Results In cultured rat neonatal cardiomyocytes it was shown that, in response to Iso and 8-CPT, PKC ϵ content was increased in particulate fractions of cell lysates independent of PKA, and PKC ϵ was translocated to the perinuclear area determined by confocal microscopy. Activation of PKC ϵ by Iso was associated with an increased pERK1/2. By down-regulation of Epac expression using Epac R279K (dominant negative), they blocked isoproterenol-induced PKC ϵ activation. Isoproterenol-induced ERK phosphorylation increase was blocked by the specific PKC ϵ inhibitor peptide.

Conclusion In cardiomyocytes, β -adrenergic receptors are able to activate PKC ϵ dependent of Epac and independent of PKA. Isoproterenol activate PKC ϵ induces ERK phosphorylation.

Hypertension

[gw22-e0176]

B-ADRENERGIC STIMULATION ACTIVATE PKC ϵ INDUCES ERK PHOSPHORYLATION IN CARDIOMYOCYTES

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Objective To evaluate PKC ϵ translocation after β -adrenergic stimulation in isolated cardiomyocytes and the cross-talk with Epac and ERK phosphorylation.

Methods Rat neonatal cardiomyocytes were cultured and treated with isoproterenol stimulation (Iso, 1 μ mol/l for 1 min) and Epac activator 8-CPT (1 μ mol/l for 10 min). After infected with a virus coding for the green fluorescent protein (GFP), dominant negative (DN) form of Epac and Adenovirus coding for rabbit muscle cAMP-dependent protein kinase inhibitor (Ad.PKI), cells were subjected to Iso. PKC ϵ content was measured in the particulate fraction of cell lysates obtained by differential centrifugation. The localisation of translocation of PKC ϵ was studied by western blot and confocal microscopy. After using of a specific PKC ϵ inhibitor peptide and a scramble peptide (negative control), cells were treated with Iso (1 μ mol/l for 10 min), pERK1/2 expression was measured by western blot.