Magnetic Resonance Investigation of the Human Brain after 6 Days of Acclimatization to 4554 m
- Preliminary Results of the EFA study -

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Objective: Hypoxia is the main trigger of acute mountain sickness (AMS). However it is not the cause of the actual symptoms of AMS. The biochemical mechanisms underlying the AMS development are not well understood what leads to a high uncertainty regarding the likeliness of AMS development in astronauts living in future moderate hypobaric hypoxic habitats on Mars or moon. The hypothesis of the EFA study (*Edema Formation in the High Alps*) was that hypoxia triggered inflammatory processes lead to a breakdown of the capillary barrier and edema formation in vulnerable tissues as the brain.

Methods: 11 subjects (5 women) ascended within 48 h from 1154 m to the Capanna Regina Margherita in 4554 m. Brain magnetic resonance imaging (MRI) was performed at sea level before the altitude exposure and within the first 12 h after descent. MRI included amongst others an anatomical 3D volumetric T1-weighted MPRAGE (magnetization-prepared rapid acquisition of gradient echo) scan, a susceptibility weighted gradient echo sequence, T2 weighted spin echo sequences and a diffusion weighted sequence to gain an apparent diffusion coefficient mapping and a trace image to test for volume changes of the different brain compartments, for hypoxic triggered brain edema and for micro-bleedings. Baseline measurements were performed at the DLR MRI lab in Cologne (77 m) whereas post line measurements were performed at the MRI department of the German Air Force in Fürstenfeldbruck (517 m) by applying identical sequences at both centers.

Results: Neither mean global intracranial volume ($p=7.97$) nor mean volumes of the particular brain compartments grey ($p=0.279$) and white matter ($p=0.758$) or cerebrospinal fluid ($p=0.586$) showed any significant differences after the altitude exposure with respect to baseline. However 6 days of altitude exposure lead to the exacerbation of pre-existing white matter lesions in one subject and the occurrence of a local hypoxic edema in the splenium of a second subjects in the sense of a reversible splenial lesion syndrome (RESLES) (1, 2).

Conclusion: Contradictory to the current literature (3) we were not able to show a general volume gain of the intracranial compartments after high altitude adaptation. However our findings of white matter lesions (4) and RESLES in two subjects not presenting any symptoms of a high altitude cerebral edema (HACE) have, as far as we know, not been described before (5).

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