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DENTAL BIOFILMS: PATHOGENESIS, CLINICAL SIGNIFICANCE, AND CONTEMPORARY STRATEGIES FOR INFECTION CONTROL

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Abstract. Background: Dental biofilms are organized microbial communities that develop on tooth surfaces and other oral structures. Their composition and metabolic activity are closely associated with the development of dental caries, gingivitis, and periodontitis. The structural organization of biofilms, their extracellular matrix, and interactions among microorganisms contribute to their persistence and reduced susceptibility to antimicrobial agents. **Objective:** This review aims to describe the biological characteristics of dental biofilms, explain their roles in major oral diseases, and evaluate established and emerging approaches to biofilm control in clinical dentistry. **Methods:** A narrative review of relevant scientific literature was conducted using publications on oral microbiology, biofilm formation, periodontal disease, antimicrobial tolerance, and emerging antibiofilm strategies. The review draws on peer-reviewed articles published in established scientific journals and professional guidance from the American Dental Association. It is not a systematic review, and no quantitative meta-analysis was performed. **Results:** Dental biofilms are dynamic, multispecies microbial ecosystems embedded in an extracellular polymeric matrix. Their pathogenic potential depends on microbial composition, environmental conditions, dietary substrate availability, host immune responses, and oral hygiene. In cariogenic biofilms, repeated carbohydrate fermentation and acid production promote enamel demineralization. In periodontal disease, dysbiotic subgingival communities interact with the host immune system and contribute to connective tissue destruction and alveolar bone loss. Mechanical biofilm disruption remains central to prevention and treatment. Fluoride-based caries prevention, professional periodontal instrumentation, and individualized oral hygiene are established interventions. Antimicrobial photodynamic therapy, biofilm-disrupting enzymes, nanoparticles, probiotics, bacteriophages, and cold atmospheric plasma are being investigated, but the strength of clinical evidence varies considerably. **Conclusion:** Effective dental biofilm management requires an understanding of microbial ecology and host–microbe interactions rather than an exclusive focus on individual bacterial species. Established preventive and therapeutic measures remain fundamental, while emerging technologies require further well-designed clinical trials to establish their efficacy, safety, and long-term benefits.

Keywords: dental biofilm; dental plaque; oral microbiome; dental caries; periodontitis; antimicrobial tolerance; periodontal therapy; oral health.

Introduction

Dental biofilms represent an important biological factor in the development of common oral diseases. Unlike microorganisms freely suspended in saliva, biofilm-associated microorganisms adhere to surfaces and form organized communities characterized by interactions among different microbial species and their surrounding environment [7; 8].

The oral cavity provides numerous ecological niches, including enamel surfaces, gingival margins, periodontal pockets, the tongue, and other mucosal surfaces. Each niche has distinct

environmental conditions, such as oxygen availability, nutrient supply, pH, and exposure to host immune factors. These differences influence the composition and activity of resident microbial communities.

Dental plaque should not be regarded simply as an accumulation of bacteria. It is a structured microbial ecosystem whose properties emerge from interactions among microorganisms, the extracellular matrix, dietary components, saliva, and host tissues [2; 8].

Under conditions compatible with oral health, microbial communities can coexist with the host without producing clinically significant tissue damage. However, changes in diet, oral hygiene, salivary function, and local environmental conditions may favor microbial communities associated with disease.

Dental caries and periodontitis illustrate two distinct consequences of biofilm-associated dysbiosis. Cariogenic biofilms promote the repeated production of organic acids and the demineralization of dental hard tissues. Periodontitis involves a dysbiotic subgingival community and an inflammatory host response that damages the periodontal ligament and alveolar bone [1; 2].

Understanding these mechanisms is essential for developing preventive strategies and selecting appropriate treatments.

Biological structure and composition of dental biofilms

1. Formation of the acquired pellicle

Biofilm development on a clean tooth surface begins with the adsorption of salivary proteins, glycoproteins, and other molecules, forming the acquired enamel pellicle.

This pellicle modifies the surface properties of enamel and provides binding sites for microorganisms. It also participates in lubrication and influences interactions between the tooth surface and the oral environment.

The pellicle is not itself equivalent to mature dental plaque. Rather, it provides an initial interface that facilitates microbial attachment.

2. Initial microbial adhesion

Early colonizers attach to the acquired pellicle through specific interactions between bacterial adhesins and salivary components. Members of the genera *Streptococcus* and *Actinomyces* commonly participate in early dental biofilm development.

Initial attachment is followed by microbial growth and the recruitment of additional species. Co-adhesion and coaggregation allow different microorganisms to establish close physical relationships within the developing community [7].

These interactions contribute to the spatial organization of oral biofilms and influence nutrient exchange, metabolism, and community stability.

3. Extracellular polymeric matrix

A defining feature of biofilms is their extracellular matrix, which contains water, polysaccharides, proteins, lipids, extracellular DNA, and other microbial or host-derived components.

The matrix provides structural cohesion, helps retain microorganisms on surfaces, and modifies the local chemical environment. Its composition varies according to the microbial community, available nutrients, and environmental conditions.

In cariogenic biofilms, extracellular polysaccharides produced by bacterial glucosyltransferases can facilitate bacterial adhesion and aggregation. Some of these polysaccharides contribute to the formation of a matrix that promotes the retention of acids near the tooth surface [5].

Extracellular DNA is another important matrix component. Research has associated bacterial extracellular DNA with biofilm architecture, microbial interactions, and responses to antimicrobial stress. Its precise contribution varies according to the organisms and conditions involved [6].

4. Maturation and dispersion

As the biofilm develops, microbial growth and matrix production create a more complex community. Different regions may have different oxygen concentrations, nutrient availability, pH values, and metabolic activity.

These microenvironments allow microorganisms with different physiological requirements to coexist. Biofilm development is therefore a dynamic process rather than a simple, uniform increase in bacterial numbers.

Cells and aggregates may subsequently detach or disperse from the community. Dispersed microorganisms can colonize other surfaces, contributing to the spread and renewal of biofilms.

The architecture and behavior of oral biofilms depend on multiple interacting factors, including salivary flow, dietary patterns, mechanical disturbance, and host conditions [7].

Dental biofilms in the pathogenesis of oral diseases

1. Dental caries

Dental caries is a biofilm-mediated, diet-modulated disease characterized by the progressive demineralization of dental hard tissues.

Frequent exposure to fermentable carbohydrates provides substrates for acid production by oral microorganisms. When acid production exceeds the buffering and remineralizing capacity of the oral environment, the mineral balance shifts toward demineralization.

Streptococcus mutans is an important cariogenic organism because it can metabolize fermentable carbohydrates, produce acids, and contribute to extracellular polysaccharide formation. Nevertheless, caries is not caused exclusively by this species. Other acidogenic and acid-tolerant organisms can participate in cariogenic communities [5].

The ecological shift toward acid-producing and acid-tolerant microorganisms is particularly important. Repeated decreases in biofilm pH select for organisms capable of surviving and remaining metabolically active under acidic conditions.

The resulting imbalance between demineralization and remineralization can lead to the development of carious lesions.

Clinical implications: Caries prevention should focus on reducing the frequency of fermentable carbohydrate exposure, maintaining effective plaque removal, using fluoride appropriately, and assessing individual caries risk. These measures address the ecological

conditions that promote disease rather than attempting to eliminate every microorganism from the oral cavity.

2. Gingivitis

Gingivitis is an inflammatory condition of the gingiva associated with dental plaque accumulation. Clinical manifestations may include erythema, edema, and bleeding during brushing or periodontal probing [3].

The inflammatory response develops as the host recognizes microbial products and activates local immune pathways. If plaque accumulation persists, the inflammatory burden may increase.

Unlike periodontitis, plaque-induced gingivitis does not involve the characteristic progressive loss of periodontal attachment or alveolar bone. In many cases, it can be reversed through effective biofilm control and professional care.

Gingivitis is clinically important because it indicates an imbalance between plaque accumulation and the host's capacity to maintain periodontal health.

3. Periodontitis

Periodontitis is a chronic inflammatory disease associated with dysbiotic subgingival biofilms and a dysregulated host response. It can result in progressive destruction of the periodontal ligament and alveolar bone, periodontal pocket formation, gingival recession, and, in advanced cases, tooth loss [1; 2].

Periodontal disease cannot be explained solely by the presence of a single pathogen. Instead, the composition and function of the subgingival microbial community interact with host susceptibility and environmental risk factors.

The inflammatory response is essential for controlling microbial challenge, but persistent inflammation can also damage host tissues. Cytokines, inflammatory mediators, and pathways regulating bone remodeling contribute to the tissue destruction observed in susceptible individuals.

Smoking, diabetes, oral hygiene, and other patient-specific factors may influence disease severity and treatment outcomes.

Clinical implications: Periodontal therapy aims to reduce pathogenic biofilm and calculus, control inflammation, address modifiable risk factors, and establish conditions that support long-term periodontal stability. Professional subgingival instrumentation and ongoing supportive periodontal care remain central components of management [1].

Why dental biofilms are difficult to control

1. Reduced antimicrobial susceptibility

Biofilm-associated microorganisms can demonstrate substantially different responses to antimicrobial agents compared with free-living, planktonic cells.

This phenomenon is often described as biofilm-associated antimicrobial tolerance. It should be distinguished from inherited antimicrobial resistance, although the two phenomena can coexist.

Several mechanisms contribute to reduced susceptibility:

- Limited or uneven penetration of certain antimicrobial agents through the biofilm matrix.
- Local differences in pH, oxygen concentration, and nutrient availability.

- Slow growth and altered physiological states in some biofilm populations.
- Stress-response mechanisms and interactions among microbial species.
- Persistence of subpopulations capable of surviving antimicrobial exposure.

The relative importance of these mechanisms depends on the biofilm and the agent being studied. The extracellular matrix does not necessarily prevent every antimicrobial from penetrating; physiological heterogeneity and the host environment can also be important [4].

Biofilm tolerance may help explain why an intervention that inhibits planktonic bacteria in laboratory testing does not always produce equivalent results against a mature biofilm.

2. Multispecies interactions

Oral biofilms contain microorganisms that can cooperate, compete, exchange metabolites, and influence one another's growth.

Some species produce substances that serve as nutrients for other organisms, while others modify environmental conditions in ways that support different community members.

These relationships may alter acid production, inflammatory potential, and responses to treatment. Consequently, studying individual species in isolation may not fully reproduce the behavior of complex oral biofilms [7].

3. Host–microbe interactions

Biofilm-associated disease emerges from interactions between microbial communities and host tissues.

Saliva provides mechanical clearance, buffering capacity, antimicrobial components, and proteins that influence microbial adhesion. The immune system also contributes to maintaining oral health.

When the balance between microbial challenge and host responses is disrupted, inflammation and tissue damage may occur. This means that effective biofilm management must consider both microbial factors and the patient's clinical condition.

Established strategies for dental biofilm control

1. Mechanical plaque removal

Mechanical biofilm disruption remains a fundamental component of oral disease prevention.

Toothbrushing and interdental cleaning help remove plaque from accessible surfaces. Professional dental procedures can remove plaque and calculus from areas that are difficult to clean effectively at home [9].

In periodontitis, professional subgingival instrumentation, including scaling and root planing where indicated, is a principal nonsurgical treatment approach [1].

The effectiveness of these procedures depends on appropriate technique, access to affected sites, patient adherence, and continuing maintenance.

Mechanical plaque control should therefore be understood as part of a long-term preventive strategy rather than a single procedure.

2. Fluoride-based caries prevention

Fluoride plays an important role in preventing dental caries by reducing demineralization and promoting remineralization under appropriate conditions.

Fluoride-based toothpastes and professionally applied fluoride preparations may be selected according to age, caries risk, and clinical indications.

Fluoride should not be described simply as an agent that eradicates dental biofilms. Its major preventive contribution involves modifying the mineral balance at the tooth surface and reducing the progression of carious lesions.

3. Chemical adjuncts

Antiseptic mouthrinses and other antimicrobial products may be useful in selected clinical situations. Chlorhexidine, for example, has established applications in particular oral care settings.

However, chemical adjuncts do not replace effective mechanical plaque removal. Their suitability depends on the indication, treatment duration, adverse effects, and patient-specific factors.

Unnecessary or prolonged antimicrobial exposure should be avoided. Systemic antibiotics are not routinely indicated for uncomplicated dental plaque accumulation or as a substitute for appropriate mechanical periodontal therapy.

4. Risk-based prevention and maintenance

The management of dental biofilms should be individualized according to the patient's caries risk, periodontal status, oral hygiene practices, dietary habits, smoking status, salivary function, and relevant systemic conditions.

Regular professional assessment allows clinicians to identify disease progression, reinforce preventive measures, and adjust treatment when necessary.

In patients with periodontitis, supportive periodontal care is particularly important because disease-associated biofilm can re-establish after active treatment.

Emerging strategies for biofilm management

1. Antimicrobial photodynamic therapy

Antimicrobial photodynamic therapy combines a photosensitizing agent with light of an appropriate wavelength to generate reactive oxygen species capable of damaging microorganisms.

This approach has been investigated as an adjunct to conventional periodontal treatment and for the control of selected oral biofilms.

Its potential advantages include localized application and a mechanism that differs from that of conventional antibiotics. Nevertheless, treatment outcomes depend on the photosensitizer, light delivery, biofilm characteristics, and clinical protocol.

Photodynamic therapy should not be considered a universal replacement for mechanical debridement. Its additional clinical benefit and appropriate indications must be evaluated from controlled clinical studies.

2. Biofilm-disrupting enzymes

Enzymatic strategies seek to degrade components of the extracellular matrix or interfere with processes required for biofilm stability.

Potential targets include extracellular polysaccharides, proteins, and extracellular DNA. By weakening matrix structure, such interventions may increase the accessibility of microorganisms to other treatments.

Recent research has highlighted extracellular DNA as a possible target for modifying dental biofilm structure [6].

However, activity demonstrated in laboratory models does not establish clinical effectiveness. Enzyme stability, delivery, tissue compatibility, effects on beneficial oral microorganisms, and the risk of unwanted tissue damage require further investigation.

3. Nanoparticle-based approaches

Nanotechnology has been explored for targeted antimicrobial delivery, localized release of active compounds, and modification of biofilm interactions.

Certain nanoparticles exhibit antimicrobial activity under experimental conditions, while others are designed to carry established therapeutic agents.

Potential benefits include improved delivery to selected sites and reduced exposure of surrounding tissues. However, nanoparticle size, composition, concentration, aggregation, and interactions with saliva may influence performance.

Before routine clinical adoption, these approaches require adequate evidence regarding biocompatibility, long-term safety, effective dosing, and superiority or added value compared with established care.

4. Probiotics and microbiome modulation

Microbiome-directed approaches aim to influence oral microbial communities rather than indiscriminately eliminate microorganisms.

Proposed mechanisms include competition for adhesion sites, competition for nutrients, production of inhibitory substances, and modulation of host responses.

The oral microbiome is complex, and an intervention that benefits one population or clinical outcome may not produce the same effect in another setting.

Consequently, probiotic products should be evaluated according to the specific strains used, dosage, treatment duration, patient population, and measured clinical outcomes. They should not be assumed to prevent or treat all biofilm-associated diseases.

5. Bacteriophages and targeted antimicrobial approaches

Bacteriophages are viruses that infect susceptible bacterial hosts. Their potential application in dentistry includes targeting selected bacterial populations while limiting effects on unrelated microorganisms.

Other experimental approaches include antimicrobial peptides and strategies directed at bacterial signaling or biofilm matrix formation.

These methods are promising research areas, but their effectiveness may be limited by host specificity, biofilm penetration, microbial diversity, and the development of resistance to the intervention itself.

More clinical evidence is needed to determine whether targeted approaches can provide reliable benefits in routine dental practice.

6. Cold atmospheric plasma

Cold atmospheric plasma has been investigated for its potential antimicrobial effects on oral microorganisms and biofilms.

Depending on the device and operating conditions, plasma-generated reactive species may damage microbial structures and affect biofilm viability.

Nevertheless, results from laboratory studies cannot be directly translated into clinical recommendations. Treatment parameters, tissue safety, reproducibility, and clinical outcomes require careful evaluation.

Cold atmospheric plasma should therefore be regarded as an investigational approach until sufficiently robust evidence supports specific indications.

Clinical and research implications

The expanding understanding of dental biofilms has important implications for preventive and therapeutic dentistry.

First, biofilm control should focus on disease-associated ecological changes and clinical outcomes rather than the complete elimination of the oral microbiome. Many oral microorganisms are compatible with health, and indiscriminate antimicrobial activity may disrupt beneficial microbial communities.

Second, the complexity of oral biofilms highlights the limitations of laboratory studies that use a single bacterial species. Multispecies models and clinically relevant conditions can provide additional insight into the interactions that influence biofilm behavior.

Third, new treatment strategies should be assessed using clearly defined outcomes. These may include plaque accumulation, gingival inflammation, probing pocket depth, clinical attachment level, caries incidence, adverse effects, and the durability of clinical improvement.

Finally, emerging technologies should be compared with established care rather than evaluated only against untreated controls. Well-designed randomized controlled trials, appropriate follow-up periods, standardized outcome measures, and transparent reporting are necessary to determine their true clinical value.

Future research should also investigate whether patient-specific characteristics, including salivary properties, microbial composition, behavioral factors, and systemic risk factors, can improve the selection of preventive and therapeutic interventions.

Limitations

This article is a narrative review and does not provide a systematic assessment of every relevant publication. The literature was selected to discuss established biological concepts, current clinical approaches, and emerging research directions.

No formal systematic search protocol, duplicate screening process, quantitative risk-of-bias assessment, or meta-analysis was conducted. Consequently, the review should not be interpreted as an exhaustive evaluation of all available evidence.

The clinical maturity of emerging technologies also varies. Some approaches are supported mainly by laboratory or preclinical research, whereas others have been studied in clinical settings.

Their efficacy and safety should therefore be interpreted according to the level and quality of the available evidence.

Conclusion

Dental biofilms are complex microbial ecosystems that contribute to the development of dental caries, gingivitis, and periodontitis. Their pathogenic potential depends on microbial composition, extracellular matrix organization, environmental conditions, and interactions with host tissues.

Biofilm-associated antimicrobial tolerance presents an important challenge because microorganisms within structured communities may respond differently to treatment than planktonic cells. Effective management consequently requires strategies that address biofilm organization, disease-associated microbial activity, and host factors.

Mechanical plaque removal, fluoride-based caries prevention, appropriate professional periodontal treatment, and individualized maintenance remain the foundation of clinical biofilm management. Emerging approaches—including photodynamic therapy, matrix-disrupting enzymes, nanoparticles, probiotics, bacteriophages, and cold atmospheric plasma—may offer additional options in the future, but their clinical applications must be supported by high-quality evidence.

A balanced approach combining established preventive measures with rigorous evaluation of new technologies offers the most reliable pathway toward improved oral health and more effective control of biofilm-associated disease.

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