Evidence of a decrease in nitric oxide-storage molecules following acute hypoxia and/or hypobaria, by means of chemiluminescence analysis.


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Nitrate, nitrite, and other nitroso compounds (NOxs) had been proposed as possible nitric oxide (NO) storage molecules. The present work examines, by means of chemiluminescence analysis, changes in NOx serum levels in rats 1 h before and 24, 48, and 72 h after exposure to acute hypobaric hypoxia (HH; barometric pressure [P(B)] 225 mmHg, oxygen partial pressure [PO2] 48 mmHg), normobaric hypoxia (NH; P(B) 716 mmHg [Jaén city], PO2 48 mmHg), hypobaric normoxia (HN; P(B) 225 mmHg, PO2 150 mmHg), and normobaric normoxia (NN; P(B) 716 mmHg, PO2 150 mmHg) the latter as a control group. Results show a decrease in NOx levels, which reached significance 24 h after exposure in HH animals, 4 h after exposure in the HN and NH groups, and persisted after 48 h of exposure in the HN group. NOx determinations were also performed in brain (cerebral cortex, hippocampus, decorticated brain [basal ganglia-brainstem] and cerebellum), liver, kidney, lung, and heart homogenates, 72 h after the experiment, to detect persistent effects when serum NOx levels had returned to basal values. Only in cerebellum (HN group) and hippocampus (HN and NH groups) were NOx levels significantly lower than in controls. We conclude that not only acute hypobaric hypoxia but also either hypobaria or hypoxia alone induce changes in NOx serum levels. Moreover, all three episodes involve a decrease in NOxs, greater and longer-lasting in hypoxia alone than in hypobaria and hypoxia together. The exhaustion of these NO-storage molecules could be critical when, as during a hypoxic episode, the L-arginine/NOS pathway is impaired.

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