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Commentary

Periodontitis Pathogenesis Immune-Regulation on an Epigenetic Basis

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DESCRIPTION

A host inflammatory response to the specific periodontal pathogens characterizes chronic periodontitis. There is an innate and adaptive phase to the immune response of the host. The TLR pathway is one of the most significant immune mechanisms that link the innate and adaptive pathways. TLR responses to the bacteria may be controlled by microRNAs. According to a recent study, ApoE$^{-/-}$ mice with experimental periodontitis had elevated miR-146a levels when infected with three periodontal pathogens: *P. gingivalis*, *Treponema denticola*, and *Tannerella forsythia*. THP 1 monocyte cell line cultures were stimulated with a combination of periodontal pathogens in the same study. There was a time-dependent increase in mi-RNA levels, which was associated with the down regulation of adaptor kinases IL-1 Receptor-Associated Kinase 1 (IRAK-1) and Tumor Necrosis Factor (TNF) Receptor-Associated Factor (TRAF-6) as well as TNF-alpha production by these cells. The creators reasoned that the raised degrees of miR-146a added to endotoxin resilience by adversely impacting IRAK-1 and TRAF-6 levels at a post transcriptional level and that miR-146a may address an objective for helpful mediation in periodontal illness.

Lipopolysaccharide (LPS) endotoxin was demonstrated that an elevated level of miR-155 down-regulates TAB2, also known as TAK1 binding protein 2, which is crucial to the IL-1 signaling pathways. The dendritic cell inflammatory responses were influenced by miR-155, which mediated the negative feedback loop. The micro RNA expression profile of chronic periodontitis and healthy gingiva was compared. Expression of the microRNAs miR-181b, miR-19b, miR-30a, miR-let 7a, and miR-301a was found to be significantly higher in the periodontitis group than in the healthy group. However, the significance of these findings has yet to be established.

The naive T cells undergo differentiation into various lineages, and during the differentiation process, changes in the chromatin structure occur through epigenetic mechanisms such as histone modification, DNA methylation, and generation of DNAse I hypersensitive sites. The primary cytokine genes that define the lineage specificity include the IFN Gamma gene for the Th1 cell lineage, the IL-4 gene for the Th2 cell lineage, and the IL-17 gene for the Th17 cell line. The various subsets of T cells have been found to have distinct patterns of methylation. Induced Treg cells have been marked with H3K4 me3 in the *FoxP3* gene, which was, however, not observed in Th17 subset and n Tregs. Taking into consideration periodontitis being an inflammatory condition whose pathogenesis involves the different T cell subsets, further studies can be conducted to analyze the role of the aforementioned epigenetic marks in disease pathogenesis.

In gingival tissue biopsies from adults with chronic periodontitis the presence of a significant number of cells that secreted antibody against Type I and, to a lesser extent, Type III collagen. Adult (chronic) periodontitis could possibly be caused by autoimmunity. After methylation inhibitors, 5' Azacytidine, were used to stop DNA methylation in mature T cells, autoreactivity by T cells was seen. Natural T regulatory cells are a subset of immune cells that have a suppressive function and also play a role in controlling immune responses and maintaining peripheral tolerance. The natural T regulatory cells represent a subset of immune cells that have a suppressive function and also play a role in controlling immune responses and maintaining peripheral tolerance. De-methylated T cells overexpression of cytokines, cell surface co-stimulatory molecules, and overstimulation of B. There may be a role for this T cell subset in the pathogenesis of periodontitis, as evidenced by the elevated levels of T regulatory cells found in gingival biopsies from periodontitis patients. T regulatory cells' growth and function depend heavily on the transcription factor Fox P3.

DNA methylation and histone modifications contribute to the establishment of a committed lineage of phenotypically stable regulatory T cells at the Foxp3 locus. It is unknown whether epigenetic pathways are involved in the autoimmune mechanisms that drive the pathogenesis of periodontitis. It is interesting to note that different models of periodontitis progression exist and that not all cases of gingivitis progress to periodontitis. Pathogen-associated molecular pattern signaling pathways, immune regulatory mechanisms, and cytokine production variability may all play a significant role in the pathogenesis of periodontitis because of their action on epithelial cells.

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