

PGC-1 α overexpression suppresses blood pressure elevation in DOCA-salt hypertensive mice

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ONLINE SUPPLEMENTAL MATERIALS

Supplemental Figures

Figure S1

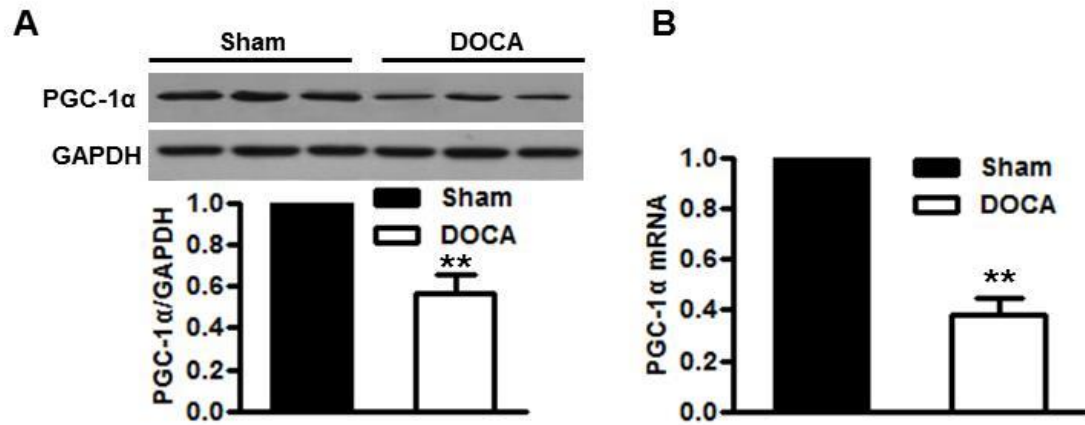


FIGURE S1 DOCA-salt treatment decreased PGC-1 α expression in mesenteric resistance arteries. (A, B) western blot (A) and quantitative PCR (B) results showed that PGC-1 α expression in mesenteric resistance arteries isolated from DOCA-salt mice was decreased. ** $P < 0.01$ vs. sham group, $n = 6$ /group.

Figure S2

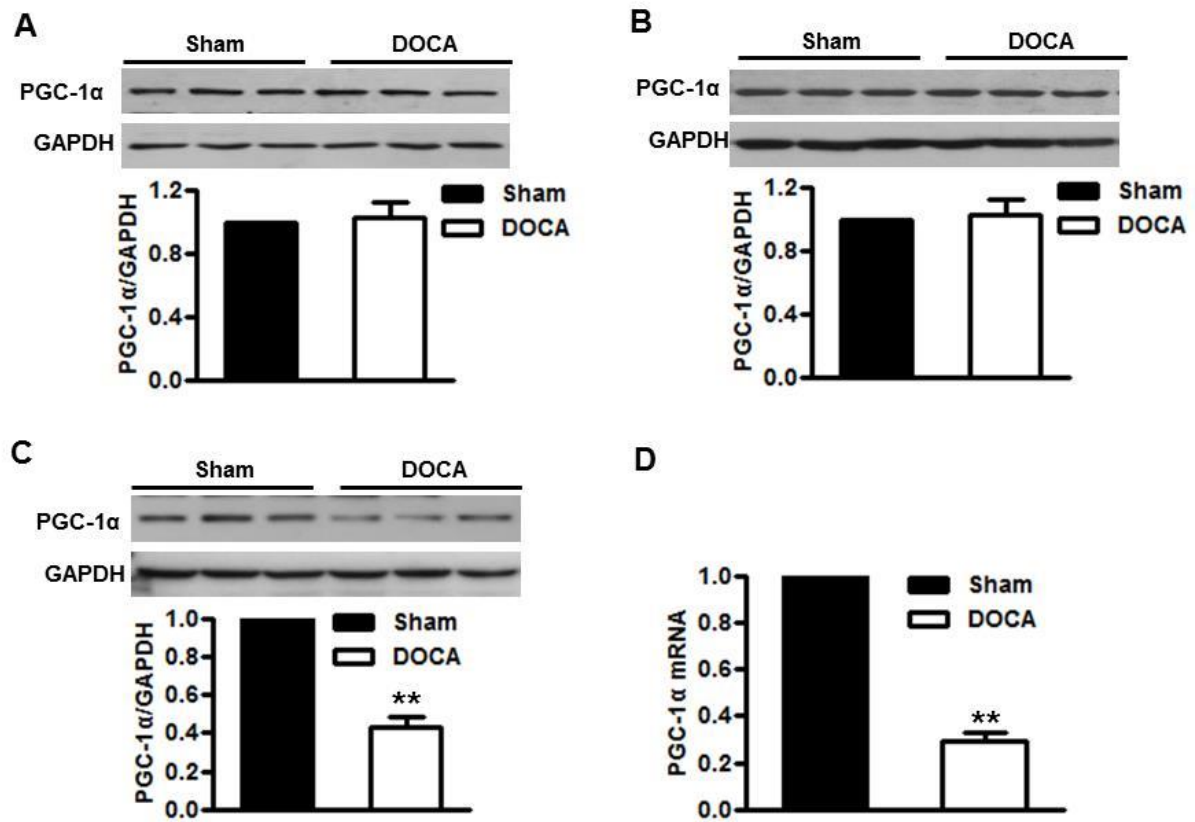


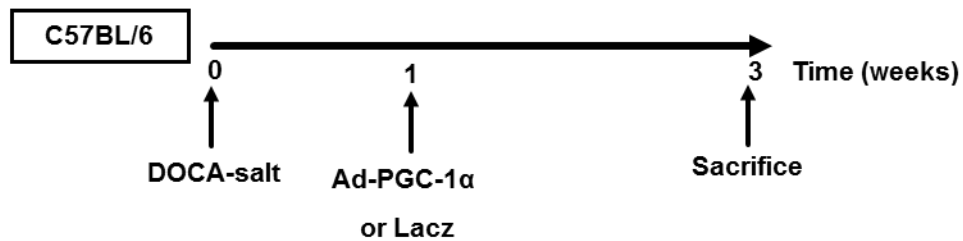
FIGURE S2 DOCA-salt treatment decreased PGC-1 α expression in MAECs. (A, B) PGC-1 α expression in aortas (A) and mesenteric resistance arteries (B) which both were stripped from endothelium following DOCA-salt treatment was analyzed by western blot. (C, D) western blot (C) and quantitative PCR (D) results showed that PGC-1 α expression in MAECs isolated from DOCA-salt mice was decreased.

** $P < 0.01$ vs. sham group, $n = 6$ /group.

Figure S3

A

Gene approach



B

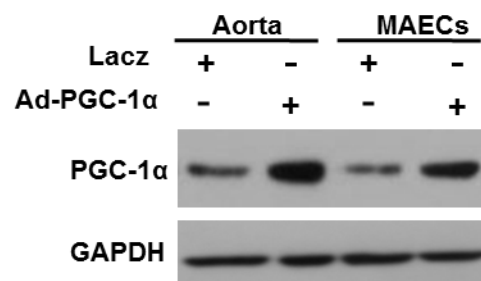


FIGURE S3 Animal experimental design. (A) A DOCA-salt hypertension model with a gene approach as schematically illustrated. (B) The infection efficiency of PGC-1α adenovirus was confirmed in aortas and MAECs by western blot. n=6/group.

Figure S4

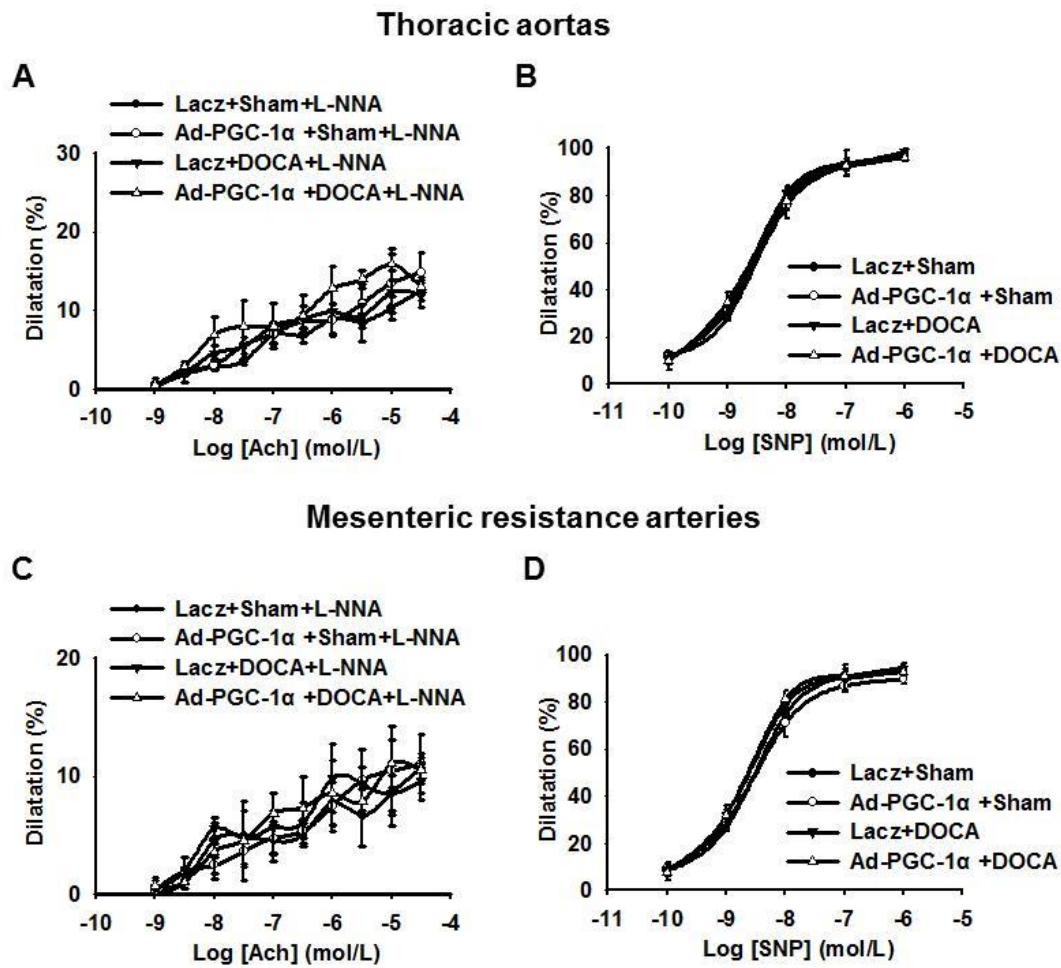


FIGURE S4 Effects of PGC-1 α on thoracic aortas and mesenteric resistance arteries reactivity. (A) Endothelium-dependent eNOS-independent relaxation in thoracic aortas was evaluated by measuring the response to acetylcholine (Ach, 10^{-9} - 10^{-5} mol/L) after incubation with the eNOS inhibitor N(G)-nitro-L-arginine (L-NNA, 10^{-4} mol/L). (B) Endothelium-independent relaxation to the NO donor sodium nitroprusside (SNP; 10^{-10} - 10^{-6} mol/L). (C, D) Endothelium-dependent eNOS-independent relaxation (C) and endothelium-independent relaxation (D) in mesenteric resistance arteries were measured as mentioned above. n=6-8/group.

Figure S5

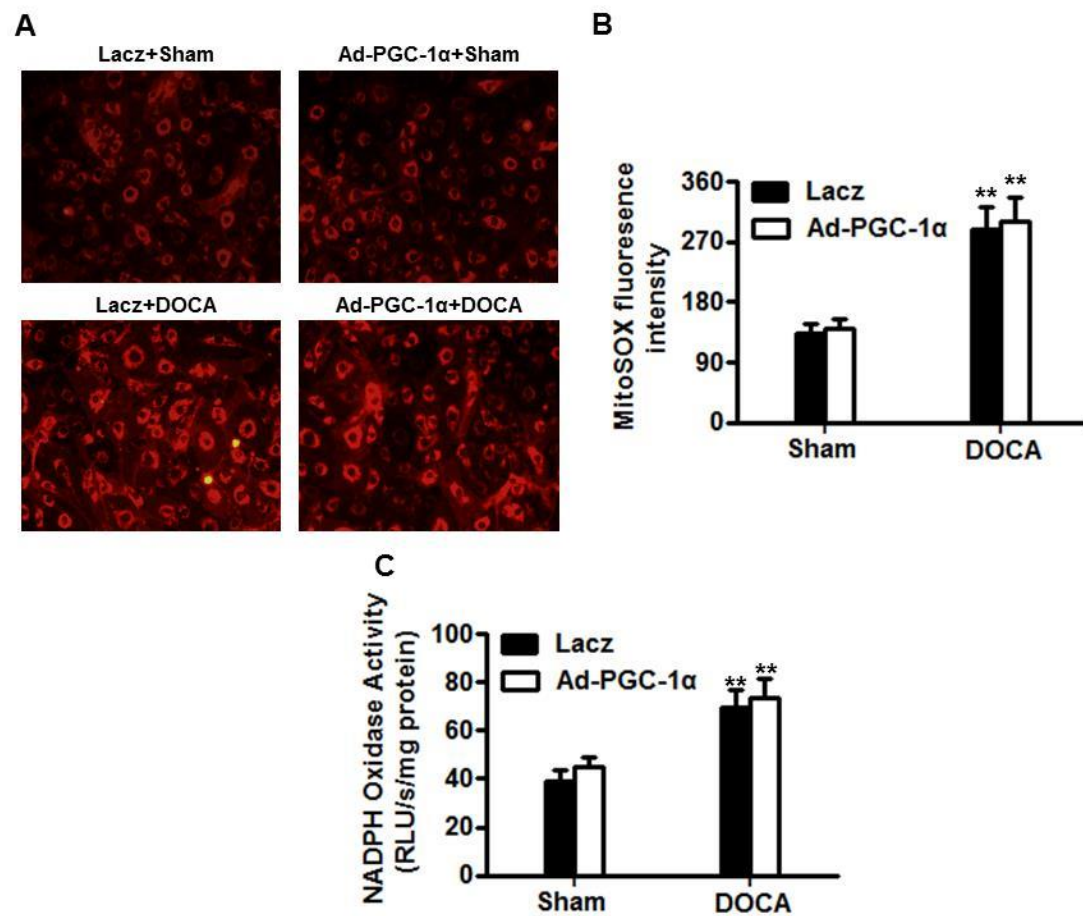


FIGURE 5 Effects of PGC-1 α overexpression on mitochondrial ROS generation and NADPH oxidase activity in MAECs. (A) Confocal microscope was used to localize mitochondrial ROS generation ($\times 200$). (B) Quantitative analysis of MitoSOX fluorescence intensity. (C) Quantitation analysis of NADPH oxidase activity measured in the presence of lucigenin (5 mmol/L) followed by treating with or without NADPH (100 μ mol/L). ** $P < 0.01$ vs. Lacz+sham.

Figure S6

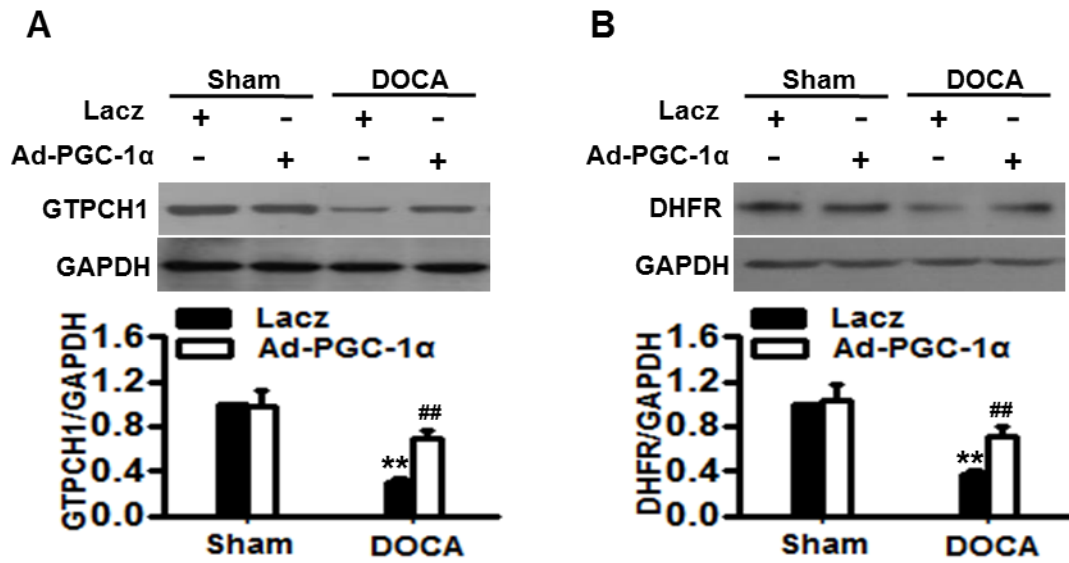


FIGURE S6 Effects of PGC-1 α on the expression of GTPCH1 and DHFR in aortas. (A, B) Western blot analysis of the expression of GTPCH1 (A) and DHFR (B) in aortas isolated from LacZ-infected and Ad-PGC-1 α -infected mice with or without DOCA-salt treatment. $n=6/\text{group}$. $^{**}P<0.01$ vs. LacZ+sham; $^{##}P<0.01$ vs. LacZ+DOCA.