Figure S7

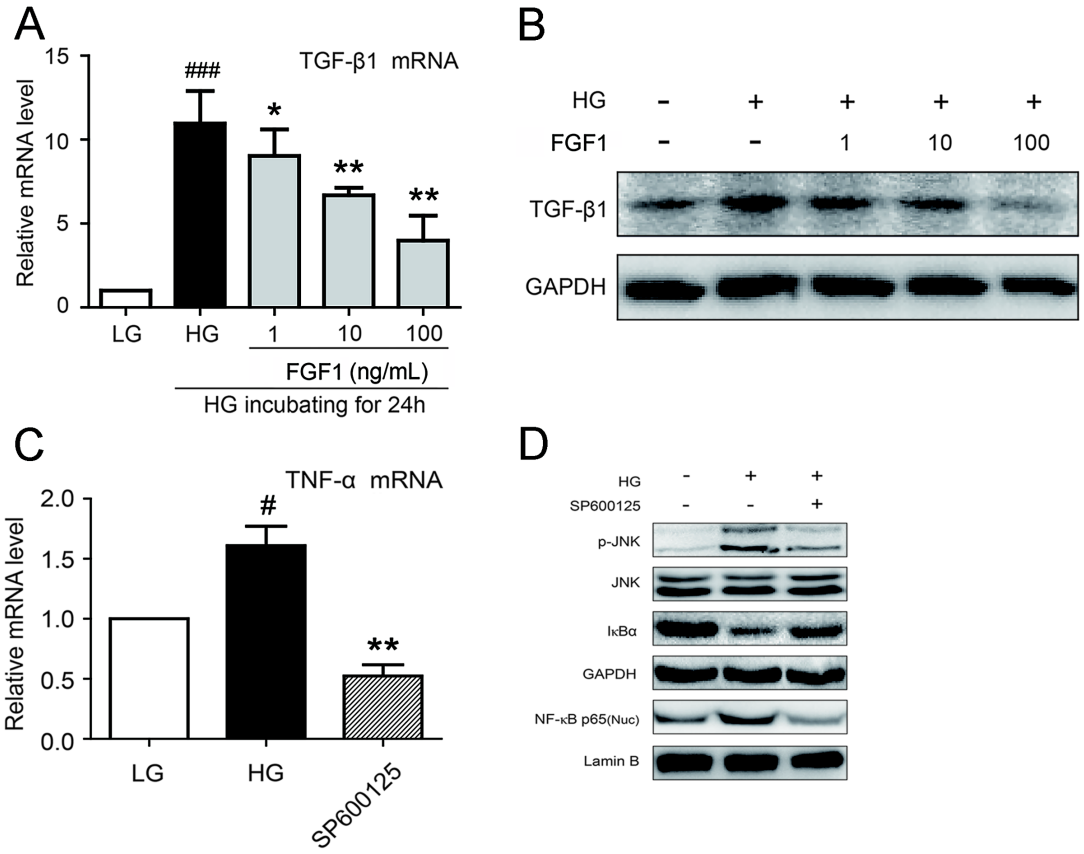

Figure S7. Effects of FGF1 and JNK-specific inhibitor SP600125 on HG-induced inflammation in mesangial cells. (A&B) Glomerular mesangial cells were pre-treated with FGF1 (1, 10, or 100 ng/mL) for 2 h, followed by treatment with high glucose (HG, 25 mM) for an additional 24 h, and (A) mRNA and (B) Western blot analysis for TGF-β1 were determined. Data are presented as mean ± SEM; $^{###}$p<0.001 vs LG (low glucose control); *p<0.05 and **p<0.01 vs HG group; n=3. **(C&D)** Glomerular mesangial cells were pre-treated with SP600125 (10 μM), followed by treatment with high glucose (HG, 25 mM) for an additional 24 h, and cells collected for the following determination: (C) TNF-α mRNA from LG, HG, and SP600125 (HG+SP600125) treatment groups. Data shown as the mean ± SEM; $^{#}$p<0.05 vs LG; **p<0.01 vs HG group; n=3; (D) Western blot analysis for JNK

phosphorylation (p-JNK), unphosphorylated JNK as loading control; IκBα degradation, GAPDH as loading control; nuclear NF-κB p65 subunit, lamin B as loading control.

Figure S8

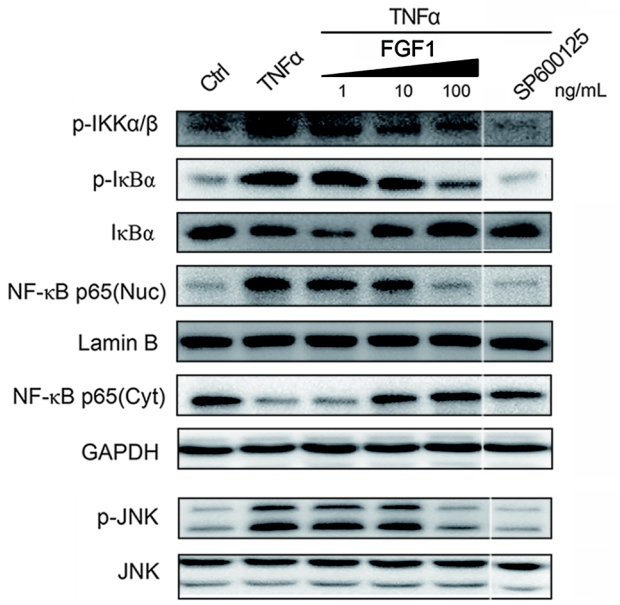

Figure S8. Effects of FGF1 and SP600125 on TNF-α-induced signaling

TNF-α-induced (1 μg/mL for 30 min) activation of NF-κB and JNK in mesangial cells that have been pre-treated with FGF1 (1, 10, or 100 ng/mL for 1 h) or SP600125 (10 μM for 1 h). The nuclear and cytosolic NF-κB p65 subunit, respectively (Nuc) and (Cyt) were detected by Western blot using the respective Lamin B and GAPDH as loading controls.