Biofilms

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KEYWORDS

• Biofilm • Chronic sinusitis • Rhinosinusitis

Chronic rhinosinusitis (CRS) is one of the most common chronic medical conditions, affecting between 14% and 16% of the US population. Direct health care costs are significant and are estimated to be more than $5.8 billion per year. According to the most recent data from the National Health Interview Survey (2007), rhinosinusitis continues to be one of the top 10 leading diagnoses of office visits in the United States. Patients with CRS demonstrate lower quality-of-life scores than those suffering from chronic obstructive pulmonary disease, congestive heart failure, back pain, or angina.[1] However, some patients with CRS suffer from persistent and recurrent infections despite maximal medical management and surgery. Since the initial description of biofilms on the sinonasal mucosa of patients with CRS, there has been a growing body of evidence supporting the contributory role of biofilms in poor disease progression and persistent sinonasal inflammation.

BIOFILMS OVERVIEW

It is currently estimated that at least 65% of all human bacterial infections may involve biofilm formation. These include a diverse range of infectious processes, including dental caries, periodontitis, musculoskeletal infections, osteomyelitis, bacterial prostatitis, endocarditis, and cystic fibrosis pneumonia. Biofilms have also been implicated in several conditions seen in an otolaryngology practice, including otitis media, chronic sinusitis, chronic tonsillitis, adenoiditis, and device infections (such as in cochlear implants, tympanostomy tubes, and tracheostomy tubes).[2] Bacterial biofilms are described as surface-associated communities of microorganisms encased in a protective extracellular matrix. The life cycle of bacterial biofilms can be divided into 5 parts (**Fig. 1**). Biofilms are initiated when free-floating, planktonic bacteria anchor to biologic or inert surfaces. The attached bacteria multiply and progress from a state of monolayer to a microcolony and then to a critical mass, in which interbacterial crosstalk occurs, triggering a phenomenon known as quorum sensing that leads to the biofilm phenotype. The bacteria respond collectively to express factors

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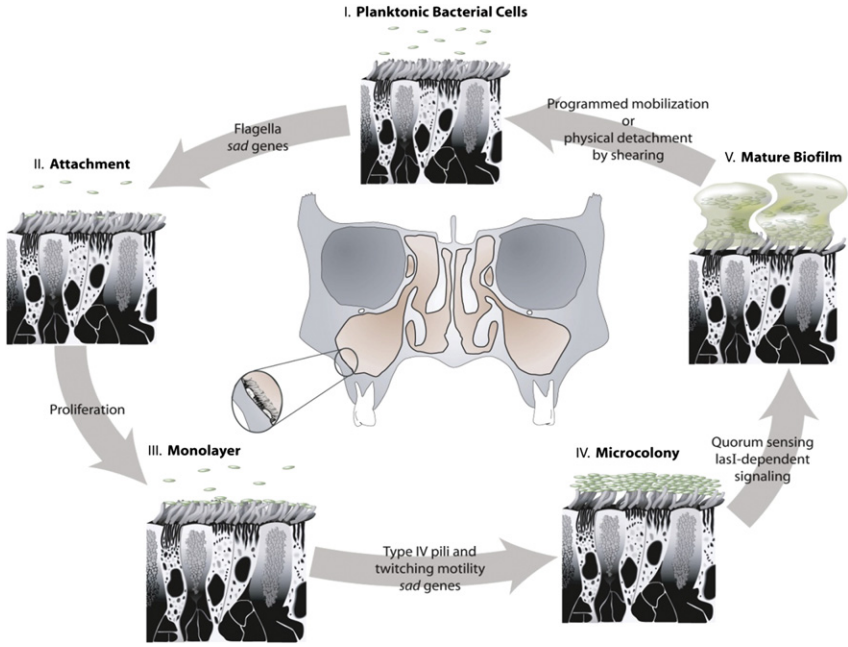

Fig. 1. Steps of bacterial biofilm formation in the paranasal sinuses. Planktonic bacterial cells (*I*) move from steps of attachment (*II*) to the formation of monolayers (*III*), microcolonies (*IV*), and a mature biofilm (*V*) through multiple progressions in gene expression.

that are specific to the biofilm phenotype, which lead to the secretion of an exopolysaccharide matrix. This biofilm phenotype is characterized morphologically by the formation of microbial towers, which are composed of layers of embedded, live bacteria with intervening water channels. Under the right environmental conditions, free-floating bacteria are released from the biofilms, and the cycle is continued at other surfaces. Approximately 80% of the world's microbial biomass resides in the biofilm state, and the National Institutes of Health estimates that more than 75% of microbial infections that occur in the human body are underpinned by the formation and persistence of biofilms.[3,4]

Biofilm formation is thought to provide a mechanism for enhanced bacterial survival. Bacteria in biofilms lack the antibiotic susceptibility of planktonic bacteria and can be up to 1000 times more resistant to antibiotic treatment.[5] Bacteria in biofilms are also more resistant to host defenses, because the extracellular matrix that makes up most of the biofilm serves to protect the bacteria against antibodies, immune-system phagocytosis, antibiotic penetration, and complement binding. There is also a decreased need for oxygen and nutrients when bacteria exist in the biofilm state, further reducing susceptibility to certain antimicrobials (**Box 1**).[6] This is especially true for bacteria within the core of the biofilm mass.[7] In addition, biofilms are environments where bacteria can share their DNA by transfer of genetic information via plasmids to encourage variability and adaptive mutations, such as antibiotic resistance. All of these properties encourage persistence of bacteria for extensive periods despite antibiotic treatment, resulting in chronic disease with intermittent acute infections. Biofilms also provide a source for recurrent infections by releasing planktonic bacteria, resulting in implantation and population of new anatomic locations.[8]