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Molecular characterization of methicillin-resistant *Staphylococcus aureus* isolates from a hospital in Ghana

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Abstract:

Background: Methicillin-resistant *Staphylococcus aureus* (MRSA) are a major cause of hospital- and community-acquired infection. They can colonize humans and cause a wide range of infections including pneumonia, endocarditis and bacteraemia. We investigated the molecular mechanism of resistance and virulence of MRSA isolates from a teaching hospital in Ghana.
Methodology: A total of 91 *S. aureus* isolates constituted the initial bacterial sample. Identification of *S. aureus* was confirmed by the VITEK 2 system. The cefoxitin screen test was used to detect MRSA and antibiotic susceptibility was determined using the VITEK 2 system. The resistance (*mecA, blaZ, aac-aph, ermC,* and *tetK*) and virulence (*lukS/F-PV, hla, hld* and *eta*) genes were amplified by polymerase chain reaction (PCR) and positive samples subjected to DNA sequencing. Pulsed field gel electrophoresis (PFGE) was used to ascertain the relatedness of the isolates.
Results: Fifty-eight of 91 (63.7%) isolates were putatively methicillin resistant by the phenotypic cefoxitin screen test and oxacillin MICs. However, 43 (47%) of the isolates were genotypically confirmed as MRSA based on PCR detection of the *mecA* gene. Furthermore, 37.9% of isolates displayed resistance to tetracycline, 19% to trimethoprim-sulphamethoxazole, 15.5% to clindamycin, 12.1% to gentamicin, 13.8% to ciprofloxacin and erythromycin, 6.9% to moxifloxacin and 7.0% to rifampicin. None of the isolates was positive for inducible clindamycin resistance. The prevalence of resistance (*mecA, blaZ, aac(6')-aph(2''), tetK, and ermC*) and virulence (*hla and lukS/F-PV*) genes respectively were 74%, 33%, 22%, 19%, 3%, 5% and 3%, with isolates organized in two highly related clades.
Conclusion: Results indicate a fairly high occurrence of MRSA, which can complicate the effective therapy of *S. aureus* infections, necessitating surveillance and stringent infection control programmes to forestall its spread.

Keywords: MRSA, *mecA*, *blaZ*, *hla*, *lukS/F-PV*

Caractérisation moléculaire d'isolats de *Staphylococcus aureus* résistants à la méthicilline provenant d'un hôpital du Ghana

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Abstrait:

Contexte: Le *Staphylococcus aureus* résistant à la méthicilline (SARM) est une cause majeure d'infection acquise à l'hôpital et dans la communauté. Ils peuvent coloniser les humains et causer un large éventail d'infections, notamment la pneumonie, l'endocardite et la bactériémie. Nous avons étudié le mécanisme moléculaire de résistance et de virulence des isolats de SARM provenant d'un hôpital universitaire au Ghana.
Méthodologie: Au total, 91 isolats de *S. aureus* constituaient l'échantillon bactérien initial. L'identification de S. aureus a été confirmée par le système VITEK 2. Le test de dépistage à la céfoxitine a été utilisé pour détecter le SARM et la sensibilité aux antibiotiques a été déterminée à l'aide du système VITEK 2. Les gènes de résistance (*mecA, blaZ, aac-aph, ermC* et *tetK*) et de virulence (*lukS/F-PV*, *hla, hld* et *eta*) ont été amplifiés par une réaction en chaîne de la polymérase (PCR) et des échantillons positifs soumis à un séquençage de l'ADN. Une électrophorèse sur gel en champ pulsé (PFGE) a été utilisée pour déterminer le caractère apparent des isolats.
Résultats: Cinquante-huit des 91 isolats (63,7%) étaient présumés résistants à la méthicilline par le test de dépistage phénotypique à la céfoxitine et par les CMI oxacillines. Cependant, 43 (47%) des isolats ont été confirmés génotypiquement comme SARM sur la base de la détection par PCR du gène *mecA*. En outre, 37,9% des isolats présentaient une résistance à la tétracycline, 19% au triméthoprime-sulfaméthoxazole, 15,5% à la clindamycine, 12,1% à la gentamicine, 13,8% à la ciprofloxacine et à l'érythromycine, 6,9% à la moxifloxacine et 7,0% à la rifampicine. Aucun des isolats n'était positif pour la résistance inductible à la clindamycine. La prévalence des gènes de résistance (*mecA, blaZ, aac(6')-aph(2''), tetK* et *ermC*) et de virulence (*hla* et *lukS/F-PV*) était respectivement de 74%, 33%, 22%, 19%, 3%, 5% et 3%, avec des isolats organisés en deux clades fortement apparentés.
Conclusion: Les résultats indiquent une présence assez élevée de SARM, ce qui peut compliquer le traitement efficace des infections à *S. aureus*, nécessitant une surveillance et des programmes de contrôle des infections rigoureux pour prévenir sa propagation.

Mots-clés: SARM, *mecA, blaZ, hla*, *lukS/F-PV*

Introduction:

Staphylococcus aureus cause a variety of infections, including mild skin infections such as boils, stye and furuncles as well as more severe infections such as meningitis, pneumonia, phlebitis, mastitis, urinary tract infections, endocarditis and osteomyelitis (1). Methicillin resistance is problematic for many healthcare facilities worldwide as is the occurrence of community strains of methicillin resistant *Staphylococcus aureus* (MRSA) which also harbor genes associated with increased virulence (2, 3). MRSA strains are produced by *S. aureus* when the *mecA* gene is acquired by methicillin susceptible *S. aureus* (MSSA). The *mecA* gene is borne on the mobile element referred to as the staphylococcal cassette chromosome (SCC) *mec* (4, 5).

There is considerable variability in the prevalence as well as the epidemiology of MRSA within and between countries (6) with limited data on the antibiotic susceptibility patterns and

molecular epidemiology of MRSA in Africa. Several African countries report a MRSA prevalence ranging from 4.8% to 20.0% (7-14). This study investigated the antibiotic susceptibility and molecular characterization of MRSA isolates from clinical samples in a Ghanaian hospital.

Materials and methods:

Ethical considerations

Ethical clearance was obtained from Biomedical Research Ethics Committee of University of KwaZulu-Natal (BE068/15) and the Committee on Human Research, Publication and Ethics of the Kwame Nkrumah University of Science and Technology, Kumasi, Ghana (CHRPE/AP/220/15).

Collection and identification of bacterial isolates

A total of 91 *S. aureus* isolates from different patients were collected from the Komfo Anokye Teaching Hospital in Ghana between May and September 2015. Presumptive staphylococci were identified by colony morphology (cream to golden yellow colour) on blood agar and the coagulase test (slide method) conducted by the Microbiology Unit of the hospital. Isolates were subjected to further tests including mannitol fermentation and the tube coagulase test. The identity of isolates was also confirmed by the automated VITEK 2 system (BioMérieux, Marcy-L'Etoile, France). Fifty-eight isolates were confirmed as MRSA (the cefoxitin screen test) and included for further study.

Detection of MRSA and antibiotic susceptibility profiles

The cefoxitin test was used to screen for MRSA according to CLSI guidelines (15). The MICs for MRSA isolates were determined using the automated VITEK 2 system for the following antibiotics: oxacillin, gentamicin, ciprofloxacin, moxifloxacin, erythromycin, clindamycin, linezolid, teicoplanin, vancomycin, tetracycline, tigecycline, fusidic acid, rifampicin and trimethoprim-sulfamethoxazole, and results interpreted according to CLSI guidelines (15). *S. aureus* ATCC 25923 was used as the reference strain. Inducible clindamycin resistance was tested by the 'D-zone' (16).

DNA extraction, PCR and sequencing of virulence and resistance genes

Genomic DNA was extracted using the Roche High Pure PCR Template Preparation Kit (Roche, Mannheim, Germany) according to the manufacturer's instructions and the concentration and purity were determined by Nanodrop™ 1000 spectrophotometer (Thermo Scientific, USA). The resistance genes *mecA, blaZ, aac-aph, ermC, tetK* conferring resistance to oxacillin, penicillin, aminoglycosides, macrolide-lincosamide-streptogramin B and tetracyclines respectively and the virulence genes *lukS/F-PV, hla, hld* and *eta* encoding Panton-Valentine Leucocidin (PVL), alpha haemolysin, delta haemolysin and exfoliative toxin A respectively were amplified. Amplification of genes was done using T100™ Thermal cycler (Bio-Rad, USA), using primers described elsewhere (17-20) (Table 1).

Genes investigated are those that confer resistance to some of the most commonly prescribed antibiotics in Ghana and virulence genes were selected based on their importance in staphylococcal infections. PCR experiments were carried out using the 2x PCR master mix (ThermoScientific, USA), 1.25 µL each of forward and reverse primers, and 50-200 ng of template DNA in a total reaction volume of 25 µL. PCR products were run on 1.5% agarose gel at 65V for 90 min and visualized by UV transillumination using Bio-Rad ChemiDoc™ MP System (Bio-Rad, UK). The PCR products were sequenced (Inqaba Biotech, Pretoria, South Africa) to confirm the identity of the genes. Analysis of the sequences was done using ChromasPro 1.9.9.1 (Technelysium, Queensland Australia), BioEdit and BLAST 2.0 available on the National Center for Biotechnology Information (NCBI) website http://www.ncbi.nhlm.nih.gov/blast/BLAST.cgi.

Table 1: Oligonucleotide primers and cycling conditions for the detection of genes in this study

Gene	Primer sequence	PCR program	Amplicon size (bp)	Reference
mecA	F-AACAGGTGAATTATTAGCACTTGTAAG	3min 95ºC, 30s 55ºC, 1min 72ºC	174	Ref 17
	R-ATTGCTGTTAATATTTTTTGAGTTGAA			
blaZ	F-ACTTCAACACCTGCTGCTTTC	3min 95ºC, 30s 55ºC, 1min 72ºC	173	Ref 17
	R-TGACCACTTTTATCAGCAACC			
tetK	F-TCGATAGGAACAGCAGTA	3min 95ºC, 30s 51ºC, 1min 72ºC	169	Ref 18
	R-CAGCAGATCCTACTCCTT			
aac (6')-aph (2'')	F-TAATCCAAGAGCAATAAGGGC	3min 95ºC, 30s 54ºC, 1min 72ºC	227	Ref 19
	R-GCCACACTATCATAACCACTA			
ermC	F-CTTGTTGATCACGATAATTTCC	3min 95ºC, 30s 52ºC, 1min 72ºC	190	Ref 17
	R-ATCTTTTAGCAAACCCGTATTC			
eta	F-GCAGGTGTTGATTTAGCATT	3min 95ºC, 30s 51ºC, 1min 72ºC	93	Ref 20
	R-AGATGTCCCTATTTTTGCTG			
lukS/F-PV	F-ATCATTAGGTAAAATGTCTGGACATGATCCA	3min 95ºC, 30s 58ºC, 1min 72ºC	443	Ref 20
	R-GCATCAAGTGTATTGGATAGCAAAAGC			
hla	F-CTGATTACTATCCAAGAAATTCGATTG	3min 95ºC, 30s 55ºC, 1min 72ºC	209	Ref 20
	R-CTTTCCAGCCTACTTTTTTATCAGT			
hld	F-AAGAATTTTTATCTTAATTAAGGAAGGAGTG	3min 95ºC, 30s 55ºC, 1min 72ºC	111	Ref 20
	R-TTAGTGAATTTGTTCACTGTGTCGA			

PFGE typing

Strain typing was conducted using pulsed field gel electrophoresis (PFGE) as described by Tenover *et al.,* (21) using contour-clamped homogeneous electric field apparatus (CHEF DR-III; BioRad, Hercules, CA). Restriction was done with the *Sma*I restriction enzyme for *S. aureus*. Analysis of results was done using BioNumerics software version 6.6 (Applied Maths NV, Belgium) using the Dice coefficient and represented by unweighted pair group method with arithmetic mean (UPGMA) with optimization settings and position tolerance set at 0.5% and 1% respectively was used to analyze the electrophoretic patterns. Clusters were defined as described by Tenover *et al*., (21).

Results:

Overall, fifty-eight (63.7%) isolates were putatively methicillin resistant as detected by the phenotypic cefoxitin screen test and oxacillin MICs. The detailed phenotypic and genotypic profiles of MRSA isolates are available in the supplementary material. Thirty-five (60.3%) isolates were from blood, 5 (8.6%) from urethral swabs, 4 (6.9%) from urine and 14 (24.1%) from unknown sources.

Table 2 delineates the percentage resistance to different antibiotics tested. Generally, 37.9% of MRSA isolates displayed resistance to tetracycline, 19% to trimethoprim-sulphamethoxazole, 15.5% to clindamycin, 12.1% to gentamicin, 13.8% to ciprofloxacin and erythromycin, 6.9% to moxifloxacin and 7.0% to rifampicin. None of the MRSA isolates tested positive for inducible clindamycin resistance. All isolates were fully susceptible to linezolid, teicoplanin, vancomycin, tigecycline and fusidic acid.

Table 3 shows the distribution of resistance and virulence genes screened. The prevalence of resistance (*mec*A, *bla*Z, *aac(6')-aph(2'')*, *tet*K and *erm*C) and virulence (*hla* and *lukS/F-PV*) genes respectively were 74%, 33%, 22%, 19%,

3%, 5% and 3%, with isolates organized into two highly related clades. None of the isolates expressed the *hld* and *eta* virulence genes (Fig 1).

Table 2: Antibiotic susceptibility profile of MRSA isolates (n=58)

Antibiotic	No sensitive (%)	No intermediate (%)	No resistant (%)
Oxacillin	0 (0.0)	0 (0.0)	58 (100.0)
Gentamicin	49 (84.5)	2 (3.4)	7 (12.1)
Ciprofloxacin	49 (84.5)	1 (1.7)	8 (13.8)
Moxifloxacin	54 (93.1)	0 (0.0)	4 (6.9)
Erythromycin	49 (84.5)	1 (1.7)	8 (13.8)
Clindamycin	18 (31.0)	31 (53.4)	9 (15.5)
Linezolid	58 (100.0)	0 (0.0)	0 (0.0)
Teicoplanin	58 (100.0)	0 (0.0)	0 (0.0)
Vancomycin	58 (100.0)	0 (0.0)	0 (0.0)
Tetracycline	36 (62.1)	0 (0.0)	22 (37.9)
Tigecycline	58 (100.0)	0 (0.0)	0 (0.0)
Fusidic acid*	15 (26.3)	42 (73.7)	0 (0.0)
Rifampicin*	37 (65.0)	16 (28.0)	4 (7.0)
Trimethoprim/sulphamethoxazole	46 (79.3)	1 (1.7)	11 (19.0)

*n=57 as MICs could not be determined for 1 isolate each

Table 3: Distribution of virulence and resistance genes (%) in MRSA strains

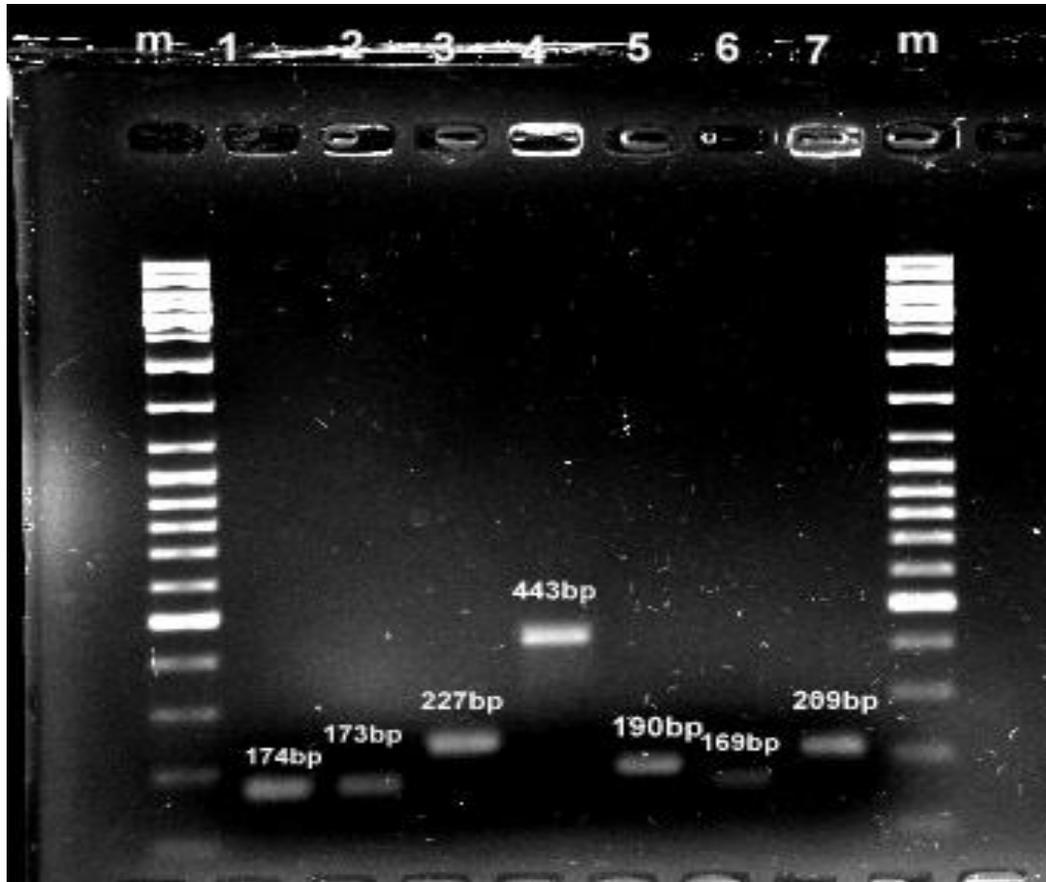| Gene	%
MecA	74
BlaZ	33
ErmC	3
aac(6')-aph(2'')	22
TetK	19
Hla	5
Hld	0
Eta	0
lukS/F-PV	3

Fig 1: PCR mapping

Lanes M: DNA molecular marker, Lane 1: *mecA*, Lane 2: *blaZ*, Lane 3: *aac(6')-aph(2'')*, Lane 4*: lukS/F-PV*,
Lane 5: *ermC*, Lane 6: *tetK*, Lane 7: *hla*

Three single nucleotide polymorphisms (SNPs) were observed for isolate S15, in the *hla* gene (C→T [12], T→A [15] and T→C [204]) as well as an insertion of G [8] and an SNP (G→C [429]) in the *lukS/F-PV* gene (KY056259). A missense mutation of N41S was observed in the *hla* protein (compared with the *hla* protein of *S. aureus* gb|AIG51324.1) Two SNPs (G→A [10] and A→C [111]) were also observed in the *blaZ* gene for isolate S31. A missense mutation of N98T was present in the *blaZ* protein (compared with the *blaZ* protein of *S. aureus* gb|ACP40660.1). Two putatively novel nucleotide sequences for *hla* and *blaZ* genes were submitted to GenBank and the following accession numbers were assigned; KY056259 (*hla*) and KY056260 (*blaZ*). Detailed report of sequence analysis can be found in supplementary material at www.afrjcem.org

PFGE analysis showed two major clades (A and B) of highly related isolates. The most resistant isolates against the most commonly prescribed antibiotics (i.e. gentamicin, ciprofloxacin, erythromycin, clindamycin, tetracycline and trimethoprim-sulfamethoxazole) belonged to clade B type (Fig 2).
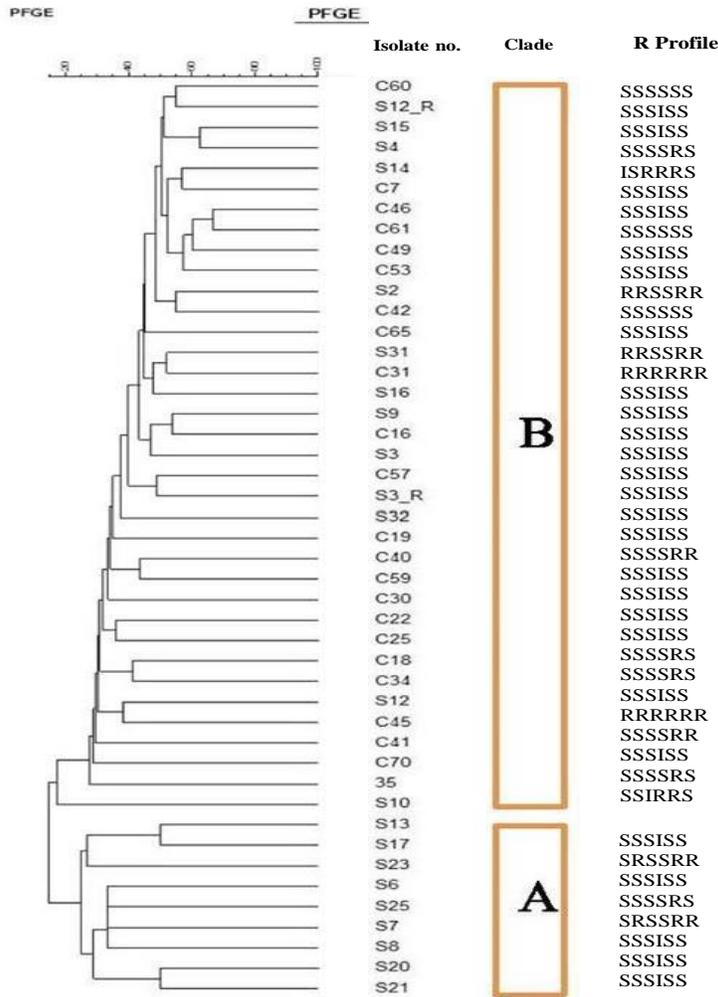
Fig 2: PFGE *SmaI* genotypic types generated from clinical MRSA isolates

The S, I and R (susceptible, intermediate and resistant) indicate resistance or susceptibility to gentamicin, ciprofloxacin, erythromycin, clindamycin, tetracycline and trimethoprim/sulfamethoxazole respectively

Discussion:

This study reports on the prevalence of MRSA from a Ghanaian hospital, with 58 (63%) of the 91 *S. aureus* isolates phenotypically methicillin or oxacillin resistant, and 43 (74%) were genotypically confirmed as MRSA. This figure is relatively high compared to the prevalence of 5.7% (11) in a Ghana study. However studies in Ethiopia, Egypt and Algeria showed an MRSA prevalence of 55.0%, 52.0% and 45.0% respectively in clinical isolates (22).

The use of tetracycline is common in Ghanaian communities, based on anecdotal evidence and may be obtained over-the-counter, which may partly explain the high resistance to this antibiotic. The resistance level to trimethoprim-sulphamethoxazole was lower in this study compared to resistance levels found in studies in Nigeria (90.9%) (9), Tanzania (50.0%) (23), and Ghana (58.3%) (24). However Egyir *et al* in 2015 reported a low level of resistance (7.0%) to trimethoprim-sulphamethoxazole in Ghana (25). Hence, MRSA from Ghana are likely to be susceptible to trimethoprim-sulphamethoxazole than those from these other countries. None of the isolates was resistant to all antibiotics tested and the

susceptibility of all isolates to linezolid, teicoplanin, tigecycline, fusidic acid and vancomycin shows that these drugs remain tenable alternatives in the treatment of MRSA infections in Ghana. However, the high costs and limited availability of these drugs in Ghana means treatment options for MRSA infections are still limited in Ghana.

Furthermore, the relatively high susceptibility levels of MRSA isolates to the quinolones, ciprofloxacin and moxifloxacin, erythromycin, gentamicin and trimethoprim-sulphamethoxazole indicates that these agents may be relied upon when the first line anti-MRSA antibiotics are not immediately available.

Of the 58 isolates identified as MRSA by phenotypic tests (disc diffusion and MIC), 43 (74.0%) carried the *mecA* gene. Methicillin resistance in staphylococcal strains that do not carry the *mecA* gene may be linked with another mechanism of methicillin resistance such as changes in affinity of penicillin-binding proteins for oxacillin (15). In addition, there are other chromosomally determined components aside *mecA* that are implicated in methicillin resistance. For example, *FemA* and *FemB,* which encode proteins involved in the formation of the pentaglycine side chain of peptidoglycan, influence the methicillin resistance expression level in *S. aureus* (26). Again, methicillin resistance in the *mecA*-negative isolates could be mediated by the *mecC* or *mecB* genes (27).

The frequency of *tetK* gene found in this study (19.0%) is close to that reported in another study in Ghana (16.3%) (28). Tetracycline resistance determinants are common and are usually found in multidrug resistant bacteria (19). *TetK* mediates resistance to tetracycline but not to minocycline, and is a more widespread tetracycline resistance gene than *tetL* and *tetO* which are uncommon (29). The *tetK* and *tetM* genes code for ribosomal target site alteration and/or efflux mechanism, limiting the effectiveness of the drug. The observation that some isolates were resistant and yet showed no *tetK* genes upon genotypic screening could be due to fact that their resistance to tetracycline may be mediated by other tetracycline resistant genes like *tetM*, which was not investigated in this study. Resistance to tetracycline is frequently due to the acquisition of new genes related to either conjugative plasmids or transposons (19). Some isolates were susceptible to tetracycline by phenotypic testing although they possessed the *tetK* gene, which suggests that the gene may be present but silent or minimally expressed in the absence of selection pressure. This assertion appears more likely given that the use of tetracycline for *S. aureus* infections in hospitals is on the decline in Ghanaian hospitals.

Of the eight isolates resistant to erythromycin and clindamycin (by phenotypic screening), only two possessed *ermC* gene upon molecular analysis. Resistance to erythromycin and clindamycin in the isolates that did not have *ermC* may be alternatively mediated by the *msrA* and *linA* genes respectively. *MrsA* encodes an efflux pump that actively pumps macrolides from the bacterial cell before they can bind their ribosomal target site. This mechanism however does not create resistance to lincosamides (16). *MsrA and linA* genes were not investigated in this study.

Clindamycin may be useful even in HA-MRSA when susceptibility testing shows it has activity, given the limited options for MRSA infections in Ghana. However, one major concern regarding the use of clindamycin in MRSA infections is the possible development of resistance in the presence of macrolide inducers such as erythromycin (30). This has resulted in some clinicians avoiding or abandoning the use of clindamycin in staphylococcal infections whenever there is resistance to erythromycin. The 'D-zone' test therefore distinguishes strains with intrinsic genetic possibility of resistance from those that are fully susceptible to clindamycin. None of the isolates tested was positive for inducible clindamycin resistance. Those that did not possess the clindamycin

resistance genes and were susceptible to clindamycin by susceptibility testing can therefore be safely reported as susceptible to clindamycin.

The prevalence of the *aac(6')-aph (2")* gene found in this study was 22.0%. Report of a previous study in Ghana showed a prevalence of 17.0% for this gene (25). The presence of the *aac(6')-aph(2")* gene in some isolates that showed no resistance to gentamicin implies the gene is not sufficiently expressed in these isolates. Again, some isolates carrying the *aac(6')-aph(2")* gene expressed intermediate susceptibility towards gentamicin and the borderline susceptibility in these isolates may be ascribed to the expression of this gene.

LukS/F-PV gene, detected in two (3.0%) of the isolates is commonly associated with community-acquired MRSA (31). Genes encoding PVL, which is a cytotoxin that causes destruction of leucocytes and tissue necrosis, were not frequently encountered until recently, being produced by less than 5% of *S. aureus* strains worldwide. The frequency of PVL-positive *S. aureus* strains associated with clinical infections in England and Wales was reported to be 1.6% by Holmes *et al* (32), whiles a study in South Africa found it to be 0.3% (12). It is not therefore surprising that just two isolates carried the gene for PVL considering they are clinical isolates. Studies in Ghana however showed PVL prevalence rates of 17.0% (5/30) and 63.8% among hospital *S. aureus* isolates (25, 28). Although PVL genes are mostly associated with community strains of MRSA, data from West and Central Africa showed that no less than 40.0% of clinical methicillin susceptible *S. aureus* (MSSA) isolates in these regions are PVL-positive (7, 13). Thus, the acquisition of *mecA* gene by PVL-positive MSSA and the potential spread of PVL-positive CA-MRSA could pose a challenge in the control of infections in countries with inadequate resources in Africa. There was an insertion of G (8) and an SNP (G→C [429]) in the *lukS/F-PV* gene.

Alpha haemolysin is encoded by *hla*

was found in 5% of the isolates and plays an important role in staphylococcal infections (20). This is in contrast to reports from Uganda (14), United States (33) and Iran (20) where the frequency of *hla* recorded were 100.0%, 100.0% and 93.15% respectively, which suggests that the geographical distribution of this gene may be lower in Ghana. Alpha haemolysin, when secreted by *S. aureus*, integrates into host cell membranes and causes lysis of eukaryotic cells, with erythrocytes being particularly susceptible (34).

None of the isolates harbored the gene for the exfoliative toxin, *eta*. This is consistent with data from another study in Ghana (25). Three SNPs were observed for isolate S15, viz, in the *hla* gene (C→T [12], T→A [15] and T→C [204]). A missense mutation of N41S was observed in the *hla* protein (compared with the *hla* protein of S. aureus gb|AIG51324.1). The effects of SNPs at different positions of the codon may affect the expression and functioning of this gene. Alpha haemolysin hyperproduction in *S. aureus* is a multifactorial process affected at both the genomic and transcriptional levels (35). A study conducted by Liang *et al.,* in 2011 identified SNPs in the *hla* gene at positions 2376, 2483 and 2484 from the start codon, associated with alpha toxin hyper-production (35).

The most resistant isolates against the most commonly prescribed antibiotics such as gentamicin, ciprofloxacin, erythromycin, clindamycin, tetracycline and trimethoprim-sulfamethoxazole belonged to clade B type. Detailed patient demographic data was unavailable, making the discussion of clonal relatedness as well as hospital-associated and community-acquired comparisons challenging.

Conclusion:

In conclusion, this study provides information on the occurrence of MRSA from a major referral hospital in Ghana over a 5-month period. Results indicated a fairly high occurrence of MRSA. Some

isolates carried the *mecA, blaZ, tetK, aac(6')-aph(2''), ermC, lukS/F-PV* and *hla* genes. The high occurrence of MRSA found in this study is worrying, considering that there are limited treatment options for antibiotic-resistant *S. aureus* in Ghana. There is an urgent need to institute effective surveillance mechanisms to monitor MRSA and implement stringent infection prevention and control programmes to forestall its spread.

Conflict of interest:

Acknowledgments:

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