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(REVIEW ARTICLE)

Biofilms: Health implications and control in clinical settings

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Abstract

Biofilms are structured communities of microorganisms that adhere to biotic or abiotic surfaces and are enclosed within a self-produced matrix of extracellular polymeric substances. The transition of microbial cells from the free-living planktonic state to the sessile biofilm mode of growth is a major survival adaptation, conferring altered physiology, reduced antimicrobial susceptibility and heightened tolerance to host defences. In clinical settings, biofilms are recognised as a central cause of persistent, chronic and recurrent infections, and most human microbial infections are thought to involve a biofilm component. This narrative review examines the structure and developmental life cycle of biofilms, the clinically important biofilm-forming microorganisms, and the mechanisms by which biofilm-associated cells resist and tolerate antimicrobial therapy, including restricted drug penetration, altered microenvironments, slow growth and persister cells. The health implications of biofilms are discussed with reference to indwelling medical device infections, chronic wounds, cystic fibrosis, infective endocarditis, chronic otitis media, periodontitis and healthcare-associated infections. Laboratory approaches for detecting biofilm production are reviewed, including the tissue culture plate method, the tube method, Congo red agar, and microscopic and molecular techniques. Prevention and control strategies are considered, ranging from aseptic device management and antimicrobial-impregnated biomaterials to emerging approaches such as quorum sensing inhibition, matrix-degrading enzymes, bacteriophage therapy, antimicrobial peptides and nanotechnology. A sound understanding of biofilm biology is essential for the medical laboratory scientist, who plays a pivotal role in the detection, surveillance and control of biofilm-associated disease and in the containment of antimicrobial resistance.

Keywords: Biofilm; Extracellular Polymeric Substances; Medical Device Infection; Antimicrobial Tolerance; Antimicrobial Resistance; Biofilm Control.

1. Introduction

A biofilm may be defined as an aggregate of microbial cells that are irreversibly attached to a surface or to one another and embedded within a self-produced hydrated matrix of extracellular polymeric substances.[1] Far from being a disorganised layer of cells, a mature biofilm is a highly structured and dynamic community in which cells communicate, cooperate, exchange genetic material and differentiate into distinct physiological states. The recognition that most microorganisms in nature and in the human body exist predominantly in this attached, sessile form, rather than as free-floating planktonic cells, has transformed the way infectious diseases are understood and managed.[2]

The existence of surface-associated microbial life was first documented by Antonie van Leeuwenhoek in the seventeenth century, when he observed the dense communities of “animalcules” scraped from his own teeth. However, the modern conceptual framework of the biofilm was established much later, largely through the work of Costerton and colleagues,

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who demonstrated that bacteria in natural, industrial and medical systems overwhelmingly adopt a matrix-enclosed, surface-attached mode of growth and that this lifestyle confers protection against environmental stress.[3,4] It is now widely cited that biofilms are associated with the majority of chronic and device-related human infections, a figure frequently placed at around sixty-five to eighty per cent of all microbial infections encountered in clinical practice.[5]

The clinical importance of biofilms arises from a single fundamental property: cells growing within a biofilm can be up to a thousand times more tolerant of antimicrobial agents than their genetically identical planktonic counterparts.[6,7] This tolerance, combined with the ability of biofilms to evade the host immune response, explains why biofilm-associated infections tend to be persistent, recurrent and extremely difficult to eradicate with conventional antibiotic therapy alone. In the hospital environment, biofilms readily colonise indwelling medical devices such as intravascular catheters, urinary catheters, prosthetic joints and heart valves, and they also underlie a range of chronic native-tissue infections. Consequently, biofilms constitute a major reservoir and driver of antimicrobial resistance and a significant source of morbidity, mortality and healthcare cost.[8,9]

This review examines the biology of biofilms and their significance in clinical settings. It describes the composition and architecture of the biofilm matrix, the sequential stages of biofilm development, and the principal biofilm-forming pathogens of medical importance. It then explores the mechanisms underlying antimicrobial tolerance, the spectrum of biofilm-associated diseases, the laboratory methods available for detecting biofilm production, and the range of preventive and therapeutic strategies currently used or under investigation, with particular attention to the practical relevance of biofilms to the medical laboratory scientist.

2. Materials and methods

This article is a narrative review of the published literature on biofilms and their significance in clinical settings. Relevant peer-reviewed publications were identified through structured searches of electronic databases, including PubMed, Google Scholar, Scopus and ScienceDirect, using combinations of the keywords “biofilm”, “extracellular polymeric substances”, “quorum sensing”, “medical device infection”, “antimicrobial tolerance”, “antimicrobial resistance”, “biofilm detection” and “biofilm control”. The reference lists of retrieved articles were also examined manually to identify additional relevant sources.

Original research articles, systematic and narrative reviews, and authoritative reference texts published in English were considered, with emphasis placed both on seminal contributions that established the biofilm concept and on recent publications reflecting current understanding. The retrieved literature was appraised for relevance and reliability, and the information was synthesised thematically under the headings presented in the following section, covering biofilm structure and formation, clinically important biofilm-forming organisms, mechanisms of antimicrobial tolerance, health implications, laboratory detection, and control strategies.

3. Results and discussion

3.1. Structure and Composition of Biofilms

The defining feature of a biofilm is the extracellular polymeric substance matrix, often referred to as the extracellular matrix, which typically accounts for the greater part of the biofilm biomass, with the resident cells constituting a smaller proportion of the total volume.[10] This matrix is a hydrated, gel-like scaffold that holds the community together, anchors it to the underlying surface, and creates the immediate microenvironment in which the cells live. It is frequently described as the “house of the biofilm cells” because it provides mechanical stability, retains nutrients and water, and shields the enclosed population from desiccation, host defences and antimicrobial agents.

The matrix is chemically heterogeneous and is composed principally of exopolysaccharides, proteins, extracellular DNA and lipids, together with a large volume of retained water. Exopolysaccharides such as the polysaccharide intercellular adhesin of staphylococci and alginate, Pel and Psl of *Pseudomonas aeruginosa* provide structural cohesion and adhesion. Extracellular DNA, released partly through the controlled lysis of a subpopulation of cells, serves as an important structural component and contributes to matrix integrity, adhesion and the exchange of genetic material. Matrix proteins include structural adhesins, lectins and a range of secreted enzymes.[2,10]

Architecturally, mature biofilms are not uniform flat layers. They commonly form heterogeneous three-dimensional structures, frequently described as mushroom- or tower-shaped microcolonies, that are interspersed with open water channels. These channels function as a primitive circulatory system, allowing the convective flow of nutrients, oxygen

and signalling molecules into the depths of the biofilm and the removal of metabolic waste products. This spatial organisation generates steep chemical gradients of oxygen, pH and nutrients across the biofilm, so that cells located in different regions experience very different microenvironments and adopt correspondingly different physiological states.[11,12] This physiological heterogeneity is of central importance to the reduced antimicrobial susceptibility of biofilms.

3.2. Biofilm Formation: The Developmental Life Cycle

Biofilm formation is not a random accumulation of cells but a regulated, sequential developmental process that follows a broadly conserved series of stages, sometimes compared to the developmental programme of a multicellular organism.[13] Although the details vary between species and environments, the classical model of the biofilm life cycle comprises reversible attachment, irreversible attachment, microcolony formation, maturation into a three-dimensional structure, and finally dispersal (Figure 1). Each stage is accompanied by characteristic changes in gene expression and cell physiology.[14]

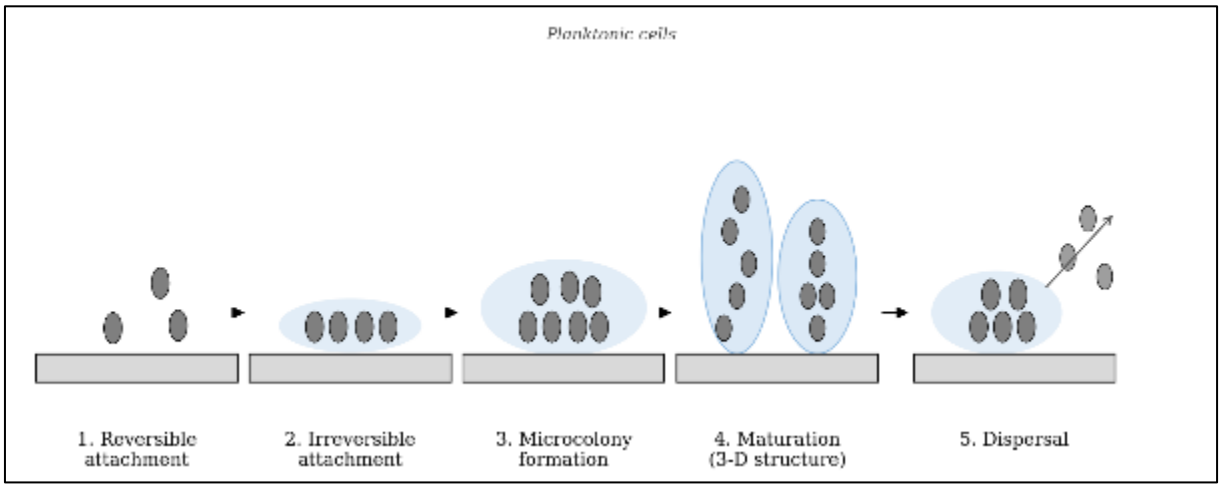

Figure 1 The five stages of the biofilm developmental life cycle, from the reversible attachment of planktonic cells to irreversible attachment, microcolony formation, maturation into a three-dimensional structure with water channels, and dispersal of cells to colonise new surfaces. Adapted from the conceptual model of Sauer et al.[14]

The process begins when planktonic cells approach and make initial, weak contact with a surface. On surfaces bathed in biological fluids, a conditioning film of host proteins such as fibronectin, fibrinogen and collagen is deposited almost immediately, and this film provides receptors that facilitate microbial adhesion. Initial attachment at this stage is reversible and is mediated by non-specific physicochemical forces, including van der Waals interactions, electrostatic forces and hydrophobic interactions, as well as by appendages such as flagella and pili, so that cells can still readily detach and return to the planktonic state.[13,15]

If cells remain in contact with the surface for long enough, the interaction becomes irreversible. Specific adhesins on the cell surface, such as the microbial surface components recognising adhesive matrix molecules of *Staphylococcus aureus*, bind firmly to the conditioning film and to the substratum. The cells begin to produce extracellular polymeric substances, which cement them permanently to the surface, so that detachment now requires strong shear forces or chemical or enzymatic disruption.[15]

The attached cells then proliferate and, together with the accumulating matrix, form multilayered clusters known as microcolonies. As the community grows, cell-to-cell communication through quorum sensing coordinates the further production of matrix and the development of the characteristic three-dimensional architecture, complete with towers, mushroom structures and water channels. During maturation, the biofilm differentiates spatially and metabolically, with actively growing cells near the oxygen- and nutrient-rich periphery and slow-growing or dormant cells in the anaerobic, nutrient-depleted interior.[12,14]

The final stage of the life cycle is dispersal, in which cells are released from the mature biofilm to colonise new surfaces and re-establish the cycle elsewhere. Dispersal may be passive, resulting from external shear forces, or active, arising from programmed changes within the biofilm such as the enzymatic degradation of the matrix and the up-regulation of motility. Clinically, dispersal is important because the periodic release of cells from a biofilm colonising an indwelling

device can seed the bloodstream, producing recurrent episodes of bacteraemia or systemic infection and acting as a persistent source of relapse.[5,14]

Underlying this coordinated behaviour is the cell-to-cell signalling system known as quorum sensing, through which bacteria regulate gene expression collectively once a threshold cell density is reached. Gram-negative bacteria typically employ acyl-homoserine lactones, whereas Gram-positive bacteria such as *Staphylococcus aureus* use autoinducing peptides through the accessory gene regulator system. Quorum sensing controls the production of the matrix, the expression of virulence factors and the initiation of dispersal, and it therefore represents an attractive target for anti-biofilm therapy.[16]

3.3. Clinically Important Biofilm-Forming Microorganisms

A wide range of bacteria and fungi are capable of forming clinically relevant biofilms, and infections frequently involve mixed communities of several species (Table 1). *Staphylococcus epidermidis* and other coagulase-negative staphylococci are the archetypal agents of device-associated biofilm infection; although they are relatively low in intrinsic virulence and form part of the normal skin flora, their outstanding capacity to adhere to and form biofilms on polymer surfaces makes them the leading cause of infections of intravascular catheters, prosthetic joints and other implanted materials.[17] *Staphylococcus aureus*, including methicillin-resistant strains, is a more virulent biofilm former responsible for device infections, infective endocarditis, osteomyelitis and chronic wound infection.

Pseudomonas aeruginosa is a paradigm biofilm-forming pathogen of great importance in cystic fibrosis, chronic wounds, burns, ventilator-associated pneumonia and contact-lens-associated keratitis, and its formation of thick, alginate-rich biofilms in the cystic fibrosis lung is a classic example of a chronic, essentially incurable biofilm infection.[8] Other clinically significant biofilm formers include *Enterococcus* species, *Klebsiella pneumoniae*, *Escherichia coli* (a major cause of catheter-associated urinary tract infection and recurrent cystitis), *Proteus mirabilis* (notable for crystalline biofilms that encrust and block urinary catheters), and streptococci associated with dental plaque and endocarditis. Among the fungi, *Candida albicans* and other *Candida* species form biofilms on catheters, prosthetic devices and mucosal surfaces and are a growing cause of device-associated and bloodstream infection.[1,18]

Table 1 Clinically important biofilm-forming microorganisms and their associated infections

Microorganism	Common biofilm-associated infections / sites
Staphylococcus epidermidis and other coagulase-negative staphylococci	Intravascular catheter infections, prosthetic joint and other implant infections, cerebrospinal fluid shunt infections
Staphylococcus aureus (including MRSA)	Device-associated infections, infective endocarditis, osteomyelitis, chronic wound infection
Pseudomonas aeruginosa	Cystic fibrosis lung infection, chronic wounds and burns, ventilator-associated pneumonia, contact-lens keratitis
Escherichia coli	Catheter-associated urinary tract infection, recurrent cystitis
Proteus mirabilis	Crystalline biofilms that encrust and block urinary catheters
Klebsiella pneumoniae and *Enterococcus* spp.	Device-associated infections, urinary tract and bloodstream infections
Streptococcus spp. (viridans group)	Dental plaque, dental caries, infective endocarditis
Candida albicans and other *Candida* spp.	Catheter and prosthetic-device infections, mucosal and bloodstream infections

3.4. Mechanisms of Antimicrobial Tolerance and Resistance

The reduced susceptibility of biofilms to antimicrobial agents is one of their most clinically consequential properties and is fundamentally different from the classical mechanisms of acquired resistance seen in planktonic bacteria. It is more accurately described as tolerance or recalcitrance, because it is largely a property of the biofilm state itself rather than of stable genetic changes, and cells dispersed from a biofilm frequently regain full antimicrobial susceptibility.[19,20]

The first mechanism is restricted penetration of antimicrobial agents through the extracellular matrix. The matrix can act as a diffusion barrier and as an adsorptive or reactive sink, binding, sequestering or chemically neutralising

antimicrobial molecules before they reach the deeper cells; positively charged aminoglycosides, for example, may be bound by the anionic matrix, and extracellular enzymes such as beta-lactamases concentrated within the matrix can degrade beta-lactam antibiotics.[6]

The second mechanism is the altered chemical microenvironment and the associated physiological heterogeneity within the biofilm. The metabolic activity of peripheral cells depletes oxygen and nutrients, creating anaerobic, nutrient-limited zones in the interior where cells grow slowly or enter a dormant, stationary-phase-like state. Because many antibiotics act preferentially on actively growing cells, these slow-growing populations survive treatment that would kill their fast-growing neighbours.[7,19]

The third and particularly important mechanism is the presence of persister cells, a small subpopulation of phenotypically dormant, highly tolerant cells that are not genetic mutants and are not killed by antimicrobial agents. After antibiotic treatment eliminates the susceptible bulk of the population, surviving persisters can regrow and repopulate the biofilm once the drug is withdrawn, giving rise to the characteristic pattern of relapse and chronicity of biofilm infection.[20,21]

Additional contributing factors include the up-regulation of multidrug efflux pumps, the induction of general and specific stress responses, and an enhanced opportunity for horizontal gene transfer within the densely packed community, which facilitates the spread of resistance determinants and helps to establish the biofilm as a reservoir for antimicrobial resistance genes.[20,22] The matrix and its architecture also impair host immune clearance by hindering phagocytosis and neutralising components of the immune response, so that biofilm infections persist even in the presence of a competent immune system and circulating antibodies.

3.5. Health Implications in Clinical Settings

The biological properties described above translate directly into a wide spectrum of persistent and difficult-to-treat human diseases. Biofilm-associated infections may be broadly divided into those forming on indwelling medical devices and other abiotic surfaces, and those forming on host tissues, although the underlying principles are the same.[5,18]

Any indwelling medical device introduced into the body provides an abiotic surface that is rapidly coated with a conditioning film of host proteins and thereby becomes an ideal substratum for biofilm formation. Central venous catheters and other intravascular devices are frequently colonised, and the resulting catheter-related bloodstream infections are a leading cause of healthcare-associated bacteraemia. Urinary catheters are colonised by biofilms that cause catheter-associated urinary tract infection, one of the most common of all hospital-acquired infections; certain organisms such as *Proteus mirabilis* produce urease and form crystalline biofilms that encrust and obstruct the catheter lumen.[9] Prosthetic joint infections and infections of other orthopaedic implants frequently require the surgical removal of the prosthesis in addition to prolonged antimicrobial therapy, while prosthetic and native heart valves may become the site of biofilm-mediated infective endocarditis. Other devices commonly affected include cardiac pacemakers and defibrillators, cerebrospinal fluid shunts, endotracheal tubes (a route to ventilator-associated pneumonia), intrauterine contraceptive devices and contact lenses.[9,23]

Biofilms also form directly on and within host tissues, giving rise to a group of chronic infections that are notoriously refractory to treatment. Chronic wounds, including diabetic foot ulcers, venous leg ulcers and pressure ulcers, are commonly colonised by polymicrobial biofilms that impair healing and perpetuate inflammation, and biofilm is now regarded as a principal reason for the failure of chronic wounds to heal. In cystic fibrosis, chronic pulmonary infection with *Pseudomonas aeruginosa* growing as biofilm in the airways is the major cause of progressive lung damage and remains essentially impossible to eradicate once established.[8] Other well-recognised biofilm-associated tissue infections include chronic and recurrent otitis media, chronic rhinosinusitis, chronic osteomyelitis, and dental biofilm diseases such as dental caries and periodontitis, in which the structured plaque biofilm is the direct cause of disease; recurrent urinary tract infection is likewise linked to biofilm-like reservoirs within the bladder epithelium.[18,24]

Beyond the patient, biofilms are also a feature of the inanimate hospital environment, where they form on surfaces, in drains and sinks, within water distribution systems, and on inadequately reprocessed instruments such as flexible endoscopes. These environmental biofilms act as persistent reservoirs of pathogens, including multidrug-resistant organisms, and have been implicated in outbreaks of healthcare-associated infection.[9]

The common thread running through all biofilm-associated infections is that they are chronic, persistent and recurrent, that they respond poorly to antimicrobial therapy at conventional doses, and that their definitive management frequently requires the physical removal of the colonised device or the surgical debridement of infected tissue.

Collectively, these infections impose a substantial burden of morbidity and mortality, prolong hospital stay, drive the repeated use of antibiotics and thereby contribute to the wider crisis of antimicrobial resistance, and generate very considerable healthcare costs.[8,24]

3.6. Laboratory Detection of Biofilms

The reliable detection of biofilm production is important both for research and for the clinical laboratory, because conventional culture of samples from biofilm infections often yields low counts or false-negative results, since the organisms remain firmly attached within the matrix rather than being freely suspended. Several phenotypic, microscopic and molecular methods are used to detect and quantify biofilm formation (Table 2).[5]

The tissue culture plate method, also known as the microtitre plate assay, is regarded as the reference standard for the quantitative detection of biofilm production. Organisms are grown in the wells of a microtitre plate, the loosely adherent cells are washed away, and the remaining adherent biofilm is fixed and stained, most commonly with crystal violet; the bound dye is then solubilised and its absorbance measured spectrophotometrically, providing a quantitative index of biofilm biomass that allows isolates to be classified as strong, moderate, weak or non-biofilm producers.[25,26] The tube method is a simple qualitative screening test in which an adherent, stained film lining the walls and base of a culture tube indicates biofilm production, while the Congo red agar method identifies biofilm- or slime-producing organisms by the formation of black, dry, crystalline colonies in contrast to the pink or red colonies of non-producers.[27]

Microscopic techniques provide direct visualisation of biofilm structure: scanning electron microscopy reveals surface topography and cellular arrangement in fine detail, while confocal laser scanning microscopy, combined with fluorescent viability dyes, permits non-destructive three-dimensional imaging of intact hydrated biofilms. Molecular methods, including the polymerase chain reaction, are used to detect genes associated with biofilm formation, such as the *ica* operon responsible for polysaccharide intercellular adhesin production in staphylococci, and to identify the organisms present within polymicrobial biofilms. In suspected device infection, sonication of the explanted device to dislodge adherent organisms, followed by quantitative culture of the resulting fluid, substantially improves microbiological diagnosis compared with conventional swab or tissue culture.[18,23]

Table 2 Laboratory methods for the detection of biofilm production

Method	Type	Principle and application
Tissue culture plate (microtitre plate) method	Quantitative	Adherent biofilm in microtitre wells is stained with crystal violet and measured spectrophotometrically; the reference standard for classifying strong, moderate, weak and non-producers
Tube method	Qualitative	An adherent stained film lining a culture tube after decanting and staining indicates biofilm production; simple but subjective
Congo red agar method	Qualitative	Biofilm/slime producers form black, dry, crystalline colonies, whereas non-producers form pink to red colonies
Scanning electron microscopy	Microscopic	High-resolution imaging of biofilm surface topography and cellular arrangement
Confocal laser scanning microscopy	Microscopic	Non-destructive three-dimensional imaging of intact hydrated biofilms with viability staining of live and dead cells
Polymerase chain reaction	Molecular	Detection of biofilm-associated genes such as the *ica* operon and identification of organisms within polymicrobial biofilms
Device sonication with quantitative culture	Culture-based	Ultrasound dislodges adherent organisms from an explanted device, improving microbiological diagnosis over conventional swab or tissue culture

3.7. Control and Management Strategies

Because established biofilms are so resistant to eradication, control rests on a combination of prevention, physical removal of the biofilm where possible, optimised conventional antimicrobial therapy, and a growing array of novel anti-biofilm approaches; no single strategy is universally effective, and management is often multimodal.[28,29]

Prevention is the most effective and cost-efficient strategy and centres on preventing the initial contamination and colonisation of surfaces. In relation to medical devices, this includes strict aseptic technique and hand hygiene during insertion and handling, maximal sterile barrier precautions, adherence to evidence-based insertion and maintenance care bundles, skin antisepsis with agents such as chlorhexidine, and the removal of devices as soon as they are no longer clinically required. Effective sterilisation and reprocessing of reusable instruments, together with rigorous environmental cleaning and disinfection, limit the establishment of environmental biofilm reservoirs.[9]

Considerable efforts have been directed towards engineering medical devices that resist biofilm formation. Catheters and other devices may be impregnated or coated with antimicrobial or antiseptic agents, such as minocycline and rifampin or chlorhexidine and silver sulfadiazine, or coated with silver nanoparticles, in order to inhibit initial colonisation, and anti-adhesive surface modifications and the selection of less colonisable biomaterials represent complementary approaches that have been shown in some settings to reduce the incidence of device-associated infection.[9,23]

Once a biofilm infection is established, the single most effective intervention is frequently the physical removal of the affected material, whether by removing an infected catheter or prosthesis or by the surgical debridement of infected and necrotic tissue. Where the device cannot be removed, antimicrobial therapy is optimised by using high doses, prolonged courses and rational combinations of agents; antibiotic lock therapy, in which a concentrated antimicrobial solution is instilled into and left within a catheter lumen, is used to treat and prevent intraluminal biofilm, and certain agents, notably rifampin in combination for staphylococcal device infection, possess useful activity against biofilm-embedded organisms.[18,21]

A wide range of novel strategies is under active investigation, many of which aim to disrupt the biofilm rather than simply to kill planktonic cells.[28,30] Quorum sensing inhibition, or quorum quenching, seeks to interfere with the signalling that coordinates matrix production and virulence, thereby preventing biofilm development or promoting dispersal without directly killing the cells, which may reduce the selective pressure for resistance.[16] Matrix-degrading agents target the structural integrity of the biofilm; enzymes such as deoxyribonuclease, dispersin B and alginate lyase can disperse the community and render the released cells more susceptible to antibiotics and immune clearance.[29]

Bacteriophage therapy exploits the ability of certain phages to penetrate and lyse biofilm cells and to produce depolymerising enzymes that degrade the matrix; antimicrobial peptides, which are small cationic host-defence molecules, show promising activity against biofilm cells including persisters; nanotechnology-based approaches, including metallic nanoparticles and targeted antimicrobial delivery, exploit both intrinsic antimicrobial activity and the ability to penetrate the matrix; and photodynamic therapy, which combines a photosensitising agent with light to generate reactive oxygen species, is being applied particularly to wound and oral biofilms.[22,30]

3.8. Challenges and Future Perspectives

Despite substantial advances in understanding, the management of biofilm-associated infection remains a formidable challenge. Standard antimicrobial susceptibility testing is performed on planktonic cells and does not reflect the tolerance of biofilm populations, so laboratory reports may seriously overestimate the likely response to therapy; the development of standardised, clinically validated biofilm susceptibility assays is therefore an important unmet need. The diagnosis of biofilm infection is itself difficult, because conventional culture frequently fails to recover the causative organisms, and many promising anti-biofilm agents remain at the experimental or preclinical stage and have yet to be translated into routine clinical practice.[20,28]

Future progress is likely to depend on the combination of several complementary strategies, on the development of biomaterials that intrinsically resist colonisation, on the rational integration of matrix-disrupting and anti-virulence agents with conventional antibiotics, and on improved rapid diagnostics. For the medical laboratory scientist, growing awareness of biofilms carries the practical implications of employing appropriate detection methods such as the microtitre plate assay and device sonication, of interpreting culture results with an appreciation of the limitations imposed by the biofilm mode of growth, and of contributing actively to surveillance and infection-control efforts.

4. Conclusion

Biofilms represent the predominant and default mode of microbial life and are central to a large proportion of persistent, chronic and device-associated human infections. The enclosure of microbial cells within a self-produced extracellular matrix, together with the physiological heterogeneity of the biofilm and the presence of tolerant persister cells, renders these communities profoundly resistant to antimicrobial agents and to host defences, and accounts for the chronic and

relapsing nature of biofilm disease. In the clinical setting, biofilms underlie infections of intravascular and urinary catheters, prosthetic joints and heart valves, chronic wounds, the cystic fibrosis lung, chronic otitis media, periodontitis and many other conditions, and they constitute a significant reservoir and driver of antimicrobial resistance. Effective control depends on prevention through aseptic device management and environmental hygiene, on the physical removal of colonised material, on optimised antimicrobial regimens, and increasingly on novel strategies that target the biofilm matrix and its signalling systems. A sound understanding of biofilm biology, together with the appropriate application of laboratory detection methods, is therefore essential to the modern medical laboratory scientist and to the wider effort to combat persistent infection and antimicrobial resistance.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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