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ORAL BIOFILM AND ITS ROLE IN THE DEVELOPMENT OF INFLAMMATORY COMPLICATIONS

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ABSTRACT

Oral biofilm represents a complex, structured microbial ecosystem that plays a key role in the development of inflammatory diseases affecting both hard and soft tissues of the oral cavity. Contemporary research confirms that it is the organized microbial biofilm, rather than isolated microorganisms, that serves as the primary etiological factor in dental caries, gingivitis, periodontitis, peri-implantitis, and complications of prosthetic treatment.

The aim of this study was to analyze the structural and functional characteristics of oral biofilm and its influence on the development of inflammatory complications in dental patients. The mechanisms of microbial adhesion, maturation, and resistance, as well as their interaction with the host immune system, are discussed. The obtained data support the necessity of a comprehensive preventive approach aimed at controlling biofilm formation.

Keywords

biofilm, oral microbiota, inflammation, dental caries, periodontitis, adhesion, resistance.

Relevance of the Topic

Over recent decades, the concept of microbial biofilm has significantly changed the understanding of the pathogenesis of dental diseases. According to the World Health Organization, oral diseases remain among the most prevalent non-communicable diseases worldwide.

Biofilm is formed as a highly organized system consisting of bacteria, fungi, and their metabolites embedded within an extracellular polysaccharide matrix. The resistance of biofilm to antimicrobial agents largely explains the chronic course of inflammatory processes and the high rate of recurrence.

The issue becomes particularly significant in prosthetic treatment, as well as in the presence of retention areas, orthodontic appliances, and dental implants, which create additional conditions for microbial colonization.

Introduction

Oral diseases occupy a leading position among chronic human pathologies. According to the World Health Organization, dental caries and periodontal diseases are among the most widespread diseases globally.

Modern research has demonstrated that the primary etiological factor of inflammatory processes is not an individual microorganism, but rather an organized microbial community — the biofilm. The transition from the concept of “specific infection” to the model of “microbial ecosystem dysbiosis” has become an important milestone in the development of dental science.

Cuticle, Pellicle, and Dental Deposits

1. Enamel Cuticle

The enamel cuticle is a dense, thin structure resistant to acid exposure. A tooth erupts with this protective covering, which disappears during the first months of life.

1.1 Primary cuticle (Nasmyth's membrane) — the final secretory product of ameloblasts, representing a thin inner layer of glycoproteins.

1.2 Secondary cuticle — a layer of reduced enamel epithelium forming a thicker outer layer.

2. Pellicle

The pellicle is a layered organic film of salivary-bacterial origin. It is an acellular structure firmly attached to the enamel surface. It forms as a result of continuous adsorption of salivary mucoproteins and facilitates the transport of substances through enamel.

3. Types of Dental Deposits

• **Dental plaque —** a structured cellular formation attached to enamel, consisting of living and dead microorganisms embedded in a polysaccharide matrix.

• **White soft plaque —** a yellowish-white cellular mass not firmly attached to enamel, composed of disorganized microorganisms and desquamated epithelial cells.

• **Food debris** — consists of freshly retained food particles and microorganisms**.**

• **Supragingival calculus —** a moderately hard mineralized deposit containing calcium phosphate salts (hydroxyapatite); yellow in color, becoming darker in smokers.

• **Subgingival calculus —** a very hard organic microbial matrix mineralized with calcium phosphate salts; typically dark brown in color.

Enamel Metabolism

Mature enamel is relatively inert and does not contain cells, as ameloblasts degenerate after tooth eruption and enamel formation. For this reason, enamel is incapable of regeneration after damage.

However, metabolic exchange (primarily of ions) occurs between enamel and saliva. The incorporation of ions into enamel is called remineralization, while the loss of ions from enamel is referred to as demineralization.

Structural Organization of Biofilm

Biofilm represents a three-dimensional structure composed of:

bacterial cells, an extracellular polysaccharide matrix, proteins, nucleic acids, metabolic products.

The matrix performs protective and structural functions, ensuring microbial resistance to mechanical and chemical influences.

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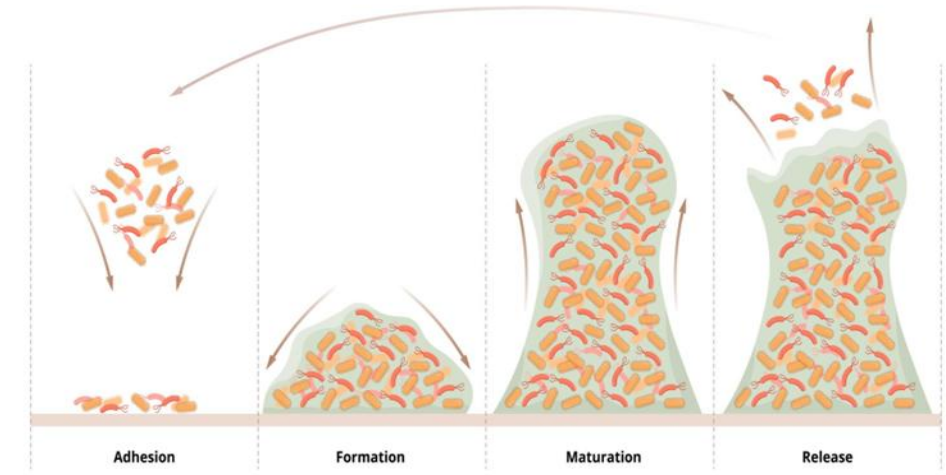


Main stages of biofilm formation

Stages of Biofilm Formation

The formation of dental biofilm occurs in several sequential stages:

Formation of the acquired pellicle on the enamel surface.

Primary adhesion of Gram-positive cocci (Streptococcus spp.).

Coaggregation and attachment of secondary colonizers.

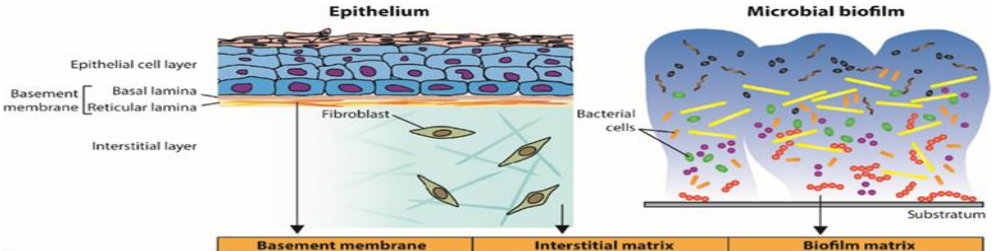
Maturation with the development of anaerobic zones.

Microbial dispersion and spread of infection.

Anaerobic microorganisms, including Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola, play a particularly significant role in the development of inflammation.

Microbial Communication and Resistance

One of the key properties of biofilm is the intercellular communication system (quorum sensing), which ensures coordinated bacterial activity.



	Basement membrane	Interstitial matrix	Biofilm matrix
Key components			
Polysaccharides			Glucans, capsules, PNAG, LTA
Nucleic acids			eDNA
Proteins	Collagen IV, nidogen, entactin	Collagen I & III	Amyloids, adhesins, enzymes, DNA binding proteins
Proteoglycans	Heparin sulfate proteoglycans	Glycosaminoglycans	Peptidoglycan fragments
Glycoproteins	Laminins	Fibronectin, elastin, tenascin C	S layers, adhesins
Others			Lipids, LPS
Key functions			
Adhesion	Anchors epithelium	Tissue support	Attachment of microbial cells
Structural support	Biomechanical resistance	Biomechanical resistance	Biomechanical resistance
Cell permeability	Cell migration (e.g. wound healing)	Cell migration (e.g. wound healing)	Restricts immune cell penetration
Signalling	Mechanosensing, integrin binding	Mechanosensing, chemokines	Quorum sensing
Homeostasis	Protect epithelial cells	Tissue homeostasis	Protection, nutrient store
Others			Gene pool

Mechanisms of resistance include:

limited diffusion of antiseptics,

altered metabolic activity,

formation of persister cells,

horizontal gene transfer of resistance determinants.

These mechanisms explain the chronic nature of inflammatory diseases and the difficulties associated with their treatment.

Role of Biofilm in the Development of Inflammatory Complications

Biofilm is involved in the pathogenesis of:

dental caries;

gingivitis;

periodontitis;

peri-implantitis;

denture stomatitis.

Chronic microbial stimulation leads to activation of immune cells and the release of pro-inflammatory cytokines (IL-1β, TNF-α), resulting in connective tissue destruction and bone resorption.

Biofilm and Prosthetic Constructions

The presence of removable and fixed prosthetic appliances creates additional retention areas that promote microbial accumulation. Prosthetic materials differ in their degree of microbial adhesion, which necessitates an individualized hygienic approach.

Modern Methods of Biofilm Control

Biofilm control includes:

Modern Methods of Biofilm Control

Biofilm control includes:

professional oral hygiene;

use of antiseptics (chlorhexidine, cetylpyridinium chloride);

mechanical plaque removal;

application of antimicrobial peptides;

photodynamic therapy.

A comprehensive approach demonstrates the highest effectiveness in preventing inflammatory complications.

Discussion

Current evidence confirms that the key factor in inflammatory diseases is disruption of microbial balance (dysbiosis), rather than the presence of a single pathogen. Biofilm functions as an integrated biological system with high adaptive capacity.

Therefore, biofilm control should be considered a priority in preventive dentistry.

Conclusion

Oral biofilm is the primary pathogenetic factor in inflammatory dental diseases. Its structural organization and resistance to antimicrobial agents contribute to the chronic course of pathological processes. Modern preventive strategies should focus on early biofilm control and maintenance of microbial homeostasis.

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