.
22. Brun-Buisson C, Legrand P. Can topical and nonabsorbable antimicrobials prevent cross-transmission of resistant strains in ICUs? Infect Control Hosp Epidemiol **1994**;15:447–55.