

Inspiratory flow resistive loading improves respiratory muscle function and endurance capacity in recreational runners

T. D. Mickleborough¹, T. Nichols², M. R. Lindley³, K. Chatham⁴, A. A. Ionescu⁵

¹Human Performance Laboratory, Department of Kinesiology, Indiana University, Bloomington, Indiana, USA, ²Cardiff School of Sport, University of Wales Institute, Cardiff, UK, ³School of Sport and Human Sciences, Human Cellular and Molecular Research Laboratory, Loughborough University, Loughborough, UK, ⁴Department of Physiotherapy, Academic Centre, University of Wales College of Medicine, University Hospital of Wales and Llandough Hospital, Cardiff and Vale NHS Trust, Penarth, UK, ⁵Section of Respiratory Medicine and Communicable Diseases, Academic Centre, University of Wales College of Medicine, University Hospital of Wales and Llandough Hospital, Cardiff and Vale NHS Trust, Penarth, UK

Corresponding author: Timothy D. Mickleborough, PhD, FACSM, Department of Kinesiology, Indiana University, 1025 E. 7th Street, HPER 112, Bloomington, IN 47401, USA. Tel: +1 812 855 0753, Fax: +1 812 855 3193, E-mail: tmickleb@indiana.edu

Accepted for publication 24 February 2009

The purpose of this study was to assess the efficacy of inspiratory flow resistive loading (IFRL) on respiratory muscle function, exercise performance and cardiopulmonary and metabolic responses to exercise. Twenty-four recreational road runners (12 male) were randomly assigned from each gender into an IFRL group ($n = 8$) and sham-IFRL group ($n = 8$), which performed IFRL for 6 weeks, or a control group ($n = 8$). Strength (+43.9% Δ), endurance (+26.6% Δ), maximum power output (+41.9% Δ) and work capacity (+38.5% Δ) of the inspiratory muscles were significantly increased ($P < 0.05$) at rest following the study period in IFRL group only. In addition, ventilation (–25.7% Δ), oxygen consumption (–13.3% Δ), breathing

frequency (–11.9% Δ), tidal volume (–16.0% Δ), heart rate (HR) (–13.1% Δ), blood lactate concentration (–38.9% Δ) and the perceptual response (–33.5% Δ) to constant workload exercise were significantly attenuated ($P < 0.05$), concomitant with a significant improvement ($P < 0.05$) in endurance exercise capacity (+16.4% Δ) during a treadmill run set at 80% $\dot{V}O_{2\max}$ in IFRL group only. These data suggest that IFRL can alter breathing mechanics, attenuate the oxygen cost, ventilation, HR, blood lactate and the perceptual response during constant workload exercise and improve endurance exercise performance in recreational runners.

In general, it has been accepted that ventilation is not a limiting factor to maximum exercise in healthy humans (Dempsey, 1986), even though a substantial portion (14–16%) of the cardiac output is directed to the respiratory muscles to support their metabolic requirements during maximum exercise in highly fit individuals (Harms et al., 1998). However, it has been shown in numerous studies that diaphragmatic fatigue occurs during high-intensity, exhaustive, constant-load running or cycling exercise of at least 80–85% $\dot{V}O_{2\max}$ or 80% W_{\max} (Babcock et al., 2002). Inspiratory muscle fatigue has been shown to occur in short-duration, high-intensity rowing (Volianitis et al., 2001), after a single 200-m freestyle swim (Lomax & McConnell, 2003) and after simulated cycling time trials (Romer et al., 2002c). Evidence suggests that inspiratory muscle training (IMT) may attenuate inspiratory muscle fatigue in healthy individuals, and thus influence exercise tolerance (Volianitis et al., 2001; Romer et al., 2002c).

Respiratory muscle training (RMT) protocols utilizing either voluntary isocapnic hyperpnea (VIH) or

IMT have been shown to improve exercise performance in rowing (Volianitis et al., 2001; Griffiths & McConnell, 2007), cycling (Stuessi et al., 2001; Romer et al., 2002a; Gething et al., 2004b) and swimming (Wells et al., 2005). Interestingly, Leddy et al., 2007 have shown reductions in breathing frequency, ventilation and $\dot{V}O_2$ during a treadmill run at 80% $\dot{V}O_{2\max}$ following 4 weeks of VIH training. Specifically, IMT has been associated with reduced exercise blood lactate concentration [Lac^-]_B (McConnell & Sharpe, 2005; Griffiths & McConnell, 2007; Brown et al., 2008), ventilation (Gething et al., 2004a) and breathing frequency (Hanel & Secher, 1991), increased diaphragm thickness (Enright et al., 2006b; Downey et al., 2007), structural changes within the inspiratory muscles (Bisschop et al., 1997) and increased inspiratory muscle strength (Witt et al., 2007; Mickleborough et al., 2008) and endurance (Mickleborough et al., 2008).

Data on the impact of RMT on running exercise performance and cardiopulmonary responses has been equivocal. While some RMT protocols utilizing

VIH (Leddy et al., 2007), pressure-threshold loading IMT (Edwards & Cooke, 2004; Edwards et al., 2008) and flow-resistive loading IMT (Chatham et al., 1999) have shown improvements in time-trial running performance, time to exhaustion during a constant load exercise test and shuttle-run performance, other studies have not observed any improvement in running performance as a consequence of IMT (Williams et al., 2002; Downey et al., 2007).

Therefore, the purpose of this study was twofold: first, to assess the efficacy of 6 weeks of inspiratory flow resistive loading (IFRL) on respiratory muscle and pulmonary function, and running time to exhaustion during a constant load exercise test in recreational runners; and secondly, to examine cardiopulmonary and metabolic responses during the same constant load exercise test. This is especially important because there is still uncertainty in the literature regarding the influence of RMT upon factors such as breathing pattern, oxygen cost, perceptual response to exercise, $[\text{Lac}^-]_{\text{B}}$ and heart-rate (HR) response during exercise.

We hypothesized that 6 weeks of IFRL would improve the performance of recreational runners during a treadmill run to exhaustion at 80% $\dot{V}O_{2\text{max}}$, increase inspiratory muscle strength, endurance, power output and work capacity, and reduce $\dot{V}O_2$, minute ventilation (\dot{V}_E), breathing frequency (f_b) and tidal volume (V_T), perception of dyspnea and $[\text{Lac}^-]_{\text{B}}$ during exercise.

Methods

Subjects

Twenty-four recreational road runners [12 male; mean age 21.5 (SD 1.9) years, mean height 174.6 (4.6) cm, mean body mass 68.9 (6.4) kg], recruited from a university population participated in this study. All participants had normal spirometry and had no history of heart and respiratory disease (as determined by questionnaire). All subjects were moderately active and engaged in 20–40 miles/week of mainly low- to moderate-intensity running three to five times per week. All subjects refrained from alcohol and physical exercise 24 h before the tests, and from drinking caffeinated beverages on test days. All females were tested during the follicular phase of their menstrual cycle (as determined by questionnaire) and four female subjects were taking tri-phasic oral contraceptives for at least 6 months before testing. The study protocols and procedures were approved by the Local Research Ethics Committee. All subjects gave written informed consent to participate in the study and agreed to maintain their usual running schedule (same intensity, duration and type of training) and to keep a log to record their physical activity for duration of the study.

Study design

Before entering the study protocol, all subjects underwent a 1-week orientation period for all laboratory-based respiratory muscle and pulmonary function tests in order to remove any potential learning effect. Before the start of the training phase

of the study all subjects reported to the laboratory to complete medical history and physical activity questionnaires, undertake pulmonary and respiratory muscle (strength and endurance) function tests and to complete an incremental treadmill exercise test to exhaustion in order to determine each subjects $\dot{V}O_{2\text{max}}$. Seventy-two hours later the subjects returned to the laboratory to complete a submaximal treadmill test to exhaustion at a workload, which had been determined to elicit $\sim 80\% \dot{V}O_{2\text{max}}$. After all baseline measurements were completed the subjects were randomly assigned from each gender into one of three groups. The experimental (IFRL) group ($n = 8$; 4 males) and sham-IFRL ($n = 8$; 4 males) performed IFRL 3 days/week for 6 weeks, while a control (CON) group ($n = 8$; 4 males) performed no IFRL, during the course of the 6-week study period. Following the 6-week IMT study period, pulmonary and respiratory muscle function tests and the submaximal treadmill test to exhaustion were completed by all subjects in the same manner and sequence as performed before the IMT study period (baseline). All these tests were completed at a similar time of day and under similar environmental conditions.

Spirometry and lung volume measurements

Spirometry was performed with the subject in the sitting position while breathing room air, with the nose being occluded by a clip. All testing was completed using a calibrated computerized spirometer (Superspiro, Micro Medical, Rochester, Kent, UK), according to the ATS/ERS Task Force recommendations on Standardization of Lung Function Testing (Miller et al., 2005), which states that an adequate test requires a minimum of three acceptable forced vital capacity (FVC) maneuvers. Acceptable repeatability is achieved when the difference between the largest and the next largest FVC is ≤ 0.150 L. The best of three consistent trials was recorded. The pulmonary function technician and spirometer were the same throughout the study. The procedure for all spirometry tests was (1) three normal tidal volume breaths, (2) maximal inhalation, (3) forced maximal exhalation and (4) maximal inhalation. The pulmonary function assessment also included a isocapnic maximal voluntary ventilation over 12-s (MVV_{12}) test, which required each subject to inspire and expire deeply as fast as possible for a period of 12 s. Residual lung volume and total lung capacity (TLC) were measured using the nitrogen dilution method with a SensorMedics V6200 Auto-box (VIASYS Healthcare, Warwick, UK).

IMT protocol

The IFRL and sham-IFRL group performed IMT three times per week in the laboratory and under supervision. The device used was the RT2 trainer and associated software (DeVilbiss Sunrise Medical Ltd., Wollaston, UK). The RT2 training device is a pressure manometer with a 2 mm leak, which utilizes an infra-red link to a computer containing the software of the “Test of Incremental Respiratory Endurance” (TIRE) regimen, as previously described (Mickleborough et al., 2008). The 2 mm leak provides a set resistance to inspiratory flow. The TIRE protocol requires each subject to forcefully exhale to residual lung volume (RV) (expiration unloaded), followed immediately by each subject breathing in maximally against the resistance (2 mm leak) from RV to TLC until task failure. This effort was recorded on a computer screen as sustained maximum inspiratory pressure (SMIP), which is the area under the curve (Fig. 1(a) and (b)). The best of three SMIP maneuvers was selected and visually redrawn on the computer screen to a training template set at 80% (IFRL group) or 30%

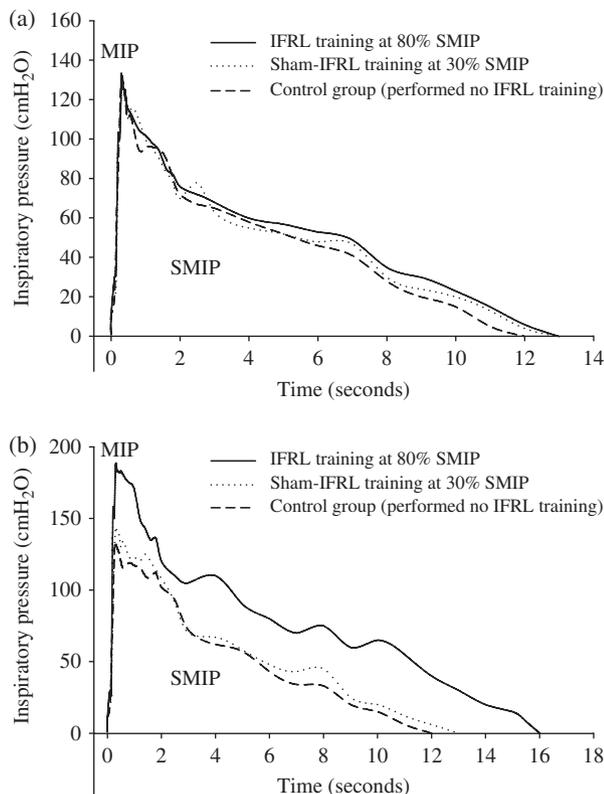


Fig. 1. (a) Tests of incremental respiratory endurance (TIRE) templates before the 6-week study period (baseline). MIP, maximal inspiratory pressure; SMIP, sustained maximal inspiratory pressure (area under the curve); IFRL, inspiratory flow resistive loading. (b) TIRE templates post 6-week study period. MIP, maximal inspiratory pressure; SMIP, sustained maximal inspiratory pressure (area under the curve); IFRL, inspiratory flow resistive loading.

(sham-IFRL group) of the peak pressure achieved over the time of inspiration. This fixed the pressure load over the full inspiratory volume range. The 80% (IFRL group) or 30% (sham-IFRL group) SMIP training template was presented on a computer screen together with a countdown clock and scores based on the pressures achieved expressed in pressure time units. This provided computerized biofeedback to each subject during training, while scores were recorded to the computer database and provided further feedback of any training progress. A set training regimen then required that the on-screen template was matched or exceeded by participants within a progressively increased work-rest ratio as previously described (Mickleborough et al., 2008).

The electronic manometer was calibrated by plotting a graph of known flow rates against different pressures in order to establish a calibration curve for the manometer. By using this data the volume of air entering the manometer was determined at a given pressure, which was then used to give conversions from pressure to energy and power. As shown previously (Mickleborough et al., 2008), a leak calibration constant was calculated, from flow rate (Q), as follows:

$$Q = 3.226 \times 10^{-6} \times \sqrt{p}$$

where pressure (p) was expressed in N/m² and Q in m³/s. Power (P) developed was derived from the assessment of p and Q , such that $P = p \times Q$, and the maximal inspiratory muscle power output (IMPO_{max}) expressed in watts. The work per

breath was derived from the power curve and expressed in Joules (J)/breath of inspiratory muscle work capacity (IMWC) (Chatham et al., 2004; Enright et al., 2006a; Mickleborough et al., 2008).

Inspiratory muscle strength

Inspiratory muscle strength was measured in all subjects (IFRL, sham-IFRL and CON groups) as the maximum negative inspiratory pressure generated at RV and sustained during a maximal inspiration using the RT2 pressure manometer. MIP and SMIP were measured by asking subjects to maximally inhale against the set resistance from RV to TLC and were again recorded on the computer screen. This was recorded as an indication of the work performed at each maximal breath, as the inspiratory muscles contracted throughout their full range. The inspiratory time of contraction (T_{cont}) during the SMIP maneuver was also recorded.

Inspiratory muscle endurance

Inspiratory muscle endurance in all subjects (IFRL, sham-IFRL and CON groups) was determined by computing the total accumulated SMIPs (\sum SMIP) generated for each training load successfully completed and recorded by the RT2 software during a TIRE-IMT session and conducted at the beginning and end of the 12-week study period. As an additional measure of inspiratory muscle endurance, all subjects were asked to match a 75% SMIP target presented every 10 s via the countdown clock, and the time to failure (T_{lim}) (i.e., unable to match at least 90% of the target template) was recorded before and after the 12-week study period.

Maximal exercise testing

An incremental exercise treadmill (Woodway Ergo ELG 2, Rhine, Germany) test to exhaustion was performed in order to determine each subject's $\dot{V}O_{2max}$. Resting measurements were taken for 3 min before the exercise test. The incremental treadmill test started with a 5-min warm-up followed by 5 min of stretching. On completion of the warm-up subjects began running at 10–13 km/h (females 10–11 km/h; males 12–13 km/h), with increments of 1 km/h every min until volitional exhaustion. Criteria used for determining whether each subject had attained $\dot{V}O_{2max}$ were an respiratory exchange ratio (RER) > 1.10, a HR within 10% of predicted HR_{max} and/or a plateau in $\dot{V}O_2$ (<150 mL/min) with an increase in treadmill grade. Ventilatory and metabolic data were collected continuously using breath-by-breath gas analysis throughout the exercise period (Pulmolab EX670, Morgan Medical, Kent, UK). The system was calibrated before each exercise test. The mass flow sensor was calibrated against a Hans Rudolf 3.0 L syringe at various flow rates and verification was accepted at ± 0.01 L. Gases were calibrated automatically from a gas cylinder containing known concentrations (14.52% O₂, 4.95% CO₂, 4.78% Ar and 75.5% N₂) (BOC Gases Ltd., Guildford, Surrey, UK). HR was recorded at rest, continuously during and following exercise and following exercise (Polar S625XTM, Polar Electro, Oy, Finland).

Submaximal endurance exercise testing

Endurance exercise capacity was determined on a motorized treadmill to volitional fatigue at a workload corresponding to $\sim 80\%$ $\dot{V}O_{2max}$, which was determined from the maximal

exercise test data. All subjects completed a 3-min warm-up directly followed by treadmill running at a 4% grade and a subsequent speed that corresponded to 80% $\dot{V}O_{2\max}$. Ventilatory and metabolic data and HR were collected in the same manner as during maximal exercise test. Endurance run time to exhaustion (ERTE) was recorded and used for data analysis. At rest, at 3 and 9 min during exercise and 1 and 5 min during recovery, a 25–50 μL capillary blood sample from the earlobe was obtained and analyzed for $[\text{Lac}^-]_{\text{B}}$ using an Analox GM7 multi-assayer (Analox Instruments Ltd., London, UK). The instrument was calibrated with a known lactate standard (8.0 mmol/L) before each test in accordance with the manufacturer's instructions. In addition, at 3 min and then at 5 min intervals during the exercise test, each subject was asked to estimate their rating of perceived dyspnea using the modified Borg scale (Borg, 1982).

Statistical analysis

Data were analyzed using SPSS version 15.0 statistical software (SPSS Inc., Chicago, USA). All data were assessed for normality using the Kolmogorov–Smirnov test, and Levene's test was used to test for homogeneity of variance between groups. Data were analyzed using a 3×2 (group \times condition) ANOVA. Where a significant F-ratio was found, Fisher's protected least-square difference *post hoc* test, with a Bonferroni adjustment (used to maintain an overall type-I error rate of 5%), was used to isolate differences in group means. Pulmonary and respiratory muscle function data are expressed as mean and their 95% confidence level. Ventilatory, metabolic, dyspnea rating and ERTE data are expressed as mean \pm standard deviation. Pearson product moment correlation coefficients were computed in order to evaluate the relationship between relative changes in selected dependent variables following IFRL. Statistical significance was accepted if $P \leq 0.05$.

Results

Subjects

There were no significant difference ($P > 0.05$) between mean height, body mass or age between groups. No significant difference ($P > 0.05$) were detected between genders in any of the measured variables, although this was to be expected because this study was not statistically powered to detect gender differences between groups ($n = 4$ from each gender in each group). There was no significant difference in baseline $\dot{V}O_{2\max}$ between groups. According to the physical activity questionnaires administered at the beginning and end of the training period subjects did not alter their physical activity status during the study.

Pulmonary and respiratory muscle function

Pulmonary and respiratory muscle function variables measured at the beginning and end of the 6-week study period were not significantly different ($P > 0.05$) within or between the sham-IFRL (Table 2) and CON (Table 3) groups. In addition, all pre-training measurements of pulmonary and respiratory muscle function for the IFRL group were not statistically different ($P > 0.05$) compared with the sham-IFRL and CON groups (Table 1, 2 and 3). However, while post-training values for FVC, forced expiratory volume in 1 s (FEV_1), residual lung volume (RV) and total lung capacity (TLC) for the IFRL group were not significantly altered ($P > 0.05$) at the end of the study period, or significantly different ($P > 0.05$)

Table 1. Respiratory muscle and pulmonary function values for experimental group (IFRL training at 80% SMIP)

	Pre	Post	Diff.	% Δ	P-value	L95% CI	U95% CI
<i>Respiratory muscle function</i>							
MIP (cmH ₂ O)	128.9 \pm 17.9	185.5 \pm 17.5	56.5*	+43.9	<0.001	37.6	75.6
SMIP (PTU)	783.1 \pm 188.4	1066.5 \pm 204.4	283.4*	+26.6	0.006	72.6	494.2
IMPO _{max} (W)	4.60 \pm 0.37	7.92 \pm 0.51	3.32	+41.9	0.013	1.23	9.64
IMWC (J/breath)	10.4 \pm 3.4	16.9 \pm 3.6	6.50	+38.5	0.006	2.97	9.65
T_{cont} (s)	13.0 \pm 2.87	16.0 \pm 2.3	3.00*	+18.8	0.019	0.21	5.80
Σ SMIP (PTU)	21735.9 \pm 6190	32142.1 \pm 5782	10406.2*	+32.4	0.002	3982.5	16830.0
T_{lim} (min)	3.15 \pm 1.23	3.90 \pm 0.97	0.75*	+23.8	0.001	0.38	1.12
MVV ₁₂ (L/min)	145.0 \pm 22.9	159.6 \pm 16.2	14.6	+9.2	0.081	-2.63	35.9
<i>Pulmonary function</i>							
FVC (L)	4.73 \pm 0.79	4.83 \pm 0.64	0.10	+2.1	0.447	-0.24	0.56
FEV_1 (L)	4.06 \pm 0.57	4.12 \pm 0.49	0.06	+1.5	0.172	-0.48	0.71
FIV_1 (L)	3.76 \pm 0.34	4.12 \pm 0.37	0.36	+9.6	0.002	0.14	0.53
RV (L)	1.56 \pm 0.31	1.32 \pm 0.28	-0.24	-15.4	0.280	-0.67	1.07
TLC (L)	6.29 \pm 1.2	6.15 \pm 1.1	-0.15	-2.2	0.227	-0.35	1.03

Values are means \pm SD.

*Significant difference from pre-value ($P < 0.05$).

Diff., difference; % Δ , percentage change; L95%CI, lower 95% confidence interval; U95%CI, upper 95% confidence interval; MIP, maximal inspiratory pressure; SMIP, sustained maximal inspiratory pressure (pressure time units); IMPO_{max}, maximal inspiratory muscle power output; IMWC, inspiratory muscle work capacity; T_{cont} , inspiratory time of contraction; Σ SMIP, the total area of SMIPs performed to the point of failure summed; T_{lim} , time to fatigue (performance test); MVV₁₂, maximal voluntary ventilation in 12 s; FVC, forced vital capacity; FEV_1 , forced expiratory volume in 1 s; FIV_1 , forced inspiratory time in 1 s; RV, residual lung volume; TLC, total lung capacity.

Table 2. Respiratory muscle and pulmonary function values for placebo group (sham-IFRL training at 30% SMIP)

	Pre	Post	Diff.	%Δ	P-value	L95% CI	U95% CI
<i>Respiratory muscle function</i>							
MIP (cmH ₂ O)	131.0 ± 29.4	142.9 ± 34.5	11.6	+9.1	0.240	- 22.8	46.0
IMPO _{max} (W)	4.70 ± 0.42	5.35 ± 0.46	0.65	+13.4	0.123	- 1.24	4.64
SMIP (PTU)	678.5 ± 211.8	741.1 ± 288.8	62.6	+9.2	0.289	- 229.0	173.8
IMWC (J/breath)	9.8 ± 2.8	10.4 ± 3.1	0.60	+6.1	0.427	- 0.96	3.65
T _{cont} (s)	11.8 ± 2.78	12.8 ± 2.76	1.0	+8.5	0.087	- 1.64	3.24
ΣSMIP (PTU)	10709.0 ± 5595	11367.3 ± 4131	658.3	+9.3	0.397	- 5932.5	4616.0
T _{lim} (min)	3.30 ± 0.99	3.49 ± 0.94	0.19	+5.8	0.359	- 0.89	1.25
MVV ₁₂ (L/min)	126.0 ± 14.5	135.8 ± 20.7	9.8	+22.1	0.147	9.4	28.9
<i>Pulmonary function</i>							
FVC (L)	4.67 ± 0.61	4.79 ± 0.60	0.12	+2.6	0.357	- 0.54	0.76
FEV ₁ (L)	3.95 ± 0.67	4.17 ± 0.56	0.17	+5.6	0.063	- 0.11	0.43
FIV ₁ (L)	3.63 ± 0.30	3.78 ± 0.35	0.15	+4.1	0.108	- 0.17	0.36
RV (L)	1.22 ± 0.55	1.28 ± 0.46	0.06	+4.9	0.406	- 0.48	0.60
TLC (L)	5.89 ± 1.6	6.07 ± 1.5	0.18	+3.1	0.108	- 0.07	0.97

Values are means ± SD.

*Significant difference from pre-value ($P < 0.05$).

Diff., difference; %Δ, percentage change; L95%CI, lower 95% confidence interval; U95%CI, upper 95% confidence interval; MIP, maximal inspiratory pressure; SMIP, sustained maximal inspiratory pressure (pressure time units); IMPO_{max}, maximal inspiratory muscle power output; IMWC, inspiratory muscle work capacity; T_{cont}, inspiratory time of contraction; ΣSMIP, the total area of SMIPs performed to the point of failure summed; T_{lim}, time to fatigue (performance test); MVV₁₂, maximal voluntary ventilation in 12 s; FVC, forced vital capacity; FEV₁, forced expiratory volume in 1 s; FIV₁, forced inspiratory time in 1 s; RV, residual lung volume; TLC, total lung capacity.

Table 3. Respiratory muscle and pulmonary function values for control group (performed no IFRL training)

	Pre	Post	Diff.	%Δ	P-value	L95% CI	U95% CI
<i>Respiratory muscle function</i>							
MIP (cmH ₂ O)	133.9 ± 23.9	133.8 ± 21.0	0.10	- 0.08	0.499	- 24.1	24.0
IMPO _{max} (W)	4.86 ± 0.34	4.87 ± 0.31	0.01	+0.21	0.431	- 0.25	0.13
SMIP (PTU)	1000.1 ± 251.7	906.5 ± 171.4	- 93.6	- 9.4	0.200	- 324.6	137.3
IMWC (J/breath)	10.1 ± 3.8	9.7 ± 3.3	- 0.4	- 4.0	0.392	- 1.32	2.96
T _{cont} (s)	11.9 ± 2.94	11.9 ± 2.97	0.01	0.0	0.497	- 3.16	3.14
ΣSMIP (PTU)	18922.8 ± 3548	22035.1 ± 4158	3112.3	+16.5	0.065	- 1032.8	7257.5
T _{lim} (min)	2.74 ± 1.17	2.71 ± 1.21	- 0.03	+1.1	0.477	- 1.32	1.23
MVV ₁₂ (L/min)	142.1 ± 19.9	147.0 ± 20.9	4.9	+3.5	0.320	- 17.0	26.8
<i>Pulmonary function</i>							
FVC (L)	4.86 ± 0.24	4.93 ± 0.20	0.07	+1.5	0.274	- 0.17	0.30
FEV ₁ (L)	3.98 ± 0.48	4.00 ± 0.45	0.02	+0.5	0.494	- 0.48	0.47
FIV ₁ (L)	3.78 ± 0.39	3.84 ± 0.36	0.06	+1.6	0.341	- 0.23	0.56
RV (L)	1.14 ± 0.60	1.13 ± 0.47	- 0.01	+0.9	0.496	- 0.48	0.47
TLC (L)	6.00 ± 1.8	6.06 ± 1.5	0.06	+1.0	0.293	- 0.84	0.65

Values are means ± SD.

*Significant difference from pre-value ($P < 0.05$).

Diff., difference; %Δ, percentage change; L95%CI, lower 95% confidence interval; U95%CI, upper 95% confidence interval; MIP, maximal inspiratory pressure; SMIP, sustained maximal inspiratory pressure (pressure time units); IMPO_{max}, maximal inspiratory muscle power output; IMWC, inspiratory muscle work capacity; T_{cont}, inspiratory time of contraction; ΣSMIP, the total area of SMIPs performed to the point of failure summed; T_{lim}, time to fatigue (performance test); MVV₁₂, maximal voluntary ventilation in 12 s; FVC, forced vital capacity; FEV₁, forced expiratory volume in 1 s; FIV₁, forced inspiratory time in 1 s; RV, residual lung volume; TLC, total lung capacity.

compared with the sham-IFRL and CON groups, forced inspiratory volume in 1 s (FIV₁) was significantly increased ($P < 0.05$) at the end of the study period by $9.6 \pm 3.1\%$ in the IFRL group (Table 1), and by $8.2 \pm 3.5\%$ and $6.8 \pm 2.9\%$ (Table 1) compared with the sham-IFRL and CON group, respectively.

No significant difference ($P > 0.05$) was observed in respiratory muscle function measures at baseline

between groups (Tables 1, 2 and 3; Fig. 1(a)). In addition, no significant change ($P > 0.05$) was observed between respiratory muscle function variables measured at baseline and at the end of the study period in the sham-IFRL (Table 2) and CON (Table 3) groups. However, in the IFRL group (Table 1) there was a significant increase ($P < 0.05$) in MIP ($43.9 \pm 7.4\%$), SMIP ($26.6 \pm 6.7\%$), IMPO_{max} ($41.9 \pm 5.1\%$), IMWC ($38.5 \pm 5.9\%$), time of contraction

($18.8 \pm 2.8\%$), sum of SMIP ($32.4 \pm 6.1\%$) and time to fatigue (performance test) ($23.8 \pm 2.4\%$) compared with baseline.

Although IFRL failed to improve MVV, significant positive relationships were observed between the relative changes in \dot{V}_{O_2} and MVV ($r = 0.62$, $P = 0.010$). In addition, in the IFRL group significant positive relationships were observed between the relative changes in MIP and \dot{V}_{O_2} ($r = 0.72$, $P = 0.008$), MIP and IMPO_{\max} ($r = 0.79$, $P = 0.004$) and IMPO_{\max} and \dot{V}_{O_2} ($r = 0.64$, $P = 0.009$). Furthermore, in the IFRL group significant positive relationships were found between the relative change in MIP and rating of perceived exertion (RPE) ($r = 0.71$, $P = 0.008$), IMPO_{\max} and RPE ($r = 0.73$, $P = 0.005$) and \dot{V}_{O_2} and RPE ($r = 0.61$, $P = 0.021$).

Responses to exercise and running time to exhaustion

Baseline measures of \dot{V}_{O_2} (Fig. 2(a)), \dot{V}_E (Fig. 2(b)), RER (Fig. 3(a)), breathing frequency (Fig. 3(b)), tidal volume (Fig. 4(a)), dyspnea ratings (Fig. 4(b)), HR (Fig. 5(a)) and $[\text{Lac}^-]_B$ (Fig. 5(b)) were not significantly different ($P > 0.05$) between groups. Following the 6-week training period, \dot{V}_{O_2} , \dot{V}_E , RER, breathing frequency, tidal volume, dyspnea ratings, HR and $[\text{Lac}^-]_B$ were unaltered from baseline during the fixed work-rate submaximal test at all time points ($P > 0.05$) in the sham-IFRL and CON groups. However, while \dot{V}_{O_2} , \dot{V}_E , RER, breathing frequency, dyspnea ratings and HR rose progressively with time, these measures were significantly lower ($P < 0.05$) at the majority of time points (e.g., 2, 5, 8, 12 and 17 min) during the submaximal exercise test after the training period in the IFRL group only. Tidal volume (Fig. 4(a)) was significantly attenuated ($P < 0.05$) following the training period in the IFRL group during the submaximal exercise test at 2, 5 and 8 min. Blood lactate concentration (Fig. 5(b)) was significantly reduced ($P < 0.05$) following the training period during the submaximal exercise test, and at 1 and 5 min post-exercise, in the IFRL group only. There were no significant differences ($P > 0.05$) between groups at maximum exercise for \dot{V}_{O_2} , \dot{V}_E , RER, breathing frequency, tidal volume, dyspnea rating and HR when comparing baseline with post-training period measures.

Running time to exhaustion was not significantly different ($P > 0.05$) between groups before training (IFRL, 20.7 ± 1.4 min; sham-IFRL, 20.0 ± 2.3 min; CON, 19.9 ± 3.31 min) (Fig. 6) or between the sham-IFRL (19.6 ± 1.8 min) and CON (20.7 ± 3.4 min) group following training. However, following the training period ERTE significantly improved in the IFRL group (24.1 ± 1.43 min) compared with the sham-IFRL and CON group. In addition, in the IFRL group there was a significant correlation

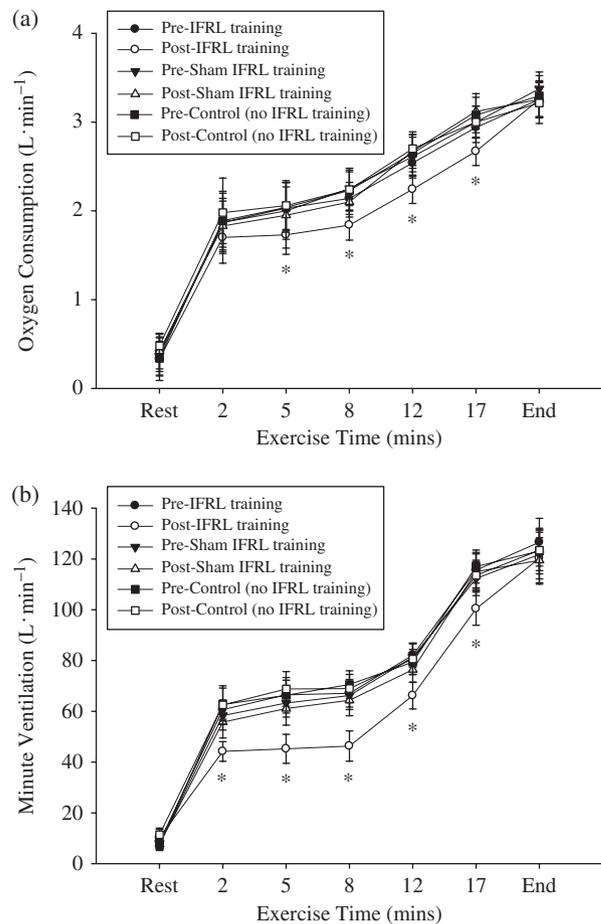


Fig. 2. (a) Oxygen consumption response during exercise before and after the 6-week study period. *Post-IFRL group significantly different ($P < 0.05$) compared with all other groups. (b) Minute ventilation response during exercise before and after the 6-week study period. *Post-IFRL group significantly different ($P < 0.05$) compared with all other groups. IFRL, inspiratory flow resistive loading.

between the change in running performance and the changes in \dot{V}_{O_2} ($r = 0.64$, $P = 0.019$), \dot{V}_E ($r = 0.68$, $P = 0.011$), RER ($r = 0.72$, $P = 0.008$), breathing frequency ($r = 0.61$, $P = 0.028$), tidal volume ($r = 0.58$, $P = 0.034$), RPE ($r = 0.64$, $P = 0.021$), HR ($r = 0.75$, $P = 0.006$) and $[\text{Lac}^-]_B$ ($r = 0.70$, $P = 0.010$) between baseline and post-intervention.

Discussion

The results of the present study have demonstrated that IFRL performed 3 days/week for 6 weeks can (1) alter breathing mechanics, lower the oxygen cost and attenuate ventilatory, HR, $[\text{Lac}^-]_B$ and the perceptual response during constant workload exercise, (2) increase inspiratory muscle strength, endurance, IMPO_{\max} and IMWC and (3) improve exercise performance of recreational runners during a laboratory treadmill running time to exhaustion test at 80%

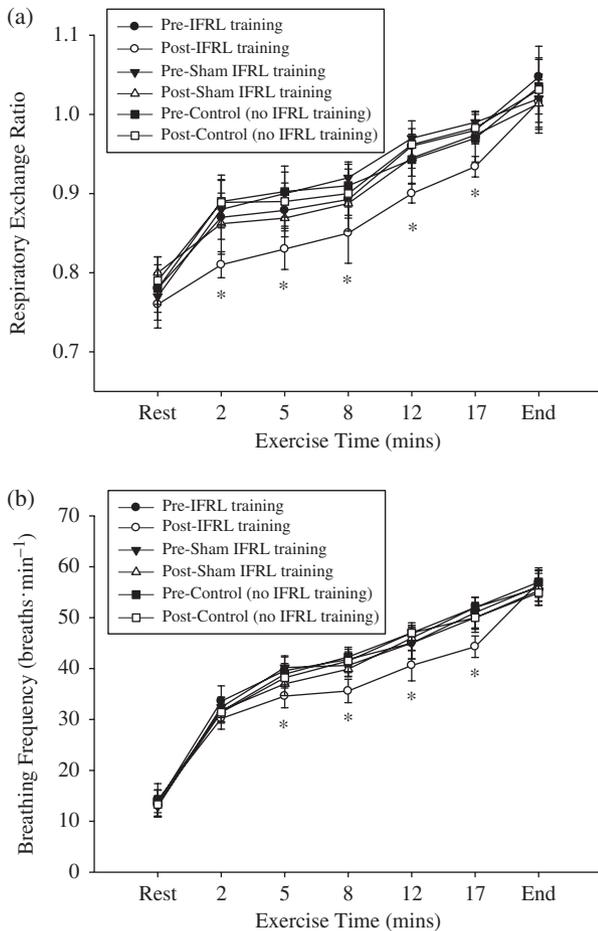


Fig. 3. (a) Respiratory exchange ratio response during exercise before and after the 6-week study period. *Post-IFRL group significantly different ($P < 0.05$) compared with all other groups. (b) Breathing frequency response during exercise before and after the 6-week study period. *Post-IFRL group significantly different ($P < 0.05$) compared with all other groups. IFRL, inspiratory flow resistive loading.

of $\dot{V}O_{2\max}$. We observed no changes in cardiopulmonary and metabolic responses to maximum running exercise as a consequence of IFRL, which is a consistent finding within the literature (Inbar et al., 2000; Williams et al., 2002; Edwards et al., 2008).

Pulmonary and respiratory muscle function

Our work supports data from other studies that showed no change in expiratory measures of pulmonary function as a consequence of either IMT or VIH in runners (Hanel & Secher, 1991; Inbar et al., 2000; Williams et al., 2002; Leddy et al., 2007) and cyclists (Gething et al., 2004b). Consistent with our previous work (Mickleborough et al., 2008) and others (Wells et al., 2005) FIV_1 increased in the IRFL group, which may be the result of an increase in the inspiratory muscle velocity of shortening as a result of improved inspiratory muscle strength. Inter-

Inspiratory muscle training and running

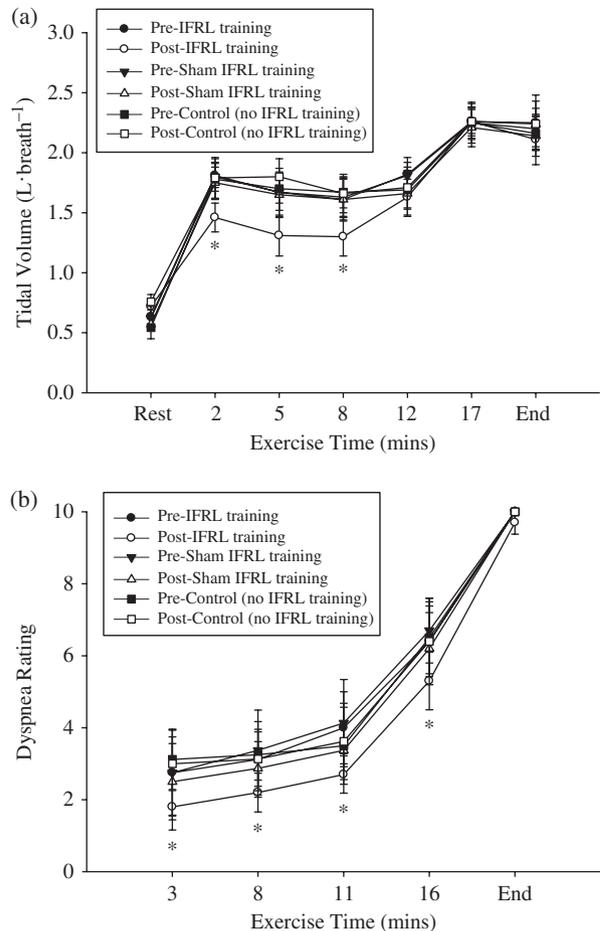


Fig. 4. (a) Tidal volume response during exercise before and after the 6-week study period. *Post-IFRL group significantly different ($P < 0.05$) compared with all other groups. (b) Dyspnea rating response during exercise before and after the 6-week study period. *Post-IFRL group significantly different ($P < 0.05$) compared with all other groups. IFRL, inspiratory flow resistive loading.

estingly, Romer et al., 2002a, b, c, p. 346) similarly found an increase in peak inspiratory flow rate following IMT, and combined with our data from the present study, these findings are in agreement with the pressure-flow specificity of IMT (Tzelepis et al., 1994).

However, similar to the findings of other studies evaluating the efficacy either IMT or VIH in elite and recreational runners (Hanel & Secher, 1991; Chatham et al., 1999; Inbar et al., 2000; Romer et al., 2002b; Williams et al., 2002; Edwards & Cooke, 2004; Downey et al., 2007; Edwards et al., 2008), in the present study inspiratory muscle strength and endurance significantly improved by 30.5% and 19.2%, respectively, in the IRFL group. The magnitude of improvement in inspiratory muscle strength and endurance is consistent with previous studies in runners using a variety of RMT devices/protocols (Hanel & Secher, 1991; Chatham et al., 1999; Inbar

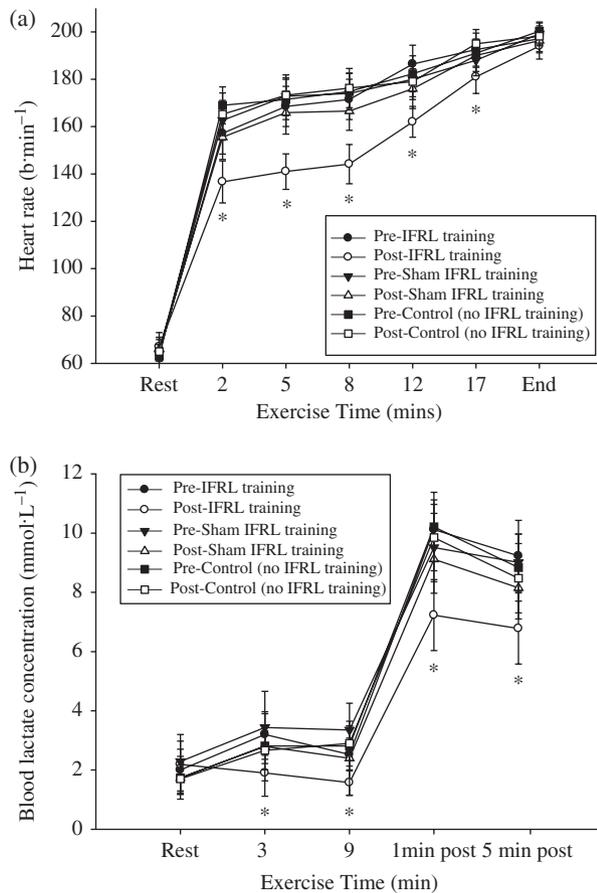


Fig. 5. (a) Heart rate response during exercise before and after the 6-week study period. *Post-IFRL group significantly different ($P < 0.05$) compared with all other groups. (b) Blood lactate response during exercise before and after the 6-week study period. *Post-IFRL group significantly different ($P < 0.05$) compared with all other groups. IFRL, inspiratory flow resistive loading.

et al., 2000; Romer et al., 2002b; Williams et al., 2002; Edwards & Cooke, 2004; Downey et al., 2007; Edwards et al., 2008). The magnitude of increase in inspiratory muscle strength following IMT varies from 12.6% in untrained individuals to 25.0% in repetitive sprint sport players (Romer et al., 2002b), 19.7% to 31% in elite runners (Inbar et al., 2000; Williams et al., 2002) and up to 45.5% in competitive rowers (Volianitis et al., 2001). However, the magnitude of change in inspiratory muscle endurance in studies is more variable. Inbar et al. (2000) demonstrated an increase of only 10%, while inspiratory muscle endurance increased by 128% in elite runners in the study by Williams et al. (2002).

Consistent with our previous findings in elite swimmers (Mickleborough et al., 2008), this study has shown that $IMPO_{max}$ and $IMWC$ increase following IFRL in recreational runners. The use of a fixed 2 mm leak allowed for conversion of SMIP measures to SI units of power and work. Unlike the measurement of MIP, $IMWC$ is a measure of pres-

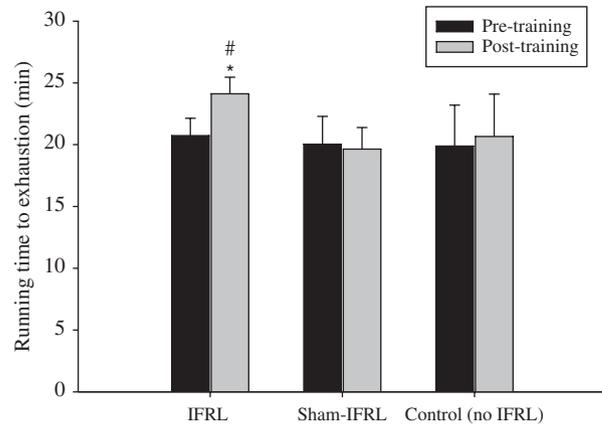


Fig. 6. Endurance running time to exhaustion (ERTE) before and after the 6-week study period. *Post-IFRL group significantly different ($P < 0.05$) compared with all other groups. #Post-IFRL ERTE significantly different ($P < 0.05$) to pre-IFRL value. IFRL, inspiratory flow resistive loading.

sure generation over the full range of lung volumes from RV to TLC (Enright et al., 2006a). The practical consequence of a longer and more powerful contraction, as shown by an improvement in T_{cont} in the IFRL group, is a functional increase in inspiratory flow. The increase in $IMPO_{max}$ in the IFRL group suggests that IMT may increase the velocity of contraction of the inspiratory muscles. As with other skeletal muscles, it has been shown that the inspiratory muscles can be trained to increase their capacity to generate force (pressure) or velocity of muscle shortening (Tzelepis et al., 1999). The improvement in the velocity of muscle shortening and $IMPO_{max}$ with training may be attributed to a change in the intrinsic contractile properties of the muscle fibers or, more possibly, to a different recruitment of muscle fibers.

Responses to exercise and running time to exhaustion

The present study identified a significant 14.1% improvement in running performance in the IFRL group. Our data are consistent with other studies that have observed an improvement in ERTE and 4-mile run time (Edwards & Cooke, 2004; Leddy et al., 2007) and 5000 m running performance (Edwards et al., 2008) after either pressure-threshold IMT or VIH.

The results of the present study have shown that IFRL can reduce $[Lac^-]_B$, perception of dyspnea, HR, $\dot{V}O_2$, (\dot{V}_E), RER in association with attenuated f_b , V_T during submaximal run, suggesting that IFRL reduced the work of breathing and/or improved ventilatory system efficiency. The reduced \dot{V}_E and $\dot{V}O_2$ during the 80% $\dot{V}O_{2max}$ treadmill run following IFRL is consistent with prior studies in runners (Leddy et al., 2007) and cyclists (Gething et al.,

2004b), and with previously reported data for the energy cost of breathing (Leddy et al., 2007). An alternative explanation for the reduction in $\dot{V}O_2$ post-IFRL may be due to improved running efficiency. In the present study, there was a significant reduction in $\dot{V}O_2$ during the submaximal run as the subjects fatigued. Previous studies have shown that respiratory muscle work has to be increased to near fatiguing levels for muscle sympathetic nerve activity to be increased (St Croix et al., 2000) or for leg blood flow to be reduced during exercise (Harms et al., 1997). In addition, exercise-induced diaphragm fatigue was only observed in healthy subjects of varying fitness levels when the intensity of exercise exceeded 80–85% $\dot{V}O_{2\max}$ and the exercise was sustained to exhaustion (Johnson et al., 1993; Babcock et al., 1998). Consistent with these observations fatiguing exercise impairs neuromuscular performance (Komi, 2000). Therefore, the combination of these factors on the performance of the limb locomotor muscles may lead to reduced running efficiency and increased energy cost of running prior to IFRL. Thus, in the present study it is possible that an IFRL-induced improvement in the performance of the limb locomotor muscles may have resulted in an enhanced running economy. At present, the mechanisms responsible for the ergogenic effect of RMT on exercise performance are uncertain. Recent evidence suggests that IMT may generate improvements in exercise performance through two main mechanisms which are most likely interrelated: (1) attenuation of effort sensations, such that exercise feels easier following IMT (Romer et al., 2002a; Gething et al., 2004b) and (2) attenuation of the inspiratory muscle metaboreflex leading to a perseveration of limb locomotor blood flow during exercise (Witt et al., 2007).

Consistent with our study, it has been shown previously that IFRL can reduce HR during exercise (Gething et al., 2004b). Witt et al. (2007) suggests that this attenuated cardiovascular response suggests a blunted sympatho-excitation to resistive inspiratory work. It is plausible that the reduction in HR during exercise as a consequence of IFRL may decrease the cardiorespiratory cost of oxygen transport (Green et al., 2000). Decreased submaximal HR concomitant with improvements in the oxygen cost of running after live-high train-low (LHTL) altitude training have been observed (Saunders et al., 2007), and there may be a commonality in underlying mechanisms between IFRL and LHTL training in lowering the oxygen cost of running.

In the present study, we have shown in runners that IFRL can attenuate f_b , V_T and perception of dyspnea during exercise. Gething et al. (2004b) has shown previously that both f_b and perception of dyspnea are reduced in cyclists following 10 weeks of IFRL, while Romer et al. (2002a) have shown a

reduction in both respiratory and peripheral effort (BorgCR10) during a cycling time trial following 6 weeks of pressure-threshold IMT. Respiratory muscle fatigue could possibly limit exercise tolerance through an inadequate ventilatory response, a detrimental change in breathing mechanics and/or an increased sensation of dyspnea, and all of which may be potentially reversed by IFRL.

The present study has shown that IFRL can reduce $[\text{Lac}^-]_B$ during constant load running, and is consistent with prior studies utilizing either IMT (McConnell & Sharpe, 2005; Griffiths & McConnell, 2007) or VIH (Spengler et al., 1999; Leddy et al., 2007) that have shown a diminution of $[\text{Lac}^-]_B$ during exercise. Interestingly, Brown et al. (2008) have shown that an increase in $[\text{Lac}^-]_B$ observed during VIH at 80% $\dot{V}_{E\max}$ was attenuated following IMT. The mechanism by which RMT can moderate $[\text{Lac}^-]_B$ remains equivocal. It has been suggested that the reductions observed in $[\text{Lac}^-]_B$ result from increased uptake by the respiratory muscles, rather than a net decrease in lactate clearance (Spengler et al., 1999). Alternatively, it is possible that IMT-mediated changes in respiratory muscle function may contribute to lowering $[\text{Lac}^-]_B$ via affecting lactate clearance by and efflux from the trained respiratory muscles. Finally, the attenuated $[\text{Lac}^-]_B$ response to IRFL may be due to an IMT-mediated increase in the oxidative and/or monocarboxylate transport protein content of the inspiratory muscles (Brown et al., 2008).

In conclusion, this study has shown that 6 weeks of IRFL significantly improved respiratory muscle function, reduced HR and $[\text{Lac}^-]_B$, attenuated the ventilatory and perceptual response to exercise and improved ERTE on a treadmill at 80% $\dot{V}O_{2\max}$ in recreational runners.

Importantly our data show that IFRL can positively influence the conscious sensation of fatigue, alter breathing mechanics and lower the oxygen cost of running at constant workload exercise.

Perspectives

A great deal research has been conducted on the response of the respiratory muscles to IMT and its effects on exercise performance (McConnell & Romer, 2004). However, there is scant data (Edwards & Cooke, 2004; Downey et al., 2007; Leddy et al., 2007; Williams et al., 2002) available pertaining to the efficacy of IMT on cardiopulmonary and metabolic measures and running performance. The present study utilized a novel approach to IMT, specifically IFRL based on the TIRE training protocol and incorporating computerized biofeedback. This type of IMT has been shown to increase diaphragm

thickness, lung volumes and cycling exercise capacity in healthy subjects (Enright et al., 2006b), improve lung function and exercise capacity (Enright et al., 2004), improve cycling time to exhaustion (Gething et al., 2004b) and enhance sputum expectoration in patients cystic fibrosis (Chatham et al., 2004). Therefore, IFRL may prove to be beneficial to inspiratory

muscle strength and endurance and whole-body exercise performance in both healthy individuals and patients with cardiopulmonary disease.

Key words: inspiratory muscle training, exercise capacity, runners, ventilation.

References

- Babcock MA, Pegelow DF, Harms CA, Dempsey JA. Effects of respiratory muscle unloading on exercise-induced diaphragm fatigue. *J Appl Physiol* 2002; 93: 201–206.
- Babcock MA, Pegelow DF, Taha BH, Dempsey JA. High frequency diaphragmatic fatigue detected with paired stimuli in humans. *Med Sci Sports Exerc* 1998; 30: 506–511.
- Bisschop A, Gayan-Ramirez G, Rollier H, Gosselink R, Dom R, de Bock V, Decramer M. Intermittent inspiratory muscle training induces fiber hypertrophy in rat diaphragm. *Am J Respir Crit Care Med* 1997; 155: 1583–1589.
- Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982; 14: 377–381.
- Brown PI, Sharpe GR, Johnson MA. Inspiratory muscle training reduces blood lactate concentration during volitional hyperpnoea. *Eur J Appl Physiol* 2008; 104: 111–117.
- Chatham K, Baldwin J, Griffiths H, Summers L, Enright S. Inspiratory muscle training improves shuttle run performance in healthy subjects. *Physiol J* 1999; 85: 676–683.
- Chatham K, Ionescu AA, Nixon LS, Shale DJ. A short-term comparison of two methods of sputum expectoration in cystic fibrosis. *Eur Respir J* 2004; 23: 435–439.
- Dempsey JA. J.B. Wolfe memorial lecture. Is the lung built for exercise? *Med Sci Sports Exerc* 1986; 18: 143–155.
- Downey AE, Chenoweth LM, Townsend DK, Ranum JD, Ferguson CS, Harms CA. Effects of inspiratory muscle training on exercise responses in normoxia and hypoxia. *Respir Physiol Neurobiol* 2007; 156: 137–146.
- Edwards AM, Cooke CB. Oxygen uptake kinetics and maximal aerobic power are unaffected by inspiratory muscle training in healthy subjects where time to exhaustion is extended. *Eur J Appl Physiol* 2004; 93: 139–144.
- Edwards AM, Wells C, Butterly R. Concurrent inspiratory muscle and cardiovascular training differentially improves both perceptions of effort and 5000-m running performance compared to cardiovascular training alone. *Br J Sports Med* 2008; 42: 523–527.
- Enright S, Chatham K, Ionescu AA, Unnithan VB, Shale DJ. Inspiratory muscle training improves lung function and exercise capacity in adults with cystic fibrosis. *Chest* 2004; 126: 405–411.
- Enright S, Unnithan VB, Davies D. Reproducibility of measurements of inspiratory work capacity in cystic fibrosis patients. *Respir Physiol Neurobiol* 2006a; 150: 35–43.
- Enright SJ, Unnithan VB, Heward C, Withnall L, Davies DH. Effect of high-intensity inspiratory muscle training on lung volumes, diaphragm thickness, and exercise capacity in subjects who are healthy. *Phys Ther* 2006b; 86: 345–354.
- Gething AD, Passfield L, Davies B. The effects of different inspiratory muscle training intensities on exercising heart rate and perceived exertion. *Eur J Appl Physiol* 2004a; 92: 50–55.
- Gething AD, Williams M, Davies B. Inspiratory resistive loading improves cycling capacity: a placebo controlled trial. *Br J Sports Med* 2004b; 38: 730–736.
- Green HJ, Roy B, Grant S, Hughson R, Burnett M, Otto C, Pipe A, McKenzie D, Johnson M. Increases in submaximal cycling efficiency mediated by altitude acclimatization. *J Appl Physiol* 2000; 89: 1189–1197.
- Griffiths LA, McConnell AK. The influence of inspiratory and expiratory muscle training upon rowing performance. *Eur J Appl Physiol* 2007; 99: 457–466.
- Hanel B, Secher NH. Maximal oxygen uptake and work capacity after inspiratory muscle training: a controlled study. *J Sports Sci* 1991; 9: 43–52.
- Harms CA, Babcock MA, McClaran SR, Pegelow DF, Nিকেle GA, Nelson WB, Dempsey JA. Respiratory muscle work compromises leg blood flow during maximal exercise. *J Appl Physiol* 1997; 82: 1573–1583.
- Harms CA, Wetter TJ, McClaran SR, Pegelow DF, Nিকেle GA, Nelson WB, Hanson P, Dempsey JA. Effects of respiratory muscle work on cardiac output and its distribution during maximal exercise. *J Appl Physiol* 1998; 85: 609–618.
- Inbar O, Weiner P, Azgad Y, Rotstein A, Weinstein Y. Specific inspiratory muscle training in well-trained endurance athletes. *Med Sci Sports Exerc* 2000; 32: 1233–1237.
- Johnson BD, Babcock MA, Suman OE, Dempsey JA. Exercise-induced diaphragmatic fatigue in healthy humans. *J Physiol (Lond)* 1993; 460: 385–405.
- Komi PV. Stretch-shortening cycle: a powerful model to study normal and fatigued muscle. *J Biomech* 2000; 33: 1197–1206.
- Leddy JJ, Limprasertkul A, Patel S, Modlich F, Buyea C, Pendergast DR, Lundgren CE. Isocapnic hyperpnea training improves performance in competitive male runners. *Eur J Appl Physiol* 2007; 99: 665–676.
- Lomax ME, McConnell AK. Inspiratory muscle fatigue in swimmers after a single 200 m swim. *J Sports Sci* 2003; 21: 659–664.
- McConnell AK, Romer LM. Respiratory muscle training in healthy humans: resolving the controversy. *Int J Sports Med* 2004; 25: 284–293.
- McConnell AK, Sharpe GR. The effect of inspiratory muscle training upon maximum lactate steady-state and blood lactate concentration. *Eur J Appl Physiol* 2005; 94: 277–284.
- Mickleborough TD, Stager JM, Chatham K, Lindley MR, Ionescu AA. Pulmonary adaptations to swim and inspiratory muscle training. *Eur J Appl Physiol* 2008; 103: 635–646.
- Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, Crapo R, Enright P, van der Grinten CP, Gustafsson P, Jensen R, Johnson DC, MacIntyre N, McKay R, Navajas

- D, Pedersen OF, Pellegrino R, Viegi G, Wanger J. Standardisation of spirometry. *Eur Respir J* 2005; 26: 319–338.
- Romer LM, McConnell AK, Jones DA. Effects of inspiratory muscle training on time-trial performance in trained cyclists. *J Sports Sci* 2002a; 20: 547–562.
- Romer LM, McConnell AK, Jones DA. Effects of inspiratory muscle training upon recovery time during high intensity, repetitive sprint activity. *Int J Sports Med* 2002b; 23: 353–360.
- Romer LM, McConnell AK, Jones DA. Inspiratory muscle fatigue in trained cyclists: effects of inspiratory muscle training. *Med Sci Sports Exerc* 2002c; 34: 785–792.
- Saunders PU, Telford RD, Pyne DB, Hahn AG, Gore CJ. Improved running economy and increased hemoglobin mass in elite runners after extended moderate altitude exposure. *J Sci Med Sport* 2009; 12: 67–72.
- Spengler CM, Roos M, Laube SM, Boutellier U. Decreased exercise blood lactate concentrations after respiratory endurance training in humans. *Eur J Appl Physiol Occup Physiol* 1999; 79: 299–305.
- St Croix CM, Morgan BJ, Wetter TJ, Dempsey JA. Fatiguing inspiratory muscle work causes reflex sympathetic activation in humans. *J Physiol* 2000; 529(Part 2): 493–504.
- Stuessi C, Spengler CM, Knopfli-Lenzin C, Markov G, Boutellier U. Respiratory muscle endurance training in humans increases cycling endurance without affecting blood gas concentrations. *Eur J Appl Physiol* 2001; 84: 582–586.
- Tzelepis GE, Kasas V, McCool FD. Inspiratory muscle adaptations following pressure or flow training in humans. *Eur J Appl Physiol Occup Physiol* 1999; 79: 467–471.
- Tzelepis GE, Vega DL, Cohen ME, Fulambarker AM, Patel KK, McCool FD. Pressure-flow specificity of inspiratory muscle training. *J Appl Physiol* 1994; 77: 795–801.
- Volianitis S, McConnell AK, Koutedakis Y, McNaughton L, Backx K, Jones DA. Inspiratory muscle training improves rowing performance. *Med Sci Sports Exerc* 2001; 33: 803–809.
- Wells GD, Pyley M, Thomas S, Goodman L, Duffin J. Effects of concurrent inspiratory and expiratory muscle training on respiratory and exercise performance in competitive swimmers. *Eur J Appl Physiol* 2005; 94: 527–540.
- Williams JS, Wongsathikun J, Boon SM, Acevedo EO. Inspiratory muscle training fails to improve endurance capacity in athletes. *Med Sci Sports Exerc* 2002; 34: 1194–1198.
- Witt JD, Guenette JA, Rupert JL, McKenzie DC, Sheel AW. Inspiratory muscle training attenuates the human respiratory muscle metaboreflex. *J Physiol* 2007; 584: 1019–1028.