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Racial and Ethnic Disparities in Periodontal Disease - Epidemiology, Biological Mechanisms and Clinical Implications

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Abstract

Periodontal disease is a chronic inflammatory condition affecting the supporting structures of the teeth, characterized by progressive clinical attachment loss and alveolar bone destruction. Although the accumulation of bacterial bio film is the primary etiologic factor, disease initiation and progression are influenced by a complex interplay of host immune responses, genetic predisposition, environmental exposures, behavioural factors, and systemic health. Emerging evidence suggests that the prevalence, severity, and progression of periodontal disease vary among different racial and ethnic populations. These disparities are likely attributable to a combination of biological, socioeconomic, cultural, environmental, and healthcare access factors rather than race alone. Furthermore, several systemic conditions, including cardiovascular disease, diabetes mellitus, and other endocrine disorders, exhibit racial and ethnic differences in prevalence and outcomes, which may further influence periodontal health through shared inflammatory and metabolic pathways. This review examines the current evidence on racial and ethnic variations in periodontal disease, explores the underlying biological and social determinants contributing to these differences, and discusses their implications for risk assessment,

prevention strategies, and personalized periodontal care. A comprehensive understanding of these disparities may facilitate the development of more equitable and targeted approaches to periodontal disease management and future research.

Keywords: Periodontitis, Racial Disparity, Race, Ethnicity, Oral Health

Introduction

Periodontitis is a common oral inflammatory disease with high global prevalence. It is characterized by the progressive destruction of the gingiva, periodontal ligament, cementum, and alveolar bone, ultimately resulting in clinical attachment loss, tooth mobility, and tooth loss if left untreated.[1] Beyond its detrimental effects on oral health, periodontitis has been increasingly recognized as a condition with significant systemic implications, owing to its association with chronic inflammatory diseases such as diabetes mellitus[2], cardiovascular disease, rheumatoid arthritis, adverse pregnancy outcomes, and metabolic syndrome.

The initiation of periodontal disease is primarily attributed to the accumulation of pathogenic bacterial bio film at the gingival margin.[3] However, the presence of microbial plaque alone is insufficient to explain the considerable variability in disease susceptibility, severity, and progression observed among individuals.

The pathogenesis of periodontitis is multi factorial, involving a dynamic interaction between periodontal pathogens, the host immune-inflammatory response, genetic predisposition, epigenetic modifications, environmental influences, behavioural factors, and systemic health conditions.[4] This complex interplay determines whether periodontal inflammation remains localized or progresses to irreversible periodontal tissue destruction.

Epidemiological studies have consistently demonstrated marked differences in the prevalence, severity, and progression of periodontal disease among racial and ethnic populations across the world.[5] While certain populations exhibit a disproportionately higher burden of periodontitis, these variations cannot be attributed solely to biological or genetic differences. Instead, they reflect the combined influence of multiple determinants, including socioeconomic status, education, access to dental care, cultural beliefs, health-related behaviours, dietary practices, tobacco use, psychosocial stress, environmental exposures, and healthcare disparities.[6] Genetic diversity, host immune response, and differences in the oral micro biome may also contribute to inter-population variability, although their relative contributions remain an area of active investigation.[7]

Importantly, many systemic diseases that are closely linked with periodontal disease, including diabetes mellitus, cardiovascular disease, obesity, and endocrine disorders, also demonstrate significant racial and ethnic disparities in their prevalence and clinical outcomes.[8] These conditions share common inflammatory and metabolic pathways with periodontitis, suggesting that population-specific differences in systemic health may further modify periodontal disease susceptibility and treatment outcomes. Understanding these inter relationships is essential for developing comprehensive strategies aimed at improving both oral and general health.

Recent advances in precision medicine and personalized healthcare have highlighted the importance of identifying population-specific risk factors for disease prevention and management. In periodontology, recognizing racial and ethnic disparities may facilitate more accurate risk assessment, improve patient-centered treatment planning, and support the development of targeted preventive

interventions.[7] However, it is equally important to distinguish between biological ancestry and the broader social construct of race, acknowledging that observed disparities often arise from the interaction of biological, environmental, and social determinants rather than race itself.

The purpose of this review is to critically examine the existing literature on racial and ethnic variations in periodontal disease, with emphasis on epidemiological trends, genetic and immunological factors, oral micro biome differences, systemic disease associations, and social determinants of health. Additionally, this review discusses the clinical implications of these disparities and identifies current knowledge gaps to guide future research toward more equitable and personalized periodontal care.

Epidemiology of Periodontal Disease Across Different Racial and Ethnic Populations

Periodontal disease has a high global prevalence rate. As per World Health Organization (WHO) reports, prevalence of severe periodontitis ranges from 10-15% of the world population. The WHO recommends the usage of Community Periodontal Index (CPI) for prevalence studies on periodontal disease for sake of assessment among varying populations. Worldwide, about 62% of individuals are affected by mild periodontitis, whereas severe periodontitis occurs in nearly 23.6% of the population. Owing to its widespread occurrence, periodontitis is documented as the seventh most prevalent disease affecting humans.[9]

Periodontitis affects nearly half of all adults, but prevalence varies significantly by race and ethnicity. According to large-scale epidemiological data like the CDC Periodontitis in Adults studies, these differences highlight deep systemic oral health disparities.

Prevalence rates among adults aged 30 and older are structured as follows[10]:

- Hispanic Americans: ~63.5% prevalence rate (the highest among major demographic groups).
- African Americans (Non-Hispanic Black): ~59.1% prevalence rate.
- Asian Americans (Non-Hispanic Asian): ~50.0% prevalence rate.
- Native Americans (American Indian / Alaska Native): ~77.2% to 84.3% reported in specific community and retrospective studies, though broader national datasets often pool them into smaller categorical datasets.
- Caucasians (Non - Hispanic White): ~40.8% prevalence rate (the lowest among major demographic groups).

These disparities are not strictly genetic. They are intensely intertwined with socioeconomic status, insurance access, frequency of dental visits, and the presence of underlying systemic conditions like diabetes.[10]

The occurrence of periodontitis varies significantly across Europe, with moderate to severe forms affecting up to half of the population. Studies consistently show that Germany reports some of the highest prevalence rates (often exceeding 50% in adults), whereas countries like Spain, Switzerland, and Sweden generally experience lower rates of the disease.[11]

Investigations conducted by the National Health and Nutrition Examination Survey (NHANES) provide the benchmark for periodontal disease epidemiology in the U.S. Overall prevalence shows approximately 42% to 46% of dentate U.S. adults aged 30 years and older have some form of periodontitis. Of the affected population, roughly 7.8% to 9% suffer from severe periodontitis.[12]

Epidemiological reviews indicate an unusually high burden of periodontal disease in the Indian subcontinent. Several studies indicate that up to 50% to 96% of Indian adults experience some form of periodontal disease (including gingivitis).[13]Systematic analyses report mild to moderate periodontitis in about 26% of adults, with severe periodontitis affecting approximately 19% of the population.[13]

Studies across Africa show a significant and often underestimated burden of periodontitis, particularly among younger demographics.[14] Epidemiological data pooling shows a cumulative periodontitis prevalence of roughly 11.2% in adolescents and up to 23% in older adults (50 years and older).[15]

Biological Basis of Racial Differences

Genetics

Periodontitis is a multifactorial disease which is associated with several risk factors such as smoking, systemic diseases, stress, obesity, genetic factors etc. Familial aggregation of periodontal disease seems to play a vital role in pathogenesis of theailment.[16]

Polymorphism is defined as the existence of at least two forms of a gene in a population, each of which occurs with a frequency greater than 1%. Polymorphism is the result of variations in DNA sequence that can be perceived by molecular biology methods. Genetic polymorphism can be the consequence of replacing a single nucleotide in a DNA sequence with additional one, removing or inserting one or more nucleotides, or inserting repetitive sequences.

IL-1 polymorphism

Interleukin-1 (IL-1) is among the most extensively investigated cytokines in studies evaluating the genetic basis of periodontal disease. It plays a pivotal role in the initiation and maintenance of the inflammatory response by promoting the expression of adhesion molecules that facilitate leukocyte recruitment to sites of inflammation. IL-1 also enhances the production of other pro-inflammatory cytokines and matrix metalloproteinases, activates T- and B-lymphocytes, stimulates osteoclast-mediated alveolar bone resorption through osteoblastic signaling, and induces apoptosis of extracellular matrix-producing cells, thereby impairing periodontal tissue regeneration. Genetic polymorphisms in the IL1A and IL1B genes have been identified as important genetic markers that may increase an individual's susceptibility to chronic periodontitis.[17]

TNF-aplha

Tumor necrosis factor (TNF) is a key pro inflammatory cytokine that plays a pivotal role in regulating immune and inflammatory responses. Numerous case–control studies conducted among both Caucasian and non-Caucasian populations have examined TNFA gene polymorphisms as potential genetic determinants of susceptibility to periodontitis.[18]

It stimulates inflammatory cell recruitment, enhances osteoclast activity, promotes connective tissue degradation, and amplifies cytokine production. Variations within the TNFA gene, particularly promoter polymorphisms such as −308 G/A and −238 G/A, have been associated with altered TNF-α expression.[19]

Individuals carrying high-expression TNFA alleles may exhibit a hyper-inflammatory phenotype characterized by excessive cytokine production following bacterial stimulation. This enhanced inflammatory response can increase the risk of connective tissue destruction and alveolar bone loss.

The distribution of TNFA polymorphisms varies among racial and ethnic populations, although their contribution to disease susceptibility remains inconsistent because of differences in study design, sample size, and population characteristics.[20]

Interleukin-6 (IL-6) Gene Polymorphisms

Interleukin-6 (IL-6) is a multifunctional cytokine that regulates both innate and adaptive immune responses. It contributes to periodontal inflammation by stimulating acute-phase protein synthesis, promoting B-cell differentiation, and facilitating osteoclast-mediated bone resorption.

Polymorphisms in the IL6 promoter region, particularly −174 G/C, have been investigated for their association with chronic periodontitis. Certain genetic variants are associated with increased IL-6 production, potentially resulting in prolonged inflammatory activity and greater periodontal tissue destruction. However, allele frequencies differ substantially among ethnic populations, and not all studies have demonstrated a significant association with disease susceptibility, indicating that IL-6 polymorphisms are only one component of a multi factorial pathogenic process.[21]

Vitamin D Receptor (VDR) Gene Polymorphisms

Vitamin D plays a critical role in bone metabolism, calcium homeostasis, and immune regulation. Its biological effects are mediated through the vitamin D receptor (VDR), which influences osteoblast differentiation, osteoclast activity, and antimicrobial peptide production.

Several polymorphisms within the VDR gene, including FokI, BsmI, ApaI, and TaqI, have been evaluated in relation to periodontal disease. These genetic variations may modify receptor activity, influencing bone mineral density and host immune responses. Differences in VDR polymorphism frequencies have been observed among Asian, European, and African populations, suggesting that genetic variation may partially contribute to inter-population differences in periodontal susceptibility. Nevertheless, environmental factors such as vitamin D status, sunlight exposure, nutrition, and lifestyle also substantially influence disease risk.[22]

Fc Gamma Receptor (FcγR) Gene Polymorphisms

Fc gamma receptors (FcγRs) are expressed on neutrophils, macrophages, and other immune cells, where they mediate antibody-dependent phagocytosis and clearance of microbial pathogens. Functional polymorphisms in FCGR2A, FCGR2B, and FCGR3A genes may alter receptor affinity for immunoglobulin G (IgG), thereby affecting bacterial clearance and inflammatory regulation.

Individuals possessing receptor variants with reduced binding affinity may exhibit impaired elimination of periodontal pathogens, leading to persistent infection and chronic inflammation. Several studies have demonstrated associations between Fcγ receptor polymorphisms and aggressive or chronic periodontitis, although findings vary among different racial and ethnic populations.[23]

Matrix Metalloproteinase (MMP) Gene Poly morphisms

Matrix metalloproteinases (MMPs) are zinc-dependent proteolytic enzymes responsible for degradation of extracellular matrix components during physiological tissue remodelling and inflammatory disease. In periodontitis, excessive expression of MMPs contributes to collagen degradation, periodontal ligament destruction, and alveolar bone loss.

Polymorphisms in genes encoding MMP-1, MMP-2, MMP-3, MMP-8, and MMP-9 have been associated with altered enzyme expression and increased susceptibility to periodontal tissue destruction. Elevated MMP activity has been linked to greater clinical attachment loss and disease severity.[24] However, considerable variability exists in the distribution of these polymorphisms among different ethnic groups, highlighting the importance of population-specific genetic studies.

Influence of Genetic Polymorphisms on the Inflammatory Response

The inflammatory response in periodontitis is determined not only by the presence of pathogenic microorganisms but also by the host's genetic capacity to regulate immune activation. Genetic polymorphisms affecting cytokines, immune receptors, and tissue-degrading enzymes can influence the intensity, duration, and resolution of inflammation. Individuals carrying high-expression variants of pro-inflammatory cytokines may generate excessive inflammatory mediators in response to bacterial biofilm, resulting in increased osteoclast activation, enhanced extracellular matrix degradation, and accelerated periodontal destruction. Conversely, polymorphisms associated with reduced immune regulation or impaired pathogen clearance may allow persistent microbial colonization, perpetuating chronic inflammation.

Although numerous candidate gene studies have identified associations between specific polymorphisms and periodontal disease, no single genetic variant fully explains differences in disease susceptibility among racial or ethnic populations. Contemporary evidence indicates that periodontal disease results from complex interactions between genetic predisposition, oral microbial ecology, environmental exposures, systemic health, and social determinants.[25]

Socioeconomic and Environmental Determinants

Although biological factors contribute to periodontal disease susceptibility, socioeconomic and environmental determinants play a pivotal role in shaping oral health disparities across racial and ethnic groups. Individuals from socioeconomically disadvantaged backgrounds often experience a greater burden of periodontitis due to limited access to preventive dental services, inadequate oral health education, financial constraints, and delayed treatment seeking. Lower educational attainment has been associated with poorer oral hygiene practices and reduced awareness of periodontal disease, resulting in increased plaque accumulation and disease progression.[26] Lifestyle behaviours such as tobacco use, unhealthy dietary patterns, psychological stress, and poor glycemic control further exacerbate periodontal destruction. Environmental influences, including neighbourhood characteristics, access to fluoridated water, availability of dental care providers, and healthcare infrastructure, also affect oral health outcomes. Importantly, racial and ethnic disparities in periodontal disease frequently reflect inequalities in social and environmental conditions rather than inherent biological differences alone.[27] Therefore, addressing these determinants through equitable health care policies, community-based preventive programmes, and improved access to oral healthcare is essential for reducing disparities.

Systemic Diseases and Shared Disparities

Periodontal disease frequently coexists with systemic conditions that disproportionately affect certain racial and ethnic populations, creating a complex network of shared health disparities.[28] Diabetes mellitus remains one of the strongest risk factors for periodontitis, with hyperglycaemia promoting chronic inflammation, impaired wound healing, and increased susceptibility to periodontal tissue destruction. Conversely, periodontal inflammation may adversely affect glycemic control, highlighting the bidirectional relationship between the two diseases.[29]

Cardiovascular disease, obesity, metabolic syndrome, and adverse pregnancy outcomes have also been linked to periodontal inflammation through common inflammatory pathways. These conditions often occur more frequently among populations experiencing socioeconomic disadvantage, limited healthcare access,

and higher exposure to modifiable risk factors.[30] Consequently, disparities in periodontal health frequently mirror broader inequalities in systemic health, emphasizing the need for integrated medical and dental care to improve overall health outcomes.

Precision Periodontology

Advances in molecular biology, genomics, and digital health technologies have introduced the concept of precision period ontology, which aims to provide individualized prevention and treatment based on each patient's biological and environmental risk profile. Rather than relying solely on clinical parameters, precision approaches incorporate genetic susceptibility, inflammatory biomarkers, microbial composition, systemic health, behavioural factors, and lifestyle characteristics to improve disease prediction and therapeutic decision-making.

Emerging technologies such as genomic analysis, salivary diagnostics, artificial intelligence, and micro biome profiling offer opportunities for early identification of high-risk individuals and more targeted periodontal interventions. While these approaches have the potential to reduce disparities by enabling personalized care, their successful implementation requires equitable access to advanced diagnostic technologies and careful consideration of population diversity to avoid widening existing healthcare inequalities.[31]

Clinical Implications

Recognition of racial and ethnic disparities in periodontal disease has important implications for contemporary clinical practice. Comprehensive patient assessment should extend beyond conventional clinical findings to include socioeconomic circumstances, systemic health status, behavioural risk factors, and barriers to accessing dental care. Treatment planning should be individualized, culturally sensitive, and supported by effective communication strategies that promote patient engagement and adherence to oral hygiene recommendations.

Preventive measures, including early risk assessment, regular periodontal screening, smoking cessation counselling, diabetes management, and patient-centred oral health education, should be prioritized for individuals at increased risk. Collaborative care involving dental professionals, physicians, and public health practitioners may further improve periodontal outcomes by addressing common risk factors shared between oral and systemic diseases.[32] Ultimately, reducing periodontal health disparities requires a comprehensive approach that integrates personalized clinical management with broader public health initiatives aimed at improving healthcare accessibility and reducing social inequalities.

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