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MEETING ABSTRACT Open Access

Plasma nitrite concentrations decrease after hyperoxia-induced oxidative stress in healthy humans

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Background

We measured plasma nitrite, the biochemical marker of endothelial nitric oxide (•NO) synthesis, before and after hyperoxia, in order to test the hypothesis that hyperoxia-induced vasoconstriction is a consequence of reduced bioavailability of •NO due to elevated oxidative stress.

Methods

Ten healthy males breathed 100% normobaric O_2 for 30 min between the 15th and 45th min of the 1 h study protocol. Plasma nitrite and malondialdehyde (MDA), arterial stiffness (indicated by augmentation index, AIx) and arterial oxygen ($P_{tc}O_2$) pressure were measured in the 1st, 15th, 45th and 60th minute of the study.

Results

Breathing of normobaric 100% oxygen during 30 min caused an increase of $P_{tc}O_2$ (from 75 ± 2 to 412 ± 25 mm Hg), AIx (from −63 ± 4 to −51 ± 3%) and MDA (from 152 ± 13 to 218 ± 15 nmol/L) and a decrease in plasma nitrite (from 918 ± 58 to 773 ± 55 nmol/L). During the 15-min recovery phase the plasma nitrite, AIx and MDA values remained altered.

Conclusions

This study suggests that the underlying mechanism of hyperoxia-induced vasoconstriction may result from reduced •NO bioavailability due to elevated and sustained oxidative stress.

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