# Preserved insulin vasorelaxation and up-regulation of the Akt/eNOS pathway in coronary arteries from insulin resistant obese Zucker rats



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| Mots-clés | Coronary [7], Endothelium [8], Insulin [9], Obesity [10] |
| Résumé en anglais | Obesity is associated with insulin resistance in the peripheral vasculature and is an important risk factor for coronary artery disease. The current study assessed whether the vascular effects and the signaling pathways of insulin are impaired in coronary arteries from a rat model of genetic obesity. Intramyocardial arteries from obese Zucker rats (OZR) and lean Zucker rats (LZR) were mounted in microvascular myographs to assess insulin vasoactive effects and the proteins of the insulin pathway were determined by Western blotting. The endothelium-dependent and nitric oxide (NO)-mediated vasorelaxant effect of insulin was similar in arteries from LZR and OZR and blunted by inhibition of phosphatidylinositol 3-kinase (PI3K) and endothelial NO synthase (eNOS), but unaltered by either mitogen activated protein kinase (MAPK) or endothelin (ET) receptor blockade. Basal levels of phospho-eNOS Ser1177 and phospho-Akt Ser473 were up-regulated in OZR, and insulin increased phosphorylation of eNOS and Akt in both LZR and OZR. Moreover, insulin enhanced Akt expression in LZR. Basal and insulin-stimulated levels of phospho-MAPK p42/p44 were lower in OZR and palmitic acid reduced these levels in LZR. Coronary arteries are protected from vascular IR. The results underscore the fact that preservation of insulin-mediated vasorelaxation along with an up-regulation of the Akt/eNOS pathway and an impairment of the MAPK cascade account for this protection. |
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