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Research Article

Interleukin Level 1 in Smokers GCF

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ABSTRACT

Smoking is an environmental factor that places individuals at high risk for negative effects on the periodontal health However, the biological mechanisms behind these detrimental effects are still obscure. Periodontal diseases are considered to be initiated by bacteria that activate pathogenic processes leading to tissue destruction(1).Basically, the same subgingival microflora is observed in smoker and non-smoker patients with periodontal disease. Hence, the effects of smoking on periodontal health seem unrelated to the composition of the sub- gingival microflora(2). Cytokines such as interleukins -1, -6 (IL-1, IL-6) and tumor necrosis factor alpha (TNF-a) are considered to be involved in the host response of periodontal disease as mediators of tissue destruction.

Keywords: Interleukin,Smoking.

INTRODUCTION

Periodontal diseases are a group of conditions affecting the supporting structures for the dentition. Periodontal disease is a chronic bacterial infection characterized by persistent inflammation, connective tissue breakdown and alveolar bone destruction. In addition to local periodontal tissue involvement, chronic infection of periodontium with continuous up regulation of pro inflammatory responses and immune mediators may contribute to systemic sequel including diabetes, pre term low birth weight infants, lung inflammation, arthritis and cardiovascular diseases. The risk factors smoking, diabetes, pathogenic bacteria, genetic factors, age, gender ,socioeconomic status, sress.

Pathogenesis of periodontal disease

The pathogenic processes of the periodontal diseases are largely the result of the host response to microbially induced tissue destruction. These destructive processes are initiated by bacteria but are propagated by host cells[1]. Thus, it is the host response that results in tissue destruction. The host produces enzymes that break down tissue. This is a necessary process that is initiated and controlled by the host in order to allow the tissues to retreat from the destructive lesions initiated by bacteria. The pathogenic processes in periodontal disease can be likened to a situation where the host strategy is to eventually expel the tooth so that the inflammation will be stopped. This is the ultimate tissue retreat from the microbial plaque, and once the tooth has been expelled, the lesion is finally defused. This is because there is no further tooth site for the plaque to build up on and simplistically, there is no periodontium left to be infected. Thus, the exfoliation of teeth during periodontal disease might be considered a host preventive strategy to protect against deeper infection such as osteomyelitis[3].

Smoking and periodontal disease

Earlier reviews of the epidemiology of periodontal disease concluded that smoking was a possible causative factor.Few studies have conclusively demonstrated any relevant microbiological changes in the periodontal tissues attributable to smoking. Some authors using self-reported smoking data, investigated the relationship between periodontal pathogens and cigarette consumption[4].They reported an increased risk for smokers to have subgingival infection with Porphyromonas gingivalis although this was not found to be statistically significant[5].

The Role of Cytokines in Periodontal Disease

Cytokines - Lipopolysaccharide (LPS) is a key microbial stimulus that will trigger the host response at periodontal disease sites(5).Locally, it triggers monocytes to release inflammatory mediators (Prostaglandin E2, Thromboxane B, Interleukins -1, -6 and -8, Tumor necrosis factor) that increase the local destruction of the connective tissues structural elements. Interleukin-1 (IL-1) is a potent bone-resorbing cytokine formerly known as the osteoclast-activating factor[6]. Interleukin-1 is primarily produced by activated macrophages or lymphocytes but it may also be released by other cells, including mast cells, fibroblasts, keratinocytes, endothelial cells and its production is stimulated by bacterial lipopolysaccharide.IL -1 has been detected in both periodontal tissues and GCF in patients with periodontal disease[7].

Biomarkers in oral and periodontal disease

GCF has been extensively investigated for the release of host response factors. It includes a mixture of molecules from blood, host tissue, and plaque biofilms, such as electrolytes, small molecules, proteins, cytokines, antibodies, bacterial antigens, and enzymes. Host cell—derived enzymes such as matrix metalloproteinases (MMPs) are an important group of neutral proteinases implicated in the destructive process of periodontal disease

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that can be measured in GCF .Porphyromonas gingivalis produces by far the greatest proteolytic activity through peptidases, elastases, trypsin like proteases, and collagenases that can be monitored by GCF analysis.In summary, GCF carries multiple molecular factors derived from the host response and is considered a significant protective mechanism in periodontal infection. These host response factors represent important mediators that can aid in the development of periodontal diagnostics.

Review of literature

The levels of several cytokines such as IL-1, IL-2, IL-6, IL-8 and TNF-α have been observed in the GCF of patients with periodontal disease (Lee et al. 1995, Tsai et al. 1995[8],Wilson et al. 1996, Hirose et al. 1997, Giannopoulou et al. 2003)[9].

Shsshik et al. (1991), Preiss et al. (1994)[10],Zhong et al. (2007)[11],that have clearly demonstrated a significant correlation between the level of IL-1β in GCF and clinical parameters such as PI, GI, and PD and also BL .

Teles et al. showed that aggressive periodontitis subjects were characterized by a higher IL-1β/IL-10 ratio than periodontally healthy subjects, suggesting an imbalance between pro- and anti-inflammatory cytokines in aggressive periodontitis[12].

CONCLUSION

Earlier studies did not provide any conclusive evidence about the level of interleukin-1 in smokers GCF .However, recent studies have provided that GCF interleukin 1 levels are higher in smokers with periodontitis .

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