Harnessing the Power of the Immune System to Treat Periodontal Disease

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Periodontal disease is characterized by destructive inflammation of the periodontium. Recent data in the literature has provided clues toward understanding how inflammation is involved with disease progression, leading to tissue destruction and bone resorption. It is thought that the pattern of chemokines expressed in periodontal tissue may determine the nature of leukocytes that migrate into the gingivae, and in turn, determine the pattern of cytokines produced in the periodontal environment. Ultimately, the overall balance between pro- and anti-inflammatory cytokines determines the severity of periodontitis through the modulation of the balance between the osteoclastogenic factor RANKL and OPG, its endogenous inhibitor. Our team’s objective is to understand and utilize the inflammatory and regulatory processes of the periodontium toward therapies that address the physiological cause of disease progression, namely superfluous host-response. We have focused on developing controlled release formulations to regulate the harmful inflammatory responses. Our strategy has initially targeted the release of CCL22 to recruit regulatory T-cells and more recently we have targeted other pathways of the inflammatory pathway. This data relating to regulation of both inflammation and the immune response will be presented highlighting these approaches as potential therapies for periodontal disease.