Nig. J. Pure & Appl. Sci. Vol. 39 (Issue 1, 2026)
e-ISSN 2756-4045
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www.njpas.com.ng

Phenotypic and Molecular Characterization of Methicillin Resistance in *Staphylococcus aureus* Strains isolated from the Urine of Patients with Urinary Tract Infections in Lagos State, Nigeria

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Date Received: 25-11-2025
Date Accepted: 28-01-2026
DOI: https://doi.org/10.48198/NJPAS/25.B45

ABSTRACT

The emergence of methicillin-resistant *Staphylococcus aureus* (MRSA) is responsible for increasing health costs, patients' morbidity and mortality. As a result, in the global priority list of antimicrobial-resistant bacteria, MRSA has been designated as Priority 2: HIGH. This study aimed to detect *mecA* and *spa* genes in MRSA, and to establish the clonal relatedness among the isolates obtained from patients with urinary tract infections (UTIs), at five (05) different hospitals in Lagos State, Nigeria. Antibiotic susceptibility testing (AST) was carried out on thirty-three (33) *Staphylococcus aureus* isolates, to screen for methicillin resistance, using cefoxitin via Kirby-Bauer disc diffusion method, while polymerase chain reaction (PCR) was used to detect *mecA* and *spa* genes in isolates resistant to cefoxitin. In addition, randomly amplified polymorphic DNA-PCR (RAPD-PCR) was carried out on the isolates confirmed to be MRSA, using three (03) oligonucleotide random primers (OPT-02, OPT-08 and OPT-17). Phenotypically, 54.5% (18/33) of the strains were confirmed to be MRSA. Of the 18 confirmed MRSA isolates, the *mecA* gene was detected in 33.3% (6/18), while 11.1% (2/18) of them harboured the *spa* gene. RAPD-PCR generated divergent banding patterns, demonstrating as a potential DNA fingerprinting technique, suitable to analyze MRSA evolutionary relationships in urine. This study showed a high prevalence of MRSA, with a low carriage rate of *mecA* and *spa* genes. As a result, the high prevalence, genetic diversity and differentiation of MRSA, including the detection of *mecA* and *spa* genes in some MRSA strains in urine samples from UTI patients raise the possibility of virulence isolates, as well as, increasing resistance to antimicrobial drugs.

Keywords: Methicillin-resistant, urinary tract infections, *mecA* and *spa* genes, prevalent, Lagos State.

1.0 Introduction

Staphylococcus aureus is ranked among the most prevailing causes of community- and hospital-acquired infections, resulting in marked incidences of illness and fatality (Tong *et al*., 2015). This bacterium makes up part of the microbiome of healthy humans and animals, and is usually found on the skin, respiratory tract, intestines and vagina (Frank *et al*., 2010). It can also become pathogenic, upon breakage of epithelial tissues, most commonly in immune-deficient individuals (Cheung *et al*., 2021), causing infections such as septic arthritis, septicaemia, pneumonia, infective endocarditis, fracture fixation, toxic shock syndrome, invasive infections, and even death (Kwiecinski and Horswill, 2020).

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S. aureus is not frequently associated with urinary tract infections (UTIs). It occurs only in 0.2 to 4% of positive urine sample cases, and 1% of uncomplicated UTIs (Al-Mohajer *et al*., 2013). These UTIs usually occur when *S. aureus* inhabiting the gut, colonizes the urethra, often by contamination, and moves upward into the bladder, via the flagella and pili (Flores-Mireles *et al*., 2015). Most *S. aureus* possess special properties that help them to survive and replicate in the bladder, and if left untreated, *S. aureus* bacteriuria can consequently move to the kidneys, cross the epithelial barrier, and finally to the bloodstream, causing bacteremia (Flores-Mireles *et al*., 2015).

S. aureus utilizes a variety of virulence factors that can evade the innate and adaptive immune responses (Falugi *et al*., 2013). One of the most significant virulence factors adopted by *S. aureus* is the staphylococcal protein A (*spa*) (Votintseva *et al*., 2014). It is a surface protein, anchored to the Fc receptor of human and animal immunoglobulins (Ig), and prevents antibody-induced opsonophagocytosis of *S. aureus*, and B cell development (Falugi *et al*., 2013). The macromolecule known as staphylococcal protein A is found in the cytoplasm, and cell walls of methicillin-resistant *Staphylococcus aureus* (MRSA). The *spa* gene encodes a 42 Da molecular weight protein, with polymorphisms in short sequence repeats (SSRs), linked to virulence and pathogenesis in staphylococcal infection (Sun *et al*., 2018). Urine samples showed diverse *spa* types among MRSA and methicillin-susceptible *Staphylococcus aureus* (MSSA) strains, with different multidrug-resistant (MDR) characteristics (Goudarzi *et al*., 2018).

The acquisition of the *mecA* gene, which is found in the staphylococcal cassette chromosome (SCCmec), is typically the cause of methicillin resistance. The *mecA* gene produces penicillin binding protein (PBP2a), a 78 kDa, with low binding affinity to all β-lactam antibiotics and other classes of antibiotics (Chambers and DeLeo, 2009). A more precise knowledge of the methicillin-resistant bacteria detected in urine samples from patients suffering from UTIs, is made possible by the presence of the *mecA* gene (Assouma *et al*., 2023).

MRSA has been reported across the world, posing as a global health challenge, with varying proportions of staphylococcal morbidity in humans (Zhu *et al*., 2025). In Europe, the prevalence of MRSA is lower, compared to other parts of the world (Uhlemann *et al*., 2014), but it is more prevalent in most developing countries (Coombs *et al*., 2009). In Africa, the prevalence of MRSA is not well described. Since year 2000, MRSA has been on the rise in several African countries, posing a significant danger to the region (Falagas, *et al*., 2013), which might be as a result of inadequate resources and a lack of formal education.

Randomly amplified polymorphic DNA (RAPD)-polymerase chain reaction (i.e. RAPD-PCR) has been widely used, as a quick and easy method to type closely related *S. aureus* strains (Kurlenda *et al*., 2007). This method allows for quick polymorphisms comparison, as a culture-dependent DNA fingerprinting technique, without the need for nucleotide sequence information in the genome, but with annealing of DNA template to short oligonucleotides at lower temperatures (Idil and Bilkay, 2014).

Antibiotic-resistant strains of *S. aureus* to commercially available antibiotics has been a source of concern for many years (Xie *et al*., 2011). The emergence of resistance has been linked to the indiscriminate use of antibiotics, longer hospitalizations, and the use of antibiotics as growth promoters in animal feeds and husbandry (Normanno *et al*., 2007). Methicillin resistance in MRSA reduces the efficacy of most β-lactam antibiotics (Blanc *et al*., 2007). Therefore, this may present a significant public health issue, and its emergence is responsible for increasing health costs, patients' morbidity, and mortality (Nakamura *et al*., 2012). As a result, MRSA was assigned Priority 2: HIGH status on a global priority list of antimicrobial-resistant bacteria (Tacconelli *et al*., 2018). Hence, in this study, *S. aureus* from urine of patients with UTIs was first, phenotypically screened, for methicillin-resistant strains, and then genotypically identified *mecA* and *spa* genes in the MRSA strains. In addition, clonal relatedness of the MRSA strains was also determined.

2.0 Materials and Methods

2.1 Collection of samples

Seventy-five (75) samples were obtained from the urine of patients with urinary tract infections (UTIs), attending five government hospitals in Lagos State, Nigeria. The hospitals are: General Hospital, Orile-Agege, Lagos University Teaching Hospital (LUTH), Idi-Araba, Gbagada General Hospital, Gbagada, General Hospital, Somolu, and General Hospital, Marina. This study protocol was approved by the Health Research Ethics Committee (GBGH/705/100) of Gbagada General Hospital, Gbagada, Lagos, Nigeria, and all procedures were performed according to best practices for clinical laboratories. Patients were fully briefed on the study objectives, and their informed consent was documented.

2.2 Phenotypic identification of Staphylococcus aureus

The urine samples were cultured on mannitol salt agar (MSA) (Oxoid, UK), and incubated at 37 °C. Bacterial growth was then examined after 24 to 48 h of incubation. Colonies on MSA were observed for *Staphylococcus aureus*, as golden yellow colouration, and were confirmed by biochemical tests, such as coagulase, catalase, and Gram staining as previously described (Benson, 2002).

2.3 Antimicrobial susceptibility test

The *S. aureus* isolates were screened for methicillin resistance by the use of cefoxitin (30 µg/disk) (Oxoid Ltd., Cambridge, UK), based on the Kirby-Bauer disc diffusion technique, and the interpretation was in line with the guidelines by the European Committee on Antimicrobial Susceptibility Testing (EUCAST) recommendation. A 0.5 McFarland standard of culture suspension of *S. aureus* culture suspension was used (Akanbi *et al*., 2017), and antibiotic susceptibility testing was conducted for MRSA with disc diffusion zone diameter breakpoints of S ≥ 22 mm and resistance of < 22 mm (EUCAST, 2021).

2.4 Molecular identification of mecA and spa genes

Genomic DNA of the *S. aureus* isolates was extracted, using the QIAamp® DNA kit (Qiagen, Germany), based on the instructions provided by the manufacturer. Molecular detection of the *mecA* gene was done using specific primers for the *mecA* gene (Abazar *et al*., 2014). Target sequences were amplified in a mixture incorporating 2.5 µL of 5x GoTaq green buffer, 0.75 µL of 25 mM $MgCl_2$, 0.75 µL of 10 mM dNTPs mix, 0.5 µL of 10 pmol each of the forward (5′-AAAATCGATGGTAAAGGTTGGC -3′) and reverse (5′-GTTCTGGAGTACCGG ATTTGC-3′) primers, 0.25 µL of Taq DNA polymerase (Promega, USA), and 18.75 µL nuclease-free water, to which 1 µL DNA template was added. The PCR amplification protocol commenced with 5 min of denaturation at 94 °C, followed by 30 cycles of 94 °C for 30 s, 49 °C for 30 s, 72 °C for 30 s, and a final extension of 72 °C for 10 min. A GeneAmp 9700 PCR system thermal-cycler (Applied Biosystems Inc., USA) was employed for the polymerase chain reaction.

The PCR for the molecular detection of the *spa* gene was done using *spa*-specific primers (Hashemizadeh, 2020), with little modifications. The reaction cocktail comprised 5 µL of 5x GoTaq, 5 µL of 25 mM $MgCl_2$, 8 µL of 10 mM dNTPs mix, 0.5 µL of 10 pmol each of forward (spa-1113: 5′-TAA AGA CGA TCC TTC GGT GAG C-3′) and reverse (spa-1514: 5′-CAG CAG TAGTGC CGT TTG CTT-3′) primers, 0.25 µL of Taq DNA polymerase (Promega, USA), and 29.75 µL nuclease-free water, to which 1 µL DNA template was added. Thermal cycling was initiated with a denaturation step at 94 °C for 5 min, followed by 30 cycles consisting of 94 °C for 30 s, 55 °C for 30 s, 72 °C for 30 s, and a final termination of 72 °C for 10 min. PCR was performed in a GeneAmp 9700 PCR system thermal-cycler (Applied Biosystems Inc., USA).

2.5 Randomly amplified polymorphic DNA-polymerase chain reaction (RAPD-PCR)

Three polymorphic RAPD markers were used for genotyping the selected bacterial isolates. RAPD-PCR amplification was performed in a 25 µL total volume as reported by Zare *et al.* (2019), with slight modifications. Initial denaturation at 94 °C for 5 min, 35 cycles of denaturation at 94 °C for 15 s, annealing at 37 °C for 90 s, extension at 72 °C for 30 s, and a final extension at 72 °C for 7 min constituted the thermal cycling conditions.

The three primers used include: OPT-02 (5′-GGAGAGACTC-3′), OPT-08 (5′-AACGGCGAC A-3′) and OPT-17 (5′-CCAACGTCGT-3′). The RAPD primer operon codes (OPT-02, OPT-08 and OPT-17) were selected from Cosmo GeneTECH operon RAPD 10 mer kit (www.cosmogenetec.com). Each primer bands were separated on a 1.5% agarose gel and electrophoresed for 45 min at 120 V.

2.6 Data analysis

For each primer, binary data was generated, and the RAPD bands were manually scored as 1 and 0 (Kumar and Gurusubramanian, 2011). Genetic distances between samples were calculated, using Jaccard's similarity coefficient (Tamura *et al*., 2013). A data matrix was created, using the generated binary data. To assess genetic relationships, a UPGMA-based dendrogram was inferred using MEGA version 6. Additionally, genetic distance was calculated using the mega6 software.

3.0 Results

The distribution of *S. aureus* infection among patients' age and sex is seen in Table 1. From the total samples (75) analyzed, 33 (44.4%) isolates of *S. aureus* were obtained; of them, 33.3% were isolated from males, and 66.7% from females. The 21-30 age group had the highest isolation rate of 45.5% *S. aureus*, which was observed in females. On the other hand, neither male nor female in the 31-40 age group had *S. aureus* infection.

Out of the 33 *S. aureus* isolates obtained, a total of 18 (54.5%) were resistant to cefoxitin (phenotypically positive methicillin-resistant *S. aureus*, MRSA), while the remaining 15 (45.5%) *S. aureus* isolates were susceptible to the antibiotics (methicillin-susceptible *S. aureus*, MSSA) (Figure 1). Isolates were regarded as resistant when the zone of inhibition was < 22 mm (Table 2).

3.1 Distribution of mecA and spa genes in MRSA isolates

Notably, only one-third (6/18, 33.3%) of the phenotypically defined MRSA isolates were found to carry the *mecA* gene. The *mecA* gene gel electrophoresis is shown in Figure 2, where six (33.3%) of the eighteen MRSA isolates displayed bands for the *mecA* gene. Additionally, a polymerase chain reaction (PCR) was performed on the eighteen MRSA isolates to detect the presence of the *spa* gene, and it was revealed that the *spa* gene was detected in only two (11.1%) isolates (Figure 3).

3.2 RAPD-PCR typing of methicillin-resistant S. aureus isolates

Among the three primers used in this study, one of them, OPT-02, showed no banding patterns. However, polymorphisms were observed in all the isolates for OPT-08 and OPT-17 primers, except isolate A26, which showed no amplified band, with primer OPT-17 (Figures 4 and 5). The two primers that yielded discriminatory patterns were used to perform the RAPD analysis. Several polymorphic bands arose from the isolates amplification, ranging from >100 to 1,200 bp in size. The primer OPT-08 showed the highest number of bands, which produced majorly three or more distinct bands in the isolates (Figure 4). In addition, all the isolates produced bands that are conserved, and could be seen in the patterns with the fragments of about 500 and 1,000 bp (Figure 4), based on OPT-08. Primer OPT-17 revealed polymorphic patterns, consisting of 4 discernible RAPD types (RT), 'RT1' (5, 27.8%), 'RT2' (9, 5%), 'RT3' (1, 5.6%) and 'RT4' (1, 5.6%) (Figure 5).

3.2.1 Cluster analysis of RAPD-PCR banding patterns

The isolates were categorized, based on similarity, to generate a dendrogram using UPGMA. They were grouped at a similarity threshold of 80% (equivalent to a Jaccard distance of 0.20), in order to define significant clusters. The analysis by UPGMA for the construction of the dendrogram of RAPD data generated, by OPT-08 and OPT-17 primers, showed that the MRSA isolates were categorized into one major group (A19, A26, A18, A14, A13, A8, A7, A1, A2, A3, A4, A10, A15, A24, A25, and A31) (Figure 6). While isolates A27 and A29 remained distinct from the major cluster. They joined the main group at a lower similarity level (approximately 70-75%) (Figure 6).

Table 1: Distribution of *S. aureus* isolated from the urine of UTI patients according to age and sex.

Age (Year)	Male (%)	Female (%)
0 -10	0 (0%)	01 (4.6%)
11-20	04 (36.4%)	03 (13.6%)
21-30	04 (36.4%)	10 (45.5%)
31-40	0 (0%)	06 (27.3%)
41-50	0 (0%)	0 (0%)
>50	03 (27.3%)	02 (9.1%)
Total	11 (33.3%)	22 (66.7%)

Table 2: Antibiotic susceptibility test of 33 *S. aureus* isolates using cefoxitin.

Number	Organism ID number	Zone of inhibition (mm)	Resistance (R)/Susceptible (S)
1	A1	0	R
2	A2	15	R
3	A3	0	R
4	A4	18.5	R
5	A5	22.5	S
6	A6	26	S
7	A7	20.5	R
8	A8	0	R
9	A9	23.5	S
10	A10	20.5	R
11	A11	23.75	S
12	A12	26.5	S
13	A13	0	R
14	A14	0	R
15	A15	15.25	R
16	A16	23.5	S
17	A17	23.5	S
18	A18	20.5	R
19	A19	19.5	R
20	A20	23.5	S
21	A21	22.5	S
22	A22	23.5	S
23	A23	22.5	S
24	A24	0	R
25	A25	0	R
26	A26	21.25	R
27	A27	21.25	R
28	A28	22.5	S
29	A29	20.25	R
30	A30	25.25	S
31	A31	0	R
32	A32	29.5	S
33	A33	28.5	S

Methicillin- resistant and methicillin -susceptible *S. aureus* strains with breakpoints zone diameter- S ≥ 22 mm, R < 22 mm.

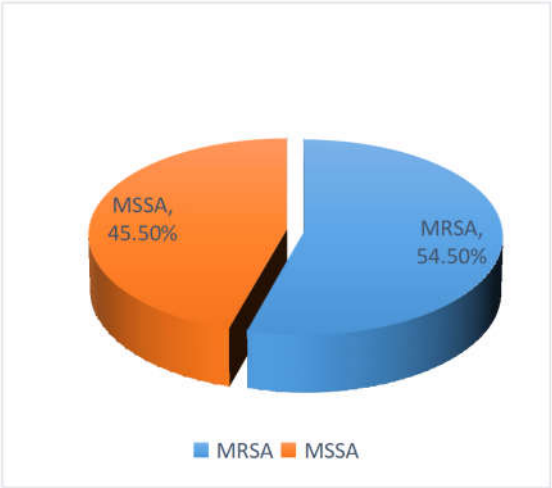

Figure 1: Distribution of *S. aureus* strains isolated from urine.

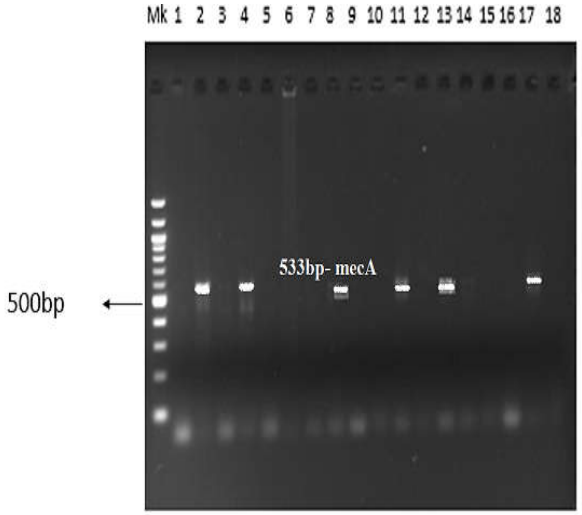

Figure 2: Gel electrophoresis for the PCR amplification products of the *mecA* gene (533 bp) of the 18 MRSA isolates.
Lanes Mk: 100 bp DNA ladder; 3, 5-8, 10, 12, 14-16 and 18: negative isolates for the mecA gene; 2, 4, 9, 11, 13 and 17: positive isolates for the mecA gene.

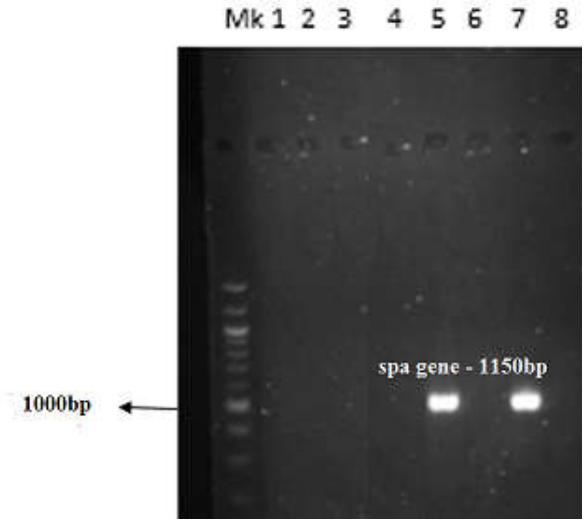

Figure 3: Gel electrophoresis showing the PCR amplification products of the two isolates positive for the *spa* gene (1,150 bp).
Lanes Mk: 1 kb DNA ladder; 1-4, 6 and 8: negative isolates; 5 and 7: positive isolates.

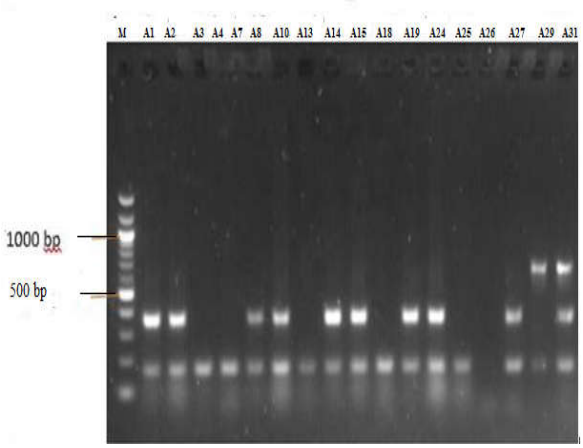

Figure 5: Agarose gel electrophoresis showing RAPD-PCR banding patterns obtained with OPT-17 primer from methicillin-resistant *S. aureus* (MRSA). M: 100 bp DNA ladder.

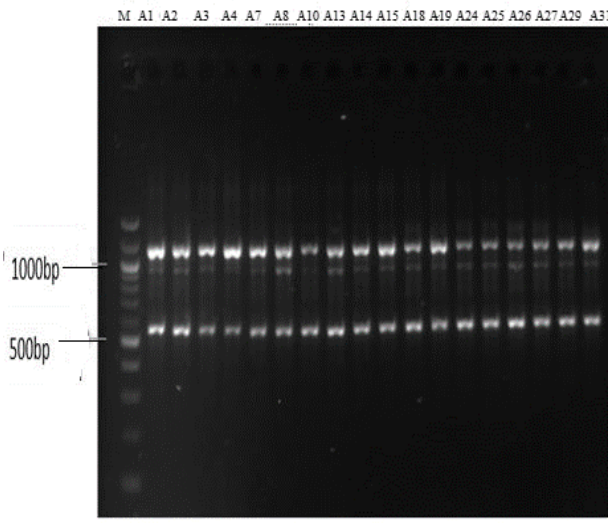

Figure 4: Agarose gel electrophoresis of RAPD-PCR banding patterns obtained with OPT-08 primer from methicillin-resistant *S. aureus* (MRSA). M: 100 bp DNA ladder.

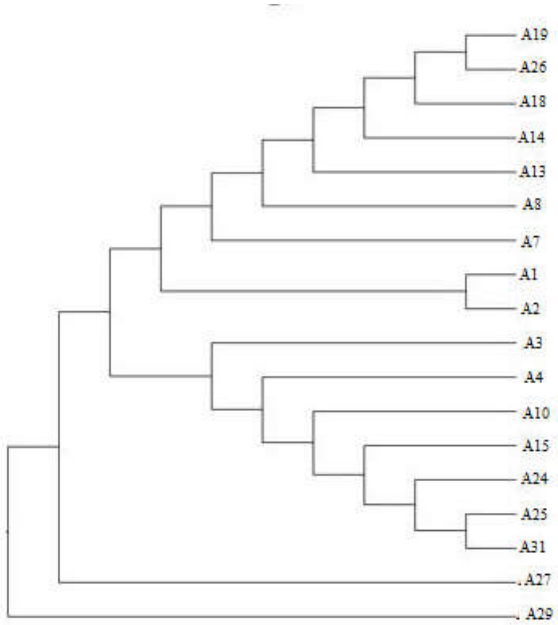

Figure 6: Dendrogram of genetic relationship among 22 strains of methicillin-resistant *S. aureus* (MRSA) obtained with two RAPD primers, OPT-08 and OPT-17.

4.0 Discussion

Methicillin-resistant *S. aureus* (MRSA) is a common cause of infections in hospitals, and the community, with clinical and economic consequences. The 54.5% MRSA revealed in the present study, corresponds to the high rate of MRSA in previous studies, which reported 54% in Egypt (Hafez *et al*., 2009), 51% in Saudi Arabia (Alghaithy *et al*., 2000), and 41.6% in China (Yu *et al*., 2012). However, a lower prevalence of 0.3%-34% has been reported in European countries (Bocher *et al*., 2010). A very high prevalence of MRSA beyond findings in this study has also been reported in Lebanon - 72% (Tokajian *et al*., 2011), and 77.65%-80% in China (Peng *et al*., 2010). Ikeh and Yakeu (2006) found an alarming percentage of 92.6% MRSA among the *S. aureus* isolates from the bacterial flora of nursing care personnel's hands. This higher prevalence might be due to overcrowding in these cities. In addition, variations in MRSA prevalence are attributed to a heterogeneity of variables, including sample nature, specimen forms, different laboratory methods, research population, and study length, among others (Dilnessa and Bitew, 2016). For instance, the increasing prevalence could be due to the usage of methicillin discs for MRSA detection (Onemu and Ophori, 2013; Gorwitz *et al*., 2006). This is due to increased penicillinase activity in *S. aureus* isolates, which brings about false-positive results, when the phenotypic approach is employed, without the presence of the *mecA* gene that might have been wrongly diagnosed as MRSA when methicillin was used to detect MRSA (Onemu and Ophori, 2013; Gorwitz *et al*., 2006).

Methicillin resistance can be caused by the expression of the *mecA* gene, the production of methicillinase, or a combination of the two, i.e., the expression of both PBP2a and methicillinase (Dominguez *et al*., 2002). In this study, it was revealed that only six (33.3%) of the isolates harboured *mecA* gene out of a total of 18 MRSA. Similar outcomes of MRSA with no concomitant possession of the *mecA* gene have been reported by other researchers in previous studies. Adesida *et al.* (2005) reported that out of the 276 *S. aureus* isolated, 26 (9.4%) were phenotypically confirmed MRSA, but only 4 out of the 26 (1.4%) have the *mecA* gene. Similarly, Shittu *et al.* (2006) found 3% phenotypically confirmed MRSA using oxacillin, but only 3 out of 200 (1.5%) possess the *mecA* gene. On the other hand, higher prevalence was observed by Adeiza *et al*. (2019), in which out of 38 phenotypically confirmed MRSA, 42.1% haboured the *mecA* gene. Also, Koosha *et al.* (2016) detected 87.6% *mecA* gene in Iran, while 94.4% *mecA* gene was detected in oxacillin-resistant *S. aureus* isolated from human urine. However, a previous study in Nigeria revealed that the isolates had neither the *mecA* gene nor its protein, PBP2a, which phenotypically were positive for MRSA (Olayinka *et al*., 2009). Variations in results might be attributed to different methods used, including whole genome sequencing, microarrays, traditional PCR, *spa*-typing, and other methods to detect *mecA* (Dilnessa and Bitew, 2016; Deyno *et al*., 2017).

The low prevalence of the *mecA* gene among MRSA reported in previous studies, may be attributed to several biochemical and genetic mechanisms, such as the presence of Borderline Oxacillin-Resistant *S. aureus* (BORSA), which arises from the hyper-expression of the *blaZ* gene, leading to an over-production of β-lactamase (Aarris *et al*., 2025). Thereby, raising the Minimum Inhibitory Concentration (MIC) to resistant level without the presence of *mecA* gene. Additionally, Modified Penicillin-Binding Proteins (MODSA) may alter the binding affinity of PBPs for β-lactam antibiotics, which may account for the low incidence of the *mecA* gene in MRSA reported in previous studies (Gitman *et al*., 2021). Furthermore, *mecA*-negative genotype may also be caused by PCR inhibitors, inadequate DNA yield, the presence of *mecC* (a genetic divergence of *mecA*), and instability of the Staphylococcal Cassette Chromosome mec (SCCmec) (Blanc *et al*., 2011; Schrader *et al*., 2012; Abdelwahab *et al*., 2023).

The pathogenic factor, protein A, of *S. aureus* is encoded by the *spa* gene, which varies in length among strains of the bacterial species. In the present study only two (11.1%) *S. aureus* strains were positive for the *spa* gene. Higher prevalence was reported in northern Nigeria, in which 47.4% of the *spa* gene was present in phenotypically confirmed MRSA isolates in Sokoto State, Nigeria (Adeiza *et al*., 2019). Further studies with

a larger sample size and *spa* types are nevertheless required. Given that the *spa* gene exhibits a high degree of polymorphism, each strain has a different replication sequence and unique number. The repetitive portion of the *spa* gene is known as the X region, and is found at the 3′ end. It is made up of 12 units, each of which has a length of 24 nucleotides. This region of the 24 nucleotides is very variable, in terms of the number and the sequence repeats. Variation is, thus, due to diversity in the X region of *S. aureus* strains (Shakeri *et al*., 2010).

Randomly amplified polymorphic DNA-polymerase chain reaction (RAPD-PCR) is one of the techniques of genotyping, which has been greatly employed to study the spread of *S. aureus.* The method is simple, rapid and economical (Amalraqibshamran *et al*., 2018). This study revealed RAPD primers with operon codes OPT-08 and OPT-17, as suitable markers for genetic diversity and differentiation among MRSA isolates found in urine. The strains that were not typed in this investigation may lack particular chromosomal locations for oligonucleotide primer binding, as reported by Zare *et al.* (2019), in their findings on non-typeable isolates. Also, point mutations and mobile genetic elements such as bacteriophages, plasmids and transposons could result to the absence of bands (El-Jakee *et al*., 2010).

In this study, the results of the dendrogram generated, showed that RAPD-PCR DNA fingerprinting can be used to group MRSA strains into clusters, which can be used to assess evolutionary relationships. The isolates with the largest cluster were obtained from the hospitals at Lagos University Teaching Hospital (LUTH), General Hospital, Shomolu and General hospital, Gbagada. The dendrogram revealed that the strains obtained from the same hospital were closely related, implying that they may have originated from a single clone. Comparable results have been previously reported by Neela *et al*. (2005), where genetically similar strains from the same hospital were found in the same cluster. Therefore, proper hygiene must be strictly adhered to, especially in all healthcare settings, to stop the spread of MRSA.

Limitations

The number of isolates obtained was relatively too small (n= 33), which may limit the generalizability of the findings. Additionally, while RAPD is rapid and economical, it lacks the discriminatory power and reproducibility of pulsed-field gel electrophoresis (PFGE) or Multilocus Sequence Typing (MLST). Furthermore, by using only three RAPD markers, the study may not have covered broader genetic variations. Therefore, future research should employ the use of PFGE or MLST, for high-resolution subtyping, and higher reproducibility and larger sample cohorts, to validate these preliminary results.

5.0 Conclusion

This study revealed that MRSA was highly prevalent in urine, with low carriage of *mecA* and *spa* genes. Therefore, there is a likelihood of increasing resistance to antimicrobial drugs and virulence strains, due to the high incidence of MRSA, and the detection of *mecA* and *spa* genes in some MRSA strains in urine samples from UTI patients. Hence, further molecular surveillance is required, to comprehend the mechanisms of methicillin resistance in phenotypically resistant but *mecA*-negative isolates. Additionally, the significant genotypic diversity of *S. aureus* strains and the spread of specific clones in hospitals were confirmed by this study. Efforts must hence be made for preventing the spread of the disease in hospitals, and the community, which include awareness and strict infection control measures.

Conflict of Interest

None

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