Run Sprint Interval Training Improves Aerobic Performance but Not Maximal Cardiac Output

REBECCA E. K. MACPHERSON[1], TOM J. HAZELL[1], T. DYLAN OLVER[1], DON H. PATERSON[2], and PETER W. R. LEMON[1]

[1]Exercise Nutrition Research Laboratory, Faculty of Health Sciences, School of Kinesiology, The University of Western Ontario, London, Ontario, CANADA; and [2]Faculty of Health Sciences, Centre for Activity and Aging, School of Kinesiology, The University of Western Ontario, London, Ontario, CANADA

ABSTRACT

MACPHERSON, R. E. K., T. J. HAZELL, T. D. OLVER, D. H. PATERSON, and P. W. R. LEMON. Run Sprint Interval Training Improves Aerobic Performance but Not Maximal Cardiac Output. *Med. Sci. Sports Exerc.*, Vol. 43, No. 1, pp. 115–122, 2011. Repeated maximal-intensity short-duration exercise (sprint interval training, SIT) can produce muscle adaptations similar to endurance training (ET) despite a much reduced training volume. However, most SIT data use cycling, and little is known about its effects on body composition or maximal cardiac output ($\dot{Q}_{max}$). **Purpose**: The purpose of this study was to assess body composition, 2000-m run time trial, $\dot{V}O_{2max}$, and $\dot{Q}_{max}$ effects of run SIT versus ET. **Methods**: Men and women ($n = 10$ per group; mean ± SD: age = 24 ± 3 yr) trained three times per week for 6 wk with SIT, 30-s all-out run sprints (manually driven treadmill), four to six bouts per session, 4-min recovery per bout, versus ET, 65% $\dot{V}O_{2max}$ for 30 to 60 min·d^{-1}. **Results**: Training improved ($P < 0.05$) body composition, 2000-m run time trial performance, and $\dot{V}O_{2max}$ in both groups. Fat mass decreased 12.4% with SIT (mean ± SEM; 13.7 ± 1.6 to 12.0 ± 1.6 kg) and 5.8% with ET (13.9 ± 1.7 to 13.1 ± 1.6 kg). Lean mass increased 1% in both groups. Time trial performance improved 4.6% with SIT ($-25.6 ± 8.1$ s) and 5.9% with ET ($-31.9 ± 6.3$ s). $\dot{V}O_{2max}$ increased 11.5% with SIT (46.8 ± 1.6 to 52.2 ± 2.0 mL·kg^{-1}·min^{-1}) and 12.5% with ET (44.0 ± 2.0 to 49.5 ± 2.6 mL·kg^{-1}·min^{-1}). None of these improvements differed between groups. In contrast, $\dot{Q}_{max}$ increased by 9.5% with ET only (22.2 ± 2.0 to 24.3 ± 1.6 L·min^{-1}). **Conclusions**: Despite a fraction of the time commitment, run SIT induces similar body composition, $\dot{V}O_{2max}$, and performance adaptations as ET, but with no effect on $\dot{Q}_{max}$. These data suggest that adaptations with ET are of central origin primarily, whereas those with SIT are more peripheral. **Key Words**: INTERVAL TRAINING, STROKE VOLUME, ARTERIAL-MIXED VENOUS OXYGEN DIFFERENCE, CARDIOVASCULAR ADAPTATIONS, MUSCLE ADAPTATIONS, BODY COMPOSITION

It is well known that physical fitness can be improved by either continuous or interval training regimens. Traditional endurance training (ET) includes some of both and requires a significant time commitment (~30–60 min·d^{-1}, several days a week [18]). Recently, sprint interval training (SIT; repeated four to six bouts of 30-s "all-out" exercise, with 4-min rest intervals three times per week over 6 wk) alone has been shown to produce similar muscle and aerobic performance adaptations as ET in less than half the time per session (10,17). This observation is intriguing and suggests that exercise intensity is a more critical aspect of aerobic training adaptations than exercise volume.

Physiological adaptations associated with ET have been studied extensively and include central (cardiac output ($\dot{Q}$)) (6) and/or peripheral (skeletal muscle arterial–venous oxygen difference (a–vO_2 difference)) adaptations (18,22), resulting in a greater maximal oxygen uptake ($\dot{V}O_{2max}$), an improved exercise capacity, and enhanced overall health (17,19). Typically, ET also results in body mass losses and enhanced body composition (35,38).

Interestingly, SIT has proven to improve not only anaerobic (glycolytic) metabolism, as expected, but also aerobic (oxidative) metabolism (7–10,17,24,28,31,33). Further, these metabolic adaptations have also produced improvements in a wide range of performances, including those that use significant aerobic metabolism (i.e., cycling time to exhaustion tests and time trials ranging in distance from 2 to 40 km) (7,17,27). These effects are somewhat surprising considering the small time commitment involved with SIT and suggest that some version of this type of training could likely benefit not only athletes but perhaps even the general adult population who seems to lack the time to exercise.

To date, the optimal type of interval training program (mode, intensity, duration, and recovery) is unclear. In addition, most SIT data have been collected with a cycling protocol that is unfortunate because running is a more

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universal exercise and, as such, can be applied to a far greater range of exercise programs and athletic activities. Moreover, ET training produces significant body fat losses and improvements in cardiovascular (i.e., $\dot{Q}$) and metabolic (i.e., oxygen consumption by muscle) systems (6,18,22). Although SIT has demonstrated significant metabolic changes as well, there is currently no data on the effect of SIT on either body composition or $\dot{Q}$. This information is critical to evaluate whether this seemingly time-efficient training protocol yields body composition and cardiovascular changes like those with traditional ET.

Therefore, the purpose of this study was to determine whether a running SIT protocol would induce similar adaptations as ET and to document any chronic effects on body mass, body composition, resting metabolic rate (RMR), run time trial performance, $\dot{V}O_{2max}$, maximal $\dot{Q}$ ($\dot{Q}_{max}$), and maximal arterial-mixed venous oxygen difference (a–$\bar{v}O_2$ difference). It was hypothesized that running SIT would result in similar aerobic performance adaptations as ET without affecting $\dot{Q}_{max}$.

METHODS

Subjects. Twenty young healthy recreationally active (11 ultimate Frisbee players, 9 university students) men ($n = 12$) and women ($n = 8$) volunteered to participate. None were training systematically before the study. All experimental procedures and potential risks were explained fully to the subjects before any testing, and the subjects provided written, informed consent. In addition, overall health/fitness was assessed via the Physical Activity Readiness Questionnaire (36). On the basis of sex, time trial performance, and $\dot{V}O_{2max}$, all subjects were matched into one of two groups: an ET group or an SIT group (Table 1). Subjects were encouraged to maintain their prestudy physical activity and diet patterns throughout the testing and training period. This study was approved by The University of Western Ontario Ethics Committee for Research on Human Subjects.

Study design. Subjects completed 6 wk of training (three sessions per week) in either the ET or the SIT group. Before and after training, subjects underwent a body composition test, an RMR test, a $\dot{V}O_{2max}$ test, a $\dot{Q}_{max}$ test, and a 2000-m run time trial performance. Body composition

was measured before the RMR test or before the $\dot{V}O_{2max}$ test on a single day. All other tests were separated by 24 h. Subjects refrained from alcohol, caffeine, and physical activity 2 h before all testing and training sessions. Recovery was standardized by allowing 48 h after the final training session before the posttesting. Posttesting was completed in the same order for all subjects (body composition/RMR/$\dot{V}O_{2max}$, performance, and $\dot{Q}_{max}$). Before baseline testing, subjects were familiarized with all testing and training procedures to ensure that any learning effect was minimized.

Baseline tests. All exercise tests were performed on separate days (6 d total for pretesting and 4 d total for posttesting) and were completed at least 2 d before training. Testing consisted of four measures:

1) Body composition. Body composition (lean mass and fat mass) was determined by whole-body densitometry using air displacement via the BodPod® (Life Measurements, Concord, CA). Testing was done in accordance with the manufacturer's instructions as detailed in the manual. Briefly, subjects were tested in approved clothing (i.e., swimsuit or compression shorts, Lycra® cap, and for the women, a sports bra). Thoracic gas volume was estimated for all subjects using the predictive equation integral to the BodPod® software. The obtained value for body density was used in the Siri (34) equation to estimate body composition. We have shown previously that this technique is reliable (no significant difference between repeat trials, $P = 0.935$; $r = 0.992$, $P < 0.001$; coefficient of variation = 0.15%; $n = 980$ duplicate measures [30]).

2) Resting metabolic rate. RMR was measured by indirect calorimetry using an online breath-by-breath gas collection system (Vmax Legacy; Sensor Medics, Yorba Linda, CA) after a 12-h overnight fast. The system was calibrated before testing using gases of known concentration and flow with a 3-L syringe. Subjects limited their activity getting to the laboratory (drove or took the bus), used the elevator upon arrival in the building, and rested supine for 30 min in an environmental chamber (21°C). All measurements were performed between 0700 and 0900 h. A Hans Rudolph silicon face mask (8940 Series; Hans Rudolph, Inc., Kansas, MO) was positioned over the subject's nose and mouth and after checking for leaks used to collect expired gases for analysis. The subjects were instructed to remain motionless and to avoid falling asleep during the test. The average of the last 15 min of collection was used to estimate the fasting energy expenditure using the energy equivalent of oxygen from a table of nonprotein respiratory quotient (32). Protein contribution was assumed to be similar across conditions.

3) $\dot{V}O_{2max}$ test. Subjects performed a 5-min warm-up on the treadmill (Desmo Pro; Woodway®, Waukesha, WI) at 5 mph (8 kph) for the women or 6 mph (9.7 kph) for the men. After the warm-up, subjects performed a

TABLE 1. Subject characteristics before and after 6 wk of either SIT or ET.

Subject Characteristics	SIT ($n = 10$)		ET ($n = 10$)	
	Pretraining	Posttraining	Pretraining	Posttraining
Age (yr)	24.3 ± 3.3	24.3 ± 3.3	22.8 ± 3.1	22.8 ± 3.1
Height (cm)	173.7 ± 9.2	173.7 ± 9.2	168.6 ± 6.8	168.6 ± 6.8
Body mass (kg)	76.0 ± 15.0	74.9 ± 13.6	68.8 ± 9.5	68.5 ± 9.1
% fat	18.4 ± 6.2	16.6 ± 7.3*	20.8 ± 9.7	19.7 ± 9.1*
Fat mass (kg)	13.7 ± 4.9	12.0 ± 4.9*	13.9 ± 5.5	13.1 ± 5.0*
Lean mass (kg)	62.3 ± 14.5	62.9 ± 14.9*	54.9 ± 12.2	55.5 ± 12.0*
$\dot{V}O_{2max}$ (mL·kg^{-1}·min^{-1})	46.8 ± 5.1	52.2 ± 6.5*	44.0 ± 5.1	49.5 ± 8.3*

Values are presented as mean ± SD. All between-group values were similar ($P > 0.05$) pretraining. Values are presented as mean ± SEM.
* Significantly different from pretraining value ($P < 0.05$); none of the observed gains were significant between groups.
SIT, sprint interval training; ET, endurance training.

progressive, continuous incremental speed treadmill test to determine $\dot{V}O_{2max}$ and run velocity max. Subjects started at 5.5 mph (8.9 kph) (women) or 6.5 mph (10.5 kph) (men), and the speed was increased by 0.5 mph (0.8 kph) every minute until volitional exhaustion (duration of the test was between 6 and 12 min). Oxygen consumption was collected continuously and analyzed using an online breath-by-breath gas collection system (Vmax Legacy, Sensor Medics), calibrated as described above. Heart rate was recorded throughout the test using a Polar HR monitor (RS200sd®; Polar Electro, Inc., Quebec, Canada). The greatest 30-s average was taken as the $\dot{V}O_{2max}$.

4) Maximal cardiac output. The test for $\dot{Q}_{max}$ was conducted in a separate laboratory with a different measurement apparatus. Subjects performed practice trials of the $\dot{Q}$ procedure at rest and then at near-maximal exercise. The test involved continuous treadmill running (Model TM310, Trackmaster; Jas Fitness System, Newton, KS) at 90% of the speed that elicited $\dot{V}O_{2max}$ until they could not continue. The duration of the exercise test was between 4 and 7 min. Oxygen consumption (see below) and HR (RS200sd®; Polar Electro, Inc.) were measured throughout the test to ensure that the $\dot{Q}$ measure was attained at or near $\dot{V}O_{2max}$. Gas exchange was measured as described previously (1). Briefly, inspired and expired flow rates were measured using a low dead space (90 mL) bidirectional turbine (VMM-110; Alpha Technologies, Laguna Beach, CA), which was calibrated before each testing session with a syringe of known volume. Inspired and expired gases were sampled continuously (every 20 ms) at the mouth and analyzed for concentrations of O_2, CO_2, and N_2 by mass spectrometry (1100 Spectrometer; Perkin-Elmer, Ontario, Canada) after calibration with precision-analyzed gas mixtures. Changes in gas concentrations were aligned with gas volumes by measuring the time delay for a square-wave bolus of gas passing the turbine to the resulting changes in fractional gas concentrations as measured by the mass spectrometer. Breath-by-breath alveolar gas exchange was calculated by using algorithms of Beaver et al. (4).

An acetylene (C_2H_2) non-rebreathing technique (breathing a C_2H_2 gas mixture in an open circuit system) was used to measure $\dot{Q}_{max}$ (5,25,39). This method provides reproducible $\dot{Q}$ measurements during submaximal to maximal exercise (5,25,29,39). Moreover, when measuring $\dot{Q}$ during maximal exercise, this technique can be better than the direct

Fick method because of the inherent risks involved and an increasing inaccuracy in later stages of vigorous exercise (39). The subjects were connected to a one-way valve assembly with a two-way valve on the inspired side. One input was room air and the other a gas mixture containing known concentrations of acetylene ((C_2H_2) (0.698%), helium (He) (8.99%), O_2 (21%), and N_2 (BAL)). A plastic bag (Douglas bag; Hans Rudolph, Inc.) was connected to the tank containing the gas mixture and to the valves at the mouth piece. The bag was emptied before each testing session to prevent contamination of inspired gas concentrations and gas dissociation due to density and then filled with the acetylene mixture. When the subjects indicated that they could not run any longer than another 30 s, HR_{max} was recorded, and the $\dot{Q}_{max}$ measure was initiated by turning the valve (at the end of expiration) so that the subjects inspired the acetylene gas mixture. Subjects were encouraged to maintain a constant respiratory rhythm and to avoid coughing, swallowing, and partial breaths. Concentrations of C_2H_2 and He were then measured continuously for 10 breaths by the mass spectrometer. Digitized signals were interpreted by a commercially available software program (BIPS; Beck Integrated Physiological Testing System, 2008). At the end of the sampling time, the input valve was switched back to room air, and the subject was allowed to stop running. Computer analysis of the rate of disappearance of acetylene (the slope between the first and the last breath of the maneuver) gives an indication of the how fast blood is circulating, and $\dot{Q}$ was calculated. Data for the ET group reflect $n = 9$ because of technical difficulties in the $\dot{Q}$ measurement for one subject.

5) Maximal stroke volume (SV) and arterial-mixed venous O_2 difference. Maximal SV and arterial-mixed venous O_2 (a–$\bar{v}O_2$) difference were calculated in accordance with a derivation of the Fick equation, where whole-body $\dot{V}O_2$ is the product of $\dot{Q}$ and the a–$\bar{v}O_2$ difference. Values for HR_{max} and $\dot{V}O_2$ from the $\dot{Q}_{max}$ test were used.

6) Performance test (2000-m run time trial). Subjects were instructed to complete 2000-m self-paced run time trials on a 200-m indoor track as quickly as possible without feedback. Exercise time was recorded. Before the study, subjects completed three run time trials on separate occasions to minimize any learning effect. The mean of the two best time trials was recorded as the baseline test.

Training. Training commenced ~48 h after the last baseline measure and consisted of three training sessions per

TABLE 2. Summary of training protocols.

Group	Protocol	Training Intensity	Total Exercise Time (h·6 wk⁻¹)	Total Rest Time (h·6 wk⁻¹)	Total Time (h·6 wk⁻¹)	Distance Covered (km·6 wk⁻¹)
SIT ($n = 10$)	30 s × 4–6, 4 min rest (3 wk⁻¹)	Maximal effort	0.75	6	6.75	10.3
ET ($n = 10$)	30–60 min (3 wk⁻¹)	65% $\dot{V}O_{2max}$	13.5	0	13.5	36.4

SIT, sprint interval training; ET, endurance training.

week over 6 wk with 1 to 2 d of recovery between sessions. Each training session was monitored in the laboratory.

Endurance group. The ET group performed 30 to 60 min of continuous running at a speed designed to elicit 65% $\dot{V}O_{2max}$ on a treadmill (Desmo Pro; Woodway®). The training progressed from 30 min in weeks 1 and 2 to 45 min during weeks 3 and 4 and 60 min during weeks 5 and 6 (Table 2).

Sprint interval group. Training for the sprint interval group consisted of repeated, 30-s maximal running efforts with 4 min of recovery (active recovery was encouraged). Each sprint was completed on a treadmill (Desmo Pro; Woodway®) set in dynamic mode so that the subject was the power source for the running belt. The number of bouts increased from four (first 2 wk) to five (weeks 3 and 4) and to six (last 2 wk of training) (Table 2). The treadmill was interfaced with a computer to allow recording of speed and HR data during each bout using MED-PRO software (Personal Trainer version 4.4; Woodway®; Fig. 1A).

Posttraining procedures. Posttraining measures were identical with the baseline testing and commenced at least 2–4 d after the final training session.

Statistical analyses. Statistical analyses were performed using Sigma Stat for Windows (version 3.5, Systat Software, Inc., Point Richmond, CA). After testing for normality and variance homogeneity, two-way (treatment × time) repeated-measures ANOVA were used to test significance between groups pretraining and posttraining, with Tukey *post hoc* testing, where necessary. The significance level was set at $P < 0.05$. All results are presented as means ± SEM.

RESULTS

Body composition. There was no significant difference in body mass pretraining or posttraining within or between groups. Interestingly, fat mass decreased significantly by 12.4% and 5.8% in the sprint and endurance groups, respectively, with no significant difference between groups (Table 1). The decreases ($P = 0.002$) in fat mass in the SIT group were due to changes in the men and not the women (men: SIT, pretraining = 13.7 ± 2.4 kg, posttraining = 10.7 ± 2.3 kg; women: SIT, pretraining = 13.7 ± 1.7 kg, posttraining = 14.0 ± 1.1 kg), whereas the decreases ($P = 0.002$) in the ET group occurred in both genders (men: ET, pretraining = 10.7 ± 1.1 kg, posttraining = 10.2 ± 1.0 kg; women: ET, pretraining = 18.7 ± 2.6 kg, posttraining = 17.3 ± 2.5 kg). Moreover, lean mass increased ($P = 0.037$) by 1.0% after training in both groups, with no significant difference between groups.

Resting metabolic rate. There was no difference ($P = 0.479$) in RMR pretraining or posttraining within or between training groups (SIT: pretraining = 6145 ± 473 kJ·d^{-1}, posttraining = 5669 ± 381 kJ·d^{-1}; ET: pretraining = 5686 ± 473 kJ·d^{-1}, posttraining = 5500 ± 314 kJ·d^{-1}).

2000-m run time trial. The 2000-m run performance was similar for both groups pretraining (~9 min). Time trial
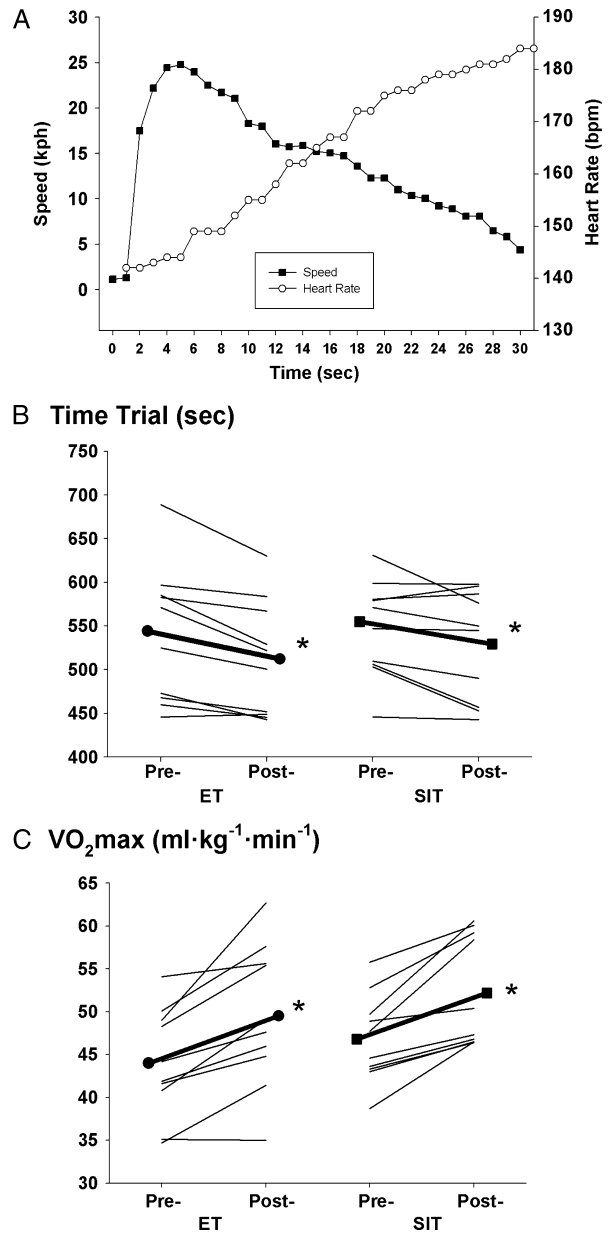
FIGURE 1—A, Speed (kph) and heart rate (bpm) over one representative 30-s maximal running bout on the manually driven treadmill. Speed (●) and heart rate (○). B, The 2000-m run time trial (s) before and after 6 wk of either SIT or ET, *Pre–post $P < 0.017$. C, $\dot{V}O_{2max}$ (mL·kg^{-1}·min^{-1}) before and after 6 wk of either SIT or ET, *Pre–post $P < 0.001$. Thick lines are means for 10 subjects in each group (thin lines are individual subjects). None of the observed gains were significantly different between groups. SIT, sprint interval training; ET, endurance training.

performance improved ($P < 0.017$) by 4.6% (−25.6 s) and 5.9% (−31.9 s) in the SIT and ET groups, respectively (Fig. 1B). These increases were not significantly different between groups.

Anaerobic performance. Top speed (peak power) achieved during the 30-s efforts increased ($P = 0.002$) by 5.1% or 0.6 mph (1 kph) from the first training session to

the last; however, there was no difference ($P = 0.455$) in average speed during the 30-s effort over the 6 wk of training (pretraining = 9.9 mph (16.0 kph); posttraining = 9.0 mph (15.6 kph)).

$\dot{V}O_{2max}$. The majority of our subjects reached a $\dot{V}O_2$ plateau toward the end of the test (29 of 40 (73%)). Seven

A **Maximal cardiac output (L·min^{-1})**

B **Maximal stroke volume (ml·beat^{-1})**

C **Maximal arterial-mixed venous O$_2$ difference (ml O$_2$·100ml^{-1} blood)**

FIGURE 2—A, Maximal cardiac output (L·min^{-1}) before and after 6 wk of either SIT or ET, *Pre–post $P = 0.01$. B, Maximal SV (mL per beat) before and after 6 wk of either SIT or ET, *Pre–post $P = 0.076$. C, Maximal a–$\bar{v}O_2$ difference (mL O$_2$·100 mL^{-1} blood) before and after 6 wk of either SIT or ET, †interaction effect $P = 0.045$. Thick lines are means for 9 subjects in the ET group and 10 subjects in the SIT group (thin lines are individual subjects). None of the observed gains were significant between groups. SIT, sprint interval training; ET, endurance training.

of the remaining 11 had an RER ≥ 1.1 or a peak HR within 10 bpm of their age-predicted maximum. Of the remaining four, HR at the end of the maximal test was within 11, 14, 15, and 21 bpm of their age-predicted maximum. In all four of these cases recorded, HR_{max} was consistent before and after training (within 1 bpm), suggesting that their estimated HR_{max} may not be accurate.
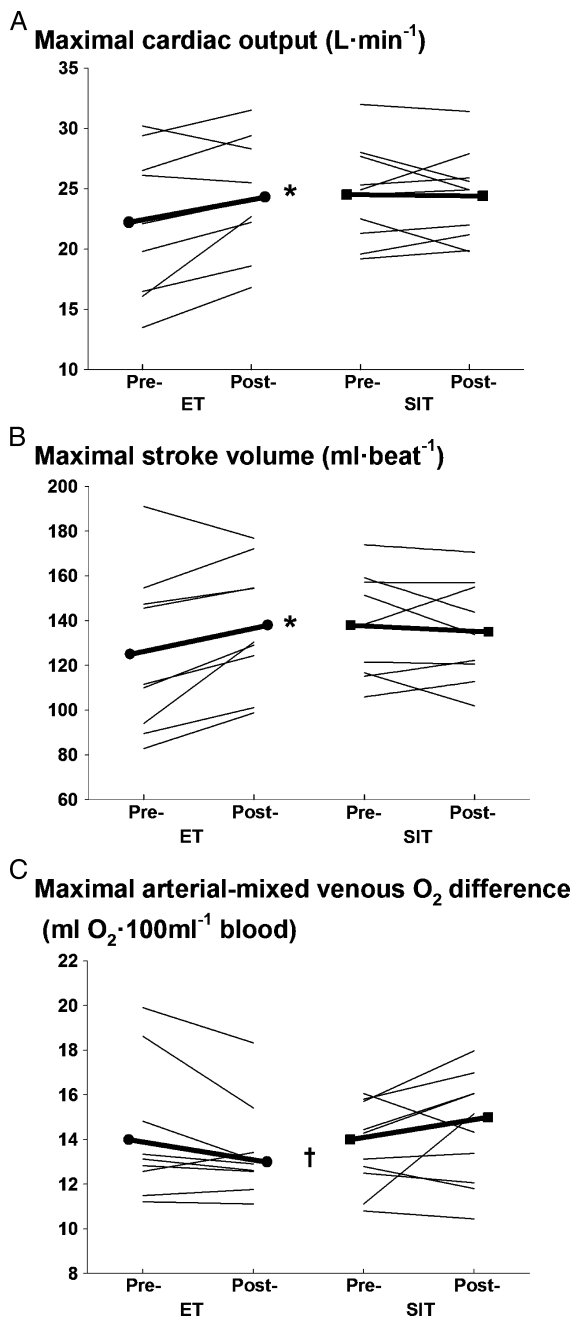
Both SIT and ET improved ($P < 0.001$) $\dot{V}O_{2max}$ by 11.5% (46.8 ± 1.6 to 52.2 ± 2.0 mL·kg^{-1}·min^{-1}) and 12.5% (44.0 ± 2.0 to 49.5 ± 2.6 mL·kg^{-1}·min^{-1}), respectively (Fig. 1C). The between-group differences in $\dot{V}O_{2max}$ were not significant. Neither training group showed a change in running economy after training (SIT: pre-submaximal, $\dot{V}O_2 = 2.46 \pm 0.14$ L·min^{-1}; post-submaximal, $\dot{V}O_2 = 2.62 \pm 0.18$ L·min^{-1}; ET: pre-submaximal, $\dot{V}O_2 = 2.18 \pm 0.17$ L·min^{-1}; post-submaximal, $\dot{V}O_2 = 2.26 \pm 0.13$ L·min^{-1}; $P = 0.523$).

Maximal cardiac output, SV, and a-$\bar{v}O_2$ difference. $\dot{Q}_{max}$ increased ($P = 0.01$) with ET by 9.5% or 2.1 L·min^{-1} (22.2 ± 2.0 to 24.3 ± 1.6 L·min^{-1}) but not after SIT (24.5 ± 1.2 to 24.4 ± 1.2 L·min^{-1}) (Fig. 2A). HR_{max} during the $\dot{Q}_{max}$ testing was similar pretraining and posttraining and between groups (SIT: pretraining = 178 ± 4 bpm, posttraining = 180 ± 3 bpm; ET: pretraining = 178 ± 4 bpm, posttraining = 177 ± 3 bpm) albeit at an HR approximately 10 bpm lower than observed on the $\dot{V}O_{2max}$ test. Measured $\dot{V}O_2$ (L·min^{-1}) during the constant load $\dot{Q}_{max}$ test also increased with training (SIT: pretraining = 3.48 ± 0.27 L·min^{-1}, posttraining = 3.66 ± 0.27 L·min^{-1}; ET: pretraining = 3.08 ± 0.23 L·min^{-1}, posttraining = 3.26 ± 0.23 L·min^{-1}) but was slightly lower (although not significantly different) compared with that attained during the incremental speed test in the performance laboratory. Thus, the $\dot{Q}$ measures were made at exercise intensities very close to $\dot{V}O_{2max}$ and at a consistent relative intensity pretraining and posttraining for both training groups (SIT: pretraining = 94% $\dot{V}O_{2max}$, posttraining = 90% $\dot{V}O_{2max}$; ET: pretraining = 99% $\dot{V}O_{2max}$, posttraining = 94% $\dot{V}O_{2max}$). The increase in $\dot{Q}$ with ET posttraining was via a 10.4% or 13 mL per beat increase ($P = 0.076$) in SV (125 ± 11 to 138 ± 9 mL per beat). In contrast, there was no change in SV with SIT (138 ± 7 to 135 ± 7 mL per beat) (Fig. 2B). Further, there was a significant ($P < 0.045$) interaction effect (group by time) between groups for a–$\bar{v}O_2$ difference (Fig. 2C); the SIT increased a–$\bar{v}O_2$ difference by 7.1%, and in the ET a–$\bar{v}O_2$ difference decreased by 7.1%.

Compliance with exercise training. All subjects completed each of the exercise training sessions, with the exception of one subject from each group (total of five missed sessions due to injury; for details, see Discussion section).

DISCUSSION

The present study is the first to examine the effects of chronic (6 wk) SIT on central ($\dot{Q}_{max}$) and peripheral (a–$\bar{v}O_2$ difference) oxygen transport/uptake adaptations as well the

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effects on body composition. Further, the majority of SIT studies to date have used cycling as the training regimen, whereas this study evaluated a running SIT protocol. Our results indicate that although 6 wk of run SIT resulted in similar body composition, performance, and $\dot{V}O_{2max}$ improvements as ET, SIT had no effect on $\dot{Q}_{max}$.

Effect on body composition. Interestingly, the effect of the running SIT protocol on body composition was similar (or perhaps greater) than that of ET. The SIT group lost 1.7 kg of body fat (12.4% of total fat mass), whereas the ET group lost 0.8 kg (5.8%). These densitometric-based observations extend the observations of several previous interval training studies that have reported a loss in fat mass after training with less intense but longer interval duration using less direct body composition measures (37,38). Further, there may be a gender training mode interaction because the women in our study did not lose fat mass with SIT but lost 1.4 kg with ET. However, a definitive conclusion on this possibility must await further study because we had only four women per group. Moreover, although not significantly different, the initial body fat values for the ET women were slightly greater (ET = 30.6% vs SIT = 22.3%). Both training groups also gained a significant amount of lean mass (SIT = 0.6 kg, ET = 0.6 kg). Finally, the observed similar over all body fat losses between training groups are remarkable given that the total 6-wk exercise time was $18\times$ longer for ET (13.5 vs 0.75 h).

Although the observed effects of ET on body composition are likely due to the energy expenditure during the training session primarily, such may not be the case with SIT. It is known that intense interval exercise causes a greater negative postexercise energy balance or increased postexercise oxygen consumption (EPOC) versus ET, and this could promote a larger lipid and overall energy deficit with SIT (2,26,38). Moreover, EPOC is influenced more by exercise intensity than duration and has been correlated with the amount of lactic acid produced during exercise (2,26). The maximal efforts during SIT in the present study would certainly accumulate high quantities of lactic acid (8,20), likely leading to an increased EPOC. Moreover, intense interval training increases the concentration and activities of enzymes and proteins involved in beta oxidation (7,31,33,38) as well as the transport of fat into the skeletal muscle cell and mitochondria (35). Consequently, chronic fat use could be altered with SIT and may have contributed to the observed body fat losses. Regardless, as mentioned, further studies are required to explain the mechanisms behind the fat loss that occurred in the present study with SIT.

Effect on 2000-m run time trial. The similar between-group time trial performance gains (here 5%–6%) are consistent with previous cycling SIT to ET comparisons showing that run SIT also results in comparable endurance performance improvements despite a much smaller training volume (10,17). Specifically, aerobic performances ranging from 2- to 40-km cycling time trials (~4- to 80-min efforts) have been reported to improve by 3.5% to 10.1% with SIT

(8,17). This ability to perform better especially on longer time trials after SIT could be a direct result of a variety of factors, including reduced carbohydrate use (i.e., glycogen sparing), decreased lactate accumulation, and/or increased muscle buffering (7,17,20,21). Although improvements in running economy could also contribute to the observed improvement in time trial performance, this is unlikely with the present study because neither training mode altered submaximal $\dot{V}O_2$ during the five minute standardized warm-up before the $\dot{V}O_{2max}$ testing.

Effect on $\dot{V}O_{2max}$. The similar improvements in $\dot{V}O_{2max}$ with both training modes are also consistent with previous studies that have compared cycling SIT and ET (10,23), indicating that run SIT, like cycling SIT, presents a significant stimulus to aerobic metabolism despite its short duration.

Effects on the cardiovascular system. Centrally, training improvements in $\dot{V}O_{2max}$ are due to increases in $\dot{Q}_{max}$ via an enhanced SV with little change in HR_{max} (6,11,14,29). Our results were collected at similar, near-maximal exercise workloads pretraining and posttraining (as indicated by HR and $\dot{V}O_2$ measures) and demonstrate a 9.5% increase in $\dot{Q}_{max}$ with ET but no change with SIT. The increased $\dot{Q}_{max}$ with ET was accompanied by a 13 mL-per-beat increase ($P = 0.076$) in maximal SV with no change in HR_{max}. We believe that this observed maximal SV increase is real because power calculations indicate that only three additional subjects would produce significance at the $P < 0.05$ level. The increase in both $\dot{Q}$ and SV in response to ET appears to depend on the prolonged (30–60 min), constant demand on the heart (6,11), with submaximal work rates providing a sufficient intensity stimulus to elicit peak SV throughout the exercise. Although high-intensity interval exercise training has been shown to increase $\dot{Q}_{max}$ (14,15,40), the exercise intensities studied previously were less than maximal and the exercise intervals longer ($8–10\times$) than our 30-s maximal efforts. Apparently, SIT, although much more intense, is of insufficient duration increase $\dot{Q}_{max}$.

Peripherally, training improvements in $\dot{V}O_{2max}$ are due to increases in a–vO_2 difference, which depend on O_2 delivery to active muscle fibers (blood flow distribution, capillary density, and arterial O_2 content), local enzymatic adaptations, and mitochondrial density/volume (6,11,15). Our results demonstrate that the $\dot{V}O_{2max}$ increases with SIT were accompanied by an increased a–$\bar{v}O_2$ difference (7.1%), whereas the a–$\bar{v}O_2$ difference was not increased with ET. This peripheral (muscle) improvement with SIT is consistent with previous studies demonstrating enzymatic (7,17,24,28,31,33) and mitochondrial adaptations (10,17). Further, our significant increase in $\dot{V}O_{2max}$ with SIT indicates that these peripheral adaptations are important functionally, which is not necessarily so because large enzymatic changes can produce only small changes in $\dot{V}O_{2max}$. The stimulus for this apparent greater peripheral adaptation versus ET could be a lower O_2 partial pressure at the muscle because of a reduction in blood flow to the exercising muscle during SIT type exercise (16). Apparently,

the ET stimulus studied was insufficient to induce similar peripheral adaptations (12,13).

Others have reported that low-to-moderate-intensity ET leads to improvements in $\dot{Q}_{max}$ via SV increases, with little or no change in a–$\bar{v}O_2$ difference, and that higher-intensity training leads to increased muscle a–vO_2 difference with no change in $\dot{Q}$ (14,15,40). Although these studies used much lower exercise intensities (~90% of $\dot{V}O_{2max}$ vs maximal SIT) and an interval training model in which the total amount of work was matched with ET, they suggest that achieving a certain total exercise volume is necessary to increase central capacity while higher-intensity exercise targets the periphery. Clearly, our SIT exercise intensity was sufficient to induce $\dot{Q}_{max}$ adaptations, but it appears that the 30-s duration used was too brief. To maximize both central and peripheral training mechanisms, we hypothesize that either longer intervals of SIT (e.g., a typical high-intensity interval training program) or a mix of SIT and ET would be ideal.

Safety. One subject from each group experienced an injury during the 6 wk of training. Both were minor. The subject in the SIT group did not complete his last four training sessions because of a hamstring muscle pull but completed the posttraining measures. The subject in the ET group did not complete his last training session because of knee discomfort but was able to complete the posttraining measures. Both subjects were included in the results. Of course, given the strenuous nature of the SIT, it is possible that cardiovascular or musculoskeletal complications could arise especially in older or less fit individuals. Therefore, it would be wise to be cautious if SIT is implemented widely. Importantly, a modest warm-up (2 min jogging in place) has been shown to eliminate or drastically reduce the incidence of heart arrhythmias with very strenuous exercise (treadmill running at 14.5 kph, 30% grade) (3). Consequently, if a proper warm-up is used, SIT may have the potential to improve both anaerobic and aerobic power quickly and safely.

CONCLUSIONS

Six weeks (three times per week) of SIT or ET produces similar improvements in $\dot{V}O_{2max}$, time trial performance, and body composition. However, the adaptations with ET are of central origin ($\dot{Q}_{max}$) primarily, whereas those with SIT are more peripheral (a–$\bar{v}O_2$ difference). Consequently, endurance athletes should consider incorporating SIT into their training regimes. Further, some version of SIT (assuming adequate warm-up and reduced intensity) may be beneficial for the general population. To maximize benefits of SIT, duration intervals longer than 30 s or some combination of SIT and ET are likely optimal. Future study is necessary to provide details.

REFERENCES

1. Babcock MA, Paterson DH, Cunningham DA, Dickinson JR. Exercise on-transient gas exchange kinetics are slowed as a function of age. *Med Sci Sports Exerc*. 1994;26(4):440–6.
2. Bahr R, Gronnerod O, Sejersted OM. Effect of supramaximal exercise on excess postexercise O₂ consumption. *Med Sci Sports Exerc*. 1992;24(1):66–71.
3. Barnard RJ, Gardner GW, Diaco NV, MacAlpin RN. Cardiovascular responses to sudden strenuous exercise—heart rate, blood pressure, and ECG. *J Appl Physiol*. 1973;34:833–7.
4. Beaver WL, Lamarra N, Wasserman K. Breath-by-breath measurement of true alveolar gas exchange. *J Appl Physiol*. 1981;51:1662–75.
5. Bell C, Monahan KD, Donato AJ, Hunt BE, Seals DR, Beck KC. Use of acetylene breathing to determine cardiac output in young and older adults. *Med Sci Sports Exerc*. 2003;35(1):58–64.
6. Blomqvist CG, Saltin B. Cardiovascular adaptations to physical training. *Annu Rev Physiol*. 1983;45:169–89.
7. Burgomaster KA, Hughes SC, Heigenhauser GJF, Bradwell SN, Gibala MJ. Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *J Appl Physiol*. 2005;98:1985–90.
8. Burgomaster KA, Heigenhauser GJF, Gibala MJ. Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *J Appl Physiol*. 2006;100:2041–7.
9. Burgomaster KA, Cermak NM, Phillips SM, Benton CR, Bonen A, Gibala MJ. Divergent response of metabolite transport proteins in human skeletal muscle after sprint interval training and detraining. *Am J Physiol Regul Integr Comp Physiol*. 2007;292:R1970–6.
10. Burgomaster KA, Howarth KR, Phillips SM, et al. Similar metabolic adaptations during exercise after low volume sprint interval training and traditional endurance training. *J Physiol*. 2008;586:151–60.
11. Clausen JP. Effect of physical training on cardiovascular adjustments to exercise in man. *Physiol Rev*. 1977;57:779–815.
12. Cunningham DA, Hill JS. Effect of training on cardiovascular response to exercise in women. *J Appl Physiol*. 1975;39:891–5.
13. Cunningham DA, McCrimmon D, Vlach LF. Cardiovascular response to interval and continuous training in women. *Eur J Appl Physiol*. 1979;41:187–97.
14. Daussin FN, Ponsot E, Dufour SP, et al. Improvement of $\dot{V}O_{2max}$ by cardiac output and oxygen extraction adaptation during intermittent versus continuous endurance training. *Eur J Appl Physiol*. 2007;101:377–83.
15. Daussin FN, Zoll J, Dufour SP, et al. Effect of interval versus continuous training on cardiorespiratory and mitochondrial functions: relationship to aerobic performance improvements in sedentary. *Am J Physiol Regul Integr Comp Physiol*. 2008;295:R264–72.
16. Essen B. Studies on the regulation of metabolism in human skeletal muscle using intermittent exercise as an experimental model. *Acta Physiol Scand Suppl*. 1978;454.
17. Gibala MJ, Little JP, van Essen M, et al. Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *J Physiol*. 2006;575(5):901–11.

APPLIED SCIENCES

18. Gollnick PD, Saltin B. Significance of skeletal muscle oxidative enzyme enhancement with endurance training. *Clin Physiol*. 1982;2:1–12.

19. Gollnick PD, Armstrong RB, Saltin B, Saubert CW IV, Sembrowich WL, Shepherd RE. Effect of training on enzyme activity and fiber composition of human skeletal muscle. *J Appl Physiol*. 1973;34:107–11.

20. Harmer AR, McKenna MJ, Sutton JR, et al. Skeletal muscle metabolic and ionic adaptations during intense exercise following sprint training in humans. *J Appl Physiol*. 2000;89:1793–803.

21. Hawley JA. Adaptations of skeletal muscle to prolonged, intense endurance training. *Clin Exp Pharmacol Physiol*. 2002;29:218–22.

22. Holloszy JO, Coyle EF. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J Appl Physiol*. 1984;56:831–8.

23. Howarth KR, Burgomaster KA, Phillips SM, Gibala MJ. Exercise training increases branched-chain oxoacid dehydrogenase kinase content in human skeletal muscle. *Am J Physiol Regul Integr Comp Physiol*. 2007;293:R1335–41.

24. Jacobs I, Esbjornsson M, Sylven C, Holm I, Jannson E. Sprint training effects on muscle myoglobin enzymes, fiber types, and blood lactate. *Med Sci Sports Exerc*. 1987;19(4):368–74.

25. Johnson BD, Beck KC, Proctor DN, Miller J, Dietz NM, Joyner MJ. Cardiac output during exercise by the open circuit acetylene washin method: comparison with direct Fick. *J Appl Physiol*. 2000;88:1650–8.

26. LaForgia J, Withers RT, Shipp NJ, Gore CJ. Comparison of energy expenditure elevations after submaximal and supramaximal running. *J Appl Physiol*. 1997;82:661–6.

27. Laursen PB, Shing CM, Peake JM, Coombes JS, Jenkins DG. Interval training program optimization in highly trained endurance cyclists. *Med Sci Sports Exerc*. 2002;34(11):1801–7.

28. MacDougall JD, Hicks AL, MacDonald JR, McKelvie RS, Green HJ, Smith KM. Muscle performance and enzymatic adaptations to sprint interval training. *J Appl Physiol*. 1998;84:2138–42.

29. Murias JM, Kowalchuk M, Paterson DH. Time course and mechanisms of adaptations in cardiorespiratory fitness with endurance training in older and younger men. *J Appl Physiol*. 2010;108:621–7.

30. Noreen EE, Lemon PWR. Reliability of air displacement plethysmography in a large, heterogeneous sample. *Med Sci Sports Exerc*. 2006;38(8):1505–9.

31. Parra J, Cadefau JA, Rodas G, Amigo N, Cusso R. The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle. *Acta Physiol Scand*. 2000;169:157–65.

32. Peronnet F, Massicotte D. Table of non-protein respiratory quotient: an update. *Can J Sport Sci*. 1991;16:23–9.

33. Rodas G, Ventura JL, Cadefau JA, Cusso R, Parra J. A short training programme for the rapid improvement of both aerobic and anaerobic metabolism. *Eur J Appl Physiol*. 2000;82:480–6.

34. Siri WE. Body composition from fluid spaces and density: analysis of methods. In: Brozek J, Henschel A, editors. *Techniques for Measuring Body Composition*. Washington (DC): National Academy of Sciences, National Research Council; 1961; p. 223–44.

35. Talanian JL, Galloway SD, Heigenhauser GJ, Bonen A, Spriet LL. Two weeks of high-intensity aerobic interval training increases the capacity for fat oxidation during exercise in women. *J Appl Physiol*. 2007;102:1439–47.

36. Thomas S, Reading J, Shephard RJ. Revision of the Physical Activity Readiness Questionnaire (PAR-Q). *Can J Sports Exerc*. 1992;17:338–45.

37. Trapp EG, Chisholm DJ, Freund J, Boutcher SH. The effects of high-intensity intermittent exercise training on fat loss and fasting insulin levels of young women. *Int J Obes*. 2008;32:684–91.

38. Tremblay A, Simoneau JA, Bouchard C. Impact of exercise on body fatness and skeletal muscle metabolism. *Metabolism*. 1994;43:814–8.

39. Warburton DE, Haykowsky MJ, Quinney HA, Humen DP, Teo KK. Reliability and validity of measures of cardiac output during incremental to maximal exercise. Part I: conventional techniques. *Sports Med*. 1999;27:23–41.

40. Wisloff U, Støylen A, Loennechen JP, et al. Superior cardiovascular effect of aerobic interval training versus moderate continuous training in heart failure patients: a randomized study. *Circulation*. 2007;115(24):3086–94.

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