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Outbreak of *Serratia marcescens* Coproducing ArmA and CTX-M-15 Mediated High Levels of Resistance to Aminoglycoside and Extended-Spectrum Beta-Lactamases, Algeria

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Serratia marcescens is one of the most important pathogens responsible for nosocomial infections worldwide. Here, we have investigated the molecular support of antibiotic resistance and genetic relationships in a series of 54 *S. marcescens* clinical isolates collected from Eastern Algeria between December 2011 and July 2013. The 54 isolates were identified by matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) mass spectrometry (MS). Antibiotic susceptibility testing was performed by disc diffusion and *E*-test methods. Antibiotic resistance genes were detected by polymerase chain reaction (PCR). The genetic transfer of antibiotic resistance was performed by conjugation using azide-resistant *Escherichia coli J53* as the recipient strain, and plasmid analysis was done by PCR-based replicon typing. The relatedness of our isolates was determined by phylogenetic analysis based on partial sequences of four protein-encoding genes (*gyrB*, *rpoB*, *infB*, and *atpD*) and then compared to MALDI-TOF MS clustering. Thirty-five out of 54 isolates yielded an extended-spectrum β-lactamase (ESBL) phenotype and carried $bla_{CTX-M-15}$ ($n=32$), bla_{TEM-1} ($n=26$), bla_{TEM-71} ($n=1$), bla_{SHV-1a} ($n=1$), and bla_{PER-2} ($n=12$). Among these isolates, we identified a cluster of 15 isolates from a urology unit that coharbored ESBL and the 16S rRNA methyltransferase *armA*. Conjugation was successful for five selected strains, demonstrating the transferability of a conjugative plasmid of incompatibility group incL/M type. Phylogenetic analysis along with MALDI-TOF clustering likely suggested an outbreak of such isolates in the urology unit. In this study, we report for the first time the co-occurrence of *armA* methyltransferase with ESBL in *S. marcescens* clinical isolates in Eastern Algeria.

Introduction

Serratia marcescens is a gram-negative rod-shaped bacterium that belongs to the Enterobacteriaceae family. Some strains of *S. marcescens* produce a red to dark pink pigmentation called prodigiosin that can easily facilitate identification.[15] While *S. marcescens* was once considered as a harmless saprophyte,[18] it is now recognized as an important emerging opportunistic pathogen associated with nosocomial infections, including wound infections, urinary tract infections, respiratory tract infections, and bacteremia.[23,42] The infections have been reported with increasing frequency, especially in the intensive care units.[42] The first report of detection of nosocomial infections caused by this organism was in 1951 at the Stanford University Hospital.[43] Outbreaks can be attributed to different sources, including contamination of disinfectant solutions, soaps, mechanical respirators, hand-to-hand transmission,[4,11] and cystoscopes.[30]

S. marcescens often exhibits multiple mechanisms of resistance.[23,37,39] Moreover, carbapenemase-encoding genes have recently been described, including class A β-lactamases (KPC and SME) and class B β-lactamases (IMP and VIM).[17] β-Lactams are usually used for treating severe infections in association with aminoglycosides.[45] Recently, the increased frequency of resistance of Enterobacteriaceae to aminoglycosides, mediated by the production of enzymes that modify the antibiotics by adenylation, phosphorylation, or acetylation, has been described worldwide.[9] Methylation of the 16S ribosomal RNA (rRNA) has emerged as a resistance mechanism to aminoglycosides in Enterobacteriaceae, including *S. marcescens*.[12]

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Several cases of outbreaks due to this bacterium and these mechanisms of resistance have been described worldwide.[6,7,17,28] However, in Algeria, only one study reported an outbreak involving extended-spectrum β-lactamases (ESBLs). This incident was caused by 14 *S. marcescens* infections in the urology ward: 12 isolates contained $bla_{CTX\text{-}M}$, and the remaining 2 isolates carried both $bla_{CTX\text{-}M}$ and bla_{TEM}.[30] Furthermore, there has been only one recent report describing an *armA* methyltransferase-producing *Klebsiella pneumoniae* in Algeria.[1] *S. marcescens* has a natural resistance to many antibiotics, including colistin.[38] The prevention of the emergence of this pathogen and the optimization of the consumption of colistin should be taken into consideration to prevent the spread and selection of this organism.

S. marcescens infection can cause mortality, especially following the use of colistin as the last therapeutic option to treat infection caused by organisms producing carbapenemases, such as *Acinetobacter baumannii*.[27] In the present study, our aim was to investigate the molecular mechanism of antibiotic resistance and a possible outbreak involving *S. marcescens* clinical isolates from Eastern Algeria.

Materials and Methods

Bacterial strains and identification

Between December 2011 and July 2013, a total of 54 consecutive and nonduplicate clinical isolates of *S. marcescens* were recovered from Eastern Algeria. All isolates were grown on MacConkey agar (Biomerieux, Marcy l'Etoile, France) and incubated at 37°C for 18–24 hr. Strains were identified using the API 20E system (Biomerieux) and confirmed by matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) mass spectrometry (MS) performed with a Bruker Daltonics Microflex using 96 spot polished-steel targets.[2]

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing was performed by the disc diffusion method on Mueller-Hinton agar (Biomerieux) as recommended by the committee of the French Society for Microbiology (CA-SFM) 2013 Guidelines. Sixteen antibiotics were used: amoxicillin, amoxicillin/clavulanic acid, cefoxitin, ceftriaxone, ceftazidime, cefotaxime, cefepime, aztreonam, imipenem, amikacin, tobramycin, gentamicin, ciprofloxacin, ofloxacin, colistin, and trimethoprim/sulfamethoxazole. *Escherichia coli* ATCC 25922 was used as a reference strain. The ESBL phenotype was detected by using the double disc synergy test as previously described.[25] Minimum inhibitory concentrations (MICs) for cefotaxime, ceftazidime, amikacin, and ciprofloxacin were determined using the *E*-test.

Screening for antibiotic resistance encoding genes

DNA was extracted using the EZ1 Advanced XL Extractor, DNA Bacteria Card, and the EZ1® DNA Tissue Kit (Qiagen, Courtaboeuf, France) according to the manufacturer's instructions. Polymerase chain reaction (PCR) was conducted to detect ESBL-encoding genes targeting $bla_{\text{CTX-M}}$, bla_{TEM}, bla_{SHV},[13,21,44] bla_{PER}, bla_{GES}, and bla_{VEB},[26] as well as aminoglycoside-modifying enzyme-encoding genes, including *aac(3)-la*, *aac(6′)-Ib*, *aph(3′)VI*,[31] *ant(2″)*-I,[20] *aadA*,[1] genes coding for 16S rRNA methyltransferase *armA*,[20] and fluoroquinolones *qnrA* and *qnrB*.[41] The cycling parameters included 15 min of denaturation at 95°C, followed by 35 cycles of denaturation at 95°C for 30 sec, annealing at 55°C for 50 sec, and extension at 72°C for 1 min and 30°sec before ending with a final extension period of 72°C for 7 min. The PCR products were examined under UV light after electrophoresis in a 1.5% agarose gel.

PCR products were purified and sequenced using BigDye terminator chemistry on an automated ABI 3130 Sequencer (Applied Biosystem, Foster City, CA) based on the Sanger's sequencing method. Analysis of the obtained sequences was performed using Sequencher Aligner 4.1.4. The antibiotic resistance genes (ARGs) were blasted against ARG-ANNOT database.[16]

Conjugation experiment and plasmid analysis

Conjugation was conducted to verify the transferability of a putative plasmid of *S. marcescens*. Five isolates producing *armA* were chosen as donors. The *E. coli J53* azide-resistant strain was used as the recipient strain.[1] Both donor and recipient strains were cultivated in tubes containing 9 ml of Trypticase Soy Broth (Becton Dickinson and Company, Pont de Claix, France). At the exponential growth phase, 1 ml of the donor strain was transferred to the recipient strain and incubated for 24 hr. Transconjugated clones were selected after cultivation on Luria–Bertani agar (Becton Dickinson and Company) containing ceftriaxone (20 μg/ml) and azide (100 μg/ml) and then identified using MALDI-TOF MS. An *E*-test was performed to determine the MICs of the transconjugants before verification of the presence of antimicrobial resistance genes by PCR amplification as described above.

To characterize the plasmids transferred to the transconjugants, total DNA was extracted from transconjugants and the plasmids were identified based on their incompatibility group using the PCR replicon typing method as previously described.[5]

Phylogenetic analysis

Due to the unavailabilty of a standard *S. marcescens* multilocus sequencing typing scheme, in this study, we selected four housekeeping genes (*gyrB*, *rpoB*, *infB*, and *atpD*) to determine the relationships between our isolates; amplification and sequencing were performed using primers retrieved from a previous study on multilocus sequencing analysis as described.[3] The obtained sequences were concatenated, aligned with ClustalW, and then a phylogenetic tree was constructed using MEGA6 software.[40] The generated phylogenetic clustering tree was then compared to the MSP dendrogram constructed using MALDI-TOF spectra of *S. marcescens* isolates through Biotyper 3.0 software (Bruker Daltonics, Bremen, Germany).

Results

Bacterial strains and identification

The 54 strains collected during this study between December 2011 and July 2013 were correctly identified by MALDI-TOF MS, with scores ranging from 1.9 to 2.3 (Fig. 1). *S. marcescens* isolates were collected from hospitals and private laboratories, including 87% collected from urine samples and 13% from pus. A total of 30 out of 54 strains produced a dark red pigmentation (prodigiosin). The distribution of *S. marcescens* according to patient sex, age, ward, and hospital is presented in Table 1.

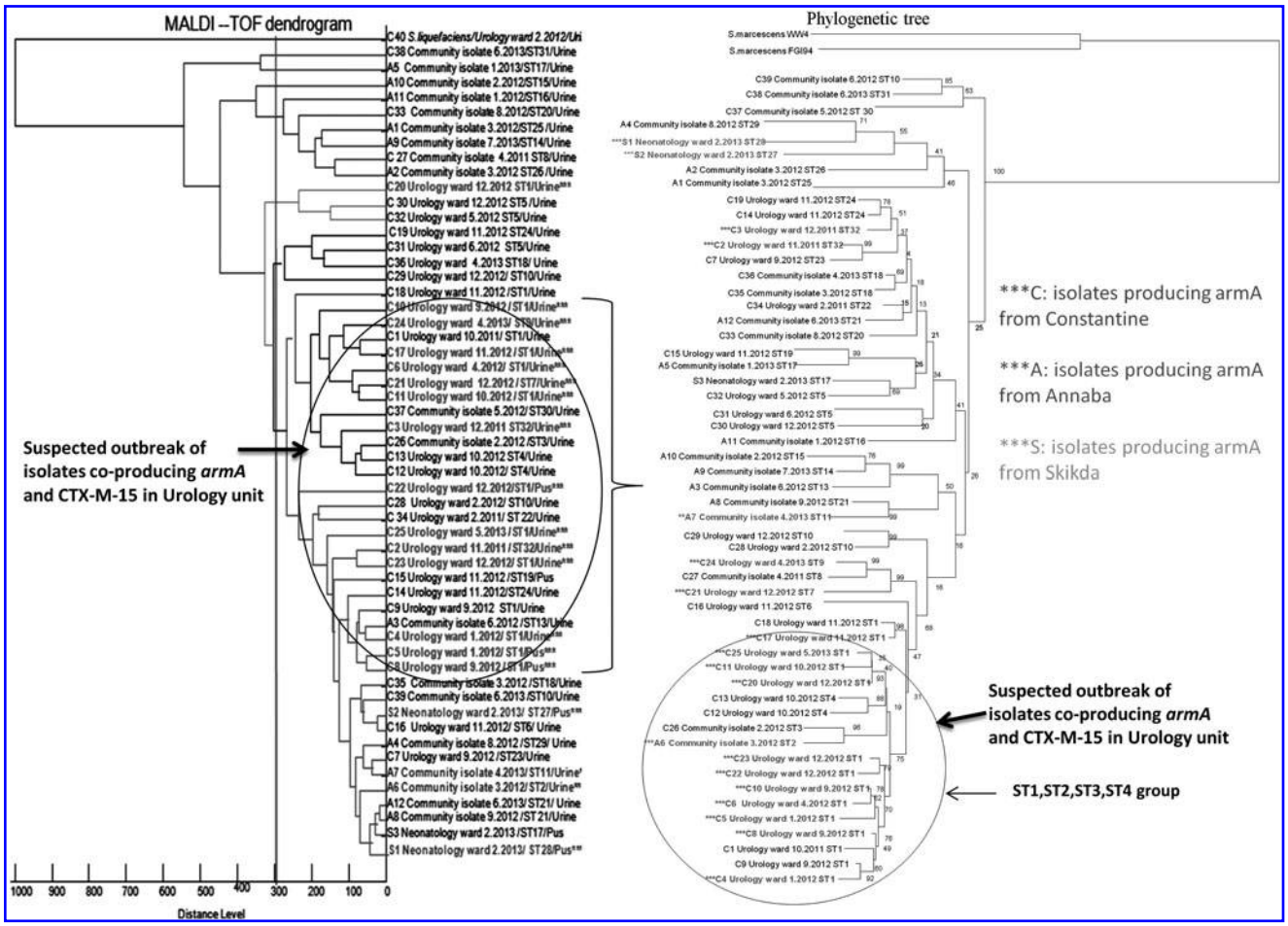

FIG. 1. MALDI-TOF dendrogram and phylogenetic tree of phylogenetic analysis of 54 isolates of *Serratia marcescens.* MSP dendrogram (*left panel*) was constructed using MALDI-TOF spectra of *S. marcescens* isolates through Biotyper 3.0 software and an arbitrary distance value of 300 was considered for clustering isolates. Phylogenetic tree of concatenated sequences of the multilocus sequence typing analysis (*right panel*) was constructed using MEGA6 software with bootstrap values of 1,000 replicates. **isolates harboring *armA*. MALDI-TOF, matrix-assisted laser desorption/ionization time-of-flight.

Antimicrobial susceptibility testing

A total of 35 out of 54 isolates of *S. marcescens* were resistant to antibiotics (65%), especially amoxicillin/clavulanic acid (100%), cefotaxime (67%), and ceftazidime (57%). MICs for ceftazidime and cefotaxime ranged from 4 to 32 μg/ml and from 12 to 256 μg/ml, respectively. High levels of resistance to fluoroquinolones and aminoglycosides were detected: 61% for gentamicin and 39% for amikacin. Twenty-four isolates were resistant to fluoroquinolones (44%), with MICs ranging from 1 to 256 μg/ml. Twenty-one pigmented isolates (39%) presented a multidrug resistance phenotype. None of the strains was resistant to imipenem (Table 2).

Screening of resistance genes

We found that 35 out of 54 resistant isolates harbored $bla_{CTX\text{-}M\text{-}15}$ ($n=32$), $bla_{TEM\text{-}1}$ ($n=26$), $bla_{TEM\text{-}71}$ ($n=1$), $bla_{SHV\text{-}1a}$ ($n=1$), and $bla_{PER\text{-}2}$ ($n=12$). None of the isolates harbored bla_{VEB} or bla_{GES}. Twenty-eight isolates presented at least one gene for resistance to aminoglycosides, including *aadA2* ($n=18$), *aadA1* ($n=2$), *armA* ($n=19$), *aac6Ib-cr* ($n=6$), and *aph(3′)VIb* ($n=1$). Finally, five isolates contained *qnr* genes: *qnrB42* ($n=3$), *qnrB6* ($n=1$), and *qnrA6* ($n=1$). Resistance profiles and ARGs of the *S. marcescens* isolates are summarized in Table 1.

Transferability and plasmid analysis

Conjugation was successful for five pigmented microbial drug resistant isolates. The antibiotic resistance profile of transconjugants corresponded to the profile of the donor. MICs of transconjugants for amikacin, gentamicin, and cefotaxime were >256 μg/ml. The presence of ARGs was verified for all transconjugants by PCR amplification as described above. As expected, the isolates contained $bla_{CTX\text{-}15}$, $bla_{TEM\text{-}1}$, *aadA2*, and *armA*.

All the transconjugants tested positive for incL/M plasmid type indicating that cephalosporin and aminoglycoside resistance genes were localized on incL/M plasmid.

Phylogenetic analysis and MALDI-TOF MS clustering

A phylogenetic tree was constructed to determine relatedness between isolates based on the concatenated sequence

Table 1. Characteristics of ESBL-Producing Aminoglycoside-Modifying Enzymes and Quinolone in *Serratia marcescens*

Sources	*Code*	*Date of isolation*	*Sex/age*	*Department*	*Sample origin*	*β-Lactamase resistance pattern*	bla *Genes detected*	*Aminoglycoside resistance pattern*	*Aminoglycoside genes detected*	*Fluoroquinolone resistance pattern*	*Quinolone genes detected*
Hopital C	C1	October 2011	M/83	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.TOB	aac6Ib-cr	CIP.OFX	qnrB42
(*n*=25)	C2	November 2011	M/30 months	Uroloogy	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1, PER-2	CN.TOB.AK	aadA2, armA	—	—
	C3	December 2011	M/1 month	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1, PER-2	CN.AK.TOB	aadA2, armA	—	—
	C4	January 2012	M/39	Urology	Urine	AMC.ATM.CTX.CRO	CTX-M-15, TEM-1	TOB.AK.TOB	aadA2, armA	CIP.OFX	—
	C5	January 2012	F/35	Urology	Pus	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.AK.TOB	aadA2, armA	CIP.OFX	—
	C6[a]	April 2012	M/67	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.AK.TOB.	aadA2, armA	OFX	—
	C7	September 2012	M/75	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	TEM-71, PER-2	CN.TOB	—	OFX	—
	C8[a]	September 2012	F/75	Urology	Pus	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.AK.TOB	aadA2, armA	CIP.OFX	—
	C9	September 2012	F/72	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.TOB	—	CIP.OFX	—
	C10	September 2012	F/68	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.TOB.AK	aadA2, armA	CIP.OFX	—
	C11	October 2012	M/58	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.TOB.AK	armA	CIP.OFX	—
	C12	October 2012	M/72	Urology	Urine	AMC.CTX	CTX-M-15	TOB	aadA2	OFX	—
	C13	October 2012	M/33	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15	TOB	—	CIP.OFX	—
	C14	November 2012	M/36	Urology	Urine	AMC.CTX.ATM.CRO	TEM-1, PER-2, SHVa2	CN.TOB.AK	—	OFX	—
	C15	November 2012	M/26	Urology	Urine	AMC.CTX	CTX-M-15, PER-2	CN.TOB	aadA1	CIP.OFX	—
	C16	November 2012	M/34	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.TOB	—	CIP.OFX	—
	C17	November 2012	F/28	Urology	Urine	CTX.CAZ.ATM.CRO	CTX-M-15, TEM-1	CN.TOB.AK	armA	CIP.OFX	—
	C18	November 2012	F/62	Urology	Urine	AMC.ATM.CTX.CRO	CTX-M-15, TEM-1	TOB	aadA2	CIP.OFX	—
	C19	November 2012	M/55	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	TEM-1, PER-2	CN.AK.TOB	aph(3′)VIb	CIP.OFX	qnrA6
	C20[a]	December 2012	M/5	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.AK.TOB	aadA2, armA	CIP.OFX	—
	C21[a]	December 2012	M/12	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.AK.TOB	aadA2, armA	CIP.OFX	—
	C22	December 2012	M/4	Urology	Pus	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.AK.TOB	aadA2, armA	CIP.OFX	qnrB6
	C23	December 2012	F/5	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1, PER-2	CN.AK.TOB	aadA2, armA	—	—
	C24[a]	April 2013	M/64	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.AK.TOB	aadA2, armA	CIP.OFX	—
	C25	May 2012	F/4	Urology	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.AK.TOB	aadA2, armA	CIP.OFX	—
Hopital S	S1	February 2013	M/2	Neonatology	Pus	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1, PER-2	CN.AK.TOB	aadA2, armA	—	—
(*n*=3)	S2	February 2013	M/2	Neonatology	Pus	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1, PER-2	CN.AK.TOB	aadA2, armA	—	—
	S3	February 2013	M/2 months	Neonatology	Pus	AMC.ATM.CTX.CAZ.CRO	CTX-M-15	CN.TOB	aac6Ib-cr	CIP.OFX	qnrB42
Community	A1	March 2012	M/58	Private laboratory	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, PER-2	CN.TOB	aac6Ib-cr	CIP	—
isolates A	A2	March 2012	M/60	Private laboratory	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1, PER-2	CN.TOB	aac6Ib-cr, aadA1	—	—
(*n*=7)	A3	June 2012	F/50	Private laboratory	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.AK.TOB	—	—	—
	A4	August 2012	M/66	Private laboratory	Urine	AMC.ATM.CTX.CRO	CTX-M-15, PER-2	CN.AK.TOB	—	CIP.OFX	—
	A5	January 2013	F/45	Private laboratory	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15	CN.TOB	aac6Ib-cr	CIP.OFX	qnrB42
	A6	March 2013	F/40	Private laboratory	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15	CN.AK.TOB	aadA2, ArmA	OFX	—
	A7	April 2013	F/65	Private laboratory	Urine	AMC.ATM.CTX.CAZ.CRO	CTX-M-15, TEM-1	CN.AK.TOB	aac6Ib-cr, ArmA	CIP.OFX	—

AK, amikacin; AMC, amoxicillin/clavulanic acid; ATM, aztreonam; CAZ, ceftazidime; CIP, ciprofloxacin; CRO, ceftriaxone; CTX, cefotaxime; CN, gentamicin; ESBL, extended-spectrum β-lactamase; F, female; M, male; OFX, ofloxacin; TOB, tobramycin.

[a]Isolates that have been conjugated in *Escherichia coli J53*.

Table 2. Resistance Rates of 54 Isolates of *Serratia marcescens*

	Resistance rate (%)		
	Hospital Constantine	*Hospital Skikda*	*Community isolates*
Antimicrobial agent	n = *39*	n = *3*	n = *12*
Amoxicillin/clavulanic acid	100	100	100
Aztreonam	77	100	100
Cefotaxime	64.1	100	58.3
Ceftazidime	59	100	58.3
Cefepime	56.4	100	58.3
Ceftriaxone	61.5	100	58.3
Gentamicin	53.8	100	58.3
Tobramicin	64.1	100	58.3
Amikacin	38.5	66.7	41.7
Ciprofloxacin	51.3	33.3	33.3
Ofloxacin	53.8	33.3	33.3
Trimethoprim/ sulfamethoxazole	64.1	100	50
Colistin	100	100	100
Imipinem	0	0	0

of the four housekeeping genes (*gyrB*, *atpD*, *rpoB*, and *infB*) (Fig. 1). We found 32 different groups, one of these refers to a group with a single locus different among isolates being associated with isolates harboring *armA* in the urology unit from Constantine between October 2011 and March 2013. Interestingly, this group corresponds to a cluster obtained in the MALDI-TOF dendrogram at the arbitrary distance value of 300 (Fig. 1).

Discussion

Dissemination of natural and acquired resistance to antibiotics in *S. marcescens* strains, especially combined resistance to ESBL and aminoglycosides, has become a remarkable concern worldwide.[25,29,30] Fifty-four *S. marcescens* isolates from private laboratories and clinical isolates were recovered between December 2011 and July 2013 and used in an epidemiological study. We found a high percentage of resistance to β-lactams and aminoglycosides, mainly due to the presence of ESBL and aminoglycoside-modifying enzyme-encoding genes. Twenty-eight out of 54 isolates (52%) from urine samples exhibited the ESBL phenotype, in agreement with previous studies showing a relationship between ESBL and urinary tract infections.[14,30] This percentage was higher than the results previously reported in Algeria by Nedjai *et al.*,[30] who showed a prevalence of $bla_{CTX\text{-}M}$ (7%) and bla_{TEM} (1%) in clinical isolates from a urology ward at Annaba in 2009 due to urinary tract infections.[30]

In our study, we confirmed that the resistance to β-lactams was mainly due to the presence of $bla_{CTX\text{-}M\text{-}15}$, a resistance gene previously reported in Algeria in *S. marcescens*.[30] In our study, we report the detection of the $bla_{PER\text{-}2}$-encoding gene, conferring the ESBL phenotype for the first time in Algeria and Africa. We identified the $bla_{TEM\text{-}71}$ gene that was detected for the first time in *S. marcescens* and was previously reported in *K. pneumoniae* in Spain.[35]

To the best of our knowledge, we report for the first time the presence of *armA* in *S. marcescens* in Algeria; this gene had only been previously reported in *K. pneumoniae* in Algeria[1] and was associated with ESBL-encoding genes localized on a transferable plasmid, described in *S. marcescens* isolates in Japan and Korea.[12,33,34] All transconjugants were found to be positive for incL/M plasmid type indicating that cephalosporin and aminoglycoside resistance genes were localized on incL/M plasmid, as already observed by Park *et al.* in Korea.[33] In addition to *armA* found in our study, we detected other aminoglycoside resistance genes: *aadA2* ($n = 18$), *aadA1* ($n = 2$), *aac6Ib-cr* ($n = 6$), and *aph(3′)VIb* ($n = 1$). However, seven strains were resistant to aminoglycosides without resistance genes detected and that will require further investigation.

Although the phylogenetic techniques used in this study are not validated in this genus, concordance between the cluster found by MALDI-TOF and phylogenetic analysis likely suggests the occurrence of a possible outbreak in the urology unit in Constantine between October 2011 and March 2013, as demonstrated by the two typing methods. However, one of the limitations of this study is the absence of a validated tool such as pulsed-field gel electrophoresis to eventually confirm the suspected outbreak. Besides this, several outbreaks of *S. marcescens* infections have been reported worldwide, including Italy, Japan, the United States, and Argentina.[6,12,24,27]

MALDI-TOF MS is increasingly used as the main tool for species identification.[2,10] Currently, some studies are focused on whether it is possible to use MALDI-TOF MS as a discriminatory tool for typing.[8,32,36] We believe the possible outbreak that occurred in this urology unit could be due to either patient-to-patient transmission or to the contamination of equipments such as the cystoscope, as previously reported in another study in Algeria[30]

This correlation suggests that MALDI-TOF MS can represent a promising tool for genotyping *S. marcescens* clinical isolates in real time and for infection control of outbreaks within hospitals, as has been confirmed for many other isolates, such as *E. coli*, *Neisseria meningitidis*, and *K. pneumoniae*.[2,8,19,32] In contrast, some studies have reported a lack of correlation between MALDI-TOF MS and genotyping for some bacterial strains.[22,36] This discrepancy may be based on differences in the principle of each technique.[2]

In conclusion, we report for the first time the co-occurrence of armA methyltransferase with ESBL in *S. marcescens* clinical isolates linked to a suspected outbreak in the urology unit in Eastern Algeria.

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Disclosure Statement

No competing financial interests exist.

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