SUPPLEMENTAL FIGURE LEGENDS

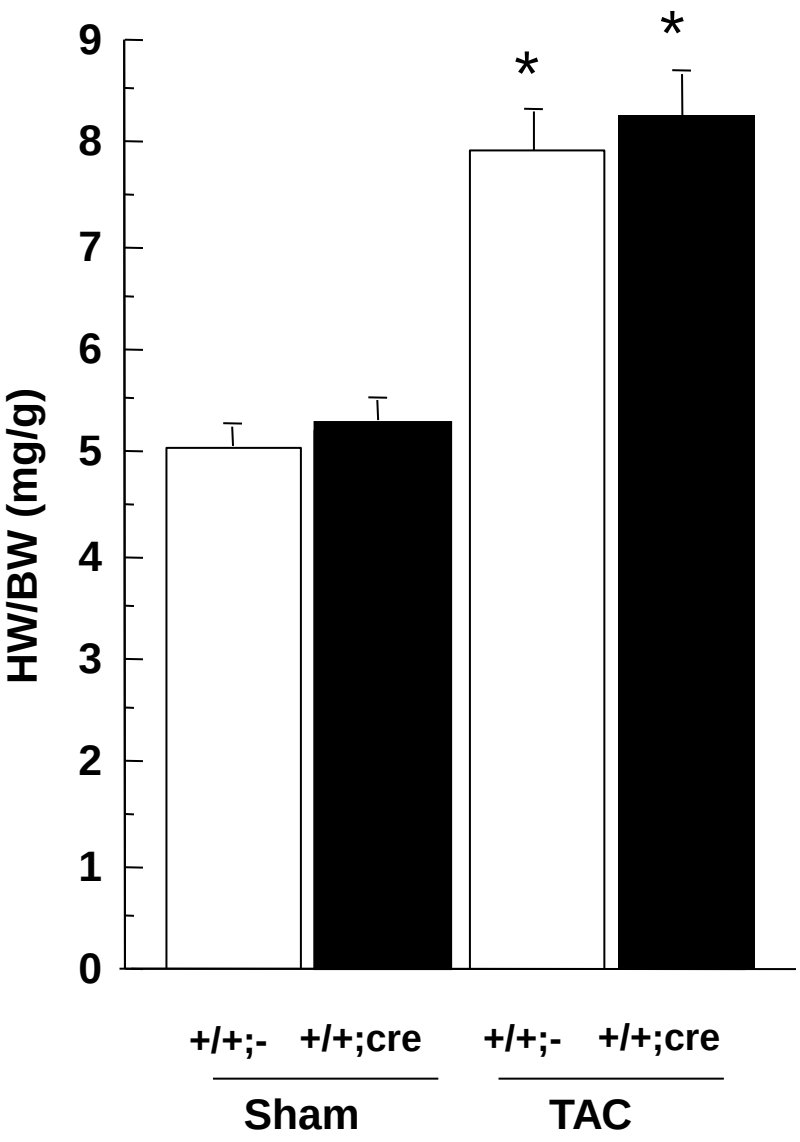
Supplemental Figure 1. The Cre transgene does not affect baseline cardiac phenotype or the response to TAC under the conditions of our assays. The ratio of heart weight to body weight (HW/BW) is presented for αMHC-*Cre*$^{-/-}$ × *Fstl3*$^{+/+}$ and αMHC-*Cre*$^{+/-}$ × *Fstl3*$^{+/+}$ mice. *P*>0.05 comparing cre-positive versus cre-negative mice at baseline or following TAC. However, TAC led to significant increases in HW/BW in both strains of mice. *P*<0.05.

Supplemental Figure 2. *Fstl3*-deficiency does not affect other Smad signaling. Phosphorylation of Smad1/5 (P-Smad1/5)(A) in the hearts of WT and KO mice at 1 week after sham operation or TAC. Representative blots of phosphorylated and total Smad1/5 are shown. Lower panels show quantitative analysis of Smad1/5 phosphorylation. Relative phosphorylated levels of Smad1/5 were normalized to control values in sham-WT mice (n=6 to 12 hearts in each group). B and C, Myocardial expression of Smad4 (B) and Smad6 (C) protein in WT and KO mice at 1 week after TAC as measured by western blot analysis. Representative blots for Smad4, Smad6 and GAPDH are shown. Band intensities were normalized to control values in sham-WT mice (n=6 to 12 hearts in each group). **P*<0.05 versus corresponding sham and #*P*<0.05 versus WT mice.

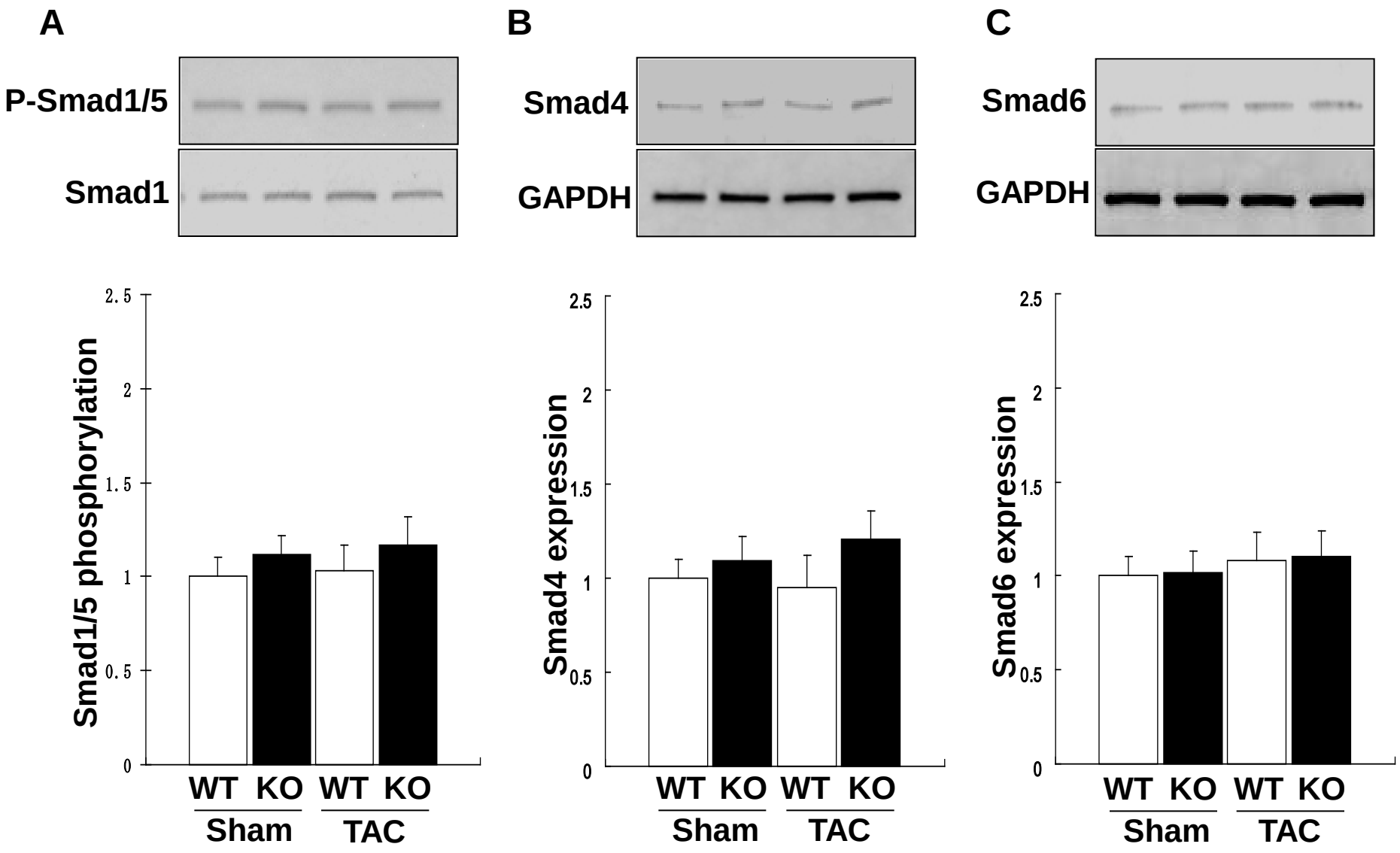
Supplemental Figure 3. Adenovirus mediated *Fstl3* overexpression, by itself, does not affect cardiac hypertrophy in vitro. A, Quantitative analysis of cell surface area after transduction with Ad-Fstl3 or Ad-βgal (control) followed by PE stimulation. B, Representative fluorescence microscope images of NRVMs transfected with Ad-Fstl3 or Ad-βgal in the absence or presence of PE. C and D, The results of real-time-PCR analysis of ANF(C) and BNP(D) expression with adenoviral vector expressing (Ad-Fstl3) or β galactosidase (Ad-βgal, control) for 8 hours in the presence or absence of PE (100μmol/L) for 24 hours. **P*<0.05 versus corresponding control without PE.

Supplemental Figure 4. The effects of Smad2 overexpression on myocyte hypertrophy. Transduction with an adenoviral vector expressing Smad2 inhibits PE-induced myocyte hypertrophy but does affect the cell surface area of myocytes at baseline. **P*<0.05 versus corresponding control without PE.

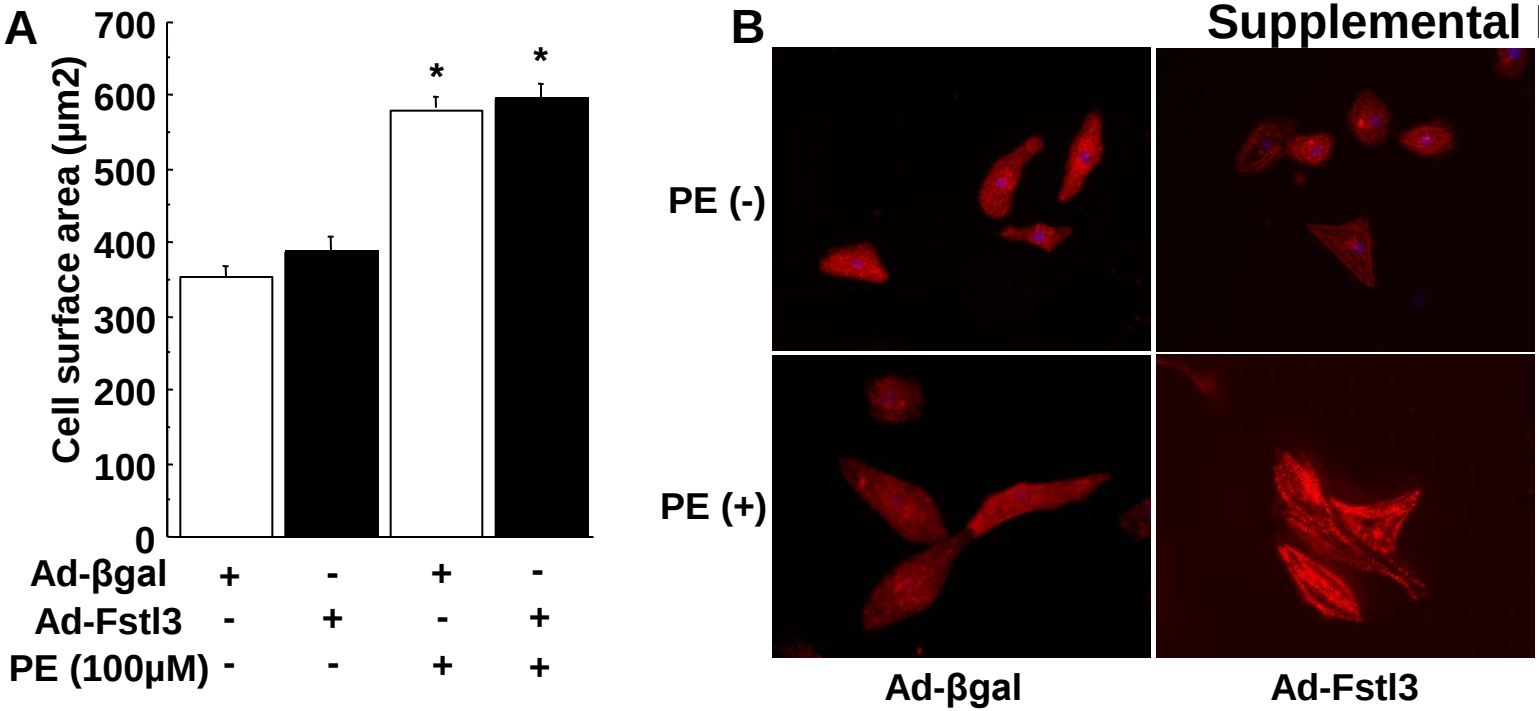
Supplemental Figure 1

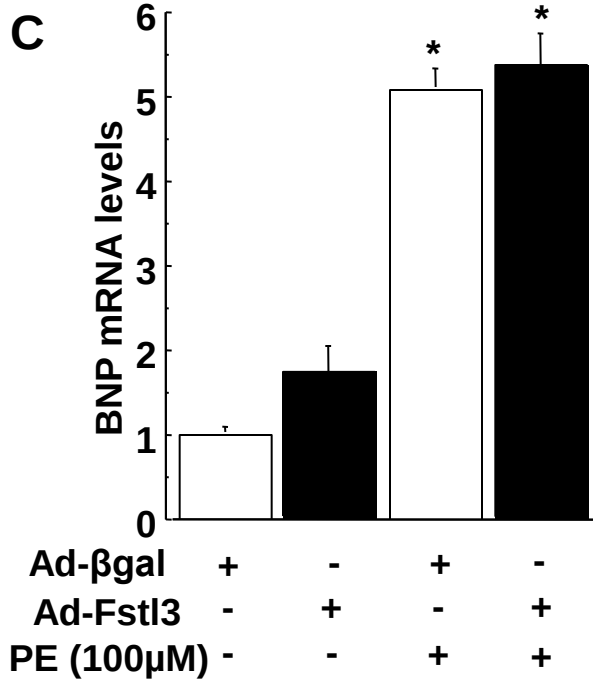


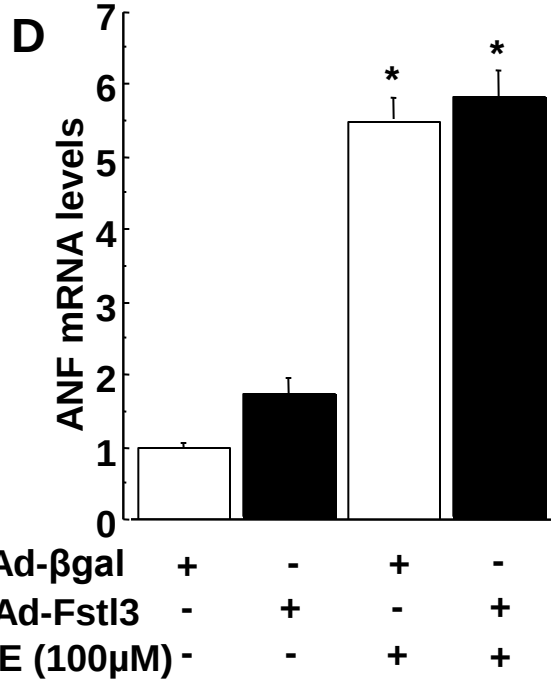
Supplemental Figure 2



Supplemental Figure 3







Supplemental Figure 4