Reorganization of Kir2.x ion channel complex under stress effects on cardiomyocytes and neuronal cells

V. Szuts1; D. Borcsok2; F. Otvos1; HR. Kozak2; M. Szuts3; L. Toth1; E. Welker1; A. Szekeres2; T. Papp2; CS. Vagvolgyi2

1Hungarian Academy of Sciences, Center for Biological Research, Szeged, Hungary; 2Department of Microbiology, Faculty of Science and Informatics, University of Szeged, Szeged, Hungary; 3Saint George Hospital, 2nd Intestinal Department, University Hospital, Szekesfehervar, Hungary

Background: Kir2.x channels are important regulators of resting membrane potential and cellular excitability of both the cardiac and neuronal cells underlying the structural base of IK1. Ophiobolins are sesterterpene-type secondary metabolites of fungi and these possess antitumor, antibacterial, anti-fungal activities. Aim and methods: In this study we examined the contribution of Kir2.x ion channels with modulator and its possible contribution to electrophysiological remodelling in the presence of ophiobolins. We compared the expression of Kir2.x ion channel complex using molecular biological techniques in cardiomyocytes and neuronal cell lines.

Results: The Kir2.x channels associate with synapse-associated protein 97 (SAP97) anchoring protein in the healthy myocytes and neuroblastoma cells. However the SAP97 binding to Kir2.x channels and distribution of their complexes are changed in the myocytes and neuronal cells. We observed that Kir2.1 expression were opposite of Kir2.2 protein densities after treatment with different ophiobolins in both cell lines. Dynamic compartmentalization of the Kir2.x channels and SAP97 altered during stress effects.

Discussion: These data provide valuable information concerning stress factor redistributes the expression of Kir2.x proteins. SAP97 has the major role in the events of the injury with other modulators of myocytes and neuronal cells in cardiomyopathy. It is possible that the conformations of the heteromeric Kir2.x channels are different in the injured cells and these changes may lead to alter in distribution of Kir2x heteromers. This work was supported by grant TAMOP-4.2.2.A-11/1/KONV-2012-0035.