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Analysis of Salivary MicroRNA-146a and IL-17A in Patients with Periodontitis and Their Association with Periodontal Tissue Destruction

A Thesis

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Abstract

Background: Periodontitis is a chronic inflammatory disease of periodontium that involving microorganism and host response that causes periodontal tissue damage. Additionally, the genetic predisposition can also participate in susceptibility to periodontitis. Immune responses to bacterial products and the subsequent production of inflammatory cytokines, are crucial in the destruction of periodontal tissue. Micro ribonucleic acids is one of the epigenetic factors, deregulation of them can contributes to the periodontitis pathogenesis. These micro ribonucleic acids regarded as attractable biomarkers and important responders that contribute to the regulation of defense and inflammatory responses of the host.

Aim of Study: The aim of the present study were to investigate the levels of gene expression of micro ribonucleic acid-146a in saliva of patients with periodontitis and control, and examine its role in regulating the inflammatory immune response by determine interleukin-17A. As well as to assess the relationship between the level of micro ribonucleic acid-146a with interleukin-17A and clinical periodontal parameters.

Materials and Methods: This case control study was performed on forty patients with ages range (23-55) years and 40 healthy controls, their ages and sexes were matched with the patients. Periodontal parameters used in present study include, plaque index, gingival index, bleeding on probing, probing pocket depth and clinical attachment level. Saliva samples were collected from all subjects (patients and controls), and then enzyme-linked immunosorbent assay was carried out to estimate the salivary level of interleukin-17A. While micro ribonucleic acid-146a in saliva samples was performed by means of reverse transcription quantitative polymerase chain reaction

Results: The current results revealed a significant elevation in median level of interleukin-17A in patients group than control group ($P< 0.05$). Moreover,

interleukin-17A shows highly significant positive association with plaque index, probing pocket depth and clinical attachment level (r=0.436, p=0.004; r=0.534, p=0.000 and r=0.595, p=0.000) respectively. On the other hand, studying of the micro ribonucleic acid-146a gene expression showed a significant down-regulation in the fold change of gene expression of the micro ribonucleic acid-146a in the patients in comparison to the control (P< 0.05). Furthermore, negative non-significant correlation also showed between salivary micro ribonucleic acid-146a level and each of plaque index, gingival index and bleeding on probing (r=-0.360, p=0.186; r=-0.082, p=0.771 and r=-0.050, p=0.859). While there were significant strong inverse correlations between micro ribonucleic acid-146a level and both of probing pocket depth and clinical attachment level (r= -0.417, p=0.024, r=-0.591, p=0.011). Finally, linear regression correlation between interleukin-17A with micro ribonucleic acid-146a in patients which revealed a weak inverse non-significant correlation (r=-0.276, P=0.319).

Conclusion: These findings indicated that increased level of interleukin-17A in periodontitis patients play a crucial role in pro-inflammatory response in periodontitis. In addition down-regulation of micro ribonucleic acid-146a with its negative correlation with clinical periodontal parameters (probing pocket depth and clinical attachment level) and interleukin-17A level in patients may aggravate the inflammatory immune response and contribute to pathogenesis of periodontitis.