Prevalence and Distribution of Superantigen Toxin Genes in Clinical Community Isolates of *Staphylococcus Aureus*

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Abstract

To investigate the distribution of twelve staphylococcal enterotoxin genes (se) and the toxic shock syndrome toxin-1 gene (tst) in *Staphylococcus aureus*, 140 community isolates from various origins were investigated. Isolates were collected, from 15 clinical laboratories located at Casablanca, between 2007 and 2008, they were identified by conventional methods, and methicillin resistance was confirmed by amplification of mecA gene by PCR. All isolates were searched using a multiplex PCR for the accessory gene regulator (agr) group, and for thirteen superantigen (SAg) toxin genes: sea, seb, sec, sed, seh, selk, sell, selm, selo, selp, selq, ser and tst.

Among all isolates, only two were methicillin-resistant and one hundred seven were shown to be positive for at least one of the tested SAg toxin genes. They were grouped in 43 genotypes. Our work showed that agr group III and agr group I *S. aureus* isolates, were highly prevalent for the presence of seh, selk, sell and/or tst genes, on one hand, and sec and/or sell genes, on the other hand (P<0.05), respectively. In addition, we found a relationship between pus/wound *S. aureus* isolates and the presence of selk + selg genes (P<0.05).

Our results suggest that agr group III isolates carried more of SAg toxin genes than agr groups I and II *S. aureus* strains.

Keywords: *Staphylococcus aureus*; MSSA; MRSA; SAg's toxin genes; agr groups; Community

Introduction

*Staphylococcus aureus* is a major cause of multiple types of infections both in and outside of the hospital setting. These infections range from superficial skin infections to deeper infections of hair follicles, abscesses, and deep tissue infections, and even to systemic infections including those of the heart, lungs, bones, and blood [1].

The organism has an array of cell-surface and secreted virulence factors that allow it to cause illnesses [1]: the surface virulence factors allow *S. aureus* to colonize the host, through adhesion to mucosal surfaces and resistance to phagocytosis, the secreted factors, including exoenzymes and exotoxins, allow the organism to interfere with normal immune system function, spread into surrounding tissues, and access nutrients through cell damage. Among the secreted virulence factors that have known roles in serious human diseases, are the staphylococcal superantigens (SAgs) [2,3]. They includes toxic shock syndrome toxin-1 (TSST-1) [4], staphylococcal enterotoxin (SE) serotypes A, B, Cn (in which n denotes that multiple variant forms exist), D, E, G, H, I, R, S, T and SE-like (SEls) serotypes J, K, L, M, N, O, P, Q, U, V [5-7]. These toxins are considered to be major virulence factors of *S. aureus* [3,5]. In addition, they exhibited superantigen activity, stimulating polyclonal T-cell proliferation through culligation between major histocompatibility complex II molecules on antigen-presenting cells (APC) and the variable portion of the T-cell antigen receptor β chain or α chain (TCR Vβ and TCR Vα, respectively), with no need for prior APC processing [2,8,9]. On the other hand, most of genes encoding these toxins are located on mobile genetic elements, such as bacteriophages, pathogenicity islands (SaPIS), genomic islands, and plasmids. This association implies a horizontal transfer of the SAg toxin genes between staphylococcal strains and an important role in the evolution of *S. aureus* as a pathogen. [10-14].

However, expression of most virulence genes in *S. aureus* is controlled by the accessory gene regulator (agr) locus. The agr locus consists of two divergent transcription units driven by promoters P2 and P3. The P2 operon encodes a two component signalling module, AgrC is the receptor and AgrA is the response regulator. It also encodes two proteins, AgrB and D, which combine to produce and secrete an autoinducing peptide (AIP) that is the ligand for AgrC. AgrA functions to activate transcription from its own promoter and from the agrP3 promoter, which drives the synthesis of RNAIII, the effector of target gene regulation [15]. Sequence variation in agrB, agrD and agrC has led to the identification of at least four *S. aureus* agr specificity groups (I to IV) [16]. Furthermore, the Agr system has been assigned a central role in *S. aureus* pathogenesis [13,17,18].

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Little information is available about the degree of superantigenic genetic variability among populations of community clinical Staphylococcus aureus in Morocco. For this reason, the purpose of the present study was to investigate the presence of the staphylococcal superantigen toxin genes in a group of S. aureus isolates in the city of Casablanca (Morocco) and to correlate them with their origin, and agr groups.

Materials and Methods

Bacterial isolates

Non-duplicate community S. aureus strains from clinical specimens were collected from outpatients of the Laboratory of Microbiology, Institute Pasteur of Morocco, and from 14 clinical laboratories located in Casablanca, between January 2007 and October 2008. Patients were assessed as to whether they had previously been hospitalised for a medical condition.

Identification of S. aureus isolates

Species were identified by colony morphology, gram staining, catalase test, coagulase activity on rabbit plasma (Bio-Mérieux, Marcy l’Etoile, France), and production of clumping factor (Pastorex Plus-Staph, Bio-Rad, Marnes-la-Coquette, France).

Antimicrobial resistance

Antimicrobial resistance to penicillin G, kanamycin, tobramycin, gentamicin, tetracycline, erythromycin, lincomycin, pristinamycin, chloramphenicol, pefloxacin, fosfomycine, cefoxitin, fusidic acid, rifampicin, vancomycin and trimethoprim-sulfamethoxazole was determined by the standard disc (Bio-Rad, Marnes-la-Coquette, France) diffusion technique. Results were interpreted according to the Committee for Antimicrobial Testing of the French Society of Microbiology guidelines (http://www.sfim.asso.fr) (Comité de l’Antibiogramme de la Société Française de Microbiologie, 2007). S. aureus ATCC 29213 and S. aureus ATCC 25923 were used as quality control organisms.

DNA extraction

All isolates of S. aureus were grown in brain heart infusion media at 37°C overnight. Their genomic DNA used for polymerase chain reaction (PCR) was extracted by using a standard phenol-chloroform procedure as described by Sambrook et al., [19]. Amplification of nuc gene which encodes an extracellular thermo stable nuclease of S. aureus was used, as described by Brakstad et al., [20], for identification and to confirm the quality of each DNA extract. Strains with phenotypic resistance to methicillin were confirmed by polymerase chain reaction detection of the mecA gene as described by Vannuffel et al. [21]. Two reference strains: U2A1585 for methicillin-resistant S. aureus (MRSA) and U2A1594 for methicillin-sensitive S. aureus for (MSSA) from the Antibacterial Agents Unit of Pasteur Institute, Paris, France, were used as controls.

Detection of staphylococcal toxin genes

Several Multiplex PCRs for the parallel detection of the presence of the following genes were performed: the classical staphylococcal enterotoxins (sea, seb, sec and sed), SEs and SEls (seh, selk, sell, sehl, selo, selp, selg and ser) and the toxic shock syndrome toxin-1 (tst) [16,22]. S. aureus ATCC19095 (sec, seh, sell, sei, sehn, selo and sed); FR913 (sec, seh, sell, selg, selo and tst); ATCC14558 (seh); were used as positive control strains. Control chromosomal DNA samples for sed, ser and selg genes were obtained from our standard laboratory controls.

Determination of agr groups

A multiplex PCR of the agr was used to determine the agr group (I-IV) [23]. S. aureus strains RN6390 (agr group I), RN6607 (agr group II), RN4845 (agr group III), and RN4850 (agr group IV) from the National Research Center of Lyon (CNR-Lyon, France) were used for agr group identification.

Statistical analysis

Chi-square test was used to study the correlation between the prevalence of the SAg toxin genes and the agr group of S. aureus isolates on one hand, and the prevalence of these SAgs toxin genes and the origin of S. aureus isolates on the other hand. Statistical analysis of the data was performed on SPSS. P < 0.05 was considered statistically significant.

| agr group (n,%), | No of S. aureus isolates with virulence SAgs gene: (genotype, n) | Total SAgs gene, n (%) |
|-----------------|---------------------------------------------------------------|-----------------------|
|                 | None  | One   | Two   | Three | Four  | Five  |
| I (66, 47.2)    |       |       |       |       |       |       |
|                 | 23    | seb, 1| seb sep, 1 | seb sem, 2 | sec sel sem seo, 9 | sec sel sem seo tst, 1 |
|                 |       |       |       |       |       |       |
| II (25,20,7)    | 10    | seoh, 1| seoh sep, 1 | seoh sem, 1 | sec sel sem seo, 1 | sed sem seo ser, 1 |
|                 |       |       |       |       |       |       |
| III (42,30,0)   | 10    | seh, 3 | seh sep, 1 | seh sem, 1 | sec seh tst, 1 | seh seh sek seq, 1 |
|                 |       |       |       |       |       |       |
|IV (3, 2,1)     | 0     | sem, 2 | sem seh, 1 | seh seh sek, 1 | seh seh sek seq, 1 | sec seh seh seq, 1 |
|                 |       |       |       |       |       |       |
|Sous total of SAgs genes/ agr (%) | 23 (34.9) | 9 (13.7) | 15 (22.7) | 8 (12.1) | 9 (13.6) | 2 (3.0) | 43 (65.15) |
|Sous total of SAgs genes/ agrI (%) | 10 (34.5) | 2 (6.9) | 9 (31.0) | 5 (17.3) | 2 (6.9) | 1 (3.4) | 19 (65.51) |
|Sous total of SAgs genes/ agrII (%) | 0     | seh, 3 | seh sep, 1 | seh sem, 1 | sec seh tst, 1 | seh seh sek seq, 1 |
|                 |       |       |       |       |       |       |
|Sous total of SAgs genes/ agrIII (%) | 0     | sem, 2 | sem seh, 1 | seh seh sek, 1 | seh seh sek seq, 1 | sec seh seh seq, 1 |
|                 |       |       |       |       |       |       |
|Sous total of SAgs genes/ agrIV (%) | 0     | 0     | 0     | 0     | 0     | 0     | 3 (100.00) |

N.B: seh, sel, sem, seo, sep and seq genes = staphylococcal enterotoxin-like

Table 1: Combination of SAgs genes in clinical isolates of S. aureus according to their agr group (n=140).
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### Table 2: Incidence of SAg genes in *S. aureus* isolates from various sources.

| SAg genes: (n,%); No of SAg genes/ agr group (%) | I (66, 47.2) | II (29, 20.7) | III (42, 30.0) | IV (1, 1.5) | Total (%) |
|------------------------------------------------|-------------|--------------|----------------|-------------|-----------|
| sea                                             | 0 (0)       | 0 (0)        | 3 (7.1)        | 0 (0)       | 3 (1.1)   |
| seb                                             | 5 (7.6)     | 1 (3.4)      | 1 (2.4)        | 0 (0)       | 8 (2.9)   |
| sec                                             | 17 (24.5)*  | 2 (6.8)      | 4 (9.5)        | 0 (0)       | 23 (6.8)  |
| sed                                             | 0 (0)       | 2 (6.8)      | 0 (0)          | 0 (0)       | 2 (0.7)   |
| seh                                             | 0 (0)       | 0 (0)        | 20 (47.6)*     | 0 (0)       | 20 (4.7)  |
| sek                                             | 11 (1.15)   | 0 (0)        | 14 (33.3)      | 0 (0)       | 15 (5.5)  |
| sel                                             | 15 (22.5)*  | 1 (3.4)      | 2 (4.8)        | 0 (0)       | 18 (6.6)  |
| sem                                             | 29 (43.9)   | 16 (55.2)    | 13 (31.0)      | 3 (100.0)   | 81 (22.4) |
| seo                                             | 33 (50.0)   | 16 (55.2)    | 23 (54.8)      | 3 (100.0)   | 75 (21.6) |
| sep                                             | 6 (9.0)     | 6 (20.7)     | 0 (0)          | 0 (0)       | 12 (4.4)  |
| seq                                             | 1 (1.5)     | 0 (0)        | 12 (28.6)*     | 0 (0)       | 13 (4.8)  |
| ser                                             | 0 (0)       | 2 (6.8)      | 2 (4.8)        | 0 (0)       | 4 (1.5)   |
| tst                                             | 2 (3.0)     | 2 (6.8)      | 1 (3.7)        | 0 (0)       | 5 (1.5)   |

### Table 3: Distribution of SAg genes by agr group of *S. aureus* isolates (n=140).

| agr group | N° of *S. aureus* isolates from: | G-U-T + | Pus/wound | Sputum | naso-pharynx* | Blood | Total |
|-----------|-------------------------------|---------|-----------|--------|---------------|-------|-------|
| I         | 29                            | 21      | 02        | 07     | 07            | 02    | 86    |
| II        | 12                            | 13      | 02        | 02     | -             | -     | 29    |
| III       | 15                            | 17      | 06        | 04     | -             | -     | 42    |
| IV        | 01                            | 01      | 01        | -      | -             | -     | 03    |
| Total     | 57                            | 52      | 16        | 13     | 02            | 02    | 140   |

### Table 4: Distribution of agr group *S. aureus* isolates by origin of samples.

| agr group | N° of *S. aureus* isolates from: | G-U-T + | Pus/wound | Sputum | naso-pharynx* | Blood | Total |
|-----------|-------------------------------|---------|-----------|--------|---------------|-------|-------|
| I         | 29                            | 21      | 02        | 07     | 07            | 02    | 86    |
| II        | 12                            | 13      | 02        | 02     | -             | -     | 29    |
| III       | 15                            | 17      | 06        | 04     | -             | -     | 42    |
| IV        | 01                            | 01      | 01        | -      | -             | -     | 03    |
| Total     | 57                            | 52      | 16        | 13     | 02            | 02    | 140   |

### Results

#### Antimicrobial resistances

A total of 140 *S. aureus* isolates were collected from clinical outpatient specimens at the Laboratory of Bacteriology, Institute Pasteur of Morocco (60% of isolates), and from 14 clinical laboratories located in Casablanca (40% of isolates). The rates of resistance to antibiotics were: penicillin (90.00%), tetracycline (30.00%), rifampicin (14.29%), fusidic acid (12.86%), kanamycin (9.29%), cotrimoxazole (9.29%), erythromycin (7.86%), pefloxacin (2.14%), gentamicin (1.43%), tobramycin (1.43%), lincomycin (1.43%), chloramphenicol (0.71%) and only two isolates (1.43%) were confirmed MRSA.

#### Prevalence of SAg toxin genes

All of 140 *S. aureus* isolates under study harboured the nuc gene. The all 13 SAg toxin genes analysed in this study were detected; the prevalence were variable from 53.6% to 1.4% (selo 53.6%, sem 43.6%, sec 17.1%, seh 14.3%, sell 12.9%, tst 12.1%, selk 10.7%, selq 9.3%, selp 8.6%, seb 5.7%, ser 2.9%, sea 2.1% and sed 1.4%). However no virulence toxin gene was diagnosed as positive in 33 (23.6%) isolates. Whereas 1 gene was detected in 18 (12.8%) strains, in all remaining isolates (63.6%) at least two SAg genes were present in the same isolate: Forty isolates carried two genes simultaneously, twenty-six isolates contained three genes, nineteen isolates were found with four genes and in four isolates five SAg genes were detected (Table 1). A total of 43 superantigen toxin genotypes were found, the toxin genotype with the highest incidence was selm+s elo (20%). This pair was detected in 57 of all isolates.

#### Distribution of SAg genes in *S. aureus* isolates from various sources

Regarding the origin of *S. aureus* isolates, two major locations could be distinguished: genital-urinary tract (40.7%), most of isolates from this location were from urine (n =30), the others were from vaginal and high vaginal swabs (n =10), urethral swabs (n =8) and sperm (n =9), the second major location was pus/wound (37.1%). In addition, two minor locations were noted: sputum (11.4%) and naso-pharynx (9.3%), strains isolated from blood were scarcely represented (3.1%) (Table 2). The most abundant genes (selo and sem) were found in 61.5% and 44.2% of pus isolates, respectively; 47.7% and 38.6% of genital-urinary tract isolates, respectively; 56.3% of respiratory tract isolates, 46.2% of naso-pharynx isolates and 50% of blood isolates (Table 2).
Distribution of SAg genes by agr group

All of S. aureus isolates were classified according to the four agr groups. 47.2% strains were found to belong to agr group I, 20.7% were agr group II, 30.0% were agr group III and only 2.1% of isolates were found to belong to agr group IV. In contrast to agr group I or II S. aureus isolates, all agr group III strains, were positive for at least one of the tested genes (Table 1 and Table 3). A total of 22 genotypes were detected among agr group III S. aureus isolates and 16.7%, 33.3%, 28.6%, 19.0%, 2.4% of these strains contained one, two, three, four and five SAg toxin genes, respectively. Nineteen SAg genotypes were observed among agr group I strains, against 10 found among agr group II isolates. On the other hand, 13.7%, 22.7%, 12.1%, 13.6% and 3% of agr group I isolates carried one, two, three, four and five SAg toxin genes, respectively. The most abundant SAg toxin gene (selo) was carried by 50.0%, 55.2% and 54.8% of isolates belonging to agr group I, II and III, respectively. The second abundant SAg toxin gene, namely sem, was carried by agr group I strains with 43.9%, agr group II with 55.2% and agr group III with 31.0%.

Distribution of agr group S. aureus isolates by origin of samples

The relationship between agr group S. aureus strains and source of isolates is summarized in (Table 4).

Discussion

Staphylococcus aureus isolates collected during the present study were originated from ambulatory patients, they were collected from 15 clinical laboratories located in Casablanca; therefore, they should represent randomly selected strains. In fact, a significant number were recovered from vaginal swabs, high vaginal swabs and urine. Isolates from high vaginal swabs are usually associated with puerperal sepsis or neglected foreign bodies, and less frequently with bacterial vaginois, and significant numbers of MRSA organisms have been found among these isolates [24,25]. While it is likely that many S. aureus organisms might be passive colonizers or due to contamination from the skin, some are associated with complications of pregnancy [24]. Although S. aureus is a rare cause of urinary tract infections, accounting for only 0.5% to 6% of all positive urine cultures [26,27], their finding is increasingly being recognized as significant, especially in patients with urinary tract catheterisation, as under-treatment or delayed treatment could lead to development of staphylococcal bacteremia [26,28,29].

It is important to discuss, even briefly, the susceptibility to the tested antibiotics of all S. aureus isolates. This study provides important data on current antimicrobial resistance, including methicillin-resistance, for a collection of 140 recent clinical isolates of S. aureus from community source in Casablanca, Morocco. We found that 90% of isolates possessed resistance to penicillin, followed by strains with resistance to one or two more antimicrobial substances. Resistance to methicillin conferred by carriage of the meca gene is rather low; it is found with only two agr group I isolates (1.43%). Epidemiological data of community-acquired methicillin-resistant S. aureus (CA-MRSA) in the countries of the Maghreb, i.e. Morocco, Algeria, Tunisia and Libya are scarce; although, recent reports suggest that Hospital-acquired MRSA epidemiology is changing, with a dramatic increase of incidences [30,31].

On the other hand, it is hypothesized that the evolution of CA-MRSA is a recent event due to the acquisition of meca DNA by previously methicillin-susceptible strains that circulated in the community [32]. Then, considering the relationship that might exist between MRSA and MSSA isolates, and the limited data on the prevalence and distribution of SAg toxin genes among S. aureus isolates in Morocco; we need more specific knowledge about the circulating S. aureus isolates, especially MSSA strains.

In a first step, we found that the overall rate of SAg toxin gene-positive isolates reached 76.4%. This is in agreement with results published by Hu et al., [33], they have reported that 75.7 % of the MSSA isolates tested carried a number of toxin genes, ranging from 1 to 11, with extensive variation between individual strains. In a second step, we found that differences in the occurrence of genes between pus/wound versus the genital-urinary tract, sputum or naso-pharynx isolates were not significant as demonstrated by the Chi-square test (P>0.05); with exception for selK and selQ genes, which were slightly frequent in pus/wound S. aureus isolates (P<0.05). In a third step, we found that 47.2% of all isolates were agr group I, this finding concurs with results of other studies [16,34]. Thereafter, we have analysed the agr group specificity of all strains, and we found that there are many SAg toxin genes in S. aureus isolates belonging to agr group I, II, III and/or IV from many clinical specimens. However this distribution was not uniform among all agr groups isolates; we found that agr group III S. aureus isolates (n=42) carried more SAg toxin genes (mean 2.55 gene/isolate), compared to 66 S. aureus isolates in agr I and 29 S. aureus isolates in agr II (mean 1.65 gene/isolate). Secondly, we found that all agr group III isolates (100%) were found with at least one of the tested virulence genes, whereas 23 and 10 strains among agr group I and agr group II isolates, respectively, had none SAg toxin genes. Thirdly, our finding showed that 50% of agr group III isolates were found with 3 to 5 virulence toxin genes, versus, only 28.9% and 27.6% of agr group I and II isolates, respectively. From these data, we can deduct that agr group III isolates were more prevalent for the presence of enterotoxin and/or tst genes than agr groups I and II isolates.

However, agr group I was prevalent in all clinical specimens whatever their origin, nevertheless it was slightly higher in genital-urinary tract samples than others. Whereas, both agr groups II and III were dominant in S. aureus isolates from pus/wound specimen. These differences were not statistically significant.

Most of virulence toxin genes under study are associated with mobile genetic elements. However, the repertoire of toxin genes encoded by SaPIs seems to be specified. These elements, as well as the majority of genetic elements encoding enterotoxins, can be horizontally transferred among S. aureus strains. But, unlike plasmids, they cannot spread autonomously. It was shown that in the presence of certain staphylococcal phages, SaPIs are excised from the genome and encapsidated. This mechanism in thought to be responsible for the transfer of pathogenicity islands [13]. For this reason the prophage ϕMu50A and ϕN315, shown to be integrated in close proximity to the TSST-1 pathogenicity island family of S. aureus Mu50 and N315, respectively, are considered to be involved in the horizontal transfer of these SaPIs [35]. These data led us to define the prevalence of each of the mobile genetic elements among all 140 S. aureus isolates under study.

In the current work, in agreement with other [22], selM and selO genes were more frequently detected in all isolates, whatever their origin and their agr group. Both genes belong to the recently described enterotoxin gene cluster (egt) that harbours 5 to 6 genes (seg, sei, selM, selO, and sometimes selH), which cluster on a staphylococcal pathogenicity island type I γSaβ [33,36,37], this cluster will be found without all of the
**Conclusions**

Despite the fact that the agr group I was the most prevalent group, among 140 community S. aureus isolates, in Casablanca, Morocco, strains belonging to the agr group III harboured more virulent superantigen toxin genes. So, we can assume that agr group III isolates may carry more enterotoxin and/or tst genes than agr groups I and II isolates. Of all thirteen SAg toxin genes, two were detected more frequently in all agr group isolates, whatever their origin: selo and selm.

Statistical analysis of the comparison of the prevalence of SAg toxin genes in the four studied agr group isolates (agr group I, II, III and VI), on one hand, and the prevalence of these virulence toxin genes according to the source of isolates, on the other hand, using the chi-square test, showed that agr group III and agr group I S. aureus isolates, whatever their origin, were more prevalent for the presence of seh, selq, selk and/or tst virulence toxin genes and selc and/or selo virulence toxin genes (P < 0.05%), respectively. Finally, it is also particularly noteworthy that, selk-positive and selq-positive isolates, were highly prevalent from pus/wound (P < 0.05%).

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