

Integrative Review of Key Molecular Markers in *Staphylococcus aureus*: Identification, Enzymes, Virulence and Antimicrobial Resistance

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Abstract:

Background: *Staphylococcus aureus* is a significant foodborne pathogen, causing illnesses through contaminated food. Its virulence, driven by enterotoxin production, and increasing antimicrobial resistance (AMR) necessitate advanced detection methods. Molecular biology tools, such as PCR and sequencing, enable precise identification of virulence and resistance markers, improving food safety strategies.

Materials and Methods: A systematic search was conducted across PubMed, SciELO, and CAPES Journal Portal using keywords like *S. aureus*, virulence genes, AMR, and PCR. Boolean operators refined results, yielding 17 articles. Data were categorized into gene markers, primer sequences, amplicon size, and biological functions (identification, virulence, AMR, enzymatic activity).

Results: The review identified critical molecular markers for *S. aureus*, organized into functional groups. Key genes linked to virulence (e.g., enterotoxins), AMR (e.g., *mecA*), and enzymatic activity were highlighted. Primer sets and amplicon sizes were documented, facilitating future diagnostics.

Conclusion: This integrative review underscores the genetic complexity of *S. aureus* and the utility of molecular markers in surveillance. The prevalence of AMR markers calls for early detection to mitigate risks. Findings support improved diagnostics and biotechnological innovations for food safety and public health.

Key Word: AMR; Diagnostic; Gene; *mecA*; Surveillance.

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I. Introduction

Staphylococcus aureus is a highly relevant pathogen in the food industry, posing serious public health risks. Responsible for various foodborne illnesses, this bacterium is often associated with the consumption of contaminated products¹. Its ability to produce enterotoxins enhances its virulence, leading to gastrointestinal disturbances in affected individuals. The widespread occurrence of *S. aureus* in different food matrices, combined with its resistance to environmental stressors, highlights the need for effective monitoring and control strategies in the context of food safety^{2,3}.

In this context, molecular biology tools have transformed the study of enteropathogens, allowing for more precise identification and characterization of microorganisms. Techniques such as polymerase chain reaction (PCR), sequencing, and genotyping enable the rapid detection of genetic markers associated with virulence and antimicrobial resistance. These methods provide valuable insights into the epidemiology of foodborne pathogens, contributing to the development of more targeted and effective interventions^{4,5}.

Understanding the molecular markers of *S. aureus* is essential for enhancing surveillance and control strategies. Primers and oligonucleotides are fundamental tools for the specific amplification of target genes related to virulence factors and resistance mechanisms. Identifying these markers allows for the differentiation

of pathogenic strains from non-pathogenic ones, as well as aiding in the assessment of the risks that *S. aureus* poses in food products^{6,7}.

This study aims to explore the main molecular markers associated with *S. aureus*, focusing on identification, virulence, antimicrobial resistance, and enzymatic activity. By systematically mapping these markers, we seek to deepen our understanding of the role of *S. aureus* in the food sector. The expected results aim to contribute to the development of more accurate diagnostic tools and more effective control measures, thereby promoting greater food safety and improved public health outcomes.

II. Material And Methods

Study Type

This study was conducted through a systematic literature review aimed at identifying molecular markers associated with *S. aureus*, particularly those related to virulence, antimicrobial resistance, and enzymatic activity.

Databases and Search Strategy

The search for scientific publications was carried out in the PubMed, SciELO, and CAPES Journal Portal databases, covering articles indexed in the fields of microbiology, biotechnology, public health, and food safety. To broaden the search and include studies published in different languages, descriptors in Portuguese, English, and Spanish were utilized. Key terms included: *Staphylococcus aureus*, molecular markers, virulence genes, antimicrobial resistance, enzymes, primers, and PCR. These descriptors were combined with Boolean operators (AND, OR, NOT), adhering to the syntax of each database to refine the results. Examples of applied combinations include: *Staphylococcus aureus* AND virulence genes AND PCR; *S. aureus* AND resistance markers OR enzymatic activity; molecular markers AND food safety NOT vaccines.

Selection Criteria and Time Frame

A time frame of 15 years was established, covering the period from 2010 to 2025, to ensure the relevance and currency of the data obtained. Inclusion criteria required that articles present experimental data describing target genes used as molecular markers in *S. aureus*, including genetic characterization and the use of specific primers.

Data Analysis and Organization

At the end of the screening process, 17 articles were selected and analyzed. The extracted information was organized into two tables. The first table included the gene name (marker), associated gene product, primer sequences (forward and reverse), amplicon size (in base pairs), and reference (author and year). The second table classified the genes based on the biological function of the gene product, grouping them into categories: identification, virulence, antimicrobial resistance, and enzymatic activity. This organization allowed for a comparative analysis of the molecular markers described in the literature, contributing to the identification of those with greater relevance and potential application in monitoring and control strategies for *S. aureus* in the context of food safety.

III. Result

Tables 1 and 2 provide a comprehensive set of genetic markers used to characterize this bacterium across various aspects, including identification, virulence, toxin production, and antimicrobial resistance. Table 1 details 39 molecular markers, along with their respective primer sequences, expected amplicon sizes, and recent literature references, while Table 2 organizes these markers according to their biological function.

In the context of identifying *S. aureus*, the genes *nuc*, *coa*, *femA*, and *spa* stand out due to their specificity and reliability. For assessing virulence, genes related to biofilm formation (*icaA*, *icaD*), adhesion factors such as *clfA*, *clfB*, *fnbA*, and *fnbB*, as well as protein A (*spa*) and the adhesin (*sdrE*), highlight the pathogenic potential of the bacterium. The presence of genes encoding toxins, such as enterotoxins (*sea*, *seb*, *sec*, *sed*, *see*), toxic shock syndrome toxin (*tst*), leukocidins (*lukS-PV/pvl*), and hemolysins (*hla*, *hly*), underscores the risk associated with infections caused by highly virulent strains.

Table no 1: Molecular Markers Applied to Identification, Virulence, Resistance and Toxins in *S. aureus*

Marker	Gene Product	Primer Sequence (5'-3')	Size	Reference
<i>nuc</i>	Nuclease	F: GCGATTGATGGTGATACGGTT R: AGCCAAGCCTTGACGAACATA	279 pb	8
<i>mecA</i>	PBP2a	F: AAAATCGATGGTAAAGGTTGGC R: AGTTCTGCAGTACCGGATTTC	533 pb	9
<i>mecC</i>	PBP2a'	F: TCACCAGGTTCAAC[Y]CAAAA R: CCTGAATC[W]GCTAATAATATTTC	356 pb	9
<i>femA</i>	Methicillin resistance	F: CGATCCATATTTACCATATCA	450 pb	10

	factor	R: ATCACGCTCTCTCGTTTAGTT		
<i>vanA</i>	VanA (VRSA)	F: GGGAAAACGACAATTGC R: GTACAATGCGGCCGTTA	732 pb	11
<i>vanB</i>	VanB	F: ATGGGAAGCCGATAGTC R: GATTTCGTTCTCGACC	635 pb	11
<i>coa</i>	Coagulase	F: GCCGCTTTAATACCAGCAAC R: CTTCCGATTGTTTCGATGCTT	2268 pb	12
<i>icaA</i>	Biofilm	F: GACCTCGAAGTCAATAGAGGT R: CCCAGTATAACGTTGGATACC	814 pb	13
<i>icaD</i>	Biofilm	F: AAACGTAAGAGAGGTGG R: GGCAATATGATCAAGATAC	318 pb	14
<i>tst</i>	TSST-1	F: TGCTAGACTGGTATAGTAGTGG R: GTTCCTTCGCTAGTATGTTGG	212 pb	15
<i>lukS-PV</i>	Toxin PVL	F: ATCATTAGGTAATAATGTCTGGACATGATCCA R: GCATCAASTGTATTGGATAGCAAAAGC	433 pb	16
<i>sea</i>	Enterotoxin A	F: GGTATCAATGTGCGGGTGG R: CGGCACTTTTCTCTTCGG	102 pb	17
<i>seb</i>	Enterotoxin B	F: GTATGGTGGTGAAGTGAAGC R: CCAAAATAGTGACGAGTTAAGG	164 pb	17
<i>sec</i>	Enterotoxin C	F: AGATGAAGTTAGTTGATGTGTATGG R: CACACTTTTAGAATCAACCG	451 pb	17
<i>sed</i>	Enterotoxin D	F: GTGGTGAAATAGATAGGACTGC R: GAAGGTGCTCTGTGGATAATG	381 pb	15
<i>see</i>	Enterotoxin E	F: AGGTTTTTTCACAGGTCATCC R: CTTTTTTTCTTCGGTCAATC	209 pb	18
<i>ermA</i>	RNA methylase	F: TATCTTATCGTTGAGAAGGGATT R: CTACACTTGGCTTAGGATGAAA	139 pb	19
<i>ermB</i>	RNA methylase	F: CTATCTGATTGTTGAAGAAGGATT R: GTTTACTCTGGTTTATGATGAAA	142 pb	19
<i>ermC</i>	RNA methylase	F: CTTGTTGATCAGGATAATTTCC R: ATCTTTTAGCAAAACCGTATTC	190 pb	19
<i>ermT</i>	RNA methylase	F: ATTGGTTCAGGGAAAGGTCA R: GCTTGATAAAATGGTTTTTGA	536 pb	19
<i>hla</i>	Alpha hemolysin	F-GCGAAGAAGGTGCTAACA R-CAATTGGTAATCATCACGAAC	569 pb	20
<i>hly</i>	Beta hemolysin	F: GTGCACTTACTGACAATAGTGC R: GTGCACTTACTGACAATAGTGC	309 pb	21
<i>clfA</i>	Aggregation factor A	F: CGCCGGTAAGTGGTGAAGCT R: TGCTCTCATTCTAGGCGCACTT	314 pb	22
<i>clfB</i>	Aggregation factor B	F: CCGGTAGTAAATGCTGCTGTA R: CACTTTGATTAGGGTCAAATGTAGTC	103 pb	22
<i>fibA</i>	Binding to fibronectin A	F: TGGTACTGATGAAGTTGATTTAGAAC R: CATTATCCCAAGTTAAGGTATATCCTC	101 pb	22
<i>fibB</i>	Binding to fibronectin B	F: GGAGCGGCCTCAGTATTCTT R: AGTTGATGTGCGCTGTATG	201 pb	22
<i>spa</i>	Protein A	F: AGACGATCCTTCGGTGAGC R: GCTTTTGCAATGTCATTACTG	330 pb	23
<i>sdrE</i>	Adhesin SdrE	F: AGGAGTGATGCTGGTTTCCA R: TTTGGTGATGCGATGTTGTC	433 pb	14
<i>pvl</i>	Leukocidin PVL	F: GCTGGACAAAACCTCTTGGAATAT R: GATAGGACACCAATAAATTCTGGATTG	87 pb	14
<i>blaZ</i>	Beta-lactamase	F: TCAAACAGTTCACATGCC R: TTCATTACACTCTGGCG	877 pb	13
<i>norA</i>	Efflux pump	F: ATCGGTTTAGTAATACCAGTCTTGC R: GCGATATAATCATTTGAGATAACGC	112 pb	24
<i>norB</i>	Efflux pump	F: AGCGCGTTGTCTATCTTTCC R: GCAGGTGGTCTTGCTGATAA	213 pb	24
<i>msrA</i>	Efflux pump	F: TCCAATCATTGCACAAAATC R: AATCCCTCTGATTTGGTGT	163 pb	19
<i>tetK</i>	Efflux pump	F: TCGATAGGAACAGCAGTA R: CAGCAGATCCTACTCCTT	169 pb	16
<i>tetL</i>	Efflux pump	F: TCGTTAGCGTGCTGTCATTC R: GTATCCCACCAATGTAGCCG	267 pb	16
<i>tetM</i>	Ribosomal protection	F: GTGGACAAAAGGTACAACGAG R: CGGTAAAGTTCGTACACAC	406 pb	16
<i>tetO</i>	Ribosomal protection	F: AACTTAGGCATTCTGGCTCAC R: TCCCACTGTTCCATATCGTCA	515 pb	16

One of the most significant points observed in the tables is the diversity and number of genes associated with antimicrobial resistance. Genes such as *mecA* and *mecC* confer resistance to methicillin, while *vanA* and *vanC* are related to vancomycin resistance. Markers for resistance to macrolides (*ermA*, *ermB*, *ermC*,

ermT), beta-lactams (*blaZ*), and tetracyclines (*tetK*, *tetL*, *tetM*, *tetO*) were also included, along with efflux pump genes (*norA*, *norB*, *msrA*), demonstrating the complexity of the genetic mechanisms involved.

The integration of information from Tables 1 and 2 provides a complete, up-to-date, and functionally distributed molecular panel that is useful for both laboratory diagnostics and epidemiological studies as well as public health surveillance. The predominance of resistance-related genes (about one-third of the total) reflects the current concern regarding multidrug-resistant strains of *S. aureus*, emphasizing the importance of genetic monitoring to guide therapeutic and control strategies.

Table no 2: Functional distribution of molecular markers in studies of *S. aureus*

Marker	Product/Description	Rating Tag
<i>nuc</i>	Nuclease	Identification
<i>femA</i>	Methicillin-Resistance-Related Factor	Identification / Antimicrobial Resistance
<i>coa</i>	Coagulase	Identification / Virulence Factor
<i>icaA</i> , <i>icaD</i>	Biofilm Formation	Virulence Factor
<i>tst</i>	Toxic Shock Syndrome Toxin	Enzymes (toxins)
<i>lukS-PV</i> , <i>pvl</i>	Leukocidin PVL	Enzymes (toxins)
<i>sea</i> , <i>seb</i> , <i>sec</i> , <i>sed</i> , <i>see</i>	Enterotoxins	Enzymes (toxins)
<i>hla</i> , <i>hly</i>	Hemolysins	Enzymes (toxins)
<i>clfA</i> , <i>clfB</i>	Clumping Factors	Virulence Factor
<i>fibA</i> , <i>fibB</i>	Fibronectin Binding	Virulence Factor
<i>spa</i>	Protein A (IgG-Binding)	Identification / Virulence Factor
<i>sdrE</i>	SdrE Adhesin	Virulence Factor
<i>mecA</i> , <i>mecC</i>	Methicillin Resistance	Antimicrobial Resistance
<i>vanA</i> , <i>vanC</i>	Vancomycin Resistance	Antimicrobial Resistance
<i>ermA</i> , <i>ermB</i> , <i>ermC</i> , <i>ermT</i>	RNA Methylases (Macrolides)	Antimicrobial Resistance
<i>blaZ</i>	Beta-Lactamase	Antimicrobial Resistance
<i>norA</i> , <i>norB</i> , <i>msrA</i> , <i>tetK</i> , <i>tetL</i>	Efflux Pumps	Antimicrobial Resistance
<i>tetM</i> , <i>tetO</i>	Ribosome Protection (Tetracyclines)	Antimicrobial Resistance

IV. Discussion

The presence of resistance genes in *Staphylococcus* highlights the remarkable ability of this bacterium to adapt and survive in the face of commonly used antimicrobials. Genes such as *mecA* and *mecC* are well-known for their association with the MRSA phenotype (methicillin-resistant *Staphylococcus aureus*). They function by modifying the proteins that bind to penicillin, thereby preventing the effective action of these antibiotics. The gene *femA* also plays a role in this process by strengthening the cell wall structure and contributing to the stability of these proteins. Meanwhile, the gene *blaZ* produces an enzyme called beta-lactamase, which degrades penicillin before it has a chance to act, further complicating treatment^{9,10,13}.

Genes such as *ermA*, *ermB*, and *ermC* provide protection against important classes of antibiotics, including macrolides, lincosamides, and streptogramins. They achieve this by altering the structure of ribosomes - the sites where antibiotics typically act - and in some cases, their effects only manifest after the bacterium has been exposed to the drug. This makes diagnosis and treatment selection even more challenging, as different strains may respond differently to these substances¹⁹.

In the case of tetracyclines, which are widely used in livestock due to their accessibility and effectiveness, resistance is primarily mediated by the genes *tetK* and *tetM*. The former encodes a pump that expels the antibiotic from the bacterial cell, while the latter protects the ribosomes from the drug's action. These mechanisms are particularly common in agricultural settings, reflecting the intensive use of this class of medications¹⁶.

The genes *vanA* and *vanB* are even more concerning as they are linked to vancomycin resistance - an antibiotic reserved for the most severe cases. These genes modify the drug's binding sites, preventing its action and posing a significant threat in both human and veterinary medicine¹¹. The gene *norA* also deserves mention. It is involved in resistance to fluoroquinolones, antibiotics commonly used in animal production. By encoding an efflux pump, this gene reduces the concentration of the drug within the bacterial cell, making treatment less effective and underscoring the importance of judicious antimicrobial use in managing infections²⁴.

The integration of information from Tables 1 and 2 reveals a comprehensive, up-to-date, and functionally distributed molecular panel, useful for both laboratory diagnostics and epidemiological studies as well as public health surveillance. The predominance of resistance-related genes (about one-third of the total) reflects the current concern regarding multidrug-resistant strains of *S. aureus*, emphasizing the importance of genetic monitoring to guide therapeutic and control strategies.

The formation of biofilms by *Staphylococcus* bacteria is not coincidental; it is regulated by a set of genes that coordinate everything from the initial adhesion of cells to the production of the extracellular matrix that protects the bacterial colony. Among these genes, the *ica* operon (composed of *icaA*, *icaB*, *icaC*, and *icaD*)

plays a crucial role, as it is directly involved in the synthesis of polysaccharide intercellular adhesin (PIA), one of the main components that keeps bacterial cells together within the biofilm. These genes have been extensively studied, particularly in clinical contexts, with the aim of finding ways to inhibit their activity and thus control biofilm formation^{13,14}.

Additionally, the genes *fmbA* and *fmbB* encode proteins that bind to fibronectin, a protein found in host tissues. This interaction is essential in the initial stage of bacterial attachment to the infected organism, laying the groundwork for biofilm development. Other important genes in this process are *clfA* and *clfB*, which produce proteins capable of binding to fibrinogen, a protein present in the host's extracellular matrix. This binding helps stabilize biofilms, particularly on surfaces such as catheters and medical implants. Therefore, these genes are also targets of studies seeking new strategies to prevent bacterial adhesion to hospital material²².

S. aureus produces enterotoxins, which are heat-stable exotoxins responsible for food poisoning and gastrointestinal illness. These enterotoxins are encoded by specific genes (*sea*, *seb*, *sec*, *sed*, *see*) and can be detected in milk, posing a significant public health risk. The study highlights the importance of identifying enterotoxin-producing *S. aureus* strains, particularly MRSA, in dairy products to prevent foodborne outbreaks. The researchers used molecular techniques to detect enterotoxin genes and methicillin resistance, emphasizing the need for rigorous food safety monitoring¹⁷.

V. Conclusion

The prospecting of key molecular markers for *S. aureus* has highlighted the diversity and genetic complexity of this pathogen, particularly regarding its identification, virulence, antimicrobial resistance, and enzyme production. The systematic analysis of the data revealed a robust set of target genes that can be applied in diagnostic, monitoring, and public health control strategies, especially in the food sector. The predominance of markers associated with antimicrobial resistance underscores the urgency of adopting molecular tools to detect multidrug-resistant strains early, contributing to food safety and outbreak prevention. Thus, the results of this study provide valuable insights for enhancing microbiological surveillance practices and encourage the development of new approaches in biotechnology applied to public health.

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