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Genomic epidemiology of invasive *Staphylococcus aureus* in southwestern Nigeria

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Abstract

Background: *Staphylococcus aureus* is a leading cause of hospital and community-associated human infections. Despite its clinical importance, molecular epidemiology data in Nigeria are limited. This study characterized bloodstream *S. aureus* isolates submitted to the Nigerian Antimicrobial Resistance Surveillance System.

Methods: Bloodstream *S. aureus* isolates from six tertiary healthcare institutions in Nigeria collected between 2016 and 2021 were characterized by antimicrobial susceptibility testing and whole-genome sequencing. Bioinformatic analyses were conducted using publicly available pipelines in addition to contextualization with publicly available sequences.

Results: Of 138 isolates, 85 (61.6%) were methicillin-resistant *S. aureus* (MRSA). A total of 31 *spa* types, 14 sequence types (STs), eight clonal complexes (CCs) - CC1, CC5, CC8, CC15, CC30,

CC45, CC88, CC121 - and two singletons (ST152 and ST11258) were identified. ST152 (n=48, 35%) was the predominant lineage, followed by ST772 (n=21, 15%), ST8 (n=16, 12%) and ST789 (n=13, 9%). These lineages were detected across sentinel sites, indicating widespread geographic dissemination. ST772-MRSA isolates possessed the highest number of antimicrobial (AMR) and virulence genes. ST789 was characterized by numerous AMR genes in both MRSA and MSSA backgrounds, whereas ST8 and ST152-MRSA isolates demonstrated abundant virulence genes, with comparatively few AMR genes. ST152, ST772, and ST789 genomes generally clustered together relative to publicly available genomes of the corresponding STs, with lineage-specific subclades differentiated by AMR profiles.

Conclusion: Predominance of ST152-MRSA, ST772-MRSA, and ST789-MRSA among bloodstream *S. aureus* isolates in Nigeria is a significant public health concern. Sustained genomic surveillance is critical to inform infection prevention and control in Nigeria.

Keywords: Bloodstream *S. aureus*, methicillin-resistant *S. aureus*, antimicrobial resistance, virulence, genomic surveillance.

Background

Staphylococcus aureus is an opportunistic pathogen responsible for a wide range of diseases, from mild skin and soft-tissue infections to life-threatening sepsis and necrotizing infections [1–3]. Globally, *S. aureus* is a leading cause of morbidity and mortality and ranks among the most common causes of infection-related deaths [4–6]. The burden is particularly pronounced in Africa, with gaps in healthcare infrastructure and deficiencies in surveillance [5, 7]. The emergence and spread of antimicrobial-resistant *S. aureus*, particularly methicillin-resistant *S. aureus* (MRSA),

have further complicated treatment and control strategies [8, 9]. Antimicrobial resistance (AMR) was estimated to account for 1.27 million deaths globally in 2019, with 130 deaths per 100,000 in West Africa. Of these, 100,000 deaths were attributable to *S. aureus*, the second most frequent cause of such antimicrobial resistance-related deaths on the continent [5]. However, the data on which these estimates are based were sparse for West Africa. Reports of extensively drug-resistant *S. aureus* continue to emerge, underscoring the urgency for surveillance and new therapeutic strategies [10, 11]. Consequently, the World Health Organization has designated *S. aureus* as a priority pathogen for antibiotic research and development [12–14].

Several virulence factors, including haemolysins, enterotoxins, leukocidins, and immune evasion factors, mediate the pathogenicity of *S. aureus* [15, 16]. Panton-Valentine Leukocidin (PVL) is a pore-forming cytotoxin associated with severe tissue damage and invasive infections [17]. The prevalence of PVL-positive *S. aureus* varies geographically, by clonal lineages and methicillin-resistance status [17]. Notably high rates have been reported in Africa, ranging from 0.3% to 100% [18, 19], including in Nigeria, where prevalence rates from 39.3% to 56.3% have been observed [20–22].

Despite its clinical significance, comprehensive data on the molecular epidemiology of *S. aureus* in Nigeria remain scarce. Existing studies on multi-drug-resistant staphylococci in Nigeria are often limited by small sample sizes or restricted geographic scope [18, 23–28]. The Nigerian Antimicrobial Resistance Surveillance Network was established in 2017 to address these gaps by monitoring WHO-priority organisms, including *S. aureus*, from invasive infections [29, 30]. In this study, isolates submitted to the reference laboratory from 2016 to 2021 were characterized to gain insights into the population structure, AMR and virulence properties of this pathogen causing life-threatening bacteremia in South-West and one site in North-Central Nigeria.

Methods

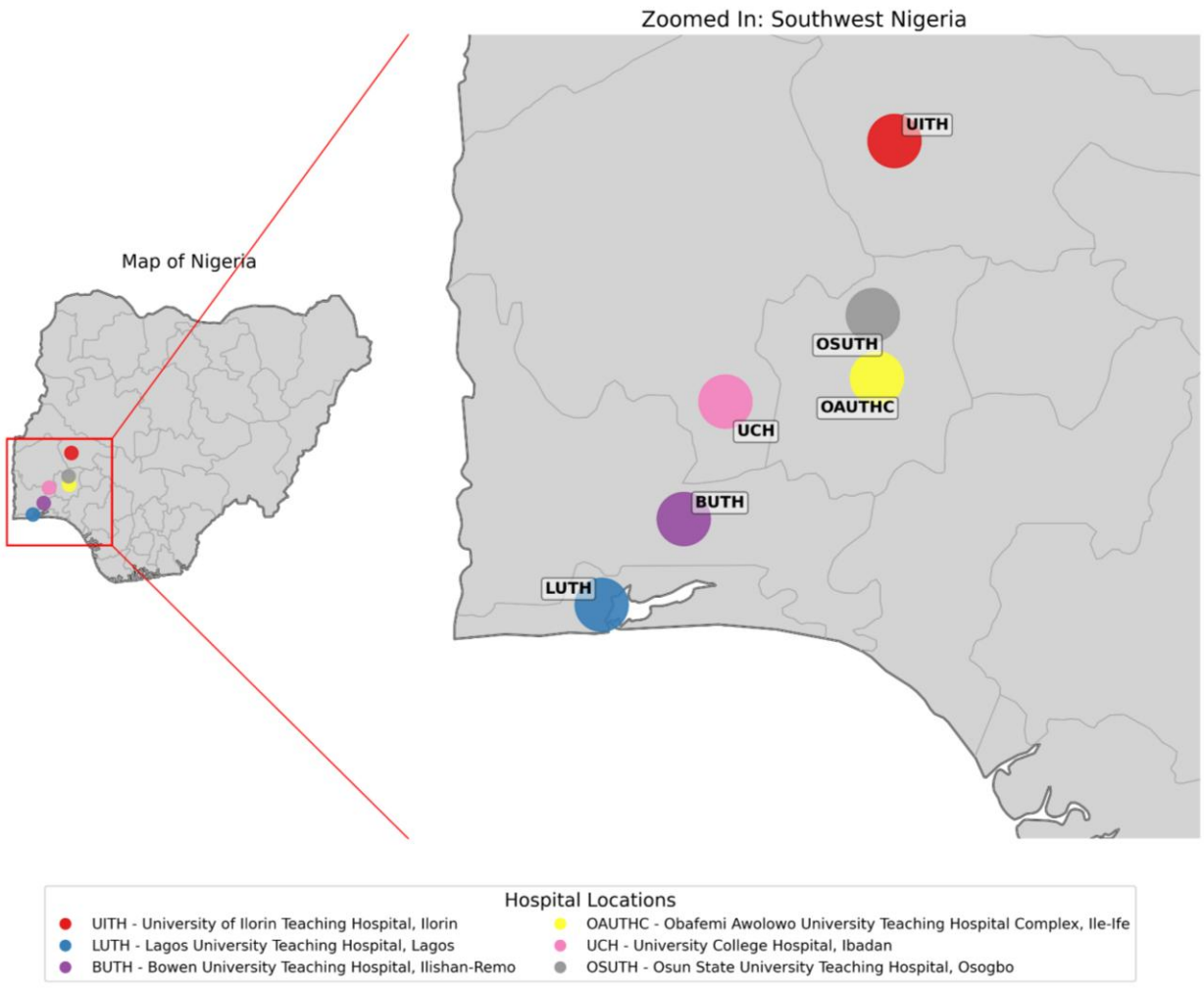


Figure 1: Map of Nigeria showing location of sentinel sites from which isolates were obtained

Strains: This study included *S. aureus* isolates recovered from positive blood cultures at sentinel laboratories participating in the Nigerian AMR Surveillance Network between 2013 and 2021. Isolates were submitted to the National Reference Laboratory (NRL) from five tertiary healthcare institutions in the South-West (SW) and one in North-Central (NC) regions in Nigeria. Those located in SW Nigeria include University College Hospital, Ibadan, Oyo State (UCH), Lagos

University Teaching Hospital, Idi-Araba, Lagos State (LUTH), Babcock University Teaching Hospital, Ilishan, Ogun State (BUTH). Others in SW Nigeria comprise Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife, Osun State (OAUTHC), and Osun State University Teaching Hospital, Osogbo, Osun State (OSUTH). The University of Ilorin Teaching Hospital, Ilorin, Kwara State (UITH) was the only institution in NC, Nigeria (Figure 1).

Antimicrobial susceptibility testing: Upon receipt at the NRL, isolates were cultured on Mannitol Salt Agar (Oxoid, England) and confirmed as *S. aureus* using the Biomerieux Vitek 2 Compact system with a GP ID card (Ref: 21342). Antimicrobial susceptibility testing (AST) was performed using the Biomerieux Vitek 2 Compact system with AST P628 card (Ref: 414534). AST results were interpreted according to the Clinical and Laboratory Standards Institute guidelines [31] and analyzed using WHONET [32]. Statistical comparisons between MRSA and MSSA resistance rates were conducted using Fisher's exact test.

Whole-genome sequencing: DNA extraction was performed using the MP Biomedicals Fast Spin Kit for Soil (Cat No: 116560-200), adapted for bacteria, with the Fast Prep Equipment. DNA was quantified with a QubitFlex Fluorometer (Invitrogen Ref: Q33326) using the dsDNA broad range kit (catalogue no. Q32853). DNA libraries were prepared by fragmentation with Covaris LC220 and thereafter with the NEBNEXT Ultra II Library Prep Kit for Illumina with 384-unique indexes (Cat. No: E6617L) following the manufacturer's instructions. The normalized libraries were then sequenced on an Illumina HiSeq X10 or Miseq platform, using 150 bp paired-end chemistry. Reads were deposited on the European Nucleotide Archive with the project ID PRJEB29739. Individual accession numbers are in additional file 1.

Sequence Analyses: Raw reads were *de-novo* assembled using publicly available GHRU pipelines (https://gitlab.com/cgps/ghru/pipelines/dsl2/pipelines/assembly), as described previously [33].

The pipeline includes workflows for adapter trimming, contamination detection, genome assembly, post-assembly quality control and species calling using the respective tools, trimmomatic, ConFindr, SPAdes, multiqc qualifyr and Bactinspector. Multi-Locus Sequence Typing was performed with the Oxford scheme, which is based on seven housekeeping genes [34, 35] using GHRU pipelines (https://www.protocols.io/view/ghru-genomic-surveillance-of-antimicrobial-resista-bpn6mmhe) and Pathogenwatch [36]. SCC*mec* and *spa* typing of the isolates were established using the *SCCmec* [37] and CGE's *spa* typer, respectively [38]. AMR genes were identified using AMRFinderPlus – v3.11.4 [39], while virulence genes and plasmid replicons were detected using VirulenceFinder – v3.2.1 [40] and PlasmidFinder – v2.1 [41], respectively, implemented with the default parameters.

Phylogeny based on single-nucleotide polymorphism was computed following the GHRU protocol (https://gitlab.com/cgps/ghru/pipelines/snp_phylogeny). The closest *S. aureus* strain to our isolates was automatically selected by the Bactinspector tool and was used as the reference (Accession number: NC_002953.3). Reads were trimmed and mapped to the selected reference. Variants were called and assembled into pseudogenomes, which were then used to construct a maximum likelihood phylogenetic tree. The phylogeny of the isolate collection was constructed, and the genomes were compared with publicly available genomes on Pathogenwatch [36]. Phylogenetic trees and isolate-associated data were visualized with Microreact [42] and iTOL [43]. Sankey plots were generated to compare the distribution of STs between isolates from this study and those from previous investigations across two sentinel sites.

Results

The Nigerian antimicrobial resistance surveillance network was recently launched and started with a few sentinel laboratories [29]. Six of 13 sentinel sites contributed *S. aureus* isolates between 2013 and 2021. OAUTHC contributed the bulk of isolates (n=46, 33%), followed by UCH (n=41, 30%), UITH (n=34, 25%), LUTH (n=14, 10%), OSUTH (n=2, 2%) and BUTH (n=1, 1%). Five of the contributing facilities are within the Southwest geopolitical zone and UITH is in the proximal North Central zone.

Antibiotic susceptibility of the isolates

Only two isolates were susceptible to all the antibiotics tested. High *S. aureus* susceptibility rates were noted for tigecycline (n=136, 98.6%), nitrofurantoin (n=135, 97.8%), teicoplanin (n=132, 95.7%), rifampicin (n=123, 89.1%), linezolid (n=121, 87.7%) and erythromycin (n=106, 76.8%). In contrast, high resistance rates were recorded for penicillin (n=133, 96.4%), trimethoprim-sulfamethoxazole (n=107, 77.5%), and oxacillin (n=92, 66.7%). Resistance rates below 50% were observed for tetracycline (n=65, 47.1%), clindamycin (60, 43.5%), daptomycin (58, 42.0%), vancomycin (57, 41.3%), ciprofloxacin (n=51, 37.0%), levofloxacin (n=50, 36.2%), and gentamicin (n=37, 26.8%). MRSA isolates exhibited significantly higher resistance rates than MSSA for some antibiotics, including ciprofloxacin, gentamicin, levofloxacin and tetracycline (Table 1).

159 **Table 1: Antimicrobial resistance rates of MSSA and MRSA isolates in Nigeria**

Antimicrobial agent	Resistance rates % (n)		
	MSSA (n=53)	MRSA (n=85)	*p*-value
Ciprofloxacin	15.1 (8)	50.6 (43)	**<0.001**
Clindamycin	39.6 (21)	45.9 (39)	0.49
Daptomycin	37.7 (20)	44.7 (38)	0.48
Erythromycin	11.3 (6)	11.8 (10)	1.00
Nitrofurantoin	0.0 (0)	1.2 (1)	1.00
Gentamicin	9.4 (5)	37.6 (32)	**<0.001**
Levofloxacin	15.1 (8)	49.4 (42)	**<0.001**
Linezolid	7.5 (4)	15.3 (13)	0.29
Oxacillin	20.8 (11)	95.3 (81)	**<0.001**
Penicillin V	92.5 (49)	98.8 (84)	0.07
Rifampicin	13.2 (7)	9.4 (8)	0.58
Tetracycline	30.2 (16)	57.6 (49)	**0.003**
Teicoplanin	1.9 (1)	3.5 (3)	1.00
Tigecycline	1.9 (1)	1.2 (1)	1.00
Trimethoprim-sulfamethoxazole	69.8 (37)	82.4 (70)	0.10
Vancomycin	37.7 (20)	43.5 (37)	0.60

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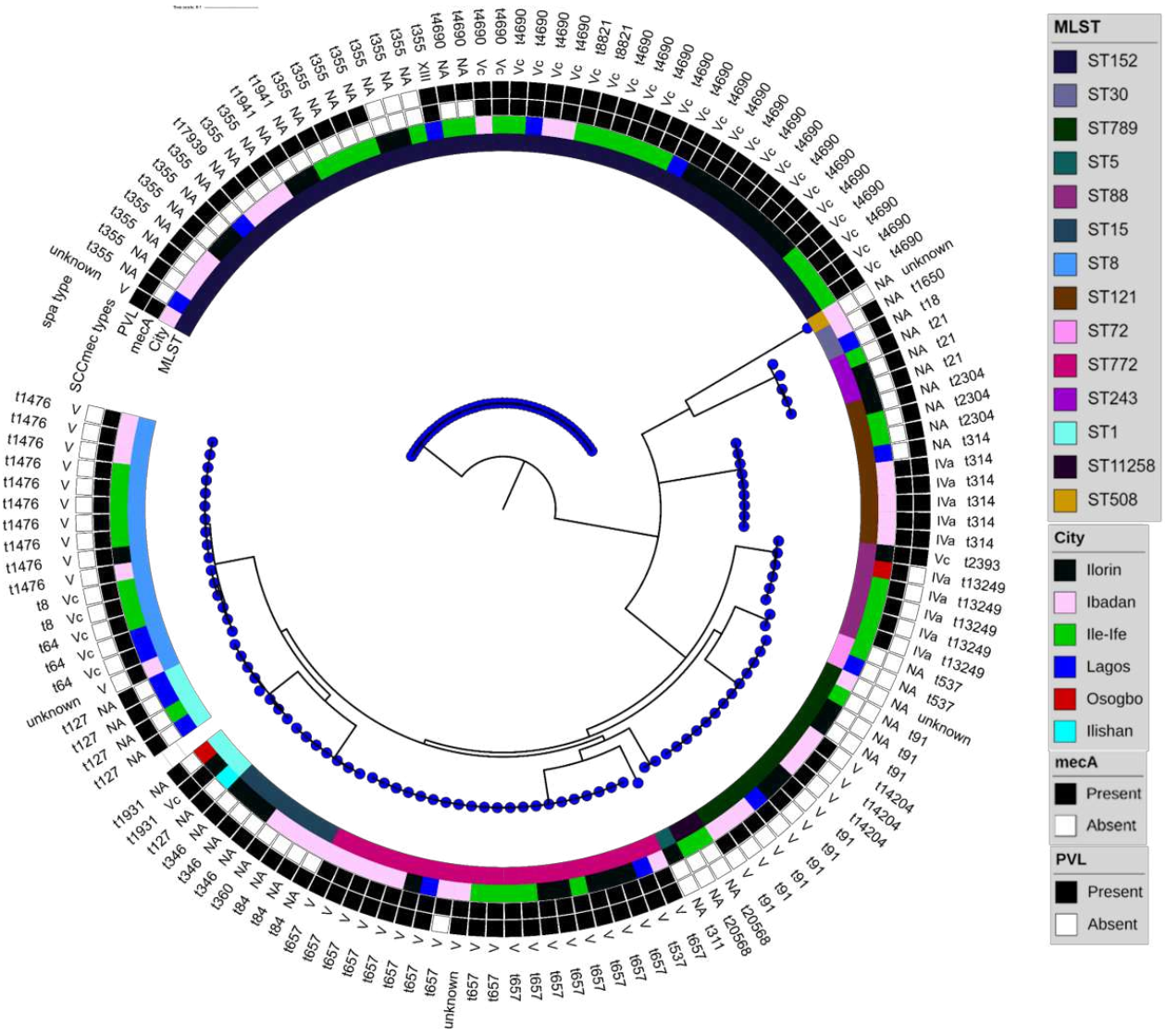


Figure 2. SNP-based maximum likelihood phylogeny of isolates with associated MLST, Location, MRSA, *PVL*, SCC*mec*, and *spa* classification.

Characterization of the isolates based on *spa* typing, MLST, SCC*mec* and plasmid typing

A total of 31 *spa* types were identified, including four new types (t22667-t22670). The most frequent *spa* type was t4690 (n=25), followed by t657 (n=19) and t355 (n=17). WGS identified 14 STs, including a new one (ST11258) from two isolates. The most common genotypes were ST152 (n=48, 35%), ST772 (n=21, 15%), ST8 (n=16, 12%) and ST789 (n=13, 9%) (Table 2). All STs

were classified into eight clonal complexes (CCs): CC1 (ST1, ST772), CC5 (ST5), CC8 (ST8, ST72, ST789), CC15 (ST15), CC30 (ST30, ST243), CC45 (ST508), CC88 (ST88), and CC121 (ST121), and two singletons: ST152 and ST11258 (Figure 2; Table 2). Methicillin resistance varied across the lineages. ST1, ST121, ST152, and ST789 comprised both MRSA and MSSA, whereas ST5, ST15, ST30, ST72, ST243, and ST508 were exclusively MSSA. In contrast, ST8, ST88, and ST772 were entirely MRSA (Table 2). Four SCC*mec* types were detected: IVa, V, Vc, and XIII. SCC*mec*V (n=39), detected in ST8, ST772, and ST789, was the most frequent, followed by SCC*mec* Vc (n=34), identified in ST1, ST8, ST88, and ST152. SCC*mec* IVa was detected in all ST121-MRSA and most of the ST88-MRSA isolates. One ST152 isolate carried the SCC*mec*XIII element and the SCC*mec* element of another isolate could not be assigned using the default thresholds (Figure 2).

Twelve plasmid replicon types were detected, with the rep5a (n=79), rep16 (n=75), and rep7a (n=74) as the most frequent. All ST1 isolates carried rep7c, while ST152 and ST789 mainly harboured rep5a and rep16. Furthermore, Rep7a, rep7c and rep5a, rep16a were common with ST8 and ST15 isolates, respectively. Multiple replicon types were observed in individual isolates, with common combinations comprising rep5–rep7a–rep16, rep5a-rep16-rep19, and rep5a-rep7a-rep20. Complete profile of plasmid replicons detected in the isolates is provided in the additional file 2.

Distribution of AMR genes among isolates across various clonal lineages

The analysis of AMR genes (n=16) revealed lineage-associated patterns, and four STs were identified as major reservoirs of multiple resistance determinants (Figure 3). ST8, ST152, ST772, and ST789 isolates harboured the highest diversity of AMR genes. ST772-MRSA exhibited the most extensive AMR gene profile, including *blaZ, dfrG, rpoB, tetK, gyrA, parC, sat4,* multiple aminoglycoside-modifying enzymes (*ant6-Ia, aph3-IIIa, aac(6′)/aph(2″)*) and macrolide

resistance genes, *mph(C),* and *msr(A)*. Similarly, ST789 isolates displayed a broad but slightly less extensive repertoire, comprising *blaZ, dfrA44, dfrG, gyrA, parC, sat4, ant6-Ia, aph3-IIIa,* and *aac(6')/aph(2")*. ST8 isolates, particularly MRSA, also carried multiple resistance determinants, including *blaZ, dfrG, dfrS1, tetK, gyrA, parC,* and *aac(6')/aph(2")*. In contrast, ST152 isolates showed a comparatively moderate resistance profile, characterized by the presence of *dfrG, tetK, aac(6')/aph(2")*, and *aph3-Ib*. Other lineages, such as ST1 and ST88, carried fewer resistance determinants and lacked the multidrug resistance profiles observed in the dominant lineages. Certain AMR genes showed restricted distribution, e.g., *mph(C)* was identified in ST772-MRSA, *aadD1* in ST15-MSSA, *ant6-Ia*, *aph3-IIIa*, and *sat4* in ST772 and ST789 isolates, respectively.

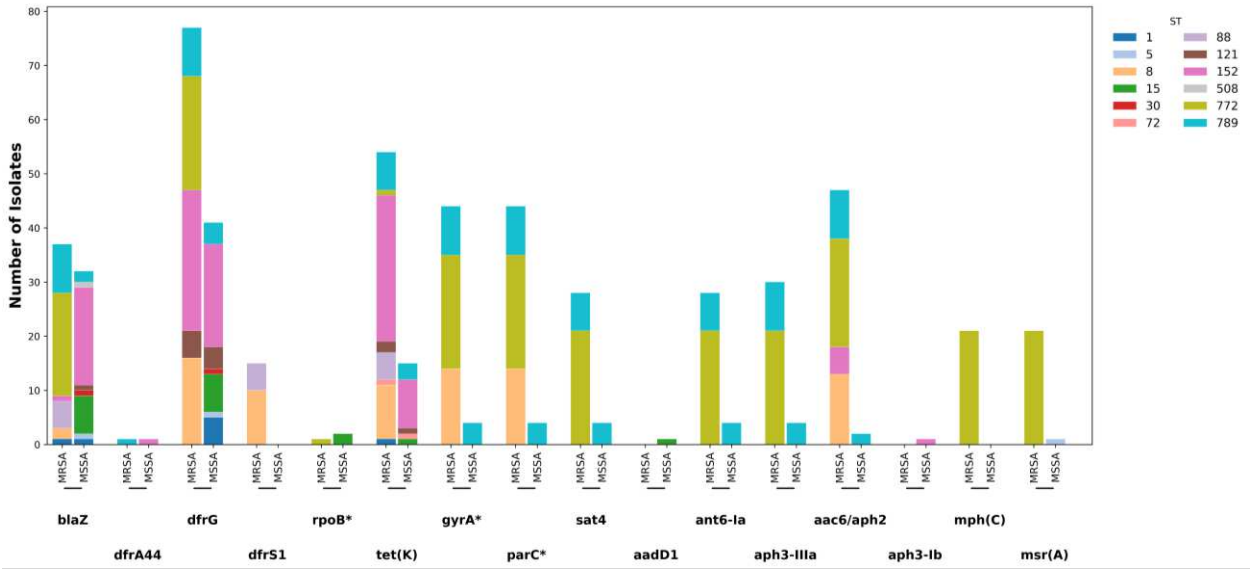


Figure 3: Proportion of antimicrobial resistance genes across MRSA and MSSA by sequence type. Bars show the number of isolates harbouring each antimicrobial resistance gene among MRSA and MSSA, with colours indicating ST distribution.

205 **Table 2: Characterization of *S. aureus* isolates in Nigeria based on *spa* typing, MLST,**

206 **SCC*mec* typing and plasmid typing**

Clonal Complex (n)	**Sequence Types (n)**	***spa* Types (n)**	**MSSA n (%)**	**MRSA n (%)**	**SCC*mec* (n)**	**Plasmid replicon types (n)**
CC1 (28)	ST1 (7)	t127 (5), **t1931** (2)	6 (86)	1 (14)	Vc (1)	**5a & 16** (2); 20 (5); 21, **7a** & US21 (1); **7c** (7)
	ST772 (21)	**t537** (1), **t657** (19), ***t22670*** **(1)**	0 (0)	21 (100)	V (21)	**10** (4); **21** (19); **7a** (5)
CC5 (1)	ST5 (1)	t311 (1)	1 (100)	0 (0)		16, 19 & 20 (1)
CC8 (31)	ST8 (16)	**t008** (2), **t64** (3), **t1476** (10), ***t22667*** (1)	0 (0)	16 (100)	Vc (6), V (9), NT (1)	**10** (1); **15** (6); **5a**, **16** & **19** (4); **20** (3); **7a** (10); **7c** (16)
	ST72 (2)	t537 (2)	2 (100)			20, 7a & 7c (2)
	ST789 (13)	**t091** (9), t14204 (3), *t22668* (1)	4 (31)	0 (0) 9 (69)	V (9)	**10b** (2); **15** (1); **5a** & **16** (13); **21** (3); **7a** (10)
CC15 (7)	ST15 (7)	t084 (3), t360 (4)	7 (100)	0 (0)		16 (6); 7a, 20 & US22 (1); 5a (7)
CC30 (5)	ST30 (2)	t018 (1), t1640 (1)	2 (100)	0 (0)		16 & 21 (2); 5a & 19 (1)
	ST243 (3)	t21 (3)	3 (100)	0 (0)		5a, 16 & 19 (3)
CC45 (1)	ST508 (1)	*t22669* (1)	1 (100)	0 (0)		5a & 16 (1)
CC121 (9)	ST121 (9)	**t314** (6), t2304 (3)	4 (44)	5 (56)	IVa (5)	**21** (2); **7a** (4)
CC88 (6)	ST88 (6)	**t2393** (1), **t13249** (5)	0 (0)	6 (100)	IVa (5), Vc (1)	**5a**, **7a** & **20** (5); **21** (6)
ST152 (48)	ST152 (48)	**t355** (17), t1941 (2), **t4690** (25), **t8821** (2), t17939 (1), t11375 (1)	21 (44)	27 (56)	Vc (26), XIII (1)	**5a** & **16** (43); **7a** (36)
***ST11258* (2)**	*ST11258* (2)	t20568	2 (100)	0 (0)		20 (2)

207 *spa* types and plasmid replicon types of MRSA in bold

208 new *spa* types and ST in italics

209 NT: Non-Typeable

Distribution of virulence genes among isolates across various clonal lineages

The analysis of virulence determinants (n=24) in the isolates revealed a combination of highly conserved virulence factors and lineage-associated accessory genes. Genes encoding haemolysins (*hla, hlb, hld, hlgAB*), polysaccharide intercellular adhesin involved in biofilm formation (*icaADBCR*), and immune evasion (*sak* and *scn*) were widely distributed across the various STs in both MRSA and MSSA populations. Some superantigen genes exhibited clonal specificity. The *seh* was only detected in ST1, *tsst-1* in ST8 (MRSA) and ST508 (MSSA), and *seb* in ST8 (MRSA) and ST121 (MRSA and MSSA). The Panton–Valentine leukocidin (PVL) genes (*lukF-PV/lukS-PV*) were common but unevenly distributed, with high prevalence observed in ST152 and ST772, and low representation in ST8. Some microbial surface components recognizing adhesive matrix molecules (MSCRAMM) genes showed notable lineage restriction. The major histocompatibility complex (*map*) gene was associated with ST8-MRSA, fibronectin B (*fnbB*) with ST1, clumping factor (*clfA*) gene in ST1 (MSSA) and ST8 (MRSA), and some MSCRAMM genes (including *clfA, clfB, fnbA,* and *fnbB*) were largely absent in ST152 isolates, despite the high prevalence of the PVL genes (Figure 4). Overall, ST772 was characterized by high levels of AMR and virulence genes. ST8 and ST152 exhibited substantial virulence gene profiles, with comparatively moderate antibiotic resistance. As for ST789, a high prevalence of AMR and virulence genes was observed in both MRSA and MSSA populations.

SNP phylogeny and population structure of *S. aureus* based on sentinel sites

Whole-genome SNP-based phylogenetic analysis revealed that the population was dominated by four major lineages: ST8, ST152, ST772 and ST789, which together accounted for over two-thirds of all isolates (Figure 2). The phylogeographic analysis revealed that these dominant lineages were distributed across all sentinel sites, suggesting the presence of widely circulating regionally

established clones (Figure 5). Despite this broad distribution, certain lineages showed site-specific associations. ST8 and ST152 were predominantly recovered from OAUTHC, the sentinel contributing the most isolates, whereas ST121 was more frequently identified at UCH.

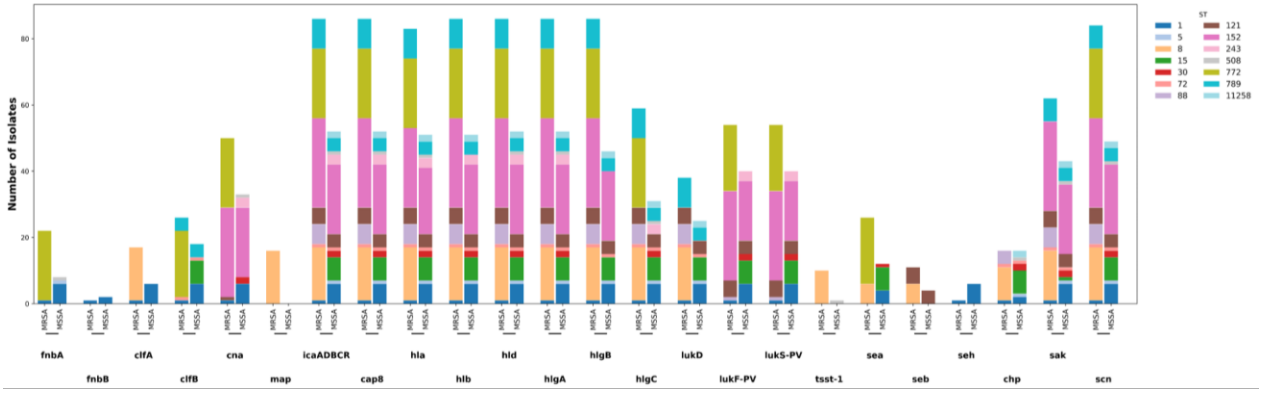


Figure 4: Proportion of virulence genes across MRSA and MSSA by sequence type. Bars show the number of isolates harbouring each virulence gene among MRSA and MSSA, with colours indicating ST distribution.

Analysis of genomes of isolates from the common STs with publicly available sequences on Pathogenwatch

Phylogenetic analysis of isolates from the predominant STs in the context of publicly available genomes on Pathogenwatch revealed that ST8 isolates from our study were divided into two distinct sub-clades, separated by others around the globe. ST8 isolates from Tanzania, The Gambia and The United Kingdom clustered closely with isolates from Nigeria. Notably, all ST8 isolates from Nigeria were *mecA* and *dfrG*-positive (Figure 6). The ST152 isolates from this study clustered into two major clades, according to methicillin susceptibility. Three ST152 isolates from other countries co-clustered with these ST152-MRSA and ST152-MSSA clades, while some ST152 isolates (n=5) from this collection were identified in other subclades (Figure 7). The ST772 isolates

from Nigeria, with similar AMR profiles, clustered in a distinct clade (Figure 8). The ST789 isolates from Nigeria were divided into two major sub-clades with a common ancestor. The major difference between these two sub-clades was methicillin resistance; the sub-clade with a higher number of isolates (n=12) was *mecA-positive*, while the other had only five isolates, all of which were *mecA*-negative. Two isolates from Ireland and one each from Germany and the United Kingdom clustered with the isolates from Nigeria (Figure 9).

The distribution and persistence of some *S. aureus* STs from previous and current studies at two sentinels, OAUTHC (Figure 10A) and UCH (Figure 10B), were investigated. In the OAUTHC dataset (Figure 10A), ST1, ST8, ST72, ST88, ST121, and ST152 were identified in both studies. ST1 and ST121 were predominant in the earlier study [20], whereas ST8, ST152, and ST88 were more common in the current dataset. The earlier OAUTHC study, which focused on colonizers rather than organisms from invasive infections, also showed a broader ST profile, with 61 strains assigned into 17 STs, compared to the 10 STs (of 46 isolates) in this current study. Interestingly, ST772 and ST789, dominant lineages in the current dataset, were not detected in the previous study. At UCH, ST8, ST15, ST30, ST121 and ST508, ST8, and ST15 were observed in both the previous and the current studies. Among these, ST30 and ST121 were found in higher proportions in the previous study, which was focused on community-acquired infections [44], while ST8 and ST15 had increased representation in the current study. Notably, ST152, the most prevalent ST in the current study, was absent in the older dataset. Across all four datasets, only ST8 and ST121 were consistently identified, highlighting their persistence over time and geographic location.

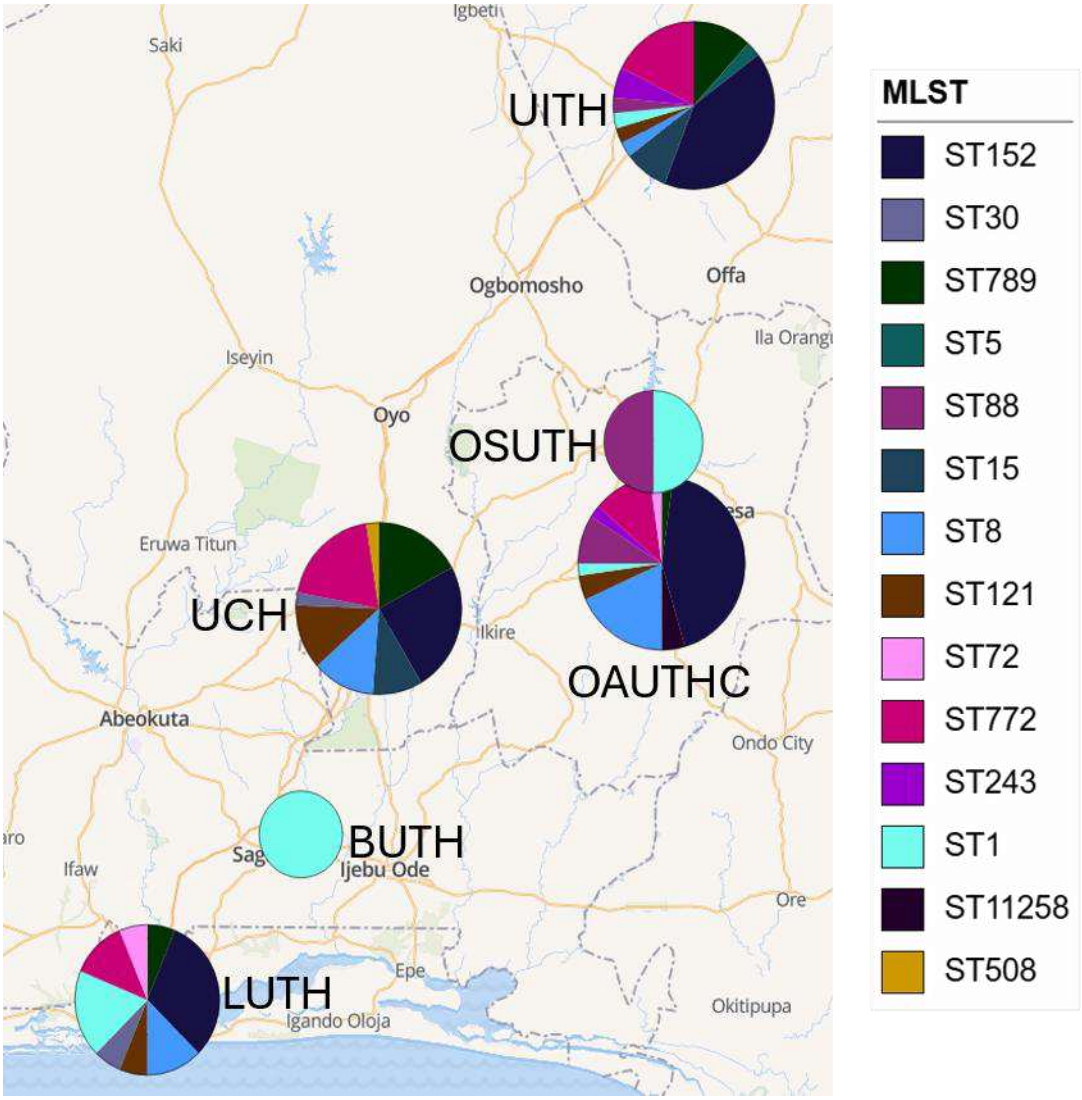


Figure 5: A map of sentinel sites. The size of the circles is to the logarithmic scale of total number isolates per sentinel site while the colours represent the sequence types.

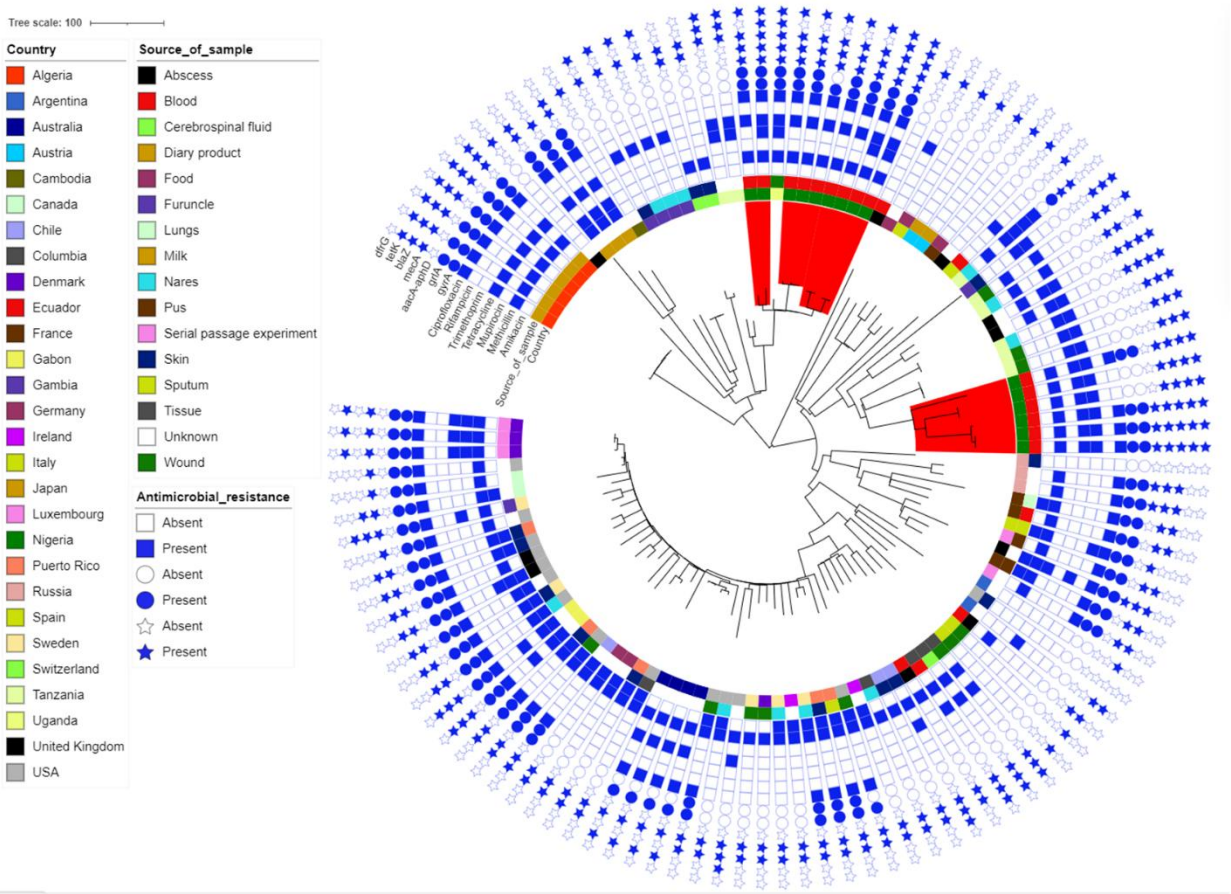
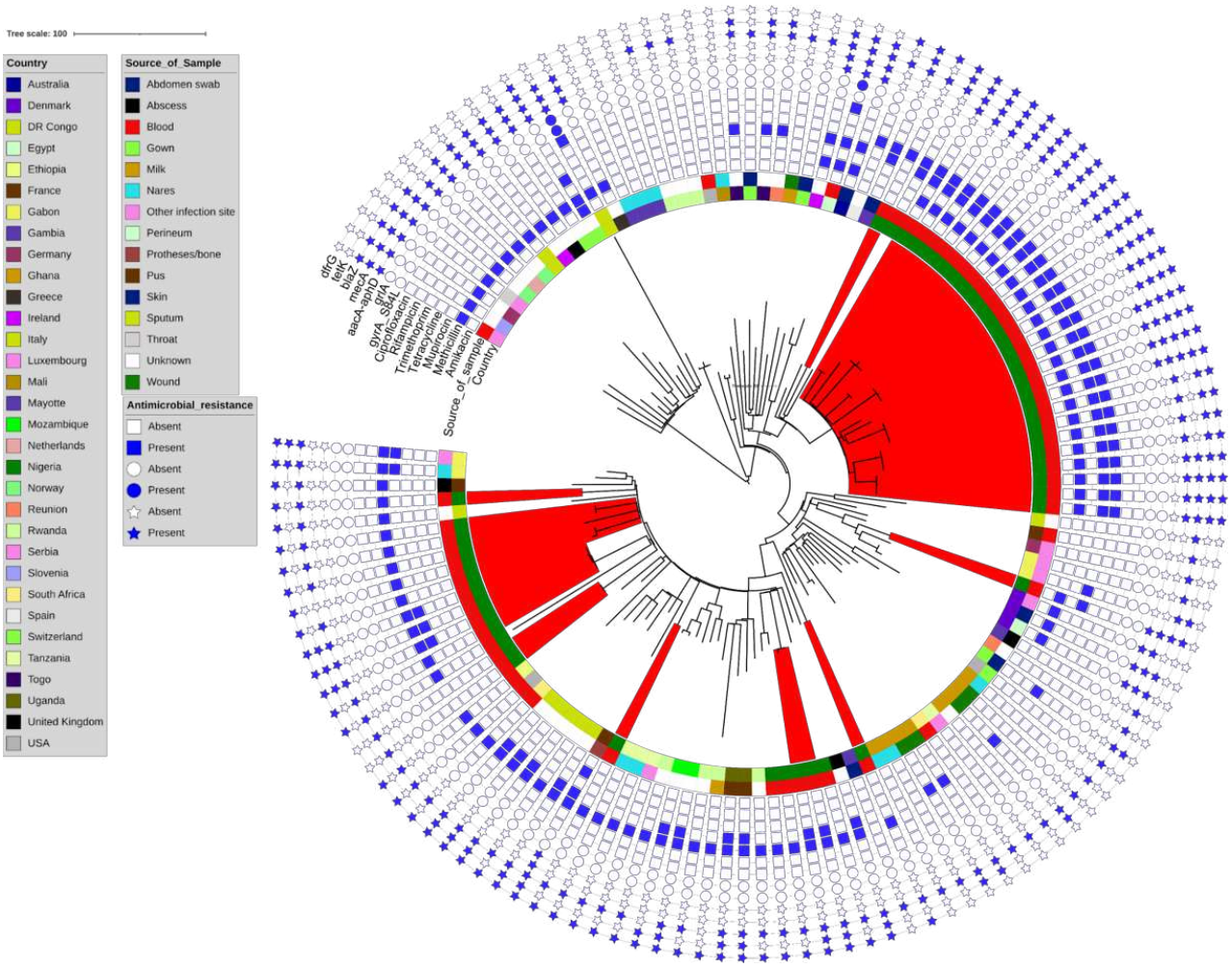
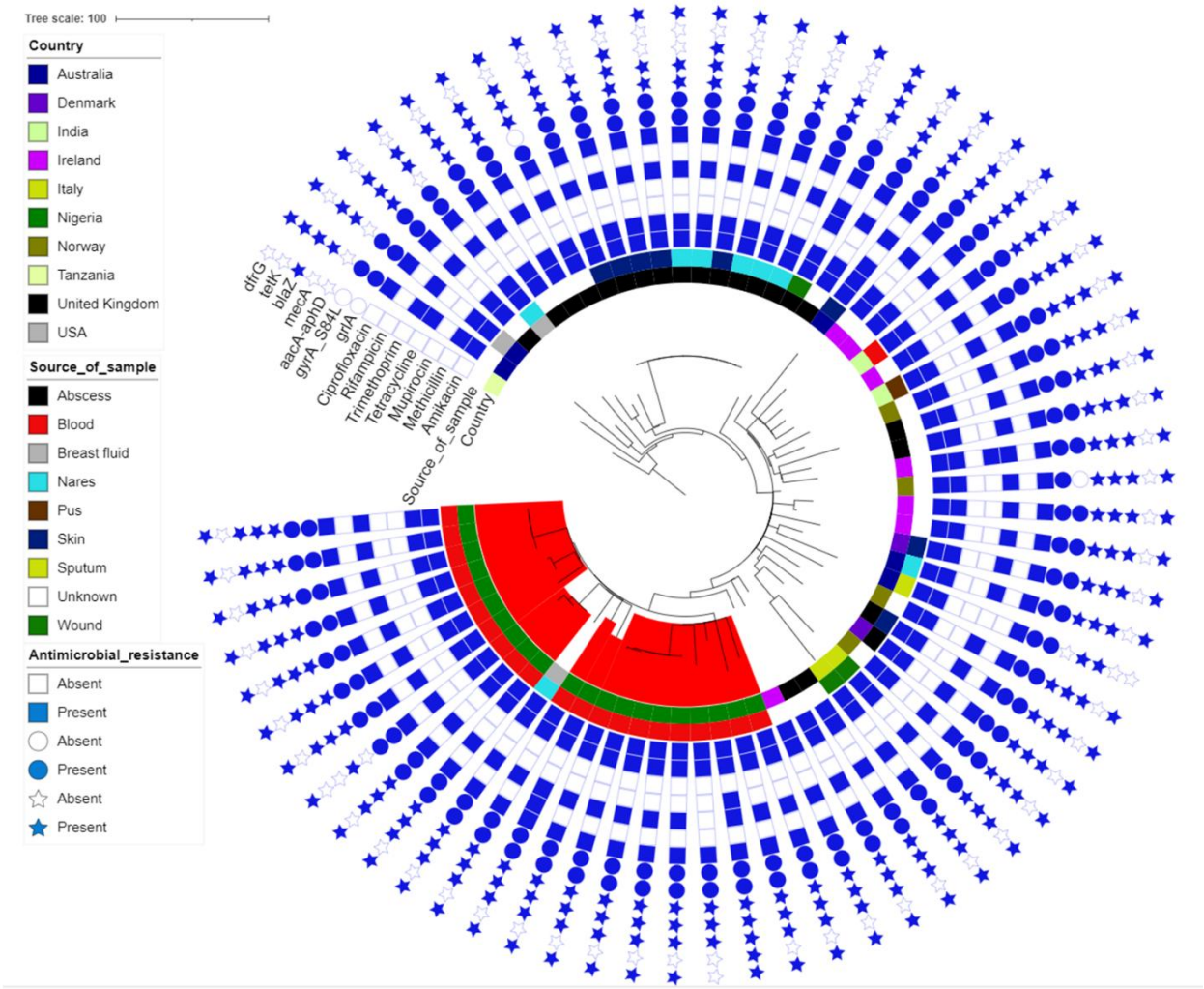


Figure 6: Phylogeny of ST8 strains from the current study (clades coloured in red) in context with selected global strains of ST8 publicly available in Pathogenwatch. The matrix shows country of isolation, sample source and antimicrobial resistance prediction- resistance to named antibiotics in squares, point mutations conferring antimicrobial resistance in circles and antimicrobial resistance genes in stars.



279

280 Figure 7: Phylogenetic tree of ST152 strains from this study (clades coloured in red) in context of
281 selected global strains of ST772 publicly available in Pathogenwatch. The matrix shows the
282 country of isolation, sample source and antimicrobial resistance prediction- resistance to named
283 antibiotics in squares, point mutations conferring antimicrobial resistance in circles and
284 antimicrobial resistance genes in stars.



285

286 Figure 8: Phylogeny of ST772 strains from the current study (clades coloured in red) in context
287 with selected global strains of ST772 publicly available in Pathogenwatch. The matrix shows
288 country of isolation, sample source and antimicrobial resistance prediction- resistance to named
289 antibiotics in squares, point mutations conferring antimicrobial resistance in circles and
290 antimicrobial resistance genes in stars.

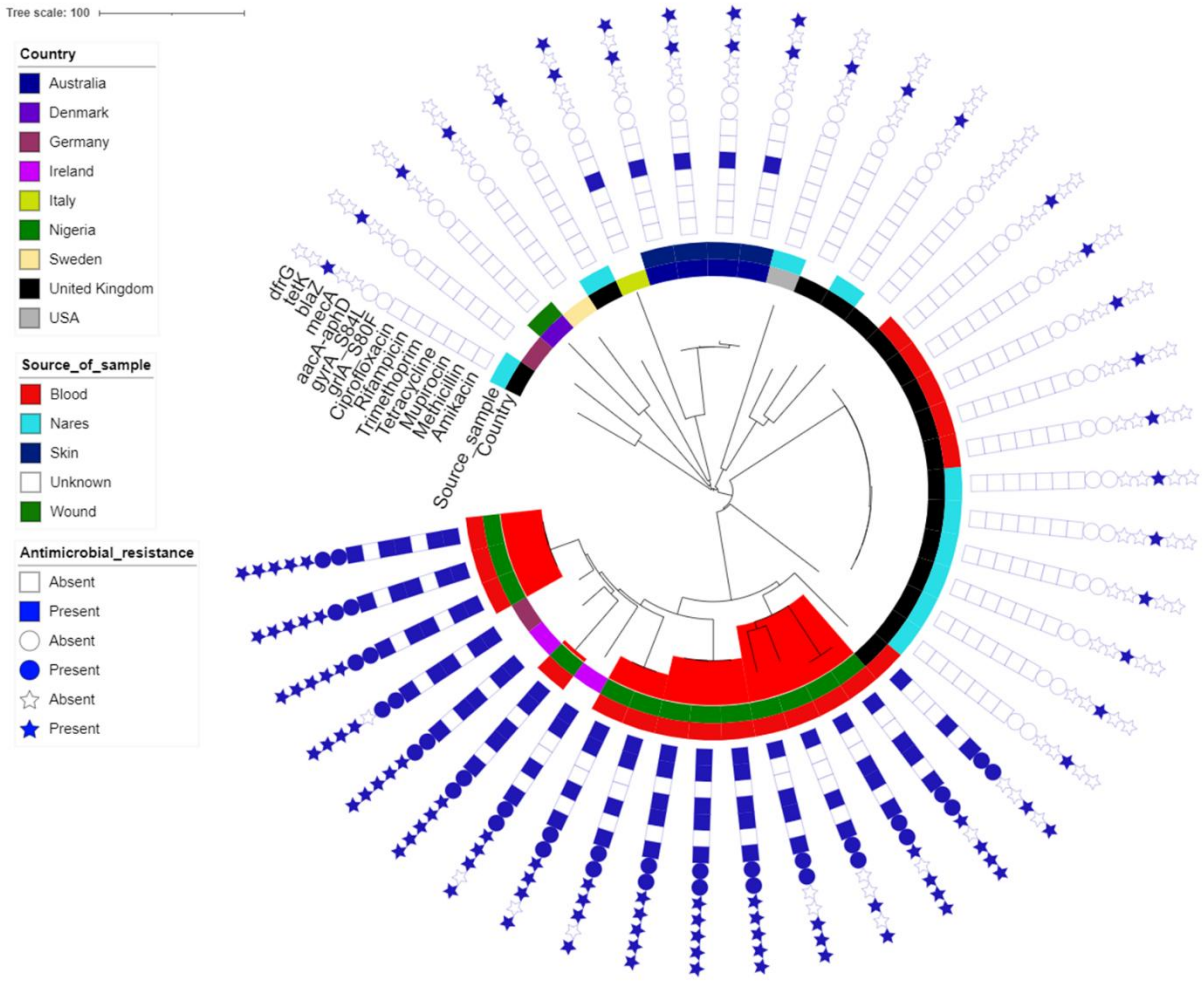
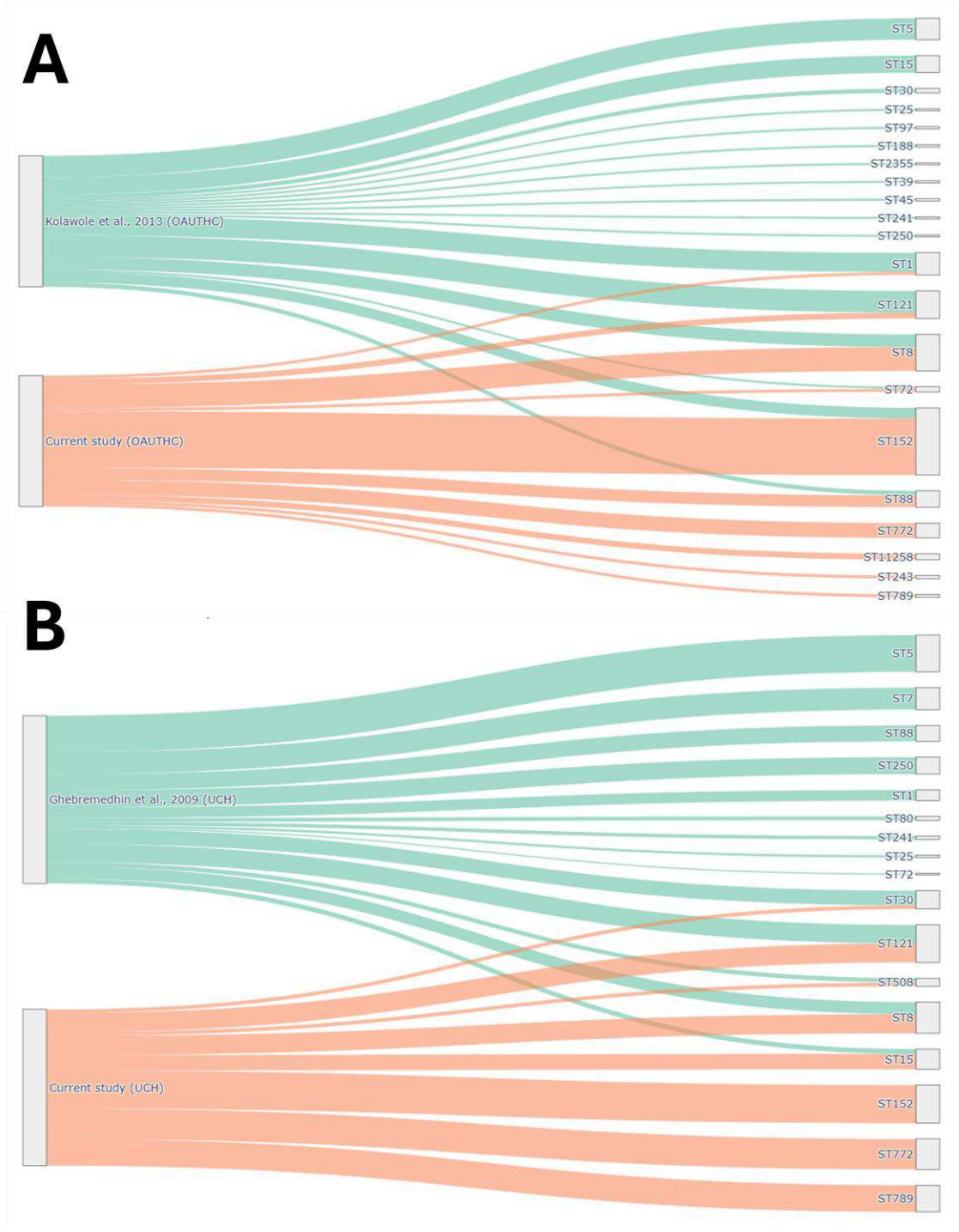


Figure 9: Phylogeny of ST789 strains from the current study (clades coloured in red) in context with selected global strains of ST789 publicly available in Pathogenwatch. The matrix shows country of isolation, sample source and antimicrobial resistance prediction- resistance to named antibiotics in squares, point mutations conferring antimicrobial resistance in circles and antimicrobial resistance genes in stars.



297

298 Figure 10: Sankey plots showing the flow of ST proportions from previous studies from (A)

299 OAUTHC, (Kolawole et al., 2013) and (B) UCH (Ghebremedhin et al., 2009) in relation to

proportions found in the current study. Flows widths represent the relative percentages of each ST in the respective studies. Shared STs are shown as connected flows.

Discussion

This study provides genomic insights into bloodstream *S. aureus* isolates from six tertiary institutions through the Nigerian AMR Surveillance Network. Molecular characterization revealed a high prevalence of MRSA, diverse clonal lineages, and dissemination of high-risk clones in the sentinel sites. All isolates were susceptible to tigecycline, and >90% were susceptible to nitrofurantoin and teicoplanin, similar to previous reports from Nigeria and Africa [45–47]. However, >40% of the isolates were resistant to daptomycin and vancomycin, but the corresponding resistance gene determinants were not detected. *S. aureus* resistance to daptomycin and vancomycin is not commonly reported in Nigeria [45, 47–49]. This discrepancy between AMR gene detection and phenotypic expression suggests the need for further validation and highlights potential limitations of phenotypic testing [50–52]. Another possible explanation for this discrepancy could be heteroresistance, caused by mutations in chromosomal genes rather than acquired resistance genes [53–56]. These mutations would not be detected by standard molecular screenings despite driving phenotypic resistance. Notably, four (4.7%) of the *mecA*-positive isolates were phenotypically susceptible to oxacillin, while 11 (20.8%) of the *mecA*-negative strains were resistant to oxacillin. This genotype-phenotype discordance is well documented in *S. aureus* [57, 58]. Oxacillin-susceptible MRSA (OS-MRSA) occur either by heteroresistance or phenotypic suppression due to unexpressed *mecA* in *in-vitro* conditions [59, 60]. Also, mutations in *mecI* and *blaI* repressors as well as in *mecA* have been associated with this phenomenon [59, 61]. Phenotypic resistance to oxacillin in the absence of *mecA* and *mecC* may be explained by

alternative resistance mechanisms including hyper production of certain variants of *blaZ* or structural point mutations in penicillin binding proteins that reduce their affinity for beta-lactam antibiotics [61–63].

The proportion of MRSA observed in this study (61.6%) is notably high compared with earlier Nigerian and African studies, where prevalence has typically ranged between 20% and 59% [20, 23, 64]. MRSA prevalence in Nigerian settings has increased over the years from 20.2% in 2007 [23, 44] to 61.6% in our study. The higher rate observed in this study could be attributed to the focus on bloodstream infections, which are often associated with more severe disease and prompt empiric administration of antibiotics [65]. The high resistance rates to penicillin and trimethoprim–sulfamethoxazole, are consistent with previous reports in Nigeria and the West African sub-region [23, 44, 47, 66–68]. Moreover, >40% of *S. aureus* isolates were resistant to tetracycline and <30% to gentamicin, similar to previous reports in the region [23, 44, 68, 69]. However, the resistance rate (>40%) to clindamycin in this study is higher than previously reported in the region (8.8-36%) [47, 70, 71]. Fluoroquinolone resistance was observed in approximately one-third of isolates, consistent with earlier reports from Nigeria [23, 47, 72]. The significantly higher resistance rates observed among MRSA compared to MSSA further highlight the limited therapeutic options available for invasive MRSA infections and reinforce the need for improved diagnostic stewardship and treatment guidelines.

The *S. aureus* population in this study was genetically diverse, and all STs, apart from ST243 and ST11258, have been previously reported in West Africa [18, 23, 47, 73–76], supporting the hypothesis of regional circulation of established lineages. Four lineages, ST8, ST152, ST772, and ST789, accounted for the majority of isolates. Of these, ST152 was the dominant lineage, occurring largely as ST152-t4690-MRSA-Vc and ST152-t355-MSSA. ST152 isolates were mainly

PVL-positive but lacked key adhesion genes (*clfA, clfB, fnbA, fnbB*) and *hlgC*, suggesting that this lineage may rely more on cytotoxin-mediated pathogenicity than adhesion [47, 64]. Historically rare or absent in earlier Nigerian studies [20, 44, 76], ST152 has transitioned to a dominant lineage, reflecting a clear temporal shift in population structure and reinforcing its growing importance in Nigeria and across Africa [23, 45, 74, 77–79].

Although ST152 is typically reported as a PVL-positive, community-associated MSSA lineage, 50% of ST152 isolates in this study were MRSA with the SCC*mec*V element, unlike the traditional SCC*mec* types (I, II and III) associated with hospital settings [80, 81]. The recent reports of PVL-positive ST152-MRSA in Nigeria, Ghana, and Kenya suggest ongoing adaptation in hospital, community, and animal settings [24, 45, 69, 70, 75]. Furthermore, the identification of an ST152-SCC*mec* XIII isolate is noteworthy, as this element is rarely reported, suggesting introduction or local evolution of uncommon SCC*mec* types in West African lineages [82]. The clustering of ST152-MRSA isolates in a different clade from ST152-MSSA in our collection supports the hypothesis that ST152-MRSA arose through clonal expansion rather than the acquisition of the SCC*mec* element from an MSSA genetic background (Figure 7).

CC1-ST772, often referred to as the "Bengal Bay clone", was first reported in the Indian subcontinent, from where it began to spread intercontinentally in the late 19th century [83]. It is a multidrug-resistant lineage that is implicated in hospital clusters and outbreaks [84]. In this study, ST772-SCC*mec*V was the second most common MRSA lineage. It was identified in four sentinel sites and exhibited the highest number of AMR and virulence genes, consistent with its classification as a high-risk clone. Limited reporting of ST772 had been observed earlier in Africa [18, 23, 72]. However, there are recent reports of this clone in Nigeria [25]. The unique clade of the ST772 genomes from this study, compared with those from other countries (Figure 8), is

noteworthy. It suggests a shift in the local epidemiology of *S. aureus*, potentially driven by antimicrobial selection pressure.

Isolates belonging to the pandemic lineages such as ST8-CC8 clustered with isolates from multiple countries, suggesting intra- and inter-country transmission, consistent with their established global dissemination [21, 23, 45, 69, 76, 85–87]. Notably, ST8 isolates formed two different clades, indicating underlying genetic diversity. One of the sub-clades, comprising ST8-CC8-MRSA-t1476, was phylogenetically distinct, aligning more closely with ST247 and UK-associated strains, and may represent a relatively uncommon or emerging variant. The second subclade was more similar to previously described ST8 lineages in Nigeria and globally [21, 23, 69]. This pattern suggests both local expansion and multiple introductions within this lineage. CC8-ST789 possessed a substantial repertoire of AMR genes in both MRSA and MSSA backgrounds, indicating its capacity to act as a reservoir for AMR genes.

ST789 was the fourth most commonly ST in this study despite being sparsely distributed worldwide. This could be due to local clonal expansion or capacity to cause invasive infections. This contrasts with previous reports from non-invasive sources in Africa where it is typically detected in small proportions [18, 67, 69, 74, 88, 89].

Plasmid replicon typing revealed multiple replicon types, with *rep*, *rep16*, *rep20*, *rep5_1*, *rep7*, and *rep7_6* being the most frequent. This distribution suggests widespread plasmid-mediated gene exchange within the population rather than lineage-specific restriction. The presence of similar replicon types across multiple STs supports the role of horizontal gene transfer in shaping the observed antimicrobial resistance profiles. However, no clear one-to-one association between specific replicons and resistance phenotypes was evident. We also did not assemble closed plasmids as only short read sequencing was employed.

The widespread distribution of dominant lineages across sentinel sites indicates the presence of regionally established clones rather than localized outbreaks. However, the identification of site-specific STs (e.g. ST8 and ST152 in OAUTHC, ST121 in UCH) suggests local transmission dynamics. The comparison with historical datasets from selected sentinel sites revealed notable shifts in lineage distribution over time. In 2009, Ghebremedhin et al reported a clone of ST88 bearing SCC*mec*IVa from Ibadan, along with ST250 and ST241 strains as predominant among patients with community-acquired infections attending UCH and another Ibadan hospital. This clone was detected predominantly in UCH in this study, suggesting that it continues to circulate in Ibadan two decades later. The emergence of ST152, ST772, and ST789 as dominant clones in the current study, with the decline or absence of previously reported lineages, suggests a dynamic and evolving *S. aureus* population. These changes may be driven by antimicrobial selection pressure, clonal replacement, or the introduction of new strains through regional or international transmission.

This study has some limitations. The isolates were obtained from a limited number of sentinel sites, all in or close to South-West Nigeria. At each site, few patients are blood cultured and therefore only a fraction of bloodstream *S. aureus* is likely recovered. Thus, the diversity seen here is unlikely to fully represent the national epidemiology of *S. aureus*. Moreover, the retrospective design and lack of key clinical metadata limit the ability to correlate genomic findings with patient outcomes, something that future studies employing the ACORN platform should be able to do [90]. Nevertheless, WGS provides insights into the molecular epidemiology of invasive *S. aureus* in Nigeria.

Conclusions

This study describes a high prevalence of MRSA, extensive genetic diversity, and widespread dissemination of high-risk *S. aureus* clones in bloodstream infections in Nigeria. The emergence of ST152-MRSA, ST772-MRSA and ST789-MRSA is of concern. Integrating genomic data into national surveillance programmes will be critical for informing infection prevention strategies, guiding antimicrobial stewardship, and mitigating the spread of high-risk clones within healthcare settings.

List of abbreviations

AMR – Antimicrobial Resistance

AST – Antimicrobial Susceptibility Testing

BUTH – Babcock University Teaching Hospital

DNA – Deoxyribonucleic Acid

ENA – European Nucleotide Archive

LUTH – Lagos University Teaching Hospital

MSCRAMM - Microbial Surface Components Recognizing Adhesive Matrix Molecules

MSSA – Methicillin-Susceptible *Staphylococcus aureus*

MRSA – Methicillin-Resistant *Staphylococcus aureus*

NC – North Central

NRL – National Reference Laboratory

OAUTHC – Obafemi Awolowo University Teaching Hospitals Complex

OS-MRSA – Oxacillin-Susceptible Methicillin-Resistant *Staphylococcus aureus*

OSUTH – Osun State University Teaching Hospital

PVL - Panton-Valentine Leukocidin

SNP – Single Nucleotide Polymorphism

ST – Sequence Type

SW – South-West

UITH – University of Ilorin Teaching Hospital

UCH – University College Hospital

WHO – World Health Organisation

Declarations

Ethics approval and consent to participate: The bacterial isolates used in this study were obtained from positive blood cultures as part of routine diagnostic services in the sentinel laboratories of the Nigerian antimicrobial resistance surveillance network coordinated by the Nigeria Centre for Disease Control and Prevention (NCDC). Ethical approval to use the isolates and genomes for research purposes, waiving the need to consent individual patients, was granted by the University of Ibadan and University College Hospital Research Ethics Committee (UI/UCH REC), with reference number UI/EC/22/0113.

Only bacterial isolates with matching deidentified metadata were handled in this study. Human samples were not handled as part of the study. The research was undertaken in strict conformation to the Declaration of Helsinki.

As there was no contact with human participants, and the study used secondary biological material (bacterial isolates) with associated de-identified metadata, the requirement for individual informed concert was waived by the UI/UCH REC.

Clinical trial number: Not applicable

Consent for publication: Not applicable

Availability of data and materials: The dataset generated and analysed during the current study are available in the European Nucleotide Archive (ENA), under the project accession PRJEB29739. Individual accession numbers are in additional file 1.

Competing interests: The authors declare that they have no competing interests

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Authors' contributions

CI, DMA and INO conceptualised the study; AOO, OOI, AOAf, EEO, AOAb, VOO, OAB, RAR, OOO, IK, AK, AAA, OVA, TOO, OFO, CE, AF, JFO, ATA, POO, VAA and INO, curated the data; AOO, IEM, GTS, DC, OOI and AOS Conducted formal analyses; CI, DMA and INO secured funding for the research; AOO, GTS, DC, VOO, OAB, RAR, OOO, IK, AK, AAA, OVA, TOO, OFO, CE, AF, JFO, ATA, POO and VAA conducted investigation; AOO, AOAf, EEO, AU, and MK contributed to the methodology; AOO, AU, SA, MK, AE, TO, DMA and INO contributed to project administration; AOAb, VOO, OAB, RAR, OOO, AK, AAA, OVA, TOO, OFO, CE, AF, JFO, ATA, POO, VAA, AE, TO, CI, DMA and INO provided resources; AOO, AOAf, EEO, AU and CI provided software; AOAb, VOO, OAB, OOO, AK, CE, AU, TO, CI, AOS, DMA and INO supervised the study; AOO, IEM, AOS and INO validated the results; AOO, IEM, DC and AOS visualised the data/results; AOO, AOS and INO wrote the first draft, AOO, IEM, GTS, DC, AOAb, OAB, SA, TO, AOS and INO edited the manuscript; All authors reviewed the final draft

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Additional Files

Additional file 1: Accession numbers of sequence data included in this study.

Additional file 2: Molecular typing, antibiogram, antimicrobial resistance, virulence and plasmid predictions for all samples included in the study.

References

Aarris, M., Hertz, F. B., Nielsen, K. L., Sato, A., Johansen, H. K., Westh, H., Kemp, M., Ellermann-Eriksen, S., Løbner-Olesen, A., Frimodt-Møller, N., & Charbon, G. (2025). Genetic Variation in the blaZ Gene Leading to the BORSA Phenotype in Staphylococcus aureus. *Antibiotics*, *14*(5). https://doi.org/10.3390/antibiotics14050449

Abdulgader, S. M., Shittu, A. O., Nicol, M. P., & Kaba, M. (2015). Molecular epidemiology of Methicillin-resistant Staphylococcus aureus in Africa: A systematic review. *Frontiers in Microbiology*, *6*(APR). https://doi.org/10.3389/fmicb.2015.00348

Ackers-Johnson, G., Kibombo, D., Kusiima, B., Nsubuga, M. L., Kigozi, E., Kajumbula, H. M., Kateete, D. P., Walwema, R., Ackers, H. L., Goodhead, I. B., Birtles, R. J., & James, C. E. (2021). Antibiotic resistance profiles and population structure of disease-associated staphylococcus aureus infecting patients in fort portal regional referral hospital, western uganda. *Microbiology (United Kingdom)*, *167*(5). https://doi.org/10.1099/MIC.0.001000

Adesida, S., Boelens, H., Babajide, B., Kehinde, A., Snijders, S., Van Leeuwen, W., Coker, A., Verbrugh, H., & Van Belkum, A. (2005). Major epidemic clones of Staphylococcus aureus in Nigeria. *Microbial Drug Resistance*, *11*(2), 115–121. https://doi.org/10.1089/mdr.2005.11.115

Afolayan, A. O., Oaikhena, A. O., Aboderin, A. O., Olabisi, O. F., Amupitan, A. A., Abiri, O. V, Ogunleye, V. O., Odih, E. E., Adeyemo, A. T., Adeyemo, A. T., Obadare, T. O., Abrudan, M., Argimón, S., David, S., Kekre, M., Underwood, A., Egwuenu, A., Ihekweazu, C., Aanensen, D. M., … Vegvari, C. (2021). Clones and Clusters of Antimicrobial-Resistant Klebsiella From Southwestern Nigeria. *Clinical Infectious Diseases*, *73*(Supplement_4), S308–S315. https://doi.org/10.1093/cid/ciab769

Algorri, M., Jorth, P., Wong-beringer, A., & Wong-beringer, A. (2021). Variable Release of Lipoteichoic Acid From Staphylococcus aureus Bloodstream Isolates Relates to Distinct Clinical Phenotypes , Strain Background , and Antibiotic Exposure Selection of Bacterial Isolates. *Frontiers in Microbiology*, *11*(January), 1–9. https://doi.org/10.3389/fmicb.2020.609280

Almanaa, T. N., Alyahya, S. A., Khaled, J. M., Shehu, M. R., Alharbi, N. S., Kadaikunnan, S., Alobaidi, A. S., & Khalid Alzahrani, A. (2020). The extreme drug resistance (XDR) Staphylococcus aureus

strains among patients: A retrospective study. *Saudi Journal of Biological Sciences*, *27*(8), 1985–1992. https://doi.org/10.1016/j.sjbs.2020.04.003

Argimón, S., Abudahab, K., Goater, R. J. E., Fedosejev, A., Bhai, J., Glasner, C., Feil, E. J., Holden, M. T. G., Yeats, C. A., Grundmann, H., Spratt, B. G., & Aanensen, D. M. (2016). Microreact: visualizing and sharing data for genomic epidemiology and phylogeography. *Microbial Genomics*, *2*(11), e000093. https://doi.org/10.1099/mgen.0.000093

Argimón, S., Yeats, C. A., Goater, R. J., Abudahab, K., Taylor, B., Underwood, A., Sánchez-busó, L., Wong, V. K., Dyson, Z. A., Nair, S., Park, S. E., Marks, F., Page, A. J., Keane, J. A., Baker, S., Holt, K. E., Dougan, G., & Aanensen, D. M. (2021). A global resource for genomic predictions of antimicrobial resistance and surveillance of Salmonella Typhi at pathogenwatch. *Nature Communications*. https://doi.org/10.1038/s41467-021-23091-2

Baig, S., Johannesen, T. B., Overballe-petersen, S., Larsen, J., Larsen, A. R., & Stegger, M. (2018). Novel SCC mec type XIII (9A) identi fi ed in an ST152 methicillin-resistant Staphylococcus aureus. *Infection, Genetics and Evolution*, *61*(January), 74–76. https://doi.org/10.1016/j.meegid.2018.03.013

Baig, S., Larsen, A. R., Simoes, P. M., Laurent, F., Johannesen, T. B., Lilje, B., Tristan, A., Schaumburg, F., Egyir, B., Cirkovic, I., Nimmo, G. R., Spiliopoulou, I., Blanc, D. S., Mernelius, S., Moen, A. E. F., David, M. Z., Andersen, S., & Stegger, M. (2020). Evolution and Population Dynamics of Clonal Complex 152 Community-Associated Methicilin-Resistant Staphylococcus aureus. *Clinical Science and Epidemiology*, *5*(4), 1–9.

Barros, E. M., Martin, M. J., Selleck, E. M., Lebreton, F., Sampaio, J. L. M., & Gilmore, M. S. (2019). Daptomycin Resistance and Tolerance Due to Loss of Function in Staphylococcus aureus dsp1 and asp23. *Antimicrobial Agents and Chemotherapy*, *63*(1), e01542-18.

Bobenchik, A. M., Hindler, J. A., Giltner, C. L., Saeki, S., & Humphries, R. M. (2014). Performance of Vitek 2 for Antimicrobial Susceptibility Testing of Staphylococcus spp. and Enterococcus spp. *Journal of Clinical Microbiology*, *52*(2), 392–397. https://doi.org/10.1128/JCM.02432-13

Buffart, B., Clevenbergh, P., Stiuliuc, A., Raftakis, I., Hing, M., Miendje Deyi, V. Y., Denis, O., Martiny, D., & Yin, N. (2025a). Borderline Oxacillin-Resistant Staphylococcus aureus (BORSA) Bacteremia—Case Report. *Antibiotics*, *14*(8). https://doi.org/10.3390/antibiotics14080809

Buffart, B., Clevenbergh, P., Stiuliuc, A., Raftakis, I., Hing, M., Miendje Deyi, V. Y., Denis, O., Martiny, D., & Yin, N. (2025b). Borderline Oxacillin-Resistant Staphylococcus aureus (BORSA) Bacteremia—Case Report. *Antibiotics*, *14*(8). https://doi.org/10.3390/antibiotics14080809

Cáceres, M. E., Ledesma, M. M., Lombarte Serrat, A., Vay, C., Sordelli, D. O., Giacomodonato, M. N., & Buzzola, F. R. (2021). Growth conditions affect biofilms of Staphylococcus aureus producing mastitis: Contribution of MALDI-TOF-MS to strain characterization. *Current Research in Microbial Sciences*, *2*. https://doi.org/10.1016/j.crmicr.2021.100073

Carattoli, A., Zankari, E., Garciá-Fernández, A., Larsen, M. V., Lund, O., Villa, L., Aarestrup, F. M., & Hasman, H. (2014). In Silico detection and typing of plasmids using plasmidfinder and plasmid multilocus sequence typing. *Antimicrobial Agents and Chemotherapy*, *58*(7), 3895–3903. https://doi.org/10.1128/AAC.02412-14

Casanova, N. G., Ruiz, M. S., & Bellido, J. L. M. (2017). Mechanisms of resistance to daptomycin in Staphylococcus aureus. *Rev Esp Quimioter*, *38*(6), 36–44. http://seq.es/wp-content/uploads/2017/12/gomez25oct2017.pdf

Chambers, H. F., & DeLeo, F. R. (2009). Waves of Resistance: Staphylococcus aureus in the Antibiotic Era. *Nature Reviews Microbiology*, *7*(9), 629–641. https://doi.org/10.1038/nrmicro2200.Waves

Cheung, G. Y. C., Bae, J. S., & Otto, M. (2021). Pathogenicity and virulence of Staphylococcus aureus. *Virulence*, *12*(1), 547–569. https://doi.org/10.1080/21505594.2021.1878688

Choi, S., Lee, J., Jung, J., Kim, E. S., Kim, M. J., Chong, Y. P., Kim, S., Lee, S., Choi, S., Woo, J. H., & Kim, Y. S. (2021). A Longitudinal Study of Adult Patients with Staphylococcus aureus Bacteremia over 11 Years in Korea. *Infectious Diseases, Microbiology and Parasitology*, *36*(16), 1–13.

CLSI. (2021). *Performance Standards for Antimicrobial* (31st ed.).

Conceição, T., Coelho, C., Santos-Silva, I., De Lencastre, H., & Aires-De-Sousa, M. (2014). Epidemiology of methicillin-resistant and -susceptible staphylococcus aureus in Luanda, Angola: First description of the spread of the MRSA ST5-IVa clone in the African continent. *Microbial Drug Resistance*, *20*(5), 441–449. https://doi.org/10.1089/mdr.2014.0007

David, M. Z., & Daum, R. S. (2010). Community-associated methicillin-resistant Staphylococcus aureus: Epidemiology and clinical consequences of an emerging epidemic. *Clinical Microbiology Reviews*, *23*(3), 616–687. https://doi.org/10.1128/CMR.00081-09

David, M. Z., Glikman, D., Crawford, S. E., Peng, J., King, K. J., Hostetler, M. A., Boyle-Vavra, S., & Daum, R. S. (2008). What is community-associated methicillin-resistant Staphylococcus aureus? *Journal of Infectious Diseases*, *197*(9), 1235–1243. https://doi.org/10.1086/533502

Egyir, B., Bentum, J., Attram, N., Fox, A., Obeng-Nkrumah, N., Appiah-Korang, L., Behene, E., Kumordjie, S., Yeboah, C., Agbodzi, B., Bentil, R. E., Tagoe, R., Tabi, B. K. A., Owusu, F., Dayie, N. T. K. D., Donkor, E. S., Nsaful, J., Asah-Opoku, K., Nyarko, E., … Letizia, A. G. (2021). Whole genome sequencing and antimicrobial resistance of staphylococcus aureus from surgical site infections in Ghana. *Pathogens*, *10*(2), 1–13. https://doi.org/10.3390/pathogens10020196

Egyir, B., Dsani, E., Owusu-Nyantakyi, C., Amuasi, G. R., Owusu, F. A., Allegye-Cudjoe, E., & Addo, K. K. (2022). Antimicrobial resistance and genomic analysis of staphylococci isolated from livestock and farm attendants in Northern Ghana. *BMC Microbiology*, *22*(1), 1–10. https://doi.org/10.1186/s12866-022-02589-9

Egyir, B., Guardabassi, L., Esson, J., Nielsen, S. S., Newman, M. J., Addo, K. K., & Larsen, A. R. (2014). Insights into nasal carriage of Staphylococcus aureus in an urban and a rural community in Ghana. *PLoS ONE*, *9*(4), 1–7. https://doi.org/10.1371/journal.pone.0096119

Egyir, B., Guardabassi, L., Monecke, S., Addo, K. K., Newman, M. J., & Larsen, A. R. (2015). Methicillin-resistant Staphylococcus aureus strains from Ghana include USA300. *Journal of Global Antimicrobial Resistance*, *3*(1), 26–30. https://doi.org/10.1016/j.jgar.2014.11.006

Egyir, B., Guardabassi, L., Sørum, M., Nielsen, S. S., Kolekang, A., Frimpong, E., Addo, K. K., Newman, M. J., & Larsen, A. R. (2014). Molecular epidemiology and antimicrobial susceptibility of clinical Staphylococcus aureus from healthcare institutions in Ghana. *PLoS ONE*, *9*(2), 3–9. https://doi.org/10.1371/journal.pone.0089716

Egyir, B., Owusu-Nyantakyi, C., Bortey, A., Rabbi Amuasi, G., Owusu, F. A., Boateng, W., Ahmed, H., Danso, J. K., Oclu, A. A. G., Mohktar, Q., Tetteh-Ocloo, G., Amegbletor, H., Fosu, K., Tetteh, F. K. M., Asante-Sefa, S., Deberu, O. N., Osei, K. M., Twasam, J., Kodom, S., … Hendriksen, R. S. (2024). Whole genome sequencing revealed high proportions of ST152 MRSA among clinical Staphylococcus aureus isolates from ten hospitals in Ghana . *MSphere*, *9*(12). https://doi.org/10.1128/msphere.00446-24

Enright, M. C., Day, N. P. J., Davies, C. E., Peacock, S. J., & Spratt, B. G. (2000). Multilocus sequence typing for characterization of methicillin-resistant and methicillin-susceptible clones of Staphylococcus aureus. *Journal of Clinical Microbiology*, *38*(3), 1008–1015. https://doi.org/10.1128/jcm.38.3.1008-1015.2000

Fatsis-Kavalopoulos, N., Kim, Y. K., Chong, Y. P., Bae, S., Lim, S. Y., Kim, Y. S., & Andersson, D. I. (2025). Vancomycin heteroresistance (hVISA) in MRSA links to treatment failure and supports a revised PAP-AUC threshold. *Nature Communications* , *16*(1). https://doi.org/10.1038/s41467-025-66118-8

Feldgarden, M., Brover, V., Gonzalez-Escalona, N., Frye, J. G., Haendiges, J., Haft, D. H., Hoffmann, M., Pettengill, J. B., Prasad, A. B., Tillman, G. E., Tyson, G. H., & Klimke, W. (2021). AMRFinderPlus and the Reference Gene Catalog facilitate examination of the genomic links among antimicrobial resistance, stress response, and virulence. *Scientific Reports*, *11*(1). https://doi.org/10.1038/s41598-021-91456-0

Friedrich, A. W., Soulanoudjingar, S., Schaumburg, F., & Ko, R. (2011). Population Structure of Staphylococcus aureus from Remote African Babongo Pygmies. *PLoS Neglected Tropical Diseases*, *5*(5). https://doi.org/10.1371/journal.pntd.0001150

Ghebremedhin, B., Olugbosi, M. O., Raji, A. M., Layer, F., Bakare, R. A., & Ko, W. (2009). Emergence of a Community-Associated Methicillin-Resistant Staphylococcus aureus Strain with a Unique Resistance Profile in Southwest Nigeria. *Journal of Clinical Microbiology*, *47*(9), 2975–2980. https://doi.org/10.1128/JCM.00648-09

Goering, R. V., Swartzendruber, E. A., Obradovich, A. E., Tickler, I. A., & Tenover, F. C. (2019). Emergence of oxacillin resistance in stealth methicillin-resistant staphylococcus aureus due to mecA sequence instability. *Antimicrobial Agents and Chemotherapy*, *63*(8). https://doi.org/10.1128/AAC.00558-19

Gostev, V., Kalinogorskaya, O., Sopova, J., Sulian, O., Chulkova, P., Velizhanina, M., Tsvetkova, I., Ageevets, I., Ageevets, V., & Sidorenko, S. (2023). Adaptive Laboratory Evolution of Staphylococcus aureus Resistance to Vancomycin and Daptomycin: Mutation Patterns and Cross-Resistance. *Antibiotics*, *12*(5). https://doi.org/10.3390/antibiotics12050928

Heidarian, S., Guliaev, A., Nicoloff, H., Hjort, K., & Andersson, D. I. (2024). High prevalence of heteroresistance in Staphylococcus aureus is caused by a multitude of mutations in core genes. *PLoS Biology*, *22*(1). https://doi.org/10.1371/journal.pbio.3002457

Hopkins, J., Lee, S. J., Waithira, N., Painter, C., Ling, C. L., Roberts, T., Miliya, T., Obeng-Nkrumah, N., Opintan, J. A., Abbeyquaye, E. P., Hamers, R. L., Saharman, Y. R., Sinto, R., Karyanti, M. R., Ibrahim, R. F., Akech, S. O., Ashley, E. A., Douangnouvong, A., Choumlivong, K., … Trinh Thi, H. P. (2026). Prospective characterisation of drug-resistant bloodstream infections in Africa and

Asia (ACORN2): a surveillance network assessment. *The Lancet Microbe*, *7*(1). https://doi.org/10.1016/j.lanmic.2025.101228

Id, W. L. J., Sohn, M. B., Taffner, S., Chatterjee, P., Dunman, M., Pecora, N., & Id, R. A. F. W. (2021). Genomics of Staphylococcus aureus ocular isolates. *PLoS ONE*, *16*(5), 1–19. https://doi.org/10.1371/journal.pone.0250975

Ide, C. J., Ogban, G. I., Ekeng, B. E., Emanghe, U. E., Onukak, A. E., Iwuafor, A. A., Udoh, U. A., Chukwuma, S. T., Jimmy, E. M., Ukpeh, I. E., Mirabeau, T. Y., & Egah, D. Z. (2025). Molecular detection of mecA and lukSF-PV in patients with Staphylococcus aureus soft tissue infections in a tertiary hospital setting, Calabar, Nigeria: a cross-sectional study . *Therapeutic Advances in Infectious Disease*, *12*. https://doi.org/10.1177/20499361251357394

Iliyasu, G., Daiyab, F. M., Tiamiyu, A. B., Abubakar, S., Habib, Z. G., Sarki, A. M., & Habib, A. G. (2016). Nosocomial infections and resistance pattern of common bacterial isolates in an intensive care unit of a tertiary hospital in Nigeria: A 4-year review. *Journal of Critical Care*, *34*, 116–120. https://doi.org/10.1016/j.jcrc.2016.04.018

Karchmer, A. W. (2000). Nosocomial Bloodstream Infections: Organisms, Risk Factors, and Implications. In *Clinical Infectious Diseases* (Vol. 31). https://academic.oup.com/cid/article/31/Supplement_4/S139/267789

Karre, T. A., Kotula, R. J., & Goering, R. V. (2026). Stealth MRSA: a diagnostic pitfall in invasive Staphylococcus aureus infection . *ASM Case Reports*. https://doi.org/10.1128/asmcr.00030-26

Katopodis, G. D., Grivea, I. N., Tsantsaridou, A. J., Pournaras, S., Petinaki, E., & Syrogiannopoulos, G. A. (2010). Fusidic acid and clindamycin resistance in community-associated, methicillin-resistant Staphylococcus aureus infections in children of Central Greece. *BMC Infectious Diseases*, *10*(1), 351. https://doi.org/10.1186/1471-2334-10-351

Kaya, H., Hasman, H., Larsen, J., Stegger, M., Johannesen, T. B., Allesøe, R. L., Lemvigh, C. K., Aerestrup, F. M., Lund, O., & Larsen, A. R. (2018). SCCmecFinder, a Web-Based Tool for Typing of Staphylococcal Cassette Chromosome mec in Staphylococcus aureus Using Whole-Genome Sequence Data. *MSphere*. https://doi.org/https:// doi.org/10.1128/mSphere.00612-17

Kolawole, D. O., Adeyanju, A., Schaumburg, F., Akinyoola, A. L., Lawal, O. O., Amusa, Y. B., Kock, R., & Becker, K. (2013). Characterization of Colonizing Staphylococcus aureus Isolated from Surgical Wards ' Patients in a Nigerian University Hospital. *PLoS ONE*, *8*(7), 6–11. https://doi.org/10.1371/journal.pone.0068721

Kyany'a, C., Nyasinga, J., Matano, D., Oundo, V., Wacira, S., Sang, W., & Musila, L. (2019). Phenotypic and genotypic characterization of clinical Staphylococcus aureus isolates from Kenya. *BMC Microbiology*, *19*(1), 1–11. https://doi.org/10.1186/s12866-019-1597-1

Letunic, I., & Gmbh, B. S. (2021). Interactive Tree Of Life (iTOL) v5 : an online tool for phylogenetic tree display and annotation. *Nucleic Acids Research*, *49*(April), 293–296.

Liu, J. L., Li, T. M., Zhong, N., Wang, X., Jiang, J., Zhang, W. X., Tang, R., Guo, Y. J., Liu, Y., Hu, J., He, L. H., Tang, J., Wu, W. J., & Li, M. (2021). Current status of oxacillin-susceptible mecA-positive Staphylococcus aureus infection in Shanghai, China: A multicenter study. *Journal of Microbiology, Immunology and Infection*, *54*(6), 1070–1077. https://doi.org/10.1016/j.jmii.2020.07.021

Maalej, S. M., Rhimi, F. M., Fines, M., Mnif, B., Leclercq, R., & Hammami, A. (2012). Analysis of borderline oxacillin-resistant Staphylococcus aureus (BORSA) strains isolated in Tunisia. *Journal of Clinical Microbiology*, *50*(10), 3345–3348. https://doi.org/10.1128/JCM.01354-12

Mahomed, T. G., Kock, M. M., Masekela, R., Hoosien, E., & Ehlers, M. M. (2018). Genetic relatedness of Staphylococcus aureus isolates obtained from cystic fibrosis patients at a tertiary academic hospital in Pretoria , South Africa. *Scientific Reports*, (March), 1–6. https://doi.org/10.1038/s41598-018-30725-x

Maiden, M. C. J., Bygraves, J. A., Feil, E., Morelli, G., Russell, J. E., Urwin, R., Zhang, Q., Zhou, J., Zurth, K., Caugant, D. A., Feavers, I. M., Achtman, M., & Spratt, B. G. (1998). Multilocus sequence typing: A portable approach to the identification of clones within populations of pathogenic microorganisms. *Proceedings of the National Academy of Sciences of the United States of America*, *95*(6), 3140–3145. https://doi.org/10.1073/pnas.95.6.3140

Medugu, N., Nwajiobi-Princewill, P. I., Shettima, S. A., Mohammed, M. M., Mohammed, Y., Wariso, K., Akujobi, C. N., Oduyebo, O. O., & Iregbu, K. C. (2021). A mini- national surveillance study of resistance profiles of Staphylococcus Aureus isolated from clinical specimens across hospitals in Nigeria. *Nigerian Journal of Clinical Practice*, *24*(2), 225–232. https://doi.org/10.4103/njcp.njcp_296_20

Mintz, P. D. (2017). *WHO publishes list of bacteria for which new antibiotics are urgently needed thereby highlighting the requirement for further vigilance for platelet transfusions*.

Moirongo, R. M., Lorenz, E., Ntinginya, N. E., Dekker, D., Fernandes, J., Held, J., Lamshöft, M., Schaumburg, F., Mangu, C., Sudi, L., Sie, A., Souares, A., Heinrich, N., Wieser, A., Mordmüller, B., Owusu-Dabo, E., Adegnika, A. A., Coulibaly, B., May, J., & Eibach, D. (2020). Regional Variation of Extended-Spectrum Beta-Lactamase (ESBL)-Producing Enterobacterales, Fluoroquinolone-Resistant Salmonella enterica and Methicillin-Resistant Staphylococcus aureus Among Febrile Patients in Sub-Saharan Africa. *Frontiers in Microbiology*, *11*(September), 1–10. https://doi.org/10.3389/fmicb.2020.567235

Murray, C. J., Ikuta, K. S., Sharara, F., Swetschinski, L., Robles Aguilar, G., Gray, A., Han, C., Bisignano, C., Rao, P., Wool, E., Johnson, S. C., Browne, A. J., Chipeta, M. G., Fell, F., Hackett, S., Haines-Woodhouse, G., Kashef Hamadani, B. H., Kumaran, E. A. P., McManigal, B., … Naghavi, M. (2022). Global burden of bacterial antimicrobial resistance in 2019: a systematic analysis. *The Lancet*, *399*(10325), 629–655. https://doi.org/10.1016/S0140-6736(21)02724-0/ATTACHMENT/B227DEB3-FF04-497F-82AC-637D8AB7F679/MMC1.PDF

Obasuyi, O., McClure, J., Oronsaye, F. E., Akerele, J. O., Conly, J., & Zhang, K. (2020). Molecular characterization and pathogenicity of staphylococcus aureus isolated from Benin-City, Nigeria. *Microorganisms*, *8*(6), 1–19. https://doi.org/10.3390/microorganisms8060912

O'Brien, T. F., & Stelling, J. M. (1995). WHONET : An Information System for Monitoring Antimicrobial Resistance. *Emerging Infectious Diseases*, *1*(2), 66–67.

Ogundipe, F. O., Ojo, O. E., Feßler, A. T., Hanke, D., Awoyomi, O. J., Ojo, D. A., Akintokun, A. K., Schwarz, S., & Maurischat, S. (2020). Antimicrobial resistance and virulence of methicillin-resistant staphylococcus aureus from human, chicken and environmental samples within live bird markets in three Nigerian cities. *Antibiotics*, *9*(9), 1–15. https://doi.org/10.3390/antibiotics9090588

Okeke, I. N., Aboderin, A. O., Egwuenu, A., Underwood, A., Afolayan, A. O., Kekre, M., Ikhimiukor, O. O., Aanensen, D. M., & Ihekweazu, C. (2022). Establishing a national reference laboratory for antimicrobial resistance using a whole- genome sequencing framework : Nigeria ' s experience. *Microbiology*, *168*, 1–11. https://doi.org/10.1099/mic.0.001208

Okon, K. O., Shittu, A. O., Kudi, A. A., Umar, H., Becker, K., & Schaumburg, F. (2014). Population dynamics of Staphylococcus aureus from Northeastern Nigeria in 2007 and 2012. *Epidemiology and Infection*, *142*(8), 1737–1740. https://doi.org/10.1017/S0950268813003117

Olalekan, A., Boutin, S., Watson, C. M., Galarion, L., Adesida, S., Iwalokun, B., Popoola, O., Esemu, S., Adegbola, R., & Nurjadi, D. (2025). Molecular landscape of methicillin-resistant Staphylococcus aureus strains in clinical infections from hospitals in Lagos, Nigeria. *JAC-Antimicrobial Resistance*, *7*(5). https://doi.org/10.1093/jacamr/dlaf161

Petit, R. A., & Read, T. D. (2024, August 25). *sccmec*. Https://Github.Com/Rpetit3/Sccmec.

Popoola, O., Kehinde, A., Ogunleye, V., Adewusi, O. J., Toy, T., Mogeni, O. D., Aroyewun, E. O., Agbi, S., Adekanmbi, O., Adepoju, A., Muyibi, S., Adebiyi, I., Elaturoti, O. O., Nwimo, C., Adeoti, H., Omotosho, T., Akinlabi, O. C., Adegoke, P. A., Adeyanju, O. A., … Okeke, I. N. (2019). Bacteremia among Febrile Patients Attending Selected Healthcare Facilities in Ibadan, Nigeria. *Clinical Infectious Diseases*, *69*, S466–S473. https://doi.org/10.1093/cid/ciz516

Raji, A., Ojemhen, O., Umejiburu, U., Ogunleye, A., Blanc, D. S., & Basset, P. (2013). High genetic diversity of Staphylococcus aureus in a tertiary care hospital in Southwest Nigeria. *Diagnostic Microbiology and Infectious Disease*, *77*(4), 367–369. https://doi.org/10.1016/j.diagmicrobio.2013.08.030

Rasigade, J., Dumitrescu, O., & Lina, G. (2014). New epidemiology of Staphylococcus aureus infections. *European Society of Clinical Infectious Diseases*, *20*(7), 587–588. https://doi.org/10.1111/1469-0691.12718

Ruimy, R., Maiga, A., Armand-Lefevre, L., Maiga, I., Diallo, A., Koumaré, A. K., Ouattara, K., Soumaré, S., Gaillard, K., Lucet, J. C., Andremont, A., & Feil, E. J. (2008). The carriage population of Staphylococcus aureus from Mali is composed of a combination of pandemic clones and the divergent Panton-Valentine leukocidin-positive genotype ST152. *Journal of Bacteriology*, *190*(11), 3962–3968. https://doi.org/10.1128/JB.01947-07

Sabat, A. J., Tinelli, M., Grundmann, H., Akkerboom, V., Monaco, M., Grosso, M. Del, Errico, G., Pantosti, A., & Friedrich, A. W. (2018). Daptomycin resistant staphylococcus aureus clinical strain with novel non-synonymous mutations in the mprF and vraS genes: A new insight into daptomycin resistance. *Frontiers in Microbiology*, *9*(NOV), 1–10. https://doi.org/10.3389/fmicb.2018.02705

Saeed, A. K., Gould, I., Espositio, S., Ahmad-saeed, N., Ahmed, S. S., Alp, E., Bal, A. M., Bassetti, M., Chan, M., Coombs, G., Dancer, S., David, M. Z., Simone, G. De, Dryden, M., Guardabassi, L., Hanitsch, L. G., Hijazi, K., Krüger, R., Lee, A., … Voss, A. (2017). Panton-Valentine Leucocidin (PVL) Staphylococcus aureus a position statement from the International Society of Chemotherapy. *International Journal of Antimicrobial Agents*. https://doi.org/10.1016/j.ijantimicag.2017.11.002

San, P., Shuan, C., Teh, J., Idris, N., & Ponnampalavanar, S. (2018). Methicillin-resistant Staphylococcus aureus (MRSA) bacteremia : Correlations between clinical , phenotypic , genotypic characteristics

and mortality in a tertiary teaching hospital in Malaysia. *Infection, Genetics and Evolution*, *59*(January), 132–141. https://doi.org/10.1016/j.meegid.2018.01.031

Sati, H., Carrara, E., Savoldi, A., Hansen, P., Garlasco, J., Campagnaro, E., Boccia, S., Castillo-Polo, J. A., Magrini, E., Garcia-Vello, P., Wool, E., Gigante, V., Duffy, E., Cassini, A., Huttner, B., Pardo, P. R., Naghavi, M., Mirzayev, F., Zignol, M., … Umubyeyi Nyaruhirira, A. (2025). The WHO Bacterial Priority Pathogens List 2024: a prioritisation study to guide research, development, and public health strategies against antimicrobial resistance. *The Lancet Infectious Diseases*, *25*(9), 1033–1043. https://doi.org/10.1016/S1473-3099(25)00118-5

Schaumburg, F., Ngoa, U. A., Kösters, K., Köck, R., Adegnika, A. A., Kremsner, P. G., Lell, B., Peters, G., Mellmann, A., & Becker, K. (2011). Virulence factors and genotypes of Staphylococcus aureus from infection and carriage in Gabon. *Clinical Microbiology and Infection*, *17*(10), 1507–1513. https://doi.org/10.1111/j.1469-0691.2011.03534.x

Schmidt, T., Kock, M. M., & Ehlers, M. M. (2015). Antimicrobial Resistance in Staphylococci at the Human– Animal Interface. In *Antimicrobial Resistance - An Open Challenge*. https://doi.org/10.5772/61785

Shittu, A. O., Adesoji, T., & Udo, E. E. (2021). DNA microarray analysis of Staphylococcus aureus from Nigeria and South Africa. *PLoS ONE*, *16*(7 July), 1–11. https://doi.org/10.1371/journal.pone.0237124

Shittu, A. O., Mellmann, A., & Schaumburg, F. (2020). Molecular characterization of Staphylococcus aureus complex from fomites in Nigeria. *Infection, Genetics and Evolution*, *85*(April), 104504. https://doi.org/10.1016/j.meegid.2020.104504

Shittu, A. O., Okon, K., Adesida, S., Oyedara, O., Witte, W., Strommenger, B., Layer, F., & Nübel, U. (2011). Antibiotic resistance and molecular epidemiology of Staphylococcus aureus in Nigeria. *BMC Microbiology*, *11*(1), 92. https://doi.org/10.1186/1471-2180-11-92

Shittu, A. O., Taiwo, F. F., Froböse, N. J., Schwartbeck, B., Niemann, S., Mellmann, A., & Schaumburg, F. (2021). Genomic analysis of Staphylococcus aureus from the West African Dwarf (WAD) goat in Nigeria. *Antimicrobial Resistance and Infection Control*, *10*(1), 1–12. https://doi.org/10.1186/s13756-021-00987-8

Shittu, A., Oyedara, O., Abegunrin, F., Okon, K., Raji, A., Taiwo, S., Ogunsola, F., Onyedibe, K., & Elisha, G. (2012). Characterization of methicillin-susceptible and -resistant staphylococci in the clinical setting: A multicentre study in Nigeria. *BMC Infectious Diseases*, *12*. https://doi.org/10.1186/1471-2334-12-286

Steinig, E. J., Duchene, S., Robinson, D. A., Monecke, S., Yokoyama, M., Laabei, M., Slickers, P., Andersson, P., Williamson, D., Kearns, A., Goering, R. V., Dickson, E., Ehricht, R., Ip, M., O'sullivan, M. V. N., Coombs, G. W., Petersen, A., Brennan, G., Shore, A. C., … Tong, S. Y. C. (2019). Evolution and global transmission of a multidrug-resistant, community-associated methicillin-resistant staphylococcus aureus lineage from the Indian subcontinent. *MBio*, *10*(6), 1–20. https://doi.org/10.1128/mBio.01105-19

Tabah, A., Lipman, J., Barbier, F., Buetti, N., & Timsit, J. F. (2022). Use of Antimicrobials for Bloodstream Infections in the Intensive Care Unit, a Clinically Oriented Review. In *Antibiotics* (Vol. 11, Number 3). MDPI. https://doi.org/10.3390/antibiotics11030362

Tetzschner, A. M. M., Johnson, J. R., Johnston, B. D., Lund, O., & Scheutz, F. (2020). *In Silico Genotyping of Escherichia coli Isolates for Extraintestinal Virulence Genes by Use of Whole-Genome Sequencing Data*. https://cge.cbs.dtu.dk/services/MyDbFinder/

Touaitia, R., Mairi, A., Ibrahim, N. A., Basher, N. S., Idres, T., & Touati, A. (2025). Staphylococcus aureus: A Review of the Pathogenesis and Virulence Mechanisms. In *Antibiotics* (Vol. 14, Number 5). Multidisciplinary Digital Publishing Institute (MDPI). https://doi.org/10.3390/antibiotics14050470

Turner, P., Ashley, E. A., Celhay, O. J., Douangnouvong, A., Hamers, R. L., Ling, C. L., Lubell, Y., Miliya, T., Roberts, T., Soputhy, C., Ngoc Thach, P., Vongsouvath, M., Waithira, N., Wannapinij, P., & van Doorn, H. R. (2020). ACORN (A Clinically-Oriented Antimicrobial Resistance Surveillance Network): a pilot protocol for case based antimicrobial resistance surveillance. *Wellcome Open Research*, *5*, 13. https://doi.org/10.12688/wellcomeopenres.15681.1

Unyime C. Essien, Samar S. Boswihi, Nneka R. Agbakoba, & Edet E. Udo. (2021). Prevalence and genotypic characteristics of Panton-Valentine Leukocidin-producing Staphylococcus aureus isolates obtained in Jos, North Central Nigeria. *World Journal of Advanced Research and Reviews*, *11*(2), 273–271. https://doi.org/10.30574/wjarr.2021.11.2.0387

Van Hal, S. J., & Paterson, D. L. (2011). Systematic review and meta-analysis of the significance of heterogeneous vancomycin-intermediate Staphylococcus aureus isolates. *Antimicrobial Agents and Chemotherapy*, *55*(1), 405–410. https://doi.org/10.1128/AAC.01133-10

Vandenesch, F., Lina, G., & Henry, T. (2012). Staphylococcus aureus hemolysins, bi-component leukocidins, and cytolytic peptides: a redundant arsenal of membrane-damaging virulence factors? In *Frontiers in cellular and infection microbiology* (Vol. 2, p. 12). https://doi.org/10.3389/fcimb.2012.00012

WHO. (2024). *WHO Bacterial Priority Pathogens List, 2024*. https://www.who.int/publications/i/item/9789240093461

WHO GLASS. (2020). *Whole-genome sequencing for surveillance of antimicrobial resistance*.

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