

Exercise-Induced Fatigue in Severe Hypoxia Is Attenuated In Healthy Humans Following an Intermittent Hypoxic Protocol

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Introduction

Severe, acute hypoxia leads to an increased development of supraspinal fatigue during whole body exercise (1). This is alleviated following acclimatisation to severe altitude (2), although the development of peripheral fatigue is not (3). Prolonged exposure to severe altitude involves substantial logistical demand, a high incidence of acute mountain sickness and immunological consequences. We assessed the mechanisms of exercise-induced fatigue in severe hypoxia before and after a protocol of intermittent hypoxic exposure and training.

Methods

Nineteen recreationally-active males performed constant-load cycling trials (186 ± 12 W) in normobaric hypoxia ($F_{I}O_2$ 0.115) before and after an intermittent hypoxic protocol (IH, $F_{I}O_2$ 0.115, $n=11$) or control protocol in normoxia ($F_{I}O_2$ 0.21, $n=8$). Groups completed ten 2 hr exposures over 14 days. Seven sessions involved cycling for 30min at 77 ± 5 W in IH, and at the same relative heart rate in control. Total haemoglobin mass (THb_{mass}) was assessed before and after IH and control using the optimised carbon monoxide rebreathing technique. Peripheral haemoglobin saturation, cerebral oxygenation and blood velocity in the left middle cerebral artery were measured throughout trials. Before and immediately after trials, responses to transcranial magnetic stimulation and supramaximal femoral nerve stimulation were obtained to assess corticospinal and neuromuscular function, respectively.

Results

Constant-load cycling performance was improved following IH (535 ± 213 vs. 713 ± 271 , $p < 0.05$), but not in control. At task failure during baseline cycling trials, maximum voluntary force (MVC), potentiated twitch force ($Q_{tw,pot}$) and cortical voluntary activation (CVA) for the knee extensors were reduced ($p < 0.05$) in both groups. The reductions in these measures were the same at task failure after IH and control ($p > 0.05$). However, following an isotime trial in the IH group, the reductions in MVC, $Q_{tw,pot}$ and CVA were attenuated ($p < 0.05$). There were no differences in THb_{mass} in IH or control ($p > 0.05$).

Discussion

Exercise-induced supraspinal fatigue, as quantified via changes in cortical voluntary activation, was alleviated after an intermittent hypoxic protocol in comparison to exacerbated levels in acute hypoxia. IH also attenuated the substantial impact of hypoxia on the development of peripheral fatigue. These novel data suggest that improvements in exercise performance in severe hypoxia after IH might, in part, be explained by an attenuated development of both central and peripheral fatigue.

References

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