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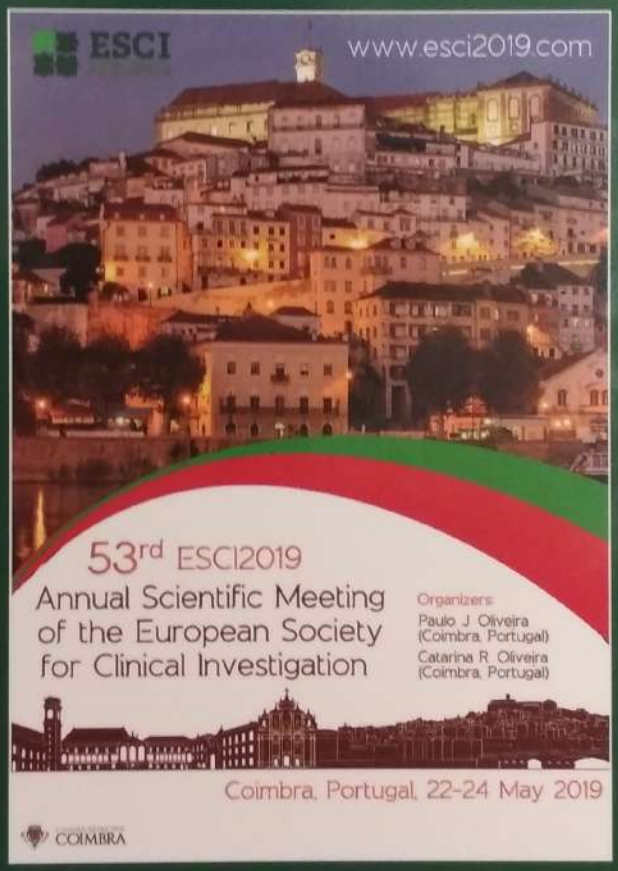

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ABSTRACT BOOK

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60-65 mm Hg. To blockade of α1A-ARs, WB4101 (Sigma) was used at a concentration of 10^{-6} mol/L. The coronary flow (CF) and heart rate (HR) were calculated along the curve. Statistical analysis was carried out with Student's *t*-test.

The selective antagonist of α1A-ARs, WB4101 10^{-6} mol/L decreased HR by 19% ($P < 0.01$) and induced an increase of CF by 15% in 20-week-old rat hearts. Selective blockade of α1A-adrenoreceptors had no effect on the isolated heart of 1-week-old rats.

The results indicated that the functional activity of the α1A-ARs in the rats heart has significant age-related features.

Work supported by Program of Competitive Growth of KFU, Russian Foundation for Basic Research and Government of the Republic of Tatarstan № 18-44-160022, RFBR № 17-04-00071.

P107-T | Effect of sodium nitroprusside on the rats myocardial contractility after NO-synthase blockade

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Background: NO-synthases present in various cardiac structures playing the important role in conduction of cardiomyocytes and contractility of coronary vasculature. NO production is regulated by NO-donors and several inhibitors of NO-synthases, which can selectively affect certain cells and tissues. Sodium nitroprusside (SNP) is a commonly use NO donor.

Material and methods: Experiments were carried out on random-bred albino rats. The contractile activity of myocardium was examined in vitro in a PowerLab setup equipped with a MLT 050/D Force Transducer (ADInstruments). The contractility of the myocardium strips of the rats atria and ventricles in the presence of the NO donor—sodium nitroprusside (SNP at a dose of 10^{-6} mol/L) and NOS inhibitor L-NAME at a dose of 10 mg/kg was studied. We evaluated the contractility of the atria and ventricular myocardium of rats to NO donor sodium nitroprusside (SNP) at a dose of 10^{-6} mol/L, and effect of SNP after administration of NOS inhibitor—L-NAME (10 mg/kg).

Results: In control group SNP increased the contractile force (CF) of ventricular myocardial strips by 24.64 ± 2.1%. In control group SNP decreased the CF of atrial strips by 12 ± 1.3%. In experimental group after inhibiting of NOS with L-NAME, SNP increased CF of ventricular myocardial strips and atrial stripes by 32.5% and 4% respectively.

Conclusions: SNP increased rat ventricular myocardial contractility and decreased the CF of atrial strips. L-NAME affected atrial contractile response to SNP.

Work supported by Program of Competitive Growth of KFU, Russian Foundation for Basic Research and Government of the Republic of Tatarstan № 18-44-160022, RFBR № 17-04-00071.

P108-T | Effects of ATP on action potentials in the atrial and ventricular myocardium of the rat heart during early postnatal ontogenesis

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Extracellular purines play major roles during embryogenesis, organogenesis, and postnatal development. Also, ATP is a sympathetic is a cotransmitter. P2X-receptors on cardiomyocytes sarcolemma are cation-selective ionic channels characterized by virtually equal permeability for Na+ and K+ ions and pronounced permeability for Ca2+ ions. These channels open in response to micromolar concentration of extracellular ATP and are responsible for rapid cell response to ATP. The research aim is to investigate dose-dependent effects of the ATP on the parameters of electrical activity right atrial and ventricular preparations of 7-, 21-, 100 day-old rats.

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: ATP at a concentration of 10^{-9} mol/L did not cause significant changes in MP and AP parameters in all age groups of animals. ATP (10^{-8} to 10^{-5} mol/L) caused a concentration-dependent change in the electrical activity of the rats right atrium and ventricular myocardium. ATP (10^{-8} to 10^{-5} mol/L) quickly reduced AP duration at 20, 50, 90% of repolarization (APD20, 50, 90) at 7-, 21, 100-days rats. The maximum effect APD20, 50, 90 on extracellular ATP was observed in 7-day rats (10^{-7} mol/L), and in 21 day rats (10^{-6} mol/L) ($P < 0.05$).

Conclusions: Our results indicate that purinoreceptors reduced the AP duration of the repolarization. The threshold concentration of the ATP from 7 to 100 days old rises, indicating a decrease in the density and sensitivity of purinoreceptors of cardiomyocytes to agonist. The effects of ATP are most pronounced in rats 7 and 21-days age, which are

characterized by the immaturity of sympathetic regulatory effects on the heart.

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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.

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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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