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SHORT COMMUNICATION

Elevation of Rat Brain Superoxide Dismutase Activity After Combined High Altitude Hypoxia and Ascorbic Acid Treatment

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Summary
An increase in brain superoxide dismutase activity was found in rats exposed to high altitude hypoxia (7000 m, 30 min daily for five days) and ascorbic acid treatment (1 $mg.g^{-1}$ daily s.c.) while no significant change was observed after high altitude hypoxia or ascorbic acid alone.

Key words
Hypoxia – High altitude – Brain – Superoxide dismutase – Ascorbic acid

Superoxide dismutase (SOD) is a metalloenzyme with several subunits protecting aerobic organisms from the harmful effects of peroxide-free radicals (Mayes 1990). Another free radical scavenger, ascorbic acid (AA), was found in previous experiments to protect rats from the lethal effect of high altitude hypoxia (Schreiber 1992). The mechanism of this protective effect remains to be elucidated. We therefore examined changes in SOD activity in the brains of rats repeatedly exposed to high altitude hypoxia with and without the simultaneous administration of ascorbic acid.

In two identical experiments, 40 and 29 male Wistar rats (VELAZ, Prague, body weight 130-160 g, aged 60 days) were divided into four groups: 1) controls, 2) exposed to high altitude hypoxia (7000 m, 30 min daily in a hypobaric chamber for five days), 3) treated s.c. with ascorbic acid in daily doses of 1 $mg.g^{-1}$ body weight, and 4) exposed to high altitude hypoxia as in group 2 combined with ascorbic acid administration (in the same dose as in group 3, one hour before exposure to hypoxia). The animals were fed on standard laboratory diet (Larsen diet, VELAZ, Prague) and during the experiments they were kept at a temperature of 22±2 °C, with a 12 h light/12 h dark regimen. Four hours after the last dark period and one hour after the last exposure to hypoxia they were decapitated.

Immediately after decapitation the brain was removed, weighed and homogenized in ice-cold Tris-HCl+EDTA buffer and its SOD activity was measured according to McCord and Fridowich (1969). The amount of SOD needed to inhibit the rate of reduction of cytochrome C by 50 % is defined as 1 unit (U) of SOD activity. The results are expressed as the number of superoxide dismutase units per gram of brain wet weight. The means ± the 95 % confidence intervals were calculated and the significance of differences between the means was determined by the analysis of variance and by Duncan's test (Duncan 1955).

The results are presented in Table 1. In both experiments a slight non-significant increase in the SOD activity occurred after repeated hypoxia; no change was found after ascorbic acid treatment, but after repeated hypoxia combined with ascorbic acid it showed a distinct increase ($p<0.01$).

The protective effect of ascorbic acid in rats exposed to high altitude hypoxia (Schreiber 1992) may thus also be related to the increase of superoxide dismutase activity of the brain after combined hypoxia and ascorbic acid treatment.

Table 1
Superoxide dismutase (U/g brain) activity in control, hypoxic, ascorbic acid treated as well as in hypoxic rats treated with ascorbic acid.

Group	1. Controls	2. Hypoxia	3. Ascorbic acid	4. Hypoxia + Ascorbic acid
Experiment 1	213.1 ± 52.7(4) n = 10	246.0 ± 27.4(4) n = 10	237.0 ± 25.4(4) n = 10	327.5 ± 27.8(1-3) n = 10
Experiment 2	160.4 ± 49.9(4) n = 5	170.2 ± 56.7(4) n = 8	134.7 ± 41.7(4) n = 6	214.9 ± 24.0(1-3) n = 12

Means ± 95 confidence intervals. The numbers in parentheses denote groups with statistically different means (Duncan's test).

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