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Biofilm: A New View of Plaque

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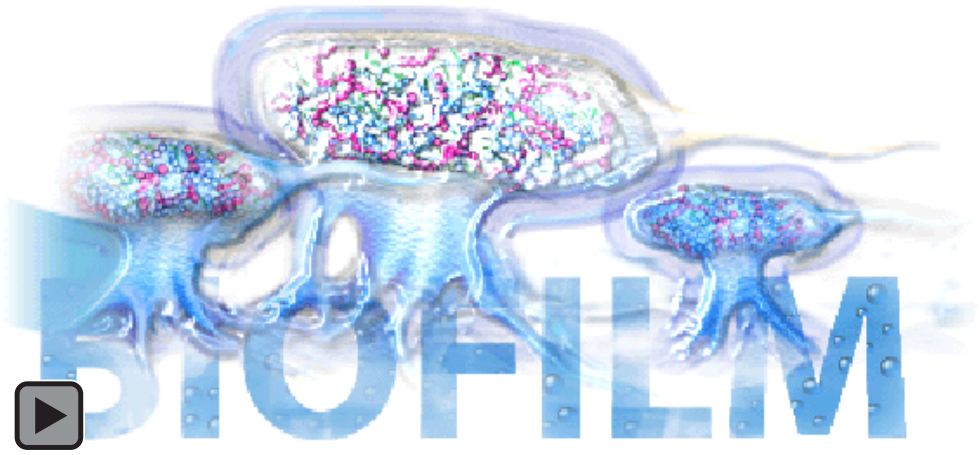


Abstract

Dental researchers have attempted to understand the microbial nature of oral diseases over the past 120 years. Their view of plaque and its constituent microorganisms has shifted from a specific plaque hypothesis to a non-specific plaque hypothesis and back again to a theory of specific periodontal pathogens in plaque. Changes in the way plaque and its microorganisms are viewed affect the strategies used to prevent and control periodontal diseases. In recent years, dental researchers have begun to view plaque as a biofilm. This shifting view of plaque has important implications for future efforts in prevention and treatment. This article describes the various ways that dental professionals have viewed plaque throughout the years and highlights the current view of plaque as a biofilm and the ramifications for periodontal therapy.

Keywords: Dental plaque, bacterial plaque, biofilm, dental biofilm

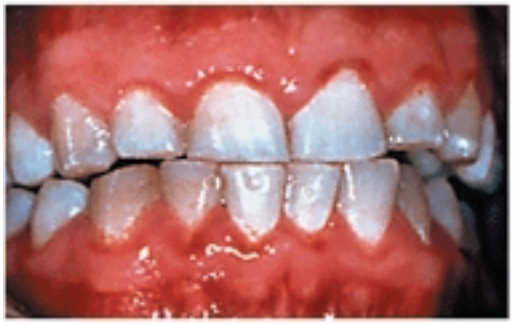


Figure 1: Gingivitis

Introduction

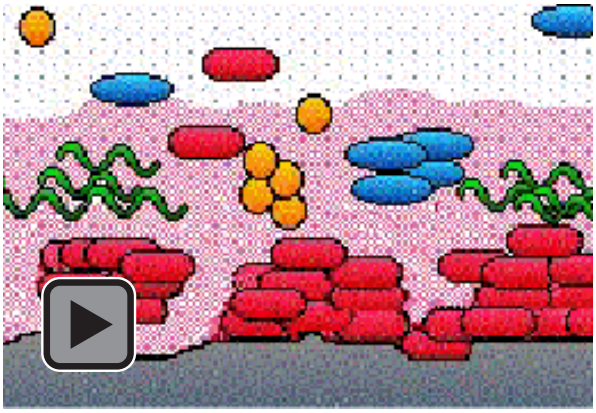
Despite the best efforts of dental health professionals, oral infections are still widespread. The average adult in the U.S. has from 10 to 17 decayed, missing or filled permanent teeth.[1] The majority of the U.S. population experiences gingivitis, with a smaller proportion experiencing moderate to severe periodontal disease (Figure 1).[2]

Figure 2: Oral microorganisms in dental plaque showing typical "corn-cob" structure of bacterium.

There is universal recognition these oral infections are multifactorial, with specific bacteria residing in intraoral plaques as a necessary, but not sufficient cause of disease. Exactly how these plaque-dwelling microorganisms (Figure 2) cause oral diseases is not completely clear. Understanding the nature of dental plaque and its resident microorganisms is dictated by the analytical tools used to study it. Consequently, this influences the strategies used to control and prevent dental diseases.[3] During the past two decades newer scientific methods have changed the view of dental plaque so dental scientists now see it as a biofilm.[1]

Biofilm

A biofilm is a well organized, cooperating community of microorganisms.[4,5] The slime layer that forms on rocks in streams is a classic example of a biofilm (Figure 3). So is the plaque that forms in the oral cavity. Biofilms are everywhere in nature. They form under fluid conditions. It is estimated over 95 percent of bacteria existing in nature are in biofilms.[5] Sometimes biofilms are seen as positive, such as their use for detoxification of waste water and sewage. More often biofilms provide a challenge for humans.[3,5]



The slime layer that forms in dental unit water lines is an example familiar to most dental professionals. Biofilms can also be found lining oil pipelines, fish tanks, indwelling catheters, internal implants, contact lenses, and prosthetic devices (Figure 4). Biofilms also can be deadly. Legionnaire's disease that killed 29 persons in Philadelphia in 1976 was ultimately traced to a bacteria in the biofilm of the air conditioning system. Millions of dollars are spent each year working to control these biofilms.[3,6]

Figure 4: Biofilm found on dental equipment.

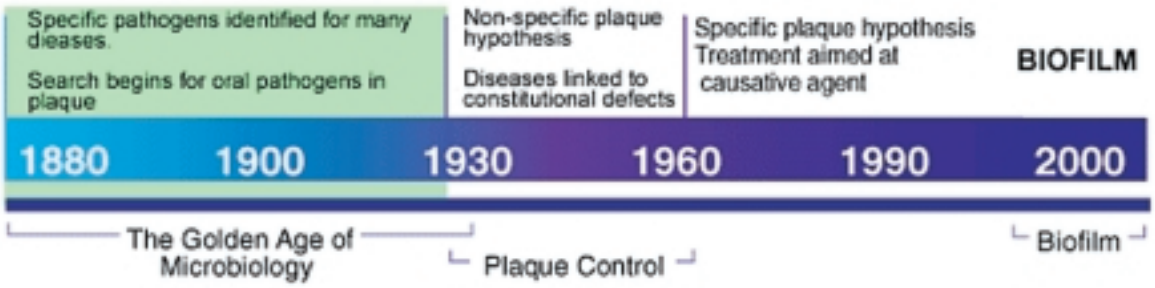


Figure 4: The Changing Views of Plaque and Periodontal Diseases (1880 to 1930)

Changing Views of Plaque

The National Institute for Dental and Craniofacial Research recently hosted an international conference on microbial ecology. This meeting focused on a new view of plaque as a biofilm. The conference highlighted the importance of this shift in thinking about dental plaque and its role in oral diseases.[1] This is not the first time in history dental professionals have shifted their thinking about plaque. Over the past 120 years the view of dental plaque has gone through several changes.

The period from 1880 to 1930 was called the golden age of microbiology (Figure 4).[7] During this period, the pathogens that caused many systemic infections of medical importance were identified. Researchers also looked for a single, specific cause of oral diseases. Assuming plaque contained the microorganism that caused periodontal disease, dental scientists studied plaque in search of the causative agent. Using the techniques available at that time (wet mounts or stained smear microscopy), scientists identified four different groups of potential etiologic agents for periodontal diseases. Amoebae, spirochetes, fusiforms and streptococci were isolated from patients with periodontal diseases and, therefore, suggested as possible etiologies. Periodontal treatments of those times varied according to the suspected causative agents and included dyes, systemic administration of an arsenic-containing antimicrobial preparations, intramuscular injection of mercury as well as vaccines.[8]

The 1930's ushered in a different view of the role of plaque and its microorganisms in the etiology of periodontal disease (Figure 5). Dental scientists believed that periodontal disease was linked with some constitutional defect in the individual.[8] Mechanical irritants such as calculus and overhanging restorations were also thought to play a major role in the pathogenesis of periodontal disease.[9]

The belief there was a single microbial agent that caused periodontal disease was replaced by non-specific plaque theories.[8] Non-specific plaque hypothesis held that the entire bacterial flora in plaque played a role in periodontal destruction rather than specific bacteria. All plaque was viewed as bad plaque. Furthermore, plaque meant more disease. Plaque control was viewed as essential to limit the production of gingival irritants that lead to inflammation and periodontal destruction.[10] Identification of specific microorganisms was not important. Stringent plaque control was important, and it became the focus of periodontal therapy.

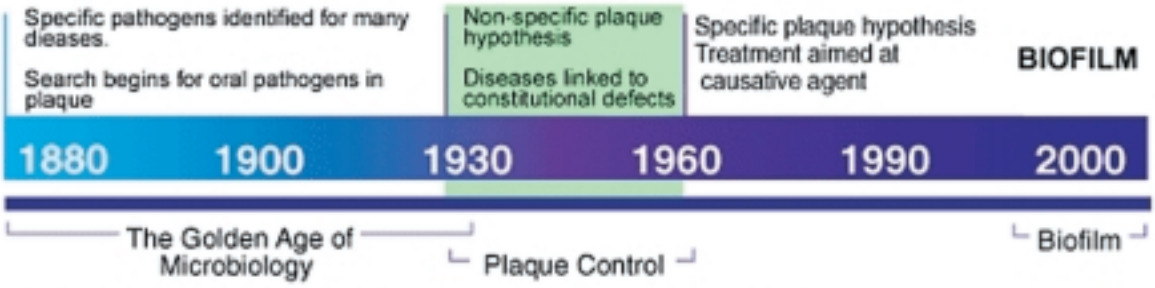


Figure 5: The Changing Views of Plaque and Periodontal Diseases (1930 - 1960)

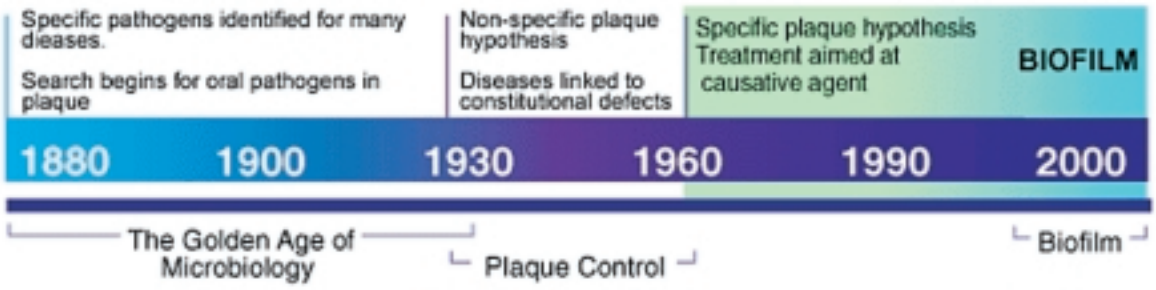


Figure 6: The Changing Views of Plaque and Periodontal Diseases (1960 to Current)

The 1960's marked a return to specific plaque hypotheses (Figure 6). Researchers were successful in showing that periodontal disease could be transmitted between hamsters.[11] The electron microscope confirmed spirochetes were in the connective and epithelial tissues of patients with acute necrotizing ulcerative gingivitis in contrast to healthy controls.[12] Believing there were differences in plaque brought about by different species, scientists again returned to the search for a specific microbial periodontal pathogen and treatment aimed at the causative agent.[8]

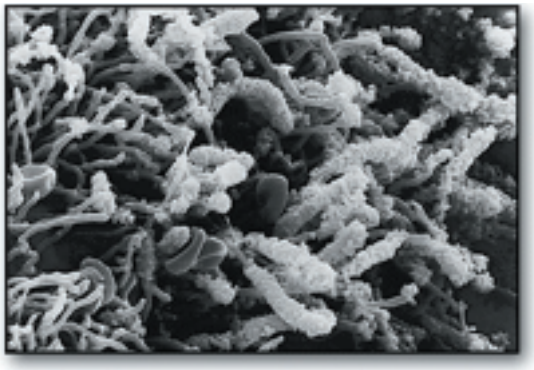


Figure 7: SEM of mature human dental plaque demonstrating corn cob formation. Bar = 10 microns at an original magnification of 2,020. Courtesy of Dr. Charles Cobb. University of Missouri-Kansas City

Newer methods of microbial analysis such as darkfield microscopy, transmission electron microscopy, scanning electron microscopy, DNA probes, BANA hydrolysis and immunoassay have aided the search.[13]

Since that time, scientists have continued to search for the specific etiologic agent with mixed success. Haffajee and Socransky[14] have detailed the reasons for the difficulties in pinpointing specific pathologic agents. Some of these difficulties are related to microbial sampling and culturing. These difficulties include: obtaining a sample from a periodontal pocket, the difficulty cultivating some organisms, and the large number of possible periodontal pathogens that may be found and cultivated from a periodontal pocket. Sampling is further complicated by the fact that periodontal pockets contain not only pathogens, but also opportunistic species. Other difficulties in pinpointing periodontal pathogens are related to the nature of periodontal diseases themselves. First, periodontal disease is not a single disease, but a collection of different diseases. Secondly, these diseases produce periods of disease activity and inactivity and variations in disease activity in different sites within an individual. A final difficulty in identifying specific periodontal pathogens is the variation in individual host response.[15]

In spite of these challenges, current researchers continue to agree that periodontal diseases are infections caused by specific pathogens. Recently, attention has turned to *Bacteroides forsythus,* as well as, *P. gingivalis* and *A. actinomycetemcomitans* as primary pathogens for most periodontal infections with moderate evidence linking another subset of microorganisms *(C. rectus, E. nodatum, F. nucleatum, P. intermedia/ nigrescens, P. micros, S. intermedium, and T. denticola)* as possible pathogens.[13,16] Researchers are working to develop diagnostic tests for detection and treatments designed to target periodontic pathogens. Systemic antibiotics such as amoxicillin, metronidazole, tetracycline, doxycycline, and augmentin have been proposed.[14] Local delivery of antimicrobials of tetracycline fibers, metronidazole and minocycline gels, chlorhexidine chips, and doxycycline polymer have also been introduced.[17] While these approaches have enhanced our ability to manage periodontal diseases, they have still failed to provide uniform success. Viewing plaque as a biofilm promises to aid in the effort to effectively manage periodontal disease.

Plaque as a Biofilm

Previously, bacteria have been studied as they grew in colonies on culture plates in the laboratory. More sophisticated microscopy, such as confocal scanning laser, has permitted examination of the biofilms in their natural states.[5]

Microorganisms in biofilm behave differently than bacteria in a culture medium (see Table 1).

Table 1. Basic Biofilm Properties[21]

- Cooperating community of various types of microorganisms
- Microorganisms are arranged in microcolonies
- Microcolonies are surrounded by protective matrix
- Within the microcolonies are differing environments
- Microorganisms have primitive communication system
- Microorganisms in biofilm are resistant to antibiotics, antimicrobials, and host response

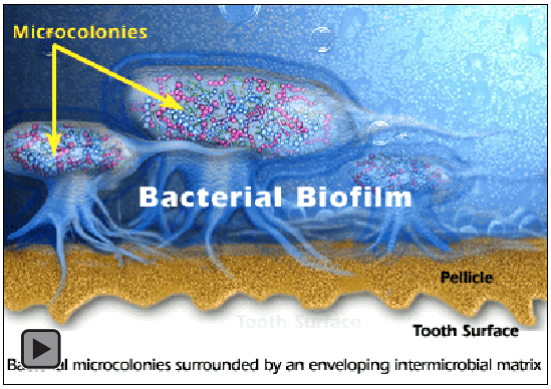


Figure 8: Artisitic Depiction of Plaque Biofilm

Seen through a microscope, bacteria in a biofilm are not distributed evenly. They are grouped in microcolonies surrounded by an enveloping intermicrobial matrix (Figure 8).

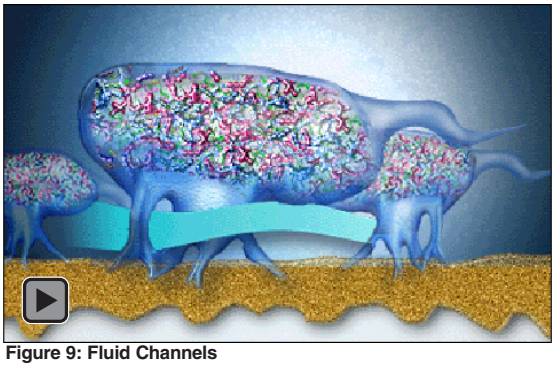


Figure 9: Fluid Channels

The matrix is penetrated by fluid channels that conduct the flow of nutrients, waste products, enzymes, metabolites, and oxygen. These microcolonies have micro environments with differing pH's, nutrient availability, and oxygen concentrations (Figure 9). The bacteria in a biofilm communicate with each other by sending out chemical signals (Figure 10). These chemical signals trigger the bacteria to produce potentially harmful proteins and enzymes.[5]

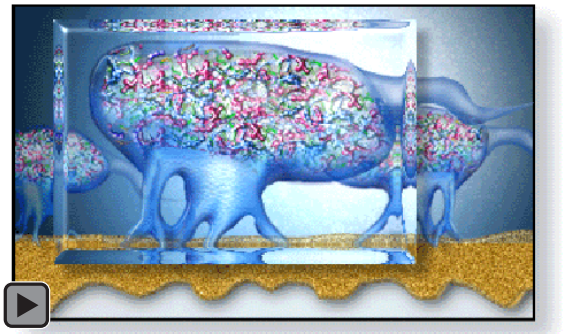
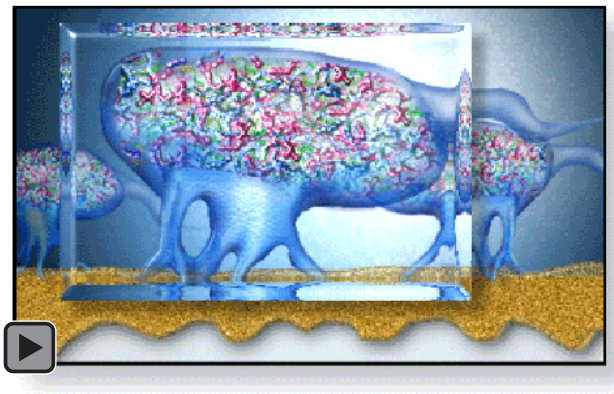


Figure 10: Biofilm bacteria communicate by sending out chemical signals.

Our previous attempts to predict and control periodontal diseases have been based on the performance of bacteria cultured under laboratory conditions.[1,5] Increased understanding of biofilms have demonstrated there are great differences between bacterial behavior in laboratory culture and in their natural ecosystems. For example, bacteria in biofilm produce compounds in biofilm that they do not produce when in culture. Also, the matrix surrounding the microcolonies serves as a protective barrier. This helps explain why systemic and locally delivered antimicrobials have not always proven successful, even when they were targeted at specific microorganisms. It also helps explain why mechanical plaque control and personal oral hygiene have continued to be an integral part of periodontal therapy.[18] Biofilms can be removed by mechanical means. However, they immediately begin to reform, so the search continues for ways to combat biofilms.



New Frontiers

Industrial researchers are pursuing new technology to combat biofilm. One approach is to interfere with the signaling between bacteria in biofilm so they can't communicate with each other. Another tact is to mimic the natural defenses developed by ocean creatures like whales and dolphins that don't accumulate bacterial biofilms.[5] Dental researchers are also pursuing new strategies to control oral biofilms[1,19] (see Table 2).

Table 2. Possible Strategies to Control Oral Biofilm[21]

Control of nutrients
- addition of base-generating nutrients (arginine)
- reduction of GCF flow through anti-inflammatory agents
- inhibition of key microbal enzymes

Control of biofilm pH
- sugar substitutes
- antimicrobial agents
- fluoride
- stimulate base production

Control of redox potential
- redox agents
- oxygenating agents

Varying the oxygen concentration, pH, and nutrient availability in plaque have been show to modulate biofilm microflora and may prove useful. For example, periodontal pathogens require a low redox potential for growth. Addition of a redox agent, such as methylene blue, to periodontal pockets has been shown to inhibit the growth of *P. Gingivalis.*[20] Since increased gingival crevicular flow (GCF) increases the nutrient supply for subgingival biofilm, control of GCF may be used in the future to control subgingival biofilm. Use of anti-inflammatory agents may not only help inhibit destructive host pathways, anti-inflammatory agents may also reduce the nutrient supply of GCF for the biofilm community. NIDCR is currently supporting research in this area with the goal of new therapies for the future.[1]

Summary
Dental researchers have attempted to understand the microbial nature of oral diseases over that past 120 years. The view of plaque and its constituent microorganisms have shifted from specific plaque hypothesis to a non-specific plaque hypothesis and back again to a theory of specific periodontal pathogens in plaque. Recently dental researchers have begun to view plaque as a biofilm. The nature of a biofilm helps explain why periodontal diseases have been so difficult to prevent and treat. An improved understanding of biofilm will lead to new strategies for management of these widespread diseases.

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