



# Staphylococcus Epidermidis: Friend Or FOE: A Commensal Bacterium but Highly Opportunistic Pathogen: A Review

Dr. Reena Kulshrestha<sup>1</sup>, Mrs. Pooja Sitholay<sup>2</sup>, Ms. Sangita Roy<sup>3</sup>

<sup>1</sup> Research Scholar, Medical Department, Rungta College of Dental Sciences and Research, Bhilai

<sup>2</sup> Lecturer, Department of Biochemistry, Rungta College of Dental Sciences and Research, Bhilai

<sup>3</sup> Lecturer, Department of Physiology, Rungta College of Dental Sciences and Research, Bhilai,

## ABSTRACT

*Staphylococcus epidermidis* is a Gram-positive bacterium that is a predominant component of the normal human skin and mucous membrane microbiota. While generally benign in its natural habitat, it has emerged as a significant opportunistic pathogen, particularly in healthcare settings. This review explores the dual nature of *S. epidermidis*, focusing on its importance as a commensal organism, its pathogenic potential, mechanisms of infection, and the challenges it poses in clinical environments.

## BACKGROUND

*Staphylococcus epidermidis* which is a ubiquitous microorganism, primarily found on the surface of skin and mucosal surfaces of humans and animals. It is usually harmless and even beneficial, contributing to the skin's defense mechanisms against pathogenic microorganisms. However, under certain conditions, particularly in immune-compromised individuals or those having medical devices implanted in their body, *S. epidermidis* can become a formidable pathogen.

## OBJECTIVES

This review is focused to discuss the dual nature of *S. epidermidis* as a commensal and a pathogen. To examine the factors that contribute to its pathogenicity; we reviewed current diagnostic, preventive and therapeutic strategies of *S. epidermidis*.

**KEY WORDS:** pathogen, immuno – compromised, commensal, implants, antibiotics, biofilm

## INTRODUCTION

A gram positive, non-motile, non – spore forming, belonging to Micro-cocceae family - *Staphylococcus epidermidis* is the commensal of surface of human skin, *Staphylococcus epidermidis* are usually found on skin, may turn opportunistic pathogens especially in Immuno-compromised patients or patients with intravascular catheters, implants, prosthetic devices, etc<sup>1,2,3</sup>. *S. epidermidis* forms Biofilm very efficiently as a defense mechanism, which helps in protection of the bacteria from any kind of environmental stress, deleterious agents and antibiotics. As a result of this Biofilm mechanism, this layer contains bacterial cell lining that allows the bacteria to lower the metabolism that results in antibiotic tolerance, which eventually leads to failure of antibiotic treatment.<sup>1,4</sup> Being a Commensal Organism, its importance in the Human Microbial flora is specific in maintaining skin health by:<sup>4,5,6</sup>

1) Competing with the pathogenic bacteria for space and resources.



- 2) Producing peptides of antimicrobial nature that restricts the growth of harmful microbes.
- 3) Modulating the immune responses of the host to prevent excessive inflammation.

## **BENEFITS TO THE HOST SKIN BARRIER FUNCTION:**

- 1) *S. epidermidis* aids to strengthen the skin barrier by reducing permeability and preventing the entry of pathogens.<sup>7,8,9</sup>

## **Immune Modulation:**

It stimulates the production of host defense molecules, enhancing the skin's immune response.<sup>10,11,12</sup>

## **S. EPIDERMIDIS AS AN OPPORTUNISTIC PATHOGEN - PATHOGENIC MECHANISMS—<sup>14,15,16</sup>**

*S. epidermidis* can cause infections when the skin barrier is breached; it also causes infection in immune-compromised individuals.

The key mechanisms behind this includes:

- 1) **Biofilm Formation:** The most crucial factors in its pathogenicity is the capacity to form biofilms on medical devices like catheters, prosthetic joints, heart valves, etc. Biofilms helps in protecting the bacteria from the host immune response against it and also antibiotics.<sup>15,16,17</sup>
- 2) **Surface Proteins and Adhesins:** These facilitate adhesion to host tissues and medical devices.
- 3) **Exopolysaccharide Production:** This helps in the creation and maintenance of biofilms.

## **INFECTIONS CAUSED DUE TO COLONIZATION OF S. EPIDERMIDIS:<sup>17,18,19</sup>**

- 1) **Nosocomial Infections:** *S. epidermidis* plays a leading role in hospital-acquired infections, particularly with patients loaded with implanted medical devices.
- 2) **Bacteremia:** It can enter the bloodstream, leading to severe infections, especially in immune-compromised patients.
- 3) **Endocarditis:** Infection of heart valves, particularly in prosthetic valves patients.

## **CLINICAL CHALLENGES : DIAGNOSIS : LABORATORY IDENTIFICATION:<sup>20,21</sup>**

*S. epidermidis* is often identified through blood cultures and tissue samples, so distinguishing between contamination and true infection can be challenging as it a commensal organism too.

### **Host range**

*S. epidermidis* infects patients showing immune-deficiency diseases that may be inherited or acquired. This includes patients undergoing immune-suppressive therapy, cancer patients, HIV patients, infants having low-birth-weight (<1500 g) and patients suffering from burn. Also, it infects individuals having any type of indwelling medical device.<sup>22,23</sup>

### **Transmission<sup>24,25</sup>**

*S. epidermidis* is transmitted through contact and surroundings from one person to another, especially during hospital visits.

### **Infection<sup>26,27</sup>**

*S. epidermidis* is very low in virulence, but is an opportunistic pathogen that has capacity to penetrate the epithelial layer in immune-compromised persons. *S. epidermidis* produces some extracellular enzymes and enterotoxins that can lead to tissue damage.



## Epidemiology<sup>28,29</sup>

*S. epidermidis* is seen associated with patients having permanent implants (e.g. prosthetic joints, vascular grafts, cardiac devices) but it also depends on type of implant, its location, co-morbidities and immunological status of the patient. In some temporary implants, such as venous catheters, the infection rate usually reaches cent percent over time. These infections may lead to prolonged hospitalization, additional surgery and increased mortality.

## Diagnosis

Molecular Methods: Advanced techniques like PCR and sequencing can aid in accurate identification.<sup>30,31</sup>

## TREATMENT:<sup>32,33,34,35</sup>

**Antibiotic Resistance:** *S. epidermidis* has developed resistance to many antibiotics, including methicillin, leading to the rise of methicillin-resistant *Staphylococcus epidermidis* (MRSE).

**Biofilm-Related Resistance:** Biofilms complicate treatment due to their inherent resistance to antibiotics and immune clearance.

**Novel Therapies:** Research is ongoing into alternative treatments, including biofilm-disrupting agents, phage therapy and immunotherapy.

## PREVENTION:

Due to the widespread emergence of antibiotic resistance associated with increased use of medical devices forms a challenge for current treatment strategies<sup>1,4</sup>. Vaccination and decolonization are failure in case of *S. epidermidis*.

Preventing *S. epidermidis* infections therefore involves:

- (1) disinfection of patient skin and sterilization of medical equipment prior to interventions; (2) regular change of temporary devices;
- (3) elimination of unnecessary contact with indwelling devices during surgery;
- (4) administration of prophylactic broad-spectrum antibiotics 60–30 min before insertion of permanent devices
- (5) appropriate usage of antimicrobial-loaded devices and topical antibiotics (such as catheter lock solutions).

Infection Control Practices: Strict adherence to hygiene and sterilization protocols in healthcare settings can reduce the incidence of *S. epidermidis* infections.<sup>18,24</sup>

**Device Coatings:** Development of anti-biofilm and antimicrobial coatings for medical devices shows efficiency in preventing infection.<sup>9,15</sup>

## CONCLUSION:

*Staphylococcus epidermidis* exemplifies the duality of commensal bacteria that can become opportunistic pathogens under certain conditions. Its ability to form biofilms and develop antibiotic resistance poses significant challenges in clinical settings.<sup>1,7</sup> Understanding its pathogenic mechanisms and developing effective diagnostic, therapeutic, and preventive strategies are crucial in managing infections caused by this adaptable microorganism.<sup>34,35</sup> This review provides a comprehensive overview of *S. epidermidis*, highlighting its complex role in human health and disease. Further research is essential to develop new strategies to combat infections caused by this adaptable bacterium.



## REFERENCES:

1. Otto, M. (2009). Staphylococcus epidermidis—the 'accidental' pathogen. *Nature Reviews Microbiology*, 7(8), 555-567.
2. Vuong, C., & Otto, M. (2002). Staphylococcus epidermidis infections. *Microbes and Infection*, 4(4), 481-489.
3. Fey, P. D., & Olson, M. E. (2010). Current concepts in biofilm formation of Staphylococcus epidermidis. *Future Microbiology*, 5(6), 917-933.
4. Kulshrestha Reena, Jayant Biswas, Soudeep Sau, Surendra Singh. The Story of Disease: An Altered History. *National Journal of Medical and Allied Sciences*: 2013. Volume2: Issue1:49-54
5. Becker, K., Heilmann, C., & Peters, G. (2014). Coagulase-negative staphylococci. *Clinical Microbiology Reviews*, 27(4), 870-926.
6. Rupp, M. E., & Archer, G. L. (1994). Coagulase-negative staphylococci: pathogens associated with medical progress. *Clinical Infectious Diseases*, 19(2), 231-243.
7. Winslow CE, Winslow AR. The Systematic Relationships of the Coccaceae: With a Discussion of the Principles of Bacterial Classification. New York: N Y John Wiley Sons; 1908. pp. 1–300.
8. Kulshrestha Reena, Verma C M, Srinivasa T S. Preventing Nosocomial Infection. *Indian Journal of Dental Research & Review*. 2012, Vol 2, Issue 1.42-43
9. Easmon, CSF and Adlam, C 1983. Staphylococci and Staphylococci Infections, London: Academic Press
10. Marrack, P and Kapple, J 1990. Staphylococcal enterotoxins and their relatives. *Science* 24, 705
11. Fairbrother RW. Coagulase production as a criterion for the classification of the staphylococci. *Journal of Pathology* 1940;50:83–88.
12. Cole K, Atkins B, Llewelyn M, Paul J. Genomic investigation of clinically significant coagulase-negative staphylococci. *Journal of Med Microbiology* 2021;70.
13. The Human Microbiome Project Consortium. Structure, function and diversity of the healthy human microbiome. *Nature* 2012;486(7402):207–214.
14. Rupp ME. Clinical characteristics of infections in humans due to Staphylococcus epidermidis. *Methods in Molecular Biology* 2014;1106:1–16.
15. Zimmerli W, Trampuz A, Ochsner PE. Prosthetic-joint infections. *New England Journal of Medicine* 2004;351:1645–1654.
16. Espadinha D, Sobral RG, Mendes CI, Méric G, Sheppard SK, et al. Distinct phenotypic and genomic signatures underlie contrasting pathogenic potential of Staphylococcus epidermidis clonal lineages. *Frontiers in Microbiology* 2019;10:1971.
17. Conlan S, Mijares LA, Becker J, Blakesley RW, Bouffard GG, et al. Staphylococcus epidermidis pan-genome sequence analysis reveals diversity of skin commensal and hospital infection-associated isolates. *Genome Biology* 2012;13:R64.
18. Lin S, Sun B, Shi X, Xu Y, Gu Y, et al. Comparative genomic and pan-genomic characterization of Staphylococcus epidermidis from different sources unveils the molecular basis and potential biomarkers of pathogenic strains. *Frontiers in Microbiology* 2021;12:770191.
19. Kulshrestha Reena, Biswas Jayant, Srinivas T S, et al. Role of Immunoglobulin G & A in Periodontitis: A Review. *Journal of Pure & Applied Microbiology*: 2013. Issue 1: Volume 7. 673 – 676
20. Paharik AE, Horswill AR. The Staphylococcal Biofilm: Adhesins, Regulation, and Host Response. In:

**Corresponding Author: Dr. Reena Kulshrestha**  
[reena.kulshreshtha@rungtacolleges.com](mailto:reena.kulshreshtha@rungtacolleges.com)

**Volume 02 Issue 01 (January) 2025**



- Kudva IT and Nicholson TL(eds). Microbiology Spectrum. 2016 March 25;4(2):4.2.06.
21. Tokars JJ. Predictive value of blood cultures positive for coagulase-negative staphylococci : implications for patient care and health care quality assurance. Clinical Infectious Diseases 2004;39:333–341.
  22. McNally M, Sousa R, Wouthuyzen-Bakker M, Chen AF, Soriano A, et al. The EBJIS definition of periprosthetic joint infection: a practical guide for clinicians. Bone Joint Journal 2021;103-B(1):18–25.
  23. Trampuz A, Zimmerli W. Antimicrobial agents in orthopaedic surgery: prophylaxis and treatment. Drugs 2006; 66:1089–1105.
  24. Lee JYH, Monk IR, Gonçalves da Silva A, Seemann T, Chua KYL, et al. Global spread of three multidrug-resistant lineages of Staphylococcus epidermidis. Nature Microbiology 2018;3: 1175–1185.
  25. Otto M. Coagulase-negative staphylococci as reservoirs of genes facilitating MRSA infection: Staphylococcal commensal species such as Staphylococcus epidermidis are being recognized as important sources of genes promoting MRSA colonization and virulence. Bioessays 2013; 35:4–11.
  26. Otto M. Staphylococcal biofilms. Microbiology spectrum 2018;6.
  27. Otto M. Staphylococcus epidermidis pathogenesis. In: Fey PD(eds). Staphylococcus Epidermidis: Methods and Protocols. Totowa, New Jersey: Humana Press; 2014. pp. 17–31. [https://doi.org/10.1007/978-1-62703-736-5\\_2](https://doi.org/10.1007/978-1-62703-736-5_2)
  28. Kulshrestha Reena, Srinivas T S, Biswas Jayant, et al. Periodontopathogens: Bacteriology of Periodontal Disease: Mini Review: Journal of Pure & Applied Microbiology 2013. Issue 1: Volume 7. 583- 586
  29. Banaszekiewicz S, Calland JK, Mourkas E, Sheppard SK, Pascoe B, et al. Genetic diversity of composite enterotoxigenic Staphylococcus epidermidis pathogenicity islands. Genome Biology and Evolution 2019;11:3498–3509.
  30. Dong Y, Speer CP, Glaser K. Beyond sepsis: Staphylococcus epidermidis is an underestimated but significant contributor to neonatal morbidity. Virulence 2018;9:621–633.
  31. Pickard JM, Zeng MY, Caruso R, Núñez G. Gut microbiota: role in pathogen colonization, immune responses, and inflammatory disease. Immunology Review 2017;279:70–89.
  32. Correa-Martinez CL, Tönnies H, Froböse NJ, Mellmann A, Kampmeier S. Transmission of vancomycin-resistant enterococci in the hospital setting: uncovering the patient -environment interplay. Microorganisms 2020;8:203.
  33. Ventola CL. The antibiotic resistance crisis. Pharmacology Therapeutics 2015;40:277–283.
  34. Kamada N, Kim Y-G, Sham HP, Vallance BA, Puente JL, et al. Regulated virulence controls the ability of a pathogen to compete with the gut microbiota. Science 2012;336:1325–1329.
  35. Narang Deepak, Kulshrestha Reena, Khan Fatima, et al. Microbes in forensic medicine: A microbiologist perspective. International Journal of Bioassays: 2016. Issue 10: Volume 5. 4913-4919