Effects of interval and continuous training on O_{2} uptake kinetics during severe-intensity exercise initiated from an elevated metabolic baseline

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Da Boit M, Bailey SJ, Callow S, DiMenna FJ, Jones AM. Effects of interval and continuous training on O_{2} uptake kinetics during severe-intensity exercise initiated from an elevated metabolic baseline. J Appl Physiol 116: 1068–1077, 2014. First published February 13, 2014; doi:10.1152/japplphysiol.01365.2013.—The purpose of this study was to test the hypothesis that VO_{2} kinetics would be speeded to a greater extent following repeated sprint training (RST), compared with continuous endurance training (ET), in the transition from moderate- to severe-intensity exercise. Twenty-three recreationally active subjects were randomly assigned to complete six sessions of ET (60–110 min of moderate-intensity cycling) or RST (four to seven 30-s all-out Wingate tests) over a 2-wk period. Subjects completed three identical work-to-work cycling exercise tests before and after the intervention period, consisting of baseline cycling at 20 W followed by sequential step increases to moderate- and severe-intensity work rates. The severe-intensity bout was continued to exhaustion on one occasion and was followed by a 60-s all-out sprint on another occasion. Phase II pulmonary VO_{2} kinetics were speeded by a similar magnitude in both the lower (ET pre, 28 ± 4; ET post, 22 ± 4 s; RST pre, 25 ± 8; RST post, 20 ± 7 s) and upper (ET pre, 50 ± 10; ET post, 39 ± 11 s; RST pre, 54 ± 7; RST post, 40 ± 11 s) steps of the work-to-work test following ET and RST (P < 0.05). The tolerable duration of exercise and the total amount of sprint work completed in the exercise performance test were also similarly enhanced by ET and RST (P < 0.05). Therefore, ET and RST provoked comparable improvements in VO_{2} kinetics and exercise performance in the transition from an elevated baseline work rate, with RST being a more time-efficient approach to elicit these adaptations.

A STEPWISE INCREMENT IN SKELETAL muscle work drives an immediate increase in ATP utilization in the contracting myocytes. In comparison, pulmonary oxygen uptake (VO_{2}), which provides a close approximation of muscle VO_{2} after an initial cardiodynamic phase (phase I) (28), rises more slowly following a step increase in work rate (62, 63). During the transition from a low-intensity unloaded baseline (typically 20 W) to a moderate-intensity work rate [below the gas exchange threshold (GET)] during leg cycle ergometry, VO_{2} increases with monoexponential kinetics in phase II until a steady-state VO_{2} is attained after approximately 2–3 min (62, 63). In contrast, a step increment to a supra-GET work rate elicits a VO_{2} slow component, which delays the attainment of a VO_{2} steady state during heavy-intensity exercise [below the critical power (CP)], or drives VO_{2} to its maximum (VO_{2} max) when severe-intensity exercise (>CP) is continued until the limit of tolerance (53, 63). When a secondary step in work rate is superimposed upon an initial work rate step (i.e., during a so-called work-to-work transition), phase II VO_{2} kinetics are slowed in the upper step compared with both the lower step and when both transitions are combined in a single step (11, 20, 33, 46, 64, 65). Therefore, the rate of oxidative energy transfer is compromised during a step increment in work rate initiated from an elevated baseline of skeletal muscle work.

The rate at which VO_{2} increases following an increment in work rate has implications for the magnitude of the O_{2} deficit incurred and, by extension, the proportion of energy derived through phosphocreatine (PCr) hydrolysis and anaerobic glycolysis. Consequently, this affects the accumulation of metabolites linked to the process of skeletal muscle fatigue [inorganic phosphate (Pi), ADP, hydrogen ions (H^{+}); 36, 52]. Moreover, the VO_{2} slow component has been shown to develop in concert with greater muscle PCr (5, 56) and glycogen (41) utilization during supra-GET exercise. Therefore, speeding phase II VO_{2} kinetics and/or attenuating the VO_{2} slow component would be predicted to attenuate the development of muscle metabolic perturbation and, therefore, improve exercise performance (13, 48, 54).

The most effective intervention to alter the dynamic VO_{2} response during a single transition to a higher rate of skeletal muscle work is a period of exercise training (7–9, 17, 47, 49, 51). Indeed, faster phase II VO_{2} kinetics and/or a reduced VO_{2} slow component have been reported following continuous endurance training (ET) (8, 47, 51), high-intensity interval training (HIT) (8, 9, 17, 47), and repeated all-out sprint training (RST) (7). These adaptations occur rapidly, with phase II VO_{2} kinetics being speeded by 20% after just two training sessions, and 40% after eight sessions of either ET or HIT (47). The VO_{2} slow component elicited during a single step to a severe-intensity work rate can also be lowered after 2 wk of RST (7), or a combination of ET and HIT (67). Recently, Williams et al. (66) showed that 2–4 wk of HIT comprising 8–12 1-min intervals at 110% of peak aerobic power, speeded phase II VO_{2} kinetics in the lower (20 W → 45% GET) and upper (45% GET → 90% GET) steps of a work-to-work exercise test in the moderate-intensity domain by a similar magnitude. However, the effects of exercise training on VO_{2} kinetics and exercise performance during a work-to-work protocol when the upper transition is >GET has yet to be investigated. In everyday living and during athletic competition, work rate changes...
abruptly from a lower to a higher metabolic rate. Therefore, it is important to investigate interventions that might improve \( \text{VO}_2 \) kinetics during work-to-work transitions.

Based on the size principle of motor unit recruitment proposed by Henneman et al. (30), skeletal muscle fibers are recruited in a hierarchical manner according to their recruitment threshold. Specifically, smaller type I (slow twitch) motor units are recruited first to meet low muscle force requirements, whereas greater muscle force requirements mandate the recruitment of larger type II (fast twitch) motor units. Accordingly, the moderate-intensity lower step of a work-to-work exercise test would be hypothesized to engage predominantly type I muscle fibers with the increased muscle force requirements imposed by the upper step being met by the additional recruitment of predominantly type II muscle fibers (29, 41, 42, 61). Therefore, training interventions that engage predominantly type I muscle fibers might be expected to improve physiological responses to a greater extent in the lower step of a work-to-work protocol, whereas training interventions that recruit a greater proportion of type II muscle fibers might be expected to augment physiological responses to a greater extent in the upper step of a work-to-work protocol. It has been reported that muscle glycogen and PCr depletion is observed in type I and II muscle fibers following repeated intervals above \( \text{VO}_2 \) max (22, 26, 27, 29, 41, 42, 60). Conversely, significant muscle glycogen and PCr depletion is observed in type I and II muscle fibers with a preferential depletion of muscle glycogen in type II muscle fibers using this method of training (25, 27, 29, 60) [also see (1) for review]. Moreover, because interval training has been shown to induce greater oxidative enzyme adaptations in type II fibers compared with continuous training (24, 31), and because type IIB fibers manifest greater training-induced elevations in oxidative capacity as training intensity increases above \( \text{VO}_2 \) max (21), oxidative metabolism may be enhanced to a greater extent in type II muscle fibers following RST compared with ET. These fiber-type-specific adaptations might be expected to result in faster \( \text{VO}_2 \) kinetics and enhanced tolerance to severe-intensity exercise initiated from an elevated baseline after RST compared with ET (15, 17, 39). However, it is currently unclear whether there is an optimal training method to speed \( \text{VO}_2 \) kinetics and improve exercise performance in the upper step of a work-to-work protocol.

The purpose of this study was to investigate the effects of ET and RST on \( \text{VO}_2 \) kinetics during moderate-intensity cycle exercise initiated from a low-intensity baseline work rate, and on \( \text{VO}_2 \) kinetics and exercise performance during severe-intensity cycle exercise initiated from a moderate-intensity baseline work rate. We administered a step increment from a low-intensity to a moderate-intensity work rate in an attempt to assess \( \text{VO}_2 \) kinetics when type I muscle fibers are predominantly recruited, and a subsequent step from a moderate-intensity to a severe-intensity work rate to assess \( \text{VO}_2 \) kinetics when a greater proportional recruitment of type II muscle fibers would be expected (41, 42). We also attempted to induce preferential physiological adaptations in type I muscle fibers via ET and in type II muscle fibers via RST (22, 27, 29, 60). We hypothesized that ET and RST would be similarly effective at speeding \( \text{VO}_2 \) kinetics in the low-to-moderate-intensity lower step (L→M), but that RST would be more effective at speeding \( \text{VO}_2 \) kinetics and improving exercise performance during the moderate-to-severe-intensity upper step (M→S) of the work-to-work exercise protocol.

**METHODS**

**Subjects.** Twenty-three healthy subjects (15 men, mean ± SD age 22 ± 5 yr, stature 1.79 ± 0.06 m, body mass 80 ± 15 kg; 8 women, age 18 ± 2 yr, stature 1.49 ± 0.08 m, body mass 59 ± 7 kg) volunteered to participate in this study. None of the subjects were tobacco smokers or users of dietary supplements. The subjects participated in exercise at a recreational level, but were not highly trained. All subjects 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 prior to the commencement of the study after 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 intake for 6 h and 24 h, respectively, before each test. All tests were performed at the same time of day (±2 h).

**Experimental design.** Subjects were required to report to the laboratory on four occasions over the 2 wk preceding the intervention period, with all tests separated by at least 24 h. After completing an initial ramp incremental exercise test, subjects completed a work-to-work exercise test during the three subsequent laboratory sessions for determination of \( \text{VO}_2 \) kinetics and exercise performance. Upon completion of the preliminary tests, subjects were randomly assigned to either the RST, ET, or control (CON) groups. Following the intervention period, subjects returned to the laboratory on three occasions and repeated the preintervention work-to-work exercise tests at the same absolute work rates to determine the effect of the respective training interventions on the physiological and performance parameters.

**Incremental test.** Before the intervention period, subjects completed a ramp incremental exercise test for determination of \( \text{VO}_2 \) max 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 by 30 W/min until the limit of tolerance. The subjects cycled at a self-selected pedal rate (between 70–90 rpm), and this pedal rate, along with saddle and handlebar height and configuration, were 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 \( \text{VO}_2 \) max was taken as the highest 30-s mean value attained prior to 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 (\( \text{VCO}_2 \)) from visual inspection of individual plots of \( \text{VCO}_2 \) vs. \( \text{VO}_2 \); 2) an increase in expired ventilation (\( \text{VE} / \text{VO}_2 \)) with no increase in \( \text{VCO}_2 / \text{VCO}_2 \); and 3) an increase in end-tidal O2 tension with no fall in end-tidal CO2 tension. The data collected during the incremental test were used to calculate the work rates, which were employed during the subsequent work-to-work step tests. Specifically, the work rates that would require 90% of the \( \text{VO}_2 \) at GET (moderate-intensity exercise) and 70% of the difference between the \( \text{VO}_2 \) at the GET and \( \text{VO}_2 \) max (70% Δ; severe-intensity exercise) were estimated with account taken of the mean response time of the \( \text{VO}_2 \) response to ramp exercise.

**Work-to-work exercise tests.** Both before and after the intervention period, subjects completed a work-to-work exercise test on three occasions for the determination of pulmonary \( \text{VO}_2 \) kinetics. Each test began with 3 min of baseline, low-intensity unloaded cycling at 20 W
before an abrupt transition to a moderate-intensity constant work rate equivalent to 90% GET (L→M). Following 4 min of moderate-intensity cycling, the work rate was abruptly increased to a severe-intensity constant work rate equivalent to 70% Δ (M→S). Of the three M→S transitions performed before and after the intervention period, the first was 6 min in duration; the second was continued to the limit of tolerance, which was recorded when the pedal rate fell more than 10 rpm below the required pedal rate; and the third M→S transition was performed for 6 min followed by a 60-s all-out sprint. The resistance on the pedals during the 60-s all-out effort was set using the linear mode of the Lode ergometer so that the subject would attain the power output calculated to be 50% Δ if they attained their preferred cadence (linear factor = power/preferred cadence). Subjects were provided with a 5-s countdown prior to the sprint and were instructed to attain the peak power as quickly as possible and to continue exercising maximally for the duration of the sprint. No time feedback was given to the subjects at any point during either the upper-step constant-work-rate test to limit of tolerance or the all-out sprint.

Training interventions. After completing the initial stage of experimental testing, the subjects were randomly assigned to either an RST group (5 men, 4 women; mean ± SD: age 20 ± 2 yr; height 1.75 ± 0.11 m; body mass 71 ± 12 kg), an ET group (5 men, 3 women; mean ± SD: age 21 ± 2 yr, height 1.76 ± 0.10 m, body mass 84 ± 17 kg), or a CON group (5 men, 1 women; mean ± SD: age 25 ± 7 yr, height 1.73 ± 0.03 m, body mass 69 ± 7 kg). Both training groups 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 (814E bicycle ergometer; Monark, Stockholm, Sweden; [12, 23]). 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 of recovery, during which subjects were permitted to cycle at a low cadence (<50 rpm) against a light resistance (a load that maintained external power output at <30 W) to reduce sensations of nausea. The ET required subjects to cycle continuously on a cycle ergometer (814E bicycle ergometer; Monark) at 80 rpm for a predetermined duration at an intensity corresponding to 90% of the GET. Subjects cycled for 60 min in the first training session, and the session duration was increased by 10 min in each subsequent session until subjects completed 110 min of continuous cycling in the final training session.

Measurements. During all tests, pulmonary gas exchange and ventilation were measured breath-by-breath with subjects wearing a nose clip and breathing through a low-dead-space, low-resistance mouthpiece and impeller turbine assembly (Jaeger Triple V). The inspired and expired gas volume and gas concentration signals were continuously sampled at 100 Hz, the latter using paramagnetic (O₂) and infrared (CO₂) analyzers (Jaeger Oxycron Pro; Hoechberg, Germany) via a capillary line connected to the mouthpiece. The gas analyzers were calibrated before each test with gases of known concentration, and the turbine volume transducer was calibrated with a 3-liter syringe (Hans Rudolph, Kansas City, MO). The volume and concentration signals were time-aligned by accounting for the delay in the capillary gas transit and the analyzer rise time relative to the volume signal. Pulmonary gas exchange and ventilation were calculated and displayed breath-by-breath.

During the exercise tests, a blood sample was collected from a fingertip into a capillary tube over the 20-s preceding the lower and upper steps of the work-to-work exercise test, the 20 s preceding the completion of 360 s of the upper step, and also as soon as possible (<10 s) following the all-out sprint and as soon as possible (<10 s) after exhaustion when the upper step was continued until the limit of tolerance. These whole blood samples were subsequently analyzed to determine blood [lactate] (YSI 1500; Yellow Springs Instruments, Yellow Springs, OH) within 30 s of collection.

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 four standard deviations 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 both the lower and upper step increment in work rate (i.e., the phase I response) were deleted, and a nonlinear least-square algorithm was used to fit the data thereafter. A single-exponential model was used to characterize the moderate exercise VO₂ responses during L→M of the work-to-work protocol, whereas a biexponential model was used for the severe exercise VO₂ responses during M→S of the work-to-work protocol, as described in the following equations:

\[ \dot{V}O_2(t) = \dot{V}O_2_{baseline} + H(t - TD_P) \times A_p \left( 1 - e^{-TD_P/\tau_p} \right) \]  

where \( \dot{V}O_2(t) \) represents the absolute \( \dot{V}O_2(t) \) at a given time \( t \); \( \dot{V}O_2_{baseline} \) represents the mean \( \dot{V}O_2(t) \) in the baseline period; \( A_p \), \( TD_p \), and \( \tau_p \) represent the amplitude, time delay, and time constant, respectively, describing the phase II increase in \( \dot{V}O_2(t) \) above baseline; and \( A_m \), \( TD_s \), and \( \tau_s \) represent the amplitude of, time delay before the onset of, and time constant describing the development of, the \( \dot{V}O_2(t) \) slow component, respectively. \( H(t) \) represents the Heaviside step function as described by Ma et al. (45):

\[ H(t) = \begin{cases} 0, & t < 0, \\ 1, & t \geq 0. \end{cases} \]  

An iterative process was used to minimize the sum of the squared errors between the fitted function and the observed values. \( \dot{V}O_2_{baseline} \) was defined as the mean \( \dot{V}O_2(t) \) measured over the final 90 s of the unloaded baseline cycling period. The \( \dot{V}O_2(t) \) at the end of the L→M was defined as the mean \( \dot{V}O_2(t) \) measured over the final 30 s of exercise. The \( \dot{V}O_2(t) \) at 360 s of M→S was taken as the mean \( \dot{V}O_2(t) \) between 330 s and 360 s, whereas the \( \dot{V}O_2(t) \) at the limit of tolerance (\( T_{lim} \)) was defined as the mean \( \dot{V}O_2(t) \) measured over the final 30 s of the exhaustive M→S exercise bout. Because the asymptotic value (\( A_m \)) of the exponential term describing the \( \dot{V}O_2(t) \) slow component may represent a higher value than is attained at the end of the exercise, the amplitude of the \( \dot{V}O_2(t) \) slow component at the end of exercise was defined as \( A_m' \). The \( A_m' \) parameter was compared at the same iso-time (360 s) for all experimental conditions. The amplitude of the \( \dot{V}O_2(t) \) slow component was also described relative to the entire \( \dot{V}O_2(t) \) response, and its trajectory was calculated by dividing \( A_m' \) by the difference between 360 s and \( TD_s \). In addition, the functional gain (G) of the fundamental (phase I + phase II) \( \dot{V}O_2(t) \) response was computed by dividing \( A_p \) by the \( \Delta \) work rate. To determine the overall kinetics of the \( \dot{V}O_2(t) \) response to both L→M and M→S, the data were also fit with a monoeponential model from 0 s to end-exercise without time delay. This mean response time (MRT) was used to calculate the \( \dot{V}O_2(t) \) deficit incurred during moderate-intensity exercise using the following equation:

\[ \Delta \dot{V}O_2 = MRT(t) \times \Delta \dot{V}O_2 \]  

where \( \Delta \dot{V}O_2 \) is the difference between end-exercise \( \dot{V}O_2(t) \) and \( \dot{V}O_{baseline} \).
interventions on the relevant physiological and performance variables. Two-way ANOVAs were also performed to compare \( \tau_p \) and the fundamental gain between the L\( \rightarrow \)M and M\( \rightarrow \)S steps of the work-to-work exercise protocol before and after the intervention period. When the analysis revealed a significant effect, the origin of the effect was explored using paired-samples \( t \)-tests with the alpha level adjusted via Fisher’s LSD. The relationship between changes in \( \dot{V}O_2 \) kinetics and performance indices were assessed with Pearson product moment correlation coefficients. All data are presented as mean \( \pm \) SD unless otherwise indicated. Statistical significance was accepted when \( P < 0.05 \).

**RESULTS**

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 the formal training for the duration of the study. The control group also self-reported that their activity patterns were consistent between the preintervention and postintervention work-to-work exercise tests. The ET group completed 510 min of moderate-intensity exercise, and the RST group completed 17.5 min of all-out sprint exercise (total session duration including recovery periods equated 157.5 min) over the 2-wk training period. The work rates applied in the moderate-intensity lower step and the severe-intensity upper step of the work-to-work exercise tests were, respectively, 79 \( \pm \) 14 W and 215 \( \pm \) 46 W for RST, 102 \( \pm \) 20 W and 246 \( \pm \) 47 W for ET, and 89 \( \pm \) 11 W and 240 \( \pm \) 17 W for CON.

**Pulmonary \( \dot{V}O_2 \) kinetics prior to the intervention.** The pulmonary \( \dot{V}O_2 \) data from the work-to-work cycle tests are reported in Table 1, with the group mean \( \dot{V}O_2 \) profiles during the L\( \rightarrow \)M and M\( \rightarrow \)S steps illustrated in Fig. 1 for the RST and ET groups. There were no significant differences in the \( \dot{V}O_2 \) phase II time constant (\( \tau_s \)), fundamental amplitude, fundamental gain, MRT, or slow component (upper step only) between the RST, ET, and CON groups prior to the intervention period.

**Table 1. Pulmonary oxygen uptake kinetics during L\( \rightarrow \)M and M\( \rightarrow \)S step increments in work rates in the RST, ET, and CON groups before and after intervention.**

<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><strong>L( \rightarrow )M</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \dot{V}O_2 ) baseline, l/min</td>
<td>0.91 ( \pm ) 0.12</td>
<td>0.88 ( \pm ) 0.10</td>
<td>1.05 ( \pm ) 0.20</td>
<td>1.03 ( \pm ) 0.20</td>
<td>0.89 ( \pm ) 0.12</td>
<td>0.90 ( \pm ) 0.10</td>
</tr>
<tr>
<td>( \dot{V}O_2 ) postexercise, l/min</td>
<td>1.44 ( \pm ) 0.18</td>
<td>1.44 ( \pm ) 0.20</td>
<td>1.77 ( \pm ) 0.31</td>
<td>1.76 ( \pm ) 0.27</td>
<td>1.54 ( \pm ) 0.20</td>
<td>1.57 ( \pm ) 0.10</td>
</tr>
<tr>
<td>Phase II time constant, s</td>
<td>25 ( \pm ) 8</td>
<td>20 ( \pm ) 7§</td>
<td>28 ( \pm ) 4</td>
<td>22 ( \pm ) 4*</td>
<td>25 ( \pm ) 3</td>
<td>23 ( \pm ) 4</td>
</tr>
<tr>
<td>Fundamental amplitude, l/min</td>
<td>0.52 ( \pm ) 0.14</td>
<td>0.52 ( \pm ) 0.19</td>
<td>0.71 ( \pm ) 0.20</td>
<td>0.70 ( \pm ) 0.23</td>
<td>0.62 ( \pm ) 0.05</td>
<td>0.64 ( \pm ) 0.03</td>
</tr>
<tr>
<td>Fundamental gain, ml-min( ^{-1} ) W( ^{-1} )</td>
<td>8.8 ( \pm ) 1.0</td>
<td>8.9 ( \pm ) 1.0</td>
<td>8.6 ( \pm ) 1.0</td>
<td>8.5 ( \pm ) 1.4</td>
<td>9.2 ( \pm ) 1.3</td>
<td>9.5 ( \pm ) 1.9</td>
</tr>
<tr>
<td>Mean response time, s</td>
<td>40 ( \pm ) 8</td>
<td>34 ( \pm ) 8*</td>
<td>40 ( \pm ) 8</td>
<td>32 ( \pm ) 6*</td>
<td>39 ( \pm ) 4</td>
<td>36 ( \pm ) 7</td>
</tr>
<tr>
<td>Oxygen deficit, liter</td>
<td>0.40 ( \pm ) 0.08</td>
<td>0.29 ( \pm ) 0.09</td>
<td>0.46 ( \pm ) 0.14</td>
<td>0.36 ( \pm ) 0.09†</td>
<td>0.40 ( \pm ) 0.05</td>
<td>0.38 ( \pm ) 0.09</td>
</tr>
<tr>
<td><strong>M( \rightarrow )S</strong></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \dot{V}O_2 ) at 360 s, l/min</td>
<td>3.02 ( \pm ) 0.62</td>
<td>2.96 ( \pm ) 0.56</td>
<td>3.48 ( \pm ) 0.61</td>
<td>3.40 ( \pm ) 0.64</td>
<td>3.37 ( \pm ) 0.22</td>
<td>3.36 ( \pm ) 0.25</td>
</tr>
<tr>
<td>Gain at 360 s, ml-min( ^{-1} ) W( ^{-1} )</td>
<td>11.6 ( \pm ) 1.0§</td>
<td>11.4 ( \pm ) 0.8§</td>
<td>11.9 ( \pm ) 1.0§</td>
<td>11.5 ( \pm ) 1.2§</td>
<td>11.9 ( \pm ) 3.1§</td>
<td>11.7 ( \pm ) 2.9§</td>
</tr>
<tr>
<td>( \dot{V}O_2 ) at exhaustion, l/min</td>
<td>3.06 ( \pm ) 0.71</td>
<td>3.05 ( \pm ) 0.58</td>
<td>3.56 ( \pm ) 0.63</td>
<td>3.50 ( \pm ) 0.65</td>
<td>3.29 ( \pm ) 0.16</td>
<td>3.48 ( \pm ) 0.31</td>
</tr>
<tr>
<td>Phase II time constant, s</td>
<td>54 ( \pm ) 7§</td>
<td>50 ( \pm ) 11§</td>
<td>50 ( \pm ) 10§</td>
<td>39 ( \pm ) 11§</td>
<td>50 ( \pm ) 16§</td>
<td>45 ( \pm ) 16§</td>
</tr>
<tr>
<td>Fundamental amplitude, l/min</td>
<td>1.30 ( \pm ) 0.38</td>
<td>1.28 ( \pm ) 0.37</td>
<td>1.40 ( \pm ) 0.30</td>
<td>1.37 ( \pm ) 0.33</td>
<td>1.48 ( \pm ) 0.22</td>
<td>1.52 ( \pm ) 0.16</td>
</tr>
<tr>
<td>Fundamental gain, ml-min( ^{-1} ) W( ^{-1} )</td>
<td>9.5 ( \pm ) 0.7</td>
<td>9.4 ( \pm ) 0.7</td>
<td>9.7 ( \pm ) 0.9</td>
<td>9.4 ( \pm ) 1.0</td>
<td>9.4 ( \pm ) 2.3</td>
<td>9.8 ( \pm ) 2.8</td>
</tr>
<tr>
<td>Slow component amplitude, l/min</td>
<td>0.29 ( \pm ) 0.17</td>
<td>0.29 ( \pm ) 0.12</td>
<td>0.32 ( \pm ) 0.08</td>
<td>0.32 ( \pm ) 0.12</td>
<td>0.40 ( \pm ) 0.11</td>
<td>0.33 ( \pm ) 0.10</td>
</tr>
<tr>
<td>Slow component trajectory, l/min</td>
<td>81 ( \pm ) 47</td>
<td>77 ( \pm ) 30</td>
<td>93 ( \pm ) 27</td>
<td>85 ( \pm ) 28</td>
<td>120 ( \pm ) 26</td>
<td>93 ( \pm ) 21</td>
</tr>
<tr>
<td>Mean response time, s</td>
<td>86 ( \pm ) 22§</td>
<td>68 ( \pm ) 5§</td>
<td>89 ( \pm ) 15§</td>
<td>69 ( \pm ) 10§</td>
<td>76 ( \pm ) 14§</td>
<td>72 ( \pm ) 12§</td>
</tr>
</tbody>
</table>

L\( \rightarrow \)M, a step increment from a low-intensity baseline to a moderate-intensity work rate; M\( \rightarrow \)S, a step increment from a moderate-intensity baseline work rate to a severe-intensity work rate; RST, repeated sprint training; ET, endurance training; CON, control. *Significantly different from Pre \( (P < 0.05) \). †Significantly different from Pre \( (P < 0.01) \). ‡Significantly different from L\( \rightarrow \)M value \( (P < 0.05) \). §Significantly different from L\( \rightarrow \)M value \( (P < 0.01) \).
Postintervention: upper step. Following the intervention periods, \( \tau_p \) was significantly lower following both RST (pre 54 \pm 7 s; post 40 \pm 11 s; \( P < 0.05 \)) and ET (pre 50 \pm 10; post 39 \pm 11 s; \( P < 0.05 \)) during M\( \rightarrow \)S. The MRT was also faster during M\( \rightarrow \)S following both RST and ET (\( P < 0.05 \); Table 1). However, despite the reduced \( \tau_p \) during M\( \rightarrow \)S after RST and ET, the M\( \rightarrow \)S \( \tau_p \) was still longer than the L\( \rightarrow \)M \( \tau_p \) following RST and ET (\( P < 0.01 \); Table 1). There were no significant changes to either the fundamental VO\(_2\) amplitude, VO\(_2\) slow component, or VO\(_2\) gain following either RST or ET (\( P > 0.05 \)). Although the two-way ANOVA revealed a significant group \( \times \) time interaction for the blood [lactate] change during M\( \rightarrow \)S (\( P < 0.01 \)), follow-up analyses indicated that this change in blood [lactate] over M\( \rightarrow \)S was not significantly different following RST (pre 3.3 \pm 2.0; post 2.8 \pm 1.3 mM) or ET (pre 3.6 \pm 2.1; post 3.6 \pm 1.8 mM; \( P > 0.05 \)).

Exercise performance. When the data of all subjects were pooled prior to the intervention period, the tolerable duration of the M\( \rightarrow \)S step was negatively correlated to the \( \tilde{V}O_2 \) MRT during M\( \rightarrow \)S (\( r = -0.42; P < 0.05 \)). There was a significant group \( \times \) time interaction effect (\( P < 0.05 \)) on the tolerable duration of M\( \rightarrow \)S, with RST (pre 550 \pm 114; post 732 \pm 162 s; \( P < 0.01 \)) and ET (pre 491 \pm 109; post 672 \pm 220 s; \( P < 0.05 \); Fig. 2) both improving exercise tolerance. The peak power output (RST pre 318 \pm 132; RST post 366 \pm 124 W; ET pre 351 \pm 114; ET post 389 \pm 127 W; \( P < 0.05 \)), mean power output (RST pre 263 \pm 84; RST post 300 \pm 88 W; ET pre 304 \pm 84; ET post 332 \pm 88 W; \( P < 0.01 \)), and total sprint work performed (RST pre 15.6 \pm 5.0; RST post 17.9 \pm 5.3 kJ; ET pre 18.0 \pm 5.0; ET post 19.7 \pm 5.3 kJ; \( P < 0.01 \)) were significantly enhanced after both RST and ET (Fig. 3). The increase in total sprint work performed was significantly correlated to the speeding of the \( \tilde{V}O_2 \) MRT during M\( \rightarrow \)S in the RST group (\( r = -0.70; P < 0.05 \)), but not in the ET group (\( P > 0.05 \)).

Fig. 1. Group mean \( \tilde{V}O_2 \) responses to a step increment from a low-intensity baseline to a moderate-intensity work rate and a subsequent step to a severe-intensity work rate. Top: the \( \tilde{V}O_2 \) responses pre and post repeated sprint training (RST). Bottom: \( \tilde{V}O_2 \) responses pre and post continuous endurance training (ET). The dashed vertical line at 0 s indicates the transition from a low-intensity work rate (20 W) to a moderate-intensity work rate (90% gas exchange threshold). The dashed vertical line at 240 s indicates the transition from a moderate-intensity work rate to a severe-intensity work rate (70% \( \Delta \)). Data are expressed as a percentage of the end-exercise \( \tilde{V}O_2 \) and shown as 5-s mean values. Note the significantly faster \( \tilde{V}O_2 \) kinetics in the lower and upper steps of the work-to-work exercise test following RST and ET.
DISCUSSION

In this study we tested the hypotheses that RST and ET would elicit a comparable reduction of H9270p during L→M, but that RST would be more effective at reducing H9270p in M→S consequent to greater training adaptations in type II muscle fibers. However, although RST and ET were similarly effective at reducing H9270p during L→M, the principal novel finding from this study was that ET and RST were also similarly effective at reducing H9270p during M→S without impacting the fundamental V˙O2 amplitude or the V˙O2 slow component. This faster adjustment of V˙O2 following the onset of exercise with ET and RST was accompanied by improved exercise tolerance during M→S and a greater amount of sprint work completed in the exercise performance test. Therefore, these findings suggest that short-term (six sessions over 2 wk) ET and RST are equally effective at improving H9270p and exercise performance when a step increment in work rate is superimposed upon an elevated baseline of skeletal muscle work (i.e., during the types of work-rate transitions that characterize many day-to-day and athletic activities). Importantly, these changes were similar despite a profound difference in the total exercise time, indicating that RST is a highly time-efficient method to improve oxidative metabolism and exercise performance.

Prior to the intervention period, V˙O2 kinetics were slower and exercise efficiency was lower (as reflected by a longer V˙O2 τp and higher V˙O2 fundamental gain, respectively) for M→S compared with L→M. Slower V˙O2 kinetics and an increased fundamental V˙O2 gain have also been observed in several other studies when a step increment in work rate was initiated from an elevated baseline work rate (11, 20, 33, 46, 64, 65). The reasons for these differences during work-to-work transitions are unclear [e.g., it is unclear whether it is the elevated baseline work rate and/or the elevated metabolic rate that is responsible (10, 18, 19)]; however, it has been suggested that a greater proportional recruitment of type II muscle fibers might play a role (11, 20, 64, 65). In support of this postulate, it has been reported that PCr utilization is lower in fast-twitch muscle fibers during a moderate-intensity exercise bout initiated from an unloaded baseline than during high-intensity exercise initiated from a moderate-intensity baseline (42). Moreover, PCr and glycolgen depletion are greater in type II muscle fibers when cycling at an intensity corresponding to 80% V˙O2 max compared with cycling at an intensity corresponding to 50% V˙O2 max (41). It has also been shown that neuromuscular blockade of type I muscle fibers slows V˙O2 kinetics and increases the O2 cost of exercise (39), and that selective glycogen depletion of type I muscle fibers, which increased...
type II muscle fiber recruitment, increased the O2 cost of exercise (40). Collectively, these and other (27, 61) findings support the principle of a progressive recruitment of type II muscle fibers as muscle force requirements are increased (30), and suggest that type II muscle fibers have slower phase II VO2 kinetics and lower contractile efficiency. Slower VO2 kinetics and an increased fundamental VO2 gain in M→S compared with L→M in the present study are therefore compatible with a greater recruitment of type II muscle fibers during M→S.

Exercise training provides a potent stimulus to speed VO2 kinetics during the transition from a low-intensity baseline to either a moderate-intensity (7, 8, 47, 49) or heavy/severe-intensity (7–9, 17) work rate. In the present study τp was reduced in L→M following RST and ET, consistent with previous findings that interval and continuous training are similarly effective at accelerating VO2 kinetics during L→M (8, 47). Furthermore, recent data suggest that HIT can also reduce τp during the transition to a moderate-intensity work rate initiated from an elevated metabolic baseline (66). However, whether exercise training can improve VO2 kinetics during M→S [i.e., during a transition when a greater recruitment of higher-order fast-twitch muscle fibers would be expected (27, 41, 42, 61)] and, if so, whether there is an optimal training method to elicit this effect has not been investigated. Because a greater proportion of type II muscle fibers are recruited during repeated intervals above VO2max (22, 25, 27, 29, 60) compared with continuous endurance exercise between 30–80% VO2max (22, 26, 27, 29, 41, 42, 60), we reasoned that changes in oxidative metabolism would be greater in type II muscle fibers following RST than ET (21, 24, 31, 57), and that this would be reflected by a reduced τp for M→S due to the RST intervention. However, our data suggest that RST and ET elicit comparable improvements in τp during M→S. Although this finding is in contrast to our experimental hypothesis, it is in accord with the finding that ET and HIT both reduce τp to a similar extent when severe-intensity exercise is initiated from a low-intensity baseline [L→S (8)]. Therefore, exercise training, provided it imposes a sufficient stimulus for adaptation (7), appears to be effective at speeding VO2 kinetics independent of the initial baseline work rate and independent of whether training is completed at a continuous submaximal intensity or as a series of short-duration, high-intensity intervals. There is evidence that τp is 20% faster after just two sessions of ET or HIT, and 40% faster after eight sessions of either ET or HIT over 19 days, with no differences in the rate at which τp improved across the groups (47). Similar improvements in τp have also been reported during L→S following 22 sessions of HIT or ET over 6 wk (8), and during high-intensity exercise following 24 sessions of HIT or ET over 8 wk (16). Taken together, these findings suggest that the trajectory with which τp is enhanced with longitudinal interval and continuous endurance training is similar, at least up to 8 wk. Conversely, whereas the VO2 slow component during L→S can be lowered by exercise training (7, 8, 67), the VO2 slow component during M→S was not affected by RST or ET in this study. Because the VO2 slow component is lower in M→S relative to L→S (20), it is possible that the reduced potential to lower the VO2 slow component with training in M→S contributed to this finding. Alternatively, it is possible that longer training periods are required to lower the VO2 slow component during M→S than in L→S.

The faster VO2 kinetics following RST and ET in L→M is unlikely to be a function of a greater bulk muscle blood flow [see (52) for review]. Instead, the reduction in τp might be linked to better matching between microvascular O2 supply and muscle O2 consumption (47, 66), improved muscle O2 extraction (7, 38), mitochondrial biogenesis (12), greater oxidative enzyme activity (12, 23), or a combination of these adaptations. In addition, greater bulk muscle blood flow is also unlikely to account for the reduced τp in M→S following RST and ET because severe-intensity exercise completed prior to a work-to-work exercise test, which would be expected to increase muscle blood flow (37), does not reduce τp during M→S (20). Williams et al. (66) have shown that HIT reduces τp during the transition to a higher moderate-intensity work rate (90% GET) from a moderate-intensity baseline (45% GET) without a change in muscle deoxyhemoglobin concentration kinetics, a noninvasive surrogate for muscle O2 extraction. These data suggest that increased microvascular O2 delivery might permit the faster increase in VO2 when a transition to a higher work rate is initiated from an elevated baseline. However, in the present study, because the moderate-intensity baseline was higher and the upper step was to a severe-intensity work rate, it is unclear whether an improved matching between muscle O2 supply and muscle O2 consumption contributed to the reduced τp in M→S following RST and ET herein.

Central to the hypothesis that RST would be more effective than ET at reducing τp in M→S was the expectation that greater activation (and therefore stimulation) of type II muscle fibers would occur during this form of training (25, 27, 29, 60). There is evidence that training comprising a series of intense intervals is more effective than ET at increasing oxidative enzymes (21, 24, 31) and mitochondrial coupling (57) in type II muscle fibers, and that intense interval training can increase capillarization of type II muscle fibers (35). Moreover, peroxisome proliferator-activated receptor-γ coactivator-1α, an important stimulus for mitochondria biogenesis (43), has been shown to increase in type I and II muscle fibers dependent upon the magnitude of metabolic perturbation (50). Finally, conversion of type IIx to type IIa muscle fibers has been reported following a similar RST protocol to that employed herein (2). Individually or in concert, these changes might explain faster VO2 kinetics in M→S following RST.

Although there is strong evidence to support predominant recruitment of type I muscle fibers during ET (22, 26, 27, 29, 41, 42, 60) and increased muscle oxidative enzyme activity in (21, 24, 31) and capillarization of (34) type I muscle fibers following ET, some subjects manifest PCr utilization in, and by inference recruitment of, type II muscles during moderate-intensity exercise (42). Furthermore, there is evidence of duration-dependent type II muscle fiber recruitment during submaximal endurance exercise (27). Therefore, it cannot be excluded that ET recruited and provoked training adaptations in type II muscle fibers in this study, which contributed to the reduced τp during M→S following ET. Indeed, ET has been shown to increase oxidative enzymes in (24) and capillarization of (34) type II muscle fibers, and to promote the conversion of type IIx to type Ia muscle fibers (4), and type II fibers to type I fibers (32). Moreover, because ET has been shown to increase slow-twitch muscle fiber maximal shortening velocity and actomyosin ATPase activity (58), it is possible that a
greater relative contribution from type I fibers (e.g., those that were previously only able to satisfy force requirements during L→M) to force production during M→S, and therefore a reduced recruitment of type II fibers, might have contributed to the lower \( \tau_p \) during M→S following ET. It is also possible that subjects were exercising at a lower relative exercise intensity after ET and RST, which might have affected the fiber populations engaged in L→M and M→S in the posttraining testing. Further research is required to unravel the effect of exercise training methods on muscle fiber energetics and recruitment patterns during a step increment in work rate initiated from an elevated baseline of skeletal muscle work.

Prior to the intervention period, we observed a significant negative correlation between the \( \nu_{O2} \) MRT and time to the limit of tolerance during M→S. This observation is consistent with the notion that \( \nu_{O2} \) kinetics is an important determinant of endurance exercise performance (13, 48, 54) and provides the first evidence to suggest that the rate at which \( \nu_{O2} \) increases during a severe-intensity work rate step initiated from an elevated metabolic baseline has important implications for exercise performance. This contention is further supported by our observation of similar improvements in the limit of tolerance (RST +34%; ET +38%), the \( \nu_{O2} \) MRT (RST −21%; ET −23%) and \( \tau_p \) (RST −26%; ET −22%) during M→S after RST and ET. Others have also shown that the improved exercise tolerance with training is accompanied by faster \( \nu_{O2} \) kinetics (7, 9, 17). However, the constant work rate that was administered in M→S does not adequately reflect the pattern of work-rate distribution that is manifest during athletic competition, which is often terminated with an end-sprint [e.g., (59)]. Therefore, we had subjects complete a 60-s all-out sprint immediately following one of the 6-min M→S steps pre- and posttraining to improve the translational potential of our data. Using this approach, we found a similar improvement in the total sprint work completed following RST (+15%) and ET (+9%) with the increased sprint work after RST, but not ET, being correlated with the speeding of the \( \nu_{O2} \) MRT. We have previously shown that a fast-start pacing strategy speeded \( \nu_{O2} \) kinetics over the initial stages of exercise and increased the total amount of sprint work completed in a 60-s all-out sprint (6), which is in line with the finding of the current study. A faster increase in \( \nu_{O2} \) over the phase II region during M→S would be expected to blunt the depletion of the finite PCR and glycogen substrates and the accumulation of metabolites (Pi, ADP, and \( H^+ \)) implicated in the process of skeletal muscle fatigue (3, 52). In conjunction with increases in muscle glycogen content and buffering capacity (23), the faster \( \nu_{O2} \) kinetics would be expected to delay the attainment of critically high muscle metabolite concentrations and low metabolic substrates that occur at the point of task failure (14), permitting improved exercise tolerance after RST and ET. With regard to the simulated exercise performance test, the reduced \( \tau_p \) during M→S post RST and ET would be expected to afford greater potential for anaerobic energy turnover and metabolite accumulation during the end-sprint, which would allow for completion of a greater amount of sprint work. Therefore, the similar improvements in exercise tolerance and simulated exercise performance after ET and RST may be linked, at least in part, to faster phase II \( \nu_{O2} \) kinetics.

The findings from the present study have important implications for improving exercise performance in recreationally active subjects and suggest that RST is a time-efficient approach to elicit these adaptations. Specifically, similar improvements in \( \nu_{O2} \) kinetics and exercise performance were observed following RST and ET despite a 69% lower total (sprint intervals and recovery periods) training duration in RST. Although RST might also have application for improving health and functional capacity in some patient populations and the elderly, the safety of this approach is unclear. Consequently, HIT comprising 10 repeats of 60 s at 90% of maximal heart rate, might be more appropriate for these populations, and this intervention has been shown to improve metabolic health in patients with type II diabetes (44). However, research suggests that ET may be more effective than HIT at improving \( \tau_p \) in patients with chronic heart failure (55). Further research is required to determine whether an optimal training intervention exists to improve \( \nu_{O2} \) kinetics and exercise tolerance during a step increment in skeletal muscle work in patient populations.

In conclusion, RST and ET were effective at improving \( \tau_p \) during L→M (RST −20%; ET −21%), consistent with previous studies, but this study has shown for the first time that 2 wk of RST and ET are similarly effective at improving \( \tau_p \) during M→S (RST −26%; ET −22%). The faster \( \nu_{O2} \) kinetics post RST and ET were accompanied by improved exercise tolerance during M→S (RST +33%; ET +37%) and increased sprint performance immediately following M→S (total sprint work performed, RST +15%; ET +9%). These findings suggest that RST is an effective and very time-efficient approach to elicit improvements in aerobic fitness compared with traditional ET.

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

AUTHOR CONTRIBUTIONS
Author contributions: M.D.B., S.J.B., F.J.D., and A.M.J. conception and design of research; M.D.B. and S.C. performed experiments; M.D.B. and S.C. analyzed data; M.D.B., S.J.B., F.J.D., and A.M.J. interpreted results of experiments; M.D.B. prepared figures; M.D.B., F.J.D., and A.M.J. drafted manuscript; M.D.B., S.J.B., F.J.D., and A.M.J. edited and revised manuscript; M.D.B., S.J.B., S.C., F.J.D., and A.M.J. approved final version of manuscript.

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