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Bacterial Biofilm And Role In The Pathogenesis Of Disease

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ABSTRACT

Background: *Bacterial biofilms may play a role in pathogenesis which has led to increased focus on identifying diseases that may be associated with biofilms. Chronic biofilm infections are usually chronic in nature, as the bacteria that live in biofilms can be resilient to both the immune system and antibiotics and other treatments. Current knowledge of how biofilms contribute to disease pathogenesis suggests a number of different mechanisms. This extends from biofilms being merely a reservoir of pathogenic bacteria, to playing a more active role, for example, by contributing to inflammation. This knowledge is important for developing effective treatment strategies for such infections.*

Keywords: **Biofilm, Pathogenesis, Infectious Diseases, Quorum Sensing.**

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INTRODUCTION

Bacterial biofilms are groups of bacteria bound to a surface and or to each other and embedded in a self-producing matrix. The matrix of biofilms is made up of substances such as proteins, polysaccharides, as well as RNA. Bacteria in biofilms can use many survival strategies to evade host defense systems. By remaining dormant and hidden from the immune system, you may damage local tissues and subsequently cause severe infection. Inside the biofilm [1,2].

Bacteria consider biofilms as one of the reasons for their survival and facing environmental conditions, the lifestyle of microorganisms of microorganisms was not of interest to medical microbiologists until the early 1970s when Niels Hobe noticed a link between persistent infections and bacterial populations in cystic fibrosis patients [3]. As biofilms have been identified as being involved in many clinical infections [4,5], and evidence is accumulating that biofilms contribute to pathogenesis, particularly in chronic infections [6]. These modifications make bacteria more resistant to antimicrobial therapy by disrupting antimicrobial targets or reducing the demands of cellular function that antimicrobials interfere with. inconsistently with antimicrobial treatments [7].

Origin and formation of biofilms

Biofilms are thought to have arisen during primitive Earth as a defense mechanism for prokaryotes, as the conditions at that time were too harsh for their survival. They can be found very early in Earth's fossil records as both Archaea and Bacteria, and commonly protect prokaryotic cells by providing them with homeostasis, encouraging the development of complex interactions between the cells in the biofilm.[8]The formation of a biofilm begins with the attachment of free-floating microorganisms to a surface.[9.10] The first colonist bacteria of a biofilm may adhere to the surface initially by the weak van der Waals forces and hydrophobic effects.[11.12 If the colonists are not immediately separated from the surface, they can anchor themselves more

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permanently using cell adhesion structures such as pili. A unique group of Archaea that inhabit anoxic groundwater have similar structures called hami. Each hamus is a long tube with three hook attachments that are used to attach to each other or to a surface, enabling a community to develop.[13][14] Hyperthermophilic archaeon Pyrobaculum calidifontis produce bundling pili which are homologous to the bacterial TasA filaments, a major component of the extracellular matrix in bacterial biofilms, which contribute to biofilm stability.[15] TasA homologs are encoded by many other archaea, suggesting mechanistic similarities and evolutionary connection between bacterial and archaeal biofilms.[16] .Some bacteria species are not able to attach to a surface on their own successfully due to their limited motility but are instead able to anchor themselves to the matrix or directly to other, earlier bacteria colonists. Non-motile bacteria cannot recognize surfaces or aggregate together as easily as motile bacteria.[17]During surface colonization bacteria cells are able to communicate using quorum sensing (QS) products such as N-acyl homoserine lactone (AHL). Once colonization has begun, the biofilm grows by a combination of cell division and recruitment. Polysaccharide matrices typically enclose bacterial biofilms. The matrix exopolysaccharides can trap QS autoinducers within the biofilm to prevent predator detection and ensure bacterial survival.[18]

Infectious diseases

Biofilms have been found to be involved in a wide variety of microbial infections in the body, by one estimate 80% of all infections.[19] Infectious processes in which biofilms have been implicated include common problems such as bacterial vaginosis, urinary tract infections, catheter infections, middle-ear infections, formation of dental plaque,[20] gingivitis, coating contact lenses,[21] and less common but more lethal processes such as endocarditis, infections in cystic fibrosis, and infections of permanent indwelling devices such as joint prostheses, heart valves, and intervertebral disc.[22][23][24] The first visual evidence of a biofilm was recorded after spine surgery.[25] It was found that in the absence of clinical presentation of infection, impregnated bacteria could form a biofilm around an implant, and this biofilm can remain undetected via contemporary diagnostic methods, including swabbing. Future studies are needed to find means of identifying and monitoring biofilm colonization at the bedside to permit timely initiation of treatment.[26] It has been shown that biofilms are present on the removed tissue of 80% of patients undergoing surgery for chronic sinusitis. The patients with biofilms were shown to have been denuded of cilia and goblet cells, unlike the controls without biofilms who had normal cilia and goblet cell morphology.[27] Biofilms were also found on samples from two of 10 healthy controls mentioned. The species of bacteria from intraoperative cultures did not correspond to the bacteria species in the biofilm on the respective patient's tissue. In other words, the cultures were negative though the bacteria were present.[28] New staining techniques are being developed to differentiate bacterial cells growing in living animals, e.g. from tissues with allergy-inflammations.[29], from an evolutionary point of view, the creation of the tragedy of the commons in pathogenic microbes may provide advanced therapeutic ways for chronic infections caused by biofilms via genetically engineered invasive cheaters who can invade wild-types 'cooperators' of pathogenic bacteria until cooperator populations go to extinction or overall population 'cooperators and cheaters ' go to extinction.[30]

Infections associated with biofilm

It is estimated that about 65% of all bacterial infections are associated with bacterial biofilms. .[31] These include both, device- and non-device-associated infections. Data for device-related infections have been estimated for several devices, such as: 2% for breast implants; 2% for joint prostheses; 4% for mechanical heart valves; 10% for ventricular shunts; 4% for pacemakers and defibrillator, and about 40% for ventricular-assisted devices. .[32] Native valve endocarditis (NVE) is an inflammation caused by interaction of bacteria with the vascular endothelium and pulmonic valves of the heart. This is usually the result of Streptococci, Staphylococci, gram negative bacteria, and/or fungal infections..[33] In this condition microbial cells gain access to the heart and blood through the gastrointestinal

tract, urinary tract and/or through the oropharynx. As the intact valve endothelium gets damaged by the microorganisms that attach to it, even after the bacteria have been cleared by the immune system a non-bacterial thrombotic endocarditis (NBTE) develops at the injury location, as a result a thrombus formation occurs, a condition where platelets, red blood cells and fibrin are aggregated. .[34]

Advancements in biofilm research

Biofilm formation of infectious significance is mostly found on "implant devices". Recently, in the last year, another saprophytic organism, the incidence of which is increased in nosocomial (hospital acquired) infections, is a risk factor for medical device-carrying patients.55P. aeruginosa is the 2nd most common reason for ventilator-associated pneumonia (VAP) and catheter-associated urinary tract infections (CAUTI). P. aeruginosa forms biofilms on endocardial tubes and catheters in CAUTI and VAP patients [35].

Another recent advancement in biofilm research has been applied to control energy crises. This approach is using microbial fuel cells (MFCs). MFCs produce electricity by utilizing chemical energy found in organic and some in-organic compounds. Electrogenic microbes play a role in this process by accepting or donating electrons to an external object (electrode), while some non-electrogenic microbes are also involved as part of a synergistic electrogenic biofilm..[30]

Quorum Sensing

Cell-to-cell signaling has recently been demonstrated to play a role in cell attachment and detachment from biofilms. dental plaque bacteria can modulate expression of the genes encoding fimbrial expression (fimA) in Porphyromonas gingivalis. P. gingivalis would not attach to Streptococcus cristatis biofilms grown on glass slides. P. gingivalis, on the other hand, readily attached to S. gordonii. S. cristatus cell-free extract substantially affected expression of fimA in P. gingivalis, as determined by using a reporter system. S. cristatus is able to modulate P. gingivalis fimA expression and prevent its attachment to the biofilm .[30]

cell-to-cell signaling systems in P. aeruginosa, lasR-lasI and rhlR-rhlI, were involved in biofilm formation. At sufficient population densities, these signals reach concentrations required for activation of genes involved in biofilm differentiation. Mutants unable to produce both signals (double mutant) were able to produce a biofilm, but unlike the wild type, their biofilms were much thinner, cells were more densely packed, and the typical biofilm architecture was lacking. In addition, these mutant biofilms were much more easily removed from surfaces by a surfactant treatment. Addition of homoserine lactone to the medium containing the mutant biofilms resulted in biofilms similar to the wild type with respect to structure and thickness..[15] , Many bacteria can switch between planktonic form and biofilm form. The planktonic bacteria have relatively high cell growth and reproduction rate. However, the biofilm state appears to be natural and predominant state of bacteria. Several reasons could account for the need for a bacterial biofilm state . First, biofilm can enhance the tolerance of bacteria to harsh environmental conditions. Bacteria can avoid being washed away by water flow or blood stream by simply attaching to a surface or tissue. The oral biofilms can stand up to repeated, strong, shear forces. Cells in biofilms are about 1000 times more resistant than their planktonic . Second, the EPS matrix protects bacteria cells, in deeper layers, against antimicrobial agentsprobably by limiting the diffusion of these agents. Biofilm restricts bacterial mobility and increases cell density, which also provides an optimal environment for eDNA (plasmid) exchange (via conjugation), some of which encode for antibiotic resistance. As reported by Hausner and Wuertz, the horizontal gene transfer rate is significantly higher in biofilms than in the planktonic cells .[35]

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