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Abstract

The purpose of this study was to determine if imposition of mitochondrial oxidative stress would lead to compromised endothelial cell bioenergetics. One of the most important roles of vascular endothelial cells involves coronary collateral growth. In an environment of increased oxidative stress, such as that present in metabolic syndrome (obesity, insulin resistance, hypertension, and hyperlipidemia), this growth has been shown to be impaired. To evaluate this, a pure primary culture of bovine aortic endothelial cells was treated with 1 µM rotenone (mitochondrial complex I inhibitor) to induce oxidative stress. A comparison of 5’ adenosine monophosphate-activated protein kinase (AMPK) levels with levels of its activated form, phosphorylated-AMPK (P-AMPK), was used as an estimate of the ratio of cellular ATP/ADP. Bovine aortic endothelial cells treated with rotenone had an increased P-AMPK/AMPK ratio implying a cellular ATP deficit. In conclusion, mitochondrial oxidative stress impairs endothelial cell bioenergetics and leads to activation of AMPK. We propose that the activation of AMPK will have a profound impact on mammalian target of rapamycin (mTOR) signaling, protein synthesis, and will compromise phenotypic switching of endothelial cells, which is necessary for coronary collateral growth.