Regulation of bone resorption factors by oral bacterial biofilms

Summary / Zusammenfassung
The perspective of this project is the elucidation of pathogenic mechanisms involved in various forms of periodontitis, a disease that destroys the tooth-supporting (periodontal tissues) as a result of chronic inflammation. This host inflammatory response is caused by bacteria that attach on the tooth surfaces in the form of polymicrobial biofilm communities. The major trait of periodontitis is the destruction of the alveolar bone supporting the teeth, eventually leading to tooth loss. The aim of these in vitro studies is to investigate the effects of oral bacterial biofilms on the expression of molecules that regulate bone resorption in a number of cell types with potential relevance to periodontitis. The RANKL-OPG bimolecular system is a major regulator of bone resorption. RANKL, a member of the TNF-ligand superfamily, is responsible for triggering the differentiation of pre-osteoclasts (cells of the monocyte/macrophage lineage), into mature osteoclasts that are responsible for resorbing bone. The action of RANKL can be blocked by its soluble decoy receptor OPG, a member of the TNF-receptor superfamily. OPG prevents the interaction of RANKL with pre-osteoclasts, thus inhibiting osteoclast differentiation and bone resorption. An elevated RANKL/OPG ratio is considered indicative of enhanced capacity of a tissue or cell to stimulate bone resorption. The major focus of these studies is how oral biofilms regulate the expression of the RANKL-OPG system in host cells.

Publications / Publikationen

Keywords / Suchbegriffe
Periodontitis, oral biofilms, bone resorption, RANKL-OPG, prostaglandin E2, interleukin-6

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