

Exploring innovative approaches and genetic roles in periodontal health care: a narrative review

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Abstract

Periodontitis is a common inflammatory disorder that causes irreversible damage to the periodontium, resulting in tooth mobility and loss, negatively affecting quality of life. It is also linked to systemic diseases like cardiovascular problems, diabetes, and rheumatoid arthritis due to common inflammatory pathways. The disease develops from a complex interplay between microbial, genetic, host, and environmental factors. Bacteria such as *Porphyromonas gingivalis* and *Treponema denticola* disrupt the immune response, promoting tissue destruction. Traditional treatments like scaling and root planing (SRP) and surgical methods reduce microbial burden but fail to address host-microbe interactions completely. New advancements in microbiome research and personalized medicine offer hope for more precise therapies targeting individual risk factors.

This review explores the pathogenesis of periodontitis, current treatment strategies, and the genetic aspects influencing the disease. It highlights the role of microbial imbalance, with harmful bacteria exacerbating inflammation and tissue damage. Non-surgical treatments such as SRP are foundational, while adjunct therapies like antibiotics, probiotics, and regenerative approaches, including bone grafting, offer additional benefits. The review also emphasizes the potential of personalized treatments based on genetic and microbial profiling, offering the possibility of more effective, targeted therapies for improved long-term patient outcomes.

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How to Cite

L. Memè, F. Bambini, P. Nardelli, S. Chieppa, F. Sabatelli, I.R. Bordea, G.V.O. Fernandes, E. Qorri, L. Almasri, M. Alkassab, M. Contaldo, G. Ferraro, A. Palermo. Exploring innovative approaches and genetic roles in periodontal health care: a narrative review. Oral and Implantology Vol. 16 No. 3 (S1) (2024), 378-393. [https://doi.org/10.11138/oi.v16i3\(S1\).86](https://doi.org/10.11138/oi.v16i3(S1).86)

Keywords: Periodontitis, Periodontal Disease, Therapeutic Strategies, Genetic Factors, Regenerative Therapy

Introduction

Periodontitis is a prevalent inflammatory condition that irreversibly damages the periodontium, leading to tooth mobility, loss, and reduced quality of life (1–7). Beyond oral health, it is linked to systemic conditions like cardiovascular disease, diabetes, and rheumatoid arthritis via shared inflammatory pathways (8–14). The disease arises from a complex microbial, host, genetic, and environmental interaction (15–21). Pathogens such as *Porphyromonas gingivalis* and *Treponema denticola* disrupt immune defenses, driving tissue destruction and bone resorption (22–30). While treatments like scaling and surgical interventions reduce microbial load, addressing host-microbial interactions remains challenging (31–37). Advances in personalized medicine and microbiome research offer promise for tailored therapies targeting individual risk factors (38–44).

Methodology

This narrative review was conducted to offer an in-depth analysis of periodontal disease, its treatment options, and the genetic factors involved. The methodology is summarized below:

Literature Search

A comprehensive search was conducted in the following academic databases:

- PubMed
- Scopus

- Web of Science
- Google Scholar

Search Terms

The search focused on terms related to periodontal disease, treatments, and genetic aspects. Key phrases included:

- Periodontitis and oral microbiota
- Periodontal therapy and adjunct treatments
- Scaling and root planing, antibiotics
- Genetic factors and periodontitis
- Probiotics, postbiotics, and periodontal health
- Regenerative therapies and periodontal outcomes

The search was confined to studies published in English from January 2004 to May 2024, prioritizing clinical studies involving humans.

Inclusion Criteria

- Studies on periodontal disease, with a particular emphasis on periodontitis.
- Research on therapeutic interventions, including scaling and root planing, antimicrobial treatments, probiotics, and regenerative methods.
- Articles investigating genetic factors and the oral microbiota in periodontitis.
- Clinical trials, observational studies, and systematic reviews that provide relevant data.

Exclusion Criteria

- Studies not published in English or before 2004.
- Animal or laboratory studies lacking clinical relevance.
- Editorials, commentaries, and case reports without substantial data.

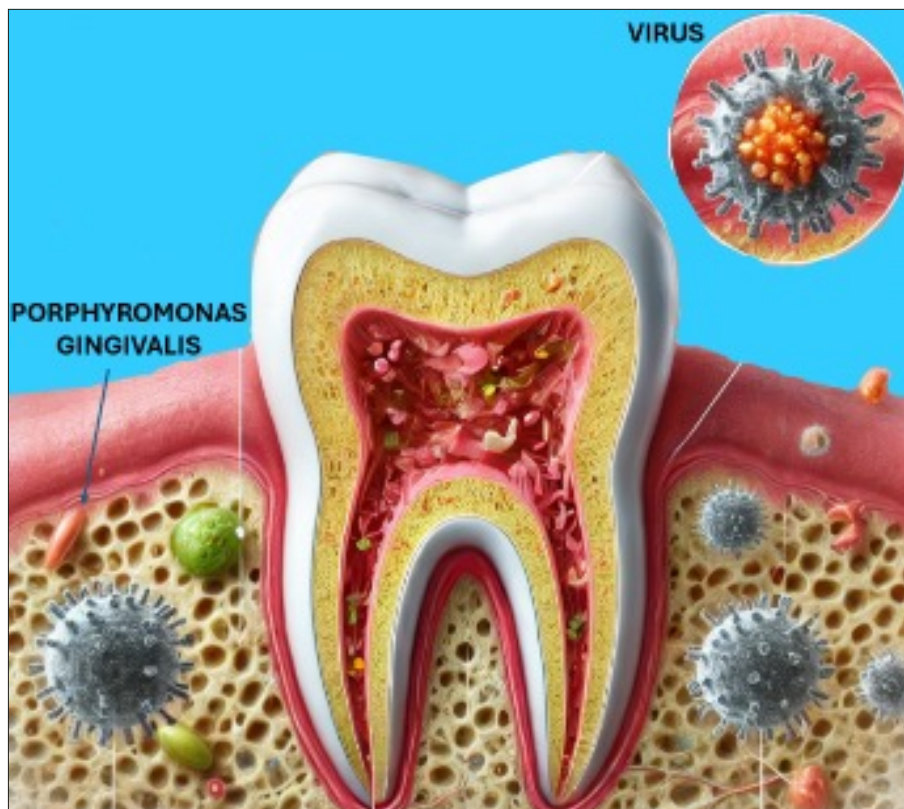


Figure 1. Illustration of bacteria and viruses affecting the tooth.

Data Extraction and Categorization

The information from the selected studies was organized into key themes:

1. The pathogenesis of periodontitis (microbial, immune, and genetic influences).
 2. Non-surgical and surgical treatment approaches.
 3. Adjunctive treatments (such as antibiotics, probiotics, postbiotics).
 4. Regenerative techniques (including enamel matrix derivatives, bone grafting, and growth factors).
 5. Personalized treatment approaches integrating genetic data and microbiome analysis.
- 3) Pathogenesis of Periodontal Disease

Microbial Dysbiosis and Oral Microbiota in Periodontitis

The oral cavity is home to one of the most diverse and dynamic microbial ecosystems in the human body, with over 700 bacterial species identified (45–53). In a healthy state, this ecosystem is dominated by commensal and symbiotic microorganisms that maintain tissue homeostasis, modulate the immune response, and prevent colonization by opportunistic pathogens (54–60). However, when this balance is disrupted—whether by poor oral hygiene, smoking, systemic conditions, or other factors—a state of dysbiosis arises, leading to disease progression (61–67).

Microbial dysbiosis in periodontitis is marked by an overrepresentation of pathogenic species such as:

- *Porphyromonas gingivalis*: This keystone pathogen manipulates the immune response by producing proteases (gingipains) that degrade complement proteins and cytokines, dampening the host's innate defenses.
- *Tannerella forsythia*: Known for its proteolytic enzymes, this organism contributes to connective tissue degradation and facilitates microbial invasion.
- *Treponema denticola*: This motile spirochete enhances biofilm maturation and produces toxins that disrupt epithelial barrier integrity.

These bacteria are often called the “red complex” due to their strong association with disease severity (68–74). The shift in microbial composition also involves the enrichment of other taxa, including *Filifactor* blocks, *Fusobacterium nucleatum*, and *Prevotella intermedia* (75–81). These organisms interact synergistically to amplify the inflammatory response and drive tissue destruction (82–88).

Functional Changes in the Microbiota

Beyond taxonomic shifts, microbial dysbiosis is characterized by functional changes in the microbiome (89–93). Pathogenic communities exhibit increased metabolic activity, producing virulence factors, proteases, and metabolites (e.g., butyrate) that enhance inflammation and tissue destruction (94–98). These changes create a self-perpetuating cycle of inflammation and dysbiosis, underscoring the need for therapeutic interventions targeting microbial composition and function (99–105).

The host's immune system is crucial in the development of periodontitis (106–110). Its primary function is to eliminate pathogens, but prolonged immune response activation can result in tissue damage (111–115). The innate immune system detects bacterial components,

such as lipopolysaccharides (LPS), through toll-like receptors (TLRs), triggering the release of cytokines like IL-1 β and TNF- α (116–122). These cytokines recruit immune cells such as neutrophils and macrophages, which produce reactive oxygen species (ROS) and matrix metalloproteinases (MMPs), leading to tissue breakdown. Additionally, RANKL plays a key role in promoting bone resorption (123–127). The adaptive immune response is also involved, where Th17 cells contribute to inflammation by producing IL-17, while T regulatory cells (Tregs) release IL-10 to counteract this inflammation (128–132). An imbalance between these two cell types contributes to the chronic inflammation seen in periodontitis (133–137).

Therapeutic Approaches for Periodontal Disease Management

Non-Surgical Periodontal Therapy

Non-surgical therapy is the foundation for managing periodontal disease and is typically the first line of treatment for controlling infection and inflammation (138–142). The principal method in this category is scaling and root planing (SRP), a mechanical intervention that removes supra- and subgingival plaque, calculus, and bacterial biofilm to reduce the microbial burden and disrupt the pathogenic environment (143–149).

Scaling and Root Planing (SRP):

SRP involves physically removing debris and biofilm from tooth surfaces and periodontal pockets (150–156). Numerous clinical trials have demonstrated significant improvements in key periodontal parameters, including reduced probing pocket depth (PPD), improved clinical attachment level (CAL), and decreased bleeding on probing (BOP) following SRP (157–163). Additionally, SRP reduces systemic inflammatory markers, such as C-reactive protein (CRP), underscoring its benefits beyond the oral cavity (164–170). However, SRP has limitations, particularly in treating advanced periodontitis with deep periodontal pockets or furcation defects, where removing pathogenic microorganisms is challenging (171–175).

Adjunctive Antimicrobial Therapy

Antimicrobial therapy is often used as an adjunct to SRP to enhance treatment efficacy by targeting residual pathogens within periodontal pockets (176–182). Depending on the severity of the condition and the treatment objectives (183–189), these agents can be administered systemically or locally.

Systemic antibiotics are frequently prescribed in conjunction with SRP to target periodontal pathogens. Common antibiotics used include:

- Amoxicillin combined with metronidazole has shown enhanced efficacy against anaerobic bacteria such as *P. gingivalis* and *T. forsythia*.
- Tetracyclines: These agents exhibit bacteriostatic effects and inhibit matrix metalloproteinase (MMP) activity, reducing connective tissue destruction.

While antibiotics are effective in improving clinical outcomes, they are associated with the risk of systemic side effects, disruption of the gut microbiota, and the emergence of antimicrobial resistance (190–196).

Consequently, their use must be judicious and limited to specific indications, such as aggressive periodontitis or refractory cases (197–203).

Locally Delivered Antimicrobials (LDAs):

LDAs offer a targeted approach to delivering antimicrobial agents directly into periodontal pockets. Examples of LDAs include:

- Chlorhexidine chips: Biodegradable chips that release chlorhexidine over time.
- Minocycline microspheres: Sustained-release formulations that reduce pathogenic bacterial loads.
- Doxycycline gels: These provide sustained concentrations of the antibiotic within pockets.

Studies have demonstrated that LDAs when used alongside SRP, improve clinical outcomes by reducing pocket depths and controlling inflammation (204–210). However, their efficacy depends on patient compliance and proper pocket debridement before placement (211–217).

Adjunctive Probiotic and Postbiotic Therapies

Probiotics and postbiotics have gained attention in periodontal therapy because they restore microbial balance, boost the immune system, and reduce inflammation (218–224). Probiotics like *Lactobacillus reuteri* and *Bifidobacterium bifidum* improve periodontal health by decreasing inflammation and bleeding on probing (225–231). They also reduce oxidative stress and inflammation (232–238). Postbiotics, such as SCFAs and bioactive peptides, enhance barriers and reduce inflammation, making them safe for immunocompromised patients and complementing treatments like scaling and root planing (SRP) (239–245).

Surgical Periodontal Therapy

When non-surgical treatments fail to produce adequate results, surgical interventions become necessary to address advanced periodontal damage. These procedures focus on removing periodontal pockets, regenerating lost tissues, and creating a more favorable environment for long-term periodontal health. Flap surgery involves lifting the gingival tissues to allow better access for cleaning and reducing pockets (246–252). This method is beneficial for deep pockets and provides improved visibility of the tooth roots and bone structures. Guided Tissue Regeneration (GTR) is another regenerative technique that uses barrier membranes to guide tissue growth while preventing the migration of epithelial cells (253–258). These membranes, like expanded polytetrafluoroethylene, can be resorbable, such as collagen, or non-resorbable. Clinical studies have shown that GTR can improve clinical attachment levels and help regenerate periodontal tissues in bone defects. Bone grafting, often combined with growth factors, involves placing bone substitutes into defects to stimulate tissue regrowth. Various graft types are used, including autografts, allografts, xenografts, and synthetic materials, each contributing to enhanced healing and regeneration.

Regenerative Techniques in Periodontal Therapy

Regenerative strategies restore lost periodontal structures by promoting new bone, cementum, and

ligament growth. Enamel matrix derivatives (EMD) from porcine tooth buds enhance fibroblast proliferation and clinical attachment gain (259–265). Growth factors like PDGF and BMPs stimulate cell proliferation and matrix synthesis, often combined with scaffolds (266–272). Tissue engineering integrates scaffolds, stem cells, and bioactive factors with innovations like 3D-printed scaffolds and mesenchymal stem cell (MSC) therapy (273–279).

Microbiological Changes Following Periodontal Therapy

Therapies like scaling and root planing (SRP) reduce pathogenic bacteria and promote health-associated species (280–284). Surgical interventions enhance microbial diversity, while probiotics and postbiotics further support beneficial microbiota shifts and suppress pathogens (285–291).

Long-Term Maintenance and Disease Prevention

Periodontal maintenance is critical for sustaining the benefits of therapy and preventing disease recurrence. Key components of maintenance therapy include:

1. Regular Professional Cleanings: To remove plaque and calculus that may accumulate over time.
2. Patient Education: Emphasizing oral hygiene practices, including proper brushing and interdental cleaning.
3. Microbial Monitoring: Periodic assessment of the subgingival microbiota using advanced diagnostic tools.
- 5) Personalized Periodontal Therapy: The Future of Treatment

Advances in genomics, proteomics, and microbiome research have paved the way for personalized periodontal therapies tailored to individual risk profiles (292–298). By integrating genetic and microbial insights into clinical decision-making, personalized approaches offer the potential for more targeted and effective treatments (299–305).

Genetic testing can identify polymorphisms associated with inflammatory cytokines, immune signaling pathways, and bone metabolism (306–312). This information can guide the selection of adjunctive therapies, such as anti-inflammatory agents or regenerative materials.

High-throughput sequencing techniques enable detailed characterization of the subgingival microbiota (313–317). Therapies can be tailored to address specific microbial imbalances, such as using bacteriophages or synthetic probiotics targeting pathogenic species (318–324).

Future Directions in Periodontal Therapy

Emerging therapeutic modalities and research avenues hold promise for transforming periodontal care. These include:

1. Microbiome-Based Therapies: Developing bacteriophage therapy, prebiotics, and synthetic probiotics to selectively target pathogenic bacteria.
2. Gene Therapy: Advances in CRISPR-Cas9 technology can potentially edit genes associated with periodontitis susceptibility.
3. Biomarker Discovery: Identifying biomarkers for early diagnosis and monitoring of treatment outcomes.

Conclusion

Periodontal disease is a complex and multifactorial condition that requires an interdisciplinary approach for effective management. While traditional therapies remain the cornerstone of treatment, integrating adjunctive modalities, regenerative techniques, and personalized approaches can revolutionize periodontal care. Advances in microbiome research and genetic profiling will play a pivotal role in shaping the future of periodontal therapy, enabling tailored interventions that address the underlying drivers of disease and improve patient outcomes.

Abbreviations

1. BOP - Bleeding on Probing
2. CRP - C-Reactive Protein
3. EMD - Enamel Matrix Derivatives
4. GTR - Guided Tissue Regeneration
5. IL - Interleukin
6. LPS - Lipopolysaccharide
7. MMPs - Matrix Metalloproteinases
8. PRP - Platelet-Rich Plasma
9. RCT - Randomized Controlled Trials
10. ROS - Reactive Oxygen Species
11. SRP - Scaling and Root Planing
12. TGF- β - Transforming Growth Factor Beta
13. TLR - Toll-Like Receptors
14. TNF- α - Tumor Necrosis Factor Alpha
15. Tregs - Regulatory T Cells
16. Th17 - T Helper 17 Cells

Funding Statement

This research received no external funding.

Data Availability Statement

Not applicable.

Conflicts of Interest

The authors declare no conflict of interest.

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