Characterization of the *Staphylococcus aureus* strains associated with food poisoning in Shenzhen, China

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Abstract

To characterize the isolates of *Staphylococcus aureus* that were associated with staphylococcal food poisoning between 2006 and 2009 in Shenzhen, Southern China, a total of 52 *Staphylococcus aureus* isolates from 11 outbreaks were analyzed using multilocus sequence typing (MLST), spa typing, and pulsed-field gel electrophoresis (PFGE). PCR analysis was used to analyze the staphylococcal enterotoxin (*se*) genes *sea* to *sei*, and antimicrobial susceptibility testing was also performed. ST6 was the most dominant ST type, constituting 63.5% (34/52) of all of the isolates in 7 outbreaks. The next most common ST type was ST943, which constituted 23.1% (12/52) of the isolates that were collected from 3 outbreaks. T701, t091, and t2360 were the most predominant spa types, constituting 67.3% (35/52) of the isolates that were collected from 11 outbreaks. Three PFGE types, type (A, B, and C) were the most frequently observed types, constituting 84.6% (44/52) of all of the isolates. The most frequent enterotoxin gene that we detected was *sea* (45/52, 86.5%). Four *se* gene profiles were observed, including *sea* (n=45), *sec-seh* (n=3), *seb* (n=2), and *seg-sei* (n=2). With respect to antibiotic resistance, penicillin resistance was the most common (96.2%, 50/52), followed by resistance to tetracycline (28.8%, 15/52). Approximately 30.8% (16/52) of the isolates were resistant to at least two antibiotics, and 7.7% (4/52) of the isolates were resistant to three or more drugs. The two predominant *S. aureus* lineages, (1) PFGE types A and B with ST type
ST6 and (2) PFGE type C with ST type ST943, were identified in the outbreaks.
Introduction

Staphylococcal food poisoning (SFP) is a frequent cause of food-borne gastroenteritis worldwide (15, 18, 26, 34). Between 2008 and 2010, a total of 371 outbreaks of bacterial foodborne diseases were reported in China, involving 20,062 individuals and leading to 41 deaths. Ninety-four outbreaks of SFP were reported to the National Monitoring Network between 2003 and 2007, involving 2,223 individuals and leading to 1,186 hospitalizations. S. aureus was the fifth most frequently observed pathogen after Vibrio parahaemolyticus, Bacillus cereus, Bacillus proteus, and Salmonella (17). In Shenzhen, eleven outbreaks of SFP were reported to the local monitoring network between 2006 and 2009, representing the second most frequent cause of bacterial food poisoning after Vibrio parahaemolyticus. Because most SFP cases are mild, the actual number of SFP cases is expected to be much higher than is reported (18).

SFP is associated with the toxinogenic S. aureus strains that express one or more of a family of genes that code for heat-stable enterotoxins (1). These genes share a common genetic relationship, structure, and function, and have a high degree of sequence homology (1). In addition to functioning as potent gastrointestinal toxins, staphylococcal enterotoxins (SE) also act as superantigens that stimulate non-specific T-cell proliferation, which can potentially cause toxic shock (1). A standard nomenclature was proposed such that only toxins that induce emesis following oral administration in a primate model are
designated as SEs. Otherwise, the toxins are referred to as staphylococcal enterotoxin-like superantigens (SAgs) (16). In addition to the five classical types of SEs (SEA through SEE), sixteen more recently described SEs or SE-like toxins (SEG through SEV) have been described (15, 20, 22, 29, 30).

To understand the epidemiology, population biology and genetic diversity of enterotoxinogenic *S. aureus*, we employed the typing methods that have been previously been used to characterize hospital- and community-acquired *S. aureus* infections. To our knowledge, there have been few molecular epidemiologic investigations of *S. aureus*-associated SFP in China. The aim of this study was to use molecular epidemiology to both characterize *S. aureus*-associated SFP and to improve our understanding of the genetic relatedness of the more pathogenic strains. These results may provide insight into the spread of the isolates that are associated with outbreaks and may ultimately improve the control of SFP in Southern China.

**MATERIALS AND METHODS**

The isolates and the patient data were collected between 2006 and 2009 from 11 outbreaks that were reported in Shenzhen, Guangdong Province, Southern China. All of the isolates were identified at the species level by coagulase production using the SlideX Staph Plus kit (Murex Biotech, Kent, France) and PCR for the *nuc* gene (2). The SFP diagnosis was confirmed by any of the following: (i) the detection of SEs in leftover food, (ii) the isolation of *S. aureus* with the same
enterotoxin type from both food and patients, and (iii) the isolation of *S. aureus* with the same enterotoxin type from different patients. An outbreak was defined by the identification of more than two epidemiologically associated cases.

**Molecular typing**

All of the isolates were characterized using pulsed-field gel electrophoresis (PFGE) and *spa* typing. Multilocus sequence typing was performed for eight isolates that included representatives of each *spa* type. PFGE was performed using the CHEF-DR III System (Bio-Rad, United States), as described previously (37). The digital images were analyzed by BioNumerics software (v. 5.10, Applied Maths) using the Dice coefficient and were generated by UPGMA with 1.5% tolerance and 1% optimization settings. A similarity cutoff of 80% and a difference of 6 bands were used to define a cluster, as described by Tenover (28). The isolates that exhibited identical or related PFGE patterns were considered to belong to the same clone. The clones were labeled with capital letters (A, B, C), and related profiles were indicated by adding a number (A1, A2, B1, B2, etc.).

The *Spa* and the MLST typing were performed as previously described (7, 10). Based Upon Repeat Pattern (BURP) analysis was used to cluster (*spa-CC*) the *spa* types (19).

**Identification of the SE genes using PCR**

The genomic DNA was extracted using the DNeasy blood and tissue kit (Qiagen GmbH, Hilden, Germany). The amplifications were performed using a
Mycycler™ thermal cycler (Bio-Rad, Hercules, California, United States). The sea, seb, sec, sed, see, seg, seh, and sei genes were detected according to the methodology of previous studies (24, 25).

Antimicrobial susceptibility

A total of 18 antimicrobial agents were tested, including penicillin G, cefoxitin, oxacillin, piperacillin-tazobactam, ampicillin-sulbactam, cefazolin, vancomycin, teicoplanin, clindamycin, erythromycin, tetracycline, minocycline, ciprofloxacin, chloramphenicol, rifampin, gentamicin, trimethoprim-sulfamethoxazole and quinupristin-dalfopristin. All of the antimicrobial agents were tested using disk diffusion (OXOID, Basingstoke, England). The CLSI zone diameter breakpoints were used to interpret the antimicrobial susceptibility of the analyzed strains.

Results

Epidemiological data and isolates

A total of 11 food poisoning outbreaks were reported between Jan 10, 2006 and Aug 24, 2009 (Table 1). Seventy-nine individuals were reported to be ill and suffered from abdominal pain (n=64), diarrhea (n=62), nausea (n=55), vomiting (n=45), giddiness (n=24) and headache (n=15). Seven outbreaks occurred in private households, two outbreaks occurred at dining halls, and the other two outbreaks occurred at a restaurant and a supermarket. The incubation period ranged from 1.5 to 11.5 hours. The S. aureus isolates that produced SEs were isolated from the food leftovers in nine outbreaks and were isolated from the
patients and from the environment in another two outbreaks. A total of 52 S.
aureus isolates were isolated from food (n=27), rectal swabs (n=15), feces (n=5),
the environment (n=4) or the hand swab of a food handler (n=1) (Table 2).
Twenty-six isolates were collected in 2006, twenty-three isolates were collected in
2007, two isolates were collected in 2008 and one isolate was collected in 2009.

MLST, spa and PFGE

Multilocus sequence typing for each of the identified spa types revealed five ST
types, namely ST1, ST5, ST6, ST188 and ST943 (Table 2). ST6 was the most
dominant ST type that was observed and was identified in 34 (63.5%) of the
isolates from 7 outbreaks. Twelve isolates from 3 outbreaks (23.1%) were found
to be ST943. Spa typing of all of the isolates yielded eight spa types and 1 spa-CC
(Table 2). T701, t091 and t2360 were the most predominant spa types,
constituting 67.3% (35/52) of all of the isolates in 11 outbreaks. The fifty-two
isolates were also typed using PFGE (Table 2). The genetic analysis revealed ten
different PFGE banding patterns (A through G) and six clusters (1 through 6)
(Figure 1). Three PFGE types (A, B, and C) were the dominant types, constituting
84.6% (44/52) of all of the isolates.

There were two outbreaks (1 and 2) for which two ST, spa and PFGE types
were observed in the same outbreak, namely ST6: t701: A1 and ST943: t091: C
(ST: spa: PFGE). A very similar clone to ST943: t091: D was also identified in
outbreak 11. Outbreaks 5 and 9 exhibited the same ST and spa types (ST6: t701)
but exhibited different PFGE types (PFGE B2 and A2). The ST6 type was also identified in outbreaks 4, 6 and 8, with different spa and PFGE types (t5777: A1/B1, t5593: A1, and t2360: B1, respectively). The ST1: t127: F, ST5: t954: G1/G2, and ST188: t189: E strains were only observed in outbreak 3, 7 and 10, respectively.

Exotoxin genes

The most frequently identified endotoxin gene was sea (45/52, 86.5%), followed by sec (4/52, 7.7%), seb (2/52, 3.8%), seh (3/52, 5.8%), seg (2/52, 3.8%) and sei (2/52, 3.8%). Four se gene profiles were observed, namely sea (n=45), sec-seh (n=3), seb (n=2), seg-sei (n=2). Seven outbreaks (1, 2, 4, 5, 6, 8 and 10) with the sea gene profile were identified. Outbreaks 3 (sec-she) and 7 (seg-sei) occurred in 2006 and 2007, each. The PFGE patterns were strongly correlated with se gene profiles (Figure 1).

Antibiotic resistance

Only two strains were observed to be susceptible to all of the drugs that were tested in this study (Table 2). No MRSA strains were detected. Penicillin resistance was the most commonly observed resistance (96.2%, 50/52) of the tested strains, followed by tetracycline resistance (28.8%, 15/52). Erythromycin and clindamycin resistance were observed less frequently (7.7%, 4/52). Two strains were found to be resistant to rifampin. Sixty-five percent of all of the isolates (34/52) were only resistant to PEN, followed by resistance to PEN-TC.
(11/52, 21.2%). Approximately 30.8% (16/52) of the isolates were resistant to at least two antibiotics, and four strains were resistant to three or more drugs.

**DISCUSSION**

In this study, the two predominant *S. aureus* lineages were identified, corresponding to (1) PFGE types A and B with the ST type ST6 and (2) PFGE type C with the ST type ST943. To our knowledge, this study is the first description of the genetic diversity of *S. aureus* isolates that have been associated with the food poisoning outbreaks that occurred between 2006 and 2009 in Shenzhen, Southern China.

Based on our results, of the five sequence types (ST1, ST5, ST6, ST188 and ST943), ST6 was the most dominant clone in Shenzhen, China in this time period. T701 (ST6) and t189 (ST188) were also observed among the SFP isolates in the isolates from two outbreaks in Ma’anshan, Anhui Province (33). However, in South Korea, the ST1, ST59, and ST30 strains were the clones that were most frequently associated with SFP (3). Within the MLST database, prior to 2004, all of the isolates with the ST type ST6 were methicillin-susceptible *Staphylococcus aureus* (MSSA) from Australia, the United Kingdom, Thailand, Japan and Gambia. The ST6 MRSA strain has rarely been isolated, but community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) clones were reported in Kuwait hospitals (32), Japan (12) and Lebanon (31). According to previous research, ST6 is not the predominant *S.aureus* lineage that is observed in
hospitals and animals in China (6, 8, 9, 35, 36, 38, 40, 41). Why ST6 MSSA
became the dominant ST type that causes SFP and whether ST6 was the unique
ST type in food products or among SFP isolates in China should be investigated
further.

Based on phylogenetic inference, the outbreaks that were analyzed in this
study were caused by several similar clones (Fig 2). The ST6-t701-A1 clone had
the same ST and spa types as did the ST-T701-A2 and ST-t701-B2 clones. The
PFGE pattern indicated several bands that differ between these strains,
suggesting certain large-scale changes in the accessory genome. The same
situation was observed with respect to the ST943-t091-C and ST943-t091-D
clones. Phylogenetic inference suggests that ST6-t701-A1/A2/B2 are the
ancestors of ST6-t5777-A1/B1 and ST6-t2360-B1, which separately acquired two
spa repeats (r24 and r25) or clonally related PFGE subtype changes. Based on
BURP analysis results, ST6-t701-A1 is also the ancestor of ST6-t5593-A1, which
acquired one spa repeat (r19) and lost another (r25). This suggested temporal
relationship should be confirmed by studies that include the analysis of additional
isolates. Further studies are required to elucidate the transmission routes of
*S. aureus* strains that are associated with SFP and to provide the tools that should
be used to prevent the spread of SFP.

**PFGE, spa** and **MLST** typing were used for the molecular epidemiological
investigation of the isolates in this study. PFGE is a technique that is still widely
used...
used for *S. aureus* isolate typing, primarily because of its excellent discriminatory power, especially with respect to the analysis of short-term epidemiology, despite the difficulty when comparing the results obtained in different laboratories (21).

Our results revealed 10 patterns that can be classified by PFGE and 8 types that can be classified by spa typing. Based on PFGE analysis, the PFGE pattern of the outbreak strains were identical within each outbreak, except for outbreaks 1 and 2, which were caused by a mixed clones. In two outbreaks (4 and 7), two similar PFGE patterns were detected in each outbreak, whereas the spa and MLST types remained indistinguishable. PFGE was more effective than sequencing with respect to identification capability. However, there were three spa types (t701, t5593, and t5777) from three outbreaks with the same PFGE type (A1). Two spa types (t2360 and t5777) were also detected in the two outbreaks that were correlated with the PFGE type B1. We could not identify any epidemiological relationship between these outbreaks. According to the spa repeat analysis, the strains that shared the same PFGE type were very closely related, indicating that these strains belong to the same clone and differed only as a result of the recombination of certain genes; these strains were indistinguishable based on PFGE analysis. This study highlights the fact that PFGE typing is effective in describing strain population structure but, due to the oligoclonality of SFP outbreaks, is limited in its epidemiological resolution.

The data in this study revealed that the sea gene is dominant in the *S. aureus*
isolates that are associated with food poisoning in Shenzhen, China. In fact, the staphylococcal enterotoxin type that is most frequently involved in food poisoning outbreaks worldwide is SEA, which is associated with other staphylococcal enterotoxins (1, 3, 5, 13, 26, 38). The SFP that is caused by sea alone maybe unique in Shenzhen. In this study, an outbreak with seg-sei profile was observed in a single family in 2007. To our knowledge, this is the first report of two S.aureus strains with the seg-sei profile that has been associated with food poisoning in China. The two strains with the closely related PFGE types G1 and G2 were ST5-spa t954 clones, which differed from the seg-sei positive strains that were observed in Korea, which carried the ST type ST20. The seg and sei genes were originally identified in two separate strains (20). The coexistence of seg and sei was unsurprising, together with sem, sen, and seo. All of these five genes belong to an enterotoxin gene cluster (egc), which is located on a genomic island; the detection of one of these genes generally indicates the presence of others (11, 14). Although the involvement of more recently identified SEs in SFP is not yet fully understood, this factor is important for the long-term monitoring of the changing epidemiology of SE and SE-encoding genes in cases of SFP.

Compared with the MRSA clones that are principally responsible for hospital- and community-acquired infections, MSSA lineages that are associated with the SFP outbreaks were more susceptible to antibiotics (35). The majority of the isolates that were examined in this study are only resistant to one or two
antibiotics. The observed high resistance rates to penicillin concurred with several previous studies of *S. aureus* isolates from food products in both China and other countries (4, 23). However, with respect to tetracycline resistance, the previously reported rates varied greatly (4,13). We also detected three multidrug resistant isolates, which were isolated from two patients and the environment. These isolates were ST1: t127: F and exhibited sec-seh enterotoxin profiles and were detected in isolates from outbreak 3, which occurred in 2006. The incidence of antimicrobial resistance in human infections is directly related to the prevalence of resistant bacteria in food products (27); antibiotic resistance to SPF case isolates must therefore be considered.

It will be necessary to better understand the population structure of MSSA carriers and clinical isolates to determine if *S. aureus* strains that cause SFP represent different lineages from those that are commonly carried and from those that cause pyogenic infections.

**Acknowledgements**

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Fig. 1. Dendrogram of the pulsed-field electrophoresis gel (PFGE) patterns of the *S.* aureus strains that were associated with SPF cases between 2006 and 2009.
Fig. 2. Phylogenetic inference of the different S. aureus clones that were examined in this study: The model was based on ST types, BURP analysis and PFGE types. The arrows indicate the directions of the changes between clones. The clone name is designated in the following way: MLST-spa-PFGE (outbreak).
<table>
<thead>
<tr>
<th>PFGE-pattern</th>
<th>PFGE Clusters</th>
<th>SpaType</th>
<th>MLST</th>
<th>se genes</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>1</td>
<td>t5593</td>
<td>ST6</td>
<td>sea</td>
</tr>
<tr>
<td>A1</td>
<td>1</td>
<td>t701</td>
<td>ST6</td>
<td>sea</td>
</tr>
<tr>
<td>A1</td>
<td>1</td>
<td>t5777</td>
<td>ST6</td>
<td>sea</td>
</tr>
<tr>
<td>A2</td>
<td>1</td>
<td>t701</td>
<td>ST6</td>
<td>sea</td>
</tr>
<tr>
<td>B1</td>
<td>1</td>
<td>t2360</td>
<td>ST6</td>
<td>sea</td>
</tr>
<tr>
<td>B1</td>
<td>1</td>
<td>t5777</td>
<td>ST6</td>
<td>sea</td>
</tr>
<tr>
<td>B2</td>
<td>1</td>
<td>t701</td>
<td>ST6</td>
<td>sea</td>
</tr>
<tr>
<td>C</td>
<td>2</td>
<td>t091</td>
<td>ST943</td>
<td>sea</td>
</tr>
<tr>
<td>D</td>
<td>3</td>
<td>t091</td>
<td>ST943</td>
<td>sea</td>
</tr>
<tr>
<td>E</td>
<td>4</td>
<td>t189</td>
<td>ST188</td>
<td>seb</td>
</tr>
<tr>
<td>F</td>
<td>5</td>
<td>t127</td>
<td>ST1</td>
<td>sec-seh</td>
</tr>
<tr>
<td>G1</td>
<td>6</td>
<td>t954</td>
<td>ST5</td>
<td>seg-sei</td>
</tr>
<tr>
<td>G2</td>
<td>6</td>
<td>t954</td>
<td>ST5</td>
<td>seg-sei</td>
</tr>
</tbody>
</table>
Table 1. Epidemiological data from the 12 food poisoning outbreaks in Shenzhen city

<table>
<thead>
<tr>
<th>Outbreak</th>
<th>Date</th>
<th>Number of ill/hospitalized/ death/ at risk</th>
<th>Site</th>
<th>Incubation period (median)</th>
<th>Symptoms (Number)</th>
<th>Food</th>
<th>SFPO assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Jan 10, 2006</td>
<td>16/13/0/200</td>
<td>dining hall</td>
<td>1.5-9.5 h (5)</td>
<td>N (15) V (13) D (9)</td>
<td>bean curd, cooked duck, cooked spareribs</td>
<td>C</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Aug 11, 2006</td>
<td>5/5/0/10</td>
<td>private</td>
<td>1.5-5 h (3.5)</td>
<td>N (5) V (2) D (4)</td>
<td>chicken, cooked squid head</td>
<td>C</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>household</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Aug 28, 2006</td>
<td>3/3/0/8</td>
<td>private</td>
<td>1.5-3 h (2)</td>
<td>N (3) V (2) D (3)</td>
<td>cake and dried meat floss cake</td>
<td>C</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>household</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Sep 15, 2006</td>
<td>3/3/0/5</td>
<td>private</td>
<td>1.5-3 h (2)</td>
<td>N (1) V (2) D (3)</td>
<td></td>
<td>C</td>
</tr>
<tr>
<td>Date</td>
<td>Location</td>
<td>Type</td>
<td>Time</td>
<td>AP</td>
<td>H</td>
<td>G</td>
<td>Notes</td>
</tr>
<tr>
<td>------------</td>
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<td>----</td>
<td>----</td>
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<td>-----------------------------------------------------------------------</td>
</tr>
<tr>
<td>Nov 15, 2006</td>
<td>private</td>
<td>2-5 h (3)</td>
<td>N (3)</td>
<td>V (3)</td>
<td>D (4)</td>
<td>raw peppery radish</td>
<td>C</td>
</tr>
<tr>
<td>Feb 20, 2007</td>
<td>private</td>
<td>1.5-3.5 h (2.3)</td>
<td>N (3)</td>
<td>V (3)</td>
<td>D (3)</td>
<td>cured meat and peanut</td>
<td>C</td>
</tr>
<tr>
<td>Sep 11, 2007</td>
<td>private</td>
<td>1.5-5 h (3)</td>
<td>N (2)</td>
<td>V (2)</td>
<td>D (5)</td>
<td>bread</td>
<td>C</td>
</tr>
<tr>
<td>Sep 18, 2007</td>
<td>private</td>
<td>1.5-5 h (3.5)</td>
<td>N (3)</td>
<td>V (2)</td>
<td>D (5)</td>
<td>steamed twisted roll, bitter</td>
<td>C</td>
</tr>
<tr>
<td>Dec 20, 2007</td>
<td>restaurant</td>
<td>1.5-3 h (2)</td>
<td>N (1)</td>
<td>V (1)</td>
<td>D (3)</td>
<td>food</td>
<td>C</td>
</tr>
<tr>
<td>Aug 14, 2008</td>
<td>supermarket</td>
<td>7.5-11.5 h (9.8)</td>
<td>N (3)</td>
<td>V (3)</td>
<td>D (3)</td>
<td>raw kelp</td>
<td>C</td>
</tr>
<tr>
<td>Date</td>
<td>Symptoms</td>
<td>Onset</td>
<td>Duration</td>
<td>Location</td>
<td>Notes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------</td>
<td>----------------</td>
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<td>------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aug 24, 2009</td>
<td>Vomiting; Diarrhea; Abdominal pain; Nausea; Headache; Giddiness</td>
<td>28/28/01500</td>
<td>1.5-6.6 h (3.5)</td>
<td>Dining hall</td>
<td>None*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* V, vomiting; D, diarrhea; AP, abdominal pain; N, nausea; H, headache; G, giddiness

** confirmed; S, suspected

Staphylococcus aureus was not isolated from food but from the patients.
Table 2. Analysis of the *Staphylococcus aureus* isolates from the food poisoning outbreaks in Shenzhen, China

<table>
<thead>
<tr>
<th>Outbreak</th>
<th>Origin (no. of isolates)$^a$</th>
<th>MLST</th>
<th>Spa (CCs)</th>
<th>PFGE pattern $^b$</th>
<th>Toxin genes $^c$</th>
<th>Resistance to $^d$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>fd (3)</td>
<td>6</td>
<td>t701 (701)</td>
<td>A1</td>
<td>sea</td>
<td>PEN, TC</td>
</tr>
<tr>
<td></td>
<td>fd (6)</td>
<td>943</td>
<td>t091 (singleton)</td>
<td>C</td>
<td>sea</td>
<td>PEN, TC/PEN</td>
</tr>
<tr>
<td></td>
<td>rs (1)</td>
<td>943</td>
<td>t091 (singleton)</td>
<td>C</td>
<td>sea</td>
<td>PEN, EM</td>
</tr>
<tr>
<td>2</td>
<td>fd (3)</td>
<td>943</td>
<td>t091 (singleton)</td>
<td>C</td>
<td>sea</td>
<td>PEN/PEN, CM, TC</td>
</tr>
<tr>
<td></td>
<td>rs (2)</td>
<td>6</td>
<td>t701 (701)</td>
<td>A1</td>
<td>sea</td>
<td>PEN, TC</td>
</tr>
<tr>
<td></td>
<td>en (1)</td>
<td>943</td>
<td>t091 (singleton)</td>
<td>C</td>
<td>sea</td>
<td>PEN, TC</td>
</tr>
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<td>3</td>
<td>rs (1), en (1)</td>
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<td>t127 (singleton)</td>
<td>F</td>
<td>sec, seh</td>
<td>PEN, CM, EM, TC, RI</td>
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<tr>
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<td>sec, seh</td>
<td>PEN, CM, EM, TC</td>
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<tr>
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<td>fd (2)</td>
<td>6</td>
<td>t5777(701)</td>
<td>A1</td>
<td>sea</td>
<td>PEN/PEN, TC</td>
</tr>
<tr>
<td></td>
<td>fc (1)</td>
<td>6</td>
<td>t5777(701)</td>
<td>B1</td>
<td>sea</td>
<td>PEN</td>
</tr>
<tr>
<td>5</td>
<td>fd (1), rs (3)</td>
<td>6</td>
<td>t701 (701)</td>
<td>B2</td>
<td>sea</td>
<td>PEN</td>
</tr>
<tr>
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<td>t5593(701)</td>
<td>A1</td>
<td>sea</td>
<td>PEN</td>
</tr>
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<td>t954 (singleton)</td>
<td>G1/G2</td>
<td>seg, sei</td>
<td>Susceptible</td>
</tr>
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<td>t2360(701)</td>
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</tr>
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<td>A2</td>
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<td>t189 (singleton)</td>
<td>E</td>
<td>seh</td>
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<td>11</td>
<td>fc(1)</td>
<td>943</td>
<td>t091</td>
<td>D</td>
<td>sea</td>
<td>PEN, TC</td>
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</table>

$^a$ A total of 52 *S. aureus* isolates were identified. The origin of the samples included food (27), rectal swabs (15), feces (5), the environment (4),...
and the food handler (1), fd, food; fcs, feces; rs, rectal swab; en, environment; fhs, food handler hand swab.

* The pulsed-field gel electrophoresis (PFGE) types are alphabetically designated, and the subtypes are numerically designated.

† Tested for the sea, seb, sec, sed, see, seg, seh, sei genes.

‡ PEN, Penicillin; TC, Tetracycline; EM, Erythromycin; CM, Clindamycin; RI, Rifampin