Occupational Medicine & Health Affairs

Short Communication Open Access

# An Overview On Dental Plaque

**Karthik Madipalli**

*Department of Human Physiology, SRMC University, Chennai, India*

Dental plaque is a structurally- and functionally-organized biofilm. Plaque forms in an ordered way and has a diverse microbial composition that, in health, remains relatively stable over time (microbial homeostasis). The predominant species from diseased sites are different from those found in healthy sites, although the putative pathogens can often be detected in low numbers at normal sites. In dental caries, there is a shift toward community dominance by acidogenic and acid-tolerating species such as mutans streptococci and lactobacilli, although other species with relevant traits may be involved. Strategies to control caries could include inhibition of biofilm development (e.g. prevention of attachment of cariogenic bacteria, manipulation of cell signaling mechanisms, delivery of effective antimicrobials, etc.), or enhancement of the host defenses. Additionally, these more conventional approaches could be augmented by interference with the factors that enable the cariogenic bacteria to escape from the normal homeostatic mechanisms that restrict their growth in plaque and out compete the organisms associated with health. Evidence suggests that regular conditions of low pH in plaque select for mutants streptococci and lactobacilli. Therefore, the suppression of sugar catabolism and acid production by the use of metabolic inhibitors and non-fermentable artificial sweeteners in snacks, or the stimulation of saliva flow, could assist in the maintenance of homeostasis in plaque. Arguments will be presented that an appreciation of ecological principles will enable a more holistic approach to be taken in caries control.

Dental plaque is the community of microorganisms found on a tooth surface as a biofilm, embedded in a matrix of polymers of host and bacterial origin [1,2]. Of clinical relevance is the fact that biofilms are less susceptible to antimicrobial agents, while microbial communities can display enhanced pathogenicity (pathogenic synergism) [3]. The structure of the plaque biofilm might restrict the penetration of antimicrobial agents, while bacteria growing on a surface grow slowly and display a novel phenotype, one consequence of which is a reduced sensitivity to inhibitors [4]. Plaque is natural and contributes (like the resident microflora of all other sites in the body) to the normal development of the physiology and defenses of the host [5].

## Development of dental plaque biofilms

Dental plaque forms via an ordered sequence of events, resulting in a structurally- and functionally-organized, species-rich microbial community. Distinct stages in plaque formation include: acquired pellicle formation; reversible adhesion involving weak long-range physico-chemical interactions between the cell surface and the pellicle, which can lead to stronger adhesin-receptor mediated attachment; co-adhesion resulting in attachment of secondary colonizers to already attached cells (Cisar – this symposium)[6]; multiplication and biofilm formation (including the synthesis of exopolysaccharides) and, on occasion, detachment. The increase in knowledge of the mechanisms of bacterial attachment and co-adhesion could lead to strategies to control or influence the pattern of biofilm formation (Cisar – this symposium).

Analogs could be synthesized to block adhesin-receptor attachment or co-adhesion, and the properties of the colonizing surfaces could be chemically modified to make them less conducive to microbial colonization. However, cells can express multiple types of adhesin [7,8], so that even if a major adhesin is blocked, other mechanisms of attachment may be invoked. Furthermore, although adhesion is necessary for colonization, the final proportions of a species within a mixed culture biofilm such as dental plaque will depend ultimately on the ability of an organism to grow and outcompete neighboring cells. Once formed, the overall composition of the climax community of plaque is diverse, with many species being detected at individual sites.

## Dental plaque and disease

Numerous studies have been undertaken to determine the composition of the plaque microflora from diseased sites in order to try and identify those species directly implicated in causing pathology. Interpretation of the data from such studies is difficult because plaque-mediated diseases occur at sites with a pre-existing diverse resident microflora, and the traits associated with cariogenicity (acid production, acid tolerance, intracellular and extracellular polysaccharide production) are not restricted to a single species. A comparison of the properties of strains representing several streptococcal species have shown considerable overlap in the expression of these cariogenic traits [9] (see below). Microorganisms in biofilms such as plaque are in close physical contact, and this can increase the probability of interactions, some of which can modulate the pathogenic potential of cariogenic bacteria (for example, Kuramitsu and Wang – this symposium). Similarly, the consequence of acid production by cariogenic species can be ameliorated by the development of food chains with Veillonella spp., or due to base production by neighboring organisms. Not surprisingly, therefore, there has been only limited success in using the presence of specific species as diagnostic or prognostic indicators of disease.

## Source of cariogenic pathogens

The origin and role of oral pathogens has been the subject of much debate. Indeed, the resolution to this debate is pivotal to the development of effective plaque control strategies. Early studies using conventional culture techniques often failed to recover the putative pathogens from healthy sites or, when pathogens were present, they comprised only a small proportion of the microflora. However, the recent application of more sensitive molecular techniques has led to the frequent detection of low levels of several pathogens (implicated in caries and periodontal diseases) at a wide range of sites [10]. Bacterial typing schemes have shown that identical strains of putative cariogenic bacteria can be found in the plaque of mother (or other close caregiver) and infants [11], implying that transmission of such bacteria can occur.

***Corresponding author:** Karthik Madipalli, Department of Human Physiology, SRMC University, Chennai, India, Tel: 7659845558; E-mail: karthikma@gmail.com

**Received** January 23, 2021; **Accepted** February 6, 2021; **Published** February 13, 2021

**Citation:** Madipalli K (2021) An Overview On Dental Plaque. Occup Med Health Aff 9: 336.

## References


1. Socransky SS, Haffajee AD. Dental biofilms: difficult therapeutic targets. Periodontology. 2002;28:12–55.
2. Marsh PD. Dental plaque as a microbial biofilm. Caries Res. 2004;38:204–211.
3. van Steenbergen TJM, van Winkelhoff AJ, de Graaff J. Pathogenic synergy: mixed infections in the oral cavity. Antonie van Leeuwenhoek. 1984;50:789–798.
4. Gilbert P, Maira-Litran T, McBain AJ, Rickard AH, Whyte FW. The physiology and collective recalcitrance of microbial biofilm communities. Adv Microb Physiol. 2002;46:203–255.
5. Marsh PD. Role of the oral microflora in health. Microb Ecol Health Dis. 2000;12:130–137.
6. Kolenbrander PE, Andersen RN, Kazmerak KM, Palmer RJ. Coaggregation and coadhesion in oral biofilms. In: Allison DG, Gilbert P, Lappin-Scott HM, Wilson M, editor. Community structure and co-operation in biofilms. Cambridge, Cambridge University Press, Society for General Microbiology Symposium 59; 2000. pp. 65–85.
7. Hasty DL, Ofek I, Courtney HS, Doyle RJ. Multiple adhesins of streptococci. Infect Immun. 1992;60:2147–2152.
8. Zhang Y, Lei Y, Nobbs A, Khammanivong A, Herzberg MC. Inactivation of Streptococcus gordonii SspAB alters expression of multiple adhesin genes. Infect Immun. 2005;73:3351–3357.
9. de Soet JJ, Nyvad B, Kilian M. Strain-related acid production by oral streptococci. *Caries Res.* 2000;**34**:486–490.
10. Tanner AC, Milgrom PM, Kent R, Mokeem SA, Page RC, Riedy CA, Weinstein P, Bruss J. The microbiota of young children from tooth and tongue samples. J Dent Res. 2002;81:53–57.
11. Tanner AC, Milgrom PM, Kent R, Jr, Mokeem SA, Page RC, Liao SI, Riedy CA, Bruss JB. Similarity of the oral microbiota of pre-school children with that of their caregivers in a population-based study. Oral Microbiol Immunol. 2002;17:379–387.