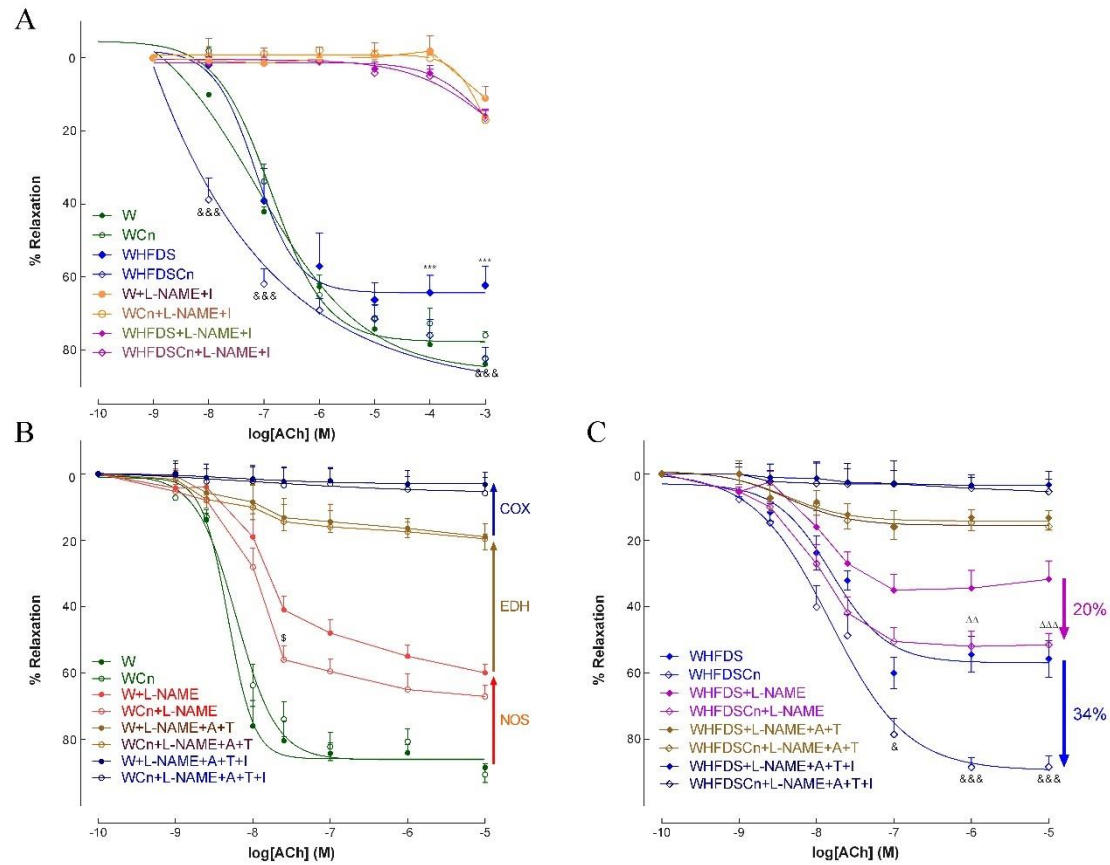


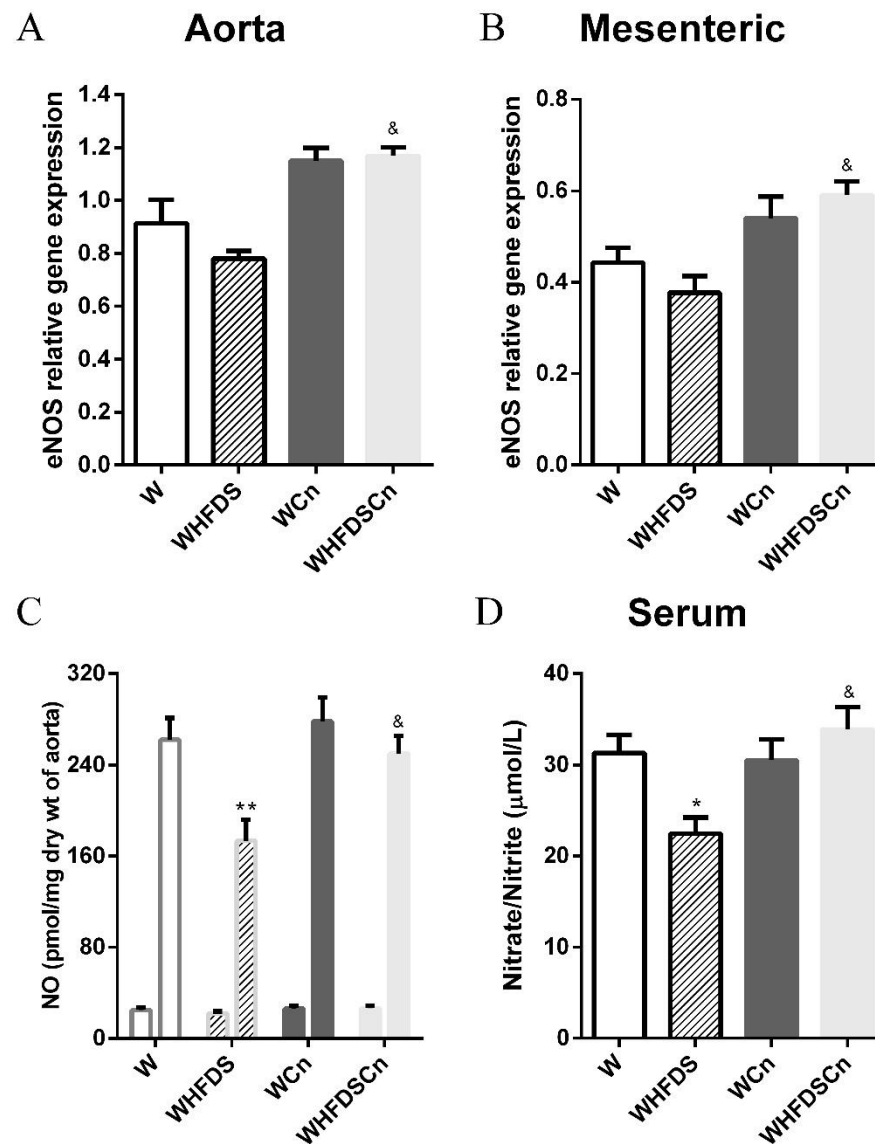
# **Cinnamaldehyde Supplementation Reverts Endothelial Dysfunction in Rat Models of Diet-Induced Obesity: Role of NF-E2-Related Factor-2**

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**Supplementary Figure S1.** Effects of cinnamaldehyde (Cn) treatment on vasodilatory responses to acetylcholine in aorta (**A**) and mesenteric arteries (**B,C**) of 8 months old Wistar (W) rats fed with sucrose and high-fat diet (WHFDS) compared with normal W rats. The specific role of endothelium-derived relaxing factors including NO, EDH, or COX-derived prostanoids was evaluated by performing concentration-response curves to acetylcholine after 30 minutes of chamber incubation with their respective inhibitors: L-NAME (100  $\mu$ mol/L), TRAM34 (T) plus apamin [(A)1  $\mu$ mol/L each], indomethacin (I, 10  $\mu$ mol/L). Data are expressed as mean  $\pm$  SE ( $n = 12$  animals in each group). &P<0.05, &&P<0.001 vs WHFDS rats; \$P<0.05, vs W+L-NAME;  $\Delta\Delta$ P<0.01,  $\Delta\Delta\Delta$ P<0.001 vs WHFDS+L-NAME.



**Supplementary Figure S2.** Effects of cinnamaldehyde (CN) on endothelial nitric oxide synthase (eNOS) expression levels in aorta (A) and mesenteric arteries (B). (C) NO metabolites were assessed in aortic homogenates and serum (D) using the Griess reaction. In each group of aortic homogenates, left and right bars represent basal and acetylcholine (ACh)-stimulated NO synthesis, respectively. Data are expressed as mean  $\pm$  SE (n = 12 animals in each group). \*P<0.05, \*\*P<0.01 vs W rats; &P<0.05 vs WHFDS rats.