30 days of normobaric hypoxia increases mitochondrial respiration

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Contrasting increases in cytochrome c oxidase and decreases in mitochondrial volume have been reported in response to stays at high altitude (Hoppeler & Vogt, 2001). However, none of these studies directly measured mitochondrial respiration and all can be criticised for a lack of control for changes in physical activity. The purpose of this study was to investigate the effects 30 days of hypoxia on directly-measured, mitochondrial respiration. Twenty Wistar rats were randomly assigned to 30 days of either normobaric normoxia (CON; 21% O_2) or hypoxia (HYP; 10% O_2). Both submaximal (0.1 mM ADP) and maximal (2 mM ADP) ADP-stimulated mitochondrial respiration were determined on both isolated mitochondria (from lungs) and permeabilised muscle fibres from the left (LV) and right ventricle (RV), and the *soleus* (SOL) and EDL. Results were analysed using one-way ANOVA ($p<0.05$). Both submaximal and maximal ADP-stimulated respiration was significantly greater in HYP for SOL and LV, and tended to be higher for RV ($p=0.06$). There were no significant differences for the EDL. The significantly greater mitochondrial respiration in the LV of HYP (26%; $p<0.05$) was similar to a previous study (16%, ns) (Novel-Chaté *et al.*, 1998). The non-significantly greater mitochondrial respiration in the RV of HYP is also consistent with previous research (Novel-Chaté *et al.*, 1998) and can probably be attributed to significantly greater mass of the RV. We have shown for the first time however, that there is a greater mitochondrial respiration in the *soleus* of rats exposed to 30 days of hypoxia.

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Novel-Chaté, V., Mateo, P., Saks, V.A., Hoerter, J.A. & Rossi, A. (1998). Chronic exposure of rats to hypoxic environment alters the mechanism of energy transfer in myocardium. *Journal of Molecular and Cellular Cardiology* **30,** 1295-1303.