Effects of nicotine and lipopolysaccharide on the expression of MMPs, PAs, and their inhibitors in human osteoblasts

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Objective: Lipopolysaccharide (LPS) from periodontopathic bacteria can initiate alveolar bone loss through the induction of host-derived cytokines. Smoking increases the risk and severity of periodontitis. We examined the effects of nicotine and LPS on the expression of matrix metalloproteinases (MMPs), plasminogen activators (PAs), and their inhibitors, including tissue inhibitors of metalloproteinases (TIMPs) and PA inhibitor-1 (PAI-1), in human osteoblasts.

Methods: The cells were cultured with or without nicotine and/or LPS for 12 days in the presence of either nicotine or LPS for 12 days with 4% paraformaldehyde and decalcified, immunostaining of collagen I andⅧ, matrix metalloproteinase (MMP)-8 and -13 was evaluated by in situ hybridization or immunohistochemistry. Total RNA was extracted from the articular cartilage and expression levels of these mRNA were measured by quantitative PCR.

Results: Expression of collagen II and MMP-8 was decreased after 3 days in the three areas, but increased after 2 weeks at hypertrophic differentiated chondrocytes in the transitional area. Immunostaining of collagen II at the transitional and contact areas was decreased. Immunostaining of collagen I was increased at hypertrophic differentiated chondrocytes in the transitional area and superficial chondrocytes in the non-contact area. Immunostaining of MMP-13 was observed at the hypertrophic differentiated chondrocytes in the transitional area. Expression levels of collagen II mRNA was decreased, however, MMP-8 and -13 mRNA was increased by quantitative PCR.

Conclusions: The mechanism of the articular cartilage degeneration after immobilization differs at the three specific areas [2, 3].

REFERENCES