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Biofilm-For We are Many Structure and Functions of Bacterial Biofilms

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Abstract

Bacterial biofilms are highly organized structures–both spatially and functionally–that represent one of the most important forms of presence of microorganisms. Cells located within biofilms are bound by joint extracellular matrix, which serves a stabilising function, allows to obtain high resistance to environmental conditions and creates a medium blocking transportation of significant amounts of toxins (among those antibiotics and antibacterial agents produced by animals' immune systems). Those features make biofilms key structures for environmental bacteria, but above all for pathogenic bacteria. This article describes the structure and main roles of biofilm with a particular consideration of biofilms created by pathogenic bacteria and membrane vesicles (MVs) involvement in its creation. Last part of the article describes an exemplary species of a pathogenic bacteria (Pseudomonas aeruginosa) which is known for the intensity of creation of multifactorial resistant biofilms and stands for one of the main development directions in medical microbiology.

Keywords: Biofilm, Pathogenic bacteria, Membrane vesicles

Introduction

During many years of research bacteria have been described mainly as single-celled organisms, that feed, grow and divide in an aqueous solution environment. Detailed analyses of the bacterial cells' movement mechanism were increasing the number of interpretations suggesting that planktonic trait of bacterial life is a crucial trait of this domain per se. However, environmental analyses and further medical microbiological research proved that under certain conditions (from the human perspective we mean more important conditions bearing direct reference to biotechnology, medicine, and interdisciplinary environmental research) bacterial cells more frequently create multicellular structures, which can be compared to animal tissues based on both structure and function [1].

Step by step

The creation of bacterial biofilm is a process in which one can distinguish several crucial stages: (I) bacterial cell adsorption to the surface in which cellular adhesins are involved; (II) increase in frequency and strength of cell-cell interaction; (III) production of extracellular matrix components; (IV) cells division, biofilm growth and increase in its density; (V) dispersion [2].

Bacterial cell adsorption to the surface is a stage that limits the amount of emerging biofilm, but also whether under certain conditions biofilm can even be formed. The efficiency of adsorption depends on many factors on the side of bacteria as well as the environment/surface [3]. To adsorb to the surface, a bacterium needs to present on its surface adhesins that will allow cell-medium interaction [4]. Considering biofilms in context of medical microbiology means we look at human tissue as such medium. Pathogenic bacteria commonly present adhesive proteins, which interact with many building components of human tissue, among them with cellular receptors and tissue ECM components [5]. Most researched biofilms of pathogenic bacteria are those created by *Pseudomonas aeruginosa* bacterium, which makes up one of the biggest risk factors connected to occurrence and development of cystic fibrosis [6,7].

Increase in frequency and strength of cell-cell interactions. Creation of biofilm requires interaction with the medium. Build-up of new layers of cells separates those newly adsorbed, or created due to the cell division, from the original biofilm medium [8]. Therefore, bacterial cells must adhere just as strongly to one another. Adhesins play just as important role in this process [9].

Most probably these interactions have been best studied among biofilms that are typical for oral cavity. Formation of such biofilm is connected to initial weak interactions of "pioneer bacteria" which are the first to colonize teeth surface. One of those tends to be singled out–*Streptococcus* spp [10]. These interactions are weak enough that a standard oral hygiene and antibacterial agents present in saliva regularly remove a thin layer of "pioneer bacteria". Subsequently, original interactions are being strengthened due to creation of specific receptor-ligand interactions. "Secondary colonizers" bind to adhesins presented by "pioneer colonists", allowing gradual increase in biofilm species diversity [11] (Figure 1).

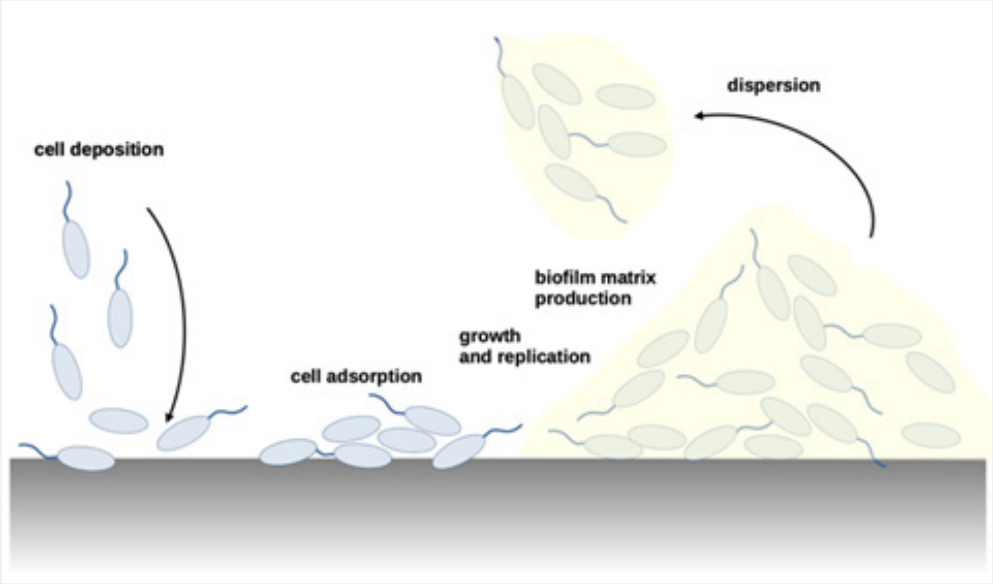

Figure 1: Steps of biofilm formation (description in text).

Creation of extracellular matrix components. With the continuous rise in number of bacteria in biofilm, biochemical level of its complexity rises as well. Depending on the species diversity biofilms can consist of a few to several dozen fundamental components that build up matrix. This matrix does not only perform the mechanical function which boosts biofilm's resistance to physical conditions (pressure, friction, fluid motion), but also increases the total number of adhesion points presented by it. Additionally, bacteria, both existing in biofilm and settling on its surface, present receptors that recognize components of biofilm matrix [12]. Further - just as crucial - function of biofilm matrix is its density augmentation through creation of steric bulks. These steric bulks effectively hinder access to biofilm for bactericidal agents – toxins, antibiotics and immune system effectors included. Moreover, matrix components allow non-specific binding of those agents to – above all – ionized functional groups presented on its surface [13]. Looking at just a fragment of a biofilm model shows extraordinary diversity of its components (Figure 2).

Growth of biofilm and increase in its density. The intensification of the stages in previously described paragraphs leads to the development of a biofilm, which becomes a heteromorphic structure. There are places with reduced (biofilm core) and increased (biofilm surface) availability of oxygen and nutrients. Such a diversity of the environment means it may lead to–and almost always does in sufficiently developed biofilms–the specialization of cells inhabiting these different niches. Conditions of reduced concentration of nutrients stimulate bacterial cells to create spores or the transition to the state of persister cells [14]. Both states make it possible to cause chronic infection in which, despite the effective degradation of the biofilm–by the immune system or specific therapies–there are still highly resistant bacteria that can recreate the population. The surface of the biofilm, apart from the much easier access to oxygen and nutrients, is also characterized by an increased exposure to bactericidal substances. Exposing the "surface population" to antibiotics and toxins can lead to selection of mutations that increase bacterial resistance to these factors. The selection of surface bacteria with an appropriate resistance ultimately leads to a state in which the further development of the biofilm can no longer be simply stopped [15]. Selection of bacteria that effectively resist immune system's activity is significantly more difficult, but most pathogenic bacteria possess a wide range of effectors that inhibit the activity of immune cells or activate their apoptosis pathways [16]. Dispersion Biofilm's specific structure and its limited strength, make it impossible for the biofilm–in most of the described examples–to grow indefinitely. The process of local biofilm breakdown leads to the release of cells which can later inhabit new environments. This is beneficial because if biofilm

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develops only locally, it can lead to nutrient depletion, resulting in halt of the growth of the entire population. Biofilms can breakdown locally due to the death of some of the cells that build it, matrix degradation, immunological activity (mainly caused by the involvement of phagocytic cells) or physical factors [17].

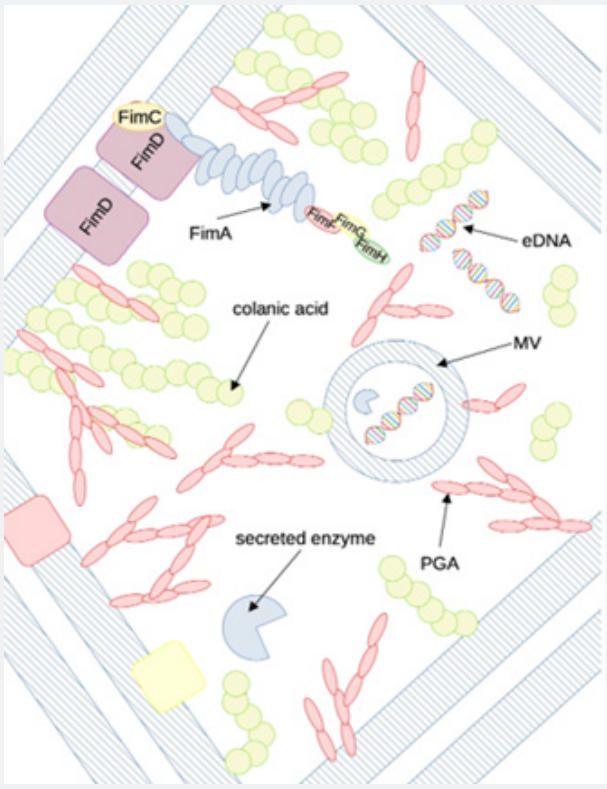

Figure 2: Extracellular matrix layout diagram in *E. coli.* EPS colanic acid, polyglucosamine (PGA), cellulose are fundamental components that facilitate interactions between bacterial cells and therefore hold them adjacent to each other. Another linking compound that also provides nutrition is eDNA. Shifts in environment are dealt with using secreted enzymes that respond by modifying extracellular polymeric substances. Bacterial aggregation that toughens the network is possible thanks to structures such as flagella and chaperone-usher pathway (CUP) pili.

East or West, biofilm's best

Creation of biofilms gives bacteria a significant advantage, but their specific structure, which heavily affects carried out functions, depends on the considered environment. In this review, the authors focused on biofilms in the context of medical microbiology based on their private interests and scientific experience.

Defense against phagocytosis. Bacteria living in planktonic form (freely swimming in aqueous solution) are particularly exposed to the activity of the immune system. Phagocytes–the first line of anti-infectious defense–effectively remove single bacterial cells by recognizing specific antigens, and phagocytosis [18]. Formation of a bacterial biofilm reduces the number of antigens available to phagocytes presented by the local bacterial population per single cell. Moreover, highly hydrophilic materials produced by bacterial cells (matrix components) in an aqueous environment take a swollen gel-like form which masks surface antigens. Any blockage of access to bacterial antigens also increases resistance to antibodies and the complement system [19]. Blockage of the penetration of antibiotics. Antibiotics are a key component of antibacterial therapies. They make it possible to remove populations sensitive to the antibiotic yet select resistant bacteria. However, spontaneous emergence of resistance is relatively rare, and only a certain pool of cells will acquire this phenotype during an infection. Therefore, the use of an antibiotic will not eliminate all cells, but the remaining (resistant) population is so small that it can be effectively removed by the immune system. Still, in the case of the production of bacterial biofilms, it turns out that it is not necessary for all bacteria to have an antibiotic-resistant phenotype. What is crucial is the presence of bacteria on the surface of the biofilm, in its less dense parts, and in "young" biofilms (thin ones, in which antibiotic can still diffuse freely). The same mechanism can describe the resistance of biofilms to heavy metals, toxins, and effectors of the immune system [15].

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Supporting horizontal gene transfer. Horizontal gene transfer (HGT) is one of the key aspects of bacterial evolution. It enables the exchange of DNA sequences between cells in a process other than cell division [20, 21]. There are three main mechanisms of HGT: (I) conjugation–made possible by a physical contact of bacteria and their cytoplasm linked with a pilus through which a plasmid is transported; (II) transduction–a form of HGT associated with the activity of bacteriophages, which during the infection cycle can collect fragments of the bacterial genome and transfer them to other bacteria through infection; (III) transformation–belongs to the least specific form of HGT, during which naked genetic material is absorbed from the environment [22]. Creation of biofilms significantly increases local cell density and at the same time the duration of their physical contact. This leads to an increase in the frequency of HGT events that require cells to be in proximity (conjugation, partly also transduction). However, even transformation can be assisted by the formation of biofilms by bacteria. Naked DNA, which is released by bacteria during cell lysis (bacterial cells especially lyse frequently in parts of biofilms that are subjected to the lack of oxygen and nutrients), is particularly sensitive to free nucleases. Binding such DNA to electrostatically positively charged matrix components–through ionic bonds–increases the stability of the genetic material [23].

MVs in biofilm–more than just a glue

Production of membrane vesicles (MVs) has been observed–and mostly well studied as well–among all domains of life [24]. More than 50 years have passed since the discovery of MVs in pathogenic bacteria, and during this time these structures have been studied for all clinically important species of bacteria. Although at first all membrane vesicles seem to be like each other, the analysis of their structure allows for the separation of key features distinguishing each type (Figure 3) [25].

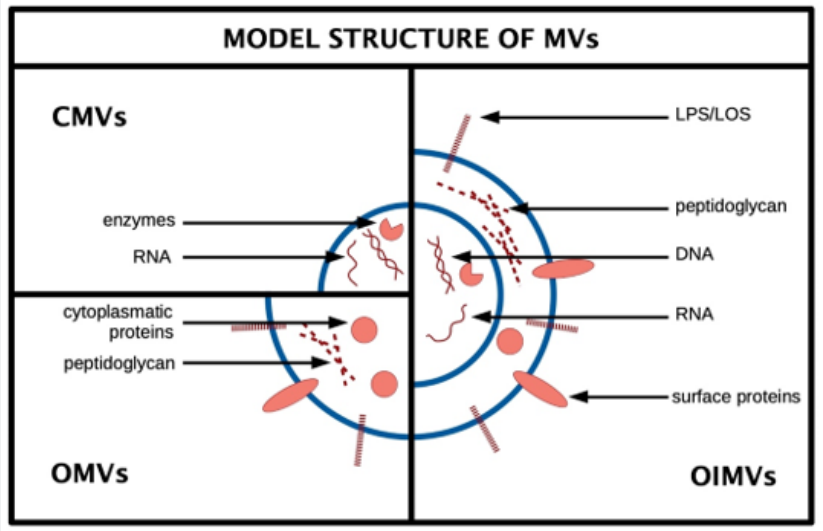

Figure 3: Main types of MVs with structure and key elements (description in text).

Commonly accepted classification of membrane vesicles is as follows:

OMVs (Outer-Membrane Vesicles): Discovered first, they are a model example of bacterial MVs. They are made of the outer membrane, contain LPS and membrane proteins. Their interior is filled with a liquid containing materials present in the periplasm of the cell: degraded peptidoglycan, proteins, periplasmic polysaccharides [26].

OIMVs (Outer-Inner Membrane Vescicles): First observed in the steady-state culture of Pseudomonas aeruginosa. They are characteristic of gram-negative bacteria. They make up a class of double membraned MVs, consisting of the outer and inner membranes separated by a layer of degraded peptidoglycan and a periplasm solution. At the center of the OIMVs there is a solution containing the components of the cytoplasm (proteins, RNA, DNA) [27].

CMVs (Cytoplasmatic Membrane Vesicles): Due to the presence of a thick cell wall, MVs secreted by gram-positive bacteria belong to the class of the least known. Their production requires local peptidoglycan degradation (e.g., due to autolysins), which makes this type of MVs present in old cultures and cultures subjected to high environmental stress [28,29].

TSMSs (Tube-Shaped Membranous Structures): These structures, also called nanotubes, were observed for several species (including the model Bacillus subtilis and Myxococcus xanthus). They are characteristic of bacteria that create complex structures such as biofilms or fruiting bodies [30].

MVs are widely studied as vectors that transport bacterial antigens, toxins, and effectors over long distances (they can cross tissue barriers and migrate in environments dense enough to rule

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out the movement of complete bacterial cells) [31]. Discovery of significant amounts of MVs in bacterial biofilms (primarily in all *M. xanthus and P. aeruginosa* biofilms) proved that they also play key roles in these multicellular communities [30,32]. MVs' qualitative and quantitative composition largely resembles a simplified bacterial cell. Thanks to this, they also present adhesins present on the bacterial cell membrane. This feature allows the MVs to function as an additional link connecting cells during initial stages of biofilm formation, when the matrix is not yet fully synthesized. Genetic material tied to the surface of the MVs or contained in its lumen is, respectively, partially, or fully protected against enzymatic degradation. As a result, MVs can be an additional store of genes and non-coding sequences in the HGT process, which (due to adhesins) are permanently located in the biofilm [33]. In the case of cells that form the surface of a biofilm, the function of MVs is even more diverse and mostly resembles planktonic bacteria vesicles. Thus, they may be antigenic decoys for capturing antibodies, a vector that transports toxins or "molecular sponges" to titrate heavy metals, antibiotics, and other antibacterial agents.

Pseudomons aeruginosa–public enemy number one

P. aeruginosa is an opportunistic gram-negative bacterium that causes chronic respiratory infections associated with the formation of complex biofilm systems that include chronic obstructive pulmonary disease, ventilator-associated pneumonia, and other nosocomial infections [34]. P. aeruginosa infections are especially dangerous for people with a mutation in the CFTR gene that causes cystic fibrosis. The accumulation of thick and sticky mucus on the surface of the mucous membranes is a characteristic symptom of this genetic disease [35]. Such a change in the environment favors local accumulation of dense populations of bacteria, which, owing to the quorum sensing (QS) system, start the process of building biofilm [36].

The formation of *P. aeruginosa* biofilm begins with the standard adhesion of singular cells. Bacteria must change the phenotype of the planktonic cell to a phenotype that can interact with solid elements of the environment, such as the apical surfaces of the epithelium or mucus proteins. Then it comes to the further aggregation of microcolony-forming bacteria and the formation of a consistent matrix structure (EPS–extracellular polymeric substances). Thanks to their polar nature, these polymers are highly hydrated and can make up as much as 85% of the mass of the entire biofilm. They mainly consist of polysaccharides, proteins and eDNA. This whole structure is figuratively called amalgam in Schooling et al work [37]. Extracellular networks of DNA derived from cell lysis were detected through: (I) specific staining with compounds showing DNA affinity; (II) showing strong absorbance at $\lambda = 260$ nm; (III) digestion with DNase; (IV) proving similarity of eDNA and genomic DNA sequences [38]. After eDNA polymers are released from the cell, they become fragmented, which results in the unraveling of superhelical strands and the formation of a loose network. Polymers of eDNA, apart from playing the role of "gene stores" in the HGT process, can additionally stabilize biofilm or serve as a food substrate for cells. The presence of phosphodiester bonds under physiological conditions ensures the nature of the polyanion of the eDNA network. This enables electrostatic interactions with metal cations, antibiotics or antibacterial proteins secreted by the host cells.

Main structural elements of EPS are: alginate, Psl and Pel. Alginate is a salt of a non-branched polymer composed of D-mannuronic acid, and L-glucuronic acid. The Psl polysaccharide is composed mainly of D-mannose, L-rhamnose and D-glucose. Chemically, the Pel polymer, containing mostly glucose, is much simpler, but its structure is still unknown. Research shows that Psl and Pel are the main components of EPS in the early stages of biofilm development. It is being proposed that LPS plays a significant role in maintaining the architecture of the biofilm. The amphiphilic nature of LPS prevents its individual presence in ESP spaces, however, membrane micelles and vesicles may constitute a specific storehouse of LPS particles existing outside the bacterial cell [39]. Described matrix immobilizes bacteria (allowing the maximum intensity of cell-cell interactions) and proteins secreted by them, increasing their local concentration. Moreover, microscopic, and immunochemical studies have shown the presence of OMVs in the spaces between local EPS concentrations. The culmination in the development of biofilm is reaching a storied, three-dimensional structure intersected by channels filled with water, with anaerobic regions and zones of local lysis of bacterial cells. It is the latter process that is associated with the formation of the OMVs fraction [40].

P. aeruginosa is a model example of bacteria that has been shown to produce large amounts of OMVs through the interaction of PQS with the outer membrane. This compound reduces the stability of the membranes, allowing them to bulge. PQS, apart from acting as an autoinducer, remains bound to OMVs and shows a high affinity for iron [41]. That way, it enables the formation of iron ions sequestering vesicles, which in this form can be more easily absorbed by bacterial cells. Detailed research on the vesicles in biofilms have shown that local cell lysis promotes the formation of eOMVs. Vesicles formed in the process of lysis were characterized by a large variation in size (with diameters larger than standard OMVs dominating) and composition. The lack of control over the transport of proteins to their matrix resulted in the presence of various OMVs fractions in terms of the types and concentrations of proteins. Additionally, cytoplasmic proteins and fragments of genetic material were found in eOMVs [42].

Conclusion

Knowledge that has been gathered on biofilm, compounds within, interactions between bacterial cells and its influence on our lives and our health is broad, however, it still leaves a huge gap of unknown as with every new discovery another pathway is being opened that needs further research. This review will allow you to take a better look at what has been uncovered, highlighting extraordinary work of MVs and what role it plays for pathogenic

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bacteria, and hopefully inspires you to look deeper into the breathtaking world of these microorganisms.

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