肺NO産生能からみた短期高地トレーニングの効果

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Effect of Short Term High-Altitude Training on the Production of Nitric Oxide in Exhaled Air

by

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ABSTRACT

Since endothelium-derived relaxing factor (EDRF) was identified as nitric oxide (NO), numerous studies have been performed for determining the cellular origin and the physiological role of NO. Up to now, it has been reported that various kinds of cells, including endothelium, neuron, mucosal epithelium, smooth muscle, skeletal muscle and macrophage can generate NO, and this endogenous NO has wide physiological roles, such as control of vascular tone,

neurotransmission, host defense, etc.

On the other hand, high altitude training has been used for the improvement of aerobic work capacity in various kinds of athletic sports, such as marathoning, swimming, skating and bicycling. It has been found that a hypoxia induces pulmonary vasoconstriction, and that endothelium derived NO reduces pulmonary vasoconstriction.

In the present study, therefore, the effect of high-altitude training both on the NO production in the exhaled air during ramp exercise in humans, and on the pulmonary circulation and an isolated vascular reaction to vasoconstrictor agents in rats were investigated.

High altitude training for 5 weeks improved arterial oxygenation during inhalation of hypoxic gas at rest, but induced no significant change in VO₂, VE and exhaled NO output (VNO) during ramp exercise in humans. In the animal study, a normoxic running training or a hypoxic running training could reduce pulmonary blood pressure to exercise or hypoxia. The constrictor tone of the isolated pulmonary artery to vasoconstrictor agents was reduced in hypoxic running trained rats.

It suggests that running training or hypoxic running training could improve pulmonary vasoconstriction to exercise or hypoxia. However, exhaled NO output may not reflect the pulmonary vasodilator activity of NO.

要旨

本研究では,肺血管内皮細胞由来の一酸化窒素 (NO)が,短期高地トレーニングによって変化するか否かを検討した.

人を対象とした実験では,5週間に渡る高地トレーニング前後にランプ負荷運動を行わせ,呼気NOが増大するか否かを検討した.その結果,高地トレーニングにより呼気NO量は変化しないことが明らかとなった。

またラットを用い、低酸素下において8週間の 持久性トレーニングを行わせた後、肺血管を摘出 し、各種血管作動物質に対する収縮反応を対照群 と比較した。その結果、低酸素トレーニングは肺 血管の収縮物質に対する反応を抑制した。このこ とは、血管内皮細胞由来の血管拡張物質(NO) の産生が、低酸素下のトレーニングにより亢進し たことを示唆するものである。

以上の結果から、低酸素下のトレーニングは、

血管内皮細胞由来の拡張物質の産生を亢進し、肺循環を改善することが示唆される.しかしながら、このような肺血管由来のNOの作用を呼気ガスから推定することには問題があると考えられる.

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