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Forum for Interface Oral Health Science

Dental plaque as a biofilm and a microbial community – implications for treatment –

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Dental plaque is an example of both a biofilm and a microbial community. Biofilms are spatially-organised and highly structured, and are often composed of consortia of interacting micro-organisms, termed microbial communities, the properties of which are more than the sum of the component species. Members of microbial communities display a broader habitat range, a greater metabolic efficiency, and are harder to treat with antimicrobial agents (cross-protection). Microbial gene expression alters markedly in biofilms; organisms communicate by gene transfer and by secretion of diffusible signalling molecules. Cells in biofilms are more tolerant of antimicrobial agents.

The microbial composition of dental plaque is highly diverse, and includes high numbers of obligate anaerobic and (currently) unculturable species. These micro-organisms form part of the human resident microbiota, and live in harmony with the host, and provide essential benefits, including (a) conferring colonisation resistance, (b) modulating the host responses, and (c) contributing to normal host physiological processes (blood pressure regulation, mucus production, etc).

On occasions, this symbiotic relationship is perturbed, and the natural balance between host and microbiota breaks down (dysbiosis), resulting in a shift in the proportions of species in the microbial community, thereby increasing the risk of disease. The microbiota from carious lesions is characterised by increased proportions of acidogenic and acid-tolerating bacteria (including, but not exclusively, mutans streptococci, lactobacilli and bifidobacteria). In contrast, periodontal diseases display an even more diverse microbiota, with increases in obligately anaerobic and proteolytic bacteria, and many unculturable taxa can be detected.

Modelling studies, using defined, mixed culture consortia grown under controlled conditions in a chemostat, with and without surfaces for biofilm formation, demonstrated unequivocally that it was possible to drive the selection of either a cariogenic or a periodontal microbiota by repeatedly exposing a community of predominantly beneficial resident bacteria to environmental cues that reflect events in disease. Thus, the competitiveness of cariogenic bacteria was favoured by repeated conditions of low pH, but not just by sugar pulses per se, while periodontal-associated organisms were selected by conditions linked to inflammation (e.g. supply of proteins/glycoproteins found in gingival crevicular fluid; a rise in pH). This led to the proposal of an 'ecological plaque hypothesis', and subsequently to an 'extended ecological plaque hypothesis' for caries.

The key feature of an ecological approach to controlling dental disease is that it is not sufficient to merely target interventions at the putative pathogens; the factors that are driving the deleterious shifts in the resident microbiota need to be identified and then removed or controlled. In this way, the selection of bacteria within the biofilm community with damaging traits is prevented while the essential benefits that are provided by the resident oral microbiota are retained.

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