Influence of priming exercise on pulmonary O₂ uptake kinetics during transitions to high-intensity exercise at extreme pedal rates

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DiMenna FJ, Wilkerson DP, Burnley M, Bailey SJ, Jones AM. Influence of priming exercise on pulmonary O₂ uptake kinetics during transitions to high-intensity exercise at extreme pedal rates. J Appl Physiol 106: 432–442, 2009. First published December 4, 2008; doi:10.1152/japplphysiol.91195.2008.—We investigated the pedal rate dependency of the effect of priming exercise on pulmonary oxygen uptake (V˙O₂) kinetics. Seven healthy men completed two, 6-min bouts of high-intensity cycle exercise (separated by 6 min of rest) using different combinations of extreme pedal rates for the priming and criterion exercise bouts (i.e., 35 → 35, 35 → 115, 115 → 35, and 115 → 115 rev/min). Pulmonary gas exchange and heart rate were measured breath-by-breath, and muscle oxygenation was assessed using near-infrared spectroscopy. When the priming bout was performed at 35 rev/min (35 → 35 and 35 → 115 conditions), the phase II V˙O₂ time constant (τ) was not significantly altered (bout 1: 31 ± 7 s vs. bout 2: 30 ± 5 s and bout 1: 48 ± 16 vs. bout 2: 46 ± 21 s, respectively). However, when the priming bout was performed at 115 rev/min (115 → 35 and 115 → 115 conditions), the phase II τ was significantly reduced (bout 1: 31 ± 7 vs. bout 2: 26 ± 5 s and bout 1: 48 ± 16 vs. bout 2: 39 ± 9 s, respectively, P < 0.05). Muscle oxygenation was significantly higher after priming exercise in all four conditions, but significant effects on V˙O₂ kinetics were only evident when muscle O₂ extraction (measured as Δ[deoxyhemoglobin]/ΔV˙O₂) was elevated in the fundamental response phase. These data indicate that prior high-intensity exercise at a high pedal rate can speed V˙O₂ kinetics during subsequent high-intensity exercise, presumably through specific priming effects on type II muscle fibers.

V˙O₂ kinetics; phase II time constant; V˙O₂ slow component; prior exercise; cadence

AN INITIAL BOUT OF HIGH-INTENSITY “PRIMING” EXERCISE profoundly alters the kinetics of pulmonary oxygen uptake (V˙O₂) during a subsequent bout of high-intensity exercise (16, 20, 27). It has been established that priming results in faster “overall” V˙O₂ kinetics, due principally to a marked reduction in the amplitude of the V˙O₂ slow component, often in association with an increase in the amplitude of the fundamental V˙O₂ response but normally with no effect on the phase II time constant (τ; Refs. 5–9, 15, 19, 30, 32, 40, 47, 59).

The precise mechanism(s) responsible for the altered V˙O₂ kinetics after priming remains to be resolved but might include changes in motor unit recruitment (5, 10), and/or O₂ availability (either increased bulk delivery consequent to greater blood flow or a more appropriate matching of regional distribution to active muscle fibers; Refs. 12, 20), and/or metabolic processes intrinsic to the involved fibers (e.g., increased activation of rate-limiting oxidative enzymes and/or greater concentration of putative regulators of mitochondrial respiration; Refs 2, 23, 26). However, a priming effect is generally only observed when both the priming and criterion exercise bouts are performed at an intensity at which the blood lactate concentration ([lactate]) is elevated [i.e., above the gas exchange threshold (GET); Refs. 9, 20]. The Henneman “size principle” posits that skeletal muscle fibers are recruited in an orderly fashion with smaller, more oxidative (type I) fibers recruited first and larger, less oxidative (type II) fibers recruited as the requirement for muscle force production increases (22). Type II fibers possess slower V˙O₂ kinetics and have lower contractile efficiency compared with type I fibers (3, 11, 55), and their recruitment has been suggested to be related to the development of the V˙O₂ slow component during exercise above the GET (1, 25, 28, 34, 42, 52, 57). It is therefore reasonable to consider that priming exercise may exert its effects on V˙O₂ kinetics (faster overall response and reduced V˙O₂ slow component) during subsequent high-intensity exercise by influencing either the recruitment or the metabolic response of type II fibers.

The deoxyhemoglobin 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 can be used to estimate O₂ extraction in the microcirculation during exercise (13, 18, 24, 59). There is evidence that both local O₂ availability and O₂ extraction are increased by priming exercise (12, 13, 24, 26). Slower V˙O₂ kinetics in type II fibers have been attributed to a reduced microvascular pressure head for O₂ and the consequent requirement for greater fractional O₂ extraction to satisfy a given increase in metabolic demand (38). It is therefore possible that oxidative function in type II fibers could benefit more from the effects of priming than could oxidative function in type I fibers.

From the above, it is clear that the use of priming exercise in conjunction with interventions that might alter the proportional contribution of type II fibers to force production at the same relative exercise intensity could be a useful way to explore the mechanistic bases for the effect of priming exercise on V˙O₂ kinetics. It is generally accepted that type II fiber contribution to force production is likely to be greater at very high pedal rates (16, 36, 46). However, previous investigations (5–9, 15, 19, 30–32, 47) that have examined the effect of priming exercise on V˙O₂ kinetics during cycle exercise have generally employed midrange pedal rates during both the priming and criterion bouts (e.g., 60–90 rev/min).

We therefore investigated the potential pedal rate (and hence fiber-type recruitment) dependency of the effect of priming...
exercise on VO\textsubscript{2} kinetics by using different combinations of extreme pedal rates (35 and 115 rev/min) for the priming and criterion exercise bouts (i.e., 35→35, 35→115, 115→35, and 115→115 rev/min). We hypothesized that the characteristic effect of priming exercise (a reduced VO\textsubscript{2} slow component and increased fundamental component amplitude with no change in the phase II τ) would be observed during transitions to high-intensity exercise at 35 rev/min, regardless of the cadence employed during the priming bout. We also hypothesized that similar effects would occur when priming exercise at 35 rev/min preceded exercise at 115 rev/min. In contrast, we hypothesized that a bout of prior exercise performed at 115 rev/min would reduce the phase II τ during subsequent exercise at 115 rev/min through specific effects on the higher order muscle fibers that would be expected to be recruited in both bouts.

**METHODS**

**Subjects**

Seven male subjects (means ± SD; age: 31 ± 8 yr, stature: 1.79 ± 0.02 m, and mass: 81.5 ± 7.5 kg) volunteered and gave written informed consent to participate in this study, which was approved by the University of Exeter Ethics Committee. The subjects were all recreationally active and were familiar with the exercise mode and experimental procedures used in the present study. On test days, subjects were instructed to report to the laboratory in a rested state, having abstained from food, alcohol, and caffeine for the preceding 3 h.

**Experimental Overview**

All testing was conducted in an air-conditioned laboratory at a temperature of 20–22°C. The subjects visited the laboratory on 10 occasions over a 4-wk period to perform exercise tests on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). This device allows for the maintenance of a prescribed constant power output across a wide range of pedal cadences by instantaneously adjusting flywheel resistance via electrical braking.

Testing was conducted at the same time of day (±2 h) for each subject. On each of the first two visits, the subjects completed a ramp incremental exercise test for determination of the cadence-specific peak VO\textsubscript{2} (VO\textsubscript{2peak}) and GET. One test was performed at a pedal rate of 35 rev/min and the other at a pedal rate of 115 rev/min, and the test order was alternated between subjects. On each of the eight subsequent visits, subjects completed two bouts of high-intensity exercise (at a work rate calculated to require 60% of the difference between the GET and VO\textsubscript{2peak}; i.e., 60% “Δ”) separated by 6 min of complete rest. Two repetitions of each priming-criterion-bout cadence combination (35→35, 35→115, 115→35, and 115→115) were performed, and the combinations were presented to subjects in random order. The initial bouts served as the unprimed control (35 unprimed and 115 unprimed). Each laboratory visit was separated by at least 48 h.

**Experimental Procedures**

The ramp incremental exercise tests consisted of 3 min of pedaling at 0 W, followed by a continuous ramped increase in work rate of 30 W/min until the subject was unable to continue. The subjects were asked to maintain the prescribed cadence, and instruction was given if/when they deviated by more than ±5 rev/min. Saddle and handlebar heights were recorded and the same settings were reproduced on subsequent tests. The VO\textsubscript{2peak} was defined as the highest 30-s mean value recorded before the subject’s volitional termination of the test.

The GET was determined from a cluster of measures including the following: 1) the first disproportionate increase in carbon dioxide output (V\textsubscript{CO\textsubscript{2}}) from visual inspection of individual plots of V\textsubscript{CO\textsubscript{2}} vs. VO\textsubscript{2}; 2) an increase in V\textsubscript{E}/VO\textsubscript{2} (where V\textsubscript{E} is expiratory ventilation) with no increase in V\textsubscript{E}/V\textsubscript{CO\textsubscript{2}}; and 3) an increase in end-tidal O\textsubscript{2} tension with no fall in end-tidal CO\textsubscript{2} tension. The work rates that would require 60% of the difference (Δ) between the cadence-specific GET and (VO\textsubscript{2peak}) were estimated for each cadence, with account taken of the mean response time of the VO\textsubscript{2} response to ramp exercise (assumed to approximate two-thirds of the ramp rate, i.e., 20 W; Ref. 53). These work rates were subsequently applied during the constant work-rate protocols.

The subjects returned to the laboratory on eight occasions to perform 3 min of “unloaded” cycling at 20 W, 6 min of cycling at 60% Δ, 6 min of passive rest, 3 min of “unloaded” cycling at 20 W, and 6 min of cycling at 60% Δ. Two repetitions of each possible combination (35→35, 35→115, 115→35, and 115→115) were completed. This protocol provided data for transitions to high-intensity exercise at both 35 and 115 rev/min in the unprimed (4 repetitions) and primed (2 repetitions by 35 rev/min cycling and 2 repetitions by 115 rev/min cycling) states. Importantly, we chose not to adjust the baseline work rate to compensate for the greater cost of internal work at the higher cadence. This approach was used to better isolate the VO\textsubscript{2} response dynamics of higher order fibers (14, 56, 57) and thus provide insight into the contribution made by these fibers under normal (midrange pedal rate) conditions (see Discussion for detail). The VO\textsubscript{2} responses from like transitions were averaged before any analysis was performed to enhance the signal-to-noise ratio and improve confidence in the parameters derived from the model fits (35, 54).

During all tests, pulmonary gas exchange and ventilation were measured continuously using a portable metabolic cart (MetaMax 3B; Cortex Biophysik, Leipzig, Germany). A DVT turbine digital transducer measured inspired and expired airflow, while an electrochemical cell O\textsubscript{2} analyzer and ND infrared CO\textsubscript{2} analyzer simultaneously measured expired gases. Subjects wore a nose clip and breathed through a low-dead-space, low-resistance mouthpiece that was securely attached to the volume transducer. The inspired and expired gas volume and gas concentration signals were continuously sampled 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 using a 3-1 syringe (Hans Rudolph, Kansas City, MO). Pulmonary gas exchange and ventilation were calculated and displayed breath-by-breath. Heart rate (HR) was calculated over the duration of each breath during all tests using short-range radiotelemetry (Polar S610; Polar Electro Oy, Kempele, Finland). During one of the trials under each condition, a blood sample from a fingertip was collected into a capillary tube over the 20 s preceding the step transitions in work rate and within the last 20 s of exercise and subsequently analyzed to determine blood [lactate] (YSI 1500; Yellow Springs Instruments, Yellow Springs, OH). 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, Higashi-ku, Japan). The system consisted of an emission probe that irradiates laser beams and a detection probe, which is positioned several centimeters from the emission probe in an optically dense rubber holder. 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. Therefore, the NIRS data represent a relative change based on the optical density measured in the first datum collected. The [HHb] signal can be regarded as being
essentially blood-volume insensitive during exercise and was therefore assumed to provide an estimate of changes in intramuscular oxygenation status and O2 extraction in the field of interrogation (17). It is presently not possible to determine the relative contribution of myoglobin (Mb) to the total NIRS signal, but it is generally believed that this is relatively small (<10%: e.g., Ref. 49).

The leg was initially cleaned and shaved around the belly of the muscle, and the probes were placed in the holder that 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 optodes did not move during exercise. Pen marks were made around the leg extended at downstroke on the cycle ergometer before the first exercise bout, and NIRS data were collected continuously throughout both bouts. The data were subsequently downloaded onto a personal computer, and the resulting text files were stored on a disk for later analysis.

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 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 (54) and a nonlinear least-square algorithm was used to fit the data, as described in the following biexponential equation:

\[ V_{\text{O2}}(t) = V_{\text{O2 baseline}} + A_{\text{p}}(1-e^{-t/TD_{\text{p}}}) + A_{\text{e}}(1-e^{-t/TD_{\text{e}}}) \]  

(1)

where \( V_{\text{O2}} \) represents the absolute \( V_{\text{O2}} \) at a given time \( t \); \( V_{\text{O2 baseline}} \) represents the mean \( V_{\text{O2}} \) in the baseline period; \( A_{\text{p}} \), \( TD_{\text{p}} \), and \( TD_{\text{e}} \) represent the amplitude, time delay, and time constant, respectively, describing the phase II increase in \( V_{\text{O2}} \) above baseline; and \( A_{\text{e}} \), \( TD_{\text{e}} \), and \( \tau \), represent the amplitude of, time delay before the onset of, and time constant describing the development of, the \( V_{\text{O2}} \) slow component, respectively. An iterative process was used to minimize the sum of the squared errors between the fitted function and the observed values. \( V_{\text{O2 baseline}} \) was defined as the mean \( V_{\text{O2}} \) measured over the final 90 s of baseline pedaling. The end-exercise \( V_{\text{O2}} \) was defined as the mean \( V_{\text{O2}} \) measured over the final 30 s of exercise. The absolute fundamental component amplitude (Absolute \( A_{\text{p}} \)) was defined as the sum of \( V_{\text{O2 baseline}} \) and \( A_{\text{p}} \). Because the asymptotic value \( (A_{\text{e}}) \) of the exponential term describing the \( V_{\text{O2}} \) slow component may represent a higher value than is actually reached at the end of the exercise, the actual amplitude of the \( V_{\text{O2}} \) slow component at the end of exercise was defined as \( A_{\text{e}} \). The amplitude of the slow component was also described relative to the entire \( V_{\text{O2}} \) response. In addition, the functional “gain” \( (G) \) of the fundamental \( V_{\text{O2}} \) response was computed by dividing \( A_{\text{e}} \) 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. A biexponential model similar to that described in Eq. 1 was used, with the exception that the fitting window commenced at the onset of exercise (i.e., at \( t = 0 \)). In addition to the fundamental [HHb] \( \tau \) and \( TD \) derived from the biexponential fit, we also used the fundamental [HHb] amplitude to determine the \( [\text{HHb}]/\Delta V_{\text{O2}} \) during this phase of the response. This ratio indicates the degree of O2 extraction required for a given increment in \( V_{\text{O2}} \) and can therefore provide insight into the dynamic balance between O2 delivery and utilization when \( V_{\text{O2}} \) kinetics are altered. We also fit a single exponential curve without time delay to the fundamental region of the HHb response, as indicated by the biexponential fit (i.e., fitting window constrained from \( t = 0 \) to the TD corresponding to the second exponential term). The “mean response time” (MRT) so derived provides an indication of overall [HHb] response dynamics during this initial phase of adaptation. We chose to express the [HHb] slow component both relative to the entire [HHb] response and as the \( \Delta [\text{HHb}] /\Delta V_{\text{O2}} \). The oxyhemoglobin concentration ([O2Hb]) response does not approximate an exponential (12) and was therefore not modeled. However, we assessed priming-induced changes in [O2Hb] by determining the average [O2Hb] over the final 60 s of baseline pedaling before the exercise transition and the last 30 s of exercise.

We also modeled the HR response to exercise in each condition. For this analysis, 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-square monoexponential model without time delay was used to fit the data, with the fitting window commenced at \( V_{\text{O2}} TD \). The HR time constant (HR \( \tau_{p} \)) so derived provides information on the overall HR response dynamics in the absence of any HR “slow component.” We also used this fit to determine the magnitude of the HR slow component, which was calculated as the difference in HR between TD and the end of exercise.

Statistics

The parameters derived from the modeling of the \( V_{\text{O2}} \), [HHb], and HR data for each cadence were analyzed using one-way repeated measures ANOVA, with Fisher’s least significant difference tests, as appropriate, to identify the location of statistically significant differences between the three conditions. Paired \( t \)-tests were used to compare parameters for 35 unprimed and 115 unprimed. Pearson product moment correlation coefficients were used to assess the relationship between changes in the \( V_{\text{O2}} \) \( \tau_{p} \), [HHb] \( \tau_{p} \), and HR \( \tau_{p} \), and significance was accepted at \( P < 0.05 \). Results are reported as means ± SD.

RESULTS

The \( V_{\text{O2peak}} \) of the subjects was not significantly different between pedal rates (41 ± 3 and 45 ± 6 ml·kg−1·min−1 at 35 and 115 rev/min, respectively, \( P > 0.05 \)). However, the GET occurred at a higher percentage of \( V_{\text{O2peak}} \) at 115 rev/min (54 ± 5 compared with 42 ± 7%; \( P < 0.05 \)). Peak work rates attained in the incremental tests were 311 ± 17 and 318 ± 36 W, and the work rates calculated for 60% \( \Delta \) were 211 ± 9 and 205 ± 26 W at 35 and 115 rev/min, respectively, (in both cases, \( P > 0.05 \)).

Pedal Rate Effect

The \( V_{\text{O2}} \) response to 35 unprimed and 115 unprimed is illustrated for a representative subject in Fig. 1, and the response parameters are reported in Tables 1, 2, 3, and 4. During baseline cycling, \( V_{\text{O2}} \), HR, and blood [lactate] were significantly higher, and [O2Hb] exhibited a greater reduction from resting values for 115 unprimed compared with 35 unprimed (\( P < 0.05 \) for all comparisons). The phase II \( V_{\text{O2}} \) \( \tau \) (48 ± 16 vs. 31 ± 7 s) and the HR \( \tau_{p} \) (51 ± 15 vs. 34 ± 9 s) were both significantly longer for 115 unprimed compared with 35 unprimed (\( P < 0.05 \)). The \( A_{\text{p}} \) and \( G_{p} \) were both significantly lower for 115 unprimed compared with 35 unprimed (\( P < 0.05 \)). The end-exercise \( V_{\text{O2}} \) was greater but the end-exercise gain was lower for 115 unprimed compared with 35
unprimed ($P < 0.05$; Tables 3 and 4). The amplitude of the $\dot{V}O_2$ slow component was not significantly different between the two conditions, although the fractional contribution of the $\dot{V}O_2$ slow component to the total increase in $\dot{V}O_2$ above baseline tended to be greater at the higher pedal rate ($17 \pm 7$ vs. $12 \pm 5\%$).

The fundamental phase $[\text{HHb}]\tau$, MRT, and $\Delta[\text{HHb}]/\Delta\dot{V}O_2$ were all similar for 35 unprimed and 115 unprimed (Tables 1 and 2), but the $[\text{HHb}]$ TD was shorter for 115 unprimed ($P < 0.05$). The $[\text{HHb}]$ slow component and $\Delta[\text{HHb}]/\Delta\dot{V}O_2$ during the slow phase of the response were also similar (Tables 1 and 2).

**Priming Effect**

The priming exercise bouts performed at the two different pedal rates required a similar fraction of cadence-specific peak $\dot{V}O_2$ (35 rev/min: $88 \pm 5$ vs. 115 rev/min: $91 \pm 7\%$). In relation to the cadence-specific unprimed control, baseline blood [lactate] was significantly elevated in all primed bouts ($P < 0.01$; Tables 1 and 2). Similarly, $\Delta\text{blood [lactate]}$ was significantly reduced in all primed bouts ($P < 0.05$) with the values for 35$\rightarrow$35 and 115$\rightarrow$35 being similar, and the values for 115$\rightarrow$115 being significantly less than for 35$\rightarrow$115 (Tables 1 and 2). In relation to the cadence-specific unprimed control, the baseline HR was also significantly elevated in all primed bouts ($P < 0.01$), but there was no significant difference between cadences. In all cases, this HR elevation persisted during exercise so that the end-exercise HR was significantly greater for all primed bouts. The end-exercise HR was similar for 35$\rightarrow$115 and 115$\rightarrow$115 but significantly greater for 115$\rightarrow$35 compared with 35$\rightarrow$35 ($P < 0.05$). Similarly, the baseline and end-exercise $[O_2\text{Hb}]$ were significantly elevated in all primed bouts ($P < 0.05$), with no significant difference between conditions (Tables 1 and 2). The group mean total hemoglobin concentration ($[\text{Hb}]$), $[O_2\text{Hb}]$, and $[\text{HHb}]$ responses are presented in Fig. 2.

The parameters of the $\dot{V}O_2$ response in both the unprimed and primed states at each cadence are reported in Tables 3 and 4, and the group mean $\dot{V}O_2$ responses are illustrated in Fig. 3.

**Thirty-five revolutions per minute.** The baseline $\dot{V}O_2$ at 35 rev/min was significantly elevated by priming exercise at both cadences, but the effect was greater for 115$\rightarrow$35 compared with 35$\rightarrow$35 rev/min (Table 3). Prior exercise at 35 rev/min resulted in a significant increase in the absolute amplitude of the fundamental $\dot{V}O_2$ response and a significant reduction in the amplitude of the $\dot{V}O_2$ slow component, with no change in the phase II $\tau$ (31$\pm$7 vs. 30$\pm$5 s) during subsequent exercise at the same pedal rate. In contrast, prior exercise at 115 rev/min resulted in a significant increase in the absolute amplitude of the fundamental $\dot{V}O_2$ response, no significant change in the amplitude of the $\dot{V}O_2$ slow component, and a significant reduction in the phase II $\tau$ (31$\pm$7 vs. 26$\pm$5 s; $P < 0.05$).

**One-hundred fifteen revolutions per minute.** The baseline $\dot{V}O_2$ at 115 rev/min was significantly elevated when priming exercise was performed at 115 but not at 35 rev/min (Table 4). Prior exercise at 35 rev/min had no significant effects on $\dot{V}O_2$ kinetics, including the phase II $\tau$ (48$\pm$16 vs. 46$\pm$21 s) during subsequent exercise at 115 rev/min. However, prior exercise at 115 rev/min resulted in a significant reduction in the

Table 1. Blood [lactate] and heart rate and oxyhemoglobin and deoxyhemoglobin kinetics during 35 unprimed, 35$\rightarrow$35, and 115$\rightarrow$35

<table>
<thead>
<tr>
<th></th>
<th>35 Unprimed</th>
<th>35$\rightarrow$35</th>
<th>115$\rightarrow$35</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline blood [lactate], mM</td>
<td>0.7$\pm$0.3</td>
<td>2.3$\pm$0.9*</td>
<td>3.0$\pm$0.8*</td>
</tr>
<tr>
<td>End-exercise blood [lactate], mM</td>
<td>3.4$\pm$0.9</td>
<td>3.6$\pm$1.4</td>
<td>4.1$\pm$1.6</td>
</tr>
<tr>
<td>$\Delta$Blood [lactate], mM</td>
<td>2.7$\pm$0.8</td>
<td>1.3$\pm$0.8*</td>
<td>1.2$\pm$1.6*</td>
</tr>
<tr>
<td>Baseline heart rate, beats/min</td>
<td>72$\pm$6</td>
<td>88$\pm$9*</td>
<td>93$\pm$11*</td>
</tr>
<tr>
<td>End-exercise heart rate, beats/min</td>
<td>140$\pm$11</td>
<td>147$\pm$13*</td>
<td>151$\pm$12†</td>
</tr>
<tr>
<td>Heart rate $\tau$, s</td>
<td>34$\pm$9</td>
<td>40$\pm$10</td>
<td>41$\pm$11</td>
</tr>
<tr>
<td>HR slow component, beats/min</td>
<td>12$\pm$8</td>
<td>7$\pm$7*</td>
<td>7$\pm$7*</td>
</tr>
<tr>
<td>Baseline $[O_2\text{Hb}], \text{AU}$</td>
<td>$-13\pm62$</td>
<td>79$\pm$72*</td>
<td>100$\pm$59*</td>
</tr>
<tr>
<td>End-exercise $[O_2\text{Hb}], \text{AU}$</td>
<td>$-152\pm47$</td>
<td>$-79\pm47*$</td>
<td>$-84\pm72*$</td>
</tr>
<tr>
<td>Primary phase $[\text{HHb}]\tau$, s</td>
<td>14$\pm2$</td>
<td>16$\pm5$</td>
<td>16$\pm5$</td>
</tr>
<tr>
<td>Primary phase $[\text{HHb}]$ time delay, s</td>
<td>6$\pm5$</td>
<td>5$\pm1$</td>
<td>5$\pm1$</td>
</tr>
<tr>
<td>Primary phase $[\text{HHb}]$ mean response time, s</td>
<td>22$\pm4$</td>
<td>23$\pm7$</td>
<td>24$\pm11$</td>
</tr>
<tr>
<td>Primary phase $\Delta[\text{HHb}]/\Delta\dot{V}O_2$, AU$^{-1} \cdot$min$^{-1}$</td>
<td>118$\pm70$</td>
<td>139$\pm65*$</td>
<td>141$\pm64*$</td>
</tr>
<tr>
<td>$[\text{HHb}]$ slow component, %</td>
<td>20$\pm7$</td>
<td>8$\pm4*$</td>
<td>8$\pm4*$</td>
</tr>
<tr>
<td>Slow component $\Delta[\text{HHb}]/\Delta\dot{V}O_2$, AU$^{-1} \cdot$min$^{-1}$</td>
<td>197$\pm99$</td>
<td>120$\pm55$</td>
<td>153$\pm88$</td>
</tr>
</tbody>
</table>

Values are means $\pm$ SD. 35 Unprimed, unprimed cycling at 35 rev/min; 35$\rightarrow$35, 35 rev/min cycling primed by 35 rev/min cycling; 115$\rightarrow$35, 35 rev/min cycling primed by 115 rev/min cycling; 115$\rightarrow$35, 35 rev/min cycling primed by 115 rev/min cycling by 115 rev/min cycling; $\tau$, time constant; $\tau_p$, time constant for the phase II increase in $\dot{V}O_2$ above baseline; AU, arbitrary units. Brackets indicate concentration. *Significantly different from 35 Unprimed condition ($P < 0.05$). †Significantly different from 35$\rightarrow$35 condition ($P < 0.05$).
phase II $\tau$ (48 ± 16 vs. 39 ± 9 s; $P < 0.05$) with no change in the other kinetic parameters. The extent of the reduction in phase II $\tau$ after priming was correlated with the difference in phase II $\tau$ between 35 unprimed and 115 unprimed ($r = 0.93; P < 0.01$); no significant correlations existed for 35→35, 115→35, or 35→115.

There were no significant differences in the fundamental phase [HHb] kinetics parameters ($\tau$, TD, or MRT) after priming at either cadence. The fundamental phase $\Delta$[HHb]/$\Delta$V\textsubscript{O2} was significantly higher after priming for 35→35, 115→35, and 115→115 ($P < 0.05$) but not for 35→115. Similarly, the [HHb] slow component was reduced by priming in all conditions except for 35→115. There was no significant change in the $\Delta$[HHb]/$\Delta$V\textsubscript{O2} during the slow component phase of the response after priming for any condition.

**DISCUSSION**

The principal finding of this investigation was that the effect of priming exercise on V\textsubscript{O2} kinetics differed according to the combination of cadences employed in the priming and criterion bouts. Specifically, consistent with previous studies, priming at 35 rev/min altered the amplitudes of the V\textsubscript{O2} fundamental and slow components during subsequent cycling at 35 rev/min without changing the phase II $\tau$. In contrast, priming at 35 rev/min had no significant effect on V\textsubscript{O2} kinetics during cycling at 115 rev/min. However, the most striking effects occurred when the priming exercise was performed at 115 rev/min: with this intervention, the phase II $\tau$ was significantly reduced during subsequent exercise irrespective of the pedal rate employed. These unexpected results contribute significantly to our understanding of the mechanistic bases of the effects of priming exercise on V\textsubscript{O2} kinetics by indicating that these effects are pedal-rate (and perhaps fiber-type recruitment) specific.

Manipulation of pedal rate at the same relative exercise intensity is potentially a useful way to explore the influence of muscle fiber type and motor unit recruitment on V\textsubscript{O2} kinetics (28). Investigations have shown that the contribution of type II fibers may be greater at high movement frequencies (36, 46) and, correspondingly, studies that have compared V\textsubscript{O2} kinetics at extreme pedal rates have reported a reduction in the gain of the fundamental component (1), an increase in the V\textsubscript{O2} slow component (39, 43, 50), and a tendency for a longer phase II $\tau$ (43) at high compared with low cadences. These differences were attributed to an increased contribution of higher order (type II) fibers to force production at higher cadences. Type II fibers are believed to possess slower V\textsubscript{O2} kinetics and have lower contractile efficiency than type I fibers (e.g., 3, 11, 28, 55).

**Table 3. O\textsubscript{2} uptake kinetics during 35 unprimed, 35→35, and 115→35**

<table>
<thead>
<tr>
<th></th>
<th>35 Unprimed</th>
<th>35→35</th>
<th>115→35</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline V\textsubscript{O2}, l/min</td>
<td>0.63±0.05</td>
<td>0.70±0.04*</td>
<td>0.74±0.07**†</td>
</tr>
<tr>
<td>Phase II $\tau$, s</td>
<td>31±7</td>
<td>30±5</td>
<td>26±5**†</td>
</tr>
<tr>
<td>Phase II time delay, s</td>
<td>13±3</td>
<td>11±3</td>
<td>13±4</td>
</tr>
<tr>
<td>Fundamental phase amplitude, l/min</td>
<td>2.03±0.17</td>
<td>2.08±0.15</td>
<td>2.07±0.17</td>
</tr>
<tr>
<td>Fundamental phase gain, ml·min$^{-1}$·W$^{-1}$</td>
<td>10.6±0.5</td>
<td>10.9±0.5</td>
<td>10.8±0.6</td>
</tr>
<tr>
<td>Fundamental phase absolute amplitude, l/min</td>
<td>2.66±0.18</td>
<td>2.77±0.17*</td>
<td>2.81±0.22*</td>
</tr>
<tr>
<td>V\textsubscript{O2} slow component time delay, s</td>
<td>139±48</td>
<td>146±41</td>
<td>149±66</td>
</tr>
<tr>
<td>V\textsubscript{O2} slow component amplitude, l/min</td>
<td>0.27±0.10</td>
<td>0.20±0.06*</td>
<td>0.20±0.12</td>
</tr>
<tr>
<td>V\textsubscript{O2} slow component relative amplitude, %</td>
<td>12.5</td>
<td>9±3*</td>
<td>9±5</td>
</tr>
<tr>
<td>End-exercise V\textsubscript{O2}, l/min</td>
<td>2.92±0.12</td>
<td>2.97±0.12</td>
<td>3.00±0.21</td>
</tr>
<tr>
<td>End-exercise gain, ml·min$^{-1}$·W$^{-1}$</td>
<td>12.0±0.4</td>
<td>11.9±0.3</td>
<td>11.8±0.4</td>
</tr>
</tbody>
</table>

Values are means ± SD. *Significantly different from 35 Unprimed condition ($P < 0.05$). †Significantly different from 35→35 condition ($P < 0.05$).
For dynamic contractions at a given power output, oxygen uptake is greater at high compared with low contraction frequencies due to the greater cost of internal work and also to a higher rate of energy turnover (1, 16, 51). In previous investigations into the effect of pedal rate on \( \dot{V}O_2 \) kinetics, high-intensity cycle transitions have been initiated either from complete rest (50) or from baseline work rates that have been adjusted to compensate for the increased \( O_2 \) cost of unloaded cycling at the highest cadence (1, 43). However, high-intensity cycle transitions from an elevated baseline metabolic rate (work-to-work transitions) are characterized by slower phase II \( \dot{V}O_2 \) kinetics, a reduced \( \dot{V}O_2 \) slow component, and a greater total response gain compared with transitions initiated from a baseline of unloaded cycling (14, 56, 57). It has been suggested that such work-to-work transitions functionally isolate the metabolic response characteristics of muscle fibers that are positioned higher in the recruitment hierarchy (4, 14, 56, 57). Therefore, it is possible that the matching of baseline \( \dot{V}O_2 \) in earlier studies masked some of the differences in \( \dot{V}O_2 \) kinetics that exist at extreme pedal rates. In the present study, we deliberately employed transitions from the same 20-W baseline for both cadences so that energy turnover during baseline cycling would be greater at 115 rev/min and the differences between the response characteristics of lower- and higher order fibers at the different pedal rates would be amplified.

**Effects of Pedal Rate on \( \dot{V}O_2 \) Kinetics in the Control (Unprimed) Condition**

A novel observation in the present study was that the phase II \( \dot{V}O_2 \) \( \tau \) was significantly longer for 115 unprimed compared with 35 unprimed. The HR \( \tau \) was also significantly longer at the higher pedal rate, which might be interpreted to indicate that limited blood flow (and, by extension, \( O_2 \) delivery) was responsible for the slower phase II \( \dot{V}O_2 \) kinetics observed at 115 unprimed compared with 35 unprimed. However, Ferguson et al. (16) have reported that muscle blood flow was actually higher at a contraction frequency of 100 compared with 60 kicks/min during knee extension exercise. Moreover, Ferreira et al. (18) have shown a similar [HHb] profile (assessed using NIRS) throughout incremental cycle exercise at 100 compared with 60 rev/min and, in the present study, the fundamental phase \( \Delta[\text{HHb}]/\Delta\dot{V}O_2 \) was similar for 35 unprimed and 115 unprimed. Similar \( O_2 \) extraction (as indicated by the [HHb] response) would not be expected if bulk \( O_2 \) delivery kinetics were limiting muscle \( O_2 \) uptake at faster pedal rates. Slower phase II \( \dot{V}O_2 \) kinetics could also be an indication of a limited capacity for the recruited muscle fibers to utilize the available \( O_2 \). Higher order fibers typically possess higher total creatine content, a greater propensity for substrate-level phosphorylation, a lower oxidative capacity, a reduced microvascular pressure head for \( O_2 \), and slower \( \dot{V}O_2 \) kinetics compared with lower order fibers (3, 11, 38, 44, 60). Therefore, it is possible that the slower phase II \( \dot{V}O_2 \) kinetics we observed at 115 unprimed compared with 35 unprimed reflected the oxidative metabolic properties of the population of muscle fibers that were principally activated. However, it is also possible that regional heterogeneities of perfusion relative to metabolic rate (3, 45) were exacerbated when the contribution of higher order fibers to force production was increased at the higher cadence.

**Effects of Priming on \( \dot{V}O_2 \) Kinetics at Extreme Pedal Rates**

A large number of previous studies have identified the characteristic effects of priming exercise (i.e., a marked attenuation of the \( \dot{V}O_2 \) slow component, often with an associated increase in the amplitude of the phase II \( \dot{V}O_2 \) response, but with no change in the phase II \( \tau \)) during upright cycle exercise in healthy young humans (e.g., 5–9, 15, 19, 24, 27, 30–32, 40, 47, 59). In some instances, reductions in the phase II \( \tau \) have also been reported, but generally only when the phase II \( \tau \) value is large (i.e., the kinetic adaptation is slow) in the control condition (21, 24, 31, 48). However, previous investigations have typically employed midrange pedal rates (e.g., 70–90 rev/min) for both the priming and criterion exercise bouts. In the present study, we elected to use extreme pedal rates (35 and 115 rev/min) in both the priming and criterion exercise bouts to investigate the potential fiber-type dependency of the effect of priming exercise on \( \dot{V}O_2 \) kinetics.

Consistent with our hypothesis and in agreement with previous observations at midrange pedal rates, priming at 35 rev/min reduced the amplitude of the \( \dot{V}O_2 \) slow component and increased the fundamental component amplitude but did not alter the phase II \( \tau \) during subsequent cycling at 35 rev/min. Priming exercise (particularly when of high intensity and resulting in a metabolic acidosis) would be expected to result in muscle vasodilatation and a rightward shift of the oxyhemoglobin dissociation curve and thus to increase both convective and diffusive components of muscle \( O_2 \) delivery (20). Indeed, priming has been shown to increase cardiac output, muscle blood flow, and muscle oxygenation before and during subsequent exercise (5, 13, 19, 26, 33, 59). Therefore, the
Fig. 2. Group mean total hemoglobin concentration ([Hb]; A), oxyhemoglobin concentration ([O$_2$Hb]; B), and deoxyhemoglobin concentration ([HHb]; C) responses during baseline cycling and following the onset of exercise at 35 (top) and 115 rev/min (bottom). , Unprimed conditions (35 unprimed and 115 unprimed); ◊, priming by 35 rev/min cycling (35→35 and 35→115); ●, priming by 115 rev/min cycling (115→35 and 115→115). Vertical dashed line represents the abrupt transition to the higher work rate. Note that baseline total [Hb] and [O$_2$Hb] are elevated in all cases after priming and that the elevation persists throughout exercise. For clarity, data are displayed as 5-s bin averages.
The priming effect we observed for 35→35 might have been caused by increased muscle O2 availability. However, VO2 kinetics were not significantly altered for 35→115, despite significant elevations of blood [lactate], HR, and [O2Hb], suggesting that factors other than changes in bulk O2 delivery might have been responsible for the priming effects observed. Another explanation for the changes in the amplitudes of the fundamental and slow components of VO2 in the 35→35 condition is that priming alters motor unit recruitment in the second compared with the first bout of high-intensity exercise. Support for this notion comes from Burnley et al. (5), who reported an increase in leg muscle integrated electromyogram during the first 2 min of high-intensity cycle exercise after priming in association with an increased phase II amplitude and decreased VO2 slow component. The authors suggested that priming increased motor unit recruitment at the onset of subsequent exercise, which allowed the phase II amplitude to project to a value that was closer to what was required later in the bout. If this phenomenon depends on the involved fibers having experienced a recent (e.g., within 45 min; Ref. 8) exercise bout involving similar motor unit recruitment patterns, it could perhaps explain the lack of effect for 35→115 despite the completion of an identical priming bout and the similar residual effects on blood [lactate], HR, and [O2Hb]. The lack of effect on VO2 kinetics when priming exercise at 35 rev/min preceded exercise at 115 rev/min was not expected and might indicate that the priming bout was not sufficiently specific to invoke the anticipated changes in the VO2 response to the subsequent exercise at the much higher pedal rate.

In accordance with our hypothesis, priming at 115 rev/min reduced the phase II τ but did not alter the VO2 fundamental or slow component amplitudes during cycling at 115 rev/min. This result is in agreement with a number of previous investigations of priming exercise where the phase II τ was large in...
the control condition. For example, the phase II $\tau$ was reduced after priming during supine cycling (unprimed $\tau$, $\sim 38$ s; Ref. 24), during arm crank exercise with the arms above heart level (unprimed $\tau$, $\sim 50$ s; Ref. 30), and in older subjects during moderate-intensity cycling (unprimed $\tau$, $\sim 50$ s; Ref. 48). However, in these investigations, $O_2$ delivery in the control condition was likely constrained (due to body position and age-related changes in the blood flow adaptation to exercise, respectively) and the effect could be attributed, in part, to improved muscle perfusion and $O_2$ delivery after priming. In the present study, it is less likely that the lengthened phase II $\tau$ observed for 115 unprimed ($\sim 48$ s) was due to restricted $O_2$ supply [see Effects of Pedal Rate on $V_O_2$ Kinetics in the Control (Unprimed) Condition]. Moreover, the significant elevation of baseline HR and $[O_2Hb]$ in the 35$\rightarrow$115 condition did not result in the same speeding of $V_O_2$ kinetics that was observed for 115$\rightarrow$115. These results suggest that the priming effect we observed for 115$\rightarrow$115 originated within the involved myocytes (e.g., priming of intramuscular oxidative mechanisms that allowed the $O_2$ being delivered to be used more rapidly; see also Effects of Priming on Muscle Oxygenation at Extreme Pedal Rates). In this respect, the increased $O_2$ availability afforded by the performance of prior exercise could be said to have facilitated, rather than caused, the reduction in the phase II $\tau$ observed (12, 27). It is also possible, however, that regional heterogeneities of perfusion relative to metabolic rate (38, 45) were alleviated only when higher order fibers were previously activated at high contraction frequencies.

An unexpected result from the present study was that, contrary to our hypothesis, the effect of priming at 115 rev/min was to reduce the phase II $V_O_2\tau$ during subsequent cycling at 35 rev/min. This latter result was surprising because the $\tau$ in the control condition at 35 rev/min was not unusually long ($\sim 31$ s). However, type II fibers would still be expected to be involved in a transition to high-intensity exercise even at a very low cadence such as 35 rev/min. If priming at the faster cadence was necessary to accelerate $V_O_2$ kinetics in these fibers, this would explain the reduced phase II $\tau$ in both the 115$\rightarrow$35 and 115$\rightarrow$115 conditions, as well as the lack of effect for 35$\rightarrow$35 and 35$\rightarrow$115 in the present study and also in previous studies where midrange pedal rates were used for priming. Although speculative, our results imply that a reduction in the phase II $\tau$ during high-intensity upright cycle exercise is possible when the priming exercise bout necessitates a significant contribution of type II fibers to force production. The precise mechanism responsible for the effects observed cannot be ascertained from the present data but prior engagement of these higher order fibers might be expected to increase $O_2$ supply and/or the activity of rate-limiting enzymes in the oxidative chain or to alter motor unit recruitment thresholds when these same fibers are recruited during subsequent exercise (10, 23, 27, 41).

Effects of Priming on Muscle Oxygenation at Extreme Pedal Rates

The NIRS data indicated that total [Hb] and $[O_2Hb]$ were increased in the area of interrogation as a consequence of priming exercise. Notably, the kinetic parameters (TD, $\tau$, and MRT) for the adjustment of [HHb] during exercise were not significantly altered by priming, indicating that muscle $O_2$ delivery was well matched to the requirements in the early minutes of exercise. DeLorey et al. (13) suggested that the observation of a slower rate of [HHb] adaptation in conjunction with a reduced phase II $\tau$ in older adults provided evidence that an $O_2$ delivery limitation in the control condition had been alleviated by priming exercise. In contrast, in the present study, the rate of [HHb] adaptation was not different in those conditions in which the phase II $\tau$ was reduced by priming. Our data are consistent with Jones et al. (26) and DeLorey et al. (12) in showing that the [HHb] MRT is not altered by prior exercise but differ from the data of Marles et al. (37) in which the [HHb] MRT was significantly reduced. However, the $\Delta[HHb]/\Delta V_O_2$, which might be used to infer muscle $O_2$ extraction, over the fundamental phase of the response was significantly increased for 35$\rightarrow$35, 115$\rightarrow$35, and 115$\rightarrow$115, although not for 35$\rightarrow$115. Interestingly, 35$\rightarrow$115 was also the only condition where there was no effect of priming on $V_O_2$ kinetics. An increased fundamental phase $\Delta[HHb]/\Delta V_O_2$ after priming has been reported previously (12) and is indicative of a greater proportional contribution of $O_2$ extraction to satisfy a given increase in $V_O_2$. The data therefore indicate that enhanced $V_O_2$ kinetics after priming exercise was contingent upon the involved muscle fibers having an improved ability to utilize the available $O_2$; when greater $O_2$ extraction was not achieved (during the 35$\rightarrow$115 condition), $V_O_2$ kinetics were not altered despite $O_2$ availability being increased to a similar extent as for the other three conditions. One interpretation is that, in the 35$\rightarrow$115 condition, type II fibers recruited in the second exercise bout were not sufficiently "primed" by the initial exercise bout.

The [HHb] slow component was significantly reduced for 35$\rightarrow$35, 115$\rightarrow$35, and 115$\rightarrow$115 (in which $V_O_2$ kinetics were altered), although not for 35$\rightarrow$115 (in which $V_O_2$ kinetics were unchanged). In keeping with earlier studies (26, 37), there was limited agreement between changes in the $V_O_2$ slow component and changes in the [HHb] slow component after prior exercise. This might not be considered surprising given that the rising $V_O_2$ during the slow phase of the response could be satisfied by increased muscle $O_2$ delivery, increased $O_2$ extraction, or both. However, the $\Delta[HHb]/\Delta V_O_2$ in the slow phase of the response was not significantly altered by prior exercise, suggesting that changes in [HHb] and $V_O_2$ were generally proportional, and that reductions in the $V_O_2$ slow component with priming might be associated with increased muscle $O_2$ availability, as has been previously suggested (58).

Some methodological issues with regard to the NIRS measurements in the present study should be highlighted. It is not clear whether changes in NIRS optical parameters such as the differential path length factor and the degree of light scattering occur during constant-work-rate exercise, during repeated bouts of exercise, and at different pedal rates. This makes it difficult to confidently quantify absolute changes in $O_2$ extraction resulting from any intervention. However, the available evidence suggests that differences in scattering between the pedal rates will be minimal at the wavelengths used in the present study (18). We expressed the $\Delta[HHb]$ data for the two pedal rates relative to the resting baseline value. The amplitude values within conditions showed good reproducibility on different days (unpublished observations) and therefore differences between conditions might be expected to have a physiological origin; this remains to be confirmed, however. It
should also be noted that the NIRS data only reflect changes within the superficial area of muscle under interrogation and as such may not be representative of the entire muscle mass. Moreover, our NIRS measurements were made at only one location, and it is known that the dynamics of muscle oxygenation express significant heterogeneity at different locations across the quadriceps muscles of healthy subjects after the onset of exercise (29).

In animal models, both Hogan (23) and Behnke et al. (2) have reported that prior contractile activity resulted in faster activation of muscle oxidative metabolism during subsequent contractions. It is unclear why pulmonary VO2 measurements only rarely demonstrate a similar effect in exercising humans (i.e., a reduced phase II τ after priming). One possibility is that the characteristic alteration in the proportional contribution of the fundamental and slow components to the total VO2 response after priming at midrange pedal rates reflects the conflation of two different priming-related phenomena. The VO2 slow component might reflect an increased activation of fibers (lower and/or higher order) as exercise proceeds (34) and/or slower VO2 kinetics of higher order fibers recruited at or close to exercise onset (57). In addition to altering the motor unit activation patterns during subsequent exercise (5), priming might accelerate oxidative phosphorylation in these fibers such that they reach (or project towards) their individual “steady-state” VO2 values more rapidly (2, 23). However, during exercise transitions typically used in priming investigations (i.e., transitions to high-intensity exercise from very low metabolic rates at midrange pedal rates), these two effects might be indistinguishable (i.e., the speeding of VO2 kinetics in higher order fibers might appear as an amplitude shift from slow to fundamental phase because the phase II response is primarily established by low-order fibers with faster kinetics). In the present study, the distinctly different effects we observed for 35→35 and 115→115 might indicate that the use of extreme pedal rates and the difference in the baseline metabolic rate allowed us to differentiate between these two different priming-induced alterations.

In conclusion, we have shown that despite similar elevations in HR and [O2Hb] after priming, the effect of prior high-intensity exercise on VO2 kinetics during subsequent high-intensity exercise differed according to the combination of pedal rates employed in the priming and criterion bouts. When the priming and criterion exercise bouts were completed at the same low pedal rate, the “classical” effects of elevated fundamental component VO2 and reduced VO2 slow component with no effect on the phase II τ were observed; when the priming bout involved a low pedal rate, no effects on VO2 kinetics during subsequent exercise at a high pedal rate were measured; however, when the priming bout involved a high pedal rate, the phase II τ was significantly reduced during subsequent exercise irrespective of the pedal rate employed. Significant effects on VO2 kinetics after priming were only evident when muscle O2 extraction (measured as Δ[HbH]/ΔVO2) was elevated. These data indicate that factors intrinsic to the recruited muscle fibers might be responsible for mediating the alterations in VO2 kinetics that attend the performance of priming exercise.

REFERENCES


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