PLOS | ONE

CORRECTION

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

Emily J. Farrar, Geoffrey D. Huntley, Jonathan Butcher

Fig 3 is incorrect in panels E through I. The authors have provided a corrected version here.

Citation: Farrar EJ, Huntley GD, Butcher J (2015) Correction: Endothelial-Derived Oxidative Stress Drives Myofibroblastic Activation and Calcification of the Aortic Valve. PLoS ONE 10(5): e0128850. doi:10.1371/journal.pone.0128850

Published: May 18, 2015

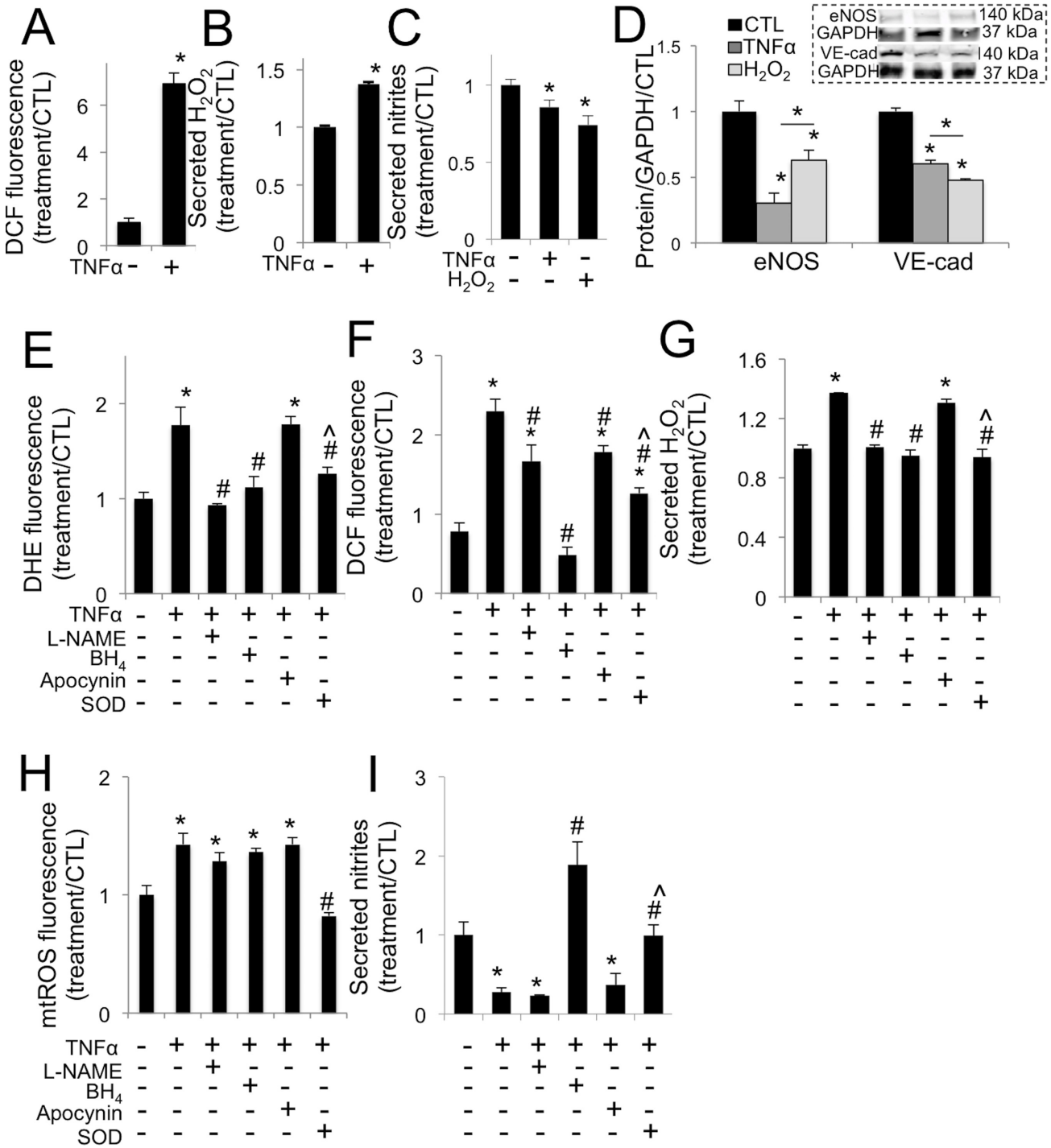

Fig 3. TNFα drives increased oxidative stress in aortic valve endothelial cells via eNOS uncoupling. A, TNFα increases oxidative stress in VEC at 30 minutes. **B**, TNFα increases hydrogen peroxide (H_2O_2) secretion from VEC at 30 minutes. **C**, TNFα or H_2O_2 decrease nitric oxide secretion from VEC at 48 hours (n = 4). **D**, TNFα or H_2O_2 decrease eNOS and VE-cadherin expression in VEC at 48 hours. Representative western blot images (inset) and blot quantification. **E**, L-NAME, BH_4, 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 BH_4 completely blocks superoxide increase, maintaining control levels. **G**, L-NAME, BH_4, or peg-SOD but not apocynin block increases in H_2O_2 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**, BH_4, 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.

doi:10.1371/journal.pone.0128850.g001

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