New horizons for cutaneous microbiology: the role of biofilms in dermatological disease

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Summary

Human skin is colonized by bacteria. The development of new genomic microbiological techniques has revealed that the bacterial ecology of human skin is far more complex than previously imagined and includes many fastidious or non-cultivable bacterial species which are found on both normal and diseased skin. In nature, the predominant bacterial phenotype on epithelial surfaces is that of organisms organized within a biofilm. This contrasts with the widely held belief that bacteria are planktonic, i.e. free-floating single cells. Biofilms are sessile bacterial communities encased in an extracellular matrix that have a well-developed communication system and can regulate bacterial growth and metabolism, confer resistance to antimicrobials and to host inflammatory cells, and alter host metabolism. Biofilms have been observed on healthy skin and in a number of dermatological conditions, including some that were previously thought not to have an infectious aetiology. Here we review the concept of biofilms and their role in cutaneous health and disease.

New genomic techniques have revolutionized microbiology and have facilitated the identification of fastidious and non-cultivable bacteria. Traditional culture approaches to skin microflora identified the major components as aerobic diphtheroids (Corynebacterium spp.), anaerobic diphtheroids (Propionibacterium acnes) and coagulase-negative staphylococci (CNS). Recent genomic analysis of disease-free skin found stable polymicrobial communities consisting predominantly of Pseudomonas and Janthinobacterium spp. Staphylococcus epidermidis and P. acnes constitute < 5% of microbiota.

New technologies have also demonstrated that bacteria form biofilms on epithelial surfaces including skin. Biofilms are complex sessile microbial communities that consist of one or more bacterial species surrounded by extracellular polymeric substances (EPS). These bacterial communities attach to biological and nonbiological surfaces and demonstrate altered phenotypes and growth characteristics. Biofilms are now thought to be the predominant bacterial phenotype on both healthy and diseased human skin.

Skin biofilms have been associated with several dermatological diseases such as acne, rosacea and atopic dermatitis (AD). Biofilms have also been shown to impair normal wound healing. These discoveries have prompted a shift toward a more targeted therapeutic approach based on the understanding of biofilm structure and organization as opposed to traditional culture-based therapies. It is becoming increasingly important for clinical dermatologists to understand the concept of biofilms, their role in cutaneous disorders and their influence on the choice of topical and systemic antimicrobials.

Biofilm

Bacteria exist in two states – planktonic and sessile – which differ significantly in their physiology, gene expression pattern and morphology. Almost all clinical microbiology is based on characterizing planktonic bacteria. In nature, however, most bacteria exist as biofilms, especially on epithelial surfaces. Transition from the planktonic form to biofilm is regulated by multiple environmental and physiological factors such as bacterial cell density and nutrient availability. Antimicrobials can have paradoxical effects. Some antibiotics in subminimum inhibitory concentrations (sub-MIC) enhance biofilm formation. For example, vancomycin and cefamandole induce biofilm production by CNS. Tobramycin, tetracycline and norfloxacin have the same effect on Pseudomonas aeruginosa cells. Gentamicin does not affect biofilm formation by Haemophilus influenzae but at sub-MIC it confers resistance to organisms within established biofilms. Conversely, other antibacterial agents such as azithromycin reduce H. influenzae biofilm thickness.
and biomass even at sub-MIC.

Biofilms occur on a variety of natural surfaces such as teeth, heart valves, lungs of patients with cystic fibrosis (CF) and middle ear mucosa in persistent otitis media. They are also found in chronic rhinosinusitis, prostatis and chronic osteomyelitis. Biofilms have been demonstrated on a number of nonbiological surfaces such as prosthetic heart valves, orthopaedic implants, intrauterine devices, contact lenses and intravenous catheters. Biofilms are also important in environmental microbiology.

Biofilm formation is a dynamic process. Many bacterial species reversibly attach to a solid surface within a few hours of inoculation (Fig. 1). Within the next few hours bacteria bind to the surface irreversibly, start multiplying and communicating with each other via quorum sensing (QS) molecules, form microcolonies and produce an extracellular matrix (ECM) around these colonies. The colonies may develop a form microcolonies and produce an extracellular matrix communicating with each other via quorum sensing (QS) molecules, bind to the surface irreversibly, start multiplying and communicating with each other via quorum sensing (QS) molecules, form microcolonies and produce an extracellular matrix (ECM) around these colonies. The colonies may develop a variety of shapes and characteristics depending on local factors and bacterial species involved (Fig. 2). For example, ‘small colony variant’ strains of several staphylococcal species seen in chronic infections such as osteomyelitis and CF demonstrate features of biofilms. Eventually, certain areas of the biofilm detach, and free bacterial cells seed to other locations where they can form new biofilms.

QS is a density-dependent form of cell–cell communication that represents a feedback loop regulating bacterial growth in which the bacterial cells synthesize and react to small signal molecules. QS allows microorganisms to sense when critical bacterial concentrations are attained. They then can suppress further multiplication by producing and releasing molecular signals that affect other bacterial cells, the host and the producer cells themselves. The QS mechanism largely comprises two distinct groups of signalling molecules. Peptide derivatives such as autoinducing peptides (AIP) are used by Gram-positive bacteria, whereas fatty acid derivatives such as acylhomoserine lactones and γ-butyrolactones are used by Gram-negative bacteria. Furanosyl diester (also known as autoinducer-2 or AI-2) is a signalling molecule that is expressed and recognized by both Gram-negative and –positive organisms; a notable exception is P. aeruginosa, which does not produce AI-2 but can respond to the molecule. The QS molecules exert their effect by regulating expression of genes involved in the production of virulence factors, sporulation, DNA uptake and biofilm formation. Unlike many QS systems described in Gram-negative biofilms, the two signalling systems of Staphylococcus aureus (agr and luxS) negatively regulate biofilm formation and virulence. The inactivation or downregulation of QS may be a crucial step in the development of S. aureus-associated infections of indwelling catheters, artificial joints, lungs of patients with CF and others.

QS may also have an effect on the eukaryotic host. For example, autoinducer signal molecule N-(3-oxododecanoyl) homoserine lactone (3O-C12-HSL) produced by P. aeruginosa can stimulate production of interleukin (IL)-8 in bronchial epithelial cells in vitro, inducing the expression of several cytokines (IL-6, IL-1α, etc.) that stimulate migration of monocytes, neutrophils and T cells in vitro and in vivo and upregulate the expression of cyclooxygenase-2 in vitro. Interestingly, in vitro, 3O-C12-HSL can also inhibit lymphocyte proliferation and IL-12 production, important for the activation of macrophages. It is possible that 3O-C12-HSL induces different immune responses depending on the environmental pressures and the type of host cells involved.

QS was originally thought to be important for bacterial biofilm formation and virulence. While it has been shown that

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**Fig 1.** Process of biofilm development. The development of biofilms involves a characteristic sequence of events. The process begins with the initial attachment of bacteria to the surface, exchange of information via quorum sensing and production of extracellular polymers leading to the formation of microcolonies. During the subsequent maturation phase the amount of extracellular material increases and biofilms acquire antibiotic resistance. These steps are physiologically distinct and require phase-specific factors. © Center for Biofilm Engineering, Montana State University, Bozeman, MT, U.S.A. Reprinted with permission.

**Fig 2.** Heterogeneity of biofilm structure. Mature biofilms often have three-dimensional structures that are commonly described as ‘towers’ or ‘mushrooms’. Fluid-filled channels found between these structures are thought to deliver nutrients to cells in deeper film layers. However, because of the wide range of microenvironments in which biofilms are found and the differences in physiological determinants such as substrates and metabolites the actual biofilm structures may vary significantly. © Center for Biofilm Engineering, Montana State University, Bozeman, MT, U.S.A. Reprinted with permission.
P. aeruginosa strains lacking a functional QS system are less virulent than wild-type strains,\textsuperscript{52,53} biofilms formed by QS-deficient bacterial strains looked identical to those produced by wild-type strains.\textsuperscript{52} These data suggest that QS is crucial for P. aeruginosa virulence but not for biofilm formation.

The composition of EPS is complex (Fig. 3) and comprises polysaccharides, proteins, extracellular DNA and even host-produced factors.\textsuperscript{9,43,54} The structure of the matrix varies considerably among species and even a single species can produce different EPS depending on growth conditions.\textsuperscript{43} In addition to structural functions, EPS also plays informative, redox-active and nutritive roles.\textsuperscript{54} Also, the cohesive strength of EPS determines the viscoelastic structure of biofilms.\textsuperscript{55} Unfortunately, the matrix is hard to image due to structural heterogeneity and the difficulty in distinguishing between microbial vs. host components of EPS.\textsuperscript{43,54}

Several in vitro studies have shown that bacteria in biofilms are 50–500 times more resistant to antibiotics than their planktonic counterparts (Table 1).\textsuperscript{56–58} Multiple factors are thought to contribute to biofilm antibiotic resistance (Fig. 4): 1 Physical barrier. The ECM of the biofilm prevents diffusion of antibiotics through the biofilm matrix.\textsuperscript{59} 2 Altered growth and metabolism. Growth, protein synthesis and metabolic activity are stratified in biofilms such that the highest level of activity happens at the surface with slow or no growth in the centre due to lack of nutrients and/or oxygen.\textsuperscript{60,61} Because most antimicrobials work by altering vital processes in dividing cells they have minimal effect on non-reproducing sessile microorganisms. 3 Phenotype switch. Sessile bacteria possess regulatory genes that integrate signals from the external environment and switch to more tolerant phenotypes upon environmental stresses such as increases in bacterial density, lack of nutrients or change in temperature and pH.\textsuperscript{62} 4 Increased mutation frequency and gene transfer. Mutation frequency in biofilm-growing bacteria is much higher than in free-floating organisms\textsuperscript{63} and is likely caused by oxidative stress resulting from slow, diffusion-limited release of reactive oxygen species\textsuperscript{64} and a deficient antioxidant system.\textsuperscript{6,65} This high mutation frequency together with increased horizontal gene transmission in biofilms\textsuperscript{66} offers an explanation for quick development of multidrug resistance by sessile bacteria. The discovery of genomic islands accounting for up to 10\% of the bacterial genome may further explain rapid changes in biofilm virulence potential.\textsuperscript{67} Genomic islands are horizontally acquired DNA segments frequently integrated in the vicinity of tRNA genes.\textsuperscript{68} When these DNA regions carry one or more virulence-associated genes they are called pathogenicity islands and they may have contributed to the evolution of the genome of multiple bacterial species.\textsuperscript{67}

![Fig 3. Biofilm structure. Fluorescence micrograph of a thin section from a human chronic wound showing biofilm formed by rod-shaped bacteria and the surrounding extracellular polymeric substances (EPS) (arrows). This specimen was stained with Sytox (green) and Phaseolus vulgaris leucoagglutinin (red). The EPS is stained green due to extracellular bacterial DNA in the matrix. The lectin stained the connective tissue red. Microscopy by Margaret Campbell, Center for Biofilm Engineering. © Center for Biofilm Engineering, Montana State University, Bozeman, MT, U.S.A. Reprinted with permission.](image)

**Table 1** Susceptibility of planktonic and biofilm bacteria to selected antibiotics

<table>
<thead>
<tr>
<th>References</th>
<th>Organism</th>
<th>Antibiotics</th>
<th>MIC/MBC of planktonic phenotype (µg mL(^{-1}))</th>
<th>Concentration effective against biofilm phenotype (µg mL(^{-1}))</th>
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<tbody>
<tr>
<td>Williams et al.</td>
<td>Staphylococcus aureus</td>
<td>Vancomycin</td>
<td>2 (MBC)</td>
<td>&gt; 1024</td>
</tr>
<tr>
<td>Ceri et al.</td>
<td>Pseudomonas aeruginosa</td>
<td>Imipenem</td>
<td>1 (MIC)</td>
<td>512</td>
</tr>
<tr>
<td>Ceri et al.</td>
<td>Escherichia coli</td>
<td>Ampicillin</td>
<td>2 (MIC)</td>
<td>800</td>
</tr>
<tr>
<td>Vorachit et al.</td>
<td>Pseudomonas pseudomallei</td>
<td>Cefazidime</td>
<td>8 (MBC)</td>
<td>3 ± 15</td>
</tr>
<tr>
<td>Larsen and Fiehn</td>
<td>Streptococcus sanguinis</td>
<td>Doxycycline</td>
<td>0·063 (MIC)</td>
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Adapted from Donlan and Costerton (2002).\textsuperscript{8} MBC, minimum bactericidal concentration; MIC, minimum inhibitory concentration.
Virulence factors are expressed during biofilm formation.\textsuperscript{70,71} Quantities of inflammatory cytokines that contribute to chronic inflammation result in the production of increased stress response and gene transfer.\textsuperscript{43} In addition, the extracellular matrix acts as a diffusion barrier to small molecules. © Center for Biofilm Engineering, Montana State University, Bozeman, MT, U.S.A. Reprinted with permission.

5 Spore-like forms. Biofilms often contain so-called ‘persister cells’ that are virtually impossible to eradicate with any antibiotic. Persisters are spore-like cells that produce proteins that shut down antibiotic targets. For example, cells that shut down the ribosome become tolerant to aminoglycosides whereas cells that prevent peptidoglycan synthesis develop resistance to cell wall-acting antibiotics.\textsuperscript{6,9}

Fig 4. Mechanisms of antimicrobial tolerance. Biofilms confer resistance to antimicrobials by several different mechanisms. An increased proportion of nondividing ‘persister cells’ resistant to most antibiotics is believed to be one of them. Biofilm represents a natural stationary phase of bacterial life cycle during which microorganisms modify their growth and metabolism as well as mutation frequency and gene transfer.\textsuperscript{43} In addition, the extracellular matrix acts as a diffusion barrier to small molecules. © Center for Biofilm Engineering, Montana State University, Bozeman, MT, U.S.A. Reprinted with permission.

Besides inducing antibiotic tolerance, biofilms may contribute to increased bacterial virulence.\textsuperscript{5} In P. aeruginosa, multiple virulence factors are expressed during biofilm formation.\textsuperscript{70,71} Conversely, S. aureus virulence factors are downregulated during biofilm growth.\textsuperscript{42} Paradoxically, this may represent a pathogenic mechanism because biofilm growth, unnoticed by the immune system, produces a higher number of nonviral bacteria that are released, and subsequently become virulent.\textsuperscript{5,1,17} Furthermore, biofilms affect phagocytosis. Polymorphonuclear leucocytes can penetrate biofilm but cannot effectively internalize individual bacteria.\textsuperscript{7,2} This dysfunctional phagocytosis results in production of increased quantities of inflammatory cytokines that contribute to chronic inflammation and destruction of the surrounding tissues.\textsuperscript{73}

Bacterial biofilms and skin

Wounds

A fertile area of study of skin biofilm pathophysiology is wounds. 16\textsubscript{S} rRNA gene-based analysis demonstrated an average of 10 different bacterial families in various chronic wounds or about four times more than estimated by cultures.\textsuperscript{74} Fastidious anaerobic bacteria belonging to the Clostridiales family were among the most prevalent organisms but were only identified by molecular analysis.\textsuperscript{74} High-throughput pyrosequencing showed increased proportions of anaerobes, Gram-negative rods and Gram-positive cocci but a decreased proportion of Propionibacterium in chronic wounds vs. normal skin (A. Han, J.M. Zenilman, J.H. Melendez et al., unpublished data).

Several studies employing molecular techniques confirmed the abundance of anaerobes such as Bacteroides, Peptostreptococcus, Finegoldia and Anaerococcus in diabetic\textsuperscript{75} and venous stasis ulcers.\textsuperscript{76} Dowd et al.\textsuperscript{75} showed that obligate anaerobes represented 62\% of the bacteria in pressure ulcers. In all the studies, cultures successfully identified only the nonfastidious, easily ‘culturable’ organisms such as S. aureus. Therefore, if an organism cannot be detected by culture, it cannot be assumed to be absent, because of the comparatively low sensitivity of standard cultures.

Other technologies have helped describe biofilm morphologies. Transmission electron microscopy revealed that S. epidermidis grows as a biofilm between the squamous cells of normal skin.\textsuperscript{8} Numerous studies have also demonstrated the presence of biofilms in chronic wounds.\textsuperscript{76–79} James et al.\textsuperscript{80} detected biofilms in 60\% of chronic wounds and only 6\% of acute wounds. Biofilms impair wound healing as they induce local immune dysfunction and apoptosis in keratinocytes, alter production of enzymes and growth factors by endothelial cells, impair neovascularization, and inhibit fibroblast synthesis, migration and proliferation.\textsuperscript{78,81} The structure of a biofilm depends on several factors including its bacterial composition. In biofilms consisting of mixed flora, anaerobes are most likely located in the centre of the biofilm while aerobes are found closer to the surface. The microelectrode profiles of tonsilloliths that are morphologically similar to biofilms demonstrated a stratification of physiological activity.\textsuperscript{82} Oxygen consumption in the top layers depleted oxygen deeper in the tonsillolith suggesting the presence of anaerobes in the centre of this biofilm-like structure.\textsuperscript{82}

Although biofilms are commonly associated with pathological processes they may also play a protective role. Donlan and Costerton\textsuperscript{8} suggested that all mammals have symbiotic relationships with specific staphylococcal species residing on their skin. The host provides staphylococci with ‘food and lodging’ while the bacteria protect their partner by competing with potential pathogens.\textsuperscript{8} Moreover lipoteichoic acid produced by S. epidermidis was shown to act on keratinocytes triggered by Toll-like receptor (TLR)-3 and hence to inhibit skin inflammation.\textsuperscript{83} In addition, a small molecule produced by S. epidermidis was found to activate TLR2 and increase production of antimicrobial peptides thus enhancing antimicrobial defence.\textsuperscript{84} A better understanding of the biofilm composition and structure will lead to the development of optimal infection management and treatment strategies. Antimicrobials should be used judiciously because antibiotic overuse especially at sub-MIC can be deleterious. As mentioned earlier, certain antibiotics at sub-MIC enhance the formation of biofilms.\textsuperscript{1,11,14} Analysis of P. aeruginosa isolated from the lungs of patients with CF revealed that treatment with amikacin, cefaz-
idime and levofloxacin, even at doses higher than MIC, selected for resistant variants. 

Reinhardt et al. prospectively collected P. aeruginosa tracheal isolates from two intubated patients treated with carbapenem, fluoroquinolone or combined β-lactam aminoglycosides. Resistance appeared within 10 days of the initiation of treatment with any class of antibiotics, suggesting that P. aeruginosa forms biofilms in the lungs of patients with CF and those who have been intubated. Persisting cells that exhibit multidrug tolerance can evolve and may be impossible to eradicate. Antibiotics eliminate both pathogenic and commensal organisms and thus allow persister cells to proliferate in the absence of competition. In an effort to limit the selective pressure of antimicrobials some authors propose treating ventilator-associated pneumonia for 8 days instead of 15.

**Biofilms in dermatological conditions**

There is a significant difference between the microbial flora of diseased and healthy skin. For example, use of broad-range 16S rDNA polymerase chain reaction demonstrated that Firmicutes was the most abundant phylum in psoriatic lesions (46.7%) as opposed to healthy skin (24.4%). A biofilm from a patient with rosacea was shown to contain more Demodex folliculorum and P. acnes than a biofilm of a healthy individual. New molecular techniques have also changed our ideas about ‘normal’ skin flora. Firstly, there is topographical diversity of the human skin microbiome. Physiologically comparable sites are colonized by similar bacterial communities, which may explain why certain dermatological disorders occur at stereotypical skin sites. Secondly, sequencing of bacterial 16S small-subunit rRNA genes from the skin of healthy volunteers revealed that the genera Pseudomonas and Staphylococcus represented the majority of the sequences. Conversely, S. epidermidis and P. acnes represented < 5% of microbiota. These findings contradict the commonly held notion that Pseudomonas is a secondary wound invader while S. epidermidis and P. acnes are the main commensal aerobes.

Certain inflammatory dermatological conditions that have traditionally been thought not to be infectious may in fact have an infectious aetiology. As mentioned above, the fact that bacteria cannot be cultured from a skin lesion does not mean that microorganisms are not present. Instead they may represent difficult-to-culture species or they may be present in biofilms. This fact can explain why certain ‘noninfectious’ diseases respond to antibiotics and relapse following the discontinuation of treatment. Acne vulgaris, miliaria and AD have long been classified as noninfectious but are now thought to be associated with biofilms.

**Acne vulgaris**

Acne vulgaris is a common cutaneous disorder affecting over 80% of teenagers. The initial step in acne pathogenesis is believed to be hyperplasia of pilosebaceous ducts with formation of keratinaceous plugs and subsequent colonization of the follicle by P. acnes. Some authors propose that P. acnes should be found in the pilosebaceous units as biofilms rather than free-floating bacteria. Although P. acnes biofilms have never been directly observed in the pilosebaceous unit this bacterium can form biofilms in vitro and in vivo on a number of medical devices. Also, the complete genome sequence of P. acnes has revealed three separate clusters of genes that encode enzymes involved in EPS biosynthesis, adhesion proteins and the homologue of luxS (ORF405), responsible for the production of the QS molecule AI-2.

**Miliaria**

Miliaria or heat rash is caused by obstruction of the eccrine ducts leading to sweat retention in different layers of the epidermis. Mowad et al. inoculated the forearms of healthy subjects with several strains of CNS under occlusion. Of all the CNS tested only the EPS-producing strains of S. epidermidis induced miliaria. Given this finding and the fact that the sweat glands were found to be obstructed by EPS, the authors concluded that S. epidermidis and its ability to form biofilms play a role in the pathogenesis of miliaria.

**Atopic dermatitis**

AD is a chronic skin disorder associated with abnormalities in skin barrier function and allergen sensitization. Although healthy skin flora only rarely contains S. aureus this pathogen is frequently isolated from AD lesions. Akiyama et al. proposed that S. aureus in these lesions could be forming biofilm-like structures. Using confocal laser scanning microscopy (CLSM) they observed the formation of biofilms by S. aureus strains isolated from AD lesions in vitro and in vivo. Katsuyama et al. collected the stratum corneum of their patients with AD and confirmed the presence of S. aureus biofilm in the lesions with scanning electron microscopy.

**Onychomycosis**

Onychomycosis is a common chronic nail infection caused by Trichophyton rubrum, T. mentagrophytes and some Candida spp. Dermatophytoma is a complication of onychomycosis that negatively affects the chance of cure. Burkhart et al. proposed that fungal biofilms contributed to resistance of dermatophytomas to antifungal therapy. The ability of dermatophytes to form biofilms has not yet been investigated but several species of yeast have been shown to form biofilms. In addition, histological examination of nail clippings in onychomycosis demonstrated resting spores mixed with actively growing hyphae. This finding is consistent with the description of biofilms in which many cells exist in a dormant state.
Impetigo and furuncles

Finally, impetigo and furuncles represent acute infectious disorders that have been shown to be associated with sessile bacterial colonies. Impetigo is a superficial infection of the skin caused by group A streptococci or S. aureus.116 Furuncles are perifollicular abscesses caused by S. aureus.117 Akiyama et al.117 observed formation of periodic acid–Schiff-positive and Ruthenium red-positive structures around S. aureus cells isolated from impetigo and furuncle lesions in vivo. Two studies by the same team later demonstrated the formation of glyocalyx by S. aureus isolated from furuncle and impetigo lesions in vivo.118,119 The researchers also studied S. pyogenes and S. aureus biofilms in nonbullous impetigo lesions in vivo. Using CLSM they found that S. pyogenes cells formed microcolonies encircled by glyocalyx in the outer walls of the lesions and that these colonies existed independently from microcolonies formed by S. aureus.120

Conclusions

Human skin is an important interface with the outside world. It is colonized by an astonishing number and variety of microorganisms. New molecular technologies have provided us with opportunities to understand the role of bacteria in cutaneous health and disease. These new methods have also demonstrated that the conventional wisdom derived from culture data is often not adequate to establish pathophysiological relationships. Recent research suggests that bacterial biofilms may be critical to our understanding of the pathogenesis of skin disorders and their treatment as biofilm appears to be the predominant bacterial phenotype on the skin that can alter host responses as well as bacterial susceptibility to antibiotics. Therefore, it is likely that in the near future biofilms will rewrite the role of bacteria in skin disease.

What’s already known about this topic?

- Human skin is colonized by bacteria.
- New molecular techniques have demonstrated that the cutaneous microflora is far more complex than previously appreciated.
- Biofilms are the predominant bacterial phenotype in nature.

What does this study add?

- Biofilms have been observed in dermatological conditions and may be important in the pathogenesis of skin disorders.
- Inappropriate use of antibiotics can enhance biofilm formation.
- Better understanding of biofilm composition and structure will lead to development of optimal treatment strategies.

References


