**Prevalence of mecA gene in methicillin-resistant Staphylococcus aureus strains isolated at the National Public Health Laboratory of Brazzaville, Congo**

**Abstract**

**Aims:** The study aims to assess the prevalence of the mecA gene in Methicillin-resistant *Staphylococcus aureus* (MRSA) strains. **Background**: MRSA is currently a serious global public health problem, responsible for both nosocomial and community-acquired infections. This resistance is due to the acquisition of the mecA gene, carried by a mobile gene cassette. **Medthod**: This study examined 120 Staphylococcus aureus isolates collected from urine samples, suppurations, and vaginal swabs between January and June 2021 at the Laboratoire National de Santé Publique in Brazzaville's bacteriology department. The cefoxitin disk diffusion test was used to detect MRSA phenotypes, while PCR was used to amplify the mecA gene. **Results**: The results revealed that 54.2 % of the strains were resistant to cefoxitin, a phenotypic marker for MRSA. The interpretation of the antibiogram showed that these strains were also highly resistant to several other antibiotics, such as kanamycin (69.7%), tobramycin (65.2%), gentamicin (65.2%) and erythromycin (62.1%).). Pristinamycin, rifampicin and lincomycin proved to be potential therapeutic alternatives because isolatedstrains presented low resistance to these antibiotic molecules with respective rates of 4.6%, 13.7% and 15.2%. The PCR results showed that 47.0% of these isolated strains carried the mecA gene, revealing a discordance with the phenotypic detection. **Conclusion** Although there is this discrepancy between the phenotypic and genotypic detection of MRSA, the high percentage of MRSA and the detection of the mecA gene could representa serious public health concern that requires increased surveillance and refined diagnostic strategies. Therefore, the acquisition of the mecA gene, which confers resistance to methicillin, enables Staphylococcus aureus (MRSA) to persist and potentially facilitates the spread of MRSA epidemics through the accumulation of additional virulence factors.

Keywords: *Staphylococcus aureus*, cefoxitin, MRSA, gene mecA, antibiotic

**1. Introduction**

*Staphylococcus aureus* is a commonbacterium found on the skin and nasal mucosa of humans and animals and is one of the main etiological agents responsible for superficial and deep suppurative infections and toxin-related syndromes [1]. This can lead to a variety of local and invasive problems, ranging from superficial skin infections to life-threatening pneumonia and bacillus infections. *Staphylococcus aureus* infections have been occurring in humans since ancient times [29]. It has a great capacity to acquire and express a wide range of virulence and antibiotic resistance factors [1, 2]. This resistance is acquired by the insertion of a mobile genetic element called SCCmec (Staphylococcal Cassette Chromosome mec) containing the mecA gene, characterised by low affinity for ß-lactamins[3, 4]. Therefore, the acquisition of the mecA gene conferring resistance to meticillin to S. aureus (MRSA) may promote the spread of MRSA epidemic through the acquisition of additional virulence factors [1]. MRSA is highly pathogenic, capable of causing severe progressive and necrotizing diseases, including bacteremia, sepsis, and toxic shock syndrome [[3](https://www.mdpi.com/2076-2607/12/1/148#B3-microorganisms-12-00148)0,31].  In fact, several epidemic and pandemic MRSA clones are expanding both in hospitals and in the community, in some cases creating new clinical syndromes and even severe infections, such as necrotising pneumonia [5, 6]. Furthermore, the emergence of vancomycin-resistant strains, once considered the antibiotic of last resort, considerably complicates the treatment of MRSA infections. This has led to the emergence of multi-resistant strains, dubbed 'superbacteria', making S. aureus infections particularly difficult to combat [7, 8, 9]. The alarming increase in antibiotic resistance to MRSAposes a growing threat to public health. Indeed, the prevalence of MRSA worldwide is highly heterogeneous and varies significantly by country and region, study period, services and living conditions of the populations concerned [10]. In the United States, approximately 1.5% of the population is infected with *S. aureus* and MRSA [8]. Approximately 478,000 hospitalizations have been associated with *S. aureus* infections [11,12]. In Africa, variability has been observed, with a high prevalence of MRSA in Togo in the early 2000 [13]. In the Republic of Congo, community-acquired MRSA infections tend to be widespread, with rates approaching 40 % [14]. The aim of our study was to assess the prevalence of the mecA gene in MRSA strains.

**2. Material and methods**

**2.1. Material**

It consisted of 120 isolates of *Staphylococcus aureus* isolated from urine samples, suppurations, and vaginal swabs over the period from January to June 2021 at the bacteriology department of the Laboratoire National de Santé Publique in Brazzaville.

**2.2 Methods**

**2.2.1. Isolation and Identification**

The bacteria were isolated by the culture of the biological product on mannitol salt agar medium (Bio-Rad), for 18 to 24 hours at 37 ° C. For identification, Gram staining was performed on suspected colonies to assess the morphological and staining characteristics. The catalase test was used to characterize the *Staphylococcus* genus, as well as the coagulase test for differentiation between *S. aureus* and other bacteria. The probable presence of *Staphylococcus aureus* was confirmed using the API® Staph gallery (Bio Mérieux) according to the manufacturer's recommendations.

**2.2.2. Antibiotic susceptibility testing**

Antibiotic susceptibility testing was carried out using the agar diffusion technique according to the recommendations of the Antibiogram Committee of the French Society of Microbiology (CA-SFM, 2022). The inoculum was prepared by suspending a well-isolated colony of a pure bacterial culture (18 to 24 hours on agar medium) in 5 ml of normal saline (0.9% NaCl), and the suspension turbidity was adjusted to 0.5 Mac Farland using Vitex Densichek. The swab technique was used to inoculate the suspension on Mueller-Hinton agar medium, as recommended by CA-SFM, 2022. Antibiotic discs were then applied to the inoculated Mueller Hinton agar medium. The plates were incubated at 37°C for 18-24 h. The diameters of the zones of bacterial growth inhibition around the disc after incubation were measured and interpreted on the basis of the threshold values published by CA-SFM, 2022 ( <https://www.sfm-microbiologie.org>). These values were transcribed into categories of susceptible (S) and resistant (R). Phenotypic detection of methicillin resistance in isolates was performed using the cefoxitin disc (30 µg). Following verification of methicillin-resistance, another sensitivity test was performed on MRSA strains using the following antibiotics: Kanamycin (K,30μg), tobramycin (T,30μg), gentamycin (G,15μg), Erythromycin (E,15UI), lincomycin (L,15μg), pristinamycin (PT,15μg), fusidic Acid (FC,10μg), Rifampicin (RD, 5μg). The reference strain of Staphylococcus aureus ATCC 29213 was used for quality control according to CASFM recommendations.

**2.2.3 Genotypic detection of the mecA gene**

**2.2.3.1. Extraction**

DNA from metillo-resistant strains was extracted using the Nucleospin DNA kit following the recommendations of the MACHEREY NAGEL protocol.

**2.2.3 2. Amplification of the mecA gene**

A 310-bp fragment of the mecA gene was amplified using primers mecA-F: 5'- AAAATCGATGGTAAAGGTTGGC-3' and mecA-R: 5' AGTTCTGCAGTACCGGATTTGC-3' [14]. The 50 µl amplification reaction mix contained 2 µl DNA template and 48 µl master mix. The master mix included 31.75 µl sterile distilled water, 2 µl forwad primer (F), 2 µl reverse primer (R), 2 µl dNTP, 10 µl PCR buffer (x5) and 0.25 µl Taq polymerase. Amplification conditions were as follows: initial denaturation at 94°C for 5 min, 30 cycles of denaturation at 94°C for 30 seconds, hybridization at 54°C for 30 seconds, elongation at 72°C for 30 seconds and final elongation at 72°C for 7 minutes. Five microliters (5μl) of PCR product were resolved on a 1.5% agarose gel containing 0.5 μg / ml of ethidium bromide in Tris- Borate-EDTA buffer, at 100 V for 40 minutes. For this molecular analysis, a reference strain of MRSA possessing the mecA gene was used as a positive control.

**3. Results**

**3.1. Distribution of strains according to the origin of the specimen**

The study included 120 strains of *S. aureus* isolated from various clinical specimens. The frequency of *S. aureus* isolation was highest in suppurations (58.3%), followed by urine (29.2%) and vaginal swabs (12.5%).

**3.2. Methicillin resistance profile**

Of the one hundred and twenty *S. aureus* strains tested for susceptibility to cefoxitin, sixty-six (54.2%) showed an inhibition diameter of less than 27 mm, an indicator of resistance to methicillin (MRSA). The distribution of MRSA according to sample type showed that the majority (60.6%) were isolated from suppurations, followed by urine (28.8%) and 10.6% vaginal swabs (figure 1).



Figure 1: Frequency of phenotypic detection of MRSA depending on their origin

**3.3. MRSA susceptibility to different antibiotics**

Sixty-six cefoxitin-resistant strains of *Staphylococcus aureus* were subsequently tested with eight different antibiotics. Antibiotic susceptibility test results revealed high levels of resistance. Aminoglycosides were the least active antibiotics, with resistance rates of 69.7% for kanamycin and 65.2% for gentamicin and tobramycin. However, pristinamycin was the most active antibiotic, with a resistance rate of 4.6% (Table 1).

**Table 1: MRSA resistance rates to different antibiotics.**

|  |  |
| --- | --- |
| Antibiotics tested | SARM (N=66)  |
|  R(%) | S (%) |
| Erythromycin (E) | 41(62.1) | 15(37.9) |
| Lincomycin (L) | 10(15.2) | 56(84.8) |
| Pristinamycin (PT) | 3(4.6) | 63(95.4) |
| Kanamycin (K) | 46(69.7) | 20(30.3) |
| Tobramycin (T) | 43(65.2) | 23(34.8) |
| Gentamycin (G) | 43(65.2) | 23(34.8) |
| Fusidique Acid (FA) | 31(47.0) | 35(53.0) |
| Rifampicin (Rf) | 9(13.7) | 57(86.3) |

R: resistance, S: sensitive

**3.4. Prevalence of mecA gene in MRSA strains**

The analysis of 66 isolated and amplified MRSA strains revealed the presence of the mecA gene in 31 of them, corresponding to a prevalence of 47.0 %. It is notable that among these 31 strains carrying the mecA gene, a majority (22 strains) were isolated from suppuration samples (Table 2).

Table 2: Distribution of mecA-positive MRSA according to their origin.

|  |  |  |
| --- | --- | --- |
| Origins |  *SARM* numbers | PCR |
| + (%) | - (%) |
| Suppurations | 40 |  22 (55.0) | 18 (45.0) |
| Urine  | 19 |  7 (36.8) | 12 (63.2) |
| vaginal swabs | 7 | 2 (28.6) | 8 (71.4) |
| Total | 66 | 31 (47.0) | 35 (53.0) |

(-): absence of the mecA gene; (+): presence of the mecA gene; %: Percentage

**4. Discussion**

In this study, 120 strains of *Staphylococcus aureus* were isolated, including 58.3% from suppurations. This result can be explained by the ability of *Staphylococcus* spp. strains to form a suppurative collection, and their classification among pyogenic germs [1]. These results corroborated with those reported by Dendi in Algeria, with a higher frequency of isolation in pus samples [15]. The cefoxitin disk diffusion test revealed that 54.2% of strains were resistant to this antibiotic, an indicator of methicillin resistance. This relatively high prevalence could be attributed to antibiotic selection pressure. This rate of resistance to cefoxitin reported in our study is higher than those observed in Brazil in 2018 with a rate of 23% [16] and in Nigeria where a rate of 46.66% of MRSA was reported from 90 strains of *S.aureus* isolated from clinical samples in Sokoto [17]. However, studies from Iran and Iraq reported MRSA rates ranging from 66 to 100% [18,19]. These differences in results could be explained by several factors, including the size and type of samples, but also the diversity of geographic locations of the studies. It is important to note that the prevalence of MRSA can vary considerably by region, country, year of study and even between different areas within the same country, as shown in numerous published studies [13, 15]. High levels of MRSA, particularly those responsible for bacteremia, lead to major complications in patient management. These are associated with a poorer vital prognosis, longer hospital stays, higher mortality and increased hospital costs [16].

Antibiotic susceptibility testing of the 66 MRSA strains revealed a high level of resistance to the various antibiotics tested, highlighting the presence of multi-resistant isolates and thus limiting therapeutic options. Among the antibiotics tested, aminoglycosides proved the least effective, with resistance rates of 69.7% for kanamycin, 65.2 % for tobramycin and 65.2 % for gentamicin. These rates of resistance to aminoglycosides are lower than those observed in Côte d'Ivoire (100%) [20] (Guessennd et al., 2013), but higher than those reported in Morocco (Elazhari et al., 2010) [21] and Congo (AHOMBO et al., 2019) [14]. This high resistance to aminoglycosides could be linked to the production of enzymes that inactivate aminoglycosides and have a high potential for dissemination, namely APH (3'), ANT (4') and APH (2')-AAC (6'), responsible respectively for the inactivation of kanamycin, (kanamycin and tobramycin) and (kanamycin, tobramycin and gentamicin) and conferring the K, KT and KTG phenotypes [22]. Of the macrolides tested, erythromycin (62.1% of resistant strains) proved the least effective, it is important to note that methicillin resistance is frequently associated with inducible resistance to MLSBs [14, 23]. This finding contrasts with studies of community MRSA [14, 24]. These discrepancies could be attributable to multiple factors such as geographical variations, antibiotic selection pressure, local epidemiology, diversity of resistance mechanisms (target modification, Erm enzymes, efflux) and local antibiotic therapy practices [25]. Our results also highlighted the efficacy of pristinamycin and lincomycin against MRSAstrains, with sensitivity rates of 95.4% and 84.8% respectively. These two antibiotics could represent promising therapeutic alternatives for the treatment of MRSA infections. For fusidic acid and rifampicin, we observed sensitivity rates of 53.0% and 86.3 %, respectively. These results are broadly in line with the literature, which reports sensitivity rates ranging from 40% to 80% [10]. Resistance to these antibiotics could be due to the selective pressure exerted by the strains, as well as inappropriate consumption of antibiotics, including self-medication and non-compliance with dosing regimens. However, it is important to note that our results differ from those reported in Congo by Ahombo, who observed higher rates of resistance to fusidic acid and rifampicin (81.6% and 67.3%, respectively), particularly in community strains of MRSA [14]. This difference could be explained by variations in the populations studied.

Analysis of the PCR results of 66 strains of MRSA revealed the presence of the mecA gene in 31 of them, corresponding to a rate of 46.96%. This mecA gene detection rate is significantly higher than the results obtained in other studies carried out in the region. For example, work carried out in the Republic of Congo reported rates of 30% to 36% [14], while in Nigeria and Algeria, the rates were 33% and 22.7% respectively [15,26]. However, wide variations were observed, with one study in Nigeria reporting a prevalence of 85.7% of MRSA harbouring the mecA gene [17] and other studies reporting the presence of the mecA gene in all MRSA isolates (100%) [19,27]. These results underline the considerable variability in the prevalence of the mecA gene depending on the contexts studied. In our study, we found that only 47.0% of MRSA strains carried the mecA gene~~, and~~ 53.0% of the phenotypically resistant MRSA did not harbour this gene. These results are consistent with studies carried out in other countries, such as Iran, Nigeria and Algeria, which have also reported the presence of MRSA strains lacking the mecA gene [15, 26]. The absence of the mecA gene in 53.0 % of MRSA strains could be explained by the fact that they are Borderline *Staphylococcus aureus* (BORSA). These strains therefore exhibit hypersecretion of penicillinase, leading to hydrolysis of penicillin M. In this case, ~~we observe~~ low resistance, associated with increased production of β-lactamases, particularlyhyperproduction of methicillinase is observed [28].

**Conclusion**

This study identified 120 strains of *Staphylococcus aureus*, 66 of which were resistant to methicillin (MRSA) and multi-resistant to other antibiotics. This multi-resistance considerably complicates the treatment of MRSA infections. However, pristinamycin, rifampicin and lincomycin proved to be potential therapeutic alternatives. A significant discrepancy was observed between the rate of MRSA detected by phenotypic method and the presence of the mecA gene, detected by PCR. This difference highlights the limitations of phenotypic MRSA detection, and underlines the reliability and accuracy of the PCR technique for MRSA isolation. Thus, given the high virulence of MRSA, it is crucial to strengthen molecular detection capabilities in laboratories. The implementation of molecular biology techniques is essential for accurate and rapid diagnosis of MRSA infections, enabling better control of their spread. Finally, it's important to note that the presence of the mecA gene can influence the antibiotic susceptibility profile of *Staphylococcus aureus* strains, contributing to an increase in antibiotic resistance. This phenomenon is exacerbated by the inappropriate use of antibiotics and the hand-carried transmission of bacteria.

**References**

1. Yehia, F. A. A., Yousef, N., & Askoura, M. (2021). Exploring *Staphylococcus aureus* Virulence Factors; Special Emphasis on Staphyloxanthin. *Microbiology and Biotechnology Letters*. <https://doi.org/10.48022/mbl.2107.07008>
2. Shore, A. C., Rossney, A. S., O’Connell, B., Herra, C. M., Sullivan, D. J., Humphreys, H., & Coleman, D. C. (2008). Detection of Staphylococcal Cassette Chromosome *mec* -Associated DNA Segments in Multiresistant Methicillin-Susceptible *Staphylococcus aureus* (MSSA) and Identification of *Staphylococcus epidermidis ccrAB4* in both Methicillin-Resistant *S. aureus* and MSSA. *Antimicrobial Agents and Chemotherapy*, *52*(12), 4407‑4419. <https://doi.org/10.1128/AAC.00447-08>
3. Santiago, C., Pang, E. L., Lim, K.-H., Loh, H.-S., & Ting, K. N. (2015). Inhibition of penicillin-binding protein 2a (PBP2a) in methicillin resistant Staphylococcus aureus (MRSA) by combination of ampicillin and a bioactive fraction from Duabanga grandiflora. *BMC Complementary and Alternative Medicine*, *15*(1), 178. <https://doi.org/10.1186/s12906-015-0699-z>
4. Rafif Khairullah, A., Rehman, S., Agus Sudjarwo, S., Helmi Effendi, M., Chasyer Ramandinianto, S., Aega Gololodo, M., Widodo, A., Hendriana Priscilia Riwu, K., & Ayu Kurniawati, D. (2022). Detection of mecA gene and methicillin-resistant Staphylococcus aureus (MRSA) isolated from milk and risk factors from farms in Probolinggo, Indonesia. *F1000Research*, *11*, 722. <https://doi.org/10.12688/f1000research.122225.3>
5. Dauwalder, O., Lina, G., Durand, G., Bes, M., Meugnier, H., Jarlier, V., Coignard, B., Vandenesch, F., Etienne, J., & Laurent, F. (2008). Epidemiology of Invasive Methicillin-Resistant *Staphylococcus aureus* Clones Collected in France in 2006 and 2007. *Journal of Clinical Microbiology*, *46*(10), 3454‑3458. <https://doi.org/10.1128/JCM.01050-08>
6. Algammal, A. M., Hetta, H. F., Elkelish, A., Alkhalifah, D. H. H., Hozzein, W. N., Batiha, G. E.-S., El Nahhas, N., & Mabrok, M. A. (2020). Methicillin-Resistant Staphylococcus aureus (MRSA) : One Health Perspective Approach to the Bacterium Epidemiology, Virulence Factors, Antibiotic-Resistance, and Zoonotic Impact. *Infection and Drug Resistance*, *Volume 13*, 3255‑3265. <https://doi.org/10.2147/IDR.S272733>
7. Foster, T. J. (2004). The Staphylococcus aureus “superbug”. *Journal of Clinical Investigation*, *114*(12), 1693‑1696. <https://doi.org/10.1172/JCI200423825>
8. Gorwitz, R. J., Kruszon‐Moran, D., McAllister, S. K., McQuillan, G., McDougal, L. K., Fosheim, G. E., Jensen, B. J., Killgore, G., Tenover, F. C., & Kuehnert, M. J. (2008). Changes in the Prevalence of Nasal Colonization with *Staphylococcus aureus* in the United States, 2001–2004. *The Journal of Infectious Diseases*, *197*(9), 1226‑1234. <https://doi.org/10.1086/533494>
9. Turner, N. A., Sharma-Kuinkel, B. K., Maskarinec, S. A., Eichenberger, E. M., Shah, P. P., Carugati, M., Holland, T. L., & Fowler, V. G. (2019). Methicillin-resistant Staphylococcus aureus : An overview of basic and clinical research. *Nature Reviews Microbiology*, *17*(4), 203‑218. <https://doi.org/10.1038/s41579-018-0147-4>
10. Forestier, E., Rémy, V., Mohseni-Zadeh, M., Lesens, O., Jauhlac, B., Christmann, D., & Hansmann, Y. (2007). Methicillin-resistant Staphylococcus aureus bacteremia: Recent epidemiological and therapeutic aspects. La Revue de Médecine Interne, 28(11), 746-755. <https://doi.org/10.1016/j.revmed.2006.11.014>
11. Klein, E., Smith, D. L., & Laxminarayan, R. (2007). Hospitalizations and Deaths Caused by Methicillin-Resistant *Staphylococcus aureus* , United States, 1999–2005. *Emerging Infectious Diseases*, *13*(12), 1840‑1846. <https://doi.org/10.3201/eid1312.070629>
12. Lakhundi, S., & Zhang, K. (2018). Methicillin-Resistant Staphylococcus aureus : Molecular Characterization, Evolution, and Epidemiology. *Clinical Microbiology Reviews*, *31*(4), e00020-18. <https://doi.org/10.1128/CMR.00020-18>
13. Da, L., Somé, D., Yehouenou, C., Somé, C., Zoungrana, J., Ouédraogo, A.-S., Lienhardt, C., & Poda, A. (2023). État des lieux de la résistance aux antibiotiques en Afrique subsaharienne. *Médecine et Maladies Infectieuses Formation*, *2*(1), 3‑12. <https://doi.org/10.1016/j.mmifmc.2023.01.003>
14. Gabriel, A., Rachel, M., Tarcisse, B. N., Christian, K. A., & Esther, O. N. N. (2019). Genetic Determinism in Community Staphylococcus and Methicillin-Resistant Clinics in Brazzaville, Republic of Congo. *International Journal of Innovative Research in Medical Science*, *4*(07). https://doi.org/10.23958/ijirms/vol04-i07/707
15. Dendi, F. Z., Allem, R., Sebaihia, M., Bensefia, S., Cheurfa, M., Alamir, H., & Obeagu, E. I. (2024). Prevalence and molecular detection of Staphylococcus aureus resistance to antibiotics. *Medicine*, *103*(24), e38562. <https://doi.org/10.1097/MD.0000000000038562>
16. Rocchetti, T. T., Martins, K. B., Martins, P. Y. F., Oliveira, R. A. D., Mondelli, A. L., Fortaleza, C. M. C. B., & Cunha, M. D. L. R. D. S. D. (2018). Detection of the mec A gene and identification of Staphylococcus directly from blood culture bottles by multiplex polymerase chain reaction. *The Brazilian Journal of Infectious Diseases*, *22*(2), 99‑105. <https://doi.org/10.1016/j.bjid.2018.02.006>
17. Umar, A. I., Manga, S. B., Baki, A. S., Uba, A., Dabai, A. I., & Department of Microbiology, Faculty of Science, Usmanu Danfodiyo University Sokoto, Nigeria. (2023). Molecular Detection of mecA gene among Methicillin Resistant Staphylococcus aureus Isolates from Clinical Samples in Sokoto, Nigeria. *International Journal Of Pharmaceutical And Bio-Medical Science*, *03*(02). <https://doi.org/10.47191/ijpbms/v3-i2-03>
18. Zahra Babaei1, Monir Doudi , Ladan Rahimzadeh Torabi. (2023). The Molecular Investigation of the *mecA* Gene and Antibiotic Susceptibility Pattern of *Staphylococcus aureus* and *Staphylococcus epidermidis* Isolated from Patients with Immune System Disorders at Omid Hospital, Isfahan, Iran . Avicenna Journal of Clinical Microbiology and Infection ; 10(3):112-119. doi:10.34172/ajcmi.3458
19. Khalil, W. L., & Al-Hayanni, H. S. (2024). Molecular Detection of the mecA and some Virulence Determinants in Methicillin-Resistant Staphylococcus aureus. *Journal of the Faculty of Medicine Baghdad*, *66*(2), 247‑253. https://doi.org/10.32007/jfacmedbagdad.6622282
20. Guessennd, N., Ouattara, M., Ouattara, N., Nevry, R., Gbanon, V., Tiekoura, K., Dosso, M., & Ger, B. (2013). Study of multidrug-resistant bacteria in hospital effluents from a university hospital (CHU) in the city of Abidjan (Côte d’Ivoire). Journal of Applied Biosciences, 69(0), 5456. <https://doi.org/10.4314/jab.v69i0.95071>
21. Elazhari M., Zerouali K., Elhabchi D., Cohen N., El malki A., Dersi N., Hassar M., Timinouni M., Saile R., 2010. Antibiotitic susceptibility of *Staphylococcus aureus* strains in the community of Casablanca (Marocco). *Revu Tunisienne d’infect*; 4(4): 134-140.
22. Cabrera-Contreras, R., Santamaría, R. I., Bustos, P., Martínez-Flores, I., Meléndez-Herrada, E., Morelos-Ramírez, R., Barbosa-Amezcua, M., González-Covarrubias, V., Silva-Herzog, E., Soberón, X., & González, V. (2019). Genomic diversity of prevalent *Staphylococcus epidermidis* multidrug-resistant strains isolated from a Children’s Hospital in México City in an eight-years survey. *PeerJ*, *7*, e8068. <https://doi.org/10.7717/peerj.8068>
23. Ghanbari, F., Ghajavand, H., Havaei, R., Jami, M.-S., Khademi, F., Heydari, L., Shahin, M., & Havaei, S. (2016). Distribution of erm genes among Staphylococcus aureus isolates with inducible resistance to clindamycin in Isfahan, Iran. *Advanced Biomedical Research*, *5*(1), 62. <https://doi.org/10.4103/2277-9175.179184>
24. Ghamba P., Mangoro Z. et Waza D., 2012. Reoccurrence and distribution of methicillinresistant *Staphylococcus aureus* (MRSA) in clinical specimens in Bauchi, North eastern Nigeria. Jrl of Med and Medical Sci ; 3(8): 506-511
25. Ngoulou, T. B., Ahombo, G., Nguimbi, E., & Ampa, R. (2019). Molecular characterization and distribution of genes encoding resistance to Macrolides, Lincosamides and Streptogramins B in community and clinical Staphylococcus in Brazzaville, Congo. Africa SCIENCE 15(5) 352 – 363.
26. Musa, I., Saadu, M., & Jibril, F. (2022). Antibiogram and detection of mecA gene among MRSA at Specialist Hospital Sokoto. *Microbes and Infectious Diseases*, *0*(0), 0‑0. <https://doi.org/10.21608/mid.2022.150608.1350>
27. Mohammed, M. M., Isa, M. A., Abubakar, M. B., Dikwa, A. S. B., & Kappo, A. P. (2025). Molecular detection of mecA gene from methicillin-resistant Staphylococcus aureus isolated from clinical and environmental samples and its potential inhibition by phytochemicals using in vitro and in silico approach. *In Silico Pharmacology*, *13*(1), 26. <https://doi.org/10.1007/s40203-024-00297-y>
28. Sawhney, S. S., Ransom, E. M., Wallace, M. A., Reich, P. J., Dantas, G., & Burnham, C.-A. D. (2022). Comparative Genomics of Borderline Oxacillin-Resistant Staphylococcus aureus Detected during a Pseudo-outbreak of Methicillin-Resistant S. aureus in a Neonatal Intensive Care Unit. *mBio*, *13*(1), e03196-21. <https://doi.org/10.1128/mbio.03196-21>
29. Abdelgader LMA, Habiballuh AMS, Altoum AZA, Mahjaf GM, Hakim NAM, Altaher TAA, Gorish BMT. Prevalence of Methicillin-Resistant Staphylococcus aureus in Nasal Cavity of Medical Students at Shendi University, Sudan. S. Asian J. Res. Microbiol. [Internet]. 2024 Feb. 3 [cited 2025 Apr. 28];18(2):9-14. Available from: <https://journalsajrm.com/index.php/SAJRM/article/view/344>
30. Xing, A., Ng, H. M., Jiao, H., Li, K., & Ye, Q. (2024). The Prevalence, Epidemiological, and Molecular Characterization of Methicillin-Resistant Staphylococcus aureus (MRSA) in Macau (2017–2022). *Microorganisms*, *12*(1), 148.
31. Hassoun, A., Linden, P. K., & Friedman, B. (2017). Incidence, prevalence, and management of MRSA bacteremia across patient populations—a review of recent developments in MRSA management and treatment. *Critical care*, *21*, 1-10.