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Survey of Methicillin-Resistant *Staphylococcus aureus* Strains from Two Hospitals in El Paso, Texas

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Seventy-one percent of 76 methicillin-resistant *Staphylococcus aureus* (MRSA) strains isolated from two medical centers in El Paso, Texas, represent three similar pulsed-field gel electrophoresis types. Overall, six pulsed-field types were identified represented by multilocus sequence/staphylococcal chromosomal cassette DNA *mec* (SCC*mec*) types: ST5-MRSA-II; ST36-MRSA-II; ST8 (untypeable SCC*mec*); and a newly described clonal cluster 8 strain, ST507-MRSA-IV. This study demonstrates the presence of multiple-antibiotic-resistant epidemic MRSA clones in El Paso.

Staphylococcus aureus is the leading cause of nosocomial infections (5). Today, methicillin-resistant *Staphylococcus aureus* (MRSA) resistant to multiple antibiotics is commonly isolated, and vancomycin-intermediate (9) and vancomycin-resistant (4) strains have been described. Only a few epidemic MRSA clones are responsible for a large percentage of MRSA disease worldwide (8, 21). Methicillin resistance in staphylococci is mediated by *mecA* (1), which is carried on a larger chromosomal element referred to as the staphylococcal chromosomal cassette DNA *mec* (SCC*mec*) (13). SCC*mec* consists of the *mec* gene complex, containing *mecA* and the *mecA* regulatory genes, and the *ccr* complex, which encodes a recombinase(s) responsible for SCC*mec* movement (12). To date, five SCC*mec* allotypes have been described on the basis of the *mec* gene complex, the *ccr* gene complex, and other genetic elements (10, 11, 15).

El Paso, Texas, is the largest United States-Mexican border city (population of ~565,000) and ~77% of the population is of Hispanic or Latino origin. Immediately south of El Paso lies the Mexican city of Ciudad Juarez (population of ~1,200,000), where antibiotics are readily purchased over the counter (3). In order to understand MRSA epidemiology in a United States-Mexican border city with a large Hispanic or Latino population, we have performed a survey of MRSA strains collected in two medical centers in El Paso.

Typing of MRSA isolates. Seventy-six clinical MRSA isolates (Table 1) were collected from the Las Palmas (27 isolates) and Del Sol (49 isolates) medical centers in El Paso, Texas, from June to August 2002. Antibiograms were performed using the Kirby-Bauer method according to the CLSI (formerly NCCLS) (17) using antibiotic disks obtained from Oxoid (Basingstoke, England) or Remel (Lenexa, KS). Resistance to fusidic acid was determined using a 10-μg disk (Oxoid). Inducible clindamycin resistance was determined by a disk diffusion assay by the method of McDougal et al. (16). Strains resistant to three or more classes of antibiotics were considered multiply antibiotic resistant (16). Vancomycin MICs were determined with AB BIODISK Etest strips (Remel, Lenexa, KS) according to the manufacturer's instructions. Pulsed-field gel electrophoresis (PFGE) of SmaI-digested chromosomal DNA was performed as previously described (18) using the CHEF DR III system (Bio-Rad Laboratories, Inc., Hercules, Calif.). Chromosomal patterns were analyzed by scanning with a Fluor-S MultiImager and analyzing them with Multi-Analyst/PC (Bio-Rad). PFGE SmaI restriction fragment length polymorphism patterns were grouped according to the criteria of Tenover et al. (23), and strains with 80% or greater pattern similarity were considered clonal. Multilocus sequence typing (MLST) was performed on a LI-COR Long Reader 4200 sequencer (LI-COR, Inc., Lincoln, NE) with primers and PCR protocols previously established (7). LI-COR sequences were collected with Base Imager software (LI-COR) and manipulated with AssemblyLIGN (Accelrys, Cambridge, England). Sequences were then submitted to http://www.mlst.net, and a sequence type (ST) was assigned. SCC*mec* allotyping (14) and phage typing (2) using the basic international, Australian MRSA, and international MRSA phage sets were determined according to published procedures.

PFGE of SmaI-digested chromosomal DNA revealed the presence of five pulsed-field type (PFT) groups and one unique strain (PFT-F) (Fig. 1). PFT-A represents the largest PFT group that was further subdivided into three subgroups, while PFT-B was the second largest group. MLST revealed that strains LP60 (PFT-A1), LP35 (PFT-E), and LP57 (PFT-F) were all ST5 (USA 100 or New York/Japan) clones, strain LP8 (PFT-B) had an ST36 (USA 200 or EMRSA-16) signature, LP13 (PFT-C) was of ST8 (USA 500 or New York V) ancestry, and LP3 (PFT-D) represents a new ST signature ST507 (3,3,74,58,4,4,3), in clonal complex 8 (CC8) (8). SCC*mec* typing revealed that strains LP60, LP8, LP35, and LP57 harbored SCC*mec* allotype II, while LP3 possessed a type IV SCC*mec*

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TABLE 1. Characteristics of MRSA strains collected from two medical centers in El Paso, Texas

Strain[a]	Pulsotype group	Medical center (source)	Antibiotype[b]	VAN MIC[c]	Phage type assigned using the following phage set:		
					Basic international	Australian MRSA	International MRSA
LP72	C	Las Palmas (sputum)	CDEGMO	2.0	75,85,(90)	90A,1648,13M	MR8,(MR25),30,33,38, M3,M5
LP13	C	Del Sol (stool)	CDEGMO	2.0	75,85,(90)	90A,1648,67R,(87M),13M	MR8,30,33,38,M5,622
LP21	C	Las Palmas (wound)	CDEGMO	2.0	85,(90)	90A,1648,13M	MR8,30,33,38,M5,622
LP23	C	Las Palmas (blood)	CDEGMO	2.0	85,90	90A,1648,13M	MR8,30,33,38,M5
LP17	C	Del Sol (stool)	CDEGMO	2.0	NT[d]	90A,1648,67R,13M	MR8,30,33,38,M5,622
LP3	D	Las Palmas (wound)	CDEGIOTm	2.5	6,47,53,54,(75),85,81	67R,87M	30,38,M3,M5
LP20	D	Del Sol (bronchial)	CDEGIOTTm	2.0	6,42E,47,(53),54,75,(83A),85,81	56B,56C,67R87M	30,(38),M3,M5,56B
LP29	D	Del Sol (bronchial)	CDEGIOTTm	2.0	6,(42E),47,54,(75),85,81	67R,87M	30,M3,M5
LP39	D	Del Sol (tissue)	CDEGIOTTm	2.0	6,(42E),(47),54,85,81	67R	30,(38),M3,M5
LP9	A1	Del Sol (urine)	CD*EO	1.5	NT	NT	NT
LP14	A1	Del Sol (blood)	CD*EO	1.5	NT	NT	NT
LP50	A1	Del Sol (wound)	CD*EO	1.5	NT	56C	NT
LP60	A1	Las Palmas (wound)	CD*EO	2.0	NT	NT	NT
LP68	A1	Las Palmas (urine)	CDEO	2.0	54	56C	NT
LP69	A1	Las Palmas (wound)	CDEO	2.0	54	56C	NT
LP30	A1	Del Sol (sputum)	CD*EO	1.5	NT	NT	NT
LP12	A1	Del Sol (wound)	CD*EO	2.0	NT	NT	NT
LP15	A1	Las Palmas (nasal)	CDEO	2.0	54	NT	NT
LP34	A1	Del Sol (wound)	CDEGO	2.0	NT	NT	NT
LP41	A1	Del Sol (wound)	CD*EO	2.0	NT	NT	NT
LP44	A2	Del Sol (urine)	CD*EOT	2.0	6,42E,54,(75),83A,81	56B,56C,67R	MR8,MR12,56B
LP24	A2	Del Sol (drainage)	CD*EOT	2.0	6,42E,54,83A	56C,67R	MR8,MR12
LP75	A2	Del Sol (nasal)	CD*EO	2.0	6,42E,54,83A,81	56B,56C,67R	MR8,MR12,56B
LP31	A2	Del Sol (wound)	CDEO	2.0	(42E),54,83A,(85),81	56B,56C,67R	MR8,MR12,56B
LP32	A2	Del Sol (wound)	CDEO	2.0	6,54,81	(56B),56C,67R	MR8,MR12,(30),(56B)
LP36	A2	Las Palmas (drainage)	CD*EO	2.0	6,54,83A,(81)	56C,67R	MR8,MR12
LP46	A2	Del Sol (unknown)	CDEO	2.0	6,54,81	56B,56C,67R	MR8,56B
LP16	A2	Las Palmas (wound)	CDEO	2.0	6,(42E),(47),54,81	56C,67R	MR8,(M3)
LP42	A2	Del Sol (surgical wound)	CDEO	1.5	NT	ND[e]	ND
LP76	A2	Del Sol (sputum)	CD*EO	1.5	6,42E,47,54,81	56B,56C,67R	56B
LP63	A2	Del Sol (wound)	CDEO	2.0	NT	NT	NT
LP64	A2	Del Sol (sputum)	CD*EO	2.0	NT	NT	NT
LP47	A2	Del Sol (unknown)	CD*EO	2.0	6,42E,47,54,81	56C	38
LP56	A2	Las Palmas (wound)	CDEO	2.0	NT	NT	NT
LP74	A2	Las Palmas (sputum)	CD*EOT	1.5	6,(42E),47,54,83A,85,81	56B,56C,67R	MR8,MR12,56B
LP48	A2	Del Sol (urine)	CDEOT	1.5	6,54,83A,(81)	(56C),67R	MR8,MR12
LP53	A2	Las Palmas (urine)	CD*EOT	2.0	47,54,83A,81	56B,56C,67R	MR8,MR12,M3,56B
LP54	A2	Las Palmas (wound)	CDEO	2.0	NT	NT	NT
LP66	A2	Del Sol (blood)	CDEGOT	2.0	NT	NT	NT
LP77	A2	Del Sol (urine)	CDEO	2.0	NT	67R	NT
LP78	A2	Del Sol (urine)	CDEO	2.0	NT	NT	NT
LP51	A2	Del Sol (respiratory)	CD*EO	2.0	54	NT	NT
LP65	A2	Las Palmas (wound)	CD*EO	2.0	6,47,54,75	56B,56C	M3,56B
LP73	A3	Las Palmas (sputum)	CDEO	2.0	54,75,83A	67R	MR8,MR12
LP38	A3	Las Palmas (abscess)	CDEO	2.0	NT	NT	NT
LP1	A3	Del Sol (urine)	CDEO	1.5	54	67R	MR8,MR12,30
LP28	A3	Del Sol (sputum)	CDEMO	2.0	NT	NT	NT
LP58	A3	Las Palmas (wound)	CDEO	2.0	ND	ND	ND
LP52	A3	Del Sol (wound)	CDEO	2.0	NT	NT	NT
LP25	A3	Las Palmas (wound)	CDEMO	2.0	NT	NT	NT
LP2	A3	Las Palmas (sputum)	CDEMO	2.0	NT	NT	NT
LP5	A3	Del Sol (stool)	CDEO	1.5	NT	NT	NT
LP4	A3	Del Sol (stool)	CD*EO	1.5	54	67R	MR12
LP7	A3	Del Sol (sputum)	CD*EO	1.5	NT	67R	NT
LP49	A3	Del Sol (unknown)	CDEOR	2.0	83A	67R	MR8,MR12,30,33,38, M3, M5,622,56B
LP40	A3	Del Sol (blood)	CDEO	2.0	NT	NT	MR8,MR12
LP11	A3	Del Sol (wound)	CDEO	2.0	NT	NT	MR8,MR12
LP45	A3	Del Sol (wound)	CDEO	2.0	NT	NT	NT
LP57	F	Las Palmas (wound)	CD*EO	1.5	6	NT	NT
LP62	E	Las Palmas (wound)	CD*EGOTTm	2.0	83A	67R	M3
LP70	E	Del Sol (sputum)	CD*EOT	2.0	NT	NT	NT
LP35	E	Del Sol (wound)	CD*EOT	2.0	NT	NT	NT
LP43	E	Del Sol (wound)	CD*EO	2.0	52,(52A),6,42E,47,53,54,75,83A, (84),85,81,(95),88,90	47T,56B,56C,90A,1648, 67R,87M,13M	MR8,MR12
LP26	B	Del Sol (wound)	CDEGIMOTm	1.5	NT	87M	M5

Continued on following page

TABLE 1—*Continued*

Strain[a]	Pulsotype group	Medical center (source)	Antibiotype[b]	VAN MIC[c]	Phage type assigned using the following phage set:		
					Basic international	Australian MRSA	International MRSA
LP6	B	Del Sol (sputum)	CDEGIOTm	0.5	NT	NT	NT
LP55	B	Del Sol (wound)	CDEGIOTm	1.5	NT	NT	NT
LP61	B	Las Palmas (urine)	CDEIO	2.0	29,6,42E,47,54,(75),81	56C,67R	NT
LP67	B	Del Sol (urine)	CDEOTm	0.75	NT	NT	NT
LP71	B	Del Sol (unknown)	CDEIMO	1.5	NT	56C	NT
LP33	B	Del Sol (bone)	CDEGIO	1.0	NT	NT	NT
LP37	B	Las Palmas (unknown)	CDEGIMOTm	1.5	NT	NT	NT
LP8	B	Las Palmas (sputum)	CDEGIOTTm	0.75	NT	NT	NT
LP10	B	Las Palmas (wound)	CDEGIMOTm	1.0	NT	NT	NT
LP18	B	Del Sol (wound)	CDEGIOTm	0.38	NT	NT	NT
LP22	B	Las Palmas (sputum)	CDEGIOTm	0.5	NT	NT	NT
LP27	B	Del Sol (wound)	CDEGIOTm	1.0	(77),95	67R	NT

[a] Bold strain names indicate that MLST and SCC*mec* allotyping were performed on these strains.
[b] Abbreviations: C, ciprofloxacin; D, clindamycin; D*, inducible clindamycin resistance; E, erythromycin; G, gentamicin; I, imipenem; M, mupirocin; O, oxacillin; R, rifampin; T, tetracycline; Tm, trimethoprim.
[c] Vancomycin (VAN) MIC determined with E test strips.
[d] NT, nontypeable.
[e] ND, not determined.

and LP13 possessed a class A *mec* complex, but the *ccr* complex could not be typed with the primer sets utilized. Phage typing was not useful in determining isolate relatedness, and many strains proved untypeable by this method (Table 1).

Therefore, 71% of the MRSA strains collected from two medical centers in El Paso, Texas, are represented by related PFTs, PFT-A, -E, and -F, which were resistant to ciprofloxacin and erythromycin and constitutively or inducibly resistant to

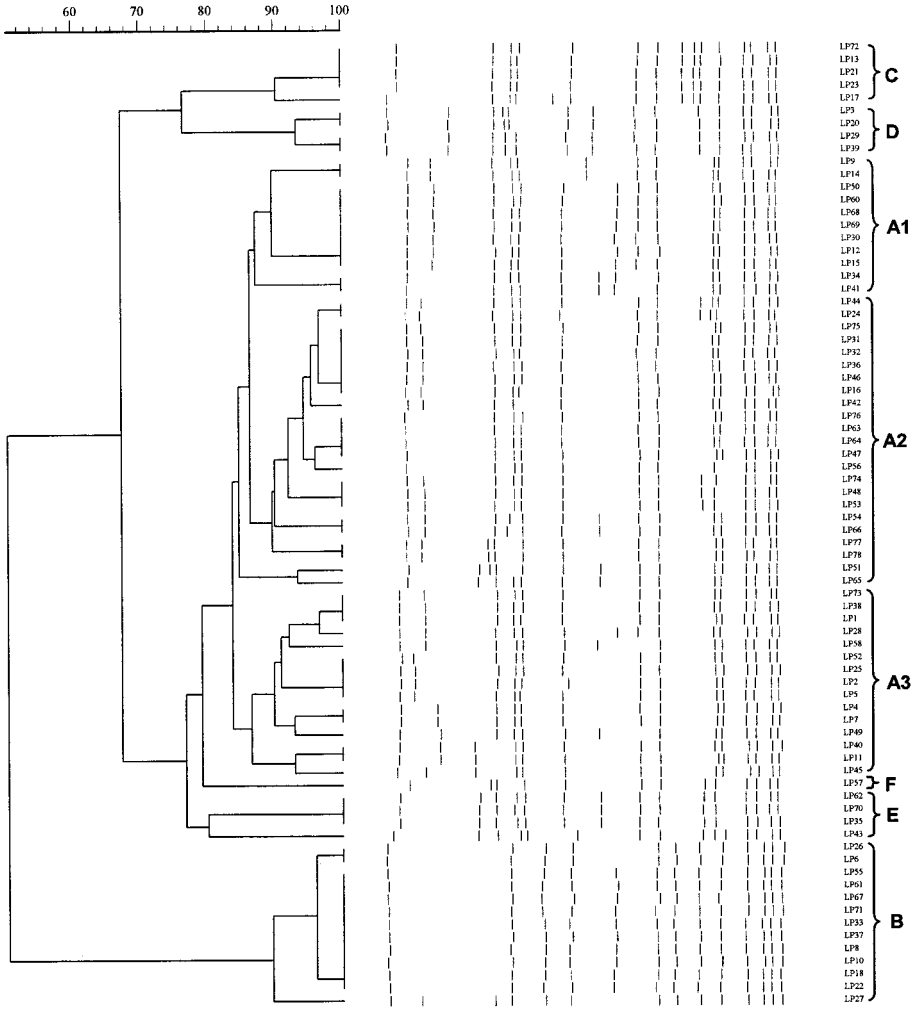

FIG. 1. PFGE patterns of SmaI-restricted chromosomal DNA and dendrogram of percent relatedness derived from the patterns.

clindamycin. Various strains in these PFT groups were also singly or multiply resistant to gentamicin, mupirocin, rifampin, tetracycline, and/or trimethoprim. Representative strains of these PFTs were all ST5-MRSA-II, and the presence of three diverged PFTs among these clones may indicate that these clones have been introduced on multiple occasions or are evolving in the medical centers investigated. ST5-MRSA-II clones represent 44% of all MRSA strains isolated in the United States (16), and in 1 year following introduction, an ST5-MRSA-II clone completely replaced an existing endemic clone in a Mexican hospital (24). Numerous vancomycin-intermediate *S. aureus* (VISA) and vancomycin-resistant *S. aureus* (VRSA) are derived from ST5-MRSA-II clones (8, 16, 17). Since this clone is endemic in El Paso, the stage is set for the selection of VISA or VRSA.

The strains in the second largest PFT group, PFT-B, were all resistant to ciprofloxacin, clindamycin, and erythromycin and represent 17% of the isolates investigated. Representative PFT-B strain LP8 is an ST36-MRSA-II clone, which is the second most commonly isolated health care-associated MRSA in the United States (16).

The PFT-C strains were resistant to ciprofloxacin, clindamycin, erythromycin, gentamicin, and mupirocin. Representative PFT-C strain LP13 is an ST8 clone with an untypeable SCC*mec*, suggesting the presence of a unique SCC*mec*. ST8 clones demonstrate variation in their SCC*mec* (6, 8), and some MRSA contain *ccr* gene complexes that do not amplify with the primers used for SCC*mec* allotyping (14, 19). ST8 clones are the second most common clone isolated in two hospitals in Miami, Florida, and 70% of MRSA strains isolated from confirmed human immunodeficiency virus-positive patients are ST8-MRSA-IV (6).

The PFT-D strains were resistant to ciprofloxacin, clindamycin, erythromycin, gentamicin, imipenem, tetracycline, and trimethoprim. PFT-D representative strain LP3 is a new ST type, ST507-MRSA-IV located in CC8, which encompasses ST8 strains as well (8). It is possible that LP3 has evolved from an ST8 strain (LP13?) endemic in our area.

It is of interest to note that the PFT-B strains demonstrated the lowest and most variable (0.38 to 1.5 mg/liter) vancomycin MICs (Table 1), while all other strains demonstrated vancomycin MICs of 1.5 to 2 mg/liter.

In conclusion, we have demonstrated the presence of multiple-antibiotic-resistant epidemic MRSA clones in two medical centers in El Paso, Texas, located at a unique ethnic crossroads. All MRSA investigated were susceptible to synercid, linezolid, and fusidic acid, and no VISA or VRSA was detected.

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