Supplementary Figure Legends

Supplementary Figure 1. Verification of cardiac-specific miR-30b transgenic mice. A. Schematic map showing the transgenic construct of miR-30b. **B.** Detection of miR-30b levels in miR-30b transgenic mice. The expression of miR-30b was analyzed by qRT-PCR from wild type and five different lines of miR-30b transgenic mice, and the results were normalized to that of U6. **C.** qRT-PCR analyzes the expression of mature miR-30b in different tissues isolated from miR-30b transgenic (Tg) mice Line#1 and wild-type mice (WT). **D.** miR-30b transgenic mice developed normally without obvious phenotype and cardiac function alterations under basal conditions. Fractional shortening (FS) was shown. (n=10 per group).

Supplementary Figure 2. miR-30b transgenic mice Line#4 suppress the expression of CypD and myocardial infarction. A. miR-30b transgenic mice Line#4 suppress the expression of CypD. CypD expression was analyzed by immunoblot from WT and miR-30b transgenic mice Line#1 and Line#4. **B.** miR-30b transgenic mice Line#4 inhibit myocardial infarction. miR-30b transgenic mice Line#4 (Tg Line#4) and wild type mice (WT) were subjected to I/R as described in methods.. Infarct size was shown. n=7. *p <0.05 versus WT+I/R.

Supplementary Figure 3. A. NF-κB and BCL-2 are not regulated in H_2O_2-induced necrotic pathway. Cardiomyocytes were treated with 750μM H_2O_2 at indicated time. NF-κB and BCL-2 levels were analyzed by immunoblot. **B.** E2F1 inhibits the

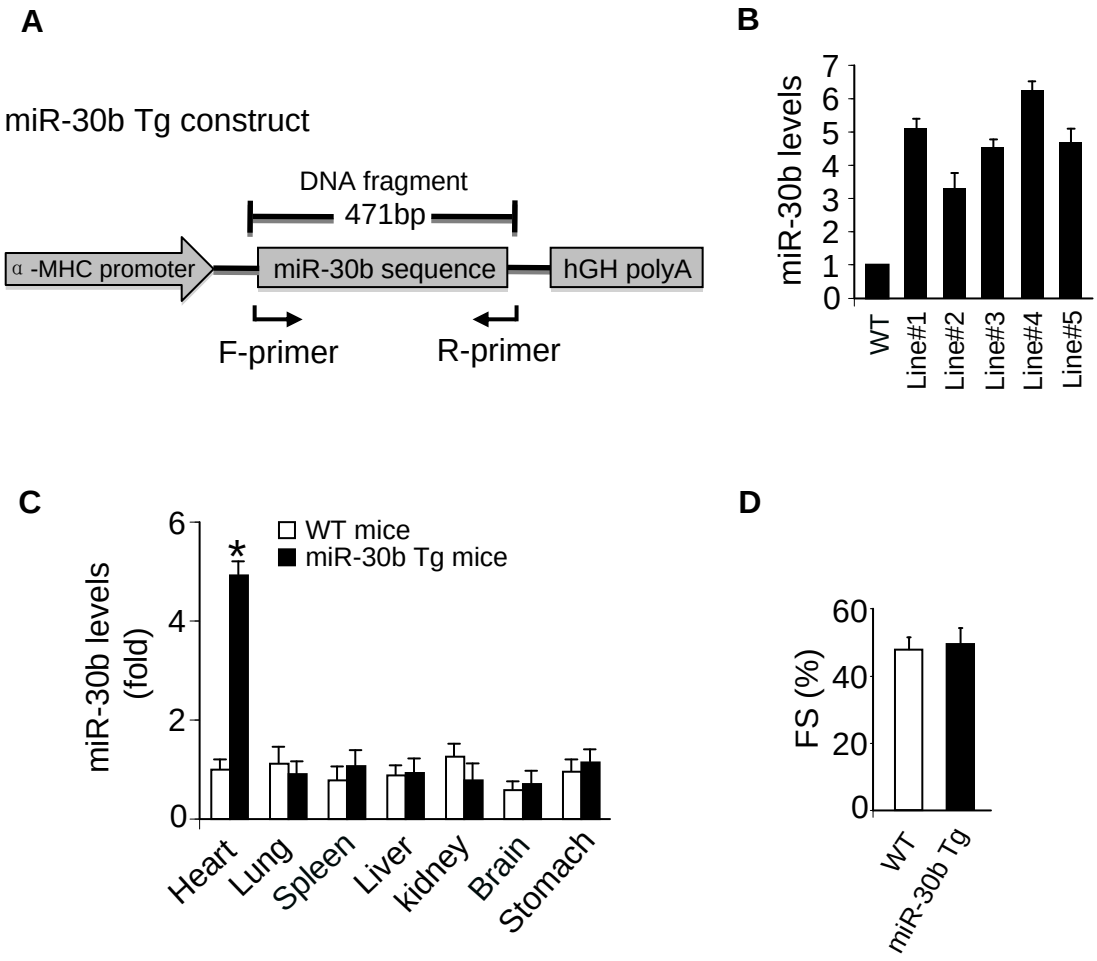
expression of miR-30b precursor (pre-miR-30b) and mature miR-30b. Cardiomyocytes were infected with adenoviral β-gal or E2F1. Pre-miR-30b and miR-30b levels were analyzed by northern blot. **C.** Pre-miR-30b and miR-30b levels in E2F1 deficient mice. Pre-miR-30b and miR-30b levels were analyzed by northern blot from E2F1 knockout mice ($E2F1^{-/-}$) and wild type littermates (WT). **D.** E2F1 has no effect on the CypD promoter activity. Cardiomyocytes were treated with the adenoviral β-gal, E2F1, E2F1-siRNA or E2F1-sc, the constructs of the empty vector (pGL-4.17), the CypD promoter, respectively. Luciferase activity was assayed.

Supplementary Figure 4. A. Representative staining of histological sections in WT and E2F1 knockout mice (E2F1 KO) by an endothelial marker, CD31 to analyze capillary density (left panel). Quantification analysis of capillaries per microscopic field is shown in right panel. **B.** Representative double TUNEL/CD31 staining in I/R treated WT and E2F1 KO mice. Immunohistochemistry was performed in hearts from WT and E2F1 KO mice subjected to ischemia/reperfusion. Endothelial cells were identified by anti-CD31 (red), apoptosis by TUNEL (green) and nuclei were counterstained with DAPI (blue). Quantitative assessment of percentage of apoptotic endothelial cells/total endothelial cells is shown in right panel.

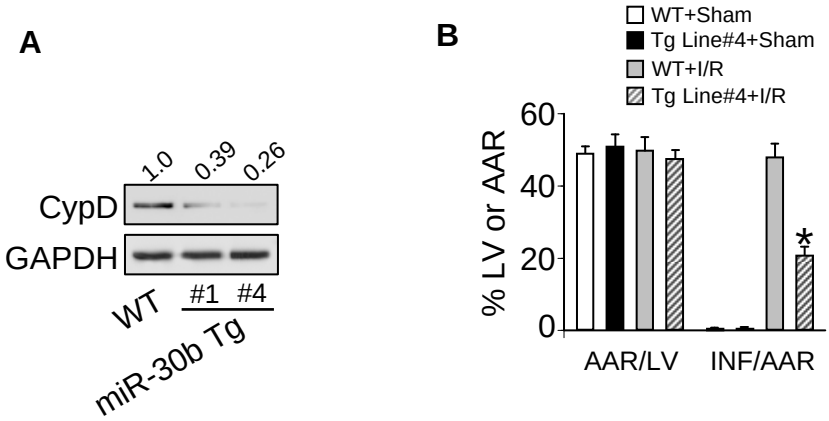
Supplementary Figure 5. The levels of miR-30b and CypD in macrophages isolated from WT and E2F1 knockout mice. A. WT and E2F1 knockout mice were subjected to I/R as described in methods. Macrophages were isolated from WT and

E2F1 KO mice, the expression levels of miR-30b were analyzed by qRT-PCR. **B.** CypD expression levels were analyzed by immunoblot.

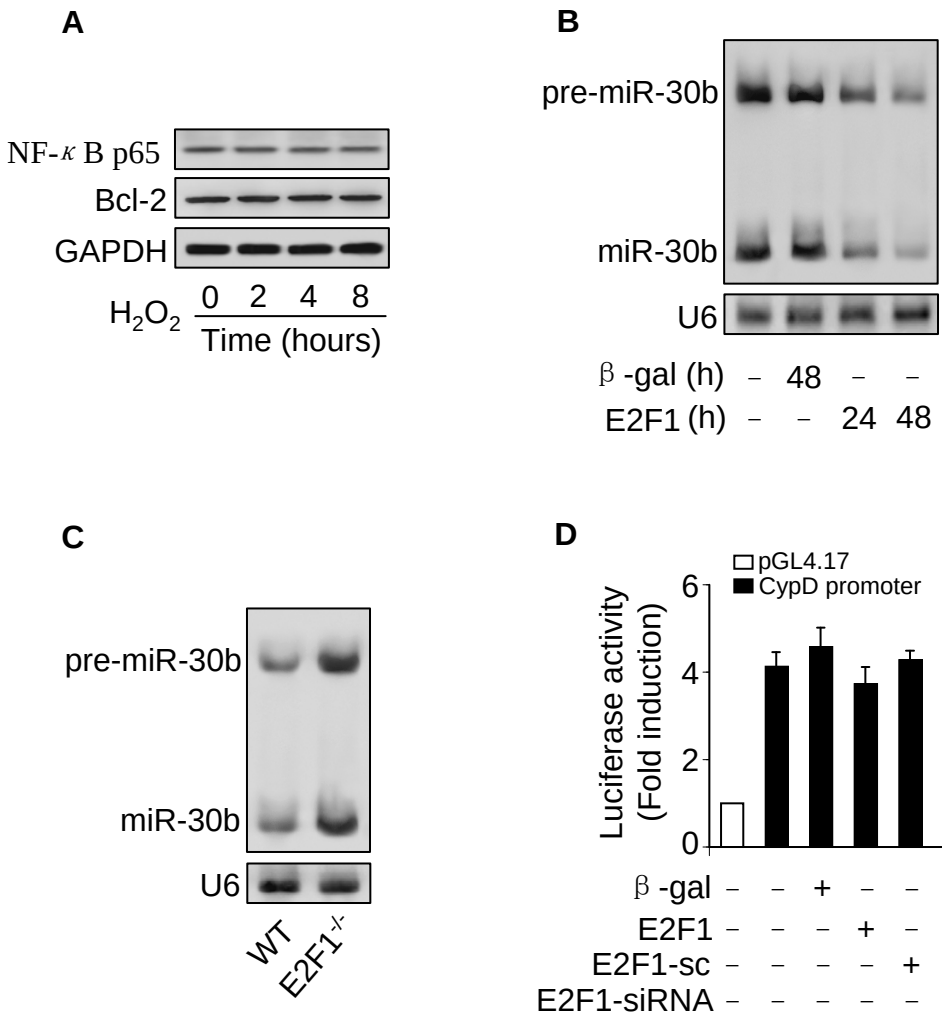
Supplementary Figure 1



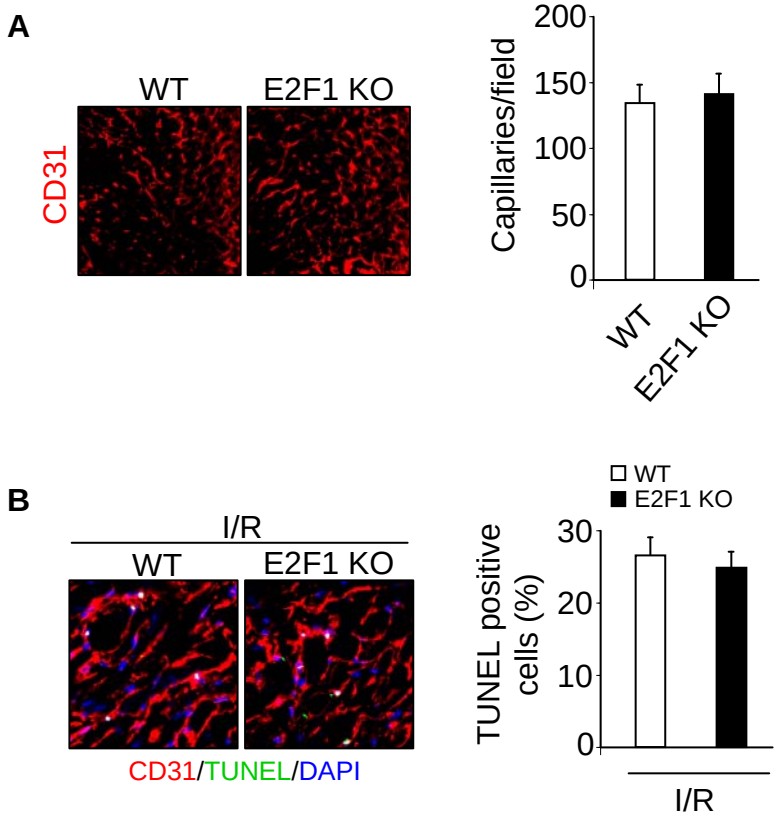
Supplementary Figure 2



Supplementary Figure 3



Supplementary Figure 4



Supplementary Figure 5