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Prevalence of *mecA* and *vanA* genes in nasal *Staphylococcus aureus* isolates from hospitalized patients in Babcock University Teaching Hospital, Ilishan-Remo, Nigeria

*¹Okunbor, H. N., ^{1,2}Otaigbe, I. I., ^{1,2}Oluwole, T. O., ^{1,2}Elikwu, C. J., and ³Bhola, A. K.

¹Department of Medical Microbiology, Babcock University Teaching Hospital, Ilishan-Remo, Ogun State, Nigeria

²Department of Medical Microbiology and Parasitology, Babcock University, Ilishan-Remo, Ogun State, Nigeria

³ISID-BMGF Research Capacity Building Mentor

*Correspondence to: okunborh@babcock.edu.ng; Tel: +2348059834262; ORCID: [0009-0001-2016-6310](https://orcid.org/0009-0001-2016-6310)

Abstract:

Background: Antimicrobial resistance (AMR) among *Staphylococcus aureus*, particularly methicillin-resistant *S. aureus* (MRSA) and vancomycin-resistant *S. aureus* (VRSA), poses a major global public health challenge. Data on the molecular epidemiology of resistance genes in Nigeria remain limited. The objectives of this study are to determine the prevalence of *mecA*, *vanA*, and *vanB* genes among nasal *S. aureus* isolates, assess antimicrobial susceptibility profiles, and explore associations of prevalence with demographic and clinical characteristics of hospitalized patients in Babcock University Teaching Hospital (BUTH), Ilishan-Remo, Nigeria.

Methodology: This descriptive cross-sectional study was conducted among 400 inpatients admitted to BUTH, Ilishan-Remo, Nigeria for various medical conditions, between June 2023 and June 2024. Sociodemographic and clinical data were obtained through interviewer-administered questionnaires and structured data extraction tools. Nasal swabs were collected from consenting participants who have been hospitalized for at least 48–72 hours. *S. aureus* isolates were identified using standard microbiological methods. Antimicrobial susceptibility testing was performed using the Kirby–Bauer disk diffusion method following CLSI 2023 guidelines. Polymerase chain reaction (PCR) was used to detect *mecA*, *vanA*, and *vanB* genes. Data were analysed using SPSS version 25.0. Associations between categorical variables were tested using Chi-square test and *p* value < 0.05 was considered statistically significant.

Results: *S. aureus* isolates were recovered from 27 (6.8%) of the 400 participants. *mecA* gene was detected in 25 of the 27 isolates (92.6%), representing 6.3% MRSA prevalence among the study participants, while *vanA* was identified in 7 of the 27 isolates (25.9%), representing 1.8% VRSA prevalence among the participants. No isolate carried *vanB* gene. Ward type showed a significant association with *mecA* carriage ($\chi^2=15.336$, $p=0.032$), while demographic characteristics and prior antibiotic use were not significantly associated with resistance gene carriage. All isolates were susceptible to linezolid, tedizolid, ceftaroline, and quinupristin–dalfopristin. High resistance rates were observed for levofloxacin (63.0%), erythromycin (44.4%), and gentamicin (37.0%).

Conclusion: This study reports a high prevalence of methicillin (*mecA*) and vancomycin (*vanA*) resistance genes carriage among *S. aureus* isolates colonizing the nostrils of hospitalized patients in BUTH, Ilishan-Remo, Ogun State, Nigeria. Strengthened infection prevention measures, antimicrobial stewardship, and confirmatory molecular sequencing are urgently needed.

Keywords: *Staphylococcus aureus*, *mecA*, *vanA*, nasal colonization, antimicrobial resistance, Nigeria

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Prévalence des gènes *mecA* et *vanA* chez les isolats nasaux de *Staphylococcus aureus* provenant de patients hospitalisés au Centre hospitalier universitaire Babcock, Ilishan-Remo, Nigéria

*¹Okunbor, H. N., ^{1,2}Otaigbe, I. I., ^{1,2}Oluwole, T. O., ^{1,2}Elikwu, C. J., et ³Bhola, A. K.

¹Département de Microbiologie Médicale, Centre Hospitalier Universitaire Babcock, Ilishan-Remo, État d'Ogun, Nigéria

²Département de Microbiologie Médicale et de Parasitologie, Université Babcock, Ilishan-Remo, État d'Ogun, Nigéria

³Mentor en Renforcement des Capacités de Recherche ISID-BMGF

*Correspondance à: okunborh@babcock.edu.ng; Tél.: +2348059834262; ORCID: [0009-0001-2016-6310](https://orcid.org/0009-0001-2016-6310)

Résumé:

Contexte: La résistance aux antimicrobiens (RAM) chez *Staphylococcus aureus*, en particulier chez les souches résistantes à la méthicilline (SARM) et résistantes à la vancomycine (VRSA), constitue un enjeu majeur de santé publique à l'échelle mondiale. Les données sur l'épidémiologie moléculaire des gènes de résistance au Nigéria restent limitées. Cette étude vise à déterminer la prévalence des gènes *mecA*, *vanA* et *vanB* parmi les isolats nasaux de *S. aureus*, à évaluer les profils de sensibilité aux antimicrobiens et à explorer les associations entre cette prévalence et les caractéristiques démographiques et cliniques des patients hospitalisés au Centre hospitalier universitaire Babcock (BUTH), à Ilishan-Remo, au Nigéria.

Méthodologie: Cette étude descriptive transversale a été menée auprès de 400 patients hospitalisés au BUTH d'Ilishan-Remo, au Nigéria, pour diverses pathologies, entre juin 2023 et juin 2024. Les données sociodémographiques et cliniques ont été recueillies au moyen de questionnaires administrés par un enquêteur et d'outils structurés d'extraction de données. Des prélèvements nasaux ont été effectués chez les participants consentants, hospitalisés depuis au moins 48 à 72 heures. Les isolats de *Staphylococcus aureus* ont été identifiés par les méthodes microbiologiques standard. L'antibiogramme a été réalisé par la méthode de diffusion sur disque de Kirby-Bauer, conformément aux recommandations CLSI 2023. La réaction en chaîne par polymérase (PCR) a été utilisée pour détecter les gènes *mecA*, *vanA* et *vanB*. Les données ont été analysées à l'aide du logiciel SPSS version 25.0. Les associations entre les variables catégorielles ont été testées par le test du χ^2 et une valeur $p < 0,05$ a été considérée comme statistiquement significative.

Résultats: Des isolats de *S. aureus* ont été retrouvés chez 27 (6,8%) des 400 participants. Le gène *mecA* a été détecté dans 25 des 27 isolats (92,6%), soit une prévalence de SARM de 6,3% parmi les participants à l'étude, tandis que le gène *vanA* a été identifié dans 7 des 27 isolats (25,9%), soit une prévalence de VRSA de 1,8% parmi les participants. Aucun isolat ne portait le gène *vanB*. Le type de service hospitalier était significativement associé au portage du gène *mecA* ($\chi^2=15,336$, $p=0,032$), tandis que les caractéristiques démographiques et les antécédents d'utilisation d'antibiotiques n'étaient pas significativement associés au portage de gènes de résistance. Tous les isolats étaient sensibles au linézolide, au tédizolide, à la ceftaroline et à la quinupristine-dalfopristine. Des taux de résistance élevés ont été observés pour la lévofloxacine (63,0%), l'érythromycine (44,4%) et la gentamicine (37,0%).

Conclusion: Cette étude révèle une forte prévalence du portage des gènes de résistance à la méthicilline (*mecA*) et à la vancomycine (*vanA*) chez les souches de *S. aureus* colonisant les narines de patients hospitalisés au BUTH d'Ilishan-Remo, dans l'État d'Ogun, au Nigéria. Le renforcement des mesures de prévention des infections, la gestion des antimicrobiens et le séquençage moléculaire de confirmation sont nécessaires de toute urgence.

Mots-clés: *Staphylococcus aureus*, *mecA*, *vanA*, colonisation nasale, résistance aux antimicrobiens, Nigéria

Introduction:

The global rise of antimicrobial resistance (AMR) represents one of the most pressing public health threats of the 21st century. Low-and-middle-income countries (LMICs) bear a disproportionate burden due to limited diagnostic capacity, weak antimicrobial stewardship, and high rates of infectious diseases (1).

Staphylococcus aureus remains a leading cause of hospital- and community-acquired infections worldwide, causing conditions ranging from mild skin and soft tissue infections to life-threatening diseases such as pneumonia, endocarditis, and sepsis (2). The emergence of methicillin-resistant *S. aureus* (MRSA), primarily mediated by *mecA* gene located on the staphylococcal cassette chromosome mec (SCCmec) has severely complicated treatment. MRSA is resistant to nearly all β -lactam antibiotics, including penicillins, cephalosporins, and carbapenems (3). The global prevalence of MRSA varies considerably, with rates exceeding 50% in many

hospital settings in Asia, Latin America, and Africa (4).

More recently, vancomycin-resistant *S. aureus* (VRSA) has emerged as a critical threat. Vancomycin has traditionally been the last-resort antibiotic for serious MRSA infections. VRSA strains often carry the *vanA* gene, which was originally described in *Enterococcus* species and confers high-level resistance to vancomycin and teicoplanin through alteration of the peptidoglycan precursor terminal D-alanyl-D-alanine to D-alanyl-D-lactate (5,6). The acquisition of both *mecA* and *vanA* genes by *S. aureus* produces a highly-resistant pathogen with extremely limited therapeutic options, significantly increasing morbidity and mortality of infections caused by these strains (7). Although VRSA remains rare globally, with fewer than 100 confirmed cases reported worldwide, its emergence in hospital environments with poor infection control practices is a sentinel event (8).

In Nigeria, the burden of AMR is substantial but poorly characterised (9). Several

studies have reported MRSA prevalence rates ranging from 30% to 80% among clinical isolates (10,11). However, most surveillance efforts have relied solely on phenotypic testing, and few have systematically examined the molecular epidemiology of resistance genes (12,13). There is a particular scarcity of data on *vanA* carriage among *S. aureus* in Nigeria, with most published studies reporting rates below 5% (14, 15). Understanding the molecular patterns of resistance and their association with patient demographics and clinical settings is essential for designing targeted interventions, strengthening infection prevention and control (IPC) policies, and informing empirical therapy guidelines.

Nasal colonisation with *S. aureus* is a well-established risk factor for subsequent infection, particularly in surgical patients and immunocompromised hosts (16), who also have the capacity to transmit *S. aureus* in the healthcare setting. This study aimed to determine the prevalence of *mecA*, *vanA* and *vanB* genes in *S. aureus* isolates colonizing the nostrils of inpatients in Babcock University Teaching Hospital (BUTH), a tertiary hospital in Nigeria, to assess antimicrobial susceptibility profiles, and to explore associations with demographic and clinical variables, including ward type and recent antibiotic use.

Materials and method:

Study design and setting:

This was a descriptive cross-sectional study integrating laboratory investigations and retrospective review of patient case notes. The study was conducted at BUTH, a 250-bed tertiary healthcare facility located in Ilishan-Remo, Ogun State, Nigeria, between June 2023 and June 2024. BUTH provides multidisciplinary services, including medical, surgical, paediatric, obstetric, and critical care.

Eight hospital wards were purposively selected to ensure representation across diverse clinical settings and antimicrobial prescribing patterns; Male Medical Ward, Female Medical Ward, Male Surgical Ward, Female Surgical Ward, Obstetric Ward, Paediatric Ward, Accident and Emergency (A &E) Unit, and Intensive Care Unit (ICU).

Ethical considerations:

Ethical approval was obtained from the Babcock University Health Research Ethics Committee (BUHREC 613/23). Written informed consent was obtained from all participants for prospective sample collection. A waiver of informed consent was granted for the retrospective component of the study.

Study population and sampling:

The study population included hospitalized individuals who had spent a minimum of 48 to 72 hours in the facility. Exclusion criteria were: patients with nasogastric tubes; individuals with incomplete records or those discharged before 48 hours of admission. A census sampling approach was used for retrospective data extraction, while 400 consenting inpatients were recruited for nasal swab collection. Overall, 421 eligible patients were identified, of whom 21 were excluded, leaving 400 participants enrolled.

Data collection:

Sociodemographic and clinical data (age, sex, marital status, educational level, occupation, ward of admission, prior antibiotic use within the past four months, and chronic medical conditions) were obtained through interviewer-administered questionnaires and structured data extraction tools. For the retrospective arm, data were sourced from case notes, medication charts, nursing records, and laboratory reports.

Specimen collection and laboratory analysis:

Nasal swab specimens were collected from participants on the third day of admission using sterile swabs transported in peptone water. Swabs sent to the medical microbiology laboratory of BUTH were processed within two hours and inoculated onto Mannitol Salt Agar for selective isolation of *S. aureus*. Isolates were identified based on colony morphology, Gram staining, catalase test, coagulase test, and confirmed using polymerase chain reaction assay.

Antimicrobial susceptibility testing:

Antibiotic susceptibility testing of *S. aureus* isolates was performed using the modified Kirby-Bauer disk diffusion method in accordance with Clinical and Laboratory Standards Institute (CLSI) 2023 guidelines (17). The antibiotics tested included cefoxitin (30µg), teicoplanin (30 µg), ceftaroline (30µg), gentamicin (10µg), erythromycin (15µg), tetracycline (30µg), levofloxacin (5µg), clindamycin (2µg), trimethoprim-sulfamethoxazole (1.25/23.75µg), quinupristin-dalfopristin (15µg), linezolid (30µg), tedizolid (2µg), and telavancin using E-test strips (0.002–32µg/mL). Inoculum density was standardized to 0.5 McFarland turbidity standard. *S. aureus* ATCC 29213 and *Enterococcus faecalis* ATCC 52199 were used as control strains.

Molecular detection of *nuc*, *mec* and *van* genes:

The molecular analysis was carried out

in the Molecular Laboratory of BUTH. DNA was extracted from 200 µL of pelleted *S. aureus* isolates using the Quick-DNA™ Miniprep Kit (Lot 221058, Cat 4060). The extracted DNA was stored at 4°C until use. PCR amplification was performed in a 5× concentrated ready-to-use solution containing FIREPol® DNA polymerase, 2.5 mM MgCl₂, 200 µM of each dNTP, 0.4 M Tris-HCl, 0.1 M (NH₄)₂, and 0.1% w/v Tween-20. Each 25µL reaction mixture contained 0.5µL forward primer, 0.5µL reverse primer, 9µL nuclease-free water, and 5µL template DNA. Primer sequences, amplicon sizes, and annealing temperatures are shown in Table 1.

Thermal cycling conditions were optimized for each gene. For *vanA*, initial denaturation at 96°C for 180 seconds, 35 cycles of 94°C for 45 seconds, 46°C for 45 seconds, and 72°C for 60 seconds, and final extension at 72°C for 360 seconds. For *vanB*; the same protocol with annealing at 50°C. For *mecA*; same protocol with annealing at 54°C. For the *nuc* gene; 37 cycles of 94°C for 60 seconds, 55°C for 30 seconds, and 72°C for 90 seconds, and final extension at 72°C for 210 seconds.

Positive controls included *E. faecium* ATCC 51559 (*vanA*), *E. faecalis* ATCC 51299 (*vanB*), and *S. aureus* ATCC 43300 (*mecA*). Negative controls (no template DNA) were included in each PCR run. PCR products (5 µL) were

resolved on 1.5% agarose gels in 1× TBE buffer (pH 8.3) at 150 V for 2 hours. Gels were stained with SafeView® dye and visualised under ultra-violet light (Table 1).

Data analysis:

Data were cleaned and coded in Microsoft Excel and analysed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics summarised demographic and clinical data. Associations between categorical variables (resistance gene carriage and demographic/clinical variables) were tested using the Chi-square test. A *p*-value less than 0.05 was considered statistically significant.

Results:

Prevalence of *nuc*, *mecA* and *vanA* genes in *S. aureus* isolates:

Of the total of 400 participants enrolled, 27 *S. aureus* isolates were recovered and confirmed by *nuc* gene amplification (Fig 1), giving 6.8% *S. aureus* nasal carriage rate in the study. *mecA* gene was detected in 25 of the 27 isolates (92.6%) (Fig 2), representing a 6.3% MRSA prevalence among the study participants. *vanA* gene was detected in 7 isolates (25.9% of isolates) (Fig 3), representing VRSA prevalence of 1.8% among the study participants. No isolate carried *vanB* gene (Fig 4).

Table 1: Primer sequences, amplicon sizes and PCR conditions

Gene	Primer sequence (5'–3')	Amplicon size (bp)	Annealing temp (°C)	Positive control
<i>mecA</i>	F: GTAGAAATGACTGAACGTCCGATAA R: CCAATTCACATTGTTTCGGTCTAA	310	54	<i>S. aureus</i> ATCC 43300
<i>vanA</i>	F: GGGAAAACGACAATTGC R: GTACAATGCGGCCGTTA	732	46	<i>E. faecium</i> ATCC 51559
<i>vanB</i>	F: GTGACAAACCGGAGGCGAGGA R: CCGCCATCCTCCTGCAAAAAA	647	50	<i>E. faecalis</i> ATCC 51299
<i>Nuc</i>	F: GCGATTGATGGTGATACGGTI R: AGCCAAGCCTTGACGAATAAGC	279	55	<i>S. aureus</i> (species control)

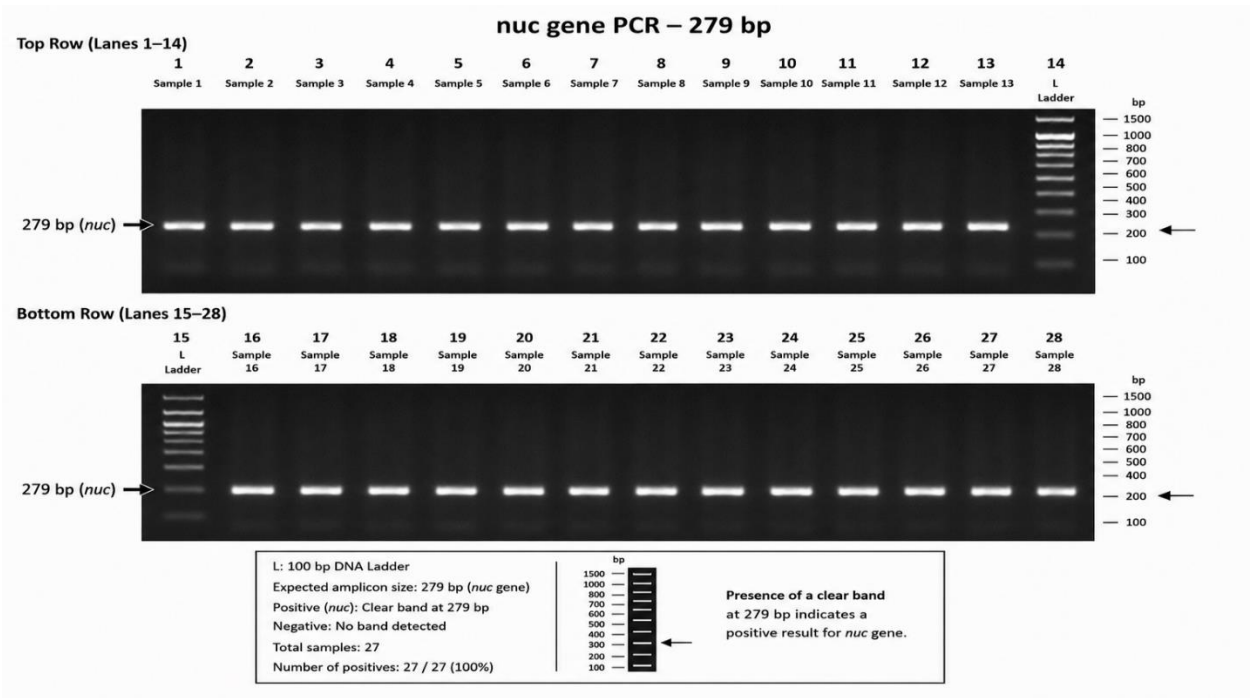


Fig. 1: Gel electrophoresis of PCR products (*nuc* gene at 279 bp)

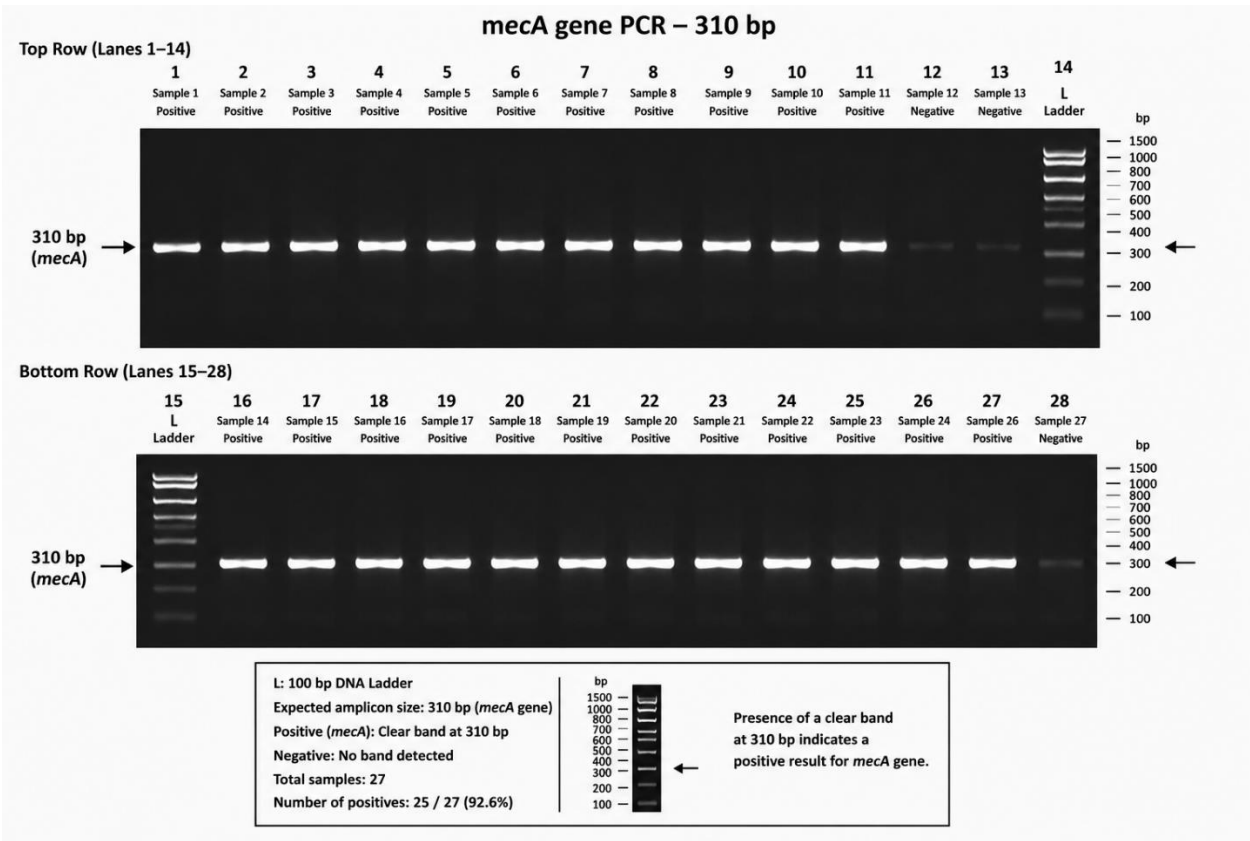


Fig 2: Gel electrophoresis of PCR products (*mecA* gene at 310 bp)

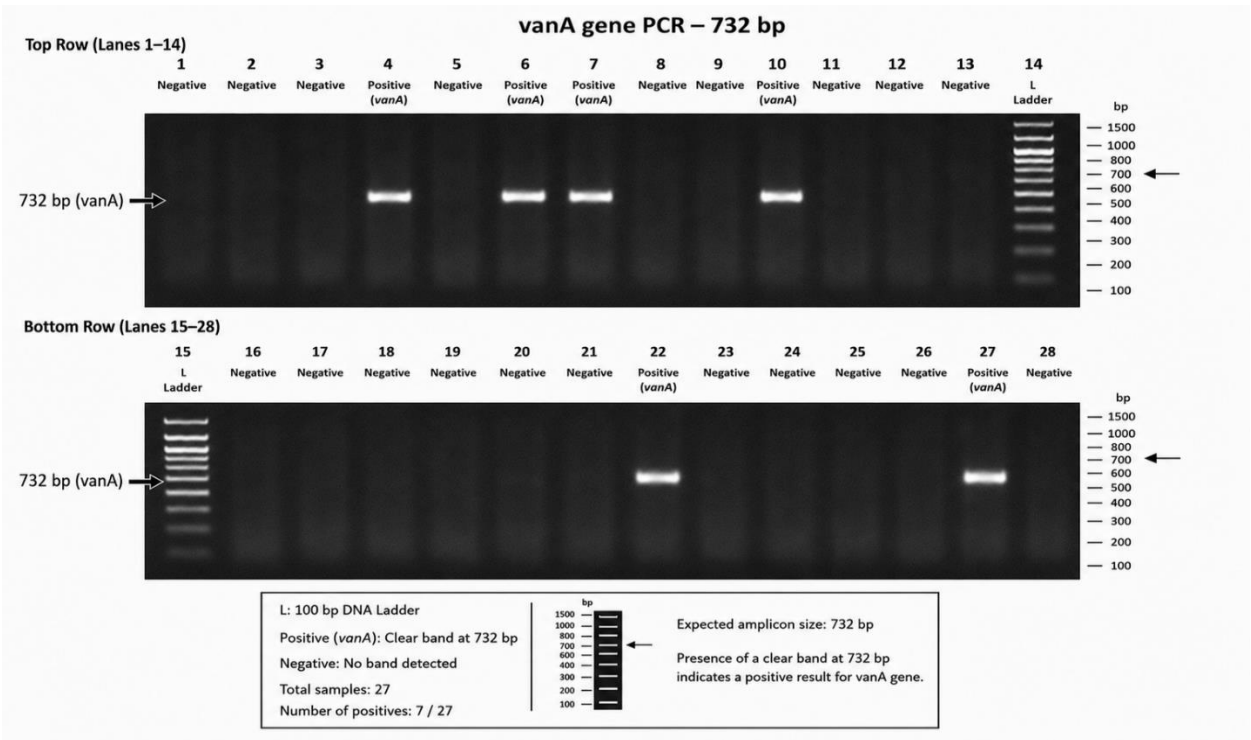


Fig 3: Gel electrophoresis of PCR products (*vanA* gene at 732 bp)

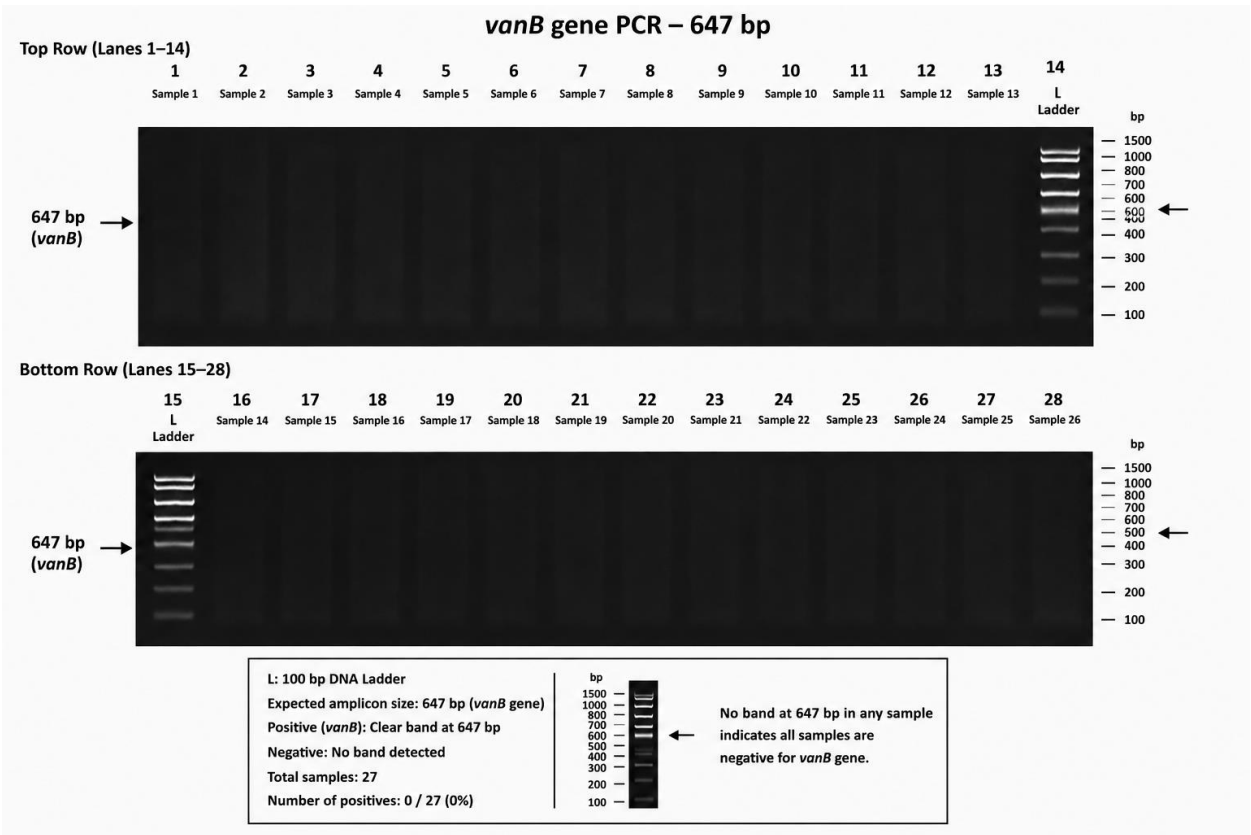


Fig 4: Gel electrophoresis of PCR products (*vanB* gene not amplified at expected 647 bp)

Association between resistance gene carriage and demographic/clinical variables of participants with *S. aureus* nasal colonization:

The highest frequency of *vanA*-positive *S. aureus* isolates was observed among participants aged 18–35 years (30.0%, n=3/10 isolates). However, age was not significantly associated with *vanA* carriage ($\chi^2=0.878$, $p=0.928$). Similarly, gender, occupation, marital status, educational level, prior antibiotic use, ward type, and chronic medical conditions showed no significant associations with *vanA* carriage (Tables 2a and 2b).

Table 2a: Association between *vanA* gene carriage and demographic characteristics of participants with *Staphylococcus aureus* nasal colonization

Variable	<i>vanA</i> -positive (n, %)	Total <i>S. aureus</i> (n)	χ^2	df	<i>p</i> -value
Age group (years)					
5–17	1 (33.3)	3	0.878	4	0.928
18–35	3 (30.0)	10			
36–45	2 (25.0)	8			
46–65	1 (25.0)	4			
>65	0	2			
Gender					
Female	5 (22.7)	22	0.686	1	0.407
Male	2 (40.0)	5			
Occupation					
Unskilled	2 (33.3)	6	0.530	2	0.767
Semi-skilled	0	1			
Skilled	5 (25.0)	20			
Marital status					
Single	3 (37.5)	8	1.037	2	0.596
Married	4 (22.2)	18			
Widowed	0	1			
Education level					
No formal education	0	1	0.791	3	0.852
Primary	0	1			
Secondary	3 (30.0)	10			
Tertiary	4 (26.7)	15			

Table 2b: Association between *vanA* gene carriage and clinical characteristics of participants with *Staphylococcus aureus* nasal colonization

Variable	<i>vanA</i> -positive (n, %)	Total <i>S. aureus</i> (n)	χ^2	df	<i>p</i> -value
Antibiotic use (past 4 months)					
Yes	5 (23.8)	21	0.032	1	0.858
No	2 (33.3)	6			
Ward type					
Accident & Emergency	1 (50.0)	2	4.319	7	0.742
Paediatric	1 (50.0)	2			
Female Medical	1 (20.0)	5			
Male Medical	0 (0.0)	1			
Female Surgical	1 (20.0)	5			
Male Surgical	1 (50.0)	2			
ICU	0 (0.0)	1			
Obstetric	2 (22.2)	9			
Chronic medical conditions					
Yes	4 (25.0)	16	0.048	1	0.827
No	3 (27.3)	11			
Total	7 (25.9)	27			

All *S. aureus* isolates (100%) from participants aged 5–17 years, 18–35 years, 46–65 years, and >65 years were *mecA*-positive, while 75.0% of isolates from the 36–45 years age group carried the gene but age was not significantly associated with *mecA* carriage ($\chi^2=5.130$, $p=0.274$). Gender, occupation, marital status, educational level, prior antibiotic use, and chronic medical conditions also showed no significant associations (Tables 3a and 3b).

Ward type demonstrated a statistically

significant association with *mecA* carriage ($\chi^2=15.336$, $p=0.032$). All *S. aureus* (100.0%) were *mecA* positive from accident and emergency (A & E), paediatric, female medical, male medical, male surgical, and obstetric wards. The female surgical ward had 80.0% (4 of 5) of the *S. aureus* positive for *mecA* while no *mecA*-positive isolate was detected in the ICU, although there was only one patient in the ICU at the time of the study (Table 3b).

Table 3a: Association between *mecA* gene carriage and demographic characteristics of participants with *Staphylococcus aureus* nasal colonization

Variable	<i>mecA</i> -positive (n, %)	Total <i>S. aureus</i> (n)	χ^2	df	p-value
Age group (years)					
5–17	3 (100.0)	3	5.130	4	0.274
18–35	10 (100.0)	10			
36–45	6 (75.0)	8			
46–65	4 (100.0)	4			
>65	2 (100.0)	2			
Gender					
Female	20 (90.9)	22	0.491	1	0.484
Male	5 (100.0)	5			
Occupation					
Unskilled	6 (100.0)	6	0.756	2	0.685
Semi-skilled	1 (100.0)	1			
Skilled	18 (90.0)	20			
Marital status					
Single	8 (100.0)	8	1.080	2	0.583
Married	16 (88.9)	18			
Widowed	1 (100.0)	1			
Education level					
No formal education	1 (100.0)	1	1.728	3	0.631
Primary	1 (100.0)	1			
Secondary	10 (100.0)	10			
Tertiary	13 (86.7)	15			

Table 3b: Association between *mecA* gene carriage and clinical characteristics of participants with *Staphylococcus aureus* nasal colonization

Variable	<i>mecA</i> -positive n (%)	Total <i>S. aureus</i> (n)	χ^2	df	p-value
Antibiotic use (past 4 months)					
Yes	19 (90.5)	21	0.617	1	0.432
No	6 (100.0)	6			
Ward type					
Accident & Emergency	2 (100.0)	2	15.336	7	0.032*
Paediatric	2 (100.0)	2			
Female Medical	5 (100.0)	5			
Male Medical	1 (100.0)	1			
Female Surgical	4 (80.0)	5			
Male Surgical	2 (100.0)	2			
ICU	0 (0.0)	1			
Obstetric	9 (100.0)	9			
Chronic medical conditions					
Yes	14 (87.5)	16			
No	11 (100.0)	11			
Total	25 (92.6)	27			

*Statistically significant at $p < 0.05$

Table 4: Antimicrobial susceptibility of *Staphylococcus aureus* nasal isolates

Antibiotic (disk content)	Susceptible n (%)	Intermediate n (%)	Resistant n (%)
Cefoxitin (30 µg) – MRSA	17 (63.0)	0	10 (37.0)
Teicoplanin (30 µg)	25 (92.6)	0	2 (7.4)
Ceftaroline (30 µg)	27 (100)	0	0
Gentamicin (10 µg)	17 (63.0)	0	10 (37.0)
Erythromycin (15 µg)	15 (55.6)	0	12 (44.4)
Tetracycline (30 µg)	19 (70.4)	0	8 (29.6)
Levofloxacin (5 µg)	10 (37.0)	0	17 (63.0)
Clindamycin (2 µg)	23 (85.2)	1 (3.7)	3 (11.1)
Trimethoprim-sulfamethoxazole (1.25/23.75 µg)	23 (85.2)	4 (14.8)	0
Quinupristin-dalfopristin (15 µg)	27 (100)	0	0
Linezolid (30 µg)	27 (100)	0	0
Tedizolid (2 µg)	27 (100)	0	0
Telavancin (E-test) – VRSA	20 (74.1)	0	7 (25.9)

Antimicrobial susceptibility profile:

All 27 isolates (100.0%) were susceptible to ceftaroline, quinupristin-dalfopristin, linezolid, and tedizolid. Teicoplanin also demonstrated high activity (92.6% of isolates susceptible) as well as clindamycin and trimethoprim-sulfamethoxazole (with 85.2% isolates susceptibility to each). Resistance rates were highest for levofloxacin (63.0%), followed by erythromycin (44.4%), gentamicin (37.0%), ceftaxitin (37%), and tetracycline (29.6%). Resistance to telavancin, suggestive of a VRSA phenotype, was detected in 7 of 27 isolates (25.9%) (Table 4).

Discussion:

Globally, *S. aureus* remains a significant pathogen, with increasing resistance to methicillin and vancomycin posing serious public health threats (3,4). Our study recorded a *mecA* prevalence of 92.6% among *S. aureus* isolates (prevalence of 6.3% among the study participants) is similar to reports from Cameroon (35) and previous Nigerian studies that reported 70–94% (36–38).

Regarding *vanA*, global participant-level carriage among *S. aureus*-colonised individuals typically ranges from 0.1–0.5% (18–41). National surveillance programmes in Europe and North America report *vanA*-positive *S. aureus* as exceedingly rare, with annual incidence rates

below 0.01% (42,22). Our finding of 1.8% participant-level *vanA* carriage is close to findings in some African countries, signalling a potential public health emergency (43,44). A Kenyan tertiary hospital study reported participant level *vanA* carriage of 1.4% among *S. aureus* carriers (34,45), and a multicentre sub-Saharan African meta-analysis yielded a pooled prevalence of 0.4–1.0% (32,46). Our 1.8% rate exceeds these averages and suggests potential regional variations for emergence of glycopeptide resistance (47,48).

Although a statistically significant association was observed between *mecA* carriage and ward type ($p=0.032$), this finding should be interpreted with caution. The apparent significance may have been influenced by the ICU category, which included only one patient and no *mecA*-positive isolate. Such a small sample size may generate a potentially misleading association. Indeed, *mecA*-positive MRSA prevalence was consistently high across the remaining wards (80–100%). This finding mirrors studies from South Africa and Tanzania, where certain hospital units were identified as MRSA colonisation hotspots (49,50). Potential reasons for this include shared healthcare workers, inadequate hand hygiene practices, environmental persistence of resistant strains, and high rate of antibiotic use in specific units.

The absence of a statistically significant association between recent antibiotic use and resistance gene carriage should also be interpreted cautiously. While antibiotic exposure is a well-established driver of antimicrobial resistance (44), the specific antibiotics used by patients were not documented in this study. Consequently, some patients may not have been exposed to antibiotics that exert selective pressure for the emergence or persistence of MRSA. This lack of detailed antibiotic exposure data may have obscured a true relationship between antibiotic use and resistance gene carriage. In addition, the relatively small sample size and potential recall bias may have further limited the ability to detect such an association.

The lack of association with demographic variables is consistent with some previous studies (31), although it contrasts with findings from others (34). All isolates remained fully susceptible to linezolid, tedizolid, ceftaroline, and quinupristin-dalfopristin, consistent with other African studies (51,52). However, access and affordability of these antibiotics remain major concerns in Nigeria's public healthcare sector (53). Resistance to levofloxacin (63%), erythromycin (44%), and gentamicin (37%) severely limits empirical treatment options.

Beyond the absence of confirmatory gene sequencing, this study has several limitations. The sample size ($n=27$) is small, restricting generalisability. Only nasal swabs were analysed, excluding other sources. Clonal typing was not performed, limiting insights into transmission dynamics. Key risk factors such as prior hospitalisation, surgical history, length of stay, and invasive device use were not assessed. Healthcare workers and environmental reservoirs were not screened. The cross-sectional design prevents determination of causality.

Pending confirmation of *vanA* findings by sequencing, the results should not change clinical practice. If confirmed, they would represent a public health emergency requiring immediate IPC interventions. Priority actions include; (i) securing funding for sequencing of biobanked isolates; (ii) sequencing of all *vanA*-positive isolates; (iii) establishing a reference laboratory confirmation pathway for VRSA in Nigeria; and (iv) conducting a larger, multi-centre study.

Even without confirmation of the *vanA*

gene findings, the high prevalence of *mecA* and its significant clustering in most wards of the hospital warrant action. Routine screening for MRSA colonisation on admission to high-risk wards, decolonisation using mupirocin, contact isolation precautions, and enhanced environmental disinfection should be considered (46).

Conclusion:

This study reports high participant-level *mecA* (6.3%) and *vanA* (1.8%) nasal carriage rates among hospitalized patients in BUTH Ilisan-Remo, Nigeria. However, these findings require independent confirmation (e. g. next-generation sequencing) before they can be accepted as valid. The biobanked isolates provide a valuable resource for this next step. The high *mecA* carriage in *S. aureus* isolates from inpatients across the major wards of the hospital suggests possible nosocomial transmission of MRSA, warranting further investigation using molecular typing. We caution against over-interpretation of the *vanA* data and recommend confirmatory studies as the immediate priority.

Contributions of authors:

OHN conceived and designed the study, participated in data collection, performed data analysis and interpretation, drafted the manuscript; OII contributed to the study design, supervised the research, critically reviewed the manuscript for intellectual content, and approved the final version; OTO contributed to data interpretation, manuscript review and revision, and approved the final version; ECJ contributed to data analysis and interpretation, reviewed the manuscript, and approved the final version; and AKB contributed to the study design, supervised the research, critically reviewed the manuscript for intellectual content, and approved the final version.

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Conflict of interest:

No conflict of interest is declared

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