TACSM Abstract

Effect of Acute Intermittent Hypoxia on Erythropoietin and Oxygen-Carrying Capacity

MERCEDES J. NAGEL, CAITLIN P. JARRARD, and SOPHIE LALANDE

Clinical Exercise Physiology Laboratory; Department of Kinesiology and Health Education; The University of Texas at Austin; Austin, TX

Category: Masters

Advisor / Mentor: Lalande, Sophie (sophie.lalande@austin.utexas.edu)

ABSTRACT

Intermittent hypoxia, defined as alternating bouts of breathing hypoxic and normoxic air, has the potential to improve oxygen-carrying capacity through an erythropoietin-mediated increase in hemoglobin mass. **PURPOSE**: To determine the effect of a single exposure of intermittent normobaric hypoxia on erythropoietin levels and hemoglobin mass in young healthy individuals. **METHODS**: Nineteen healthy individuals (10 women and 9 men, age: 24 ± 4 years, height: 174 ± 11 cm, weight: 72.2 ± 12.2 kg) participated in the study. Participants were randomly assigned to an intermittent hypoxia group (Hyp, n = 10) or a placebo intermittent normoxia group (Norm, n = 9). Intermittent hypoxia consisted of five 4-min hypoxic cycles at a targeted arterial oxygen saturation of 90% interspersed with 4-min normoxic cycles. Air was made hypoxic by titrating nitrogen to a breathing circuit connected to a tank of compressed air. Nitrogen was not added to the breathing circuit in the intermittent normoxia condition. Pulmonary gas exchange, arterial oxygen saturation, and hemodynamics, using finger plethysmography, were continuously assessed during the intervention. Erythropoietin levels were measured before and two hours following the completion of the protocol. Hemoglobin mass was assessed using the carbon monoxide rebreathing technique the day before and seven days after exposure to intermittent hypoxia or normoxia. **RESULTS**: As anticipated, the intermittent hypoxia group had a lower arterial oxygen saturation than the intermittent normoxia group during the intervention (Hyp: 89 ± 1 vs. Norm: 99 ± 1%, $p < 0.01$), which was equivalent to a lower fraction of inspired oxygen (Hyp: 0.119 ± 0.008, Norm: 0.209 ± 0.001, $p < 0.01$). Erythropoietin levels did not significantly increase following exposure to intermittent hypoxia (Hyp: 8.2 ± 4.5 to 9.0 ± 4.8, Norm: 8.9 ± 1.7 to 11.1 ± 2.1 mU/ml, $p = 0.56$). Hemoglobin mass did not change following exposure to intermittent hypoxia (Hyp: 10.6 ± 1.4 to 10.2 ± 1.4, Norm: 9.7 ± 1.6 to 9.4 ± 1.5 g/kg, $p = 0.48$). Exposure to intermittent hypoxia did not affect mean arterial pressure (Hyp: 91 ± 6 to 90 ± 6, Norm: 93 ± 12 to 93 ± 12 mmHg, $p = 0.84$) or heart rate (Hyp: 68 ± 8 to 74 ± 9, Norm: 68 ± 8 to 68 ± 8 mmHg, $p = 0.27$). Respiratory rate, tidal volume, end-tidal CO_2 and total minute ventilation were not affected by intermittent hypoxia. **CONCLUSION**: A 40-min session of intermittent hypoxia was not sufficient to elicit a rise in erythropoietin levels or oxygen-carrying capacity in young healthy individuals. A longer exposure to intermittent hypoxia at a lower arterial oxygen saturation may be necessary to trigger an erythropoietin-mediated increase in hemoglobin mass in young healthy individuals.