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Commentary

Levels of GCF IL 35 as a Diagnostic Marker for Site Evaluation of Periodontal Disease

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DESCRIPTION

A chronic inflammatory condition called periodontitis is a major factor in the degeneration of the tissues that support teeth. Periodontal disease rarely advances and does so episodically in most people. Healthy gingiva changes from gingivitis to periodontitis throughout the progression of periodontal disease. Periodontitis affects some people more than others. Recurrent periodontal disorders affect 30% of cases. Therefore, diligent patient observation is required to spot problem areas before more tissue damage occurs. To distinguish between disease locations that are active and inactive at the moment, clinical, microbiologic, and radiographic data are employed to identify early symptoms of disease activity. All of these diagnostic techniques have one drawback, though, which is that it is impossible to predict when healthy gingiva will turn into gingivitis and when gingivitis will turn into periodontitis. This hinders efforts to diagnose the illness and forecast future disease locations from both the clinical and research perspectives. Therefore, a problem unique to the discipline of periodontics is the difficulty to spot areas that are already degrading and at risk of being destroyed in the future as well as patients who are particularly vulnerable.

Changes in the immune system carried by periodontitis include the infiltration of the tissues by neutrophils, macrophages, B cells, and T cells as well as the local production of high concentrations of cytokines and other mediators. The soluble proteins known as cytokines attach to particular receptors on target cells to initiate and stop intracellular signaling cascades. T cells contribute significantly to the control of the immune response at the site of inflammation by secreting both pro and anti-inflammatory cytokines. In numerous investigations, the role of cytokines in periodontitis has been examined. When it comes to promoting osteoclast activation, cytokines including GMCSF, IL-1 beta, IL-6, IL-17, and TNF-alpha are among the more crucial pro inflammatory mediators.

However, a significant gap exists in this field because the majority of studies have focused on anti-inflammatory cytokines that control inflammation by acting on Th1 and Th2 cells (IL-4, IL-10, IL13 etc.) and there are very few research focused on how anti-inflammatory interleukins affect Treg cells (IL-35) Finding clinical diagnostic and prognostic cutoff values for interleukins is problem number. Future research is warranted given the paucity of previous studies in this field in order to confirm diagnostic and prognostic recommendations and investigate recommendations for unstudied interleukin disease connections.

Interleukin-12 (IL-12) is one of the cytokines that seems to support the inflammatory response in a variety of physiological and pathological processes. IL-12, IL-23, IL-27, and IL-35.4 make up the IL-12 family, which is evolutionarily related to the IL-6 cytokine superfamily. The cytokines that are linked to IL-12 are heterodimeric proteins made up of a p19, p28, or p35 chain (p40 or Ebi3). A novel anti-inflammatory cytokine, IL-35 is made up of the IL-27 chain Ebi3 and the IL-12 chain p35. It is the newest member of the IL-12 family. The most potent suppressive cytokine produced by Treg cell populations is IL-35, which is an efficient inhibitory cytokine. T cell proliferation is inhibited by IL-35 by stopping mitosis in the G1 phase without causing apoptosis. Treg cell subsets known as iTr35 cells can form when IL-35 is present. Target T cells that have been repressed are converted into iTr35 cells by inhibition mediated by n Treg cells, which supports the regulatory environment in inflammatory areas. Additionally, several investigations have shown that IL-35 appears to play an immunological modulatory role in a variety of illness states. High inflammatory areas and as a powerful stimulator of nTreg cells are where IL-35 can be most useful.

The process to collect GCF is minimally intrusive, and the examination of particular GCF ingredients provides quantitative cellular metabolism that indicates the state of the periodontium. As a result, efforts to create novel diagnostic methods have largely concentrated on Gingival Crevicular Fluid (GCF). Studies on GCF composition have raised the likelihood of developing trustworthy diagnostic tests that could circumvent the current clinical constraints by providing important information for a better understanding of disease process. Due

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to the analysis of immune components, connective tissue degradation products, acute phase proteins, and other inflammatory mediators inside GCF for these purposes, it would seem more acceptable to design biochemical diagnostic tests based on the presence of GCF.