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PTP4A1 Knockout Attenuates Renal Fibrosis in a Mouse Model of Unilateral Ureteral Obstruction

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Objectives : Inhibitors targeting protein tyrosine phosphatases (PTPs) have been investigated as potential anti-fibrotic agents. PTP4A1, a member of the prenylated PTP subfamily, is known for its role in promoting tumor cell growth and migration. However, its function in renal physiology remains largely unclear. This study aimed to evaluate whether PTP4A1 could be a therapeutic target for reducing renal fibrosis.
Methods : Ten-week-old male PTP4A1 knockout (KO) and wild-type mice were divided into four groups: wild-type, PTP4A1 KO, wild-type with unilateral ureteral obstruction (UUO), and PTP4A1 KO with UUO. Seven days after surgery, mice were sacrificed, and kidney tissues were collected for molecular analyses and histological examinations.
Results : PTP4A1 KO mice with UUO exhibited reduced renal tubulo-interstitial damage and fibrosis compared to wild-type UUO mice. The loss of PTP4A1 in UUO kidneys was associated with decreased renal expression of α-SMA and TGF-β compared to wild-type UUO mice. Additionally, while wild-type UUO kidneys showed reduced E-cadherin expression relative to sham mice, PTP4A1 KO UUO kidneys exhibited increased E-cadherin expression. In vitro, PTP4A1 silencing in TGF-β-treated HK2 cells led to enhanced E-cadherin expression and decreased phosphorylation of AKT and GSK3B.
Conclusions : PTP4A1 KO mitigates renal fibrosis in UUO-afflicted kidneys, suggesting that PTP4A1 may serve as a potential therapeutic target for renal fibrosis.