AltitudeOmics: Enhanced brain blood flow control with high altitude acclimatisation and re-exposure

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Introduction

Maintaining cerebral blood flow (CBF) and oxygen transport is vital.
CBF responsiveness to CO_2, termed cerebrovascular CO_2 reactivity, provides a useful, non-invasive index of cerebrovascular function.

Few studies investigated acclimatisation effect to high altitude on cerebrovascular CO_2 reactivity. Interpretation of findings from these studies is difficult due to: timing of measurement at altitude; confounding effect of relative deacclimatisation from higher altitude; artificial normobaric hypoxia; method used to assess reactivity.

Aims:

- Assess effect of altitude acclimatisation and re-exposure on cerebrovascular CO_2 reactivity
- Compare steady-state and modified rebreathing methods.

Methods

- 24 healthy non-smoking physically active subjects.
- We measured ventilation (VE), arterial blood pressure (ABP), middle cerebral artery velocity (MCAv), arterial PCO_2 ($PaCO_2$) and PO_2 (PaO_2), cerebrovascular conductance index (CVCi = MCAv/ABP) at:
  - sea level (SL)
  - acute exposure to 5,260 m (ALT1)
  - after 14 days at 5,260 m (ALT16)
  - upon re-exposure to 5,260 m following either 7 (POST7) or 21 (POST21) days at 1,500 m
- 3 step steady-state hypercapnia ($PETCO_2$ = 20, 40 and 50 mmHg) in background hyperoxia ($PETO_2$ = 300 mmHg).
- Modified rebreathing ($PETCO_2$ = 20 to 50 mmHg, $PETO_2$ > 300 mmHg).

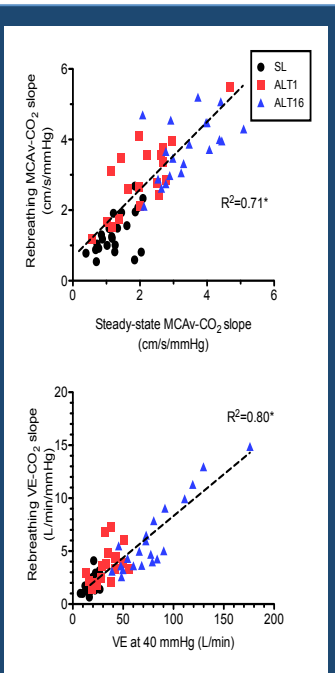

Figure 3. Comparison of steady-state and rebreathing estimate of cerebrovascular and ventilatory responsiveness to CO_2 with acclimatisation to altitude. * significant correlations (P<0.05).

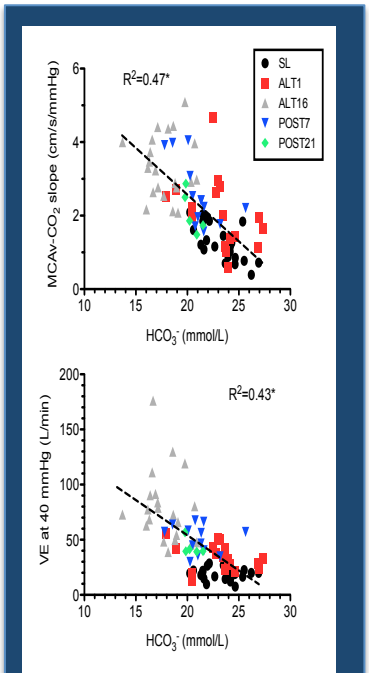

Figure 4. Relationship between arterial bicarbonate concentration and steady-state cerebrovascular and ventilatory responsiveness to CO_2 with acclimatisation and re-exposure to altitude. * significant correlations (P<0.05).

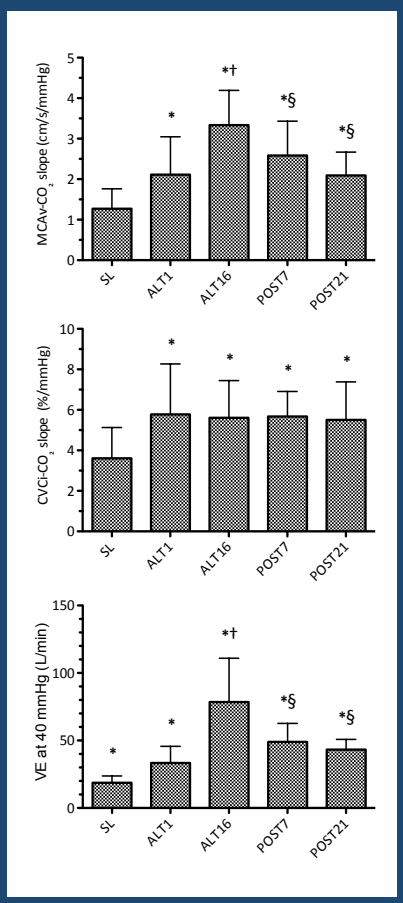

Figure 1. Changes in steady-state cerebrovascular and ventilatory responsiveness to CO_2 with acclimatisation and re-exposure to 5,200 m. Values expressed as mean ± SD. * different from SL (P<0.05), † different from ALT1 (P<0.05), § different from ALT16 (P<0.05).

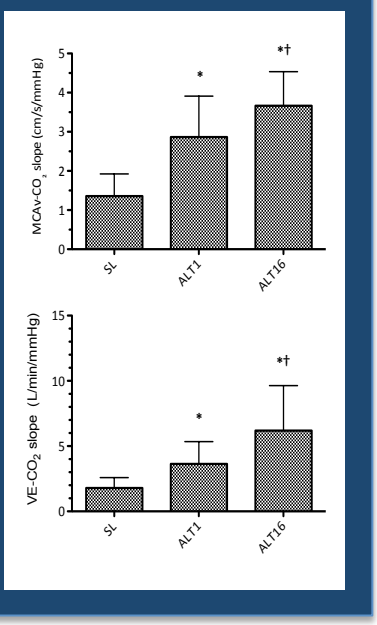

Figure 2. Changes in rebreathing cerebrovascular and ventilatory responsiveness to CO_2 with acclimatisation to 5,200 m. Values expressed as mean ± SD. * different from SL (P<0.05), † different from ALT1 (P<0.05).

Discussion

This study is the first to assess the effect of altitude acclimatisation and re-exposure on cerebrovascular and ventilatory responsiveness to CO_2 using both the steady-state and modified rebreathing methods.

We demonstrate that:

1) Cerebrovascular CO_2 reactivity is further elevated following 16 days at 5,260m compared to the initial increase found upon arrival, regardless of the method of assessment.

2) The cerebrovascular and ventilatory responsiveness to CO_2 remained elevated upon re-exposure to altitude despite deacclimatisation period of 7 and 21 days at a lower altitude.

3) This increase in cerebrovascular CO_2 reactivity with acclimatisation coincided with an elevated ventilatory responsiveness to CO_2 (mainly reflecting the central chemoreflex), both of which correlated with the changes in resting arterial [HCO_3^-].

Our data indicate that these increases in cerebrovascular and ventilatory responsiveness to CO_2 might be accounted for, by the changes acid-base balance associated with high altitude exposure. We found the effect of acclimatisation on these physiological parameters is partly retained despite a deacclimatisation period of 7 and 21 days. We speculate that an enhanced cerebrovascular CO_2 reactivity could help maintain cerebral O_2 delivery during perturbations in breathing stability at high altitude.

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