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Effects of inspiratory muscle training on time–trial performance in trained cyclists

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We evaluated the effects of specific inspiratory muscle training on simulated time–trial performance in trained cyclists. Using a double–blind, placebo–controlled design, 16 male cyclists ($\dot{V}O_{2max} = 64 \pm 2$ ml·kg^{-1}·min^{-1}; mean ± $s_{\bar{x}}$) were assigned at random to either an experimental (pressure–threshold inspiratory muscle training) or sham–training control (placebo) group. Pulmonary function, maximum dynamic inspiratory muscle function and the physiological and perceptual responses to maximal incremental cycling were assessed. Simulated time–trial performance (20 and 40 km) was quantified as the time to complete pre–set amounts of work. Pulmonary function was unchanged after the intervention, but dynamic inspiratory muscle function improved in the inspiratory muscle training group ($P \leq 0.05$). After the intervention, the inspiratory muscle training group experienced a reduction in the perception of respiratory and peripheral effort (Borg CR10: 16 ± 4% and 18 ± 4% respectively; compared with placebo, $P \leq 0.01$) and completed the simulated 20 and 40 km time–trials faster than the placebo group [66 ± 30 and 115 ± 38 s (3.8 ± 1.7% and 4.6 ± 1.9%) faster respectively; $P = 0.025$ and 0.009]. These results support evidence that specific inspiratory muscle training attenuates the perceptual response to maximal incremental exercise. Furthermore, they provide evidence of performance enhancements in competitive cyclists after inspiratory muscle training.

Keywords: cycling, dyspnoea, placebo, respiratory muscle.

Introduction

Improvements in whole–body endurance capacity (Boutellier and Piwko, 1992; Boutellier *et al.*, 1992; Spengler *et al.*, 1999; Markov *et al.*, 2001; Stuessi *et al.*, 2001) and short–duration high–intensity time–trial performance (Volianitis *et al.*, 2001) have been reported in healthy individuals after specific respiratory muscle training. However, several studies have failed to demonstrate an ergogenic effect (Morgan *et al.*, 1987; Belman and Gaesser, 1988; Fairbarn *et al.*, 1991; Hanel and Secher, 1991; Kohl *et al.*, 1997; Inbar *et al.*, 2000; Sonetti *et al.*, 2001). The explanation for these divergent findings is unclear, but may be due to differences in the type of exercise test used to evaluate performance, the relative intensity and duration of exercise, differences in experimental design and the fitness of participants.

Perhaps the most important concern is that most previous studies of respiratory muscle training have evaluated outcome, almost without exception, using time–to–fatigue tasks at a fixed percentage of maximal oxygen uptake ($\dot{V}O_{2max}$) (Morgan *et al.*, 1987; Belman and Gaesser, 1988; Fairbarn *et al.*, 1991; Boutellier and Piwko, 1992; Boutellier *et al.*, 1992; Kohl *et al.*, 1997; Spengler *et al.*, 1999; Markov *et al.*, 2001; Stuessi *et al.*, 2001). Procedures that use volitional end–points have been criticized for not representing accurately competitive endurance performance (low external validity) and for being unreliable (Hopkins *et al.*, 1999). A large inter–individual variance in exercise measures coupled with a small sample size may explain in part why some studies have reported improvements in exercise capacity but failed to achieve statistical significance (Fairbarn *et al.*, 1991; Hanel and Secher, 1991; Sonetti *et al.*, 2001). Several previous studies have also failed to use

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carefully matched control groups in their experimental design (Boutellier and Piwko, 1992; Boutellier *et al.*, 1992; Spengler *et al.*, 1999). Where control groups were used, they were not true placebo groups (Morgan *et al.*, 1987; Fairbarn *et al.*, 1991; Kohl *et al.*, 1997; Markov *et al.*, 2001; Stuessi *et al.*, 2001) and the treatments were not presented to participants in a double-blind manner (Morgan *et al.*, 1987; Fairbarn *et al.*, 1991; Hanel and Secher, 1991; Kohl *et al.*, 1997; Inbar *et al.*, 2000; Markov *et al.*, 2001; Sonetti *et al.*, 2001; Stuessi *et al.*, 2001; Volianitis *et al.*, 2001). Studies without a double-blind, placebo-controlled design have been criticized for having weak internal validity and being vulnerable to the potential influence of participant and experimenter bias. Furthermore, most previous studies of respiratory muscle training can be criticized for not adequately monitoring participants' adherence to such training and, in particular, failing to exclude the potential influences of participants' routine training. Both factors could lead to uncertainty about the causal influences responsible for any changes that are observed after the intervention.

In view of the deficiencies and discrepancies described above, the aim of the present study was to determine the effects of specific inspiratory muscle training on endurance performance in trained cyclists using a double-blind, placebo-controlled research design in which all training was quantified objectively. We wished to evaluate the effects of inspiratory muscle training on two discrete intensities of exercise using relevant, valid (externally and internally) and reliable performance measures, a procedure that would enable us to use a reasonable sample size to identify meaningful treatment effects.

Methods

Participants

After receiving local ethics committee approval and written informed consent, 16 healthy trained male road cyclists (5 triathletes) volunteered to participate in the study (see Table 1). The participants competed regularly in local races and were considered 'trained' based on the unifying classification proposed by Jeukendrup *et al.* (2000). All participants were non-smokers (self-report), with no evidence of respiratory restriction or obstruction upon examination of maximum flow–volume loops.

General design

The participants were assigned at random to either an experimental (inspiratory muscle training) or sham-training control (placebo) group using a double-blind, placebo-controlled design. Participation required seven visits to the laboratory. Visit 1 was used to familiarize the participants with the test procedures. Visit 2 required the assessment of inspiratory muscle and pulmonary function and the physiological response to maximal incremental cycling. Visits 3 and 4 required participants to complete simulated 20 and 40 km time-trials, the order of which was counterbalanced. Pre-intervention trials (visits 2–4) were separated by at least 48 h, completed within 2 weeks and repeated in the same order (visits 5–7) after 6 weeks of inspiratory muscle training. Thus, the overall duration of the study was ~10 weeks.

Procedure

Pre-test preparation. Testing took place during a maintenance phase of normal training so that confounding influences were minimized. The participants performed only a light recovery training session (less than 50 km at less than 75% maximal heart rate) 24 h before testing and maintained their regular diet in the days preceding physiological assessment. On a test day, the participants were instructed not to eat in the 2 h before testing, to avoid drinking alcohol or caffeinated beverages and the taking of any other substances that are known to affect, or may be suspected to affect, human physiological functions. The participants kept detailed records of all exercise and food intakes in the 48 h before the first test and used these records to replicate their activities before subsequent tests. The tests were conducted at the same temperature (18–22°C) and relative humidity (< 70%). For each participant, testing was scheduled at a similar time of day (±1 h) so that diurnal fluctuation effects were minimized (Atkinson and Reilly, 1996).

Pulmonary function. Pulmonary function (forced flow–volume loops) was assessed using an on-line turbine spirometer (Mijnhardt Oxycon Alpha, Bunnik, Netherlands). Measurements were made according to European Respiratory Society recommendations (Quanjer *et al.*, 1993), although the participants assumed their normal riding position on a cycle ergometer throughout testing. The following variables were derived from the flow–volume profiles: forced vital capacity, forced expiratory volume in 1 s and peak expiratory flow rate. A measurement of 15 s maximum voluntary ventilation was also obtained.

Maximum dynamic inspiratory muscle function. The pressure–flow relationship for inspiratory muscles working in synergy was assessed using maximal inspiratory efforts performed against a pressure-threshold

Table 1. Descriptive characteristics of the participants for the pre-intervention trials (mean ± $s_{\bar{x}}$)

	Inspiratory muscle training (n = 8)	Placebo (n = 8)
Anthropometry		
Age (years)	29.5 ± 3.3	30.3 ± 2.6
Training history (years)	8.9 ± 2.0	9.3 ± 1.9
Height (m)	1.78 ± 0.02	1.80 ± 0.02
Body mass (kg)	70.1 ± 2.3	74.5 ± 2.3
Sum of 4 skinfolds (mm)	24.8 ± 1.6	31.0 ± 3.4
Estimated body fat (%)	12.3 ± 1.1	15.0 ± 1.5
Pulmonary function		
FVC (l)	5.53 ± 0.26 (108 ± 3)	6.13 ± 0.3 (117 ± 5)
FEV_1 (l)	4.71 ± 0.29 (109 ± 5)	5.17 ± 0.32 (118 ± 7)
FEV_1/FVC (%)	84.8 ± 1.8 (101 ± 2)	84.0 ± 2.1 (101 ± 2)
PEF ($l \cdot s^{-1}$)	10.1 ± 0.8 (110 ± 9)	10.6 ± 0.5 (107 ± 6)
MVV ($l \cdot min^{-1}$)	195.4 ± 16.3 (142 ± 12)	197.1 ± 11.3 (130 ± 7)
Maximal incremental exercise		
$\dot{W}_{max}$ (W)	355 ± 10	380 ± 17
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	4.58 ± 0.17	4.70 ± 0.08

Abbreviations: FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; PEF, peak expiratory flow; MVV, 15 s maximal voluntary ventilation; $\dot{W}_{max}$, maximal external power; $\dot{V}O_{2max}$, maximal oxygen uptake.
Note: Values in parentheses represent the percent of predicted value based on age, height and sex (Quanjer *et al.*, 1993).

valve arrangement (Caine and McConnell, 2000). Inspiratory mouth pressure was measured with a pressure transducer (Mercury M14, Glasgow, UK) connected by polyethylene tubing to a vent (4 mm internal diameter) located near the mouthpiece of the breathing circuit. Inspiratory airflow was measured with an ultrasonic phase-shift flow meter (Birmingham Flowmetrics Ltd, Birmingham, UK) located distal to the pressure-threshold valve. The pressure and flow signals were amplified, passed through a 12-bit analog-to-digital converter at a sampling rate of 200 Hz, recorded on a computer and processed using bespoke software (Labview 3, National Instruments, Austin, TX, USA).

Mean pressure at zero flow (P_0) was measured with complete closure of the threshold valve. A 1 mm orifice was exposed to prevent the cyclist from producing artificially high inspiratory pressures with the muscles of the buccal cavity (Black and Hyatt, 1969). To ensure that inspiratory efforts were performed at the same lung volume (residual volume), changes in vital capacity were measured with a pneumotachograph spirometer (Vitalograph 2120, Buckingham, UK) connected in series to the expiratory port of the pressure-threshold device. After the determination of P_0, individuals performed maximal inspiratory manoeuvres against six discrete load settings (~0, 20, 25, 35, 50 and 65% P_0), which were assigned at random using a balanced Latin square for an even number of treatment conditions. The order of treatments was retained throughout the remaining trials. Three technically correct trials were performed at each of the loading intensities and 30 s was permitted between efforts. Maximal pressure and unloaded flow were re-evaluated at the end of each measurement session. No changes from initial values were observed, suggesting the absence of pressure and flow fatigue. All manoeuvres were performed while seated and were completed within ~15 min. The participants received visual feedback of pressure and flow to maximize respiratory efforts, and were consistently instructed to contract maximally and as rapidly as possible.

Pressure and flow measures were obtained from the single inspiratory effort at each level of %P_0 that gave the largest product of mean inspiratory pressure and flow (i.e. power). Pressure–flow data for the different %P_0 trials were fitted by curves drawn according to a linear

least squares representation [$P = a\dot{V} + b$], where P is pressure (cmH_2O), $\dot{V}$ is flow ($l \cdot s^{-1}$) and a and b are constants. Maximal flow ($\dot{V}_{max}$) was derived for each participant from the experimental data by extrapolation. Inspiratory muscle power ($\dot{W}_I$) was calculated from the product of mean inspiratory pressure and mean inspiratory flow rate. Maximal power of the inspiratory muscles ($\dot{W}_{Imax}$; $cmH_2O \cdot l^{-1} \cdot s^{-1}$) was calculated by differentiation from a zero grade tangent to the flow–power data. Optimal flow ($\dot{V}_{opt}$; $l \cdot s^{-1}$ and $\%\dot{V}_{max}$) and pressure (P_{opt}; cmH_2O and $\%P_0$) were defined as the flow and pressure values corresponding to maximal power of the inspiratory muscles on the power–flow curve, respectively (see Fig. 1). The maximal rate of pressure development, which occurs during the initial incline of the maximal inspiratory pressure curve, was assessed and defined as the positive peak of the pressure derivative as a function of time.

Maximal incremental exercise. The participants performed an incremental exercise test to volitional exhaustion on a calibrated electromagnetically braked cycle ergometer (Lode Excalibur, Groningen, Netherlands) set in the 'hyperbolic' mode (i.e. independent of pedal rate). The cycle ergometer was modified with racing handlebars and seat and the cyclist's own pedals for cleated shoes. The ergometer was configured to match the dimensions of each athlete's racing bicycle. Power was increased by 35 W every 3 min starting from 95 W. The participants selected their preferred pedalling frequency during the incremental exercise test and this cadence was kept constant in subsequent tests by inspection of a digital cadence output. The test was terminated when pedal cadence fell below 60 $rev \cdot min^{-1}$ and maximal power output ($\dot{W}_{max}$) was calculated by interpolation from the total time completed during the final stage of the test.

Ventilatory and pulmonary gas exchange indices were measured breath-by-breath at rest and throughout incremental exercise using an on-line system (Mijnhardt Oxycon Alpha, Bunnik, Netherlands). A maximal inspiratory capacity measurement was obtained during the last 30 s of each 3 min workload from the best of at least two measurements. A mean of 10–20 tidal breaths taken at the end of each workload were averaged using a computer program to provide a representative tidal flow–volume loop for that workload. The end-expiratory lung volume was determined by subtracting the maximal inspiratory capacity for each workload from the resting forced vital capacity (FVC) (Hyatt, 1961) and expressed relative to FVC. End-inspiratory lung volume was calculated as the sum of end-expiratory lung volume and tidal volume (Hyatt, 1961) and also expressed relative to FVC.

Arterialized venous blood samples were collected during the final 30 s of each incremental exercise stage from a superficial vein on the dorsal surface of a heated hand for subsequent determination of plasma lactate concentration using an NAD^+/NADH linked enzymatic method (Sigma Diagnostics, Poole, UK). A short-range telemetry system (Polar Accurex Plus, Polar Electro, Kempele, Finland) was used to monitor heart rate continuously throughout each test. The arteriovenous oxygen content difference, cardiac output and stroke volume were estimated non-invasively from the measured $\dot{V}O_2$ and heart rate during exercise (Stringer *et al.*, 1997). Ratings of respiratory and peripheral exertion were recorded during the final 30 s of each submaximal exercise stage using Borg's modified CR10 scale (Borg, 1998).

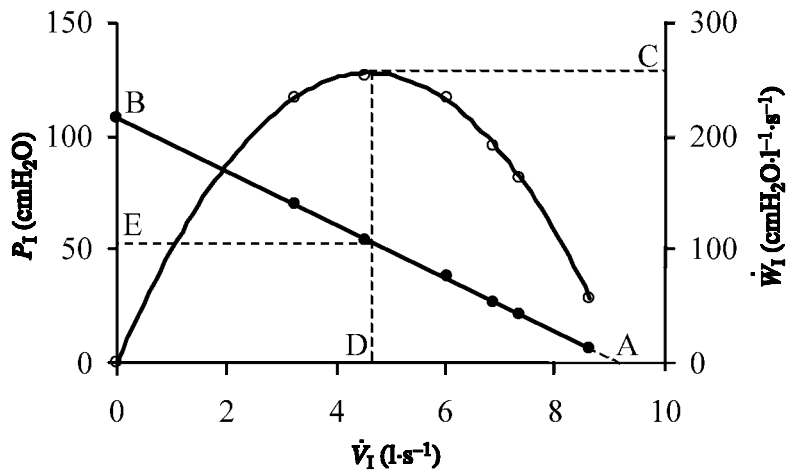

Fig. 1. Schematic representation of inspiratory pressure–flow–power calculations. $\dot{V}_I$, inspiratory flow rate; P_I, inspiratory mouth pressure; $\dot{W}_I$, inspiratory muscle power. *Note*: A = maximal flow ($\dot{V}_{max}$); B = maximal pressure at zero flow (P_0); C = maximal power (W_{Imax}); D = optimal flow ($\dot{V}_{opt}$); E = optimal pressure (P_{opt}).

Time-trial performance. Exercise performance was evaluated as the time taken to complete simulated 20 and 40 km time-trials, the order of which was counter-balanced. Full details of the procedure have been reported previously (Jeukendrup *et al.*, 1996). Briefly, the cycle ergometer was set in the 'linear' mode (i.e. pedal rate dependent) according to the formula: $\dot{W} = L \cdot (\text{rev} \cdot \text{min}^{-1})^2$. The value for L was chosen such that it would elicit a pedalling rate of 95 $\text{rev} \cdot \text{min}^{-1}$ at 82 and 75% maximal power ($\dot{W}_{max}$) for the 20 and 40 km time-trials, respectively (i.e. L was dependent on a participant's maximal power output). Thus, 82 and 75% $\dot{W}_{max}$ could be achieved at 95 $\text{rev} \cdot \text{min}^{-1}$, which was the pedalling rate most of the participants chose voluntarily during the maximal incremental exercise test. After a warm-up (5 min, 40% $\dot{W}_{max}$), the participants performed a pre-determined amount of work (equal to ~30 or ~60 min cycling) in the fastest time possible. The measure of performance was the time to complete the target amount of work. This target was based on the maximal power achieved during incremental exercise ($\dot{W}_{max}$), and was calculated according to the formula: target work (J) = 0.82 (or 0.75) $\cdot \dot{W}_{max} \cdot$ 1800 (or 3600) for the 20 km (or 40 km) time-trials. Although the participants were informed visually about the percentage of the total pre-set work that had been performed during the tests, they received no information on exercise intensity, pedalling rate, time and heart rate. Furthermore, the participants received no verbal encouragement during the time-trial tests (although the principal investigator was present at all times). The only variable measured throughout the trials was heart rate. Water was available *ad libitum*. The post-inspiratory muscle training target work was identical to pre-training values. Simulated time-trial performance is reliable (Jeukendrup *et al.*, 1996; Bishop, 1997), correlates with actual time-trial performance in the field (Coyle *et al.*, 1991) and is sensitive to changes in endurance performance (Lindsay *et al.*, 1996).

Inspiratory muscle training. The participants were ranked according to maximal inspiratory muscle power ($\dot{W}_{Imax}$) and divided subsequently into matched pairs. One individual of each pair was assigned at random to the inspiratory muscle training group by an independent observer and the other to the placebo group. The principal investigators were therefore blinded to the training condition. The inspiratory muscle training group performed 30 dynamic inspiratory efforts twice daily for 6 weeks (84 sessions) against a pressure-threshold load equivalent to $\dot{W}_{Imax}$ (~50% P_0), a protocol known to be effective in eliciting an adaptive response (Caine and McConnell, 1998). The placebo group trained using 60 slow protracted breaths once daily for 6 weeks at 25% $\dot{W}_{Imax}$ (~15% P_0), a protocol known to elicit negligible changes in inspiratory muscle function (Caine and McConnell, 1998). Loading characteristics of the inspiratory muscle training device (POWERbreathe®, IMT Technologies Ltd, UK) have been documented (Caine and McConnell, 2000). After the initial setting of training loads, the participants in the inspiratory muscle training group were instructed by an independent observer to increase periodically the load to a value that would permit them to only just complete 30 manoeuvres; the placebo group was not given these instructions. The participants were told they were participating in a study to compare the influence of strength (inspiratory muscle training group) versus endurance (placebo group) protocols and, as a consequence, were blinded to the true purpose of the study and the expected outcomes. They were instructed to cease training 48 h before the post-intervention trials.

Adherence to training. The number of inspiratory efforts completed by participants throughout the intervention was monitored using a thermistor suspended within the main body of the training device that sensed acute drops in air temperature associated with changes in airflow. The cumulative number of temperature changes over the course of an intervention was stored on a microchip attached outside the main body of the training device. Data were retrieved by reading the pin voltages on a 12-bit binary counter, which corresponded to the total number of breaths at the prescribed load.

Detailed physical activity diaries were used to monitor training volume and intensity. Training impulse, which is a measure of the quantity of training in any given session, was calculated week-by-week as the product of training volume (duration of training in minutes) and intensity (delta heart rate ratio = [exercise heart rate – resting heart rate]/[maximum heart rate – resting heart rate]) (Banister and Calvert, 1980). As a guard against allotting disproportionate importance to long-duration activity at low heart rates (compared with intense but short-duration activity), the delta heart rate ratio was weighted in a manner that reflected the intensity of the effort (Banister and Calvert, 1980). A factor based upon the increase in blood lactate in trained individuals was used to weight the delta heart rate ratio proportionately higher the higher its elevation during the exercise period. The multiplying factor was generated from the equation for males: $y = 0.64e^{1.92x}$, where y = multiplying factor, x = delta heart rate ratio during exercise and e is the natural logarithm having a value of 2.712 (Green *et al.*, 1983). The delta heart rate ratio was also weighted according to exercise modality. Cycling and swimming, with factors of 1.0, were considered the baseline modalities to which other sports

were related. Running was assigned a factor of 0.9, since individuals would have to work ~10% harder in cycling than in running to reach the same heart rate.

Data analyses

All data collected during the maximal incremental exercise test were aligned to fixed percentages of maximal power output by assuming linear relationships between consecutive data points and interpolating between these points. Mixed factorial analyses of variance were used to test for between-group effects due to 'treatment' (inspiratory muscle training or placebo) and within-group effects due to 'intervention' (pre- and post-treatment), 'distance' (20 and 40 km) and 'relative workload' (50, 60, 70, 80, 90, 95 and 100% $\dot{W}_{max}$) or 'time' (25, 50, 75 and 100% total test time) on each of the dependent variables. Mauchly's sphericity test was used to check homogeneity of covariance. Violations of the assumption of sphericity were corrected using the Greenhouse–Geisser adjustment. The residuals from the analyses of variance were checked for normality. Planned pairwise comparisons were made with repeated-measures *t*-tests and the Bonferroni adjustment was used to modify the *per family* Type I error rate per comparison. Pearson product–moment correlation coefficients were computed to assess the relationship between the relative changes in selected physiological variables after inspiratory muscle training. Stepwise multiple regression analysis was used to determine which variables accounted for the largest amount of variation in the time-trial performance expressed relative to pre-intervention values. The results are expressed as the mean ± standard error of the mean ($s_{\bar{x}}$) unless stated otherwise. Significance was set at $P < 0.05$. All statistical analyses were performed using the 8.0 release version of SPSS for Windows (SPSS Inc., Chicago, IL, USA).

Results

Routine physical exercise and compliance with inspiratory muscle training

The participants' physical activity did not vary between (inspiratory muscle training *vs* placebo) or within groups (1 to 6 weeks) as evidenced by non-significant differences in frequency, duration, intensity and training impulse scores (see Table 2). Furthermore, the total training impulse score was not correlated with changes in $\dot{W}_{Imax}$ or 20 and 40 km time-trial performance ($r = 0.13$, 0.18 and 0.20, respectively). Excellent compliance with the prescribed inspiratory muscle training regimen was found for both groups. The inspiratory muscle training group completed 81 ± 2 of the 84 training sessions (96% adherence), whereas the placebo group completed 40 ± 2 of the 42 sessions (95% adherence).

Pulmonary and respiratory muscle function

All baseline pulmonary function values were within normal limits (Table 1). For the placebo group, none of the pulmonary or respiratory muscle function measures were different after the 6 weeks of sham training (Table 3). In contrast, significant improvements in P_0 (28 ± 7%), $\dot{V}_{max}$ (17 ± 4%), $\dot{W}_{Imax}$ (49 ± 16%), P_{opt} (25 ± 15%), $\dot{V}_{opt}$ (17 ± 8%) and maximal rate of pressure development (18 ± 8%) were observed in the inspiratory muscle training group ($P \leq 0.01$), as indicated by the significant interaction effect. No other measures changed significantly after the inspiratory muscle training.

Physiological and perceptual response to maximal incremental exercise

Changes in performance. Pre-intervention measures for $\dot{W}_{max}$ did not differ between the inspiratory muscle training and placebo groups (5.07 ± 0.14 and 5.10 ± 0.23 $W \cdot kg^{-1}$, respectively). Similarly, $\dot{W}_{max}$ remained unchanged after the intervention for both the inspiratory muscle training and placebo groups (5.11 ± 0.12 and 5.04 ± 0.27 $W \cdot kg^{-1}$, respectively).

Changes in metabolism. There was a tendency for blood lactate concentration to be reduced during the middle and later stages of incremental exercise in the inspiratory muscle training group after the intervention (−8 ± 2% from pre-intervention values), although this failed to reach statistical significance (see Fig. 2). Maximal oxygen uptake ($\dot{V}O_{2max}$) remained unchanged in both groups. However, the average $\dot{V}O_2$ across all workloads was reduced slightly in the inspiratory muscle training group after the intervention (−3 ± 1% from pre-training values), but this was not statistically significant (see Fig. 2). In line with the change in $\dot{V}O_2$, estimated stroke volume (and, therefore, cardiac output) was slightly reduced during all exercise stages in the inspiratory muscle training group after the intervention (− 4 ± 1% on average), but this was not statistically significant (see Fig. 3).

Changes in ventilation and breathing pattern. When working at power outputs of 90 and 100% $\dot{W}_{max}$, a significant increase in tidal volume was observed in the inspiratory muscle training group after the intervention (pre- *vs* post-: 3.14 and 2.93 l *vs* 3.30 and 3.10 l;

Table 2. Frequency, duration, intensity and quantity of physical activity for the inspiratory muscle training and placebo groups (mean ± $s_{\bar{x}}$)

	Inspiratory muscle training ($n = 8$)				Placebo ($n = 8$)			
	Frequency (days)	Duration (min)	Intensity (heart rate ratio)	Quantity (TRIMP)	Frequency (days)	Duration (min)	Intensity (heart rate ratio)	Quantity (TRIMP)
Week 1	3.4 ± 0.5	316 ± 55	0.74 ± 0.03	634 ± 143	3.8 ± 0.7	292 ± 59	0.76 ± 0.02	597 ± 123
Week 2	3.6 ± 0.6	324 ± 91	0.73 ± 0.02	633 ± 196	3.6 ± 0.3	310 ± 56	0.74 ± 0.01	618 ± 112
Week 3	4.0 ± 0.6	329 ± 73	0.75 ± 0.01	651 ± 160	3.6 ± 0.6	358 ± 72	0.73 ± 0.01	659 ± 124
Week 4	4.0 ± 0.6	321 ± 57	0.76 ± 0.01	646 ± 112	3.8 ± 0.6	360 ± 79	0.73 ± 0.02	649 ± 139
Week 5	4.1 ± 0.7	299 ± 58	0.80 ± 0.03	692 ± 150	3.9 ± 0.5	374 ± 53	0.73 ± 0.02	704 ± 116
Week 6	4.0 ± 0.8	360 ± 84	0.73 ± 0.02	696 ± 168	3.9 ± 0.5	366 ± 71	0.73 ± 0.02	684 ± 113
Mean 1–6	3.8 ± 0.1	325 ± 20	0.75 ± 0.02	659 ± 28	3.7 ± 0.1	343 ± 34	0.74 ± 0.01	652 ± 40

Abbreviation: TRIMP, training impulse (dimensionless unit) corrected for exercise intensity and modality.
Note: No between- or within-group differences ($P > 0.05$).

Table 3. Inspiratory muscle function for the inspiratory muscle training and placebo groups pre- and post-intervention (mean ± $s_{\bar{x}}$)

	Inspiratory muscle training ($n = 8$)		Placebo ($n = 8$)	
	Pre-	Post-	Pre-	Post-
P_0 (cmH_2O)	102 ± 6	126 ± 5**	100 ± 6	99 ± 6
$\dot{V}_{max}$ ($l \cdot s^{-1}$)	9.15 ± 0.32	10.71 ± 0.30**	8.99 ± 0.32	9.20 ± 0.30
$\dot{W}_{Imax}$ ($cmH_2O \cdot l^{-1} \cdot s^{-1}$)	237 ± 23	329 ± 22**	230 ± 18	233 ± 15
P_{opt} (cmH_2O)	52.9 ± 3.6	66.0 ± 2.5**	50.4 ± 2.9	50.7 ± 2.6
P_{opt} (%P_0)	52.2 ± 0.9	52.5 ± 0.3	51.1 ± 0.7	51.9 ± 0.8
$\dot{V}_{opt}$ ($l \cdot s^{-1}$)	4.35 ± 0.14	5.08 ± 0.15**	4.41 ± 0.18	4.43 ± 0.17
$\dot{V}_{opt}$ (%$\dot{V}_{max}$)	47.8 ± 0.9	47.5 ± 0.3	49.0 ± 0.7	48.1 ± 0.8
MRPD ($cmH_2O \cdot ms^{-1}$)	0.537 ± 0.032	0.643 ± 0.021**	0.525 ± 0.029	0.500 ± 0.030

Abbreviations: P_0, maximal inspiratory pressure at zero flow; $\dot{V}_{max}$, maximal inspiratory flow; $\dot{W}_{Imax}$, maximal inspiratory muscle power; P_{opt}, optimal pressure; $\dot{V}_{opt}$, optimal flow; MRPD, maximal rate of pressure development.
** Significant interaction effect ($P \leq 0.01$).

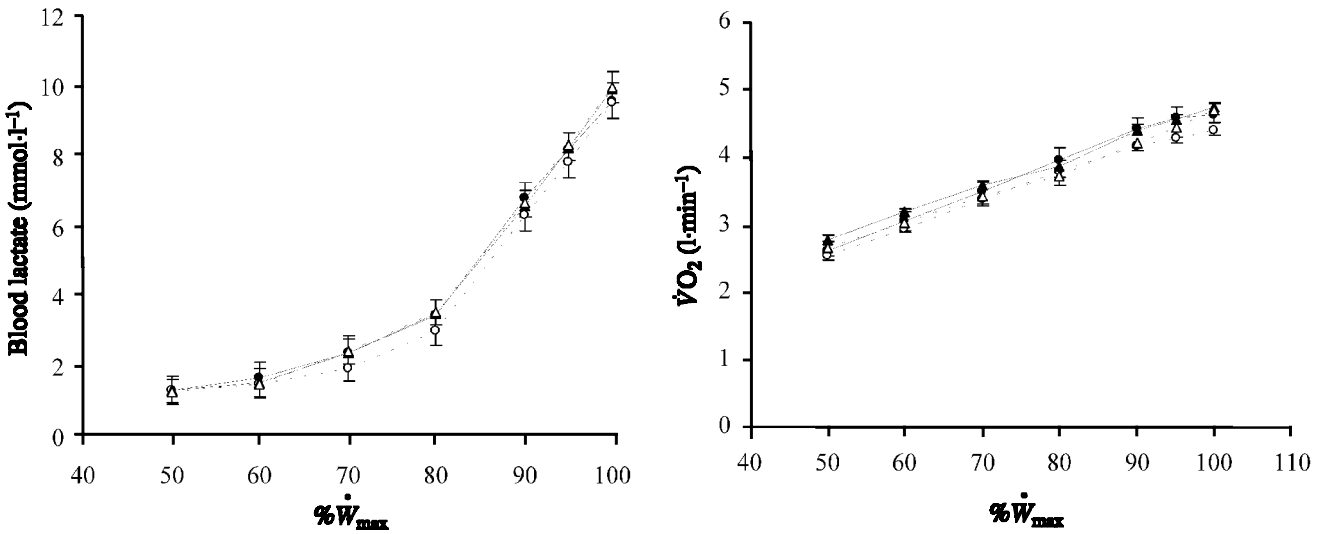

Fig. 2. Metabolic response to maximal incremental exercise for the inspiratory muscle training and placebo groups pre- and post-intervention (mean ± $s_{\bar{x}}$). ●, before inspiratory muscle training; ○, after inspiratory muscle training; ▲, before placebo; Δ, after placebo.

$P \leq 0.05$) (see Fig. 4). This was primarily the result of a non-significant increase in end-inspiratory lung volume (see Fig. 5). As a consequence of the change in tidal volume, minute ventilation at 100% $\dot{W}_{max}$ was increased from pre-intervention values in the inspiratory muscle training group (171.3 ± 11.3 *vs* 176.6 ± 9.7 $l \cdot min^{-1}$, $P \leq 0.05$), although a significant interaction effect was not observed. No other ventilatory parameters changed during incremental exercise after inspiratory muscle training. The placebo group did not exhibit any post-intervention changes, although there was a tendency towards a more tachypnoeic pattern that was characterized by a lower tidal volume at 100% than at 90% $\dot{W}_{max}$ (3.17 *vs* 3.49 l).

Perceptual changes. Substantial effects were observed for the inspiratory muscle training group during maximal incremental exercise for the perception of both respiratory and peripheral effort when averaged across all workloads (16 ± 4% and 18 ± 4%, respectively; $P \leq 0.01$). However, peripheral effort at the end of exercise was unchanged after inspiratory muscle training. The change in amplitude for these perceptions was similar for both respiratory and peripheral ratings

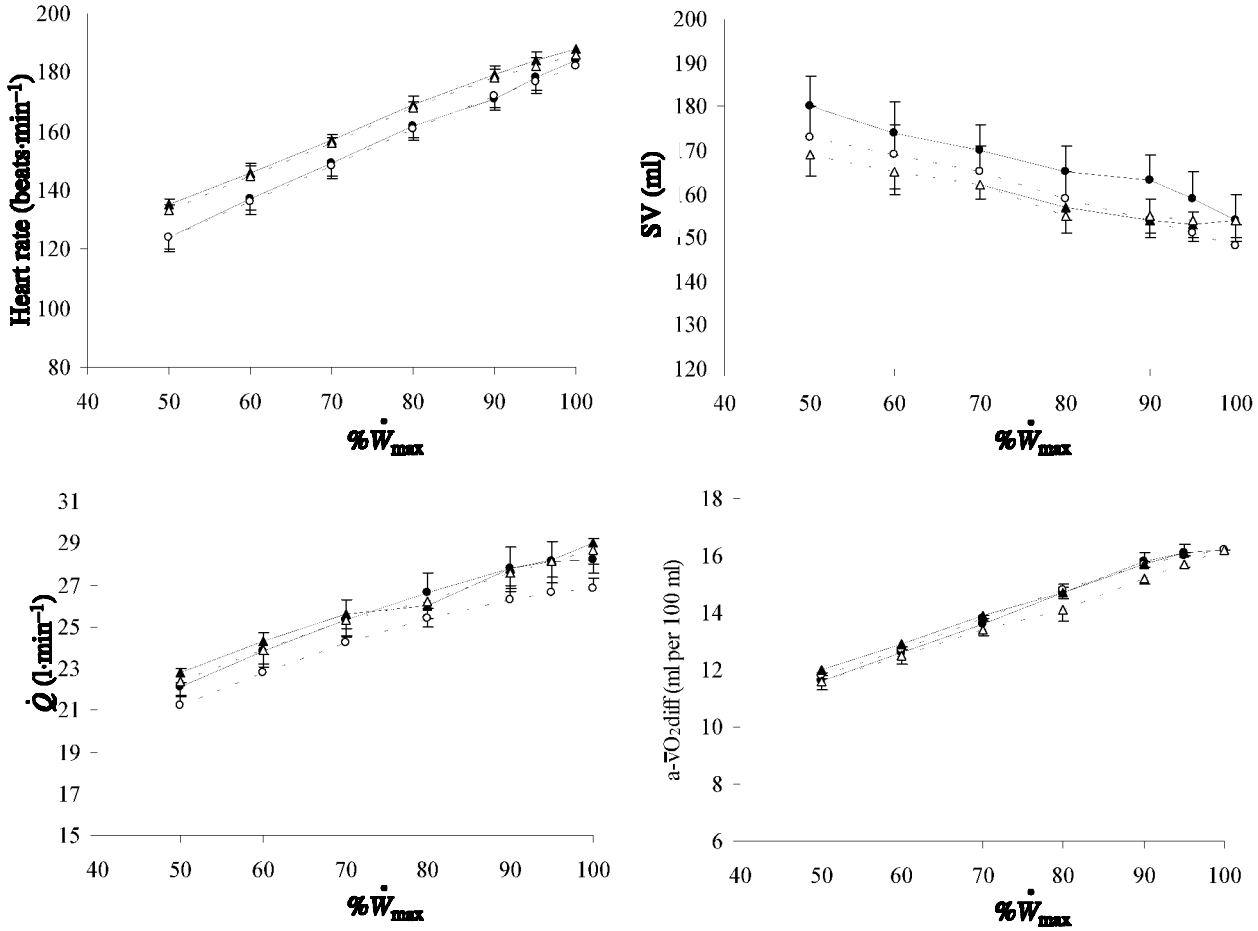

Fig. 3. Cardiovascular response to maximal incremental exercise for the inspiratory muscle training and placebo groups pre- and post-intervention (mean ± $s_{\bar{x}}$). *Note*: SV = stroke volume; $\dot{Q}$ = cardiac output; a-$\bar{v}O_2$diff = arterial mixed-venous oxygen difference. ●, before inspiratory muscle training; ○, after inspiratory muscle training; ▲, before placebo; Δ, after placebo.

(see Fig. 6). No changes were observed for the placebo group.

Time-trial performance

Group mean results for the 20 and 40 km simulated time-trial performances are presented in Table 4. Figure 7 shows the relative changes in 20 and 40 km time-trial performance for the inspiratory muscle training and placebo groups. After the intervention, the inspiratory muscle training group completed the simulated 20 and 40 km time-trials faster than the placebo group (65 ± 30 and 114 ± 38 s faster, respectively; $P = 0.025$ and 0.009). This represents an improvement of 3.8 ± 1.7% and 4.6 ± 1.9% for 20 and 40 km time-trial performance, respectively. The 95% confidence intervals for the relative changes in 20 and 40 km time-trial performance were −0.7 to −6.9% and −0.8 to −8.4%, respectively. Between-group differences were most pronounced during the later stages of the time-trial rides (see Fig. 7).

Individual data for changes in 20 and 40 km time-trial performance for the two groups are shown in Fig. 8. The 20 km time-trial was completed more quickly by 7 of 8 inspiratory muscle training participants post-intervention compared with 2 of 8 placebo participants. Completion time for the 40 km time-trial was quicker in 7 of 8 inspiratory muscle training participants post-intervention compared with 3 of 8 placebo participants. The regression lines of pre- versus post-intervention 20 and 40 km time-trial performances in the inspiratory muscle training group were parallel to the lines of identity, indicating that the effects of inspiratory muscle training upon absolute time-trial performance were similar across all baseline times.

Correlations among variables with inspiratory muscle training

Inter-individual differences with inspiratory muscle training for several variables were significantly

interrelated (Table 5). The relative change in the maximal rate of pressure development after inspiratory muscle training accounted for ~31% of the variance in performance time expressed relative to pre-intervention values.

Discussion

Main findings

The aim of the present study was to determine the influence of a 6 week specific inspiratory muscle training programme on simulated 20 and 40 km time-trial performance in trained male cyclists using a double-blind, placebo-controlled experimental design. The main finding was that inspiratory muscle training attenuated the perceptual response to maximal incremental exercise and improved performance in both the simulated 20 and 40 km time-trials.

Changes in inspiratory muscle function

Inspiratory muscle function was improved in the inspiratory muscle training group but not the placebo group. The significant 28% increase in maximal pressure at zero flow (P_0) is consistent with the 25–45% improvements reported previously in healthy individuals using pressure-threshold inspiratory muscle training (Inbar *et al.*, 2000; Volianitis *et al.*, 2001), but somewhat larger than the 8% improvement reported by others (Sonetti *et al.*, 2001). The small but significant improvement in inspiratory muscle strength noted by Sonetti and colleagues might have been due in part to strength being constrained by an initial high baseline

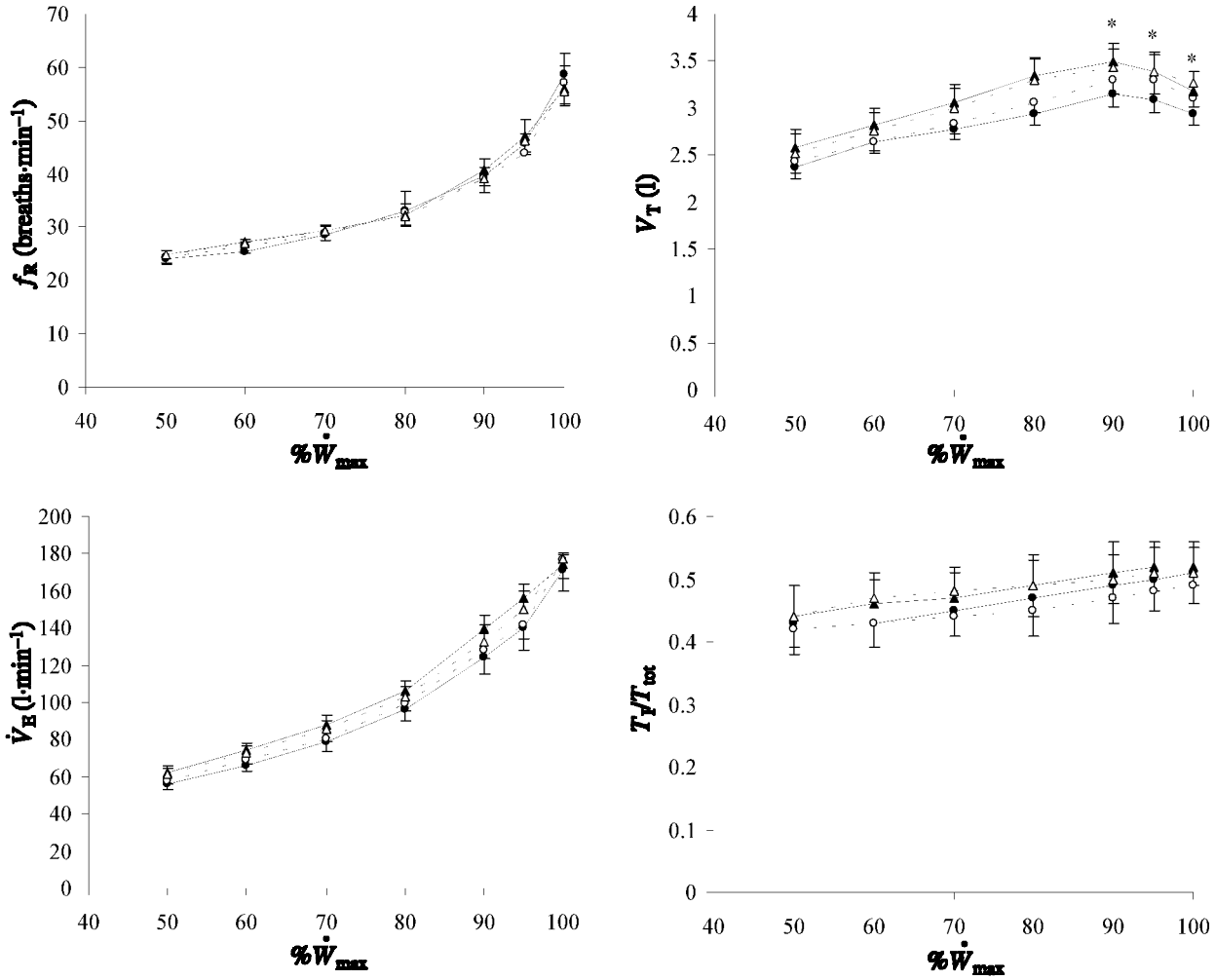

Fig. 4. Ventilatory response to maximal incremental exercise for the inspiratory muscle training and placebo groups pre- and post-intervention (mean ± $s_{\bar{x}}$). *Note*: f_R = respiratory frequency; V_T = tidal volume; $\dot{V}_E$ = minute ventilation; T_I/T_{tot} = inspiratory time/total breath time. ●, before inspiratory muscle training; ○, after inspiratory muscle training; ▲, before placebo; Δ, after placebo. * Significant interaction effect ($P \leq 0.05$).

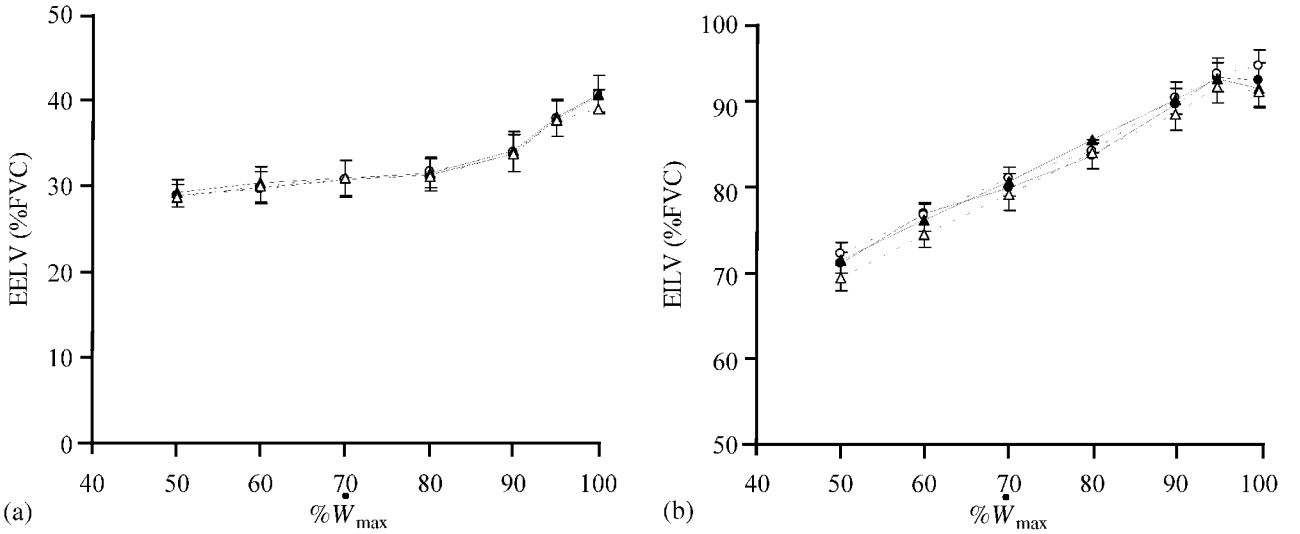

Fig. 5. End-expiratory lung volume (EELV) (a) and end-inspiratory lung volume (EILV) (b) responses to maximal incremental exercise for the inspiratory muscle training and placebo groups pre- and post-intervention (mean ± $s_{\bar{x}}$). ●, before inspiratory muscle training; ○, after inspiratory muscle training; ▲, before placebo; Δ, after placebo.

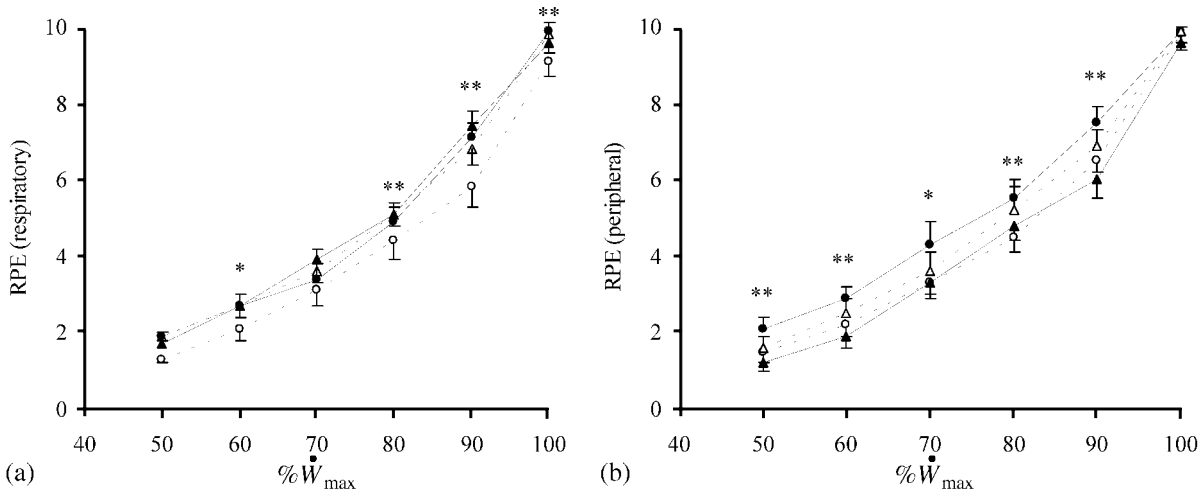

Fig. 6. Ratings of perceived exertion (RPE) for respiratory (a) and peripheral (b) effort during maximal incremental exercise for the inspiratory muscle training and placebo groups pre- and post-intervention (mean ± $s_{\bar{x}}$). ●, before inspiratory muscle training; ○, after inspiratory muscle training; ▲, before placebo; Δ, after placebo. *Significant interaction effect ($P \leq 0.05$). **Significant interaction effect ($P \leq 0.01$).

value in the respiratory muscle training group (Sonetti *et al.*, 2001). An alternative explanation is that the concurrent strength (pressure-threshold loading) and endurance (voluntary isocapnic hyperpnoea) respiratory muscle training performed by individuals might have inhibited strength development (Leveritt *et al.*, 1999). Maximal inspiratory flow rate for the inspiratory muscle training group increased by 17%. The training manoeuvre undertaken by the inspiratory muscle training group was characterized by a combination of moderate inspiratory pressure and flow rate. Thus, the finding of a significant increase in both maximal pressure at zero flow and maximal inspiratory flow rate is in accordance with the previously documented changes with inspiratory muscle training (Tzelepis *et al.*, 1994) and confirms the results from previous investigations using pressure-threshold inspiratory muscle training (Caine and McConnell, 1998). Maximal power of the inspiratory muscles was increased in line with the observed changes in maximal pressure at zero flow and maximal inspiratory flow rate. The increase in maximal rate of pressure development for the inspiratory muscle training group is in accordance with a recent study that documented an increase in the rate of pressure

Table 4. Results of 20 and 40 km time-trial performances for the inspiratory muscle training and placebo groups pre- and post-intervention (mean ± $s_{\bar{x}}$)

	Inspiratory muscle training (n = 8)		Placebo (n = 8)	
	Pre-	Post-	Pre-	Post-
20 km time-trial				
Time (s)	1777 ± 28	1716 ± 38*	1813 ± 15	1817 ± 14
Power (W)	294 ± 8	305 ± 9*	308 ± 13	307 ± 12
	(83 ± 1)	(85 ± 2)	(82 ± 1)	(81 ± 1)
Heart rate (beats · min^{-1})	170 ± 4	170 ± 4	173 ± 2	173 ± 2
	(93 ± 1)	(94 ± 1)	(92 ± 1)	(93 ± 1)
40 km time-trial				
Time(s)	3540 ± 92	3419 ± 97**	3602 ± 61	3595 ± 60
Power (W)	271 ± 8	280 ± 9**	284 ± 13	284 ± 12
	(77 ± 2)	(78 ± 2)	(75 ± 1)	(76 ± 1)
Heart rate (beats · min^{-1})	166 ± 4	166 ± 4	171 ± 4	170 ± 3
	(90 ± 2)	(91 ± 2)	(91 ± 2)	(91 ± 1)

Note: Values in parentheses represent percentage of maximum.
* Significant interaction effect ($P \leq 0.05$). ** Significant interaction effect ($P \leq 0.01$).

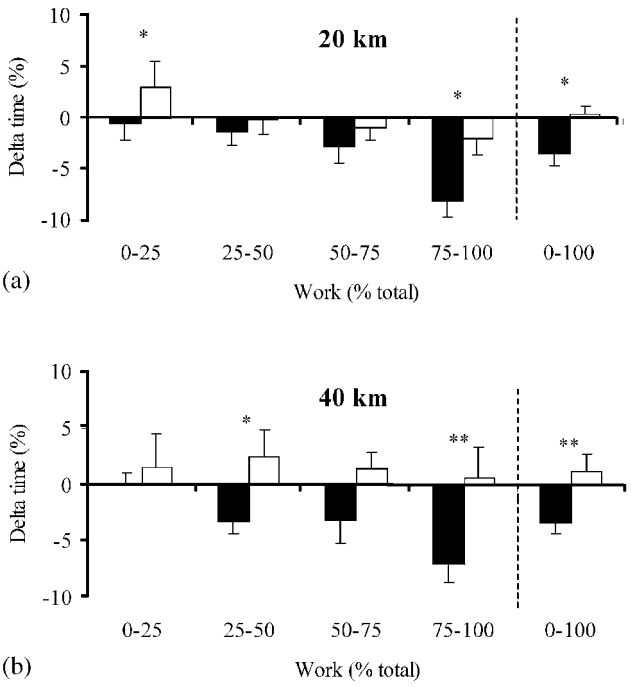

Fig. 7. Relative changes in 20 km (a) and 40 km (b) time-trial performance for the inspiratory muscle training (closed bars) and placebo (open bars) groups (mean ± $s_{\bar{x}}$). *Significant interaction effect ($P \leq 0.05$). **Significant interaction effect ($P \leq 0.01$).

production after both pressure- and flow-based inspiratory muscle training (Tzelepis *et al.*, 1999). The relative change in maximal rate of pressure development accounted for ~32% of the variance in time-trial performance expressed relative to pre-intervention values. The functional benefits of an improvement in maximal rate of pressure development are unclear. We speculate that a faster rate of pressure development might result in a faster inspiratory time and allow expiratory time to be prolonged. This could help to maintain end-expiratory lung volume, thereby allowing the diaphragm to contract nearer its optimal length and reducing the elastic recoil forces to be overcome. However, although our results suggest that inspiratory time/total breath time was reduced slightly post-intervention, there were no accompanying changes in end-expiratory lung volume. A longer rest period between inspirations could also enhance muscle perfusion (Bellemare *et al.*, 1983) and reduce the extent of inspiratory muscle fatigue (Bellemare and Grassino, 1982). Although no gross changes in breathing pattern were observed in the present study during incremental exercise, we cannot discount the possibility that subtle changes may have profound effects upon inspiratory muscle function. The tendency for the inspiratory muscle training group to maintain tidal volume during the later stages of the incremental test, at a time when the placebo group resorted to a tachypnoeic breathing pattern, supports the notion that inspiratory muscle fatigue was attenuated after inspiratory muscle training (Romer *et al.*, 2002).

Changes in time-trial performance

The present study identified significant (−3.5% and −3.4%) effects in performance for the 20 and 40 km

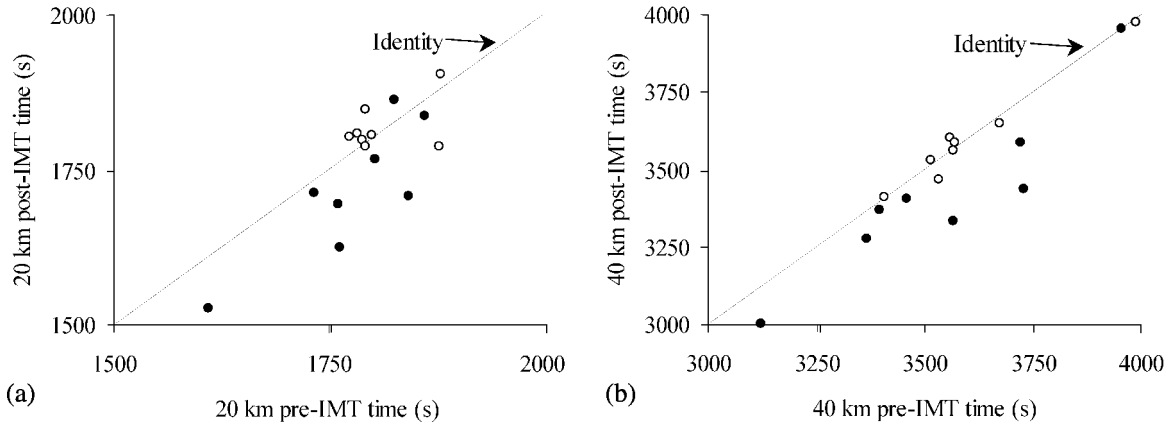

Fig. 8. Relationship between pre-IMT and post-IMT 20 km (a) and 40 km (b) time-trial performance for the IMT (●) and placebo (○) conditions. Note that 20 and 40 km time-trial performances were shorter in 7 of 8 post-IMT trials. IMT = inspiratory muscle training.

Table 5. Correlation matrix of the relative changes from baseline with inspiratory muscle training for pre-exercise measurements of inspiratory muscle function

	$\%\Delta P_0$	$\%\Delta \dot{V}_{max}$	$\%\Delta \dot{W}_{Imax}$	%Δ MRPD
%Δ Performance time	0.48	0.39	0.33	0.56*
$\%\Delta P_0$		0.86**	0.92**	0.63*
$\%\Delta \dot{V}_{max}$			0.83**	0.48
$\%\Delta \dot{W}_{Imax}$				0.56*

Abbreviations: P_0, maximal inspiratory pressure at zero flow; $\dot{V}_{max}$, maximal inspiratory flow; $\dot{W}_{Imax}$, maximal inspiratory muscle power; MRPD, maximal rate of pressure development.
Note: $n = 16$ (pooled 20 and 40 km time-trial data for inspiratory muscle training group). On the basis of stepwise multiple regression analysis, changes in MRPD accounted for 32% of the variance in %Δ performance time: %Δ performance time = 0.06(%Δ MRPD) − 4.74 (SEE = 2.5%). * $P \leq 0.05$; ** $P \leq 0.01$.

time-trials (−3.8 and −4.6% above control) and the likely ranges of the true effect of the treatment on the average participant (i.e. 95% confidence intervals) for these outcomes included enhancements of almost twice these magnitudes. The smallest enhancement of performance that would make a difference to an athlete's chance of winning is about half the typical variation in an athlete's performance between events (Hopkins *et al.*, 1999). Although there are no published data on the typical variation of cyclists from event to event, preliminary data suggest that elite male cyclists have typical variation of time of ~1% for time-trials lasting ~1 h (Paton and Hopkins, 2001). Thus, the improvements in performance observed in the present study would likely constitute worthwhile changes. The effect sizes identified for 20 and 40 km time-trial performances (0.31 and 0.40, respectively) were considered 'moderate-to-large' and 'large' based on the classification of Cohen (1988).

The finding of an ergogenic effect with respiratory muscle training is consistent with some (Boutellier and Piwko, 1992; Boutellier *et al.*, 1992; Spengler *et al.*, 1999; Markov *et al.*, 2001; Stuessi *et al.*, 2001; Volianitis *et al.*, 2001), but not all (Morgan *et al.*, 1987; Belman and Gaesser, 1988; Fairbarn *et al.*, 1991; Hanel and Secher, 1991; Kohl *et al.*, 1997; Inbar *et al.*, 2000; Sonetti *et al.*, 2001), previous studies. The relative changes in exercise performance for the inspiratory muscle training group alone (3.4–3.5%) are comparable with the results of previous respiratory muscle training studies in competitive rowers (3.1–3.5%; Volianitis *et al.*, 2001) and cyclists (1.8%; Sonetti *et al.*, 2001), although only the former study obtained statistical significance when comparisons were made with the sham-training placebo groups.

Our rigorous experimental design excluded the major potential confounding influences on the participants' responses to inspiratory muscle training. It is unlikely that either the improvements in inspiratory muscle function or whole-body performance observed in the present study can be ascribed to a whole-body training response. The routine physical activity of the

participants did not change during the study and no significant relationships were observed between the quantity of whole-body training, inspiratory muscle function and time-trial performances. Similarly, we do not believe that between-trial differences in participant motivation or experimenter bias were responsible for the observed changes in time-trial performance, or for the physiological alterations post-intervention, since the study had a double-blind, placebo-controlled design. The mechanism by which inspiratory muscle training improves whole-body exercise performance is, as yet, unclear. We believe the reason for the improvement is multifactorial, but may include the effects of inspiratory muscle training on the intensity with which both respiratory and peripheral efforts are perceived.

Changes in respiratory effort sensations

The present study provides evidence that inspiratory muscle training leads to overall dyspnoea abatement during maximal incremental cycling. This outcome is in line with that reported in clinical populations (ACCP/AACVPR, 1997) and endurance-trained rowers (Volianitis *et al.*, 2001). Because of the association between respiratory muscle function and respiratory effort sensation (ATS, 1999), an improvement in the contractile properties of inspiratory muscles with specific inspiratory muscle training would be expected to result in reduced dyspnoea (Kellerman *et al.*, 2000). Improvement in the force-generating capacity of inspiratory muscles decreases the relative tension for a given level of ventilation. The effect would probably be to reduce the intensity of respiratory effort during periods of elevated ventilation (e.g. exercise). An alternative hypothesis to explain the reduction in dyspnoea with inspiratory muscle training is that the repeated generation of large inspiratory pressures (as occurs during inspiratory muscle training) has a desensitizing effect upon the sensory input from the inspiratory muscles to the brain during exercise (El-Manshawi *et al.*, 1986; Revelette and Wiley, 1987; Wilson and Jones, 1990).

In the inspiratory muscle training group, tidal volume increased significantly above pre-intervention values at the higher intensities of incremental exercise, while the placebo group continued to adopt a more tachypnoeic breathing pattern, characterized by a reduction in tidal volume and a slight increase in respiratory frequency, for the maintenance of minute ventilation. Although breathing pattern was not measured during the time-trial rides (to minimize interference with the participant's performance), many of the participants described their breathing as more controlled during the later stages after inspiratory muscle training. Indeed, when the relative improvement in time-trial performance was considered based on the percentage of total work completed, it was in the final 25% that participants experienced the greatest improvement (see Fig. 7). Thus, the onset of severe breathlessness might have been delayed, allowing a higher external power to be maintained throughout the tests for the same respiratory and peripheral effort.

Changes in peripheral effort sensations

In addition to the effects of specific inspiratory muscle training on respiratory effort sensations during whole-body exercise, inspiratory muscle training may mediate the intensity of exertional perceptions via peripheral mechanisms. A previous study has observed a significant reduction in peripheral effort sensations after inspiratory muscle training (Caine and McConnell, 1998). This observation is in agreement with the results of the present study that inspiratory muscle training attenuates the perception of peripheral effort during maximal incremental cycling. The peripheral physiological processes potentially influenced by inspiratory muscle training include those linked to metabolic acidosis and respiratory muscle blood flow.

Specific inspiratory muscle training may facilitate a reduction in the intensity of peripheral effort sensations via favourable changes in acid–base balance. The results of the present study indicate a trend towards a reduced blood lactate concentration during incremental exercise after inspiratory muscle training, a finding that concurs with the results of previous investigations (Boutellier *et al.*, 1992; Kohl *et al.*, 1997; Spengler *et al.*, 1999). Experimental and correlational investigations support blood pH as a potent mediator of exertional perceptions, especially at exercise intensities that equal or exceed the lactate threshold (Kostka and Cafarelli, 1982).

Blood flow to exercising muscle determines the availability of energy substrates for exercise metabolism. Inadequate perfusion of tissue limits metabolism, inducing fatigue and intensifying the peripheral perceptual signal (Cain, 1973). Conversely, improvements in blood flow to exercising muscle would be expected to reduce the intensity of peripheral effort sensations. Recent evidence suggests that unloading the respiratory muscles via proportional assist ventilation improves exercise capacity (Harms *et al.*, 2000). A redistribution of blood flow from respiratory muscles to locomotor muscles was deemed responsible for the improvement in exercise capacity. Although speculative, inspiratory muscle training may lower the relative intensity of inspiratory muscle work or delay the fatigue of the inspiratory muscles resulting in the production of fewer metabolic stimuli by these muscles. These events could signal a decreased need for vasodilatation in the

inspiratory muscles and allow a greater blood supply to be directed to the locomotor muscles (Wetter and Dempsey, 2000). Alternatively, a reduction in chemical stimuli could reduce the output from respiratory muscles to the working limbs via reflex pathways (type III/IV afferents), reducing vasoconstriction in the limbs (Wetter and Dempsey, 2000). In either case, increased limb blood flow may result in greater oxygen delivery to the limbs, reduced limb fatigue and perhaps attenuated peripheral effort sensations. Since the total proportion of cardiac output that is directed to the respiratory muscles during maximal exercise is about 14–16% (Harms *et al.*, 1998), any effects of inspiratory muscle training on blood flow redistribution are likely to be small. Our results hint at a change in oxygen uptake (−3%) and thus cardiac output (−4%) but lack the statistical power and direct cardiovascular measurements to demonstrate this unequivocally.

In conclusion, the results of the present study support previous evidence that specific inspiratory muscle training attenuates the perceptual response to maximal incremental exercise. The study provides new evidence of performance enhancements in competitive cyclists after inspiratory muscle training. Further studies are required to clarify the mechanisms by which changes in inspiratory muscle function modify endurance performance.

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