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From Symbiosis to Dysbiosis: Microbial Dynamics in Periodontal Disease Progression

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KEYWORDS

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ABSTRACT:

Periodontitis is a chronic multifactorial inflammatory disease characterized by progressive destruction of the tooth-supporting structures and is closely associated with dysbiotic alterations in the oral microbiome. The transition from microbial symbiosis to dysbiosis plays a critical role in disease initiation and progression, shifting the oral ecosystem from a health-associated community dominated by Gram-positive facultative bacteria to a pathogenic community enriched with Gram-negative anaerobes such as Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola. Contemporary concepts, including the ecological plaque hypothesis, microbial shift hypothesis, and polymicrobial synergy and dysbiosis model, emphasize that periodontitis arises from complex interactions within a polymicrobial biofilm rather than the action of individual pathogens. These dysbiotic microbial communities exhibit enhanced virulence, metabolic cooperation, and the ability to evade and modulate host immune responses, resulting in persistent inflammation, connective tissue breakdown, and alveolar bone loss. Furthermore, host immune dysregulation, genetic susceptibility, and environmental factors contribute to the establishment and maintenance of this pathogenic state. Understanding periodontitis through the lens of dysbiosis provides a comprehensive framework integrating microbial and host factors, highlighting the need for advanced therapeutic strategies that extend beyond conventional mechanical debridement to include microbiome modulation and host-targeted approaches for improved clinical outcomes.

INTRODUCTION

"**Our lives are intertwined with trillions of unseen microorganisms that help shape our health and survival**". Periodontitis is a chronic multifactorial inflammatory disease associated with dysbiotic plaque biofilm and characterized by progressive destruction of tooth supporting apparatus[1] . Bacteria are by far the predominant group of organisms in the oral cavity.

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Microbial communities constitute complex and dynamic systems that exhibit resilience and maintain a fundamental state of ecological harmony. A complex polymicrobial associations found between the bacteria known as Biofilm . Bacteria form microcolonies on tooth surface and secrete a sticky extracellular polymeric substance that helps the bacteria adhere to the surface, and to each other. Once the extracellular polymeric substance is secreted, the biofilm undergoes maturation, expanding in size and developing a characteristic structure.

Typically, this architecture comprises distinct zones of rapidly and slowly proliferating cells, water channels that facilitate the flow of metabolites, and nutrient gradients. This intricate structural arrangement results in functional diversity within the biofilm, enabling it to adapt metabolically and phenotypically to various conditions. As a result, the biofilm gains several new traits and advantages that enhance its resilience and survival.

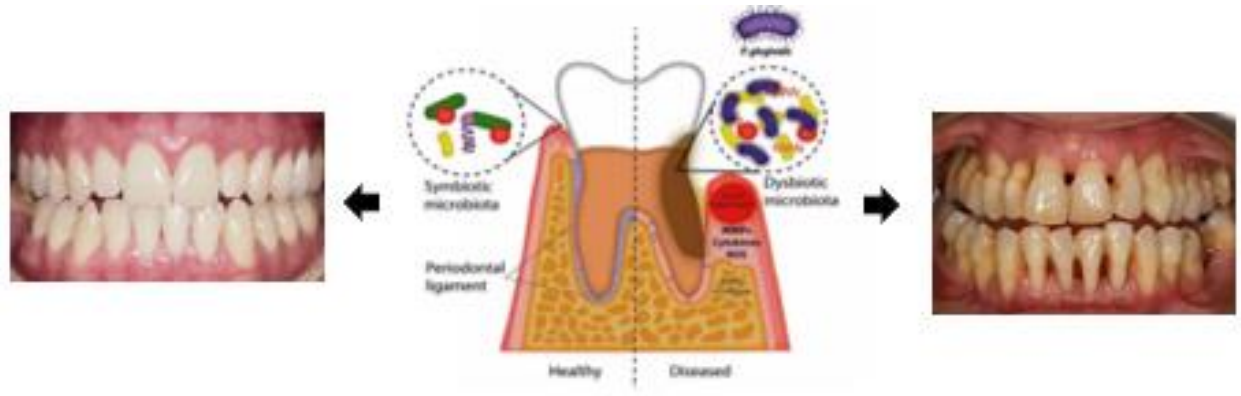
Dysbiosis refers to change in the normal composition and function of the microbiota, resulting in a state that promotes disease rather than health. In the case of periodontitis, dysbiosis disrupts the homeostatic relationship between the host and its oral microbiota, initiating and perpetuating chronic inflammation and tissue destruction[2]

THE ORAL MICROBIOME IN HEALTH

The oral microbiome is a complex and dynamic assemblage of microorganisms that normally exist within the oral cavity contributing to host defense mechanisms. Oral dysbiosis refers to an imbalance or disruption in microbial community, which arise from various factors such as illness, inadequate oral hygiene, certain medications, or dietary habits.[3] Dysbiosis of the oral microbiome can contribute to the development of numerous oral and systemic diseases, including dental caries, periodontitis, cardiovascular disease and diabetes. It is associated with an increase in pathogenic bacteria and a corresponding decrease in beneficial bacteria

MICROBIAL SHIFT AND DISEASE-ASSOCIATED BIOFILMS

Recent advances in microbiome research have shifted the focus from individual pathogens to the role of entire microbial communities in both health and disease. Concept of "**one germ, one disease**" may need modification because with a particular disease more than one bacterial species may be associated. However, this model has proven insufficient for explaining complex polymicrobial conditions such as periodontitis.[4] Contemporary studies suggest that disease may result not only from the presence of pathogenic organisms but also from the **absence or depletion of beneficial microbial species** that help maintain microbial balance and host immune homeostasis. This understanding has given rise to the **"Microbial Shift Hypothesis,[5]"** which proposes that certain diseases arise from a disruption or shift in the composition of the local microbiota—favoring a dysbiotic community that promotes inflammation and tissue damage.



Microbial shift, commonly referred to as **dysbiosis**, describes a transition within the microbial community where beneficial symbiotic organisms decline and pathogenic species proliferate. In the context of periodontitis, this shift represents a well-established paradigm in which the oral microbiota evolves from a community dominated by **Gram-positive, facultative aerobes**—typically associated with periodontal health—to one dominated by **Gram-negative, obligate anaerobes**, which are strongly linked to inflammation and tissue destruction. According to recent studies, oral dysbiosis is essential to the development and course of

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periodontitis[6]. The microbial ecology experiences a gradual shift in the balance of the microbiome, which disrupts the delicate symbiotic relationship between the host and its resident microenvironment. The microbial community changes in a more pathogenic composition, as this balance worsens, which eventually leads to periodontal tissue damage and persistent inflammation.

Concept / Term	Researches	Key Points
Dysbiosis and Dysbiotic microbial Communities	General Terminology	Microbial imbalance leading to harmful communities driving disease
Ecological plaque hypothesis **Or Polymicrobial synergy and dysbiosis concept**	George Hajishengallis & Richard Lamont	• Different bacterial species play distinct ,cooperative roles . • Together they form a disease – causing , stable microbiota that trigger chronic inflammation
Keystone pathogen	Hajishengallis & Lamont[7]	Highlights P. gingivalis as a key orchestrator that shift microbiota

MICROBIAL CHARACTERISTICS OF PERIODONTAL DYSBIOSIS

The dysbiotic microbiome in periodontitis is typically enriched with a cluster of anaerobic, gram-negative bacteria, including *P. gingivalis*, *Tannerella forsythia*, *Treponema denticola*, *Fusobacterium nucleatum*, and *Prevotella intermedia*. These organisms exhibit virulence traits such as the ability to invade host tissues, evade immune detection, and produce toxins and enzymes that degrade extracellular matrix components.

Several features characterize the dysbiotic biofilm in periodontitis:

1. **Increased Diversity and Load**: Contrary to earlier beliefs, disease-associated biofilms are often more diverse than those found in health, with higher total bacterial loads.
2. **Metabolic Synergy**: Pathogenic communities exhibit metabolic cooperation, such as cross-feeding, which enhances survival and pathogenicity.
3. **Host Modulation**: Keystone pathogens like *P. gingivalis* can interfere with innate immune signaling pathways, subverting host defenses and promoting a non-resolving inflammatory response.

This dysbiotic state does not occur in isolation but is facilitated and sustained by the host's own inflammatory response, creating a self-reinforcing cycle of microbial dysregulation and tissue destruction.

PERIODONTITIS AS THE RESULT OF POLYMICROBIAL ACTIVITY

In the **late 19th century**, bacteria were regarded as the primary causative agents of **periodontal disease**. However, during the **early to mid-20th century**, the prevailing view shifted, emphasizing the role of **occlusal trauma and parafunctional forces** as the principal etiological factors contributing to the initiation and progression of periodontal disease. In the late 1960's that the role of bacteria as an etiologic factor in periodontal disease was actually accepted During this period, **Loesche (1976)**[8] introduced the **Non-Specific Plaque Hypothesis**, which proposed that **periodontal disease** results primarily from an overall **increase in the quantity of dental plaque**, rather than the presence of specific pathogens. According to this hypothesis, all plaque bacteria were considered to have a similar potential to induce disease, with the **cumulative bacterial load** being the critical factor in the onset of periodontal inflammation.

This theory was challenged by **Socransky's** specific-plaque hypothesis (1976)[9] Socransky's postulates included that (i) The bacterium must be found more frequently and in higher numbers in disease sites (ii) Can

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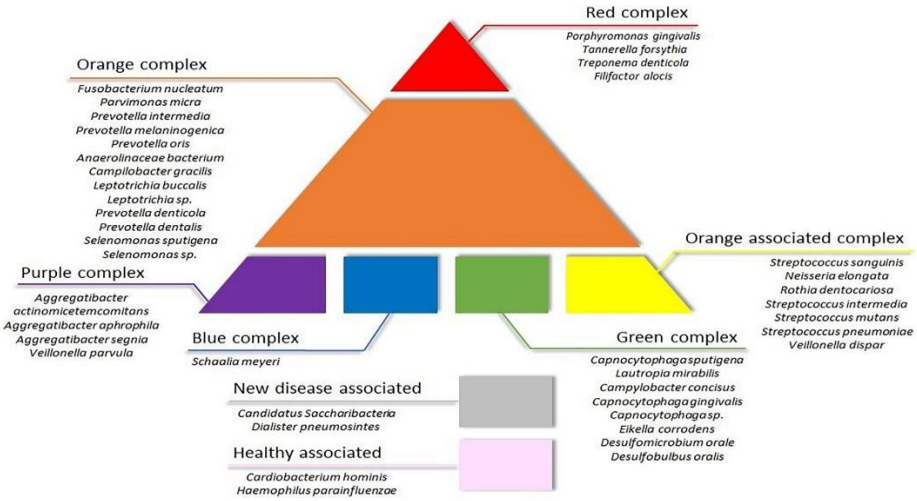
be isolated from diseased sites; (iii) Upon elimination of the bacterium, the tissue returns to health ; (iv) The host mount a response against this specific pathogen; (v) The pathogen expresses virulence factor; and (vi) Animal studies should prove its pathogenicity and potential for disease. in which he stressed that certain species are more involved in periodontal destruction . Sigmund Socransky classified subgingival bacteria in six color-coded distinctive microbial complexes, based on their association with periodontal health or periodontal disease. The most well-known bacterial group associated with periodontal disease is the “red complex,” which includes the Gram-negative anaerobic bacteria *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola*. These microorganisms are typically present in higher numbers and concentrations in periodontal pockets associated with active disease.



Updated scheme of oral microbial complexes. Color coding of the bacterial groups was based on their association with microbial complexes involved in oral pathogenesis, indicating their potential contribution to oral health (Socransky et al., 1998; Haffajee et al., 2008; Colombo and Tanner, 2019)[10]

Hajishengallis and Lamont proposed a model in which periodontitis arises from a dysbiotic and cooperative microbial community, rather than being driven by a single pathogen or a limited group like the "red complex." They suggested that a variety of bacterial species—or specific gene combinations within the community—play complementary roles that collectively establish a stable, disease-inducing microbiota. Within this biofilm, the microbes act synergistically to evade host defenses and promote inflammation, leading to heightened proteolytic activity and cytokine production, ultimately resulting in tissue damage.[11]

PERIODONTITIS AS A CHRONIC DYSBIOTIC DISEASE

The term dysbiosis was first mentioned in a 1959 paper addressing gastrointestinal disturbance in infants. Interestingly, recent longitudinal studies using both metagenomic and metatranscriptomic approaches to compare healthy and diseased states—or sites with progressing versus stable conditions-have found that many different types of bacteria increase in people with periodontitis compared to those with periodontal health. These results reinforce the idea of synergistic virulence among metabolically active commensal bacteria, rather than the expansion of a few specific pathobionts, which typically characterizes most dysbiotic conditions. Microbial shifts occur when inflammation in certain individuals changes the microbiome composition by modifying the conditions that affect microbial growth. A positive feedback loop amplifies the inflammatory response exacerbating the inflammatory response and additional changes in the microbiome composition. This cascade results in the release of powerful host proteases and ultimately leads to the destruction of tooth connective tissue attachment and the resorption of alveolar bone.

HOST RESPONSE AND IMMUNE DYSREGULATION

The pathogenesis of periodontitis is not solely microbial but results from a complex interplay between dysbiotic biofilms and the host immune system. The host's

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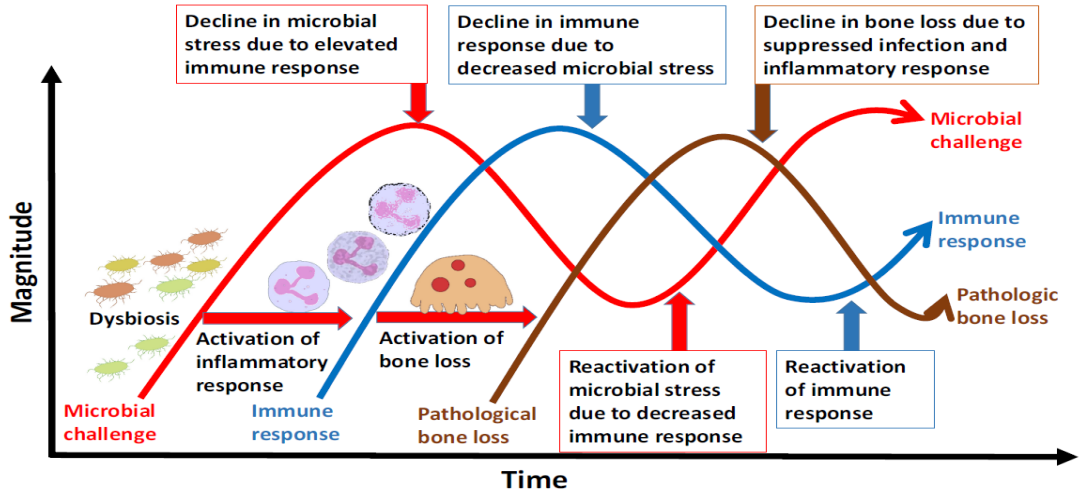
response to microbial challenge is critical in determining whether the inflammatory process will be resolved or progress to chronic tissue damage.

In health, the innate immune system efficiently detects and eliminates pathogens through mechanisms such as phagocytosis, antimicrobial peptides, and complement activation. However, in periodontitis, the presence of dysbiotic microbiota leads to an aberrant immune response characterized by:

- **Persistent Inflammation**: Elevated levels of pro-inflammatory cytokines such as interleukin-1β (IL-1β), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6) contribute to chronic inflammation.
- **Neutrophil Dysregulation**: Although neutrophils are essential for microbial clearance, their hyperactivation in periodontitis leads to the release of proteolytic enzymes and reactive oxygen species that damage host tissues.
- **Bone Resorption**: Cytokines such as receptor activator of nuclear factor kappa-B ligand (RANKL) promote osteoclastogenesis, resulting in alveolar bone loss.

Importantly, the host immune response does not effectively eliminate the dysbiotic biofilm but instead exacerbates tissue damage, fueling the progression of disease.



Implementation of the sequential and inter-dependent changes to the episodic periodontitis pathogenesis model. The current figure is modified from the original hypothesis, reproduced under the terms of the Creative Commons Attribution License from Gursoy et al[12]

PERIODONTAL DYSBIOSIS AND HOST GENETICS

Host genetic factors that increase susceptibility to periodontal disease may shape the subgingival microbiome in ways that promote dysbiosis. For instance, individuals with Leukocyte Adhesion Deficiency exhibit a subgingival microbiome with markedly reduced diversity, characterized by fewer, unique, and more invasive bacterial species linked to severe periodontal damage[13]. This microbial profile differs from that seen in typical forms of periodontitis in otherwise healthy individuals and aligns more closely with a dysbiotic state. Likewise, reduced microbial diversity has been observed in patients with aggressive periodontitis, where tissue-invasive bacteria are believed to dominate and suppress other members of the microbial community.

THERAPEUTIC IMPLICATIONS OF DYSBIOSIS

Understanding dysbiosis as a central factor in periodontitis has significant implications for treatment strategies. Traditional periodontal therapy focuses on mechanical debridement to disrupt biofilms and reduce bacterial load. While effective to a degree, this approach may not fully address the underlying dysbiotic state or modulate the host immune response. Adjunctive antimicrobial therapy to disrupt the structural integrity of the subgingival biofilm.[14]

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FUTURE PERSPECTIVES TARGETING THE BIOFILM MATRIX :

Matrix-degrading enzymes like Dispersin B and deoxyribonucleases are emerging as promising therapies to disrupt biofilms and enhance treatment effectiveness.[15]

CONCLUSION

Periodontitis is a complex disease rooted in the disruption of the delicate balance between the host and its oral microbiota. As our knowledge of the pathogenesis of periodontitis increases, with a better understanding that the host immune response is the main culprit for the tissue destruction observed with periodontal disease, as well as its role in the dysbiosis of the microbial community, we are drawn to consider alternative methods to aid in the treatment of periodontitis. The concept of dysbiosis provides a unifying framework that integrates microbial, immunological, and environmental factors in the pathogenesis of periodontal disease.

By shifting the focus from specific pathogens to the overall ecological and immunological context, researchers and clinicians can develop more comprehensive and effective strategies for diagnosis, treatment, and prevention. These include microbiome-targeted therapies, host-modulation interventions, and personalized care models. As our understanding of the oral microbiome and its interactions with the host continues to evolve, so too will our ability to manage and ultimately prevent periodontitis—improving not only oral health but systemic health and quality of life.

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