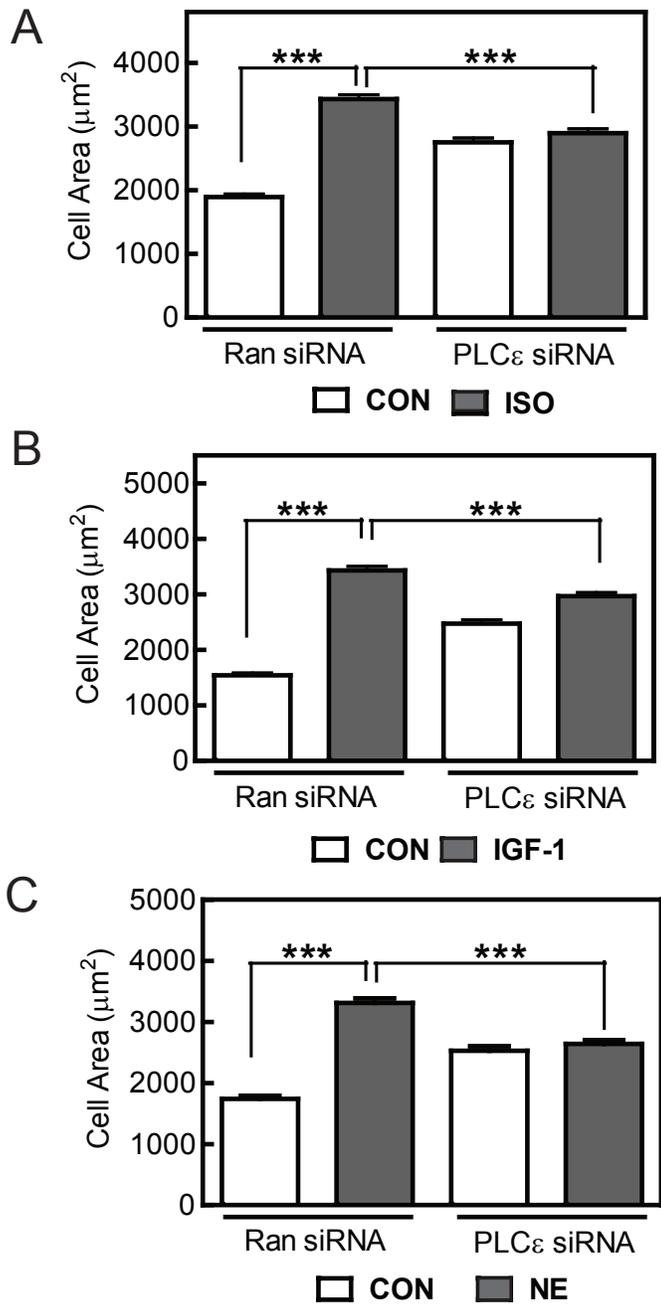
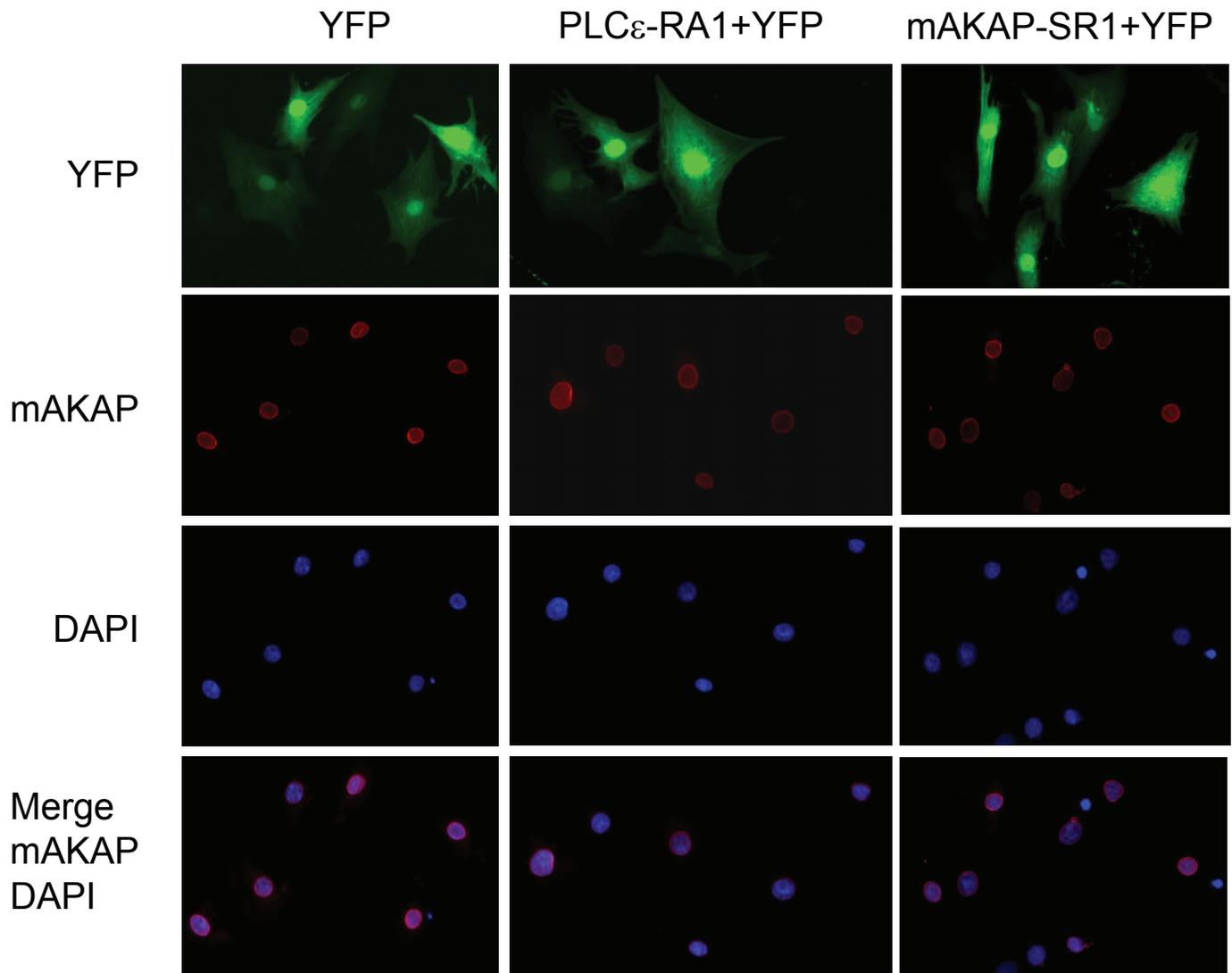


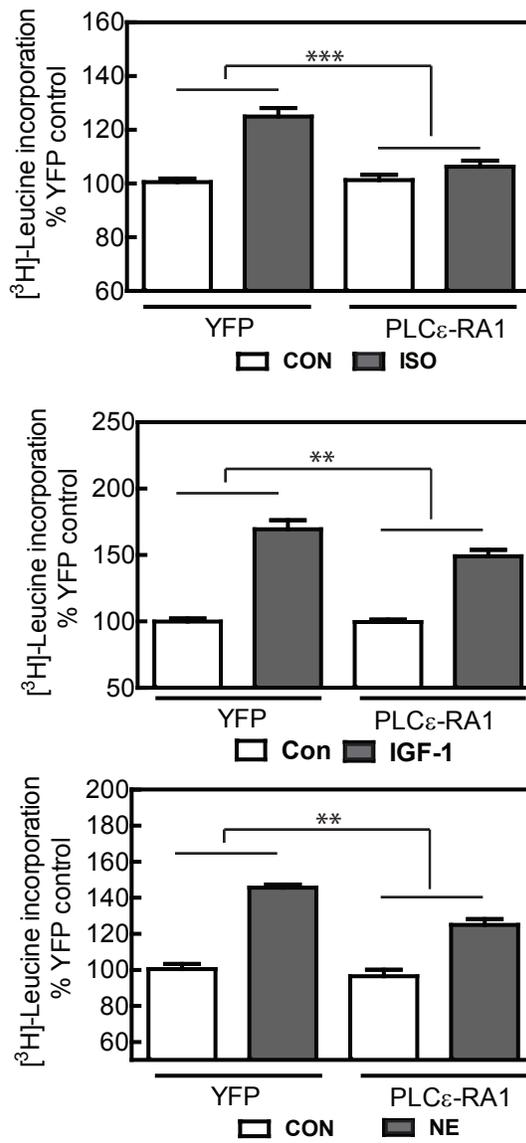
Supplemental Figure 1. siRNA of PLCε. A) Neonatal myocytes were infected for 3 days or 6 days with the indicated adenoviruses. mRNA was extracted and assessed for PLCε or GAPDH mRNAs by semi-quantitative jRT-PCR. Shown are duplicate determinations. The images were analyzed by densitometry and PLCε data, normalized to GAPDH expression, are shown in the bottom panels. B) NRVMs were infected with the indicated siRNAs for 3 days. Soluble protein was extracted and PLCε was immunoprecipitated and immunoblotted. Cell lysate from PLCε transfected HEK 293 cells is shown as a control. C) NRVMs were infected with the indicated adenoviruses, soluble protein extracted and Western blotted for the indicated PLC isoforms. D) NRVMs were co-infected with viruses expressing the indicated proteins where PLCε (res) is PLCε with a single non-coding point mutation that confers resistance to the PLCε siRNA. [³H]-leucine incorporation was measured as in figure 1 and methods.



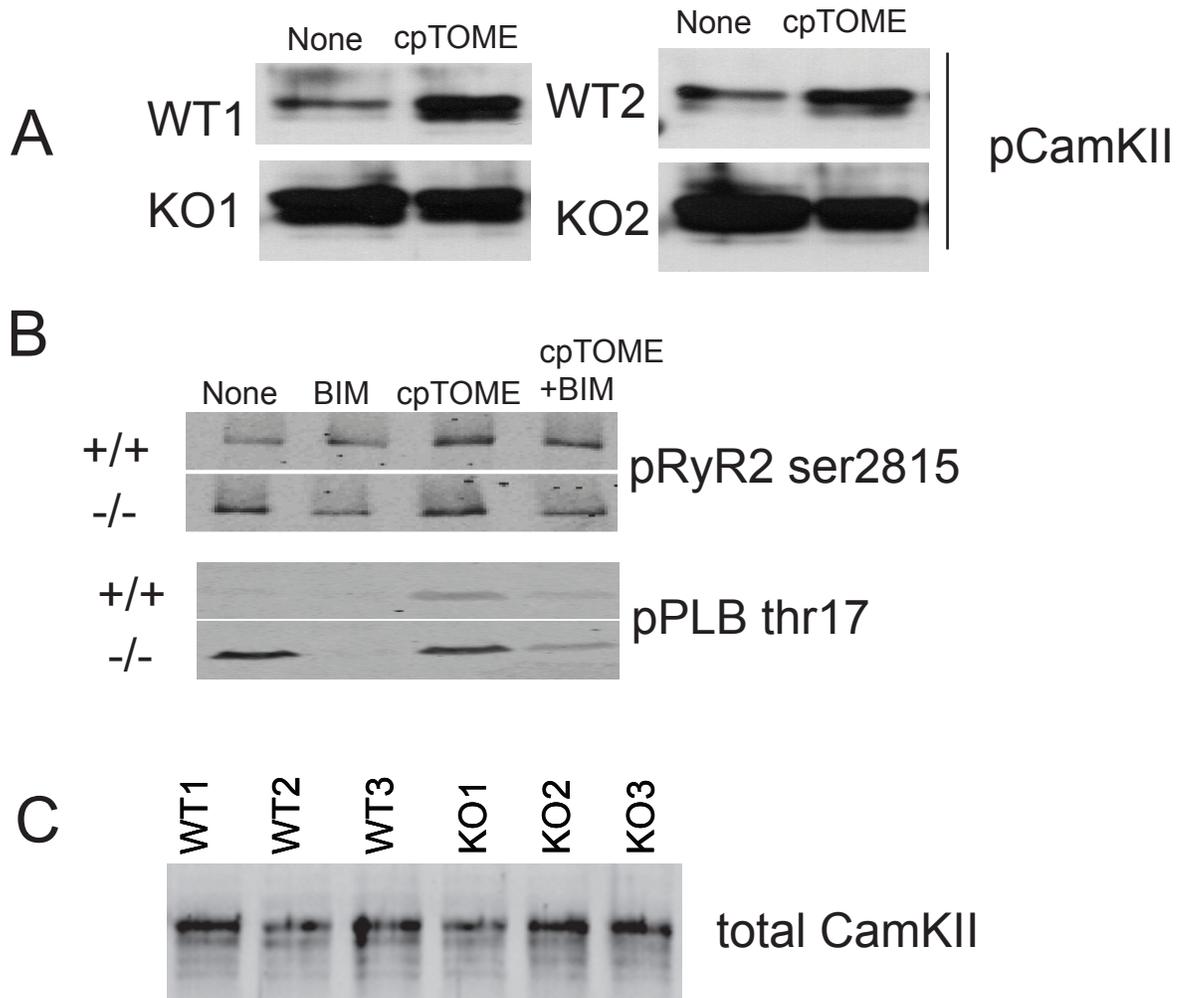
Supplemental Figure 2. NRVMs were stimulated with A) Iso, B) IGF-1 or C) NE as in Figure 2. Cell area was measured as in Fig 1B and in methods.



Supplemental Figure 3. PLC ϵ RA1 or mAKAP-SR1 expression in NRVMs does not disrupt mAKAP β localization. NRVMs were infected with adenoviruses expressing PLC ϵ -RA1+YFP, mAKAP-SR1+YFP or YFP. Cells were stained and analyzed as in Fig 7 C. Cells expressing PLC ϵ -RA1 or mAKAP-SR1 have similar mAKAP β localization to uninfected cells.



Supplemental Figure 4. NRVMs were infected with PLC ϵ -RA1 expressing adenovirus as in Figure 8 and treated with the indicated agonists as in Figure 2. Leucine incorporation was measured as indicated in methods.



Supplemental Figure 5. CamKII activity is elevated in $PLC\epsilon^{-/-}$ mice. A) Adult ventricular myocytes were isolated from 2 separate wild type and knockout mice and treated with or without 10 $\mu\text{mol/L}$ cpTOME, an Epac specific activator for 3 min. Extracts were prepared and assayed by Western blotting for phospho-CamKII. B) AVMs were isolated and treated with 1 $\mu\text{mol/L}$ bisindoylmaleimide (BIM), a PKC inhibitor for 30 min, or 10 $\mu\text{mol/L}$ cpTOME for 5 min. Extracts were prepared and western blotted for CamKII phosphor-specific sites on Ryr2 or phospholamban. C) Heart extracts from mice as indicated were western blotted for total CamKII.

