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Editorial

Periodontal Inflammation

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The etiology of periodontal diseases is host response against dental biofilm. The human oral cavity harbors a substantial and continuously evolving load of microbial species. The ecological interactions between the host and microbes determine the severity of the disease. Unlike many infectious diseases, periodontal diseases appear to be infections mediated by the overgrowth of commensal organisms, rather than by the acquisition of an exogenous pathogen. As microorganisms evolve more rapidly than their mammalian hosts, immune mechanisms that determine the ecological balance of commensal organisms also need to change to preserve homeostasis.

The pathogenesis of periodontal diseases is mediated by the inflammatory response to bacteria in the dental biofilm. The presence of these microorganisms in individuals with no evidence of disease progression suggests that the disease is the net effect of the immune response and the inflammatory processes, not the mere presence of the bacteria. Regulation of immune–inflammatory mechanisms governs patient susceptibility and is modified by environmental factors.

In susceptible individuals, periodontal inflammation fails to resolve and chronic inflammation becomes the periodontal pathology. Periodontal disease results from excess inflammation and may be considered a failure of resolution pathways. Inadequate resolution and failure to return tissue to homeostasis results in neutrophil mediated destruction and chronic inflammation, with destruction of both extracellular matrix and bone, and scarring and fibrosis. Scarring and fibrosis in periodontitis prevent the return to homeostasis.

Physiologic inflammation is a well-orchestrated network of cells, mediators and tissues. It is very important to consider the inflammatory/immune response as a whole, rather than many different modules working separately. As disease appears to be the result of loss of regulation and a failure to return to homeostasis, it is important to achieve a more complete understanding of the molecular and cellular events in this complex system. The paradigm shift in our understanding of inflammatory disease, such as periodontitis, is that resolution of inflammation is an active, rather than a passive, process that activates specific biochemical programs of resolution. It is increasingly evident that future treatment modalities of periodontal infections and periodontal surgical patients will rely on clinicians having a detailed map and molecular appreciation of the resolution programs for inflammation and tissue injury.

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