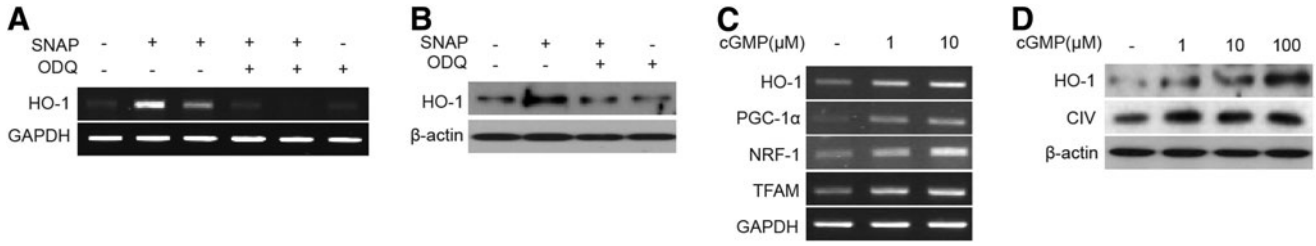

SUPPLEMENTARY FIG. S2. NO induces HO-1 expression through cGMP-dependent pathway. (A, B) HepG2 cells were pretreated in the absence or presence of ODQ (1 μM), an inhibitor of sGC, and then treated with SNAP (100 μM) for 12 h. **(A)** HO-1 mRNA levels **(B)** HO-1 protein **(C, D)** HepG2 cells were treated with the indicated concentrations of 8-Br-cGMP (cGMP) for 12 h. **(C)** PGC-1α, NRF-1, TFAM, and HO-1 mRNA levels. **(D)** CIV (complex IV) and HO-1 protein levels. All experiments were performed in triplicate, and representative data are shown. GAPDH and β-actin were used as a loading control in each experiment. 8-Br-cGMP, 8-bromoguanosine 3′,5′-cyclic monophosphate; cGMP, guanosine 3′,5′-monophosphate; GAPDH, glyceraldehyde 3-phosphate dehydrogenase; ODQ, 1H-[1,2,4]oxadiazolo[4,3-a]quinoxalin-1-one; sGC, soluble guanylate cyclase.