Name of research institute or organization:

Institute of Physiology, University of Zürich

Title of project:

Human adaptation to high altitude

Project leader and team:

Prof. Carsten Lundby

Project description:

From our 2014 campaign not all results have been analyzed yet, but two manuscripts have been submitted.

The first study was performed to elucidate the response of hemoglobin mass to high altitude:

Altitude exposure facilitates a rapid contraction of plasma volume (PV) and a slower occurring expansion of hemoglobin mass (Hb_{mass}). This study investigated the kinetics of the Hb_{mass} expansion and the mechanisms mediating the PV contraction.

Nine sea-level (SL) residents sojourned for 28 days at 3,454 m. Hb_{mass} and PV were estimated by carbon monoxide re-breathing at SL, on every fourth day at altitude, and again one and two weeks upon return to SL.

After four weeks at altitude Hb_{mass} was increased by 5.26 % (range 2.5 – 11.1 %; $p < 0.001$). The individual increases commenced with up to 12 days delay and reached a maximal rate of 4.04 ± 1.02 $g.d^{-1}$ after 14.9 ± 5.21 days. The probability for Hb_{mass} to plateau increased steeply after 20 - 24 days of exposure. Upon return to SL Hb_{mass} decayed at a rate of -2.46 ± 2.3 $g.d^{-1}$ reaching values similar to baseline after two weeks. PV, aldosterone concentration and renin activity were reduced at altitude (all $p < 0.001$) while the total circulating protein mass remained unaffected.

In conclusion, the Hb_{mass} response to 3,454 m followed a sigmoidal pattern as characterized by a delayed start and a plateau after ~ 3 weeks. The observed decay of Hb_{mass} upon descent to SL does not suggest major changes in the rate of erythrolysis. Our data furthermore supports a role of the renin-angiotensin-aldosterone axis to facilitate hypoxia induced PV contraction, whereas changes in oncotic pressure do not seem of relevance here for.

The second study was performed to investigate the cerebral blood flow response to high altitude:

Controversy exists on the effect of high altitude exposure on cerebrovascular function as determined by cerebrovascular CO_2 reactivity (CVR). Confounding factors in previous studies include ascent profile, duration and severity of exposure and likely also environmental factors associated with altitude exposure. In the current study, middle cerebral artery mean velocity ($MCAv_{mean}$) CVR was assessed with hyperventilation (hypocapnia) and CO_2 administration (hypercapnia) in background normoxia (SL) and hypoxia (3,454 m) in nine healthy volunteers [26 ± 4 years (mean ± SD)] at sea-level (SL), and after 30 min (HA0), 3 (HA3) and 22 (HA22) days of high altitude (3,454 m) exposure. Capillary blood was analysed for pH and bicarbonate. At altitude, ventilation was increased whereas $MCAv_{mean}$ was not altered. Hypercapnic CVR was reduced at HA0 (1.16 ± 0.16 %/mmHg, mean ± SEM) whereas both hyper- and hypocapnic CVR were elevated at HA3 (3.13 ± 0.18 and 2.96 ± 0.10 %/mmHg) and HA22 (3.32 ± 0.12 and 3.24 ± 0.14 %/mmHg) compared with SL (1.98 ± 0.22 and 2.38 ± 0.10 %/mmHg) regardless of background oxygenation. Cerebrovascular conductance ($MCAv_{mean}$/mean arterial pressure) CVR was determined to account for blood pressure changes and revealed an attenuated response. When controlling for confounding

factors this collectively demonstrates that hypocapnic and hypercapnic CVR are elevated with acclimatization to high altitude.

Key words:

Human, physiology, blood, brain, cerebral, muscle, exercise, sympathetic nervous activity

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