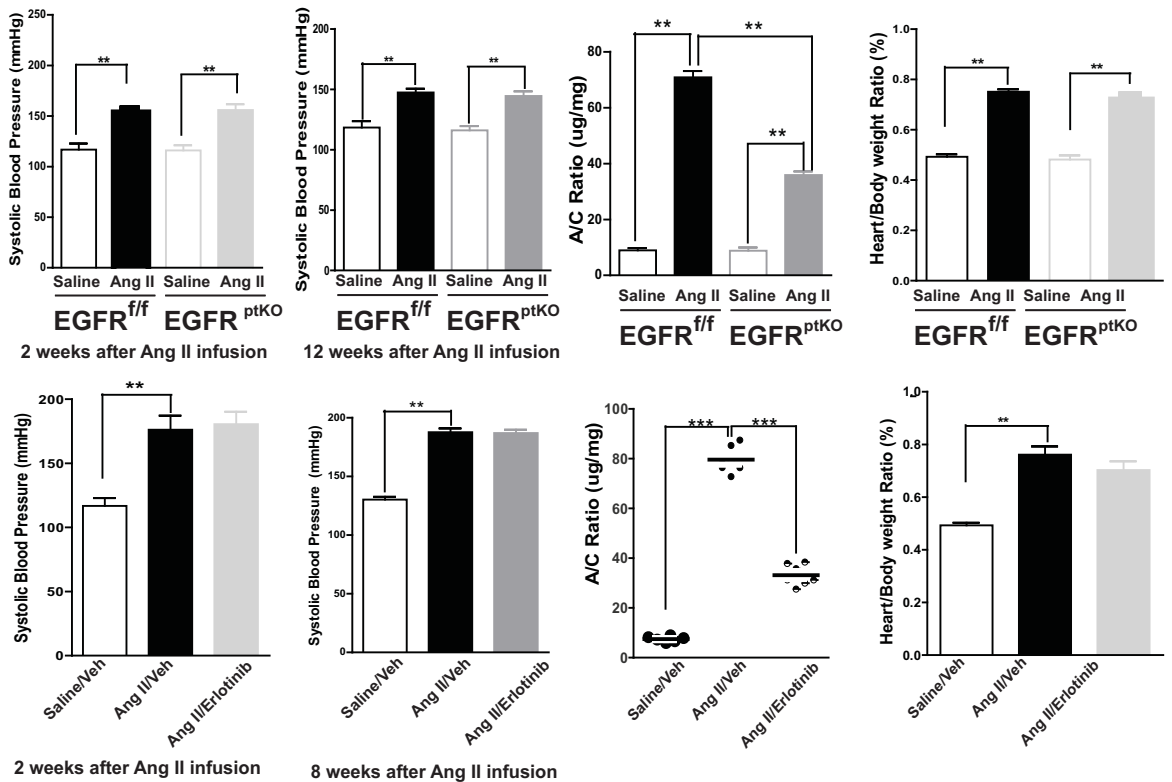


Supplementary data

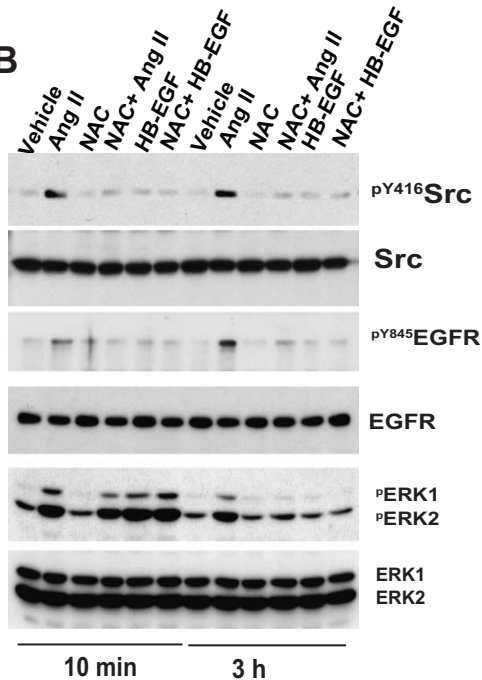
Figure S. (A) Elevation of systolic blood pressure, and heart hypertrophy, Albuminuria (indicated by albumin/creatinine ratios) in mice subjected to chronic Ang II administration. (B) In AT1R/C14 cells, Ang II increased Y416 phosphorylation of Src, a marker of kinase activation, which was inhibited by the antioxidant, n-acetylcysteine (NAC). NAC also inhibited the sustained Ang II-mediated expression of ^{Y845}EGFR and ERK_{1/2} phosphorylation. (C) Kidney cortex lysates from mice chronically exposed to Ang II or saline controls were subjected to immunoprecipitation with anti-EGFR antibodies and immunoblotting with antibodies against phospho-EGFR (either Y845 or Y1173), Shc, GRB2, or total EGFR, respectively. (D) Quiescent AT1R/C14 cells were pretreated with vehicle alone or filipin III (5 µg/ml) for 30 min before treatment with Ang II (10⁻⁷ M), HB-EGF (15 ng/ml) or vehicle for 10 min or 3 h. Cell lysates were subjected to immunoprecipitation with anti-PY antibodies, followed by immunoblotting with an antibody against EGFR or direct immunoblotting with an antibody against phospho-ERK_{1/2}, followed by stripping and reprobing with an antibody against total ERK_{1/2}. (n = 4-8 per group; as Mean ± SEM; **P<0.01, ***P<0.001)

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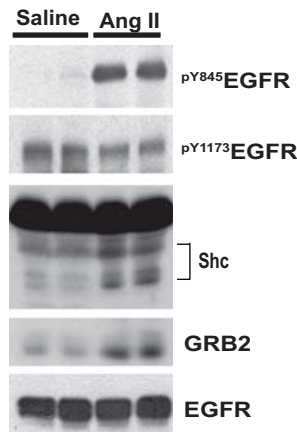
A



B



C



D

