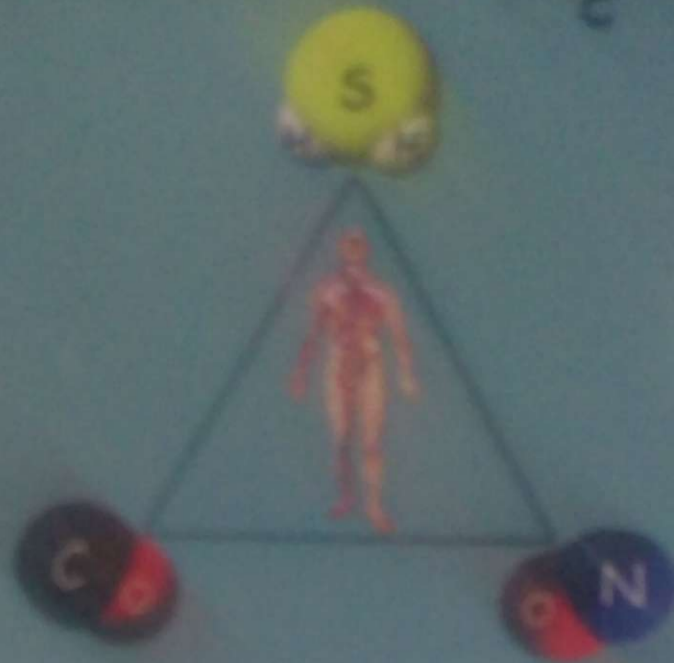


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of exocytosis does not change, which indicates the sensitivity at the slow muscle fibers to the effects of long-term hyperglycemia. Pre-exposure of the drug in a solution containing flounder 100 M LNAME both in normal and in experimental diabetes did not lead to significant changes in the emission reduction of nerve terminals. Thus, the blocking of NO-synthase did not lead to a change in synaptic transmission of nerve terminals in experimental diabetes.

The results show a decrease in synthesis of nitric oxide in the neuromuscular junction of the mouse diabetes.

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THE ROLE OF HYDROGEN SULFIDE IN REGULATION OF RAT VENTRICLE CONTRACTILITY

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Hydrogen sulfide (H₂S) is a substance with a specific smell known for its toxic properties. At the end of the last century, it was found out that in addition to nitric oxide (NO) and carbon monoxide (CO) H₂S has a number of physiological functions mediated by different targets: ion channels, intracellular mediators. Despite the fact that in recent decades its physiological role in various tissues and organs, different classes of living organisms was actively studied, many questions on the targets and mechanisms of its action are not disclosed.

In the cardiovascular system showed that H₂S affects the vascular tone in all classes of vertebrates (fish, amphibians, reptiles) and includes both vasoconstriction and vasodilation, all point to the phylogenetic antiquity of this gaseous mediator and the versatility of its actions. According to various sources, targets of H₂S in the cardiovascular system are adenylate cyclase system, voltage-dependent L-type Ca-channels, various types of K-channels.

It is known that a number of K-currents contribute to the repolarization of the membrane of cardiomyocytes in different phases of the action potential. One of the known mechanisms of action of H₂S in vascular smooth muscle and rat cardiomyocytes is the activation of ATP-dependent K-channels (K(ATP)-channels).

Methods. The object of the study was the right ventricle myocardium of the *Rattus norvegicus*. Experiments to determine the myocardium contraction were carried out using Biopac Systems, Inc. (USA). During the experiment, the preparation was perfused by Krebs solution. NaHS was used as H₂S donor. Also glibenclamide, diazoxide, minoxidil were obtained Sigma.

Results and discussion.

Application of NaHS at concentrations of 200 mcM dose-dependently reduced the force of contraction of rat heart ventricle cardiomyocytes and force of contraction was $70,1 \pm 9,2\%$ ($n = 3$, $p < 0.05$) and 27 ± 6 ($n = 7$, $p < 0.05$) relative to the control values.

Minoxidil, the activator of K (ATP) channels, at a concentration of 100 mcM resulted in a slight decrease in the force of contraction of the ventricular myocardium to $92,6 \pm 1\%$ ($n = 4$, $p < 0.05$) relative to the control values. On the background of activation of K (ATP) channels

negative inotropic effects of NaHS at a concentration 200 mcM were preserved and force of contraction was $22,8 \pm 10,8\%$ ($n = 5$, $p < 0.05$) relative to the control values.

Diazoxide, the selective activator of mitochondrial K (ATP) channels, at a concentration 100 mcM also led to a slight decrease in the force of contraction of cardiomyocytes. Negative inotropic effect of NaHS at a concentration of 200 mcM on a background of diazoxide preserved and amounted to $43,8 \pm 9\%$ ($n = 3$, $p < 0.05$), although it was less expressed than in control and on the background of the minoxidil application.

On the background of blocking sarcoplasmic and mitochondrial K (ATP) channels concentration of 50 mcM by glibenclamide, negative inotropic effects in concentrations of NaHS at 200 mcM were preserved and achieved $40,5 \pm 10,6\%$ ($n = 4$, $p < 0.05$) relative to control values.

Possible that the role of K (ATP) channels in the effect of hydrogen sulfide on contractility of rat ventricular myocardium cardiomyocytes is insignificant and mainly concerns the mitochondrial K (ATP) channels.

It was suggested that K(ATP)-channels are one of the targets of NaHS action in rat heart ventricle.

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THE EFFECTS OF HYDROGEN SULFIDE ON TRANSMITTER RELEASE WHEN CHANGING THE INTRACELLULAR CA²⁺ CONCENTRATION BY RYANODINE AND DANTROLENE AT MOUSE NEUROMUSCULAR JUNCTION

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Hydrogen sulfide (H₂S) belongs to a class of signal molecules endogenously synthesized in human and animal tissues and affecting the cardiovascular system, gastrointestinal tract and nervous system [1, 2]. Mammalian tissues contain high H₂S concentrations: 46 μM in the plasma of rats and 50–100 μM in the brain tissues [2, 3]. Some neurodegenerative processes in cardiovascular diseases are accompanied by changes in the endogenous H₂S levels [4]. High concentration in the smooth musculature of water vertebrates achieves 300 μM [5]. These data suggest a physiological role of this gas. A number of researchers believe that H₂S is related to a specific group of gaseous mediators, along with nitrogen oxide (II) (NO) and carbon monoxide (CO) [4, 6, 7]. H₂S is involved in a relaxation of mammalian smooth muscles like NO and CO [8]. This gas, in physiological concentrations, enhances the activity of NMDA receptors, facilitates an induction of long-term potentiation in the hippocampus [2]. H₂S was shown to increase the intracellular calcium concentration in astrocytes and induce calcium waves that mediate the interaction between the neurons and glia [9]. The known targets of H₂S are adenylate cyclase [10] and ATP-dependent K channels [3, 4]. In this work, we studied the role of ryanodine receptors (RyR) Ca²⁺-channels in the effects of hydrogen sulfide on neurotransmitter release at mouse motor nerve ending.

The experiments were performed on neuromuscular preparations of diaphragm from mouse using extracellular microelectrode recording. All the experiments were performed under conditions of constant perfusion of the preparation by Krebs's solution. To eliminate