

Sineque et al. Afr. J. Clin. Exper. Microbiol. 2026; 27 (4): 471 - 480

<https://www.africem.org>African Journal of Clinical and Experimental Microbiology. ISSN 1595-689X
AJCEM/2487. <https://www.ajol.info/index.php/ajcem>

Oct 2026; Vol.27 No.4

Copyright AJCEM 2026: <https://dx.doi.org/10.4314/ajcem.v27i4.7>

Original Article

Open Access

Nasal colonization of methicillin and glycopeptide resistant *Staphylococcus aureus* among outpatients at a clinic facility in Maputo, Mozambique: A cross-sectional study

^{*1,2}Sineque, A., ¹Mabasso, A. A., ¹Chongo, A. E., and ¹Mussagy, A.¹Department of Biological Sciences, Eduardo Mondlane University, Maputo, Mozambique²Centre for Functional Ecology – One Health, Department of Life Sciences, University of Coimbra, Coimbra, Portugal*Correspondence to: sineque.alberto@uem.ac.mz; ORCID: <https://orcid.org/0000-0003-2468-9925>

Abstract:

Background: Nasal colonization by *Staphylococcus aureus* is a major source of community and hospital associated staphylococcal infections, including methicillin-resistant *S. aureus* (MRSA) infections. Screening of MRSA nasal colonization is important in the prevention and control of infection and may provide useful information to guide antimicrobial therapy. The study aimed to determine the prevalence of nasal colonization by methicillin-resistant *S. aureus* and its glycopeptide susceptibility pattern among symptomatic and asymptomatic individuals in Maputo, Mozambique.

Methodology: A cross-sectional study of symptomatic outpatients and asymptomatic students at Eduardo Mondlane University in Maputo, was conducted by collecting nasal specimens for *S. aureus* isolation. Using culture techniques such as Mannitol salt and HiCrome™ Rapid MRSA agar, *S. aureus* was isolated based on the colonial characteristics and confirmed by Gram staining, catalase, and Microgen®Staph tests. The antibiotic susceptibility test was performed by the Kirby-Bauer disk diffusion method on Salt Mueller Hinton agar for methicillin (cefoxitin). Vancomycin and teicoplanin antibiotics were tested as an initial screening for glycopeptide inhibition patterns by the disc diffusion method.

Results: A total of 50 (39.1%) *S. aureus* isolates were obtained from nasal samples collected from 128 participants, of whom 104 (31.3%) were from university students and 24 (7.8%) from outpatients in the university clinic. The prevalence of MRSA (resistance to cefoxitin) in the study population was 7.8% (n=24/128). For the glycopeptides, 14.1% and 11.7% showed reduced susceptibility to teicoplanin and vancomycin, respectively.

Conclusion: In this study, the prevalence of nasal colonization by *S. aureus* was 39%, while the prevalence of MRSA was relatively low (7.8%) in the population studied. However, the reduced glycopeptide susceptibility highlights the need for surveillance, to prevent the spread of potential glycopeptide-resistant *S. aureus* in vulnerable populations.

Keywords: *Staphylococcus aureus*, Nasal colonization, antibiotic susceptibility, Maputo.

Received Jan 17, 2026; Revised Mar 14, 2026; Accepted Mar 16, 2026

Copyright 2026 AJCEM Open Access. This article is licensed under the terms of the Creative Commons Attribution 4.0 International License [rel="license" href="http://creativecommons.org/licenses/by/4.0/">](http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution and reproduction in any medium, provided credit is given to the original author(s) and the source. Editor-in-Chief: Prof. S. S. Taiwo

Colonisation nasale par *Staphylococcus aureus* résistant à la méthicilline et profil de sensibilité aux glycopeptides chez des individus symptomatiques et asymptomatiques à Maputo, Mozambique

^{*1,2}Sineque, A., ¹Mabasso, A. A., ¹Chongo, A. E., et ¹Mussagy, A.¹Département des Sciences Biologiques, Université Eduardo Mondlane, Maputo, Mozambique²Centre d'Ecologie Fonctionnelle – Une seule Santé, Département des Sciences de la Vie, Université de Coimbra, Coimbra, Portugal*Correspondance à: sineque.alberto@uem.ac.mz; ORCID: <https://orcid.org/0000-0003-2468-9925>

Résumé:

Contexte: La colonisation nasale par *Staphylococcus aureus* est une cause majeure d'infections staphylococciques

communautaires et nosocomiales, notamment d'infections à *S. aureus* résistant à la méthicilline (SARM). Le dépistage de la colonisation nasale par le SARM est important pour la prévention et le contrôle de l'infection et peut fournir des informations utiles pour orienter l'antibiothérapie. L'étude visait à déterminer la prévalence de la colonisation nasale par le SARM et son profil de sensibilité aux glycopeptides chez des individus symptomatiques et asymptomatiques à Maputo, au Mozambique.

Méthodologie: Une étude transversale a été menée auprès de patients externes symptomatiques et d'étudiants asymptomatiques de l'Université Eduardo Mondlane de Maputo, par prélèvement nasal en vue de l'isolement de *S. aureus*. L'isolement de *S. aureus* a été réalisé par culture sur gélose au mannitol et au sel et sur milieu HiCrome™ Rapid MRSA, en se basant sur les caractéristiques des colonies. L'identification a été confirmée par coloration de Gram, test de la catalase et test Microgen® Staph. La sensibilité aux antibiotiques a été déterminée par la méthode de diffusion sur disque de Kirby-Bauer sur gélose Mueller-Hinton salée pour la méthicilline (céfoxitine). La vancomycine et la teicoplanine ont été testées par la méthode de diffusion sur disque pour évaluer l'inhibition de la croissance par les glycopeptides.

Résultats: Cinquante isolats de *S. aureus* (39,1%) ont été obtenus à partir d'échantillons nasaux prélevés chez 128 participants, dont 104 (31,3%) étaient des étudiants et 24 (7,8%) des patients externes d'un centre hospitalier universitaire. La prévalence du SARM (résistance à la céfoxitine) dans la population étudiée était de 7,8% (n=24/128). Concernant les glycopeptides, 14,1% et 11,7% des souches ont présenté une sensibilité réduite à la teicoplanine et à la vancomycine, respectivement.

Conclusion: Dans cette étude, la prévalence de la colonisation nasale par *S. aureus* était de 39 %, tandis que la prévalence du SARM était relativement faible (7,8 %) au sein de la population étudiée. Toutefois, la sensibilité réduite aux glycopeptides souligne la nécessité d'une surveillance accrue afin de prévenir la propagation de *S. aureus* potentiellement résistant aux glycopeptides aux populations vulnérables.

Mots-clés: *Staphylococcus aureus*, colonisation nasale, sensibilité aux antibiotiques, Maputo

Introduction:

Staphylococcus aureus (*S. aureus*) is a versatile commensal microorganism found in the human body and is associated with a wide variety of infections with high morbidity, mortality, and healthcare-associated costs (1,2). *S. aureus* can efficiently adapt to multiple niches, establishing solid interactions with epithelial cells (2,3) and able to overcome these defenses and colonize this microenvironment (2,4,5). The most frequent carriage site is the nose, with approximately 20 to 40% of the general population being permanently but asymptomatically colonized, and another 30% carrying the bacterium intermittently (2,6).

Intermittent nasal colonization by *S. aureus* represents the major risk factor for infections such as cellulitis, endocarditis, impetigo, pneumonia, urinary tract infections, osteomyelitis, pneumonia, septicemia, and toxic shock syndrome (TSS) (2,4,7,8). Its pathogenicity is multifaceted, encompassing antimicrobial resistance (AMR) and a high proficiency for biofilm formation (2,9,10). *S. aureus* has become resistant to various antimicrobial agents, including the commonly used penicillin-related antibiotics such as methicillin, designated methicillin-resistant *S. aureus* (MRSA). It is considered one of the principal pathogens responsible for 100,000 global deaths annually associated with AMR (11). For instance, most MRSA isolates have also shown resistance to vancomycin, a glycopeptide that was introduced and widely used as a preferred option for treating MRSA infections (12–14).

Globally, including in the African context, the prevalence rates of MRSA and multi-drug-resistant (MDR) *S. aureus* strains reach as high as 61.6% (15–22). However, data from sub-Saharan Africa, particularly in Mozambique, remain scarce. A few studies conducted in both hospital and community settings have reported *S. aureus* infection rates of up to 12.3% (22, 23), with MDR and MRSA accounting for as much as 33.3% of these infections (23–25). This has escalated into a global health challenge, as bacterial infections once easily treated have become increasingly difficult to manage (17,26).

Nasal colonization of both *S. aureus* and MRSA plays a key role in the epidemiology and pathogenesis of both MDR *S. aureus* and MRSA infections in various populations (13,17,27). Screening for and eliminating nasal carriage of *Staphylococcus aureus* and MRSA has been associated with a reduction in infection rates (28–30). On the other hand, understanding antimicrobial patterns of *S. aureus* is crucial for developing effective prevention and control measures, particularly in developing countries, where healthcare infrastructure and access to medical services remain limited (13,30,31).

This study therefore aimed to estimate the prevalence of nasal colonization by *S. aureus* and its methicillin-resistance profile among symptomatic and asymptomatic individuals in Maputo, Mozambique, while additionally characterizing the glycopeptide susceptibility patterns of the isolates.

Materials and method:

Study type and population:

This was a cross-sectional study carried out from June to November 2022 (during Covid-19 pandemic), in a group of symptomatic outpatients attending the Eduardo Mondlane University (EMU/UEM) clinic, and asymptomatic students from the Department of Biological Sciences, Eduardo Mondlane University (DBC-EMU/UEM).

Study participants and sample collection:

Nasal specimens were collected aseptically from 128 participants, comprising outpatients (n=24) and students (n=104). Willingness of the participants to participate in the study was a strong criterion for selection. Additionally, participants were asked if they had undergone antibiotic treatment within the last two weeks prior to sample collection; and those who had received were excluded. The nasal specimens were collected with labelled sterile swab sticks and taken within 2 hours of collection for inoculation on selective media in the Microbiology laboratory at the DBC-EMU/UEM.

Ethical approval:

Before the study, ethical approval was obtained from the Mozambican National Ethical Committee on June 06, 2022 (Approval No. 347/CNBS/22). The study was explained to the participants, and only those who provided written informed consent and informed assent in the case of minors under 18 years of age, were included. Confidentiality and privacy were maintained throughout the study.

Isolation and characterization of *S. aureus*:

Each of the swab samples was inoculated onto sterilized Mannitol salt agar (MSA) and HiCrome™ Rapid MRSA agar (HiMedia, India) plates and incubated aerobically at 37°C for 24 to 48 hours. The culture media were prepared and sterilized according to the manufacturer's instructions. The isolates were characterized using standard established microbiological methods.

Isolates with golden yellow-coloured colonies on MSA agar plates and greenish yellow-

coloured colonies on HiCrome™ Rapid MRSA agar were presumptively identified as *S. aureus*, and subsequently sub-cultured on MSA plates to obtain pure colonies, followed by confirmation by Gram staining, catalase, and agglutination (Microgen®Staph) tests. All isolates that were Gram positive cocci (grape-like clusters) and positive for catalase and agglutination tests, were confirmed as *S. aureus* and submitted for antibiotic susceptibility testing (AST).

Antibiotic susceptibility testing:

Antibiotic susceptibility testing was performed in accordance with the Clinical and Laboratory Standards Institute (CLSI) guidelines (32), and from previous studies by Roy et al., (33) and Deepak et al., (34). The testing was performed using the Kirby-Bauer disk diffusion method on Mueller Hinton agar (MHA) supplemented with salt (HiMedia, India) for detection of methicillin resistance in *S. aureus* isolates using cefoxitin. Vancomycin and teicoplanin discs were additionally included as a preliminary screening approach to observe glycopeptide inhibition patterns among the isolates, while recognizing that definitive susceptibility categorization for glycopeptides is recommended using minimal inhibitory concentration (MIC)-based methods (broth microdilution or E-test) (35,36).

Briefly, a suspension of colonies of the pure culture of *S. aureus* isolate was prepared with normal sterile saline solution and adjusted to a turbidity of 0.5 McFarland standards. The cell suspension was inoculated with a sterile swab onto the surface of MHA and left briefly to dry at room temperature. Three antibiotic discs (Oxoid, UK): cefoxitin (30µg), vancomycin (30 µg) and teicoplanin (30µg) were placed on the surface of the inoculated MHA plates, and allowed to stand for at least 30 minutes before incubation for 24 hours at 30-35°C, for the growth of methicillin resistant strains (26,37).

The diameter of the inhibition zone (in mm) of the isolate was measured (in mm) with a ruler. The interpretation of AST result as sensitive, intermediate or resistant was in accordance with CLSI guidelines, as the shown in Table 1. MRSA strain (ATCC 25923), provided by José Sumbana (Laboratory of Microbiology, Faculty of Medicine, UEM), was used as a control.

Table 1: Antibiotic discs and the inhibition zone diameters used for the measurement/interpretation of the results

Antibiotic disc contents	Zone diameter of inhibition (mm)			References
	R (resistant)	I (intermediate)	S (sensitive)	
Cefoxitin (30µg)	≤ 21	-	≥ 22	CLSI (33,35)
Vancomycin (30µg)	-	-	≥15*	Roy et al., (34)
Teicoplanin (30µg)	-	-	≥14*	CLSI (33), Deepak et al., (37)

*All *S. aureus* isolates with inhibition zones below the specified threshold, which is indicative of reduced antimicrobial susceptibility, were re-tested to confirm the observed results; CLSI – Clinical and Laboratory Standard Institute.

Statistical analysis:

Statistical data analysis was performed using SPSS 21 software (IBM Corp., Armonk, NY). Frequencies were obtained and percentages were calculated for study variables. Demographic characteristics were compared with the use of the Chi-square test. All reported *p*-values of less than or equal to 0.05 were considered to be statistically significant ($p < 0.05$).

Results:

Nasal samples were collected from 128 participants: 24 outpatients from EMU/UEM clinic representing the symptomatic participants, and 104 were students from DBC-EMU/UEM, representing the asymptomatic participants. The modal age group for both groups was 18–30 years with a total of 116 participants. The age range of the participants was 1–62 years (Table 2).

Distribution of *S. aureus* and CoNS by participant's characteristics:

A total of 50 (39.1%) out of 128 participants had *S. aureus* isolated from their nasal samples while 54 (42.2%) had coagulase-negative *Staphylococcus* (CoNS) isolated. EMU/UEM clinic outpatients had higher colonization rates by *S. aureus* (41.7%, $n=10/24$) and CoNS (45.8%, $n=11/24$) than the DBC-EMU/UEM students with *S. aureus* rate of 38.5% ($n=40/104$) and CoNS rate of 41.3% ($n=43/104$) respectively, but the differences were not statistically significant ($p=0.81$).

Regarding the sex distribution, male participants had higher *S. aureus* colonization rate (57.5%, $n=27/47$) than female participants (28.4%, $n=23/81$) ($p=0.002$), while the female participants had higher CoNS colonization rate (50.6%, $n=41/81$) than the male participants (27.7%, $n=13/47$) ($p=0.02$). No statistically significant difference was found for *S. aureus* ($p=0.153$) and CoNS ($p=0.285$) colonization prevalence between the age groups.

Table 2: Prevalence of *S. aureus* and coagulase-negative *Staphylococcus* nasal colonization among the study population

Category	Sample Number (N)	<i>S. aureus</i>		<i>P</i> value	CoNS		<i>P</i> value
		N	(%)		n	(%)	
Overall	128	50	39.1	-	54	42.2	-
Type of participants							
EMU/UEM clinic Outpatients	24	10	41.7	0.819	11	45.8	0.819
DBC-EMU/UEM Students	104	40	38.5		43	41.3	
Sex							
Female	81	23	28.4	0.002*	41	50.6	0.02*
Male	47	27	57.5		13	27.7	
Age group (years)							
≤17	2	2	100.0	0.153	0	0.0	0.285
18-30	116	42	36.2		51	43.9	
31-45	4	3	75.0		1	25.0	
46-60	3	2	66.7		0	0.0	
≥61	3	1	33.3		2	66.7	

DBC – Department of Biological Sciences; EMU/UEM – Eduardo Mondlane University; CoNS – Coagulase-Negative *Staphylococcus*; *statistically significant

Distribution of MRSA and isolates with reduced zone of inhibition for glycopeptides:

Table 3 presents the frequencies of MRSA and *S. aureus* isolates with observed reduced zones of inhibition for glycopeptides. Methicillin resistance, assessed using cefoxitin, was detected in 7.8% of the study population ($n=10/128$) and 20.0% of *S. aureus* isolates ($n=10/50$), occurring in 10.0% ($n=1/10$) of *S. aureus* from outpatients and 22.5% ($n=9/40$) of *S. aureus* from students ($p=0.6631$). Reduced vancomycin susceptibility was more frequent, with 30.0% among *S. aureus* isolates ($n=15/50$) and 11.7% among the study population ($n=15/128$), showing inhibition zones below the adopted threshold for vancomycin, including 40.0% ($n=4/10$) among *S. aureus* isolates from outpatients and 27.5% ($n=11/40$) among *S. aureus* isolates from students ($p=0.4616$).

Similarly, reduced teicoplanin susceptibility was also frequent, with 36.0% among *S.*

aureus isolates ($n=18/50$) and 14.1% among the study population ($n=18/128$), exhibiting inhibition zones below the adopted threshold for teicoplanin, with higher (but insignificant) rates in outpatients (60.0%, $n=6/10$) compared with students (30.0%, $n=12/40$) ($p=0.1381$).

The simultaneous detection of methicillin resistance and reduced glycopeptide susceptibility in *S. aureus* isolates was also evaluated (Fig 1). Overall, 28.0% of the isolates ($n=14/50$) demonstrated decreased susceptibility to at least two of the antibiotics tested. Among these, 12.0% ($n=6/14$) showed reduced susceptibility to methicillin with either one of the glycopeptides, 4.0% ($n=2/14$) showing reduced susceptibility to all three antibiotic agents (methicillin, vancomycin, and teicoplanin), and 12.0% ($n=6/14$) exhibiting reduced susceptibility specifically to both glycopeptides (vancomycin and teicoplanin).

Table 3: Frequencies of MRSA and *S. aureus* isolates with reduced zone of inhibition to glycopeptides

Antibiotic disc contents and susceptibility profile/pattern	Overall (n=50)		EMU/UEM clinic Outpatients (n=10)		DBC-EMU/UEM Students (n=40)		p value
	Number	(%)	Number	(%)	Number	(%)	
Methicillin-resistance							
Cefoxitin (30µg)	10	20.0	1	10.0	9	22.5	0.6631
Reduced zone of inhibition/susceptibility for glycopeptides							
Vancomycin (30µg)	15	30.0	4	40.0	11	27.5	0.4616
Teicoplanin (30µg)	18	36.0	6	60.0	12	30.0	0.1381

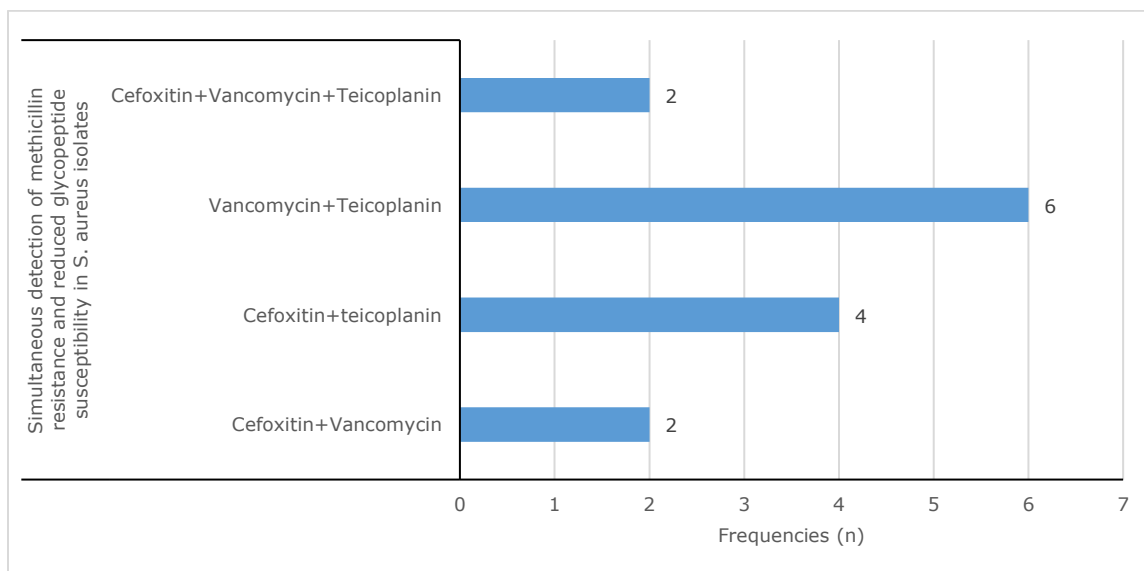


Fig 1: Frequency of simultaneous methicillin resistance and reduced glycopeptide susceptibility in *S. aureus* isolates

Discussion:

Staphylococcus aureus colonization has long been recognized as an important risk factor for subsequent infection caused by the colonizing strain (16,27). This pathogen can cause a wide range of clinical manifestations, from minor skin and soft tissue infections to severe invasive diseases such as pneumonia, bacteraemia, and sepsis. Its clinical relevance is further heightened by its remarkable capacity to acquire resistance to multiple classes of antibiotics (26,33). Of particular concern is the global spread of multidrug-resistant (MDR) strains, especially MRSA, which complicates treatment and reduces available therapeutic options. Nasal colonization of *S. aureus* has been implicated in both community-associated infections, such as skin and soft tissue infections, and hospital-associated infections, including bacteremia and surgical site infections (17,27,38).

The present study assessed the prevalence of nasal carriage of *S. aureus* and MRSA among symptomatic and asymptomatic individuals, while also examining glycopeptide inhibition zone patterns that may suggest reduced susceptibility. Our findings demonstrate an overall *S. aureus* nasal carriage rate of 39.1%, which aligns with previous studies conducted in other geographic regions. For instance, Onanuga and Temedie (26) in Nigeria, Kuehnert et al. (39) in the United States and Uemura et al. (40) in Japan, reported prevalence rates of 33.3%, 32.4% and 36.0%, respectively, among healthy adults. These findings support the notion that approximately one-third of the population may carry *S. aureus* in the anterior nares across different populations.

Nevertheless, variations in prevalence have been reported. Higher colonization rates exceeding 43% were documented in both hospital and non-hospital populations in Abia State, Nigeria (41), as well as among medical students (42) and hospital personnel (43) in Nepal. In contrast, substantially lower colonization rates have also been observed. For instance, Adesida et al., (44) reported a prevalence of only 14.0% among students in Lagos, Nigeria, while Abie et al., (45) found 23.2% prevalence among janitors in Addis Ababa, Ethiopia. The recent study by Taqveem et al., (46) in Pakistan similarly noted low prevalence among students. Such discrepancies highlight that nasal carriage is influenced by multiple epidemiological and environmental factors, including antibiotic usage, healthcare exposure, population demographics, and even sampling and laboratory techniques (17, 26).

Antibiotic exposure appears to be par-

ticularly influential. Populations receiving antibiotic treatment at the time of sampling often exhibit lower *S. aureus* prevalence due to suppression of colonization by susceptible strains. Conversely, hospital settings typically report higher colonization rates because of increased opportunities for bacterial transmission in environments with high infection burdens (44). In contrast, non-hospitalized populations not under antibiotic treatment may exhibit relatively higher prevalence, reflecting natural colonisation patterns. This has been confirmed in studies of community populations where nasal colonization typically ranges between 20–30% (2,6). These findings collectively demonstrate that *S. aureus* colonization is shaped by a complex interplay between microbial ecology, host factors, and healthcare exposure.

In the present study, the anterior nares were confirmed as a major reservoir for *S. aureus*, consistent with previous reports (27). Importantly, we found no significant difference in colonization rates between symptomatic (outpatients) and asymptomatic individuals (students) ($p=0.819$), suggesting that carriage is not necessarily associated with acute infection risk at the individual level. However, colonisation serves as a latent risk, providing a reservoir for potential infection and onward transmission. Interestingly, colonization was significantly associated with sex ($p=0.002$), with males exhibiting higher carriage rates (57.5%) compared to females (28.4%). This observation echoes findings by Taqveem et al., (46), where male students demonstrated higher prevalence of both *S. aureus* (57.7%) and MRSA (21.4%) compared to females (42.3% and 17.9%). Potential explanations include behavioral differences (e. g., hygiene practices, sports participation, or crowding exposures), as well as biological differences such as hormonal or immune factors.

Age-related colonization patterns have also been described globally, with higher rates observed shortly after birth and during adolescence (16). In African populations, additional risk factors have been identified, including HIV infection, frequent handwashing with harsh detergents, rural residence, and hospitalization, particularly in surgical wards (47,48). Conversely, higher educational status and male sex have been reported as protective in some settings (16), suggesting that risk factors may be context-specific and interact with socioeconomic, environmental, and cultural variables.

Antibiotic susceptibility testing in our study revealed low overall methicillin-resistance rates; 20.0% of the nasal *S. aureus* isolates, representing 7.8% of the study population. These findings are consistent with both local and

inter- national studies. For example, Ceccarelli et al., (49) reported 4.0% MRSA prevalence, van der Meeren et al., (23) found 15.1%, and Vubil et al., (24) documented 8.0% in Mozambique. Comparable results were also seen in Pakistan, with Taqveem et al., (46) reporting 5.5%. While MRSA prevalence appears relatively low in healthy carriers, its presence remains clinically significant because colonization can lead to difficult-to-treat infections, especially in healthcare environments.

Comparisons with international studies underscore regional variability. Chen and Huang (50) reported MRSA prevalences from <5% to >35% in Asian countries, Olonitola et al., (51) 14.85% in Nigeria, Rijal et al., (52) 56.1% in Nepal, and Onanuga and Temedie (26) 27.5% in Nigeria. The variability may reflect differences in infection control practices, antibiotic prescribing patterns, and genetic background of circulating strains. Notably, our MRSA estimation was based on phenotypic resistance to cefoxitin rather than molecular detection of the *mecA* gene, which encodes the altered penicillin-binding protein (PBP2a). This methodological limitation means that some "phenotypic MRSA" isolates may not carry *mecA*, as reported in previous studies (49,53).

The public health implications of these findings are profound. Nasal carriage of *S. aureus*, particularly MRSA, is a major driver of subsequent infections, with estimates suggesting 7% of hospitalized patients and 2% of community populations carry MRSA (17). Furthermore, healthcare-associated MRSA strains often display extensive multidrug resistance, whereas community-acquired strains remain more susceptible to non- β -lactam antibiotics (54). This distinction is critical for guiding empirical therapy and infection control interventions.

Beyond methicillin resistance, reduced inhibition zones to glycopeptides were also observed among several isolates. Overall, 30.0% (11.7% of the study population) and 36% (14.1% of the study population) of *S. aureus* isolates showed inhibition zones below the adopted threshold for vancomycin and teicoplanin, respectively. These findings warrant attention because glycopeptides remain key therapeutic agents for MRSA infections (26,55). However, the use of disc diffusion for glycopeptide susceptibility testing has limitations as these large molecules diffuse poorly in agar and may lead to underestimation or misclassification of resistance (56).

While most studies report low glycopeptide resistance ($\leq 11.1\%$), usually reflecting intermediate susceptibility phenotypes (34,55), higher rates of intermediate vancomycin resis-

tance have been reported in African settings ($\leq 14.5\%$) (14,57). The emergence of vancomycin-resistant *S. aureus* (VRSA) remains a serious global concern, particularly because vancomycin has long served as a last-line therapy against MRSA infections, with teicoplanin increasingly used as an alternative (26,34,55). These observations highlight the importance of continued surveillance and improved laboratory capacity for confirmatory testing in resource-limited settings such as Mozambique.

This study has some limitations. First, susceptibility to vancomycin and teicoplanin was tested using disc diffusion, which is not recommended by the current CLSI guidelines (35–37,58). Therefore, the classification of isolates as presumptively resistant should be interpreted cautiously and ideally confirmed using MIC-based methods such as broth microdilution or E-test (35–37). Nevertheless, previous studies suggest that disc diffusion and E-test methods may yield broadly comparable results, supporting the pragmatic use of disc diffusion in laboratories with limited resources (34). Secondly, the relatively small and specific study population limits the generalizability of the findings, emphasizing the need for larger studies involving more diverse populations across Mozambique.

Conclusion:

The findings of this study underscore the importance of nasal carriage of *S. aureus* and MRSA as key reservoirs for subsequent infection and transmission. With carriage rates of 39.1% for *S. aureus* and 7.8% for MRSA in the study population, alongside reduced glycopeptide susceptibility, our findings highlight both the persistent burden of colonization and the growing challenge of antimicrobial resistance in Mozambique. Addressing these threats requires strengthened surveillance, efficient antibiotic stewardship, and reliable confirmatory testing. Future investigations should expand to larger cohorts, integrate molecular approaches for resistance detection, and assess the effectiveness of decolonization strategies in lowering infection risk.

Acknowledgements:

The authors acknowledge the Mozambican National Investigation Fund (FNI) for the research funding and are grateful to the UEM University clinic and DBC for the cooperation during sample collection. We are also thankful to the students and patients from whom we collected our community samples.

Contributions of authors:

All authors made significant contributions to the conceptualization, design and implementation of the study, and to the drafting of the manuscript.

Source of funding:

This study received funding from the National Research Fund (FNI) under the project category "Special Projects".

Conflicts of interest:

No conflict of interest is declared

Data availability:

The data that support the findings made in this study can be made available from the corresponding author on request.

References:

- Thampi, N., Showler, A., Burry, L., et al. Multicenter study of health care cost of patients admitted to hospital with *Staphylococcus aureus* bacteremia: Impact of length of stay and intensity of care. *Am J Infect Control*. 2015; 43 (7): 739–744. doi:10.1016/j.ajic.2015.01.031.
- Sakr, A., Brégeon, F., Mège, J. L., Rolain, J. M., and Blin, O. *Staphylococcus aureus* Nasal Colonization: An Update on Mechanisms, Epidemiology, Risk Factors, and Subsequent Infections. *Front Microbiol*. 2018; 9. doi:10.3389/fmicb.2018.02419.
- Bartlett, A. H., and Hulten, K. G. *Staphylococcus aureus* Pathogenesis. *Pediatr Infect Dis J*. 2010; 29 (9): 860–861. doi:10.1097/INF.0b013e3181ef2477.
- Tong, S. Y. C., Davis, J. S., Eichenberger, E., Holland, T. L., and Fowler, V. G. *Staphylococcus aureus* Infections: Epidemiology, Pathophysiology, Clinical Manifestations, and Management. *Clin Microbiol Rev*. 2015; 28 (3): 603–661. doi:10.1128/CMR.00134-14.
- Kluytmans, J., van Belkum, A., and Verbrugh, H. Nasal carriage of *Staphylococcus aureus*: epidemiology, underlying mechanisms, and associated risks. *Clin Microbiol Rev*. 1997; 10 (3): 505–520. doi:10.1128/CMR.10.3.505.
- Wertheim, H. F., Melles, D. C., Vos, M. C., et al. The role of nasal carriage in *Staphylococcus aureus* infections. *Lancet Infect Dis*. 2005; 5 (12): 751–762. doi:10.1016/S1473-3099(05)70295-4.
- Wertheim, H. F., Vos, M. C., Ott, A., et al. Risk and outcome of nosocomial *Staphylococcus aureus* bacteraemia in nasal carriers versus non-carriers. *Lancet*. 2004; 364 (9435): 703–705. doi:10.1016/S0140-6736(04)16897-9.
- Nouwen, J., Schouten, J., Schneebergen, P., et al. *Staphylococcus aureus* Carriage Patterns and the Risk of Infections Associated with Continuous Peritoneal Dialysis. *J Clin Microbiol*. 2006; 44 (6): 2233–2236. doi:10.1128/JCM.02083-05.
- Krismer, B., and Peschel, A. Does *Staphylococcus aureus* Nasal Colonization Involve Biofilm Formation? *Future Microbiol*. 2011; 6 (5): 489–493. doi:10.2217/fmb.11.37.
- Zacher, A. T., Mirza, K., Thieme, L., Nietzsche, S., Senft, C., and Schwarz, F. Biofilm formation of *Staphylococcus aureus* on various implants used for surgical treatment of destructive spondylodiscitis. *Sci Rep*. 2024; 14 (1): 19364. doi:10.1038/s41598-024-70244-6.
- Murray, C. J. L., Ikuta, K. S., Sharara, F., et al. Global burden of bacterial antimicrobial resistance in 2019: a systematic analysis. *Lancet*. 2022; 399 (10325): 629–655. doi:10.1016/S0140-6736(21)02724-0.
- Foster, T. J. Antibiotic resistance in *Staphylococcus aureus*. Current status and future prospects. *FEMS Microbiol Rev*. 2017; 14: 430–449. doi:10.1093/femsre/fux007.
- Gnanamani, A., Hariharan, P., and Paul-Satyaseela, M. *Staphylococcus aureus*: Overview of Bacteriology, Clinical Diseases, Epidemiology, Antibiotic Resistance and Therapeutic Approach. In: *Frontiers in Staphylococcus aureus*. InTech; 2017. doi:10.5772/67338.
- Adeoye-Isijola, M., Olajuyigbe, O., Adebola, K., Cooposamy, R., and Afolayan, A. Vancomycin intermediate resistant *Staphylococcus aureus* in the nasal cavity of asymptomatic individuals: A potential public health challenge. *Afr Health Sci*. 2020; 20 (3): 1109–1117. doi:10.4314/ahs.v20i3.12.
- Conceição, T., de Lencastre, H., and Aires-de-Sousa, M. Carriage of *Staphylococcus aureus* among Portuguese nursing students: A longitudinal cohort study over four years of education. *PLoS One*. 2017; 12 (11): e0188855. doi:10.1371/journal.pone.0188855.
- Schaumburg, F., Alabi, A. S., Peters, G., and Becker, K. New epidemiology of *Staphylococcus aureus* infection in Africa. *Clin Microbiol Infect*. 2014; 20: 589–596. doi:10.1111/1469-0691.12690.
- Lee, A. S., Huttner, B. D., Catho, G., and Harbarth, S. Methicillin-Resistant *Staphylococcus aureus*: An Update on Prevention and Control in Acute Care Settings. *Infect Dis Clin North Am*. 2021; 35: 931–952. doi:10.1016/j.idc.2021.07.001.
- Lawal, O. U., Ayobami, O., Abouelfetouh, A., et al. A 6-Year Update on the Diversity of Methicillin-Resistant *Staphylococcus aureus* Clones in Africa: A Systematic Review. *Front Microbiol*. 2022; 13. doi:10.3389/fmicb.2022.860436.
- Ezeh, C. K., Eze, C. N., Dibua, M. E. U., and Emencheta, S. C. A meta-analysis on the prevalence of resistance of *Staphylococcus aureus* to different antibiotics in Nigeria. *Antimicrob Resist Infect Contr*. 2023; 12 (1): 40. doi:10.1186/s13756-023-01243-x.
- Diop, M., Bassoum, O., Ndong, A., et al. Prevalence of multidrug-resistant bacteria in healthcare and community settings in West Africa: systematic review and meta-analysis. *BMC Infect Dis*. 2025; 25 (1): 292. doi:10.1186/s12879-025-10562-w.
- Hasanpour, A. H., Sepidarkish, M., Mollalo, A., et al. The global prevalence of methicillin-resistant *Staphylococcus aureus* colonization in residents of elderly care centers: a systematic review and meta-analysis. *Antimicrob Resist Infect Contr*. 2023; 12 (1): 4. doi:10.1186/s13756-023-01210-6.
- Abdulgader, S. M., Shittu, A. O., Nicol, M. P., and Kaba, M. Molecular epidemiology of Methicillin-resistant *Staphylococcus aureus* in Africa: a systematic review. *Front Microbiol*. 2015; 6: 348. doi:10.3389/fmicb.2015.00348.
- van der Meeren, B. T., Millard, P. S., Scacchetti, M., et al. Emergence of methicillin resistance and Pantón-Valentine leukocidin positivity in

- hospital- and community-acquired *Staphylococcus aureus* infections in Beira, Mozambique. Trop Med Int Health. 2014; 19 (2): 169–176. doi: 10.1111/tmi.12221.
24. Vubil, D., Garrine, M., Ruffing, U., et al. Molecular Characterization of Community Acquired *Staphylococcus aureus* Bacteremia in Young Children in Southern Mozambique, 2001–2009. Front Microbiol. 2017; 8: 730. doi: 10.3389/fmicb.2017.00730.
 25. Garrine, M., Quintó, L., Costa, S. S., et al. Epidemiology and clinical presentation of community-acquired *Staphylococcus aureus* bacteraemia in children under 5 years of age admitted to the Manhica District Hospital, Mozambique, 2001–2019. Euro J Clin Microbiol Infect Dis. 2023; 42 (5): 653–659. doi:10.1007/s10096-023-04580-2.
 26. Onanuga, A., and Temedie, T. C. Nasal carriage of multi-drug resistant *Staphylococcus aureus* in healthy inhabitants of Amassoma in Niger delta region of Nigeria. Afr Health Sci. 2011; 11 (2):176–181.
 27. von Eiff, C., Becker, K., Machka, K., Stammer, H., and Peters, G. Nasal Carriage as a Source of *Staphylococcus aureus* Bacteremia. New Engl J Med. 2001; 344 (1): 11–16. doi:10.1056/NEJM200101043440102.
 28. Robotham Robotham, J. V., Graves, N., Cookson, B. D., et al. Screening, isolation, and decolonisation strategies in the control of methicillin resistant *Staphylococcus aureus* in intensive care units: Cost effectiveness evaluation. BMJ. 2011; 343 (7827): d5694. doi:10.1136/bmj.d5694.
 29. Lee, A. S., de Lencastre, H., Garau, J., et al. Methicillin-resistant *Staphylococcus aureus*. Nat Rev Dis Primers. 2018; 4 (1): 18033. doi:10.1038/nrdp.2018.33.
 30. Abebe, A., and Birhanu, A. Methicillin Resistant *Staphylococcus aureus*: Molecular Mechanisms Underlying Drug Resistance Development and Novel Strategies to Combat. Infect Drug Resist. 2023; 16: 7641–7662. doi:10.2147/IDR.S428103
 31. Tarai, B., Das, P., and Kumar, D. Recurrent Challenges for Clinicians: Emergence of Methicillin-resistant *Staphylococcus aureus*, Vancomycin Resistance, and Current Treatment Options. J Lab Physicians. 2013; 5 (02): 071–078. doi:10.4103/0974-2727.119843
 32. CLSI. Performance Standards for Antimicrobial Susceptibility Testing. 26th ed. CLSI supplement M100S. Wayne, PA: Clinical and Laboratory Standards Institute; 2016. Clinical and Laboratory Standards Institute; 2016. 252.
 33. Roy, P. C., Shaheduzzaman, Md., Sultana, N., and Jahid, I. K. Comparative Antibiotic Sensitivity Pattern of Hospital and Community Acquired *Staphylococcus aureus* Isolates of Jessore, Bangladesh. J Biosci Med (Irvine). 2015; 03 (10): 17–23. doi:10.4236/jbm.2015.310003
 34. Deepak, K., Ravi, T., Nayak, J., Manish Kumar, P., and Prakash, V. In-Vitro Activity of Teicoplanin against Clinical Methicillin Resistant *Staphylococcus aureus* Isolates. Open Access J Microbiol Biotechnol. 2017; 2 (1): 1–4. doi: 10.23880/oajmb-16000112.
 35. CLSI. Performance Standards for Antimicrobial Susceptibility Testing. 26th ed. CLSI supplement M100S. Wayne, PA: Clinical and Laboratory Standards Institute; 2020. Clinical and Laboratory Standards Institute; 2019. 282 p.
 36. EUCAST. European Committee on Antimicrobial Susceptibility Testing Breakpoint tables for interpretation of MICs and zone diameters Version 16.0, valid from 2026-01-01. <https://www.eucast.org/bacteria/clinical-breakpoints-and-interpretation/clinical-breakpoint-tables/>. (Accessed Mar 14, 2026)
 37. Ennab, R., Al-Momani, W., Al-Titi, R., and Elayan, A. Antibiotic profile of pathogenic bacteria isolated from postsurgical site infections in public hospitals in northern Jordan. Infect Drug Resist. 2022; 15: 359–366. doi:10.2147/IDR.S350406.
 38. Techasupaboon, T., Vasikasin, V., Varothai, N., Raknaisil, N., and Nasomsong, W. *Staphylococcus aureus* nasal carriage and bloodstream infection among conventional hemodialysis patients in Thailand: a prospective multicenter cohort study. BMC Res Notes. 2022; 15 (1): 290. doi: 10.1186/s13104-022-06185-y.
 39. Kuehnert, M. J., Kruszon-Moran, D., Hill, H. A., et al. Prevalence of *Staphylococcus aureus* Nasal Colonization in the United States, 2001–2002. J Infect Dis. 2006; 193 (2): 172–179. doi:10.1086/499632.
 40. Uemura, E., Kakinohana, S., Higa, N., Toma, C., and Nakasone, N. Comparative Characterization of *Staphylococcus aureus* Isolates from Throats and Noses of Healthy Volunteers. Jpn J Infect Dis. 2004; 57 (1): 21–24.
 41. Chigbu, C. O., and Ezeronye, O. U. Antibiotic resistant *Staphylococcus aureus* in Abia State of Nigeria. Afr J Biotechnol. 2003; 2 (10): 374–378. doi:10.5897/AJB2003.000-1077.
 42. Rongpharpi, S. R., Hazarika, N. K., and Kalita, H. The prevalence of nasal carriage of *Staphylococcus aureus* among healthcare workers at a tertiary care hospital in Assam with special reference to MRSA. J Clin Diagn Res. 2013; 7 (2): 257–260. doi:10.7860/JCDR/2013/4320.2741.
 43. Mukhiya, R. K., Shrestha, A., Rai, S. K., et al. Prevalence of Methicillin-Resistant *Staphylococcus aureus* in Hospitals of Kathmandu Valley. Nepal J Sci Technol. 2013; 13 (2): 185–190. doi:10.3126/njst.v13i2.7734.
 44. Adesida, S. A., Abioye, O. A., Bamiro, B. S., et al. Associated risk factors and pulsed field gel electrophoresis of nasal isolates of *Staphylococcus aureus* from medical students in a tertiary hospital in Lagos, Nigeria. Braz J Infect Dis. 2007; 11 (1): 63–69. doi: 10.1590/s1413-86702007000100016.
 45. Abie, S., Tiruneh, M., and Abebe, W. Methicillin-resistant *Staphylococcus aureus* nasal carriage among janitors working in hospital and non-hospital areas: a comparative cross-sectional study. Ann Clin Microbiol Antimicrob. 2020; 19 (1): 47. doi:10.1186/s12941-020-00391-x.
 46. Taqveem, A., Rasool, M. H., Aslam, B., et al. Methicillin-Resistant *Staphylococcus aureus* among Students: Nasal Carriage Rate, Contributing Factors, and Antimicrobial Susceptibility. Medicina (B Aires). 2024; 60 (10): 1590. doi:10.3390/medicina60101590.
 47. Olalekan, A.O., Schaumburg, F., Nurjadi, D., Dike, A.E., Ojurongbe, O., et al. Clonal expansion accounts for an excess of antimicrobial resistance in *Staphylococcus aureus* colonising HIV-positive individuals in Lagos, Nigeria. Int J Antimicrob Agents. 2012; 40 (3): 268–272. doi: 10.1016/j.ijantimicag.2012.05.016.
 48. Kinabo, G. D., van der Ven, A., Msuya, L. J., et al. Dynamics of nasopharyngeal bacterial colonisation in HIV-exposed young infants in Tanzania. Trop Med Int Health. 2013; 18 (3): 286–295. doi:10.1111/tmi.12057.
 49. Ceccarelli, D., Mondlane, J., Sale, M., et al. Sporadic methicillin resistance in community acquired *Staphylococcus aureus* in Mozambique. New Microbiol. 2005; 28 (4): 327–336.
 50. Chen, C. J., and Huang, Y. C. New epidemiology of *Staphylococcus aureus* infection in Asia. Clin

- Microbiol Infect. 2014; 20 (7): 605–623.
doi:[10.1111/1469-0691.12705](https://doi.org/10.1111/1469-0691.12705).
51. Olonitola, O. S., Olayinka, B. O., and Sani, F. D. Antibiotic susceptibility of *Staphylococcus aureus* isolates from a Nigerian Federal Medical Center. Cameroon J Exper Biol. 2008; 3 (2).
doi:[10.4314/cajeb.v3i2.37968](https://doi.org/10.4314/cajeb.v3i2.37968)
 52. Rijal, K. R., Pahari, N., Shrestha, B. K., et al. Prevalence of methicillin resistant *Staphylococcus aureus* in school children of Pokhara. Nepal Med Coll J. 2008; 10 (3): 192–195.
 53. Olayinka, B. O., Olayinka, A. T., Obajuluwa, A. F., Onaolapo, J. A., and Olurinola, P. F. Absence of *mecA* gene in methicillin-resistant *Staphylococcus aureus* isolates. Afr J Infect Dis. 2010; 3 (2): 49–56. doi:[10.4314/ajid.v3i2.55081](https://doi.org/10.4314/ajid.v3i2.55081).
 54. Fey, P. D., Saïd-Salim, B., Rupp, M. E., et al. Comparative Molecular Analysis of Community- or Hospital-Acquired Methicillin-Resistant *Staphylococcus aureus*. Antimicrob Agents Chemother. 2003; 47 (1): 196–203.
doi:[10.1128/AAC.47.1.196-203.2003](https://doi.org/10.1128/AAC.47.1.196-203.2003)
 55. Shariati, A., Dadashi, M., Moghadam, M. T., van Belkum, A., Yaslianifard, S., and Darban-Sarokhalil, D. Global prevalence and distribution of vancomycin resistant, vancomycin intermediate and heterogeneously vancomycin intermediate *Staphylococcus aureus* clinical isolates: A systematic review and meta-analysis. Sci Rep. 2020; 10 (1): 12689.
doi:[10.1038/s41598-020-69058-z](https://doi.org/10.1038/s41598-020-69058-z)
 56. Cavenaghi, L. A., Biganzoli, E., Danese, A, and Parenti, F. Diffusion of teicoplanin and vancomycin in agar. Diagn Microbiol Infect Dis. 1992; 15 (3): 253–258. doi:[10.1016/0732-8893\(92\)90121-9](https://doi.org/10.1016/0732-8893(92)90121-9).
 57. Belete, M. A., Gedefie, A., Alemayehu, E., et al. The prevalence of vancomycin-resistant *Staphylococcus aureus* in Ethiopia: a systematic review and meta-analysis. Antimicrob Resist Infect Control. 2023; 12 (1): 86. doi:[10.1186/s13756-023-01291-3](https://doi.org/10.1186/s13756-023-01291-3).
 58. Humphries, R. M., Abbott, A. N., and Hindler, J. A. Understanding and Addressing CLSI Breakpoint Revisions: a Primer for Clinical Laboratories. J Clin Microbiol. 2019; 57 (6): e00203-19.
doi:[10.1128/JCM.00203-19](https://doi.org/10.1128/JCM.00203-19).