Effects of arterial hypoxemia and work intensity on exercise-induced diaphragmatic fatigue in elite cyclists
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Introduction
Diaphragmatic fatigue occurs in highly-trained athletes during exhaustive exercise. Since approximately 50% of these athletes also exhibit arterial hypoxemia during high-intensity exercise, the present study sought to test the hypothesis that arterial hypoxemia contributes to exercise-induced diaphragmatic fatigue in this population.

Methods
Twelve cyclists (VO2max: 68.0±1.4 ml·kg⁻¹·min⁻¹) randomly completed one normoxemic [arterial O2 saturation (SaO2): 96±1%] and one hypoxemic (SaO2: 90±2%) 5-min exercise test at intensities equal to 90±2% and 85±2% of normoxic maximal work rate (WRmax), respectively, producing the same tidal volume (VT) and breathing frequency (fb) throughout exercise. These work rates were chosen specifically to match tidal volume and frequency, and thus ventilation, between the two conditions to assure the same respiratory muscle loads. We reasoned that under these conditions, if the hypoxic run produced more fatigue this would point to hypoxia as a prime cause for fatigue. However, if there was more fatigue in the normoxemic run, this would be compatible with the hypothesis that fatigue was related more to respiratory muscle blood flow limitations. This in turn is based on the presumption that a higher leg effort (90% versus 85%) would require greater leg blood flow, potentially reducing flow available to the respiratory muscles. Cervical magnetic stimulation was used to determine reduction in transdiaphragmatic pressure generation (Pdi) during recovery.

Results
Normoxemic exercise at 90% WRmax induced significantly (p=0.02) greater post-exercise reduction in Pdi (15±2%) than did hypoxic exercise at 85% WRmax (10±2%), despite the similar mean ventilation (VE: 128±6 and 126±5 L·min⁻¹, respectively) breathing pattern (VT: 2.74±0.1 and 2.70±0.1 L, fb: 46.2±1.5 and 48.6±1.4 breaths·min⁻¹), mean exercise Pdi (Pdi: 39.4±0.6 and 40.7±0.4 cm H2O, respectively) (Fig. 1) and end-exercise arterial lactate (La: 12.5±0.8 and 13.0±1.2 mmol·L⁻¹, respectively).

Discussion/Conclusion
The greater fatigue observed in normoxemia (at higher leg work rate) suggests that neither arterial hypoxemia nor lactic acidosis per se are predominant causative factors in diaphragmatic fatigue in this population, at least at the level of SaO2 tested (Fig. 2). Rather, this result leads us to hypothesize that blood flow competition with the legs is an important contributor to diaphragmatic fatigue in heavy exercise, assuming that higher leg work required greater blood flow.