JOURNAL OF CLINICAL MICROBIOLOGY, Mar. 2005, p. 1415–1419 Vol. 43, No. 3
0095-1137/05/$08.00+0 doi:10.1128/JCM.43.3.1415–1419.2005

The *pls* Gene Found in Methicillin-Resistant *Staphylococcus aureus* Strains Is Common in Clinical Isolates of *Staphylococcus sciuri*

Katri Juuti,[1]* Salha Ibrahem,[2] Anni Virolainen-Julkunen,[2]
Jaana Vuopio-Varkila,[2,3,4] and Pentti Kuusela[3,4]

*General Microbiology, Faculty of Biosciences,[1] and Department of Bacteriology and Immunology,[3]
Haartman Institute, University of Helsinki, Division of Clinical Microbiology,
Helsinki University Central Hospital,[4] and Department of Microbiology,
National Public Health Institute,[2] Helsinki, Finland*

Received 12 October 2004/Accepted 17 November 2004

***pls*, a gene found in type I staphylococcal cassette chromosome *mec* (SCC*mec*) regions of methicillin-resistant *Staphylococcus aureus* strains, was present in 12 of the 15 human clinical *Staphylococcus sciuri* isolates studied. Pls was expressed in the *S. sciuri* isolates, although at a lower level than in *S. aureus*. Other parts of SCC*mec* could also be found in the *S. sciuri* genome.**

Methicillin-resistant *Staphylococcus aureus* (MRSA) is an increasing problem in hospital and community environments worldwide. Resistance towards methicillin is encoded by the *mecA* gene, carried by a mobile genetic element, staphylococcal cassette chromosome *mec* (SCC*mec*). Five types of SCC*mec* elements, containing slightly differing combinations of *mecA*, its regulators, and site-specific recombinase (*ccr*) genes, which enable a site-specific integration into the chromosome, as well as additional type-specific DNA, have so far been characterized (4, 11–13, 20). New MRSA strains are thought to emerge by acquisition of SCC*mec* by sensitive clones. The origin of *mecA*, however, has been suggested to be another staphylococcal species, possibly *Staphylococcus sciuri* or a close evolutionary relative, since a *mecA* homologue with 88% amino acid sequence similarity (32) is uniformly found in *S. sciuri* strains (2). *S. sciuri* is a taxonomically primitive staphylococcal species found on the skins of several animals as well as environmental sources (16, 17). It has occasionally been isolated from humans but has only rarely been associated with infections (6, 9, 21, 28–30).

pls is a part of the type I SCC*mec* element (11). Pls is a large surface protein with an LPXTG peptidoglycan-anchoring sequence and a so-far-uncharacterized carbohydrate-containing portion (7, 18, 27). Its expression results in reduced adhesiveness to host proteins (27) and decreased cellular invasiveness (14). On the other hand, it mediates bacterial aggregation and binding to glycolipids (10) and to human desquamated nasal epithelial cells (25). It acts as a virulence factor in a mouse model of septic arthritis and sepsis (13a).

The *pls* gene is common in *S. sciuri* isolates. Fifteen *S. sciuri* clinical human isolates of three subspecies, isolated from various sites (Table 1) (21), were studied for the presence of the *pls* and *mecA* genes by Southern hybridization. SmaI-digested DNA separated by pulsed-field gel electrophoresis (PFGE) (26, 27) was hybridized with a digoxigenin-labeled *pls* probe (Table 1) at 68°C and a *mecA* probe (Table 1) at 55°C to allow hybridization to both the *S. aureus* and *S. sciuri mec* genes. All the strains differed from each other by their PFGE patterns (Fig. 1A). *pls* was present in 12 out of 15 *S. sciuri* strains. Three strains, N900165, N900228, and S A8b, all of which are *S. sciuri* subsp. *rodentium*, lacked the gene (Fig. 1B). *mecA* hybridization resulted in a single weak band in 7 of 15 *S. sciuri* strains (Fig. 1C). All of these strains are oxacillin and methicillin sensitive (Table 1), and primers specific for *S. aureus mecA* are unable to amplify their DNA or the amplification is weak (21). Eight of 15 of the strains showed either a single strong band (4 of the 8 strains) or a strong and a weak band (4 of the 8 strains). Seven of these eight strains are oxacillin resistant, and their *S. aureus mecA* genes can be detected by PCR (21). A *mecA* probe has been shown to hybridize with a large and a smaller fragment in methicillin-resistant *S. sciuri* strains (2). Based on these data and the better homology of our probe with *S. aureus mec* than *S. sciuri mec*, we conclude that the strong bands represent *S. aureus mecA* and that the weak bands represent its *S. sciuri* homologue, which is slightly different in sequence. In 10 strains, a *mecA*-hybridizing fragment, usually the weak one, also hybridized with the *pls* probe (Fig. 1B and C). The sizes of these fragments were at least 400 kb, and consequently, it is difficult to estimate how near to each other the two genes are in the *S. sciuri* genome.

The *pls* genes of five *S. sciuri* strains (N960509, N960546, N970234, N970555, and S 29) were characterized in more detail by hybridization and PCR analyses and found to be similar but not identical to *S. aureus pls*. DNA of each strain, isolated as described previously for *S. aureus* (27) and digested with StuI and PstI, hybridized with enhanced-chemiluminescence-labeled probes for repeat regions R1, R2, and R3 and the nonrepeat region A of *S. aureus* strain 1061 Pls (27; results not shown). However, only the R2 region of all five strains and the A region of strain N970555 were amplifiable by PCR with the primers used to prepare these probes (27; data not shown).

Pls is expressed by *S. sciuri*. To study the expression of Pls in these 15 *S. sciuri* strains, surface proteins were solubilized by incubating stationary-phase cells with lysostaphin in the presence of raffinose essentially as described previously for *S. au-*

* Corresponding author. Mailing address: Faculty of Biosciences, General Microbiology, P.O. Box 56 (Viikinkaari 9), University of Helsinki, FIN-00014 Helsinki, Finland. Phone: 358-9-191 59246. Fax: 358-9-19159262. E-mail: katri.juuti@helsinki.fi.

TABLE 1. Probes used for dot blot and Southern hybridizations and results of the dot blot hybridization

Probe	Probe sequence[a]	Primer(s) used for probe amplification[b]	Template DNA in probe amplification[c]	SCC*mec* type(s) in which the primer sequence(s) is found[d]	Results of dot blot hybridization at 68°C of *S. sciuri* strains[e]: Oxa^r with *S. aureus mecA*$^{+f}$ N900109, *sc.*	N900165, *rod.*	N930262, *sc.*	S A8b, *rod.*	S 2832j, *sc.*	N960509, *car.*	S 29, *sc.*	With Oxa *S. aureus mecA*$^+$ N900228, *rod.*	*Oxa*^{s} without *S. aureus mecA* S 106j, *rod.*	N920212, *sc.*	N950120, *rod.*	N960546, *sc.*	N970234, *sc.*	N970555, *ear.*	N950282, *sc.*	1061, *S. aureus*	Controls[g]
A	Upstream of *pls* gene	Loci A to H	IA	I	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+	+/−
B	*kdp* operon	Loci A to H	II	II	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+/−
C	*mec*I	Loci A to H	II	II, III	+	−	+	+	+	+	+	−	−	−	−	−	−	−	−	−	+/−
D	*ORF* 5′ to *orfX*	Loci A to H	IA	I, II, IV	−	−	−	+	+	+	+	−	−	−	−	−	−	−	−	+	+/−
E	Between integrated pI258 and Tn*554*	Loci A to H	IIIA	III (not IIIB)	−	−	−	−	−	+	−	+	−	−	−	−	−	−	−	−	+/−
F	Between Tn*554* and *orfX*	Loci A to H	IIIA	III (not IIIB)	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+/−
G	Left junction between IS*431* and pUB110	Loci A to H	II	II, IA	+	+	+	+	+	+	+	+	+	−	−	+	−	+	+	+	+/+
H	Left junction between IS*431* and pT181	Loci A to H	III	III (not IIIA/B)	+	+	+	+	+	+	+	+	+	−	−	+	−	+	+	+	+/+
ccr1	Part of *ccrA1* and *-B*	β2	IA	I	−	−	−	−	−	−	−	−	+	−	−	+	−	−	+	+	+/−
ccr2	Part of *ccrA2* and *-B*	β2	II	II	+	+	+	−	−	−	−	+	+	−	−	+	−	−	+	+	+/−
ccr3	Part of *ccrA3* and *-B*	β2	IIIA	III	+	+	+	+	+	+	+	+	+	−	−	+	−	−	+	+	+/+
pls	Nonrepetitive A region of *pls*	27	1061	I	+	−	+	−	+	+	+	−	+	+	+	+	+	+	+	+	+/−
mecA	Part of *S. aureus mecA*	477 nt	1061	All types	+	−	+	+	+	+	+	−	−	−	−	−	−	−	−	+	+/−

[a] ORF, open reading frame.
[b] Loci A to H, primers to amplify loci A to H in a multiplex PCR (24); β2, primers β2, specific for the *ccrB* gene, used together with either primer α2, primer α3, or primer α4, specific for the *ccrA1, -2,* and *-3* genes, respectively (11); 27, primers reported in reference 27; 477 nt, primers 5′ AAGATGGCAAAGATATTCAAC3′ and 5′TTCTTACTGCCTAATTCGAG3′ to amplify an internal 477-nt region of *S. aureus mecA.*
[c] IA, Iberian; II, UK-EMRSA-16; IIIA, Brazilian; III, Helsinki IV; 1061, clinical MRSA isolate (7, 27).
[d] The strains of these types produce an amplification product in a multiplex PCR (24).
[e] *sc., S. sciuri* subsp. *sciuri; rod., S. sciuri* subsp. *rodentium; car., S. sciuri* subsp. *carnaticus.*
[f] Strains for which the MIC was >2 μg/ml were considered to be resistant to oxacillin (21).
[g] MRSA DNAs positive and negative for the primer sequences were used as the controls. The symbol to the left side of the slash shows the hybridization signal by the strain used as a positive control, and that to the right side of the slash shows the signal by a negative control.

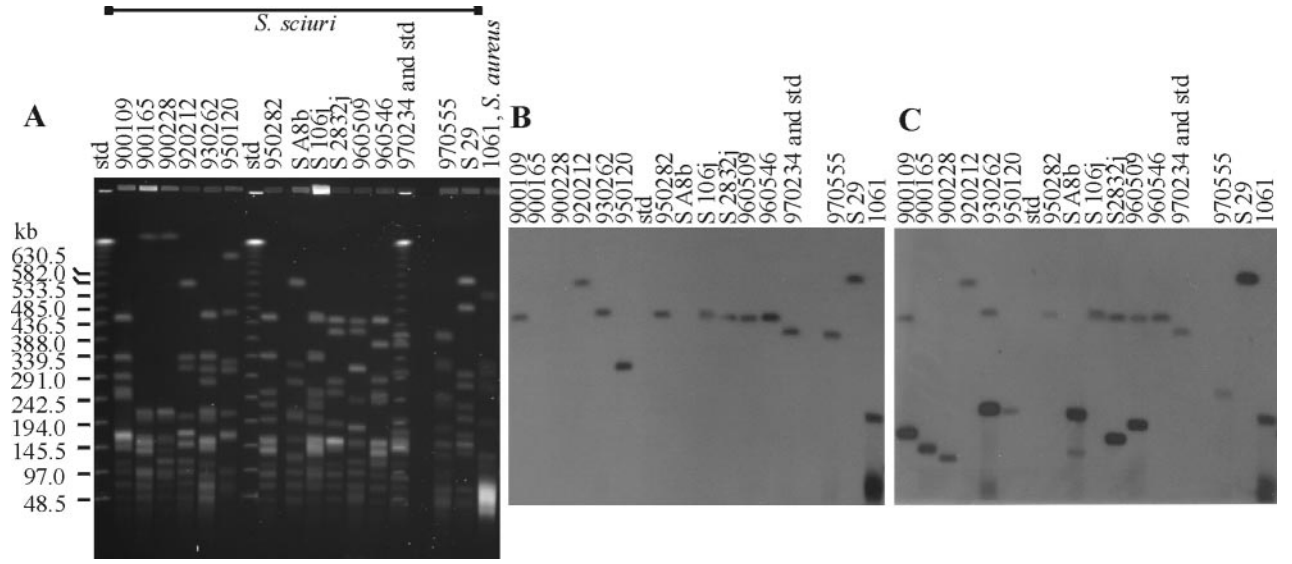

FIG. 1. SmaI-digested DNA of *S. sciuri* strains separated by PFGE (A) was capillary blotted onto a nylon membrane (Roche) and probed with the digoxigenin-labeled MRSA strain 1061 *pls* gene A region (B) and an internal region of MRSA *mecA* (C). The lambda ladder PFG (Biolabs) was used as a marker. Molecular size standards (std) were run in three lanes, one of which by mistake was combined with a sample lane (N970234). Marker sizes are shown on the left.

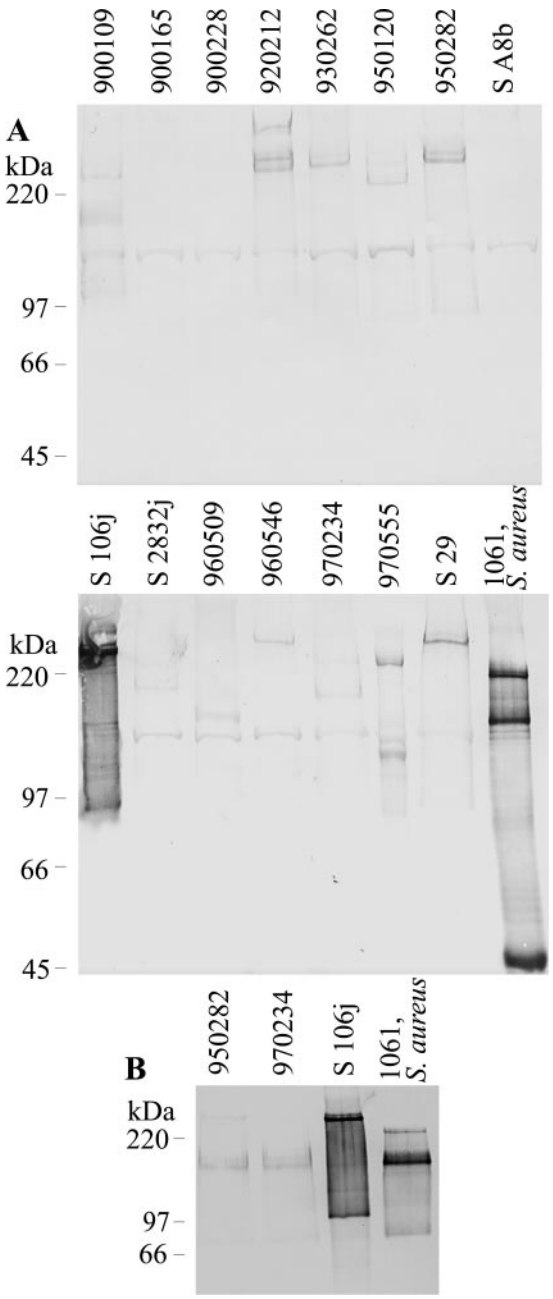

FIG. 2. Western analysis of lysostaphin-extracted surface proteins (A) or WGA-purified Pls proteins (B) of *S. sciuri* and *S. aureus* strains separated in a sodium dodecyl sulfate–8% polyacrylamide gel and blotted on 0.2-μm-pore-size nitrocellulose membranes (Protran). The membranes were blocked with 2% (wt/vol) bovine serum albumin–phosphate-buffered saline. Pooled mouse monoclonal anti-Pls antibodies (14) followed by alkaline phosphatase conjugated rabbit anti-mouse antibodies (Dako) and alkaline phosphatase substrate were used for detection. (A) The samples originate from 100 μl (40 μl for strain 1061) of a stationary-phase culture. (B) Twenty microliters of the peak fractions that eluted from strains N950282 and N970234 and 3 μl from strains S 106j and 1061 were used.

reus (1) and analyzed by Western blotting for Pls. Pls was expressed by all of the 12 *pls*$^+$ strains (Fig. 2A). However, based on both Coomassie staining (data not shown) and the Western analysis, the level of expression (except in one strain, S 106j) was clearly lower than in *S. aureus* strain 1061, whose level of Pls is typical of Pls$^+$ *S. aureus* strains studied (27). In most of the *S. sciuri* strains, Pls was even larger than the 230 kDa found for *S. aureus* strain 1061. In some cases more than one band was seen, possibly due to proteolytic cleavage, as was previously suggested for *S. aureus* 1061 Pls (7). A band of approximately 150 kDa was seen in all strains, including the ones without *pls* genes, and probably represents a cross-reactive protein.

Pls proteins of three *S. sciuri* strains and *S. aureus* strain 1061 were affinity purified from lysostaphin digests by using wheat germ agglutinin (WGA) Sepharose and elution with 100 mM *N*-acetylglucosamine (7) and then analyzed by Western blotting for Pls (Fig. 2B). Pls of *S. sciuri*, like that of *S. aureus*, bound to WGA and thus seems to have a carbohydrate moiety containing *N*-acetylglucosamine.

Analysis of the "SCC*mec* region" in *S. sciuri*. The presence of areas homologous to different types of *S. aureus* SCC*mec* regions in *S. sciuri* DNA was examined. Dot blot hybridization was used for all 15 strains, and additionally, Southern hybridization was used for strains N920212, N950120, and N960546. Thirteen digoxigenin-labeled probes designed to differentiate among type I to IV SCC*mec* regions were PCR amplified (Table 1). Four micrograms of total DNA was dot blotted, or SmaI-digested DNA separated by PFGE (5, 23) was capillary blotted, onto positively charged nylon membranes. The dot blot hybridizations were performed at 68°C (Table 1), and the Southern hybridizations were performed at 55°C (results not shown). To make sure that hybridization to *S. aureus* SCC*mec* regions was not examined, it was safest to look closely only at the *S. sciuri* strains not containing the *S. aureus mecA* gene. All the *S. sciuri* strains, even if positive for *pls*, gave negative results with probe A binding to a region upstream of *pls* in type I SCC*mec* regions. At 68°C, the strains without *S. aureus mecA* bound the *mecA* probe very weakly and not at all probe C, containing the *mecI* sequence. In a PCR analysis of 28 human *S. sciuri* isolates by Couto et al., the presence of *mecI* was always connected to the presence of a copy of *S. aureus mecA* (3). Similarly, *mecA* regions sequenced from four *S. sciuri* strains by Wu et al. revealed *mecR1* and *mecI* adjacent to *mecA* genes of the *S. aureus* type but not adjacent to those of the *S. sciuri* type (31). It seems that *mecI* is not a part of the native *S. sciuri mec* region. Probes G, H, and *ccr3* bound to negative-control DNA and were thus unable to differentiate between the SCC*mec* types. There were, however, three to four strains that did not bind to these probes at all, suggesting that IS*431* or plasmids pUB101 and pT181 sometimes but not always are a part of the *S. sciuri* genome. All the other strains except the four *S. sciuri* strains that did not bind to any of the *ccr* probes probably have some kind of site-specific recombinase genes. Probes B, D, and F did not bind to the DNA of any strains having only the *S. sciuri* copy of *mecA*, suggesting that these regions of SCC*mec* were not a part of the S. *sciuri* genome.

The Southern analysis (data not shown) of strain N920212 at a low stringency gave a positive signal with *pls*, *mecA*, and the C and A probes, all of which hybridized with the largest fragment. One fragment of strain N950120 DNA hybridized with *mecA* and probe C, and another fragment hybridized with *pls*. This strain was one of the few strains having *pls* and *mecA* in different SmaI fragments (Fig. 1). One fragment of strain

N960546 DNA hybridized with *pls*, *mecA*, and C, another one hybridized with the *ccr* probes and G, another one hybridized with the *ccr* probes and A, and yet another one hybridized with the H probe. Thus, sequences related to *mecI* and the upstream region of *pls* seem to exist in *S. sciuri*.

The idea that *pls*, like *mecA*, might originate from *S. sciuri* is supported by the much greater frequency of *pls* in *S. sciuri* strains than in *S. aureus* strains. The strains used in this study were human clinical isolates and may not represent the *S. sciuri* populations in their natural habitats. There is a possibility that *pls* was a part of an *S. aureus* SCC*mec* region in some of the *S. aureus mecA*$^+$ *S. sciuri* strains. Half of the strains, however, did not contain an *S. aureus mecA* gene. We do not know what the function of Pls in *S. sciuri* is, but the low level of expression suggests that the effects may be different from those in *S. aureus*.

Some of the SCC*mec* sequences were present in *S. sciuri* strains and even localized to the same chromosomal areas. Whether *mec* genes are a part of an SCC*mec* element also in species other than *S. aureus* is not known. SCC elements without *mecA* have recently been found in *S. aureus* as well as some other staphylococcal species, and they are thought to act as mobile genetic elements transferring any useful genetic information between species (8, 15, 19, 22). The presence of *pls* in *S. sciuri* as well as *S. aureus* is yet another piece of evidence that there is constant genetic exchange between staphylococcal species.

Jérôme Etienne is thanked for kindly providing the *S. sciuri* strains.

The study was financially supported by the Academy of Finland (projects 173854 and 1206356 to P.K. and K.J.) and The Paulo Foundation (a grant to K.J.)

REFERENCES

1. **Cheung, A. L., and V. A. Fischetti.** 1988. Variation in the expression of cell wall proteins of *Staphylococcus aureus* grown on solid and liquid media. Infect. Immun. **56:**1061–1065.
2. **Couto, I., H. de Lencastre, E. Severina, W. Kloos, J. A. Webster, R. J. Hubner, I. Santos Sanchez, and A. Tomasz.** 1996. Ubiquitous presence of a *mecA* homologue in natural isolates of *Staphylococcus sciuri*. Microb. Drug Resist. **2:**377–391.
3. **Couto, I., I. Santos Sanchez, R. Sá-Leão, and H. de Lencastre.** 2000. Molecular characterization of *Staphylococcus sciuri* strains isolated from humans. J. Clin. Microbiol. **38:**1136–1143.
4. **Daum, R. S., T. Ito, K. Hiramatsu, F. Hussain, K. Mongkolrattanothai, M. Jamklang, and S. Boyle-Vavra.** 2002. A novel methicillin-resistance cassette in community-acquired methicillin-resistant *Staphylococcus aureus* isolates of diverse genetic backgrounds. J. Infect. Dis. **186:**1344–1347.
5. **Goering, R. V., and M. A. Winters.** 1992. Rapid method for epidemiological evaluation of Gram-positive cocci by field inversion gel electrophoresis. J. Clin. Microbiol. **30:**577–580.
6. **Hedin, G., and M. Widerström.** 1998. Endocarditis due to *Staphylococcus sciuri*. Eur. J. Clin. Microbiol. Infect. Dis. **17:**673–675.
7. **Hildén, P., K. Savolainen, J. Tyynelä, M. Vuento, and P. Kuusela.** 1996. Purification and characterisation of a plasmin-sensitive surface protein of *Staphylococcus aureus*. Eur. J. Biochem. **236:**904–910.
8. **Holden, M. T. G., E. J. Feil, J. A. Lindsay, S. J. Peacock, N. P. J. Day, M. C. Enright, T. J. Foster, C. E. Moore, L. Hurst, R. Atkin, A. Barron, N. Bason, S. D. Bentley, C. Chillingworth, T. Chillingworth, C. Churcher, L. Clark, C. Corton, A. Cronin, J. Doggett, L. Dowd, T. Feltwell, Z. Hance, B. Harris, H. Hauser, S. Holroyd, K. Jagels, K. D. James, N. Lennard, A. Line, R. Mayes, S. Moule, K. Mungall, D. Ormond, M. A. Quail, E. Rabbinowitsch, K. Rutherford, M. Sanders, S. Sharp, M. Simmonds, K. Stevens, S. Whitehead, B. G. Barrell, B. G. Spratt, and J. Parkhill.** 2004. Complete genomes of two clinical *Staphylococcus aureus* strains: evidence for the rapid evolution of virulence and drug resistance. Proc. Natl. Acad. Sci. USA **101:**9786–9791.
9. **Horii, T., Y. Suzuki, T. Kimura, T. Kanno, and M. Maekawa.** 2001. Intravenous catheter-related septic shock caused by *Staphylococcus sciuri* and *Escherichia vulneris*. Scand. J. Infect. Dis. **33:**930–932.
10. **Huesca, M., R. Peralta, D. N. Sauder, A. E. Simor, and M. J. McGavin.** 2002. Adhesion and virulence properties of epidemic Canadian methicillin-resistant *Staphylococcus aureus* strain 1: identification of novel adhesion functions associated with plasmin-sensitive surface protein. J. Infect. Dis. **185:**1285–1296.
11. **Ito, T., Y. Katayama, K. Asada, N. Mori, K. Tsutsumimoto, C. Tiensasitorn, and K. Hiramatsu.** 2001. Structural comparison of three types of staphylococcal cassette chromosome *mec* integrated in the chromosome in methicillin-resistant *Staphylococcus aureus*. Antimicrob. Agents Chemother. **45:**1323–1336.
12. **Ito, T., Y. Katayama, and K. Hiramatsu.** 1999. Cloning and nucleotide sequence determination of the entire *mec* DNA of pre-methicillin-resistant *Staphylococcus aureus* N315. Antimicrob. Agents Chemother. **43:**1449–1458.
13. **Ito, T., X. X. Ma, F. Takeuchi, K. Okuma, H. Yuzawa, and K. Hiramatsu.** 2004. Novel type V staphylococcal cassette chromosome *mec* driven by a novel cassette chromosome recombinase, *ccrC*. Antimicrob. Agents Chemother. **48:**2637–2651.

13a. **Josefsson, E., K. Juuti, M. Bokarewa, and P. Kuusela.** Infect. Immun., in press.

14. **Juuti, K. M., B. Sinha, C. Werbick, G. Peters, and P. I. Kuusela.** 2004. Reduced adherence and host cell invasion by methicillin-resistant *Staphylococcus aureus* expressing the surface protein Pls. J. Infect. Dis. **189:**1574–1584.
15. **Katayama, Y., F. Takeuchi, T. Ito, X. X. Ma, Y. Ui-Mizutani, I. Kobayashi, and K. Hiramatsu.** 2003. Identification in methicillin-susceptible *Staphylococcus hominis* of an active primordial mobile genetic element for the staphylococcal cassette chromosome *mec* of methicillin-resistant *Staphylococcus aureus*. J. Bacteriol. **185:**2711–2722.
16. **Kloos, W. E., D. N. Ballard, J. A. Webster, R. J. Hubner, A. Tomasz, I. Couto, G. L. Sloan, H. P. Dehart, F. Fiedler, K. Schubert, H. de Lencastre, I. Santos Sanchez, H. E. Heath, P. A. Leblanc, and Å. Ljungh.** 1997. Ribotype delineation and description of *Staphylococcus sciuri* subspecies and their potential as reservoirs of methicillin resistance and staphylolytic enzyme genes. Int. J. Syst. Bacteriol. **47:**313–323.
17. **Kloos, W. E., K. H. Schleifer, and R. F. Smith.** 1976. Characterization of *Staphylococcus sciuri* sp. nov. and its subspecies. Int. J. Syst. Bacteriol. **26:**22–37.
18. **Kuusela, P., P. Hildén, K. Savolainen, M. Vuento, O. Lyytikäinen, and J. Vuopio-Varkila.** 1994. Rapid detection of methicillin-resistant *Staphylococcus aureus* strains not identified by slide agglutination tests. J. Clin. Microbiol. **32:**143–147.
19. **Luong, T. T., S. Ouyang, K. Bush, and C. Y. Lee.** 2002. Type 1 capsule genes of *Staphylococcus aureus* are carried in a staphylococcal cassette chromosome genetic element. J. Bacteriol. **184:**3623–3629.
20. **Ma, X. X., T. Ito, C. Tiensasitorn, M. Jamklang, P. Chongtrakool, S. Boyle-Vavra, R. S. Daum, and K. Hiramatsu.** 2002. Novel type of staphylococcal cassette chromosome *mec* identified in community-acquired methicillin-resistant *Staphylococcus aureus* strains. Antimicrob. Agents Chemother. **46:**1147–1152.
21. **Marsou, R., M. Bes, M. Boudouma, Y. Brun, H. Meugnier, J. Freney, F. Vandenesch, and J. Etienne.** 1999. Distribution of *Staphylococcus sciuri* subspecies among human clinical specimens, and profile of antibiotic resistance. Res. Microbiol. **150:**531–541.
22. **Mongolrattanothai, K., S. Boyle, T. V. Murphy, and R. S. Daum.** 2004. Novel non-*mecA*-containing staphylococcal chromosomal cassette composite island containing *pbp4* and *tagF* genes in a commensal staphylococcal species: a possible reservoir for antibiotic resistance islands in *Staphylococcus aureus*. Antimicrob. Agents Chemother. **48:**1823–1836.
23. **Murchan, S., M. E. Kaufmann, A. Deplano, R. de Ryck, M. Struelens, C. E. Zinn, V. Fussing, S. Salmenlinna, J. Vuopio-Varkila, N. El Solh, C. Cuny, W. Witte, P. T. Tassios, N. Legakis, W. van Leeuwen, A. van Belkum, A. Vindel, I. Laconcha, J. Garaizar, S. Haeggman, B. Olsson-Liljequist, U. Ransjo, G. Coombes, and B. Cookson.** 2003. Harmonization of pulsed-field gel electrophoresis protocols for epidemiological typing of strains of methicillin-resistant *Staphylococcus aureus*: a single approach developed by consensus in 10 European laboratories and its application for tracing the spread of related strains. J. Clin. Microbiol. **41:**1574–1585.
24. **Oliveira, D. C., and H. de Lencastre.** 2002. Multiplex PCR strategy for rapid identification of structural types and variants of the *mec* element in methicillin-resistant *Staphylococcus aureus*. Antimicrob. Agents Chemother. **46:**2155–2161.
25. **Roche, F. M., M. Meehan, and T. J. Foster.** 2003. The *Staphylococcus aureus* surface protein SasG and its homologues promote bacterial adherence to human desquamated nasal epithelial cells. Microbiology **149:**2759–2767.
26. **Salmenlinna, S., O. Lyytikäinen, P. Kotilainen, R. Scotford, E. Siren, and J. Vuopio-Varkila.** 2000. Molecular epidemiology of methicillin-resistant *Staphylococcus aureus* in Finland. Eur. J. Clin. Microbiol. Infect. Dis. **19:**101–107.
27. **Savolainen, K., L. Paulin, B. Westerlund-Wikström, T. J. Foster, T. K. Korhonen, and P. Kuusela.** 2001. Expression of *pls*, a gene closely associated with the *mecA* gene of methicillin-resistant *Staphylococcus aureus*, prevents bacterial adhesion in vitro. Infect. Immun. **69:**3013–3020.
28. **Shittu, A., J. Lin, D. Morrison, and D. Kolawole.** 2004. Isolation and mo-

lecular characterization of multiresistant *Staphylococcus sciuri* and *Staphylococcus haemolyticus* associated with skin and soft-tissue infections. J. Med. Microbiol. **53:**51–55.
29. **Stepanović, S., I. Dakić, S. Djukić, B. Lozuk, and M. Švabić-Vlahović.** 2002. Surgical wound infection associated with *Staphylococcus sciuri*. Scand. J. Infect. Dis. **34:**685–686.
30. **Tsakris, A., E. Papadimitriou, J. Douboyas, F. Stylianopoulou, and E. Manolis.** 2002. Emergence of vancomycin-intermediate *Staphylococcus aureus* and *S. sciuri*, Greece. Emerg. Infect. Dis. **8:**536–537.
31. **Wu, S. W., H. de Lencastre, and A. Tomasz.** 1998. Genetic organization of the *mecA* region in methicillin-susceptible and methicillin-resistant strains of *Staphylococcus sciuri*. J. Bacteriol. **180:**236–242.
32. **Wu, S. W., C. Piscitelli, H. de Lencastre, and A. Tomasz.** 1996. Tracking the evolutionary origin of the methicillin resistance gene: cloning and sequencing of a homologue of *mecA* from a methicillin susceptible strain of *Staphylococcus sciuri*. Microb. Drug Resist. **2:**435–441.