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RGS2, Regulator of G Protein Signaling 2 Inhibits the Progression of Fibrosis in Kidney

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Background: RGS2, a regulator of G protein signaling 2, is the most potent negative regulator of Gq protein-coupled receptor such as angiotensin II type 1 receptor which is well-known contributor in the progression of chronic kidney disease. However, the role of RGS2 in kidney fibrosis remains to be defined. Here, we investigated the role of RGS2 in renal interstitial fibrosis following unilateral ureteral obstruction (UUO).
Methods: RGS2 deficient (RGS2 -/-) and wild type (RGS+/+) mice were subjected to UUO or sham-operation and kidneys were harvested at 3 and 5 days after operation. Expression of RGS2 mRNA and protein was determined by RT-PCR and Western blot analysis, respectively. Angiotensin II type 1 receptor (AT1R) and α-smooth muscle actin (α-SMA) expressions, and collagen deposition were evaluated by Western blot analysis and trichrome staining, respectively.
Results: UUO elevated the levels of RGS2 mRNA and protein, α-SMA and collagen deposition in the kidney. RGS2 gene deletion accelerated these increases in the kidneys. Increases of AT1R expression after UUO were significantly higher in RGS2-/- than in RGS+/+ mice. Expression of PAI-1 and phosphorylated-ERK (p-ERK) was greater in the kidney of RGS2 -/- than in the kidney of RGS+/+ mice.
Conclusion: These results suggest that RGS2 mitigates the progression of kidney fibrosis, proposing that the possibility of RGS2 as a therapeutic target of chronic kidney disease.

Key Words: 콩팥 섬유화, 요관폐쇄, RGS2
Kidney fibrosis, Ureteral obstruction, RGS2