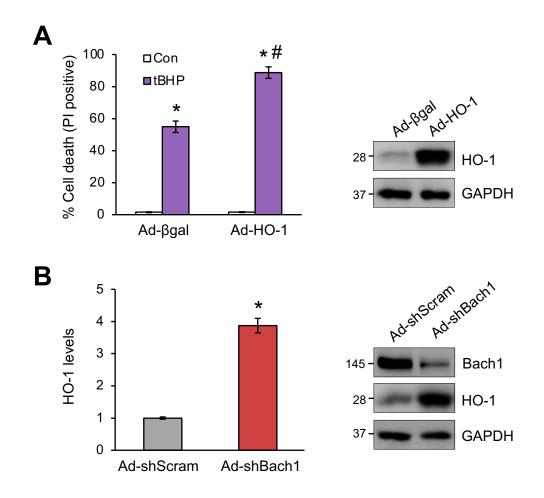
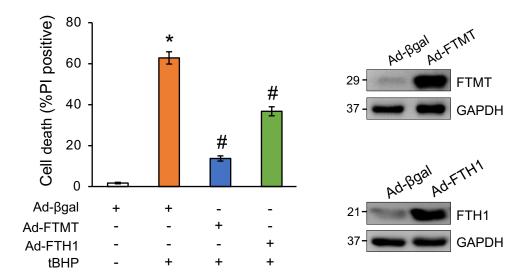


Supplemental Figure 1. Inactivation of HO-1 reduced tBHP-induced labile iron levels and cell death in cardiomyocytes. A. Assessment of labile iron pool in neonatal rat cardiomyocytes treated with tBHP in the presence and absence of ZnPP for 8 h. B. Quantification of cell death from cells treated as in A for 12 h.  $^*P$ < 0.05 vs Control;  $^*P$ < 0.05 vs tBHP. n = 3.

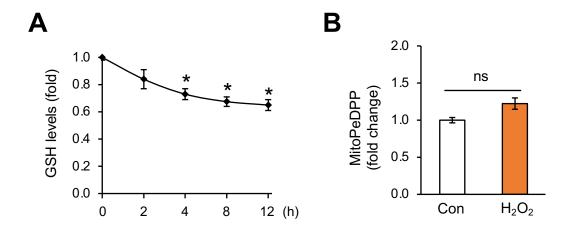


Supplemental Figure 2. Overexpression of HO-1 promoted tBHP-induced cell death in cardiomyocytes. A. Cell death was assessed from cardiomyocytes infected with Ad- $\beta$ gal or Ad-HO-1 for 24 h followed by treatment with tBHP or vehicle control for 12 h. P< 0.05 vs Control; #P< 0.05 vs Ad- $\beta$ gal + tBHP. n = 3. B. HO-1 expression in cardiomyocytes infected with adenoviral vectors for Bach1 shRNA (Ad-shBach1) or scrambled control (Ad-shScram) for 24 h. P< 0.05 vs Ad-shScram. n = 3.

## **Supplemental Figure 3**



Supplemental Figure 3. Overexpression of mitochondrial ferritin (FTMT) inhibited tBHP-induced cell death in cardiomyocytes. Cell death was assessed from cardiomyocytes infected with Ad- $\beta$ gal, Ad-FTMT, or Ad-FTH1 for 24 h followed by treatment with tBHP or vehicle control for 12 h. Ad-FTH1, adenovirus expressing ferritin heavy chain-1. FTMT and FTH1 expression levels were assessed by Western blotting. P< 0.05 vs Ad- $\beta$ gal; #P< 0.05 vs Ad- $\beta$ gal + tBHP. n = 3.



Supplemental Figure 4. Effect of  $H_2O_2$  on GSH levels and mitochondrial lipid peroxidation in cardiomyocytes. A. GSH levels in cardiomyocytes treated with 200  $\mu$ M  $H_2O_2$  for the indicated time periods. \*P< 0.05 vs 0 h. n = 3. B. Mitochondrial lipid peroxidation assessed with MitoPeDPP in cardiomyocytes treated with 200  $\mu$ M  $H_2O_2$  for 8 h. ns, non-significant. n = 3.

## Full length blots

