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Review Article

Opportunism Versus True Pathogenicity in *Staphylococcus Epidermidis* is an Ongoing Dilemma Areview Article

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ABSTRACT

Staphylococcus epidermidis is a gram-positive coccus considered as the major skin inhabitant, it is accountable for the contamination challenge in the clinical specimens Interestingly, the ability of *S. epidermidis* to initiate an infection, mostly those associated with medical equipment and instruments such as prosthetic joints, indwelling catheters, and others, is mainly owing to the synthesis of virulence factors such as adhesins, exotoxins, and enzymes as well as their antibiotic resistance. The *S. epidermidis* pathogenicity is amplified by its ability to establish biofilms that gave protection against antimicrobial agents and host defenses. Acquisition awareness of the fundamental mechanisms, which enhance the pathogenicity of *S. epidermidis* has the ability to participate in an inclusive comprehension of its pathogenic nature, permitting its categorizing as either an opportunistic pathogen or true pathogen. Besides, this review may participate in the establishment of innovative tactics for the prevention and management of infections initiated by this particular species.

Keywords: *Staphylococcus epidermidis*, pathogenesis, opportunistic, biofilms.

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INTRODUCTION

Staphylococcus epidermidis is a Gram-positive coccus, which is mainly encountered as a commensal microorganism on the human being's skin and mucosa. Even though *S. epidermidis* is frequently considered a harmless bacterium, it is more likely prone to initiate opportunistic infections, particularly in immunocompromised individuals or patients with implanted medical prosthetics. The aptitude of *S. epidermidis* to produce biofilms is complicatedly related to its emergence as an opportunistic pathogen (Severn and Horswill, 2023).

Staphylococcus epidermidis significantly participates in keeping the harmony and stability of its niche that is principally the human skin. However, our understanding of its precise function in this context is still limited (Grice and Segre, 2011). A local study has shed light on the potential impact of *S. epidermidis* in inhibiting the colonization of *Staphylococcus aureus*, a more pathogenic species, in a dual biofilm (Karam and Al-Mathkhury, 2017). This inhibition is achieved through the expression of a specific serine-type protease known as Esp, which disrupts the colonization mechanisms of *S. aureus*, particularly biofilm formation (Sugimoto *et al.*, 2013). Another local study done by Shaker and Lafta (2018) revealed that *S. epidermidis* accounted for the highest occurrence rate (50.6%) among the isolated bacteria from breast cancer patients. Of interest, Hussein and Luti (2020) utilized viable cells of *S. epidermidis* to cure wound infections caused by pathogenic bacteria, owing this effect to bacteriocin production.

Due to its widespread presence on the skin (AL-Mashaykhi *et al.*, 2020), *S. epidermidis* is frequently encountered as a contaminant in clinical samples. This poses a significant challenge for medical microbiologists who need to reliably differentiate between true invasive isolates and those that are mere contaminants (Mack *et al.*, 2009). Nevertheless, *S. epidermidis* has emerged as one of the most important bacteria associated with hospital-acquired infections (Muhammad and Al-Mathkhury, 2014; Flayyih and Rabaa, 2015, Al-Amara, 2021), highlighting the need for improved methods to accurately discriminate clinically relevant *S. epidermidis* isolates.

According to one viewpoint, the invasive behavior exhibited by *S. epidermidis* is posited to be an inadvertent outcome rather than a deliberate approach. According to this perspective, some mechanisms and traits that commonly facilitate commensalism can unintentionally foster virulence and result in the occurrence of infections, given certain circumstances. This perspective posits that the progression towards a pathogenic state could potentially arise from environmental stimuli or alterations in host-related elements, rather than being a deliberate and preplanned tactic (Otto, 2009). Conversely, one alternative viewpoint posits that *S. epidermidis* exhibits distinct mechanisms and characteristics that facilitate its deliberate transition into a pathogenic state, hence enhancing its capacity for colonisation and the induction of infections. This perspective posits that specific genetic variables and regulatory systems may play a role in the intentional transition towards increased virulence (Severn and Horswill, 2023).

This review seeks to acquire a thorough comprehension of the underlying mechanisms and contributing elements that contribute to the pathogenic potential of *S. epidermidis*, hence substantiating its actual pathogenicity as opposed to being merely accidental or opportunistic.

Staphylococcus epidermidis infections

Urinary tract infections

Urinary tract infections caused by *S. epidermidis* is more frequent in females than males that get entrance to urinary tract via urethra. Such female-prone infections might be taking place due many reasons; among which the presence of *S. epidermidis* on the area surrounding the urethra, the sexual intercourse, shorter female urethra, and high estrogen level (Geerlings, 2016; Mussa and Al-Mathkhury, 2018). Several local studies revealed that *S. epidermidis* is responsible for 4.6 up to 55% of UTI cases especially in females. Fadhel *et al.* (2013) reported that 55% of UTI patients were infected with *S. epidermidis*. (Hindi *et al.*, 2013) found that this pathogen was the causative

agent in 4.6% of UTI cases of honeymoon women. Whereas, Abbas and Al-Mathkhury (2020) stated that *S. epidermidis* was responsible for 12.5% of recurrent UTI cases in Iraqi females.

Catheter-related infections

Catheter-associated bloodstream infections represent a significant contributor to both morbidity and mortality among individuals receiving inpatient care. The occurrence of these infections arises from the colonisation of bacteria on the catheter surface, leading to the formation of a biofilm that exhibits resistance to antibiotic treatment. *Staphylococcus epidermidis* is a primary etiological factor in bloodstream infections associated with catheters, particularly among those with venous catheters in place. In certain instances, germs have the potential to infiltrate the circulatory system, leading to the development of sepsis, a condition that poses a significant risk to an individual's life (Pitiriga *et al.*, 2020). Nevertheless, it is worth noting that bacteria have the ability to cling to a catheter and commence the process of biofilm formation through four distinct pathways. These pathways include direct contact with the catheter, the act of inserting the catheter, and the maintenance of the catheter, as well as sources that are not directly associated with the catheter (Wolcott, 2021).

Locally, *S. epidermidis* was the most identified organism in catheter tip infection as indicated by a cross-sectional study done by Jaudah and Musa (2017) on hemodialysis patients in Basrah General Hospital, Iraq. Similarly, Nuradeen *et al.* (2018) reported that *S. epidermidis* was the most frequent causative agent of catheter-related infections from patients receiving hemodialysis in Sulaimani, Iraq.

Prosthetic joint infections

Staphylococcus epidermidis, *S. aureus*, and *P. aeruginosa* are the primary causative agents of almost three-quarters of bacterial biofilms detected on a range of medical equipment, including catheters, shunts, pacemakers, and orthopedic devices. The bacteria in question have acquired an infamous notoriety due to their capacity to adhere to foreign substances (Visperas *et al.*, 2022). It is worth mentioning that *S. epidermidis*, followed by *S. aureus*, are the primary causal agents responsible for periprosthetic joint infections (Patel, 2023).

Virulence factors

In addition to biofilm formation, *S. epidermidis* can produce a range of virulence factors that contribute to its pathogenicity. These virulence factors include adhesins, exotoxins, enzymes, siderophores, and phenol-soluble modulins (PSMs).

Biofilm

The establishment of biofilms is a significant contributing element to the pathogenicity of *S. epidermidis*. Biofilms are intricate formations consisting of bacterial cells that are encased within a matrix generated by the bacteria themselves. Biofilms possess the ability to protect bacteria from both the human immune system and antibiotics, hence rendering illnesses challenging to manage. *S. epidermidis* biofilms have been associated with catheter-related bloodstream infections and prosthetic joint infections, specifically (Pouget *et al.*, 2023).

The matrix of *S. epidermidis* biofilms is composed of a complex blend of nucleic acids, proteins, and polysaccharides. One of the initial molecules discovered to play a role in *S. epidermidis* biofilm formation is the polysaccharide intercellular adhesin (ica) that is synthesized via the ica operon over time, other factors involved in accumulation of ica and biofilm were indicated, like the accumulation-associated protein (Aap) (Rohde *et al.*, 2005), the small basic protein, and the extracellular matrix-binding protein (Oliveira, *et al.*, 2021).

The penetration of reactive oxygen species, antimicrobial agents, phagocytes, and more is hindered by the *S. epidermidis* biofilm matrix, which partially explains the difficulty in eradicating or recurring infections associated with *S. epidermidis* biofilms. However, the notion that the extracellular polymeric substance (EPS) of biofilm solely represents a physical barricade is

questionable as time passes. Singh *et al.* (2016) indicated that the bactericidal efficacy of various antibiotics of different classes in killing cells of biofilms developed by many bacterial species including *S. epidermidis* is not reliant on penetration. The same authors highlighted one more significant aspect of infections caused by biofilm is evading the immune defenses through various mechanisms. Previously, Cerca *et al.* (2006) documented that the antibodies can diffuse through *S. epidermidis* biofilms without any EPS hindrance. Alternatively, the diffused antibodies can bind to specific receptors within the EPS, reducing the availability of antibodies throughout opsonophagocytic.

Biofilms developed by *S. epidermidis* have the ability to inactivate the complement components, and antimicrobial peptides (Kristian *et al.*, 2008), as well as impairing the activation of macrophage cells. Another important aspect related to biofilms is that cells implementing this growth style display a reduced metabolic rate, resulting in reduced susceptibility to antibiotics that rely on actively growing cells. Additionally, dormant *S. epidermidis* biofilm cells have been attributed to low pro-inflammatory properties (Schommer *et al.*, 2011).

Pouget *et al.* (2023) investigated the characteristics of biofilm development and virulence of a group of *S. epidermidis* strains collected from different clinical sources, interestingly, most of these strains demonstrated reduced virulence possibilities in a Caenorhabditis elegans model and exhibited a limited capability to establish biofilms. Yet, a subset of strains displayed a wide virulence arsenal, challenging the notion that *S. epidermidis* is solely an "accidental pathogen" as formerly believed (Otto, 2009).

Adhesins

Adhesins are a class of proteins that facilitate the attachment of bacteria to both host cells and other surfaces. *Staphylococcus epidermidis* is capable of producing many adhesins, such as the cell-surface protein Aap (accumulation-associated protein) and the extracellular protein Bhp (biofilm-associated protein). Aap plays a crucial role in the formation of biofilms, facilitating the adherence of *S. epidermidis* to various biological surfaces such as tissues and medical devices like catheters and prosthetic joints. Bhp was associated with the production of biofilm and, perhaps, contributes to inhabiting host tissues (Giormezis *et al.*, 2014; Azara *et al.*, 2022).

Exotoxins and enzymes

Numerous exotoxins are elaborated by *Staphylococcus epidermidis*, among which, are delta- and gamma-toxins. The first one has a cytolytic action which stimulates the pore formation in host cells, resulting in cell lysis. Contrariwise, the other toxin can induce apoptosis in host cells. Both of these toxins potentially participated in damaging host tissues as well as inflammation, thereby aggravating *S. epidermidis* infections (Otto, 2009).

Lipases might be defined as carboxylesterases; which stimulate the production and hydrolysis of long-chain acylglycerols (Essamri *et al.*, 1998).

Regarding the extracellular protease of *S. epidermidis* is a serine protease that has the ability to hinders the establishment of *S. aureus* biofilm. In addition, it able to degrade human fibrogen and C5 component of the complement (Vandecandelaere *et al.*, 2014)

Pei and Flock (2001) and Cunha *et al.* (2007) separately reported an elevation in the expression of enterotoxin-encoding genes (*sea* and *sec*) in *S. epidermidis*. Similarly, Giormezis *et al.* (2014) discovered a greater occurrence of tsst-positive isolates among *S. epidermidis* obtained from Greek hospitals.

Enzymes produced by *S. epidermidis* also contribute to its pathogenicity. For example, *S. epidermidis* produces lipases and proteases, which can break down host tissues and contribute to the spread of infection (Martínez-García *et al.*, 2018).

Moreover, Pouget *et al.* (2023) detected the presence of cytotoxins and hemolysins in the strains, although their significance in *S. epidermidis* infections remains unclear. While these toxins have been implicated in the pathogenesis of *S. aureus*, unfortunately, the part played by these toxins

in infections caused by *S. epidermidis* is not well understood. Reports on the presence and expression of cytotoxin-encoding genes, such as *hla* as well as *hld* genes (encoding α- and δ-hemolysin, respectively), in coagulase-negative staphylococci (CoNS) are limited (Okee *et al.*, 2012). α-hemolysin is known to induce hemolysis, dermonecrosis, and neurotoxic effects (Coelho *et al.*, 2009), whereas β- hemolysin displays phosphorylase activity and with a maximum affinity towards various cytoplasmic membranes, leading to membrane instability (Pinheiro *et al.*, 2015). Furthermore, in hospital-acquired infections, *hld* gene was predominant with significant toxigenicity, suggesting a possible action performed by these genes in *S. epidermidis* pathogenicity (de Lastours *et al.*, 2014). However, additional experimental work is required to fully comprehend the various influencing elements contributing to *S. epidermidis* virulence. Outcomes of Pinheiro *et al.* (2015) and those reached by Pouget *et al.* (2023) demonstrated that 91% of the sequenced strains of *S. epidermidis* tested positive for the *hld* gene. Additionally, these results highlight the importance of these genes in establishing *S. epidermidis* as a pathogen alongside probable contribution to the initiation of CoNS-related infections (de Lastours *et al.*, 2014).

Siderophores

In addition to these virulence factors, *S. epidermidis* can also produce other molecules that contribute to its pathogenicity. For example, the bacteria can produce siderophores, which are molecules that act as scavengers of iron from the host. Iron is an essential nutrient for the growth of bacteria, and siderophores allow *S. epidermidis* to obtain iron from the host and grow more effectively (Oliveira *et al.*, 2021).

The notion that systems of iron sequestration in bacteria, mostly siderophores, act as virulence factors is based on the remark stating that when these systems are deactivated, there is a noticeable decrease in virulence (Runci *et al.*, 2019). Holden and Bachman (2015) documented that the involvement of siderophores in other activities besides the acquisition of iron, including influencing metabolic pathways of the host, provides an adaptive benefit against the immunity of the host in addition to its contribution to the pathogenesis of bacteria. However, addressing specific activities to various siderophores would be considered a challenging issue because of their chemical and structural variety, as well as variations in their biosynthesis pathways even among strongly-linked species. Beasley *et al.* (2009) indicated that *S. aureus* elaborates two distinct siderophores (staphyloferrin A and staphyloferrin B); on the other hand, *S. epidermidis* only produces staphyloferrin A (Oliveira *et al.*, 2021). Brozyna *et al.* (2014) demonstrated that *S. lugdunensis* acquires siderophores from other staphylococci rather than elaborating its own.

phenol-soluble modulins

Several virulence genes known to be associated with *S. epidermidis* were found in both commensal and pathogenic strains (Severn and Horswill, 2023). Specifically, Pouget *et al.* (2023) identified the presence of PSMs in all *S. epidermidis* strains examined in their study. PSMs are "pro-inflammatory peptides" can be extracted by hot phenol from the culture supernatant of *S. epidermidis*. PSMs are involved in inflammatory responses and leukocyte lysis (Peschel and Otto, 2013). Additionally, the ESAT-6 secretion system (ESS), consisting of *esaAB*, *esaABC*, and *esaAC* genes, plays a role in circumvention of host immunity and eradication of neutrophils (Even *et al.*, 2010; Wang *et al.*, 2016).

Antibiotic resistance

Several reports stated that *S. epidermidis* developed multidrug resistance. Flayyih and Rabaa (2015) demonstrated that *S. epidermidis* showed total resistance to penicillin and amoxicillin-clavulanic acid, imipenem (95%), and vancomycin (40%). Interestingly, *S. epidermidis* isolated by Shaker and Lafta (2018) from benign and malignant tumors were multi-drug resistant as well as methicillin-resistant. Recently, Hashosh *et al.* (2022) stated that *S. epidermidis* isolated from various locations in Al-Basrah and Al-Muthanna provinces were resistant to cefalexin, ceftriaxone, ampicillin, and vancomycin. Nonetheless, Ferreira *et al.* (2021) indicated that the prevalence of methicillin-resistant *S. epidermidis* was 100%.

CONCLUSION

Staphylococcus epidermidis is a prevalent commensal microorganism found on the skin and mucous membranes of humans. Owing to its arsenal of virulence factor; among which, adhesins, lipase, protease, siderophores, biofilm, and antibiotic resistance, *S. epidermidis* is also capable of causing infections in other anatomical locations, such as catheter associated infections and prosthetic associated infections notably in individuals with implanted medical devices. Hence, it can be classified as a genuine pathogen rather than an incidental one. Treating infections caused by *S. epidermidis* can provide challenges, primarily attributed to the bacteria's capacity to develop biofilms.

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الانتهازية مقابل الامراضية الحقيقية لبكتريا *S. epidermidis* معضلة مستمرة

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الملخص

بكتريا *Staphylococcus epidermidis* هي مكورات موجبة لصبغة غرام وتعد من مستوطنات الجلد الرئيسة وبذلك تكون مسؤولة التلوث الحاصل في العينات السريرية. من المثير للاهتمام، ان قدرة بكتريا *S. epidermidis* على احداث اصابة وخصوصا تلك المرتبطة بالمعدات والاجهزة الطبية مثل المفاصل الصناعية والقثطرة الساكنة وغيرها تعود بشكل اساسي الى تصنيع عوامل الفوعة مثل اللواصق والذيفانات الخارجية والانزيمات وكذلك مقاومة المضادات الحياتية. تتضخم امراضية بكتريا *S. epidermidis* من خلال تكوين الاغشية الحياتية التي تحميها من العوامل المضادة للمايكروبات ودفاعات المضيف. اكتساب الوعي بالآليات الاساسية التي تعزز من امراضية بكتريا *S. epidermidis* كان له القدرة في المساهمة في الفهم الشامل للطبيعة الممرضة لهذه البكتريا، مما يسمح بتصنيفها الى ممرضات انتهازية وممرضات حقيقية. علاوة على ذلك، مقال المراجعة هذا قد يساهم في ارساء تكتيكات مبتكرة للوقاية ومنع العدوى التي يسببها هذا النوع بالذات.

الكلمات الدالة: المكورات العنقودية، الامراضية، الانتهازية، الاغشية الحياتية.