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

Hypertension

[gw22-e0176] **Β-ADRENERGIC STIMULATION ACTIVATE PKCΕ INDUCES ERK PHOSPHORYLATION IN CARDIOMYOCYTES**

Lin Li, Hua Liu, Hongyan Cai, Tao Guo *First Affiliated Hospital of Kunming Medical University, Kunming, China*

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Objective To evaluate PKCε 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 μmol/l for 1 min) and Epac activator 8-CPT (1 μmol/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. PKCε content was measured in the particulate fraction of cell lysates obtained by differential centrifugation. The localisation of translocation of PKCε was studied by western blot and confocal microscopy. After using of a specific PKCε inhibitor peptide and a scramble peptide (negative control), cells were treated with Iso (1 μmol/l for 10 min), pERK1/2 expression was measured by western blot.