Ibn Al-Haitham Journal for Pure and Applied Sciences
Journal homepage: jih.uobaghdad.edu.iq
PISSN: 1609-4042, EISSN: 2521-3407
IHJPAS. 2026; 39(1)

Molecular Detection of Teichoplanin Associated Genes in *Staphylococcus aureus*

Massara Abbas Hameed [1*], Rasmiya Abd Aburesha [2]
[1,2]Department of Biology, College of Sciences, University of Baghdad, Baghdad, Iraq
*Corresponding Author

Received: 23/May/2025.
Accepted: 7/September/2025
Published: 20/January/2026
doi.org/10.30526/39.1.4207

Abstract

Staphylococcus aureus is one of the causes of serious diseases that cause a wide range of infections, including urinary, skin, and respiratory infections. Thus, it has shown strains resistant to antibiotics, so that studying the molecular basis of virulence in staphylococcal bacteria becomes more important. The aim of this study is to investigate the presence of teicoplanin-associated genes (A, B, R) in *S. aureus* bacteria taken from clinical samples by PCR technique. 25 clinical samples from patients with respiratory diseases, skin ulcers, and urinary tract infections were collected from Yarmouk Teaching Hospital for a period ranging from October 2024 to January 2025. The presence of *S. aureus* in these samples has been identified by using standard biochemical methods and confirmed by the Vitek system. The resistance results showed that the highest percentage of resistance to the antibiotic was in benzylpenicillin (100%) and oxacillin (100%), and the lowest percentage of resistance was in the antibiotic (moxifloxacin 0%, linezolid 0%, tigecycline 0%, inducible clindamycin 0%, and trimethoprim 0%). purified the genomic DNA that was done by amplifying the polymerase chain reaction by specific prefixes of the target teicoplanin-associated genes (*tca*). The results indicated that all 25 isolates proved the presence of all *tcaA*, *tcaB*, and *tcaR* genes, and this was confirmed by gel electrophoresis. The relationship between the presence of genes with antibiotic resistance was detected; the result was shown especially for glycopeptides (vancomycin and teicoplanin).

Keywords: Clinical isolates, Molecular detection, PCR, *Staphylococcus aureus*, Virulence genes.

1. Introduction

Staphylococcus aureus is a Gram-positive bacterium found on human and animal skin and mucous membranes. It contributes significantly to hospital-acquired infections and is a leading contributor to illnesses ranging from minor skin conditions to life-threatening diseases like sepsis, pneumonia, and endocarditis [1–4]. This bacterium, Staphylococcus aureus, has a high pathogenic potential due to its ability to produce a broad range of virulence factors, including adhesives, immune evasion proteins, and various toxins, which facilitate its survival and proliferation within the host [5–8]. The emergence of antibiotic-resistant strains, particularly methicillin-resistant *S. aureus*, has represented a significant challenge to global public health [9,10]. *S. aureus* is associated with increased mortality and morbidity rates and increased hospitalizations, and thus health care costs will increase [11]. Due to the ability of staphylococci to resist multiple antibiotics, increasing attention has been directed toward understanding their virulence mechanisms, with the aim of identifying potential therapeutic targets [10]. These genes (*tcaA*, *tcaB*, and *tcaR*) have been implicated in enhancing the virulence of *S. aureus*, contributing to its ability to cause various infections. These genes participate in various cellular activities such as the production of biofilms, the release of toxins, and the immune evasion, as they play an

important role in the survival and development of bacteria inside the host [12]. Since *S. aureus* bacteria were able to escape the host's immunity and colonize surfaces and resist antimicrobials by producing biofilms, which is considered an important virulence trait [13,14]. Molecular work on the following genes (*tcaA, tcaB,* and *tcaR*) has become a valuable tool for elucidating bacterial virulence mechanisms and guiding the development of targeted antibiotics [12]. Molecular identification methods were used to swiftly and specifically identify the necessary genes in these bacterial samples that were diagnosed by the polymerase chain reaction (PCR) [15,16]. Other molecular techniques for evaluating the expression of regulatory genes related to antibiotic resistance Scientists were able to pinpoint the underlying genetic components using this method [17]. The infection caused by these bacteria can be identified by finding the virulence genes (*tcaA, tcaB, tcaR*) and [15,16]. The *tcaRAB* operon is essential for the proper function of the *tcaA, tcaB,* and *tcaR* genes, which have been associated with antibiotic resistance—particularly to teicoplanin, biofilm formation, and immune evasion [18,19]. The *tcaA* modifies the bacterial cell wall to reduce the binding of teicoplanin to peptidoglycan chains, while simultaneously *tcaB* facilitates the production of proteins that help bacteria combat the effects of antibiotics [20]. The expression and overexpression of *tcaA, the tcaB* are regulated by *tcaR*. Resistance teicoplanin may develop. The consistent presence of these genes in clinical specimens highlights their importance in the pathogenesis and survival of *S. aureus* [1]. One of the primary mechanisms behind the failure of antibiotic therapy in *S. aureus* is the production of persister cells, which are transiently inactive cells that can tolerate high antibiotic concentrations without harboring inheritable resistance traits. Recent studies have indicated that genes associated with glycopeptide resistance, such as *tcaA,* may play a role in this persistent phenotype. Inactivation of the *tcaA* gene in *S. aureus* isolates resulted in a significant increase in persister cell production, indicating that this gene plays a dual role in both teicoplanin resistance and persistence [20]. This discovery highlights the significance of understanding the genetic interaction between resistance and persistence in bacteria, given the rise in therapeutic failure. The main goal of this research is to determine the presence of virulence genes in these bacterial isolates from clinical samples. According to this, the research will look into the molecular mechanisms behind how these bacteria (*S. aureus*) cause infection. This research will surely contribute to the development of diagnostic instruments. **A**dditionally, this research will contribute to the development of innovative therapies.

2. Materials and Methods

2.1. Sample Collection

Twenty-five urine samples were collected from 160 patients with urinary tract infections and skin ulcers at Yarmouk Teaching Hospital from October 2024 to January 2025. Isolation and identification of *Staphylococcus aureus* was done by culturing the sample on mannitol salt agar and blood agar and identification by coagulase and catalase tests and confirmed by the Vitek kit.

2.2. Antibiotic Resistance

Resistance to 19 antibiotics was studied using the Vitek kit, and the results were illustrated according to CLSI 2023 [(21)].

2.3. Molecular Methods

2.3.1. DNA Extraction

Genomic DNA from the confirmed *Staphylococcus aureus* 25 isolates was extracted by the EASY PURE BACTERIA GENOMIC DNA KIT according to the manufacturer's guidelines. Briefly, a single colony of each isolate was resuspended in 200 µL lysis buffer and allowed to sit at 56°C for 10 minutes. For lysis of the bacterial cells, after lysis, 20 µL proteinase K was added, and to break down proteins, the mixture was incubated for 10 minutes at 70°C before being combined with the lysate. The column was centrifuged at 12,000 rpm after being poured into a spin column with 200 µL of binding buffer. The column was rinsed twice with RPM for one minute to bind the DNA to the membrane. The lysate was mixed and incubated at 70°C for 10

minutes to aid in protein breakdown before being eluted in 50 µL of wash buffer to eliminate impurities and DNA. Following placement in a spin column containing 200 µL of binding buffer, the column was centrifuged at 12,000 rpm. To let the DNA bind to the membrane, the column was rinsed twice with RPM for one minute. The contaminants were removed by 500 microliters of wash, and the DNA was subsequently eluted in 50 µL of elution buffer. The NanoDrop spectrophotometer was used to determine concentration and purity. DNA samples with an A260/A280 ratio of 1.8-2.0 were considered suitable for PCR amplification and stored at -20°C for subsequent use.

2.3.2. Polymerase Chain Reaction Amplification

The genes (*tcaA*, *tcaB*, *tcaR*) were detected in bacterial isolates by polymerase chain reaction (PCR). Primers were designed using the Primer BLAST, which was used to precisely design the new primers' exact gene sequence for optimization, as in **Table 1**.

Table 1. Primer Sequences and Product Size of the Genes Used in the Study.

Target Gene	Sequence Of Primer (5'—3')	Product Size/ bp	Reference
tcaA (R) *tcaA* (F)	CTGCAAAATCACCGTTCTCA GCACCTACCAAGCAACCAAT	178	This Study
tcaB (R) *tcaB* (F)	TGCCATTGGAGCATTATCAA CGCCCAGTTGAATCTGAAAT	165	This Study
tcaR (R) *tcaR* (F)	GGTGTAAATAAGGCCGCAGT GCGCAATATCTGTCATAATCG	172	This Study

By electrophoresis at 70 volts for 2 hours on a 1.5% agarose gel, the polymerase reaction products were resolved. The agarose gel was stained with ethidium bromide dye, and the agarose gel was monitored by ultraviolet light to confirm the presence of the target genes according to their sizes (*tcaR* = 172), (*tcaB* = 165), (*tcaA* = 178).

2.4. Statistical Analysis

The impact was identified using the statistical analysis IBM SPSS Statistics 29 software. The study parameters were influenced by multiple variables.

3. Results

3.1. Isolation and identification

From 160 samples, *Staphylococcus aureus* was identified and isolated. This resulted in 25 isolates. On mannitol salt agar, the bacteria constituted 15% of the total sample, which was cultured after 24 hours. After 24 hours of incubation at 37°C, colonies appeared golden yellow when cultured on mannitol salt agar, and conventional biochemical tests and a Vitek apparatus were used to confirm the presence of *S. aureus*. When cultured on blood agar, *S. aureus* exhibited beta-hemolytic activity, meaning it lyses red blood cells in the surrounding environment. A transparent halo appeared around the colony as a result of hemolysis of hemoglobin, indicating that it lyses red blood cells in the surrounding environment.

3.2. Antibiotic susceptibility

Antibiotic susceptibility tests were performed using the Vitek system, and the results indicated resistance to moxifloxacin (0%), inducible clindamycin resistance (0%), linezolid (0%), tigecycline (0%), trimethoprim/sulfamethoxazole (0%), tobramycin (4%), levofloxacin (4%), nitrofurantoin (4%), gentamicin (8%), rifampicin (68%), erythromycin (72%), fusidic acid (76%), teicoplanin (80%), tetracycline (80%), clindamycin (88%), vancomycin (92%), cefoxitin screen (100%), benzylpenicillin (100%), and oxacillin (100%), as shown in **Figure 1**.

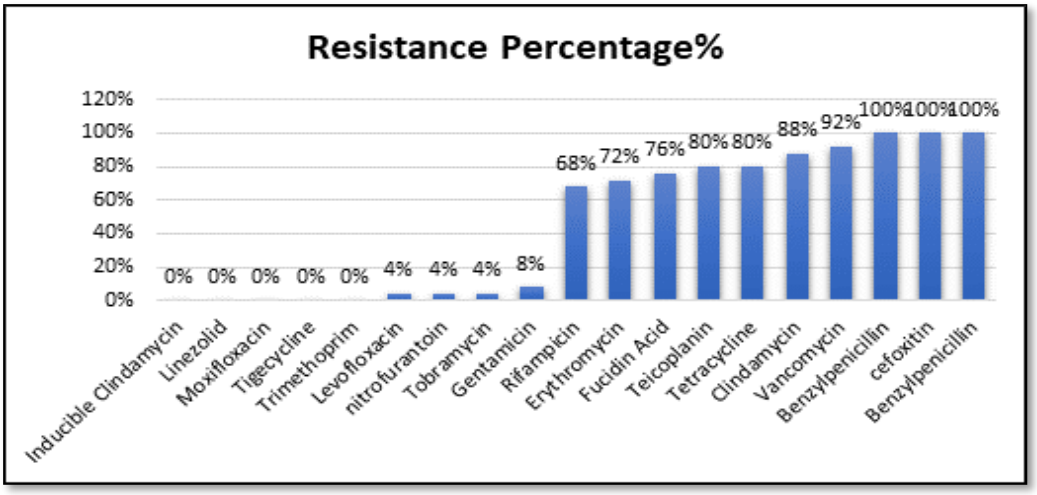

Figure 1. Resistance percentage of isolates for each antibiotic.

3.3. Detection of TCA Genes

Electrophoresis was used to detect the presence of teicoplanin-related genes *(tcaA, tcaB),* and polymerase chain reaction (PCR) results showed that all *S. aureus* isolates were positive for both *tcaA* and *tcaB* genes (**Figures 2-4).**

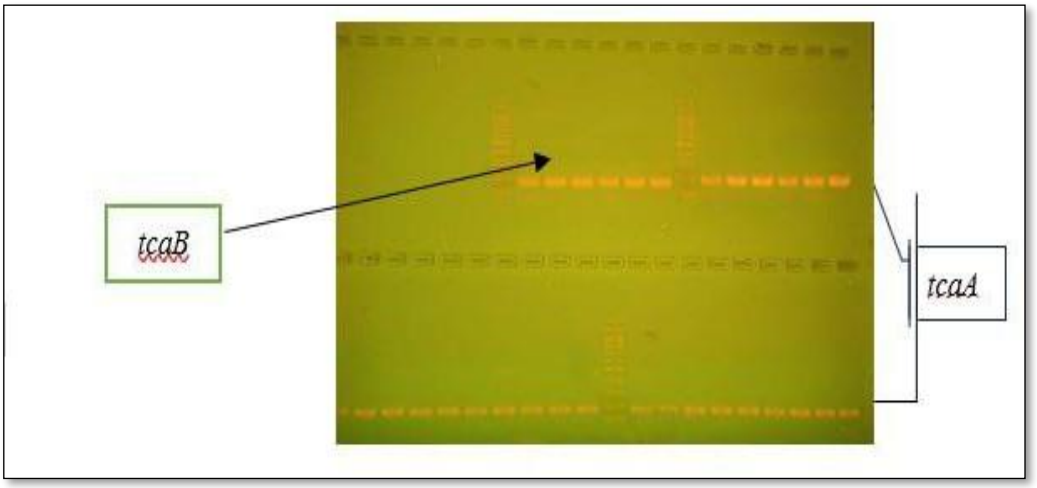

Figure 2. Gel Electrophoresis of the *tcaA* Gene of *Staphylococcus Aureus* Isolates Using 1.5% Agarose Gel Electrophoresis (70 Volts For 2 Hours). M:100 Pb Ladder Marker, Lanes 1-25 Resemble PCR Products.

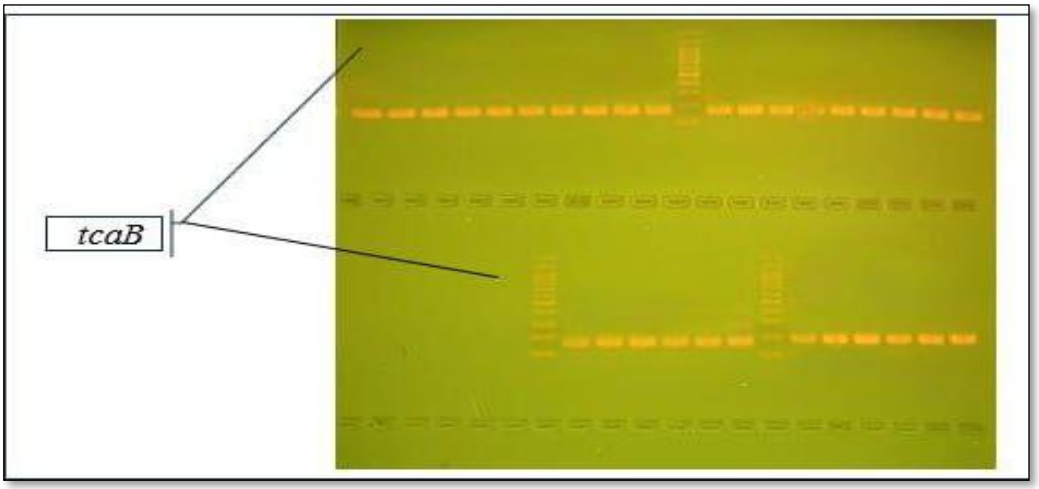

Figure 3. Gel Electrophoresis of the *tcaB* Gene of *Staphylococcus Aureus* Isolates Using 1.5% Agarose Gel Electrophoresis (70 Volts For 2 Hours). M: 100 Pb Ladder Marker, Lanes 1-25 Resemble PCR Products.

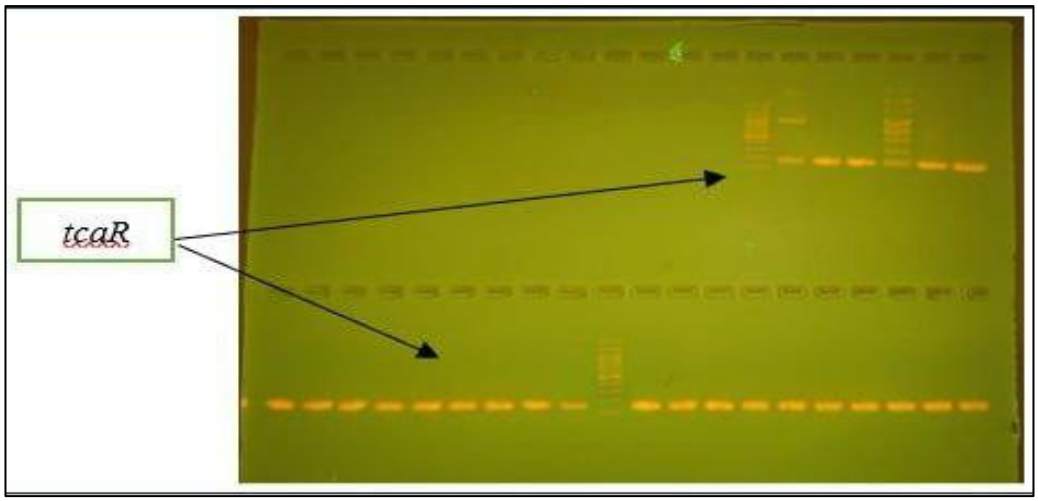

Figure 4. Gel Electrophoresis of the *tcaR* Gene of *Staphylococcus Aureus* Isolates Using 1.5% Agarose Gel Electrophoresis (70 Volts For 2 Hours). M:100 Pb Ladder Marker, Lanes 1-25 Resemble PCR Products.

The results showed that all 25 isolates (100%), and that the genes (*tcaA, tcaB, tcaR*) were present in all *S. aureus* isolates.

3.4. Correlation between antibiotic resistance especially glycopeptide antibiotic and presence of TCA genes

A relationship was also demonstrated between the presence of antibiotic resistance of the isolate and the presence of the genes. The results showed that all genes were found in the isolates and showed resistance to the isolates, but the highest resistance was to teicoplanin and vancomycin, as shown in **Table 2.**

Table 2. Correlation Between the Presence of *tcaA, tcaB,* and *tcaR* Regulated Genes and the Number of Antibiotic-Resistant Isolates.

Benzylpenicillin	Oxacillin	Gentamicin	Tobramycin	Levofloxacin	Moxifloxacin	Erythromycin	Clindamycin	Linezolid	Teicoplanin	Vancomycin	Tetracycline	Tigecycline	nitrofurantio	Fusidic Acid	Rifampicin	Trimethop	Cefoxitin screen	Inducible Clindamycin	TCA A	TCA B	TCA R
100%	100 %	8 %	4 %	4 %	0 %	72 %	88 %	0 %	80 %	92 %	80 %	0 %	4 %	76 %	68 %	0 %	100 %	0%	100 %	100 %	100 %

4. Discussion

Through the results obtained, it was found that all isolates of *Staphylococcus aureus* possessed the *tcaA* resistance genes. The presence of these genes in all isolates indicates the importance and role of these genes in the resistance of these bacteria. This is what was observed, as most of the isolates showed multiple resistance to most of the antibiotics used in the study, and most of the isolates were resistant to the glycopeptide group, such as teicoplanin and vancomycin, meaning that most of the isolates were of the VRSA type [1,10,18,20,21]. It was found that all 25 (*tcaA, tcaB, tcaR*) genes in *S. aureus* (100%) were identified, confirming their important role in bacterial virulence. These genes are part of the *tcaRAB* operon associated with immune evasion, biofilms, and teicoplanin resistance [20]. The complete identification of these genes in our analysis is consistent with the results of other studies ,*tcaA* and *tcaB* were detected in 98% and 95% of isolates, respectively, while *tcaR* was found in 90% [22]. Similar molecular detection methods were used to study resistance genes in MRSA [22,23]. If there is a difference in the distribution of bacterial strains or differences in the sources of clinical samples, those factors may lead to a slight difference in the prevalence of *tcaR*. concluded that *tcaA* and *tcaB* are responsible for the formation of biofilms, which is consistent with their results [24]. Similar

findings were reported in Iraq, where the presence of biofilm-related genes such as icaA and icaD in MRSA isolates was confirmed, This was demonstrated in a study published in the Iraqi Journal of Biotechnology, where all MRSA isolates harbored both icaA and icaD genes, indicating their role in biofilm formation.[24,25]. It has also been shown that these genes are essential in the production of extracellular matrix components for the structure of biomembranes [26]. Due to the presence of these genes in all isolations, these bacteria in this study are characterized by high abilities in the occurrence of harm, as well as because of the biological membranes and the immune response of the host [20]. The detection of these genes by the use of polymerase chain reaction has been shown to be a guaranteed way to identify potential *S. aureus* strains of antibiotics and resistance [27]. The disruption of *tcaRAB* leads to an increase in the resistance of teicoplanin, indicating that these genes play an important regulatory role in antibiotic sensitivity [28]. In another local study, *S. aureus* isolates resistant to most antibiotics were detected, with a ratio of 93.30% for penicillin and tetracycline and 53.30% for vancomycin [29]. The complete presence of these *tcaRAB* genes is considered to mean that these strains have a stable virulence profile, and this affects their ability to continue in clinical infection [30]. Overall, these studies contribute to the importance of these molecular methods in causing bacterial diseases. The full explanation of these genes suggests that they are likely to be potential targets for new treatments in order to control *S. aureus* infection [22,31]. The genes *tcaA, tcaB,* and tcaR and their role in teicoplanin resistance. The *tcaA* gene plays an important role in modifying the structure of the bacterial cell wall. This gene reduces the ability of teicoplanin to bind to peptidoglycan chains. This makes bacteria less sensitive to antibiotics. Disruption of this gene, as shown by studies, leads to an increase in the sensitivity of bacteria to teicoplanin; this makes the *tcaA* gene important in resistance to antibiotics [31,32]. While the *tcaB* gene has a role in the production of proteins, by changing the structure of the cell wall or by pumping the extracellular antibiotic that helps bacteria to avoid the effects of teicoplanin. This is part of the mechanisms developed by bacteria to resist antibiotics. Studies have also shown that this gene helps bacteria survive in the presence of teicoplanin and that the role of the *tcaR* gene is an important regulator of the expression of both *tcaA* and *tcaB* genes and that the increase in excess of *tcaR* increases the resistance of bacteria to the antibiotic teicoplanin [20,22]. Studies have also shown that disrupting this gene leads to an increase in bacterial sensitivity to the antibiotic, indicating that *tcaR* plays a regulatory role in antibiotic resistance [1,24].

5. Conclusion

We conclude from the results of these studies that *tca* genes are related to the burden of resistance of *Staphylococcus aureus* to antibiotics, especially the glycopeptide group, as most of the isolates containing these genes were resistant to glycopeptide antibiotics because these genes play a role in modifying the target area on which these antibiotics act.

Acknowledgment

The authors would like to express their sincere gratitude to the Department of Biology, College of Sciences, University of Baghdad, for their continuous support, valuable resources, and guidance throughout this research. Thanks to the patients who participated in this study, which was essential to its success.

Conflict of Interest

The authors state that there are no competing interests.

Funding

Study No funding was received for conducting this study.

Ethical Approval

The Ethical and Scientific Committee of the College of Sciences granted the study its approval. Ethical clearance with the number (Ref.: CSEC/0125/0006) from the University of Baghdad, Iraq.

References

1. Turner NA, Sharma-Kuinkel BK, Maskarinec SA, Eichenberger EM, Shah PP, Carugati M, Holland TL, Fowler VG Jr. Methicillin-resistant *Staphylococcus aureus*: an overview of basic and clinical research. Nat Rev Microbiol. 2019;17(4):203–218. https://doi.org/10.1038/s41579-018-0147-4
2. Taylor TA, Unakal CG. Staphylococcus aureus Infection. 2025. PMID: 28722898
3. Touaitia R, Mairi A, Ibrahim NA, Basher NS, Idres T, Touati A. Staphylococcus aureus: A Review of the Pathogenesis and Virulence Mechanisms. Antibiotics. 2025 May 6;14(5):470. https://doi.org/10.3390/antibiotics14050470
4. Liu A, Garrett S, Hong W, Zhang J. Staphylococcus aureus Infections and Human Intestinal Microbiota. Pathogens. 2024 Mar 24;13(4):276. DOI: https://doi.org/10.3390/pathogens13040276
5. Alkuraythi D. Virulence Factors and Pathogenicity of Staphylococcus aureus. In 2024. https://doi.org/10.5772/intechopen.1006633
6. Girma A. Staphylococcus aureus: Current perspectives on molecular pathogenesis and virulence. The Cell Surface. 2025 Jun; 13:100137. https://doi.org/10.1016/j.tcsw.2024.100137
7. Kavanagh N, Ryan EJ, Widaa A, Sexton G, Fennell J, O'Rourke S. Staphylococcal osteomyelitis: disease progression, treatment challenges, and future directions. Clin Microbiol Rev. 2018;31(2):e00084-17. **https://doi.org/10.1128/CMR.00084-17**
8. de Jong NWM, van Kessel KPM, van Strijp JAG. Immune Evasion by *Staphylococcus aureus*. Microbiol Spectr. 2019 Apr 12;7(2). https://doi.org/10.1128/microbiolspec.GPP3-0061-2019
9. Flemming HC, Wingender J, Szewzyk U, Steinberg P, Rice SA, Kjelleberg S. Biofilms: an emergent form of bacterial life. Nat Rev Microbiol. 2016 Sep 11;14(9):563–75. https://doi.org/10.1038/nrmicro.2016.94
10. Antimicrobial Resistance Collaborators. Global burden of bacterial antimicrobial resistance in 2019: a systematic analysis. ***Lancet***. 2022;399(10325):629–655. **https://doi.org/10.1016/S0140-6736(21)02724-0**
11. Poudel AN, Zhu S, Cooper N, Little P, Tarrant C, Hickman M, Yao G. The economic burden of antibiotic resistance: a systematic review and meta-analysis. *PLoS One*. 2023;18(5):e0285170. **https://doi.org/10.1371/journal.pone.0285170**
12. Hobbs AM, Kluthe KE, Carlson KA, Nuxoll AS. Interruption of the tricarboxylic acid cycle in Staphylococcus aureus leads to increased tolerance to innate immunity. AIMS Microbiol. 2021;7(4):513–27. https://doi.org/10.3934/microbiol.2021031
13. Patel H, Rawat S. A genetic regulatory see-saw of biofilm and virulence in MRSA pathogenesis. Front Microbiol. 2023 Jun 22;14. https://doi.org/10.3389/fmicb.2023.1204428
14. Pai L, Patil S, Liu S, Wen F. A growing battlefield in the war against biofilm-induced antimicrobial resistance: insights from reviews on antibiotic resistance. Front Cell Infect Microbiol. 2023 Dec 19;13. https://doi.org/10.3389/fcimb.2023.1327069
15. Chiba M, Aoyagi T, Yoshida M, Katsumi M, Fujimaki S-I, Ishii Y, Tateda K, Kaku M. Evaluation of the performance of GeneSoC®, a novel rapid real-time PCR system, to detect ***Staphylococcus aureus*** and methicillin resistance in blood cultures. ***J Infect Chemother***. 2023;29(7):718–721. **https://doi.org/10.1016/j.jiac.2023.03.006**
16. Nadiya S, Kolla HB, Reddy PN. Optimization and evaluation of a multiplex PCR assay for detection of Staphylococcus aureus and its major virulence genes for assessing food safety. Brazilian Journal of Microbiology. 2023 Mar 23;54(1):311–21. https://doi.org/10.1007/s42770-023-00906-6
17. Abdrabaa MK, Abd Aburesha R. Gene Expression Evaluation of Intracellular Adhesins and Regulatory Genes among Biofilm Producing MRSA Isolates. Iraqi Journal of Science. 2023 Jan 30;75–83. https://doi.org/10.24996/ijs.2023.64.1.8
18. Bakthavatchalam YD, Babu P, Munusamy E, Dwarakanathan HT, Rupali P, Zervos M, Victor PJ, Veeraraghavan B. Genomic insights on heterogeneous resistance to vancomycin and teicoplanin in methicillin-resistant ***Staphylococcus aureus***: a first report from South India**.** *PLoS One*. 2019;14(12):e0227009. **https://doi.org/10.1371/journal.pone.0227009**

19. Salgueiro V, Manageiro V, Bandarra NM, Ferreira E, Clemente L, Caniça M. Genetic Relatedness and Diversity of *Staphylococcus aureus* from Different Reservoirs: Humans and Animals of Livestock, Poultry, Zoo, and Aquaculture. Microorganisms. 2020 Sep 3;8(9):1345. https://doi.org/10.3390/microorganisms8091345
20. Habib G, Gul H, Ahmad P, Hayat A, Rehman MU, Mohamed Moussa I, Elansary HO. Teicoplanin-associated gene tcaA inactivation increases persister cell formation in Staphylococcus aureus. Front Microbiol. 2023 Oct 13;14:1241995. https://doi.org/10.3389/fmicb.2023.1241995
21. CLSI. Performance Standards for Antimicrobial Susceptibility Testing. 33rd edition. Wayne, PA: Clinical and Laboratory Standards Institute; 2023.
22. Brandenberger M, Tschierske M, Giachino P, Wada A, Berger-Bächi B. Inactivation of a novel three-cistronic operon tcaR-tcaA-tcaB increases teicoplanin resistance in Staphylococcus aureus. Biochimica et Biophysica Acta (BBA) - General Subjects. 2000 Oct;1523(2–3):135–9. https://doi.org/10.1016/S0304-4165(00)00133-1
23. Study the Expression of msrA,msrB and linA/linA' genes in Presence of Some Antibiotics in Methicillin Resistance Staphylococcus aureus. Iraqi Journal of Science [Internet]. 2018 Oct 31;59(4A):1811–25. https://ijs.uobaghdad.edu.iq/index.php/eijs/article/view/528
24. Otto M, Süssmuth R, Jung G. Role of tcaA and tcaB in biofilm formation. Infect Immun. 2006;74(4):2145–53. https://doi.org/10.1128/IAI.74.4.2145-2153.2006
25. Mussa AA, Al-Mathkhury HJF. Estimation of the expression of glucose-dependent biofilm-encoding icaA and icaD genes in methicillin-resistant *Staphylococcus aureus* isolates. Iraqi J Biotechnol. 2025; 24(1): 270–282.
26. Miao J, Lin S, Soteyome T, Peters BM, Li Y, Chen H, Su J, Li L, Li B, Xu Z, Shirtliff ME, Harro JM. Biofilm formation of Staphylococcus aureus under food heat processing conditions: First report on CML production within biofilm. Sci Rep. 2019 Feb 4;9(1):1312. https://doi.org/10.1038/s41598-018-35558-2.
27. El Seedy FR, Samy AA, Salam HSH, Khairy EA, Koraney AA. Polymerase chain reaction detection of genes responsible for multiple antibiotic resistance Staphylococcus aureus isolated from food of animal origin in Egypt. Vet World. 2017;10(10):1205–11. https://doi.org/10.14202/vetworld.2017.1205-1211
28. Novick RP, Geisinger E. Quorum Sensing in Staphylococci. Annu Rev Genet. 2008;42(1):541–64. https://doi.org/10.1146/annurev.genet.42.110807.091640
29. Shakur JA, Aburesha RA. Investigating the presence of eap and spa genes of *Staphylococcus aureus* and their relation to antibiotic resistance. Ibn Al-Haitham J Pure Appl Sci. 2024;37(3). https://doi.org/https://doi.org/10.30526/37.3.3436
30. Grundmann H, Aires-de-Sousa M, Boyce J, Tiemersma E. Emergence and resurgence of meticillin-resistant Staphylococcus aureus as a public-health threat. The Lancet. 2006;368(9538):874–85. https://doi.org/10.1016/S0140-6736(06)68853-3
31. Foster TJ. Antibiotic resistance in *Staphylococcus aureus*. Current status and future prospects. FEMS Microbiol Rev. 2017 May 1;41(3):430–49. https://doi.org/10.1093/femsre/fux007
32. Munita JM, Arias CA. Mechanisms of Antibiotic Resistance. Microbiol Spectr. 2016;4(2). https://doi.org/10.1128/microbiolspec.VMBF-0016-2015