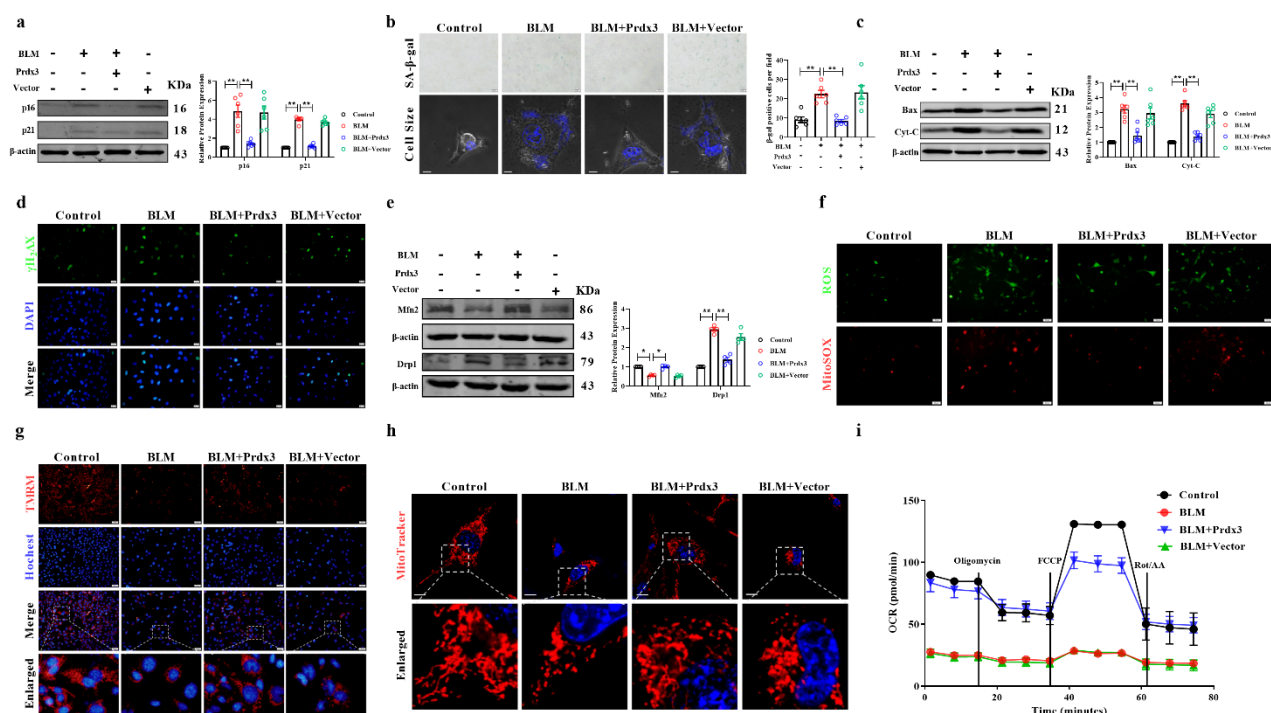
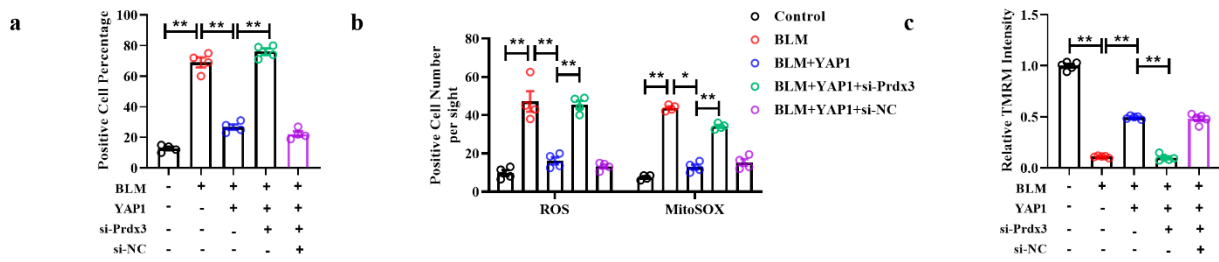


Supplementary Materials



Supplementary Fig. 1 Overexpression of Prdx3 inhibits BLM-induced senescence and mitochondrial dysfunction.

(a) Western blot results of protein expression of p16 and p21 in MLE-12 cells transfected with Prdx3 after BLM treatment, $n=6$. (b) SA- β -gal staining for cellular senescence of MLE-12 cells transfected with Prdx3 after BLM treatment, $n=6$. Scale bars, 50 μ m. The cell size was determined by confocal microscopy; nuclei were stained with Hoechst 33342 (blue). Scale bars: 10 μ m. (c) Western blot results of protein expression of Bax and Cyt-C in MLE-12 cells transfected with Prdx3 after BLM treatment, $n=6$. (d) Immunofluorescence staining assay of γ H₂AX in MLE-12 cells treated with Prdx3 plasmid. Scale bars: 20 μ m. (e) The protein level of Drp1, and Mfn2 was determined by western blot, $n=4$. (f) Cytosolic and mitochondrial ROS in Prdx3 over-expressing MLE-12 cells after BLM administration was assessed by DCFH-DA probe and MitoSOX. Scale bars: 50 μ m. (g) Representative images obtained from MLE-12 cells stained with TMRM (red) and Hoechst 33342 for nuclei (blue). Scale bars: 50 μ m. (h) Representative images acquired by confocal microscopy showing the morphological change of mitochondria. Scale bars: 10 μ m. (i) O₂ consumption rate was assessed using a seahorse mitochondrial stress test assay. Data are presented as the mean \pm SEM. ** $P < 0.01$ (Ordinary one-way ANOVA).



Supplementary Fig. 2 Silencing Prdx3 restores cellular senescence and mitochondrial dysfunction inhibited by YAP1.

(a) Statistics of γ H₂AX in MLE-12 cells treated with YAP1 or silencing Prdx3 following BLM induction. *n*=4. (b) Statistics of ROS and MitoSOX in Prdx3 over-expressing MLE-12 cells after BLM administration. *n*=4. (c) Statistics of MLE-12 cells stained with TMRM. *n*=5. **P* < 0.05, ***P* < 0.01.