TACSM Abstract

Impact of Intermittent Hypoxia on Glycemic Response to a 2-Hour Oral Glucose Tolerance Test

SAHAR D. MASSOUDIAN, FRANK WOJAN, JIAHUI ZHAO, SOPHIE LALANDE

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

Category: Masters

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

ABSTRACT

Hypoxia triggers glucose uptake independently from the action of insulin. **PURPOSE:** The purpose of this study was to determine the acute effect of intermittent hypoxia, defined as alternating short bouts of breathing hypoxic and room air, on plasma glucose levels during an oral glucose tolerance test in healthy individuals. We hypothesized that exposure to intermittent hypoxia would attenuate the increase in glucose levels in response to an oral glucose tolerance test. **METHODS:** Nine individuals (5 men, age: 24 ± 4 years, height: 175 ± 9 cm, weight: 71.0 ± 13.5 kg, HbA1c: 5.4 ± 0.1%) participated in the study. Participants visited the laboratory on two occasions. On both visits, a 2-hour oral glucose tolerance test was performed, with venous blood samples collected 0, 30, 60, 90 and 120 minutes following the ingestion of a 75 g glucose drink. On visit 1, an intermittent hypoxia (IH) protocol, consisting of eight 4-minute hypoxic cycles at a targeted arterial oxygen saturation of 80% interspersed with breathing room air to resaturation, was performed following ingestion of the glucose drink. On visit 2, an intermittent normoxia protocol consisting of eight 4-minute normoxic cycles interspersed with breathing room air was performed following ingestion of the glucose drink. Visit order was randomized and participants were blinded to the condition. **RESULTS:** As expected, intermittent hypoxia resulted in a lower arterial oxygen saturation than intermittent normoxia (IH: 83 ± 3, IN: 98 ± 1%, $p < 0.01$) which corresponded to lower levels of inspired oxygen (IH: 10.9 ± 0.7, IN: 20.9 ± 0.3%, $p < 0.01$). Plasma glucose responses to the oral glucose tolerance test were not different between conditions (IH vs. IN: 0: 90 ± 7 vs. 89 ± 6; 30: 135 ± 21 vs. 137 ± 24; 60: 110 ± 28 vs. 108 ± 25; 90: 96 ± 18 vs. 88 ± 14; and 120: 101 ± 19 vs. 83 ± 14 mg/dl, $p = 0.29$). Intermittent hypoxia triggered an increase in cardiac output (6.1 ± 0.9 to 6.8 ± 1.3 L/min, $p < 0.01$) caused by an increase in heart rate (67 ± 10 to 79 ± 12 bpm, $p < 0.01$). Contrary to our hypothesis, intermittent exposure to hypoxia did not attenuate the increase in plasma glucose levels during an oral glucose tolerance test in individuals with normal glycemic control. It remains to be determined whether intermittent hypoxia can attenuate the increase in plasma glucose levels in response to an oral glucose tolerance test in individuals with impaired glucose tolerance.