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Pathogenic Oral Biofilms

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Editorial

Biofilms are microbial colonies embedded in an extracellular matrix that produce a highly organised structure that is responsible for a variety of human illnesses. Dental caries (tooth decay) is a polymicrobial biofilm disease caused by a solid surface's diet and microbiota-matrix interactions. Sugars promote ecological changes and concerted multispecies efforts that are conducive to acid damage of the mineralized tooth tissue by fueling the emergence of pathogens, the assembly of the matrix, and the acidification of the biofilm microenvironment, promoting ecological changes and concerted multispecies efforts that are conducive to acid damage of the mineralized tooth tissue. Because it provides a diverse nutrient input, high humidity, and changing oxygen concentrations, the mouth cavity is a unique habitat that allows for colonisation of a wide diversity of commensal microbial species. Furthermore, the presence of non-shedding soft (gingiva) and hard (tooth) tissues offers microbes with possible surfaces for adhesion and subsequent interaction with diverse host cells. In healthy people, oral cavity colonisation is predicated on a balance of bacteria-host and interbacterial interactions. Periodontitis is caused by the presence of dental plaques in gingival tissues, as well as interactions between pathobionts and host cells (PD). Biofilms are responsible for or exacerbate many infectious illnesses. Mouth infectious diseases are prime instances of the outcomes of dynamic interactions between microorganisms, their hosts, and the host's food, which result in microbial colonisation of oral surfaces and the formation of pathogenic biofilms (or dental plaque). Advances in DNA and RNA sequencing technologies are providing crucial details regarding the biofilm microbiota's variety in composition, genomic content, and activities at various oral locations.

Many variables influence the composition of the microbiota present on various surfaces of the mouth, particularly when teeth erupt, offering new, non-shedding sites for commensals and opportunistic pathogens to colonise. Age, food, dental hygiene, systemic and immunological diseases, and the use of some medications that cause hyposalivation, for example, are only a few of them. The importance of nutrition in microbial colonisation has been thoroughly demonstrated in people and experimental animals. The structure and composition of biofilms formed on teeth changes significantly when hosts are over-exposed to dietary sugars, and the residing microbial communities become highly fit to metabolise carbohydrates and produce acids, resulting in dental caries. Sequencing of ancient dental plaque provides additional evidence that shifts in oral microbiota are associated with dietary changes. For example, *Streptococcus mutans* (a cariogenic bacterium) was not detected in plaque sam. Mutans first appeared in the fossil record and became increasingly common as the consumption of refined sugars in the diet increased, as did the prevalence of dental caries [1-3].

Although early research focused on the microbial makeup of biofilms, it is now evident that microorganisms in biofilms are embedded in a matrix of extracellular polymeric molecules (EPS). The matrix's relevance in collective microbial activity and pathogenicity, as well as antibiotic tolerance, is being increasingly acknowledged and regarded vital to the biofilm lifestyle. Microbial attachment to a surface and cell-to-cell adhesion are directly mediated by EPS synthesis, which also forms a polymeric matrix that improves biofilm mechanical durability. Furthermore, the diffusion-modifying properties of the EPS matrix cause chemical/nutrient gradients to form, resulting in microenvironments within biofilms that differ significantly from other sites in key environmental inputs known to influence microbial behaviour, such as pH, redox, and nutrient availability. As a result, the matrix enables cells to form cohesive multicellular ecosystems in which cooperative and antagonistic interactions occur within a heterogeneous chemical and physical milieu, allowing for the formation of localised niches with varying pathogenic potentials.

Gram-negative bacteria with a lower amount of pellicle adhesion, such as representatives of the genera *Veillonella* and *Fusobacterium*, can coaggregate thanks to the surface molecules of these early invaders. Bridge species are bacteria from the genus *Fusobacterium*, such as *Fusobacterium nucleatum*, that may coaggregate with both early and late colonisers. They are known to promote the successful establishment of dental biofilm. *F. nucleatum* uses surface molecules such RadD, an arginine-inhibitable adhesin, and Fap2, a fusobacterial apoptosis protein, to bridge nearby bacteria. The genus *Corynebacterium* is notably specialised for supragingival and subgingival plaque, according to habitat analysis of the oral microbiome, with *Corynebacterium matruchotii* dominating among six species deposited in the Human Oral Microbiome Database [4,5].

Conflict of Interest

None.

References

1. Bowen, William H., Robert A. Burne, Hui Wu, and Hyun Koo. "Oral biofilms: pathogens, matrix, and polymicrobial interactions in microenvironments." *Trends Microbiol* 26 (2018): 229-242.
2. Radaic, Allan, Changchang Ye, Brett Parks and Li Gao, et al. "Modulation of pathogenic oral biofilms towards health with nisin probiotic." *J Oral Microbiol* 12 (2020): 1809302.
3. Szafrański, Szymon P., Jørgen Slots, and Meike Stiesch. "The human oral phageome."*J Periodontol* 86 (2021): 79-96.
4. Do, Thuy, Deirdre Devine, and Philip D. Marsh. "Oral biofilms: molecular analysis, challenges, and future prospects in dental diagnostics." *Clin Cosmet Investig Dent* 5 (2013): 11.
5. Palmer, Robert J., Richard Darveau, Richard J. Lamont and Bente Nyvad, et al. "Human oral bacterial biofilms: Composition, dynamics, and pathogenesis. "*Biofilm Infec* (2011) 35-68.

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