ЖУРНАЛ ЭВОЛЮЦИОННОЙ БИОХИМИИ И ФИЗИОЛОГИИ, 2020, том 56, № 7, с. 798

МАТЕРИАЛЫ КОНФЕРЕНЦИИ И ШКОЛЫ

NITRIC OXIDE DEFICIENCY DURING PRENATAL DEVELOPMENT DELAYS THE FORMATION OF VASCULAR SMOOTH MUSCLE CONTRACTILE PHENOTYPE IN RATS

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DOI: 10.31857/S004445292007267X

The disruption of nitric oxide (NO) pathway is a key cause of the development of a dangerous pathology during pregnancy – preeclampsia. The effects of preeclampsia on the circulatory system of newborn offspring have been little studied. The stimulating effect of NO on the expression of specific for smooth muscle cells (SMC) contractile proteins was shown in experiments on cell culture. We tested the hypothesis that a deficiency of NO in the mother's body during pregnancy delays the differentiation of arterial SMC of newborn rat offspring.

Preeclampsia in female rats was modeled using an NO synthase inhibitor L-NAME (intake with drinking water from the 10th day of gestation till delivery, daily dose 78 mg/kg). Females of the control group consumed water without L-NAME throughout pregnancy. Serum nitrites/nitrates (Griess method), vasomotor aortic reactions in isometric conditions and gene mRNA expression in aortic tissue (real-time PCR) were studied in the offspring 1-2 days after birth.

The consumption of L-NAME by pregnant females led to a threefold drop in the content of NO metabolites in the blood of newborn offspring, as well as to a decrease in endothelium-dependent aortic relaxation in response to acetylcholine and the contribution of NO to this reaction. The aortic contractile responses to an increase in the extracellular Ca^{2+} concentration did not differ between rats of two groups, while the content of L-type Ca^{2+} channels mRNA was reduced, and mRNA of the sarcoplasmic reticulum proteins (RyR2 and SERCA2A) was increased in rat pups of L-NAME group compared to control. mRNA content of mature SMC specific proteins (alpha-actin, smooth muscle isoform of myosin heavy chains and SM22) in rat pups of L-NAME group was lower than in the control.

Thus, a deficiency of NO during prenatal development leads to a change in vasomotor reactions and delays the formation of a contractile phenotype of arterial smooth muscle.

Supported by RSF 19-15-00210.