B-6. MMP-13 regulation in rat periodontal ligament cell

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Background

Periodontal inflammation characterized by collagen degradation results in attachment loss around tooth. Matrix metalloproteinases (MMPs) play an important role both in the maintenance and degradation of extracellular matrix within the periodontium. MMP-13 has been shown to be positively correlated with periodontal disease. Mitogen activated protein (MAP) Kinases play an important role in the induction of proteins associated with inflammatory response such as IL-1β and TNF-α. This study is accomplished as an initial step towards a better understanding of the role of MMP-13 in the pathogenesis of periodontal disease and possible regulation of MMP-13 through MAP Kinase signaling by different stimuli. MMP-13 mRNA expression in PDL cells was determined by semi-quantitative RT-PCR and MAP Kinases expression were determined by western blot.

Results

In semi-quantitive RT-PCR analysis, IL-1β, TNF-α, and PTH-induced MMP-13 expression was increased to 320%, 180%, 380%, respectively. But rh BMP-7 had little effect on MMP-13 expression (120%). When PDL cells were pre-treated with SB203580, the p38 inhibitor, IL-1β-induced MMP-13 mRNA level was reduced by ~40% in compared to IL-1β-treated cells only. There was no effect on PTH pre-treated cells. IL-1βincreased the half-life of MMP-13 mRNA with ~120 minutes, but in the presence of SB203580, the half life was decreased to ~60 minutes following normalization to GAPDH. In western blot analysis, IL-1β activates both phosphorylated form of p38 MAP Kinase and JNK, but not of ERK. Neither BMP-7 nor PTH activates p38, JNK, and ERK MAP Kinase. IL-1β-induced p38 MAP Kinase phosphorylation is inhibited by SB203580.

Conclusions

This results suggest that p38 MAP Kinase pathway would be a central signalling pathway controlling MMP-13 expression induced by IL-1β.