Supplementary Figure Legends:

**Supplementary Figure 1:** Overexpression of PPARγ inhibits TGF-β1-stimulated expression of ECM molecule mRNA in mouse CFs. A) to C) CFs were infected with PPARγ adenovirus (Ad-PPARγ), empty vector adenovirus (Ad-null) or vehicle (Control group) for 72 hours. Serum starved CFs were then treated with TGF-β1 (1 ng/ml) or vehicle for an additional 24 hours. CTGF, persiotin, α-SMA and GAPDH mRNA expression was determined by real-time RT-PCR analysis. (n) = number of samples. * p < 0.05 compared with respective vehicle groups; # p < 0.05 compared with TGF-β1 alone groups.
Supplementary Figure 2: Activation of PPARγ by agonists inhibits TGF-β1-stimulated expression of CTGF and periostin mRNA by interfering with Smad3 phosphorylation in mouse CFs. Serum starved CFs were pretreated with the PPARγ agonists rosiglitazone (Rosi, 1.0 μmol/l), pioglitazone (Pioglitz, 10 μmol/l) or vehicle for 24 hours and then treated with TGF-β1 (1 ng/ml) or vehicle for an additional 24 hours (A and B) or 30 min (C and D). CTGF, persiotin, α-SMA and GAPDH mRNA expression was determined by real-time RT-PCR analysis. Smad3 phosphorylation was assessed by Western blot analysis using a monoclonal anti-pSmad3 antibody (Cat. 9514, Cell Signaling Tech). (n) = number of samples. * p < 0.05 compared with respective vehicle groups; #p < 0.05 compared with TGF-β1 alone groups.

Supplementary Figure 3: Neither rosiglitazone nor T0070907 changed afterload in the pressure overloaded LV of wild type mice. Three weeks after TAC, LV systolic pressure (LVSP, mmHg) was measured as described in Methods. Results are means ± SEM; (n) = number of animals. * p < 0.01 compared with treated sham control.