Enhanced pulmonary and active skeletal muscle gas exchange during intense exercise after sprint training in men

Michael J. McKenna *, George J. F. Heigenhauser †, Robert S. McKelvie †, George Obminski †, J. Duncan MacDougall ‡ and Norman L. Jones †

*Departments of † Medicine and ‡ Kinesiology, McMaster University, Hamilton, Ontario, Canada and * Department of Human Movement, Recreation and Performance, Centre for Rehabilitation, Exercise and Sports Science, Victoria University of Technology, Melbourne, Victoria 8001, Australia*

1. This study investigated the effects of 7 weeks of sprint training on gas exchange across the lungs and active skeletal muscle during and following maximal cycling exercise in eight healthy males.

2. Pulmonary oxygen uptake ($\dot{V}_{O_2}$) and carbon dioxide output ($\dot{V}_{CO_2}$) were measured before and after training during incremental exercise ($n = 8$) and during and in recovery from a maximal 30 s sprint exercise bout by breath-by-breath analysis ($n = 6$). To determine gas exchange by the exercising leg muscles, brachial arterial and femoral venous blood O_2 and CO_2 contents and lactate concentration were measured at rest, during the final 10 s of exercise and during 10 min of recovery.

3. Training increased ($P < 0.05$) the maximal incremental exercise values of ventilation ($\dot{V}_E$, by $15.7 \pm 7.1\%$), $\dot{V}_{CO_2}$ (by $9.3 \pm 2.1\%$) and $\dot{V}_{O_2}$ (by $15.0 \pm 4.2\%$). Sprint exercise peak power ($3.9 \pm 1.0\%$ increase) and cumulative 30 s work ($11.7 \pm 2.8\%$ increase) were increased and fatigue index was reduced (by $-9.2 \pm 1.5\%$) after training ($P < 0.05$). The highest $\dot{V}_E$, $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ values attained during sprint exercise were not significantly changed after training, but a significant ($P < 0.05$) training effect indicated increased $\dot{V}_E$ (by $19.2 \pm 7.9\%$), $\dot{V}_{CO_2}$ (by $9.3 \pm 2.1\%$) and $\dot{V}_{O_2}$ (by $12.7 \pm 6.5\%$), primarily reflecting elevated post-exercise values after training.

4. Arterial O_2 and CO_2 contents were lower after training, by respective mean differences of 3.4 and 21.9 ml l^{-1} ($P < 0.05$), whereas the arteriovenous O_2 and CO_2 content differences and the respiratory exchange ratio across the leg were unchanged by training.

5. Arterial whole blood lactate concentration and the net lactate release by exercising muscle were unchanged by training.

6. The greater peak pulmonary $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ with sprint exercise, the increased maximal incremental values, unchanged arterial blood lactate concentration and greater sprint performance all point strongly towards enhanced gas exchange across the lungs and in active muscles after sprint training. Enhanced aerobic metabolism after sprint training may contribute to reduced fatigability during maximal exercise, whilst greater pulmonary CO_2 output may improve acid–base control after training.

Rapid increases in ATP turnover during intense, short-term exercise result from both anaerobic and aerobic metabolism (Hermansen & Medbø, 1984; Jones *et al.* 1985; Cheetham, Boobis, Brooks & Williams, 1986; McCartney, Spriet, Heigenhauser, Kowalchuk, Sutton & Jones, 1986; Kowalchuk, Heigenhauser, Lindinger, Obminski, Sutton & Jones, 1988*a*; Medbø & Tabata, 1989; Nevill, Boobis, Brooks & Williams, 1989; Spriet, Lindinger, McKelvie, Heigenhauser & Jones, 1989; Stathis, Febbraio, Carey & Snow, 1994; Bogdanis, Nevill, Boobis & Lakomy, 1996). These metabolic processes together necessitate rapid increases in gas exchange within the active skeletal musculature and across the lung.

The extent of aerobic metabolism by contracting skeletal muscle during intense sprinting exercise has been approximated by measurements of pulmonary O_2 uptake ($\dot{V}_{O_2}$). Breath-by-breath respiratory measurements during a maximal 30 s sprint bout indicate at least a 10-fold rise in pulmonary $\dot{V}_{O_2}$ in untrained males (Kowalchuk *et al.* 1988*a*). When measured with Douglas bags, pulmonary $\dot{V}_{O_2}$ during a

30 s sprint reached 54% of $\dot{V}_{O_2,max}$ (Withers *et al.* 1991). Thus, aerobic metabolism has been estimated to supply between 28 and 40% of the total energy requirements during such exercise (Hermansen & Medbø, 1984; Medbø & Tabata, 1989; Withers *et al.* 1991; Bogdanis *et al.* 1996).

Substantial CO_2 production occurs in contracting skeletal muscle as a consequence of increased mitochondrial oxidation of glycogen and fatty acids (Jones, 1980; McCartney *et al.* 1986). CO_2 is also evolved from intramuscular carbamino compounds (Kowalchuk *et al.* 1988*a* and references therein). Intense exercise also increases intramuscular [HCO_3^-] and [H^+], due to increases in lactate concentration and loss of intramuscular potassium, reducing the strong ion difference, and thereby generating carbonic acid, which dissociates, producing additional CO_2 (Kowalchuk *et al.* 1988*a*; Kowalchuk, Heigenhauser, Lindinger, Sutton & Jones, 1988*b*). Inactive skeletal muscle may provide another source of CO_2 during intense exercise, resulting from lactate uptake and oxidation, acidosis-induced dissociation of carbamino compounds and carbonic acid dissociation (Kowalchuk *et al.* 1988*a*). Additional CO_2 will be produced through carbonic acid formation and dissociation in blood and inactive tissues, again due to the acidosis of exercise. Together these result in a marked increase in CO_2 production during sprint exercise, evident in the large increases in venous P_{CO_2}, which may exceed 100 mmHg in blood draining active muscle (Kowalchuk *et al.* 1988*a*; Lindinger, Heigenhauser, McKelvie & Jones, 1992) and 70 mmHg in blood draining inactive muscle (Kowalchuk *et al.* 1988*a*), coupled with the 10-fold increase in pulmonary CO_2 output ($\dot{V}_{CO_2}$) (Kowalchuk *et al.* 1988*a*).

To accommodate the rapidly increased demand for O_2 supply and CO_2 removal during intense sprinting exercise, pulmonary ventilation ($\dot{V}_E$) rises more than 10-fold (Kowalchuk *et al.* 1988*a*). This is sufficient to maintain arterial O_2 saturation, P_{O_2} and P_{CO_2} constant during sprint exercise; whereas post-exercise, the increase in $\dot{V}_E$ exceeds CO_2 production with a consequent sharp decline in arterial P_{CO_2} (Kowalchuk *et al.* 1988*a*). Despite this respiratory compensation, arterial plasma [H^+] may rise more than 2-fold during intense exercise, due almost entirely to marked ionic concentration changes, dominated by the rise in plasma lactate concentration ([Lac^-]) to more than 20 mmol l^{-1} (Hermansen & Osnes, 1972; Kowalchuk *et al.* 1988*a*,*b*; Lindinger *et al.* 1992). Thus, the rapid increase in pulmonary ventilation during intense exercise enables rapid increases in aerobic metabolism but also serves as a vital means of acid–base control through the rapid elimination of CO_2 from muscle.

To our knowledge, no studies have investigated the effects of high-intensity exercise training on aerobic metabolism, CO_2 output, $\dot{V}_E$ and acid–base regulation during and following sprint exercise. Although numerous studies have demonstrated that sprint training may increase $\dot{V}_{O_2,max}$ (Cunningham & Faulkner, 1969; Saltin *et al.* 1976; Sharp, Costill, Fink & King, 1986), the functional importance of this adaptation during sprint exercise remains controversial, with reports of both unchanged and increased $\dot{V}_{O_2}$ during intense exercise (Cunningham & Faulkner, 1969; Nevill *et al.* 1989).

The present study investigated the effects of 7 weeks of sprint training on the contributions of the lungs and active skeletal muscle to sprint exercise metabolism, CO_2 output and acid–base control. Pulmonary $\dot{V}_E$, $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ were measured before and after training using open-circuit spirometry during incremental exercise to determine individual maxima and, in a separate test, they were measured on a breath-by-breath basis to determine the functional importance of these changes during and following intense sprint cycling exercise. To investigate the role of the active musculature in exercise gas exchange, arterial and femoral venous blood gas contents and lactate concentrations were measured during and following a separate intense cycling exercise test, to indicate net O_2 extraction, net CO_2 and lactate release by the exercising leg muscles. The effects of sprint training on arterial and femoral venous plasma and whole blood ions and acid–base balance during sprint exercise are reported in the accompanying paper (McKenna, Heigenhauser, McKelvie, MacDougall & Jones, 1997). We hypothesized that sprint training would augment pulmonary and skeletal muscle gas exchange during and following intense exercise, as indicated by greater pulmonary $\dot{V}_E$, $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$, as well as by wider arteriovenous O_2 and CO_2 content differences across the exercising leg.

METHODS

Subjects and overview

Eight healthy males (means ± S.E.M.: age, 20·9 ± 0·6 years; height, 178·2 ± 1·9 cm; body mass, 75·3 ± 3·2 kg) participated in the study after being informed of the associated risks and giving written informed consent. All procedures were approved by the McMaster University Ethics Committee. The subjects were not highly trained and none participated in regular physical activities outside the training programme. Subjects completed three exercise tests before and after 7 weeks of sprint training, comprising an incremental exercise test ($n = 8$) and a maximal 30 s sprint test on an isokinetic cycle ergometer ($n = 6$) conducted on two separate days (Fig. 1). Ventilation and gas exchange were measured by breath-by-breath analysis during the first 30 s sprint test (respiratory test); brachial arterial and femoral venous blood samples were drawn during the second 30 s sprint test (invasive test). Separate sprint tests were performed for practical reasons as well as to reduce the stressful effects of the complex procedures on the subject; these tests were conducted in the same order before and after training (Fig. 1).

Sprint training protocol

Subjects completed 7 weeks of sprint training, undertaking three sessions per week, as described in detail in the accompanying paper (McKenna *et al.* 1997). Briefly, training comprised a series of maximal 30 s sprint bouts performed on mechanically braked cycle ergometers (Monark, Varberg, Sweden), with the flywheel tension set at 0·075 kg (kg body mass)$^{-1}$. Training progressed from four to ten bouts per session in weeks 1 to 4, with 4 min recovery periods between bouts; thereafter ten bouts per session were performed

with recovery time progressively reduced to 3 min during weeks 5 and 6 and unchanged in week 7.

Incremental exercise

Incremental exercise (60 r.p.m., 24·5 W min^{-1}) was conducted on an electrically stabilized cycle ergometer (Elema AM 370) and continued until volitional fatigue. Ventilation and gas exchange were measured every 15 s using a previously validated (Jones, 1984) open-circuit gas analysis system using a mixing chamber (Metabolic Measurement Cart; Sensor Medics, Pasadena, CA, USA), as previously described in detail (Kowalchuk *et al.* 1988*a*). The turbine system was calibrated before each test using a manually operated standard syringe; the gas analysers were calibrated before and rechecked at the completion of every test against room air and two precision gases, each checked against gases analysed by the Lloyd–Haldane procedure. $\dot{V}_{O_2,max}$ was defined as the mean of the two highest consecutive (15 s) oxygen uptake measurements at the highest work rate. The incremental respiratory data were analysed for two ventilatory thresholds, based on changes in $\dot{V}_E/\dot{V}_{O_2}$ and $\dot{V}_E/\dot{V}_{CO_2}$ relative to $\dot{V}_{O_2}$. The first ventilatory threshold (VT_1) was defined as the $\dot{V}_{O_2}$ beyond which a progressive rise in $\dot{V}_E/\dot{V}_{O_2}$ occurred, with no concomitant rise in $\dot{V}_E/\dot{V}_{CO_2}$. A second ventilatory threshold (VT_2) was defined as the $\dot{V}_{O_2}$ beyond which a progressive rise in $\dot{V}_E/\dot{V}_{CO_2}$ occurred.

Maximal sprint performance

Subjects performed maximal sprint exercise at 100 r.p.m. before and after the sprint training programme on an isokinetic cycle ergometer. Full details of the ergometer, test procedure and measurements taken are described in the accompanying paper (McKenna *et al.* 1997). Briefly, torque was sampled every 10 ms from instrumented pedal cranks; peak torque, peak instantaneous power, maximal average power and work done were calculated for every pedal stroke and the latter summed for all pedal strokes to give total 30 s cumulative work output. The percentage decline in power output from the peak attained in the first few pedal strokes to the final power at the end of the test was expressed as the fatigue index.

Respiratory test: ventilation and gas exchange with sprint exercise

Pulmonary gas exchange was measured at rest, during and following the first 30 s sprint test, as previously described in detail (Kowalchuk *et al.* 1988*a*). Resting $\dot{V}_E$, $\dot{V}_{CO_2}$, $\dot{V}_{O_2}$ and respiratory exchange ratio (RER) were measured using the open-circuit gas analysis system described above, with the subject seated on the cycle ergometer for 10 min before the test. Heart rate (HR) was measured by ECG. Breath-by-breath measurement of $\dot{V}_E$, $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ commenced at the beginning of sprint exercise and continued until 9·5 min after the end of exercise (Kowalchuk *et al.* 1988*a*). Two separate respiratory measurement systems were used during the test because of data storage limitations with the breath-by-breath system. Very close matching between these two systems has been previously demonstrated during steady-state and non-steady-state conditions over the range of respiratory responses encountered with incremental and sprint exercise (Kowalchuk, 1985). Expired air flow was measured by a pneumotachograph (Fleisch no. 3) and a differential pressure transducer (Hewlett Packard 270) and volume was derived by integration (Validyne). Expired O_2, CO_2 and N_2 concentrations were determined by mass spectrometer (Perkin Elmer MG-1100) from gas sampled from the mouthpiece at a rate of 60 ml min^{-1}. Breath temperature was measured by a thermometer placed between the expired air valve and the pneumotachograph. The air flow and gas concentration signals were sampled every 50 ms by computer (Digital Equipment PDP 11-03). Flow signals were computed to single breath data, and matched to gas concentrations identified as single breaths using the peak end-tidal CO_2, after accounting for a 250 ms delay in gas concentration measurements. The corresponding O_2 uptake and CO_2 output for each breath were calculated from inspired–expired gas concentration differences, and by expired ventilation, with inspired ventilation being calculated by nitrogen correction (Jones, 1984). Air flow and gas concentrations were calibrated immediately before each test. Briefly, air flow was calibrated using a rotameter and power vacuum system with a range of steady-state flows between 0 and 300 l min^{-1}. Gas concentrations were calibrated against room air and three precision gases, each checked against gases analysed by the Lloyd–Haldane procedure. A range of flow and gas concentration measurements was checked against constructed calibration curves. To reduce breath-by-breath fluctuations and to allow pre- *vs.* post-training statistical comparisons, all individual breath $\dot{V}_E$, $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ data during 15 s intervals were averaged, during exercise and for the first 2 min following exercise ($n = 6$). Thereafter, all breaths within 30 s intervals were averaged ($n = 5$). HR ($n = 6$) was averaged over each 30 s from the end of exercise until 4·5 min post-exercise and over every 60 s thereafter. Data are presented with different subject numbers in these measurements because of technical difficulties; however, only paired pre- and post-training data are reported for each individual, thus allowing comparisons of training effects. Data are presented as the means ± S.E.M. of the mid-point of each sampling period (i.e. 22·5 s post-exercise data represent data collected between 15 and 30 s post-exercise).

Invasive test: blood sampling with sprint exercise

Full details of subject preparation, catheterization and postural controls are given in the accompanying paper (McKenna *et al.* 1997). Briefly, blood samples were drawn simultaneously from brachial arterial (a) and femoral venous (v) catheters at rest, during the final 10 s of the 30 s sprint bout and at 1, 2, 5 and 10 min of recovery, in both pre- and post-training tests. Blood samples were analysed for P_{CO_2}, P_{O_2}, pH, oxygen saturation (S_{O_2}), haematocrit (Hct), haemoglobin concentration ([Hb]) and whole blood lactate concentration ([Lac^-]), as described in the accompanying paper (McKenna *et al.* 1997). In two of the six subjects completing the invasive test it proved impossible to obtain venous blood samples within the last 10 s of exercise; thus for this observation point $n = 4$; for all other times $n = 6$.

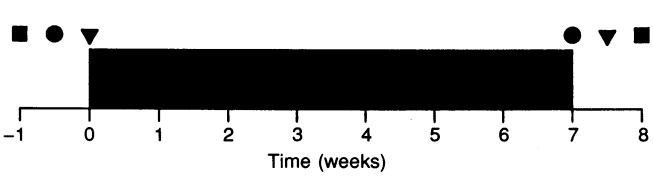

Figure 1. Experimental overview

Filled bar represents duration of sprint training protocol. ■, $\dot{V}_{O_2,max}$ test; ●, respiratory sprint test; ▼, invasive sprint test.

Table 1. Peak cardiorespiratory responses to maximal incremental exercise (30 s average, $n = 8$) and a maximal 30 s sprint bout (15 s average, $n = 6$) before and after sprint training

	HR (beats min^{-1})	$\dot{V}_E$ (l min^{-1})	$\dot{V}_{O_2}$ (l min^{-1})	$\dot{V}_{CO_2}$ (l min^{-1})	RER
Maximal incremental exercise test					
Pre-training	189 ± 4	132·6 ± 7·4	3·53 ± 0·19	4·19 ± 0·18	1·20 ± 0·04
Post-training	188 ± 4	151·0 ± 6·2*	4·02 ± 0·16*	4·56 ± 0·15*	1·14 ± 0·01
Maximal 30 s sprint test					
End exercise					
Pre-training	173 ± 4	121·1 ± 2·6	1·964 ± 0·227	2·535 ± 0·213	1·32 ± 0·08
Post-training	172 ± 6	134·0 ± 12·0	2·038 ± 0·219	2·460 ± 0·198	1·24 ± 0·07
Peak post-exercise					
Pre-training	—	—	2·183 ± 0·181	3·154 ± 0·252	2·36 ± 0·11
Post-training	—	—	2·352 ± 0·172	3·408 ± 0·251	2·10 ± 0·20

*$P < 0·05$.

Calculations

Blood CO_2 content (C_{CO_2}) was calculated from P_{CO_2}, pH, S_{O_2} and [Hb], using the equation of Douglas, Jones & Reed (1988). Blood oxygen content (C_{O_2}, in ml l^{-1}) was calculated as: $C_{O_2} = [Hb] \times S_{O_2} \times 0·134$. Values for P_{CO_2}, pH, [Hb] and plasma ions have been reported in the companion paper (McKenna *et al.* 1997). The decline in blood volume in venous compared with arterial blood (ΔBV_{a-v}) was calculated during and following exercise from changes in [Hb] and Hct (McKenna *et al.* 1997). The arteriovenous differences for whole blood [Lac$^-$] ([Lac$^-$]$_{a-v}$, in mmol l^{-1}) were corrected for ΔBV_{a-v} (%) using the equation: $[Lac^-]_{a-v} = ([Lac^-]_a/(1 + \Delta BV_{a-v})) - [Lac^-]_v$ (McKenna *et al.* 1997). Excess post-exercise O_2 uptake was calculated as the total recovery $\dot{V}_{O_2}$ less the resting $\dot{V}_{O_2}$. Similarly, the excess post-exercise CO_2 output was determined as the total recovery $\dot{V}_{CO_2}$ less the resting $\dot{V}_{CO_2}$. Leg respiratory exchange ratio was calculated as $C_{a-v,CO_2}/C_{a-v,O_2}$, with the pulmonary respiratory exchange ratio calculated as $\dot{V}_{CO_2}/\dot{V}_{O_2}$.

Statistics

All data are reported as means ± S.E.M. Training effects on paired incremental and sprint test data were analysed by Student's paired two-tailed t tests. Sprint exercise performance measures (trial, training and subject), respiratory and blood data (measurement time, training and subject) and submaximal exercise responses (work rate, training and subject) were analysed using three-way analysis of variance, with subsequent Newman–Keuls *post hoc* analyses. Interactions between time and training are referred to only when significant, and direct comparisons of pre- *vs.* post-training at a given time were performed only when a significant interaction effect was found. A significance level of $P < 0·05$ was accepted for all statistical analyses.

RESULTS

Incremental exercise

The maximal power output achieved during incremental exercise increased by 10% from 269·9 ± 18·1 W before training to 327·7 ± 16·4 W following sprint training ($P < 0·05$); the corresponding cumulative work outputs were 17·71 ± 0·90 and 19·44 ± 0·87 kJ ($P < 0·05$). Peak incremental $\dot{V}_E$, $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ (pre-training, 47·1 ± 2·6 ml kg^{-1} min^{-1} *vs.* post-training, 53·0 ± 2·6 ml kg^{-1} min^{-1}) were increased after training ($P < 0·05$, Table 1). Body mass was unchanged after training (pre-training, 75·25 ± 3·40 kg *vs.* post-training, 76·34 ± 3·28 kg).

Small but significant training main effects ($P < 0·05$) were found for submaximal $\dot{V}_E$, $\dot{V}_{CO_2}$, $\dot{V}_{O_2}$, RER and $\dot{V}_E/\dot{V}_{O_2}$, when compared across all submaximal work rates (24·5–220·5 W) common to all subjects, in pre- and post- training tests. After training, submaximal $\dot{V}_{O_2}$ was higher (by 0·02 ± 0·03 l min^{-1}), whilst lower values were found for submaximal $\dot{V}_E$ (−1·5 ± 1·2 l min^{-1}), $\dot{V}_{CO_2}$ (−0·06 ± 0·04 l min^{-1}), RER (−0·04 ± 0·03), $\dot{V}_E/\dot{V}_{O_2}$ (−1·0 ± 0·6) and HR (−5 ± 2 beats min^{-1}).

VT_1 was increased from a $\dot{V}_{O_2}$ of 1·61 ± 0·14 l min^{-1} before training to 1·94 ± 0·17 l min^{-1} after training ($P < 0·06$), corresponding to 46 ± 4% of the pre-training $\dot{V}_{O_2,max}$ and 48 ± 3% of the post-training $\dot{V}_{O_2,max}$, respectively (n.s.). VT_2 was increased from 2·50 ± 0·08 l min^{-1} before to 2·73 ± 0·10 l min^{-1} after training ($P < 0·05$), corresponding to 72 ± 3% of the pre-training $\dot{V}_{O_2,max}$ and 68 ± 3% of the post-training $\dot{V}_{O_2,max}$, respectively (n.s.).

Maximal sprint performance

Order effects. Small but statistically significant trial-order effects were found, with higher peak power (increased by 3·8 ± 1·3%, $P < 0·05$), maximal average power (increased by 6·3 ± 1·6%, $P < 0·01$), peak torque (increased by 4·2 ± 1·2%, $P < 0·01$) and a higher fatigue index (increased by 2·9 ± 1·4%, $P < 0·05$) in the invasive tests, carried out several days after the respiratory tests (Table 2). However, no order effect was found for cumulative work output. A trial order by training interaction effect was found for fatigue index, with the fatigue index during the pre-training invasive test being higher than in all other trials ($P < 0·001$). Despite the significant order effects, differences between the two trials in the same training status were

Table 2. Peak performance data during maximal 30 s sprint tests before and after 7 weeks of sprint training

	Peak instantaneous power (W)	Maximal average power (W)	Maximal peak torque (N m)	Fatigue index (%)	Cumulative work (kJ)
Pre-training					
Respiratory test	1427·1 ± 80·0	879·4 ± 59·8	132·5 ± 6·8	48·9 ± 2·1	18·8 ± 1·2
Invasive test	1452·1 ± 64·9†	949·2 ± 47·9††	136·3 ± 5·7††	55·8 ± 2·6†‡	19·5 ± 0·8
Post-training					
Respiratory test	1456·0 ± 74·2*	963·8 ± 46·2**	135·2 ± 6·7*	43·6 ± 1·5**‡	20·9 ± 1·2**
Invasive test	1536·5 ± 78·0*†	1007·0 ± 55·5**††	142·1 ± 6·5*††	42·6 ± 1·5**†‡	21·9 ± 1·2**

Subjects completed two separate sprint tests in each training status, comprising: (1) respiratory measurements; and (2) invasive procedures involving arterial and venous blood sampling (both $n = 6$). Significant trial order effect: † $P < 0·05$, †† $P < 0·01$; significant training effect: * $P < 0·05$, ** $P < 0·01$; significant trial order by training interaction effect: ‡ $P < 0·001$.

small, with low coefficients of variation (pre-training, post-training) for peak power (4·0%, 3·5%), maximal average power (5·5%, 4·2%), maximal peak torque (3·7%, 2·7%), cumulative work (7·9%, 3·9%) and fatigue index (5·7%, 6·1%).

Training effects. Sprint performance was enhanced following training (Table 2), with increased peak power (increased by 3·9 ± 1·0%, $P < 0·05$), maximal average power (increased by 8·1 ± 2·3%, $P < 0·005$), peak torque (increased by 3·2 ± 1·1%, $P < 0·05$) and work output (increased by 11·7 ± 2·8%, $P < 0·001$) and with a lower fatigue index (−9·2 ± 1·5%, $P < 0·001$).

Cardiorespiratory changes with sprint exercise

Time main effects. $\dot{V}_E$, $\dot{V}_{CO_2}$, $\dot{V}_{O_2}$, RER and HR were all elevated during exercise and until 9·5 min post-exercise ($P < 0·05$, Figs 2–4). Before training, $\dot{V}_E$, $\dot{V}_{CO_2}$, $\dot{V}_{O_2}$ and HR during sprint exercise reached 91, 61, 55 and 92% of their respective maximal incremental exercise values (Table 1). Both $\dot{V}_E$ and HR declined immediately following exercise (Fig. 2). $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ continued to increase after exercise; $\dot{V}_{CO_2}$ reached a peak pre-training value of 3·154 ± 0·252 l min^{-1} at 13·8 ± 1·3 s post-exercise and $\dot{V}_{O_2}$ reached a peak pre-training value of 2·183 ± 0·181 l min^{-1} at 7·5 ± 1·9 s post-exercise (Table 1 and Fig. 3). These peak

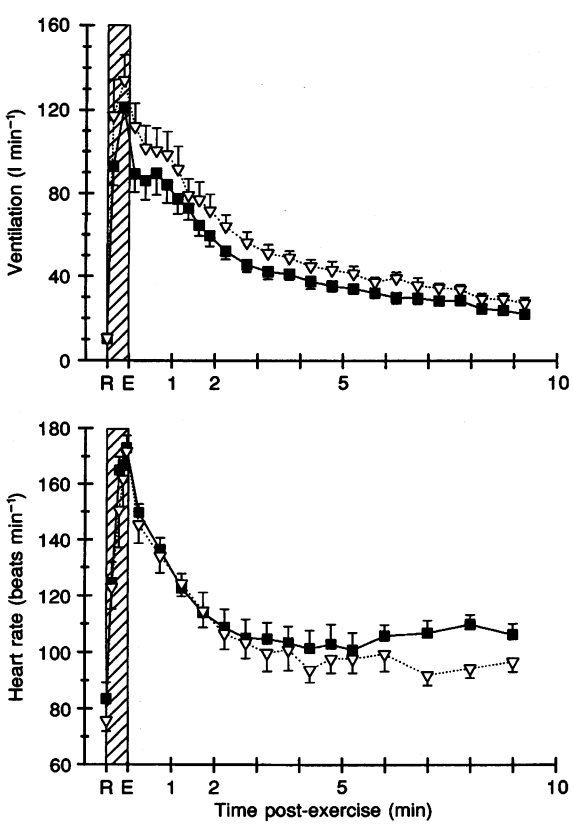

Figure 2. Ventilation (top) and heart rate (bottom)
Measurements were obtained at rest (R), during (denoted by hatched bar) and following maximal exercise (E), conducted before (■) and after (▽) 7 weeks of sprint training. $\dot{V}_E$ was averaged over 15 s from rest until 2 min post-exercise and then over 30 s until 9·5 min post-exercise; $n = 6$ during rest and up to 4·5 min post-exercise and $n = 5$ thereafter. HR ($n = 6$) was averaged over 30 s between 0 and 4·5 min post-exercise and over 60 s thereafter. Ventilation was elevated and heart rate reduced after training (both $P < 0·05$).

post-exercise values represented 75 and 62% of the maximal incremental values for $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$, respectively. The peak RER was also observed post-exercise and before training it exceeded 2·0 in all subjects (Fig. 4).

Training main effects. Training effects were significant for $\dot{V}_E$, $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ ($P < 0·05$, Figs 2 and 3), which were increased after training by mean differences of 10·5 ± 4·6 l min^{-1} (19·2 ± 7·9%), 0·12 ± 0·13 l min^{-1} (9·8 ± 9·5%) and 0·11 ± 0·06 l min^{-1} (12·7 ± 6·5%), respectively. HR was lower after training by a mean difference of 4·9 ± 4·5 beats min^{-1} (−3·9 ± 3·8%) ($P < 0·05$, Fig. 2), whereas RER was unaltered by training (Fig. 4).

After training, the peak $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ occurred post-exercise, being, respectively, 3·408 ± 0·251 l min^{-1} at 15·0 ± 1·9 s post-exercise and 2·352 ± 0·172 l min^{-1} at 11·3 ± 1·7 s post-exercise (Table 1 and Fig. 3). Peak $\dot{V}_{O_2}$ occurred slightly later after training ($P < 0·08$). Although higher in four of six subjects, neither peak $\dot{V}_{CO_2}$ nor peak $\dot{V}_{O_2}$ were different after training. The excess post-exercise $\dot{V}_{O_2}$ tended to be higher after training (by 20%), with increases in four out of five subjects (pre-training, 3·775 ± 0·379 l *vs.* post-training, 4·546 ± 0·448 l, $P < 0·2$, n.s.). The calculated total excess CO_2 output also tended to be higher, with respective pre- and post-training values being 9·853 ± 0·670 and 11·098 ± 0·765 l (13%, $P > 0·3$, n.s.).

Blood gas contents and leg respiratory exchange ratio

Arterial O_2 content (C_{a,O_2}) increased during exercise, remained elevated during recovery ($P < 0·05$) and was lower across all times after training, by a mean difference of 3·4 ml l^{-1} ($P < 0·05$, Fig. 5 and Table 3), reflecting a slightly lower S_{a,O_2} ($P < 0·05$). Venous O_2 content (C_{v,O_2}) decreased sharply during exercise ($P < 0·05$), returned to resting levels thereafter and was unchanged after training ($P < 0·07$, Table 3). The C_{a-v,O_2} increased during exercise ($P < 0·05$), indicating increased net O_2 extraction, but was unchanged after training (Fig. 5). Arterial CO_2 content (C_{a,CO_2}) decreased during and following exercise ($P < 0·05$) and was lower after training by a mean difference of 21·9 ml l^{-1} ($P < 0·05$, Fig. 6 and Table 3). In contrast, C_{v,CO_2} rose during exercise ($P < 0·05$), fell progressively during recovery ($P < 0·05$) and was also lower across all times after training, by a mean difference of 24·0 ml l^{-1} ($P < 0·05$, Table 3). A large net CO_2 release from muscle (negative C_{a-v,CO_2}) was found during and following exercise ($P < 0·05$, Fig. 6), but this was unchanged following training. Leg RER was increased above rest at 1 and 2 min following exercise ($P < 0·05$), peaking in excess of 3·0 at 1 min post-exercise, but no significant training effect was found (Fig. 4).

Blood lactate

Both $[Lac^-]_a$ and $[Lac^-]_v$ increased sharply during exercise and early recovery ($P < 0·05$), peaking at 5 min post-exercise (Fig. 7 and Table 3). Net muscle Lac^- release (negative $[Lac^-]_{a-v}$) was found at all time points, with the greatest $[Lac^-]_{a-v}$ occurring during exercise ($P < 0·05$) at −3·5 ± 1·0 mmol l^{-1} before and −4·1 ± 1·9 mmol l^{-1} after training (n.s., Fig. 7 and Table 3). No training effects were found for $[Lac^-]_a$, $[Lac^-]_v$ or $[Lac^-]_{a-v}$.

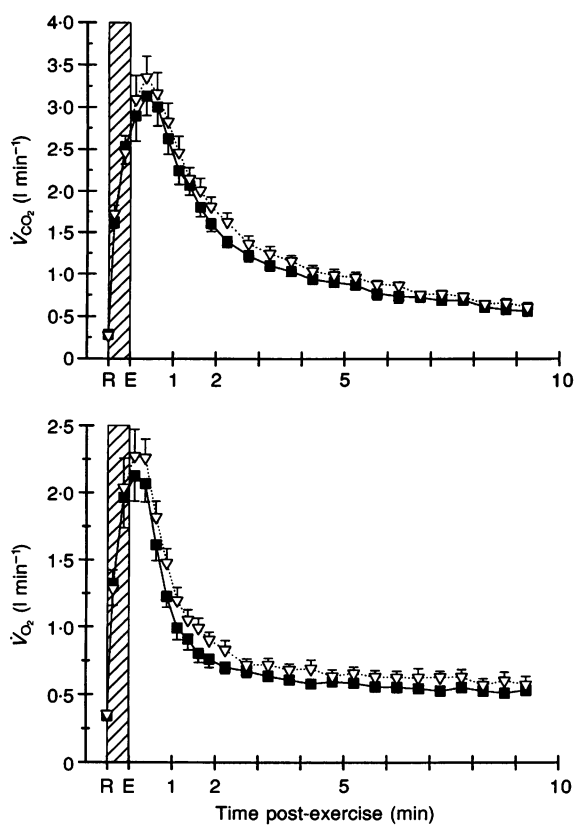

Figure 3. $\dot{V}_{CO_2}$ (top) and $\dot{V}_{O_2}$ (bottom)
Measurements were obtained at rest (R), during (hatched bar) and following maximal exercise (E), conducted before (■) and after (▽) sprint training. Sample size and times as for ventilation in Fig. 2. Both $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ were elevated after training (both $P < 0·05$).

Table 3. Arterial values, femoral venous values and arteriovenous differences in blood oxygen content (C_{O_2}), carbon dioxide content (C_{CO_2}) and lactate concentration ([Lac$^-$]) at rest, during sprint exercise and for 10 min of recovery, before and after sprint training

	Rest	Exercise	1 min	2 min	5 min	10 min
Pre-training						
$[Lac^-]_a$ (mmol l^{-1})*	1·5 ± 0·1	5·1 ± 0·5	13·2 ± 0·7	15·0 ± 0·8	16·2 ± 1·0	15·4 ± 1·3
$[Lac^-]_v$ (mmol l^{-1})*	1·7 ± 0·2	8·9 ± 1·3	16·9 ± 0·8	18·2 ± 1·0	18·5 ± 0·8	16·8 ± 1·2
$[Lac^-]_{a-v}$ (mmol l^{-1})*	−0·1 ± 0·1	−3·5 ± 1·0	−3·3 ± 0·4	−3·0 ± 0·7	−2·0 ± 0·8	−0·7 ± 0·5
C_{a,O_2} (ml l^{-1})*	205·2 ± 4·1	218·3 ± 4·4	223·8 ± 4·1	225·1 ± 4·1	221·3 ± 3·2	215·2 ± 2·9
C_{v,O_2} (ml l^{-1})*	169·0 ± 8·6	82·6 ± 4·5	174·6 ± 7·9	188·2 ± 7·3	179·5 ± 7·4	174·9 ± 5·7
C_{a-v,O_2} (ml l^{-1})*	36·2 ± 7·9	136·7 ± 2·4	49·2 ± 10·2	36·9 ± 8·8	41·8 ± 9·2	40·3 ± 7·7
C_{a,CO_2} (ml l^{-1})*	513·2 ± 6·7	434·4 ± 11·2	308·1 ± 15·0	261·8 ± 11·5	234·5 ± 10·6	245·5 ± 13·8
C_{v,CO_2} (ml l^{-1})*	546·0 ± 10·2	669·6 ± 21·2	447·0 ± 20·4	362·1 ± 10·0	304·7 ± 6·9	292·7 ± 12·5
C_{a-v,CO_2} (ml l^{-1})*	−32·7 ± 10·9	−237·5 ± 5·6	−138·9 ± 19·4	−100·3 ± 15·7	−70·2 ± 7·3	−47·2 ± 8·0
Post-training						
$[Lac^-]_a$ (mmol l^{-1})	1·1 ± 0·1	5·0 ± 0·6	14·2 ± 1·0	15·3 ± 0·9	16·4 ± 1·0	15·6 ± 1·2
$[Lac^-]_v$ (mmol l^{-1})	1·3 ± 0·1	8·6 ± 2·2	17·1 ± 1·6	18·7 ± 1·4	19·1 ± 1·3	16·8 ± 1·3
$[Lac^-]_{a-v}$ (mmol l^{-1})	−0·2 ± 0·1	−4·1 ± 1·9	−2·8 ± 0·6	−2·9 ± 0·6	−2·5 ± 0·6	−1·1 ± 0·4
C_{a,O_2} (ml l^{-1})†	202·6 ± 3·0	214·6 ± 2·3	220·9 ± 3·8	220·1 ± 2·8	215·4 ± 1·8	215·0 ± 3·0
C_{v,O_2} (ml l^{-1})	172·0 ± 9·5	75·3 ± 5·5	171·9 ± 5·4	177·4 ± 3·7	167·2 ± 6·6	168·8 ± 4·0
C_{a-v,O_2} (ml l^{-1})	30·6 ± 8·7	137·6 ± 6·5	49·1 ± 6·1	42·7 ± 4·7	48·3 ± 5·7	46·2 ± 5·7
C_{a,CO_2} (ml l^{-1})†	498·4 ± 13·2	421·5 ± 10·6	278·6 ± 7·5	233·7 ± 6·4	207·6 ± 11·7	226·7 ± 12·0
C_{v,CO_2} (ml l^{-1})†	535·8 ± 15·4	635·5 ± 19·5	425·4 ± 15·9	321·0 ± 5·4	276·1 ± 12·1	284·8 ± 13·4
C_{a-v,CO_2} (ml l^{-1})	−37·4 ± 6·5	−205·1 ± 12·1	−146·8 ± 13·9	−87·3 ± 7·0	−68·4 ± 10·9	−58·1 ± 10·5

$n = 6$ except for venous samples and arteriovenous differences during exercise, where $n = 4$. The $[Lac^-]_{a-v}$ values have been corrected for the arteriovenous decline in blood volume. * Significant time main effect ($P < 0·05$); † significant training main effect ($P < 0·05$). Details are reported in the text.

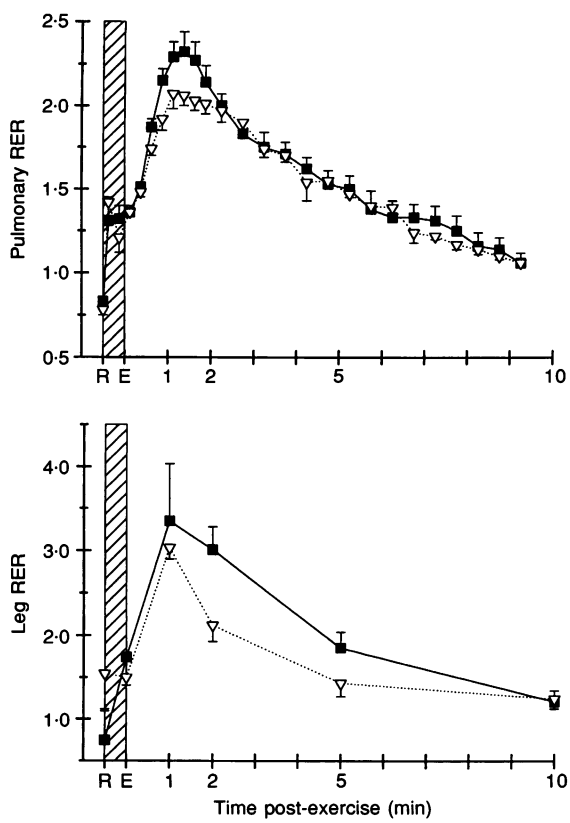

Figure 4. Pulmonary RER (top) and leg RER (bottom)
Measurements were obtained at rest (R), during (hatched bar) and following maximal exercise (E), conducted before (■) and after (▽) sprint training. Sample size and times for pulmonary RER as for ventilation in Fig. 2 and for leg RER as in Table 3. Training main effects were not significant.

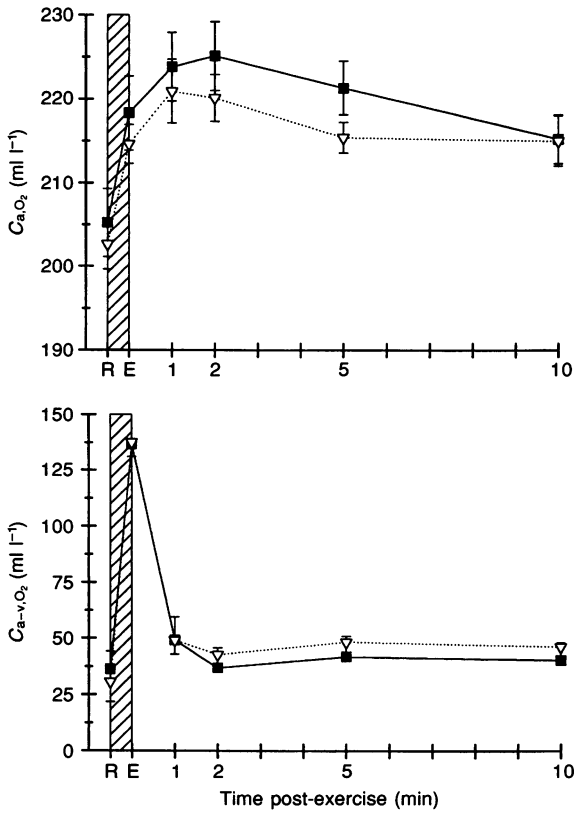

Figure 5. Arterial O_2 content (top) and arterio-femoral venous O_2 content difference (bottom)

Measurements were obtained at rest (R), during (hatched bar) and following maximal exercise (E), conducted before (■) and after (▽) sprint training. Data are means ± S.E.M., $n = 6$, except arteriovenous C_{O_2} difference during exercise, where $n = 4$. Arterial O_2 content was lower across all times after training ($P < 0{\cdot}05$).

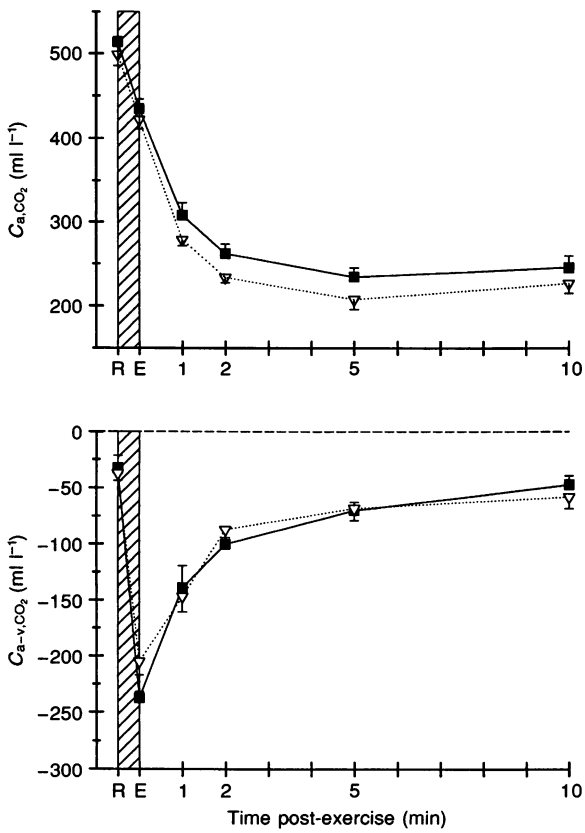

Figure 6. Arterial CO_2 content (top) and arterio-femoral venous CO_2 content difference (bottom)

Measurements were obtained at rest (R), during (hatched bar) and following maximal exercise (E), conducted before (■) and after (▽) sprint training. Data are means ± S.E.M., $n = 6$, except arteriovenous C_{CO_2} difference during exercise, where $n = 4$. Arterial CO_2 content was lower across all times after training ($P < 0{\cdot}05$).

DISCUSSION

Sprint training, comprising repeated 30 s bouts of maximal cycling exercise, with only brief recovery periods, increased maximal incremental exercise performance, ventilation and gas exchange. Work during sprint exercise increased and fatigue was lessened. Peak pulmonary O_2 uptake and CO_2 output were greater, suggesting that O_2 consumption and CO_2 production during sprint exercise were augmented after sprint training. Arterio-femoral venous O_2 and CO_2 contents and lactate concentration differences across the exercising leg during and following sprint exercise were all unchanged after training, suggesting that higher muscle blood flow may have accounted for the greater pulmonary gas exchange after training.

Enhanced sprint exercise performance with training

Sprint training increased maximal incremental exercise work rate by 10%, accompanied by similar relative increases in $\dot{V}_E$, $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$. Training also enhanced performance during maximal 30 s exercise, with a 12% increase in work output, a 4% increase in peak power and a 9% lower fatigue index. An order effect independent of training was found such that exercise performance was greater in the second, invasive trial than in the first, respiratory trial. Although a learning influence cannot be discounted, the effect was qualitatively and quantitatively similar in tests conducted pre- and post-training. Since the order effect was present in both pre- and post-training tests, our interpretations of training effects on either respiratory or blood metabolite data are not adversely affected. The greater performance in the invasive trial may have resulted from higher levels of subject arousal due to the invasive procedures employed, as well as a slightly reduced performance during the respiratory trial, resulting from the encumbrance of breathing through respiratory circuitry. Despite this, the differences between the two trials in the same training status were also relatively small, with coefficients of variation similar to values of ~5% previously reported for intense cycle exercise (Coggan & Costill, 1984). Thus, on the basis of performance measures, it is reasonable to compare respiratory changes in the first test with metabolic and gas exchange variables across the exercising leg in the second test. Further validation of the appropriateness of matching between the two tests was the close correspondence between the measured pulmonary $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ and values calculated from arterio-femoral venous gas content differences, assuming a leg blood flow of 6 l min^{-1} at the end of exercise (Lindinger *et al.* 1992). Before training, the increase in pulmonary $\dot{V}_{O_2}$ above rest measured during the final 15 s of exercise was 1·62 l min^{-1}, identical to the $\dot{V}_{O_2}$ (1·64 l min^{-1}) calculated from C_{a-v,O_2} for both legs over the final 10 s of exercise. The respective post-training values, assuming the same blood flow, were also identical (1·65 and 1·68 l min^{-1}). These calculations also indicate that the rise in pulmonary $\dot{V}_{O_2}$ could be attributed to the exercising leg muscles. The matching between trials for $\dot{V}_{CO_2}$ was reasonable, being 2·25 l min^{-1} for measured increase in pulmonary $\dot{V}_{CO_2}$ *vs.* 2·85 l min^{-1} for calculated two-legged $\dot{V}_{CO_2}$ before training and 2·18 *vs.* 2·46 l min^{-1}, respectively, after training. Slight differences between pulmonary and muscle gas exchange data reflect errors due to the 15 s averaging of pulmonary data *vs.* a single time point measure for blood data, subject

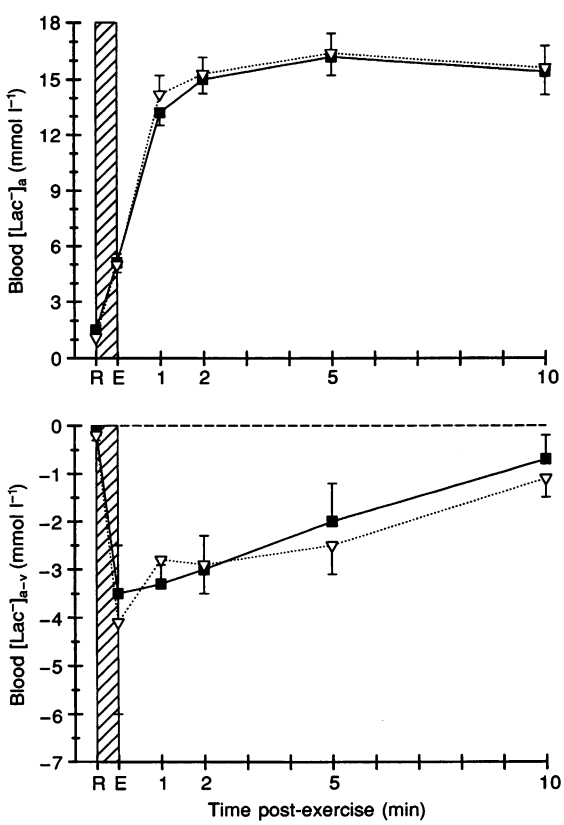

Figure 7. Arterial blood lactate concentration (top) and arterio-femoral venous blood lactate concentration difference (bottom)

Meaurements were obtained at rest (R), during (hatched bar) and following maximal exercise (E), conducted before (■) and after (▽) sprint training. Data are means ± S.E.M., $n = 6$, except arteriovenous [Lac$^-$] difference during exercise, where $n = 4$. Training main effects were not significant.

variability in the invasive and respiratory tests, slight delays in peak pulmonary responses after cessation of exercise (i.e. post-exercise) and the use of the same muscle blood flow for estimation of muscle gas exchange in pre- and post-training tests.

Enhanced aerobic metabolism following sprint training

Submaximal exercise. A small but significant increase in submaximal $\dot{V}_{O_2}$ was found after training, suggesting improved O_2 kinetics during non-steady-state exercise. This, together with lower submaximal $\dot{V}_E$, $\dot{V}_{CO_2}$ and RER, as well as increased respiratory thresholds, suggests that sprint training induces qualitatively similar aerobic adaptations to those reported after endurance training (Hickson, Bomze & Holloszy, 1978; Taylor & Jones, 1979). The greater VT_1 and VT_2 after training suggests a delayed rise in blood [Lac^-] and improved acid–base balance during incremental exercise. However, neither of these possibilities can be confirmed in the present study, since no blood samples were drawn during the incremental exercise test.

Increased $\dot{V}_{O_2,max}$. The 15% greater $\dot{V}_{O_2,max}$ in progressive exercise after training is consistent with increases reported in earlier studies using untrained males (Cunningham & Faulkner, 1969; Saltin *et al.* 1976). By the Fick equation the increased $\dot{V}_{O_2,max}$ must be due to an increased maximal cardiac output and/or maximal O_2 extraction, but neither variable has yet been studied in humans during maximal incremental exercise after sprint training. An increased maximal cardiac output has been found in sprint-trained rats (Hilty, Groth, Moore & Musch, 1989), suggesting that a similar adaptation may occur in humans. Muscle capillarization and mitochondrial volume density each affect O_2 extraction, but to our knowledge no longitudinal studies have investigated the effects of sprint training on these variables in human muscle. Increased capillarization has been reported in gastrocnemius muscle of sprint and middle distance runners (Olesen, Raabo, Bangsbo & Secher, 1994). It seems reasonable that acute elevations in oxidative metabolism during repeated maximal sprint bouts would stimulate mitochondrial adaptation. However, equivocal findings have been reported after sprint training on changes in the activities of human skeletal muscle mitochondrial enzymes, such as succinate dehydrogenase and citrate synthase (Saltin *et al.* 1976; Sharp *et al.* 1986; Jacobs, Esbjörnsson, Sylven, Holm & Jansson, 1987), with muscle myoglobin (and therefore most probably total O_2 stores) being unchanged (Jacobs *et al.* 1987). Further studies are clearly required to investigate the effects of intense training on oxidative peripheral adaptations and O_2 extraction during intense exercise.

Pulmonary $\dot{V}_{O_2}$ and skeletal muscle O_2 extraction during sprint exercise. The large increase in $\dot{V}_{O_2}$ during sprint exercise is consistent with other reports (Hermansen & Medbø, 1984; Kowalchuk *et al.* 1988*a*; Medbø & Tabata, 1989; Withers *et al.* 1991; Bogdanis *et al.* 1996). In all our subjects $\dot{V}_{O_2}$ continued to increase following cessation of exercise, reaching a peak at ~8–15 s post-exercise (Fig. 3). This is probably due to the circulatory lag in the return of muscle venous blood to the lung, previously estimated at ~6–15 s (Krogh & Lindhard, 1913; Medbø & Tabata, 1989), as well as the averaging of breaths over a 15 s interval. The decline in femoral venous O_2 content evident during the final 10 s of sprint exercise was no longer present 1 min post-exercise, reflecting not only a rapid decline in O_2 extraction, but also sustained post-exercise hyperaemia; it is likely that O_2 consumption by the active musculature remained high following exercise.

After training, the peak $\dot{V}_{O_2}$ was further delayed and tended to be higher (by 8%, n.s.), suggesting that aerobic metabolism during sprint exercise was increased after training. This conclusion contrasts with a previous finding that sprint training did not improve $\dot{V}_{O_2}$ during 30 s sprint exercise (Nevill *et al.* 1989), a discrepancy which can be explained by methodological differences. In the study by Nevill *et al.* (1989), expired gas was collected during the sprint bout and therefore may have failed to measure the peak $\dot{V}_{O_2}$ in the immediate post-exercise period, so any possible training effect may have been missed. This lag effect in $\dot{V}_{O_2}$ would be less the longer the sprint duration, thereby also accounting for the unchanged $\dot{V}_{O_2}$ reported with training during the first 30 s of sprint exercise, but greater $\dot{V}_{O_2}$ at 45 s exercise (Cunningham & Faulkner, 1969). An increase of 8% in O_2 consumption during sprint exercise represents a substantial energetic advantage after training, consistent with our findings of lower fatigue index and greater work production after training.

The reasons that we were unable to detect an increased $\dot{V}_{O_2}$ *during* sprint exercise may be partly due to a lag in return of deoxygenated blood to the lungs, the variable individual respiratory responses to sprint exercise, the low subject numbers and our inability to measure muscle blood flow. Insufficient sensitivity of our methods should not have been a problem; the mean differences during sprint exercise after training in $\dot{V}_E$, $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ were 12·9 l min^{-1}, 75 ml min^{-1} and 74 ml min^{-1}, respectively, compared with the previously validated 95% confidence limits of 0·9 l min^{-1}, 35 ml min^{-1} and 42 ml min^{-1}, respectively (Kowalchuk *et al.* 1988*a*).

Despite evidence that $\dot{V}_{O_2}$ during sprint exercise was increased after training, the arterio-femoral venous O_2 content difference during and following exercise was unchanged by training. Interpreted alone, this might suggest that leg muscle O_2 uptake was unchanged by training, but such a conclusion conflicts with the elevated peak and recovery pulmonary $\dot{V}_{O_2}$ after training. This conflict would be resolved by a small increase in muscle blood flow during maximal exercise after training, which could generate a substantial increase in muscle $\dot{V}_{O_2}$. We were unable to test this possibility, since no known method for measuring muscle blood flow in humans is adequate for the high-intensity, non-steady-state exercise conditions used in this experiment. Whilst Laughlin, Korthuis, Sexton & Armstrong (1988) and

Musch, Terrell & Hilty (1991) showed increases in hindlimb blood flow and vascular capacity in sprint-trained rats, hindlimb blood flow during maximal exercise was not increased after training in a small group of rats (Musch *et al.* 1991). However, the most likely interpretation of our data is that increased pulmonary $\dot{V}_{O_2}$ was associated with increased muscle perfusion after training. The 12% increase in post-exercise $\dot{V}_{O_2}$ after training is consistent with previous reports of increased 'oxygen debt' after sprint training (Cunningham & Faulkner, 1969; Nevill *et al.* 1989). Of this increase, approximately 30% is probably explained by an increase in muscle temperature caused by the greater work output, and 25% by increased total Na^+,K^+-ATPase activity (McKenna, Schmidt, Hargreaves, Cameron, Skinner & Kjeldsen, 1993).

Enhanced muscle and pulmonary CO_2 output after training

The increase in pulmonary $\dot{V}_{CO_2}$ with sprint exercise and the sustained elevated post-exercise $\dot{V}_{CO_2}$ are consistent with our earlier study (Kowalchuk *et al.* 1988*b*). After sprint training, pulmonary CO_2 output was increased by 8%, with most of this additional CO_2 output occurring post-exercise. The $\dot{V}_{CO_2}$ during sprint exercise was unchanged, whilst the peak $\dot{V}_{CO_2}$, which occurred ~20–25 s post-exercise, tended to be higher after training (by 7%, n.s.); the subsequent $\dot{V}_{CO_2}$ was clearly elevated after training. The pulmonary RER exceeded 1·2 during the 30 s of exercise, indicating excess total CO_2 output relative to aerobic CO_2 production of 0·57 and 0·42 l min^{-1} before and after training, respectively. This discrepancy increased further to ~1 l min^{-1} during recovery, with RER peaking at 2·36 and 2·10 before and after training, respectively. The source of the greater $\dot{V}_{CO_2}$ after training may include increased CO_2 release from active skeletal muscle or from other inactive tissues, or greater hyperventilation and depletion of body CO_2 reserves after training. The total increase in $\dot{V}_{CO_2}$ after training was 1·1 l min^{-1}, almost identical to the greater $\dot{V}_{O_2}$, suggesting that the source of the additional CO_2 released after training was most probably related to increased aerobic metabolism. However, part of the increased $\dot{V}_{CO_2}$ after training must also have been due to a lowering of total body CO_2 content, consistent with the higher $\dot{V}_E$ and with the lower arterial P_{CO_2} and $[HCO_3^-]$ described in the preceding paper (McKenna *et al.* 1997).

Femoral venous P_{CO_2} and CO_2 content increased dramatically with maximal cycling exercise, consistent with earlier reports (Kowalchuk *et al.* 1988*b*; McKelvie, Lindinger, Heigenhauser & Jones, 1991; Lindinger *et al.* 1992). The C_{a-v,CO_2} remained substantially elevated for several minutes post-exercise, such that the leg RER peaked at over 3·0, indicating continuing CO_2 release from muscle following cessation of exercise, in excess of aerobic CO_2 production. After training, the arterio-femoral venous CO_2 content differences during and following exercise were unchanged, despite a significantly greater work production, probably due to increases in muscle blood flow as discussed above. A possible source of CO_2 during recovery from intense exercise is the uptake and oxidation of lactate by muscle that had not taken part in the exercise. However, the large CO_2 release from inactive skeletal muscle with exercise (Kowalchuk *et al.* 1988*a*) was probably unchanged after training, since the arterio-antecubital venous CO_2 content difference during and following leg exercise was unchanged after sprint training (M. J. McKenna, G. J. F. Heigenhauser, R. S. McKelvie & N. L. Jones, unpublished observations).

Effects of training on muscle glycolysis

One of the most commonly reported changes after sprint training is a greater rise in arm venous blood or plasma $[Lac^-]$ following heavy exercise (Saltin *et al.* 1976; Sharp *et al.* 1986; Jacobs *et al.* 1987; Nevill *et al.* 1989; Stathis *et al.* 1994). In contrast to an increase in 30 s maximal work output, no changes were found after sprint training in the present study, in the arterial or femoral venous blood $[Lac^-]$, or the arterio-femoral venous blood $[Lac^-]$ differences. Further, neither the femoral venous plasma $[Lac^-]$, nor the arterio-femoral venous plasma $[Lac^-]$ differences were altered after training, with the only change in circulating $[Lac^-]$ being a 1 mmol l^{-1} greater arterial plasma $[Lac^-]$, reported in the accompanying paper (McKenna *et al.* 1997). It is therefore unlikely that muscle glycolysis was substantially greater after sprint training in the present study. In the studies cited above neither arterial plasma nor blood $[Lac^-]$ was measured and it is possible that contracting arm muscles may have contributed to the increase in venous $[Lac^-]$ in these studies. Consistent with this, when subjects minimized contraction of the forearm muscles during maximal leg exercise, no changes in antecubital venous $[Lac^-]$ were found after sprint training (Snow, McKenna, Carey & Hargreaves, 1992; M. J. McKenna, G. J. F. Heigenhauser, R. S. McKelvie, J. D. MacDougall & N. L. Jones, unpublished observations); when the forearm muscles were freely contracted during exercise, antecubital venous plasma $[Lac^-]$ was increased after sprint training, despite unchanged muscle Lac^- content at fatigue (Stathis *et al.* 1994). However, it is likely that muscle lactate release during maximal exercise was increased after training in view of the probable increase in maximal muscle perfusion after training. A greater muscle Lac^- release after sprint training is consistent with findings of increased muscle lactate transport capacity in sprint-trained muscle in rats and in humans, probably reflecting an increased content of monocarboxylic transport proteins (Pilegaard, Juel & Wibrand, 1993; Pilegaard, Bangsbo, Richter & Juel, 1994).

Enhanced ventilation after training

The elevated $\dot{V}_E$ with sprint exercise after training has important energetic and acid–base advantages, helping to minimize muscular fatigue through increases in O_2 intake and constraining large increases in arterial $[H^+]$ through reductions in arterial P_{CO_2}. Potential factors responsible for the greater $\dot{V}_E$ after sprint training might include increased contributions from neural and/or humoral respiratory control mechanisms (Waldrop, Eldridge, Iwamoto & Mitchell, 1996; Kaufman & Forster, 1996). Increased neural contributions

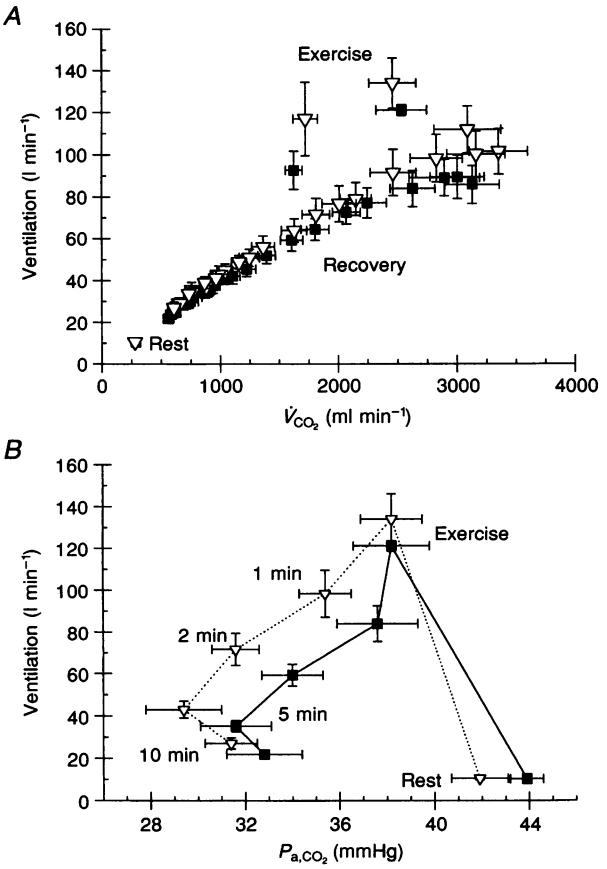

Figure 8. Relationships between pulmonary ventilation and CO_2 variables

Pulmonary ventilation plotted against pulmonary $\dot{V}_{CO_2}$ (*A*) and arterial P_{CO_2} (*B*) at rest, during maximal exercise and during recovery from maximal exercise, before (■) and after (▽) sprint training. Sample size and times for ventilation as in Fig. 2; $n = 6$ for P_{a,CO_2} except during exercise, where $n = 4$.

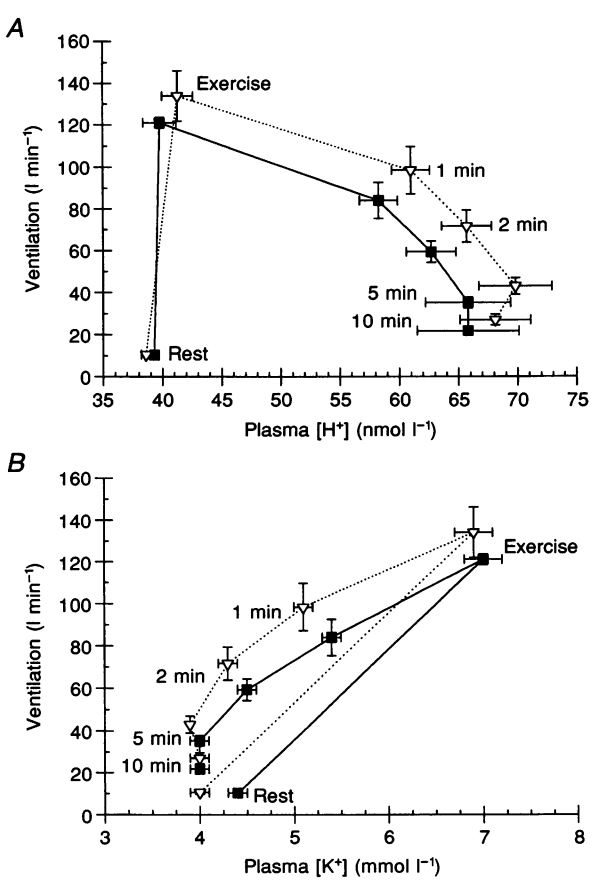

Figure 9. Relationships between pulmonary ventilation and arterial ion concentrations

Pulmonary ventilation plotted against arterial plasma $[H^+]$ (*A*) and arterial plasma $[K^+]$ (*B*) at rest, during maximal exercise and during recovery from maximal exercise, before (■) and after (▽) sprint training. Sample size and times for ventilation as in Fig. 2; $n = 6$ for $[H^+]$ and $[K^+]$ except during exercise, where $n = 4$.

may result from greater central command, potentiation of medullary neuronal output, or increased feedback via skeletal muscle group III and IV afferent nerves. Increased humoral contributions may result from greater CO_2 production and resultant $\dot{V}_{CO_2}$, or increased arterial plasma $[H^+]$ or $[K^+]$. Of these factors, it is possible to analyse the importance of CO_2 output and arterial plasma $[H^+]$, $[K^+]$ and P_{CO_2}, since these variables were also measured during maximal 30 s exercise in these subjects (McKenna *et al.* 1997). The relationships between $\dot{V}_E$ and these variables are presented in Figs 8 and 9.

As might be expected, a close relationship existed between $\dot{V}_{CO_2}$ and $\dot{V}_E$ before and after training, with the exception of the exercise data points (see below), suggesting that the augmented $\dot{V}_E$ after training might be primarily linked with the greater $\dot{V}_{CO_2}$ after training (Fig. 8*A*). The exact mechanism linking CO_2 production and $\dot{V}_E$ during exercise remains obscure. However, additional factors must also contribute to the higher $\dot{V}_E$ after training, since the post-training $\dot{V}_E$ is greater relative to $\dot{V}_{CO_2}$, particularly at high values of $\dot{V}_{CO_2}$. Furthermore, this increased ventilatory response occurred despite a significantly lower P_{a,CO_2} after training, which might be expected to inhibit $\dot{V}_E$. Thus, the post-training $\dot{V}_E$ was greater at a given P_{a,CO_2} (Fig. 8*B*). A likely factor contributing to the increased $\dot{V}_E$ after training is the significant increase in arterial plasma $[H^+]$ after training, although $\dot{V}_E$ after training appeared greater at any given $[H^+]$ (Fig. 9*A*). It is noteworthy that two data points for the $\dot{V}_E$ *vs.* $\dot{V}_{CO_2}$ relationship clearly differ from the remainder of the data; these are the data collected during exercise. This greater hyperventilation relative to $\dot{V}_{CO_2}$ occurred at a time when arterial $[H^+]$ was unchanged from rest and arterial P_{CO_2} was reduced, but it coincided with a large increase in arterial plasma $[K^+]$, which peaked at 7 mmol l^{-1} at the end of 30 s maximal exercise (McKenna *et al.* 1997). The exercise-induced hyperkalaemia may contribute to the rapid increase in $\dot{V}_E$ with heavy exercise via stimulation of carotid body chemoreceptors; additionally, elevated interstitial $[K^+]$ may stimulate muscle chemoreceptors associated with group III and IV muscle afferent nerves (Paterson, 1992). Despite this, the augmented post-training $\dot{V}_E$ was not mediated through elevated plasma $[K^+]$, since arterial plasma $[K^+]$ was reduced after training; this is further indicated by the greater $\dot{V}_E$ after training at a given $[K^+]$ (Fig. 9*B*). Although increased carotid chemoreceptor sensitivity to K^+ or H^+ might explain these findings, it seems more plausible that the increased $\dot{V}_E$ after training may result from greater neural contributions.

Conclusion

Ventilation and gas exchange during maximal incremental exercise were increased after sprint training. Although we did not detect any change in gas exchange during 30 s maximal exercise, post-exercise ventilation, CO_2 output and O_2 uptake were elevated after training. This strongly suggests that aerobic metabolism and CO_2 production during sprint exercise were enhanced after sprint training. The peak pulmonary $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ occurred following exercise, probably because of a circulatory lag in the return of muscle venous blood to the lung. Enhanced aerobic metabolism is consistent with the increased maximal exercise work output and reduced fatigability found after training, whereas enhanced pulmonary CO_2 excretion is consistent with improved acid–base regulation after sprint training.

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Acknowledgements

This paper is dedicated to the memory of our friend and colleague, Professor John R. Sutton, whom we also acknowledge for assistance with the study and for valuable comments. We thank T. Chypchar, Dr M. Ganagarajah, G. Harpur, Dr G. L. Jones, Dr N. McCartney and J. Otis for their help with the study and Dr S. L. Skinner for valuable comments. This study was supported by the Canadian Medical Research Council, and the Heart and Stroke Foundation of Ontario. G.J.F.H. is a Career Investigator, Heart and Stroke Foundation of Ontario. R.S.McK. is a Career Scientist, Ontario Ministry of Health.

Author's email address

M. J. McKenna: michaelmckenna@vut.edu.au

Received 26 November 1996; accepted 11 March 1997.