GLP-1 promotes mitochondrial metabolism in vascular smooth muscle cells by enhancing endoplasmic reticulum-mitochondria coupling

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Incretin GLP-1 has important metabolic effects on several tissues, mainly through the regulation of glucose uptake and usage. One mechanism for increasing cell metabolism is modulating endoplasmic reticulum (ER)-mitochondria communication, as it allows for a more efficient transfer of Ca2+ into the mitochondria, thereby increasing activity. Control of glucose metabolism is essential for proper vascular smooth muscle cell (VSMC) function. GLP-1 has been shown to produce varied metabolic actions, but whether it regulates glucose metabolism in VSMC remains unknown. In this report, we show that GLP-1 increases mitochondrial activity in the aortic cell line A7r5 by increasing ER-mitochondria coupling. GLP-1 increases intracellular glucose and diminishes glucose uptake without altering glycogen content. ATP, mitochondrial potential and oxygen consumption increase at 3 h of GLP-1 treatment, paralleled by increased Ca2+ transfer from the ER to the mitochondria. Furthermore, GLP-1 increases lev