52 Viola HM, Arthur PG, Hool LC: Transient exposure to hydrogen peroxide causes an increase in mitochondria-derived superoxide as a result of sustained alteration in L-type Ca\(^{2+}\) channel function in the absence of apoptosis in ventricular myocytes. Circ Res 2007;100:1036–1044.

In J Vasc Res 2011:48:67–78, a p value for the black column (4) in figure 5b is missing. The corrected figure and legend should be as follows:

**Fig. 5.** H\(_2\)O\(_2\) production by mesenteric arteries under different conditions. a Endothelial (E) damage with 0.3% CHAPS prevented the angiotensin II- (0.5 μM) stimulated increase in H\(_2\)O\(_2\) production. Both apocynin (100 μM) and catalase (2,000 U/ml) inhibited angiotensin II-induced elevation of H\(_2\)O\(_2\) production. b Angiotensin II- (0.5 μM) stimulated increase of H\(_2\)O\(_2\) production was inhibited by NAD(P)H oxidase inhibitor gp91ds-tat (10 μM), but not by scrambled-tat (10 μM). The number of different experiments performed is indicated in each column of the bar graph. Apocynin alone had no significant effect on H\(_2\)O\(_2\) production. * p < 0.001, # p < 0.05 compared with control. + p < 0.001 compared with angiotensin II.