Brief Communication

Genetic factors in pathogenesis of chronic periodontitis

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Abstract

Chronic periodontitis is a global health problem that affects the majority of adult population worldwide. The inflammatory destruction of tooth supporting structure is multifactorial in nature. It results from interaction between microbial, environmental, immunologic, and genetic factors. The exact mechanism of action by which genetic factors alter the course of chronic periodontitis and aid in pathogenesis is yet not well understood. Studies suggest association genetic polymorphism and susceptibility to chronic periodontitis. It is recommended that further investigation of a larger sample size with consideration of interaction with other factors that contribute to the pathogenesis of chronic periodontitis.

Keywords: Chronic periodontitis; Genetic factors; Periodontal disease

Introduction

Chronic periodontitis is an inflammatory destruction of tooth supporting structures that, if left untreated, may lead to tooth loss.1,2 Chronic periodontitis is the most prevalent inflammatory disease worldwide. It affects nearly 50% of adult population and 60% of aged population globally.3

The pathogenesis of chronic periodontitis is multifactorial in nature. It results from interaction between bacterial, environmental, immunologic and genetic factors.3,4 Subgingival bacteria are the main factor responsible for chronic periodontitis. Bacterial plaque activates inflammatory response, which in turn activates a cascade of immune reactions.3 Porphyromonas gingivalis (P. gingivalis) and Fusobacterium nucleatum (F. nucleatum) are highly associated with periodontitis development. However, periodontal pockets often harbor a mixture of anaerobic bacteria.4 Periodontitis sites are characterized by a large volume of inflammatory cells infiltrate and vascular proliferations. Immune response to bacterial plaque and virulence factors released by pathogenic bacteria is B-cell mediated. Plasma cells and lymphocytes are the predominant inflammatory cells at the periodontitis sites.4,5 Smoking is a recognized risk factor for chronic periodontitis. Smoking and smoke components produce a hypo-oxygenated environment that favors anaerobic bacterial growth and reduces the level of
vascular supply, which aids to promote tissue destruction. The role of genetics in the development of periodontitis is controversial. This paper aims to review the genetics factors that play a role in the pathogenesis of chronic periodontitis.

The role of matrix metalloproteases gene (MMPs)

MMPs are a group of proteolytic enzymes responsible for extracellular matrix and basement membrane destruction. MMP-1 is abundant in periodontitis sites. It is responsible for types I and III collagen degradation. Types I and III collagen are the predominant types of interstitial collagen in the periodontium. Scientific data suggests that 2G allele, instead of 1G allele, at MMP-1-1607 gene created a new 59-GGA-39 core recognition sequence, which results in increased potency of bone resorption and connective tissue destruction. MMPs are a group of proteolytic enzymes responsible for extracellular matrix and basement membrane destruction. MMP-1 is abundant in periodontitis sites. It is responsible for types I and III collagen degradation. Types I and III collagen are the predominant types of interstitial collagen in the periodontium. Scientific data suggests that 2G allele, instead of 1G allele, at MMP-1-1607 gene created a new 59-GGA-39 core recognition sequence, which results in increased potency of bone resorption and connective tissue destruction. The role of genetics in the development of periodontitis is controversial. This paper aims to review the genetics factors that play a role in the pathogenesis of chronic periodontitis.

The role of interleukins (IL) gene

Interleukins (IL) are a class of cytokines produces by immunocompetent cells in local inflammatory tissue. It is released into periodontitis sites in response to bacterial presence and their virulence factors that aids in bone resorption and connective tissue destruction. The role of IL genes polymorphism in the pathogenesis of chronic periodontitis is largely obscured. Studies indicate IL-1α, IL-1β, IL-1RN, IL-6, and IL-10 gene variations are significantly associated with chronic periodontitis. IL-1β is associated with increased potency of bone resorption and connective tissue destruction. IL-1β+3953 C > T polymorphism is significantly associated with chronic periodontitis. The carriage of T allele increases the risk of periodontitis. Scientific literature reports conflicting results in the role of IL-17R gene polymorphism in chronic periodontitis pathogenesis. Studies that investigated the role of IL gene polymorphism are inconclusive because the interaction between gene polymorphism and environmental and microbial factors that contribute to periodontitis pathogenesis. Nevertheless, no information was provided on the effect of coexistence of systemic conditions and the interaction between oral and systemic factors.

The role of vitamin D receptor (VDR Taq1) on genetic polymorphism

Vitamin D plays a key role in bone metabolism. VDR Taq1 polymorphism, however, increases the susceptibility to chronic periodontitis despite the fact that the exact mechanism through which gene polymorphism exerts its effect is yet not well understood. The sample size remained a significant limitation in studies that investigated the role of VDR Taq1 polymorphism in the pathogenesis of periodontitis. Therefore, establishing a proper animal model is necessary to understand the mechanism of action and determine preventive methods.

Beta-defensin-2 genomic copy number variation

Human beta-defensin-2 (hBD-2) is a small 3- to 5-kDa cationic peptide produced by several epithelial cells including oral epithelium. It regulates immune cell chemotaxis and cytokines release, links innate and adaptive immunity, and promotes wound healing. Low DEFB 4CN is associated with increased susceptibility to severe chronic periodontitis because it alters hBD-2 production, which leads to imbalanced host immune response. Therefore, DEFB 4CN can be used as a genetic marker. However, further studies with a larger sample size are required to confirm the sensitivity of DEFB 4CN as a biomarker and determine whether its level is affected by environmental and/or systemic conditions.

Conclusion

Chronic periodontitis is initiated by bacterial infection. However, interaction between microbial, environmental, immunological, and genetic factors plays a major role in the pathogenesis and progression of chronic periodontitis. The role of genetic factors is largely obscured and the exact mechanism of which it exerts its effect is not well understood yet. Further studies that investigate the mechanism of genetic factors and its interaction with microbial, environmental and immunologic factors are necessary.

Conflict of interest

The authors have no conflict of interest to declare.

References


