Cellular immunity of periodontitis and OPG/RANKL/RANK system.

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Abstract

A new era in the field of bone biology has been explored during the last decade. The identification and characterization of OPG/RANKLIRANK system have also played an important role in the landscape of bone biology. Osteoblasts may express RANKL on cell surface and regulate the differentiation and activation of osteoclasts through cell receptor RANK by cell to cell contact. On the other hand, osteoblasts may also express OPG to bind RANKL in an autocrine modulation and hinder osteoclastogenesis. In the study of functional human T-cell immunity and RANKL controlled bone resorption, it implies that the induction of alveolar bone resorption in periodontitis is triggered by exotoxin or endotoxin of microorganism through the RANKL expression on CD4+ T cells and the consequent activation of osteoclasts. On the contrary, the CD8+T cells and gingival fibroblast may express OPG to conjugate RANKL and inhibit osteoclastogenesis. The explosion in the field of unraveling OPG/RANKL/RANK system will high-light a complete understanding of the relation between osteoc1astogenesis and periodontal destruction. In addition to providing fundamental insight in bone biology, the detailed characterization of OPG/RANKL/RANK system will open up entirely new areas for clinical research and therapeutic approach in periodontology.