Effect of short-term high-intensity interval training vs. continuous training on O₂ uptake kinetics, muscle deoxygenation, and exercise performance

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Pulmonary O₂ uptake (VO₂p) does not increase instantaneously at the onset of exercise but increases exponentially toward a new steady state, with VO₂p kinetics reflecting those of muscle O₂ utilization to within ~10% (2, 21). It has been suggested that VO₂p kinetics (and thus muscle O₂ utilization) are limited by activation of rate-limiting enzymes and provision of oxidative substrate [i.e., acetyl CoA, reducing equivalents (NADH, FADH₂) ADP, P₃] to the mitochondrial tricarboxylic acid cycle and electron transport chain (ETC) (20), by muscle blood flow and O₂ availability (31), or by a combination of both (23, 24). Exercise training induces adaptive changes, which should reduce the “sluggish” activation of metabolic processes within muscle (e.g., by increasing mitochondrial enzymes) and increase O₂ delivery (9, 35, 46, 47, 56); however, few studies have examined the effects of HIT on VO₂p kinetics during the transition to exercise (4, 35, 45).

Previous studies examining the effect of training on VO₂p kinetics typically have used a more “traditional” exercise training regime [i.e., continuous endurance exercise at ~60–65% maximal O₂ uptake (VO₂ max) for 30–120 min] (1, 9, 46). Endurance exercise training (4–6 wk) resulted in faster VO₂p kinetics during the transition to moderate-intensity exercise (4, 46), with a significant reduction in rVO₂ reported after only 4 training days (46). Furthermore, the kinetics of femoral arterial blood velocity were faster after 10 days training [i.e., the earliest measure taken during training (56)], suggesting that adaptations to blood flow and O₂ delivery to the active muscles occurred earlier than adaptations to muscle oxidative capacity (22, 46, 56). More recent data, however, suggest that muscle oxidative capacity may increase after only six sessions of endurance training spread over 2 wk, which was not different from adaptations found with short-term interval training over a similar training period (18).

Studies comparing both (low-volume) HIT and lower-intensity (high-volume) continuous (END) training regimes have demonstrated similar adaptations for increases in oxidative potential (18) and speeding of VO₂p kinetics (4, 45) in the two types of training programs. However, VO₂p kinetics were not...
measured until after 6 or 10 wk of HIT and END (4, 45), whereas increases in oxidative capacity [i.e., protein content of cytochrome-C oxidase subunit 4 (COX4)] were found after 1 wk of HIT (6). Therefore, although a speeding of V\textsubscript{O\textsubscript{2p}} kinetics appears to accompany HIT (4, 45), it is still unknown as to how soon after the start of the training program the faster V\textsubscript{O\textsubscript{2p}} kinetics become evident. Understanding the time course of these early adaptations may provide a better understanding of the relationship between biochemical and physiological responses to exercise training. Thus the primary purpose of the present study was to determine the early time course of the training-induced adaptation of the phase II V\textsubscript{O\textsubscript{2p}} kinetics (reflecting muscle O\textsubscript{2} utilization) throughout eight sessions of (low-volume) HIT. A secondary purpose was to compare the time course of this response between HIT and a more “traditional” lower-intensity (high-volume) END program. The kinetics of V\textsubscript{O\textsubscript{2p}} and muscle deoxygenation during the transition to moderate-intensity exercise were studied before and throughout a 3-wk training program consisting of eight HIT training sessions or eight END sessions. We hypothesized that 1) eight sessions of HIT would result in faster V\textsubscript{O\textsubscript{2p}} kinetics compared with END and 2) the adaptation of local muscle deoxygenation will become faster with training consequent to a faster rate of muscle O\textsubscript{2} utilization.

**METHODS**

**Subjects**

This study was approved by The University of Western Ontario Ethics Committee for Research on Human Subjects. Twelve young adult males [age = 25 ± 4 yr (means ± SD), body mass = 83 ± 7 kg, V\textsubscript{O\textsubscript{2max}} = 3.68 ± 0.47 l/min] volunteered and gave written informed consent to participate in the study. Participants were randomly assigned to either a high-intensity (low-volume) interval training (HIT) group (n = 6) or a low-intensity (high volume) continuous endurance training (END) group (n = 6). All subjects were healthy with no known musculoskeletal or cardiopulmonary disease, and none were taking medications known to affect the cardiopulmonary system. Subjects were all recreationally active but not currently involved in a training program and were instructed to continue normal daily activities and to refrain from beginning any other training until the completion of the study.

**Experimental Protocol**

**Preexperimental procedures.** Subjects reported to the laboratory three times 1 wk before baseline testing to become familiarized with the testing used to evaluate training status. Subjects performed 1) a ramp incremental (RI) test to the limit of tolerance, 2) a constant-load, moderate-intensity exercise test at a work rate (WR) corresponding to ~90% of their estimated lactate threshold, and 3) a timed, constant-load performance test to volitional fatigue at a WR corresponding to the highest WR achieved during the RI test; all testing was done on a cycle ergometer.**

**Exercise testing.** Subjects reported to the laboratory on three separate occasions separated by at least 24 h and at approximately the same time of day (+2 h) before beginning exercise training. Pretraining V\textsubscript{O\textsubscript{2max}}, estimated lactate threshold (from gas-exchange variables), and measured lactate threshold (with the use of measured plasma lactate concentration; [Lac\textsuperscript{-}]) were determined during an RI test (described below) on the first day of testing (preperiod) at least 1 wk after the familiarization visits. V\textsubscript{O\textsubscript{2p}} kinetics were determined by a series of five-step transitions at a WR of moderate intensity (described below) on the second visit. Endurance performance was determined by a timed performance ride to fatigue (described below) on the third visit. These testing procedures were repeated after four training sessions (mid-training) and again after completion of the eight-session training program (posttraining). In addition, the sequence of five moderate-intensity, step transitions in WR were also repeated after 2 and 6 days of training to establish a time course for any training-induced change in V\textsubscript{O\textsubscript{2p}} kinetics. The last exercise tests were conducted ~72 h after the last training bout to minimize any influence of the last exercise bout on the muscle metabolic environment (38).

**RI test.** An RI exercise test (20 W/min) was performed to the limit of tolerance on an electromagnetically braked cycle ergometer (model H-300-R, Lode, Groningen, The Netherlands) for determination of V\textsubscript{O\textsubscript{2max}}, estimated lactate threshold, and measured lactate threshold. The gas exchange estimated lactate threshold was defined as the V\textsubscript{O\textsubscript{2p}} at which V\textsubscript{CO\textsubscript{2}} began to increase out of proportion relative to V\textsubscript{O\textsubscript{2p}}, combined with a systematic rise in the ventilatory equivalent for V\textsubscript{O\textsubscript{2}} (VE/V\textsubscript{O\textsubscript{2p}}) and the end-tidal PO\textsubscript{2} without a concomitant rise in the ventilatory equivalent for V\textsubscript{CO\textsubscript{2}} (VE/V\textsubscript{CO\textsubscript{2}}) or fall in end-tidal PC\textsubscript{O\textsubscript{2}}. The measured lactate threshold was defined as the WR where the plasma [Lac\textsuperscript{-}] began to increase progressively above baseline values. The V\textsubscript{O\textsubscript{2max}} was calculated as the average V\textsubscript{O\textsubscript{2}} of the last 15 s of the RI test. V\textsubscript{O\textsubscript{2max}} was verified using the RI step exercise protocol described by Rossiter et al. (53), with the post-RI step exercise intensity equal to 105% of the RI WR\textsubscript{max}. Based on RI testing, a moderate-intensity WR was selected to elicit a V\textsubscript{O\textsubscript{2}} equivalent to ~90% estimated lactate threshold.

**Blood sampling.** Throughout the RI test, arterialized venous blood was sampled at specific times for determination of plasma [Lac\textsuperscript{-}]. Before the RI test was started, with the subject resting in a supine position, a percutaneous Teflon catheter (BD Angiocath, 21 gauge; Becton Dickinson) was placed into a dorsal hand vein. The hand and forearm were wrapped in a heating pad with additional heating provided by a heat lamp to “arterialize” the blood. Arterialized venous blood was sampled at baseline, every minute throughout the RI test, and immediately before and after the step exercise test and analyzed for plasma [Lac\textsuperscript{-}].

**Moderate-intensity step test.** Before training and after every 2 training days, subjects completed a sequence of five repeated step transitions in WR of moderate intensity. Each step transition test consisted of 6 min of cycling on an electromagnetically braked cycle ergometer (model H-300-R, Lode) at a baseline of 20 W followed by an instantaneous step increase in WR (corresponding to ~90% estimated lactate threshold); each step transition was separated by at least a 20-min resting recovery to allow muscle blood flow and metabolism to return back toward resting levels.

**Time-to-fatigue performance test.** Subjects performed a time-to-fatigue (TTF) performance ride that was adapted from Burgomaster et al. (8). Subjects cycled on an electromagnetically braked cycle ergometer (model H-300-R, Lode) at 50 W for 5 min as a warm-up followed by an instantaneous increase in WR to 100% WR\textsubscript{max} obtained in the pretraining RI test. Subjects cycled at a self-selected cadence above 70 rpm until they were unable to maintain a cadence above 60 rpm, at which time the test was stopped and the endurance time was recorded. Subjects were not given any visual, physiological, or temporal feedback during the test. A control group (n = 6) completed a similar timed performance test on two occasions separated by 1 wk. Subjects in this group were asked to maintain normal daily activities during the week separating the tests.

**Exercise Training Protocols**

Training was initiated 2–3 days after the baseline testing and consisted of either eight sessions of HIT or 8 sessions of END performed over a 19-day period, with each training session separated by 1–2 days of rest. Training was conducted on a friction-braked cycle ergometer (Monark Ergomedic 874E, Monark, Vansbro, Sweden) and was monitored by one of the investigators.

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HIT session. Each HIT session consisted of a 5-min warm-up followed by 1-min exercise at 120% of the pretraining WRmax followed by 1-min “loadless” cycling. This interval was repeated 8 times on training days 1 and 2 and progressed to 12 repeated intervals by the eighth session. Subjects were given strong verbal encouragement, and resistive loads were altered in accordance with the average pedal cadence to ensure that the average WR for each 1-min bout was 120% of the pretraining WRmax.

END session. The END protocol was adapted from a protocol described previously (22, 46). Briefly, each training session consisted of 90–120 min of cycling at an intensity equivalent to 65% of the pretraining VO2max. Subjects were monitored by the investigators and given verbal encouragement when required. Subjects were allowed brief rest pauses (30–90 s) if they were unable to perform the 90-min exercise continuously, as described by others (22). Average cadence was recorded to allow calculation of average work completed per session and total work completed for the entire training program.

Dietary considerations. Subjects were asked to record their diet on the day before the RI and TTF performance tests and to replicate this same diet before the mid- and posttraining tests. Subjects were instructed to abstain from caffeine or alcohol for at least 8 h before testing.

Data Collection

Gas-exchange measurements were similar to those described previously (55). Briefly, inspired and expired flow rates were measured using a low dead space (90 ml) bi-directional turbine (Alpha Technologies VMM 110), which was calibrated before each test with the use of a 3.0-liter syringe. Inspired and expired gases were sampled continuously at the mouth and analyzed for concentrations of O2, CO2, and N2 by mass spectrometry (Amis 2000, Innovision, Odense, Denmark) after calibration with precision-analyzed gas mixtures. Breath-by-breath alveolar gas exchange was calculated using the algorithms of Beaver et al. (3). Beat-by-beat heart rate (HR) was recorded continuously by a three-lead electrocardiogram.

Local muscle oxygenation of the vastus lateralis muscle of the quadriceps muscle group was monitored by near-infrared spectroscopy (NIRS) (NIRO 300, Hamamatsu Photonics, Hamamatsu City, Japan) using the method described by Delorey et al. (13). The theory of NIRS is described in detail by Elwell (16). The inter-optode spacing was 5 cm, and the differential pathlength factor was assumed to be 3.83; however, because of the uncertainty of this value during exercise, NIRS data are reported as “arbitrary units”. Changes in oxy- (ΔO2Hb), deoxy- (ΔHHb), and total-hemoglobin-myoglobin (ΔHbtot) are reported as a change in concentration (in arbitrary units) from the pretransition (20 W cycling) baseline. The ΔHHb signal can be regarded as being essentially blood volume insensitive during exercise (10, 17); thus it was assumed to reflect muscle O2 extraction within the field of interrogation (11, 17).

Data Analysis

Curve fitting. Kinetic analysis of breath-by-breath VO2p, beat-by-beat HR, and NIRS-derived oxygenation data have been described previously (13, 23, 24, 40, 55). VO2p and HR data obtained during each step transition were filtered for aberrant data points and linearly interpolated to 1-s intervals. Each transition was time aligned and ensemble averaged to yield a single profile and then averaged into second-by-second data and corresponded to the time of the first point demonstrating a consistent increase above the nadir of the ΔHHb signal. The ΔHHb data between the ΔHHbTD and 90 s (corresponding to the duration of the phase II VO2p response) were modeled with a monoexponential function of the form given in Eq. 1 to determine the time course of muscle ΔHHb (τΔHHb). The effective time constant (τ' = ΔHHbTD + τΔHHb) was calculated to provide a description of the overall time course for muscle ΔHHb.

Plasma [Lac−] analysis. Arterialized venous blood samples from the RI test were analyzed for plasma [Lac−] with an ion-selective electrode (StatProfile 9 Plus blood gas-electrolyte analyzer, Nova Biomedical Canada). The electrodes were calibrated before each test and at regular intervals throughout the analysis.

Statistical Analysis

Statistical analysis was performed using SigmaStat 3.0 analysis software (Systat). Differences between groups for total and average work completed during training were analyzed by one-way ANOVA. The parameter estimates for VO2p, HR, ΔHHb, ΔO2Hb, ΔHbtot, peak exercise responses, [Lac−], and body mass were analyzed with a two-way ANOVA for repeated measures (one factor for time and one factor for training program). Correlations for rVO2p, vs. time and ΔVO2p vs. pre-τVO2p were analyzed using the Pearson product moment correlation. Statistical significance was accepted at P < 0.05. Significant interactions and main effects were analyzed using the Tukey’s honestly significant difference post hoc test. All results are presented as means ± SD.

RESULTS

A descriptive summary of the two exercise training protocols used in this study is presented in Table 1. The total exercise time for the eight training sessions was ~90% lower (P < 0.001) in HIT (80 min) than in END (825 min), and the total exercise volume in HIT (~1,800 kJ) was ~80% lower (P < 0.001) than in END (~8,500 kJ).

Table 1. Training protocols

<table>
<thead>
<tr>
<th>Parameter</th>
<th>HIT</th>
<th>END</th>
</tr>
</thead>
<tbody>
<tr>
<td>Work intensity</td>
<td>~120% VO2max (~390 W)</td>
<td>~65% VO2max (~175 W)</td>
</tr>
<tr>
<td>Exercise protocol</td>
<td>60 s x 8–12 repeats, 60 s rest between each repetition</td>
<td>90–120 min</td>
</tr>
<tr>
<td>Total exercise time</td>
<td>80 min</td>
<td>825 min*</td>
</tr>
<tr>
<td>Total time including rest</td>
<td>160 min</td>
<td></td>
</tr>
<tr>
<td>Exercise volume per session</td>
<td>~200 kJ</td>
<td>~1,050 kJ*</td>
</tr>
<tr>
<td>Total exercise volume</td>
<td>~1,800 kJ</td>
<td>~8,500 kJ*</td>
</tr>
</tbody>
</table>

HIT, high-intensity interval training; END, continuous endurance training; VO2max, maximal O2 uptake. Total exercise volume and exercise volume per session results are based on average workloads sustained during training sessions, excluding rest intervals (loadless cycling). *Significantly different (P < 0.001) from HIT.
Response to the RI Test

Table 2 summarizes cardiorespiratory fitness changes assessed during RI testing at pre-, mid-, and posttraining for the HIT and END protocols. Absolute VO$_{2\text{max}}$ (l/min) did not change over the course of eight training sessions, but relative VO$_{2\text{max}}$ (ml·kg$^{-1}$·min$^{-1}$) increased ($P < 0.05$) by ~4.5% and ~7.0% in HIT and END, respectively (Table 2). There was a main effect for time (exercise training per se; $F = 8.503$); however, there was no difference between groups. The WR$_{\text{max}}$ reached during the RI test increased at mid-training (by ~4.5%; $P = 0.013$), with a further ~4% increase in WR$_{\text{max}}$ seen posttraining ($P = 0.009$). There was a main effect for time ($P = 0.001, F = 27.43$), but no differences were observed between training groups.

The VO$_{2p}$ corresponding to the gas-exchange estimated lactate threshold increased ($P = 0.004$) ~13% by the end of training (main effect for time; $F = 9.571$), with no difference observed between training groups (Table 2). The WR corresponding to estimated lactate threshold increased by ~4–5% ($P = 0.01$) and ~8–9% ($P = 0.02$) at mid- and posttraining, respectively (main effect for time only; $F = 0.001, F = 23.96$). The training-induced increases in measured lactate threshold were similar to those of the gas-exchange estimated lactate threshold (Table 2). Also, baseline and peak plasma [Lac$^-$] results measured during the RI step exercise tests were not affected by training or by training protocol ($1.65 \pm 0.43$ and $12.78 \pm 2.28$ mmol/l, respectively).

**VO$_{2p}$ Kinetics**

Table 3 shows the effects of training on the parameter estimates for VO$_{2p}$ kinetics during the transition to moderate-intensity exercise. Figure 1 presents the VO$_{2p}$ response during the transition to moderate-intensity exercise for representative subjects in the HIT (Fig. 1, A and B) and the END training programs (Fig. 1, C and D). The WR for the moderate-intensity exercise was 88 ± 19 W in HIT and 92 ± 10 W in END; pretraining, this WR corresponded to 91 ± 4% estimated lactate threshold (or 42 ± 5% VO$_{2\text{max}}$) and 90 ± 5% estimated lactate threshold (or 40 ± 3% VO$_{2\text{max}}$) in HIT and END, respectively.

Baseline and end-exercise VO$_{2p}$ were lower ($P < 0.05$) posttraining than pretraining in both HIT and END (Table 3), with a main effect for time only ($P = 0.018, F = 3.81$ baseline; $P = 0.014, F = 4.12$). The VO$_{2p}$ amplitude and VO$_{2p}$ gain ($\Delta$V$\text{O}_2p/\Delta$WR) were not affected by either training program. The $\tau$VO$_{2p}$ decreased ($P < 0.05$) by ~20% after only 2 days of training and by ~40% posttraining, with no difference between HIT and END (Table 3). There was a main effect for time only ($P < 0.001, F = 23.01$). Regression analysis determined the slope of the training adaptation of $\tau$VO$_{2p}$ for both HIT and END to be ~2.4 s for every 2 training days (Fig. 2A) with individuals with the slowest pretraining kinetics (i.e., >25 s) showing the greatest reduction in $\tau$VO$_{2p}$ over the course of the training program, regardless of the training mode (Fig. 2B).

**HR Kinetics**

Table 4 presents parameter estimates for HR during the step transition tests throughout the course of training. Figure 3 presents the HR response to moderate-intensity exercise pre- and posttraining for a representative subject in the HIT group (Fig. 3A) and in the END group (Fig. 3B). The time constant for the HR response ($\tau$HR) during the transition to moderate-intensity exercise was reduced after training ($P < 0.05$); however, no changes were observed in $\tau$HR until end of training. For $\tau$HR, there was a main effect for time only ($P = 0.003, F = 5.88$). End-exercise HR was reduced by ~4.5% ($P < 0.05$) in both groups after training, with a main effect for time only ($P = 0.006, F = 5.024$). There were no between-group differences for any of the HR parameters during the transition to moderate-intensity exercise.

**NIRS-derived ΔO$_2$Hb, ΔHbtot, and ΔHHb**

Steady-state baseline and end-exercise NIRS-derived ΔO$_2$Hb and ΔHbtot were not different between groups, and they were not changed by training (data not shown).

Table 5 shows parameter estimates for deoxygenated hemoglobin-myoglobin (ΔHHb) kinetics throughout the course of training. There were no training-induced changes or group differences in the time course (TD, $\tau$ΔHHb, $\tau^\prime$) or amplitude of the ΔHHb response (Fig. 4).

**Exercise Performance**

The TTF during the constant-load test increased ($P < 0.001$) ~55% for HIT (pretraining: 200 ± 46 s; posttraining: 310 ± 88 s) and ~43% for END (pretraining: 221 ± 47 s; posttraining, 316 ± 55 s) as a consequence of training, with a greater ($P < 0.05$) TTF observed in mid- than in pretraining in both groups. No change in TTF was observed in the control group.

**DISCUSSION**

This study compared the effects of eight sessions of either a low-volume HIT program or a high-volume END program on the...
early adaptation of phase II $V_{\dot{O}_2p}$ kinetics and muscle deoxygenation kinetics. The present design allowed training adaptations to be studied after every 2 training days, thus providing insight into the early time course of training adaptations in two groups whose training program differed in total volume (i.e., HIT: 1,800 kJ, END: 60% $V_{\dot{O}_2 max}$), and total exercise time (i.e., HIT: 80 min, END: 825 min). The major novel findings of the present study are as follows (1) phase II $\tau V_{\dot{O}_2p}$ (reflecting the adaptation of muscle O2 utilization) was reduced by ~20% after only two training sessions and by ~40% after 8 days of training, with similar changes occurring in both the HIT and END groups; 2) the reduction in $\tau V_{\dot{O}_2p}$ (i.e., faster $V_{\dot{O}_2p}$ kinetics) occurred in a progressive, linear fashion over the eight training sessions in both groups; 3) ΔHtHb kinetics were not changed by training despite a training-induced speeding of $V_{\dot{O}_2p}$ kinetics. To our knowledge, this is the first study to directly compare the early temporal adaptation of $V_{\dot{O}_2p}$ kinetics and muscle deoxygenation kinetics during the first 8 days of HIT and END.

Previous studies employing measurements early in training have shown $V_{\dot{O}_2p}$ kinetics were faster after 4 training days (46) and blood velocity kinetics were faster after 10 days of training (56). However, in these studies, no measures were taken before 4 training days and only the effects of continuous training were examined (46, 56). In recreationally active subjects, eight sessions of HIT or END was sufficient to induce positive changes in $V_{\dot{O}_2p}$ kinetics, with the average $\tau V_{\dot{O}_2p}$ being reduced to those observed in highly trained athletes (~15 s) (32, 33, 44).

**Effect of Short-Term Interval or Continuous Training on Phase II $V_{\dot{O}_2p}$ Kinetics**

In the present study, the early training-induced speeding of $V_{\dot{O}_2p}$ kinetics during the transition to moderate-intensity exercise occurred after only 2 days of training with both HIT and END. In a previous study, Phillips et al. (46) reported faster $V_{\dot{O}_2p}$ kinetics after 4 days of continuous endurance training (2 h/day at 60% $V_{\dot{O}_2 max}$), which represented the first assessment after the start of the training program. In that study (46), the phase II $\tau V_{\dot{O}_2p}$ was reduced ~22% after 4 days of training (i.e., from 37 to 29 s), which was similar to the ~30% (HIT) and ~17% (END) reduction in $\tau V_{\dot{O}_2p}$ observed after 4 days of training in the present study. However, in the present study, there was a significant reduction in $\tau V_{\dot{O}_2p}$ after only 2 training days (~17–20% for both HIT and END). The ~40% overall reduction in $\tau V_{\dot{O}_2p}$ after 8 days of training in both HIT and END in the present study is somewhat higher than reported by Berger et al. (4), who observed an ~26–34% reduction in $\tau V_{\dot{O}_2p}$ after 6 wk of interval (at 90% $V_{\dot{O}_2 max}$) or continuous (at 60% $V_{\dot{O}_2 max}$) training or the 15% reduction of the mean response time reported by Overend et al. (45) for two types of interval training compared with continuous training of 10-wk duration. Furthermore, Krustup et al. (35) observed a greater increase in muscle O2 uptake and femoral arterial blood flow in the trained leg during the first 1–2 min of the transition to moderate- and heavy-intensity single-leg knee-extension exercise after 7 wk (29 training sessions) of intense single-leg knee-extension interval training. However, in that study, the time course of these changes was not established. Although single-leg interval training induced a speeding of O2 uptake kinetics in the heavy-intensity domain, a speeding of O2 uptake kinetics in the moderate-intensity domain was not observed in that study (35). This, in part, may be due to differences in the measurement of O2 uptake. In that study, muscle O2 uptake was determined during single-leg knee-extension exercise using arteriovenous catheterization and thermodilution techniques, with O2 uptake calculated at discrete time points (5–7 time points during the exercise transition) during only a single exercise trial. In the present study, $V_{\dot{O}_2p}$ (reflecting muscle O2 utilization) was measured breath-by-breath during each of five repetitions of the step transition in WR, which provided more data points and thus greater confidence in the O2 uptake signal, with which to estimate the time course of O2 uptake. Differences in exercise modality (cycling vs. single leg knee-extension exercise) and thus differences in active muscle volume and recruitment may also have contributed to the different findings between studies.

In the present study, the baseline and end-exercise $V_{\dot{O}_2p}$ was ~70–100 ml/min lower post- than pretraining in both training
groups. This was surprising as there was no obvious tendency for the baseline and end-exercise $V_{O2p}$ to decrease over the course of the training program. Also, a lower absolute $V_{O2p}$ post-compared with pretraining is not supported by findings in other training studies (e.g., Refs. 4, 35). Although it is possible that there was an increase in cycling efficiency as a consequence of the time spent cycling during the training program, this is unlikely considering that the lower $V_{O2p}$ was observed in both groups, each having markedly different training volumes; however, an increase in cycling efficiency cannot be completely ruled out. Also, the increase in $V_{O2p}$ for a given change in WR (i.e., $V_{O2p}$ gain) was remarkably consistent throughout the training program in both groups, suggesting that the $V_{O2p}$ response to an increase in WR (the inverse of efficiency) was not changed. Finally, subjects from both groups were recruited into the study at different times, thus testing was “staggered” over the course of many weeks. It is very unlikely that a “calibration” problem could be responsible for the lower posttraining $V_{O2p}$ for all subjects. Thus the mechanism responsible for the lower steady-state posttraining $V_{O2p}$ is not known.

In agreement with previously reported findings (4, 46), the activation of oxidative phosphorylation is faster after training, regardless of the training mode (interval vs. continuous), and this adaptation in oxidative phosphorylation may precede increases in mitochondrial enzymes (4, 5, 35, 46). Although it has been reported that mitochondrial CS activity was not increased in the early stages of END (46, 47), recent data suggest that the maximal CS activity may increase ~11–38% after 2 wk of HIT (7, 8) and the protein content of the mitochondrial COX4 may increase 35% after only 1 wk of HIT (6). Furthermore, 2 wk of HIT resulted in an increased mitochondrial PDH activation compared with pretraining levels (7). Together, these findings suggest that oxidative capacity of the muscle increases in as little as 1 wk during HIT. In the present study, oxidative enzymes were not measured; however, a comparison of the $V_{O2p}$ profiles in response to the HIT program used in the present study and the Wingate-HIT used by Burgomaster et al. (6–8) indicates that the peak $V_{O2p}$ within an interval and the overall (or average) $O_2$ cost of a series of intervals may have been higher in our HIT protocol than that
V̇O₂ max for 1 min each with 1-min rest intervals vs. 4–6 training for the Wingate-HIT protocol (data not shown). The HIT protocol used in the present study resulted in subjects repeatedly approaching and/or reaching V̇O₂ max during each of the training intervals, which thus represented a greater proportion of the training duration (i.e., 8–12 repeated bouts at training intervals, which thus represented a greater proportion and likely an integration of both enhanced metabolic control and O₂ delivery. A faster activation of PDH (as well as other oxidative enzymes) during the transition to moderate-intensity exercise would provide a greater substrate availability to the mitochondrial tricarboxylic acid cycle and ETC earlier in the exercise transition and may result in a more rapid achievement of steady state, as evidenced by a reduction in the τV̇O₂p. After short-term training, it is likely that the increase in PDH activity is a consequence of an upregulation or greater sensitivity of the regulators of PDH (PDH phosphatase and/or PDH kinase) (37, 50). Importantly, an elevated PDH activation that exists after a bout of heavy-intensity priming exercise was associated with a “speeding” of V̇O₂ kinetics during the transition to a subsequent bout of moderate-intensity exercise (23).

In addition, increased COX4 protein content and increased COX activity observed as early as 1 wk during training (6, 18) would allow for an increased flux through the ETC. Neuf and Dohm (43) reported that CS gene transcription was elevated at 3 and 24 h after a single “acute” bout of treadmill running in rats. They also reported that, after 1 wk of treadmill running, CS mRNA content and CS protein content were, respectively, ~15% and 1.3-fold greater than that shown in untrained control animals (43).

ADP concentration, as with NADH and O₂, must be in sufficient concentrations to allow for adequate ATP turnover from oxidative phosphorylation, and these concentrations must increase proportionately during the transition to exercise. ADP is produced immediately at the onset of exercise via the cellular ATPase; however, the rise in ADP concentration (both cytosolic and mitochondrial) is buffered by the creatine kinase reaction. Evidence to support the effect of PCR breakdown to buffer increases in ADP have shown that, when creatine kinase is inhibited by iodoacetamide in rats, the rate of oxidative phosphorylation increases rapidly during the transition to exercise (28, 34). Further investigations using creatine kinase knock-out mice show similar results, indicating that, when there is an increase in ADP without buffering by PCR breakdown, oxidative phosphorylation will increase rapidly (25). In a recent study by Glancy et al. (19), alterations in the total creatine pool strongly influenced the rate of aerobic flux. Increased total creatine induced an increase in the time constant of the aerobic flux in their in vitro rat muscle preparation (19). ADP appears to be a key signaling molecule of PDH (57); without an increase in ADP, there would be minimal drive to increase oxidative phosphorylation, and thus ADP is likely the determining substrate for oxidative phosphorylation at the onset of exercise. Combined, these factors might overcome any limitation to oxidative phosphorylation imposed by inadequate availability of oxidative substrate (i.e., metabolic limitation).

In the present study, bulk muscle blood flow and O₂ delivery to the muscle were not measured. However, NIRS-derived ΔO₂Hb, ΔHbtot, and ΔHHb provide information on local muscle O₂ availability (ΔO₂Hb, ΔHbtot) and local O₂ extraction (ΔHHb) (10–12, 15). The NIRS-derived ΔHHb reflects the balance between local muscle O₂ consumption and muscle O₂ delivery; when used in combination with measures of V̇O₂ kinetics (reflecting the kinetics of muscle O₂ consumption), it also provides information on the profile of local muscle (microvascular) blood flow adaptation. In the present study, no changes to τΔHHb or the τ' (or mean response time = τΔHHb + TD) were observed over the course of training in either group despite a reduction in V̇O₂p after only 2 days of training with further reductions in τV̇O₂p seen over the course of the remaining sessions, suggesting that the faster rate of increase in muscle O₂ utilization was not accompanied by a
faster and/or greater muscle O₂ extraction. Together, these findings suggest that the distribution of local microvascular blood flow at the onset of moderate-intensity exercise became faster as a consequence of the HIT and END programs. However, whether the early adaptation of microvascular blood flow is accompanied by a faster adaptation of conduit artery blood flow has not been established. To our knowledge, Shoemaker et al. (56) have provided the only information on early training-induced adaptations to conduit artery blood flow. In that study, they showed that the kinetics of conduit artery blood velocity and arterial conductance became faster after 10 days of END training, which represented the first reported measure of the HIT and END groups for pretraining, TR2, mid-training, TR6, and posttraining.

Values are means ± SD. Fixed time delay = 0 for all subjects. HR, heart rate; rHR, time constant of heart rate response; BSL, baseline (20 W cycling); AMP, amplitude; End-Ex, end exercise HR; ΔHR/ΔWR, efficiency. No differences between HIT and END groups. *Significant (P < 0.05) difference from Pre. †Significant (P < 0.05) difference from TR2. ‡Significant (P < 0.05) difference from Mid.

Table 4. Average parameter estimates for HR on-transients to moderate-intensity cycle ergometer exercise in the HIT and END groups for pretraining, TR2, mid-training, TR6, and posttraining

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pre</th>
<th>TR2</th>
<th>Mid</th>
<th>TR6</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>BSL, beats/min</td>
<td>91±11</td>
<td>92±11</td>
<td>89±7</td>
<td>91±12</td>
<td>87±11</td>
</tr>
<tr>
<td>AMP, beats/min</td>
<td>20±4</td>
<td>20±4</td>
<td>19±3</td>
<td>19±4</td>
<td>20±5</td>
</tr>
<tr>
<td>End-Ex, beats/min</td>
<td>112±11</td>
<td>112±10</td>
<td>109±8</td>
<td>110±12</td>
<td>107±9†‡</td>
</tr>
<tr>
<td>rHR, s</td>
<td>18±7</td>
<td>19±7</td>
<td>20±7</td>
<td>16±9</td>
<td>14±7†‡</td>
</tr>
</tbody>
</table>

HIT

END

Table 5. Average parameter estimates for NIRS HHb on-transients to moderate-intensity cycle ergometer exercise in the HIT and END groups for pretraining, TR2, Mid, TR6, and posttraining

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pre</th>
<th>TR2</th>
<th>Mid</th>
<th>TR6</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔHHb BSL, AU</td>
<td>-1.1±4.5</td>
<td>-0.6±2.2</td>
<td>-0.7±1.9</td>
<td>-1.0±2.5</td>
<td>-2.8±3.7</td>
</tr>
<tr>
<td>ΔHHb AMP, AU</td>
<td>4.7±2.9</td>
<td>4.7±2.6</td>
<td>4.4±2.4</td>
<td>4.7±2.4</td>
<td>4.4±2.5</td>
</tr>
<tr>
<td>ΔHHb End-Ex, AU</td>
<td>4.4±1.9</td>
<td>5.4±2.6</td>
<td>5.9±2.5</td>
<td>4.7±2.6</td>
<td>4.5±2.7</td>
</tr>
<tr>
<td>ΔHHb TD, s</td>
<td>11±2</td>
<td>10±3</td>
<td>12±2</td>
<td>12±2</td>
<td>11±1</td>
</tr>
<tr>
<td>ΔHHb rΔHHb, s</td>
<td>13±6</td>
<td>14±8</td>
<td>10±4</td>
<td>13±6</td>
<td>10±6</td>
</tr>
<tr>
<td>ΔHHb τ, s</td>
<td>24±7</td>
<td>24±10</td>
<td>21±3</td>
<td>26±4</td>
<td>20±4</td>
</tr>
</tbody>
</table>

END

Fig. 3. Beat-by-beat heart rate (HR) response for the transition to constant-load, moderate-intensity exercise. bpm, Beats/min. A: representative subject for HIT (rHR = 16 s pretraining; 8 s posttraining). B: representative subject for END (rHR = 19 s pretraining; 8 s posttraining). ○, pretraining; ●, posttraining.
the activation of muscle O2 utilization also did not change. If the training stimulus increases the early hyperemic response and subsequent local vasodilatory response adequately to meet the increasing O2 demand during the transition to exercise, then the time course of O2 extraction need not become faster. In the present study, the adaptation of HR was used to reflect the adaptation of cardiac output and bulk blood flow (40). HR kinetics (and thus, possibly, bulk blood flow) did not become faster until after eight training sessions. Therefore, these data suggest that the kinetics of bulk blood flow may not be altered by short-term training but that the distribution of blood flow and subsequent adaptation of microvascular blood flow may be improved with training (26).

In the present study, individuals with relatively fast pretraining V\textsubscript{2}O\textsubscript{p} kinetics (i.e., τ\textsubscript{V2Op} < 25 s) showed less of a training-induced reduction in τ\textsubscript{V2Op} than those with slower V\textsubscript{2}O\textsubscript{p} kinetics (i.e., τ\textsubscript{V2Op} > 25 s) (Fig. 2B). These results are similar to those of Gurd et al. (23, 24), in which individuals with slower V\textsubscript{2}O\textsubscript{p} kinetics had a greater reduction in τ\textsubscript{V2Op} after a bout of heavy-intensity exercise than those with faster V\textsubscript{2}O\textsubscript{p} kinetics. Thus individuals with faster pretraining V\textsubscript{2}O\textsubscript{p} kinetics may require a longer training duration (>8 sessions) or a greater training stimulus (higher intensity) to achieve “measurably” faster V\textsubscript{2}O\textsubscript{p} kinetics.

**Effect of Interval or Continuous Training on Lactate Threshold and Maximal O2 Uptake**

In the present study, the estimated lactate threshold and blood lactate threshold (as determined using plasma [Lac−]) increased ~13% in both training groups. The increase in estimated lactate threshold found in this study was similar to the 10-wk changes with interval and continuous training groups reported by Overend et al. (45) but less than the 24–45% increase in estimated lactate threshold found in other previous studies (48, 49) that employed much longer training durations than those used in this study (3 vs. 8 wk). Lower catecholamine release during submaximal exercise as a consequence of training would decrease the stimulation of glycogenolysis, reducing pyruvate production and thereby reducing Lac− production (51), whereas increased peripheral uptake of Lac− would increase clearance from the blood during submaximal exercise, thus prolonging exercise before an accumulation of Lac− is sustained (14). Additionally, faster activation of mitochondrial oxidative phosphorylation (see above) would reduce the requirement for substrate-level phosphorylation (30).

In the present study, both HIT and END failed to increase the absolute V\textsubscript{O}\textsubscript{2max}, although the relative V\textsubscript{O}\textsubscript{2max} increased by a small, but significant, amount: 2–3 ml·kg\textsuperscript{-1}·min\textsuperscript{-1} (related in part to a decrease in body mass). This is in agreement with several short-term training studies (i.e., 1–3 wk), which demonstrated an increase in endurance performance and oxidative capacity and/or WR\textsubscript{max} without significant increases in V\textsubscript{O}\textsubscript{2max} (7, 8, 18, 22, 46). It is likely that longer training interventions than those used in the present study are required before changes in V\textsubscript{O}\textsubscript{2max} are observed (36, 39, 42, 45).

**Conclusions**

In summary, this study demonstrated that V\textsubscript{2}O\textsubscript{p} kinetics was faster during moderate-intensity exercise after only 2 days of training, regardless of the type of exercise training program (HIT or END). The τ\textsubscript{V2Op} kinetics was progressively reduced throughout training. The extent to which V\textsubscript{2}O\textsubscript{p} kinetics was reduced is influenced by the initial training status of the individual, with individuals who have slower initial kinetics responding to the training with a greater magnitude. Both short-term HIT and END protocols showed similar training-induced adaptations with respect to exercise performance, V\textsubscript{2}O\textsubscript{p} kinetics, HR kinetics, measured lactate threshold, and estimated lactate threshold, with no differences occurring between training programs. The time course of ΔHHb (reflecting fractional O2 extraction) was not changed with training, despite a speeding of V\textsubscript{2}O\textsubscript{p} kinetics, suggesting that local muscle (microvascular) O2 delivery was faster and remained “matched” to muscle O2 utilization during the transition to exercise after training. These results suggest that V\textsubscript{2}O\textsubscript{p} kinetics (and muscle O2 utilization kinetics) is influenced similarly by both training programs and that the early adaptation begins as early as after 2 training days.

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HIGH-INTENSITY INTERVAL TRAINING, VO2, AND MUSCLE DEOXYGENATION KINETICS

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