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Deletion of Exoc5 Exacerbates Kidney Fibrosis following Unilateral Ureteral Obstruction

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Objectives : Exoc5, a central component of exocyst complex, regulates cell differentiation, which is critical for the repair process after kidney injury. Here, we investigated the role of Exoc5 on kidney fibrosis, which is a major feature of impaired repair process and chronic kidney disease.
Methods : Kidney proximal tubule cell-specific Exoc5 deletion (PT–Exoc5^{KO}) mice were generated by crossing Exoc5$^{f/f}$ mice with PEPCK-Cre mice. Wild-type (PT–Exoc5^{WT}) and PT–Exoc5^{KO} mice were subjected to unilateral ureteral obstruction (UUO) for 7 days and the fibrosis of the kidney was evaluated. In HK-2 cells, cultured human proximal tubule cells, Exoc5 was down-regulated using siRNA, and the fibrosis was evaluated after TGF-β treatment.
Results : Exoc5 deletion did not cause functional and structural kidney damage. UUO led to kidney fibrosis with decreased Exoc5 expression and Exoc5 deletion exacerbates the fibrosis. UUO induced epithelial to mesenchymal transition (EMT) and paired box 2 (Pax2) expression in both groups of mice, with greater induction in PT–Exoc5^{KO} compared to PT–Exoc5^{WT} mice. Exoc5 deletion alone increased Yes-associated protein (YAP) expression in the kidney and exacerbated UUO-induced YAP activation compared to PT–Exoc5^{WT} mice. In HK-2 cells, human proximal tubule cells, Exoc5 knockdown increased YAP expression. Exoc5 knockdown augmented YAP activation and EMT following TGF-β treatment compared with control cells.
Conclusions : These findings demonstrate that Exoc5 deletion exacerbates kidney fibrosis and EMT, suggesting that Exoc5 may be a novel therapeutic strategy for kidney fibrosis.