Relationship of multidrug-resistant gene and extended-spectrum carbapenem-resistance in *Staphylococcus aureus*

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**Key words:** MRSA, BlaTEM, BlaNDM, SCCmec type II, Type IVA, Extended-spectrum carbapenems

**Abstract:** The aim of this study was to determine the relationship between phenotypic antimicrobial susceptibility patterns and extended-spectrum, carbapenem-resistance genes. A total of 109 clinical *Staphylococcus aureus* strains were subjected to 19 antimicrobial susceptibility tests. Resistance to methicillin (mecA), penicillin (blaTEM), and tetracycline (tetM) was detected. We compared the presence of the blaTEM genes with extended-spectrum, carbapenem-related genes and identified the types of SCCmec genes. Of 109 clinical *S. aureus* strains, 62 (56.88%) had methicillin resistance and 60 strains carried mecA. The prevalence of blaTEM and tetM genes was 81.65% and 37.61%, respectively. The most predominant SCCmec type was SCCmec type II 28/60 (46.67%), in 60 mecA-positive methicillin-resistant *S. aureus* (MRSA) isolates. The SCCmec prevalence rates were type IVA 30.00% (18/60), type IVb 8.33% (5/60), type IVd 6.67% (4/60), and non-typable 8.33% (5/60). Sixty of the 109 (55.05%) MRSA isolates were positive for extended-spectrum carbapenems (31/60) (51.67%), cefepim 40/60 (66.67%) and carbapenems 31/60 (51.67%). The predominant SCCmec type II demonstrated more carbapenem-resistance than the IVA, IVb and IVd types.

**Introduction**

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a prominent pathogen that causes severe infections in both healthcare and community settings (Inomata et al., 2015). MRSA in hospital settings is more prevalent in Asian countries, such as South Korea, China, and Japan, with reported rates of 70-80%, than in Europe (25.1%) (Reinert et al., 2015). In one recent study, the proportion of MRSA in healthcare-associated (HA) isolates was very high (73.3%) (Moon et al., 2014). Although the rates of community-associated (CA) MRSA infections are still very low in South Korea, the recently reported rates of MRSA isolates have been unclear identify those from CA settings (Kim et al., 2007; Moon et al., 2014).

MRSA infections are difficult to treat because they are resistant to many groups of antibiotics, such as β-lactams, tetracyclines, aminoglycosides, and macrolides. MRSA is resistant to all penicillins, including semi-synthetic penicillinate-resistant congeners, carbapenems, cephalosporins, and penems. One mechanism of penicillin resistance is the expression of penicillinase, which hydrolyzes the β-lactam ring of penicillin, inactivating it (Olsen et al., 2006).

The principal mechanism of aminoglycoside resistance in *S. aureus* is drug inactivation mediated by aminoglycoside-modifying enzymes (AMEs) encoded by various genes such as *aac(6’)-aph(2'*) and *ant(4')-Ia* (Ramirez and Tolmasky, 2010). The most prevalent AME in *S. aureus* is bifunctional enzyme AAC(6’)-APH(2’), which is encoded by *aac(6’)-aph(2’*) (Martineau et al., 2000). In addition, ANT(4’)-I, encoded by *ant(4’)-Ia*, has been found in *S. aureus* (Schmitza et al., 1999).

Meanwhile, erythromycin resistance is associated with *erm(A)* and *erm(C)* genes, and the main gene associated with the tetracycline resistance of *S. aureus* is tetM (Trzcinski et al., 2000; Varaldo et al., 2009).

*S. aureus* can acquire antibiotic resistance genes through horizontal gene transfer using mobile genetic elements, including SCCmec, plasmids, transposons, insertions, and bacteriophage (McCarthy et al., 2014). SCCmec elements are important for MRSA because they usually serve as determinants of antibiotic resistance patterns. Healthcare-associated MRSA strains usually harbor type I-III SCCmec elements that confer multidrug resistance (MDR) (Uhlemann et al., 2014), while community-associated strains...
are generally non-MDR strains carrying small SCCmec elements, most of which are types IV and V (Kondo et al., 2007; Orlin et al., 2017).

The objective of this study was to evaluate the relationship between phenotypic antimicrobial susceptibility patterns and extended-spectrum carbapenem genes in isolated bacterial strains. We also compared the prevalence of genes with SCCmec resistance with βlTET and extended-spectrum carbapenem antibiotic resistance among isolated clinical S. aureus strains.

**Material and Methods**

**Bacterial isolates**

A total of 109 S. aureus strains were obtained from clinical patients at Gachon University Gil Medical Center in South Korea between April 2016 and June 2018. The research was approved by the Ethics Committee of Gil Hospital, Gachon University of Medicine. The S. aureus strains were streaked onto sheep blood agar (Sinyang Diagnostics, Seoul, Korea) and transported to our laboratory after culture. One colony was picked from each blood agar plate and incubated in lysogeny broth with shaking (80 rpm) at 37°C overnight. The isolates were preserved in 20% glycerol (vol/vol) and stored at -80°C until further use.

**Antimicrobial susceptibility testing**

Antimicrobial susceptibility was determined using the Kirby-Bauer disc diffusion method described by the Clinical and Laboratory Standard Institute (CLSI) guidelines, 2015 (Wayne, 2015). Each bacterial suspension was adjusted to McFarland 0.5 turbidity, swabbed onto lysogeny broth agar, and incubated in the presence of antibiotic discs at 37°C for 18 h. We tested the following 19 antibiotic Liofilchem discs (Liofilchem, Roseto degli Abruzzi, Italy): penicillin G (10 IU), methicillin (5 μg), kanamycin (30 μg), gentamicin (10 μg), streptomycin (10 μg), tetracycline (30 μg), erythromycin (15 μg), vancomycin (30 μg), chloramphenicol (30 μg), amoxicillin (25 μg), ticarcillin (75 μg), piperacillin (100 μg), cefepime (30 μg), cefotaxime (30 μg), ceftazidime (30 μg), imipenem (10 μg), ertapenem (10 μg), and meropenem (10 μg). The diameters of inhibition zones were measured, and each isolate was identified as resistant or susceptible to the antimicrobial agents based on CLSI guidelines. We obtained the S. aureus control strain Staphylococcus aureus ATCC 29213 from the Korean Culture Center of Microorganisms (Seodaemun-gu, Seoul, Korea).

**TABLE I**

| Primers | Oligonucleotide sequence (5’→3’) | Amplicon size (bp) | Specificity | Reference | GenBank no. |
|---------|---------------------------------|-------------------|------------|-----------|-------------|
| MecA147-F | GTCAGATATACAACTGATT | 147 | mecA | Zhang |
| MecA147-R | ATGCCTATAGATGAAAGGAT | | | |
| Type I-F | GCTTTAACGATGTTTACAGG | 613 | SCCmec I | Zhang |
| Type I-R | GTCTCTCATAGATGACGGC | | | |
| Type II-F | CGTGTAGATGTAAGGCG | 398 | SCCmec II | Zhang |
| Type II-R | CGAATCTGATGTTAACTGAC | | | |
| Type III-F | CCTATGTTTACAGATCG | 280 | SCCmec III | Zhang |
| Type III-R | CCTATGTTTACAGATCG | | | |
| Type IVa-F | GCCTTATCGAGAAACCG | 766 | SCCmec | Zhang |
| Type IVa-R | CTACTCTTCTGAAAAAGGGTGG | | | |
| Type IVb-F | TCTGGAATTACTTCAGCTGC | 493 | SCCmec | Zhang |
| Type IVb-R | AAACAGATTTGCTCCTCATC | | | |
| Type IVc-F | ACAATTTTTGATATCCGAGAGC | 200 | SCCmec | Zhang |
| Type IVc-R | TGGTATGAGGATTGCTGG | | | |
| Type IVd-F | CTCAAAATCGACCCCAATACA | 881 | SCCmec | Zhang |
| Type IVd-R | TGGCTCAGTATCTGAAAG | | | |
| Type V-A-F | TTACCTGCTTTTATGAATTGTA | 1752 | SCCmec | This study EU437549.2 |
| Type V-A-R | ACAATGATCCGAAATGACGCTGTA | | | |
| Type V-F | GAAATTTGACTTAAATGACG | 325 | SCCmec V | Zhang |
| Type V-R | TGGAAATGACTACCCCTGCACCC | | | |
Genomic DNA isolation
Genomic DNA was isolated after alkaline cell lysis, phenol-chloroform DNA extraction, and ethanol DNA precipitation (Sambrook and Russell, 2006). A single colony was picked from each blood agar plate and incubated in lysogeny broth at 37°C overnight. Then, 1.5 mL of the bacterial suspension was harvested by centrifugation at 14000 rpm for 30 s. The harvested bacterial pellet was proceeded protocol alkaline phenol-chloroform method using fresh tubes and phenol-chloroform (1:1) solution (Bioneer, Daejeon, Korea). The DNA pellet was then dissolved in 30 μL autoclaved tri-distilled water. DNA concentrations were determined using a NanoDrop™ spectrophotometer (Thermo Fisher Scientific, Waltham, MA, USA).

Identifying mecA, blaTEM and SCCmec genes by multiplex real-time PCR
The PCR primers used to detect the mecA and blaTEM genes are listed in Tabs. 1 and 2 (Strommenger et al., 2003; Zhang et al., 2005; Varaldo et al., 2009). The following reaction mixture was added to each sample: 10 pmol of each primer, 2 μL DNA (100 ng), and 10 μL iQ™ SYBR Green supermix (2X reaction buffer with dNTPs, iTaq DNA polymerase, SYBR® Green I, fluorescein, and stabilizers (Bio-Rad, Hercules, CA, USA)). The volume was adjusted to 20 μL by adding autoclaved triple-distilled water. The PCR cycling conditions on a thermal cycler (iQ5, Bio-Rad and TC-512, TECHNE, Cambridge, UK) were as follows: 94°C for 3 min, followed by 35 cycles of denaturation at 94°C for 30 s, annealing at 56°C for 30 s, and extension at 72°C for 45 s. The reaction was ended with a final extension step at 72°C for 10 min. Multiplex PCR was carried out for SCCmec typing using nine pairs of primers specific for SCCmec types I, II, III, IVa, IVa, IVb, IVc, IVd, and V (Zhang et al., 2005). The PCR products were subjected to electrophoresis using 2% agarose gel in 1X TBE buffer at 100 V for 25 min. A 100 bp DNA ladder (Bioneer, Daejeon, Korea) was used as a molecular size marker. The PCR products in gels were then visualized with Safe Green loading dye (Applied Biological Materials, Inc, Vancouver, BC, Canada).

Detecting antibiotics resistance genes
PCR was performed to detect genes associated with antimicrobial resistance. The oligonucleotide primers sequences and specific genes are listed in Tab. 2 (Strommenger et al., 2003; Varaldo et al., 2009). The examined genes were the tetracycline resistance gene (tetM), aminoglycoside-modifying enzyme (AME) genes (ant4'-Ia, aac6'-aph2''), and macrolide resistance genes (ermA, ermC). These products were determined the existence of PCR result and DNA sequencing.

Results
We tested for antimicrobial susceptibility using Kirby-Bauer disc diffusion and determined the isolates to be resistant or susceptible to the antimicrobial agents based on the diameters of the inhibition zones. The susceptibility testing showed that 56.88% (62/109) of the S. aureus strains were resistant to methicillin. The results also showed high rates of susceptibility to chloramphenicol 107/109 (98.16%) and vancomycin 107/109 (98.16%), however, most of the S. aureus strains were resistant to streptomycin 70/109 (64.22%) and penicillin 71/109 (65.14%). The overall rates of resistance to kanamycin, gentamicin, erythromycin, and tetracycline were 54.13%, 52.29%, 37.61%, and 26.61%, respectively (Tab. 3).

| Antibiotic       | Primer   | Oligonucleotide sequence | Amplicon specific gene | Reference | GenBank. |
|------------------|----------|--------------------------|------------------------|-----------|----------|
| β-lactams        | TEM-F    | GCA CGA GTG GTG TAC ATC GA | 311                    | blaTEM    | This study NG_050162.1 |
|                  | TEM-R    | GGT CCT CGG ATC GTG GTG AG | 200                    | etEM      | This study LS483319.1 |
| Tetracyclines    | tet(M)-F | GGT TGG AAT GTG AGC GAC TG | 200                    | tetM      | This study LS483319.1 |
|                  | tet(M)-R | ATC GTT GTA TGC TCG TGA AAG A | 390                  | ant(4')-Ia | This study CSp19563.1 |
| Aminoglycosides  | kan-F    | GAA GCA GAG TAC AGC CAT GA | 222                    | aac(6')-aph(2') | Strommenger |
|                  | kan-R    | CGA AGC GCT CGT CGT ATA AC | 222                    | aac(6')-aph(2') | Strommenger |
| Macrolides       | erm(A)-F | AAG CGG TAA ACC CCT CTG A | 199                    | ermA      | Varaldo  |
|                  | erm(A)-R | ACAATGATGGACAATGACGTGGA | 299                    | ermC      | Varaldo  |
|                  | erm(C)-F | AAT CGT CAA TCC CTG CAT GT | 299                   |           |          |
|                  | erm(C)-R | TAA TCG TGG AAT AGC GGT TTG | 299                   |           |          |
### TABLE 3

| Antibiotic               | Resistant strains No. (%) | Susceptible strains No. (%) | PCR positive strains No. (%) | PCR negative strains No. (%) |
|--------------------------|---------------------------|----------------------------|-----------------------------|-----------------------------|
| Methicillin              | 62 (56.88%)               | 47 (43.12%)                | mecA 60 (55.05%)            | mecA 49 (44.95%)            |
| Penicillin G             | 71 (65.14%)               | 38 (34.86%)                | blaTEM 89 (81.65%)          | blaTEM 20 (18.35%)          |
|                          |                           |                            | ant(4')-Ia 32 (29.36%)      |                             |
| Kanamycin                | 59 (54.13%)               | 50 (45.87%)                | ant(4')-Ia/ant(6')-aph(2') 12 (11.01%) | ant(4')-Ia/ant(6')-aph(2') 32 (29.36%) |
|                          |                           |                            | total 52 (47.41%)           |                             |
| Erythromycin             | 41 (37.61%)               | 68 (62.36%)                | ermC2 1 (1.33%)             | ermC73 (66.97%)             |
|                          |                           |                            | total 36 (33.03%)           |                             |
| Gentamicin               | 57 (52.29%)               | 52 (47.71%)                | ant(6')-aph(2') 32 (29.35%) | ant(6')-aph(2') 77 (70.64%) |
| Tetracycline             | 29 (26.61%)               | 80 (73.39%)                | tetM 41 (37.61%)            | tetM 68 (62.39%)            |
| Streptomycin             | 70 (64.22%)               | 39 (35.78%)                |                             |                             |
| Vancomycin               | 2 (1.83%)                 | 107 (98.16%)               | vanA, vanB (not detected)   |                             |
| chloramphenicol          | 2 (1.83%)                 | 107 (98.16%)               |                             |                             |

**FIGURE 1.** mecA, laTEM, ant(4')-Ia, and ant(6')-aph(2') detection by polymerase chain reaction (PCR). The PCR results were visualized on 2% agarose gel stained with Safe Green loading dye. Lane M, 100 bp DNA ladder. (a) Multiplex PCR detected line no 1-3, mecA (147 bp) and laTEM (311 bp), (b) line no 4, 6, 8: AME genes ant(4')-Ia (390 bp), and line 2, 3: ant(6')-aph(2') (222 bp), line 1, 5, 7 was not detected in S. aureus strains. (c) Multiplex PCR detected line no 2, 3: tetM (200bp), line 1 was not detected in S. aureus strains. (d) Multiplex PCR forSCCmec typing, Lane M:100 bp DNA ladder; Lane 1: type IVa (881 bp) SCCmec-positive strain; Lanes 2, 3, 5, 8: type II (398 bp) SCCmec-positive strains; Lanes 4, 6: type IVA (1752bp) SCCmec-positive strains; Lane 7: non-typable S. aureus strains.
The susceptibility testing also showed that 81/109 (74.3%) of the S. aureus strains were susceptible to amoxicillin (AML) and resistance to piperacillin 42/109 (38.53%) and cefotaxime 27/109 (24.77%) were found, as well. Tab. 3 displays the correlations between methicillin-resistance and the presence of mecA. A total of 62 MRSA strains were resistant to methicillin, 60 strains were mecA-positive, and two strains were mecA-negative (Tab. 3). Forty-seven (43.12%) strains of S. aureus were susceptible to S. aureus-negative positive, and two strains were mecA-negative based on disc diffusion, and 89 of them were positive for blaTEM (Tab. 3, Fig. 1(A)).

Tab. 3 shows the correlations between kanamycin resistance and the presence of ant(4')-Ia and aac(6')-aph(2') genes in S. aureus. A total of 52/109 (47.41%) strains carried at least one of the genes. Fifty-nine S. aureus strains were resistant to kanamycin, including 52 that carried resistance genes, and 12 strains were positive for ant(4')-Ia and aac(6')-aph(2') by PCR. Fifty-seven (52.29%) of the S. aureus strains were resistant to gentamycin as determined by disc diffusion, and 32 of these were positive for aac(6')-aph(2') (Tab. 3).

Correlations between erythromycin resistance and the presence of ermA and ermC are summarized in Tab. 3. A total of 41 (37.61%) S. aureus isolates were resistant to erythromycin determined by disc diffusion, including 34 that were positive for ermA and two that carried ermC (Tab. 3). Tetracycline-resistance correlated with the presence of tetM (Tab. 3). Twenty-nine (26.61%) S. aureus strains were resistant to tetracycline in the susceptibility tests, but 41 were positive for tetM by PCR (Tab. 3, Fig. 1(B)).

We used multiplex PCR to determine the SCCmec types in 60 mecA-positive strains (Fig. 1(C)). The predominant type was SCCmec type II 28/60 (46.67%). The prevalence of type IVA, type IVb, type IVd, and non-typable were 30.00% (18/60), 8.33% (5/60), 6.67% (4/60), and 8.33% (5/60), respectively, by multiplex PCR.

The correlation between carbapenem-resistance and the presence of SCCmec types is shown in Tab. 4. A total of 28/60 (46.67%) SCCmec type II strains were resistant to piperacillin 21/28 (75.00%), cefotaxime 22/28 (78.57%), and imipenem 22/28 (78.57%), and 18/60 (30.0%) SCCmec type IVA strains were resistant to piperacillin 11/18 (61.11%), cefotaxime 9/18 (50.00%), and imipenem 5/18 (27.78%). Fourteen (14/60, 23.33%) IVb, IVd, and non-typable strains were resistant to ticarcillin 5/14 (35.71%), cefepime 5/14 (35.71%), and meropenem 3/14 (21.42%) (Tab. 4). SCCmec type II had higher carbapenem-resistance than the IVA, IVb, IVd, and not-typable strains.

Discussion

The present study compared the results of antimicrobial susceptibility determined for S. aureus strains (Tab. 3). Although the results of the present study showed an almost perfect correlation between phenotypic methicillin susceptibility and mecA, two strains presented discrepancies between the genotype and phenotype, as did two methicillin-resistant mecA-negative strains. Previous researchers have reported that S. aureus isolates carrying mecA were sensitive to oxacillin, and thus, mecA might be heterogeneously expressed, and some S. aureus strains carrying mecA might

### Table 4

| Antibiotics | mecA-positive MRSA (n=60) |
|-------------|----------------------------|
|              | type II (n = 28, 46.67%) | type IVA (n = 18, 30.00%) | IVb, IVd, Non-typable (n = 14, 23.33%) |
|             | Susceptible number (n, %) | Resistance number (n, %) | Susceptible number (n, %) | Resistance number (n, %) | Susceptible number (n, %) | Resistance number (n, %) |
| Amoxicillin | 22 (78.57%)               | 6 (21.43%)               | 15 (83.33%)               | 3 (16.67%)               | 8 (57.14%)               | 6 (42.86%)               |
| Ticarcillin | 9 (32.14%)                | 19 (67.86%)              | 14 (77.78%)               | 4 (22.22%)               | 9 (64.29%)               | 5 (35.71%)               |
| Piperacillin| 7 (25.00%)                | 21 (75.00%)              | 7 (38.89%)                | 11 (61.11%)              | 6 (42.86%)               | 8 (57.14%)               |
| Cefepime    | 6 (21.43%)                | 22 (78.57%)              | 8 (44.44%)                | 10 (55.56%)              | 9 (64.29%)               | 5 (35.71%)               |
| Cefotaxime  | 6 (21.43%)                | 22 (78.57%)              | 9 (50.00%)                | 9 (50.00%)               | 8 (57.14%)               | 6 (42.86%)               |
| Cefazidine  | 6 (21.43%)                | 22 (78.57%)              | 7 (38.89%)                | 11 (61.11%)              | 7 (50.00%)               | 7 (50.00%)               |
| Imipenem    | 6 (21.43%)                | 22 (78.57%)              | 13 (72.22%)               | 5 (27.78%)               | 11 (78.57%)              | 3 (25.43%)               |
| Ertapenem   | 7 (25.00%)                | 21 (75.00%)              | 13 (72.22%)               | 5 (27.78%)               | 10 (71.43%)              | 4 (27.57%)               |
| Meropenem   | 6 (21.43%)                | 22 (78.57%)              | 13 (72.22%)               | 5 (27.78%)               | 11 (78.57%)              | 3 (25.43%)               |
not be detectable using phenotypical methods (Kolbert et al., 1998; Martineau et al., 2000). The possibility of selecting resistant cells from originally-susceptible strains has been demonstrated. Some strains do not express mecA unless they are provided with selective pressure via increasing gradients of the antibiotic agent (Baym et al., 2016). The second case of discrepancy occurred in two mecA-negative S. aureus strains that were phenotypically-resistant to methicillin, but the mecA gene was not detected in these isolates. Further investigations into these two isolates are on-going (our result type mecC). Penicillin-resistance in S. aureus has been reported to be mediated by the expression of penicillinase encoded by blaZ, which hydrolyzes the β-lactam ring and contributes to the inactivation of penicillin (Bradford PA, 2001; Olsen et al., 2006; Xu et al., 2014; Ferreira et al., 2017).

However, the results of other investigations between penicillin-resistance and blaTEM were unclear. Our PCR results showed that 81.65% (89/109) of the S. aureus strains carried blaTEM- blaTEM is known to encode a series of class A plasmid-mediated enzymes belonging to the extended-spectrum β-lactamases that are associated with penicillin resistance and are frequently present in Klebsiella pneumoniae and Escherichia coli (Chong et al., 2011; Dahms et al., 2015). Recent clinical trial results demonstrated the efficacy of beta-lactams and carbapenems against S. aureus (Lee et al., 2018; Saeki et al., 2018).

The S. aureus strains in this study were analyzed for the presence of AME genes ant(4')-Ia and aac(6')-aph(2") by multiplex PCR. In accordance with previous studies, aac(6')-aph(2") was the most prevalent AME gene encoded in S. aureus (Martineau et al., 2000). The aac(6')-aph(2") gene encodes a bifunctional enzyme AAC(6')-APh(2") that catalyzes both acetyltransferase and phosphotransferase reactions, thereby inactivating an extensive range of clinically-useful aminoglycosides (Ramirez et al., 2010). In this study, strains that harbored ant(4')-Ia were resistant to kanamycin, and all strains that carried aac(6')-aph(2") were clearly resistant to gentamicin and kanamycin in susceptibility testing. Some S. aureus strains were phenotypically-resistant to kanamycin, including three that showed kanamycin resistance in susceptibility testing but did not carry ant(4')-Ia or aac(6')-aph(2") genes.

The S. aureus strains were also analyzed for the prevalence of erythromycin-resistance genes ermA and ermC. The S. aureus strains carried at least one of these erm genes, and the majority carried ermA. Our results were consistent with findings from previous studies reporting that the incidence of ermA was higher than that of ermC in clinical S. aureus strains (Martineau et al., 2000). However, a higher prevalence of S. aureus carrying ermC in Denmark has been reported (Westh et al., 1995). The five samples with discrepant results between resistance genotypes and phenotypes indicated that some strains might have harbored other erythromycin-resistance genes. The prevalence of phenotypic tetracycline-resistance in 12 S. aureus samples was different than the tetM results. These discrepancies also suggest that some strains might harbor tetracycline-resistance genes and the diameters of inhibition zones ≤ 13 mm were incorrectly measured.

In the present study, we evaluated the prevalence of different types of SCCmec by multiplex PCR. Commonly, HA-MRSA strains carry SCCmec types I-III with multidrug resistance, while CA-MRSA strains harbor types IV and V. Previous research in South Korea reported that SCCmec type II was the most prevalent among the HA-MRSA strains, while SCCmec type IVA was predominant in the CA-MRSA strains, however, another study reported a higher prevalence of type IV (Kim et al., 2007; Park et al., 2007). Excessive therapeutic use of antimicrobial agents in hospital environments might have contributed to the development of resistance and the widespread distribution of SCCmec type II MRSA strains.

Conclusions

This study showed that SCCmec type II was more predominant than type IV. A total of 22/28 SCCmec type II strains were resistant to imipenem, and five SCCmec type IVA strains were resistant to imipenem. The other SCCmec type IVb, type IVd, and non-typable strains showed lower resistant to imipenem. The SCCmec type II strains had higher carbapenem-resistance than type IVA strains.

Conflict of Interest

The authors declare that they have no conflicts of interest.

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