Correction: Endothelial-Derived Oxidative Stress Drives Myofibroblastic Activation and Calcification of the Aortic Valve

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Fig 3 is incorrect in panels E through I. The authors have provided a corrected version here.
Fig 3. TNFs drives increased oxidative stress in aortic valve endothelial cells via eNOS uncoupling. A, TNFα increases oxidative stress in VEC at 30 minutes. B, TNFα increases hydrogen peroxide (H₂O₂) secretion from VEC at 30 minutes. C, TNFα or H₂O₂ decrease nitric oxide secretion from VEC at 48 hours (n = 4). D, TNFα or H₂O₂ decrease eNOS and VE-cadherin expression in VEC at 48 hours. Representative western blot images (inset) and blot quantification. E, L-NAME, BH₄, or peg-SOD but not apocynin block increases in superoxide (DHE) in VEC caused by TNFα, at 30 minutes. F, L-NAME, apocynin, and peg-SOD mitigate increases in general oxidative stress (DCF) caused by TNFα at 30 minutes, but only BH₄ completely blocks superoxide increase, maintaining control levels. G, L-NAME, BH₄, or peg-SOD but not apocynin block increases in H₂O₂ secreted by VEC at 30 minutes caused by TNFα at 30 minutes. H, TNFα drives increased mtROS, mitigated only by co-treatment with SOD. I, BH₄, or peg-SOD but not L-NAME or apocynin block decreases in nitric oxide secretion in VEC caused by TNFα at 48 hours. * indicates p < 0.05 versus control. # indicates p < 0.05 versus TNFα. ^ indicates p < 0.05 versus apocynin. N = 4.

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Reference

1. Farrar EJ, Huntley GD, Butcher J (2015) Endothelial-Derived Oxidative Stress Drives Myofibroblastic Activation and Calcification of the Aortic Valve. PLoS ONE 10(4): e0123257. doi: 10.1371/journal.pone.0123257 PMID: 25874717