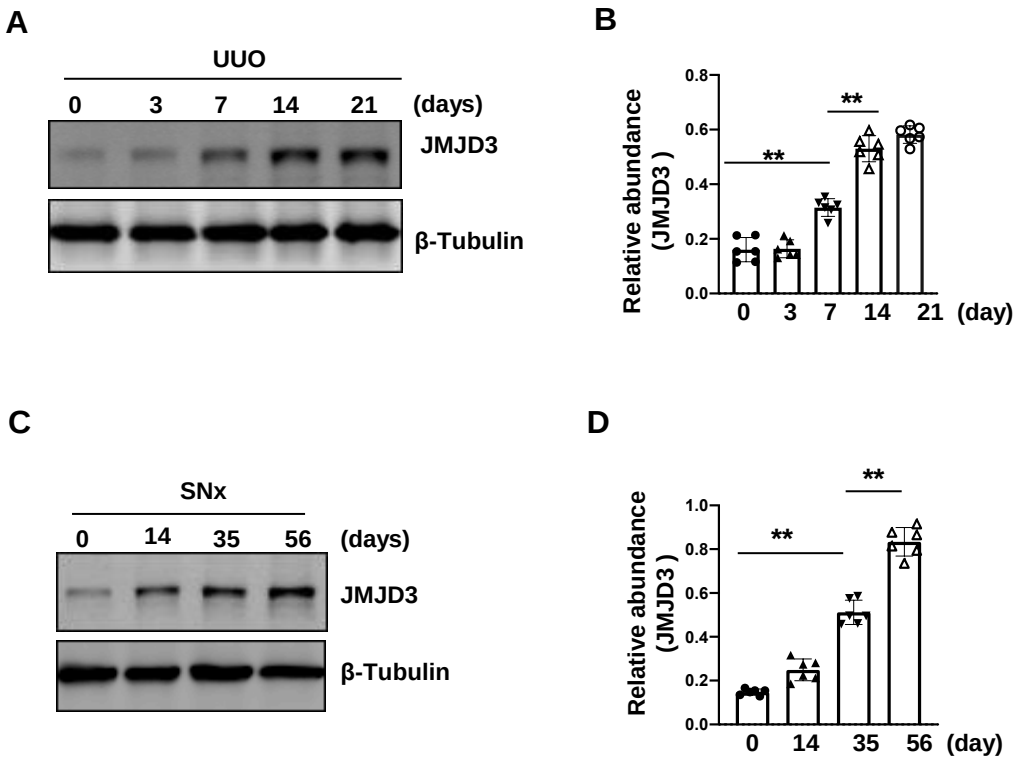


**Figure S1. The time course of UUO or 5/6 nephrectomy (SNx)-induced expression of JMJD3**. After UUO (A) or SNx (B), kidneys were collected at different time points as indicated. The whole kidney lysates were subject to immunoblot analysis with specific antibodies to JMJD3 or β-tubulin. Expression levels of JMJD3 and β-tubulin were quantified by densitometry analysis. (B, D) JMJD3 was normalized with β-tubulin. Values are the means ± sem of 6 samples. ***P*<0.01

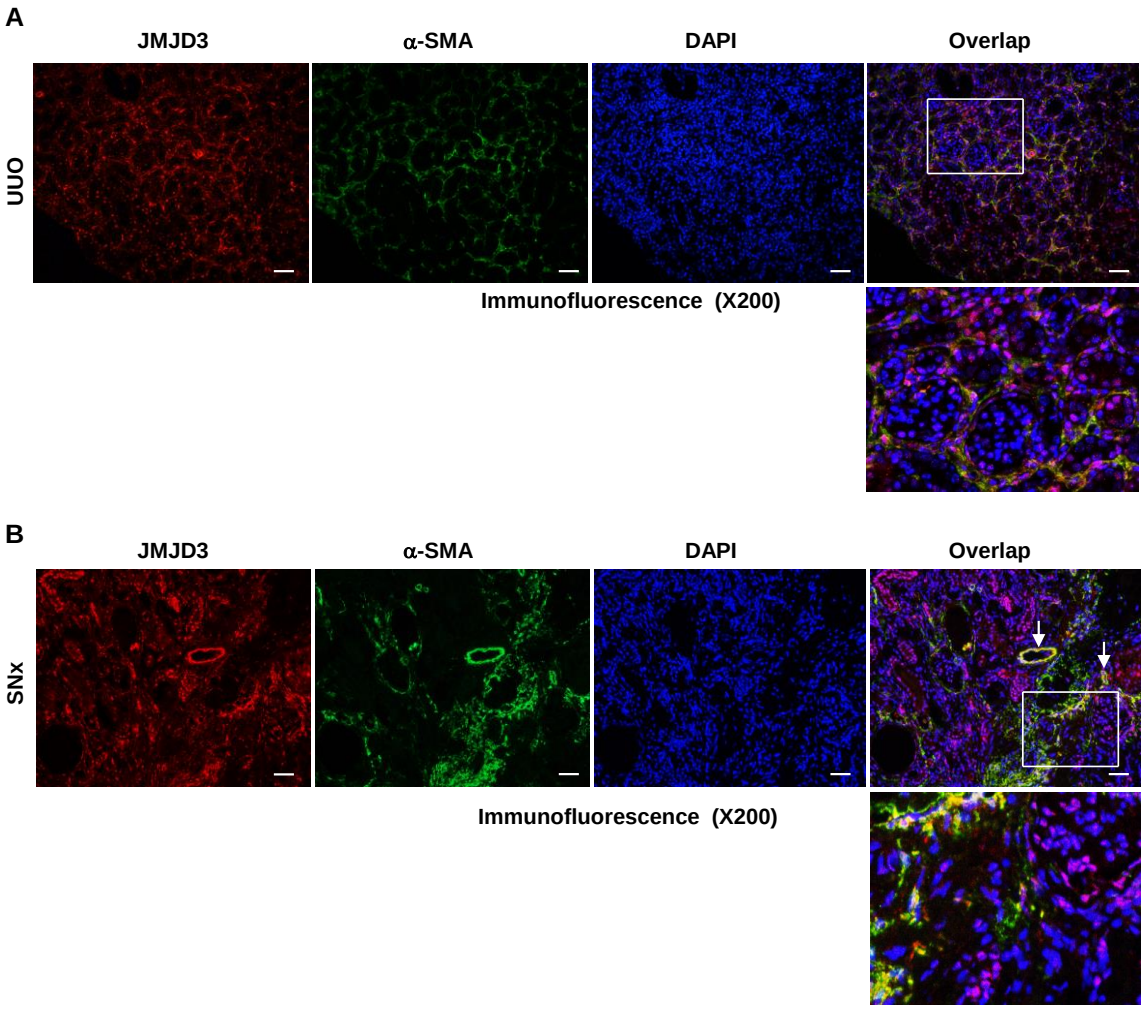


**Figure S2**. **Expression of JMJD3 in the kidney after UUO (A) and SNx (B).** The kidney was collected at 7 days after UUO or 56 days after SNx. Photomicrographs illustrating immunofluorecent co-staining of JMJD3 with α-SMA (original magnification, ×200). JMJD3 is expressed in the nucleus of both renal tubular cells and renal interstitial fibroblasts. JMJD3 was also co-stained with α-SMA (Yellow) in the cytosol of some interstitial fibroblasts and small arteries (white arrows), indicating its expression in the cytoplasm in this cell type and renal arteries. (Original magnification ×200)

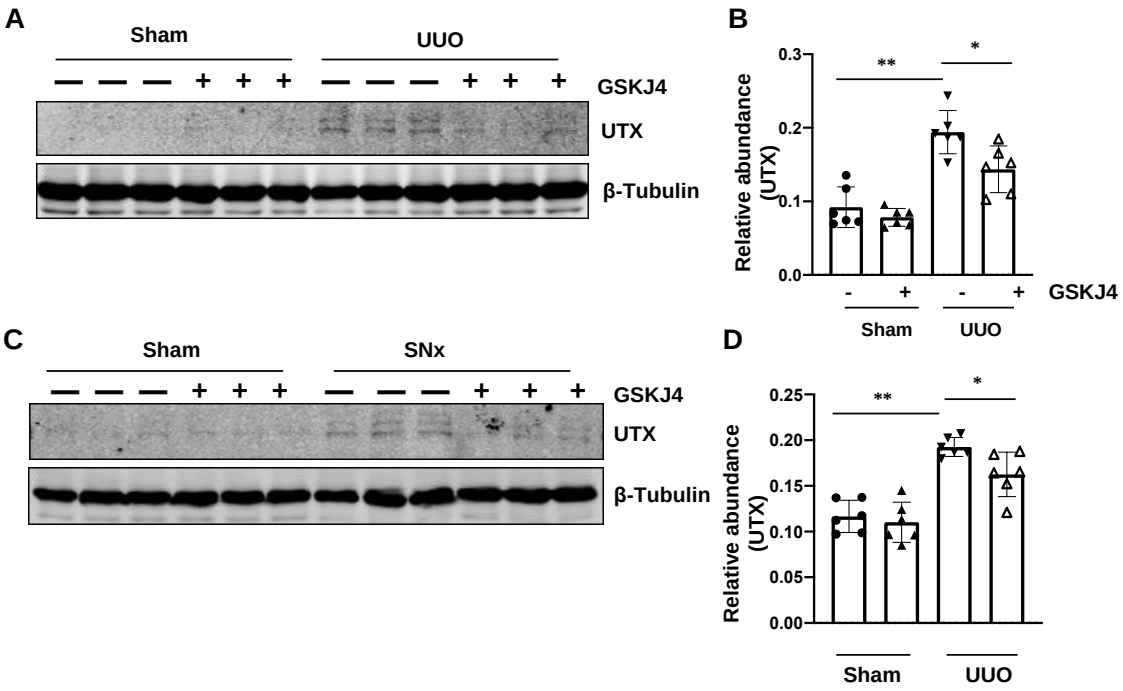


**Figure S3. Effect of GSKJ4 on the expression of UTX expression in the kidney after UUO and 5/6 nephrectomy (SNx).** The kidney tissue lysates from Sham-operated, UUO injured (UUO) (A) or remnant kidneys after surgery (SNx)(C) with and without administration of GSKJ4 were subjected to immunoblot analysis with specific antibodies to UTX or β-Tubulin. (B, D) Expression levels of UTX were quantified by densitometry analysis and then normalized with β-Tubuin. Values are the means ± sem of 6 samples. ***P*<0.01

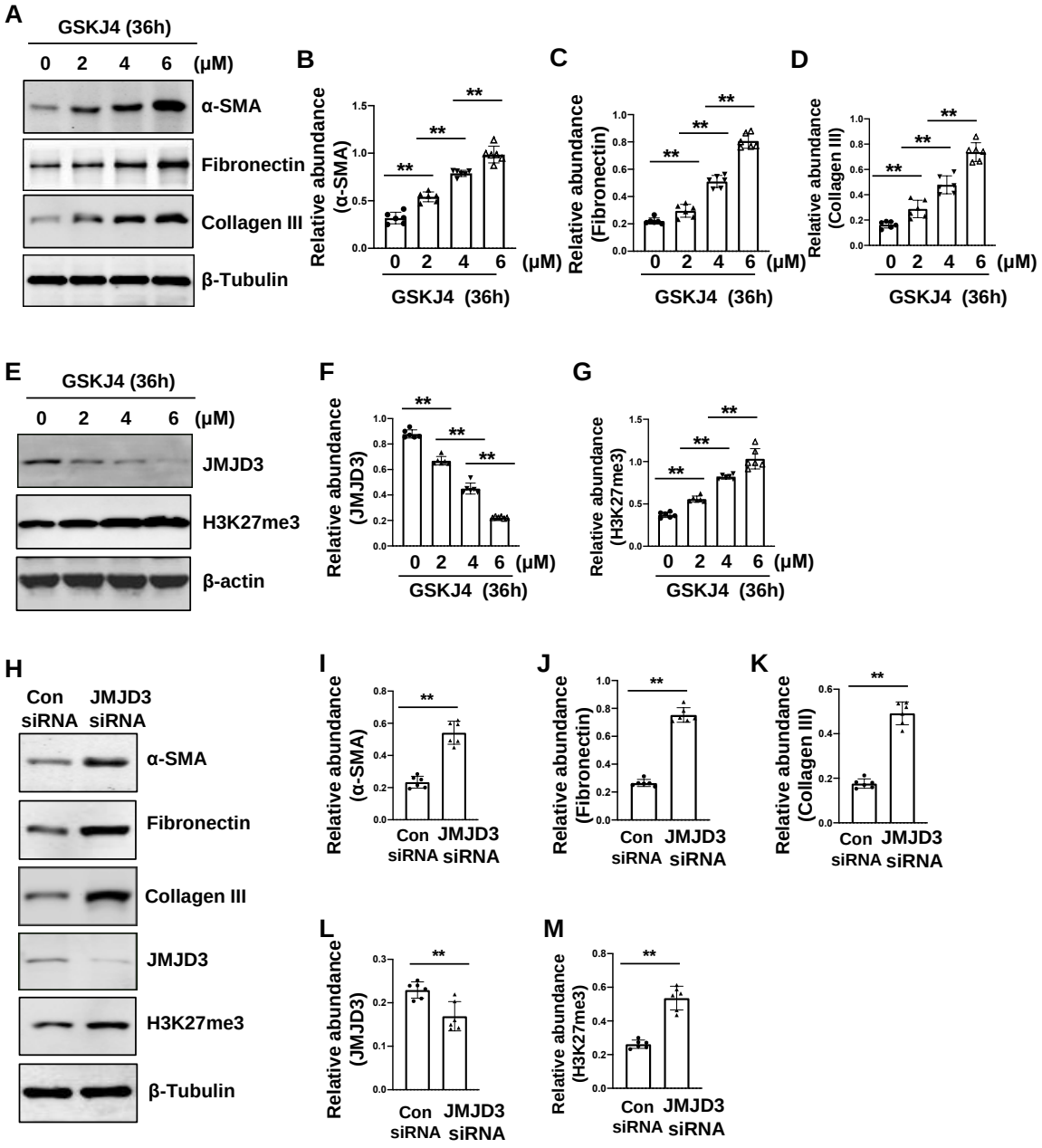


**Figure S4. Inhibition of JMJD3 by GSKJ4 or siRNA promotes activation of renal interstitial fibroblasts induced by serum.** (A, E) NRK-49F cells were incubated with medium containing 5 % serum in the presence or absence of GSKJ4 (0-6 μM) for 36 h. Cell lysates were prepared and subjected to immunoblot analysis with antibodies against α-SMA, fibronectin, collagen III, β-tubulin (A), JMJD3, H3K27me3, β-actin (E). Expression levels of α-SMA (B), fibronectin (C), collagen III (D), JMJD3 (F), H3K27me3 (G) were quantified by densitometry analysis and then normalized with β-tubulin or β-actin, respectively, as indicated. (H) NRK-49F cells were transfected with control siRNA or JMJD3 siRNA and then incubated with medium containing 0.5 % serum for 36 h. Cell lysates were prepared and subjected to immunoblot analysis with antibodies against α-SMA, fibronectin, collagen III, JMJD3, H3K27me3, β-tubulin. Expression levels of α-SMA (I), fibronectin (J), collagen III (K), JMJD3 (L), H3K27me3 (M), β-

tubulin were quantified by densitometry analysis and then normalized with β-tubulin. Values are the means ± sem of at ≥3 independent experiments. **P*<0.05; ***P*<0.01

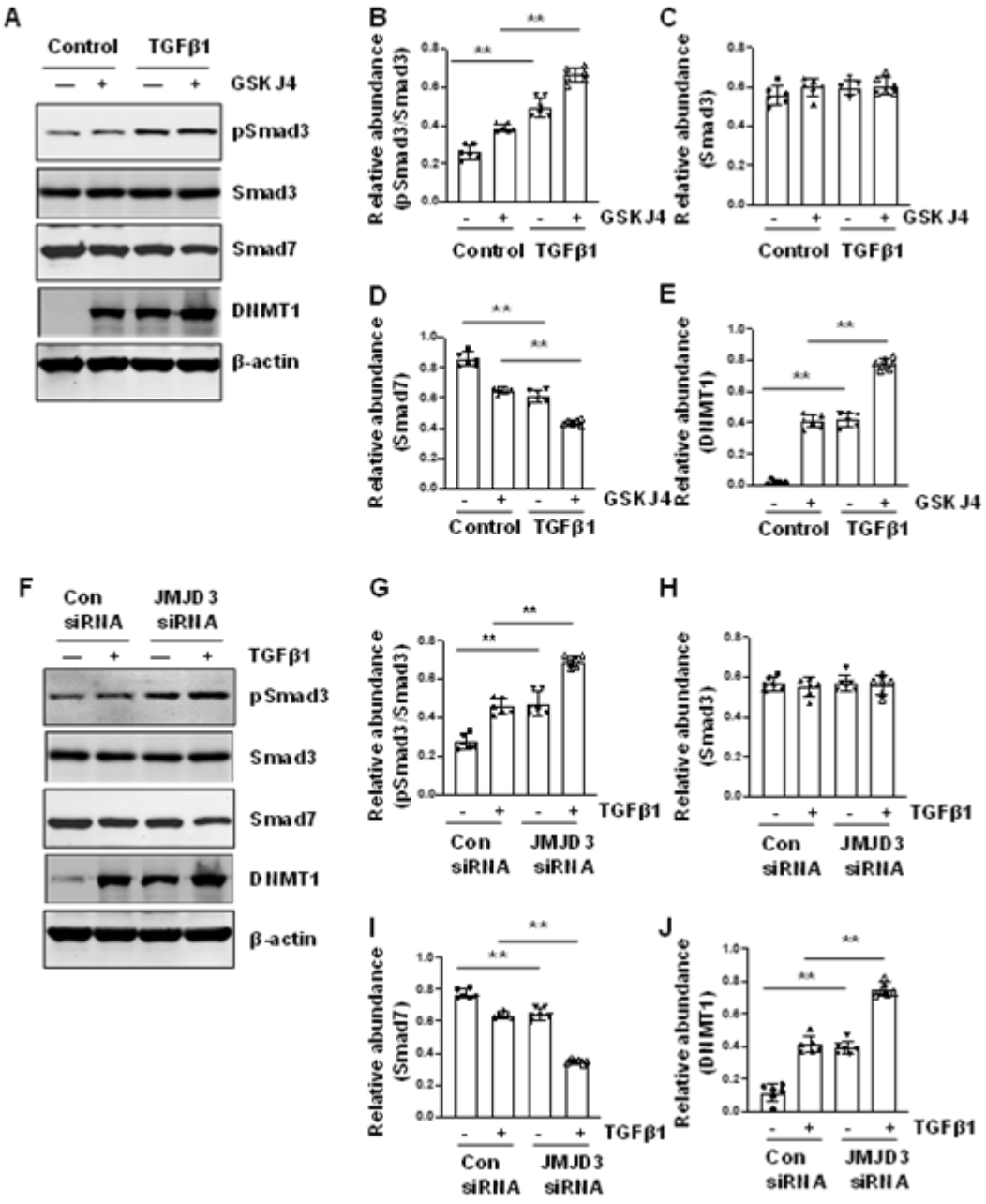


**Figure S5. Inhibition of JMJD3 by GSKJ4 or siRNA promotes Smad3 phosphorylation and expression of DNMT1, as well as reduces Smad7 expression in cultured renal interstitial fibroblasts exposed to TGFβ1.** (A) NRK-49F cells were incubated with medium containing 0.5% serum or treated with TGFβ1 (2 ng/ml) in the presence or absence of GSKJ4 (6 μM) for 36 h. Cell lysates were prepared and subjected to immunoblot analysis with antibodies against pSmad3, Smad3, Smad7, DNMT1, β-actin. Expression levels of pSmad3 (B), Smad3(C), Smad7 (D),

DNMT1 (E) or β-actin were quantified by densitometry analysis and then normalized with Smad3 or β-actin, respectively, as indicated. (F) NRK-49F cells were transfected control siRNA or JMJD siRNA and then incubated with medium containing 0.5% serum or treated with TGFβ1 (2 ng/ml) for 36 h. Cell lysates were prepared and subjected to immunoblot analysis with antibodies against pSmad3, Smad3, Smad7, β-actin. Expression levels of pSmad3 (G), Smad3 (H), Smad7 (I), DNMT1 (J) or β-actin were quantified by densitometry analysis and then normalized with Smad3 or β-actin as indicated in the figures. Values are the means ± sem of at ≥3 independent experiments. ***P*<0.01

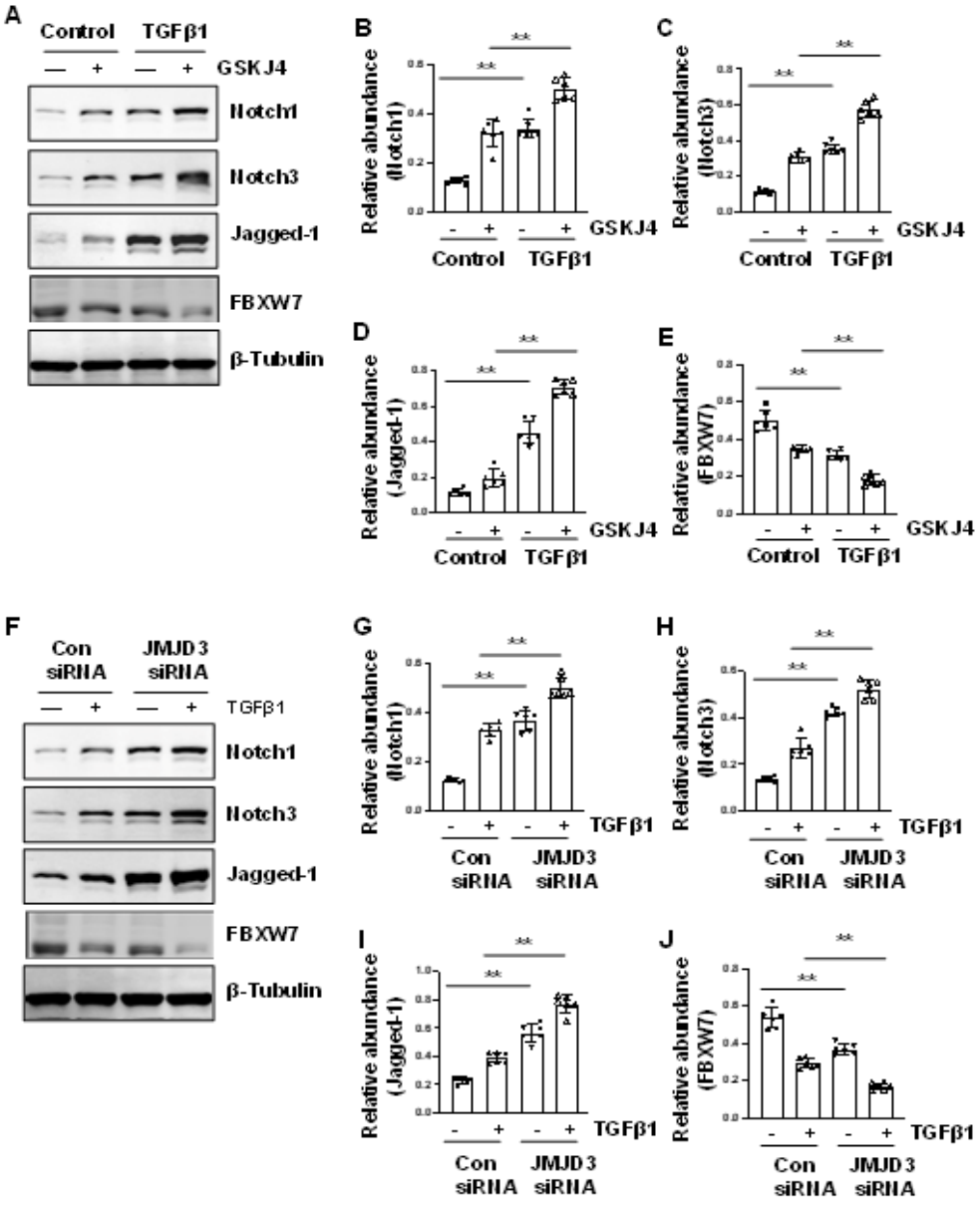


**Figure S6. Inhibition of JMJD3 by GSKJ4 or siRNA enhances TGFβ1-induced expression of Notch1, Notch3 and Jagged-1 and reduced expression of FBXW7 in cultured mouse renal epithelial cells.** (A) mTECS cells were incubated with medium containing 0.5% serum or treated with TGFβ1 (2 ng/ml) in the presence or absence of GSKJ4 (6 μM) for 36 h. Cell lysates were

prepared and subjected to immunoblot analysis with antibodies against Notch1, Notch3 and Jagged-1, FBXW7 or β-tubulin. Expression levels of Notch1 (B), Notch3 (C) and Jagged-1 (D), FBXW7 (E) or β-tubulin were quantified by densitometry analysis and then normalized with β-tubulin, respectively, as indicated in the figures. (F) mTECS cells were transfected control siRNA or JMJD siRNA and then incubated with medium containing 0.5% serum or treated with TGFβ1 (2 ng/ml) for 36 h. Cell lysates were prepared and subjected to immunoblot analysis with antibodies against Notch1, Notch3 and Jagged-1, β-tubulin. Expression levels of Notch1 (G), Notch3 (H) , Jagged-1 (I) or FBXW7 (J), β-tubulin were quantified by densitometry analysis and then normalized with β-tubulin, respectively, as indicated in the figures Values are the means ± sem of at ≥3 independent experiments. **P*<0.05; ***P*<0.01