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Nitric oxide generation in red blood cells induced by exercise

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Objective Vascular endothelial nitric oxide synthase (NOS) is considered to be the main enzyme source for NO production in blood vessels, and studies have shown that RBC may also express NOS and produce NO. The purpose of this study was to summarize the expression of NOS in vascular red blood cells caused by changes in hemodynamics, and to improve the bioavailability of NO, and to lay a theoretical foundation for exploring the mechanism of exercise to improve vasodilation.

Methods A literature review method was used to analyze related studies on exercise and RBC-NOS published in recent years.

Results Intravascular NO is one of the most important vascular signaling molecules, which has the function of relaxing blood vessels. NO is produced during the conversion of L-arginine into L-citrulline, which is mainly dependent on the regulation of vascular eNOS. RBC can express NOS under certain action, and RBC-NOS is mainly located on RBC membrane and cytoplasm; The regulatory mechanisms of RBC-NOS and eNOS have similarities and differences: RBC-NOS and eNOS are both dependent on Ca^{2+} regulation and phosphorylation of Serine 1177 via the PI3K pathway; however, since red blood cells do not have nuclei, endoplasmic reticulum and Golgi, they do not have other mechanisms of action of eNOS. Therefore, the vascular endothelium is not the only source of NO production. Red blood cells, white blood cells and platelets can produce NO. The amount of NO produced by red blood cells is significantly higher than that of white blood cells and platelets, it is another major source of NO production in blood vessels. The level of wall shear stress is the main determinant of NOS expression in blood vessels: On the one hand, exercise training can cause hemodynamic changes, increased shear stress, and induce changes in eNOS and RBC-NOS levels, increase NO bioavailability, and participate in the regulation of vasodilation. On the other hand, moderate-intensity exercise causes NO produced by RBC to increase red blood cell deformability and participate in vascular regulation.

Conclusions 1. Erythrocyte is an enzyme source that relies on hemodynamics to release NO from the blood vessel wall. It is regulated by Ca^{2+} and phosphorylates ser¹¹⁷⁷ through the PI3K pathway to participate in the regulation of the body.

2. Hemodynamic changes caused by exercise training can simultaneously induce the expression of eNOS and RBC-NOS, increase the bioavailability of NO, and jointly mediate vasodilation.