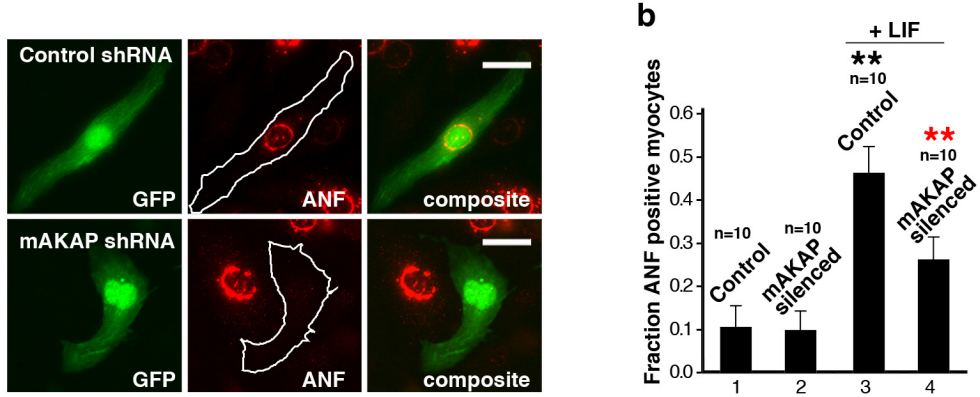
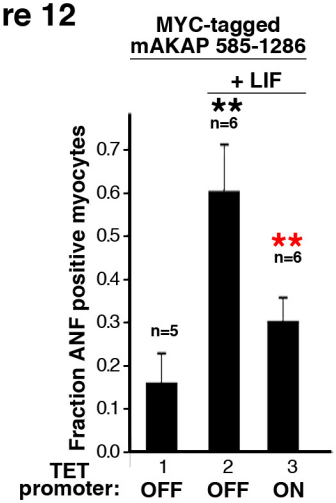
Supplementary Figure 11



Supplementary Figure 11. mAKAP plays a role in LIF-induced cardiac hypertrophy. **(a)** RNV were co-transfected with a GFP expression vector and either control shRNA (top panels) or mAKAP shRNA (bottom panels). Cells were treated with LIF (1000U/ml) before immunostaining with anti-ANF antibody (middle panels). Cells positive for GFP transfection (left panels) are demarcated by white outline (middle panels). The composite images (right panels) demonstrate that mAKAP knockdown inhibits ANF expression. Scale bar indicates 20μm. **(b)** A graph representing the fraction of cells expressing ANF in cultures treated without (1 & 2) and with LIF (3 & 4) in control (1 & 3) and mAKAP silenced (2 & 4) RNV. Error bars indicate S.E.M. P values <0.01 (**) are indicated relative to control (black) and sample (red).

Supplementary Figure 12



Supplementary Figure 12. Displacement of mAKAP inhibits LIF-induced expression of ANF, an indicator of hypertrophy. RNV were infected with the TET OFF inducible mAKAP-585-1286-MYC adenovirus to conditionally express this construct. Cells cultured with 50ng/ml doxycycline repressed expression of the fragment (TET OFF, 1 & 2) while those cultured in 0.75ng/ml doxycycline permitted moderate expression of the fragment (TET ON, 3) and displacement of mAKAP. Infected RNV were identified with antibodies to mAKAP-585-1286 (anti-MYC) and ANF. ANF expression was monitored in control and LIF treated cultures. Displacement of mAKAP inhibited LIF induced hypertrophy as measured by ANF expression. Error bars indicate S.E.M. P values <0.01 (**) are indicated relative to control (black) and sample (red).