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Microbial Dysbiosis in Periodontitis: A Paradigm Shift in Understanding Oral Health

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Introduction

Periodontitis is an inflammatory condition caused by complex interactions between the host immune response and the microbial community within the oral cavity. Traditionally, a "specific plaque hypothesis" dominated the understanding of periodontal disease, attributing its pathogenesis to a few key pathogens, such as Porphyromonas gingivitis, Tannerella forsythia, and Treponema denticola. However, the emergence of the "polymicrobial synergy and dysbiosis" (PSD) model has shifted the focus to the entire microbial community and its disrupted balance (dysbiosis) [1, 2].

Dysbiosis results from an imbalance between commensal (healthy) and pathogenic microbes, often triggered by environmental changes such as poor oral hygiene, smoking, or systemic diseases. This imbalance alters the microbial ecosystem, leading to the establishment of a pathogenic biofilm and subsequent periodontal destruction.

The Oral Microbiome: A Complex Ecosystem

The oral cavity is home to over 700 bacterial species, along with fungi, viruses, and archaea, that coexist in a dynamic equilibrium. These microbes form a biofilm on oral surfaces, maintaining homeostasis and preventing colonization by pathogens.

In health, the oral microbiome is dominated by commensals such as Streptococcus, Actinomyces, and Veillonella. These bacteria contribute to metabolic activities, immune modulation, and ecological stability. However, environmental shifts-such as an increase in gingival inflammation, changes in pH, or nutrient availability—can favour the growth of pathogenic bacteria, disrupting the microbial equilibrium [3].

Microbial Dysbiosis and Periodontitis

The transition from health to periodontitis involves three key stages of microbial dysbiosis:

Initiation Phase

Dysbiosis begins with environmental changes, such as increased gingival inflammation, which alter the biofilm composition. Early colonizers such as Streptococcus species decrease, while facultative anaerobes such as Prevotella intermedia proliferate.

Transition to Pathogenic Dysbiosis

Keystone pathogens like P. gingivitis disrupt the host immune response by evading phagocytosis, impairing neutrophil function, and releasing virulence factors like gingipains. These pathogens synergize with other bacteria to amplify inflammation and tissue damage [4].

Advanced Dysbiosis

Advanced dysbiosis is characterized by the dominance of pathogenic species, including T. denticola, Fusobacterium nucleatum, and Aggregatibacter actinomycetemcomitans. The biofilm becomes more resilient, and chronic inflammation leads to irreversible destruction of periodontal tissues.

Mechanisms of Dysbiosis in Periodontitis

Immune Dysregulation

Dysbiosis disrupts immune homeostasis by inducing a hyper-inflammatory response. Pathogenic bacteria release lipopolysaccharides (LPS), gingipains, and other toxins, activating pro-inflammatory cytokines like interleukin-1β (IL-1β) and tumour necrosis factor-α (TNF-α).

Nutritional Competition

Dysbiotic bacteria exploit host-derived nutrients, such as home and peptides, favouring their survival and outcompeting commensals.

Biofilm Resilience

Pathogenic biofilms are more resistant to antimicrobial agents and immune clearance. The synergistic interaction between bacteria enhances their virulence and resilience [5, 6].

Implications for Periodontal Therapy

Understanding microbial dysbiosis in periodontitis opens new avenues for therapeutic interventions:

Probiotics and Prebiotics

Probiotics like Lactobacillus reuteri and prebiotics that promote commensal growth can help restore microbial balance and reduce inflammation.

Host-Modulation Therapies

Targeting the host immune response with agents like anti-inflammatory drugs, cytokine inhibitors, or resolving can mitigate the effects of dysbiosis.

Antimicrobial Strategies

Narrow-spectrum antimicrobials and bacteriophages targeting

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keystone pathogens offer precise methods for managing dysbiosis without disrupting the commensal microbiota.

Microbiome Transplantation

Emerging research explores microbiome transplantation from healthy individuals to restore microbial homeostasis in periodontitis patients [7-10].

Discussion

Microbial dysbiosis in periodontitis represents a pivotal shift in the understanding of the disease, highlighting the complexity of the oral microbiome and its interplay with host factors. This discussion explores the broader implications of dysbiosis, the challenges it presents in clinical practice, and the future directions for research and therapy.

Revisiting the Aetiology of Periodontitis

Traditional theories, such as the "specific plaque hypothesis," focused on individual pathogens as the primary cause of periodontal destruction. However, the polymicrobial synergy and dysbiosis model emphasizes the importance of microbial interactions and ecological changes rather than the presence of specific bacteria alone. This holistic approach challenges diagnostic practices and necessitates a shift toward identifying dysbiotic patterns instead of detecting isolated pathogens.

Conclusion

Microbial dysbiosis represents a significant paradigm shift in the understanding of periodontitis. By moving beyond a pathogen-centric view to an ecosystem-based approach, we can better address the complexities of this disease. Future therapeutic strategies targeting dysbiosis hold the promise of not only managing periodontal disease but also improving overall oral and systemic health.

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