SL01

Neurovascular Effects of Saturated Fatty Acids

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Utilizing a superfusion bioassay cascade technique, we have shown that a vasodilator, which is more potent than nitric oxide (NO), is released in the rat superior cervical ganglion (SCG) upon electrical or chemical depolarization. Release of this vasodilator was enhanced by arginine analogues including N^{ω}-nitro-L-arginine (a NO synthase inhibitor). Analysis by gas chromatography/mass spectrometry (GC/MS) identified palmitic acid methyl ester (PAME) being released from the SCG upon depolarization in the presence of arginine analogues. Exogenous PAME induced significant aortic dilation (EC_{50}=0.19 nM), suggesting that PAME is the vasodilator. Denervation study demonstrated that PAME was released from the preganglionic neurons of the SCG. Furthermore, release of PAME was calcium- and myosin light chain kinase-dependent, suggesting that it was released from axoplasmic vesicular stores. Electrophysiological studies further demonstrated that PAME inhibited nicotine-induced inward currents in cultured SCG and the α7-nicotinic acetylcholine receptor (α7-nAChR)-expressing Xenopus oocytes. Endogenous PAME appears to play a role in modulation of the autonomic ganglionic transmission, and to complement the vasodilator effect of NO in the SCG. Possible roles of PAME in the SCG and retinal circulation will be discussed.