

Supplemental Figure 1. GSK-3 α activity and early infarct size in post-MI conditional KO hearts. (A) Immunoblot shows GSK-3 α activation and expression in sham and 3 wk post-MI WT and KO hearts. Blot quantification shows increase GSK-3 α phosphorylation (inhibition) (B) in WT post-MI and comparable GSK-3 α levels (C) in sham vs MI. n=3 for each group. (D) The LAD was permanently ligated and 48 hours post ligation, hearts were harvested. Representative images of a triphenyltetrazolium chloride (TTC)-stained heart show infarct zones (white/brown areas). (E) Infarct size is expressed as a percentage of the total area of LV. n=5 for each group. Data are presented as mean±SEM, ** *P*<0.01



Supplemental Figure 2. Role of GSK-3 α in post-MI hypertrophy. Hypertrophy was analyzed in 8wk post-MI hearts. (A) Heart weight to body weight and (B) heart weight to tibia length ratios show comparable hypertrophy in KO vs WT, n= 10-17 for MI and n=4 for sham group. (C) Representative images show trichrome-stained heart sections from remote myocardium (D) Cardiomyocyte cross sectional area in WT and KO shows comparable hypertrophy. n=5-9 for WT and KO hearts.



Supplemental Figure 3; Role of GSK-3 α in post-MI remote fibrosis. (A) Representative images of Masson's trichrome-stained post-MI heart sections (8 wks). (B) Quantification shows comparable remote fibrosis and is presented as percent of fibrotic area measured, n=10-14 WT and KO hearts. (C) Immunoblotting for fibrotic markers, α -smooth muscle actin (α -SMA) and vimentin in post-MI LV lysates (3 wks). (D) Results show no significant changes in either markers between KO and controls, n=4-5 for WT and KO.

Supplemental Fig. 4



Supplemental Figure 4. GSK-3 α does not regulate post-MI NF-kB signaling. (A) Immunoblot from post-MI WT and *Gsk3* α KO hearts for activity and expression of NF-kB (P65). (B) Immunoblot quantification shows comparable NF-kB activity in WT and KO post-MI hearts. n=4-5 for WT and KO.

Supplemental Fig. 5



Supplemental Figure 5. Cardiac specific conditional deletion of $Gsk3\alpha$ does not affect post-MI m-TORC1 signaling; Immunoblot of WT and $Gsk3\alpha$ KO post-MI hearts for the m-TORC1 targets, including (A) p70S6 kinase (B) S6 ribosomal protein (C) 4E-BP1 show comparable activation. n=4-5 for WT and KO.



Supplemental Figure 6; Cyclin dependent kinases (Cdks), Cyclin D1 and Wnt beta catenin pathways in *Gsk3*α KO heart. (A) Immunoblotting for Cdk2, Cdk4 and cyclin D1, (B) Beta catenin and Axin-2 level in 3 wk post-MI LV lysate. (C) There were no significant changes in these markers between KO and controls. n=4-5 for WT and KO.