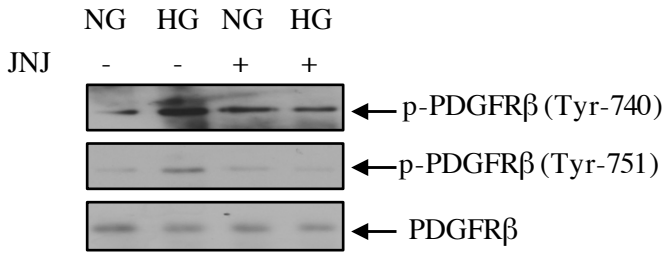
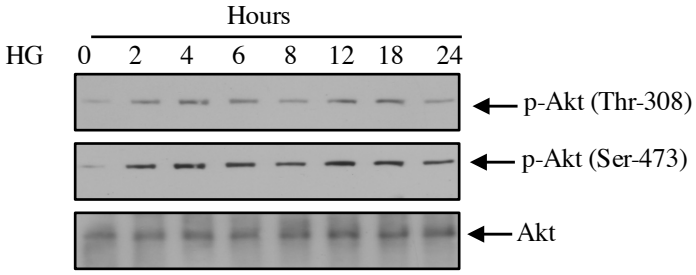
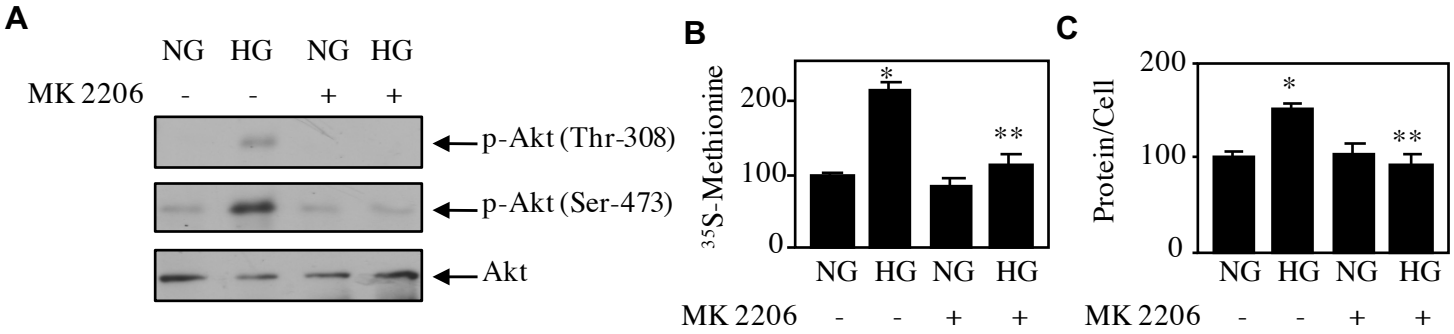


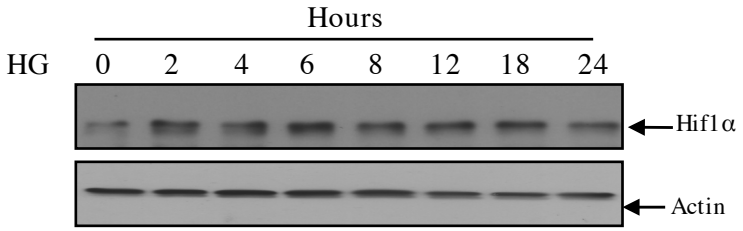
Supplementary Fig. S1. The PDGFRβ inhibitor JNJ blocks tyrosine phosphorylation of PDGFRβ at residues 740 and 751. Mesangial cells were treated with 0.1 μM JNJ prior to incubation with normal glucose (NG) or high glucose (HG) for 24 hours. The cell lysates were immunoblotted with phospho-PDGFRβ (Tyr-740), phospho-PDGFRβ (Tyr-751) and PDGFRβ antibodies as indicated.



Supplementary Fig. S2. High glucose increases the phosphorylation of Akt in a time-dependent manner. Serum-starved mesangial cells were incubated with 25 mM glucose (HG) for indicated periods of time. The cell lysates were immunoblotted with phospho-Akt (Thr-308), phospho-Akt (Ser-473) and Akt antibodies.



Supplementary Fig. S3. Akt inhibitor MK 2206 blocks Akt phosphorylation and high glucose-induced mesangial cell protein synthesis and hypertrophy. Serum-starved mesangial cells were treated with 0.1 μM MK 2206 (MK) for one hour prior to incubation with normal glucose (NG) or high glucose (HG). (A) The cell lysates were immunoblotted with phospho-Akt (Thr-308), phospho-Akt (Ser-473) and Akt antibodies. (B and C) Protein synthesis as the incorporation of ^{35}S-Methionine (panel B) and hypertrophy as the ratio of protein to cell number (panel C) were determined. Mean ± SE of 3 – 6 measurements is shown. *$p < 0.01$ vs NG; **$p < 0.01$ vs HG.



Supplementary Fig. S4. High glucose increases the expression of Hif1α in a time-dependent manner. Serum-starved mesangial cells were incubated with 25 mM glucose (HG) for indicated periods of time. The cell lysates were immunoblotted with Hif1α and actin antibodies.