Z406 Role of Protein Kinase C in α_1-adrenergic Regulation of a^i_{Na} in Guinea Pig Ventricular Myocytes

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We investigated the role of protein kinase C (PKC) in α_1-adrenergic regulation of intracellular Na^+ activity (a^i_{Na}) in guinea pig ventricular myocytes. a^i_{Na} and membrane potentials were measured with the Na^+-sensitive fluorescent indicator, SBFI and conventional microelectrodes, respectively, while myocytes were stimulated at the rate of 0.25 - 0.3 Hz. Stimulation of the α_1-adrenoceptor with 50 μM phenylephrine decreased the a^i_{Na} from 6.1 ± 0.3 to 4.6 ± 0.3 mM. The PKC activator, 4β-phorbol 12-myristate 13-acetate (PMA), also decreased a^i_{Na} in a concentration-dependent manner. 100 nM PMA produced a maximal decrease in a^i_{Na} of 1.5 mM from 6.5 ± 0.4 to 5.0 ± 0.4 mM. The PMA concentration required for a half-maximal decrease in a^i_{Na} was 0.46 ± 0.13 nM. PMA decreased the a^i_{Na} to a similar extent when the membrane potential of the myocytes was held at -40 mV or -85 mV. An inactive phorbol, 4α-phorbol 12-myristate 13-acetate, did not decrease the a^i_{Na}. The decrease caused by PMA could be blocked by PKC inhibitors, such as staurosporine and bisindolylmaleimide I (GF109203X). The decrease in a^i_{Na} produced by phenylephrine was blocked by pretreatment with PMA, staurosporine, or GF109203X. The decrease in a^i_{Na} produced by PMA was not prevented by pretreatment with tetrodotoxin, but it was blocked by pretreatment with either strophanthidin or high $[K^+]_o$. The results suggest that α_1-adrenergic receptor activation results in a decrease in a^i_{Na} via PKC-induced stimulation of the Na^+-K^+ pump in cardiac myocytes.

Z501 Morphological Recovery from Aging in Endothelial Cells

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Peroxynitrite ($ONOO^-$), a reactive nitrogen species (RNS) produced by oxidative stress, can cause aging by damaging cells. The aging promoting chemicals (t-butylhydroperoxide (t-BHP), 4-hydroxynonenal (HNE), 3-morpholinosydnonimine (SIN-1)) have a toxicity by producing peroxynitrite. In this study, the effect of aging promoting chemicals on bovine aortic endothelial cell (BAEC) and cell of pulmonary artery endothelium (CPAE) was examined. The cell-damage-recovery effects that 2,3,6-tribromo-4,5-dihydroxybenzyl methyl ether (TDB) and phloroglucinol, anti-aging substances analyzed by our coworkers, were investigated on the damaged cells. The TDB and phloroglucinol were analyzed from *Symphyocladia latiuscula* and *Echloria stolonifera* respectively. After the treatments of t-BHP (10 μM), HNE (0.2 μM), SIN-1 (500 μM) to the cells, damage in the cytoplasm and nucleus occurred. Especially, the necrosis was occurred in the cytoplasm. After the treatments of these chemicals, the cells were treated with TDB (150 μM) and phloroglucinol (150 μM), we detected cell-damaged recovery through the time course. These results suggest that anti-aging substances are scavenger of peroxynitrites aging.