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Conference Abstract

P.27 Mechanisms of NADPH Oxidase Participation in the Regulation of Diaphragm Artery Contractile Responses

Anna Borzykh[1,*], Ilya Kuzmin[2], Olga Vinogradova[1,2], Olga Tarasova[1,2]

[1]*SRC RF – Institute for Biomedical Problems RAS*
[2]*M.V. Lomonosov Moscow State University*

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ABSTRACT

Reactive oxygen species (ROS) produced by NADPH-oxidase (NOX) participate in vascular tone control, but their effects in the arteries of respiratory muscles is poorly understood. Possible targets of vasoregulatory ROS influence are NO-pathway in the endothelium and Rho-kinase pathway in smooth muscle cells. Therefore, the aim of this study was to evaluate the interaction of NOX-dependent control with NO- and Rho-kinase signaling pathways in rat diaphragm arteries (DA).

Methods: The segments of DA were isolated from male Wistar rats and mounted in wire myograph (DMT A/S). We studied the effects of NOX inhibitor VAS2870 (1 μM) on contractile responses to α_1-adrenergic agonist methoxamine in the absence and in the presence of NO synthase (L-NNA 100 μM) or Rho-kinase (Y27632, 3 μM) inhibitors as well as in the presence of NO donor DEA/NO.

Results: VAS2879 prominently attenuated the contractile responses of DA to methoxamine (30% decrease of the area under the concentration-response curve). L-NNA and Y27632 increased and decreased methoxamine-induced contraction of DA, respectively. L-NNA did not change the effects of VAS2870 and the sensitivity to DEA/NO did not differ in arteries with active and inhibited NOX. Along with that Y27632 eliminated the effects of VAS2879 on DA contractile responses to methoxamine.

Conclusions: We showed that NOX-produced ROS potentiate contractile responses of DA. ROS did not affect the activity of NO-pathway in either endothelial or smooth muscle cells of DA. However, ROS modulate the activity of the Rho-kinase pathway in DA smooth muscle cells. Supported by RSF (project No 19-75-00060).

*Corresponding author. Email: borzykh.anna@gmail.com