وقائع المؤتمر العلمي السنوي الثالث لكلية التربية الاساسية
في مجال العلوم الصرفة والموسوم:
(مؤتمر كلية التربية الاساسية في مجال العلوم الصرفة)
وتحت شعار
(بالعلوم الصرفة نبني واقعا معرفيا متجددا)
يومي الاحد والاثنين 21-2026/6/22

Staphylococcus aureus: An Overview on Taxonomy, Epidemiology, Pathogenicity and Antibiotic Resistance

Shahbaa Hameed Majeed
Department of Science, College of Basic Education, AL-Mustansiriyah University, Baghdad, Iraq.
Shahbaa.edbs@uomustansiriyah.edu.iq.

Abstract:
This review summarizes the taxonomy, epidemiology, pathogenicity, virulence determinants, and antibiotic resistance mechanisms of *S. aureus*. The bacterium is a Gram-positive coccus that typically appears in grape-like clusters and exhibits characteristic biochemical properties like catalase and coagulase positivity. Epidemiological evidence indicates that *S. aureus* colonizes a considerable proportion of the global population, particularly in the nasal cavity. Methicillin-resistant *S. aureus* (MRSA) has become a significant public issue of health because of its increasing prevalence and association with severe Infectious illnesses including bacteremia, pneumonia, endocarditis, osteomyelitis, and soft tissue and skin infectious illnesses. The pathogenicity of *S. aureus* is attributed to numerous virulence factors like adhesins, enzymes, toxins, and immune-evasion proteins that facilitate colonization, tissue invasion, immune evasion, and biofilm formation. The bacterium also possesses complex regulatory systems, including the *agr* and *sar* regulatory networks, that control the expression of virulence genes in response to environmental conditions. In addition, *S. aureus* has developed multiple mechanisms of antibiotic resistance, including β-lactamase production. Understanding the epidemiology, pathogenic mechanisms, and resistance strategies of *S. aureus* is essential for improving diagnostic approaches, Infectious illness control strategies, and the development of effective therapeutic interventions.

Keywords: Antibiotic resistance, Epidemiology, Pathogenicity, *S. aureus*, Virulence factors, Methicillin.

Introduction:

The bacteria belonging to the genus *Staphylococcus* are Gram-positive cocci that are usually present in an irregular cluster, often resembling a grape bunch. These bacteria are an integral part of the normal flora of humans as well as animals. They are present on the skin surfaces as well as the mucosal surfaces of the urogenital, alimentary, and respiratory tracts of different birds and mammals. Although these bacteria are often considered opportunistic pathogenic organisms, they can cause different infections illnesses in humans as well as animals **(Fayisa & Tuli, 2023)**.

Of all coagulase-positive staphylococci isolated from human specimens, *S. aureus* is considered the most important **(Sigudu *et al.*, 2023)**. In veterinary medicine, other coagulase-positive staphylococci are also common and may be of great importance **(Sulikowska *et al.*, 2024)**. *S. aureus* is recognized to be a key contributory agent of intramammary infections illnesses in dairy ruminants, resulting in clinical mastitis and subclinical mastitis. Such infections illnesses have been shown to cause economic losses in dairy farming because of reduced milk production and deterioration in milk quality. *S. aureus* can be isolated from different parts of the body in addition to milk in dairy cattle. It is believed that the distribution of intramammary infection in dairy cattle occurs through the milking process **(Fayisa & Tuli, 2023)**. *S. aureus* has been linked with a broad range of illnesses in human populations, from mild infectious illnesses of skin to serious systemic illnesses like bacteremia and pneumonia. The illnesses resulted from this bacterium have been reported to occur in community and healthcare settings. While the bacterium has been reported to colonize healthy and normal skin, its presence in the bloodstream and other body parts has been linked with serious and life-threatening illnesses **(Huang *et al.*, 2022)**. In terms of clinical relevance, *S. aureus* has been linked with toxic shock syndrome, pneumonia, poisoning of food, and suppurative illnesses. Furthermore, MRSA is one of the major causes of hospital-acquired infectious illnesses, while community-linked MRSA has been increasingly found to cause severe illnesses like pneumonia **(Abdallah *et al.*, 2024; Abdallah & Sulieman, 2024; Pal *et al.*, 2024)**. The pathogenic potential of *S. aureus* has been

attributed to its capability to synthesize a range of virulence determinants like hemolysins, leukocidins, enterotoxins, exfoliative toxins, and a range of immune modulators. The expression of these virulence determinants is tightly regulated via a range of regulatory systems, including the accessory gene regulator quorum-sensing system. Using these systems, *S. aureus* can cause tissue adhesion, cell damage, and evade the host's immune response. The MRSA, which has been identified as an important pathogenic organism in humans and animals and commonly found in clinical cases, has been recognized globally in the last few years because of the increasing number of livestock-linked MRSA with multidrug resistance phenotypes. The presence of MRSA has created therapeutic problems, especially with resistant strains. In spite of different measures taken to control MRSA in animal husbandry, *S. aureus* has been found to persist in environmental sources, dairy products, animal surfaces, and humans **(Douglas *et al.*, 2023; Fayisa & Tuli, 2023)**.

From a public perspective of health, the appearance of Livestock-linked MRSA has raised concerns regarding zoonotic transmission potential. Humans in close contact with animals, like dairy farm workers, are at a higher risk of acquiring these strains directly, and there is a potential for the distribution via the food chain into the community. Animals that are colonized with MRSA can serve as a reservoir, enabling the transmission of MRSA to humans. Moreover, the use of antibiotics in animal husbandry has been implicated in the emergence of antimicrobial resistance, which has been recognized as one of the major health concerns worldwide. In this regard, efforts have been made to explore the use of natural antimicrobial substances in the multidrug-resistant *S. aureus* infections illnesses' management **(Khairullah *et al.*, 2023; Roy *et al.*, 2024; Y. Wang *et al.*, 2023)**.

Despite extensive knowledge on the epidemiology, virulence determinants, and antimicrobial resistance mechanisms of *S. aureus*, knowledge gaps still exist. This comprises a need for extensive surveillance on resistant strains, like MRSA, and a better understanding of virulence regulation systems, which play a role in pathogenicity and adaptation to hosts. Moreover, the

emergence of drug-resistant strains emphasizes the need to design more effective therapeutic strategies and alternative antimicrobial therapy to effectively combat infections illnesses.
illnesses.

1. Methodology of the Review

The review was based on a narrative review where Staphylococcus aureus was emphasized in relation to its taxonomy, epidemiology, pathogenesis, virulence factors, clinical manifestations, foodborne transmission, and antimicrobial resistance. The relevant literature on *S. aureus* as outlined in the review was examined for the purpose of categorization. The reviewed sources included recent primary research articles, review articles, epidemiological information, and literature regarding the detection, incidence, virulence control, resistance, foodborne disease, and carriage of S. aureus by food handlers. Information related to the subject was extracted and classified under various headings, including general characteristics, current classification, epidemiology, clinical manifestation, virulence genes and their control, virulence, resistance to antibiotics, and foodborne diseases. Conventional and contemporary techniques like microscopy, culture, biochemical analysis, RapID STAPH, Sensititre, and API Staph assays have been discussed, drawing upon knowledge obtained from the references. Knowledge obtained from international and local resources was utilized wherever deemed necessary to highlight the importance of S. aureus and MRSA within the context of public health care. Ethical approval was unnecessary since there were no studies conducted that involved sample acquisition, lab work, human subjects, or animal testing.

2. *Staphylococcus aureus*

2.1 General Characteristics

In term of bacterium description and identification, various tests are used, as follows(Table 1):

Table 1: Microscopic field, cultural characteristics and biochemical characteristics of S. aureus (Ahmed et al., 2026; Becker, 2023; Brakstad et al., 1993; Essers & Radebold, 1980; Fayisa & Tuli, 2023; Thaker et al., 2013).

Category	Test / Observation	Result for *S. aureus*
Microscopic field	Gram stain	Gram-positive cocci
	Cell arrangement	Grape-like clusters
	Cell shape	Spherical (cocci)
	Cell size	0.5–1.5 μm in diameter
	Spore formation	Non-spore forming
	Motility	Non-motile
Cultural characteristics	Nutrient agar	Smooth, circular, convex colonies
	Blood agar	β-hemolytic colonies with 1-3 mm colony size
	Mannitol Salt Agar (MSA)	Yellow or golden-yellow, round, smooth colonies with colonies size of 1–3 mm
	Brain heart infusion agar	medium-sized, circular, creamy-white to golden-yellow colonies.
	Colony size	The typical staphylococcal phenotype is characterized by moderately sized colonies measuring approximately 1–3 mm in diameter after 24 hours of incubation.
Biochemical characteristics	Catalase test	Positive
	Coagulase test	Positive (the presence of free and/or bound coagulase)
	DNase	Positive
	Oxidase	Negative
	Thermonuclease (heat-stable nuclease)	Positive
	Latex agglutination (detecting clumping factor/protein A)	Positive

In addition to these conventional examinations, advanced system for identification also utilized as follows:

(i) Rapidic STAPH system

Recent studies reported that the RapID STAPH PLUS system is a commercial identification method used for the species-level confirmation of staphylococci including *S. aureus*. The system is based on the detection of specific biochemical reactions generated by bacterial metabolism, and identification results are interpreted according to the manufacturer's database. Several recent investigations stated that *S. aureus* isolates were identified or confirmed using the RapID STAPH system prior to molecular verification **(Al-Nsour *et al.*, 2024; Al-Tameemi *et al.*, 2023)**

(i) Sensititre system

Recent scientific reports described the Sensititre system as an automated or semi-automated platform used for antimicrobial susceptibility testing and minimum inhibitory concentration (MIC) determination of *S. aureus* and other bacterial pathogens. The method employs microdilution panels containing defined antimicrobial concentrations, and MIC values are interpreted using standardized reading software or automated readers. In the retrieved recent studies, this system was applied after phenotypic or biochemical identification to evaluate resistance profiles of isolates of *S. aureus* **(Kerek *et al.*, 2025; Seo *et al.*, 2025)**.

(ii) API Staph system

Recent reviews and primary research articles reported that the API Staph system is a standardized biochemical identification method designed for the differentiation and confirmation of staphylococcal species including *Staphylococcus* aureus. The system consists of a panel of miniaturized biochemical substrates that detect enzymatic activity and carbohydrate utilization patterns, and identification is achieved by comparing the reaction profile with reference databases. Recent studies stated that the API Staph system was used either alone or together with automated platforms and molecular assays to confirm the identity of *S. aureus* isolates **(Al-Khfaji *et al.*, 2025; Mairi *et al.*, 2025; Mustafa, 2024)**.

2.2 Current taxonomic state

The genus *Staphylococcus* has more than 40 species, with 20 being of veterinary interest. These species vary significantly with respect to their ecological niches, epidemiological distribution, pathogenicity, and resistance patterns against antimicrobial agents. In addition, there are differences observed at the strain level as well. One of these species is *S. aureus*, whereas this species' classification as follows **(Fayisa & Tuli, 2023)**:

Domain: Bacteria
Phylum: Firmicutes
Class: Bacilli
Order: Bacillales
Family: Staphylococcaceae
Genus: *Staphylococcus*
Species: *S. aureus*

2.3 Epidemiology

The S. aureus has emerged as one of the most common bacterial pathogenic organisms across the globe and is considered one of the primary causes of not only community-acquired but also health care-linked Infectious illnesses. It has been identified through various epidemiological studies across the globe that one out of every four individuals across the globe is asymptomatically colonized with S. aureus in the nasal passage or other body sites **(Yang *et al.*, 2024)**. In fact, based on a present meta-analysis, the worldwide prevalence of *S. aureus* colonization in the nasal or throat area is about 26.4%, and the prevalence of MRSA among *S. aureus* colonizing strains is about 15%; however, there are significant regional variations **(Locke *et al.*, 2025)**. Additionally, a large systematic review that comprised data from over 160,000 participants from different countries estimated the overall prevalence of MRSA globally and found that it was approximately 14.69%, which shows the prevalence and distribution of this particular pathogenic organism **(Hasanpour *et al.*, 2023)**. From an epidemiological perspective, MRSA has emerged as a significant public issue of health owing to its association with severe clinical syndromes like bacteremia, pneumonia, and soft tissue and skin infectious illnesses, coupled with its

rising antimicrobial resistance profile, which makes treatment and control strategies against this pathogenic organism complicated **(Shoaib *et al.*, 2023)**.

The issue of *S. aureus* infectious illness is considered one of the main challenges in Iraq, especially considering the high rates of antimicrobial resistance and the prevalence of MRSA. Several studies carried out in Iraq have identified high rates of prevalence of *S. aureus* in different samples. For instance, research carried out on the clinical isolates of the pathogenic organism in Iraq identified the prevalence rate of 27.9% of the total bacterial isolates obtained from the patient samples **(Ghareeb *et al.*, 2023)**. In a similar vein, studies conducted on the nasal carriage of healthy persons in Iraqi society have shown that the levels of colonization were significant, with 27.3% of MRSA strains comprising *S. aureus* strains from samples collected from the anterior nasal cavity **(AL-Salihi *et al.*, 2023)**. Studies have shown that a relatively high percentage of healthcare workers in Iraq have a high carriage rate of MRSA, which reaches 22.5% **(Hussein *et al.*, 2019)**. In addition, surveillance studies carried out in Iraqi hospitals have indicated that *S. aureus* continues to be an important cause of skin Infectious illnesses, wound infectious illnesses, and other conditions, with molecular studies detecting the presence of *mecA* resistance genes, which are responsible for the persistence of MRSA strains **(Abood, 2022)**.

2.4 Clinical Manifestations of *S. aureus*

Various clinical manifestations are resulted from *S. aureus* and illustrated in

Table 2: Clinical Manifestations of S. aureus (Rallis et al., 2025; Tong et al., 2015).

No.	Clinical manifestation	Brief description
1	**Staphylococcal food poisoning (SFP)**	Food-borne illness resulted from ingestion of enterotoxins produced via *S. aureus*, resulting in gastrointestinal symptoms like vomiting and diarrhea.
2	**Staphylococcal scalded skin syndrome (SSSS)**	Toxin-mediated skin disorder characterized via fever, erythema, superficial blistering, and skin peeling, mainly affecting infants and young children.
3	**Toxic shock syndrome (TSS)**	Severe toxin-mediated systemic illness presenting with fever, rash, vomiting, diarrhea, and potential organ failure and shock.
4	***S. aureus* bacteremia (SAB)**	Bloodstream infectious illness that can disseminate to multiple organs, leading to serious complications and high morbidity and mortality.
5	**Infective endocarditis**	Infectious illness of the heart valves resulting from bacterial colonization of damaged cardiac endothelium and thrombotic vegetations.
6	**Osteomyelitis**	Inflammatory Infectious illness of bone tissue that leads to bone destruction and may occur via hematogenous distribution or direct inoculation.
7	**Staphylococcal pneumonia**	Severe lung Infectious illness that may lead to necrotizing pneumonia, bacteremia, or sepsis with high fatality in some cases.
8	**Soft tissue and skin infectious illnesses**	Comprises conditions like boils, abscesses, impetigo, and cellulitis resulting from invasion of skin or soft tissues.

2.5 Virulence factor of *Staphylococcus aureus*

2.5.1 Virulence genetics

The genome of *S. aureus* can be classified into 2 main portions: the core genome and the accessory genome. The first portion comprises about 75% of the total genome, and it contains genes that are highly conserved and are necessary for metabolism and replication of the bacterium **(Kläui *et al.*, 2019; Prosperi *et al.*, 2019)**. On the other hand, the accessory genome, which comprises about 25% of the total genome, makes a significant contribution to genetic variation in *S. aureus*, and it often contains genes

related to virulence, immune evasion, and antibiotic resistance, especially in MRSA and other *S. aureus* pathogenic strains **(Seder *et al.*, 2023)**. *S. aureus*'s virulence genes of are found in the accessory genome, and this section of the genome contains mobile genetic elements (MGEs) of *S. aureus*, including staphylococcal cassette chromosomes, pathogenicity islands, insertion sequences, prophages, plasmids, and transposons **(Prosperi *et al.*, 2019)**. MGEs are capable of distributing virulence genes and resistance genes, and in some instances, these genes may code for virulence factors of specific illnesses, including enterotoxins and toxic shock syndrome toxin-1 (TSST-1), which are responsible for food poisoning **(Patel & Rawat, 2023)**. The most important toxins and virulence factors are located in the phage-linked regions and pathogenicity islands, thus indicating the importance of horizontal gene transfer (HGT)in the pathogenic evolution of *S. aureus* **(Cervera-Alamar *et al.*, 2018)**. For example, staphylokinase, enterotoxins, immune evasion proteins like SCIN and Chemotaxis Inhibitory Protein of *Staphylococcus* (CHIPS), epidermolytic toxins A and B, Panton-Valentine leukocidin (PVL), among many others, are located in prophages **(Kong *et al.*, 2016)**. Moreover, the *hlb* gene encode the beta toxin, which has a critical function in the pathogenicity and colonization of the pathogenic organism **(Loftus *et al.*, 2022)**. Similarly, pathogenicity islands encode enterotoxins and TSST, among other toxins, whereas other virulence genes located in genomic islands, including LukDE, α-toxin, lipoprotein-like toxins (LPLs), peptide spectrum match peptides (PSMs), SSL, and other enterotoxins, may differ in expression in various strains, thus affecting their pathogenic potential **(Cheung *et al.*, 2021)**.

2.5.2 Virulence regulation

The virulence of *S. aureus* is regulated via an advanced networks of metabolic and genetic regulatory systems that are sensitive to environmental conditions, which comprise temperature, pH, availability of nutrient, and other host-related conditions **(Bernardos *et al.*, 2019)**. The virulence genes' expression is regulated via several regulatory systems, which comprise the global regulators that regulate the expression of various virulence determinants, in addition to the locus-specific regulators, which comprise the

gene of *icaR* that regulates the operon of *ica* and the PmtR regulator that regulates the PSM exporter operon. One of the most significant regulatory systems is the system of accessory gene regulator (*agr*), which contains *agrA*, *agrB*, *agrC*, and *agrD* genes, forming a two-component system. The previous mentioned system is induced in response to the autoinducing peptides accumulation secreted via the bacteria, which act as a quorum-sensing mechanism. Once this system is activated, the *agr* system has been observed to down-regulate the expression of adhesion surface proteins and capsule polysaccharides, and to up-regulate the expression of several virulence determinants, thereby making the bacteria more invasive and capable of immune evasion **(Bashabsheh *et al.*, 2024)**.

Another significant regulatory mechanism is the staphylococcal accessory regulator system, which comprises the sarA, sarR, and sarS genes. These genes encode regulatory proteins which play an important role in the regulation of the synthesis of toxins, adhesins, etc. **(Albalbaki *et al.*, 2024; Cheung *et al.*, 2001; Tiwari *et al.*, 2020)**. Other global regulators which were found to be significant in the regulation of virulence comprise the ferric uptake regulator, which responds to iron starvation signals and regulates the production of virulence factors and iron acquisition systems, and the Staphylococcal Respiratory Response System (SrrAB), which is an oxygen-responsive two-component system which is significant in the regulation of virulence factor production and the enhancement of resistance to oxidative stress, especially under anaerobic conditions **(Balasubramanian *et al.*, 2017; Bashabsheh *et al.*, 2024; Tiwari *et al.*, 2020)**.

Transcription regulation in *S. aureus* is also influenced via the existence of sigma factors which direct the enzyme RNA polymerase to the target promoters. The bacterium contains four different sigma factors, denoted as σA, σB, σH, and σS, which are responsible for the regulation of different target gene sets **(Shaw *et al.*, 2008)**. Among these, the alternative sigma factor σB (SigB) is critical for stress response, which regulates the expression of over 250 target genes, including those responsible for biofilm formation and stress tolerance **(Kinkel *et al.*, 2013; Wang *et al.*, 2022)**.

Other regulatory systems, like CodY, Catabolite Control Protein A (CcpA), and the two-component systems Sae and ArlRS, also responsible for the regulation of virulence gene expression along with the agr and sar systems **(Cheung *et al.*, 2011)**. The overall regulation via these systems allows *S. aureus* to rapidly adapt the virulence factors' production in responses to changes in the environment, which is critical for the pathogenicity and ability of the bacterium to evade the host immune response **(Touaitia *et al.*, 2025)**.

2.5.3 Virulence factors

There are diversity of virulence factors responsible for pathogenicity of *S. aureus*, as follows (Table 3):

Table 3: Virulence factors of S. aureus (Bashabsheh et al., 2024).

Category	Virulence Factor	Target	Main Effect
Cell wall–linked factors	Capsular polysaccharides	Mucosal surfaces	Protect bacteria from phagocytosis
	PIA/PNAG polysaccharides	Bacterial cells, tissues	Biofilm formation and bacterial aggregation
	Serine-aspartate repeat protein (SdrE)	Complement proteins	Suppresses complement-mediated opsonizations
	Determinant proteins of iron-regulated surface (IsdA, IsdB)	Heme-iron	Acquisition of iron; promotes tissue invasion and abscess formation
	Clumping factors (ClfA, ClfB)	Fibrinogen, platelets, cytokeratin	Promote adhesion and platelet activation
	Collagen-binding protein (Cna)	Cartilage, collagen	Facilitates adhesion to host tissues; inhibits complement activation
	Fibronectin-binding proteins (FnBPA, FnBPB)	Fibronectin, fibrinogen, elastin	Adhesion to extracellular matrix proteins and host tissues
	Lipoteichoic acid, teichoic acid, and peptidoglycan	Immune tissues ad cells	Stimulate response of inflammation; responsible for biofilm and formation adhesion
	Staphylococcal protein A (SpA)	Complement, IgM and IgG,	Attaches region of Fc of antibodies; suppresses phagocytosis and opsonization
Enzymes	Catalase	Hydrogen peroxide	Breaks down reactive oxygen species

	Coagulase	Prothrombin	Promotes clot formation; protects bacteria from phagocytosis
	Staphylokinase	Plasminogen	Activates plasmin, enabling degradation of extracellular matrix
	Lipases	Lipids	Degrade host lipids and promote bacterial distribution
	Nucleases	Nucleic acids	Degrade DNA in neutrophil extracellular traps
	Proteases (SspA, ScpA, SspB, Aur)	ECM proteins, complement	Degrade host proteins and inhibit immune responses
	Superoxide dismutases	Superoxide radicals	Protect bacteria from oxidative stress
Toxins	Hemolysins (α, β, γ, δ)	Erythrocytes, leukocytes	Cell lysis and immune evasion
	Enterotoxins	Intestinal cells, T cells	Cause food poisoning and cytokine release
	Exfoliative toxins	Skin desmosomes	Cause skin blistering in scalded skin syndrome
	Toxic shock syndrome toxin-1 (TSST-1)	T lymphocytes	Massive cytokine production causing toxic shock syndrome
	Panton-Valentine leukocidin (PVL)	Macrophages, neutrophils,	Leukocyte destruction and tissue necrosis
Secreted immune-evasion proteins	Extracellular fibrinogen-binding protein (Efb)	Fibrinogen, complement	Interferes with platelet aggregation and complement
	SCIN	Complement proteins	Blocks complement activation
	SSL-5	Neutrophils, platelets	Inhibits neutrophil activation
	SSL-7	IgA, complement C5	Prevents antibody and complement function
	CHIPS	Neutrophils	Inhibits neutrophil chemotaxis

* PIA = Polysaccharide Intercellular Adhesin; IgM = Immunoglobulin M; PNAG = Poly-N-Acetylglucosamine; SdrE = Serine–Aspartate Repeat Protein E; IsdA = Iron-regulated Surface Determinant Protein A; IsdB = Iron-regulated Surface Determinant Protein B; ClfA = Clumping Factor A; ClfB = Clumping Factor B; Cna = Collagen-Binding Protein; FnBPA = Fibronectin-Binding Protein A; SSL-7 = Staphylococcal Superantigen-Like Protein 7; FnBPB = Fibronectin-Binding Protein B; SpA = Staphylococcal Protein A; IgG

= Immunoglobulin G; ECM = Extracellular Matrix; SspA = Serine Protease A; ScpA = Staphylococcal Cysteine Protease A; SspB = Staphopain B; Aur = Aureolysin; PVL = Panton-Valentine Leukocidin; TSST-1 = Toxic Shock Syndrome Toxin-1; Efb = Extracellular Fibrinogen-Binding Protein; SCIN = Staphylococcal Complement Inhibitor; SSL-5 = Staphylococcal Superantigen-Like Protein 5; CHIPS = Chemotaxis Inhibitory Protein of *Staphylococcus*.

2.6 Pathogenicity

The *S. aureus* is portion of the normal flora of the mucous membranes and skin, but it can be pathogenic once it gains access to the body, especially through skin injuries, and to people with weakened immune systems. The development of systemic Infectious illnesses is linked with two main mechanisms: toxin production and bacterial colonization, which aids in tissue invasion and destruction (Figure 2) **(Fayisa & Tuli, 2023)**.

2.7 Pathogenesis of *S. aureus* infectious illnesses

The pathogenesis of *S. aureus* Infectious illness starts with the adhesion of the bacteria, which is a critical step for the colonization of the tissues of the host. This adhesion occurs with the help of various adhesin proteins, especially the microbial surface components recognizing adhesive matrix molecule (MSCRAMM) family, which comprises fibronectin-binding proteins (FnBPs), fibrinogen-binding protein (ClfA) and collagen-binding protein (Cna), that allow the bacteria to attach to the tissues, especially the skin, nasal mucosal surfaces, and heart valves, and so on, via adhering to the extracellular matrix components of the tissues **(Bonsaglia *et al.*, 2025; Jafaar & Kandala, 2025; Touaitia *et al.*, 2025)**. In addition, the structural components, like teichoic acid and lipoteichoic acid, also allow the bacteria to attach, while the capsule helps the bacteria adhere and also protects the bacteria **(Park *et al.*, 2025)**.

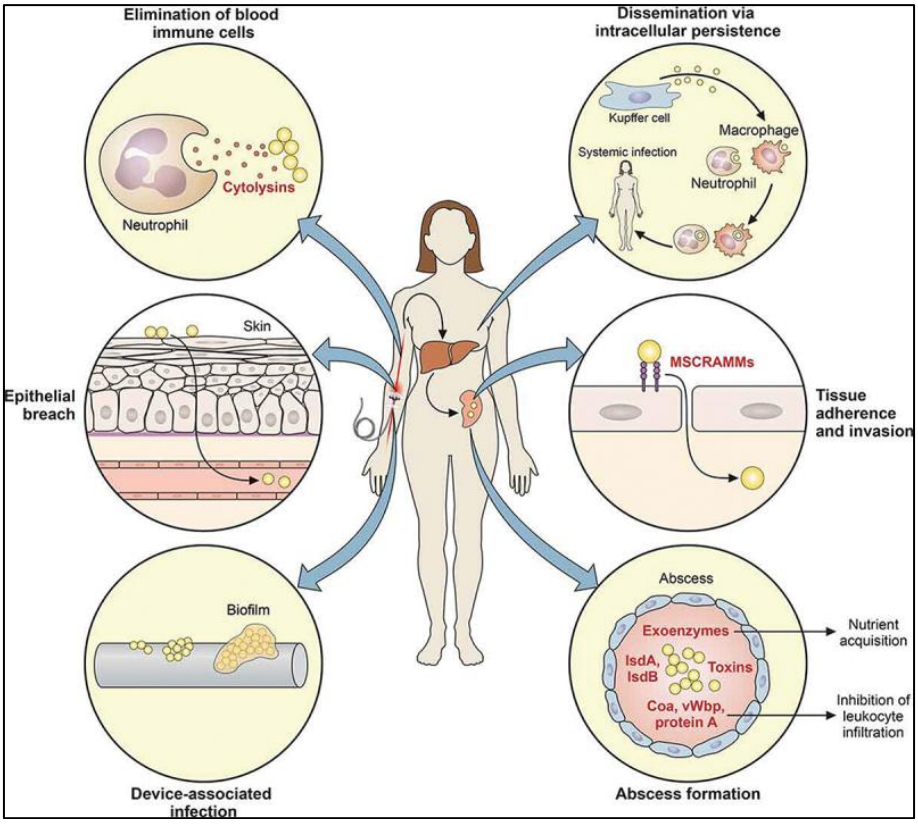


Figure 1: The pathogenesis of S. aureus systemic Infectious illnesses is initiated via the bacterium overcoming the body's natural barriers, like the skin, or distributing from biofilms that form on medical devices. Once inside the body, the bacterium is able to evade the immune system via secreting cytolytic toxins that destroy immune cells, like neutrophils. The bacterium can also remain inside these immune cells, which allows it to distribution to different parts of the body. The bacterium can also distribution via the bloodstream, which allows it to invade different tissues of the body. The formation of abscesses is influenced via different bacterial adhesins, toxins, exoenzymes, and other virulence factors **(Cheung *et al.*, 2021)**.

After adhesion, *S. aureus* invades the tissues of the host via the secretion of various enzymes and virulence factors. Enzymes like hyaluronidase and fibrinolysin cause the degradation of tissues, which helps the distribution of the bacteria, while proteases like aureolysin and staphopain degrade the

proteins, like immunoglobulins and complements, of the host, which helps the bacteria overcome the system of immunity **(Linz *et al.*, 2023; Prosperi *et al.*, 2019)**. In addition, the bacteria can form abscesses that shield the bacteria from the host's immune system and provide them with nutrients, as well as biofilm, which helps the bacteria resist the effects of antibiotics and the system of host's immunity **(Albalbaki *et al.*, 2024; G. Cheung *et al.*, 2021; Mlynarczyk-Bonikowska *et al.*, 2022)**.

In addition, *S. aureus* uses various strategies to escape the host immune system. These strategies involve the secretion of chemotaxis inhibitory proteins, which inhibit the phagocytic activity of neutrophils, the secretion of anti-opsonizing proteins, which inhibit the phagocytic activity of neutrophils, protein A, which affects the antibody-mediated immune response, Panton-Valentine leukocidin, which destroys superantigens like enterotoxins and toxic shock syndrome toxins-1, and leukocytes, which affect the immune response via inducing abnormal activation of T cells **(Bashabsheh *et al.*, 2024)**.

2.8 Antibiotic Resistance

Several mechanisms have been adopted via S. aureus that confer resistance to methicillin and other antibiotics. Among the most important mechanisms adopted via S. aureus, the production of β-lactamase, which has the ability to cleave the β-lactam ring and inhibit the action of methicillin, can be comprised. Another mechanism adopted via S. aureus that makes it resistant to methicillin and other β-lactam antibiotics is the modification of the penicillin-binding proteins, which are very vital for the synthesis of the cell walls of the bacterium **(Brawley *et al.*, 2022; Lade *et al.*, 2022)**. Furthermore, some strains of *S. aureus* decrease the cell membrane permeability and increase the activity of efflux pumps, which reduces the ability of antibiotics to enter the bacterial cell and decreases the effectiveness of the antibiotic via actively pumping out the drugs from the cell **(Bashabsheh *et al.*, 2024)**. Another major route through which resistance is established is through horizontal transfer of gene via mobile genetic elements (MGEs), which comprise transposons and plasmids that are known to carry genes responsible for resistance to antibiotics **(Bitrus *et al.*,**

2017). The nature of the resistance genes carried via the plasmids depends on their sizes; small-sized plasmids carry resistance genes for tetracycline and erythromycin, while large-sized plasmids carry resistance genes for β-lactam antibiotics and macrolides, which together with other MGEs are responsible for resistance to vancomycin, erythromycin, β-lactam antibiotics, and trimethoprim **(Jian *et al.*, 2021; Rasheed & Hussein, 2021)**. Traditionally, the development of resistance to antibiotics has been a feature of *S. aureus* since the development of antimicrobial agents, with the first reports of penicillin resistances occurring in the 1940s, followed via the identification of MRSA in the United Kingdom in the early 1960s, which has become a serious public health issue with severe soft tissue and skin infectious illnesses worldwide **(Bashabsheh *et al.*, 2024)**.

Besides MRSA, other resistant strains that have been identified comprise vancomycin-intermediate *S. aureus* (VISA), vancomycin-resistant *S. aureus* (VRSA), and heterogeneous vancomycin-intermediate *S. aureus* (hVISA), which display reduced or complete resistance to vancomycin **(Shariati *et al.*, 2020)**. The first case of VISA was identified in Japan in 1997, while the first case of VRSA was reported in the United States in 2002 in a diabetic patient **(Mondal *et al.*, 2024)**. The rising incidence of resistant strains, especially in Asian and American countries, warrants close monitoring of vancomycin treatment and the adoption of measures that can control the distribution of resistant *S. aureus* strains.

2.9 *Staphylococcus aureus* and Food posing

2.9.1 *Staphylococcus aureus* as a Common Foodborne Pathogen

Staphylococcus aureus has been recognized as a significant foodborne pathogen because of its potential for producing heat-stable staphylococcal enterotoxins (SEs) that are active after exposure to usual cooking temperatures **(Elshebrawy *et al.*, 2025; Shrestha *et al.*, 2025)**. These toxins are produced by classical enterotoxin genes like *sea*, *seb*, *sec*, *sed*, and *see*, and are potentially produced in the food before it is ingested, thus resulting in acute onset of illness after exposure, even at low concentrations of the toxin **(Li *et al.*, 2024)**.

The prevalence of *S. aureus* in the healthy human population, especially in those dealing with food, allows for cross-contamination during processing and handling **(Das *et al.*, 2025)**. Continuous colonization, even in the absence of symptoms, allows for the constant transfer of the pathogen to food products, especially in environments with poor hygiene practices **(Bencardino & Vitali, 2019)**. Apart from foodborne intoxication, *S. aureus* is also recognized as an important pathogen in the development of both healthcare-associated and community-acquired infections **(Gherardi, 2023)**. The extensive use of antibiotics in the treatment of these infections has also led to the development of resistance, complicating the treatment and possibly leading to failure **(Liu *et al.*, 2021)**.

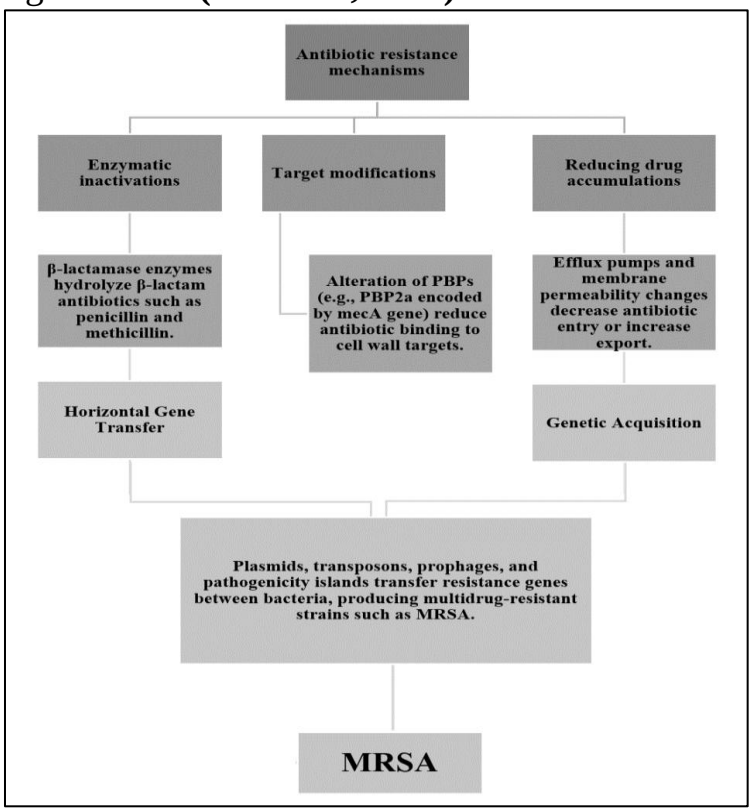


Figure 2: Mechanisms of antibiotic resistance in S. aureus. MRSA = Methicillin-Resistant S. aureus; PBP2a = Penicillin-Binding Protein 2a; mecA = Methicillin Resistance Gene A; PBP = Penicillin-Binding Protein; β-lactam = Beta-lactam Antibiotics; S. aureus = Staphylococcus aureus.

2.9.2 Colonization and Transmission Dynamics Related to Food Handlers

Nasal carriage has been viewed as an essential aspect of the environment-mediated spread of *Staphylococcus* spp. across food-processing facilities. In environments with inadequate sanitation, microorganisms from colonized workers may contaminate shared utensils, equipment, and preparation surfaces. Findings have shown that a considerable number of hand contamination events, especially among workers who are not colonized, may be related to strains from persisting colonized co-workers, thus supporting the concept of indirect transferring via shared workplaces **(Viana *et al.*, 2025)**. The mode of spread is particularly significant in situations wherein surfaces that are frequently touched, such as preparation tables or cutting tools, are not subjected to disinfection **(Mahros *et al.*, 2021)**. Moreover, the ability of *Staphylococcus* aureus to produce biofilms on human skin surfaces as well as on various environmental surfaces is a significant factor that makes it difficult to eradicate this pathogen in food processing plants . Food handlers may also be infected with *Staphylococcus* spp. over direct attachments with undercooked or raw products of animals **(Kebede & Getu, 2023)**.

Molecular epidemiological studies have demonstrated that the pathogens isolated from foods as well as from food handlers may be of the same clonal complexes and may carry virulence determinants as well as antimicrobial resistance determinants **(Argudín *et al.*, 2012)**. For example, the Spa type t127, frequently found in animal sources, has also been found in food handlers, representing that the handling of polluted dairy or meat products may facilitate the introduction of strains of animal origin into the carriers human **(Baptistao *et al.*, 2016)**. In addition, asymptomatic cases, especially with intermittent and persistent colonization of the nose, are an important reservoir of contamination. Furthermore, some of the handlers may transmit this bacterium on their hands instead of the nose, as they may not carry the bacteria nasally, probably because of contact with polluted surface as opposed to self-inoculation, despite inadequate hygiene practices **(Viana *et al.*, 2025)**.

Regarding the level at which food becomes dangerous, an outbreak investigation reported that several studies consider *S. aureus* counts greater than 10^5 CFU/g in food to be toxic and sufficient for staphylococcal enterotoxin production. The same report also stated that enterotoxin A was nevertheless detected in outbreak foods with lower counts, including up to 10^3 CFU/g, and cited a Belgian investigation where SEA was detected in mashed potato at about 200 CFU/g and in chicken and sausage at up to 10^3 CFU/g **(Sanad *et al.*, 2025)**.

2.9.3 Prevalence of *Staphylococcus aureus* among Iraqi food handlers

In Baghdad, Iraq, **(Obaid, 2025)** examined 325 nasal swabs from food handlers without clinical symptoms working in restaurants and cafeterias and found that 30.15% carried *S. aureus* as well as at least one classical enterotoxin gene was detected in 46.93% of the isolates, with single-gene carriage reported as sed 3%, sec 4%, seb 6.1%, and sea 9.1%, while see was not detected. Recent Iraqi food data also show the presence of *S. aureus* in foods. In Basrah, **Aziz *et al.* (2024)** reported 27 *S. aureus* isolates from 135 locally made food samples, giving an overall prevalence of 20%; the distribution reported in that study was 10.7% in meat samples, 6.2% in milk samples, 1% in cake samples, and 2.1% in cheese samples. In Duhok Governorate, **Issa and Khidhir (2023)** found *S. aureus* in 37.5% of raw milk samples, and the reported density ranged from 1×10^3 to 4.0×10^4 cfu/mL in one sampling group, with other positive groups showing counts from 2.7×10^4 to 3.0×10^4 cfu/mL.

Conclusion:

The *S. aureus* is one of the most important human pathogenic bacteria, both in the hospital and the community. Its ability to infect the human body and produce a multitude of virulence factors, along with its capability to avoid the system of human immunity, is responsible for the broad range of illnesses linked with this pathogenic organism. The bacterium has a complex regulatory mechanism that controls the production of enzymes, toxins, and adhesins, which enables the pathogenic organism to rapidly adapt to different human environments. In addition, the emergence and distribution of antibiotic-resistant strains of *S. aureus*, like MRSA, VISA, and VRSA,

have made the treatment of *S. aureus* Infectious illnesses and the control of this illness a major challenge. The acquisition of resistant genes via mobile genetic elements and the possession of multiple mechanisms of resistance have made *S. aureus* a successful pathogenic organism. Therefore, it is critical to continue surveillance of this illness, improve Infectious illness control measures, and find new ways of combating this illness to control the distribution of resistant strains of *S. aureus*.

Further Work

The monitoring of S. aureus infections, especially infections with MRSA and MDR bacteria, must always be carried out in hospitals, communities, food processing plants, and animals. The sensitivity of the tests used for diagnosis and antibiogram must be enhanced. Improvement is needed in relation to the approaches for infections control, proper hygiene practices in the handling of food-by-food handlers, antibiotic use, and surveillance of food products in order to avoid the incidence of infections and contamination. Besides, more research can also be done in regard to the issues raised herein.

References

1. Abdallah, E. M., & Sulieman, A. M. E. (2024). *Staphylococcus* aureus. *Microbial Toxins in Food Systems: Causes, Mechanisms, Complications, and Metabolism, 235*.

2. Abdallah, E. M., Sulieman, A. M. E., & Saleh, Z. A. (2024). New discoveries in toxins from gram-positive bacteria *Staphylococcus* aureus. In *Microbial toxins in food systems: Causes, mechanisms, complications, and metabolism* (pp. 235–252). Springer.

3. Abood, H. H. K. Z. H. (2022). *Staphylococcus* aureus Incidence in Some Patients with a Topic Dermatitis in Baghdad City. *Iraqi Journal of Biotechnology*, *21*(2).

4. Ahmed, M. E., Shaghaleh, H., Ahmed, A. F., & Alhaj Hamoud, Y. (2026). Eco-Friendly Selenium Nanoparticle Strategy Against *Staphylococcus* Aureus Biofilm Formation. *Applied Biochemistry and Biotechnology*, 1–23.

5. Albalbaki, M. M., Aburayyan, W., Seder, N., Al-Sanabra, O. M., AL-Tahrawe, L., & Shatnawi, M. N. (2024). The correlation of gene mutation of coagulopathy cascade with elevated D-dimer levels in COVID-19 patients. *Journal of Applied Pharmaceutical Science*, *14*(1), 54–63.
6. Al-Khfaji, Z. A., Jalil, B. S., & Mahdi, Z. H. (2025). Assessment of Environmental Contamination with Methicillin-Resistant *Staphylococcus* Aureus at Some Hospitals in Kerbala City, Iraq. *Kerbala Journal of Nursing and Health Sciences.*, *1*(1), 60–72.
7. Al-Nsour, E. H., T AL-Hadithi, H., Al-Groom, R. M., Abushattal, S., Naser, A. Y., Al Nsour, A. H., Sallam, R. A., Kollab, L. M., Alswalha, L., & Khan, M. S. A. (2024). Increased incidence of methicillin resistant *Staphylococcus* aureus and methicillin resistant *Staphylococcus* epidermidis in the skin and nasal carriage among healthcare workers and inanimate hospital surfaces after the COVID-19 pandemic. *Iranian Journal of Microbiology*, *16*(5), 584.
8. AL-Salihi, S. S., Karim, G. F., Al-Bayati, A., & Obaid, H. M. (2023). Prevalence of Methicillin-Resistant and Methicillin Sensitive *Staphylococcus* aureus Nasal Carriage and their Antibiotic Resistant Patterns in Kirkuk City, Iraq. *Journal of Pure & Applied Microbiology*, *17*(1).
9. Al-Tameemi, A. I., Masarudin, M. J., Rahim, R. A., Timms, V., Neilan, B., & Isa, N. M. (2023). Antibacterial properties of zinc oxide nanoparticles synthesized by the supernatant of weissella confusa upm22mt04. *Iraqi Journal of Agricultural Sciences*, *54*(5), 1209–1222.
10. Argudín, M. A., Mendoza, M. C., González-Hevia, M. A., Bances, M., Guerra, B., & Rodicio, M. R. (2012). Genotypes, exotoxin gene content, and antimicrobial resistance of *Staphylococcus* aureus strains recovered from foods and food handlers. *Applied and Environmental Microbiology*, *78*(8), 2930–2935.
11. Aziz, S. N., Huseein, K. A., Mohammed, A. A., & Najm, T. A. (2024). Isolation and Identification of Methicillin-Resistant *Staphylococcus* aureus (MRSA) From Different Sources of Food in Basra Province/Iraq. *Egyptian Journal of Medical Microbiology*, *33*(4), 91–97.

12. Balasubramanian, D., Harper, L., Shopsin, B., & Torres, V. J. (2017). *Staphylococcus* aureus pathogenesis in diverse host environments. *Pathogens and Disease*, *75*(1), ftx005.
13. Baptistao, L. G., Silva, N. C. C., Bonsaglia, E. C. R., Rossi, B. F., Castilho, I. G., Junior, A. F., & Rall, V. L. M. (2016). Presence of immune evasion cluster and molecular typing of methicillin-susceptible *Staphylococcus* aureus isolated from food handlers. *Journal of Food Protection*, *79*(4), 682–686.
14. Bashabsheh, R. H. F., AL-Fawares, O., Natsheh, I., Bdeir, R., Al-Khreshieh, R. O., & Bashabsheh, H. H. F. (2024). *Staphylococcus* aureus epidemiology, pathophysiology, clinical manifestations and application of nano-therapeutics as a promising approach to combat methicillin resistant *Staphylococcus* aureus. *Pathogens and Global Health*, *118*(3), 209–231.
15. Becker, K. (2023). Detection, identification and diagnostic characterization of the staphylococcal small colony-variant (SCV) phenotype. *Antibiotics*, *12*(9), 1446.
16. Bencardino, D., & Vitali, L. A. (2019). *Staphylococcus* aureus carriage among food handlers in a pasta company: pattern of virulence and resistance to linezolid. *Food Control*, *96*, 351–356.
17. Bernardos, A., Piacenza, E., Sancenon, F., Hamidi, M., Maleki, A., Turner, R. J., & Martínez- Máñez, R. (2019). Mesoporous silica- based materials with bactericidal properties. *Small*, *15*(24), 1900669.
18. Bitrus, A. A., Zunita, Z., Bejo, S. K., Othman, S., & Nadzir, N. A. A. (2017). In vitro transfer of methicillin resistance determinants mec A from methicillin resistant *Staphylococcus* aureus (MRSA) to methicillin susceptible *Staphylococcus* aureus (MSSA). *BMC Microbiology*, *17*(1), 83.
19. Bonsaglia, E. C. R., Rossi, B. F., Possebon, F. S., Silva, N. C. C., Gonçalves, J. L., Castilho, I. G., Fernandes Junior, A., Santos, M. V. dos, & Rall, V. L. M. (2025). In Vitro Adhesion and Invasion Rates of *Staphylococcus* aureus Isolated from Mastitic Cows Are Modulated by the agr System and MSCRAMM Genes. *Veterinary Sciences*, *12*(3), 270.
20. Brakstad, O. G., MÆLAND, J. A., & TVETEN, Y. (1993). Multiplex polymerase chain reaction for detection of genes for *Staphylococcus* aureus

thermonuclease and methicillin resistance and correlation with oxacillin resistance. *Apmis*, *101*(7- 12), 681–688.
21. Brawley, D. N., Sauer, D. B., Li, J., Zheng, X., Koide, A., Jedhe, G. S., Suwatthee, T., Song, J., Liu, Z., & Arora, P. S. (2022). Structural basis for inhibition of the drug efflux pump NorA from *Staphylococcus* aureus. *Nature Chemical Biology*, *18*(7), 706–712.
22. Cervera-Alamar, M., Guzmán-Markevitch, K., Žiemytė, M., Ortí, L., Bernabé-Quispe, P., Pineda-Lucena, A., Pemán, J., & Tormo-Mas, M. Á. (2018). Mobilisation mechanism of pathogenicity islands by endogenous phages in *Staphylococcus* aureus clinical strains. *Scientific Reports*, *8*(1), 16742.
23. Cheung, A. L., Schmidt, K., Bateman, B., & Manna, A. C. (2001). SarS, a SarA homolog repressible by agr, is an activator of protein A synthesis in *Staphylococcus* aureus. *Infection and Immunity*, *69*(4), 2448–2455.
24. Cheung, G. Y. C., Bae, J. S., & Otto, M. (2021). Pathogenicity and virulence of *Staphylococcus* aureus. *Virulence*, *12*(1), 547–569.
25. Cheung, G. Y. C., Wang, R., Khan, B. A., Sturdevant, D. E., & Otto, M. (2011). Role of the accessory gene regulator agr in community-associated methicillin-resistant *Staphylococcus* aureus pathogenesis. *Infection and Immunity*, *79*(5), 1927–1935.
26. Das, M., Ojha, A. K., Dolma, K. G., Majumdar, T., Sarmah, P., Hazarika, S., Modi, D., Gogoi, D., Das, S., & Ramamurthy, T. (2025). Monitoring the potential dissemination of antimicrobial resistance in foods, environment, and clinical samples: a one health prospective. *Food Science and Biotechnology*, *34*(3), 803–813.
27. Douglas, E. J. A., Wulandari, S. W., Lovell, S. D., & Laabei, M. (2023). Novel antimicrobial strategies to treat multi- drug resistant *Staphylococcus* aureus infections. *Microbial Biotechnology*, *16*(7), 1456–1474.
28. Elshebrawy, H. A., Kasem, N. G., & Sallam, K. I. (2025). Methicillin- and vancomycin-resistant *Staphylococcus* aureus in chicken carcasses, ready-to-eat chicken meat sandwiches, and buffalo milk. *International Journal of Food Microbiology*, *427*, 110968.

29. Essers, L., & Radebold, K. (1980). Rapid and reliable identification of *Staphylococcus* aureus by a latex agglutination test. *Journal of Clinical Microbiology*, *12*(5), 641–643.
30. Fayisa, W. O., & Tuli, N. F. (2023). Review on *Staphylococcus* aureus. *Int. J. Nurs. Care Res*, *1*, 1–8.
31. Ghareeb, N. H., Obaid, S. S., & Jumaah, I. A. M. (2023). The prevalence of multidrug-resistant *Staphylococcus* aureus isolated from different clinical samples. *Medical Journal of Babylon*, *20*(Supplement 1), S185–S187.
32. Gherardi, G. (2023). *Staphylococcus* aureus infection: pathogenesis and antimicrobial resistance. In *International journal of molecular sciences* (Vol. 24, Number 9, p. 8182). MDPI.
33. Hasanpour, A. H., Sepidarkish, M., Mollalo, A., Ardekani, A., Almukhtar, M., Mechaal, A., Hosseini, S. R., Bayani, M., Javanian, M., & Rostami, A. (2023). The global prevalence of methicillin-resistant *Staphylococcus* aureus colonization in residents of elderly care centers: a systematic review and meta-analysis. *Antimicrobial Resistance & Infection Control*, *12*(1), 4.
34. Huang, F., Shen, T., Hai, X., Xiu, H., Zhang, K., Huang, T., Chen, J., Guan, Z., Zhou, H., & Cai, J. (2022). Clinical characteristics of and risk factors for secondary bloodstream infection after pneumonia among patients infected with methicillin-resistant *Staphylococcus* aureus. *Heliyon*, *8*(12).
35. Hussein, N., Salih, R. S., & Rasheed, N. A. (2019). Prevalence of methicillin-resistant *Staphylococcus* aureus in hospitals and community in Duhok, Kurdistan region of Iraq. *International Journal of Infection*, *6*(2), e89636.
36. Issa, F. A., & Khidhir, M. A. (2023). BACTERIOLOGICAL ANALYSIS OF UNTREATED RETAIL RAW MILK COLLECTED FROM RANDOM SUPPLIERS AT DOHUK GOVERNORATE–KURDISTAN REGION–IRAQ. *Science Journal of University of Zakho*, *11*(3), 376–385.
37. Jafaar, F. A., & Kandala, N. J. (2025). Molecular detection of adhesive matrix molecules for *Staphylococcus* aureus isolated from different samples. *Iraqi Journal of Agricultural Sciences*, *56*(2), 784–798.

38. Jian, Z., Zeng, L., Xu, T., Sun, S., Yan, S., Yang, L., Huang, Y., Jia, J., & Dou, T. (2021). Antibiotic resistance genes in bacteria: Occurrence, spread, and control. *Journal of Basic Microbiology*, *61*(12), 1049–1070.
39. Kebede, M. T., & Getu, A. A. (2023). Assessment of bacteriological quality and safety of raw meat at slaughterhouse and butchers' shop (retail outlets) in Assosa Town, Beneshangul Gumuz Regional State, Western Ethiopia. *BMC Microbiology*, *23*(1), 403.
40. Kerek, Á., Szabó, Á., & Jerzsele, Á. (2025). Antimicrobial susceptibility profiles of *Staphylococcus* aureus and Streptococcus spp. Isolates from clinical cases of waterfowl in Hungary between 2022 and 2023. *Antibiotics*, *14*(5), 496.
41. Khairullah, A. R., Sudjarwo, S. A., Effendi, M. H., Ramandinianto, S. C., Gelolodo, M. A., Widodo, A., Riwu, K. H. P., & Kurniawati, D. A. (2023). Pet animals as reservoirs for spreading methicillin-resistant *Staphylococcus* aureus to human health. *Journal of Advanced Veterinary and Animal Research*, *10*(1), 1.
42. Kinkel, T. L., Roux, C. M., Dunman, P. M., & Fang, F. C. (2013). The *Staphylococcus* aureus SrrAB two-component system promotes resistance to nitrosative stress and hypoxia. *MBio*, *4*(6), 10–1128.
43. Kläui, A. J., Boss, R., & Graber, H. U. (2019). Characterization and comparative analysis of the *Staphylococcus* aureus genomic island v Saβ: an in silico approach. *Journal of Bacteriology*, *201*(22), 10–1128.
44. Kong, C., Neoh, H., & Nathan, S. (2016). Targeting *Staphylococcus* aureus toxins: a potential form of anti-virulence therapy. *Toxins*, *8*(3), 72.
45. Lade, H., Joo, H.-S., & Kim, J.-S. (2022). Molecular basis of non-β-lactam antibiotics resistance in *Staphylococcus* aureus. *Antibiotics*, *11*(10), 1378.
46. Li, Q., Dou, L., Zhang, Y., Luo, L., Yang, H., Wen, K., Yu, X., Shen, J., & Wang, Z. (2024). A comprehensive review on the detection of *Staphylococcus* aureus enterotoxins in food samples. *Comprehensive Reviews in Food Science and Food Safety*, *23*(1), e13264.

47. Linz, M. S., Mattappallil, A., Finkel, D., & Parker, D. (2023). Clinical impact of *Staphylococcus* aureus skin and soft tissue infections. *Antibiotics*, *12*(3), 557.
48. Liu, W.-T., Chen, E.-Z., Yang, L., Peng, C., Wang, Q., Xu, Z., & Chen, D.-Q. (2021). Emerging resistance mechanisms for 4 types of common anti-MRSA antibiotics in *Staphylococcus* aureus: A comprehensive review. *Microbial Pathogenesis*, *156*, 104915.
49. Locke, T. E., Keeley, A. J., Laundy, N., Keil, C., Hamilton, J., Pandor, A., de Silva, T. I., & Darton, T. C. (2025). Prevalence and risk factors for *Staphylococcus* aureus colonisation among healthy individuals in low-and middle-income countries: A systematic review and meta-analysis. *Journal of Infection*, *90*(4), 106462.
50. Loftus, M. J., Young-Sharma, T. E. M. W., Wati, S., Badoordeen, G. Z., Blakeway, L. V, Byers, S. M. H., Cheng, A. C., Jenney, A. W. J., Naidu, R., & Prasad, A. (2022). Epidemiology, antimicrobial resistance and outcomes of *Staphylococcus* aureus bacteraemia in a tertiary hospital in Fiji: a prospective cohort study. *The Lancet Regional Health–Western Pacific*, *22*.
51. Mahros, M. A., Abd-Elghany, S. M., & Sallam, K. I. (2021). Multidrug-, methicillin-, and vancomycin-resistant *Staphylococcus* aureus isolated from ready-to-eat meat sandwiches: An ongoing food and public health concern. *International Journal of Food Microbiology*, *346*, 109165.
52. Mairi, A., Ibrahim, N. A., Idres, T., & Touati, A. (2025). A Comprehensive review of detection methods for *Staphylococcus* aureus and its Enterotoxins in Food: From traditional to emerging technologies. *Toxins*, *17*(7), 319.
53. Mlynarczyk-Bonikowska, B., Kowalewski, C., Krolak-Ulinska, A., & Marusza, W. (2022). Molecular mechanisms of drug resistance in *Staphylococcus* aureus. *International Journal of Molecular Sciences*, *23*(15), 8088.
54. Mondal, R. K., Karmakar, D., Pal, O., & Samanta, S. K. (2024). AVR/I/SSAPDB: a comprehensive & specialised knowledgebase of antimicrobial peptides to combat VRSA, VISA, and VSSA. *World Journal of Microbiology and Biotechnology*, *40*(11), 348.

55. Mustafa, S. N. (2024). Detection of the *Staphylococcus* aureus icA gene Isolated from some clinical cases. *Samarra Journal of Pure and Applied Science*, *6*(4), 150–161.
56. Obaid, W. A. (2025). Molecular characterization of classical enterotoxin genes among *Staphylococcus* aureus Isolates from food handlers in Baghdad City, Iraq. *Iraqi Journal of Science*.
57. Pal, M., Rebuma, T., Regassa, T., & Zende, R. (2024). Methicillin-resistant *Staphylococcus* aureus (MRSA) remains a major threat to public health. *Am J Public Health Res*, *12*(3), 48–53.
58. Park, Y., Sisley, T. A., Stone, M. C., Corrigan, R. M., Vickery, C. R., Gründling, A., & Walker, S. (2025). SpbR controls lipoteichoic acid length by directly inhibiting signal peptidase SpsB in *Staphylococcus* aureus. *Proceedings of the National Academy of Sciences*, *122*(27), e2426464122.
59. Patel, H., & Rawat, S. (2023). A genetic regulatory see-saw of biofilm and virulence in MRSA pathogenesis. *Frontiers in Microbiology*, *14*, 1204428.
60. Prosperi, M., Azarian, T., Johnson, J. A., Salemi, M., Milicchio, F., & Oliva, M. (2019). Unexpected predictors of antibiotic resistance in housekeeping genes of *Staphylococcus* aureus. *Proceedings of the 10th ACM International Conference on Bioinformatics, Computational Biology and Health Informatics*, 259–268.
61. Rallis, D., Atzemoglou, N., Kapetaniou, K., Giaprou, L.-E., Baltogianni, M., & Giapros, V. (2025). Molecular epidemiology clinical Manifestations, decolonization Strategies, and treatment options of Methicillin-Resistant *Staphylococcus* aureus infection in neonates. *Pathogens*, *14*(2), 155.
62. Rasheed, N. A., & Hussein, N. R. (2021). *Staphylococcus* aureus: an overview of discovery, characteristics, epidemiology, virulence factors and antimicrobial sensitivity. *European Journal of Molecular & Clinical Medicine*, *8*(3), 1160–1183.
63. Roy, M. C., Chowdhury, T., Hossain, M. T., Hasan, M. M., Zahran, E., Rahman, M. M., Zinnah, K. M. A., Rahman, M. M., & Hossain, F. M. A. (2024). Zoonotic linkage and environmental contamination of Methicillin-

resistant *Staphylococcus* aureus (MRSA) in dairy farms: A one health perspective. *One Health*, *18*, 100680.

64. Sanad, M. Y., Mohamed, A. G., & Mohamed, A. M. (2025). Investigation on Food Poisoning Outbreak Caused by *Staphylococcus* aureus Enterotoxin A and Incidence of Staphylococcal Food Poisoning in the Kingdom of Bahrain. *Health*, *17*(5), 557–568.

65. Seder, N., Rayyan, W. A., Al-Fawares, M. H. O., & Bakar, A. (2023). Pseudomonas aeruginosa virulence factors and antivirulence mechanisms to combat drug resistance; A systematic review. *Mortality*, *10*(11).

66. Seo, Y.-R., Choi, S., Kim, S., Kang, K., Ro, C., & Hyeon, J.-Y. (2025). Antimicrobial resistance profiles of *Staphylococcus* spp. and Escherichia coli isolated from dogs and cats in Seoul, South Korea during 2021–2023. *Frontiers in Veterinary Science*, *12*, 1563780.

67. Shariati, A., Dadashi, M., Moghadam, M. T., van Belkum, A., Yaslianifard, S., & Darban-Sarokhalil, D. (2020). Global prevalence and distribution of vancomycin resistant, vancomycin intermediate and heterogeneously vancomycin intermediate *Staphylococcus* aureus clinical isolates: a systematic review and meta-analysis. *Scientific Reports*, *10*(1), 12689.

68. Shaw, L. N., Lindholm, C., Prajsnar, T. K., Miller, H. K., Brown, M. C., Golonka, E., Stewart, G. C., Tarkowski, A., & Potempa, J. (2008). Identification and characterization of σS, a novel component of the *Staphylococcus* aureus stress and virulence responses. *PloS One*, *3*(12), e3844.

69. Shoaib, M., Aqib, A. I., Muzammil, I., Majeed, N., Bhutta, Z. A., Kulyar, M. F.-A., Fatima, M., Zaheer, C.-N. F., Muneer, A., & Murtaza, M. (2023). MRSA compendium of epidemiology, transmission, pathophysiology, treatment, and prevention within one health framework. *Frontiers in Microbiology*, *13*, 1067284.

70. Shrestha, S., Riemann, M., Juneja, V. K., & Mishra, A. (2025). Evaluating the Growth of *Staphylococcus* aureus during Slow Cooking of Beef and Turkey Formulations from 10 C to 54.4 C for an Extended Time. *Journal of Food Protection*, *88*(2), 100445.

71. Sigudu, T. T., Oguttu, J. W., & Qekwana, D. N. (2023). Prevalence of *Staphylococcus* spp. from human specimens submitted to diagnostic laboratories in South Africa, 2012–2017. *Southern African Journal of Infectious Diseases*, *38*(1), 477.
72. Sulikowska, M., Marek, A., Jarosz, Ł. S., Pyzik, E., Stępień-Pyśniak, D., & Hauschild, T. (2024). Pathogenic Potential of Coagulase-Positive *Staphylococcus* Strains Isolated from Aviary Capercaillies and Free-Living Birds in Southeastern Poland. *Animals*, *14*(2), 295.
73. Thaker, H. C., Brahmbhatt, M. N., Nayak, J. B., & Thaker, H. C. (2013). Isolation and identification of *Staphylococcus* aureus from milk and milk products and their drug resistance patterns in Anand, Gujarat. *Veterinary World*, *6*(1), 10–13.
74. Tiwari, N., López-Redondo, M., Miguel-Romero, L., Kulhankova, K., Cahill, M. P., Tran, P. M., Kinney, K. J., Kilgore, S. H., Al-Tameemi, H., & Herfst, C. A. (2020). The SrrAB two-component system regulates *Staphylococcus* aureus pathogenicity through redox sensitive cysteines. *Proceedings of the National Academy of Sciences*, *117*(20), 10989–10999.
75. Tong, S. Y. C., Davis, J. S., Eichenberger, E., Holland, T. L., & Fowler Jr, V. G. (2015). *Staphylococcus* aureus infections: epidemiology, pathophysiology, clinical manifestations, and management. *Clinical Microbiology Reviews*, *28*(3), 603–661.
76. Touaitia, R., Mairi, A., Ibrahim, N. A., Basher, N. S., Idres, T., & Touati, A. (2025). *Staphylococcus* aureus: A review of the pathogenesis and virulence mechanisms. *Antibiotics*, *14*(5), 470.
77. Viana, G. G. F., Cardozo, M. V., Pereira, J. G., & Rossi, G. A. M. (2025). Antimicrobial resistant *Staphylococcus* spp., Escherichia coli, and Salmonella spp. in food handlers: a global review of persistence, transmission, and mitigation challenges. *Pathogens*, *14*(5), 496.
78. Wang, H., Hou, X., Shen, J., Wang, W., Ye, Y., Yu, J., & Xue, T. (2022). Alternative sigma factor B reduces biofilm formation and stress response in milk-derived *Staphylococcus* aureus. *Lwt*, *162*, 113515.
79. Wang, Y., Zhang, P., Wu, J., Chen, S., Jin, Y., Long, J., Duan, G., & Yang, H. (2023). Transmission of livestock-associated methicillin-resistant

Staphylococcus aureus between animals, environment, and humans in the farm. *Environmental Science and Pollution Research*, *30*(37), 86521–86539.
80. Yang, L., Dharmaratne, P., Zhu, C., Sapugahawatte, D. N., Rahman, N., Barua, N., Li, C., Kwok, K. O., Luo, M., & Liyanapathirana, V. (2024). Global epidemiology of asymptomatic colonisation of methicillin-resistant *Staphylococcus* aureus in the upper respiratory tract of young children: a systematic review and meta-analysis. *Archives of Disease in Childhood*, *109*(4), 267–274.

المكورات العنقودية الذهبية: نظرة عامة على التصنيف، والوبائيات، والإمراضية، ومقاومة المضادات الحيوية

شهباء حميد مجيد
قسم العلوم، كلية التربية الأساسية، الجامعة المستنصرية، بغداد، العراق.
Shahbaa.edbs@uomustansiriyah.edu.iq.

مستخلص البحث:
تلخص هذه المراجعة التصنيف، والوبائيات، والإمراضية، وعوامل الضراوة، وآليات مقاومة المضادات الحيوية في المكورات العنقودية الذهبية .تُعد هذه البكتيريا مكورات موجبة لصبغة غرام، وتظهر عادةً على شكل تجمعات تشبه عناقيد العنب، كما تمتلك خصائص كيميائية حيوية مميزة مثل إيجابية اختباري الكاتالاز والتخثر. تشير الأدلة الوبائية إلى أن المكورات العنقودية الذهبية تستعمر نسبة ملحوظة من سكان العالم، ولا سيما في التجويف الأنفي. وقد أصبحت المكورات العنقودية الذهبية المقاومة للميثيسيلين مشكلة صحية عامة مهمة بسبب تزايد انتشارها وارتباطها بأمراض إنتانية شديدة، تشمل تجرثم الدم، والالتهاب الرئوي، والتهاب شغاف القلب، والتهاب العظم والنقي، والتهابات الجلد والأنسجة الرخوة. وتُعزى إمراضية المكورات العنقودية الذهبية إلى العديد من عوامل الضراوة، مثل عوامل الالتصاق، والإنزيمات، والسموم، وبروتينات التهرب المناعي، التي تسهّل الاستعمار، وغزو الأنسجة، والتهرب من الاستجابة المناعية، وتكوين الأغشية الحيوية. كما تمتلك هذه البكتيريا أنظمة تنظيمية معقدة، تشمل شبكتي التنظيم sarوagr، التي تتحكم في التعبير عن جينات الضراوة استجابة للظروف البيئية. بالإضافة إلى ذلك، طورت المكورات العنقودية الذهبية آليات متعددة لمقاومة المضادات الحيوية، بما في ذلك إنتاج إنزيمات البيتا-لاكتاماز. إن فهم وبائيات المكورات العنقودية الذهبية وآلياتها الإمراضية واستراتيجيات مقاومتها يُعد ضروريًا لتحسين طرائق التشخيص، واستراتيجيات السيطرة على الأمراض الإنتانية، وتطوير تدخلات علاجية فعالة.

الكلمات المفتاحية : مقاومة المضادات الحيوية، الوبائيات، الإمراضية، المكورات العنقودية الذهبية، عوامل الضراوة، الميثيسيلين.
ملاحظة: هل البحث مستل من رسالة ماجستير او اطروحة دكتوراه؟ كلا