

# Dental plaque as a biofilm

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## COMMENTARY

Dental plaque is a biofilm of microorganisms that forms on the surface of a tooth and is immersed in a matrix of host and bacterial polymers. Biofilms are less vulnerable to antimicrobial drugs, whereas microbial colonies can have increased pathogenicity. This has clinical implications (pathogenic synergism). The plaque's construction Biofilm may limit antimicrobial agent penetration, but bacteria living on a surface grow slowly and exhibit a unique phenotypic, one of which is lower susceptibility to inhibitors. Plaque is natural and contributes to the normal development of the host's physiology and defenses (together with the resident microflora of all other places in the body). Dental plaque is formed by a series of activities that culminate in a species-rich, structurally, and functionally structured microbial population. 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 involving secondary colonizers attaching to already attached cells (Cisar - this symposium); multiplication and biofilm formation (including the synthesis of exopolysaccharides) and, on occasion, detach.

The irreversible solubilization of tooth mineral by acid produced by bacteria adhering to the tooth surface in bacterial populations known as dental plaque causes dental decay. The bacteria *Streptococcus mutans* is the most common cause of tooth decay. The evolution of the lesion is linked to several *lactobacilli* followed by the ingestion of foods containing fermentable carbohydrates, the tooth surface loses some tooth mineral due to the acid produced by plaque bacteria. Saliva replenishes this mineral in between meals. When fermentable foods are consumed often, however, the low pH in the plaque is maintained, resulting in a net loss of mineral from the tooth. This low pH favors aciduric organisms like *S. mutans* and *lactobacilli*, which store polysaccharide and produce acid long after the food has been swallowed (particularly *S. mutans*). Although dental decay has been known from the beginning of time, it was not a major health concern until sucrose became a common component of the human diet. When sugar is consumed often, a bacteria called *Streptococcus mutans* becomes the dominant organism, and it is this bacterium that has been linked to dental disease. *S. mutans* was

first isolated from human caries lesions in 1924, but it was not researched extensively until the 1960s, when it was re-identified as the causative agent of a transmissible caries infection in rodent models. In these experiments, all of Koch's infectivity postulates were met in animal models. Because *S. mutans* were a part of the natural flora on the teeth, it was difficult to show that an increase in *S. mutans* truly preceded and/or corresponded with the early clinical lesion.

Because the severe discomfort that sends the patient to the dentist is usually always resolved by a dental restoration or extraction, a microbiologic diagnosis for a *S. mutans/lactobacilli* infection is rarely sought. As a result, the presence of an underlying *S. mutans* infection would have no effect on the treatment. Microbiologic diagnosis, on the other hand, might be beneficial in the patient's therapy to avoid or limit further degradation. When an expensive procedure, such as orthodontic therapy or the insertion of dental crowns and bridges to replace missing teeth, is planned, such scenarios may arise. A microbiologic examination would be beneficial at the conclusion of any restorative therapy to determine the extent of *S. mutans* and *lactobacilli* colonization on the teeth. An ordered sequence of events results in a physically and functionally organized, species-rich microbial population in dental plaque. 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 involving secondary colonizers attaching to already attached cells (Cisar - this symposium); multiplication and biofilm formation (including the synthesis of exopolysaccharides); and, on occasion, detach. Several investigations have been conducted to evaluate the makeup of plaque microflora from sick areas to identify the species solely responsible for pathology. Because plaque-mediated diseases occur at sites with a diverse resident microflora, and the traits associated with carcinogenicity (acid production, acid tolerance, intracellular and extracellular polysaccharide production) are not limited to a single species, interpreting the data from such studies is difficult. When the qualities of strains representing different streptococcal species were compared, it was discovered that there was a lot of overlap in the expression of these cariogenic traits.

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