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Epigenetics in Periodontal Diseases

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Abstract

Genetics and epigenetics is the process by which genes were expressed or suppressed through polymorphism, demethylation, and deacetylation. Many Environmental factors such as race, gender, diabetes, education, smoking etc. can have profound effects on the epigenetic changes and induce susceptibility to disease. Periodontal diseases are bacterial infections in which predominantly gram negative anaerobic bacteria colonize the sub gingival areas and triggers an inflammatory response that affect the periodontium; periodontitis is a periodontal disease that destroys the tissue and bone surrounding the tooth. Periodontitis is responsible for tooth loss in adults.

Keywords: Epigenetics; Chronic periodontitis; Genetics; Inflammatory disorders

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Introduction

There are three types of epigenetic modifications seen as DNA methylation, histone modification and RNA-associated silencing (micro-RNA) [1,2]. It was found that with methylation, demethylation and acetylation, deacetylation or combination of these worked to either express or repress genes during replication. DNA methylation is one of the most broadly studied and well characterised epigenetic modification dating back to studies done by Griffith and Mahler in 1969 which suggested that DNA methylation may be important in long term memory function [3]. These processes triggered by environmental influences regulated cytokines such as IL6 and IL1 which were responsible for inflammatory tissue destruction. Susceptibility of individual to periodontitis depends on his immune response. Decrease in number of neutrophils also increases the susceptibility of infections.

Cytokines noted as inflammatory response influence the host response in periodontitis [4]. Cytokines are reported as powerful regulatory proteins released by the immune cells influence the behaviour of other cells [5]. SNPs of interleukin (IL-) 1a, IL-1B, IL-4, IL-6, IL-8, IL-18 located in different regions of the cytokine have been shown to affect the risk of the disease in several populations [6]. A study was carried on and seen the polymorphism in IL 1 due to major part that IL 1B played in the development of periodontitis. Studies suggested that individuals who had higher level of IL1 have a greater chance of periodontitis then those having less level [7].

Modification of Histone Proteins and DNA Methylation

A gene is reported as the instructional portion of DNA that signals biological activity within our cells. DNA methylation is noted as chemical reactions that add a methyl group to a DNA molecule (hypermethylation) and (demethylation) as the removal of methyl group from DNA [8]. Histone proteins are responsible for packing and unpacking chromosomes for transcription process. Histone modification results from enzymatic acetylation or deacetylation. Acetylation creates a less compact DNA complex that allows for gene expression in contrast deacetylation creates a more compact DNA complex that suppresses gene expression. It is seen that there is a strong correlation between gene expression with both histone modification and DNA methylation [9]. These reaction leads to change in gene expression which leads to change in biological activity while maintaining the DNA sequence as it is. These types of reactions are reversible and are called as epigenetic regulation. It was found that with methylation, de methylation and acetylation, deacetylation or any combination of these worked to either express or repress genes during replication. It was found that these processes were key factors in inflammatory diseases. These processes triggered by environmental influences regulated cytokines such as IL1 and IL6 which are responsible for periodontitis [9]. The two common epigenetic factors are nutrition and aging. Effects on epigenetics

can be seen in deficiency of folic acid and deficiencies of selenium, arsenic and polyphenols. These deficiencies can cause diseases like neural tube defect and malignancies. Wilson reported on epigenetic factor aging methylation and acetylation process were carried out in mono zygotic twins and very similar results were seen in early life and substantial differences in later life. Several Environmental influences contribute to the progression of periodontitis. Alterations in DNA methylation status as a result of environmental stressors have been reported to begin before birth though some epigenetic marks are potentially reversible, many epigenetic changes appear to persist throughout the cell lineage and life of the organism several factors responsible for epigenetic modification are race, gender, diabetes, BMI etc. One of the important environmental risk factors for progression of periodontitis is smoking. Smoking causes long term hypo and hypermethylation changes in the DNA. Haffajee and Socransky in a study found that smokers had more severe form of periodontitis with more attachment loss and deeper pockets compared with non-smokers.

Epigenetics and Susceptibility of Disease

The person is susceptible to disease due to the process of hyper/hypo methylation, histone acetylation or combination of any of these. Disease is generally inflammatory in nature like rheumatoid, arthritis, periodontitis. In chronic periodontitis hypermethylation of gene E cardherin and cyclo oxygenase 2 takes place which increases the susceptibility of individual to periodontitis. In a study from the journal of translational medicine few DNA were extracted from the gingival of 108 systematically healthy non periodontitis patient, blood samples from 110 periodontitis patients and neoplastic tissues from 106 breast cancer patient and then all are tested for the methylation of E-cardherin and cyclo-oxygenase 2. In periodontitis patients detection rate of hypermethylation of E cadherin and cyclooxygenase was 25 and 19% and nothing was found in systematically healthy non periodontitis patient [10]. These findings were obvious enough to establish the relation between hypermethylation and periodontitis.

Conclusion

Susceptibility of individuals to periodontitis is increased when there is increased level of IL1 and IL1 gene polymorphism. Epigenetics and environmental factors changed the expression of DNA transcription through these factors (diet, diabetes, aging, gender, BMI, race, smoking) an individual's predisposition for inflammatory response is determined.

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