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TGF-β1 stimulates collagen content, pro-collagen I and TIMP-1 production of human dental pulp cells is regulated by MEK/ERK and ALK5/Smad Signaling

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Introduction: In order to clarify the role of TGF-β1 in pulp repair/regeneration, we investigated the differential signaling pathways responsible for effects of TGF-β1 on collagen turnover, metalloproteinase-3 (MMP-3) and tissue inhibitor metalloproteinase-1 (TIMP-1) production in human dental pulp cells.

Materials and Methods: Pulp cells were exposed to TGF-β1 with/without pretreatment and co-incubation by U0126 (a MEK/ERK inhibitor) and SB431542 (an ALK5/Smad signaling inhibitor). Sircol collagen assay was used to measure cellular collagen content. Culture medium pro-collagen I, TIMP-1 and MMP-3 levels were determined by enzyme-linked immunosorbant assay (ELISA). Phosphorylation of Smad2 and ERK1/2 were analyzed by western blotting or Pathscan p-ELISA.

Results: TGF-β1 increased the collagen content, pro-collagen I and TIMP-1 production, but slightly decreased MMP-3 production of pulp cells. TGF-β1 stimulated ERK1/2 and Smad2 phosphorylation within 60 min of exposure. SB431542 and U0126 prevented the TGF-β1-induced increase of collagen content and TIMP-1 production of dental pulp cells.

Conclusions: These results indicated that TGF-β1 may be involved in the healing/regeneration processes of dental pulp in response to injury by stimulation of collagen and TIMP-1 production. These events are associated with ALK5/Smad2/3 and MEK/ERK signaling. (This study is supported by National Science Council, Taipei, Taiwan, ROC and Chang Gung Memorial Hospital)

Biography

Me-Chi Chang received his master and Ph.D degree in Department of Pharmacology, National Taiwan University Medical College (1995). Currently he is a professor, researcher and teaching staff in the Chang Gung University of Science and Technology. He had published many papers in scientific journals including Blood, Thrombosis & Haemostasis, BBA, Biomaterials, Acta Biomaterialia, J Endod, Int Endod J, Carcinogenesis etc. he also served as a reviewer and editorial board member of many journals such as Toxicology, J Dent Res, Carcinogenesis, Int Endod J, Austin Dent J.

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