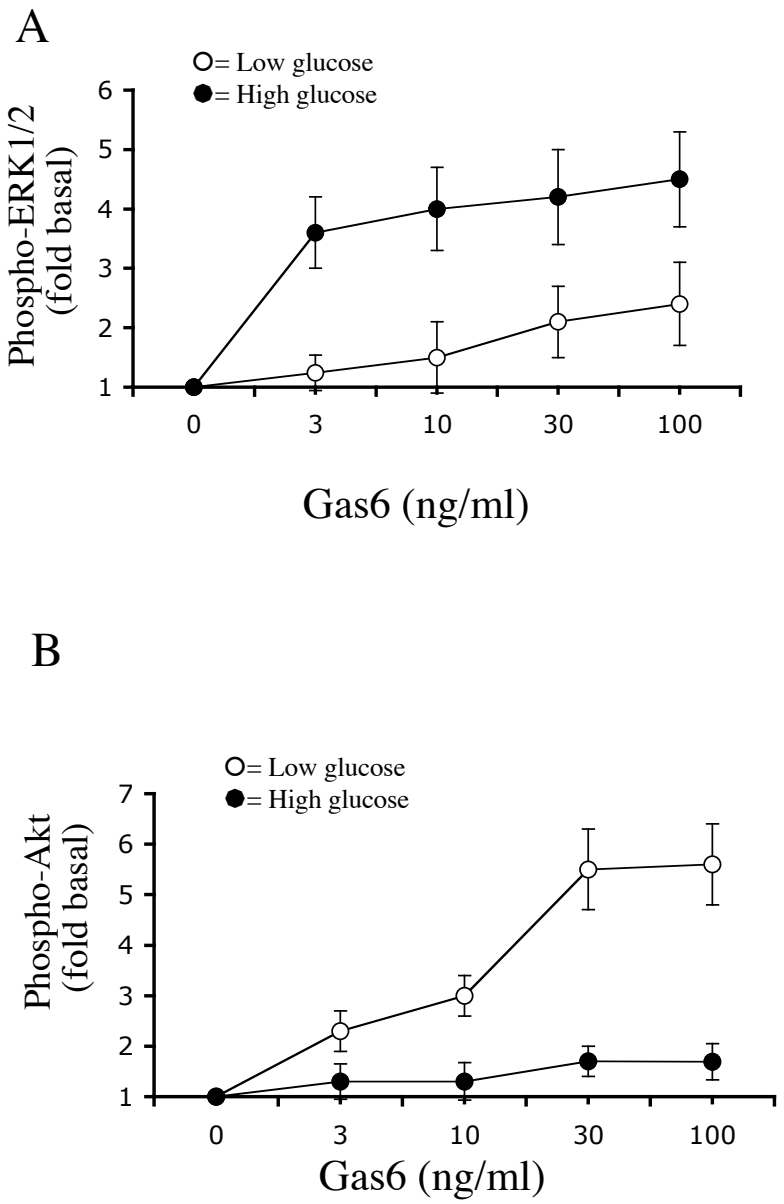
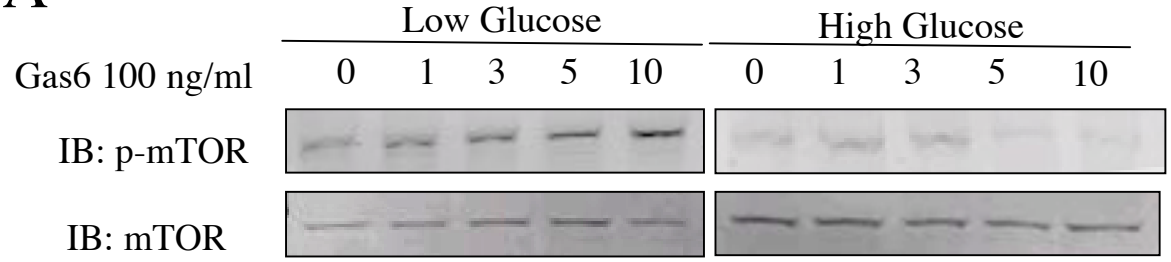
Figure I



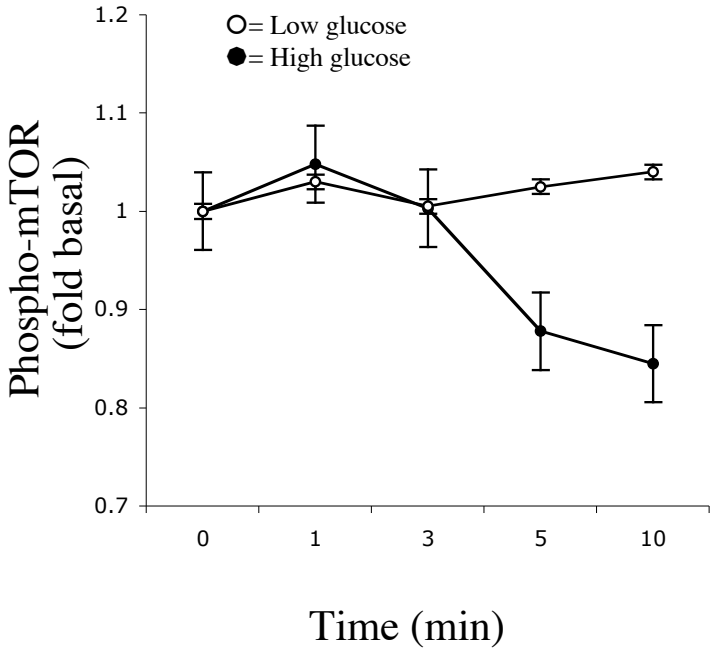
Supplemental Figure I. Quantification of ERK 1/2 phosphorylation and AKT phosphorylation. VSMC were cultured in serum free LG or HG DMEM for 24 hr and treated with increasing concentrations of Gas6 for 10 mins. A. In HG ERK 1/2 phosphorylation increased in response to Gas6 stimulation. B. In LG Akt phosphorylation increased in response to Gas6 stimulation. Quantification of data from n=7.

Figure II

A

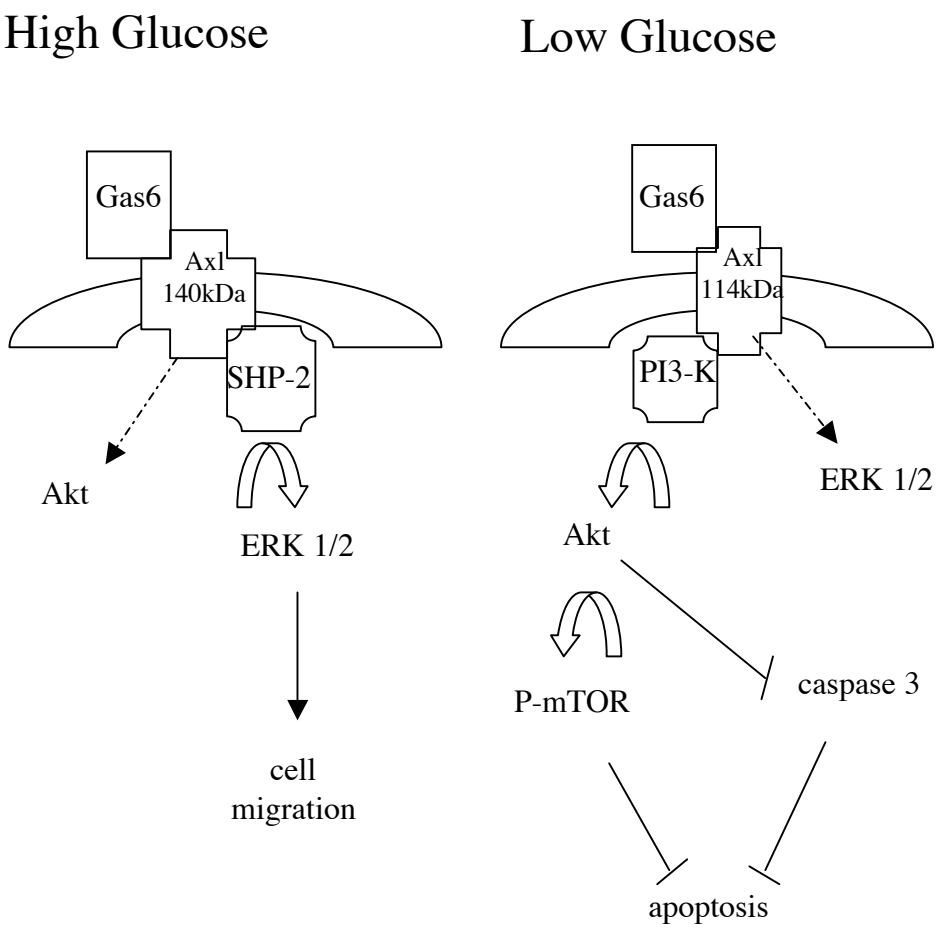


B



Supplemental Figure II. Low glucose modulates phosphorylation of mTOR upon stimulation with Gas6. VSMC were cultured in serum free LG or HG DMEM for 24hr. Cells were treated with Gas6 for the indicated times. A. Lysates were immunoblotted and probed with mTOR and phosphospecific mTOR antibodies. Gas6 induced a significant time dependent decrease in phosphorylation of mTOR at Ser 2448 in HG compared to a slight increase in phosphorlyation of mTOR in LG. B. Quantification of data from n=3.

Figure III



Supplemental Figure III. Proposed mechanism of Gas6 activation of Axl VSMC in High Glucose and Low Glucose. High Glucose: Gas6 stimulation of the 140 kDa Axl receptor results in Axl association with SHP-2, down stream activation of ERK 1/2, resulting in cell migration. Low Glucose: Gas6 stimulation of the 114 kDa Axl receptor results in Axl association with the p85 subunit of PI3K activating Akt and its downstream anti-apoptotic target mTOR as well as inhibition of caspase 3 activity.