**Supplementary Figure Legends**

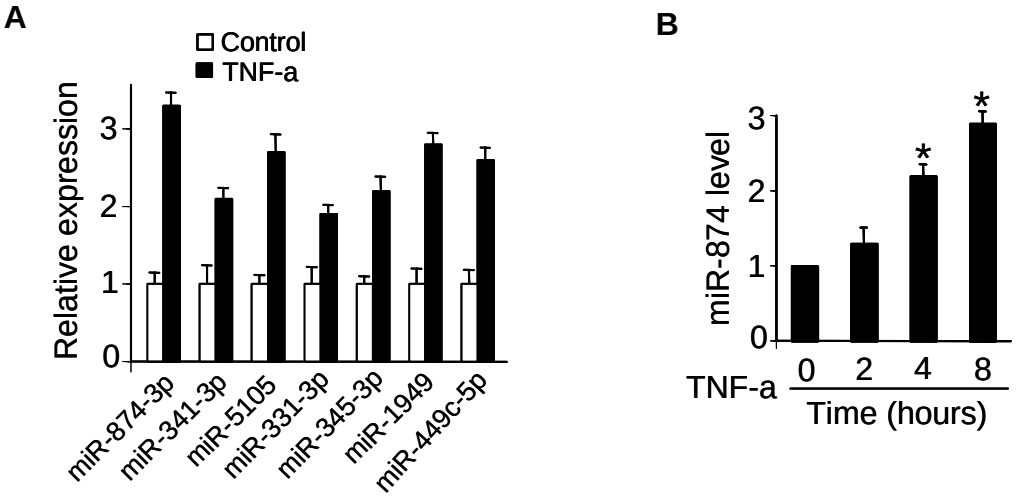
**Supplementary Figure 1. The expression levels of miRNAs in TNFα induced necrotic cell death in caridomyocytes. A.** Upregulated miRNAs upon TNFα treatment. Cardiomyocytes were treated with 10 ng/ml human TNFα for 24 hr. The expression levels of miRNAs were analyzed by qRT-PCR. **B.** qRT-PCR analysis of miR-874. Cardiomyocytes were treated with 10 ng/ml human TNFα at the indicated time, and the expression level of miR-874 was analyzed. *p<0.05 vs control.

**Supplementary Figure 2. The RIPK1/RIPK3 is involved in H2O2 induced necrotic cell death. A** and **C.** The expression levels of RIPK1 or RIPK3 upon $H_2O_2$ treatment. Cardiomyocytes were treated with $H_2O_2$ at the indicated time, and the expression of RIPK1 or RIPK3 was analyzed by western blot. **B** and **D**. Knockdown of RIPK1 or RIPK3 can attenuate $H_2O_2$ induced necrotic cell death. Cardiomyocytes were infected with adenovirus RIPK1-siRNA (B) or RIPK3-siRNA (D) and their scramble form (RIPK1-sc or RIPK3-sc), then exposed to $H_2O_2$. Cells were harvested at 48h after treatment for the analysis of RIPK1 or RIPK3 levels and PI exclusion. *$p$<0.05 vs $H_2O_2$ alone.

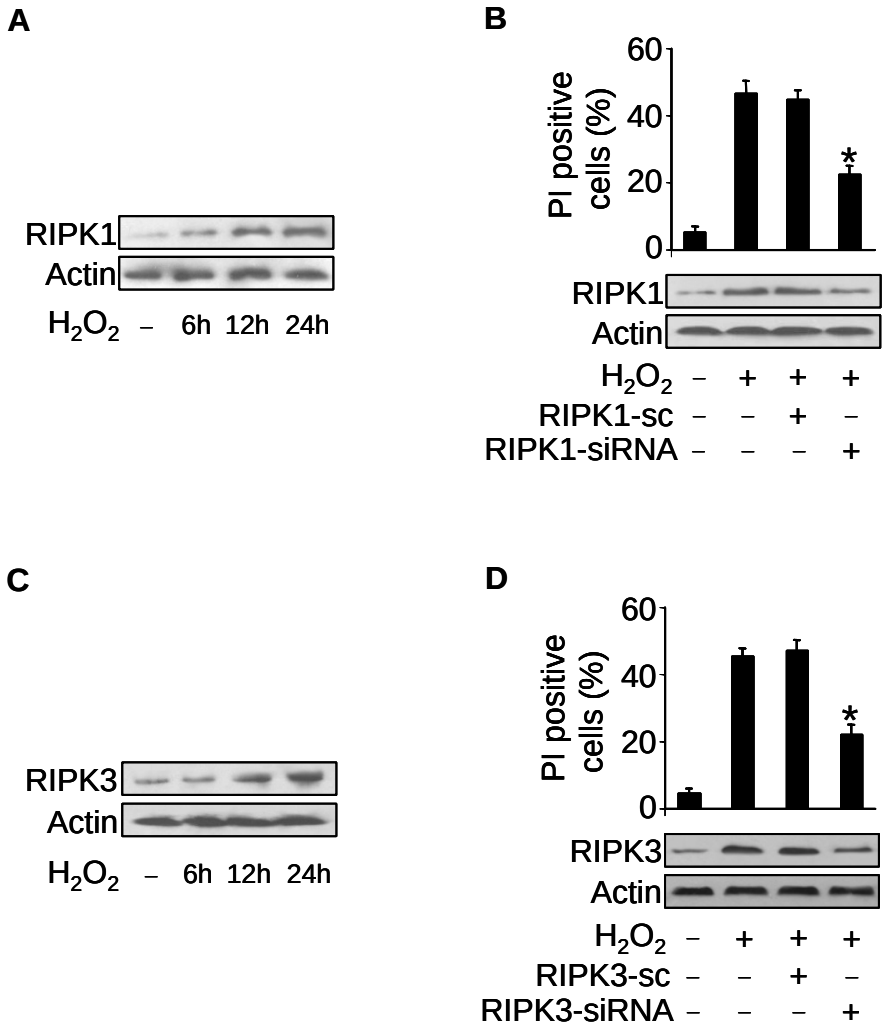
**Supplementary Figure 3. Foxo3a KO mice exhibit increased I/R injury.** WT and Foxo3a KO mice were subjected to I/R as described in methods. **A**. Caspase-8 level were analyzed by western blot. **B**. Foxo3a KO mice increased myocardial infarction

upon I/R. *p<0.05 vs WT+I/R. **C**. SDS-soluble lysates were immunoblotted with antibody specific for the N-terminus of CYLD; the unprocessed CYLDp107 and the CYLDp25 cleavage product generated from the endogenous CYLD protein were shown.

**Supplementary Figure 1**



**Supplementary Figure 2**



**Supplementary Figure 3**