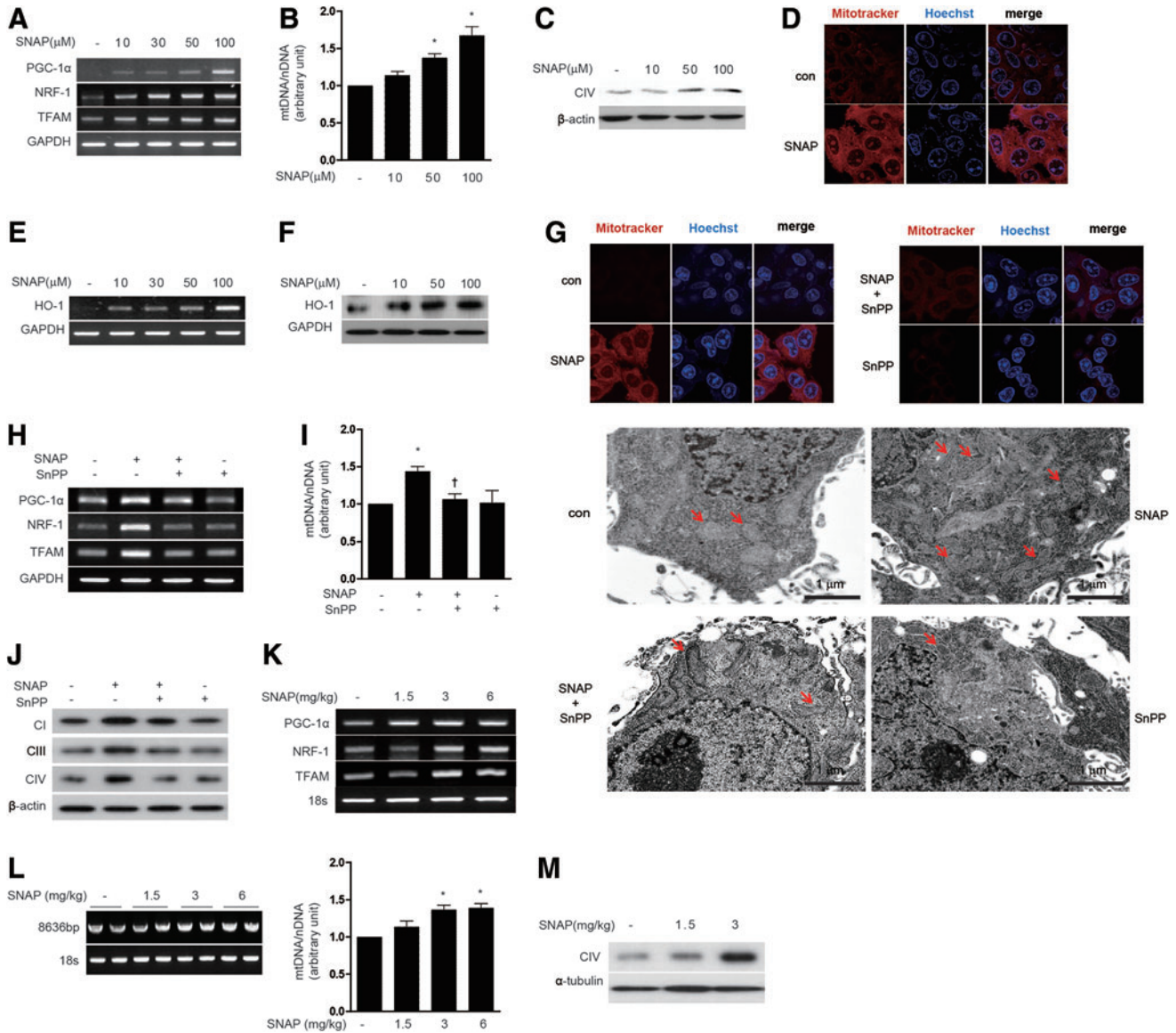


Supplementary Data



SUPPLEMENTARY FIG. S1. NO induces mitochondrial biogenesis through the induction of HO-1. (A–F) HepG2 cells were treated with SNAP at the indicated concentrations (0–100 μ M) for 12 h. (G) HepG2 cells were pretreated in the absence or presence of SnPP (20 μ M), and then treated with 100 μ M SNAP for 12 h. (H–J) AML12 cells were pretreated in the absence or presence of SnPP (20 μ M), and then treated with 100 μ M SNAP for 12 h. (A, H) Expression levels of PGC-1 α , NRF-1, and TFAM were measured by RT-PCR. (B, I) The relative mtDNA content was measured by real time-PCR. (C, J) The expression of CI (complex I), CIII (complex III), and CIV (complex IV) protein was analyzed by western blotting. β -actin served as the standard. (D, G) Mitochondrial mass was assessed by using MitoTracker[®] Red CMXRos staining (red). Nuclei were stained with Hoechst dye (blue). Images of fluorescence were analyzed by confocal microscopy. Mitochondrial biogenesis and morphology was shown by electron microscopy study. Scale bar, 1 μ m (E) HO-1 mRNA levels were measured by RT-PCR (F) HO-1 protein levels were measured by western blotting. (K–M) C57BL/6 mice were injected with SNAP at the indicated doses. (K) Expression of PGC-1 α , NRF-1, and TFAM mRNA were measured by RT-PCR. (L) The mtDNA content was measured by Expand Long Template PCR. (M) The expression of CIV (complex IV) was analyzed by western blotting. All experiments were performed in triplicate, and representative data are shown. Data are expressed as mean \pm SEM. * p < 0.05 compared with untreated control cells (or un-injected control group); [†] p < 0.05 compared with cells treated with SNAP alone. HO-1, heme oxygenase-1; mtDNA, mitochondrial DNA; NO, nitric oxide; NRF-1, nuclear respiratory factor-1; PGC-1 α , peroxisome proliferator-activated receptor gamma coactivator-1 alpha; SNAP, S-nitroso-N-acetylpenicillamine; SnPP, tin-protoporphyrin-IX; TFAM, mitochondrial transcription factor-A; PCR, polymerase chain reaction.