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Keywords: lncRNA-H19, extracellular vesicles, splice variants

55ASM-0016 ST | Neuropeptide Y changes potassium currents in rat atrium cardiomyocytes

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Background: NPY is mainly found in post-ganglionic sympathetic neurons, from which it is released together with norepinephrine in response to sympathetic stimulation, functioning as a "co-transmitter." Among the six identified Y family GPCR, Y1R, Y2R, and Y5R are the main cardiovascular regulators. Earlier we showed, that selective NPY1 receptor agonist [Leu31, Pro34]NPY and a non-selective NPY agonist cause a shortening of the repolarization phase at the DAP50 and DAP90 levels in the working cardiomyocytes of the atria and ventricles. The research aim is to study the effect of the selective Y1 [Leu31, Pro34]NPY agonist on the electrical activity parameters of the rat right atrial preparation during blockade of potassium currents.
Materials and Methods:The study was carried out on rats. Membrane potential (MP) and action potential (AP) were recorded using glass microelectrodes. The stimulus duration (1ms) and repetition rate (3Hz). Statistical significance was assessed using Student's t-test.
Results: The [Leu31, Pro34]NPY at a concentration of 10-6mol/L did not cause significant changes in MP and duration of depolarization. [Leu31, Pro34]NPY increase AP duration at 50, 90% of repolarization. [Leu31, Pro34]NPY 10-6mol/L and 4-aminoperedin at a concentration of 10-4M, the effect of the Y1 receptor agonist was preserved. [Leu31, Pro34]NPY 10-6mol/L against the background of blockade with 4-aminoperedin at a concentration of 3*10-6mol/L did not lead to significant changes in the membrane potential and action potential.
Conclusions: The combined effect of the Y1 receptor agonist 10^{-6}mol/L and the IKur blocker (4-aminoperedine) 10^{-4}mol/L does not change the duration of the repolarization phase relative to one agonist. When Ito current is blocked, the duration of the repolarization phase against the background of [Leu31, Pro34]NPY is not saved. The reported study was funded by RFBR according to the research project № 18-34-00567 and of Kazan Federal University Strategic Academic Leadership Program.

55ASM-0036 ST | Influence of dopamine to rat right atrial strips contractility

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Background: Effects of dopamine on the heart can be realized, both through adrenergic and dopamine receptors. It is shown that dopamine agonists can, both inhibit and stimulate Adenylyl cyclase through metabotropic D1-D5 receptors. In the mammalian heart, dopamine receptors have been identified in the atria, mainly at the endings of sympathetic and parasympathetic postganglionic neurons.
The aim of the study is to investigate changes in spontaneous contractile activity of rat atrial myocardium strips under the influence of various concentrations of dopamine.
Materials and Methods: In the experiments, mongrel white rats were used, conducted in compliance with the rules of the bioethics committee. Isometric contraction of right atrial myocardial strips with preserved sinus node was recorded on the «Power Lab» system (AD Instruments, Australia).
Results: Application of dopamine in a perfused solution at a concentration (10^{-10} - 10^{-6} mol/L) causes a decrease in the frequency of spontaneous activity ($p < 0.05$; $n = 10$) and an increase in contraction force ($p < 0.05$; $n = 10$). Dopamine in a concentration (10^{-5} mol/L) causes an increase in the frequency of spontaneous activity and a decrease in the force of contraction. After dopamine is removed from the solution, the isometric contraction parameters are restored in the third minute.
Conclusions: The results prove the participation of dopamine receptors in the regulation of isometric contraction. The activation of dopamine receptors increase contractile force and decrease the frequency of spontaneous activity. Increasing the frequency of spontaneous contractions and decreasing in contractile force may be associated with activation of adrenergic receptors. Further research will determine the role of various subtypes of dopamine receptors in the regulation of myocardial contractility.
This work is part of Kazan Federal University Strategic Academic Leadership Program