

Multiorganism Biofilms: A New Challenge for Ecto- and Endoprostheses

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Infection may be defined as invasion by and multiplication of pathogenic microorganisms in a bodily part or tissue, which may produce subsequent tissue injury and progress to overt disease through a variety of cellular or toxic mechanisms [1,2]. However, as our understanding of infection and the behaviour of microbiota expands then we can take issue with many of the tenets in this definition.

Bacteria do not need to invade tissue in order to cause a host reaction or cause to a pathological state within a patient. The presence of bacteria within dental plaque can lead to dental caries and bacteria within a chronic wound can lead to delayed wound healing [3-6]. Similarly microorganisms which in and of themselves are not inherently pathogenic can cause disease states or predispose to infection in otherwise healthy individuals.

Using dental plaque as an example we can see it represents a multiorganism adherent population of microbiota. This is often referred to as a biofilm. A more complete definition of a biofilm would be "an adherent population of microorganisms within a polysaccharide matrix of its own creation that demonstrates increased resistance to chemical and physical attacks" [7].

The advent of electron microscopy has allowed the adherent biofilms to be identified in a many different environments from ice-cream factories through to healthcare related devices [8-13]. There are many aspects to the biofilm that enhance its pathogenicity. A multiorganism biofilm, may have non-pathogenic bacteria that set-up the first crucial adherence step [14], or may even have candidal hyphae within them to enhance and strengthen the polysaccharide matrix within which they reside, while candida can certainly be pathogenic, it resides within the oral cavity of 25% of the population with no obvious ill-effects [15].

It is felt that the adherence step plays a role in the recidivism of biofilm infections, and why radical surgical debridement is often cited as a cornerstone of their effective management [16,17]. One of the other aspects that make biofilms so difficult to treat is the matrix within which they reside. This matrix has demonstrated increased resistance to many antibiotics, using both physical methods of resistance, such as a negatively polarized surface of the biofilm to repel antibiotics, and chemical methods, where bacteria within the body of the biofilm exist at different levels of metabolic activity and oxygen consumption, to render many antibiotics less effective [18-20]. This is further enhanced by the ready transmission of bacterial DNA within the biofilm allowing horizontal transfer of genetic bacterial resistance [21].

The biofilm polysaccharide matrix also provides a physical barrier against attack, such as debridement, and the action of the body's own host-defense peptides and neutrophils [22-24]. Indeed, the "fall-out" from these host defenses often results in damage to the surrounding tissues worsening existing ulceration [24-26].

While it is clear that many bacterial populations are highly developed and that biofilms represent a sophisticated phenotype with serious healthcare consequences, biofilms are not a new evolution of bacteria, and have been present since long before the discovery of penicillin [27]. Our growing understanding of this bacterial phenotype allows us to target therapies and co-ordinate management strategies

in a more focused fashion. Where ecto- and endoprostheses are used in diverse areas of healthcare, practitioners need to be cognizant of bacteria biofilms and to tailor their therapy stratagems accordingly.

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