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Anti-fibrotic effects of synthetic oligodeoxynucleotide for the regulation of TGF-β1 and Smad expression in kidney fibrosis

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Kidney fibrosis is a common process of various kidney diseases leading to end-stage renal failure irrespective of etiology. Myofibroblasts are crucial mediators in kidney fibrosis through production of extracellular matrix (ECM), but their origin has not been clearly identified. Many study proposed that epithelial and endothelial cells become myofibroblasts by epithelial dedifferentiation and endothelial-mesenchymal transition (EndoMT). TGF-β1/Smad signaling plays a crucial role in partly epithelial-mensencymal transition (EMT) and EndoMT. Thus, we designed the TGF-β1/Smad oligodeoxynucleotide (ODN), a synthetic short DNA containing complementary sequence for Smad transcription factor and TGF-β1 mRNA. Therefore, this study investigated the anti-fibrotic effect of synthetic TGF-β1/Smad ODN on UUO-induced kidney fibrosis in vivo model and TGF-β1-induced in vitro model. To examine the effect of TGF-β1/Smad ODN, we performed various experiments to evaluate kidney fibrosis. The results showed that UUO induced inflammation, ECM accumulation, epithelial dedifferentiation and EndoMT processes, and tubular atrophy. However, synthetic TGF-β1/Smad ODN significantly suppressed UUO-induced fibrosis. Furthermore, synthetic ODN attenuated TGF-β1-induced epithelial dedifferentiation and EndoMT program via blocking TGF-β1/Smad signaling. In conclusion, this study demonstrated that administration of synthetic TGF-β1/Smad ODN attenuates kidney fibrosis, epithelial dedifferentiation, and EndoMT processes. The findings propose the possibility of synthetic ODN as a new effective therapeutic tool for kidney fibrosis.