R-17. The role of p 38 MAP Kinase on RANKL regulation in mouse pereiodontal ligament fibroblasts

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background

The signal transduction systems involved in mediating the actions of hormones and cytokines involved with RANKL production have been studied. Understanding these pathways is essential to aid in generating therapeutics that can treat disease mediated by RANKL. So the purpose of this study focused on the role of MAP kinase in IL-1β-induced RANKL expression.

results & conclusion

Soluble RANKL (sRANKL) production was dose-dependently inhibited by SB203580, p38 MAP kinase inhibitor when stimulated with IL-1β. sRANKL production was not inhibited with SP600125, PD98059, and NF-kB inhibitor. IL-1β increased steady-state RANKL mRNA levels in mPDL cells. However, p38 MAP kinase inhibitor decreased steady-state RANKL mRNA levels in mPDL cells. In addition, inhibitory effects of RANKL mRNA required de novo protein synthesis. p38 MAP kinase inhibitor accelerated RANKL mRNA rate of decay in mPDL cells.

These results suggest that p38 MAP kinase regulate IL-1β-stimulated RANKL in mouse periodontal ligament cells and would be provide therapeutic targets in the management of periodontitis.