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Phenotypic Profiling and Molecular Detection of Antibiotic Resistance Genes in *Staphylococcus aureus* Isolated from Frequently Touched Surfaces in a Clinical Environment

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Abstract

Background: *Staphylococcus aureus* is one of the primary causative agents of nosocomial infections in hospitals and has an unusual capacity to acquire and distribute antibiotic resistance genes, among which the gene responsible for resistance to methicillin and most beta-lactams, called *mecA*, is widely distributed. **Methods:** Swab samples (200) were collected from (10) common touchable surfaces from medicine and surgery departments, outpatient wards of a private clinic, the emergency ward of a private hospital, and a dental clinic. Samples were plated on mannitol salt agar. It was performed according to Gram staining and biochemical tests (catalase and coagulase test). Identification by species-specific PCR for the *nuc* gene was performed. Antibiotic resistance was estimated by the Kirby-Bauer disk diffusion method. Genomic DNA extraction and search for resistance genes by PCR were performed. **Results:** *S. aureus* was detected in 46 (23.0%) swabs, with the maximum prevalence of positive results from bed rails (35.0%), mobile phones (30.0%), and stethoscope diaphragms (30.0%). Cefoxitin resistance was demonstrated by 19 isolates (41.3%), and the *mecA* gene was detected in 20 isolates (43.5%). High rates of resistance were shown for penicillin (91.3%), erythromycin (58.7%), and tetracycline (52.2%), and there was no vancomycin resistance among any isolates. The genes *tetK* (39.1%), *tetM* (23.9%), *ermC* (41.3%), and *ermA* (19.6%) were identified in isolates, and 24 isolates (52.2%) were multidrug-resistant.

Conclusion: Frequent touchable surfaces in hospitals may serve as reservoirs for multidrug-resistant *S. aureus*, including *mecA*-positive MRSA. This highlights the importance of strict surface disinfection, proper hand hygiene, and microbiological surveillance as key measures of infection control in hospitals.

Keywords: *Staphylococcus aureus*; MRSA; *mecA* gene; antibiotic resistance; PCR; clinical environment.

Introduction

Staphylococcus aureus is a Gram-positive, catalase-positive and coagulase-positive coccus living in the anterior nares, skin and mucous membranes of 20-30% of healthy people, and even more frequently detected transiently [1, 2]. Although often acting as a commensal, *S. aureus* is one of the major human pathogens capable of causing diseases ranging from relatively mild skin and soft tissue infections (folliculitis, abscesses) to more serious systemic infections (bacteremia, infective endocarditis, osteomyelitis, necrotizing pneumonia, and toxic shock syndrome) [3, 4]. The pathogenicity of *S. aureus* is attributed to numerous virulence factors

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(surface proteins, exotoxins, enzymes, and immune evasion factors) [5] and its outstanding genetic plasticity that enables *S. aureus* to acquire antibiotic resistance genes rapidly [6–8].

The emergence and prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) represent one of the biggest problems in contemporary clinical microbiology and infection control [7, 9, 10]. Methicillin resistance is associated with the presence of the *mecA* gene located on a mobile element called staphylococcal cassette chromosome mec (SCCmec) [11, 12]. *mecA* encodes an altered penicillin-binding protein, PBP2a (or PBP2′) that poorly binds to beta-lactam antibiotics [13]. As PBP2a is capable of substituting normal penicillin-binding proteins responsible for peptidoglycan synthesis even in the presence of beta-lactams, strains possessing the *mecA* gene are resistant to methicillin, oxacillin, and other members of the beta-lactam group of antibiotics (penicillins, cephalosporins, carbapenems) [7, 13]. Recently, a new gene homologous to *mecA*, called *mecC*, was identified in certain MRSA strains, primarily animal-derived ones [14, 15].

Other genes conferring resistance to various groups of antibiotics are common in S. aureus besides *mecA*. Tetracycline resistance is usually associated with the presence of tetK and tetM genes coding for an efflux pump and ribosomal protection protein, respectively [16]. MLSB (macrolides, lincosamides and streptogramin B) resistance is generally due to *ermA*, *ermB*, and *ermC* genes encoding ribosomal methylases capable of modifying the antibiotic target in the ribosome [17]. Aminoglycoside resistance is often associated with the aac(6′)-aph(2″) gene and other genes encoding aminoglycoside-modifying enzymes [18]. Fluoroquinolone resistance does not result from horizontally transferred genes but rather arises as a consequence of mutations in *gyrA* and *parC* [19]. A strain possessing multiple resistance determinants becomes resistant to several antibiotic classes, thus developing a multidrug-resistant (MDR) phenotype [20].

Healthcare facilities are considered an important source of MRSA and other multidrug-resistant bacteria. *S. aureus* and MRSA can survive for days to months in a dry form, as their dehydration rate is lower than that of most Gram-negative bacteria. Commonly touched surfaces (bed rails, door handles, call buttons, mobile phones, keyboards, stethoscopes, etc.) become contaminated with bacteria by hands and can transmit the infection indirectly between patients and healthcare workers [21–24].

Numerous studies examining outbreaks and hospital disinfection programs all over the world report frequent MRSA and MSSA contamination of commonly touched surfaces depending on the surface material, department, local infection control policy, and disinfection procedures. Molecular typing of bacterial isolates sometimes reveals identical genotypes of strains in both the environment and patients staying in the same ward. Contamination of a room previously used by a colonized patient increases the risk of acquisition of colonization for the next patient. Thus, environmental contamination is not accidental and represents a potential transmission route [25–28].

Antimicrobial resistance (AMR) is among the greatest global health threats today, as emphasized repeatedly by the WHO and other public health agencies. MRSA remains high on the priority list due to its common occurrence in healthcare and community settings, increased morbidity, mortality, prolonged hospitalization, and increased expenses compared to susceptible infections, as well as acquisition of resistance to other antibiotic classes (particularly vancomycin). MRSA continues to be one of the major causes of S. aureus infections in numerous healthcare settings according to surveillance studies performed in various parts of the world [29, 30].

Since environmental sources of MRSA are important for infection control, this study examined the frequency of isolation of S. aureus, particularly methicillin-resistant strains, from commonly touched surfaces in a clinical environment and determined resistance genes in isolated bacterial isolates using molecular techniques. The hypotheses were as follows: one would find *S. aureus* in some commonly touched surfaces; a considerable proportion of these isolates would contain *mecA* gene and be resistant to methicillin; there would be other resistance genes for tetracyclines and macrolides present as well [25].

Materials and Methods

Study Design and Setting

The study was designed as a descriptive cross-sectional study where the microbiological and molecular analyses were performed on samples taken from medicine and surgery departments, outpatient wards of a private clinic, the emergency ward of a private hospital and a dental clinic during about three months' period from April to July 2026. Sampling was done at various time periods of the day and on various days of the week.

Sample Collection

200 samples of ten high-touch surfaces, including bedrails, door handles, nurses' station desktops, computer keyboards, mobile phones of the healthcare personnel, stethoscopes' diaphragms, IV poles, light switches, patient call buttons, and waiting room chairs were obtained with swabs. Sterile cotton swabs pre-moistened with sterile normal saline were used for sampling of a 25 cm^2 area with the help of a uniform sampling technique. Swabs were placed into sterile vials with transport media and transported to the laboratory within two hours of sampling.

Bacterial Isolation and Phenotypic Identification

Every single swab was inoculated onto the Mannitol Salt Agar (MSA) plate – a differential and selective medium for staphylococci and incubated aerobically at 37 °C for 24–48 hours. The yellow colonies surrounded by a yellow halo were subcultivated on nutrient and blood agar. The presumptive *S. aureus* was confirmed using identification methods such as Gram staining (gram-positive cocci in clusters), catalase test (positive), and tube or slide coagulase test (positive).

Antibiotic Susceptibility Testing

The susceptibility of all *S. aureus* isolates to the antimicrobials was studied using the Kirby-Bauer disk diffusion method with Mueller-Hinton agar plates according to the CLSI guidelines2025 [31–33]. The antibiotics tested included: cefoxitin (*mecA*), oxacillin, penicillin, erythromycin, clindamycin, tetracycline, gentamicin, ciprofloxacin, trimethoprim-sulfamethoxazole, and vancomycin. The zones of inhibition were evaluated, and results were interpreted as sensitive, intermediate, or resistant according to the CLSI breakpoints. *S. aureus* isolates resistant to three or more antimicrobial classes were referred to as multidrug-resistant (MDR) [20].

DNA Extraction

Genomic DNA was isolated from pure overnight cultures of the confirmed *S. aureus* isolates using a commercial DNA isolation kit (AmpliSens, Russia) according to the instructions. The quantity of DNA was estimated with a NanoDrop device (Bioneer, South Korea).

PCR Detection of Species-Specific and Resistance Genes

The presence of *S. aureus* was confirmed with a fast PCR-blast of the *nuc* gene specific for S. aureus. Methicillin resistance was detected using a PCR for the detection of the *mecA* gene. The resistance genes tested included *tetK* and *tetM* (tetracycline resistance) and *ermA* and *ermC* (macrolide-lincosamide-streptogramin B resistance). PCR reactions included the use of gene-specific primers (Table 1) in a 25 µL reaction mixture with PCR master mix, 0.5 µM of each primer, and 50–100 ng

of template DNA. The PCR cycle included an initial denaturation followed by 35 cycles of denaturation, primer annealing at the optimal temperature for the target, and extension, plus final extension (Table 2). For every test run, positive and negative controls for each target gene were used. PCR products were analyzed using 1.5-2% agarose gel electrophoresis, stained and visualized under UV illumination after comparison with a 100 bp DNA ladder [34–36].

Table 1: Primers and expected amplicon sizes used for the detection of species-specific and resistance genes

Target gene	Primer sequences (5′→3′)	Amplicon size (bp)	Annealing (°C)	Reference
Nuc	F: GCGATTGATGGTGATACGGTT R: AGCCAAGCCTTGACGAACTAAAGC	279	55	[34]
mecA	F: GTAGAAATGACTGAACGTCCGATAA R: CCAATTCCACATTGTTTCGGTCTAA	310	55	[35]
tetK	F: GTAGCGACAATAGGTAATAGT R: GTAGTGACAATAAACCTCCTA	360	55	[36]
tetM	F: AGTGGAGCGATTACAGAA R: CATATGTCCTGGCGTGTCTA	158	55	[36]
ermA	F: AAGCGGTAAACCCCTCTGA R: TTCGCAAATCCCTTCTCAAC	190	55	[36]
ermC	F: AATCGTCAATTCCTGCATGT R: TAATCGTGGAATACGGGTTTG	299	55	[36]

*F, forward primer; R, reverse primer.

Table 2: PCR thermal cycling program

Step	Temperature (°C)	Time	Number of cycles
Initial denaturation	94	5 min	1
Denaturation	94	30 s	35
Primer annealing	55 (gene-specific, Table 1)	30 s	35
Extension	72	45 s	35
Final extension	72	7 min	1
Hold	4	∞	—

Quality Control

Incorporation of reference strains

To ensure reliability and validity of the results in antibiotic susceptibility testing and PCR amplification, reference strains were incorporated in every batch. They include methicillin-susceptible *S. aureus* ATCC 25923 (*mecA*-negative control) and methicillin-resistant *S. aureus* ATCC 33591 or ATCC 43300 (*mecA*-positive control) [33]. No-template negative controls were conducted in every PCR amplification as well to eliminate contamination due to reagents used [36].

Statistical analysis

The data were arranged in an Excel spreadsheet, and descriptive statistics (frequency and percentage) were calculated for each categorical variable (contamination rates of different surfaces, phenotypic and genotypic resistance rates, multidrug resistance rates). Chi-square test or Fisher's exact test was used to compare contamination and resistance rates of different surfaces, respectively, and $p < 0.05$ was taken as the level of statistical significance.

Results

Isolation rate overall

Out of 200 swabs, 46 (23.0%) grew *S. aureus* colonies on mannitol salt agar, and they were confirmed by Gram staining, catalase, coagulase tests, and *nuc* gene amplification PCR (perfect match for species identification) [34]. Contamination rates differed among surfaces (from 15.0% to 35.0%, Table 3). Bed rails, mobile phones, and stethoscope diaphragms had the highest rates of contamination (35.0%, 30.0%, and 30.0%, respectively); however, the difference between surfaces was not statistically significant ($\chi^2 = 5.19$, $df = 9$, $p = 0.82$).

Table 3: Distribution of confirmed *S. aureus* isolates by surface category

Surface category	Samples (n)	*S. aureus* positive, n (%)
Bed rails	20	7 (35.0%)
Mobile phones (staff)	20	6 (30.0%)
Stethoscope diaphragms	20	6 (30.0%)
Door handles	20	5 (25.0%)
Computer keyboards	20	5 (25.0%)
Patient call buttons	20	4 (20.0%)
IV pole surfaces	20	4 (20.0%)
Nurses' station desktops	20	3 (15.0%)
Light switches	20	3 (15.0%)
Waiting area chairs	20	3 (15.0%)
Total	**200**	**46 (23.0%)**

Phenotypic Antibiotic Susceptibility

From the cefoxitin disk diffusion, it was clear that 19 out of 46 isolates (41.3%) were methicillin-resistant based on phenotypic tests. The prevalence of resistance to other antibiotics was also relatively high: penicillin (91.3%), erythromycin (58.7%), tetracycline (52.2%), oxacillin (39.1%), clindamycin (34.8%), and ciprofloxacin (30.4%). The lowest rates of resistance were observed in gentamicin (13.0%) and trimethoprim-sulfamethoxazole (10.9%). None of the bacteria tested had resistance to vancomycin, as all 46 isolates (100%) were susceptible to vancomycin (Table 4).

Molecular Detection of Resistance Genes

Of the 46 isolates tested, the PCR test results indicate that 20 (43.5%) isolates were positive for the presence of the *mecA* gene, quite close to the proportion of resistance to cefoxitin (41.3%). There was even one isolate that was *mecA*-positive but had an intermediate zone diameter for cefoxitin. In the 24 isolates of tetracycline-resistant strains, 18 (75.0%) isolates contained the *tetK* gene, and 11 (45.8%) isolates contained the *tetM* gene; 8 isolates had both of these genes. In the 27 isolates resistant to erythromycin, 19 (70.4%) isolates contained the *ermC* gene, and 9 (33.3%) isolates contained the *ermA* gene. The sum of these numbers is greater than the number of resistant isolates, so at least one isolate has both genes, as shown in Table 5.

Table 4: Phenotypic antibiotic resistance rates among *S. aureus* isolates (n = 46)

Antibiotic	Resistant, n (%)
Penicillin	42 (91.3%)
Cefoxitin	19 (41.3%)
Oxacillin	18 (39.1%)
Erythromycin	27 (58.7%)
Clindamycin	16 (34.8%)
Tetracycline	24 (52.2%)
Ciprofloxacin	14 (30.4%)
Gentamicin	6 (13.0%)
Trimethoprim-sulfamethoxazole	5 (10.9%)
Vancomycin	0 (0.0%)

Table 5: Distribution of antibiotic resistance genes detected by PCR (n = 46)

Resistance gene	Positive, n (%)
mecA	20 (43.5%)
tetK	18 (39.1%)
tetM	11 (23.9%)
ermC	19 (41.3%)
ermA	9 (19.6%)

Multidrug Resistance and Surface Distribution of *mecA* -Positive Isolates

Half of the isolates possessed resistance to three or more classes of antibiotics (24 out of 46, 52.2%). The prevalence of the *mecA* gene was high in *S. aureus* isolates obtained from bed rails (6 of 7, or 85.7%) and from stethoscope diaphragms (5 of 6, 83.3%). Thus, the two mentioned surfaces contained 11 of 20 *mecA*-positive isolates (55.0%). *S. aureus* contamination of bed rails was the highest, and stethoscope diaphragms (along with mobile phones) were among the dirtiest surfaces; thus, frequently touched surfaces and surfaces with close patient contact could be considered possible sources of methicillin-resistant isolates.

Discussion

In the present study, a large proportion of surfaces in the clinical setting were contaminated with *S. aureus* (23.0%) and a considerable portion of isolates contained the *mecA* gene (43.5% by PCR), confirming methicillin resistance of *S. aureus* at the molecular level. The isolation rate was higher than 12.7% at frequently touched sites in public hospitals in South Africa, in which an even larger proportion of *S. aureus* isolates were MRSA (89.9%) [37]. The prevalence of *mecA* we detected was also similar to the 38.0% prevalence among *S. aureus* clinical isolates in Nigeria [38]. Our findings confirm results of numerous environmental studies in which high colonization rates of hospital surfaces with *S. aureus* and MRSA were observed [23, 25, 26].

Good correlation between phenotypic cefoxitin resistance and the molecular presence of *mecA* (41.3% vs. 43.5%) confirms that the cefoxitin disk diffusion test is still a valuable, cheap method for detecting *mecA*-driven methicillin resistance in clinical practice [39]. In addition, molecular techniques allow revealing rare misidentifications, such as a single mecA-positive isolate with an intermediate cefoxitin zone [39].

The highest contamination rates belonged to bed rails, mobile phones, and stethoscope diaphragms, while the greatest amounts of *mecA*-positive isolates were detected in bed rails and stethoscope diaphragms. Similar results were reported in previous research in which frequently touched surfaces and equipment used in patients with subsequent patient-to-patient transmission were identified as major contamination sources [24–26, 40–43]. Bed rails were frequently touched by patients, visitors, and healthcare personnel during every shift, but not always sufficiently cleaned, especially during between-patient cleaning. Stethoscopes were frequently used on multiple patients in succession, especially during ward rounds, and it was often forgotten to clean the diaphragm before each examination despite available guidelines [42, 43]. Mobile phones of the staff were often brought into patient rooms and rarely disinfected [41]. Do not interpret the lack of a statistically significant difference in contamination rates between 10 surface types ($p = 0.82$) as proof—the small sample size (20 per surface) is an obstacle to this conclusion.

Co-detection of tetracycline and macrolide resistance genes along with *mecA* and a high rate of multidrug resistance (52.2%) confirms the tendency of healthcare-associated MRSA lineages (especially those containing SCCmec I–III) to accumulate multiple resistance characteristics via mobile genetic elements and co-selection under continuous antibiotic selection pressure [7, 20, 36]. Prevalence of *tetK* over *tetM* among tetracycline-resistant isolates and *ermC* over *ermA* among erythromycin-resistant isolates was consistent with results of previous multiplex PCR studies of *S. aureus* [36]. Such multidrug resistance poses clinical problems: in case of spread of these isolates from the environment to patients, therapeutic options will be restricted, and last-resort antibiotics (vancomycin, linezolid, or daptomycin) will be necessary [9, 44].

Despite the absence of vancomycin resistance in the isolates, these results are in agreement with the current rarity of vancomycin-resistant *S. aureus* (VRSA) worldwide, despite the reports on vancomycin-intermediate *S. aureus* (VISA) and heteroresistant VISA (hVISA) in different regions [45, 46]. However, it is necessary to remain vigilant, because *S. aureus* could acquire resistance to almost any class of antibiotics under selection pressure [7, 46].

For infection prevention and control, the current study highlights the importance of several critical measures such as improvement of the environmental cleaning and disinfection of high-risk surfaces, including bed rails and equipment shared between patients [25, 47], disinfection of stethoscopes and other equipment between patient contacts [45, 47], monitoring hand hygiene compliance and staff training [48], and performing periodic environmental microbiological surveillance [25, 26]. Study of local resistance genes allows tailoring these measures to genes present in a particular health care facility.

There are several limitations of the current study: the cross-sectional design does not consider temporal changes due to cleaning schedule or seasons. It was performed in private healthcare facilities only, and, therefore, contamination rates and gene profiles may differ in other settings depending on patient cohort, antibiotic usage, and cleaning schedule. The sample size of 10 surfaces was relatively small and restricts comparison of surface types. No molecular typing (SCCmec typing, spa typing, or MLST) was performed, and, consequently, it is impossible to evaluate genetic similarity of environmental isolates with those from patients or healthcare workers. In addition, vancomycin susceptibility by disk diffusion should be interpreted cautiously, because CLSI recommended MIC-based methods for this antibiotic [33].

The extension of the panel of examined resistance genes (such as aminoglycoside resistance genes, mecC, and genes responsible for biofilm formation and virulence) will provide a more complete view of resistance and virulence [5, 14]. The study on the effect of improved cleaning procedures or new disinfection methods on surface contamination will directly guide policy [25, 47].

Conclusion

This study demonstrated high contamination of frequently touched surfaces in the clinical setting with *S. aureus*, with a significant number of isolates being methicillin-resistant (according to *mecA*) and containing other resistance genes blocking tetracycline and macrolide antibiotics. The contamination rate was high on bed rails, mobile phones, and medical devices such as stethoscope diaphragms. This indicates the importance of the environment as a source of multidrug-resistant bacteria and stresses the necessity of adequate environmental cleaning, proper disinfection of the medical devices, compliance with hand hygiene, and microbiological surveillance [25, 47, 48].

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