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Editorial :

HOST MODULATION

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Specific microorganisms initiate the immunoinflammatory processes that destroy tissue in periodontitis. Recent work has demonstrated, in addition to bacterial control, that modulation of the host immunoinflammatory response is also capable of controlling periodontitis. Matrix metalloproteinases (MMPs) destroy collagen and other matrix components, and the osteoclastic bone remodeling determines the periodontal bone response to a bacterial challenge. Other components of the biology, including cytokines and prostanoids, regulate MMPs and bone remodeling and are also involved in regulating the production of defensive elements, such as antibody. Agents directed at blocking MMPs or osteoclastic activity is effective in reducing periodontitis. Agents that inhibit prostaglandin E2 and selective blockage of specific cytokines have also been effective. Improved knowledge of bacterium- host interactions and of the processes leading to tissue destruction will help to identify targets for host modulation to reduce periodontitis in selected situations.

Periodontal diseases are initiated by Gram-negative tooth associated microbial biofilms that elicit a host response, with resultant osseous and soft tissue destruction. In response to endotoxins derived from periodontal pathogens, several osteoclast- related mediators target the destruction of alveolar bone and supporting connective tissues. Major drivers of this aggressive tissue destruction are matrix metalloproteinases (MMPs), cathepsins, and other osteoclast-derived enzymes.

In periodontal diseases, bacteria trigger inflammatory host responses which, along with the direct destructive effects of the bacteria, cause most of the tissue destruction. Periodontal inflammatory responses are, by and large, immunologic, and our understanding of these reactions has been advanced by the explosion of knowledge in immunobiology. Understanding the role of immune cells and their regulatory cell surface molecules such as the MHC, CD antigens, and receptors, as well as knowledge of effector systems set into motion such as phagocytes and cytotoxic T-cells, and the effector molecules such as antibodies, complement, and cytokines, have led to better understanding of the complex

pathogenesis of periodontal disease. The role of mediators including the matrix metalloproteinases, proteoglycans, the kinins and anaphylatoxins, and low molecular weight mediators including products of arachidonic metabolism is beginning to be elucidated in periodontal disease.

Important avenues of research for development of diagnostic tests based upon host response are apparent. For example, tissue products released during periodontal inflammation including the metalloproteinases, elastase, cytokines, Prostaglandins, antibodies, and complement components may provide the basis for future diagnostic indicator tests.

Host modulation has emerged as a valid treatment concept for the management of periodontal disease. Various host modulating agents have been investigated for prevention and treatment of periodontal disease. Sub-Antimicrobial dose doxycycline is the only agent which is at present recommended by FDA and marketed for the purpose of host modulation in management of periodontal disease.

Use of systemic HMTs for treatment of patients' periodontal condition may also provide benefits for other inflammatory disorders, such as arthritis, cardiovascular disease, dermatologic conditions, diabetes and osteoporosis. Also, patients who are currently taking host modulatory agents such as NSAIDs, bisphosphonates or tetracyclines, as well as newer agents targeting specific cytokines for the management of medical conditions, may be experiencing periodontal benefits from these systemic medications.

As for now, HMT is not a routinely used treatment modality but it is surely going to be a treatment concept where natural pathways of resolution of inflammation can be used to limit inflammation and promote healing and regeneration with minor risk of side effects.

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