DERMATOLOGICAL SURGERY

KNOCKDOWN OF FOXM1 INHIBITS EXTRACELLULAR MATRIX EXPRESSION IN KELOID FIBROBLASTS VIA INHIBITION OF TRANSFORMING GROWTH FACTOR-β1/SMAD SIGNALING PATHWAY

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Keloid is a pathologic fibro-proliferative disease characterized by excessive extracellular matrix (ECM) component deposition and hyperproliferation of fibroblasts. Forkhead box M1 (FOXM1) has been implicated in the development of fibrosis. However, the role of FOXM1 in keloid remains unknown. Therefore, the aim of this study was to examine the effects of FOXM1 on keloid fibroblasts (KFs) and explored the underlying mechanism. Our results showed that the expression of FOXM1 was highly expressed in KFs. Knockdown of FOXM1 suppressed TGF-β1-induced KFs proliferation. Silencing FOXM1 also inhibited the expression of type I collagen, CTGF and α-smooth muscle actin (α-SMA) in human KFs induced by TGF-β1. Furthermore, knockdown of FOXM1 suppressed the expression of TGF-β1-induced Smad2 and Smad3 phosphorylation in human KFs. In conclusion, these findings suggest that silencing FOXM1 reduced ECM expression in KFs, at least in part, via inhibiting the TGF-β1/Smad signaling pathway. Thus, FOXM1 may represent a promising target for treatment of keloid.