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Bacterial Colonization on Human Skin

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Abstract

Bacterial surface colonizers are depended to a variety of physical stresses. During the colonization of human epithelia such as on the skin or the elementary canal, bacteria mainly have to suffer the mechanical stress of being removed by fluid flow, discarding, or epithelial turnover.

To that end, they express a series of molecules to establish firm attachment to the epithelial surface, such as febrile projections which is also called pile and surface-anchored proteins that bind to human matrix proteins. In addition, some bacteria-in particular gut and urinary tract pathogens-use internalization by epithelial cells and other methods such as directed inhibition of epithelial turnover to ascertain continued association with the epithelial layer. Furthermore, many bacteria produce multilayered collections called biofilms with a sticky extracellular matrix, providing additional protection from removal.

It will give an overview over the mechanisms human bacterial colonizers have to withstand physical stresses with a focus on bacterial adherence. Staphylococci are the most abundant skin-colonizing bacteria and the most important causes of nosocomial infections and community-associated skin infections. Molecular determinants of staphylococcal skin colonization include surface polymers and proteins that promote fixing and accumulation, and a wide variety of mechanisms to avoid received and natural host defenses.

Keywords: Physical stresses; Colonization of human epithelia; Internalization; Staphylococcal skin colonization

Introduction

Antimicrobial peptides (AMPs) likely play a central role in providing immunity to bacterial colonization on human epithelia. Recent research has shown that staphylococci have a broad depository to combat AMP activity, and can regulate expression of AMP-resistance mechanisms depending on the presence of AMPs. While direct in vivo evidence is still lacking, this suggests that the interplay between AMPs and AMP resistance mechanisms during evolution had a crucial role in rendering staphylococci efficient colonizers of human skin. The microorganisms that inhabit hospitals may influence patient recovery and outcome, although the complexity and diversity of these bacterial communities can confound our ability to focus on potential pathogens in isolation.

To develop a community-level understanding of how microorganisms colonize and move through the hospital environment, we characterized the bacterial dynamics among hospital surfaces, patients, and staff over the course of 1 year as a new hospital became operational. The bacteria in patient rooms, particularly on bedrails, consistently resembled the skin micro biota of the patient occupying the room. Bacterial communities on patients and room surfaces became increasingly similar over the course of a patient's stay [1].

Temporal correlations in community structure demonstrated that patients initially acquired room-associated taxa that predated their stay but that their own microbial signatures began to influence the room community structure over time. The α- and β-diversity of patient skin samples were only weakly or non-significantly associated with clinical factors such as chemotherapy, antibiotic usage, and surgical recovery and no factor except for ambulatory status affected microbial similarity between the micro biotas of a patient and their room. Metagenomes analyses revealed that genes conferring antimicrobial resistance were consistently more abundant on room surfaces than on the skin of the patients inhabiting those rooms. In addition, persistent unique genotypes of Staphylococcus and Propionibacterium were identified. Dynamic Bayesian network analysis suggested that hospital staff were more likely to be a source of bacteria on the skin of patients than the reverse but that there were no universal patterns of transmission across patient rooms [2].

The skin is the human body's largest organ, colonized by a diverse milieu of microorganisms, most of which are harmless or even beneficial to their host. Colonization is driven by the ecology of the skin surface, which is highly variable depending on topographical location, endogenous host factors and exogenous environmental factors. The cutaneous innate and adaptive immune responses can modulate the skin micro biota, but the micro biota also functions in educating the immune system. The development of molecular methods to identify microorganisms has led to an emerging view of the resident skin bacteria as highly diverse and variable. An enhanced understanding of the skin micro biome is necessary to gain insight into microbial involvement in human skin disorders and to enable S.epidermis were isolated from human skin and subjected to a taxonomic study. Amended descriptions of S. epidermis's and S. saprophytic we are also given. The main characters for the distinction of staphylococci and micrococci are mentioned. Staphylococci were classified on the basis of cell wall composition, lactic acid configuration, and a variety of morphological and physiological characters. There are some key differential characters of these species which can be determined by simple laboratory procedures. The failure to ferment trehalose and manifold is typical for S. epidermises.

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Methodology

Fermentation of xylose

The fermentation of xylose and/or arabinose is a characteristic of S. xylosus. The failure to ferment sucrose and Tura nose is typical for S. Connie. Strains of S. saprophyticus do not reduce nitrate, but most of them produce acetylmethylcarbinol and ferment xylitol. S. haemolyticus is usually hemolysis positive, like S. aurous, but it does not produce coagulase, does not have strong phosphatase and deoxyribonuclease activities, and does not ferment mannose. Staphylococcus epidermis is the most important member of the coagulase-negative staphylococci and one of the most abundant colonizers of human skin. While for a long time regarded as innocuous, it has been identified as the most frequent cause of device-related infections occurring in the hospital setting and is therefore now recognized as an important opportunistic pathogen.

S.epidermis produces a series of molecules that provide protection from host defenses. Specifically, many proteins and exopolymers, such as the exopolysaccharide PIA, contribute to biofilm formation and inhibit phagocytosis and the activity of human antimicrobial peptides. Furthermore, recent research has identified a family of pro-inflammatory peptides in, the phenol-soluble modulins (PSMs), which have multiple functions in immune evasion and biofilm development, and may be catalytic [3].

However, in accordance with the relatively benign relationship that *S.* epidermises have with its host, production of aggressive members of the PSM family is kept at a low level. Interestingly, in contrast to *S.* aurous with its large arsenal of toxins developed for causing infection in the human host, most if not all "virulence factors" of *S.* epidermises appear to have original functions in the commensal lifestyle of this bacterium. The opportunistic human pathogen Staphylococcus epidermises have become the most important cause of nosocomial infections in recent years. Its pathogenicity is mainly due to the ability to form biofilms on indwelling medical devices. In a biofilm, *S.* epidermises is protected against attacks from the immune system and against antibiotic treatment, making *S.* epidermises infections difficult to eradicate. The coagulase-negative staphylococci and, in particular, Staphylococcus epidermises, have emerged as major nosocomial pathogens associated with infections of implanted medical devices.

These organisms, which are among the most prevalent bacteria of the human skin and mucous membrane micro flora, present unique problems in the diagnosis and treatment of infections involving biofilm formation on implanted biomaterials. Epidemiological data that address whether invasive S. epidermises strains can be traced to commensal organisms or an endemic occurrence of distinct strains with enhanced virulence have important implications for the implementation of appropriate infection control measures. An extracellular polysaccharide adhesion represents a key virulence determinant in S. epidermises and is required for biofilm formation [4].

Polysaccharide adhesion synthesis

Recent advances in understanding the molecular events controlling polysaccharide adhesion synthesis and the potential clinical implications of its phase variable regulation are outlined. Further research in this area may contribute to the development of novel strategies for therapeutic intervention. Finally, in addition to antibiotic prophylaxis, preventive strategies to control S. epidermises medical device-related infections are focusing on the development of improved biomaterials and physical electrical barriers to impede bacterial colonization. Functioning as the exterior interface of the human body with the environment, skin acts as a physical barrier to prevent the invasion of foreign pathogens while providing a home to the commensal micro biota. The harsh physical landscape of skin, particularly the desiccated, nutrient-poor, acidic environment, also contributes to the adversity that pathogens face when colonizing human skin [5].

Despite this, the skin is colonized by a diverse micro biota. In this Review, we describe amp icon and shotgun metagenomes DNA sequencing studies that have been used to assess the taxonomic diversity of microorganisms that are associated with skin from the kingdom to the strain level. We discuss recent insights into skin microbial communities, including their composition in health and disease, the dynamics between species and interactions with the immune system, with a focus on Propionibacterium acnes, Staphylococcus epidermises and Staphylococcus aurous. Staphylococcus epidermis is a biofilm-producing commensal organism found ubiquitously on human skin and mucous membranes, as well as on animals and in the environment. Biofilm formation enables this organism to evade the host immune system [6].

Colonization of percutaneous devices or implanted medical devices allows bacteria access to the bloodstream. Isolation of this organism from blood cultures may represent either contamination during the blood collection procedure or true bacteremia. *S.* epidermises bloodstream infections may be indolent compared with other bacteria. Isolation of *S.* epidermises from a blood culture may present a management quandary for clinicians. Over-treatment may lead to patient harm and increases in healthcare costs. There are numerous reports indicating the difficulty of predicting clinical infection in patients with positive blood cultures with this organism. No reliable phenotypic or genotypic algorithms currently exist to predict the pathogenicity of a S.epidermis bloodstream infection. This review will discuss the latest advances in identification methods, global population structure, pathogenicity, biofilm formation, antimicrobial resistance and clinical significance of the detection of S.epidermis in blood cultures.

Discussion

Previous studies that have attempted to discriminate between invasive and contaminating strains of S.epidermis in blood cultures will be analyzed. Staphylococcus aurous is both a major bacterial pathogen as well as a common member of the human skin micro biota. Due to its widespread prevalence as an asymptomatic skin colonizer and its importance as a source of skin and soft tissue infections, an improved understanding of how S. aurous attaches to, grows within, and breaches the stratified layers of the epidermis is of critical importance. Three-dimensional organotypic human skin culture models are informative and tractable experimental systems for future investigations of the interactions between S. aurous and the multifaceted skin tissue. It proposes that S. aurous virulence factors, primarily appreciated for their role in pathogenesis of invasive infections, play alternative roles in promoting asymptomatic bacterial growth within the skin [7].

Experimental manipulations of these cultures will provide insight into the many poorly understood molecular interactions occurring at the interface between S. aurous and stratified human skin tissue. Current research on the complex interplay between the micro biota, the barrier function and the innate immune system of the skin indicates that the skin's micro biota have a beneficial role, much like that of the gut micro flora. As a consequence, interest in strategies beyond

antibiotic that allow a more selective modulation of the skin micro flora is constantly growing. This review wills briefly summarize our current understanding of the cutaneous micro biota and summarize existing information on pre- and probiotic strategies for skin.

Commensal organisms that constitute the skin micro biota play a pivotal role in the orchestration of cutaneous homeostasis and immune competence. This balance can be promptly offset by the expansion of the opportunistic pathogen Staphylococcus aurous, which is responsible for the majority of bacterial skin infections. *S. aurous* carriage is also known to be a precondition for its transmission and pathogenesis. Recent reports suggest that skin-dwelling coagulase-negative staphylococci (CoNS) can prime the skin immune system to limit the colonization potential of invaders, and they can directly compete through production of antimicrobial molecules or through signaling antagonism. This transition enables the investigation of the full diversity of microorganisms inhabiting human skin. The skin provides a range of habitats with different micro biota associated with the three major regions of the skin, namely the moist axilla, perineum, and toe webs; oily or sebaceous head, neck, and trunk; and dry forearm and legs [8].

These new culture-independent tools are revealing the diversity of the human skin micro biota in the different locations of the body and with skin depth. These tools should lead to a better understanding of the state of homeostasis between the micro biota and the host and the overall functionality of that micro biota. Staphylococcus epidermises are a usually harmless symbiotic bacterium highly abundant on the human skin. Under defined reactivating conditions, most importantly implantation of a medical device, S. epidermises, however, can switch from a colonizing to a presumptuous life style. The emergence of S. epidermises as an opportunistic pathogen is closely linked to the biofilm forming capability of the species. During the past decades, tremendous advance regarding our understanding of molecular mechanisms contributing to surface colonization has been made, and detailed information is available for several factors active during the priory attachment, accumulative or dispersal phase of biofilm formation.

Conclusion

A picture evolved in which different factors, though appearing to be unnecessarily organized, take over specific and exclusive functions during biofilm development. In this review, these mechanisms are described in molecular detail, with a highlight on recent insights into multi-functional S. epidermises cell surface proteins contributing to surface adherence and intercellular adhesion. The integration of distinct biofilm-promoting factors into regulatory networks is summarized, with an emphasis on mechanism that could allow S. epidermises to flexibly adapt to changing environmental conditions present during colonizing or invasive life-styles. S.epidermis is a ubiquitous commensal of human skin. The widespread use of inhabiting medical devices in modern medicine provides an opportunity for it to cause infections. Disease causing isolates can come from many different genetic backgrounds. Multiply antibiotic resistant strains have spread globally. S. epidermises has a smaller repertoire of cell wall anchored (CWA) surface proteins than S. aurous. Nevertheless these CWA proteins promote adhesion to components of the extracellular matrix including collagen, fibrinogen and fibronectin, and to the formation of biofilm. The A domain of the accumulation associated protein can promote adhesion to unconditioned biomaterial but must be removed proteolytically to allow accumulation to proceed by hemophilic Zn2+ dependent interactions. Mature biofilm contain starchy structures formed by the small basic protein. The latter contribute to the integrity of both protein and polysaccharide biofilm matrices. Several other CWA proteins are also involved in S. epidermises biofilm formation.

Acknowledgement

None

Conflict of Interest

None

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