

## *Staphylococcus lugdunensis* Infections: High Frequency of Inguinal Area Carriage

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Following a change in surgical practice, we noted that the rate at which *Staphylococcus lugdunensis* was isolated from samples from the plastic surgery unit of our hospital increased considerably. We investigated the sources of these *S. lugdunensis* strains, and we found that in the case of drain colonization or surgical site infection, the strain was more likely to have come from the patient's skin bacteria when the pubic site had been shaved preoperatively. To test the hypothesis of pubic site colonization, we evaluated the prevalence of *S. lugdunensis* carriage among the cutaneous flora of the inguinal area. We found that 22% of 140 incoming patients carried *S. lugdunensis* in this area and that carriage at both inguinal folds was frequent (68% of carriers). A study of the genetic structure of the total population, including the clinical ( $n = 18$ ) and the commensal ( $n = 53$ ) strains, revealed that the diversity of the species was low and that the population was composed of two major groups that diverged at a distance of 35%. No particular characteristics made it possible to distinguish between clinical and commensal strains. Only isolates producing  $\beta$ -lactamase were homogeneous; six of the eight  $\beta$ -lactamase-positive strains displayed the same pulsed-field gel electrophoresis pattern.

*Staphylococcus lugdunensis* is a coagulase-negative *Staphylococcus* (CoNS) that was first described by Freney et al. (9) in 1988 and that has the potential to be an opportunistic pathogen. *S. lugdunensis* is an unusually virulent CoNS and can cause many types of infection, ranging from superficial skin infections to life-threatening endocarditis. Most laboratory isolates are collected from colonized patients or patients with primary skin infections or minor postoperative wound infections. Nevertheless, *S. lugdunensis* has been shown to be associated with serious infections such as breast abscesses (18, 34), peritonitis (19, 28), infected joint prostheses (26, 35), osteomyelitis (22), discitis (2), septic arthritis (12), and pacemaker infections (1, 3, 16). Unlike *S. epidermidis*, which usually results in indolent subacute infections, *S. lugdunensis* results in acute infections, similar to *S. aureus*. *S. lugdunensis* infections typically resemble *S. aureus* infections in terms of the virulence of the organism and the clinical course of infection, which is often highly destructive (28, 31, 33).

*S. lugdunensis* can act as an etiologic agent of infective endocarditis. It may infect both prosthetic and native valves (31). Patel et al. (24) found that *S. lugdunensis* accounted for 18% of CoNS strains causing infective endocarditis and 44% of CoNS strains causing native valve endocarditis. The mortality rate as a result of endocarditis caused by *S. lugdunensis* is high (7, 15, 31). Few studies have looked at the epidemiology and ecology of *S. lugdunensis*. Similar to other CoNS strains, *S. lugdunensis* is considered part of the resident flora of the entire surface of the human skin and mucous membranes (11). No detailed studies have been carried out on the distribution of *S. lug-*

*dunensis* on the human body. Skin infections due to *S. lugdunensis* occur at a number of different sites, although the lower limbs are most commonly involved (38%) (11, 32). Overall, most (73%) of the infections involved sites below rather than above the waist (27%). These results are consistent with the suggestion that the preferred site of *S. lugdunensis* carriage may be the perineum rather than the entire skin surface. Associations have been reported between *S. lugdunensis* endocarditis and inguinal skin breaks occurring in the context of vasectomy (7), scrotal wounds (24), renal transplantation (24), continuous ambulatory peritoneal dialysis (14), femoral arterial catheterization, total-knee arthroplasty (24), prostatic cancer (8), and inguinal furuncle (17, 25). Several cases of ventriculoperitoneal shunt infection caused by *S. lugdunensis* have also been reported (5, 27). All these reports are consistent with a perineal, pelvic, or inguinal cutaneous source of *S. lugdunensis*. We decided to investigate this hypothesis, as it could explain the reason for *S. lugdunensis* infections following invasive procedures in the perineal, pelvic, or inguinal area.

Following a change in surgical practice, the microbiology laboratory of our hospital noted that the rate at which *S. lugdunensis* was isolated from samples from the plastic surgery unit of our hospital increased considerably. To investigate the sources of these *S. lugdunensis* strains, we conducted a case-control study to identify the risk factors for *S. lugdunensis* infection in the patients studied and used molecular typing methods to determine whether the strains isolated were epidemiologically linked. In a second step, with the aim of testing the hypothesis of pubic site colonization, we evaluated the prevalence of *S. lugdunensis* carriage as part of the inguinal cutaneous flora of 140 incoming patients. In a third step, we studied the genetic structure of the total population including the clinical ( $n = 18$ ) and the commensal ( $n = 53$ ) strains to

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identify characteristics that may make it possible to distinguish between the two groups of strains.

#### MATERIALS AND METHODS

**Plastic surgery unit investigation.** Trousseau Hospital is a 600-bed teaching hospital located in Tours, France. The plastic surgery unit has 30 beds.

(i) **Bacteriology.** Surgical sites are routinely monitored for signs of infection, and wounds are sampled with sterile cotton swabs whenever infection is suspected. Surgical drains are systematically cultured after removal by the technique described by Maki et al. (20). All specimens are cultured on sheep blood (Trypticase soy agar with 5% sheep blood; bioMérieux, Marcy l'Etoile, France). The plates were incubated for 48 h at 35°C in 5 to 7% CO<sub>2</sub>. The bacteria were identified as *S. lugdunensis* on the basis of their characteristic growth pattern, colony morphology, Gram staining characteristics, catalase test results, coagulase production (rabbit plasma; Difco Laboratories, Elancourt, France), and the results of the slide test for clumping factor (Pastorex Plus-Staph; Bio-Rad, Marnes-la-coquette, France) and by using ID 32 STAPH strips, which are part of a system for the identification of the genera *Staphylococcus*, *Micrococcus*, *Stomatococcus*, and *Aerococcus*. The procedures recommended by the manufacturer (API system; bioMérieux) were followed. Eighteen *S. lugdunensis* isolates were recovered from 11 patients with superficial incisional surgical site infections that had occurred in the plastic surgery unit over 4 months.

(ii) **Case-control study.** A retrospective case-control study was carried out to determine the risk factors for *S. lugdunensis* infection and to identify possible sources of surgical site infection or drain colonization. A case was defined as a person for whom *S. lugdunensis* was isolated from specimens taken after surgery in the plastic surgery unit. Two control patients were enrolled for each case patient. Each control patient underwent surgery during the same week as the case patient. Thus, the study included 11 case patients and 22 noninfected, colonized patients. The variables studied included (i) patient characteristics such as age, sex, and weight; (ii) preoperative management; (iii) operators (7 surgeons [surgeons S1 to S7], 7 instrument or dresser nurses); (iv) perioperative antibiotic treatment; (v) drainage tubes (number of drains, volume of fluid that drained, duration of tube placement); (vi) postoperative care (12 different nurses); and (vii) duration of hospital stay. Data were analyzed with Epi Info (version 6) software by comparing case patients with control patients. Univariate analysis was conducted, and odd ratios were calculated. The significance of differences was tested by statistical tests.

**Inguinal area carriage study.** Specimens for culture (e.g., swabs of the left and right inguinal folds) were collected from 140 consecutive patients evaluated at the accident and emergency unit of our hospital over a 3-month period. The samples taken from the inguinal folds were cultured for *S. lugdunensis* on sheep blood (Trypticase soy agar with 5% sheep blood; bioMérieux). The plates were incubated for 48 h at 35°C in 5 to 7% CO<sub>2</sub>. Laboratory technicians subcultured any colonies that resembled *S. lugdunensis*. Isolates from subcultures were identified as described above.

**Microbial typing.** *S. lugdunensis* reference strain ATCC 103642<sup>T</sup> was included in the microbial typing study.

(i) **Antimicrobial susceptibility testing.** Antimicrobial susceptibility testing was performed with ATB STAPH strips according to the recommendations of the manufacturer (bioMérieux). The antibiotics tested were penicillin G, oxacillin, erythromycin, lincomycin, pristinamycin, tetracycline, kanamycin, tobramycin, gentamicin, rifampin, fusidic acid, fosfomycin, pefloxacin, co-trimoxazole, vancomycin, and teicoplanin. The cefinase test (bioMérieux) was used to detect β-lactamase production.

(ii) **DNA macrorestriction and PFGE.** Genomic DNA was extracted from the isolates and digested with *Sma*I before being subjected to pulsed-field gel electrophoresis (PFGE) as described previously for *S. aureus* (4). Gels were stained with ethidium bromide and photographed with instant Polaroid camera equipment. The PFGE patterns were compared visually and interpreted according to the guidelines of Tenover et al. (29). Patterns were considered indistinguishable if all bands were shared. Strains were considered to belong to the same pulsotype if they differed by a maximum of one band. Each pulsotype is designated by a roman numeral. The subtypes of a single pulsotype (e.g., pulsotype X) are designated with a suffix (e.g., subtypes X-1 and X-2). Strains that differed by more than one band were considered different pulsotypes.

(iii) **Numerical pattern analysis.** Numerical pattern analysis was conducted by using the Taxotron package (Taxolab; Institut Pasteur, Paris, France). The images were transferred to a microcomputer, and the distances that each band migrated in each lane were determined in pixel units with RestrictoScan software. The molecular size of each fragment was calculated from the distance migrated by using cubic s&s algorithms with RestrictoTyper software. This soft-

ware also generates a schematic representation of the electrophoretic patterns and produces a distance matrix. The relationships between pulsotypes were calculated by the unweighted pair group method with arithmetic means with the Adanson clustering program (dissimilarity). A dendrogram was drawn with Dendrograf software.

(iv) **DI.** The discrimination index (DI) of PFGE was calculated by applying Simpson's numerical index of diversity (13).

#### RESULTS

***S. lugdunensis* infection.** (i) **Clinical data.** Eighteen *S. lugdunensis* isolates were recovered from 11 patients (patients P1 to P11). The mean and median age of the patients was 43 years. Eight of these patients underwent abdominoplasty, two underwent mammoplasty and one underwent inguinal ganglion excision. The isolates were obtained from an abscess ( $n = 1$ ) or from purulent drainage from the affected site ( $n = 10$ ). A single isolate was obtained from each of six patients, two isolates were obtained from each of three patients, and three isolates were obtained from each of two patients. Nosocomial acquisition was confirmed by the definitions of the Centers for Disease Control and Prevention.

(ii) **Case-control study.** The data yielded only one risk factor with an odds ratio greater than 4 for arbitrarily selected risk thresholds, i.e., two members of the operating room staff (surgeons S1 and S4). Eight of the 11 patients were treated by at least one of these two surgeons. The three other patients (patients P8, P10, and P11) were exposed to other surgeons. None of the other risk factors differed between case patients and control patients and between surgeons. Interviews with medical staff revealed that surgeons S1 and S4 had recently modified the surgical technique used for abdominoplasty. The incision site chosen by surgeons S1 and S4 was lower on the abdomen than that used by the other surgeons. For esthetic reasons, their incision sites were totally concealed in the pubic area, which was shaved preoperatively. Furthermore, patients were shaved the day before the operation. The incision made by the other surgeons was above the pubic hairline in a hair-free area, thus requiring no prior shaving.

(iii) **Determination of antibiotic resistance patterns.** None of the 18 isolates studied were oxacillin resistant; 12 (67%) strains isolated from nine patients were sensitive to all of the antibiotics tested. The three isolates recovered from patient P5 were resistant to penicillin (17%), and the three isolates recovered from patient P4 were resistant to both penicillin and erythromycin (17%).

(iv) **Pulsotyping.** According to the criteria of Tenover et al. (29), seven different pulsotypes were obtained for the 18 *S. lugdunensis* strains (Fig. 1). In all cases, when multiple isolates were taken from a given patient, they all shared the same pulsotype. Four pulsotypes clustered strains from different patients: strains from patients P1 and P3 were of pulsotype I, strains from patients P4 and P5 were of pulsotype IV, strains from patients P8 and P10 were of pulsotype VI, and strains from patients P9 and P11 were of pulsotype VII.

***S. lugdunensis* inguinal area carriage.** The origin of drain colonization and surgical site infection with *S. lugdunensis* was probably the patient's skin bacteria when the pubic site had been shaved preoperatively. To test the hypothesis of pubic site colonization, we evaluated the prevalence of *S. lugdunensis*

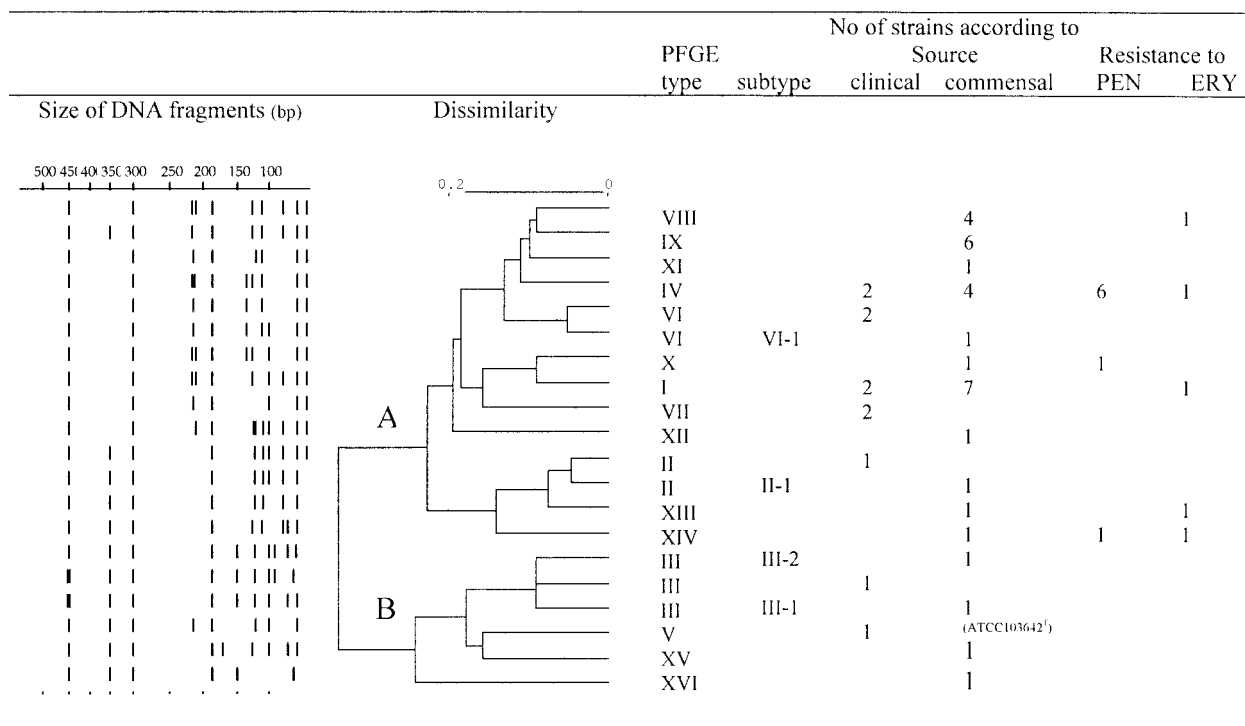


FIG. 1. Schematic representation of the PFGE restriction patterns obtained by macrorestriction with *Sma*I and the genetic relationships among 72 *S. lugdunensis* strains. The distribution of the strains according to their origins (clinical or commensal) and antibiotic susceptibility patterns are indicated on the right of the dendrogram. PEN, penicillin; ERY, erythromycin.

carriage as part of the inguinal cutaneous flora of 140 incoming patients.

(i) **Prevalence of inguinal area carriage.** The 140 patients investigated (patients C1 to C140) included 63 men and 77 women. We found that 19 women (23%) and 12 men (19%) were *S. lugdunensis* carriers. Twenty-one of these patients (68%) carried *S. lugdunensis* in both the left and the right inguinal folds. The frequency of carriage did not differ with respect to sex or body mass index when male and female patients were considered together. Overweight patients were not more likely to be carriers than other patients (23% of patients younger than age 65 years were overweight, and 19% of patients age 65 years or older were overweight). None of the 16 male patients age 65 years or older tested positive for *S. lugdunensis*. The highest frequencies of carriage were in non-overweight female patients age 65 years or older (50%) and nonoverweight male patients younger than age 65 years (41%).

(ii) **Antibiotic resistance patterns.** We determined the antibiotic resistance patterns of the 53 isolates recovered from 31 patients and *S. lugdunensis* ATCC 103642<sup>T</sup>. None were oxacillin resistant; strains from 22 patients (71%) were sensitive to all of the antibiotics tested. Isolates from five patients (16%) were resistant to penicillin, isolates from three patients (10%) were resistant to erythromycin, and isolates from one patient (3%) were resistant to both penicillin and erythromycin. When both the left and the right inguinal regions from a given patient were positive, both isolates yielded the same antibiotic type.

(iii) **Pulsotyping.** *Sma*I macrorestriction revealed 15 different pulsotypes for the 54 strains (Table 1). Pulsotypes I to VI,

which characterized the strains isolated from infections, were also found for carriage strains, and new patterns (pulsotypes VIII to XVI) were identified. Pulsotype VII was not observed for the carriage strains. According to Tenover et al. (29), four subtypes were found for pulsotypes II, III, and IV; these were named subtypes II-1, III-1, III-2, and VI-1, respectively. When cultures were positive for both the left and right inguinal folds for a given patient, the two strains were found to have the same pulsotype in all cases. For patient C120, the strains from the left and right inguinal folds differed by one band; the two subtypes were named III-1 and III-2, respectively. Eleven  $\beta$ -lactamase-positive strains were isolated from six patients (patients C30, C42, C45, C70, C140, and C15), and 8 of these 11 strains belonged to pulsotype IV.

**Genetic diversity and population structure of *S. lugdunensis* species as defined by PFGE.** To study the genetic diversity of *S. lugdunensis* species, one strain was selected from each infected patient or carrier for 42 of the 43 patients, as strains from patients that gave multiple isolates all exhibited identical pulsotypes or subtypes. The strains recovered from the left and right inguinal folds of patient C120 (strains 120L and 120R, respectively) yielded distinct patterns (subtypes III-1 and III-2, respectively). Thus, both strains from this patient were included in this analysis. Furthermore, ATCC 103642<sup>T</sup> was included in the study.

Figure 1 shows a schematic representation of all pulsotype and subtype patterns, as well as a dendrogram showing the deduced genetic relationships among the strains tested.

According to the genetic features determined by PFGE, *S.*

TABLE 1. Medical data for the 31 *S. lugdunensis* carriers and molecular typing results for 53 commensal isolates and strain ATCC 103642<sup>T</sup>

Strain no. <sup>a</sup>	Patient	Sex <sup>b</sup>	Age (yr)	Antibiotype <sup>c</sup>	PFGE group	Type	Subtype
4L, 4R	C4	M	35	S	A	I	
10L, 10R	C10	F	44	S	A	I	
38L	C38	F	88	S	A	I	
62L, 62R	C62	F	55	S	A	I	
79L	C79	F	17	E	A	I	
113L, 113R	C113	F	72	S	A	I	
124L, 124R	C124	M	57	S	A	I	
102L	C102	F	42	S	A	II	II-1
120R	C120	F	19	S	B	III	III-1
120L	C120			S	B	III	III-2
42L, 42R	C42	M	39	β-Lactamase positive	A	IV	
45L, 45R	C45	F	72	β-Lactamase positive	A	IV	
70L, 70R	C70	F	16	β-Lactamase positive	A	IV	
140L, 140R	C140	F	24	β-Lactamase positive	A	IV	
ATCC 103642 <sup>T</sup>				S	B	V	
61L, 61R	C61	M	29	S	A	VI	VI-1
9L, 9R	C9	M	44	S	A	VIII	
52L, 52R	C52	F	69	S	A	VIII	
74L, 74R	C74	F	56	E	A	VIII	
132L	C132	M	17	S	A	VIII	
6L, 6R	C6	M	49	S	A	IX	
65L, 65R	C65	F	18	S	A	IX	
76L	C76	M	18	S	A	IX	
83R	C83	F	65	S	A	IX	
108L	C108	M	42	S	A	IX	
110L, 110R	C110	M	35	S	A	IX	
30L, 30R	C30	M	18	β-Lactamase positive	A	X	
54R	C54	F	52	S	A	XI	
135L, 135R	C135	F	25	S	A	XII	
41L, 41R	C41	M	21	E	A	XIII	
15R	C15	F	15	β-Lactamase Positive, E	A	XIV	
51L, 51R	C51	F	80	S	B	XV	
100L	C100	F	33	S	B	XVI	

<sup>a</sup> L, left inguinal fold; R, right inguinal fold.

<sup>b</sup> M, male; F, female.

<sup>c</sup> S, susceptible to all antibiotics tested; E, erythromycin resistance.

*lugdunensis* species exhibit a low degree of diversity (Fig. 1). Two populations (populations A and B) were identified at a level of 35% dissimilarity. Most of the strains (37 of 44 [84%]) were clustered in group A. Each group was genetically homogeneous (maximum dissimilarity, 25%). The 11 strains characterized by a resistant phenotype (e.g., β-lactamase-positive and/or erythromycin-resistant strains) were all found in group A. The strains producing β-lactamase were strongly homogeneous; six of the eight β-lactamase-positive strains were of pulsotype IV. Reference strain ATCC 103642<sup>T</sup> was in group B. The proportions of clinical strains in groups A and B were comparable, with 24% in group A (9 of 37 strains) and 29% in group B (2 of 7 strains).

**Discriminatory power of PFGE.** To evaluate the discriminatory power of a typing method, the strains selected must not be related. Therefore, the DI of our PFGE method was calculated first by using the strains isolated from the 31 carriers who were sampled upon their arrival at the accident and emergency unit. These strains were clearly not epidemiologically related. To avoid the selection of twin strains and according to the guidelines of Tenover et al. (29), we considered strains to be twins when they belonged to similar pulsotypes and were from the same patient. For this carrier population, the DI was 0.90 (Table 2). Furthermore, the DIs were similar when they were calculated for the strains isolated from infected patients and

for all 43 representative strains studied (0.93 and 0.91, respectively) (Table 2). In contrast, the PFGE method used was shown to be an inappropriate method for the typing of β-lactamase-positive strains, with a DI of 0.46.

**DISCUSSION**

The rate at which *S. lugdunensis* is isolated from samples from the plastic surgery unit by the microbiology laboratory of our hospital has increased considerably. We conducted a case-control study to investigate the sources of the *S. lugdunensis* strains and to identify risk factors for *S. lugdunensis* infection in the case patients studied. We also used molecular typing methods to determine whether the strains isolated were epidemiologically linked. Our results raised questions about the inguinal

TABLE 2. Discriminatory power of PFGE

Subject or strain	No. of strains	No. of pulsotypes	DI
Carriers	31	14	0.90
Infected patients	11	7	0.93
All strains (including ATCC 103642 <sup>T</sup> )	43	16	0.91
β-Lactamase positive strains	8	3	0.46

area carriage of *S. lugdunensis* and the diversity of the species. Thus, these two points were also investigated.

**Investigation of *S. lugdunensis* infection.** We found that the strains from 11 patients belonged to seven distinct pulsotypes, demonstrating that a single clone did not occur over the study period. Strains from three patients (patients P2, P6, and P7) exhibited specific pulsotypes, suggesting that these patients were infected with strains from their own flora. For the other patients, the conclusions differed. The strains from four pairs of patients had identical pulsotypes: the strains from patients P1 and P3 belonged to pulsotype I, the strains from patients P4 and P5 belonged to pulsotype IV, the strains from patients P9 and P11 belonged to pulsotype VII, and the strains from patients P8 and P10 belonged to pulsotype VI. For the first three pairs, the pairs of patients were not hospitalized at the same time. It is likely that the similarities of the PFGE patterns were coincidental and that the sources of the *S. lugdunensis* strains differed in these three cases. The similarities of the patterns may indicate a certain degree of homogeneity in the species rather than an epidemiological link between the strains. In contrast, patients P8 and P10 were hospitalized simultaneously; thus, we cannot exclude the possibility of an epidemiological link between the strains involved.

The case-control study provided a potential explanation for these unrelated repetitive cases of *S. lugdunensis* infection. We reviewed procedures and techniques in collaboration with surgeons to determine whether any particular practice was associated with an increased risk of *S. lugdunensis* infection. Interviews with medical staff revealed that surgeons S1 and S4 had recently modified the surgical technique used for abdominoplasty. These two surgeons operated on 7 of the 11 infected patients. The incision sites chosen by surgeons S1 and S4 were totally within the pubic area, which was shaved preoperatively. However, the other surgeons cut above the pubic hairline, in an area with no hair. Patients were shaved the day before the operation. This procedure is known to cause large skin cuts, which increases the risk of surgical site infection by disrupting the skin barrier and allowing the invasion of microorganisms. This observation demonstrates the validity of Centers for Disease Control and Prevention recommendations that indicate that "if hair removal is necessary, it should be done either by clipping or by using a depilatory lotion rather than shaving" (21).

**Prevalence of *S. lugdunensis* inguinal area carriage.** The investigation of repetitive cases of *S. lugdunensis* infection indicated that the origin of these cases was more likely to be from the patient's skin, after shaving of the inguinal and pubic areas, rather than from cross-colonization. This suggests that the carriage of *S. lugdunensis* is usual in these anatomic regions. We evaluated the prevalence of *S. lugdunensis* inguinal area carriage as part of the cutaneous flora of 140 incoming patients to study this point. Twenty-two percent of the patients carried *S. lugdunensis* in this area, and carriage at both inguinal folds was frequent (68% of carriers). The highest frequency of carriage was observed in women aged 65 years and older (51%). This may partly explain the observation of Haile et al. (10), who showed that the rate of occurrence of *S. lugdunensis* in cultures of urine from women of this age range was high: 53% of cultures of urine from this population were *S. lugdunensis* positive. Surprisingly, among men, the highest rate

was found in men younger than age 65 years (41%). In addition, obesity did not seem to play a role, regardless of sex.

The frequency of inguinal area carriage of *S. lugdunensis* measured here is consistent with the fact that *S. lugdunensis* is an etiologic agent of skin and soft tissue infections of the lower body. Vandenesch et al. (32) described 63 patients with *S. lugdunensis* skin or soft tissue infections. Most (73%) of these infections were below the waist. In the past few years, infections located in the pubic area or perineum have repeatedly been described (e.g., a Bartholin gland abscess [23], a pubic cutaneous abscesses [23], and a cesarean delivery scar abscess [30]).

Inguinal area carriage may explain the physiopathology of invasive *S. lugdunensis* infections following the performance of invasive procedures near the inguinal region. *S. lugdunensis* peritonitis has been reported in a patient undergoing continuous ambulatory peritoneal dialysis (14) and following delivery by cesarean section (8). Several cases of *S. lugdunensis* endocarditis have also been reported: in a patient on hemodialysis (14), in a patient after renal transplantation (24), in patients after femoral arterial catheterization, after vasectomy (24), and in association with a scrotal wound (24). Recently, a case of ventriculoperitoneal shunt infection caused by *S. lugdunensis* was described (5, 27). All these clinical reports strongly suggest that pubic-inguinal area carriage is the source for iatrogenic *S. lugdunensis* infections.

Inguinal area carriage does not explain the two cases of *S. lugdunensis* infections that we observed following mammaplasty (in patients P2 and P8). In addition, several cases of breast abscess caused by *S. lugdunensis* have been reported (18, 34). These observations suggest that the breast region or the area under the mammary folds is another site of *S. lugdunensis* carriage. Further studies are required to explore this hypothesis. Unfortunately, the axilla site was not sampled in our study.

**Antibiotic susceptibility.** In accordance with previously published data (11, 32), most of the strains studied (49 of 72 [68%]) were found to be sensitive to all of the antibiotics tested. Seventeen strains (24%) produced  $\beta$ -lactamase, and nine strains (12%) were found to be resistant to erythromycin. Clinical and commensal strains showed similar antibiotic susceptibilities.

**Genomic diversity of *S. lugdunensis*.** Our knowledge of the genetic diversity of *S. lugdunensis* is limited and concerns only a limited number of strains (6). On the basis of the genetic features obtained by *Sma*I macrorestriction, we established the relationships between 72 *S. lugdunensis* strains (Fig. 1). This type of genetic study is a first step (i) toward evaluating the diversity of a species and determining the link between subpopulations of strains, especially between commensal strains and strains isolated from patients with infectious diseases or between strains that are susceptible or resistant to an antibiotic, and (ii) toward calculating the discriminatory power of a typing method (PFGE in this study).

The degree of genetic diversity of *S. lugdunensis* appears to be low. Most of the strains clustered in two homogeneous groups (groups A and B) that diverged at a dissimilarity of 35% (Fig. 1). In our population, the strains responsible for infections exhibited a degree of diversity similar to that exhibited by commensal strains, and strains isolated from patients with in-

fections did not cluster in particular subgroups. The genetic structure of this population suggests that most commensal strains have the ability to cause infectious disease, at least locally. Our data indicate that the species may be more diverse than the degree of diversity reported by Etienne et al. (6), who cut chromosomal DNA from 30 *S. lugdunensis* strains with three enzymes (*EcoRI*, *PstI*, and *PvuII*) and showed that 23 strains exhibited the same patterns with the three enzymes. Nevertheless, the difference between the findings of diversity from the study of Etienne et al. (6) and our results may be explained by the different methods used.

In our population of *S. lugdunensis* strains, strong homogeneity was found only for isolates producing  $\beta$ -lactamase, a subpopulation that was very homogeneous; six of the eight  $\beta$ -lactamase-positive strains displayed by the same PFGE pattern.

The diversity of *S. lugdunensis* species by macrorestriction analysis with *SmaI* was limited but was sufficient to consider that PFGE with *SmaI* is suitable for epidemiological investigations. Indeed, the DI of Hunter and Gaston (13) calculated for epidemiologically unrelated strains was 0.90, a value considered to be acceptable. In contrast, and because of the strong genetic homogeneity observed in our study with most *S. lugdunensis* strains producing  $\beta$ -lactamase, PFGE with *SmaI* should not be used for these strains (for which the DI was 0.60). Further studies that use multiple molecular methods to characterize the genetic diversity of a larger collection of  $\beta$ -lactamase-positive *S. lugdunensis* strains may be indispensable for proposing adequate tools that can be used to type this population.

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#### REFERENCES

- Bobin, S., A. Durand-Dubief, D. Bouhour, G. Kirkorian, F. Vandenesch, J. Etienne, M. Ballet-Mechain, and D. Peyramond. 1999. Pacemaker endocarditis due to *Staphylococcus lugdunensis*: report of two cases. *Clin. Infect. Dis.* **28**:404–405.
- Camacho, M., S. Guis, J. P. Mattei, R. Costello, and J. Roudier. 2002. Three-year outcome in a patient with *Staphylococcus lugdunensis* discitis. *Joint Bone Spine* **69**:85–87.
- Celard, M., H. Lelievre, J. F. Obadia, P. Chevalier, F. Forey, F. Vandenesch, and J. Etienne. 1997. Long-standing bacteremia and endocarditis caused by *Staphylococcus lugdunensis* in a patient with an implantable cardioverter defibrillator. *Clin. Microbiol. Infect.* **3**:387–388.
- Deplano, A., A. Schuermans, J. Van Eldere, W. Witte, H. Meugnier, J. Etienne, H. Grundmann, D. Jonas, G. T. Noordhoek, J. Dijkstra, A. van Belkum, W. van Leeuwen, P. T. Tassios, N. J. Legakis, A. van der Zee, A. Bergmans, D. S. Blanc, F. C. Tenover, B. C. Cookson, G. O'Neil, M. J. Struelens, and The European Study Group on Epidemiological Markers of the ESCMID. 2000. Multicenter evaluation of epidemiological typing of methicillin-resistant *Staphylococcus aureus* strains by repetitive-element PCR analysis. *J. Clin. Microbiol.* **38**:3527–3533.
- Elliott, S. P., R. Yogev, and S. T. Shulman. 2001. *Staphylococcus lugdunensis*: an emerging cause of ventriculoperitoneal shunt infections. *Pediatr. Neurosurg.* **35**:128–130.
- Etienne, J., F. Poitevin-Later, F. Renaud, and J. Fleurette. 1990. Plasmid profiles and genomic DNA restriction endonuclease patterns of 30 independent *Staphylococcus lugdunensis* strains. *FEMS Microbiol. Lett.* **55**:93–97.
- Fervenza, F. C., G. E. Contreras, K. N. Garratt, and J. M. Steckelberg. 1999. *Staphylococcus lugdunensis* endocarditis: a complication of vasectomy? *Mayo Clin. Proc.* **74**:1227–1230.
- Fleurette, J., M. Bes, Y. Brun, J. Freney, F. Forey, M. Coulet, M. E. Reverdy, and J. Etienne. 1989. Clinical isolates of *Staphylococcus lugdunensis* and *S. schleiferi*: bacteriological characteristics and susceptibility to antimicrobial agents. *Res. Microbiol.* **140**:107–118.
- Freney, J., Y. Brun, M. Bes, H. Meugnier, F. Grimont, P. A. D. Grimont, C. Nervi, and J. Fleurette. 1988. *Staphylococcus lugdunensis* sp. nov. and *Staphylococcus schleiferi* sp. nov., two species from human clinical specimens. *Int. J. Syst. Bacteriol.* **38**:168–172.
- Haile, D. T., J. Hughes, E. Vetter, P. Kohner, R. Snyder, R. Patel, and F. R. Cockerill III. 2002. Frequency of isolation of *Staphylococcus lugdunensis* in consecutive urine cultures and relationship to urinary tract infection. *J. Clin. Microbiol.* **40**:654–656.
- Herchline, T. E., and L. W. Ayers. 1991. Occurrence of *Staphylococcus lugdunensis* in consecutive clinical cultures and relationship of isolation to infection. *J. Clin. Microbiol.* **29**:419–421.
- Hernandez, J. L., J. Calvo, X. Antolinez, F. Gutierrez-Rubio, and M. C. Farinas. 2001. Septic arthritis due to *Staphylococcus lugdunensis*. *Enferm. Infect. Microbiol. Clin.* **19**:414.
- Hunter, P. R., and M. A. Gaston. 1988. Numerical index of the discriminatory ability of typing systems: an application of Simpson's index of diversity. *J. Clin. Microbiol.* **26**:2465–2466.
- Kamaraju, S., K. Nelson, D. N. Williams, W. Ayenew, and K. S. Modi. 1999. *Staphylococcus lugdunensis* pulmonary valve endocarditis in a patient on chronic hemodialysis. *Am. J. Nephrol.* **19**:605–608.
- Koh, T. W., S. J. Brecker, and C. A. Layton. 1996. Successful treatment of *Staphylococcus lugdunensis* endocarditis complicated by multiple emboli: a case report and review of the literature. *Int. J. Cardiol.* **55**:193–197.
- Laguno, M., O. Miro, C. Font, and A. de la Sierra. 1998. Pacemaker-related endocarditis. Report of 7 cases and review of the literature. *Cardiology* **90**:244–248.
- Lessing, M. P., D. W. Crook, I. C. Bowler, and B. Gribbin. 1996. Native-valve endocarditis caused by *Staphylococcus lugdunensis*. *Q. J. Med.* **89**:855–858.
- Lina, B., F. Vandenesch, M. E. Reverdy, T. Greenland, J. Fleurette, and J. Etienne. 1994. Non-puerperal breast infections due to *Staphylococcus lugdunensis*. *Eur. J. Clin. Microbiol. Infect. Dis.* **13**:686–687.
- Ludlam, H., and I. Phillips. 1989. *Staphylococcus lugdunensis* peritonitis. *Lancet* **ii**:1394.
- Maki, D. G., C. E. Weise, and H. W. Sarafin. 1977. A semiquantitative culture method for identifying intravenous-catheter-related infection. *N. Engl. J. Med.* **296**:1305–1309.
- Mangram, A. J., T. C. Horan, M. L. Pearson, L. C. Silver, and W. R. Jarvis. 1999. CDC guideline for prevention of surgical site infection. *Infect. Control Hosp. Epidemiol.* **20**:247–278.
- Murdoch, D. R., R. J. Everts, S. T. Chambers, and I. A. Cowan. 1996. Vertebral osteomyelitis due to *Staphylococcus lugdunensis*. *J. Clin. Microbiol.* **34**:993–994.
- Ortiz de la Tabla, V., F. Gutierrez-Rodero, C. Martin, A. Zorraquino, and I. Belinchon. 1996. *Staphylococcus lugdunensis* as a cause of abscesses in the perineal area. *Eur. J. Clin. Microbiol. Infect. Dis.* **15**:405–407.
- Patel, R., K. E. Piper, M. S. Rouse, J. R. Uhl, F. R. Cockerill III, and J. M. Steckelberg. 2000. Frequency of isolation of *Staphylococcus lugdunensis* among staphylococcal isolates causing endocarditis: a 20-year experience. *J. Clin. Microbiol.* **38**:4262–4263.
- Polenakovik, H., T. Herchline, C. Bacheller, and J. Bernstein. 2000. *Staphylococcus lugdunensis* endocarditis after angiography. *Mayo Clin. Proc.* **75**:656–657.
- Sampathkumar, P., D. R. Osmon, and F. R. Cockerill III. 2000. Prosthetic joint infection due to *Staphylococcus lugdunensis*. *Mayo Clin. Proc.* **75**:511–512.
- Sandoe, J. A., and C. M. Longshaw. 2001. Ventriculoperitoneal shunt infection caused by *Staphylococcus lugdunensis*. *Clin. Microbiol. Infect.* **7**:385–387.
- Schnitzler, N., R. Meilicke, G. Conrads, D. Frank, and G. Haase. 1998. *Staphylococcus lugdunensis*: report of a case of peritonitis and an easy-to-perform screening strategy. *J. Clin. Microbiol.* **36**:812–813.
- 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.
- Uriel, J. A., M. L. Aisa, M. L. Marco, B. Fortunato, and L. Torres. 1996. Cesarean scar abscess caused by *Staphylococcus lugdunensis*. *Enferm. Infect. Microbiol. Clin.* **14**:631–632.
- Vandenesch, F., J. Etienne, M. E. Reverdy, and S. J. Eykyn. 1993. Endocarditis due to *Staphylococcus lugdunensis*: report of 11 cases and review. *Clin. Infect. Dis.* **17**:871–876.
- Vandenesch, F., S. J. Eykyn, J. Etienne, and J. Lemozy. 1995. Skin and post-surgical wound infections due to *Staphylococcus lugdunensis*. *Clin. Microbiol. Infect.* **1**:73–74.
- Vandenesch, F., S. J. Projan, B. Kreiswirth, J. Etienne, and R. P. Novick. 1993. Agr-related sequences in *Staphylococcus lugdunensis*. *FEMS Microbiol. Lett.* **111**:115–122.
- Waghorn, D. J. 1994. *Staphylococcus lugdunensis* as a cause of breast abscess. *Clin. Infect. Dis.* **19**:814–815.
- Weightman, N. C., K. E. Allerton, and J. France. 2000. Bone and prosthetic joint infection with *Staphylococcus lugdunensis*. *J. Infect.* **40**:98–99.