A reduction in blood lactate concentration ([lac]B) during whole body endurance exercise has been consistently reported following inspiratory muscle training (IMT) (McConnell and Romer 2004). This is surprising since respiratory muscle function is deemed unlikely to affect [lac]B (Wetter & Dempsey 2000). Furthermore, it is unclear whether a reduced [lac]-B following IMT is directly effected by the respiratory muscles, working locomotor, and/or other metabolically active tissues. To further investigate these issues, we investigated the effects of specific inspiratory muscle training (IMT) on [lac]B during volitional hyperpnoea at rest. 22 healthy, active males were assigned to either an IMT (n=1) or a control group (n=1). Prior to and following a 6 wk intervention, subjects completed 10 min isocapnic hyperpnoea using the breathing pattern associated with 95% maximal exercise minute ventilation (VE). The [lac]B of arterialized venous blood was determined every 2 min. Breathing frequency was controlled by an audio metronome and subjects received visual, breath-by-breath feedback of VE. There was no difference in VE between groups during hyperpnoea (IMT vs. control: 133 ± 6 vs. 145 ± 7 L/min). IMT was performed using a pressure-threshold loading device and comprised 30 dynamic inspiratory efforts twice daily, at an intensity of 50% maximal inspiratory mouth pressure (MIP). The control group performed no IMT. MIP increased significantly following IMT (mean ± SEM) pre vs. post: 147 ± 8 vs. 188 ± 8 cmH2O (interaction, group x trial; P<0.01) with no change observed in the control group (162 ± 6 vs. 165 ± 6 cmH2O). The absolute increase in [lac]B during 10 min of hyperpnoea was reduced following IMT (pre: 1.18 ± 0.19 [range: 0.50 to 2.50] vs. post: 0.46 ± 0.10 [range: 0.00 to 0.95] mmol/L; P<0.01) with no change observed in the control group (0.74 ± 0.13 [range: 0.20 to 1.70] vs. 0.83 ± 0.60 [range: 0.10 to 1.80] mmol/L). [lac]B was significantly reduced at all time points following IMT (relative changes: 2 min, -53%; 4 min, -54%; 6 min, -50%; 8 min -47% and 10 min -53%; interaction: group x time x trial P<0.01) with no change in the control (relative changes: 2 min, +19%; 4 min, +11%; 6 min, +19%; 8 min +14% and 10 min +10%). The IMT-mediated reduction in [lac]B observed during volitional hyperpnoea at rest suggests that the inspiratory muscles are the source of at least part of the reduction in [lac]B during endurance exercise following IMT. The precise mechanisms responsible for this reduction are unclear, but may include a reduced rate of lactate production and/or an increased rate of lactate clearance by inspiratory musculature. These notions are supported by the IMT-mediated increases in diaphragmatic oxidative capacity in animals (Akiyama et al. 1994) and the proportion of type I muscle fibres in human external intercostal muscles (Ramirez-Sarmiento et al. 2002).

Keywords: Lactate, Ventilation, Inspiratory Muscle Function