



Effects of intravenous iron on hypoxic pulmonary vasoconstriction and maximal exercise capacity during sustained (8 h) hypoxia in healthy volunteers.

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Abstract

Background: Hypoxic pulmonary vasoconstriction (HPV) increases pulmonary artery systolic pressure (PASP) and right ventricular afterload during sustained hypoxia. This could limit cardiac output and contribute to exercise limitation at high altitude, or in patients with respiratory disease. Intravenous (IV) iron has been shown to inhibit HPV. We hypothesised that IV iron would therefore enhance maximal exercise capacity after sustained hypoxia.

Methods: During visit 1, 12 healthy volunteers (age 25 ± 5 yr, mean \pm SD; 8 male, 4 female) each underwent an incremental exercise test to exhaustion on a cycle ergometer, breathing 12% oxygen. During visit 2, volunteers were randomised to receive a single-blinded IV infusion of iron sucrose (200 mg; $n=6$) or placebo (0.9% saline; $n=6$), immediately prior to an 8-h exposure to eucapnic hypoxia (end-tidal P_{O_2} 7.3 kPa). This exposure was followed within 30 min by an incremental exercise test breathing 12% oxygen.

Results: In the placebo group, sustained hypoxia increased resting PASP by 12.6 ± 1.5 mmHg (mean \pm SEM), compared with an increase of 3.4 ± 1.0 mmHg in the iron group ($p < 0.001$, Student's *t*-test). However, IV iron did not significantly influence the change in peak oxygen uptake or maximal work rate induced by sustained hypoxia, compared with placebo ($p > 0.1$ for each parameter).

Conclusions: Sustained hypoxia produces a substantial rise in PASP in healthy volunteers at rest, which is inhibited by IV iron. However, the rise in PASP during hypoxia appears not to influence maximal exercise capacity, suggesting that it does not significantly impede right ventricular function in healthy young volunteers.

Footnotes

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