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Inspiratory muscle training lowers the oxygen cost of voluntary hyperpnea

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Abstract

The purpose of this study was to determine if inspiratory muscle training (IMT) alters the oxygen cost of breathing ($\dot{V}_{O_{2RM}}$) during voluntary hyperpnea. Sixteen male cyclists completed 6 wk of IMT using an inspiratory load of 50% (IMT) or 15% placebo (CON) of maximal inspiratory pressure ($P_{I_{max}}$). Prior to training, a maximal incremental cycle ergometer test was performed to determine \dot{V}_{O_2} and ventilation (\dot{V}_E) at multiple workloads. Pre- and post-training, subjects performed three separate 4-min bouts of voluntary eucapnic hyperpnea (mimic), matching \dot{V}_E that occurred at 50, 75, and 100% of $\dot{V}_{O_{2max}}$. $P_{I_{max}}$ was significantly increased ($P < 0.05$) by $22.5 \pm 8.7\%$ from pre- to post-IMT and remained unchanged in the CON group. The $\dot{V}_{O_{2RM}}$ required during the mimic trial corresponded to 5.1 ± 2.5 , 5.7 ± 1.4 , and $11.7\% \pm 2.5\%$ of the total \dot{V}_{O_2} ($\dot{V}_{O_{2T}}$) at ventilatory workloads equivalent to 50, 75, and 100% of $\dot{V}_{O_{2max}}$, respectively. Following IMT, the $\dot{V}_{O_{2RM}}$ requirement significantly decreased ($P < 0.05$) by 1.5% ($4.2 \pm 1.4\%$ of $\dot{V}_{O_{2T}}$) at 75% $\dot{V}_{O_{2max}}$ and 3.4% ($8.1 \pm 3.5\%$ of $\dot{V}_{O_{2T}}$) at 100% $\dot{V}_{O_{2max}}$. No significant changes were shown in the CON group. IMT significantly reduced the O_2 cost of voluntary hyperpnea, which suggests that a reduction in the O_2 requirement of the respiratory muscles following a period of IMT may facilitate increased O_2 availability to the active muscles during exercise. These data suggest that IMT may reduce the O_2 cost of ventilation during exercise, providing an insight into mechanism(s) underpinning the reported improvements in whole body endurance performance; however, this awaits further investigation.

THE OXYGEN COST of breathing or energy requirement of the respiratory muscles are shown to increase relative to the level of ventilation (\dot{V}_E) and the work of breathing (W_b) (1, 8). During moderate-intensity exercise the respiratory musculature requires ~3–6% of total oxygen consumption ($\dot{V}_{O_{2T}}$), increasing to ~10–15% at maximal exercise (1, 3). The W_b is derived from a combination of elastic work, the work done to overcome the elastic recoil of the lung (during inspiration) and chest wall (during expiration), and resistive work that is required to overcome airway resistance (4, 15). Reducing the W_b by ~50% via a proportional assist-ventilator during whole body exercise has been shown to result in an ~7% decrease in pulmonary oxygen consumption (17) and a significant improvement in exercise tolerance (~90% $\dot{V}_{O_{2max}}$) (18).

Inspiratory muscle training (IMT) is an intervention that has been associated with improvements in whole body exercise performance (24, 31, 34), enhanced pulmonary oxygen uptake kinetics (5), reduced blood lactate concentrations (6, 24), diaphragmatic fatigue, and cardiovascular responsiveness (37). In addition, recent evidence suggests that improved exercise performance following a period of IMT is associated with reduced W_b (28). Based on the relationship between W_b and \dot{V}_{O_2} (1, 3, 16), it is plausible to suggest that if IMT does act to decrease respiratory muscle work during exercise, then the energy requirement for exercise hyperpnea would presumably decrease, potentially leading to an increase in O_2 availability to the limb locomotor muscles during exercise and the favorable changes previously described. However, this postulate remains to be elucidated. Eucapnic voluntary hyperpnea (EVH) is a measurement technique previously used to determine the O_2 cost of breathing, thereby assessing the energy requirements of the respiratory musculature for any given ventilation (1, 3). Accordingly, by determining the O_2 cost of breathing using EVH at rest, rather than whole body \dot{V}_{O_2} during exercise, it is possible to assess changes in the

metabolic demand of the respiratory muscles following IMT. Therefore, the purpose of this study was to evaluate the influence of IMT on the O_2 cost of breathing during EVH, mimicking the ventilator volumes and rates obtained during moderate-to-maximal exercise intensities. We hypothesized that following a period of IMT, the O_2 cost of breathing for a given ventilatory workload during voluntary hyperpnea would be reduced.

METHODS

Subjects.

Sixteen healthy, highly trained male cyclists [mean \pm SD; age 24 ± 5 yr, height 1.80 ± 0.05 m, body mass 77 ± 8 kg, maximal oxygen consumption ($\dot{V}O_{2\max}$) $61.8 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$] who competed regularly in local cycle races were recruited for participation in the study. All subjects were free from cardiovascular and pulmonary disease as determined from a self-reporting medical questionnaire. All subjects had normal pulmonary function (**Table 1**). Subjects were instructed to adhere to their normal diet and training regimen throughout the duration of the study and to abstain from participating in any strenuous exercise 24 hr prior to any tests. All tests and procedures were approved by the Indiana University Institutional Review Board for Human Subjects, and all subjects provided written informed consent to participate in the study.

Table 1.

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Pulmonary and respiratory muscle function

Study design.

Prior to commencing the study, subjects were familiarized with all test procedures, including all respiratory muscle and pulmonary function tests. Upon entering the study, subjects were required to visit the laboratory on three separate occasions. During *session 1*, subjects completed a medical and physical activity questionnaire, baseline pulmonary function tests (PFTs), and an incremental cycle ergometer test to the limit of exercise tolerance to determine $\dot{V}O_{2\max}$ and peak power output (W_{\max}). *Sessions 2* and *3* each consisted of a voluntary hyperpnea test and were completed pre- and post-training intervention. Following the completion of the pretraining voluntary hyperpnea test, subjects were randomly assigned in a double-blind manner to either an inspiratory muscle training group (IMT) or a placebo-controlled sham-training group (CON). Both groups completed the prescribed daily training (IMT or CON) for 6 wk.

Pulmonary function and maximal inspiratory pressure measurements.

Pulmonary function tests were performed on all subjects in triplicate according to the American Thoracic Society recommendations, which requires the subject to perform at least three acceptable spiograms, where two of the largest forced vital capacity (FVC), forced expiratory volume in 1 s FEV_1 , and forced expiratory flow at 25–75% of vital capacity ($FEF_{25-75\%}$) values may not differ by more than 10% (4a). The highest recorded value was reported. Maximal inspiratory pressure ($P_{I_{\max}}$) was measured as an index of inspiratory muscle strength using a portable hand-held mouth pressure meter (Micro Medical, Kent, UK). All maneuvers were conducted in an upright-seated position, and were initiated from residual lung volume (RV). Measurements of $P_{I_{\max}}$ were conducted at 30-s intervals, where the variability of the best values was 5% or within 5 cmH_2O (**36**); the largest value was reported.

Maximal incremental exercise testing.

A maximal incremental cycling ergometer test was performed using a Monark cycle ergometer (model 828E, Varberg, Sweden) to determine $\dot{V}_{O_2 \max}$ and W_{\max} . The test was initiated at a workload of 150 W and increased by 50 W every 3 min until volitional exhaustion, or was terminated when the subject's cadence dropped by more than 10 rpm below their self-selected pedal rate. Verification that $\dot{V}_{O_2 \max}$ was achieved was determined by a plateau (<150 ml) or a decrease in \dot{V}_{O_2} during the final 2 min of the incremental exercise test, respiratory exchange ratio of greater than 1.1, or maximal heart rate exceeding 85% of predicted ($220 - \text{age}$). At least two of these criteria were required for a valid $\dot{V}_{O_2 \max}$ test. The $\dot{V}_{O_2 \max}$ reported was the highest 60-s average value attained prior to volitional exhaustion.

Ventilatory and metabolic data were continuously monitored using open-circuit, indirect calorimetry. Subjects breathed through a low-resistance two-way non-breathing valve (Hans Rudolph 2700, Kansas City, MO) that was connected on the expired side to a 5-liter mixing chamber. Dried expired gases were continuously sampled at a rate of 300 ml/min for fractional concentrations of O_2 and CO_2 using an Applied Electrochemistry S-3A oxygen analyzer and a CD-3A carbon dioxide analyzer (Ametek, Thermo Instruments, Pittsburgh, PA). Inspired and expired ventilation were measured using a pneumotachometer on both the inspired and expired side (Hans Rudolph 3813). The pneumotachometer on the expired side was heated to 37°C (Hans Rudolph pneumotachometer heater control). \dot{V}_E was calculated from the inspired ventilation.

Flow volume loops were collected during the final 30 s of each minute during the $\dot{V}_{O_2 \max}$ test and during the final 30 s of each minute throughout each voluntary hyperpnea stage. Tidal volume loops were computer averaged for ~12–15 breaths. The average tidal volume loop was placed within a maximum flow volume loop, obtained at rest (pre-exercise), based on a measurement of end-expiratory lung volume (EELV). EELV was determined by subtracting an inspiration to maximal lung capacity (IC maneuver) from forced vital capacity (FVC). Subjects performed an IC maneuver at 30 s and 50 s of each minute during the exercise test.

Voluntary hyperpnea.

The voluntary hyperpnea protocol required subjects at rest (seated on the cycle ergometer) to mimic the exercise \dot{V}_E from three workloads, each corresponding to 50, 75, and 100% of $\dot{V}_{O_2 \max}$. Each target \dot{V}_E was maintained for 4 min. To mimic the mechanical parameters of exercise hyperpnea, the target \dot{V}_E was achieved by matching the exercise tidal volume (V_T) and breathing frequency (f_b), which were held constant for both pre- and post-training (IMT and CON). Subjects were paced for f_b using an audio metronome, and real-time visual feedback was provided for V_T . Eucapnia was maintained through inspiration of a premixed gas (5% CO_2 , 21% O_2 , balance N_2) from a Douglas bag that was connected to a two-way breathing valve via tubing containing a humidifier. The O_2 cost of hyperpnea (\dot{V}_{O_2RM}) was calculated by subtracting the \dot{V}_{O_2} measured during passive rest from the values obtained during the mimic trial.

Training intervention.

Following baseline testing, subjects were assigned to either the IMT or CON group. The IMT group completed 30 dynamic inspiratory maneuvers twice daily (AM and PM session) at a pressure-threshold load of 50% of PI_{\max} for 6 wk; this protocol has previously been shown to elicit improvements in inspiratory muscle function (7, 29, 30, 32). Subjects in the CON group completed a sham training intervention, which consisted of 60 breaths, once daily (AM or PM session) for 6 wk at ~15% of PI_{\max} ; this protocol has been shown to exhibit no changes in inspiratory muscle

function (7, 30). Subjects were instructed to initiate each breath from residual lung volume and to continue until total lung capacity. Breathing frequency during the inspiratory efforts was reduced to prevent hyperventilation-induced hypocapnea. All inspiratory training was performed using a pressure-threshold training device (POWERbreathe, HaB International, Southam, UK).

Compliance to training was assessed by monitoring the cumulative number of breaths completed during the intervention using a pressure sensor suspended within the main body of the inspiratory muscle trainer (30). An inspiratory effort was registered and counted when the negative pressure generated during inspiration exceeded the set point on the pressure switch. The cumulative number of pressure threshold changes were recorded and computed into total number of breaths. Daily physical activity and IMT training logs were kept by all subjects throughout the duration of the study to monitor training volume/intensity and adherence to training, respectively.

Data analysis.

Data were analyzed using SPSS version 17.0 statistical software. The data were assessed for normality using the Kolmogorov-Smirnov test, and Levene's test was used to test for homogeneity of variance between tests. Within and between group interactions were analyzed using a split-plot 2 × 2 [time (pre vs. post) by group (IMT vs. CON)] ANOVA. A priori simple main effects were computed to determine time and group differences. Pearson product moment correlation coefficients were used to assess the relationship between the ventilatory parameters (\dot{V}_E , f_b , V_T , and EELV) attained during the maximal exercise test and voluntary hyperpnea. The relationship between $\dot{V}_{O_{2RM}}$ and \dot{V}_E during voluntary hyperpnea was determined from regression analysis. Statistical significance was set at $P < 0.05$. Values are reported as means ± SD.

RESULTS

There was no significant difference in age, height, or weight between the CON and IMT groups. Furthermore, pretraining measures of $\dot{V}_{O_{2\max}}$ and power output (\dot{W}_{\max}) were not significantly different between the IMT and CON groups (64.3 ± 9.1 vs. 59.1 ± 4.2 ml·kg⁻¹·min⁻¹ and 369 ± 46 vs. 331 ± 26 W, respectively). Physical activity levels were not significantly different between (IMT and CON) or within (pre to post) groups. Adherence to training was also high in both groups as demonstrated by a compliance rate of 90% for both the IMT and CON groups.

Pulmonary and respiratory muscle function.

All pulmonary function measures were within normal predictive values (26; **Table 1**). There was no significant difference in pulmonary function between CON and IMT and pre- or post-training in either group. Baseline values for respiratory muscle strength ($P_{I_{\max}}$) were not significantly different between the IMT and CON groups. Following 6 wk of training, the IMT group demonstrated a significant increase ($P < 0.05$) in $P_{I_{\max}}$ of $22 \pm 13.2\%$. There was no significant change in $P_{I_{\max}}$ values from pre- to post-training in the CON group.

Comparison of ventilatory parameters during exercise and voluntary hyperpnea.

The ventilator parameters attained during maximal exercise and voluntary hyperpnea are presented in **Table 2**. \dot{V}_E was significantly increased ($P < 0.05$) across all exercise intensities through a significant increase ($P < 0.05$) in both f_b and V_T . There was no significant difference in EELV at any exercise intensity. The time spent on inspiration expressed

as a percentage of total time of one breathing cycle or duty cycle (T_i/TT) was not significantly different between 50 and 75% $\dot{V}_{O_{2\max}}$, but was significantly increased ($P < 0.05$) at 100% $\dot{V}_{O_{2\max}}$. \dot{V}_E and breathing patterns (f_b , V_T , EELV) were closely matched between the maximal incremental exercise test and mimic trial (prior to training) for both the IMT and CON groups as shown by significant correlations in \dot{V}_E (IMT $r = 0.98$, $P < 0.05$; CON $r = 0.99$, $P < 0.05$), f_b (IMT $r = 0.99$, $P < 0.05$; CON $r = 0.99$, $P < 0.05$), V_T (IMT $r = 0.95$, $P < 0.05$; CON $r = 0.90$, $P < 0.05$), and EELV (IMT $r = 0.63$, $P < 0.05$; CON $r = 0.80$, $P < 0.05$).

Table 2.

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Ventilatory responses for exercise and voluntary hyperpnea (mimic)

Oxygen cost of voluntary hyperpnea.

The curvilinear relationship between oxygen uptake ($\dot{V}_{O_{2RM}}$) and \dot{V}_E for all subjects (IMT and CON) at all levels of voluntary hyperpnea ($r = 0.88$) is shown in **Fig. 1**. At higher levels of \dot{V}_E , it is evident that an increase in \dot{V}_E was disproportionate to the increase in $\dot{V}_{O_{2RM}}$. There was no significant difference in pre- and post-training \dot{V}_E between the IMT and CON groups at 50 or 75% $\dot{V}_{O_{2\max}}$, but was significantly higher ($P < 0.05$) in the IMT group at 100% $\dot{V}_{O_{2\max}}$ both pre- and post-training. Pre- and post-training measures of \dot{V}_E during the mimic trials were not significantly different within groups (IMT or CON) at 50, 75, or 100% $\dot{V}_{O_{2\max}}$. The O_2 cost of voluntary hyperpnea data for the IMT and CON groups, pre- and post-training are shown in **Table 3**. At ventilatory workloads equivalent to 50% $\dot{V}_{O_{2\max}}$ ($\dot{V}_E \sim 50\text{--}55$ l), the O_2 cost of ventilation per liter of \dot{V}_E ($\dot{V}_{O_{2RM}}/\dot{V}_E$) averaged ~ 2.0 ml $O_2 \cdot l^{-1} \cdot \text{min}^{-1}$ in both the IMT and CON groups. When \dot{V}_E increased to ~ 150 l/min at 100% $\dot{V}_{O_{2\max}}$ in the IMT group, $\dot{V}_{O_{2RM}}/\dot{V}_E$ increased to 3.7 and to 2.6 ml $O_2 \cdot l^{-1} \cdot \text{min}^{-1}$ in the CON group ($\dot{V}_E \sim 135$ l/min).

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Fig. 1.

Individual subject values for the oxygen cost of voluntary hyperpnea ($\dot{V}_{O_{2RM}}$) at low (50% $\dot{V}_{O_{2max}}$), moderate (75% $\dot{V}_{O_{2max}}$), and high (100% $\dot{V}_{O_{2max}}$) levels of \dot{V}_E for all subjects pretraining. •, Preinspiratory muscle training (IMT); ▲, precontrol (CON). The equation for the line: $\dot{V}_{O_{2RM}} \text{ (ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}) = 0.0003\cdot V_E \text{ (l/min)}^2 - 0.0045\cdot V_E \text{ (l/min)} + 0.9787$ ($r = 0.88$, $P < 0.05$). Dashed lines represent 95% confidence intervals.

Table 3.

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Oxygen cost of voluntary hyperpnea

Assuming that the O_2 cost of ventilation does not differ between voluntary hyperpnea and exercise, it is possible to determine the percentage of the total \dot{V}_{O_2} ($\dot{V}_{O_{2T}}$) that is used to achieve a given \dot{V}_E during exercise. During exercise at 50% $\dot{V}_{O_{2max}}$ pre-IMT, the ventilatory load required $5.1 \pm 2.5\%$ of $\dot{V}_{O_{2T}}$ and increased to $5.7 \pm 1.4\%$ of $\dot{V}_{O_{2T}}$ at 75% $\dot{V}_{O_{2max}}$ and $11.7\% \pm 2.5\%$ of $\dot{V}_{O_{2T}}$ at 100% $\dot{V}_{O_{2max}}$ (**Fig. 2A**). Post-IMT, the ventilatory requirement for the same absolute \dot{V}_E significantly decreased ($P < 0.05$) to 4.2 ± 1.4 and $8.1 \pm 3.5\%$ of $\dot{V}_{O_{2T}}$ at 75% and 100% $\dot{V}_{O_{2max}}$, respectively. There was no significant change in $\dot{V}_{O_{2T}}$ at 50% $\dot{V}_{O_{2max}}$ from pre- to post-IMT or at any level of \dot{V}_E from pre- to post-CON (**Fig. 2B**). Individual changes in $\dot{V}_{O_{2RM}}$ at 100% $\dot{V}_{O_{2max}}$ in response to training are shown in **Fig. 3**, A and B. Six subjects in the IMT group demonstrated decreased $\dot{V}_{O_{2RM}}$ following training. No significant change was observed in $\dot{V}_{O_{2RM}}$ from pre- to post-training in the CON group.

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Fig. 2.

The oxygen cost of voluntary hyperpnea ($\dot{V}_{O_{2RM}}$) and $\dot{V}_{O_{2RM}}$ expressed as a percentage of total oxygen consumption ($\dot{V}_{O_{2T}}$) graphed against \dot{V}_E at low (50% $\dot{V}_{O_{2max}}$), moderate (75% $\dot{V}_{O_{2max}}$), and high (100% $\dot{V}_{O_{2max}}$) exercise intensities for both IMT (A) and CON (B) groups, pre- and post-training (means \pm SE). \bullet , Pre-IMT; \circ , post-IMT; \blacktriangle , pre-CON; \triangle , post-CON. *Significant difference ($P < 0.05$) from pre-IMT.

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Fig. 3.

Individual pre- and post-training $\dot{V}_{O_{2RM}}$ values obtained during maximal voluntary hyperpnea (100% $\dot{V}_{O_{2max}}$) for both IMT (A) and CON (B) groups. \bullet , Individual IMT subjects; \circ , mean IMT values; \blacktriangle , individual CON subjects; \triangle , mean CON values. *Significant difference ($P < 0.05$) from pre-IMT.

Ventilatory parameters during voluntary hyperpnea pre- and post-intervention.

The ventilatory parameters attained during voluntary hyperpnea for both the IMT and CON groups are shown in **Table 4**. There was no significant difference in \dot{V}_E , V_T , f_b , Ti/TT , EELV, end-inspiratory lung volume (EILV) at any level of voluntary hyperpnea from pre- to post-training in either the IMT or CON group. Peak inspiratory (PF_I) and expiratory

flow (PF_E) rates significantly increased ($P < 0.05$) during all mimic trials as \dot{V}_E increased. Following training, there was a significant decrease ($P < 0.05$) in PF_I or PF_E at 50% $\dot{V}_{O_{2\max}}$ in both the IMT and CON groups. There was no significant change in PF_I or PF_E at 75% $\dot{V}_{O_{2\max}}$ or 100% $\dot{V}_{O_{2\max}}$ in either the IMT or CON group.

Table 4.

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Ventilatory parameters during voluntary hyperpnea

Heart rate (HR) during voluntary hyperpnea, prior to training, increased to 77 ± 8 and 79 ± 7 beats/min at 75% $\dot{V}_{O_{2\max}}$ in the IMT and CON groups, respectively, and further increased to 93 ± 7 (IMT) and 88 ± 11 beats/min (CON) at 100% $\dot{V}_{O_{2\max}}$. Following IMT, HR significantly decreased ($P < 0.05$) to 73 ± 6 beats/min at 75% $\dot{V}_{O_{2\max}}$ and to 86 ± 4 beats/min at 100% $\dot{V}_{O_{2\max}}$. There was no significant change in HR at any level of voluntary hyperpnea following training in the CON group.

DISCUSSION

To our knowledge this study is the first to investigate the influence of IMT on the oxygen cost of voluntary hyperpnea. The main findings of the present study are that the relationship between increasing ventilatory workloads and the O_2 cost of voluntary hyperpnea is curvilinear in trained cyclists and that 6 wk of pressure threshold IMT significantly reduced the O_2 cost of \dot{V}_E at high ventilatory workloads. Importantly, the finding that $\dot{V}_{O_{2RM}}$ is reduced at a \dot{V}_E above 50% $\dot{V}_{O_{2\max}}$ suggests that IMT may reduce the energy requirements of the respiratory musculature in maintaining a given \dot{V}_E .

The oxygen cost of voluntary hyperpnea prior to training ranged from ~4% of $\dot{V}_{O_{2T}}$ at low intensity exercise to ~11% of $\dot{V}_{O_{2T}}$ at maximal intensity exercise, thus supporting previous evidence that the respiratory muscles require 10–15% of total oxygen consumption during maximal exercise (3, 17). The increase in the percentage of $\dot{V}_{O_{2T}}$ devoted to the O_2 cost of voluntary hyperpnea from low- to high-intensity exercise demonstrates a curvilinear relationship ($r = 0.88$), with the O_2 cost per liter of \dot{V}_E increasing with higher levels of hyperventilation. This finding is consistent with previous data that showed that the respiratory muscle demand for O_2 is positively correlated to \dot{V}_E ($r = 0.81$) and the W_b ($r = 0.76$) (1). At submaximal ventilatory workloads (~75% $\dot{V}_{O_{2\max}}$), the pretraining O_2 cost per liter of \dot{V}_E was 2.13 ± 0.65 ml O_2/l for all subjects ($n = 16$) and significantly increased to 3.14 ± 0.98 ml O_2/l ($P < 0.05$) at maximal ventilatory workloads (~100% $\dot{V}_{O_{2\max}}$). These findings are comparable to those reported in the study by Aaron et al. (1) that reported mean values of 1.79 ml O_2/l for $\dot{V}_{O_{2RM}}/\dot{V}_E$ at moderate-intensity exercise (~70% $\dot{V}_{O_{2\max}}$) and 2.85 ml O_2/l at high intensity (100% $\dot{V}_{O_{2\max}}$).

Prior to training, the O_2 cost of \dot{V}_E was significantly higher in the IMT group compared with the CON group at ventilatory workloads corresponding to 100% $\dot{V}_{O_{2\max}}$. Importantly, the level of \dot{V}_E achieved during voluntary hyperpnea in the IMT group was also significantly higher than the CON group, which may explain the pretraining difference in the O_2 cost of \dot{V}_E between the IMT and CON groups. Specifically, at higher levels of \dot{V}_E the increase in the O_2 cost of \dot{V}_E is disproportionate to the increase in \dot{V}_E , where small changes in \dot{V}_E result in greater changes in the O_2 cost of \dot{V}_E ; thus the higher levels of \dot{V}_E as demonstrated in the IMT would lead to greater O_2 cost. The increase in energy expenditure as \dot{V}_E increases can be attributed to a variety of sources of respiratory muscle work, including the elastic recoil of the

chest and lung wall, airway resistance (4, 15), increased EELV (9), and high muscle shortening velocities (19, 23). It has been suggested that as tidal breathing approaches the maximal limits for inspiratory muscle pressure development and expiratory flow rates, energy expenditure may increase to overcome the additional respiratory muscle work (3). Conversely, if one or more of the additional sources of respiratory muscle work are reduced as a result of IMT, it is reasonable to suggest that the increase in the O_2 cost may be attenuated.

In the present study, following 6 wk of IMT, $\dot{V}_{O_{2RM}}$ was significantly reduced from pretraining values at submaximal and maximal levels of ventilation. The O_2 cost of voluntary hyperpnea expressed as a percentage of $\dot{V}_{O_{2T}}$ was reduced by 1.5% at a \dot{V}_E corresponding to 75% $\dot{V}_{O_{2max}}$ following IMT. The greatest reduction in the O_2 cost of voluntary hyperpnea was observed at $\dot{V}_{O_{2max}}$, where $\dot{V}_{O_{2RM}}$ was significantly reduced from 11% of $\dot{V}_{O_{2T}}$ to 8% $\dot{V}_{O_{2T}}$ following IMT. These data therefore suggest that IMT may decrease the respiratory muscle work associated with voluntary hyperpnea. Harms et al. (17) showed that by unloading the respiratory muscles (via a proportional-assist ventilator) and consequently decreasing the W_b by ~50%, whole body O_2 consumption was reduced by ~7% during exercise. Recently, Ray et al. (28) showed that 4 wk of respiratory muscle training (inspiratory and expiratory) resulted in a significant decrease in both the inspiratory and expiratory work of breathing during underwater swimming at 70% $\dot{V}_{O_{2max}}$, suggesting that an IMT-mediated reduction in the W_b may consequently lead to a reduction in O_2 consumption during exercise. The findings from this study provided novel evidence to support this premise by demonstrating a reduction in the O_2 demand of the respiratory muscles during high levels of voluntary hyperpnea following IMT.

Oxygen consumption during exhaustive treadmill running at a workload corresponding to ~80% $\dot{V}_{O_{2max}}$ was previously shown to be attenuated following respiratory muscle training (21, 24). Reduced \dot{V}_{O_2} during whole body exercise was associated with reduced f_b and \dot{V}_E , indicating that IMT may reduce the W_b and/or improve ventilatory efficiency (21, 24). Increased ventilatory demand was previously shown to elicit a sympathetically mediated metaboreflex (33), which increases heart rate and mean arterial pressure (MAP), reducing blood flow to the limb locomotor muscles during exercise (16) and potentially reducing whole body endurance performance (18). Furthermore, Witt et al. (37) showed that IMT attenuates this increase in HR and MAP, presumably by reducing or delaying the sympathetically mediated reflex. The finding that IMT can reduce HR during voluntary hyperpnea in the present study is consistent with previous studies that demonstrated a reduced cardiovascular response following IMT (13, 37). Furthermore, our data are novel in demonstrating that IMT can reduce the O_2 demands of the respiratory musculature in combination with a reduction in HR, indicating that IMT may facilitate increased O_2 availability to the active limb locomotor muscles through an attenuated metaboreflex.

Although the work of breathing was not directly measured in the present study, we showed that IMT reduced the O_2 demand of voluntary hyperpnea. One possible explanation for the IMT-induced reduction in the O_2 cost of ventilation could relate to changes in dynamic lung volumes during hyperpnea. Specifically, if voluntary hyperpnea is performed at an increased EELV, the respiratory muscles will likely operate at a less than optimal length and thus increase the elastic work and elevate the O_2 cost of \dot{V}_E (10). It is plausible that stronger respiratory muscles may operate at a more optimal length of the force-tension spectrum and therefore lower the O_2 cost of \dot{V}_E . However, in the current study, the mechanical parameters of voluntary hyperpnea were controlled by matching V_T and f_b , which resulted in no significant difference in EELV from pre- to post-IMT. Therefore, these data suggest that changes in the O_2 cost of hyperpnea following IMT did not occur as a function of changes in EELV.

The 22% increase in respiratory muscle strength shown in the present study is similar in magnitude to those previously reported using pressure-threshold IMT (**11, 22, 30, 32, 37**). Respiratory muscle structure has also been shown to change following IMT, with an increase in diaphragm thickness (**11, 12**) and hypertrophy of type II muscle fibers of the external intercostal muscles (**27**) being reported. Therefore, the relationship between muscle fiber cross-sectional area and muscle strength may, in part, explain the reduction in the O_2 cost of \dot{V}_E . Specifically, for a given ventilatory demand, an increase in inspiratory muscle strength may result in the recruitment of fewer muscle fibers or delay the recruitment of accessory respiratory muscles, thereby lowering the O_2 requirement for voluntary hyperpnea.

While the present study has shown that IMT decreases \dot{V}_{O_2} during voluntary hyperpnea, determining the mechanism responsible for this reduction is complex, due to an array of factors that contribute toward the energy requirements of ventilation. These sources have been shown to include both maximal and operational flow rates. Specifically, it was demonstrated that by increasing the muscle shortening velocity for a given inspiratory pressure (increased inspiratory flow rate) oxygen consumption was increased (**19, 23**); thus it is possible that by decreasing the shortening velocity of the respiratory muscle during contraction, O_2 consumption may be accordingly reduced. However, the findings from the present study showed no change in inspiratory flow rates at high levels of voluntary hyperpnea following IMT, indicating that the reduction in \dot{V}_{O_2} following IMT was not related to a change in inspiratory muscle shortening velocity. It is possible that the reduction in \dot{V}_{O_2} following IMT may be related to a change in resistance to airflow, where energy expenditure is increased when tidal breathing encroaches on the maximal limits for flow (**3, 4, 15**). Aaron et al. (**3**) demonstrated that individuals who reached their reserve for expiratory flow and inspiratory muscle pressure development required 13–15% of $\dot{V}_{O_{2T}}$ compared with ~10% of $\dot{V}_{O_{2T}}$ for non-flow-limited individuals. Thus, an increase in maximal expiratory flow rates or inspiratory pressure development would increase the ventilatory reserve, thereby increasing the maximal limits for ventilation. The findings of this study are consistent with previous studies that showed that maximal expiratory flow rates remained unchanged following a period of IMT (**14, 20, 21, 24**). In contrast, some studies demonstrated an increase in maximal inspiratory flow rates following IMT (**24, 25, 35**); however, this change would presumably be a consequence of increased inspiratory muscle shortening velocity, which would increase \dot{V}_{O_2} (**19, 23**). Thus the findings of the present study suggest that the observed reduction in \dot{V}_{O_2} following IMT is not related to a change in either inspiratory or expiratory flow rates.

Conclusion.

The present study provides novel evidence that IMT reduces the O_2 cost of voluntary hyperpnea in highly trained cyclists. This IMT-mediated reduction in the O_2 cost of voluntary hyperpnea suggests that reducing the O_2 requirements of the respiratory muscles may facilitate an increase in O_2 availability to the active muscles during exercise. Thus these data may provide an insight into the possible mechanisms underpinning the previously reported improvements in whole body endurance performance following IMT.

DISCLOSURES

The inspiratory muscle training devices were provided by POWERbreathe, HaB International Ltd., Southam, UK.

AUTHOR CONTRIBUTIONS

Author contributions: L.A.T., R.F.C., J.M.S., and T.D.M. conception and design of research; L.A.T., S.L.T.-L., and D.P.W. performed experiments; L.A.T., S.L.T.-L., R.F.C., J.M.S., D.P.W., and T.D.M. analyzed data; L.A.T., S.L.T.-L., R.F.C., J.M.S., and T.D.M. interpreted results of experiments; L.A.T. prepared figures; L.A.T., S.L.T.-L., R.F.C., J.M.S., D.P.W., and T.D.M. drafted manuscript; L.A.T., S.L.T.-L., R.F.C., J.M.S., D.P.W., and T.D.M. edited and revised manuscript; L.A.T., S.L.T.-L., R.F.C., J.M.S., D.P.W., and T.D.M. approved final version of manuscript.

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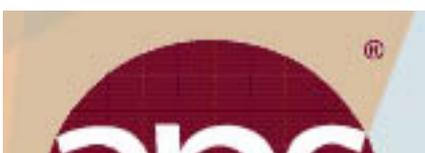
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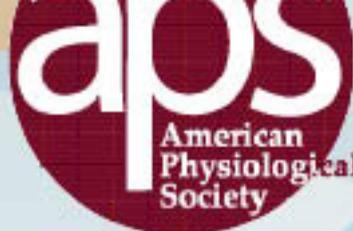
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