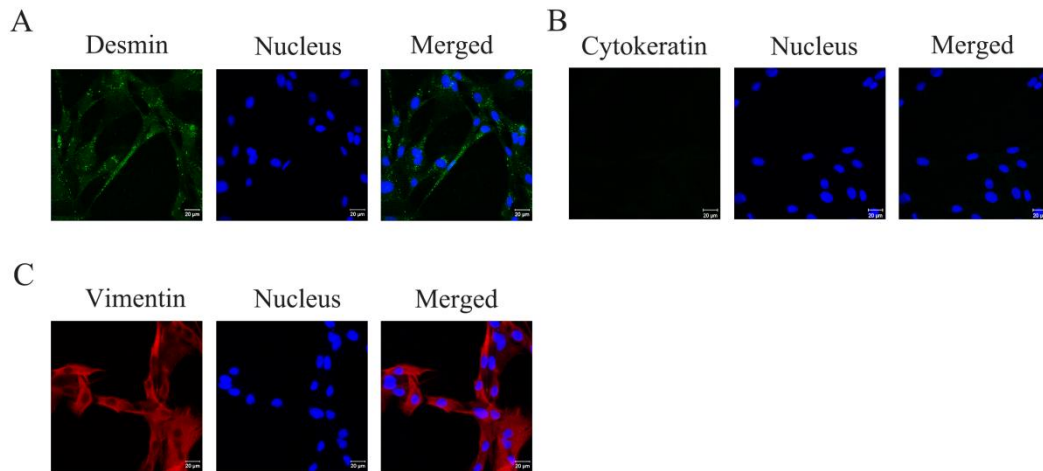
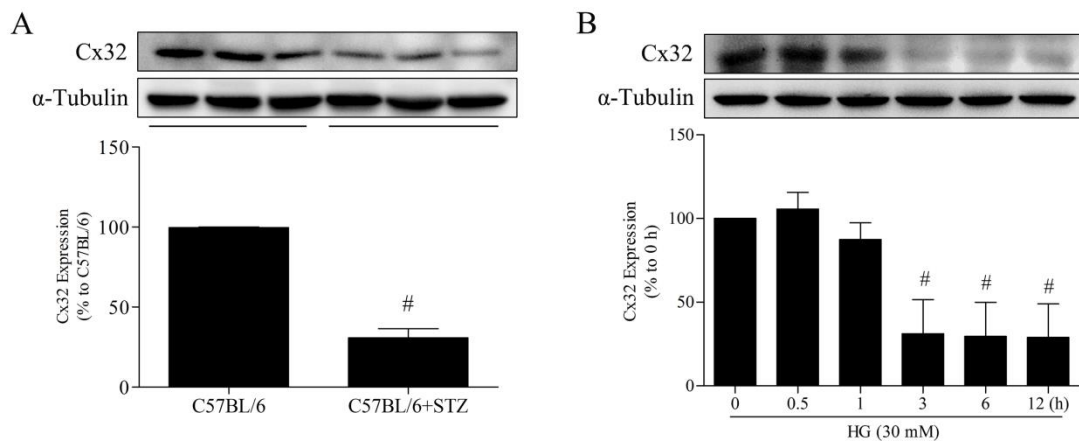


1 **Supporting Information**

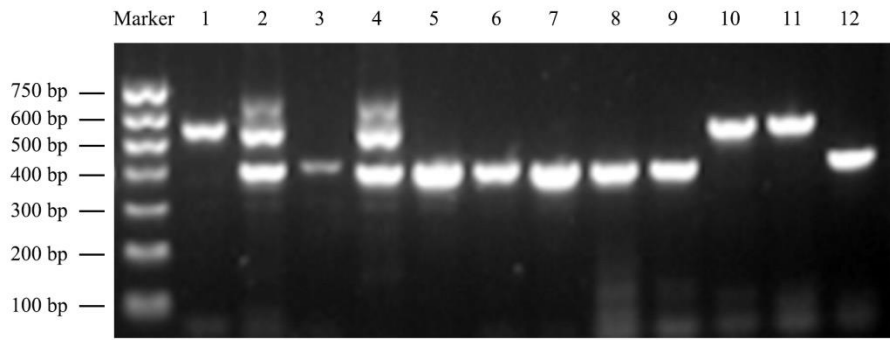


2 **Fig. S1. Immunofluorescence identification of primary GMCs.** Antibodies of
 3 Desmin (A) and Vimentin (C), markers of glomerular mesangial cells, as well as
 4 Cytokeratin (B), a marker of epithelial cells were used for the immunofluorescence
 5 assays.

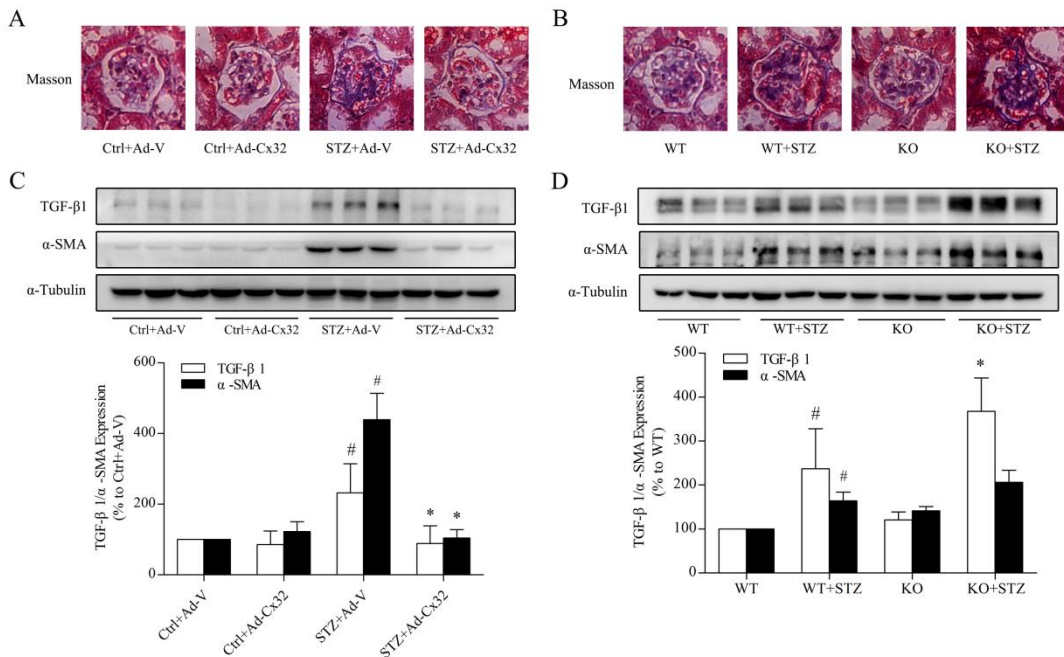


6 **Fig. S2. The expression of Cx32 is downregulated in the kidney of diabetic mice**
 7 **and HG-induced GMCs.** The expression of Cx32 was detected using western
 8 blotting analysis. (A) Cx32 expression in the kidney of STZ-induced diabetic mice. [#]*P*
 9 < 0.01 vs. C57BL/6, n = 5. (B) Cx32 expression in HG-induced GMCs. [#]*P* < 0.05 vs.
 10 0 h, n = 5. The data are presented as the means ± SD.

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15 **Fig. S3. Genotyping is performed by PCR analysis of DNA from mice tail-tips.**
 16 Primers used for detection of the Cx32 WT alleles generate a PCR product of 550
 17 base pairs. Primers used for detection of the Cx32 KO alleles generate a PCR product
 18 of 414 base pairs.
 19



20 **Fig. S4. The effects of Cx32 on renal fibrosis in STZ-induced diabetic mice.** (A
 21 and B) Glomerular histopathology in the kidney of diabetic mice was analyzed by
 22 masson staining (400 × magnification), blue indicates collagen fibers. (C)
 23 Overexpression of Cx32 reduced the expressions of TGF-β1 and α-SMA in the kidney
 24 of STZ-induced diabetic mice as shown by western blotting. [#]*P* < 0.05 vs. Ctrl + Ad-V;
 25 ^{*}*P* < 0.05 vs. STZ + Ad-V, n = 5. (D) Cx32 deficiency promoted the expressions of

- 26 TGF- β 1 and α -SMA in the kidney of STZ-induced diabetic mice. [#] $P < 0.05$ vs. WT;
- 27 ^{*} $P < 0.05$ vs. WT + STZ, n = 5. The data are presented as the means \pm SD.