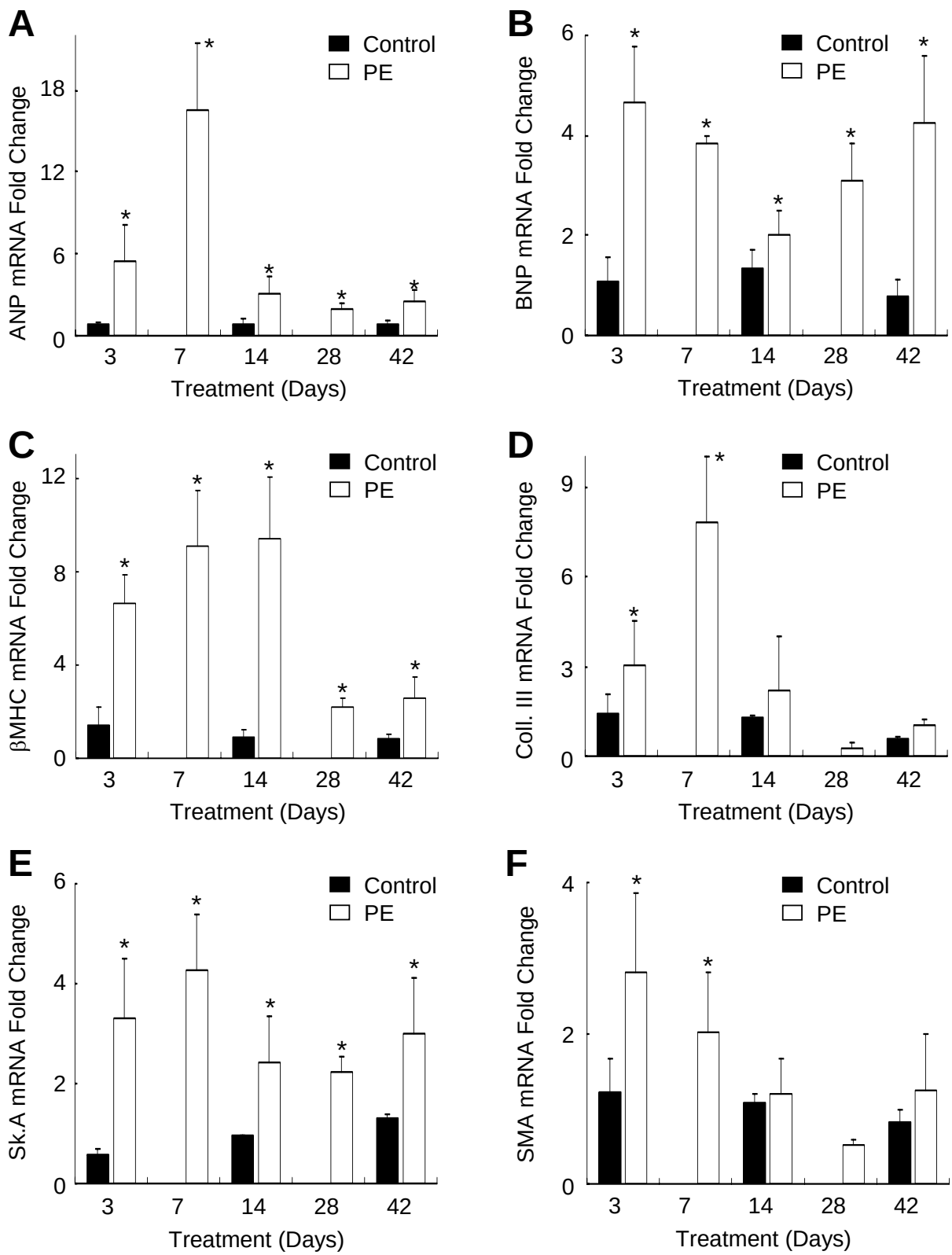
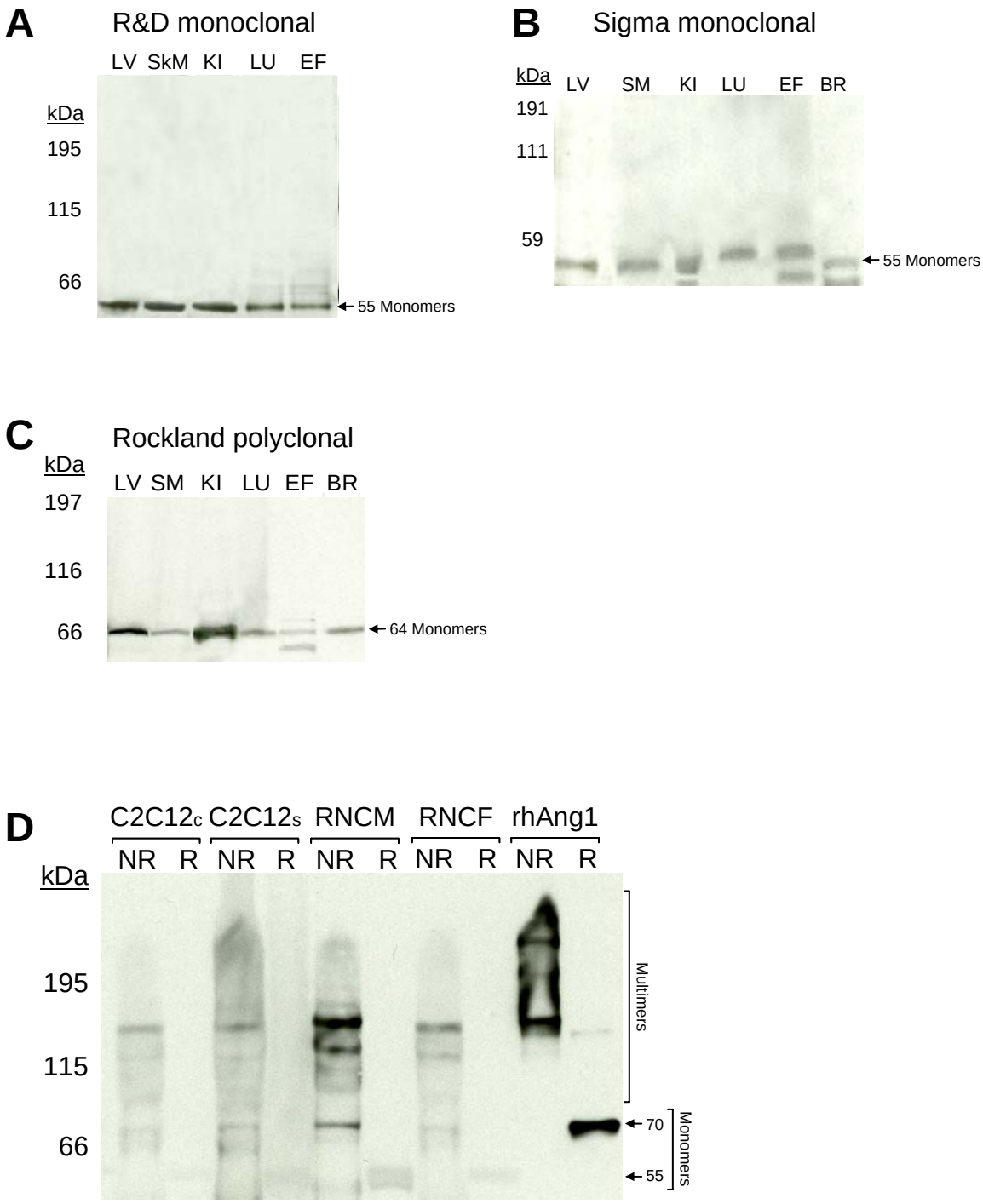
Supplemental Figure Legends

Supp Fig 1. Fetal Gene Expressions Increase with PE-Induced Cardiac Hypertrophy. LV (A) ANP, (B) BNP, (C) βMHC, (D) collagen III (Coll. III), (E) skeletal actin (Sk. A) and (F) SMA mRNA levels (real-time PCR) increased with hypertrophy. *n*=3/group; studies done in triplicate. *$p \leq 0.05$ versus controls.

Supp Fig 2. Protein lysates from mouse LV, skeletal muscle (SM), kidney (KI), lung (LU), epididymal fat tissue (EF), and brain (BR) were treated with PNGase F and evaluated under reduced (βMe) conditions via SDS-PAGE (3-8% gel)/Western blotting. Blots were probed with anti-ang1 (A) R&D or (B) Sigma monoclonal, or (C) Rockland polyclonal (studies done in duplicate). PNGase-F-treated lysates probed for ang1 showed monomers at (A, B) 55kDa (R&D and Sigma monoclonal) or (C) 64kDa (Rockland polyclonal). (D) Cell media was collected, precleared, incubated with anti-ang1 Sigma monoclonal, and agarose beads (studies done in duplicate). Immunoprecipitated lysates were analyzed by SDS-PAGE (3-8% gels), and Western blots were probed with anti-ang1 R&D monoclonal and examined under nonreduced (NR) and reduced (R) conditions. Cell production of ang1 forms was compared with rhAng1/R&D (rhAng1). C2C12 were differentiated into myocytes by confluency ($C2C12_c$) or a change in serum ($C2C12_s$). Rat neonatal CM (RNCM) and cardiac fibroblasts (RNCF) and C2C12 skeletal myocytes produce ang1 monomers and multimers.



Supplementary Figure 1



Supplementary Figure 2