



Review

Antibacterial surfaces developed from bio-inspired approaches

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ABSTRACT

Prevention of bacterial adhesion and biofilm formation on the surfaces of materials is a topic of major medical and societal importance. Various synthetic approaches based on immobilization or release of bactericidal substances such as metal derivatives, polyammonium salts and antibiotics were extensively explored to produce antibacterial coatings. Although providing encouraging results, these approaches suffer from the use of active agents which may be associated with side-effects such as cytotoxicity, hypersensitivity, inflammatory responses or the progressive alarming phenomenon of antibiotic resistance. In addition to these synthetic approaches, living organisms, e.g. animals and plants, have developed fascinating strategies over millions of years to prevent efficiently the colonization of their surfaces by pathogens. These strategies have been recently mimicked to create a new generation of bio-inspired biofilm-resistant surfaces. In this review, we discuss some of these bio-inspired methods devoted to the development of antibiofilm surfaces. We describe the elaboration of antibacterial coatings based on natural bactericidal substances produced by living organisms such as antimicrobial peptides, bacteriolytic enzymes and essential oils. We discuss also the development of layers mimicking algae surfaces and based on anti-quorum-sensing molecules which affect cell-to-cell communication. Finally, we report on very recent strategies directly inspired from marine animal life and based on surface microstructuring.

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1. Introduction

It is now accepted that microbial populations use cell attachment to solid substrates to survive, forming structured communities called biofilms. Biofilms are defined as biopolymer matrix-enclosed microbial populations adhering to each other and/or surfaces [1]. Consequently, two bacterial living states exist in natural environments: planktonic (free floating) and sessile (attached) states. While the planktonic growth mode is important for the bacterial spread, biofilms are necessary to allow bacteria to persist and to resist adverse environmental conditions. Therefore, biofilms occur on inert and living supports in natural environments and in industrial installations [2] (Fig. 1). Bacterial biofilms are responsible for a wide range of human infections, including otitis media, osteomyelitis, native valve endocarditis and cystic fibrosis pneumonia [1,3]. Bacterial biofilm infections are particularly problematic because sessile bacteria can withstand host immune responses and are drastically more resistant to antibiotics (up to 1000-fold), biocides and hydrodynamic shear forces than their

planktonic counterparts [4]. In humans, individuals with implanted medical devices, e.g. prostheses or catheters, and those with compromised immune systems, are considered to be most at risk of biofilm infections, and even humans with competent immune defenses often fail to resolve these infections independently. The protective mechanisms at work in biofilms appear to be distinct from those that are responsible for conventional antibiotic resistance. Although several mechanisms have been postulated to explain this reduced susceptibility of sessile organisms to antimicrobials, it is becoming evident that biofilm resistance is multifactorial [5]. Poor antibiotic penetration, nutrient limitation, slow growth, adaptive stress responses and the formation of multi-resistant cells are hypothesized to constitute a multi-layered defense [6]. Genetic and biochemical details of the biofilm defenses are now beginning to emerge. The biofilm phenotype of *Pseudomonas aeruginosa* appears to be regulated more at the translational and perhaps post-translational levels than at the transcriptional level, as highlighted by the discrepancies between microarray and proteomic experiments [7]. Whereas transcriptome analyses led to the conclusion that few genes showed differential expression in planktonic and biofilm cells [8,9] protein-based approaches suggested that a large number of proteins are differentially regulated during biofilm development [10,11]. Considering the high resistance of sessile micro-organisms to inhibitors, the eradication of biofilms needs

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