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Use of Pulsed-Field Gel Electrophoresis Typing To Study an Outbreak of Infection Due to *Serratia marcescens* in a Neonatal Intensive Care Unit

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Serratia marcescens **is a well-known cause of nosocomial infections and outbreaks, particularly in critically ill neonates and immunocompromised patients. Numerous methods have been proposed for typing. We used pulsed-field gel electrophoresis (PFGE) typing to analyze an outbreak in a neonatal intensive care unit (NICU). We included 23 patient isolates from an outbreak (March to July 1995), and 10 patient isolates from different wards during the same time period. PFGE of whole-cell DNA digested by *Spe*I was used as a marker of strain identity. The most common presentation of the infection was sepsis in 18 of 23 (78%) neonates. Only four different biotypes were identified; biotype A8d accounted for 84% of the strains. PFGE typing revealed two clones responsible for two different clonal strain dissemination outbreaks from March to July, with 24 patient isolates being pattern A and 4 patient isolates being pattern E. PFGE typing suggests cross transmission between patients in the NICU and other wards. The isolates from 5 other patients showed distinct PFGE patterns. Extensive investigation and cultures failed to identify any environmental or staff reservoir of *S. marcescens*. This is one of the first reports applying PFGE to the study of *S. marcescens*, and this method was a useful marker of strain identity. PFGE typing distinguished strains which appeared to be the same by biotyping.**

Serratia marcescens has been implicated in many types of nosocomial infection (1). Outbreaks of *S. marcescens* have been frequently reported (19, 20, 22, 24). Studies from the 1970s suggested that neonatal colonization and infection were rare events; however, from 1981 to 1995 the number of publications that document epidemics of *S. marcescens* infections in neonatal intensive care units (NICU) has increased (8, 11, 16, 21, 25, 26). Environmental sources of *S. marcescens* in the hospital are widely described (1, 5, 19, 22). This microorganism has a predilection for a moist environment. Some of the environmental sources mentioned previously have been sponges, fiberoptic bronchoscopes, adhesive tape, distilled water, plastic caps of saline bottles, catheters, floors, inhalation therapy equipment, electrocardiogram bulbs, transducers, intravenous solutions, irrigating fluids, hand lotions, shaving brushes and soap, sink traps, and flowers and plants (1, 19, 20, 22, 24). In neonates, colonized symptom-free patients have been described as an important reservoir (8, 18). Multiple resistance of *S. marcescens* to antibiotics causes a problem in treating infected patients and controlling outbreaks (5, 7, 19).

Studies of epidemiological markers are important in an attempt to trace the source of contamination or to prevent patient to patient dissemination of strains. Numerous methods have been proposed to type *S. marcescens* for epidemiological purposes: antibiograms, biotyping, serotyping, phage typing, bacteriocin typing, plasmid analysis, DNA restriction endonuclease analysis, ribotyping, and PCR. The molecular typing techniques are preferred in differentiating strains for epidemiological studies (2, 3, 14). The other techniques cannot discriminate between strains, or they are either lengthy and labor-intensive or available through only a few reference laboratories (6, 15, 19). We used pulsed-field gel electrophoresis (PFGE) of whole-cell DNA digested with *Spe*I to analyze an outbreak in an NICU.

MATERIALS AND METHODS

Background. The Pediatric Hospital, National Medical Center (NMC) in Mexico City, Mexico, is a 200-bed tertiary care center opened in 1992. The NICU has 16 beds admitting critically ill neonates. When they no longer require intensive care, they are transferred to the Department of Infants in the same floor. Twenty-three patient isolates from an outbreak (March to July 1995) in the NICU and 10 patient isolates from different wards during the same time period were analyzed in this study. A patient was considered to have a severe infection if clinical signs of sepsis, pneumonia, and/or meningitis were present.

Before this outbreak, only 5 patients in the Pediatric Hospital (NMC) had infection due to *S. marcescens* (May 1992 to February 1995). During this epidemic, the most common presentation of infection in the neonates was sepsis in 18 of 23 (78%). Seven of these patients died (30% mortality), and patients from the other wards presented with catheter-related bacteremia (50%) and abdominal sepsis (20%) (pancreatic abscess, complicated apendicitis, necrotizing enterocolitis). None of the patients in other wards died.

Bacterial isolates. Twenty-three isolates were from blood, five were from catheter tips, three were from peritoneal fluid, one was from pleural fluid, and one was from an abdominal abscess. The clinical isolates were identified as *S. marcescens* by VITEK (Bio-Merieux, Hazelwood, Mo.). The biochemical profile number were also noted. Susceptibilities of the microorganisms to antimicrobial agents were determined by agar diffusion according to the procedures suggested by the National Committee for Clinical Laboratory Standards (17). The antimicrobial agents included were ampicillin, gentamicin, amikacin, netilmicin, isepamicin, chloramphenicol, cefotaxime, cefepime, ceftazidime, aztreonam, imipenem, pefloxacin, and trimethoprim/sulfamethoxazole.

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Environmental samples were taken from incubators, humidifiers, suction jars, antiseptics, soap, irrigation fluids, distilled water, inhalation therapy equipment, intravenous solutions, sinks, and stethoscopes. Hand cultures were taken from members of the staff on several occasions by means of a damp cotton swab, which was rubbed over both hands. All the samples were incubated overnight in brain heart infusion (BHI) broth at 37°C and subcultured on MacConkey agar.

Biotyping and biogrouping. Biotyping and biogrouping were performed according to the method of Grimont and Grimont (9, 10), including carbon source utilization tests (*m*-erythritol, trigonelline, quinate, 3-hydroxybenzoate, 4-hydrozybenzoate, benzoic acid, DL-carnitine, D-rhamnose, lactose, adonitol, L-arabinose, D-sorbitol), tetrathionate reduction, and prodigiosine production. A biogroup is formed by strains of closely related biotypes.

PFGE typing. A single colony from an 18- to 24-h subculture plate was inoculated into a tube containing 5 ml of BHI broth and incubated without shaking overnight at 37°C. These tubes were then centrifuged at 4,000 × *g* for 20 min; then the supernatant was removed and the pellets were resuspended in 2 ml of PIV buffer (1.0 M Tris [base], 1.0 M NaCl, pH 7.6) and allowed to equilibrate to 50°C, after which 2 ml of 1.6% low-melting-point agarose (Boehringer Mannheim, Indianapolis, Ind.) at 50°C was added to each cell suspension and the mixture was pipetted into Bio-Rad (Richmond, Calif.) plug molds and refrigerated at 4°C for 45 min. Plugs were removed and incubated in 5 ml of lysis buffer (1.0 M Tris, 1.0 M NaCl, 100 mM EDTA, 0.5% Brij 58, 0.2% deoxycholate, 0.5% Na lauroyl sarcosine, 50 μg of ribonuclease A [Sigma, St. Louis, Mo.] per ml, and 1 mg of lysozyme [Sigma] [pH 7.6] per ml) overnight at 37°C. Then the lysis buffer was replaced by ESP buffer (0.4 M EDTA, 1% sodium lauroyl sarcosine [pH 9.0] and 0.5 mg of proteinase K [Sigma] per ml) and the plugs were incubated for 24 h at 50°C. Plugs were washed once with 1× TE at 37°C (5 mM Tris, 5 mM EDTA [pH 7.5]) containing 1 mM phenylmethylsulfonyl fluoride and three times with 0.1× TE, each change for at least 30 min. For restriction endonuclease digestion, plug slices were equilibrated in 300 μl of the appropriate 1× restriction endonuclease buffer for 2 to 3 h at room temperature, after which 30 U of *Spe*I (Boehringer Mannheim) were added and the mixture was incubated at the manufacturer-suggested incubation temperature. Plug slices were then loaded into and run on a 0.8% PFGE agarose gel in 1× TBE buffer (0.089 M Tris [base], 0.089 M borate, 2.5 mM disodium EDTA) (ICN Biomedicals, Aurora, Ohio). PFGE was performed with a CHEF-DRII system (Bio-Rad). The pulse time was 5 to 60 s over 15 h to 17 h at 200 V. Lambda concatemers (Bio-Rad) were used as the size standard. Gels were stained for 20 min in 1 μg of ethidium bromide per ml, decolorized in distilled water for 10 min, and photographed by filtered UV illumination.

Based on recent guidelines, isolates were considered to be the same strain if their PFGE patterns were identical. An isolate was considered to be a closely related strain if the PFGE pattern differed from the outbreak pattern by two or three bands. An isolate was considered to be possibly related if its PFGE pattern differs from the outbreak pattern by four to six bands. An isolate was considered distinct if the isolate differed from the outbreak pattern by seven or more bands (23).

RESULTS

Five different biochemical profiles were identified by using the Vitek system. Based on the biotyping/biogrouping system, four different biotypes were identified, with A8d being the most common (84.8%); others were A6a (9%), A2a (3%), and ND2 (3%). The three biogroups noted were A5/8 (84.8%), A8c (12%), and A2/6 (3%) (see Table 2).

All the isolates were resistant to aztreonam, trimethoprim/sulfamethoxazole, and ampicillin; 100% were susceptible to isepamicin, 97% were susceptible to amikacin, 90% were susceptible to pefloxacin, 87% were susceptible to imipenem, 72% were susceptible to cefotaxime, and cefepime, 55% were susceptible to chloramphenicol, 51% were susceptible to ceftazidime, 25% were susceptible to netilmicin, and 22% were susceptible to gentamicin. We did not find a characteristic susceptibility pattern, and in comparison with the results by biotyping and PFGE typing there was no correlation between the biotypes, the PFGE patterns, and the susceptibility profiles (Table 1).

By PFGE typing, 24 patients had the same strain (pattern A), and this clone was responsible for the outbreak from March to July (Fig. 1), suggesting cross-transmission between patients in the NICU (20 patients) and other wards (4 patients). A separate clone (pattern E, E_1, E_2) was responsible for infection in four other patients (two in the NICU and two on other wards). The isolates from five other patients showed distinct patterns (patterns B, C, D, F, G) (Fig. 2).

TABLE 1. Antimicrobial susceptibility testing of *S. marcescens*

Agent	No. (%) of resistant isolates
Aztreonam	33 (100)
Trimethoprim/sulfamethoxazole	33 (100)
Ampicillin	33 (100)
Gentamicin	26 (78.7)
Netilmicin	25 (75.5)
Ceftazidime	16 (49)
Chloramphenicol	15 (45.4)
Cefepime	9 (28)
Cefotaxime	9 (28)
Imipenem	4 (13)
Pefloxacin	3 (10)
Amikacin	1 (3)
Isepamicin	0 (0)

Table 2 illustrates the distribution of the PFGE patterns during March to July, as well as the location of the patients, site of infection, biochemical profile, biotype, biogroup, and date of isolation. Extensive investigation and cultures failed to identify any environmental or staff reservoir of *S. marcescens.*

During the outbreak a number of control measures were introduced. Strict hand washing by personnel between their handling of patients was stressed. Attempts were made to group infected and noninfected patients by cohort. Nurses were encouraged to wear gloves when suctioning and during any catheter manipulation of the patients.

DISCUSSION

Severe illness due to *S. marcescens* is generally seen in immunocompromised patients. In newborns these organisms can be responsible for sepsis and meningitis, which have a high case fatality rate (5). Before this outbreak *S. marcescens* infections were rarely seen at the Pediatric Hospital, even though 60% of the admitted patients are immunocompromised (patients with underlying diseases such as cancer, AIDS, and nephrotic syndrome, etc.).

The index case (patient 1) was transferred from another

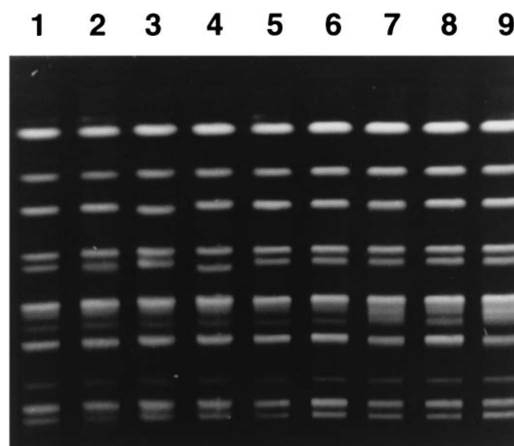

FIG. 1. PFGE of whole-cell DNA from *S. marcescens* isolates digested with *Spe*I. The pulse time was 5 to 60 s over 17 h at 200 V. The PFGE patterns in lanes 1 to 9 are the same pattern (A), suggesting a common strain during the outbreak (March to July 1995) in patients 1, 3, 4, 5, 6, 9, 12, 27, and 29.

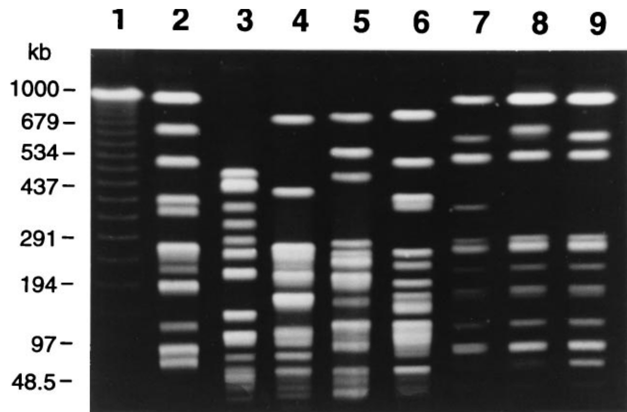

FIG. 2. PFGE of whole-cell DNA from *S. marcescens* isolates digested with *Spe*I. The pulse time was 5 to 60 s over 15 h at 200 V. Lane 2 shows the PFGE pattern of the outbreak strain (pattern A, patient 1). Lanes 3 to 6 show distinct PFGE patterns (B, C, D, F) from patients 8, 16, 19, and 21, respectively. Lanes 7 to 9 show the PFGE patterns E, E_1, and E_2 from patients 22, 26, and 31, respectively. Lambda ladder size standard is shown in lane 1.

hospital, and *S. marcescens* was isolated from cultures taken upon admission. Most of the infections in the neonates were severe, while infections of patients from the other wards were related to intravascular devices or a previous abdominal problem.

A great variety of typing methods have been applied to the study of infections due to *S. marcescens*. Some clinical bacteriologists have attempted to use API20E or VITEK numerical profiles as epidemiological tools. Unfortunately, although they are satisfactory systems for species identification, reproducibility of the numerical profile biotype for enterics showed that only 56% of the strains gave identical biotypes when testing was repeated (14, 19). The biochemical profile numbers found in this study did not correlate with the results by PFGE typing. Strains with the same biochemical profile number had a distinct pattern by PFGE, and also strains with distinct pattern showed the same biochemical profile number. Similar findings have been pointed out by other authors (1, 12), and it has been proposed not to use biotyping to differentiate strains from the same hospital.

Even though a biotyping system by Grimont and Grimont (9, 10) has been described as a useful epidemiological method for typing *S. marcescens*, the results are contradictory among different authors, who have compared this method with serotype, bacteriocin type, ribotyping (2), DNA restriction endonuclease analysis (3, 15), and polyacrylamide gel electrophoresis (13). Biotyping is lengthy and labor-intensive, and the reproducibility and discriminatory power are poor, even though it is an easy method to perform. In this study biotyping failed to distinguish correctly five strains that had a PFGE pattern distinct from that of the outbreak strain.

Molecular typing methods have been used more recently. Ribotyping (2, 4), PCR fingerprinting (14), and DNA typing by

TABLE 2. Location of the patients, site of infection, and characteristics of *S. marcescens* isolates during the outbreak

Patient no.	Location	Infection	PFGE pattern	Biotype	Biogroup	Date of isolation (mo/day/yr)
1	NICU	Sepsis	A	A8d	A5/8	03/03/95
2	NICU	Sepsis	A	A8d	A5/8	03/06/95
3	NICU	Catheter-related bacteremia	A	A8d	A5/8	03/07/95
4	4th floor	Catheter-related bacteremia	A	A6a	A2/6	03/09/95
5	NICU	Catheter-related bacteremia	A	A8d	A5/8	03/13/95
6	NICU	Sepsis/meningitis	A	A8d	A5/8	03/20/95
7	NICU	Sepsis/meningitis	A	A8d	A5/8	05/19/95
8	3rd floor	Catheter-related bacteremia	B	ND2	A8/c	05/24/95
9	3rd floor	Abdominal sepsis	A	A8d	A5/8	05/02/95
10	NICU	Sepsis	A	A8d	A5/8	05/15/95
11	NICU	Sepsis	A	A8d	A5/8	05/16/95
12	4th floor	Sepsis	A	A8d	A5/8	05/18/95
13	NICU	Sepsis	A	A8d	A5/8	05/25/95
14	NICU	Catheter-related bacteremia	A	A8d	A5/8	05/28/95
15	NICU	Sepsis	A	A8d	A5/8	06/08/95
16	PICU[a]	Sepsis	C	A2a	A2/6	06/15/95
17	NICU	Sepsis	A	A8d	A5/8	06/16/95
18	NICU	Sepsis	A	A8d	A5/8	06/22/95
19	5th floor	Catheter-related bacteremia	D	A8d	A5/8	06/22/95
20	NICU	Sepsis	A	A8d	A5/8	06/28/95
21	5th floor	Abdominal sepsis	F	A6a	A2/6	06/27/95
22	NICU	Sepsis	E	A8d	A5/8	06/27/95
23	NICU	Sepsis	A	A8d	A5/8	06/24/95
24	NICU	Sepsis	A	A8d	A5/8	06/23/95
25	4th floor	Catheter-related bacteremia	E	A8d	A5/8	07/03/95
26	PICU	Pneumonia	E_1	A6a	A2/6	07/06/95
27	NICU	Sepsis	A	A8d	A5/8	07/06/95
28	NICU	Sepsis	A	A8d	A5/8	07/08/95
29	NICU	Sepsis	A	A8d	A5/8	07/24/95
30	NICU	Sepsis	G	A8d	A5/8	07/25/95
31	NICU	Abdominal sepsis	E_2	A8d	A5/8	07/17/95
32	4th floor	Catheter-related bacteremia	A	A8d	A5/8	07/05/95
33	NICU	Abdominal sepsis	A	A8d	A5/8	07/26/95

[a] PICU, Pediatric intensive care unit.

restriction endonuclease analysis (3, 15) have been considered optimal methods for differentiation of strains in epidemiological investigation. A single method may be used alone for this purpose, but none of them has been adopted universally. Ribotyping is slower and more labor-intensive and involves working with probes, PCR is a rapid method but requires development of primers for each organism and expensive start-up equipment, and the complex banding patterns produced by restriction endonuclease analysis are difficult to interpret.

PFGE of whole-cell DNA digested with restriction endonucleases is a relatively rapid and very reproducible reliable method that can be used to type a broader array of bacterial species (23). To our knowledge this is one of the first reports of applying PFGE typing to the study of *S. marcescens*. In our study it demonstrated cross-transmission between patients in the NICU and other wards. It seems that two different strains were responsible for two outbreaks, one from March to July (pattern A, 24 patients) and the other during June and July (pattern E, 4 patients). Five other patients were infected by distinct strains. Biotyping and susceptibility profile did not successfully distinguish distinct strains.

The environmental investigation failed to identify a source of *S. marcescens* from surfaces, water, solutions, ventilation systems, stethoscopes, or hands of the staff. The most likely reservoir for the epidemic in the NICU was the gastrointestinal tract of infants, as has been reported before (16, 18). Transient contamination of hands of personnel has been suggested as playing an important part in transmission of *S. marcescens* in newborns infants (5) and we believe that this, as well as the patients being transferred from the NICU to the fourth floor ward, could have contributed to the dissemination of the strains within the hospital. PFGE typing results were useful in education of the staff regarding cross transmission, to avoid ongoing spread of the organism.

In conclusion, PFGE typing of whole-cell DNA is useful to study outbreaks of *S. marcescens*. PFGE typing was useful in distinguishing strains which appeared to be the same by biotyping and biogrouping. Results facilitated prompt evaluation of the outbreak so that effective preventive measures could be instituted.

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REFERENCES

1. **Acar, J. F.** 1986. *Serratia marcescens* infections. Infect. Control. **7:**273–278.
2. **Alonso, R., H. M. Aucken, J. C. Perez-Diaz, B. D. Cookson, F. Baquero, and T. L. Pitt.** 1993. Comparison of serotype, biotype and bacteriocin type with rDNA RFLP patterns for the type identification of *Serratia marcescens*. Epidemiol. Infect. **111:**99–107.
3. **Bale, M., M. Sanford, R. Hollis, and M. A. Pfaller.** 1993. Application of a biotyping system and DNA restriction fragment analysis to the study of *Serratia marcescens* from hospitalized patients. Diagn. Microbiol. Infect. Dis. **16:**1–7.
4. **Bingen, E. H., P. Mariani-Kurkdjian, N. Y. Lambert-Zechovsky, P. Desjardins, E. Denamur, Y. Aujard, E. Vilmer, and J. Elion.** 1992. Ribotyping provides efficient differentiation of nosocomial *Serratia marcescens* isolates in a pediatric hospital. J. Clin. Microbiol. **30:**2088–2091.
5. **Bollman, R., E. Halle, W. Sokolowka-Kohler, E. L. Gravel, P. Buchholz, I. Klare, H. Tschape, and W. Witte.** 1989. Nosocomial infections due to *Serratia marcescens*. Clinical findings, antibiotic susceptibility patterns and fine typing. Infection **17:**294–300.
6. **Coria-Jimenez, V. R., L. Villa-Tanaka, and C. Ortiz-Torres.** 1991. Partial characterization of *Serratia marcescens* nosocomial strains. Arch. Invest. Med. **22:**273–278.
7. **Coria-Jimenez, V. R., and C. Ortiz-Torres.** 1994. Aminoglycoside resistance patterns of *Serratia marcescens* strains of clinical origin. Epidemiol. Infect. **112:**125–131.
8. **Gordon, D., M. D. Christensen, and B. Sheldon.** 1982. Epidemic *Serratia marcescens* in a neonatal intensive care unit. Importance of the gastrointestinal tract as a reservoir. Infect. Control. **3:**127.
9. **Grimont, P. A. D., and F. Grimont.** 1978. Biotyping of *Serratia marcescens* and its use in epidemiological studies. J. Clin. Microbiol. **8:**73–83.
10. **Grimont, P. A. D., F. Grimont, S. Le Minor, B. Davis, and F. Pigache.** 1979. Compatible results obtained from biotyping and serotyping in *Serratia marcescens*. J. Clin. Microbiol. **10:**425–432.
11. **Hammer, H.** 1984. *Serratia marcescens* in a neonatal intensive care unit—a 5-year analysis. Padiatr. Grenzgeb. **23:**299–303.
12. **Hartstein, A. I., V. H. Morthland, J. W. Rourke, R. Sykes, and A. L. Rashad.** 1993. Plasmid DNA analysis, biotyping, and antimicrobic susceptibility as subtyping tests for *Klebsiella pneumoniae* and *Klebsiella oxytoca*. Diagn. Microbiol. Infect. Dis. **16:**35–41.
13. **Larose, P., B. Picard, M. Thibault, F. Grimont, and P. H. Goullet.** 1990. Nosocomial *Serratia marcescens* individualized by five typing methods in a regional hospital. J. Hosp. Infect. **15:**167–172.
14. **Liu, Y., Y. Lau, B. S. Hu, J. Shir, M. Cheung, Z. Shi, and W. Tsai.** 1994. Use of PCR to study epidemiology of *Serratia marcescens* isolates in nosocomial infection. J. Clin. Microbiol. **32:**1935–1938.
15. **McGeer, A., D. E. Low, J. Penner, J. NG, C. Goldman, and A. E. Simor.** 1990. Use of molecular typing to study the epidemiology of *Serratia marcescens*. J. Clin. Microbiol. **28:**55–58.
16. **Montanaro, D., G. M. Grasso, I. Annino, N. De Ruggiero, A. Scarcella, and F. Schioppa.** 1984. Epidemiological and bacteriological investigation of *Serratia marcescens* epidemic in a nursery and in a neonatal intensive care unit. J. Hyg. **93:**67–78.
17. **National Committee for Clinical Laboratory Standards.** 1994. Methods for dilution antimicrobial susceptibility tests for bacteria that grow aerobically. 3rd ed. Approved standard. M7-A3. National Committee for Clinical Laboratory Standards, Villanova, Pa.
18. **Newport, M. T., F. F. John, Y. M. Michel, and A. H. Leukoff.** 1985. Endemic *Serratia marcescens* infection in a neonatal intensive care nursery associated with gastrointestinal colonization. Pediatr. Infect. Dis. **4:**160–167.
19. **Sifuentes-Osornio, J., G. M. Ruiz-Palacios, and D. H. Gröschel.** 1986. Analysis of epidemiologic markers of nosocomial *Serratia marcescens* isolates with special reference to the Grimont biotyping system. J. Clin. Microbiol. **23:** 230–234.
20. **Simor, A. E., L. Ramage, L. Wilcox, S. B. Bull, and H. Bialkowska-Hobrzanska.** 1988. Molecular and epidemiologic study of multiresistant *Serratia marcescens* infections in a spinal cord injury rehabilitation unit. Infect. Control. Hosp. Epidemiol. **9:**20–27.
21. **Smith, P. J., D. S. K. Brookfield, D. A. Shaw, and J. Gray.** 1984. An outbreak of *Serratia marcescens* infection in a neonatal unit. Lancet **ii:**151–153.
22. **Solaski, S. J., M. A. Jewell, A. C. Asmus-Shillington, J. Mulcahy, and J. Segreti.** 1992. An outbreak of *Serratia marcescens* in 14 adult cardiac surgical patients associated with 12-lead electrocardiogram bulbs. Arch. Intern. Med. **152:**841–844.
23. **Tenover, F. C., R. D. Arbeit, R. V. Goering, P. A. Mickelsen, B. E. Murray, D. H. Persing, and B. Swaminathan.** 1995. Interpreting chromosomal DNA restriction patterns produced by pulsed field gel electrophoresis. Criteria for bacterial strain typing. J. Clin. Microbiol. **33:**2233–2239.
24. **Villarino, M. E., W. R. Jarvis, C. O'Hara, J. Bresnahan, and N. Clark.** 1989. Epidemic of *Serratia marcescens* bacteremia in a cardiac intensive care unit. J. Clin. Microbiol. **27:**2433–2436.
25. **Wake, C., H. Lees, and A. B. Cull.** 1986. The emergence of *Serratia marcescens* as a pathogen in a newborn unit. Aust. Paediatr. J. **22:**323–326.
26. **Zaidi, M., J. Sifuentes, M. Bobadilla, D. Moncada, and S. Ponce de Leon.** 1989. Epidemic of *Serratia marcescens* bacteremia and meningitis in a neonatal unit in Mexico City. Infect. Control. Hosp. Epidemiol. **10:**14–20.