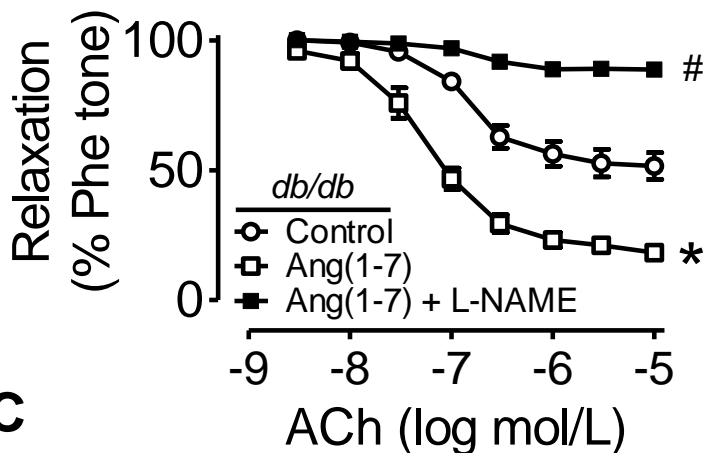
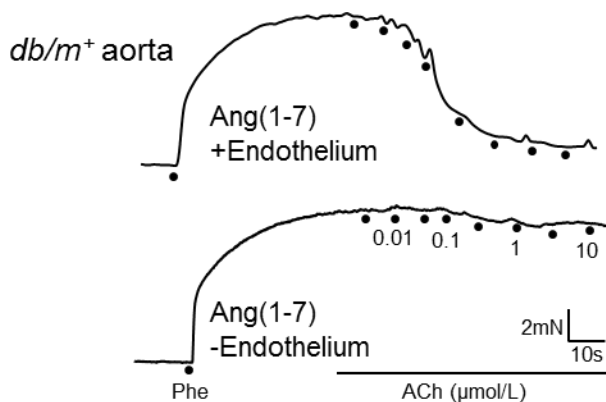


# Supplementary Fig. S3

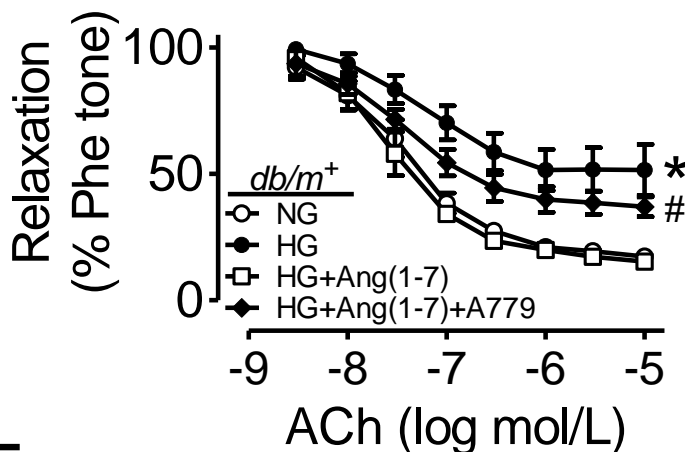
**A**



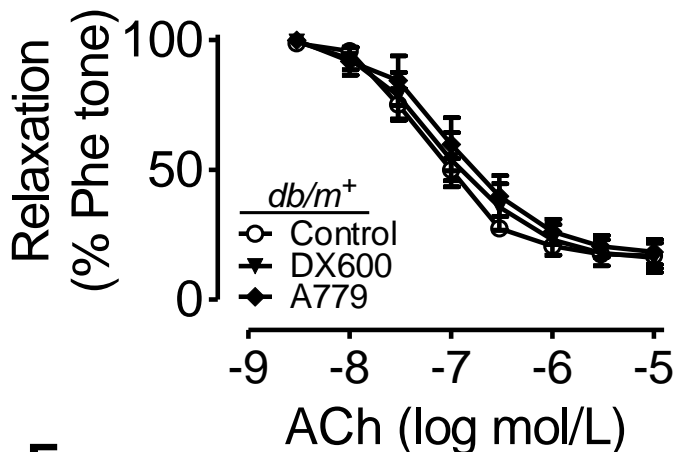
**B**



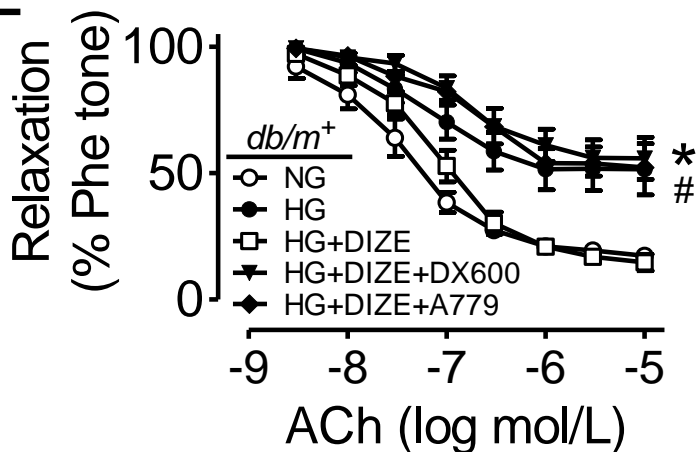
**C**



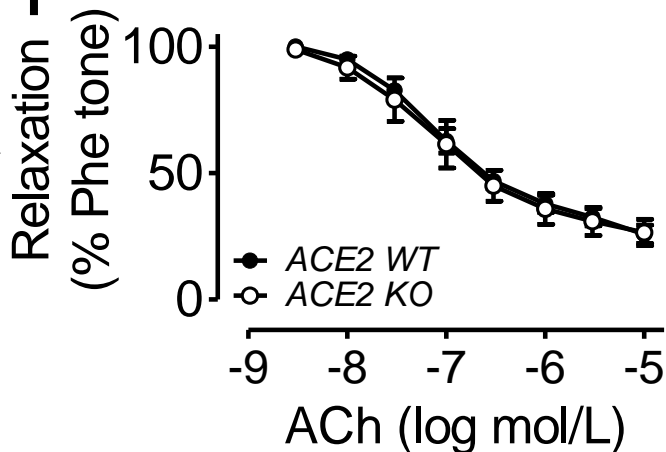
**D**



**E**



**F**



**Supplementary Fig. S3.** (A) ACh-induced EDRs in isolated mouse aortas from *db/db* mice following 24-hour exposure to Ang(1-7) (1  $\mu$ mol/L) and Ang(1-7) plus L-NAME (5  $\mu$ mol/L). (B) The representative traces of ACh-induced EDRs in *db/m+* mouse aortas with/without endothelium following 24-hour exposure to Ang(1-7) (1  $\mu$ mol/L). ACh-induced EDRs in *db/m+* mice following (C, E) 48-hour exposure to normal glucose (NG, 5 mmol/L), high glucose (HG, 30 mmol/L), HG plus relative drugs (DIZE: putative activator of ACE2) and (D) 48-hour exposure to ACE2 inhibitor DX600 (1  $\mu$ mol/L) or Ang(1-7) antagonist A779 (1  $\mu$ mol/L). (F) EDRs in aortas from ACE2 wild type (ACE2 WT) and ACE2 knockout (ACE2 KO) mice. Data are means  $\pm$  SEM of 5-6 mice. (A) \* $p$ <0.05 vs Control, # $p$ <0.05 vs Ang(1-7); (C) \* $p$ <0.05 vs NG, # $p$ <0.05 vs HG+Ang(1-7). (E) \* $p$ <0.05 vs NG, # $p$ <0.05 vs HG+DIZE.