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April 2019 Vol 49 Suppl

European Journal of Clinical Investigation

53rd Annual Scientific Meeting of the
European Society for Clinical Investigation

Coimbra, Portugal
22nd – 24th May 2019

ABSTRACT BOOK

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EUROPEAN SOCIETY FOR CLINICAL INVESTIGATION
Official journal of the European Society for Clinical Investigation

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Acknowledgments: The reported study was funded by Program of Competitive Growth of KFU.

P109-T | NPY regulates electrical activity atrial and ventricle cardiomyocytes in postnatal ontogenesis of rats

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Background: Neuropeptide Y (NPY) is released from sympathetic neurons and exerts short-term effects on prejunctional nerve terminals and postjunctional cardiac ion channels. NPY also exerts trophic effects on angiogenesis, cardiac hypertrophy, autonomic signaling, cardiac ion channels, including effects on L-type Ca2+ and pacemaker channels. Results suggest a long-term influence of NPY to modify the autonomic sensitivity of the heart and/or the ionic channels that are the target of NPY agonists. The research aim is to investigate dose-dependent effects of the non-selective NPY on the parameters of electrical activity of rat right atrial and ventricular preparations.

Methods: The study was carried out on 7-, 21-and 100-day rats. Membrane potential (MP) and action potential (AP) were recorded using glass microelectrodes. The stimulus duration (1 ms) and repetition rate (5 Hz). Statistical significance was assessed using Student's *t* test.

Results: NPY at a concentration of 10^{-9} mol/L did not cause significant changes in MP and AP parameters in all age groups of animals. NPY reduced AP duration at 20, 50, 90% of repolarization (APD20, 50, 90) at 7-days rats at a concentration of 10^{-8} and 10^{-7} mol/L. NPY reduced APD 50, 90 at 21-days rats at a concentration—10^{-8} and 10^{-6} mol/L and in 100-days rats—10^{-6} mol/L ($P < 0.05$).

Conclusions: Our results indicate that NPY-receptors changes the AP duration of the repolarization. The threshold concentration of the peptide from 7 to 100 days old rises, indicating a decrease in the density and sensitivity of NPY receptors of right atrial and ventricular cardiomyocytes to agonist. The effects of NPY are most pronounced in rats 7 and 21-days age, which are characterized by the immaturity of sympathetic regulatory effects on the heart.

Acknowledgments: The reported study was funded by RFBR according to the research project № 18-34-00567 and Program of Competitive Growth of KFU.

P110-T | The effect of blockade VIP-receptors on myocardial contractility in rats

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Background: The 28-amino acids vasoactive intestinal peptide (VIP) was initially isolated from the intestine and identified soon thereafter as a neuropeptide localized both in the central and peripheral nervous system. VIP belongs to a family of structurally related neuropeptides and hormones that include secretin, glucagon and the closely related with pituitary adenylate cyclase-activating polypeptide PACAP. VIP is expressed by neurons in various brain areas, - and stored and released from nerve fibers innervating numerous organs, including heart, lung, thyroid, kidney, urogenital and gastrointestinal tracts. There are differences in the localization of the three VIP/PACAP receptors. VPAC1 is expressed in brain and in peripheral tissues such as liver, lung and intestine. VPAC2 is expressed in the CNS and in a number of peripheral tissues, including the heart, blood vessels, skeletal muscle and others. PAC1 is present predominantly in brain, in the adrenal medulla. The wide distribution of these receptors indicates that VIP/PACAP affect many different targets, both in the CNS and in the periphery. The research aim is to investigate dose-dependent effects of the blockade of VIP/PACAP receptors in the heart contraction.

Methods: Registration of isometric contraction of right atrial preparations with their own rhythm was carried out on a PowerLab device with a force sensor MLT 050/D (ADInstruments).

Results: The non-selective antagonist of VIP-receptors (10^{-10} mol/L) [Ac-Tyr1, D-Phe2]-VipAntagonist-GRF produced decrease in own rhythm frequency and myocardial contractility ($P < 0.05$). VipAntagonist 10^{-9} mol/L, 10^{-8} mol/L produced a biphasic effect: in first the increase ($P < 0.05$) and then the decrease in own rhythm frequency and myocardial contractility ($P < 0.05$). VipAntagonist-GRF 10^{-7} mol/L did not significantly affect the studied parameters.

Conclusions: Our results indicate that the blockade of VIP-receptors causes significant changes of own rhythm frequency and myocardium contractility.

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