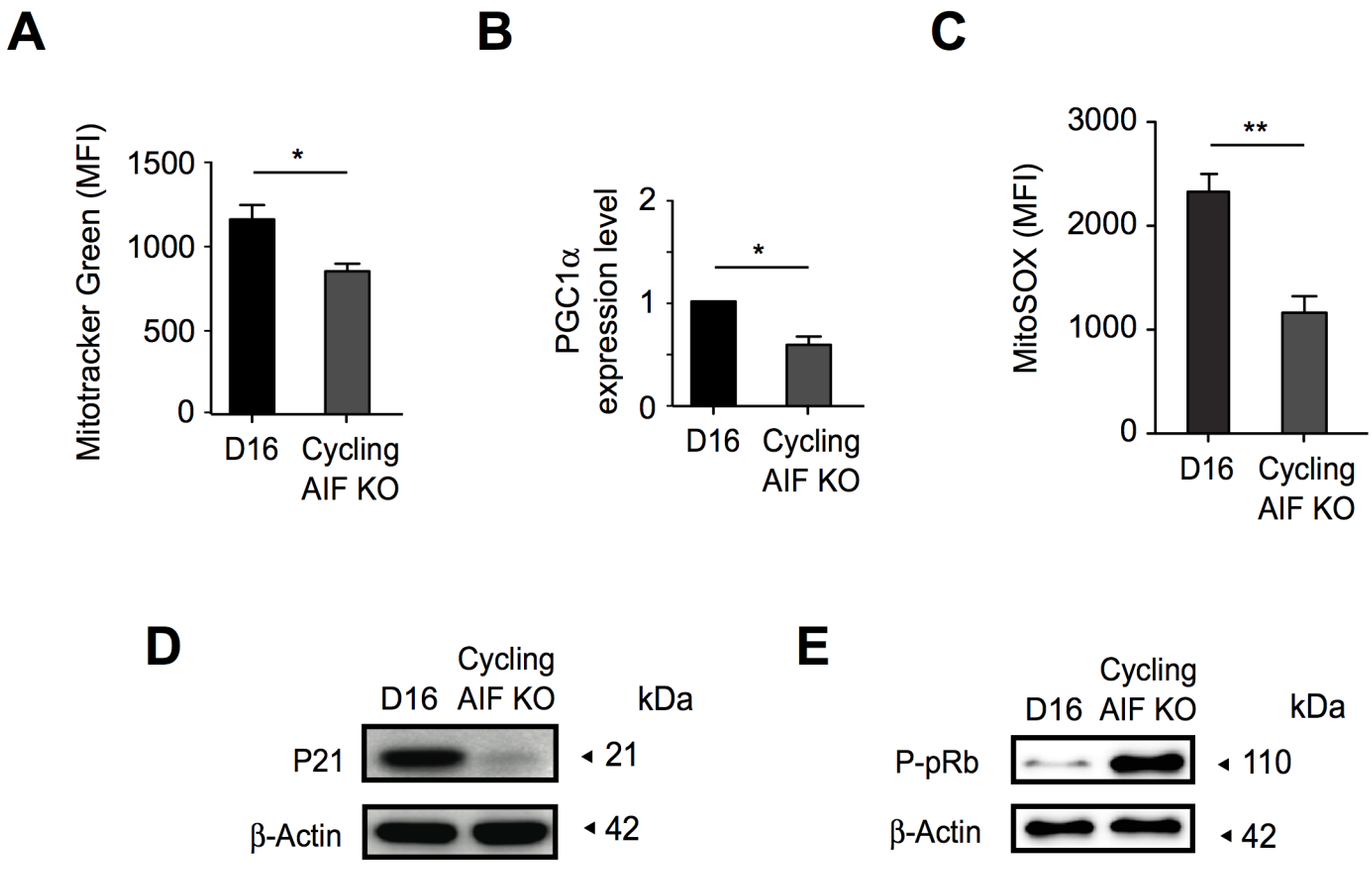

Supplemental Figure 4. Cycling AIF KO cells inactivated cell cycle arrest and P21-mediated senescence by diminishing mitochondrial mass and mitochondrial ROS generation. (**A**) Mitochondrial mass of senescent AIF-/Y MEFs (D16) and cycling AIF KO cells assessed in a flow cytometer by Mitotracker Green labeling (n = 5).(**B**) PGC1α mRNA levels determined by qPCR in senescent AIF-/Y MEFs (D16) and cycling AIF KO cells (n = 6). The 18S mRNA expression was used to normalize data. The results were graphed as a ratio relative to D16 cells (mean of data set at 1.0). (**C**) Mitochondrial ROS levels recorded by flow cytometry in senescent AIF-/Y MEFs (D16) and cycling AIF KO cells (n = 7). Data were obtained in 10,000 cells and ex pressed as mean fluorescence intensity (MFI). (**D**) Representative immunoblot of senescent AIF-/Y MEFs (D16) and cycling AIF KO cells revealing the cell cycle inhibitor P21. Equal cellular loading was confirmed by β-Actin probing (n = 3 experiments with similar results). (**E**) Representative immunoblot of senescent AIF-/Y MEFs (D16) and cycling AIF KO cells revealing the levels of pRb phosphorylation. Equal cellular loading was confirmed by β-Actin probing (n = 3 independent experiments with similar results). Statistical significance was calculated by Mann Whitney (**A**) or student t (**B**, **C**) tests. Bars represent mean ± SEM.