Influence of repeated sprint training on pulmonary O₂ uptake and muscle deoxygenation kinetics in humans

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Bailey SJ, Wilkerson DP, DiMenna FJ, Jones AM. Influence of repeated sprint training on pulmonary O₂ uptake and muscle deoxygenation kinetics in humans. J Appl Physiol 106: 1875–1887, 2009. First published April 2, 2009; doi:10.1152/japplphysiol.00144.2009.—We hypothesized that a short-term training program involving repeated all-out sprint training (RST) would be more effective than work-matched, low-intensity endurance training (ET) in enhancing the kinetics of oxygen uptake (VO₂) and muscle deoxygenation ([deoxyhemoglobin concentration ([HHb])] following the onset of exercise. Twenty-four recreationally active subjects (15 men, mean ± SD: age 21 ± 4 yr, height 173 ± 9 cm, body mass 71 ± 11 kg) were allocated to one of three groups: RST, which completed six sessions of four to seven 30-s RSTs; ET, which completed six sessions of work-matched, moderate-intensity cycling; and a control group (CON). All subjects completed moderate-intensity and severe-intensity “step” exercise transitions before (Pre) and after the 2-wk intervention period (Post). Following RST, [HHb] kinetics were speeded, and the amplitude of the [HHb] response was increased during both moderate and severe exercise (P < 0.05); the phase II VO₂ kinetics were accelerated for both moderate (Pre: 28 ± 8, Post: 21 ± 8 s; P < 0.01) and severe (Pre: 29 ± 5, Post: 23 ± 5 s; P < 0.05) exercise; the amplitude of the VO₂ slow component was reduced (Pre: 0.52 ± 0.19, Post: 0.40 ± 0.17 l/min; P < 0.01); and exercise tolerance during severe exercise was improved by 53% (Pre: 700 ± 234, Post: 1,074 ± 431 s; P < 0.01). None of these parameters was significantly altered in the ET and CON groups. Six sessions of RST, but not ET, resulted in changes in [HHb] kinetics consistent with enhanced fractional muscle O₂ extraction, faster VO₂ kinetics, and an increased tolerance to high-intensity exercise.

The commencement of muscular exercise mandates an immediate increase in ATP turnover in the recruited myocytes. Transiently, the bulk of this additional energy requirement is met through intramuscular phosphocreatine (PCr) degradation and the anaerobic catabolism of glycogen, and, as such, an “oxygen deficit” is incurred (42). Simultaneously, oxygen uptake (VO₂) rises in an exponential fashion, such that oxidative phosphorylation makes a progressively greater contribution to ATP resynthesis as exercise proceeds. Pulmonary VO₂ reaches a “steady-state” within 2–3 min of the onset of moderate-intensity exercise [below the gas exchange threshold (GET); Refs. 67, 68]; during >GET exercise, however, a “steady state” is either delayed (for heavy exercise performed below the critical power) or is unattainable (for severe exercise performed above the critical power) due to the emergence of an additional VO₂ “slow component” (68). This slow component rise in VO₂ is associated with a commensurate fall in muscle PCr concentration ([PCr]) (57, 58) and greater glycogen utilization (43). Thus features of the dynamic adjustment of VO₂ following the onset of exercise have been suggested to be related to the process of muscular fatigue (9, 37, 66). Interventions that accelerate the phase II VO₂ kinetics and/or reduce the amplitude of the VO₂ slow component should theoretically result in enhanced exercise tolerance (36, 66).

A potent stimulus to enhance the dynamics of VO₂ during exercise is endurance training (ET). Indeed, a period of ET has been shown to accelerate phase II VO₂ kinetics (4, 18, 31, 43) and reduce the amplitude of the VO₂ slow component (4, 10, 11, 59), adaptations that can be discerned after only a few days of training (54, 71). These adaptations might, therefore, account, at least in part, for the enhanced exercise tolerance typically observed after a period of ET (e.g., Refs. 13, 18, 35). However, whether there is an “optimal” training strategy to elicit improvements in VO₂ kinetics is presently unclear. Berger et al. (4) reported that adaptations in VO₂ kinetics were similar when the ET consisted of continuous, low-intensity exercise or as a series of repeated high-intensity exercise bouts interspersed with a short recovery period. In contrast, the data of Daussin et al. (13) suggest that interval training may be a more efficacious intervention for accelerating phase II VO₂ kinetics compared with continuous lower intensity ET.

A novel, and time-efficient, approach to elicit the adaptations associated with ET has recently been introduced (6–8, 22). Specifically, six sessions of repeated sprint training (RST) comprising four to six repeats of an all-out 30-s Wingate cycle test, separated by 4-min recovery, induced strikingly similar increases in muscle oxidative enzyme activity, buffering capacity, glycogen content, and exercise tolerance to those observed following six sessions of 90–120 min of continuous cycling at ~65% peak VO₂ (VO₂peak) (22). Earlier work revealed that this RST protocol resulted in increases in cycle endurance capacity (time to exhaustion at ~80% VO₂peak), citrate synthase activity (8), and reductions in glycogenolysis and lactate accumulation (6), compared with a control group (CON), who remained sedentary. Moreover, RST and ET (40- to 60-min cycling continuously at 65% VO₂peak) resulted in similar reductions in glycogen and PCr degradation during 1 h of exercise at 65% VO₂peak (7, 23). Collectively, these findings indicate that RST is as effective as more prolonged ET for increasing the capacity for oxidative metabolism, sparing the contribution of substrate level phosphorylation to energy turnover, and enhancing exercise tolerance. However, to what extent RST enhances VO₂ kinetics (by speeding the phase II VO₂ response and/or reducing the VO₂ slow component) relative to traditional ET, and to what extent any improvements in VO₂ kinetics are related to enhanced exercise tolerance following RST, are presently unclear.
While it has been known for some time that ET subjects exhibit rapid VO2 response dynamics (3, 28, 40, 56), the factors that regulate the dynamic VO2 response during exercise continue to be debated (37, 55, 62). Depending on the circumstances, VO2 kinetics might be principally limited by muscle O2 delivery or its distribution, or by an intrinsic inertia of the muscle metabolic machinery (37, 55, 62). Exercise training results in rapid adaptations in muscle blood flow (46, 61) and mitochondrial enzyme activity (6 – 8, 22, 43, 54), but information on the impact of these changes on the faster VO2 kinetics following training is presently limited. The available evidence indicates that, while training results in enhanced muscle blood flow and muscle O2 extraction, the latter might be particularly important in the speeding of VO2 kinetics observed following training during high-intensity exercise (43). The deoxyhemoglobin/myoglobin concentration ([HHb]) signal derived from near-infrared spectroscopy (NIRS) measurements reflects the balance between O2 delivery and O2 utilization in the field of interrogation and has been used to provide a noninvasive estimate of fractional O2 extraction in the microcirculation during exercise (16, 17, 21, 27, 34, 41). The assessment of [HHb] kinetics (to reflect muscle fractional O2 extraction; Refs. 26, 27) and heart rate (HR) kinetics (as a crude estimate of muscle blood flow kinetics; Ref. 50) might, therefore, facilitate investigation of the mechanisms by which VO2 kinetics are altered with different types of training. Unaltered muscle [HHb] dynamics [as assessed by changes in the time delay (TD), time constant (τ), and amplitude of the response] following training, despite evidence of faster VO2 kinetics, would be interpreted to indicate that local muscle blood flow remained well matched to the increased muscle O2 utilization following training. A longer TD and/or τ or a smaller [HHb] amplitude following training would indicate that microvascular blood flow increased more than muscle O2 extraction, whereas a shorter TD and/or τ or a greater [HHb] amplitude would indicate that O2 extraction had increased more than local blood flow. The direction of any changes in [HHb] kinetics as a consequence of training, therefore, has implications for the mechanism(s) by which VO2 kinetics are accelerated.

The purpose of the present study was, therefore, to assess the effect of work-matched RST and ET on the kinetics of VO2, HR, and muscle deoxygenation during moderate- and severe-intensity exercise in recreationally active subjects, as well as the impact of these training methods on severe-intensity exercise tolerance. We reasoned that simultaneous assessment of the kinetics of VO2, HR, and [HHb] might provide insight into the mechanistic bases (i.e., increased muscle blood flow and/or metabolic adjustments) for the enhanced VO2 kinetics observed following exercise training. We hypothesized that both the phase II VO2 τ and the VO2 slow-component amplitude would be reduced, and exercise tolerance enhanced, by training in both groups. We also hypothesized that the magnitude of these changes would be greater for the RST group, particularly during severe-intensity exercise, consequent to a greater improvement in muscle O2 extraction (as estimated by changes in [HHb]) kinetics.

**METHODS**

**Subjects**

Twenty-four healthy subjects (15 male, mean ± SD: age 21 ± 4 yr, height 173 ± 9 cm, body mass 71 ± 11 kg) volunteered to participate in this study. The subjects participated in exercise at a recreational level, but were not highly trained, and were familiar with laboratory exercise testing procedures, having previously participated in studies employing cycle ergometry in our laboratory. The procedures employed in this study were approved by the University of Exeter Research Ethics Committee, and all subjects were required to give their written, informed consent before the commencement of the study, once the experimental procedures, associated risks, and potential benefits of participation had been explained. Subjects were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 3 h postprandial, and to avoid strenuous exercise in the 24 h preceding each testing session. Each subject was also asked to refrain from caffeine and alcohol 6 and 24 h before each test, respectively. All tests were performed at the same time of day (±2 h).

**Experimental Design**

The subjects were required to report to the laboratory on 12 occasions over a 4- to 5-wk period (six occasions for subjects assigned to the control condition), and all tests were interspersed with at least 24-h recovery. Subjects underwent a number of preliminary tests for the determination of VO2peak, GET, VO2 kinetics, and exercise tolerance. Upon completion of the preliminary tests, subjects were randomly assigned to one of the following training interventions: 30-s RST, continuous ET, or CON. Following completion of the training protocols, subjects repeated all of the baseline tests at the same absolute work rates to determine the effect of the respective training interventions on the physiological and performance parameters.

**Incremental Test**

Both before and after the intervention period, the subjects completed a ramp incremental exercise test for determination of the VO2peak and GET. All cycle tests were performed on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, the Netherlands). Initially, subjects performed 3 min of baseline cycling at 0 W, after which the work rate was increased at a rate of 25 W/min until the limit of tolerance. The subjects cycled at a self-selected pedal rate (70 – 90 rpm), and this pedal rate, along with saddle and handle bar height and configuration, was recorded and reproduced in subsequent tests. Breath-by-breath pulmonary gas-exchange data were collected continuously during the incremental tests and averaged over consecutive 10-s periods. The VO2peak was taken as the highest 30-s average value attained before the subject’s volitional exhaustion in the test. The GET was determined from a cluster of measurements, including 1) the first disproportionate increase in CO2 production (VCO2) from the posttransitional (post-VCO2) increase in VCO2; 2) an increase in expired ventilation (VE)/VO2 with no increase in VCO2; and 3) an increase in end-tidal O2 tension with no fall in end-tidal CO2 tension. The work rates that would require 90% of the GET (moderate exercise) and 70% Δ (GET plus 70% of the difference between the work rate at the GET and VO2peak; severe exercise) were subsequently calculated, with account taken of the mean response time (MRT) for VO2 during ramp exercise (i.e., two-thirds of the ramp rate was deducted from the work rate at GET and peak; Ref. 64).

**Step Exercise Tests**

On two occasions, both before and after the intervention period, the subjects also completed three “step” tests for the determination of VO2 kinetics. The protocol comprised two moderate-intensity and one severe-intensity cycle transition, each of 6-min duration. Each transition began with 3 min of baseline pedaling at 0 W before an abrupt transition to the target work rate. A passive recovery of 8 min separated the transitions. On one occasion, both before and after the intervention period, the severe-intensity transition was continued until task failure as a measure of exercise tolerance. The time to task failure was recorded when the pedal rate fell by >10 rpm below the required...
pedal rate. Therefore, all subjects performed a total of four bouts of moderate-intensity exercise and two bouts of severe-intensity exercise, both before and after the intervention period. The VO₂ responses from these like transitions were averaged before any analysis to enhance the signal-to-noise ratio and improve confidence in the parameters derived from the model fits (45, 65).

Training Interventions

After completing the initial stage of experimental testing, the male and female subjects were randomly assigned to either a RST group (mean ± SD: age 21 ± 5 yr, height 172 ± 8 cm, body mass 74 ± 9 kg), an ET group (age 20 ± 4 yr, height 1.76 ± 12 cm, body mass 71 ± 11 kg), or a CON group (age 20 ± 1 yr, height 171 ± 7 cm, body mass 66 ± 9 kg). All three groups contained five male and three female subjects. Both training groups performed a total of six training sessions over a 2-wk period, with at least 24-h recovery separating sessions, while the CON group maintained their habitual levels of physical activity.

The RST group performed a number of “all-out” 30-s cycle sprints (Wingate test) against a resistance equivalent to 0.075 kg/kg body mass on a mechanically braked cycle ergometer (model 814E bicycle ergometer, Monark, Stockholm, Sweden; Refs. 6–8, 22). The subjects completed a total of four repetitions of the 30-s sprint in the first training session, five repetitions in the second session, six repetitions in the third and fourth sessions, and seven repetitions in the fifth and sixth sessions. Subjects were instructed to pedal maximally against the ergometer’s inertial resistance −2 s before the appropriate load was applied. Strong verbal encouragement was provided to the subjects during all sprints to ensure a maximal effort was achieved. All sprints were separated by 4-min recovery, during which subjects were permitted to cycle at a low cadence (<50 rpm) against a light resistance (<30 W) to reduce sensations of nausea. The online data-acquisition system determined peak power, mean power, and rate of fatigue for each test, and these data were stored on a personal computer for subsequent analysis.

The ET required subjects to cycle continuously on a cycle ergometer (model 814E bicycle ergometer; Monark, Stockholm, Sweden) at 80 rpm for a predetermined duration at an intensity corresponding to 90% of the GET. The exercise duration was calculated so that the total work performed per training session for each subject was matched to the mean work performed by the RST group as a whole in the corresponding session. The mean work done and the duration of exercise completed by the RST and ET groups are presented in Table 1 for each of the six training sessions.

Measurements

During all tests, pulmonary gas exchange and ventilation were measured continuously using a portable metabolic cart (MetaMax 3B, Cortex Biophysik, Leipzig, Germany), as described previously (19). Pulmonary gas exchange and ventilation were calculated and displayed breath by breath. HR was measured during all tests using short-range radiotelemetry (Polar S610, Polar Electro Oy, Kempele, Finland).

During one of the transitions to moderate and severe exercise, pre- and posttraining, a blood sample was collected from a fingertip into a capillary tube over the 20 s preceding the step transition in work rate and within the last 20 s of exercise. A capillary blood sample was also collected at the limit of tolerance for the severe-intensity bout. These whole blood samples were subsequently analyzed to determine blood lactate concentration ([lactate]) (YSI 1500, Yellow Springs Instruments, Yellow Springs, OH) within 30 s of collection. Blood lactate accumulation (Δ blood [lactate]) was calculated as the difference between blood [lactate] at end exercise and blood [lactate] at baseline.

The oxygenation status of the m. vastus lateralis of the right leg was monitored using a commercially available NIRS system (model NIRO 300, Hamamatsu Photonics KK, Hiugasuki-ku, Japan). The sensor consisted of an emission probe that irradiates laser beams and a detection probe. Four different wavelength laser diodes provided the light source (776, 826, 845, and 905 nm), and the light returning from the tissue was detected by a photomultiplier tube in the spectrometer. The intensity of incident and transmitted light was recorded continuously at 2 Hz and used to estimate concentration changes from the resting baseline for oxygenated, deoxygenated, and total tissue hemoglobin/myoglobin. Therefore, the NIRS data represent a relative change based on the optical density measured in the first datum collected. The [Hb] signal can be regarded as being essentially blood-volume insensitive during exercise (14, 27, 41) and was, therefore, assumed to provide an estimate of changes in O₂ extraction in the field of interrogation (17, 21, 27). It should be noted here that the contribution of deoxygenated myoglobin to the NIRS signal is presently unclear, and, as such, the terms [Hbo₂], [HbO₂], and [Hb] used in this paper should be considered to refer to the combined concentrations of total, oxygenated, and deoxygenated hemoglobin and myoglobin, respectively.

The leg was initially cleaned and shaved around the belly of the muscle, and the probes were placed in the holder, which was secured to the skin with adhesive at 20 cm above the fibular head. To secure the holder and wires in place, an elastic bandage was wrapped around the subject’s leg. The wrap helped to minimize the possibility that extraneous light could influence the signal and also ensured that the optical diodes did not move during exercise. Pen marks were made around the probes to enable precise reproduction of the placement in subsequent tests. The probe gain was set with the subject at rest in a seated position with leg extended at 90° and wires in place. The intensity of incident and transmitted light was recorded continuously at 2 Hz and used to estimate concentration changes from the resting baseline for oxygenated, deoxygenated, and total tissue hemoglobin/myoglobin, respectively.

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Table 1. Total work done and duration of exercise completed by the RST and ET groups for each training session

<table>
<thead>
<tr>
<th>Session No.</th>
<th>RST Total Work Done, kJ</th>
<th>RST Exercise Duration, min</th>
<th>ET Exercise Duration, min</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>63±15</td>
<td>2</td>
<td>14.9±2.8</td>
</tr>
<tr>
<td>2</td>
<td>76±17</td>
<td>2.5</td>
<td>18.0±3.3</td>
</tr>
<tr>
<td>3</td>
<td>92±20</td>
<td>3</td>
<td>21.6±4.0</td>
</tr>
<tr>
<td>4</td>
<td>93±21</td>
<td>3</td>
<td>21.8±4.0</td>
</tr>
<tr>
<td>5</td>
<td>107±23</td>
<td>3.5</td>
<td>25.1±4.6</td>
</tr>
<tr>
<td>6</td>
<td>106±22</td>
<td>3.5</td>
<td>25.0±4.6</td>
</tr>
</tbody>
</table>

Values are means ± SD. The subjects in the endurance training (ET) group completed cycle exercise at 90% gas exchange threshold (GET) until they had completed the same amount of work as the repeated sprint training (RST) group for the equivalent session.

Data Analysis Procedures

The breath-by-breath VO₂ data from each test were initially examined to exclude errant breaths caused by coughing, swallowing, sighing, etc., and those values lying more than 4 SDs from the local mean were removed. The breath-by-breath data were subsequently linearly interpolated to provide second-by-second values, and, for each individual, identical repetitions were time aligned to the start of exercise and ensemble averaged. The first 20 s of data after the onset of exercise (i.e., the phase I response) were deleted (65), and a nonlinear least squares algorithm was used to fit the data thereafter. A single-exponential model was used to characterize the VO₂ responses to moderate exercise, and a biexponential model was used for severe exercise, as described in the following equations:

\[
\text{VO}_2(t) = \text{VO}_{2\text{baseline}} + A_p \left(1 - e^{-t/\text{TD}_{p/e}}\right) \quad (\text{moderate})
\]

\[
\text{VO}_2(t) = \text{VO}_{2\text{baseline}} + A_p \left(1 - e^{-t/\text{TD}_{p/e}}\right) + A_w \left(1 - e^{-t/\text{TD}_{w/e}}\right) \quad (\text{severe})
\]
\[ \dot{V}O_2(t) = \dot{V}O_{2\text{baseline}} + A_p [1 - e^{-t/\tau_p}] + A_s [1 - e^{-t/\tau_s}] \] (severe) (2)

where \( \dot{V}O_2(t) \) represents the absolute \( \dot{V}O_2 \) at a given time \( t \); \( \dot{V}O_{2\text{baseline}} \) represents the mean \( \dot{V}O_2 \) in the baseline period; \( A_p, T D_p, \) and \( \tau_p \) represent the amplitude, TD, and \( \tau \) respectively, describing the phase II increase in \( \dot{V}O_2 \) above baseline; and \( A_s, T D_s, \) and \( \tau_s \) represent the amplitude of, TD before the onset of, and \( \tau \) describing the development of the \( \dot{V}O_2 \) slow component, respectively.

An iterative process was used to minimize the sum of the squared errors between the fitted function and the observed values. \( \dot{V}O_{2\text{baseline}} \) was defined as the mean \( \dot{V}O_2 \) measured over the final 90 s of baseline pedaling. The end-exercise \( \dot{V}O_2 \) was defined as the mean \( \dot{V}O_2 \) measured over the final 30 s of exercise. The absolute fundamental component amplitude (absolute \( A_s \)) was defined as the sum of \( \dot{V}O_{2\text{baseline}} \) and \( A_p \). Because the asymptotic value (\( A_0 \)) of the exponential term describing the \( \dot{V}O_2 \) slow component may represent a higher value than is actually reached at the end of the exercise, the actual amplitude of the \( \dot{V}O_2 \) slow component at the end of exercise was defined as \( A' \). The \( A_0 \) parameter was compared at the same iso-time (360 s) pre- and posttraining. The amplitude of the slow component was also described relative to the entire \( \dot{V}O_2 \) response. The rate at which the \( \dot{V}O_2 \) slow component developed (i.e., in ml/min/s) was also calculated. In addition, the functional “gain” of the fundamental \( \dot{V}O_2 \) response was computed by dividing \( A_p \) by the \( \Delta \)work rate. The functional gain of the entire response (i.e., end-exercise gain) was calculated in a similar manner.

To provide information on muscle oxygenation, we also modeled the [HHb] response to exercise. Mono- and bieponential models, similar to those described above, were applied to the ensemble-averaged data with the exception that the fitting window commenced at the time at which the [HHb] signal increased 1 SD above the baseline mean. Moderate-intensity [HHb] kinetics were determined by constraining the fitting window to the point at which monoexponentiality became distorted, consequent to a gradual fall in [HHb] (41), as determined by visual inspection of the residual plots. Severe-intensity [HHb] kinetics were determined by fitting a bieponential model from the first data point, which was 1 SD above the baseline mean through the entire response. It should be noted that, although the [HHb] response can be conveniently described by an exponential function, it has not been established that an exponential function best reflects the underlying physiological response. The [HHb] TD and \( \tau \) values were summed to provide information on the overall [HHb] response dynamics in the fundamental phase of the response. In addition to the [HHb] \( \tau \) and TD derived from the model fits for both moderate and severe exercise, we also used the fundamental [HHb] amplitude to determine the \( \Delta[HHb]/\Delta\dot{V}O_2 \) during this phase of the response. We chose to express the [HHb] slow component, observed during severe-intensity exercise, both relative to the entire [HHb] response and as the \( \Delta[HHb]/\Delta\dot{V}O_2 \). The [HbO2] and [Hb] responses do not approximate an exponential (15) and were, therefore, not modeled. Rather, we assessed training-induced changes in these parameters by determining the [HbO2] and [Hb] at baseline (90-s preceding step transition), 120 s, and [HHb] TD, for severe-intensity exercise, and end exercise (average response over the final 30 s).

We also modeled the HR response to exercise in each condition. For this analysis, HR data were linearly interpolated to provide second-by-second values, and, for each individual, identical repetitions from like transitions were time aligned to the start of exercise and ensemble averaged. A nonlinear least squares monoeponential model without TD was used to fit the data to moderate-intensity exercise, with the fitting window commencing at \( t = 0 \) s. The fitting window was constrained to the \( \dot{V}O_2 \) TD, in the case of severe-intensity exercise (see above). The HR \( \tau_p \) so derived provides information on the overall HR response dynamics in the absence of any HR “slow component.” The HR slow component was computed as the difference between the HR attained at the \( \dot{V}O_2 \) TD, and the HR attained at end exercise. In addition to this analysis, we also modeled the HR response with a bieponential model from the onset of exercise without constraining the fit to the \( t < \dot{V}O_2 \) TD, region.

### Statistical Analysis

A 2 \times 3 (time by group) ANOVA with repeated measures for time was employed to determine the effects on the relevant physiological variables elicited by the differing training protocols. Where the analysis revealed a significant difference, individual paired t-tests were employed with a Bonferroni correction to determine the origin of such effects. Pearson’s product-moment correlation was employed to examine the interrelationships between the parameters of \( \dot{V}O_2 \) kinetics and [HHb] and exercise tolerance. All data are presented as means \( \pm \) SD. Statistical significance was accepted when \( P < 0.05 \).

### RESULTS

All subjects were recreationally active on recruitment to the study and the physiological parameters of interest (i.e., \( \dot{V}O_{2\text{peak}}, \dot{V}O_2 \) at GET, \( \dot{V}O_2 \) \( \tau_p \), \( \dot{V}O_2 \) slow-component amplitude) were similar between the three groups before the commencement of the study (Tables 2 and 3). Each subject completed 100% of the training sessions required by the specific training groups and self-reported that they did not alter their activity levels outside of training for the duration of the study. The ET group completed \( \sim 126 \) min of low-intensity exercise, and the RST group completed 17.5 min of high-intensity exercise over the 2-wk training period.

### Incremental Test

The \( \dot{V}O_2 \) and work rate attained at the GET and at the limit of tolerance before (Pre) and following (Post) the 2-wk intervention period are presented in Table 2. The RST group demonstrated a significant (\( P < 0.05 \)) increase in both absolute (Pre: 3.06 \pm 0.60, Post: 3.29 \pm 0.65 l/min) and relative (Pre: 42 \pm 6, Post: 45 \pm 6 ml·kg\(^{-1}\)·min\(^{-1}\)) \( \dot{V}O_{2\text{peak}} \) following

### Table 2. Work rate and oxygen uptake at the GET and at peak during ramp incremental exercise in the RST, ET, and control groups pre- and postintervention

<table>
<thead>
<tr>
<th></th>
<th>RST Pre</th>
<th>RST Post</th>
<th>ET Pre</th>
<th>ET Post</th>
<th>CON Pre</th>
<th>CON Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}O_2 ) at GET, l/min</td>
<td>1.45 \pm 0.64</td>
<td>1.47 \pm 0.37</td>
<td>1.41 \pm 0.34</td>
<td>1.44 \pm 0.45</td>
<td>1.38 \pm 0.33</td>
<td>1.39 \pm 0.41</td>
</tr>
<tr>
<td>WR at GET, W</td>
<td>103 \pm 18</td>
<td>129 \pm 16</td>
<td>82 \pm 16</td>
<td>90 \pm 16</td>
<td>88 \pm 11</td>
<td>98 \pm 12</td>
</tr>
<tr>
<td>( \dot{V}O_{2\text{peak}}, ) l/min</td>
<td>3.06 \pm 0.62</td>
<td>3.29 \pm 0.77*</td>
<td>3.16 \pm 0.73</td>
<td>3.14 \pm 0.74</td>
<td>3.09 \pm 0.81</td>
<td>3.00 \pm 0.85</td>
</tr>
<tr>
<td>( \dot{V}O_{2\text{peak}}, ) ml·kg(^{-1})·min(^{-1})</td>
<td>42 \pm 6</td>
<td>45 \pm 6*</td>
<td>43 \pm 5</td>
<td>43 \pm 5</td>
<td>47 \pm 8</td>
<td>46 \pm 8</td>
</tr>
<tr>
<td>Peak WR, W</td>
<td>299 \pm 63</td>
<td>313 \pm 58</td>
<td>284 \pm 49</td>
<td>288 \pm 52</td>
<td>294 \pm 64</td>
<td>289 \pm 61</td>
</tr>
</tbody>
</table>

Values are means \( \pm \) SD. Pre, preintervention; Post, postintervention; CON, control; \( \dot{V}O_2 \), oxygen uptake; \( \dot{V}O_{2\text{peak}}, \) peak \( \dot{V}O_2 \); WR, work rate. *Significantly different from RST Pre (\( P < 0.05 \)); †significantly different from RST Pre (\( P < 0.01 \)).
training, while these parameters were not significantly altered in the ET and CON groups (Table 2). Similarly, peak work rate was only improved significantly following RST (Pre: 299 ± 63, Post: 313 ± 58 W; P < 0.01). The work rate at the GET demonstrated a significant main effect for time (P < 0.05); however, further analysis revealed no effect in any individual group. The VO2 associated with the GET was not altered significantly following any of the interventions.

**VO2 Kinetics**

The parameters derived from the monoexponential model fits, including the 95% confidence intervals surrounding the τp estimate, are presented in Table 3. The VO2 response of a representative subject from each training group is illustrated in Fig. 1 for moderate-intensity exercise and in Fig. 2 for severe-intensity exercise.

**Moderate-intensity exercise.** Neither the baseline nor the amplitude of the VO2 response was affected by any of the interventions. For τp, however, there was a significant main effect for time (P < 0.01). Further analyses revealed that only the RST τp was significantly reduced following training (Pre: 28 ± 8, Post: 21 ± 8 s; P < 0.01), whereas τp was not significantly altered following ET and CON (Table 3). Likewise, significant reductions in the MRT (Pre: 45 ± 7, Post: 36 ± 6 s) and O2 deficit (Pre: 0.45 ± 0.10, Post: 0.36 ± 0.10 liter) were observed following RST (both P < 0.05), but not ET or CON (Table 3). There was a strong correlation between the initial τp value and the reduction in τp observed with RST (r = 0.80; P < 0.05). The Δblood [lactate] was significantly reduced in the RST group, but not in the ET or CON groups (Table 3).

**Severe-intensity exercise.** The baseline VO2 and primary VO2 amplitude were not altered in RST, ET, or CON. Only the RST intervention elicited a significant speeding of the τp (Pre: 29 ± 5, Post: 23 ± 5 s; P < 0.05) and MRT (Pre: 62 ± 11, Post: 54 ± 12 s; P < 0.05). There was a strong correlation between the initial τp value and the reduction in τp observed with RST (r = 0.71; P < 0.05). The A*p (Pre: 0.52 ± 0.19, Post: 0.40 ± 0.17 l/min; P < 0.01) and the rate at which the slow component developed were significantly reduced following RST, but were not altered in ET or CON (Table 3). The end-exercise and Δblood [lactate] values were significantly reduced in the RST group, but not in the ET or CON groups (Table 3).

**NIRS Measurements**

[ΔHbO2] and [ΔHba] were not significantly different before or during exercise in the three groups before the intervention, and the responses were not altered by the training or control periods. The parameters derived from modeling the [ΔHb] response are shown for each group pre- and posttraining in Table 4. The [ΔHb] dynamics of a representative subject from each training group are illustrated in Fig. 3 for moderate-intensity exercise and in Fig. 4 for severe-intensity exercise.

**Moderate-intensity exercise.** A significant interaction effect was observed for the TD of the [ΔHb] response (P < 0.05). Follow-up analyses revealed that the [ΔHb] TD was significantly reduced in RST (Pre: 14 ± 4, Post: 10 ± 2 s; P < 0.05), but not in ET or CON (Table 4). The [ΔHb] τ did not differ significantly between groups and was not altered following the intervention period. However, the [ΔHb] TD + τ was significantly reduced following RST only (Pre: 25 ± 8, Post: 20 ± 3 s; P < 0.05). The amplitude of the [ΔHb] response and the prime.
V\text{\textsubscript{O}}2 were also significantly increased in RST, but not in ET or CON (Table 4). The changes in V\text{\textsubscript{O}}2\text{\textsubscript{p}} with RST were significantly correlated with changes in the [HHb] $\tau$ ($r = 0.78; P < 0.05$), the [HHb] TD + $\tau$ ($r = 0.78; P < 0.05$), and the [HHb] amplitude ($r = -0.81; P < 0.05$; Fig. 5). The reduction in the V\text{\textsubscript{O}}2 slow component with RST was not correlated with changes in [HHb] kinetics.

HR Kinetics

HR kinetics were not altered in the RST, ET, or CON groups for either moderate- or severe-intensity exercise (Table 5). The only significant difference observed was in the end-exercise HR, which was reduced in the RST condition (Table 5). When the fitting window for severe exercise was not constrained (see METHODS), the $\tau$ values were slightly shorter ($\sim 25$ s), and the amplitude of the slow component was slightly larger ($\sim 25$ beats/min), but again there was no difference between groups before or after the intervention period.

**Severe-intensity exercise.** The TD of the [HHb] response was not different before or after the intervention period in any group. However, a significant interaction effect was observed for the $\tau$ of the [HHb] response ($P < 0.05$). Follow-up analyses revealed that the [HHb] $\tau$ was significantly reduced in RST (Pre: $12 \pm 3$, Post: $9 \pm 3$ s; $P < 0.05$), but not in ET or CON (Table 4). The [HHb] TD + $\tau$ also tended to be faster following RST (Pre: $18 \pm 6$, Post: $15 \pm 3$ s; $P = 0.12$). The amplitude of the [HHb] response and the $\Delta$[HHb]/$\Delta$V\text{\textsubscript{O}}2 for the fundamental phase were significantly increased only in the RST group (Table 4). The [HHb] $A_c$ and the $\Delta$[HHb]/$\Delta$V\text{\textsubscript{O}}2 in the slow phase of the response were not altered in any condition (Table 4).

HR Kinetics

HR kinetics were not altered in the RST, ET, or CON groups for either moderate- or severe-intensity exercise (Table 5). The only significant difference observed was in the end-exercise HR, which was reduced in the RST condition (Table 5). When the fitting window for severe exercise was not constrained (see METHODS), the $\tau$ values were slightly shorter ($\sim 25$ s), and the amplitude of the slow component was slightly larger ($\sim 25$ beats/min), but again there was no difference between groups before or after the intervention period.

Fig. 1. Pulmonary oxygen uptake (V\text{\textsubscript{O}}2) response to a step increment from an unloaded baseline to a moderate-intensity work rate in a representative subject from the repeated sprint training (RST) group (top) and the endurance training (ET) group (bottom). These data are expressed as a percentage of the overall response. The preintervention (Pre) responses are shown as open circles, and the postintervention (Post) responses are shown as solid squares. The vertical line represents the abrupt transition to the higher work rate. The solid lines represent the monoexponential model fits to the data. Note the significantly faster phase II V\text{\textsubscript{O}}2 kinetics following RST. $\tau$\text{\textsubscript{p}} phase II time constant.

Fig. 2. Pulmonary V\text{\textsubscript{O}}2 response to a step increment from an unloaded baseline to a severe-intensity work rate in a representative subject from the RST group (top) and the ET group (bottom). These data are expressed as a percentage of the overall response. The Pre responses are shown as open circles, and the Post responses are shown as solid squares. The vertical line represents the abrupt transition to the higher work rate. The solid lines represent the monoexponential model fits to the data. Note the significantly faster phase II V\text{\textsubscript{O}}2 kinetics following RST.
Exercise Tolerance

The alterations in exercise tolerance are illustrated in Fig. 6. A significant time and interaction effect was indicated (P < 0.01), and further analyses revealed that, of the three groups, only the RST group demonstrated a significant increase in the time to exhaustion during severe-intensity exercise (Pre: 700 ± 284, Post: 1,074 ± 331 s; P < 0.01; Fig. 6). Before RST, exercise tolerance was correlated with A1 (r = -0.83; P < 0.05), but not τp (r = -0.40; P > 0.05). After RST, exercise tolerance was correlated with τp (r = -0.81; P < 0.05), but not A1 (r = -0.70; P = 0.06). However, the improvement in exercise tolerance with RST was not significantly correlated with either the Δτp (r = -0.58; P > 0.05) or the ΔA1 (r = -0.44; P > 0.05). The change in exercise tolerance was not significantly correlated with the changes in VO2peak (r = 0.13) or GET (r = 0.68).

DISCUSSION

To our knowledge, this investigation is the first to have compared the influence of six sessions of RST (6–8, 22) and work-matched, continuous, moderate-intensity ET on exercise tolerance and the kinetics of VO2, [HHb], and HR in young adults. In contrast to our first hypothesis, VO2 kinetics were speeded, and exercise tolerance was enhanced, only following RST. However, in support of our second hypothesis, the enhanced VO2 kinetics following RST were associated with a greater muscle O2 extraction (as estimated by changes in [HHb] kinetics; Refs. 17, 26, 27). Indeed, the faster phase II VO2 kinetics following RST were significantly correlated with changes in [HHb] kinetics, suggesting that the training intervention impacted VO2 kinetics by enhancing muscle fractional O2 extraction.

Improvements in VO2peak (8%) and peak work rate (5%) in the incremental test were only observed following the RST intervention in the present study. Likewise, previous investigators have reported a 7–8% increases in VO2peak following 30-s RST (2, 7, 49), although others have reported no effect (6, 8, 33). The VO2/work rate slope during incremental exercise and the gain of the fundamental phase of the VO2 response to moderate and severe exercise (~10 ml·min−1·W−1) were not altered by training, consistent with other studies that have shown that short-term training interventions do not alter cycling efficiency (4, 35, 53). The GET was not altered by either RST or ET, suggesting that longer term training might be required to impact measurably on this parameter of aerobic function (4, 31, 35, 53).

VO2 Kinetics

A novel finding of the present investigation was that just six sessions of RST resulted in 20–25% reductions in both the VO2 τp and the VO2 slow-component amplitude, whereas VO2 kinetics were not altered by six sessions of work-matched ET and were also unchanged in the CON group (Fig. 6). A more rapid approach toward, or attainment of, the requisite VO2 "steady state" would reduce the O2 deficit and result in corresponding reductions in energy supply from substrate-level phosphorylation (9, 18, 38, 66) and the accumulation of fatigue-related metabolites, such as HC and Pi (1). Moreover, a reduction in the VO2 slow component should attenuate the depletion of the muscles’ finite PCR and glycogen stores (39, 43, 57, 58). Our results contrast with those of Berger et al. (4), who reported that 6 wk of interval training (3–4 sessions/wk of 20 × 1-min exercise bouts at 90% VO2peak separated by 1-min rest) and ET (3–4 sessions/wk of 30-min continuous exercise at 60% VO2peak) were equally effective in speeding VO2 kinetics and reducing the VO2 slow component. These authors concluded that the type of exercise training performed (i.e., high-intensity intermittent or low-intensity continuous) was less important than the total volume of training completed. The present study demonstrates that just six sessions of RST (amounting to a total of 17.5 min of exercise over 2 wk) has a greater effect on VO2 kinetics than six sessions of work-matched ET (present study), but a similar effect as 6 wk of conventional ET (4). RST, therefore, clearly repre-

Table 4. Deoxyhemoglobin/myoglobin kinetics during moderate-intensity and severe-intensity exercise in the RST, ET, and control groups pre- and postintervention

<table>
<thead>
<tr>
<th></th>
<th>RST Pre</th>
<th>RST Post</th>
<th>ET Pre</th>
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<th>CON Pre</th>
<th>CON Post</th>
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<td></td>
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</tr>
<tr>
<td>[HHb] baseline, AU</td>
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<td>-81 ± 57</td>
<td>-82 ± 62</td>
<td>-77 ± 52</td>
<td>-83 ± 50</td>
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<td>[HHb] amplitude, AU</td>
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<td>73 ± 41*</td>
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<td>41 ± 29</td>
<td>44 ± 33</td>
<td>45 ± 26</td>
</tr>
<tr>
<td>Phase II Δ[Hhb]/ΔVO2, AU·1−1·min−1</td>
<td>92 ± 69</td>
<td>114 ± 60*</td>
<td>93 ± 39</td>
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</tr>
<tr>
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<td>-106 ± 60</td>
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<tr>
<td>[HHb] primary time constant, s</td>
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</tr>
<tr>
<td>[HHb] time delay × time constant, s</td>
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<td>17 ± 5</td>
<td>19 ± 6</td>
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<tr>
<td>[HHb] primary-phase amplitude, AU</td>
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<td>220 ± 137*</td>
<td>127 ± 76</td>
<td>138 ± 92</td>
<td>145 ± 92</td>
<td>139 ± 89</td>
</tr>
<tr>
<td>Primary-phase Δ[Hhb]/ΔVO2, AU·1−1·min−1</td>
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<td>113 ± 52*</td>
<td>89 ± 46</td>
<td>99 ± 42</td>
<td>84 ± 40</td>
<td>80 ± 38</td>
</tr>
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<td>[HHb] slow-phase amplitude, AU</td>
<td>32 ± 20</td>
<td>37 ± 24</td>
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<td>26 ± 17</td>
<td>32 ± 13</td>
</tr>
<tr>
<td>Slow-phase Δ[Hhb]/ΔVO2, AU·1−1·min−1</td>
<td>63 ± 54</td>
<td>94 ± 46</td>
<td>52 ± 34</td>
<td>63 ± 42</td>
<td>52 ± 38</td>
<td>68 ± 39</td>
</tr>
</tbody>
</table>

Values are means ± SD. [HHb], deoxyhemoglobin/myoglobin concentration. *Significantly different from RST Pre (P < 0.05); †significantly different from RST Pre (P < 0.01).
sents a potent and time-effective strategy for enhancing $V\dot{O}_2$ kinetics, at least in the recreationally active young subjects who participated in the present study.

In contrast to RST, six sessions of ET had no significant effect on the kinetics of $V\dot{O}_2$, HR, or [HHb], or exercise tolerance. Whether or not ET impacts upon $V\dot{O}_2$ kinetics might be dependent on the precise nature of the training program performed (intensity, volume, frequency), as well as its duration (35). Several earlier studies demonstrated that ET, which typically involves three to four sessions per week of continuous exercise at 60–80% $V\dot{O}_2$peak for 4–8 wk, resulted in significant reductions in the $t_p$ and/or the $V\dot{O}_2$ slow component (4, 10, 11, 18, 31, 52, 54, 59, 71). One explanation for the lack of effect might be that our subjects were recreationally active in various sports upon their recruitment to the present study. It has been suggested that, once a given level of aerobic fitness has been attained, high-intensity intermittent training, rather than continued ET, is required to elicit further performance gains (47). Moreover, since the RST and ET were work matched in the present study, the combination of a limited number of training sessions ($n = 6$), short session duration (10–30 min), and low training intensity (90% GET, equivalent to 40–45% $V\dot{O}_2$peak) may have represented an insufficient stimulus to elicit improvements in the parameters of aerobic fitness and exercise tolerance. The lack of effect of ET on parameters of aerobic fitness and exercise performance in the present study was striking, and it is clear that the program of training completed did not exceed the minimum “threshold” stimulus (i.e., intensity and/or volume and/or frequency and/or duration) necessary to enable $V\dot{O}_2$ kinetics to be altered (4).

**Muscle Oxygenation and Estimated $O_2$ Extraction**

We used NIRS to assess the effects of RST and ET on muscle oxygenation during exercise. The [HbO₂] and [Hbtot] values were not different in the three groups before or during moderate or severe intensity, and they were not altered by the intervention period. The observed training adaptations might, therefore, be considered to be independent of changes in blood volume and $O_2$ content in the area of interrogation. HR kinetics were also not altered by RST or ET in the present study. If HR kinetics broadly reflect leg blood flow kinetics during cycling exercise, as has been suggested for knee-extension exercise (50), these data suggest that bulk $O_2$ delivery to muscle was not appreciably altered by the training interventions. Longer dura-

Fig. 3. Muscle deoxyhemoglobin/myoglobin (HHb) response to a step increment from an unloaded baseline to a moderate-intensity work rate in a representative subject from the RST group (top) and the ET group (bottom). The Pre responses are shown as open circles, and the Post responses are shown as solid squares. The vertical line represents the abrupt transition to the higher work rate. The solid lines represent the monoexponential model fits. [HHb], HHb concentration; AU, arbitrary units.

Fig. 4. Muscle HHb response to a step increment from an unloaded baseline to a severe-intensity work rate in a representative subject from the RST group (top) and the ET group (bottom). The Pre responses are shown as open circles, and the Post responses are shown as solid squares. The vertical line represents the abrupt transition to the higher work rate. The solid lines represent the monoexponential model fits.
tions of continuous ET (61) and intense interval training (43) have been reported to result in faster conduit artery blood flow kinetics and greater vascular conductance. The HR kinetics and [HbO₂] data in the present study collectively suggest that alterations in bulk O₂ delivery to muscle were not responsible for the effects of RST on V̇O₂ kinetics. This might imply that V̇O₂ kinetics were not limited by muscle O₂ availability in our subjects, a position that has support in the literature (16, 26, 34, 55, 69, 70). However, it should be acknowledged that our measurements were indirect, and we are, therefore, unable to rule out the possibility that faster muscle blood flow kinetics contributed, in part, to the faster V̇O₂ kinetics observed following RST. Indeed, even if bulk muscle blood flow was not altered, it is possible that microvascular adaptations with training enabled a better matching of perfusion to local metabolic rate, perhaps especially in the type II fiber population, which would have received particular stimulus with RST (25, 43, 49). Preferential alterations in blood flow to fibers that were relatively underperfused before training might facilitate greater O₂ extraction and faster V̇O₂ kinetics after training (46).

Fig. 5. Relationship between RST-induced changes in phase II V̇O₂ kinetics and [HHb] dynamics. Relationships are shown between difference (Δ) in TP and Δ[Hb] time delay (TD) during moderate- (A) and severe-intensity exercise (B); between ΔTP and Δ[Hb] TP during moderate- (C) and severe-intensity exercise (D); between ΔTP and Δ[Hb] TD + 0.05 during moderate- (E) and severe-intensity exercise (F); and between ΔTP and Δ[Hb] amplitude during moderate- (G) and severe-intensity exercise (H). The relationships illustrated in D–H were statistically significant (P < 0.05).
The rate and magnitude of [HHb] changes during exercise provide information on the dynamic balance between O2 delivery and O2 utilization within the area of interrogation (15–17, 21, 27, 34). To our knowledge, the present study is the first to document the influence of RST and ET on [HHb] kinetics following the onset of exercise. The [HHb] TD and the [HHb] TD + τ were significantly reduced during moderate exercise, the [HHb] τ was significantly reduced during severe exercise, and the [HHb] primary amplitude and Δ[HHb]/ΔV˙O2 were significantly increased for both moderate and severe exercise in the RST group (but not the ET or CON groups). Assuming that changes in [HHb] dynamics accurately reflect changes in muscle fractional O2 extraction (17, 21, 27), these data indicate that RST enabled O2 extraction to be initiated more rapidly, and to a greater extent, following the onset of exercise. Although complicated by possible changes in blood volume following the onset of contractions, the [HHb] TD has been interpreted to represent a period of time during which O2 delivery to the muscle fibers in the area of interrogation is adequate to meet the augmented O2 demand following the initiation of contractions (17, 27). A shorter [HHb] TD during moderate exercise following RST, therefore, implies that the active muscle fibers extracted O2 more rapidly in the metabolic transient. Similarly, the reduced [HHb] τ during severe exercise following RST indicates that, following the initial TD, muscle O2 extraction increased more rapidly towards the “steady-state” requirement for the metabolic rate. Assuming similar muscle O2 availability (see earlier discussion), the reduced [HHb] TD and/or τ, along with the greater [HHb] primary amplitude following RST, would be expected to enable muscle O2 consumption to rise more rapidly following the onset of exercise. Consistent with this interpretation, the faster phase II V˙O2 kinetics observed following RST was significantly correlated with changes in both the amplitude and some temporal aspects of the kinetics of the [HHb] response during both moderate-intensity and severe-intensity exercise (Fig. 5).

It was of interest that RST had a somewhat different effect on [HHb] kinetics during moderate exercise (reduced TD, unchanged τ) and severe exercise (unchanged TD, reduced τ). These data might imply that training resulted in intensity-dependent adaptations in factors that influence the balance between muscle O2 availability (e.g., muscle pump vs. regulation of vasodilatation; Ref. 63) and O2 utilization. However, the net result of RST was for [HHb] kinetics to be generally faster and for the amplitude of the response (reflecting the magnitude of O2 extraction) to be greater. Collectively, the results indicate that the adaptation of muscle O2 extraction was greater than the adaptation of local muscle blood flow, such that the former contributed more than the latter to the faster V˙O2 kinetics following RST.

Models of respiratory control predict that a speeding of V˙O2 kinetics is contingent upon increased mitochondrial volume (e.g., Ref. 51), and, at least in young, physically active subjects, the phase II V˙O2 response appears to be limited by an inertia of the intracellular oxidative metabolic processes (26, 34, 55, 69, 70). It has been well documented that RST results in increased mitochondrial enzyme activity, including cytochrome-c oxidase (22), citrate synthase (8), and pyruvate dehydrogenase (6). Increases in succinate dehydrogenase, malate dehydrogenase, and hexokinase activity have also been reported using a similar training intervention (49). Similar enzymatic adaptations in the present study would be expected to increase the capacity for muscle fractional O2 extraction, which, in turn, might have facilitated the acceleration of V˙O2 kinetics that we observed.

The increased [HHb] amplitude (and Δ[HHb]/ΔV˙O2) in the primary response phase observed following RST corroborates the findings of Krustrup et al. (43) during severe-intensity exercise. Specifically, those authors reported that supramaximal, intermittent, one-legged, knee-extensor exercise training resulted in an increased thigh O2 extraction between 20 and 70 s of exercise, accompanied by an elevated thigh V˙O2 between 20 and 110 s of exercise. Importantly, during the first 75 s of exercise, thigh blood flow was not significantly enhanced postraining, indicating that the increased V˙O2 was consequent to an increased muscle O2 extraction. Our results are also consistent with Daussin et al. (13), who recently reported that V˙O2 kinetics were accelerated by interval training (which resulted in increased muscle oxidative capacity), but not by continuous ET (which did not alter oxidative capacity).

Table 5. Heart rate kinetics during moderate-intensity and severe-intensity exercise in the RST, ET, and control groups pre- and postintervention

<table>
<thead>
<tr>
<th></th>
<th>RST Pre</th>
<th>RST Post</th>
<th>ET Pre</th>
<th>ET Post</th>
<th>CON Pre</th>
<th>CON Post</th>
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<tr>
<td>Baseline HR, beats/min</td>
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<tr>
<td>End-exercise HR, beats/min</td>
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<td>115±14</td>
<td>110±11</td>
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<td>Primary time constant, s</td>
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<td>34±16</td>
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<tr>
<td>HR primary amplitude, beats/min</td>
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<tr>
<td>Baseline HR, beats/min</td>
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<td>End-exercise HR, beats/min</td>
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</table>

Values are means ± SD. HR, heart rate. *Significantly different from RST Pre (P < 0.01).
that the $\tau_p$ values reported by Daussin et al. (13) were surprisingly long (>60 s) for healthy subjects (e.g., Ref. 16).

**Exercise Tolerance**

Six sessions of RST resulted in a 53% improvement in exercise tolerance (determined as the time to “exhaustion”) during severe-intensity exercise, whereas six sessions of ET resulted in a nonsignificant 13% increase. It has previously been reported that RST resulted in a doubling of exercise time at 80% $VO_2$peak (8) and 10% improvements in time trial performance (6, 22). The available evidence indicates that the ergogenic effect of RST may be attributed, in part, to alterations in muscle oxidative capacity and substrate metabolism (6–8, 22, 23). For example, the rates of glycogen and PCr degradation were reduced, and the rates of carbohydrate and fat utilization were reduced and increased, respectively, during 60 min of cycle exercise at 65% $VO_2$peak following RST (7). A greater energetic contribution from oxidative phosphorylation and a reduced reliance on substrate-level phosphorylation, with an attendant reduction in muscle PCr depletion and $H^+$ and $Pi$ accumulation, are consistent with the faster phase II $VO_2$ kinetics and reduced $VO_2$ slow-component amplitude observed in the present study. That $VO_2$ kinetics have the potential to impact upon exercise tolerance is demonstrated by the significant correlations observed between the $V\dot{O}_2$ parameters and exercise tolerance. The Pre responses are shown as open bars, and the Post responses are shown as solid bars. Note that only RST resulted in improvements in $VO_2$ kinetics and exercise tolerance. *$P < 0.05$; $\#P < 0.01$.

Fig. 6. Group mean ± SD alterations in the parameters of $VO_2$ kinetics and exercise tolerance following the control, ET, and RST intervention periods. A: changes in moderate exercise phase II $VO_2$ kinetics. B: changes in severe exercise phase II $VO_2$ kinetics. C: changes in the $VO_2$ slow component amplitude. D: changes in exercise tolerance. The Pre responses are shown as open bars, and the Post responses are shown as solid bars. Note that only RST resulted in improvements in $VO_2$ kinetics and exercise tolerance. *$P < 0.05$; $\#P < 0.01$. 

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**A**

Moderate Intensity $\dot{V}\!O_2$, (s)  
Control | Endurance Training | Repeated Sprint Training

**B**

Severe Intensity $\dot{V}\!O_2$, (s)  
Control | Endurance Training | Repeated Sprint Training

**C**

$\dot{V}\!O_2$ Slow Component Amplitude (L·min$^{-1}$)  
Control | Endurance Training | Repeated Sprint Training

**D**

$Tlim$ (s)  
Control | Endurance Training | Repeated Sprint Training
small elevation in $\text{VO}_{2\text{peak}}$, which, when combined with the reduced $\text{O}_{2}$ slow-component amplitude, would have delayed the attainment of $\text{VO}_{2\text{peak}}$ and thus the point of rapid substrate depletion and fatiguing metabolite accumulation (9); and 2) a possible increased anaerobic energy yield, resulting from an improved muscle buffering capacity (22).

The features of RST that promote the profound improvements in parameters of aerobic fitness and exercise tolerance documented in this and previous studies (2, 6–8, 22, 49) are not entirely clear. However, in addition to providing near-maximal stress to the energy pathways during the repeated 30-s sprints, the perturbations in substrate availability and metabolite accumulation would require substantial oxidative energy transfer to restore homeostasis following each sprint (5). Moreover, the severe fluctuations in work rate and ATP turnover with RST, and the associated profound perturbations to cellular homeostasis, including changes in oxygen tension and the redox and phosphorylation potentials, might be a potent stimulus to the signaling pathways, resulting in mitochondrial biogenesis (32). One adaptation associated with RST is an increase in the protein content of peroxisome proliferator-activated receptor-$\gamma$ coactivator-1a (7). A corollary of peroxisome proliferator-activated receptor-$\gamma$ coactivator-1a expression is the modulation of the skeletal muscle phenotype via a fast- to slow-twitch fiber conversion (48). However, whether this could occur to any appreciable extent following just six training sessions is unclear. Alternatively, given the profound differences in the cadence and force generation requirements of continuous low-intensity ET and RST, it is probable that fiber recruitment differed considerably in the two training groups (25, 60). Specifically, the all-out nature of the RST would have required the recruitment of a large proportion of type II fibers, whereas ET would require the recruitment of predominantly type I fibers (29). It has been shown that interval training induces greater oxidative enzyme adaptations in type II fibers than continuous training (24, 30), and that type IIb fibers manifest greater training-induced elevations in oxidative capacity as training intensity increases above $\text{VO}_{2\text{peak}}$ (20). These fiber-type-specific adaptations would be expected to result in faster $\text{VO}_{2}$ kinetics and enhanced tolerance to high-intensity exercise (12, 18, 38, 44).

Conclusion
Six sessions of RST resulted in an acceleration of $\text{VO}_{2}$ kinetics during step transitions to both moderate-intensity and severe-intensity exercise and enhanced exercise tolerance. In contrast, six sessions of work-matched ET did not alter $\text{VO}_{2}$ kinetics or exercise tolerance. HR kinetics were not altered by either training intervention. However, the acceleration of $\text{VO}_{2}$ kinetics with RST was associated with an augmented change in the NIRS-derived muscle deoxygenation signal. These data indicate that RST provokes rapid adaptations of estimated muscle $\text{O}_{2}$ extraction that facilitated an acceleration of $\text{VO}_{2}$ kinetics and improved exercise performance.

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