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Panton-Valentine Leukocidin-Positive and Toxic Shock Syndrome Toxin 1-Positive Methicillin-Resistant *Staphylococcus aureus*: a French Multicenter Prospective Study in 2008▽

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The epidemiology of community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) differs from country to country. We assess the features of the ST80 European clone, which is the most prevalent PVL-positive CA-MRSA clone in Europe, and the TSST-1 ST5 clone that was recently described in France. In 2008, all MRSA strains susceptible to fluoroquinolones and gentamicin and resistant to fusidic acid that were isolated in 104 French laboratories were characterized using *agr* alleles, *spa* typing, and the staphylococcal cassette chromosome *mec* element and PCR profiling of 21 toxin genes. Three phenotypes were defined: (i) kanamycin resistant, associated with the ST80 clone; (ii) kanamycin and tobramycin resistant, associated with the ST5 clone; and (iii) aminoglycoside susceptible, which was less frequently associated with the ST5 clone. Among the 7,253 MRSA strains isolated, 91 (1.3%) were ST80 CA-MRSA (89 phenotype 1) and 190 (2.6%) were ST5 CA-MRSA (146 phenotype 2, 42 phenotype 3). Compared to the latter, ST80 CA-MRSAs were more likely to be community acquired (80% versus 46%) and found in young patients (median age, 26.0 years versus 49.5 years) with deep cutaneous infections (48% versus 6%). They were less likely to be tetracycline susceptible (22% versus 85%) and to be isolated from respiratory infections (6% versus 27%). The TSST-1 ST5 clone has rapidly emerged in France and has become even more prevalent than the ST80 European clone, whose prevalence has remained stable. The epidemiological and clinical patterns of the two clones differ drastically. Given the low prevalence of both among all staphylococcal infections, no modification of antibiotic recommendations is required yet.

Community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infection was first recognized about a decade ago. Initially causing outbreaks in various well-defined populations, such as children, incarcerated persons, or sports teams, it now has spread worldwide (15). Several CA-MRSA clones have been identified throughout the world, mainly on the basis of molecular typing tools (15). They differ from the classical health care-associated (HA) MRSA in several important ways, including the following: (i) the size of the staphylococcal cassette chromosome *mec* element (SCC*mec*) containing the *mecA* gene, which is usually smaller and of type IV or V (7); (ii) some virulence factors that are frequently detected in these various clones, mainly Panton-Valentine leukocidin (PVL) and toxic shock syndrome toxin 1 (TSST-1) (20, 23); and (iii) specific susceptibility patterns with fewer resistances to antimicrobial drugs that could ease their recognition (16). Of interest, some authors have shown that phenotypic rules based on these specific antimicrobial resistance patterns are useful in routine tests for CA-MRSA strains and even for clone assignment (8, 10, 12, 16, 22).

The high prevalence of CA-MRSA in the United States is due mainly to the spread of the PVL-positive USA300 clone. Conversely, in Europe the prevalence remains low, except in Greece, and epidemics are due mainly to the European PVL-positive ST80 clone (15). In France, this clone emerged 10 years ago, but a new CA-MRSA clone containing the *tst* gene (encoding TSST-1) was discovered 6 years ago. It contains an SCC*mec* type I variant that is truncated for the *pls* gene, and it was named the ST5 Geraldine clone. The antimicrobial resistance patterns of these two clones are relatively stable. Both are susceptible to fluoroquinolones (FQ) (9, 19), whereas most nosocomial MRSA strains isolated in French hospitals are FQ resistant (2, 21). The European ST80 clone is resistant to kanamycin, tetracycline, and fusidic acid, and the ST5 Geraldine clone is resistant to fusidic acid and possibly to kanamycin and tobramycin (9, 23).

In 2004, an initial prospective study based on antimicrobial susceptibility patterns demonstrated that the European ST80 clone represented only 1.4% of clinical MRSA strains in France, confirming findings of a previous retrospective study (18, 19). The prevalence of the ST5 Geraldine clone currently is not known. To evaluate the recent epidemiology of these two

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CA-MRSA clones in France, we performed a large prospective study in 2008 in which the ST80 and ST5 clones were searched for by microbiological laboratories throughout France on the basis of their specific antibiotic susceptibility patterns. Corresponding strains then were sent to the French National Reference Center for Staphylococci (FNRCS) for molecular characterization using *agr* alleles, *spa* typing, and SCC*mec* and PCR profiling of 21 toxin genes.

MATERIALS AND METHODS

The study was promoted by ONERBA (National Observatory for Epidemiology of Bacterial Resistance to Antibiotics; http://www.onerba.org), a nonprofit organization established in 1997 and composed of 15 French surveillance networks for the surveillance of bacterial resistance to antibiotics in collaboration with the FNRCS. During a 6-month period in 2008, all laboratories participating in one of ONERBA's networks were asked to include all patients harboring a MRSA strain that was susceptible to fluoroquinolones and gentamicin and resistant to fusidic acid. In addition, MRSA strains had to match one of the following antibiotic susceptibility patterns: (i) resistant to kanamycin and susceptible to tobramycin (phenotype 1; considered typical for ST80 CA-MRSA in Europe); (ii) resistant to kanamycin and tobramycin (phenotype 2; considered to be frequently associated with the ST5 Geraldine clone); and (iii) susceptible to kanamycin and tobramycin (phenotype 3; less frequently associated with the ST5 Geraldine clone).

Only clinical specimens were considered, and duplicates were excluded. Susceptibility testing was performed in each participating laboratory by following guidelines from the French Society for Microbiology for antibiotic susceptibility testing (CA-SFM) (4). For each patient harboring one of the three defined MRSA patterns, basic demographic and clinical information were collected. In addition, each laboratory recorded the total number of patients harboring *S. aureus* that were susceptible or resistant to methicillin during the study period.

When available, the strains were sent to the FNRCS to be further characterized. The strains were screened for genes encoding methicillin resistance (*mecA*), staphylococcal enterotoxins A, B, C, D, H, K, L, M, O, P, Q, and R (*sea* to *sed*, *seh*, *sek* to *sem*, and *seo* to *ser*, respectively), toxic shock syndrome toxin 1 (*tst*), exfoliative toxins A, B, and D (*eta*, *etb*, and *etd*, respectively), Panton-Valentine leukocidin (PVL; *luk-PV*), class F LukM leukocidin (*lukM*), beta-hemolysin (*hlb*), and epidermal cell differentiation inhibitor (*edinA*, *edinB*, and *edinC*), as previously described (13, 22). Multiplex PCR amplification was used to determine the *agr* allelic group (13). *spa* typing was performed with the Ridom Staph Type standard protocol and using the Ridom SpaServer, which automatically analyzes *spa* repeats and assigns *spa* types (http://spa.ridom.de/index.shtml); the multilocus sequence type was inferred from the *spa* type according to the Ridom SpaServer (11). SCC*mec* types were determined by PCR with a simplified version of Kondo's typing system, including M-PCR-1 and M-PCR-2, without determining differences in the junkyard region (14).

MRSA strains with *pvl*, *etd*, and *edin* genes, as well as *agr* allele type 3, and that belonged to *spa*-CC t044 were classified as the European CA-MRSA ST80 clone. MRSA strains with *tst*, *sec*, *sel*, *sem*, *seo* toxin genes, *agr* allele type 2, and SCC*mec* type I and that belonged to *spa*-CC t002 were classified as the ST5 Geraldine clone.

Data were computerized using EpiData software and were analyzed using Stata 10 (Stata Corp, College Station, TX). Categorical variables were compared using the χ^2 test or the Fischer's exact test when appropriate, and the Mann-Whitney test was used for continuous variables. *P* values are two tailed, and $P < 0.05$ was considered statistically significant.

RESULTS

A total of 104 laboratories distributed throughout the French territory (including La Réunion Island) and Monaco participated in the 2008 study. There were 60 general hospital laboratories, 32 university hospitals, 7 army hospitals, and 5 private laboratories working for ambulatory patients and private hospitals.

A total of 34,970 patients harboring *S. aureus* in a clinical specimen were diagnosed, including 7,253 with MRSA (20.7%). Among the latter, 391 (5.4%) strains displayed one of the three predefined phenotypes, and 354 were sent to the FNRCS (37 strains were lost). Among those sent to the NRC, 14 belong to a species other than *S. aureus* and 7 were *mecA*-negative *S. aureus*. Finally, 333 MRSA strains were analyzed, including 93 (1.3% of all MRSA strains) displaying phenotype 1, 176 (2.4%) with phenotype 2, and 64 (0.9%) having phenotype 3.

TABLE 1. Microbiological characteristics of MRSA strains according to the three predefined phenotypes

Microbiological characteristic	Phenotype		
	1 (n = 93) (%)	2 (n = 176) (%)	3 (n = 64) (%)
Antibiotic susceptibility			
Fusidic acid	0	0	0
Fluoroquinolones	93 (100)	176 (100)	64 (100)
Gentamicin	93 (100)	176 (100)	64 (100)
Kanamycin	0	0	64 (100)
Tobramycin	93 (100)	0	64 (100)
agr allele type			
1	0	11 (6)	5 (8)
2	3 (3)	163 (93)	56 (88)
3	90 (97)	1 (1)	3 (5)
Protein encoded			
PVL	89 (96)	0	3 (5)
EDIN	90 (97)	0	4 (6)
ETD	90 (97)	0	2 (3)
TST	3 (3)	152 (86)	47 (73)
SEA	0	13 (7)	7 (11)
SEC	3 (3)	152 (86)	47 (73)
SED	3 (3)	143 (81)	44 (69)
SEL	3 (3)	152 (86)	47 (73)
SEM	3 (3)	167 (89)	56 (88)
SEO	3 (3)	167 (89)	56 (88)
SEP	0	4 (2)	2 (3)
SEQ	0	0	3 (5)
SER	3 (3)	141 (80)	44 (69)
ETA, ETB, LUKM	0	0	0
HLB	1 (1)	14 (8)	8 (13)
spa type			
Clonal complex 2	3 (3)	153 (87)	50 (78)
Clonal complex 44	89 (96)	2 (1)	2 (3)
Other *spa* types	0	20 (11)	12 (19)
Indeterminate	1 (1)	1 (1)	0

Phenotypic analysis. A total of 90 of the 93 phenotype 1 MRSA (97%) strains had *agr* allele type 3, including 89 *pvl*$^+$, *etd*$^+$, and *edin*$^+$ strains (Table 1). One of the *pvl*-positive strains also was *hlb* positive. The most frequent *spa* type among the 89 *pvl*-positive *agr* allele type 3 MRSA strains was *spa* t044 (n = 77; 87%). Among the 12 remaining strains, 11 presented *spa* types belonging to the same *spa*-CC as *spa* t044 but differed from the main *spa* type by one (*spa* t042, *spa* t131, *spa* t376, and *spa* t4725) or two repeats (*spa* t639 and *spa* t5088). The last strain had an indeterminate *spa* type, although it shared all other microbiological characteristics of the ST80 European clone, and MLST confirmed (data not shown) that it belonged to ST80. Therefore, a total of 89 of the 93 phenotype 1 MRSA belonged definitively to the ST80 CA-MRSA European clone. Moreover, one strain shared all of the other features of the ST80 clone (*agr* allele type 3, *etd*$^+$, *edin*$^+$, and *spa* t376) except for the absence of *pvl*, which likely was due to the excision of

the phage harboring *pvl*. Because the latter strain did not strictly match the ST80 European clone definition, it was not further considered to belong to this clone. The three remaining phenotype 1 MRSA strains were *tst* positive, harbored *sec*, *sel*, *sem*, and *seo* genes, had *agr* allele type 2, and belonged to *spa* t002. In addition, two of the latter were SCC*mec* type I and therefore were considered to belong to the ST5 Geraldine clone. The third remaining strain was SCC*mec* type IV.

Of the 176 phenotype 2 MRSA strains, 163 (92.6%) had *agr* allele type 2, including 151 *tst*-positive strains harboring the *sec*, *sel*, *sem*, and *seo* toxin genes. Among these 151 *tst*-positive strains, 145 had SCC*mec* type I, and one strain had a variant of SCC*mec* type I that included an additional *ccrC* recombinase gene. All 146 SCC*mec* type I MRSA strains had *spa* types belonging to the same *spa*-CC: 113 (77.4%) *spa* type 2, 19 (13.0%) differing from *spa* t002 by one repeat (*spa* t010, *spa* t067, *spa* t088, *spa* t105, *spa* t242, *spa* t269, *spa* t306, *spa* t447, *spa* t856, *spa* t3230, and *spa* t5046), 6 (4.1%) differing by two repeats (*spa* t509, *spa* t1300, *spa* t1683, and *spa* t1815), and 8 (5.5%) differing by more than two repeats (*spa* t045, *spa* t062, *spa* t688, *spa* t1340, *spa* t3152, *spa* t4971, and *spa* t5094). Therefore, 146 of the 176 (83.0%) phenotype 2 MRSA strains matched the characteristics of the ST5 Geraldine clone. Among the five remaining *tst*-positive strains, two had SCC*mec* type II, two SCC*mec* type IV, and one had a nontypeable SCC*mec* element. Of the 12 *agr* allele type 2 *tst*-negative strains, 11 harbored *sed*, *sem*, *seo*, and *ser* toxin genes and one harbored only *sem* and *seo* toxin genes. Finally, 11 of the 176 phenotype 2 MRSA strains had *agr* allele type 1 and were *tst* and *pvl* negative, one had *agr* allele type 3 and was *tst* and *pvl* negative, and one had a nontypeable *agr* allele and was *tst* positive and *pvl* negative.

Among the 64 phenotype 3 MRSA strains, 56 (87.5%) had *agr* allele type 2, including 47 *tst*-positive strains harboring the *sec*, *sel*, *sem*, and *seo* toxin genes. Of the 47 *tst*-positive strains, 41 had SCC*mec* type I and 2 had a variant of SCC*mec* type I, including an additional *ccrC* recombinase gene. All 42 SCC*mec* type I MRSA strains had *spa* types belonging to the same *spa*-CC: 31 isolates (73.8%) with *spa* t002, 7 (16.7%) with spa types differing from *spa* t002 by one repeat (*spa* t010, *spa* t105, *spa* t242, *spa* t447, *spa* t2487, and *spa* t5037), 1 with a *spa* type differing by two repeats (*spa* t1815), and 3 with *spa* types differing by more than two repeats (*spa* t045 and *spa* t4909). Therefore, 42 of the 64 (65.6%) phenotype 3 MRSA strains matched the features of the ST5 Geraldine clone. The five remaining *tst*-positive strains had SCC*mec* type IV. Among the nine *agr* allele type 2 *tst*-negative strains, six harbored *sem* and *seo* toxin genes and three had *sem*, *seo*, *sed*, and *ser* toxin genes. Finally, five of the strains were *agr* allele type 1 and three were *agr* allele type 3. Among the latter, two were *pvl* positive and had *spa* t044, therefore belonging to the ST80 clone.

ST80 European clone. The 91 patients with an ST80 strain (phenotype 1, $n = 89$; phenotype 3, $n = 2$) had a median age of 27 (range, 0.1 to 83) years and were male in 53% of the cases. The ST80 clone represented 0.26% of all *S. aureus* and 1.25% of all MRSA strains found. ST80 MRSA-positive samples were drawn in the outpatient clinic or within 2 days following admission in 82 (91%) cases. However, in 7 of these 82 cases, infection was considered hospital acquired by the physician in charge due to associated risk factors; in 6 additional cases, the origin of acquisition was doubtful, resulting in at least 73 (80%) of the strains being community acquired. Only six (7%) patients had recognized links with other patients harboring similar strains. Most patients had either deep (48%) or superficial (34%) cutaneous infections. Less than half (45%) of the patients were treated with antibiotics, and 40% had surgery. Details of resistance profiles and antibiotic treatment are presented in Table 2.

ST5 Geraldine clone. Strains belonging to the ST5 Geraldine clone were distributed into phenotype 2 ($n = 146$), phenotype 3 ($n = 42$), and phenotype 1 ($n = 2$). No statistical difference was observed between microbiological features of strains or between patient characteristics according to the phenotype (Table 2). Therefore, the three populations were merged for further analysis (Table 2).

The 190 patients had a median age of 49.5 (range, 0.1 to 96) years and were male in 49% of the cases. This clone represented 0.54% of all *S. aureus* and 2.61% of all MRSA strains isolated. A total of 135 (71%) of the strains were drawn within the first 48 h following admission; the strain was considered hospital acquired by the physician in charge in 45 of these 135 cases, and the origin of the strain was doubtful for 12 cases. Therefore, only 87 (46%) of the cases were considered community acquired. A majority of patients (37%) had superficial cutaneous infections, while only 6% had deep cutaneous infections. Among other patients, 51 (27%) had respiratory infections, 12 (6%) had urinary tract infections, and 33 (17%) displayed other types of infections. Toxic shock syndrome was reported for 19 (10%) patients, but only 3 of the 19 patients had clinical signs matching the CDC definition of TSS (http://www.cdc.gov/ncphi/disss/nndss/casedef/toxicsscurrent.htm). Details of resistance profiles and antibiotic treatment are presented in Table 2.

Comparison of patient populations. Compared to patients harboring a strain belonging to the ST5 Geraldine clone, those harboring a strain belonging to the ST80 European clone were younger (27.0 versus 49.5; $P < 0.001$). They also were more likely to have deep cutaneous infections (48% versus 6%; $P < 0.001$), a community-acquired infection (80% versus 46%; $P < 0.001$), and a recognized contact case (7% versus 1%; $P = 0.02$), to have received a beta-lactam-containing regimen (16% versus 11%; $P = 0.03$), and to have benefited from surgical treatment (42% versus 11%; $P < 0.001$) (Table 2). On the other hand, they were less likely to have bloodstream (0% versus 6%; $P = 0.01$), respiratory (8% versus 27%; $P < 0.001$), or urinary tract infections (0% versus 6%; $P = 0.01$) and to have a strain susceptible to tetracycline (22% versus 85%; $P < 0.001$).

DISCUSSION

The principal epidemiological characteristics of two CA-MRSA clones, the ST80 European clone and the ST5 Geraldine clone, are reported for France using a screening method based on typical antimicrobial susceptibility patterns that subsequently was complemented by molecular analysis. The participation of 104 laboratories throughout France during a 6-month period, with the screening of 34,970 *S. aureus* strains, including 7,253 MRSA strains, gives a national perspective to the results. We highlight that (i) the use of specific antimicro-

TABLE 2. Characteristics of patients harboring defined clones of toxin-producing MRSA

Characteristic	ST80 European clone (n = 91)	ST5 Geraldine clone			P^b
		Phenotype 2 (n = 146)	Phenotype 3 (n = 42)	Total (n = 190[c])	
Proportion (%)					
Among *S. aureus* strains (n = 34,970)	0.26	0.42	0.12	0.54	<.001
Among MRSA strains (n = 7,253)	1.25	2.01	0.58	2.61	<.001
Days in hospital before sampling, median (10p-90p[a])	0 (0–2)	0 (0–21)	1 (0–12)	0 (0–19)	0.001
Age, median (10p-90p)	26.0 (2–64)	48.5 (3–94)	53.5 (1–84)	49.5 (2–84)	<.001
Male gender	48 (53%)	68 (47%)	24 (57%)	94 (49%)	0.61
Type of sample					
Deep cutaneous	44 (48%)	8 (5%)	3 (7%)	11 (6%)	<.001
Superficial	31 (34%)	54 (37%)	17 (41%)	71 (37%)	0.69
Bloodstream infection		9 (6%)	3 (7%)	12 (6%)	0.01
Respiratory	7 (8%)	39 (27%)	10 (24%)	51 (27%)	<.001
Urine		11 (8%)	1 (2%)	12 (6%)	0.01
Other	9 (10%)	25 (17%)	8 (19%)	33 (17%)	0.11
Toxic shock syndrome[d]	1 (1%)	13 (9%)	6 (14%)	19 (10%)	0.007
Community acquired	73 (80%)	65 (45%)	21 (50%)	87 (46%)	<.001
Linked to another case	6 (7%)	2 (1%)	0	2 (1%)	0.01
Antibiotic treatment	41 (45%)	85 (62%)	20 (48%)	107 (56%)	0.06
Surgical treatment	36 (40%)	16 (11%)	5 (12%)	21 (11%)	<.001
Associated susceptibility of strains					
Erythromycin	64 (70%)	105 (72%)	33 (79%)	140 (74%)	0.57
Tetracycline	18 (22%)	121 (83%)	38 (90%)	161 (85%)	<.001
Rifampin	91 (100%)	136 (93%)	41 (98%)	179 (94%)	0.02

[a] 10p-90p, 10th and 90th percentiles.
[b] P values after comparison between ST80 and total ST5 patients.
[c] Two strains harbored phenotype 1 (see Results).
[d] As reported by physicians, even if the classification was incomplete according to CDC criteria (http://www.cdc.gov/ncphi/disss/nndss/casedef/toxicsscurrent.htm).

bial resistance patterns allowed for the efficient screening of the two clones; (ii) the ST5 Geraldine clone (0.54% of *S. aureus*, 2.61% of MRSA) is more prevalent than the ST80 European clone (0.26% of *S. aureus*, 1.26% of MRSA), with the presence of the latter clone being stable since 2004; and (iii) the two CA-MRSA clones have highly different epidemiological features.

As suggested by others (10, 16, 22), phenotypic rules based on antimicrobial resistance patterns are useful tools for suspecting *pvl*- and *tst*-positive MRSA strains in France: phenotype 1 was highly predictive of the ST80 European clone (97%), whereas phenotype 2 and phenotype 3 were highly predictive of the ST5 Geraldine clone (83 and 66%, respectively). This is of major importance, because no method, except for specific PCR, currently is available to easily obtain information about the toxins present in *S. aureus* strains. Nevertheless, CA-MRSA clones have revealed worrisome and gradually increasing multidrug resistance (1, 8), indicating that genetic drift is associated with a wide dissemination of these clones observed in the community and/or in hospitals. Such evolution demonstrates that the susceptibility pattern, although currently very helpful, must be regarded with caution when used as a single tool for CA-MRSA surveillance over time. In any case, phenotypic rules must be periodically reassessed to be accurate.

The large scale of the present study gave us the opportunity to clearly define and compare microbiological and clinical features of these two CA-MRSA clones that differ drastically. Surprisingly, it appears that, among CA-MRSA, the circulation of the Geraldine clone in France is higher than that of the ST80 European clone. This clone was reported for the first time in France in 2003 and was involved in neonatal toxic shock-like exanthematous disease (NTED) (9). According to the database of the FNRCS, this clone has been present in France since at least 2000 (one strain; unpublished data). Since then, it has been described as an emerging CA-MRSA clone in France (5, 9, 17). Two susceptibility patterns, probably related by acquisition or the loss of a mobile genetic element harboring the 4′-4″-aminoglycoside nucleotidyltransferase gene (ant4′) responsible for kanamycin and tobramycin resistance, are spreading in France. No statistical difference regarding epidemiological, clinical, or microbiological characteristics was observed between strains and patients harboring the two phenotypes, suggesting that they represent only resistance phenotype variants. The second CA-MRSA clone, namely, the ST80 European clone, initially considered to be the European counterpart of the USA300 clone, has not had the same epidemiological success. First described in France in 1999, its prevalence remains low (1.26% of all MRSA strains) and stable (on the basis of data from a study performed in 2004 with the same design as the present one; 1.4% of all MRSA strains [18]). Conversely, the PVL-positive USA300 clone has spread rapidly in the U.S. community since 2000. The genetic background of the two clones is different and could explain the epidemiological differences observed between the two PVL-positive clones. In fact, the reason for this favorable but unexpected

stagnation of the ST80 European clone in France is unclear. Indeed, the ST80 clone has spread widely throughout Greece, Tunisia, and Algeria, and there is a high flow of travel and migrants toward France from the latter two countries. In these countries, more than 50% of staphylococcal infections in the community and the hospital are due to *pvl*-positive ST80 MRSA (1) (F. Laurent, personal data).

The present study highlighted many differences between the two CA-MRSA clones. Infections due to the ST5 Geraldine clone (i) were acquired equally in the community and in the hospital, whereas those due to the ST80 European clone were mainly community acquired (80%), and(ii) involved an older population than that affected by the ST80 European clone. The ST80 European clone was associated with a limited range of cutaneous infections and, to a lesser extent, with pulmonary infections. The sites involved are in agreement with the reported literature and match the polynuclear cytolytic and/or apoptotic activity of PVL (3, 6). Conversely, the ST5 Geraldine clone was associated with a wider variety of clinical manifestations, including a few cases of toxic shock syndrome. This large panel of infections and the low proportion of toxic shock syndrome cannot be directly related to the superantigenic activity of TSST-1. However, it suggests that TSST-1 is not a major virulence determinant but is mainly a highly linked epidemiological marker for the ST5 Geraldine clone. The diversity of infection sites may be linked to the fact that a large proportion of infections due to the ST5 Geraldine clone were hospital acquired. According to the low prevalence of ST80 and the Geraldine MRSA clone, no modification of antibiotic recommendations for staphylococcal disease is required yet.

The present study demonstrated the stability of the prevalence of the ST80 European clone in France and the rapid spread of the ST5 Geraldine clone. On the basis of the number of strains in the present study and the respective prevalence of community-acquired and hospital-acquired strains, one can estimate that the number of strains of the two CA-MRSA clones circulating in the community is roughly the same. These results contrast with those from some other European countries, where the ST80 European clone is more prevalent and the ST5 Geraldine clone has not been reported. The increase in reports of local and large-scale outbreaks over the past few years stresses the need for the surveillance of CA-MRSA throughout the world. Additional studies with different methodological approaches may be of interest to confirm our data. For instance, community-based surveys of CA-MRSA infections with active case findings may be of interest to confirm the level of circulation of these CA-MRSA clones in France.

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