Letter to the Editor


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RE: Hypobaric Hypoxia and Reoxygenation Induce Proteomic Profile Changes in the Rat Brain Cortex (Hernandez et al. 2012)

We would like to thank Hernandez et al. for their interesting paper describing changes in the rat brain-cortex proteomic profiles in an in vivo model of hypobaric hypoxia/reoxygenation (Hernandez et al. 2012). Hypobaria, at the pressure altitudes used in this study, is associated with the risk of decompression sickness (DCS) in humans. The US Air Force have developed the Altitude Decompression Sickness Risk Assessment Computer (ADRAC) model to predict this risk (Pilmanis et al. 2004) for resting humans breathing 100% oxygen. Oxygen breathing reduces tissue nitrogen levels in the individual and therefore, in itself, reduces the DCS risk at a given altitude. For individuals breathing 100% oxygen exposed to 30,000 feet pressure altitude for 60 minutes, ADRAC predicts a 15% risk of DCS. This risk would be increased if the subjects were breathing air.

In aviation there is increasing recognition that hypobaric DCS may be more common than previously appreciated (Hundemer et al. 2012). U-2 pilots fly at high altitudes and are exposed to hypobaria with cabin pressure altitudes of 28,000-29,000 feet (8,534-8,839 m), which are similar to those in this study, but for longer periods (8-12 hours). Duration of exposure and repeat exposures both appear to increase the risk of DCS in this scenario (Hundemer et al. 2012). Unlike the reported rat model, the U-2 pilots breathe 100% oxygen to protect them both from hypoxia and to reduce (but not eliminate) their risk of DCS (McGuire et al. 2012). Recent reports have included cases of severe neurological decompression sickness in U-2 pilots (Jersey et al. 2013) and radiological evidence of neurological damage with an increase in volume of hyperintense white matter lesions in U-2 pilots who have suffered from neurological DCS (McGuire et al. 2012).

We appreciate these data on DCS relate to humans rather than rats, but wonder if the authors have considered the potential for an element of the pathophysiological changes reported to be related to DCS rather than purely hypoxia? Lillo and Parker (Lillo and Parker 2000) describe possible observable symptoms of DCS in rats after provocative “dives”. Further, if DCS is a confounder in this model, then the authors should be aware of the proteomic and genomic changes DCS can provoke (Mountcalm-Smith et al. 2007). In view of this, we wonder if the authors should reconsider whether hypobaric hypoxia is the best model to shed light on therapeutic targets for hypoxia/re-oxygenation related damage in clinical conditions that would generally occur in normobaria?

As a secondary comment, for man breathing air at 30,000 feet (8,810 m) the time of useful consciousness would be 60-90 secs and this probably represents a fatal exposure for humans. Therefore, there will be additional issues in translating data from this model to humans. The authors...
give no information about the condition of the rats at the end of the exposure or during their "recovery" phase and, more importantly, whether any died.


