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# Role of Interleukin 1-ALPHA in GCF with Aggressive Periodontitis

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## 1. Introduction

Periodontitis is an infectious disease which is characterized by the inflammation of periodontium in response to the bacterial flora in the oral cavity. Imbalance between the protective and destructive host mechanism which are initiated with infection is the reason for the tissue destruction in periodontitis[1]. Periodontal tissue damage is detected by two means – 1) probing which can measure the depth of the pocket and loss of attachment. 2) radiographs which shows alveolar bone loss[2]. Molecular messengers are the determinants of the efficient innate and adaptive immune response which helps in better understanding of interplay among the various components of the immune system[3]. Aggressive periodontitis occurs in patients who are clinically healthy otherwise, has features like rapid attachment loss, bone destruction and familial aggregation[4].

Among the three fluids in the oral cavity – saliva, GCF and serum, saliva and GCF are the most commonly used fluids for diagnostic purposes because of their ease of collection and minimally invasive collecting procedure. GCF is an inflammatory exudate that seeps in to the gingival crevices or the periodontal pockets around the tooth with gingival inflammation[5]. It contains a rich array of cellular and biochemical factors which helps in indicating the metabolic status of various components of periodontium. They are composed of enzymes and locally generated materials like tissue breakdown products, antibodies against dental plaque bacteria and inflammatory mediators[6].

## 2. Components of GCF

GCF is a transudate starting from the gingival plexus of veins in the gingival connective tissue, near the epithelium coating of the dentogingival space[7]. It consist of cells and microorganisms in the microbial dental plaque[8]. GCF gives a precise portrayal of tissue and serum concentrations[9]. Up to now, no less than 90 distinct segments in GCF have been assessed as conceivable biomarkers for conclusion of periodontal sickness [10].

Components of GCF are of three groups and they are the markers for the progression of periodontitis. They are
1) Host derived enzymes and inhibitors.
2) Tissue breakdown products.
3) Inflammatory mediators[11].

Collection of GCF is a minimally invasive procedure and its analysis provides quantitative biochemical indicators which are used for the evaluation of local cellular metabolism reflected in the periodontal health status[12]. A considerable number of studies all through the 1980s and the 1990s investigated the prescient capacity of GCF segments for distinguishing proof of progressive periodontal lesions[13]. While individual GCF segments delivered positive predictive qualities that were better than individual clinical measures, these reviews concentrated generally on the expectation of periodontitis at the site level as opposed to the recognizable proof of high-hazard gatherings and people[14]. An early multi-focus study by Lamster et al. (1995) examined the prescient estimation of b-glucuronidase (bG) at the patient level, in a population of patients, and showed that subjects with persistently elevated levels of GCF bG at pattern, 2-week and 3-month reviews had in the vicinity of 7 and 14 times expanded hazard proportion for periodontitis progression[15].

Curtis et al, stated that the markers of disease encloses three groups,
1) Indicators of current disease.
2) Predictors of future disease initiation.
3) Predictors of future disease progression[16].

## 3. Cytokines

Cytokines are small glycoproteins produced bynumber of cells, predominantly lymphocytes that helps in regulation of immunity, inflammation and hematopoiesis. Interleukin-1,interferon and nerve growth factors are the early discovered cytokines. They are released during non-immune events and a play a role which is not related to immune response in many tissues[17].

Interleukin 1 is responsible for inducing fever, damage to joins and regulation of bone marrow cells and lymphocytes. There are two groups of proteins – IL-1 alpha and IL-1 beta. In IL-1, terms from IL-1F1 to IL-1F10[18].

## 4. Mechanism

Matrix metalloproteinase (MMP) are known zinc and calcium dependent neutral endopeptidaseenzymes which are important in resorption of extracellular matrix in both normal and pathological state[19]. Collagen degradation is associated with the initiation of MMP secretion during physiological and pathological events like wound healing, bone remodeling, tumor invasion and metastasis[20]. Plasmin is one of the physiological MMP activator which acts as a pro-inflammatory agents that helps in inducing neutrophils

aggregation, platelet degranulation and stimulate the release of the inflammatory cytokine[21]. Plasmin stimulates secretion of pro-inflammatory cytokines such as tumor necrosis factor-alpha, IL-1 alpha and IL-1 beta[22].

**ROLE OF IL-1 IN AGGRESSIVE PERIODONTITIS:**

***M Suzuki et al, 2008*** stated the Interleukin (IL)-1 is closely related to the initiation and progression of periodontal disease. IL-1 levels in the gingival crevicular fluid (GCF) of subjects with periodontitis are higher than those in periodontally healthy controls, and the levels of IL-1 correlate with disease severity[23].

## 5. Review of the Literature

There is no article reviewing the role of interleukin 1 alpha in GCF with aggressive periodontitis.

***Wilton, J. M. A. et al (1992)***

The results indicate that IL-1 is present in the GCF from a proportion of sites with evidence of previous periodontal destruction[24].

***Toker et al. (2008)***

Stated that Interleukin 1 beta is high and decreased after SRP.[25]

***Rescala et al. (2010)***

Concluded that Interleukin 1 beta,elastase were higher in deep sites than shallow sites in periodontitis groups[26].

***SiamakYaghobee (2013)***

The findings of the present study indicated that the level of IL-1β may be an important supplement to clinical findings in measuring the health status of gingival or peri-implant tissues[27].

## 6. Discussion/Conclusion

Generous changes have been made in the comprehension in the pathogenesis, and progression of periodontitis. Assessment of the markers in GCF is viewed as a decent technique in the assurance of a man's hazard for periodontal disease. In GCF the concentration of interleukin- 1β was higher in chronic periodontitis group which acts as a diagnostic marker and gives information about the progression of periodontal disease. Therefore, interleukin 1 beta plays a major role in aggressive periodontitis than interleukin 1 alpha .

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