

## **DIFFERENT VASOACTIVE EFFECT OF ADHERENT ADIPOSE TISSUE DURING HYPOXIA IN MICE AORTA AND MESENTERIC ARTERIES**

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Recent studies propose a paracrine role for perivascular adipose tissue in the regulation of vascular tone. The potential influence of hypoxia on the influence of brown and white adipose tissue was investigated using isometric tension recording of isolated mice aorta and mesenteric arteries with or without adherent adipose tissue. Aorta and mesenteric arteries from male Swiss mice with or without adipose tissue were mounted in a wire myograph for isometric tension recording. Hypoxia (bubbling with 95% N<sub>2</sub>, 5% CO<sub>2</sub>) relaxed precontracted (NOR, 5 μM) aorta with brown adipose tissue, while a biphasic response was seen in precontracted (NOR, 10 μM) mesenteric arteries with white adipose tissue. Only a minimal vasorelaxing effect was observed in both arteries without adipose tissue. Indomethacin (10 μM) significantly impaired the hypoxic vasocontractile effect in mesenteric arteries. Precontraction with 60 mM K<sup>+</sup> significantly impaired the hypoxic response in both arteries while glibenclamide (30 μM) significantly blocked the hypoxic response in aorta. 8-(p-sulfophenyl)theophylline (0.1 mM) did not influence the hypoxic response in aorta. Also removal of the endothelium did not influence the hypoxic relaxation in aorta, while in mesenteric arteries removal of the endothelium almost completely blocked the hypoxic relaxation. From these results we conclude that in mice aorta hypoxia has a relaxing influence in the presence of adherent brown adipose tissue. This relaxation is at least in part mediated by opening K<sub>ATP</sub> channels and independent of the endothelium and functional adenosine receptors. These findings are in line with the involvement of the as yet unidentified "adipocyte-derived relaxing factor" (ADRF). In mice mesenteric arteries, hypoxia induces a biphasic response in the presence of adherent white adipose tissue. The hypoxic vasoconstriction is in part mediated by COX metabolites, while the hypoxic vasodilating response is endothelium-dependent and in part mediated by opening K<sup>+</sup> channels.