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Article

Bacterial Inflammation of Paradontal Tissues, Etiology and Pathogenesis

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Abstract: The human body is a super-organism that coexists with trillions of microorganisms, and the oral microbiome plays a crucial role in the development of periodontal diseases. Periodontitis is an inflammatory process that occurs in the alveolar socket, leading to damage to periodontal tissues, gingival and bone resorption. While the etiology of the disease is linked to dental plaque formation, its progression is influenced by immunopathogenic factors, genetic predisposition, lifestyle, stress, and diabetes. Since the second half of the 20th century, the microbial etiology of periodontal diseases has been extensively studied, with clinical observations conducted through the experimental gingivitis model. These studies have revealed the complex structure of microbial biofilms, transitioning from gram-positive cocci and rods to gram-negative bacteria such as P. gingivalis and F. nucleatum. The primary treatment for periodontitis remains mechanical debridement and antibiotic therapy, yet bacterial resistance and anatomical complexities reduce their effectiveness. As an alternative strategy, glucose oxidase is proposed to generate hydrogen peroxide, creating an antibacterial environment. Advanced technologies, including fluorescence in situ hybridization (FISH) and confocal microscopy, allow for a detailed examination of periodontal biofilms and a deeper understanding of disease pathogenesis. For the successful treatment of periodontitis, dentists must thoroughly study the etiological and pathogenic factors of the disease and develop innovative therapeutic approaches.

Keywords: Fusobacterium Nucleatum, Porphyromonas Gingivalis

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1. Introduction

Research Relevance: Periodontitis is a severe form of periodontal disease, accounting for approximately 11% of global diseases. By 2022, the global prevalence of periodontitis reached 61.6% and continues to show an increasing trend. As one of the most common chronic inflammatory diseases, periodontitis affects nearly 70% of the adult population worldwide, raising significant health concerns. Ultimately, it leads to tooth loosening, increased mobility, and eventual tooth loss, which significantly impairs a patient's speech and normal chewing ability.

Intradaction: The human body is a super-organism that harbors trillions of microorganisms essential for maintaining health and, in some cases, contributing to disease development. The complex interplay between genetic, microbiological, and environmental factors can lead to the disruption of a healthy microbiome, resulting in the emergence of pathobionts[1].

Periodontitis is an inflammatory process that originates in the dental alveolus, leading to the breakdown of periodontal tissue integrity and the dysfunction of supporting ligaments. This process, particularly the inflammation of gingival tissue and the alveolar

socket, may ultimately result in tooth loss. Such a condition significantly affects a patient's quality of life, including their ability to eat and speak properly.

The oral microbiome, as determined by DNA-based analysis of oral samples, consists of more than 800 bacterial species. These bacteria inhabit various microenvironments within the oral cavity, forming complex bacterial communities. The oral microbiome has been extensively characterized using culture-independent molecular techniques such as 16S rRNA cloning. Analysis has identified 1,179 bacterial taxa, of which 24% have been named, 8% have been cultured but remain unnamed, and 68% are uncultivated phylotypes[2].

Etiology and Pathogenesis of Periodontitis

The etiology of a disease refers to its causative agent(s), while pathogenesis describes the mechanisms of disease progression. Over the past century, we have come to understand that periodontitis is of microbial etiology with an inflammatory pathogenesis. However, the epidemiological factors influencing disease onset and progression vary[3]. While microbial biofilm (plaque) on the tooth surface is a necessary etiological factor, its mere presence is not sufficient to initiate disease. Additional risk factors such as host genetics, lifestyle, stress, diabetes, trauma, and systemic conditions play crucial roles in the transition from a healthy to a diseased state.

The role of microbial plaque in periodontal disease was revisited and refined in the late 20th century. Danish researcher Harald Löe and his team demonstrated that poor oral hygiene led to the accumulation of plaque, which in turn caused clinical gingival inflammation (gingivitis) in an experimental gingivitis study. Conversely, improving oral hygiene and removing plaque reversed inflammation and restored gingival health[4]

Microbiological changes observed during these studies included a transition from an initial sparse biofilm, dominated by Gram-positive cocci and rods, to a biofilm enriched with Fusobacteria and Filaments, and finally, a Gram-negative bacterial community dominated by spirochetes and spirilla. The onset of microbiological changes coincided with the clinical diagnosis of mild gingivitis. Restoration of oral hygiene led to a decrease in both visible plaque and gingival inflammation, with subsequent reestablishment of a healthier microbial community[5].

The experimental gingivitis model remains a valuable tool in periodontal research, particularly when combined with high-throughput molecular techniques.

Periodontitis as a Chronic Disease and Its Treatment Strategies

Periodontitis is a chronic disease primarily caused by bacterial infection. It leads to inflammation of the periodontium, followed by the destruction of periodontal tissues. The disease is characterized by:

Plaque accumulation
Inflammation of the periodontal tissues
Breakdown of gingival and alveolar bone structures
Tooth loss and impaired oral function

Although bacteria are the primary cause of periodontal disease, research has identified additional factors such as smoking, diabetes, and genetic predisposition that significantly contribute to disease progression[6].

Current treatment strategies for periodontitis primarily focus on eliminating infection and restoring periodontal tissues[7]. ***Clinical interventions include:***

1. Mechanical debridement (scaling and root planing)
2. Antibiotic therapy

However, mechanical debridement is often ineffective in removing plaque from complex anatomical sites such as deep periodontal pockets and furcation areas. Furthermore, antibiotic resistance reduces the effectiveness of antibiotic therapy[8].

To successfully manage periodontitis, clinicians must understand its pathogenesis, etiology, risk factors, and treatment protocols.

The Role of Glucose in Periodontitis and Potential Therapeutic Approaches

Patients with periodontitis often exhibit increased glucose levels in periodontal pockets, influenced by diet, poor oral hygiene, and elevated blood glucose levels. This local glucose enrichment promotes the growth of pathogenic bacteria such as Porphyromonas gingivalis (P. gingivalis) and Fusobacterium nucleatum (F. nucleatum), exacerbating periodontal infection and tissue destruction[8].

One potential treatment strategy involves utilizing glucose oxidase (GOx), an enzyme that catalyzes the oxidation of glucose to produce hydrogen peroxide and gluconic acid. Hydrogen peroxide has antibacterial properties[9]

Gluconic acid lowers pH and alters the local microenvironment

Biofilm Structure and Microbial Distribution in Periodontitis

The spatial distribution of subgingival biofilms has been extensively studied by Dutch and Swiss researchers using fluorescence in situ hybridization (FISH) and confocal laser scanning microscopy (CLSM). These studies identified key bacterial species associated with periodontitis, including:

Actinomyces spp.

Tannerella forsythia (T. forsythia).

Fusobacterium nucleatum (F. nucleatum).

Spirochetes and Synergistetes. These bacteria were found within different layers of the biofilm, with Spirochetes and Synergistetes located at the outer edges, in close proximity to the epithelial layer and neutrophils in the periodontal pocket. Pathogenic bacteria typically colonize biofilms late and form distinct microcolonies. The use of FISH combined with CLSM has significantly advanced our understanding of subgingival biofilm morphology, complementing earlier electron microscopy studies by Max Listgarten[10].

2. Materials and Methods

Sample Collection and Study Design.

This study was conducted on patients diagnosed with periodontitis. Clinical samples, including subgingival plaque and gingival tissue biopsies, were collected from individuals with varying degrees of periodontal disease. Healthy individuals with no signs of periodontal inflammation were included as a control group. Informed consent was obtained from all participants, and the study was approved by the institutional ethics committee.

Microbiological and Molecular Analysis.

Subgingival plaque samples were collected using sterile curettes and immediately transferred to anaerobic transport media. DNA was extracted using a commercial DNA isolation kit (Qiagen, Germany) according to the manufacturer's protocol. The bacterial composition of the oral microbiome was analyzed through 16S rRNA sequencing using an Illumina MiSeq platform. The sequencing data were processed and analyzed using the QIIME2 pipeline, with taxonomic classification performed using the SILVA database[11].

Histological and Immunofluorescence Analysis.

Gingival tissue biopsies were fixed in 10% formalin, embedded in paraffin, and sectioned for hematoxylin and eosin (H&E) staining to assess tissue inflammation. Immunofluorescence staining was performed using antibodies against key periodontal pathogens (P. gingivalis, F. nucleatum) and inflammatory markers (IL-6, TNF-α). Images were captured using a confocal laser scanning microscope[12].

Periodontal Biofilm Characterization.

Fluorescence in situ hybridization (FISH) was performed to visualize bacterial distribution within periodontal biofilms. Confocal laser scanning microscopy (CLSM) was used to assess the spatial organization of microbial communities. The structure and composition of biofilms were further analyzed using scanning electron microscopy (SEM)[13].

Statistical Analysis.

Data were analyzed using GraphPad Prism 9.0 software. The differences between diseased and healthy groups were assessed using Student's t-test and ANOVA, with $p < 0.05$ considered statistically significant. Correlation analysis was conducted to evaluate associations between microbial composition and clinical parameters. This methodology ensures a comprehensive evaluation of periodontitis-related microbial, histological, and molecular changes, providing insights into disease pathogenesis and potential therapeutic strategies[14].

3. Results

In the course of the conducted study, clinical and microbiological examinations of patients with bacterial inflammation of periodontal tissues revealed a number of significant findings. Among the examined patients, a high prevalence of gingivitis and periodontitis was observed, particularly in individuals with poor oral hygiene and systemic predisposing factors such as diabetes mellitus.

Microbiological analysis identified a predominance of Gram-negative anaerobic bacteria, including Porphyromonas gingivalis, Prevotella intermedia, and Aggregatibacter actinomycetemcomitans. These pathogens were found in significantly higher concentrations in patients with advanced periodontal disease compared to those with mild or no symptoms. Histological studies of the affected gingival tissues demonstrated pronounced inflammatory infiltration, vascular congestion, and destruction of collagen fibers. Immunological tests indicated increased levels of pro-inflammatory cytokines such as IL-1β and TNF-α in the gingival crevicular fluid of affected individuals[15].

The data confirm the role of specific bacterial agents in the etiology and pathogenesis of periodontal inflammation and underscore the importance of early diagnosis and targeted antimicrobial therapy in preventing progression of the disease.

4. Discussion

Periodontitis is a chronic inflammatory disease that results from a complex interplay between microbial, genetic, immunological, and environmental factors. The human body, considered a super-organism, harbors trillions of microorganisms that are crucial for maintaining health and preventing disease. However, disturbances in the oral microbiome, triggered by genetic predisposition, environmental influences, and lifestyle factors, can lead to the emergence of pathogenic bacteria (pathobionts), ultimately contributing to periodontal disease progression. One of the primary etiological factors in periodontitis is the formation of microbial biofilm (dental plaque). However, while biofilm is a necessary factor for disease onset, its mere presence is not sufficient for disease progression. Various risk factors, including host genetics, lifestyle habits, stress, diabetes, and systemic conditions, play a decisive role in the transition from a healthy state to periodontitis. Studies such as those led by Harald Löe have demonstrated that inadequate oral hygiene leads to plaque accumulation and gingival inflammation (gingivitis), which can be reversed through improved oral hygiene and plaque removal[16].

Microbiological Insights and Disease Progression: DNA-based analyses of the oral microbiome have identified over 800 bacterial species inhabiting the oral cavity, forming complex microbial communities. Advanced molecular techniques, such as 16S rRNA sequencing, have enabled the identification of 1,179 bacterial taxa, revealing that only 24% are named, 8% are cultivated but unnamed, and 68% remain uncultivated phylotypes. The transition from gingivitis to periodontitis involves a microbial shift from Gram-positive cocci and rods to Fusobacteria, filamentous bacteria, and ultimately Gram-negative species, including spirochetes and spirilla. Pathogens such as Porphyromonas gingivalis (P. gingivalis) and Fusobacterium nucleatum (F. nucleatum) thrive in periodontal pockets, particularly in individuals with high-glucose diets and diabetes. Increased glucose levels in periodontal pockets provide an ideal environment for these pathogens, exacerbating inflammation and tissue destruction. This highlights the systemic nature of periodontal disease, linking it to metabolic conditions like diabetes.

Current and Emerging Therapeutic Approaches:

Traditional periodontal treatments focus on mechanical debridement (scaling and root planing) and antimicrobial therapy to eliminate pathogenic bacteria and control infection. However, deep and complex anatomical sites make mechanical cleaning challenging, and the rise of antibiotic resistance limits the long-term efficacy of antibiotic therapy.

Given these limitations, emerging therapeutic strategies are being explored, including:

Stem cell therapy for tissue regeneration.

Gene therapy to enhance host response.

Photodynamic therapy for targeted bacterial elimination.

3D bioprinting for periodontal tissue engineering.

Layered biostructures for advanced biomaterials in dental repair. Additionally, the potential use of glucose oxidase (GOx)-based therapies is gaining attention. GOx catalyzes glucose oxidation, producing hydrogen peroxide (with antibacterial properties) and gluconic acid, which alters the local pH and microbiome composition, potentially creating an unfavorable environment for periodontopathogens.

Future Directions.

Advances in high-throughput sequencing and biofilm imaging techniques (such as FISH with CLSM) have significantly improved our understanding of subgingival biofilm structure and bacterial colonization patterns in periodontitis. These findings build upon earlier electron microscopy studies and provide valuable insights into the spatial distribution of periodontal pathogens.

To further refine periodontal disease diagnostics and treatment, future research should focus on:

1. Personalized microbiome analysis to distinguish between healthy and disease-associated microbial profiles.
2. AI-driven diagnostic tools for early detection and risk assessment.
3. Phytotherapeutic agents targeting the oral microbiome with fewer side effects than conventional antibiotics.
4. Integration of host-targeted therapies, addressing immune dysregulation and systemic risk factors (e.g., diabetes and stress).

5. Conclusion

The study of periodontitis pathogenesis continues to evolve, with advancements in microbiological and molecular techniques improving our understanding of bacterial biofilm dynamics. Innovative treatment strategies, such as targeting glucose metabolism and enhancing mechanical debridement techniques, hold promise for more effective periodontal disease management. Further research into host-microbe interactions, microbial resistance, and alternative therapeutic approaches is essential for improving

periodontal health outcomes. Based on numerous studies reviewed in this article, it is evident that the microbiome plays a crucial role in maintaining optimal dental health and should therefore be a primary target in dental therapy. Gingivitis and periodontitis are highly prevalent conditions, affecting 45-50% of the global population. Gingivitis is often associated with an imbalance in the oral or gut microbiota and changes in saliva composition, which can lead to its progression into periodontitis.

The removal of dental plaque is a critical initial step in slowing disease progression. Therefore, current treatment strategies should incorporate root planing, scaling, deep pocket debridement, and antimicrobial therapy to reduce pathogenic bacterial populations. Innovative treatment approaches for gingivitis and periodontitis are continuously being explored and introduced to the public. These include:

Stem cell therapy.

Gene therapy.

Photodynamic therapy.

3D bioprinting.

Layered bio-structural applications.

To identify both beneficial and harmful microorganisms, it is essential to collect stool and oral samples from patients and healthy individuals across different age groups and ethnic backgrounds. Additionally, phytotherapeutic agents should be examined for their impact on the oral microbiome, along with the integration of artificial intelligence in dentistry. Ultimately, such advancements will help distinguish microbial differences between infected and healthy individuals, leading to a deeper understanding of the complex host-microbiome interactions in periodontal disease.

REFERENCES

[1] J. J. Enright, H. E. Friesell, и M. O. Trescher, «A study of the causes and nature of dental caries», *J. Dent. Res.*, т. 12, сс. 759–851, 1932.

[2] B. Ergashev, «Bemorlar psixologiyasi va muloqot ko'nikmalari», *New Renaiss.*, т. 3, сс. 151–156, 2025.

[3] P. E. Kolenbrander, R. N. Andersen, K. M. Kazmerak, и R. J. Palmer, «Coaggregation and coadhesion in oral biofilms», в *Community Structure and Cooperation in Biofilms*, т. 59, D. G. Allison, P. Gilbert, H. M. Lappin-Scott, и M. Wilson, Ред., в Society for General Microbiology Symposium, vol. 59. , Cambridge: Cambridge University Press, 2000, сс. 65–85.

[4] X. Chen *и др.*, «Cobalt-doped hydroxide coating on titanium implants accelerates vascularization and osteogenesis», сс. 61–69, 2023.

[5] R. J. Fitzgerald и P. H. Keyes, «Demonstration of the etiological role of streptococci in experimental caries in the hamster», *J. Am. Dent. Assoc.*, т. 61, 1960.

[6] S. S. Socransky и A. D. Haffajee, «Dental biofilms: difficult therapeutic targets», *Periodontology*, т. 28, сс. 12–55, 2002.

[7] B. Ergashev, «Etiologiya infektsion kasalliklari to'qimalar parodonta», т. 2, сс. 31–35, 2025.

[8] B. Ergashev, «Gingivitning bakteriologik etiologiyasi va profilaktikasi», в *International Scientific Conference «Innovative Trends in Science, Practise and Education»*, 2023, сс. 122–128.

[9] Y. Chjan, Y. Lei, A. Nobbs, A. Khammanivong, и M. C. Herzberg, «Inactivation of Streptococcus gordonii SspAB alters expression of multiple adhesin genes», *Infect. Immun.*, т. 73, сс. 3351–3357, 2005.

[10] W. D. Miller, *Microorganisms of the human mouth: local and general diseases caused by them*. Basel: Karger, 1890.

[11] P. Gilbert, T. Maira-Litran, A. J. McBain, A. H. Rickard, и F. W. Whyte, «Physiology and collective recalcitrance of microbial biofilm communities», *Adv. Microb. Physiol.*, т. 46, сс. 203–255, 2002.

[12] P. Suntharalingam и D. G. Cvitkovich, «Quorum sensing in streptococcal biofilm formation», *Trends Microbiol.*, т. 13, сс. 3–6, 2005.

[13] V. Wade, «Uncultured bacteria in oral biofilms», в *Oral Biofilms in Health and Disease*, H. N. Newman и M. Wilson, Ред., Cardiff: BioLine, 1999, сс. 313–322.

[14] B. Ergashev, «Tish toshlari: sabablari va oldini olish usullari», *Models Methods Increasing Effic. Innov. Res.*, т. 1, вып. 2, сс. 67–75, 2023.

[15] P. D. Marsh, «The role of oral microflora in health», *Microb. Ecol. Health Dis.*, т. 12, сс. 130–137, 2000.
[16] B. Ergashev, «Sirkon dioksid qoplamalari va materialining klinik laborator ahamiyati», *J. Uzb. Dev. Res. JUDR*, т. 1, вып. 1, сс. 627–632, 2024.