

Oral Microbiota Dysbiosis in Periodontal and Peri-implant Diseases

Linares Degener*

Department of Oral Diseases, National Clinical Research Center for Oral Diseases, Sichuan University, Chengdu, China

Introduction

The human oral cavity harbors a complex and dynamic microbial community that plays a crucial role in maintaining oral health. However, disturbances in this microbial balance, known as dysbiosis, have been implicated in the pathogenesis of various oral diseases, including periodontal disease and peri-implantitis. These conditions are characterized by inflammation and destruction of the supporting structures of teeth and dental implants, respectively. Understanding the mechanisms underlying oral microbiota dysbiosis and its association with these diseases is essential for developing effective preventive and therapeutic strategies. The oral microbiota comprises a diverse array of microorganisms, including bacteria, fungi, viruses, and archaea. Bacteria are the most abundant and are predominantly anaerobic species that colonize various niches within the oral cavity, such as the teeth, gums, tongue, and mucosal surfaces. Under healthy conditions, these microorganisms exist in a balanced state, contributing to the maintenance of oral health through various mechanisms, including the production of antimicrobial peptides and competition with potential pathogens [1].

Dysbiosis refers to an imbalance in the microbial community, where pathogenic microorganisms proliferate at the expense of beneficial ones. This shift can be triggered by various factors, including poor oral hygiene, smoking, diet, systemic diseases, and genetic predisposition. Dysbiosis disrupts the homeostasis of the oral microbiota, leading to an increased abundance of pathogenic species that can initiate and sustain inflammatory processes [2].

Description

Periodontal disease encompasses a spectrum of conditions, from gingivitis to periodontitis, characterized by inflammation of the periodontal tissues. The progression from gingivitis to periodontitis involves the accumulation of dental plaque, a biofilm composed of microorganisms, on the tooth surface. In susceptible individuals, pathogenic bacteria within this biofilm can invade the gingival tissues, leading to inflammation, tissue destruction, and eventual tooth loss if left untreated. A Gram-negative anaerobe that can disrupt host immune responses and promote inflammation. A spirochete associated with severe periodontal conditions. Often found in deep periodontal pockets and associated with advanced periodontal disease. This plays a role in the formation of dental plaque and the progression of periodontal disease. These pathogens possess various virulence factors, such as proteases and toxins that facilitate tissue invasion and immune evasion, contributing to the pathogenesis of periodontal disease. Dysbiosis in the oral microbiota leads to an overrepresentation of

pathogenic bacteria and a reduction in beneficial species. This imbalance results in an inflammatory environment that promotes the destruction of periodontal tissues. Factors such as obesity have been shown to influence the composition of the subgingival microbiota, with obese individuals exhibiting a higher prevalence of periodontal disease due to microbial dysbiosis [3].

Peri-implantitis is an inflammatory condition affecting the tissues surrounding dental implants, leading to bone loss and potential implant failure. Similar to periodontal disease, peri-implantitis is associated with microbial dysbiosis at the implant site. The biofilm formed on dental implants differs from that on natural teeth, with distinct microbial communities contributing to the disease process. Commonly found in both periodontal and peri-implant diseases, indicating its role in oral biofilm formation. Part of the "red complex," these bacteria are strongly associated with periodontal disease and are also prevalent in peri-implantitis. Opportunistic pathogens that can colonize the implant surface and contribute to inflammation. The presence of these pathogens disrupts the local immune response, leading to inflammation and bone resorption around the implant. The microbial communities in periodontal pockets and peri-implant sites differ, with unique species predominating in each environment. The host immune response to microbial invasion may vary between periodontal tissues and peri-implant sites, influencing disease progression and severity [4].

Mechanical debridement, including scaling and root planing, is a standard treatment for both periodontal disease and peri-implantitis. This procedure removes plaque and calculus from tooth and implant surfaces, reducing the microbial load. However, it may not be sufficient to eliminate all pathogenic microorganisms, and adjunctive therapies are often necessary. The use of systemic or local antibiotics can help reduce the population of pathogenic bacteria. However, the emergence of antibiotic-resistant strains poses a challenge, necessitating the development of alternative therapies. Administration of beneficial microorganisms to outcompete pathogens and restore microbial balance. Compounds that promote the growth of beneficial bacteria. Utilization of bacteriophages to target and eliminate specific pathogenic bacteria. Although primarily used for gut dysbiosis, this approach is being explored for its potential in oral health. Modulating the host immune response through the use of anti-inflammatory agents or immune modulators can help control the inflammatory processes associated with periodontal disease and peri-implantitis [5].

Conclusion

Oral microbiota dysbiosis plays a pivotal role in the pathogenesis of periodontal disease and peri-implantitis. Understanding the complex interactions between pathogenic microorganisms and the host immune system is essential for developing effective preventive and therapeutic strategies. While traditional treatments remain fundamental, emerging microbiome-based therapies offer promising avenues for restoring microbial balance and improving patient outcomes. Continued research into the oral microbiome will enhance our ability to manage and treat these prevalent oral diseases effectively.

*Address for Correspondence: Linares Degener, Department of Oral Diseases, National Clinical Research Center for Oral Diseases, Sichuan University, Chengdu, China; E-mail: degenerlinares@esn.cn

Copyright: © 2025 Degener L. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

Received: 03 March, 2025, Manuscript No. OHCR-25-165591; Editor Assigned: 05 March, 2025, PreQC No. P-165591; Reviewed: 17 March, 2025, QC No. Q-165591; Revised: 22 March, 2025, Manuscript No. R-165591; Published: 29 March, 2025, DOI: 10.37421/2471-8726.2025.11.183

Acknowledgement

None.

Conflict of Interest

None.

References

1. Sakima, Vinicius Tatsuyuji, Paula Aboud Barbugli, Paulo Sérgio Cerri and Marlus Chorilli, et al. "Antimicrobial photodynamic therapy mediated by curcumin-loaded polymeric nanoparticles in a murine model of oral candidiasis." *Molecules* 23 (2018): 2075.

2. Czerninski, Rakefet, Anna Pikovsky, Irith Gati and Michael Friedman, et al. "Comparison of the efficacy of a novel sustained release clotrimazole varnish and clotrimazole troches for the treatment of oral candidiasis." *Clin Oral Investig* 19 (2015): 467-473.
3. Wen, Jianchuan, Fuguang Jiang, Chih-Ko Yeh and Yuyu Sun. "Controlling fungal biofilms with functional drug delivery denture biomaterials." *Colloids Surf* 140 (2016): 19-27.
4. Sankar, Vidya, Vanessa Hearnden, Katrisha Hull and D. Vidovic Juras, et al. "Local drug delivery for oral mucosal diseases: Challenges and opportunities." *Oral Dis* 17 (2011): 73-84.
5. Millsop, Jillian W. and Nasim Fazel. "Oral candidiasis." *Clin Dermatol* 34 (2016): 487-494.

How to cite this article: Degener, Linares. "Oral Microbiota Dysbiosis in Periodontal and Peri-implant Diseases." *Oral Health Case Rep* 11 (2025): 183.