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# GALLIC ACID OF THE PRODUCTS RAT AORTA SMOOTH MUSCLE CONTRACTION TO BE STUDIED

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Gallic acid ( also known as with known as 3,4,5-trihydroxybenzoic acid )a trihydroxybenzoic with acid formula It is classified as $C_6H_2(OH)_3CO_2H$. phenolic acid . Partially oxidation because of samples usually brown although , it is white hard Salts and Esters time acid " gallates " is called . Molecular mechanisms rules shrinking physiological and pathological under the circumstances smooth muscle (SM) properties modern science current problem However , some issues still satisfactory solution not found , that's it including : in 2002 publication done research to the results according to , Gallic acid rat in the aorta contractile reaction to increase and vasodilator reactions inhibition take come on This effect is mainly due to endothelial by working issued NO . own inside received nitrogen nitric oxide (NO) inactivation through happened It will be . except , Gallic acid molecule functional groups change contractile of the answer increase eliminate to be able and vasodilator reactions inhibition weaken Also , Gallic acid separated rat thoracic in the aorta to phenylephrine vasoconstrictor reaction but this impact endothelial decreased in the arteries no that implementation done .

Hypoxia and reoxygenation sedative impact proven depolarization as a result come came out rat aortic SMCs contractile activity hyperkalemia solution or α1-adrenergic activation with membranes receptors . GM hurry their in the background more precisely that was found . New data have been obtained showing that under conditions of hypoxia and reoxygenation the calming effect of carbon monoxide weakens the contractions of vascular segments, induced by hyperkalemia solution or phenylephrine. For the first time, it was found that hydrogen sulfide causes relaxation of the GM during hypoxia and reoxygenation, pre-contraction

hyperkalemia solution And, conversely, the contraction of SMC caused by phenylephrine is multidirectional: it makes them mechanically tense during hypoxia and increases during reoxygenation. Data on the role of preferential gaseous mediators (carbon monoxide and hydrogen sulfide) in isolated smooth muscle segments of the aorta of male Wistar rats were studied by scientists using the mechanographic method. This method allows you to directly study the mechanical activity of smooth muscle, assessed by the radionuclide method, changes in the concentration of monovalent sodium and potassium cations in the culture medium of SMC of the rat aorta. Determined by the ATP content inside the cell.

According to modern concepts, oxygen starvation, in First, it begins to suppress the synthesis of macroergs in reactions, combined with oxidative phosphorylation in the inner membrane mitochondria. LD. According to Lukyanova, mainly "bioenergetic hypoxia" is a false consistent change in the activity of enzymes of the respiratory chain (RC) mitochondria, depending on the intensity and/or duration of hypoxic effects. Thus, the first (compensatory) stage is characterized by the accumulation and transfer of reduced pyridine nucleotides associated with the inactivation of the NADH-dependent oxidative pathway. This process causes compensatory activation. In severe cases, hypoxia is characterized by a violation of the electron transport function of the DC in the region of the second stage. The long-term level allows cytochrome oxidase to work up to oxygen. Compensatory activation of glycolysis as an ATP supplier in conditions hypoxia, As a result of the increase in glycolysis reactions, their inhibition occurs, In addition, the increased intensity of glycolytic processes leads to the accumulation of lactic acid. Excessive concentration of lactate in the cytoplasm raises intracellular pH and the development of metabolic acidosis, which is further aggravated by the excessive accumulation of acidic products of the metabolism of fatty acids and amino acids. It is clear that under conditions of hypoxia, a decrease in the synthesis of macroergs occurs, on the basis of which energy-dependent processes are disrupted, which provide maintenance of functional activities of various intracellular systems . In addition, the earliest violations are observed in the work of ATP-dependent ion pumps [122, 126]. One of them is Na,K-

ATPase, which provides transmembrane ion transport against the K concentration gradient and thereby maintains the value. In conditions of ATP deficiency, its suppression leads to an increase in the intracellular concentration of ions + + Na ([Na ]i), a decrease in the ion content + + K ([K ]i) and an increase in the ratio [ + Na ]i/[K ]. The accumulation of + Na in the cytoplasm of cells leads to the appearance of first partial, and then permanent depolarization of cells, the disruption of which causes repolarization and functional activity. The increase in the content of Ca2+ ions in the cytosol of the membranes [97, 54] is also of great importance in the mechanism of hypoxic disorders, the redistribution of ions is a regulator of cellular metabolism, the consequences of which are associated with the exchange of Ca2+. In addition, the suppression of the electron transport function of DC in the background leads to a decrease in the difference. Another important factor that plays an important role in the damage to cells during hypoxia is the activation of free radical oxidation. The sources of free radicals can be various processes. Reactive oxygen species (ROS) can be synthesized as in the incomplete 4-electron reduction of oxygen (the final acceptor), and when interrupted by the electron transport chain, the transfer of electrons to oxygen dissolved in the matrix. It is clear that even under normoxic conditions, mitochondrial complexes I and III are capable of generating the superoxide anion radical (O -2). Increased ROS formation, similar to the blocking conditions observed during hypoxia, is dependent on NADH. ROS production release , particularly superoxide anion also occurs will be membrane with by NADPH oxidase (NOX) bound catalyzed react . TO real time V cells 7 isoforms of mammalian NADPH oxidase identified , of which the most many studied of which NOX2 (gp91phox). DUOX1/2 subtypes determined hydrogen peroxide working to release able . This has been shown selective The non- NOX inhibitor DPI ( diphenyleneiodonium ) contributes added ROS production in cells of release decrease and with him related growth . Hypoxia ROS production during of release increase with dependency shown antioxidant enzymes activity reduction ( superoxide dismutase , Studies were conducted on isolated preparations of rat aortic smooth muscle. For experiments, 11-13-week-old rats were used. Males of the

Wistar line were killed by achadon neck (190). dislocation after intraperitoneal administration of sodium pentobarbital (Nembutal, at a dose of 70 mg/kg). Isolated rat thoracic aorta was placed in a physiological Krebs balanced salt solution, prepared fat and connective tissue at room temperature (20-25 ° C), after which vascular segments 2-3 mm wide were prepared. Deendothelization of isolated smooth muscle segments was carried out directly, by mechanical rotations in their lumen with a wooden spatula. Prepared smooth muscle preparations were used immediately, the rest; the remaining vascular material was stored in a refrigerator at a temperature of 4 ° C. Determination of intracellular content of monovalent cations and ATP was performed on rat aortic SMCs used for no more than 10 min (Lonza, Walkersville, MD, USA). Cells were cultured in Dulbecco's modified medium (DMEM, Invitrogen, Carlsbad, CA, USA) containing 10% bovine serum, 100 U/ml penicillin, and 100 μg/ml streptomycin, in a specialized incubator. Contractile activity of smooth muscle segments was studied by the method of mechanography. Changes in mechanical tension (MN) of vascular segments under conditions close to isometric, were recorded using a four-channel Myobath II system and a hardware and software complex LAB-TRAX-4/16 (Germany) for conducting electrophysiological studies. The obtained vascular smooth muscle segments were placed in aerated chambers of an experimental device with a volume of 10 ml, stretched with a 500 mg load and secured with iron hooks. The chambers were then filled with Krebs physiological salt solution and thermostated at 37.0±0.5ºC. Before the start of the study, the aortic segments of rats were washed with saline solution (pH 7.35-7.4) for 40-50 minutes, after which they were twice reduced to hyperkalemia by Krebs solution (30 mM NaCl equimolarly replaced with KCl). In a series of experiments, the contractile responses of VSMCs were normalized by the addition of the α1-adrenergic agonist phenylephrine to the Krebs solution (PE, 1 μM). To study its effect, the contractile responses of VSMCs were modeled by depleting oxygen in the washing solution. Before exposure to the solution under investigation, they were saturated with gaseous nitrogen (N2, purity 99.95%). The oxygen content in the solution did not exceed 10.0±0.2 vol.% and was controlled by

a portable oximeter HI 9146-04 (HANNA, Germany). The saturation process was carried out in a glass vessel with an inert gas heated, isolated from atmospheric air. thermostat, after which it was sent to the working chambers.

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