High-intensity interval training speeds the adjustment of pulmonary O$_2$ uptake, but not muscle deoxygenation, during moderate-intensity exercise transitions initiated from low and elevated baseline metabolic rates

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High-intensity interval training speeds the adjustment of pulmonary O$_2$ uptake, but not muscle deoxygenation, during moderate-intensity exercise transitions initiated from low and elevated baseline metabolic rates. J Appl Physiol 114: 1550–1562, 2013. First published March 21, 2013; doi:10.1152/japplphysiol.00575.2012.—During step transitions in work rate (WR) within the moderate-intensity (MOD) exercise domain, pulmonary O$_2$ uptake (V$\dot{O}_2$p) kinetics are slowed, and V$\dot{O}_2$p gain ($\Delta$V$\dot{O}_2$p/ΔWR) is greater when exercise is initiated from an elevated metabolic rate. High-intensity interval training (HIT) has been shown to speed V$\dot{O}_2$p kinetics; near-infrared spectroscopy (NIRS); O$_2$ deficit; ATP; exercise performance.

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COINCIDENT WITH A STEP INCREASE in exercise intensity there is an immediate increase in ATP turnover in the working muscle (52). Pulmonary O$_2$ uptake (V$\dot{O}_2$p), however, does not increase instantaneously, but rather rises with an exponential time course toward a new steady-state level, after allowing for the transport delay between the active muscle and the pulmonary circulation (4, 25, 35). The kinetics of the fundamental phase II V$\dot{O}_2$p response predominantly reflects the adjustment of mitochondrial O$_2$ utilization in the working muscle (4, 25, 35). Additional energy requirements not met by oxidative phosphorylation are met through substrate-level phosphorylation with consequent breakdown of phosphocreatine (PCr) and glycogen (25, 33, 52).

When exercise transitions to work rates (WR) within the moderate- (MOD) (8, 9, 32, 40, 55), or the heavy- or severe-intensity domains (17, 32, 60, 61), are initiated from an elevated compared with lower baseline WR, and thus metabolic rate, the time course of adjustment of V$\dot{O}_2$p is slower than when starting from a low or resting metabolic rate. Additionally, the fundamental V$\dot{O}_2$p gain ($\Delta$V$\dot{O}_2$p/ΔWR), a measure of the O$_2$ cost of the change in WR (reflecting exercise efficiency), is greater for exercise initiated from a higher compared with lower metabolic rate (8, 9, 40, 55, 60, 61). Although the specific mechanisms governing the observed slowed V$\dot{O}_2$p kinetics and lower efficiency for exercise transitions initiated from an elevated baseline metabolic rate within the MOD domain have not been established, the differing responses may reflect intrinsic properties of newly recruited muscle fibers (i.e., having a slower activation and lower efficiency) (9); a less favorable cellular energetic state of the active fibers (lower cellular PO$_2$, PCr concentration ([PCr]) and [ATP]; higher [ADP] and [Pi]; less negative free energy of ATP hydration ($\Delta$G$_{ATP}$) (42); a slower adjustment of central (cardiac output) and peripheral conduit artery bulk blood flow and O$_2$ delivery, and/or a lower steady-state increase in blood flow relative to muscle O$_2$ utilization (32, 40); or a combination of these factors.

The performance of low-volume, high-intensity interval training (HIT) [or low-volume, high-intensity sprint interval training (SIT)] promotes rapid adaptations in maximal V$\dot{O}_2$p ($V_{O_2,max}$), work capacity, muscle oxidative capacity, and vascular function (3, 12, 22, 30, 36, 41, 49), with consequent improvements in exercise performance (3, 13, 22, 41). For example, Burgomaster et al. (11, 13) observed increases in resting muscle glycogen, mitochondrial citrate synthase and pyruvate dehydrogenase activity, and improved time trial performance following 6 sessions (2 wk) of SIT. Gibala et al. (23) reported immediate increases in markers of mitochondrial biogenesis following a single session of SIT (total of 2 min; <80 kJ total work). Recently, McKay et al. (41) reported that the adjustment of V$\dot{O}_2$p during single-step (SS) transitions into the upper part of the MOD domain [20 W → 90% lactate threshold ($\theta_L$)] became faster after only two sessions of HIT ($\tau V_{O_2)p}$ reduced by $\sim$20%, where $\tau$ is the time constant), with an even faster adjustment of V$\dot{O}_2$p seen after eight training sessions.
sessions ($\dot{V}O_2p$ reduced by $\sim 40\%$). Additionally, the speeding of $V_{O2p}$ kinetics (and presumably muscle $O_2$ utilization kinetics) with HIT occurred in the absence of any changes in muscle deoxygenation kinetics (41), implying that faster adjustments of local muscle (microvascular) blood flow and $O_2$ delivery accompanied the faster rate of adjustment of muscle $O_2$ utilization. These findings clearly illustrate the effectiveness of HIT in producing rapid improvements to skeletal muscle oxidative potential and exercise performance, and faster adjustments in oxidative ATP production.

Although a faster adjustment of $V_{O2p}$ kinetics during exercise initiated from a low baseline metabolic rate is expected following HIT (7, 41, 47), the effect of HIT (or more traditional types of endurance training) on exercise transitions initiated from an elevated baseline metabolic rate has not been examined. Additionally, previous reports from our laboratory reported greater acute speeding of $V_{O2p}$ kinetics within the MOD domain following a priming bout of heavy-intensity exercise for those individuals presenting with slower $V_{O2p}$ kinetics in an “unprimed” condition (27–29, 53). Thus we questioned whether the rapid HIT-induced adaptations in muscle oxidative capacity and vascular function seen in prior reports (12, 22, 30, 36, 49) would result in a greater speeding of $V_{O2p}$ kinetics, without affecting muscle deoxygenation kinetics (i.e., reflecting the dynamic relationship between local microvascular blood flow and muscle $O_2$ utilization), during exercise transitions in the upper step (US) compared with the lower step (LS).

The primary goal of this study was to investigate the effects of HIT on $V_{O2p}$ and muscle deoxygenation kinetics during transitions from low and elevated metabolic rates, within the MOD domain. SS transitions from the low metabolic rate to the higher WR and time-to-fatigue (TTF) endurance trials were additionally examined, to compare (and confirm) both to prior training studies and to the training-induced changes in the LS and US of the present study. The following hypotheses were tested: 1) HIT would speed $V_{O2p}$ kinetics in the US and LS without affecting muscle deoxygenation ($\{\text{deoxygenation of deoxyhemoglobin concentration (Hb)}\}$) kinetics; 2) HIT-induced speeding of $V_{O2p}$ kinetics would be greater in the US compared with LS, as a consequence of attenuated $V_{O2p}$ kinetics in the US in the untrained state; and 3) a transient overshoot in the ratio of muscle deoxygenation to $V_{O2p}$ would be present before, but not after, HIT as a consequence of an improved dynamic relationship between the adjustment of microvascular blood flow and muscle $O_2$ utilization.

METHODS

Subjects

Eight young, healthy adult men were recruited to complete the HIT program [27 ± 6 yr (mean ± SD), 82 ± 5 kg]. An additional five young, healthy men served as control subjects (CON; 23 ± 3 yr, 79 ± 9 kg) and completed all aspects of the experimental protocol except for the HIT. All participants were untrained, recreationally active (other light-intensity to MOD activities, up to 2–3 times per week), and were asked to continue their regular daily activities for the duration of the study. All participants reported being healthy, without current or history of cardiovascular, respiratory, metabolic, or musculoskeletal disease, and none was a smoker or taking any medications that might affect the cardiovascular or hemodynamic responses to exercise. The protocol, including possible risks and discomforts related to the testing and exercise training, was provided to the subjects both verbally and in writing before commencement of data collection. Participants were instructed to maintain their normal diets over the course of the study. Subjects provided written, informed consent before voluntary participation in this study. All procedures in this study were approved by The University of Western Ontario Ethics Committee for Research on Human Subjects.

Experimental Protocol

**HIT protocol.** The HIT program used in the present study was slightly modified from that used previously in our laboratory (41). All training was completed on a friction-braked cycle ergometer (Monark Ergomedic 874E, Monark, Vansbro, Sweden), with an investigator always present. Each HIT session began with 5 min of “loadless” cycling, with no external resistance applied. Then training participants cycled for 1 min at 110% of the maximal WR ($WR_{max}$) attained during the maximal ramp incremental (RI) test (see below). These work intervals were followed by 1 min of “loadless” cycling. Work intervals were repeated eight times during the first training session, progressing to a total of 12 intervals by the final (12th) training session; each training session was separated by 1–2 days of recovery (Fig. 1). Participants cycled at a self-selected cadence between 80 and 100 revolutions/min (rpm) and maintained that cadence across all work intervals of the training sessions. Total work and adherence to the selected cadence were monitored by the investigator and recorded for each HIT session. After every two training sessions, the cycling load was increased by $\sim 2–4\%$, and an additional training interval was added (Fig. 1), to promote continuous improvements in training performance. During training sessions, participants were provided with strong verbal encouragement, were allowed water ad libitum, and remained on the cycle ergometer for the entire duration of the session.

**Exercise testing.** At the start of the study [pretraining (PRE)], all participants visited the laboratory for an initial RI plus constant-load, step exercise (SE) test (RISE-105) to volitional fatigue to determine their $V_{O2max}$ and estimated $\dot{V}O_2p$ (50). Over the following two visits, they completed step transitions in WR from a baseline of 20 W to a final intensity representing 90% $\dot{V}O_2p$ (i.e., within the MOD exercise domain) performed either as a 1) SS protocol (SS, 20 W → 90%), or...
HIT Speeds $\dot{V}O_2$ Adjustments from Elevated Metabolic Rates • Williams AM et al.

2) as a double-step (DS) protocol, where the increases in WR were performed as two identical step transition increments in WR from 20 W to ~45% $\dot{V}O_2_L$ (LS), followed by a step transition from ~45% $\dot{V}O_2_L$ to 90% $\dot{V}O_2_L$ (US). Each step transition lasted 6 min, and each step protocol was repeated either three (SS) or five times (DS). The effectiveness of the HIT program was assessed by means of a TTF performance test consisting of a constant-load cycling test performed at the same absolute WR, which corresponded to the WRmax achieved during the PRE RI exercise test. For the HIT group, the RISE-105 exercise test, the MOD step transitions, and the TTF test were repeated following 6 (after ~2 wk; MID) and 12 training sessions (after ~4 wk; POST). The CON group performed identical sets of testing separated by 2 (MID) and 4 wk (POST). For both the HIT and CON groups, a final “verification” (VER) testing session was performed 4–5 days following POST testing and consisted of a RISE-105 test, SS-MOD transitions (total of 3 transitions), DS-MOD transitions (total of 5 transitions), and a TTF test. Testing and training time lines for both groups are illustrated in Fig. 1. Testing at the PRE, MID, POST, and VER times was intended to establish a time course for physiological adaptations and performance enhancements over the course of the 12 HIT sessions. The MOD exercise transitions were performed at VER to confirm that any changes in $\dot{V}O_2p$ kinetics were a result of longer-term HIT training, and not a consequence of short-term effects resulting from the previous (final) exercise training sessions. Participants abstained from caffeine for at least 3 h, and from alcohol for at least 12 h, before all testing.

RI exercise (RISE-105) test. Participants completed a RI exercise test (20 W/min) to their limit of tolerance at each of the PRE, MID, POST, and VER time points. The RISE-105 test was performed on an electromagnetically braked leg cycle ergometer (H-300-R Lode; Lode BV, Groningen, Holland) and was used to determine both $V_{O2\max}$ and estimated $\dot{V}O_2_L$. The $\dot{V}O_2_L$ was determined visually as the $V_{O2\max}$ at which the rise in CO2 output ($V_{CO2}$) and ventilation ($V_{E}$) became disproportionate to the rise in $V_{O2p}$, along with a systematic increase in the $V_{E}$-to-$V_{O2p}$ ratio ($V_{E}/V_{O2p}$) and end-tidal PCO2, and where the $V_{E}/V_{CO2}$ and end-tidal PCO2 remained stable (6). A value for $\dot{V}O_2_L$ was identified for each of the listed criteria, and values were averaged together to attain a final estimate for $\dot{V}O_2_L$. $V_{O2\max}$ was determined as the average $V_{O2p}$ measured during the final 20 s of each of the RI and SE portions of the RISE-105 protocol (50): at volitional fatigue at the end of the RI test, the WRmax was noted, and WR was returned to 20 W. Participants continued cycling at 20 W for 5 min, after which the WR was increased as a step function to 105% WRmax (SE-105). Participants cycled at this higher WR until they could no longer maintain a cadence above 50 rpm, despite strong verbal encouragement.

MOD step transition tests. Five repetitions of the DS MOD transition tests (DS-MOD) were completed on a cycle ergometer (H-300-R Lode; Lode BV) at each of the PRE, MID, POST, and VER testing points. Additionally, three repetitions of the SS MOD transition tests (SS-MOD) were completed at both PRE and VER time points. The SS-MOD consisted of 6-min cycling at a baseline of 20 W, followed by an instantaneous step transition to a WR corresponding to 90% $\dot{V}O2_L$ for an additional 6 min. For the DS-MODs, participants cycled for 6 min at 20 W, followed by two 6-min step transitions; the first LS involved an instantaneous increase to a WR midway between 20 W and the WR corresponding to 90% $\dot{V}O2_L$ ($\DeltaWR_{LS}$), and the second US involved an instantaneous increase in WR from $\DeltaWR_{LS}$ to a WR corresponding to 90% $\dot{V}O2_L$ ($\DeltaWR_{US}$). The change in WR for LS and US were identical (i.e., $\DeltaWR_{LS} = \DeltaWR_{US}$). Transitions lasted 6 min to allow the achievement of steady-state $V_{O2p}$ ($\dot{V}O2_{ps}$). Participants maintained a cadence of ~70 rpm during these tests. Because HIT was performed on alternate days, it was not possible to perform only single transitions per day; therefore, multiple transitions were performed on the same day, with each MOD transition separated by 10 min of resting recovery. It has been previously shown that parameter estimates [r, time delay (TD), amplitude] for $\dot{V}O2p$ and deoxygenation ([HHb]) kinetics do not differ when transitions to MOD are completed, either on separate occasions, or sequentially as a series of transitions (2 or 6), each separated by 6-min baseline cycling (56).

TTF performance test. Participants completed a constant-load TTF performance test on a cycle ergometer (H-300-R Lode; Lode BV) at each of the PRE, MID, POST, and VER testing points. The test involved an initial 5 min of cycling at 20 W, followed by an instantaneous increase in WR to 100% of the WRmax attained during the PRE RI test. Participants were instructed to maintain a cadence of at least 70 rpm; the test was stopped when subjects could not maintain a cadence of at least 60 rpm, despite strong verbal encouragement.

Training and testing time lines. PRE testing was completed during three visits, totaling ~2.5 h. After 1–2 days, the HIT group began training with 1–2 days rest between sessions. Each training session required 20–30 min for completion. After completion of six HIT sessions (~2 wk), the HIT group completed MID testing during two visits, totaling ~2 h. The HIT group completed another six training sessions (~2 wk), followed by POST testing. POST tests were completed during two visits, totaling ~2 h. VER testing was performed ~4–5 days after the POST testing and required ~2.5 h during two visits. On average, participation in this study required 5–6 wk for completion.

CON participants completed all testing along a time line identical to the HIT group, but did not complete any HIT training sessions. The CON group was asked to maintain their regular recreational activity (2–3 times per week), without additional training.

Data Collection

Gas exchange measurements were similar to those previously described (1). Briefly, inspired and expired flow rates were measured with a low dead space (90 ml) bidirectional turbine (Alpha Technologies VMM 110), which was calibrated before each test with a 3.0-liter syringe. The inspired and expired gases were sampled continuously (~20 ms) at the mouth and analyzed for concentrations of O2, CO2, and N2 by mass spectrometry (Innovation, AMIS 2000, Lindvedvæg, Denmark) following calibration with precision-analyzed gas mixtures. Changes in gas concentrations were aligned with gas volumes by measuring the TD for a square-wave bolus of gas passing through the turbine to the resulting changes in fractional gas concentrations (measured by the mass spectrometer). Data were transferred to a computer, which aligned the concentration and volume information to build a profile of each breath. Breath-by-breath alveolar gas exchange was calculated using algorithms (5). Heart rate was continuously monitored by three-lead electrocardiogram using PowerLab (ML132/ML880; ADInstruments, Colorado Springs, CO) on a separate computer.

Local muscle deoxygenation ([HHb]) of the quadriceps vastus lateralis muscle was measured using near-infrared spectroscopy (NIRS) (Oxiplex TS, Model 95205, ISS, Champaign, IL). The rigid sensor was placed on the belly of the muscle, midway between the lateral epicondyle and greater trochanter of the femur. Emitter fibers and detector were housed in a rigid, plastic sensor casing, ensuring that their positions remained fixed. The sensor was clamped on to a Velcro strap, which was wrapped around the participant’s leg to secure its position. Additionally, an optically dense black vinyl sheet was placed over top and around the sensor casing (minimizing the intrusion of extraneous light and loss of near infrared light). The thigh, with attached sensor and covering, was wrapped with an elastic bandage to minimize any movement of the sensor.

The theory of tissue spectroscopy has been previously described by Elwell (20). Briefly, the Oxiplex TS, providing multidistance frequency-domain spectroscopy, provided a single channel of eight laser diode light sources operating at two different wavelengths (690 nm and 828 nm, per wavelength). Light from the diodes was coupled to fibers in a photomultiplier tube and pulsed in rapid succession (110
MHz. The diodes were set at source-detector distances of 2.0, 2.5, 3.0, and 3.5 cm. The light was received by another detector fiber and then carried back and detected by a photodiode in the spectrometer. After the rigid sensor was secured on the leg, detector gain was adjusted for an optimal signal as the subject rested on the cycle ergometer (detector “gain” is dependent on detector bias voltage, where a larger voltage produces a larger signal). The OxiplexTS produced 25 measurements per second; averaged measurements were displayed and recorded at a frequency of 1 Hz. The concentration of NIRS-derived muscle [HHb] is presented as arbitrary units.

**Data Analysis**

Breath-by-breathe $\dot{V}O_2$ data were edited by removing aberrant data points that lay outside 4 SD of the local mean (37, 50). The remaining points that lay outside 4 SD of the local mean (37, 50). The ensemble-averaged [HHb] responses were ensemble averaged and further time averaged into 5-s bins to yield a single profile for each subject at each testing period. The phase I-phase II transition was identified as previously described (29, 52). On-transient phase II $\dot{V}O_2p$ kinetics were modeled using the following equation:

$$Y(t) = Y_{\text{BLN}} + \text{Amp}[1 - e^{(-\text{TD})t}]$$

where $Y(t)$ represents $\dot{V}O_2p$ at any time (t); $Y_{\text{BLN}}$ is the average $\dot{V}O_2p_{\text{ss}}$ measured during the period immediately before the change in WR; Amp (amplitude) is the steady-state increase in $\dot{V}O_2p$ above the baseline $\dot{V}O_2p$ ($\dot{V}O_2p_{\text{bas}}$); TD represents the time required to attain 63% of the steady-state amplitude; and TD is mathematically generated as the point at which the exponential model is predicted to intersect the baseline. Steady-state $\dot{V}O_2p_{\text{ss}}$ was established from data 60 s before each change in WR. Data were modeled from the phase I-phase II transition to the end of the 6 min exercise transition using Origin data fitting software (OriginLab). The 95% confidence interval for the estimated $\tau$ was determined following a preliminary fit with $Y_{\text{BLN}}$. Amp, and TD constrained to best fit values, with the $\tau$ allowed to vary. The mean response time (MRT) (39) of $\dot{V}O_2p$. described the overall time course of $\dot{V}O_2p$ during the exercise transition and was estimated using the function described in Eq. 1, but with inclusion of all $\dot{V}O_2p$ data from the onset of exercise, and the TD constrained to 0 s. This approach allowed for an estimate of the O2 deficit (52) for each WR transition. The O2 deficit provides information on nonoxidative energy transfer and was calculated as:

$$O_2 \text{ deficit (I)} = \text{MRT(s)} \times \Delta \dot{V}O_2p_{\text{ss}}/(l/min) \times \text{min}/60s$$

The functional gain of the fundamental $\dot{V}O_2p$ response was calculated as $\Delta \dot{V}O_2p/\Delta WR$ (mL·min$^{-1}$·W$^{-1}$), where $\dot{V}O_2p_{\text{ss}}$ is steady-state increase in $O_2$ uptake above baseline.

NIRS-derived data were time aligned and ensemble averaged into 5-s bins to yield a single profile for each subject. The time course of adjustment for [HHb] has been described to consist of a TD following the onset of exercise, with a subsequent “exponential-like” increase in the signal with time of exercise (17). The TD for the [HHb] ([HHb]$_{\text{TD}}$) response was determined visually using second-by-second data and was identified as time between the step increase in WR and the time when the [HHb] signal began to rise systematically above the nadir in the signal response (28). [HHb]$_{\text{TD}}$ was determined for individual trials and averaged for each LS, US, and SS, at each testing point, for each subject. The ensemble-averaged [HHb] response was modeled from [HHb]$_{\text{TD}}$ to 90 s of the transition, with a monoexponential function of the form in Eq. 1 to determine the time course of muscle [HHb] (τ[HHb]). A systematic decline in [HHb] did not occur before 90 s of the transition in any subjects. Baseline [HHb] was determined for each of the US and LS as the mean value in the 60 s before a transition. The effective $\tau$ ($\tau = [\text{HHb}]_{\text{TD}} + \tau[\text{HHb}]$) was calculated to describe the overall time course for muscle [HHb]. The steady-state value for [HHb] was determined as the end point for the fitting of the monoexponential function ([HHb]$_{\text{ss}}$). The steady-state increase in muscle deoxygenation in relation to the change in muscle $O_2$ utilization was calculated as $\Delta [\text{HHb}]_{\text{ss}}/\Delta \dot{V}O_2p$.

Second-by-second [HHb] and $\dot{V}O_2p$ data were normalized for each subject (0–100% of response). The normalized phase II $\dot{V}O_2p$, data were modeled (from 0–100% of response) using the $\tau$ values for individual subjects for LS, US, and SS transitions. The normalized adjustment of muscle $O_2$ utilization was estimated by shifting the normalized phase II $\dot{V}O_2p$, response profile toward the start of each of the LS, US, and SS step transitions (i.e., $t = 0$ for the LS and SS, and $t = 360$ s for the US) by a time corresponding to the estimated phase II TD for each transition, thereby making the normalized $\Delta \dot{V}O_2p$ at the immediate onset of the transition equal to “zero”. Data were further averaged into 5-s bins, and a $\Delta [\text{HHb}]-to-\Delta \dot{V}O_2p$, ratio ($\Delta [\text{HHb}]_1/\Delta \dot{V}O_2p$) was calculated for each of the LS, US, and SS exercise transitions, with a value of 1.0 corresponding to “steady-state” conditions for each of the $\Delta [\text{HHb}]$, and $\Delta \dot{V}O_2p$, variables. An “overshoot” in the $\Delta [\text{HHb}]$-to-$\Delta \dot{V}O_2p$, response profile was estimated by integrating the area bordered by the $\Delta [\text{HHb}]$-to-$\Delta \dot{V}O_2p$, profile and a ratio value equal to 1.0. The area was determined for the period 20–180 s of each transition, at each testing point for each subject, as this time window incorporated data beyond the NIRS-derived $[\text{HHb}]_{\text{TD}}$ and was of sufficient duration for the $\dot{V}O_2p$, and [HHb] signals to reach 100% of their steady-state amplitudes.

**Statistical Analysis**

Data are presented as means ± SD. Repeated measures ANOVA was used to determine statistical significance for the dependent variables: repeated factor of time (PRE, MID, POST, and VER), and between factor of group (HIT, CON). Where group × time interactions were identified, a Tukey post hoc analysis was used to identify differences between conditions. ANOVA was analyzed using SPSS version 17.0 (SPSS, Chicago, IL). Statistical significance was accepted at $P < 0.05$.

**RESULTS**

**Exercise Performance (TTF and Training)***

Relative training intensity remained constant across the 12 training sessions (~110% $\dot{V}O_2_{\text{max}}$), but training interval power output and exercise volume were increased ($P < 0.05$) during the final 6 training sessions (Table 1).

Exercise performance, measured as the TTF and peak WR during the RISE-105 test, increased ($P < 0.05$) with HIT over the course of the 12 training sessions, while CON group showed no changes (Table 2). The TTF when exercising at the constant WR$_{\text{max}}$ achieved in the initial (PRE) RI test increased by 85% ($P < 0.01$) from PRE to VER, while the WR$_{\text{max}}$ achieved during RI testing increased by ~17% ($P < 0.01$).

Table 1. Training protocol, average work, and time per training session

<table>
<thead>
<tr>
<th>HIT Sessions 1–6</th>
<th>HIT Sessions 7–12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relative intensity, %$\dot{V}O_2_{\text{max}}$</td>
<td>110</td>
</tr>
<tr>
<td>Absolute intensity, W</td>
<td>315 ± 45</td>
</tr>
<tr>
<td>Total exercise volume, kJ</td>
<td>168 ± 25</td>
</tr>
<tr>
<td>Total exercise time, min</td>
<td>9</td>
</tr>
<tr>
<td>Total time (rest + warm-up), min</td>
<td>23</td>
</tr>
</tbody>
</table>

HIT, high-intensity interval training; $\dot{V}O_2_{\text{max}}$, maximal $O_2$ uptake. Total exercise volume and relative intensity per session results are based on average work loads sustained during work intervals and do not include loadless cycling. *Significantly different ($P < 0.01$) from sessions 1–6.
V̇O₂p parameter estimates (no differences between the CON and HIT groups for any of the group at PRE and VER. Before the start of training, there were difference from HIT for corresponding testing point. B (Fig. 2) step transitions within the MOD domain for the HIT P 0.01) and 35% (P 2); the increases from PRE to POST training were 20% (the study. The V̇O₂ max and Lactate Threshold

Table 2. Training responses for aerobic and performance parameters assessed during ramp incremental and time-to-fatigue testing

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PRE</th>
<th>MID</th>
<th>POST</th>
<th>VER</th>
</tr>
</thead>
<tbody>
<tr>
<td>V̇O₂max, l/min</td>
<td>3.41 ± 0.54</td>
<td>3.61 ± 0.64*</td>
<td>3.88 ± 0.69†</td>
<td>4.00 ± 0.66‡</td>
</tr>
<tr>
<td>V̇O₂max, ml·kg⁻¹·min⁻¹</td>
<td>43 ± 4</td>
<td>46 ± 6*</td>
<td>49 ± 5†</td>
<td>50 ± 5†</td>
</tr>
<tr>
<td>WRmax, W</td>
<td>291 ± 45</td>
<td>316 ± 49*</td>
<td>336 ± 51†</td>
<td>340 ± 48‡</td>
</tr>
<tr>
<td>Estimated 쇄, l/min</td>
<td>1.99 ± 0.26</td>
<td>2.15 ± 0.34*</td>
<td>2.38 ± 0.46†</td>
<td>2.35 ± 0.45‡</td>
</tr>
<tr>
<td>Estimated 쇄, W</td>
<td>117 ± 21</td>
<td>133 ± 23*</td>
<td>158 ± 30†</td>
<td>158 ± 25‡</td>
</tr>
<tr>
<td>Time to fatigue, s</td>
<td>209 ± 51</td>
<td>283 ± 38*</td>
<td>370 ± 78‡</td>
<td>386 ± 63‡</td>
</tr>
<tr>
<td>V̇O₂max, l/min</td>
<td>3.95 ± 0.37‡</td>
<td>3.89 ± 0.35</td>
<td>3.89 ± 0.41</td>
<td>3.98 ± 0.38</td>
</tr>
<tr>
<td>V̇O₂max, ml·kg⁻¹·min⁻¹</td>
<td>48 ± 4‡</td>
<td>47 ± 4</td>
<td>48 ± 5</td>
<td>49 ± 4</td>
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<tr>
<td>WRmax, W</td>
<td>319 ± 38</td>
<td>321 ± 44</td>
<td>320 ± 48</td>
<td>318 ± 42</td>
</tr>
<tr>
<td>Estimated 쇄, l/min</td>
<td>2.15 ± 0.21</td>
<td>2.16 ± 0.16</td>
<td>2.16 ± 0.11</td>
<td>2.15 ± 0.11</td>
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<tr>
<td>Estimated 쇄, W</td>
<td>116 ± 18</td>
<td>122 ± 17</td>
<td>129 ± 23‡</td>
<td>128 ± 15‡</td>
</tr>
<tr>
<td>Time to fatigue, s</td>
<td>207 ± 58</td>
<td>207 ± 292</td>
<td>198 ± 283</td>
<td>195 ± 32‡</td>
</tr>
</tbody>
</table>

Values are means ± SD. CON, control; WRmax, work rate (WR) at maximal O2 uptake; 쇄, lactate threshold; PRE, pretraining; MID, midtraining; POST, posttraining; VER, verification. *Significant (P < 0.05) difference from PRE. †Significant (P < 0.05) difference from PRE and MID. ‡Significant (P < 0.05) difference from HIT for corresponding testing point.

during this same period. Improvements for both TTF and RI WRmax were observed from PRE to MID (P < 0.05), and MID to POST (P < 0.05); no differences occurred between POST and VER. There were no differences present at baseline (PRE) TTF or WRmax between the HIT and CON groups.

V̇O₂max and Lactate Threshold

In the HIT group, absolute V̇O₂max increased by 6% (P < 0.05) from PRE to MID, and by 11% (P < 0.05) from MID to VER, while relative V̇O₂max increased by 7 and 9%, respectively, for these same training periods (P < 0.05) (Table 2); V̇O₂max remained unchanged in CON at all testing times.

In the HIT group, the WR and V̇O₂p corresponding to the estimated 쇄 increased from PRE to MID (P < 0.05), and MID to POST (P < 0.05), with no changes POST to VER (see Table 2); the increases from PRE to POST training were 20% (P < 0.01) and 35% (P < 0.01), respectively, relative to V̇O₂p and WR (Table 2). The 쇄 remained unchanged in CON throughout the study. The 쇄 was different between groups at the start of training.

During PRE, the end-step, V̇O₂p ss for the LS and US represented 72 and 98% of PRE 쇄, and 42 and 58% of the PRE V̇O₂ max. In the US, the slope of the V̇O₂p-time relationship during the latter part of the transition was not different from “zero”, indicating the attainment of steady-state conditions with no evidence of a “slow-component”. This confirms that the exercise intensity was confined within the MOD domain during the US.

V̇O₂p Kinetics

Tables 3 (LS, US) and 4 (SS) outline V̇O₂p kinetics parameters for both HIT and CON groups, at PRE, MID, POST, and VER testing points. Figure 2 shows the group mean, ensemble-averaged V̇O₂p response profiles for the DS (Fig. 2A) and SS (Fig. 2B) step transitions within the MOD domain for the HIT group at PRE and VER. Before the start of training, there were no differences between the CON and HIT groups for any of the V̇O₂p parameter estimates (τV̇O₂p, V̇O₂p ss, V̇O₂p bsl, V̇O₂p amplitude, TD), the V̇O₂p gain (ΔV̇O₂p ss/ΔWR), or O2 deficit. Changes in LS or US V̇O₂p kinetics parameters were not seen in CON across testing periods.

US vs. LS. Despite the same ΔWR in the US and US, the τV̇O₂p (P < 0.05) and V̇O₂p gain (P < 0.01) were greater in the US than in the LS at all time points for both the HIT and CON groups. In the HIT group, the O2 deficit was ~60% greater in the US than in the LS at PRE (423 ± 125 vs. 265 ± 58 ml; P < 0.01). At VER, however, the O2 deficit was greater in the US than in the LS (303 ± 100 vs. 246 ± 73 ml), consequent to a greater reduction (P < 0.05) of the O2 deficit in the US. In the CON group, the O2 deficit was greater in US than in LS, and there were no changes with time.

LS. In the HIT group, the τV̇O₂p was reduced by 38% from PRE to POST (24 ± 6 and 15 ± 2 s, respectively; P < 0.01). No differences in τV̇O₂p occurred between POST (15 ± 2 s) and VER (14 ± 3 s). TD in the HIT group increased from PRE to POST (13 ± 4 and 20 ± 2 s, respectively; P < 0.05), with no differences occurring between POST and VER. No changes in V̇O₂p ss, V̇O₂p bsl, V̇O₂p gain, or O2 deficit occurred as a consequence of the training program. No significant changes occurred over time in CON subjects.

US. In the HIT group, the τV̇O₂p was reduced by 38% from PRE to POST (45 ± 5 and 28 ± 7 s, respectively; P < 0.01). No change in τV̇O₂p occurred between POST and VER. The HIT group TD increased from PRE to POST (5 ± 10 and 11 ± 6 s, respectively; P < 0.05), with no differences occurring between POST and VER. O2 deficit was reduced by 22% from PRE to POST (423 ± 125 and 329 ± 76 ml, respectively; P < 0.05), with no differences between POST and VER. No changes in V̇O₂p ss, V̇O₂p bsl, V̇O₂p gain, or O2 deficit occurred as a consequence of the training program. There were no changes in the CON group over time.

SS. In the HIT group, the τV̇O₂p was reduced by 41% from PRE to VER (32 ± 7 and 19 ± 3 s, respectively; P < 0.01). The O2 deficit decreased by 20% in the HIT group (661 ± 110 and 527 ± 102 ml, respectively; P < 0.01). There were no changes in V̇O₂p ss, V̇O₂p bsl, V̇O₂p gain, or O2 gain. In the CON group, τV̇O₂p, V̇O₂p gain, and O2 deficit remained
unchanged over time; however, both VO2p,bsl and VO2p,ss were reduced (P < 0.05) at VER.

NIRS-Derived [HHb] Kinetics and Muscle Oxygenation Parameters

Tables 5 (LS, US) and 6 (SS) show [HHb] kinetics parameters for HIT and CON groups, at PRE, MID, POST, and VER time points. The τ'[HHb] was consistently larger in the US compared with the LS, in both the HIT and CON groups (P < 0.05 at all time points). Despite a significant lowering of τVO2p following 12 sessions of HIT, there was no change in the

Table 3. VO2p kinetics parameters for LS and US moderate-intensity exercise transitions at PRE, MID, POST, and VER testing points

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PRE</th>
<th>MID</th>
<th>POST</th>
<th>VER</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO2p,bsl, l/min</td>
<td>1.00 ± 0.09</td>
<td>1.50 ± 0.29</td>
<td>1.09 ± 0.12</td>
<td>0.99 ± 0.10</td>
</tr>
<tr>
<td>VO2p,ss, l/min</td>
<td>1.44 ± 0.14</td>
<td>1.96 ± 0.24</td>
<td>1.45 ± 0.15</td>
<td>1.97 ± 0.28</td>
</tr>
<tr>
<td>Amp, l/min</td>
<td>0.44 ± 0.12</td>
<td>0.52 ± 0.11</td>
<td>0.47 ± 0.13</td>
<td>0.52 ± 0.13</td>
</tr>
<tr>
<td>TD, s</td>
<td>13 ± 4</td>
<td>5 ± 10</td>
<td>16 ± 5*</td>
<td>6 ± 6*</td>
</tr>
<tr>
<td>τVO2p, s</td>
<td>24 ± 6</td>
<td>45 ± 5*</td>
<td>18 ± 3*</td>
<td>35 ± 8*</td>
</tr>
<tr>
<td>C50,s</td>
<td>5 ± 1</td>
<td>5 ± 2</td>
<td>4 ± 2</td>
<td>5 ± 2</td>
</tr>
<tr>
<td>ΔVO2p,ml-min^{-1}-W^{-1}</td>
<td>8.4 ± 1.2</td>
<td>10.0 ± 1.08</td>
<td>8.9 ± 1.3</td>
<td>9.9 ± 1.3</td>
</tr>
<tr>
<td>O2 deficit, ml</td>
<td>265 ± 58</td>
<td>423 ± 125</td>
<td>249 ± 89</td>
<td>347 ± 117</td>
</tr>
</tbody>
</table>

Values are means ± SD. LS, lower step; US, upper step; VO2p, pulmonary O2 uptake; VO2p,bsl, baseline VO2p; VO2p,ss, steady-state VO2p; Amp, amplitude of VO2p response; TD, time delay; τVO2p, time constant for VO2p response; C50, 95% confidence interval of τVO2p, ΔVO2p, ΔWR, functional gain. *Significant (P < 0.05) difference from PRE. †Significant (P < 0.05) difference from PRE and MID. §Significant (P < 0.05) difference from LS. ¶Significant (P < 0.05) difference from HIT for corresponding testing point.

adjustment of [HHb] (τ[HHb] and τ'[HHb]) in the LS, US, and SS transitions. The CON group also did not experience any changes over time in τ[HHb] and τ'[HHb]. In both the HIT and CON groups, there were no changes in [HHb] kinetics parameters ([HHb]b, [HHb]s, [HHb]e, and [HHb]n) end-step, and Δ[HHb]/ΔVO2p,ss for LS, US, or SS at any of the training-related testing times.

Figure 3 shows ensemble-averaged absolute and normalized [HHb] responses to MOD step transitions (LS, US, and SS) in the HIT group, at PRE and VER. As evidenced in Fig. 3a, the [HHb] profile exhibited a transient overshoot during the LS before training that was not seen during VER. This overshoot was observed in six of eight HIT subjects and was not evident at MID, POST, or VER; in CON, the overshoot was observed in three of five subjects at both PRE and VER.

Normalized Δ[HHb]/ΔVO2p

Figure 4 shows the mean profile for Δ[HHb]/ΔVO2p in LS and US for the HIT group. At PRE, the Δ[HHb]/ΔVO2p for LS and US were 1.05 and 1.20, respectively (both significantly different from 1.0, P < 0.05); by MID this “overshoot” was no longer apparent in either the LS or US (i.e., the ratio was no longer different from 1.0). These ratios remained at ~1.0 throughout the remainder of the study (MID to VER), consistent with steady-state conditions for both variables. Similarly, Fig. 5 illustrates mean profile for Δ[HHb]/ΔVO2p during SS at PRE and VER in the HIT group. Before training (PRE), the overall ratio from 20 to 180 s of the transition averaged 1.09 (significantly different from 1.0, P < 0.05); following training (VER), no overshoot was observed.

DISCUSSION

This study investigated the effects of 12 sessions of HIT on phase II VO2p and muscle deoxygenation ([HHb]) kinetics...
HIT Speeds $\dot{V}O_2p$ Adjustments from Elevated Metabolic Rates • Williams AM et al.

During transitions to constant-load, MOD exercise initiated from a lower (LS) and higher (US) baseline WR and metabolic rate. In agreement with previous reports (9, 32, 40, 55), we observed that, for similar increments in WR ($\Delta$WR), there was a slower adjustment of both $\dot{V}O_2p$ and [HHb] in the US compared with the LS, as well as a greater steady-state $O_2$ cost of exercise (gain, $\Delta$O2p/ΔWR) in the US, reflecting lower metabolic efficiency. As hypothesized, there was a speeding of $\dot{V}O_2p$ kinetics in both the LS and US following 4 wk (i.e., 12 sessions) of HIT training. The absolute reduction of $\dot{V}O_2p$ in the US ($\sim$20 s) was greater than that seen in the LS ($\sim$10 s); however, these changes represented a similar relative speeding in both steps ($\sim$40%). Despite the training-induced speeding of $\dot{V}O_2p$, the adjustment of muscle deoxygenation ([HHb]), reflecting fractional $O_2$ extraction and the ratio of microvascular blood flow-to-muscle $O_2$ utilization, did not change. The observed speeding of $\dot{V}O_2p$ kinetics without changes in the adjustment muscle deoxygenation agree with the findings of McKay et al. (41), who reported a faster adjustment of $\dot{V}O_2p$ in transitions from 20 W to a WR corresponding to 90% $\theta_t$ (performed as a SS). Overall, these findings suggest that the training-induced adjustments of $\dot{V}O_2p$ (and muscle $O_2$ utilization) are independent of the baseline metabolic rate, and also that microvascular blood flow kinetics matched those of muscle $O_2$ utilization. To our knowledge, this is the first study to observe training-induced speeding of $\dot{V}O_2p$ kinetics initiated from a lower and higher baseline metabolic rate within the MOD domain.

**Effect of HIT on $\dot{V}O_2p$ and [HHb] Kinetics**

Following 12 sessions of HIT, $\tau\dot{V}O_2p$ was reduced by $\sim$40% when the exercise transition was performed as a SS of 20 W to $\sim$90% $\theta_t$; $\dot{V}O_2p$ decreased from 32 to 19 s. These changes in $\tau\dot{V}O_2p$ mirror those seen by McKay et al. (41), where eight sessions of HIT resulted in a 12-s reduction in $\tau\dot{V}O_2p$ during SS transitions. In agreement with McKay et al. (41), the $\dot{V}O_2p$ gain was not affected by training, remaining at $\sim$9–10 ml-min$^{-1}$-W$^{-1}$. As a consequence of similar $\dot{V}O_2p$ gain but faster $\dot{V}O_2p$ kinetics, the $O_2$ deficit was reduced from 661 ml (PRE) to 527 ml (VER). In agreement, Bailey et al. (3) and Berger et al. (7) used a short-term interval training model and reported reductions in SS-MOD $\tau\dot{V}O_2p$ from 28 to 21 s (3) and 32 to 21 s (7).

As previously reported (9, 32, 40, 55), the adjustment of $\dot{V}O_2p$ was slower, and the $\dot{V}O_2p$ gain was greater when the exercise transition was initiated from an elevated baseline metabolic rate (US vs. LS). There was a speeding of $\dot{V}O_2p$ kinetics following 12 sessions of HIT during transitions in both LS and US, as $\tau\dot{V}O_2p$ decreased by $\sim$20 s (US) and $\sim$10 s (LS). Several factors have been suggested to contribute to the slowed adjustment of $\dot{V}O_2p$ within the upper region of the MOD domain: $O_2$ availability to the working muscle (32, 40), hierarchical recruitment of additional muscle fibers (9), and intramuscular energetic state (8). Prior work has described slowed adjustments of femoral artery (bulk) blood flow and $O_2$ delivery in the US compared with LS, suggesting that the greater $\tau\dot{V}O_2p$ in the US occurred as a consequence of a slowed adjustment of bulk $O_2$ delivery (32). Recently Bowen et al. (8) suggested that, when exercise transitions are initiated from an elevated baseline metabolic rate, factors other than slowed blood flow adjustment and intrinsic metabolic properties of newly recruited muscle fibers could account for the slower adjustment of $\dot{V}O_2p$ in the US compared with LS. They proposed that, during transitions from a raised metabolic rate, a lowered metabolic energy state (i.e., elevated muscle concentrations of free ADP and $P_i$; lower ATP) and lowered free energy release from ATP hydrolysis (“less negative” $\Delta G_{ATP}$) would require greater activation of mitochondrial oxidative energy production and muscle $O_2$ utilization, compared with when exercise was initiated from a lower metabolic rate (8, 24). While this hypothesis remains to be tested, the findings do suggest that slowed $\dot{V}O_2p$ kinetics in the upper region of the MOD domain likely are a consequence of limitations imposed...
by a combination of adjustments to metabolism, blood flow, and O₂ delivery.

Despite the faster HIT-induced adjustments in Vₑ₂p (and muscle O₂ utilization) in the LS, US, and SS, the overall adjustment (TD + τ) of muscle deoxygenation ([HHb]) was unchanged over the course of the 12 HIT sessions. These results agree with previous studies from our laboratory (41, 44), but differ from those of Bailey and coworkers (3), who reported faster kinetics for both Vₑ₂p and muscle deoxygenation during transitions into MOD after repeated sprint training. In the present study, the speeding of the Vₑ₂p kinetics without accompanying changes in muscle deoxygenation kinetics suggests that the adjustment of microvascular blood flow also became faster with training. Consistent with this is the transient overshoot in the profile of the normalized Δ[HHb]/ΔVₑ₂p, which reflects a transient decrease in microvascular blood flow-to-muscle O₂ utilization and increased fractional muscle O₂ extraction. This overshoot was present in the exercise transition at PRE, but not seen at MID, POST, and VER. Using a lower intensity END training program, Murias et al. (43) reported a reduction in the normalized Δ[HHb]/ΔVₑ₂p overshoot after 3-wk training, and its elimination after 6-wk training, with these changes being closely associated with reductions in τVₑ₂p (see Fig. 4 in Ref. 43). In the present study, however, Vₑ₂p kinetics continued to speed over the entire 12 HIT sessions, while the reduced Δ[HHb]/ΔVₑ₂p overshoot was seen only between PRE and MID. These observations suggest that training-induced adaptations contributing to the elimination of an overshoot do not entirely account for the speeding of Vₑ₂p kinetics seen throughout the HIT program. Possible mechanisms for the speeding of Vₑ₂p kinetics are discussed below.

Potential Mechanisms Contributing to Speeding of Vₑ₂p Kinetics

Table 6. [HHb] kinetics parameters for SS moderate-intensity exercise transitions at PRE, MID, POST, and VER testing points

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PRE SS</th>
<th>VER SS</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>[HHb]₀sb, AU</td>
<td>27.0 ± 5.2</td>
<td>30.5 ± 2.4</td>
</tr>
<tr>
<td>[HHb]₀ss, AU</td>
<td>39.6 ± 10.3</td>
<td>44.6 ± 7.1</td>
</tr>
<tr>
<td>[HHb] End-Stp, AU</td>
<td>40.4 ± 9.7</td>
<td>45.5 ± 7.3</td>
</tr>
<tr>
<td>TD, s</td>
<td></td>
<td></td>
</tr>
<tr>
<td>[HHb]</td>
<td>10 ± 2</td>
<td>10 ± 2</td>
</tr>
<tr>
<td>τ[HHb], s</td>
<td>10 ± 4</td>
<td>11 ± 6</td>
</tr>
<tr>
<td>τ'[HHb], s</td>
<td>19 ± 5</td>
<td>21 ± 7</td>
</tr>
<tr>
<td>Δ[HHb]₀/ΔVₑ₂pₓₓ</td>
<td>15 ± 9</td>
<td>17 ± 10</td>
</tr>
</tbody>
</table>

CON

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PRE SS</th>
<th>VER SS</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>[HHb]₀sb, AU</td>
<td>26.7 ± 3.8</td>
<td>27.0 ± 8.3</td>
</tr>
<tr>
<td>[HHb]₀ss, AU</td>
<td>38.9 ± 8.4</td>
<td>36.0 ± 12.1</td>
</tr>
<tr>
<td>[HHb] End-Stp, AU</td>
<td>39.9 ± 9.2</td>
<td>35.3 ± 8.5</td>
</tr>
<tr>
<td>TD, s</td>
<td></td>
<td></td>
</tr>
<tr>
<td>[HHb]</td>
<td>8 ± 3</td>
<td>8 ± 2</td>
</tr>
<tr>
<td>τ[HHb], s</td>
<td>12 ± 3</td>
<td>12 ± 3</td>
</tr>
<tr>
<td>τ'[HHb], s</td>
<td>20 ± 2</td>
<td>20 ± 2</td>
</tr>
<tr>
<td>Δ[HHb]₀/ΔVₑ₂pₓₓ</td>
<td>13 ± 4</td>
<td>14 ± 10</td>
</tr>
</tbody>
</table>

Values are means ± SD. No significant changes occurred over time (P > 0.05). No significant differences existed among any variable between HIT and CON.
Training adaptations in the adjustment of microvascular blood flow likely contributed, in part, to the speeding of $\dot{V}O_2p$ kinetics, especially early in the training period (between the PRE and MID), as evidenced by the elimination of a $\Delta [HHb]/\Delta \dot{V}O_2p$ overshoot by MID. However, adaptations to microvascular blood flow dynamics likely continued with training because, as $\tau_{\dot{V}O_2p}$ continued to fall significantly in both LS (by 22%) and US (by 29%) from MID to VER (Table 3), the $\tau'_{[HHb]}$ did not change significantly (Table 5). This suggests that the dynamic relationship between microvascular blood flow and muscle $O_2$ utilization, and thus muscle deoxygenation, was maintained. Green et al. (26) reported increases in muscle capillarity with a 6-day training model (at $\sim 67\%$ peak $O_2$ uptake for 2 h/day): increases in capillary contacts per muscle fiber area and capillary density (without changes in muscle oxidative potential) were reported after 3 training days, while increases in capillary contacts per muscle fiber, capillary contacts per muscle fiber area, and capillary density were reported after 6 training days. These increases in muscle capillarity would be expected to increase the functional capillary surface area and thus the potential for microvascular convective and diffusive $O_2$ delivery to the muscle mitochondria. Using a similar training program, Shoemaker et al. (54) reported faster leg conduit artery blood velocity and vascular conductance kinetics after 10 training days, while Phillips et al. (47) reported faster $\dot{V}O_2p$ kinetics after 4 training days, before any increases in muscle oxidative potential.

In the present study, the continued speeding of $\dot{V}O_2p$ kinetics seen late in the HIT program likely suggest that mechanisms in addition to microvascular blood flow adaptations may have gradually contributed to the training adaptations (between MID and VER). Using an intense, intermittent sprint-training protocol, Gibala and coworkers (10, 12, 22, 23) observed increases in maximal activities of cytochrome oxidase (COX) (22), citrate synthase (12), and $\beta$-hydroxyacyl CoA dehydrogenase (12); pyruvate dehydrogenase (12) and COX II and COX IV (10, 22) protein content; peroxisome proliferator-activated receptor-$\gamma$ coactivator-1 mRNA (23) and protein content (12); and phosphorylation of AMP kinase (22), p38 mitogen-activated protein kinase (22), and calcium/calmodulin-dependent protein kinase (22). Altogether, alterations in these markers reflect increases in cell signaling leading to mitochondrial biogenesis and increases in muscle oxidative capacity, after only one to six training sessions. Given the HIT program used in the present study, similar training-induced adaptations might be expected, although the somewhat lower training intensity used in the present study (repeated exercise bouts at 110% of PRE $WR_{max}$ vs. repeated “all-out” exercise bouts) may have induced a differing rate of metabolic adaptation. A higher oxidative capacity of the trained muscle thus may have contributed to the continued speeding of $\dot{V}O_2p$ kinetics seen in the present study.

**Effect of HIT on $\dot{V}O_2_{max}$ Estimated Lactate Threshold and Exercise Performance**

Improvements in $\dot{V}O_2_{max}$ and exercise performance (TTF and $WR_{max}$) occurred following 12 sessions of HIT, demonstrating the effectiveness of this training program in improving...
training status and overall fitness. In the present study, improvements in $V_{O2\max}$ (both relative and absolute), WR$_{max}$ during RI testing, and the WR and $V_{O2p}$ corresponding to $\theta_L$, occurred from PRE to MID testing, and further from MID to POST. The greatest improvements occurred in TTF, with an 85% increase (209 to 386 s) following 12 sessions of training. These findings agree with the findings of McKay et al. (41), whose similar HIT protocol promoted improvements in relative (but not absolute) $V_{O2\max}$, WR$_{max}$ during RI testing, and the WR and $V_{O2p}$ corresponding to $\theta_L$. Additionally, several investigations employing short-term HIT [2-wk (3, 22) and 6-wk (10) programs] have reported improvements in exercise performance, with reduced time to complete time trials (10, 22), increased power output during the time trial (22), and greater TTF during a severe-intensity exercise test (3).

The improvements in $V_{O2\max}$ (both relative and absolute), exercise tolerance, and performance in the present study likely occurred, at least in part, due to improved muscle oxidative capacity, faster adjustment of mitochondrial oxidative metabolism, and reduced reliance on substrate-level phosphorylation (possibly with slower rates of depletion of “anaerobic” energy stores, glycogen, and PCR; slower rates of accumulation of free ADP, Pi, and H$^+$; and maintenance of a higher $\Delta G_{ATP}$), enhanced buffering, and transport of fatigue-inducing metabolites. Increases in resting muscle glycogen and glycolytic capacity have also been associated with HIT, and these adaptations may also have contributed to the improvements in performance (11).

**Limitations**

In the present study, there is some uncertainty when using the noninvasive measure of phase II $\Delta V_{O2p}$ profile as a proxy for muscle O$_2$ utilization in the calculation of the $\Delta [HHb]/\Delta V_{O2p}$. It is possible that, by eliminating the phase I $V_{O2p}$ response and focusing only on the phase II response, this approach may not reflect the actual response at the muscle. Krustup and colleagues (35) reported that, after the onset of exercise, there is a shortening of the muscle capillary-to-lung transit time, resulting in deoxygenated blood from the exercising muscle appearing at the lung after a delay of $\sim 10$–15 s. They concluded, however, that, despite the early arrival of deoxygenated blood from contracting muscle, its contribution to the $V_{O2p}$ response was “quantitatively small” and did not distort the fidelity of the relationship between muscle O$_2$ uptake and $V_{O2p}$ kinetics. Additionally, it is reasonable to assume that the increase in muscle ATP turnover and initial activation and increase in the rate of muscle O$_2$ utilization (and $V_{O2p}$) should coincide with, or occur very soon after, the transition to a higher WR taking place (i.e., with minimal TD) (for review, see Ref. 51), and that the increase in muscle O$_2$ utilization would be “exponential-like” (with a time course reflecting the phase II $V_{O2p}$ response) (25, 35, 51). Therefore, we believe that this adjustment then provides a reasonable estimation of events occurring in the recruited muscle at the immediate onset of the exercise transition.

*References*

- McKay et al. (41)
- Krustup and colleagues (35)
- Ref. 51

**Fig. 5.** Left: HIT group mean profiles for adjustments of [HHb] and $V_{O2p}$ (modeled and averaged from individual subject $r$) in the initial 180 s of SS moderate-intensity exercise transitions, shown for PRE and POST (at VER). *Significant difference of overall $\Delta [HHb]/\Delta V_{O2p}$ (20–180 s) from 1.0 ($P < 0.05$). Right: HIT group mean profiles for the relative adjustment of [HHb]/$V_{O2p}$ in the initial 180 s of SS moderate-intensity exercise transitions, shown for PRE and POST (at VER).
Additionally, direct measures of HIT-induced adaptations in the muscle were not made, and we can only speculate on adaptations based on published literature. However, given the low-volume, high-intensity nature of the HIT program used in this study was modeled after Gibala and coworkers (10–13, 22, 23) where muscle biopsy data are presented, we are confident that similar muscle adaptations will occur. These adaptations require further study.

Conclusions

In summary, the results of the present study demonstrated that low-volume HIT (12 training sessions over 4 wk; 8–12 intervals per session at 110% VO2max; 60 s exercise: 60 s recovery) was associated with a speeding of VO2p kinetics during transitions into the MOD exercise domain, regardless of whether the exercise transition was initiated from a low or elevated baseline metabolic rate. Also, muscle deoxygenation kinetics were unchanged, despite the faster rate adjustment of VO2p. While the relative speeding of the LS and US did not differ, the absolute speeding of the US was twice that of the LS following the training program. Speeding of VO2p kinetics in both the US and LS occurred without parallel changes in the adjustment of muscle deoxygenation, suggesting a role for improved coordination of microvascular blood flow with O2 utilization, particularly in the early stages of training, with training-induced adaptations in muscle oxidative capacity likely contributing more with the duration of HIT. While the specific physiological mechanisms controlling the adjustment of O2 uptake for each of the lower and upper regions of the MOD exercise domain remain elusive, a prominent body of evidence suggests that training-induced improvements in \( \tau \text{VO2p} \) for LS and US transitions may involve metabolic re-modeling of the active muscle.

ACKNOWLEDGMENTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


REFERENCES


