

Figure. S1. Volcano plot highlighting specific examples of proteins changing between ACi and Control: yellow nodes demonstrate proteins that were significantly upregulated in ACi vs Control ; Blue nodes demonstrate proteins that were significantly downregulated in ACi vs control

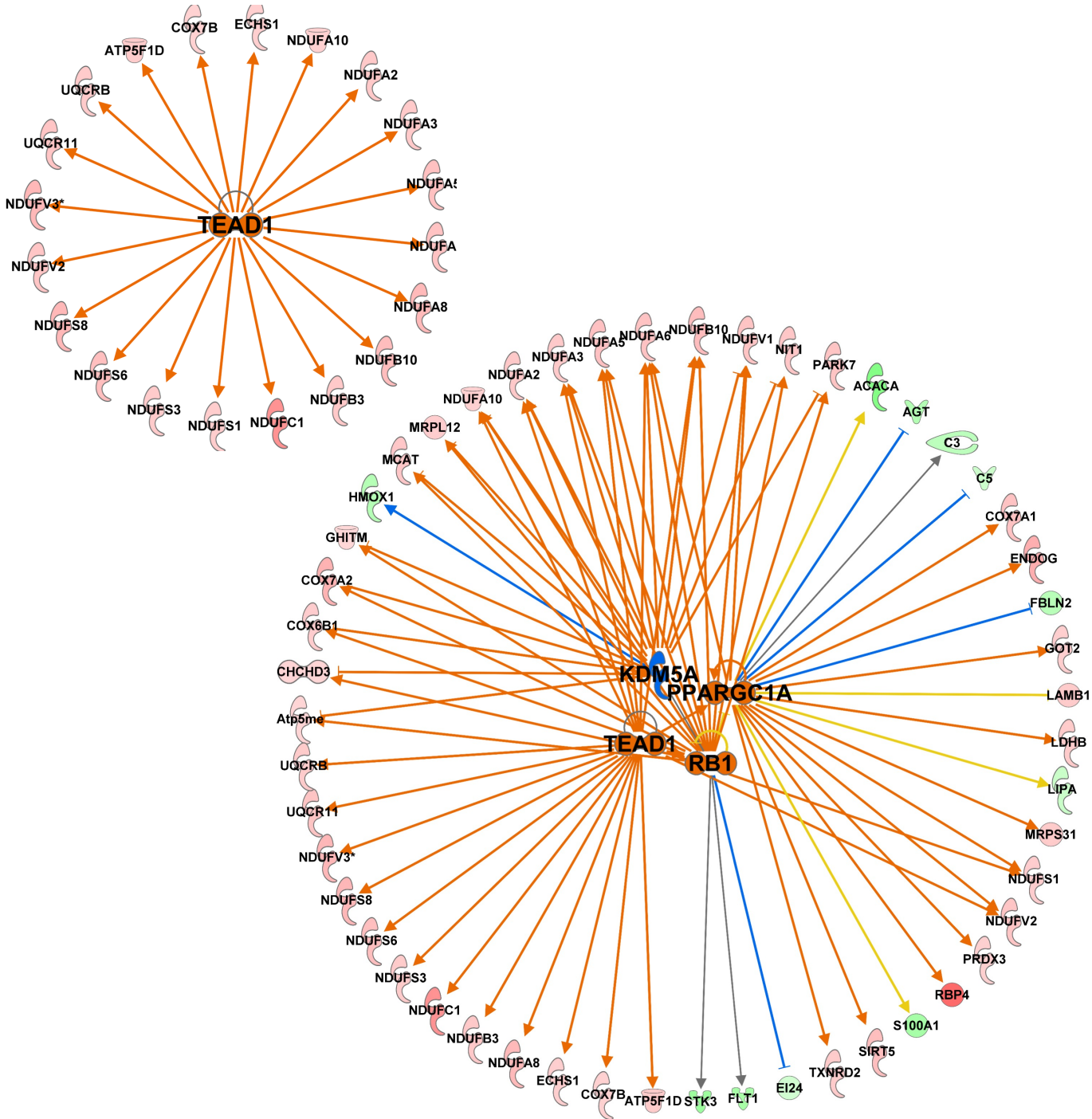


Figure. S2. A network diagram of transcriptional regulators, including TEAD1, KDM5A, PPARGC1A, and RB1 previously shown in Figure 2.G.

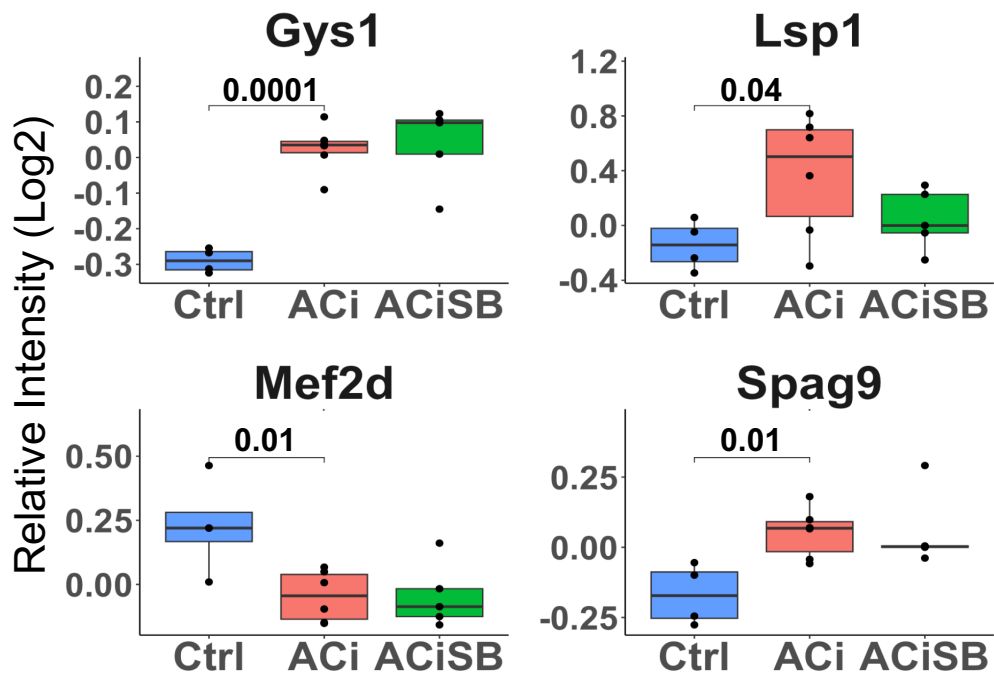


Figure S3. Expression of many p38 MAPK substrates does not change in ACi: Global significance ($p < 0.05$) established via F-test, with p-values derived from LIMMA contrast matrices for inter-group comparisons. P-values < 0.05 are indicated by faceted brackets.

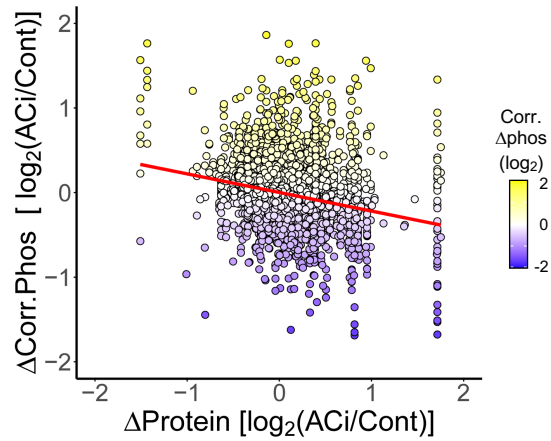


Figure S4. Correcting phosphorylation signals for underlying protein abundance substantially reduces the dependence of phosphorylation on protein dynamics in HF.

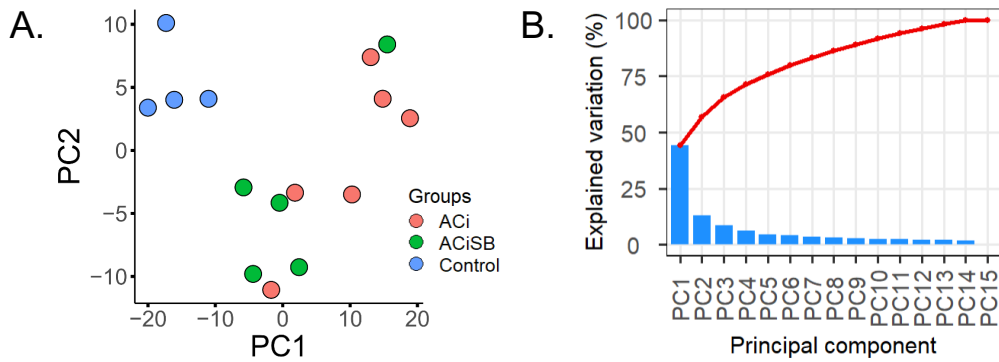


Figure S5. Phosphoproteome, uncorrected for protein abundance. A. PCA biplot indicates of the ACi phosphoproteome from control. SB mitigates remodeling, though changes in protein abundance accounts for a large measure of the variance. B. Scree plot indicating the contribution of each principal component to experimental variance.

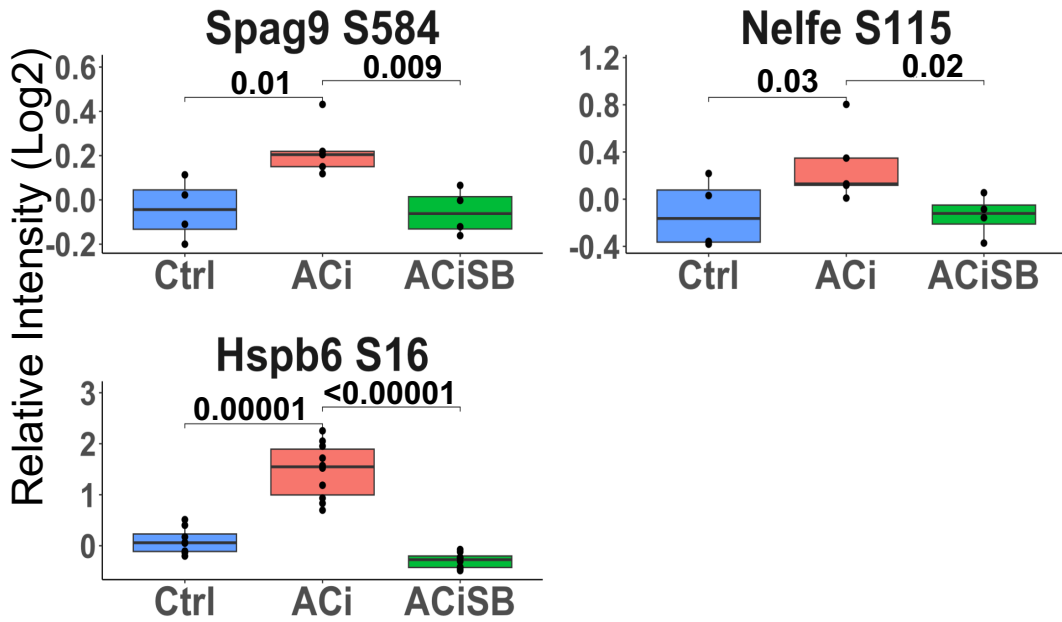


Figure S6. Phosphorylation of known p38 MAPK substrates and targets : Global significance ($p < 0.05$) established via F-test, with p-values derived from LIMMA contrast matrices for inter-group comparisons. P-values < 0.05 are indicated by faceted brackets.

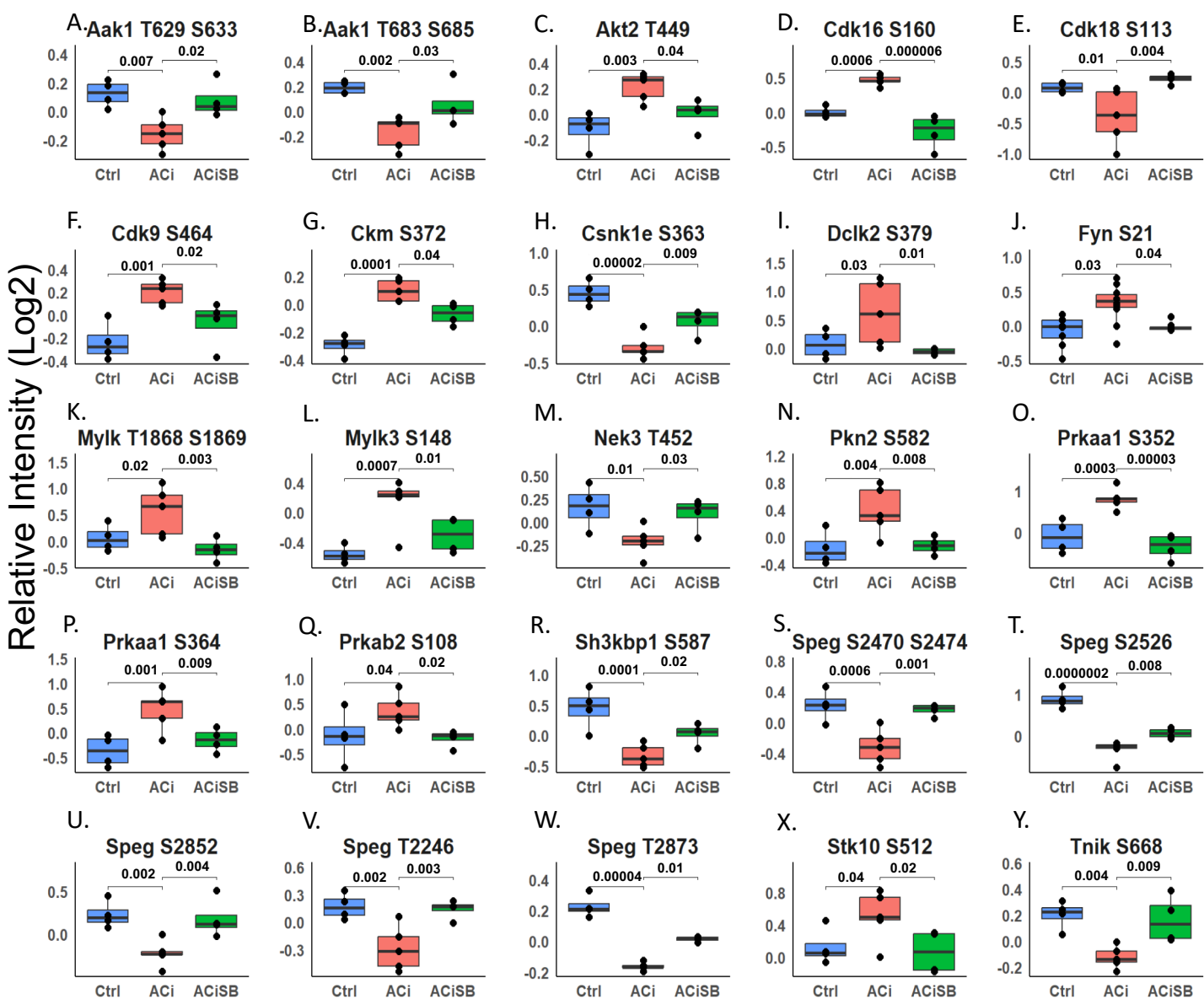


Figure S7. ACISB-Responsive Kinase Phosphorylation Global significance ($p < 0.05$) established via F-test, with p-values derived from LIMMA contrast matrices for inter-group comparisons. Only a subset of results is shown in the figure. P-values < 0.05 are indicated by faceted brackets.