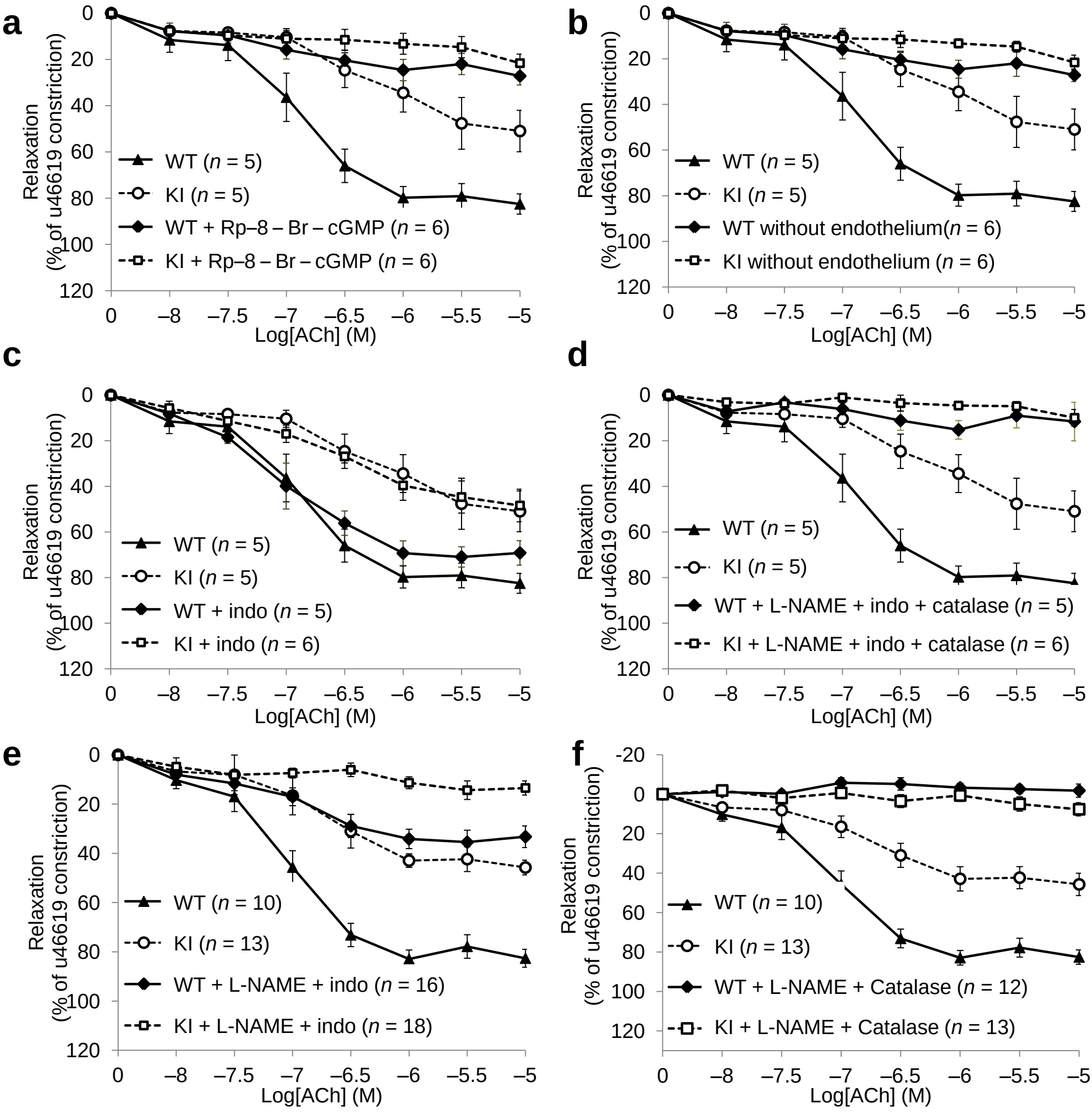

Supplementary Figure 4. Characterisation of the responses of WT and KI mesenteries to acetylcholine. (**a**) KI vessel relaxations are markedly less sensitive to acetylcholine compared to WT mesenteries (EC_{50} WT= 148.9±48.9 μM, EC_{50} KI = 614.2±162.9 μM). The acetylcholine–induced relaxation in WT was efficiently attenuated by the PKG inhibitor Rp-8-Br-cGMP, but had little effect on the KI vessel. (**b**) Removal of the endothelium from WT mesenteries greatly impaired their acetylcholine-induced relaxation, but did not alter (the already impaired) relaxation profile of KI. (**c**) Indomethacin did not alter the relaxation profiles induced by acetylcholine in WT or KI mice. (**d**) L-NAME and indomethacin in combination attenuated the relaxation induced by acetylcholine. (**e**) L-NAME, indomethacin and catalase in combination were highly effective in attenuating the relaxation induced by acetylcholine. (**f**) When the L-NAME was supplemented solely with catalase, acetylcholine-dependent relaxation was also fully blocked.