

UC Irvine

UC Irvine Previously Published Works

Title

Strain-Relatedness of Methicillin- Resistant Staphylococcus aureus Isolates Recovered from Patients with Repeated Infection

Permalink

<https://escholarship.org/uc/item/48v0z5k1>

Journal

Clinical Infectious Diseases, 46(8)

ISSN

1058-4838

Authors

Huang, Susan S
Diekema, Daniel J
Warren, David K
[et al.](#)

Publication Date

2008-04-15

DOI

10.1086/529381

Copyright Information

This work is made available under the terms of a Creative Commons Attribution License, available at <https://creativecommons.org/licenses/by/4.0/>

Peer reviewed



Published in final edited form as:

Clin Infect Dis. 2008 April 15; 46(8): 1241–1247. doi:10.1086/529381.

Strain-Relatedness of Methicillin-Resistant *Staphylococcus aureus* Isolates Recovered from Patients with Repeated Infection

Susan S. Huang^{1,2}, Daniel J. Diekema^{4,5,6}, David K. Warren⁷, Gianna Zuccotti⁸, Patricia L. Winokur⁴, Shailesh Tendolkar⁵, Linda Boyken⁵, Rupak Datta³, Rebecca M. Jones⁷, Melissa A. Ward⁶, Tanya Aubrey⁸, Andrew B. Onderdonk², Christian Garcia³, and Richard Platt^{2,3}

¹Division of Infectious Diseases, University of California Irvine School of Medicine, Orange

²Channing Laboratory, Department of Medicine, Brigham and Women's Hospital, Boston, Massachusetts

³Department of Ambulatory Care and Prevention Harvard Medical School and Harvard Pilgrim Healthcare, Boston, Massachusetts

⁴Division of Infectious Diseases, Department of Internal Medicine, Iowa City

⁵Department of Pathology, University of Iowa College of Medicine, Iowa City

⁶Program of Hospital Epidemiology, University of Iowa Hospitals and Clinics, Iowa City

⁷Division of Infectious Diseases, Department of Internal Medicine, Washington University School of Medicine, St. Louis, Missouri

⁸Division of Infectious Diseases, Department of Internal Medicine, Memorial Sloan Kettering Cancer Center, New York, New York

Abstract

Invasive disease following methicillin-resistant *Staphylococcus aureus* (MRSA) detection is common, regardless of whether initial detection involves colonization or infection. We assessed the genetic relatedness of isolates obtained ≥ 2 weeks apart representing either repeated infections or colonization-infection sets to determine if infections are likely to be caused by previously harbored strains. We found that MRSA infection following initial colonization or infection is caused by the same strain in most cases, suggesting that a single successful attempt at decolonization may prevent the majority of later infection.

Methicillin-resistant *Staphylococcus aureus* (MRSA) causes >278,000 hospitalizations and 56,000 septic episodes annually [1]. Hospitalized patients with newly identified MRSA colonization or infection have a 35% risk of subsequent invasive disease within 1 year [2,3]. Strategies to prevent infection have included decolonization [–11], administration of anti-staphy-lococcal antibodies [12], and vaccination [13–14].

Decolonization has had variable success [4]. Its benefit may depend on whether subsequent MRSA infections are caused by the original infecting or colonizing strain, indicating persistent carriage or repeated acquisition from the local environment. Under these scenarios, a single successful decolonization of patients and potentially their immediate surroundings could

prevent future infection. However, if subsequent infections are caused by different strains, decolonization would be of limited value unless sustained over long periods.

Colonizing strains are responsible for infections occurring soon after detected colonization [18]. Less is known about the association between colonizing strains and subsequent infections occurring in the intermediate or distant future, but 40%–60% of carriers demonstrate prolonged carriage with the same strain [14–17]. We assessed the genetic relatedness of MRSA isolates obtained >2 weeks apart. We evaluated isolates representing both repeated infections and asymptomatic carriage followed by infection.

Methods

We conducted a multicenter cohort study in 4 academic hospitals identifying patients with MRSA invasive disease following prior MRSA infection or colonization (table 1). Hospitals selected a 1–2-year study period during which all patients with sterile site MRSA cultures >2 weeks apart were identified (the infection-infection group). Associated MRSA isolates were retrieved. Two institutions additionally collected isolates from patients with MRSA-positive nares surveillance cultures followed by invasive clinical cultures (the colonization-infection group). Patients could contribute to both groups. Decolonization was not routinely employed. This study was approved by each hospital's institutional review board.

Medical records were reviewed to confirm infection by Centers for Disease Control and Prevention (CDC) criteria [19]. Sequential infections were required to be discrete and unrelated, as determined through review by an infectious diseases physician. Sequential device-related bacteremia required device removal between events and complete resolution of the initial infection.

Data collected included age, sex, hospitalization dates, length of hospital stay(s), hospital day of cultures, pre-admission location(s), and postdischarge disposition(s). Risk factors for MRSA infection were collected, such as recent surgery, wounds, and comorbidities (hemodialysis, diabetes).

Isolates were sent to the Molecular Epidemiology and Fungus Testing Laboratory at the University of Iowa (Iowa City) for confirmation of methicillin resistance (*mecA* gene probe), antibiotic susceptibility testing, and SCCmec typing [20]. PCR assays for the Pantone-Valentine leukocidin (PVL) gene were performed. Antibiotic susceptibility testing involved broth microdilution [21,22] for oxacillin, erythromycin, clindamycin, tetracycline, rifampin, moxifloxacin, gentamicin, vancomycin, linezolid, and quinupristin-dalfopristin; mupirocin, tetracycline, and trimethoprim-sulfamethoxazole susceptibility were determined by Etest (AB Biodisk) [23]. D-tests for inducible clindamycin resistance were performed when isolates were resistant to erythromycin but susceptible to clindamycin.

Strain types based on PFGE [24] were classified as indistinguishable, similar, or different [25]. Identical banding patterns were considered to be indistinguishable, those differing by ≤ 3 bands were considered to be similar (includes indistinguishable strains), and those differing by > 3 bands were considered to be different. PFGE patterns were compared to defined USA types [26] to distinguish common hospital and community-associated MRSA strains (BioNumerics). Total and hospital-specific dendrograms were created to display strain relatedness.

Patients' characteristics were reported as proportions. Repeated infections were summarized by culture site, infection type by CDC criteria [14], duration between events, and location between events (inpatient, home, or skilled nursing facility or rehabilitation center [SNF/rehab]). Location between events was characterized by the highest acuity center.

Within the infection-infection group, strain relatedness was assessed by the proportion of the first 2 infection events per person that had indistinguishable, similar, or different strain types. Strain relatedness among the colonization-infection group was similarly assessed. Associations between strain relatedness and patient location or duration between events were assessed using Fisher's exact test.

MRSA isolates were additionally described by SCCmec type and presence of PVL. We also assessed whether subsequent infecting strains exhibited increased antibiotic resistance, compared with original strains.

Results

Thirty-seven patients had ≥ 2 discrete MRSA infections, for a total of 83 infections. By study design, nearly all infections (79 [95%] of 83) involved bacteremia. Twenty-nine patients had sets of isolates representing colonization followed by infection, 27 (93%) of which involved bacteremia. Eight of the 29 had another subsequent MRSA infection and contributed to both groups. Patient characteristics and infection types are found in table 2 and table 3.

We found considerable strain diversity, with 97%–100% of patients having PFGE subtypes that were unique within each institution (example institution shown in figure 1). Nevertheless, despite population diversity, strains from a single patient representing either sequential infection or colonization-infection were highly related (table 4). Among the infection-infection group, only 4 (11%) of 37 patients had subsequent MRSA infections caused by a different strain. Patients with intervening SNF/rehab stays were more likely to have infections caused by different strains than were those who went home ($P=.02$) or remained in the hospital ($P=.02$). Longer durations between MRSA infections were not predictive of differing strain types (table 5).

Six patients had >2 MRSA infections. These infections involved 20 bacteremic events spanning 30–302 days (median, 182.5 days). Five of 6 patients' infections were repeatedly caused by indistinguishable strain types.

USA100 was the most common strain type in either group (54 of 66 sets), followed by USA500 (3 sets) and USA200 (2 sets). One patient each had repeated infection with USA300 and USA400, accounting for the 2 PVL positive, SCCmec IV isolates.

The percentage of initial invasive MRSA isolates that were susceptible to the following antibiotics is as follows: moxifloxacin, 6% of isolates; erythromycin, 6%; clindamycin, 33%; trimethoprim-sulfamethoxazole, 92%; tetracycline, 95%; gentamicin, 92%; rifampin, 100%; vancomycin, 100%; linezolid, 100%; quinupristin-dalfopristin, 100%; and mupirocin, 79%. Nine strains had inducible clindamycin resistance. Among the 26 repeated infections caused by identical strains, 4 strains (15%) showed increased antibiotic resistance (moxifloxacin, intermediate to resistant [1 strain]; gentamicin, susceptible to resistant [1 strain]; rifampin, susceptible to intermediate [1 strain]; and clindamycin, susceptible to resistant [1 strain]).

Discussion

With a 35% risk of infection in the year following detection [2,3], there is a need to prevent disease among MRSA carriers. We show that most MRSA infections following initial colonization or infection are caused by identical strains. This likely represents ongoing risks of infection from persistently carried strains, because diverse strain types at participating institutions made repeated acquisition of an identical strain unlikely from any source other than a patient's immediate surroundings. It also supports lack of strain-specific immunity. This

finding suggests that single successful decolonization of patients and possibly their immediate surroundings may obviate the majority of later MRSA infections.

Patients who remained hospitalized and those discharged to home were significantly more likely to experience repeated infections due to identical strains, compared with patients who had intervening SNF/rehab stays. Given the low frequency (<1%) with which healthy people carry MRSA [27], this was not unexpected for MRSA carriers who returned home, where there was unlikely to be a preexisting reservoir of diverse strains among family members. This is in contrast to those with an intervening stay at an SNF/rehab, where MRSA prevalence is often high and where social networking is common and encouraged. Among hospitalized patients, contact precautions and the lack of patient interaction may explain the tendency for a single strain to cause repeated infections despite evidence that hospital strains are diverse. Thus, decolonization of patients in their home or hospital room may be more successful at preventing infection than similar attempts at an SNF/rehab, where repeated screening and decolonization may be needed to ensure lack of carriage.

In 24% of subsequent infections, strains were not identical, although most differed by <3 bands. These strains may represent new environmental acquisition. Another possibility is that the duration between infections was sufficiently long for minor genetic changes to produce highly related, but non-identical, strains.

We found that a patient's location between infections was more predictive of strain relatedness than was time between infections. Nearly all isolates recovered from persons with repeated infection were of similar strain types, even when infections were separated by >6 months.

MRSA strains causing repeated infections generally retained the same susceptibility profile. Of the 15% of strains with a more resistant antibiotic profile at the time of the second infection, only 2 had a substantial change in their MIC, and 1 was confirmed to be related to inducible macrolide-lincosamide-streptogramin resistance.

There are several limitations to this study. First, the sample size is small, despite multiple participating centers. Second, all institutions were academic centers, and results may not be generalizable to other settings. In addition, these results are largely based on health care-associated MRSA strains. Further data are needed to assess the relatedness of strain types in recurrent community-associated MRSA infections. Third, because we did not perform environmental cultures, we do not know the contribution of a patient's immediate surroundings in maintaining carriage with the same strain. Nevertheless, we recognize that several studies have shown that MRSA easily contaminates the environment [28–30].

Repeated MRSA infection and MRSA infection occurring after colonization are not uncommon. We show that the majority of these infections are due to strains identical to the initial strain, even when the events occur many months apart. This evidence of persistent carriage suggests that time-limited decolonization regimens for patients, possibly coupled with disinfection of their surroundings, may substantially reduce the risk of subsequent infection.

Acknowledgments

Financial support. The CDC Prevention Epicenters Program and the National Institutes of Health (K23AI64161-01).

Potential conflicts of interest. D.K.W. receives research support from Sage Products, and has served as a consultant to Enturia and 3M Healthcare. R.P. has received research grants from Sanofi-Aventis, GlaxoSmithKline, Pfizer, and TAP Pharmaceuticals in the past 2 years. All other authors: no conflicts.

References

1. Klein E, Smith DL, Laxminarayan R. Hospitalizations and deaths caused by methicillin-resistant *Staphylococcus aureus*, United States, 1999–2005. *Emerg Infect Dis* 2007;13:1840–1846. [PubMed: 18258033]
2. Huang SS, Platt R. Risk of methicillin-resistant *Staphylococcus aureus* infection after previous infection or colonization. *Clin Infect Dis* 2003;36:281–285. [PubMed: 12539068]
3. Huang, SS.; Hinrichsen, VL.; Stulgis, L., et al. Methicillin-resistant *Staphylococcus aureus* infection in the year following detection of carriage [abstract 157]. Programs and abstracts of the 16th Society of Healthcare Epidemiology of America Annual Meeting (Chicago); Arlington, VA. Society of Healthcare Epidemiology of America; 2006.
4. Loeb M, Main C, Walker-Dilks C, Eady A. Antimicrobial drugs for treating methicillin-resistant *Staphylococcus aureus* colonization. *Cochrane Database Syst Rev* 2003;4CD003340
5. Simor AE, Phillips E, McGeer A, et al. Randomized controlled trial of chlorhexidine gluconate for washing, intranasal mupirocin, and rifampin and doxycycline versus no treatment for the eradication of methicillin-resistant *Staphylococcus aureus* colonization. *Clin Infect Dis* 2007;44:178–185. [PubMed: 17173213]
6. Parras F, Guerrero C, Bouza E, et al. Comparative study of mupirocin and oral co-trimoxazole plus topical fusidic acid in eradication of nasal carriage of methicillin-resistant *Staphylococcus aureus*. *Antimicrob Agents Chemother* 1995;39:175–179. [PubMed: 7695302]
7. Kimura A, Mochizuko T, Nishizawa K, Mashiko K, Yamamoto Y, Otsuka T. Trimethoprim-sulfamethoxazole for the prevention of methicillin-resistant *Staphylococcus aureus* pneumonia in severely burned patients. *J Trauma* 1998;45:383–387. [PubMed: 9715201]
8. Roccaforte JS, Bittner MJ, Stumpf CA, Preheim LC. Attempts to eradicate methicillin-resistant *Staphylococcus aureus* colonization with the use of trimethoprim-sulfamethoxazole, rifampin, and bacitracin. *Am J Infect Control* 1988;16:141–146. [PubMed: 3263821]
9. Perl TM, Cullen JJ, Wenzel RP, et al. Intranasal mupirocin to prevent postoperative *Staphylococcus aureus* infections. *N Engl J Med* 2002;346:1871–1877. [PubMed: 12063371]
10. Kauffman CA, Terpenning MS, He X, et al. Attempts to eradicate methicillin-resistant *Staphylococcus aureus* from a long-term-care facility with the use of mupirocin ointment. *Am J Med* 1993;94:371–378. [PubMed: 8475930]
11. Harbarth S, Dharan S, Liassine N, Herrault P, Auckenthaler R, Pittet D. Randomized, placebo-controlled, double-blind trial to evaluate the efficacy of mupirocin for eradicating carriage of methicillin-resistant *Staphylococcus aureus*. *Antimicrob Agents Chemother* 1999;43:1412–1416. [PubMed: 10348762]
12. Weisman, LE.; Thackray, HM.; Gracia-Prats, JA., et al. Phase I/II double blind, placebo controlled, dose escalation, safety and pharmacokinetics study in very low birth weight neonates of BSX-A110, an antistaphylococcal monoclonal antibody for the prevention of staphylococcal bloodstream infections [abstract LB14]. Programs and abstracts of the Pediatric Academic Societies Annual Meeting (San Francisco); Woodlands, TX. Pediatric Academic Societies; 2004.
13. Shinefield H, Black S, Fattom A, et al. Use of a *Staphylococcus aureus* conjugate vaccine in patients receiving hemodialysis. *N Engl J Med* 2002;346:491–496. [PubMed: 11844850]
14. Fattom A, Fuller S, Propst M, et al. Safety and immunogenicity of a booster dose of *Staphylococcus aureus* types 5 and 8 capsular polysaccharide conjugate vaccine (StaphVAX) in hemodialysis patients. *Vaccine* 2004;23:656–663. [PubMed: 15542186]
15. Herwaldt LA, Pottinger JM, Coffman S, Tjaden J. Molecular epidemiology of methicillin-resistant *Staphylococcus aureus* in a Veterans Administration medical center. *Infect Control Hosp Epidemiol* 2002;23:502–505. [PubMed: 12269446]
16. Maslow JN, Brecher S, Gunn J, Durbin A, Barlow MA, Arbeit RD. Variation and persistence of methicillin-resistant *Staphylococcus aureus* strains among individual patients over extended periods of time. *Eur J Clin Microbiol Infect Dis* 1995;14:282–290. [PubMed: 7649190]
17. Lim MSC, Marshall CL, Spelman D. Carriage of multiple subtypes of methicillin-resistant *Staphylococcus aureus* by intensive care unit patients. *Infect Control Hosp Epidemiol* 2006;27:1063–1067. [PubMed: 17006813]

18. Von Eiff C, Becker K, Machka K, Stammer H, Peters G. Nasal carriage as a source of *Staphylococcus aureus* bacteremia. *New Engl J Med* 2001;344:11–16. [PubMed: 11136954]
19. Horan, TC.; Gaynes, RP. Surveillance of nosocomial infections. In: Mayhall, CG., editor. *Hospital epidemiology and infection control*. Vol. 3rd ed.. Philadelphia: Lippincott Williams & Wilkins; 2004. p. 1659-1702.
20. Oliveira DC, de Lencastre H. Multiplex PCR strategy for rapid identification of structural types and variants of the mec element in methicillin-resistant *Staphylococcus aureus*. *Antimicrob Agents Chemother* 2002;46:2155–2161. [PubMed: 12069968]
21. Clinical and Laboratory Standards Institute (formerly National Committee for Clinical Laboratory Standards [NCCLS]). Approved standard M7-A5. Vol. 5th ed.. Wayne, PA: NCCLS; 2000. Methods for dilution antimicrobial susceptibility tests for bacteria that grow aerobically.
22. Clinical and Laboratory Standards Institute. CLSI document M100-S16. Wayne, PA: CLSI; 2004. Performance standards for antimicrobial susceptibility testing: 16th informational supplement.
23. Palepou MF, Johnson AP, Cookson BD, et al. Evaluation of disc diffusion and Etest for determining the susceptibility of *Staphylococcus aureus* to mupirocin. *J Antimicrob Chemother* 1998;42:577–583. [PubMed: 9848440]
24. Diekema DJ, Pfaller MA, Turnidge J, et al. Genetic relatedness of multidrug- resistant, methicillin (oxacillin)-resistant *Staphylococcus aureus* bloodstream isolates from SENTRY antimicrobial resistance surveillance centers worldwide, 1998. *Microb Drug Resist* 2000;6:213–221. [PubMed: 11144421]
25. Tenover FC, Arbeit RD, Goering RV. How to select and interpret molecular strain typing methods for epidemiological studies of bacterial infections: a review for healthcare epidemiologists: molecular typing working group of the society for healthcare epidemiology of america. *Infect Control Hosp Epidemiol* 1997;18:426–439. [PubMed: 9181401]
26. McDougal LK, Steward CD, Killgore GE, Chaitram JM, McAllister SK, Tenover FC. Pulsed-field gel electrophoresis typing of oxacillin-resistant *Staphylococcus aureus* isolates from the United States: establishing a national database. *J Clin Microbiol* 2003;41:5113–5120. [PubMed: 14605147]
27. Kuehnert MJ, Kruszon-Moran D, Hill HA, et al. Prevalence of *Staphylococcus aureus* nasal colonization in the United States 2001–2002. *J Infect Dis* 2006;193:172–179. [PubMed: 16362880]
28. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect Control Hosp Epidemiol* 1997;18:622–627. [PubMed: 9309433]
29. Sexton T, Clarke P, O’Neill E, et al. Environmental reservoirs of methicillin-resistant *Staphylococcus aureus* in isolation rooms: correlation with patient isolates and implications for hospital hygiene. *J Hosp Infect* 2006;62:187–194. [PubMed: 16290319]
30. French GL, Otter JA, Shannon KP, et al. Tackling contamination of the hospital environment by methicillin-resistant *Staphylococcus aureus* (MRSA): a comparison between conventional terminal cleaning and hydrogen peroxide vapour decontamination. *J Hosp Infect* 2004;57:31–37. [PubMed: 15142713]

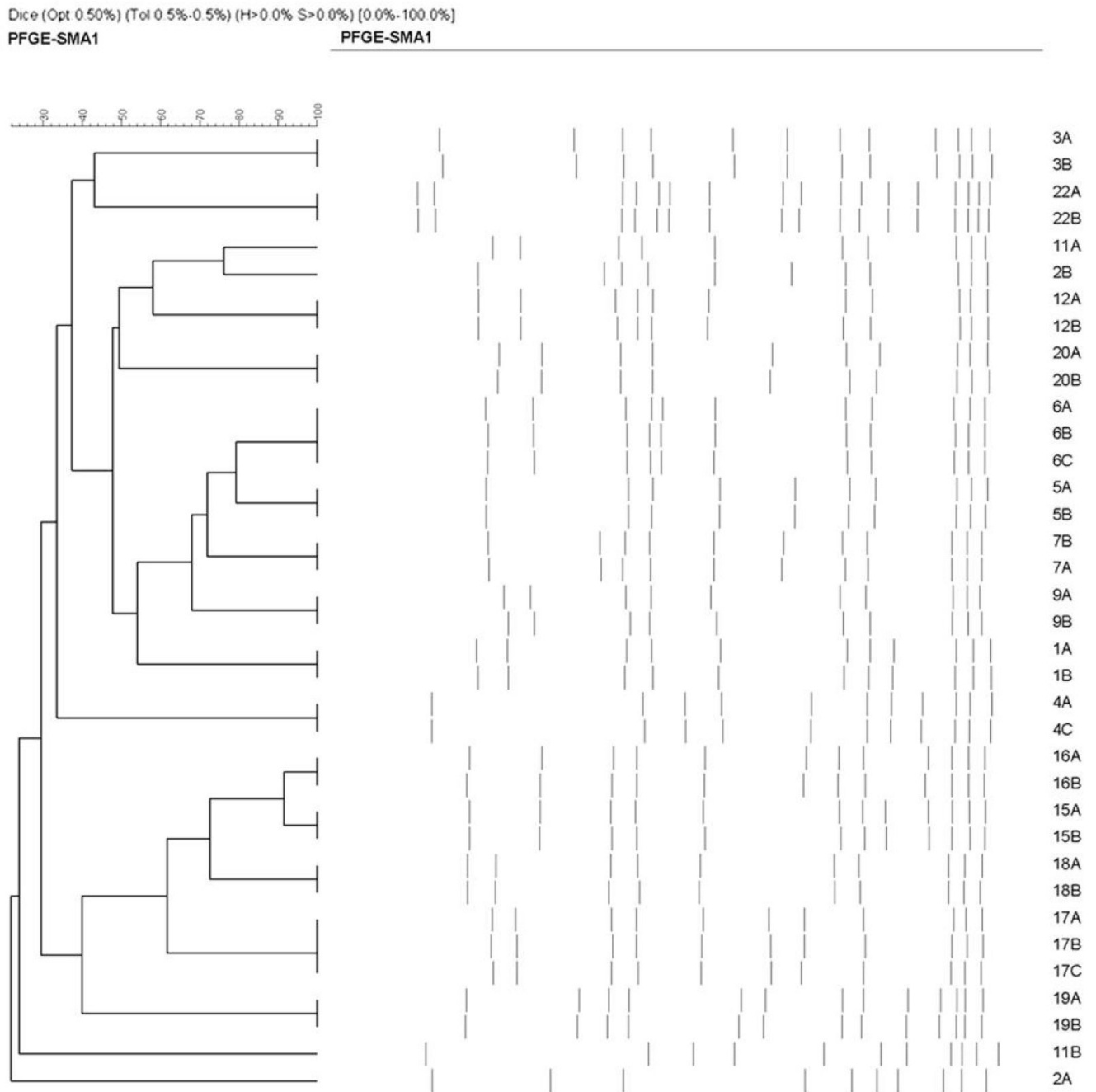


Figure 1.

Dendrogram demonstrating the broad diversity of methicillin-resistant *Staphylococcus aureus* (MRSA) strains across patients at a single participating institution. The dendrogram is based on PFGE of MRSA isolates from discrete events. Seventeen patients are represented, each identified by a number on the right side of the figure. Fourteen patients had multiple discrete bacteremic events, and 3 patients had bacteremia following surveillance isolates. Letter designations within a given number refer to isolates from the same patient. In this example institution, no 2 patients have identical isolates based on PFGE. Identical strain types may be slightly offset in pattern as a result of small differences in lane alignment or gel characteristics when grouping strains for dendrogram analysis.

Table 1

Participating institutions and cohorts.

Institution	Study design	Collected specimens	Study period
1	Retrospective cohort, hospital-wide	Repeated bacteremia	1 January 2002 to 31 December 2003
2	Retrospective cohort, hospital-wide	Repeated invasive disease ^a	1 January 2002 to 31 December 2003
3	Retrospective cohort, hospital-wide	Repeated bacteremia	1 September 2003 to 31 August 2004
3	Retrospective cohort, surgical intensive care unit	Nares-bacteremia	1 September 2003 to 31 August 2004
4	Prospective cohort, intensive care units (8) ^b	Repeated invasive disease ^a ,nares-invasive disease ^a	1 September 2003 to 30 June 2005

^aDefined by Centers for Disease Control criteria (see methods).

^bIntensive care units included cardiac (1), medical (1), surgical (1), burn/trauma (1), cardiac surgery (2), neurosurgery (1), and thoracic surgery (1).

Table 2Characteristics of patients with methicillin-resistant *Staphylococcus aureus* infections.

Characteristic	No. (%) of patients	
	Infection-infection (n = 37)	Colonization-infection (n = 29)
Hospital		
1	10 (27)	0 (0)
2	2 (5)	0 (0)
3	14 (38)	3 (10)
4	11 (30)	26 (90)
Male sex	18 (49)	17 (59)
Age group, years		
18–34	5 (14)	4 (14)
35–44	4 (11)	1 (3)
45–54	5 (14)	4 (14)
55–64	10 (27)	3 (10)
65–84	11 (30)	17 (59)
≥85	2 (5)	0 (0)
Diabetes	13 (35)	8 (28)
Hemodialysis	5 (14)	3 (10)
Recent surgery ^a	21 (57)	26 (90)
Wound ^b	17 (46)	22 (76)

^aDefined as surgery within 6 months of the initial isolate. The high proportion of surgery in the colonization-infection group reflects sampling in surgical intensive care units.

^bAt time of initial isolate.

Table 3Characteristics of repeated methicillin-resistant *Staphylococcus aureus* events.

Characteristic	Infection-infection ^a (n = 37)	Colonization-infection ^a (n = 29)
Time between events, median days (range)	74 (14–482)	39 (15–250)
Source of first isolate		
Primary bacteremia		
Total	28 (76)	29 (100) ^b
Catheter-related	23 (82)	...
Unknown source	5 (18)	...
Secondary bacteremia		
Total	6 (16)	...
Surgical site infection	3 (50)	...
Lung infection	2 (33)	...
Bone/joint infection	1 (17)	...
Other (nonbacteremic)	3 (8) ^c	...
Source of second isolate ^d		
Primary bacteremia		
Total	24 (65)	21 (72)
Catheter-related	21 (88)	11 (52)
Unknown source	3 (12)	...
Secondary bacteremia		
Total	11 (30)	6 (21)
Surgical site infection	3 (27)	0 (0)
Lung infection	2 (18)	3 (50)
Bone/joint infection	3 (27)	1 (17)
Vascular	3 (27)	1 (17)
Soft-tissue infection	0 (0)	1 (17)
Other (nonbacteremic)	2 (5) ^e	2 (7) ^f
Patient disposition between isolates		
Not discharged	13 (35)	15 (52)
Home ^g	13 (35)	4 (14)
Skilled nursing facility or rehabilitation center	11 (30)	10 (34)

NOTE. All data are no. (%) of patients, unless otherwise indicated.^aDescription pertains to first 2 events per person.^bAll primary bacteremia events in the colonization-infection group were nares-bacteremias.^cUrinary tract infection (1), lung infection (1), and gastrointestinal infection (1).^dNinety percent of patients experiencing serial bacteremia had an echocardiogram performed to exclude endocarditis as the source.^eUrinary tract infection (1) and bone and joint infection (1).^fSurgical site infection (1) and bone and joint infection (1).

^gRestricted to patients whose only disposition between events was to home.

Genetic relatedness of methicillin-resistant *Staphylococcus aureus* (MRSA) strains isolated from individual patients.

Table 4

Source	Indistinguishable strain type ^a	No. (%) of patients			PVL positive ^c	SCCmec IV ^d
		Similar strain type ^{a,b}	Different strain type ^a			
Infection-infection						
Total (n = 37)	28 (76)	33 (89)	4 (11)		2 (5) ^d	4 (11)
Bacteremia-bacteremia ^e (n = 34)	26 (76)	30 (88)	4 (12)		2 (6) ^d	4 (12)
Colonization-infection						
Total (n = 29)	21 (72)	29 (100)	0 (0)		0 (0)	2 (7)
Nares-bacteremia ^f (n = 27)	19(70)	27 (100)	0 (0)		0 (0)	2 (7)
Total (n = 66)	49(74)	62 (94)	4 (6)		2 (3) ^d	6 (9)

NOTE. PVL, Pantone-Valentine leukocidin.

^aFirst 2 events per patient.

^bIncludes indistinguishable and similar strains. Strains differ by ≤ 3 bands by PFGE.

^cNo. of sets with any strain meeting this criterion.

^dOne set was USA300, and one was USA400.

^eMRSA isolates from 2 discrete MRSA bloodstream events in a single patient.

^fMRSA isolate from a nares culture and a subsequent MRSA bloodstream event in a single patient.

Table 5Similarity of methicillin-resistant *Staphylococcus aureus* strain types, by interval days and location.

Characteristic	No. (%) of patients		
	Indistinguishable strain type	Similar strain type ^a	Different strain type
Infection-infection sets ^b			
Interval time, months			
<2 (<i>n</i> = 14)	11 (79)	13 (93)	1 (7)
2–5 (<i>n</i> = 17)	13 (76)	15 (88)	2 (12)
≥6 (<i>n</i> = 6)	4 (67)	5 (83)	1 (17)
Interval location			
Not discharged (<i>n</i> = 13)	11 (85) ^c	13 (100) ^c	0 (0) ^c
Home (only) (<i>n</i> = 14)	14 (100) ^c	14 (100) ^c	0 (0) ^c
Skilled nursing facility or rehabilitation center (<i>n</i> = 10)	3 (30)	6 (60)	4 (40)
Colonization-infection sets ^d			
Interval time, months			
<2 (<i>n</i> = 20)	13 (65)	20 (100)	0 (0)
2–5 (<i>n</i> = 7)	6 (86)	7 (100)	0 (0)
≥6 (<i>n</i> = 2)	2 (100)	2 (100)	0 (0)
Interval location			
Not discharged (<i>n</i> = 15)	12 (80)	15 (100)	0 (0)
Home (only) (<i>n</i> = 7)	5 (71)	7 (100)	0 (0)
Skilled nursing facility or rehabilitation center (<i>n</i> = 7)	4 (57)	7 (100)	0 (0)

^aIncludes indistinguishable and similar strains. Strains differ by ≤3 bands by PFGE.^bFirst 2 events per patient.^c*P* < .05 when comparing proportions with those whose interval location included a skilled nursing facility or rehabilitation center. Since similar and different PFGE types compose the whole set and are mutually exclusive, the tests of proportions occurring in these groups are identical.^dInfection reflects first invasive event after detection of colonization.