

Actin alpha 2, smooth muscle, a transforming growth factor- β 1-induced factor, regulates collagen production in human periodontal ligament cells via Smad2/3 pathway

ファカタバ, ナーティ

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氏 名 : Fakatava Naati

論 文 名 : Actin alpha 2, smooth muscle, a transforming growth factor- β 1-induced factor, regulates collagen production in human periodontal ligament cells via Smad2/3 pathway
(Transforming growth factor- β 1 で誘導される Actin alpha2, smooth muscle は、Smad2/3 経路を介してヒト歯根膜細胞のコラーゲン産生を制御する)

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論 文 内 容 の 要 旨

Actin alpha 2, smooth muscle (ACTA2) is an actin isoform that forms the cytoskeleton. Actin plays a crucial role in numerous cellular functions. ACTA2 is a marker of functional periodontal ligament (PDL) fibroblasts and is upregulated by transforming growth factor- β 1 (TGF- β 1); however, the underlying function of ACTA2 in PDL tissue is unknown. We aimed to examine the localization and potential function of ACTA2 in PDL tissues and cells.

In this research, RNA expression was determined using semi-quantitative reverse transcription–polymerase chain reaction (RT-PCR) and quantitative RT-PCR. Protein expression was determined using immunofluorescence staining and western blot analysis. Small interfering RNA (siRNA) was used for knockdown assay to examine the effect of ACTA2 in human periodontal ligament cells. Cell proliferation was determined using WST-1 assay and migration was determined using cell migration assay. Soluble and insoluble collagen production was examined using the Sircol collagen assay and picrosirius red staining, respectively.

From the results, ACTA2 expression was observed in human primary PDL cells and PDL cell line, 2-23 cells. 2-23 cells treated with scramble siRNA exhibited a fibroblast-like morphology with a spindle-shaped cell form, while 2-23 cells treated with ACTA2 siRNA showed round appearance. ACTA2 knockdown also significantly suppressed the proliferation and migration of 2-23 cells. TGF- β 1 upregulated ACTA2, collagen type I alpha1 chain (COL1A1), periostin (POSTN), and fibrillin-I (FBN1) expression and soluble and insoluble collagen production in 2-23 cells. However, ACTA2 depletion by siRNA strongly suppressed PDL-related gene expression and soluble and insoluble collagen production compared with those of TGF- β 1–stimulated control cells. Furthermore, ACTA2 knockdown significantly suppressed the phosphorylation of Smad2 and Smad3.

In summary, ACTA2 is involved in the cell morphology, proliferation, and migration process in PDL cell. ACTA2 also plays a crucial role in PDL-related marker expression and collagen production via Smad2/3 phosphorylation. These results suggest that ACTA2 may be a critical factor involved in homeostatic maintenance of PDL tissue.