Short-term interval training at both lower and higher intensities in the severe exercise domain result in improvements in \( \dot{V}O_2 \) on-kinetics

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Abstract

Purpose Although high-intensity interval training (HIT) seems to promote greater improvements in aerobic parameters than continuous training, the influence of exercise intensity on \( \dot{V}O_2 \) on-kinetics remains under investigation.

Methods After an incremental test, twenty-one recreationally trained cyclists performed several time-to-exhaustion tests to determine critical power (CP), and the highest intensity (I\(^{\text{HIGH}}\)) and the lowest exercise duration (T\(^{\text{LOW}}\)) at which \( \dot{V}O_2\text{max} \) is attained during constant exercise. Subjects also completed a series of step transitions to moderate- and heavy-intensity work rates to determine pulmonary \( \dot{V}O_2 \) on-kinetics. Surface electromyography (EMG) of vastus lateralis muscle and blood lactate accumulation (\( \Delta \text{BLC} \)) was measured during heavy exercise. Subjects were assigned to one of two 4-week work-matched training groups: the lower [105 \% CP: \( n = 11; 4 \times 5 \text{ min at } 105 \% \text{ CP} \) (218 ± 39 W), 1 min recovery] or the upper [I\(^{\text{HIGH}}\): \( n = 10; 8 \times 100 \% \) I\(^{\text{HIGH}}\) (355 ± 60 W), 1:2 work:recovery ratio] intensity of the severe exercise domain.

Results The two interventions were similarly effective in reducing the phase II \( \dot{V}O_2 \) time constant during moderate (105 \% CP: 34 ± 13 to 25 ± 8 s; I\(^{\text{HIGH}}\): 31 ± 9 to 23 ± 6 s) and heavy exercise (105 \% CP: 25 ± 7 to 18 ± 5 s; I\(^{\text{HIGH}}\): 27 ± 7 to 16 ± 5 s) and in reducing the amplitude of \( \dot{V}O_2 \) slow component, EMG amplitude, and \( \Delta \text{BLC} \) during heavy exercise.

Conclusion In conclusion, the short-term adjustments in response to step transitions to moderate and heavy exercise were independent of training intensity within the severe exercise domain.

Keywords Exercise intensity domains · Cycling · Lactate threshold · Exercise tolerance · \( \dot{V}O_2 \)

Abbreviations

\( \tau \) Time constant
\( \Delta 50 \% \) Intensity requiring 50 \% of the difference between the LT and \( \dot{V}O_2\text{max} \)

BLC Blood lactate concentration
CP Critical power
EMG Electromyography
HIT High-intensity interval training
I\(^{\text{HIGH}}\) The highest constant intensity at which \( \dot{V}O_2\text{max} \) is attained
LT Lactate threshold
MRT Mean response time
RMS Root mean square
TD Time delay
T\(_{\text{LIM}}\) Time-to-exhaustion
T\(_{\text{LOW}}\) The lowest exercise duration at which \( \dot{V}O_2\text{max} \) is attained
\( \dot{V}O_2 \) Pulmonary oxygen uptake
\( \dot{V}O_2\text{max} \) Maximum oxygen uptake
\( \dot{V}O_{2\text{SC}} \) \( \dot{V}O_2 \) slow component
\( W^\prime \) Work capacity above critical power

Introduction

After the onset of moderate-intensity exercise, i.e., performed below the lactate threshold (LT), pulmonary \( O_2 \)
uptake ($\dot{V}O_2$) rises with a near exponential kinetics to achieve a steady state within 2–3 min in healthy adults (Whipp and Wasserman 1972). In this regard, the time constant ($\tau$) describing the rate at which $\dot{V}O_2$ rises in the fundamental phase (phase II) to attain the steady state may represent an important determinant of $O_2$ delivery and utilization (Powers et al. 1985). In addition, in healthy participants, the $\dot{V}O_2$ on-kinetics are similar to the kinetics of oxygen uptake measured across the exercising limb (Koga et al. 2014), and have, therefore, been used to resolve the essential control mechanisms of muscle energetics and oxidative function.

At exercise intensities above the LT, but below the critical power (CP), i.e., heavy-exercise domain, the phase II is supplanted by a delayed-onset $\dot{V}O_2$ slow component ($\dot{V}O_{2SC}$), which delays the attainment of the predictable steady state (Berger et al. 2006; Burnley and Jones 2007; Jones et al. 2011). This phenomenon has been partly associated with additional motor unit recruitment (Krøstrup et al. 2004; Jones et al. 2011; Grassi et al. 2015) as exercise proceeds and the initially recruited fibres become fatigued. Accordingly, $\dot{V}O_{2SC}$ has been related to increased metabolic inefficiency within the active muscles and may be associated to the dynamics of blood lactate accumulation (Roston et al. 1987; Berger et al. 2006). For a given metabolic demand, fast $\dot{V}O_2$ on-kinetics mandates a smaller oxygen deficit, less substrate-level phosphorylation, and high exercise tolerance (Murgatroyd et al. 2011). As $\dot{V}O_2$ on-kinetics determine aerobic energy provision during exercise below maximum oxygen uptake ($\dot{V}O_{2max}$), understanding the mechanisms by which $\dot{V}O_2$ on-kinetics may be speeded by physical training is relevant for both athletic populations and subjects with slowed $\dot{V}O_2$ on-kinetics (Rossiter 2011; Poole and Jones 2012).

Accordingly, it is not surprising that endurance training has been shown to speed up the fundamental $\dot{V}O_2$ response (McKay et al. 2009; Berger et al. 2006; Burnley and Jones 2007; Bailey et al. 2009) and to reduce the $\dot{V}O_{2SC}$ amplitude (Berger et al. 2006; Burnley and Jones 2007; Bailey et al. 2009; Jones et al. 2011). Among these interventions, sprint interval training (SIT), high-intensity interval training (HIT), and continuous endurance training are effective methods of improving endurance capacity (Laursen and Jenkins 2002; Burgomaster et al. 2005). However, while HIT has been suggested to induce superior improvements in $\dot{V}O_{2max}$ than continuous endurance training (Wenger and Bell 1986; Buchheit and Laursen 2013; Milanovic et al. 2015), the optimal intervention to enhance $\dot{V}O_2$ on-kinetics remains under investigation. When compared, 6 weeks of continuous training vs. HIT, matched in terms of total work done, resulted in similar reductions in phase II kinetics during moderate- and severe exercise and $\dot{V}O_{2SC}$ amplitude in sedentary subjects (Berger et al. 2006). In contrast, other work-matched studies observed that 2-week SIT (Bailey et al. 2009) or 8-week HIT (Daussin et al. 2008) speeded up phase II kinetics and/or attenuated $\dot{V}O_{2SC}$ compared with continuous endurance training in untrained subjects. Taken together, these findings suggest that the intensity of training is an important stimulus for changes in $\dot{V}O_2$ on-kinetics. However, it is not known whether higher exercise intensities are needed to elicit superior improvements in $\dot{V}O_2$ on-kinetics. Since the severe domain comprises exercise intensities ranged from slower (submaximal) to faster (supramaximal) $\dot{V}O_2$ responses to $\dot{V}O_{2max}$ (Hill et al. 2002; Caputo and Denadai 2008; Turnes et al. 2016), two different HIT regimens performed at the boundaries of the severe domain would be instrumental in understanding the influence of exercise intensity on $\dot{V}O_2$ on-kinetics adjustments. Furthermore, this experiment may elucidate whether faster $\dot{V}O_2$ response to $\dot{V}O_{2max}$ could be an optimal training stimulus in terms of $\dot{V}O_2$ on-kinetics improvements.

Thus, the purpose of the present study was to compare the effects of two different 4-week HIT programs performed at the lower (i.e., 105 % CP) or upper ($I_{\text{HIGH}}$) intensity of the severe domain, matched in terms of total work, on $\dot{V}O_2$ on-kinetics during moderate- and heavy-intensity exercise in recreationally trained cyclists. In addition to the gas exchange measurements, electromyography (EMG) signal of vastus lateralis muscle and blood lactate accumulation was analyzed during heavy-intensity transitions. We hypothesized that while both interventions would result in a reduction in the phase II kinetics and attenuation of the amplitude of $\dot{V}O_{2SC}$ (for heavy exercise), superior improvements are expected after HIT at $I_{\text{HIGH}}$ due to the repeated stimulation of faster $\dot{V}O_2$ on-kinetics during acute training sessions.

Methods

Subjects

Twenty-one subjects volunteered and gave written informed consent to participate in this study, which had been approved by the Institutional Ethics Committee for Research on Human Subjects and was performed according to the Declaration of Helsinki. Subjects were regarded as recreationally trained cyclists (De Pauw et al. 2013), and were instructed to continue normal daily activities and to refrain from beginning any other training until the completion of the study. Inclusion criteria were: (1) actively training for at least 3 days per week during the 3 months prior to the intervention, (2) no musculoskeletal or cardiorespiratory disease, and (3) older than 18 years. The subjects were engaged in recreational cycling, but not for competitive purposes. Following the completion of the pre-training period,
the subjects were matched in terms of their $\dot{V}O_{2\text{max}}$ and assigned to an HIT group with training performed either at the lower (105 % CP; $n = 11$; 10 males; 22 ± 2 years; 76 ± 6 kg; 175 ± 6 cm; $\dot{V}O_{2\text{max}}$: 47.0 ± 5.4 ml kg min$^{-1}$) or at the upper ($I_{\text{HIGH}}$; $n = 10$; 9 males, 23 ± 3 years; 78 ± 8 kg; 174 ± 7 cm; $\dot{V}O_{2\text{max}}$: 48.5 ± 5.4 ml kg min$^{-1}$) intensity of the severe exercise domain. The subject’s characteristics and training effects on aerobic parameters, such as $\dot{V}O_{2\text{max}}$, lactate threshold, CP, and $I_{\text{HIGH}}$ were published elsewhere (Turmes et al. 2016).

**Procedures**

All subjects visited the laboratory for 6 days of experimentation before and after the 4 weeks of training. On the first day, the subjects performed an incremental test to determine their lactate threshold (LT) and $\dot{V}O_{2\text{peak}}$. During their second, third, and fourth visits, subjects performed two constant time-to-exhaustion ($T_{\text{LIM}}$) tests per day to determine $I_{\text{HIGH}}$, CP, and the finite work capacity above CP ($W'$), to prescribe HIT programs. On any given test day, the subjects completed a $T_{\text{LIM}}$ test for $I_{\text{HIGH}}$ determination and a $T_{\text{LIM}}$ test for CP and $W'$ determination, separated by a 1-h passive rest. Each $T_{\text{LIM}}$ test was preceded by a 6-min moderate-intensity warm up performed at the power output requiring 100 % of the LT. In their fifth and sixth visits, subjects performed two step transitions from a 20 W baseline to heavy-intensity power output with one transition per day. A summary of the experimental design is presented in Fig. 1.

Following the training intervention, all subjects completed the same tests as described above. In addition to tests at the same absolute moderate and heavy intensities, all subjects completed step tests to the intensities corresponding to 100 % LT and Δ50 % calculated from the post-training incremental test.

All tests were performed at the same time of day (±2 h) and separated by ≥24 h, but within a period of 14 days. All participants were instructed not to perform strenuous exercise and to abstain from alcohol on the day before each session and to refrain from consuming caffeine for at least 2 h before each trial.

**Materials**

All exercise tests were conducted using an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands), and subjects were instructed to maintain their preferred cadence between 70 and 90 revolutions per minute (rpm). Throughout each testing protocol, cyclists were a facemask, and respiratory gas exchange was measured breath-by-breath using an automated open-circuit gas analysis system (Quark CPET, COSMED Srl, Rome, Italy). The volume transducer was calibrated before each test with a 3-L calibration syringe, and the analyzers were calibrated with gases of known concentration.

Ear lobe blood samples (25 μL) were collected into a capillary tube during incremental test and heavy-exercise bouts for blood lactate concentration (BLC) using an automated analyzer (YSI 1500 Sport, Yellow Springs, Ohio, USA). Surface EMG signals of the vastus lateralis muscle were recorded during the heavy-intensity transitions. EMG data were sampled at 2 kHz using a 14-bit analog-to-digital converter with a four-channel EMG system (Miotool 400 USB, Miotec Ltda., Porto Alegre, Brazil).

**Incremental test**

The initial power output for the incremental test was set at 0.5 W kg$^{-1}$ for 3 min and then increased by 0.5 W kg$^{-1}$ every 3 min until voluntary exhaustion. At rest and in the end of each stage, capillary blood samples were collected from the non-hyperaemic ear lobe to measure BLC. $\dot{V}O_{2\text{peak}}$ was determined as the highest average $\dot{V}O_2$ over a 15-s period. LT was defined as the first sudden and sustained increase in blood lactate above baseline levels. The power output halfway between the LT and the $\dot{V}O_{2\text{max}}$ (Δ50 %) was calculated.

**Step tests**

On separate days following completion of the initial incremental test, the subjects completed six moderate-exercise transitions at 100 % of LT. Two transitions, separated by 1 h of rest, were performed per day. For heavy intensity, two...
transitions were performed at Δ50 % intensity in two different days (Fig. 1). BLC was determined immediately before and after one of the heavy-exercise bouts to calculate the difference between the pre- and post-exercise BLC values (i.e., ΔBLC). Each step exercise test comprised 6 min of exercise and was preceded by 3 min of 20 W baseline.

Training period

Both HIT groups trained three times per week for 4 weeks. All training sessions were monitored by one of the investigators. To prescribe the HIT training intensities, CP and \( I_{\text{HIGH}} \) were determined first (Turnes et al. 2016). Briefly, CP and \( W' \) were estimated from three predictive trials in a random order, at intensities yielding \( T_{\text{LIM}} \) between 3 and 9 min, and then calculated according to the linear model
\[
P = [W'/T_{\text{LIM}}] + \text{CP}.
\]
\( I_{\text{HIGH}} \) was determined from 2 to 3 \( T_{\text{LIM}} \) tests and defined for each subject as the highest power output at which \( \dot{V}O_{2\text{max}} \) was attained (Caputo and Denadai 2008; Turnes et al. 2016). The lowest exercise duration in which \( \dot{V}O_{2\text{max}} \) was attained \( (t_{\text{LOW}}) \) was \( T_{\text{LIM}} \) performed at \( I_{\text{HIGH}} \). At each interval training session, cyclists in the 105 % CP group completed a single series of four intervals at 105 % CP \((218 \pm 39 \text{ W})\) for a duration of 5 min, alternating with 1 min of passive recovery. Cyclists in the \( I_{\text{HIGH}} \) group completed two series of four intervals at 100 % \( I_{\text{HIGH}} \) \((355 \pm 60 \text{ W})\), for a duration equal to 60 % \( T_{\text{LOW}} \) \((79 \pm 16 \text{ s})\), with a 1:2 recovery ratio (Turnes et al. 2016). The amount of training was increased by including a single extra exercise interval per series per week in both groups. The total work completed was calculated as a product of the amount of time completed at the assigned power output in each HIT session, including recovery bouts.

\( \dot{V}O_{2} \) on-kinetics

The pulmonary \( \dot{V}O_{2} \) data from each test were initially examined to exclude occasional errant breaths caused by coughing, swallowing, sighing, etc., which were considered not to be reflective of the underlying kinetics; i.e., only values greater than three SDs from the local mean were omitted. The breath-by-breath data were first linearly interpolated to provide the second-by-second values, and, for each individual, identical repetitions of each intensity were time aligned to the start of exercise and then ensemble averaged. The first 20 s of data after the onset of exercise (i.e., the phase I response) were deleted, and a nonlinear least squares algorithm was used to fit the data thereafter. A single-exponential model was used to analyze \( \dot{V}O_{2} \) responses to moderate exercise, and a biexponential model was used for heavy exercise as described in the following equations:

\[
\dot{V}O_{2}(t) = \dot{V}O_{2}b + Ap(1 - e^{-t/TDp/\tau p})(\text{moderate})
\]

\[
\dot{V}O_{2}(t) = \dot{V}O_{2}b + Ap(1 - e^{-t/TDp/\tau p})(\text{heavy}) + As(1 - e^{-t/TDs/\tau s})(\text{heavy})
\]

where \( \dot{V}O_{2}(t) \) represents the absolute \( \dot{V}O_{2} \) at a given time \( t \), \( \dot{V}O_{2}b \) represents the mean \( \dot{V}O_{2} \) in the final 60 s of the baseline period; \( Ap, TDp, \) and \( \tau p \) represent the amplitude, time delay, and time constant, respectively, describing the phase II increase in \( \dot{V}O_{2} \) above baseline; and \( As, TDs, \) and \( \tau s \) represent the amplitude of, time delay before the onset of, and time constant describing the development of the \( \dot{V}O_{2} \) slow component, respectively. The amplitude of the \( \dot{V}O_{2} \) slow component was also described as the difference between \( \dot{V}O_{2} \) of the sixth and third minutes of exercise \( (\Delta\dot{V}O_{2}) \).

To provide information on the “overall” \( \dot{V}O_{2} \) on-kinetics, with no distinction made for the various phases of the response during heavy-intensity exercise, the \( \dot{V}O_{2} \) mean response time (MRT) was also fitted by a single-exponential curve without time delay to the data from the onset to the end of exercise.

EMG measurements

During the heavy-intensity transitions, quadriceps electromyograms were recorded from the right vastus lateralis muscle using bipolar 10 mm diameter Ag–AgCl surface electrodes (Tyco Healthcare Group LP, Massachusetts, USA). The placement and location of the electrodes followed the standard recommendations of SENIAM (Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles). The reference electrode was placed on skin surface above the ulnar head. The positioning of the electrodes was marked with indelible ink and recorded along with other marks (angiomas and/or scars) on the subject’s skin to ensure that they were placed in the same location at the subsequent tests. The raw digital EMG signal was processed using MATLAB 7.12 (MathWorks Inc., Natick, USA). The signal was initially filtered using a 10–500 Hz bandpass, fifth-order Butterworth filter. The root mean square (RMS) of Δ50 % exercise transition was calculated over 20-s intervals. The values were normalized to the average RMS measured during the 120–180 s interval of baseline cycling phase before the heavy-intensity transition.

Statistical analysis

Descriptive data are presented as arithmetic means and standard deviations (±SD). Comparisons of \( \dot{V}O_{2} \) on-kinetics parameters, EMG measurements, and BLC were performed with the mixed linear modeling procedure of the IBM SPSS statistics (Version 19.0, IBM Corporation, New York, USA). The subject term was used as a random effect. Group (105 % CP and \( I_{\text{HIGH}} \)) and time (pre- and
post-training) were fixed effects. Analysis was performed in the log-transformed, and effects on data were expressed as percent change of back-transformed values and 95 % confidence limits (±95 % CL). Mean of pre-training values was always utilized as a covariate. Where a significant interaction effect was obtained, a post hoc analysis was performed to identify the source of differences using Bonferroni correction. The Pearson product moment correlation coefficient (r) was used to examine the relationship between variables. All tests were analyzed at an alpha level of 0.05.

**Results**

The mixed modeling analysis of all measures of interest at baseline revealed no significant differences between the two groups. In addition, there were no significant group × time interactions in all the VO₂ kinetics parameters (P > 0.05). All the subjects adhered completely to the protocols of training sessions. The total work performed during the training period in I_HIGH (4420 ± 653 kJ) and 105 % CP (4426 ± 744 kJ) groups was similar (P = 0.98).

**Moderate step exercise**

The parameters of the V̇O₂ on-kinetic in the transition to the same absolute and relative moderate intensity in the two groups before and after the intervention period are shown in Table 1. V̇O₂ responses to moderate exercise of representative subjects are illustrated in Fig. 2. There was a significant reduction in τp post-training during the absolute and relative moderate exercise in the two interventions (P < 0.01), although there was no significant interaction between the groups for τp for absolute (P = 0.86) and relative intensities (P = 0.55). In the two groups, Ap was significantly increased post-training for relative (P < 0.01), but not for absolute intensity (P = 0.86).

**Heavy step exercise**

The parameters of the V̇O₂ on-kinetic in the transition to the same absolute and relative heavy intensity in the two groups before and after the intervention period are shown in Table 2. V̇O₂ responses to heavy exercise of representative subjects are illustrated in Fig. 3. There were a significant reduction in τp and MRT post-training during the absolute and relative heavy exercise in the two interventions (P < 0.01), although there was no significant interaction between the groups for absolute (P = 0.22 and 0.07, respectively) and relative intensities (P = 0.37 and 0.97,
In both groups, As and $\Delta \dot{V}O_2$ were significantly reduced post-training for absolute heavy intensity, with no interaction between the groups ($P = 0.91$ and $0.89$, respectively). There was no significant change in the parameters associated with $\dot{V}O_{2SC}$ in the two groups ($P > 0.05$).

The mean EMG responses to heavy intensity for each group are illustrated in Fig. 4. The RMS amplitude of the vastus lateralis muscle was significantly reduced within the two groups from pre- to post-training (change

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**Table 2** Oxygen uptake on-kinetics during heavy exercise before and after 105 % CP and $I_{\text{HIGH}}$ training

<table>
<thead>
<tr>
<th></th>
<th>105 % CP</th>
<th>$I_{\text{HIGH}}$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post-absolute</td>
</tr>
<tr>
<td>Work rate (W)</td>
<td>171 ± 41</td>
<td>198 ± 37*</td>
</tr>
<tr>
<td>Baseline $\dot{V}O_2$ (mL min$^{-1}$)</td>
<td>974 ± 136</td>
<td>969 ± 110</td>
</tr>
<tr>
<td>Primary amplitude (mL min$^{-1}$)</td>
<td>1584 ± 300</td>
<td>1649 ± 302</td>
</tr>
<tr>
<td>Primary time delay (s)</td>
<td>9 ± 6</td>
<td>10 ± 3</td>
</tr>
<tr>
<td>Primary time constant (s)</td>
<td>25 ± 7</td>
<td>18 ± 5*</td>
</tr>
<tr>
<td>$\dot{V}O_{2SC}$ amplitude(mL min$^{-1}$)</td>
<td>84 ± 81</td>
<td>41 ± 43*</td>
</tr>
<tr>
<td>$\dot{V}O_{2SC}$ time delay (s)</td>
<td>242 ± 84</td>
<td>295 ± 60*</td>
</tr>
<tr>
<td>$\dot{V}O_{2SC}$ time constant (s)</td>
<td>139 ± 42</td>
<td>119 ± 58</td>
</tr>
<tr>
<td>Mean response time (s)</td>
<td>52 ± 11</td>
<td>40 ± 7*</td>
</tr>
<tr>
<td>$\Delta \dot{V}O_2$ 6th to 3rd min (mL min$^{-1}$)</td>
<td>163 ± 85</td>
<td>98 ± 68*</td>
</tr>
<tr>
<td>$\Delta$Blood lactate (mmol L$^{-1}$)</td>
<td>3.3 ± 0.7</td>
<td>2.6 ± 1.2*</td>
</tr>
</tbody>
</table>

Data are in Mean ± SD

* Significant difference from pre-training ($P < 0.05$)
in measure $\pm 95\%$ CL, $105\%$ CP: $-12 \pm 3\%$; $I_{\text{HIGH}}$: $-9 \pm 3\%$; $P < 0.01$), although there was no significant interaction between the groups ($P = 0.13$).

There were no difference in the pre-exercise blood lactate concentration between the groups ($I_{\text{HIGH}}$: $1.4 \pm 0.3$ mMol L$^{-1}$; $105\%$ CP: $1.4 \pm 0.3$ mMol L$^{-1}$; $P = 0.95$). In the two groups, the $\Delta BLC$ was significantly reduced post-training for absolute ($P < 0.01$), but not for relative intensity ($P = 0.38$).

Discussion

The main finding of the present study was that short-term HIT performed in the boundaries of the severe exercise domain was similarly effective in speeding the $\dot{V}O_2$ responses at the onset of both moderate and heavy exercise. In addition to the faster phase II $\dot{V}O_2$ kinetics, the reductions in $\dot{V}O_{2\text{SC}}$ were accompanied by lower quadriceps myoelectric signal and $\Delta BLC$ during heavy-intensity exercise in the two groups. The results supported the first hypothesis that both training interventions would enhance $\dot{V}O_2$ on-kinetics. However, the results did not support the second hypothesis that the stimulus from the faster $\dot{V}O_2$ response to $\dot{V}O_{2\text{max}}$ during acute training sessions would be more effective in speeding $\dot{V}O_2$ on-kinetics.

Regardless of the training intensity, the phase II $\dot{V}O_2$ on-kinetics was similarly speeded after both interventions in the present study, corroborating with previous studies (Berger et al. 2006; McKay et al. 2009). McKay et al. (2009) verified that eight sessions of HIT and continuous endurance training accelerated the phase II kinetics, attributing such changes to enhanced mitochondrial enzymatic activity and faster local microvascular blood flow at the onset of moderate exercise in both groups of active subjects. In contrast, Bailey et al. (2009) observed that only SIT speeded $\dot{V}O_2$ on-kinetics in active subjects, assigning this to a faster and greater $O_2$ extraction. Similarly, Daussin et al. (2008) reported that 8 weeks of HIT enhanced $\dot{V}O_2$ on-kinetics and skeletal muscle oxidative capacity in sedentary subjects, with no modifications in such parameters after continuous endurance training. It has been well-documented that HIT increases the activity of key metabolic enzymes within the active fibres (MacDougall et al. 1998; Burgomaster et al. 2008; Lundby and Jacobs 2016) and that the upregulation of some mitochondrial enzyme activity seems to be dependent on exposure to higher exercise intensities during training (Jacobs and Lundby 2013; Lundby and Jacobs 2016; Granata et al. 2016). While the possible mechanisms involved in the speeding of $\dot{V}O_2$ on-kinetics throughout $105\%$ CP or $I_{\text{HIGH}}$ exercise training program are complex and likely an integration of both enhanced metabolic control and $O_2$ delivery, it is possible to suppose that the differences in the exercise intensity, $\dot{V}O_2$ response, or power output fluctuations during the training sessions employed herein were not sufficient to induce superior $\dot{V}O_2$ on-kinetics improvements in recreationally trained cyclists.

Although the lower and upper intensities of the severe domain induced different $\dot{V}O_{2\text{SC}}$ responses during training sessions, similar reductions in $\dot{V}O_{2\text{SC}}$ were observed after both interventions. This finding agrees with previous studies comparing the influence of different training intensities on $\dot{V}O_{2\text{SC}}$ (Berger et al. 2006; Bailey et al. 2009). The active skeletal muscle is the predominant site of the development of the $\dot{V}O_{2\text{SC}}$ (Poole et al. 1991; Jones et al. 2011). Accordingly, $\dot{V}O_{2\text{SC}}$ has been associated with additional motor unit recruitment as exercise proceeds (Grassi et al. 2015; Jones et al. 2011), and some evidence indicate that a greater recruitment of type II fibres leads to a higher $\dot{V}O_{2\text{SC}}$ (Perrey et al. 2001; Borrani et al. 2001; Osborne and Schneider 2006; Jones et al. 2011). In addition, while HIT improves skeletal muscle respiratory capacity (Daussin et al. 2008; Jacobs and Lundby 2013; Lundby and Jacobs 2016), increases in mitochondrial volume density are somewhat more pronounced in type IIa when compared to type I or type IIx fibres (Howald et al. 1985). Since a large pool of muscle fibres might already be recruited during long intervals (considering a minimal recruitment threshold at >75–85% of $\dot{V}O_{2\text{max}}$ for type II fibres)(Altenburg et al. 2007; Buchheit and Laursen 2013; Gollnick et al. 1974), likely improving their oxidative capacity, it was not surprising that both interventions promoted similar reductions in the $\dot{V}O_{2\text{SC}}$ and EMG signal.

In addition to the lower EMG signal after both interventions, reductions in $\Delta BLC$ accompanied the lower $\dot{V}O_{2\text{SC}}$ after training. Although BLC accumulation and muscle acidity have been suggested to be of little importance for $\dot{V}O_{2\text{SC}}$ development (Gaesser et al. 1994; Poole et al. 1994; Jones et al. 2011), they have a strong association with muscle fatigue and endurance performance (Fitts 1994; Beneke et al. 2011). In addition, as $\dot{V}O_{2\text{SC}}$ reflects increased skeletal muscle inefficiency, exercise tolerance can be improved by interventions that reduce or eliminate $\dot{V}O_{2\text{SC}}$ (Jones et al. 2011). When combined with the reduced EMG signal and the speeded $\dot{V}O_2$ on-kinetics, the present findings indicate that both training interventions used in the present study may lead to an improved level of “metabolic stability” during exercise (e.g., decreases in the Gibbs free energy of ATP hydrolysis and muscle phosphocreatine, increases in BLC, $H^+$, ADP, $P_i$, IMP, NH3, etc.), owing to an enhanced exercise tolerance.

In healthy participants, a faster $\dot{V}O_2$ on-kinetics occurred in a few days of endurance training (Phillips et al. 1995; McKay et al. 2009; Murius et al. 2016), with improvements continuing up to 3–4 weeks after the start of the training program (Phillips et al. 1995; Murius et al. 2010, 2011).
Based on that, a 4 week period was applied in the present study, with robust $\dot{V}O_2$ on-kinetics enhancements occurring after this short-term period. Although no further changes have been observed after 6, 8, or 12 weeks of endurance training (Fukuoka et al. 2002; Murias et al. 2010, 2011), additional benefits caused by HIT on $\dot{V}O_2$ on-kinetics during longer interventions may not be ruled out. Further studies are warranted to clarify the long-term time course of adaptation of phase II kinetics in well-trained athletes exposed to different HIT regimens.

In addition to training period, total work completed may also determine the longitudinal responses caused by training. There is evidence that increases in training volume, measured by weekly kcal/kg expenditure, contributed to a greater magnitude of training response in healthy women (Sisson et al. 2009). Accordingly, controlling total work is widely employed in studies comparing the influence of training interventions on $\dot{V}O_2$ on-kinetics (Berger et al. 2006; Daussin et al. 2008; Bailey et al. 2009). Despite work matching groups may not be consistent with how athletes typically approach their training (Seiler et al. 2013), the present study preferred to match HIT groups in terms of total work completed to isolate intensity as a training variable. Thus, although the differences between training session length and exercise intensity between 105 % CP and $I_{HIGH}$, the two work-matched groups resulted in similar adaptations on $\dot{V}O_2$ on-kinetics in recreationally trained cyclists.

Some limitations of the present study should be taken into consideration. The accurate estimate of the boundaries of the severe intensity domain is laborious and time consuming, which in part contributed for the absence of an endurance training group that could strength the present experimental design. However, $I_{HIGH}$ and 105 % CP, two intensities with large difference in absolute power output, indicated that higher intensities not necessarily resulting in more gain in terms of $\dot{V}O_2$ on-kinetics improvement. Indeed, with moderate changes in cardiorespiratory fitness, old and middle-aged males may be able to reach values for $\tau_p$ similar to those of active young men (Babcock et al. 1994; Fukuoka et al. 2002). On the other hand, it is possible that trained cyclists could not be responsive to the short-term training in terms of $\dot{V}O_2$ on-kinetics improvement. In support to this notion, previous studies have observed a positive relationship between the baseline $\tau_p$ and the magnitude of $\tau_p$ reduction with training (Berger et al. 2006; Bailey et al. 2009; McKay et al. 2009). In a cross-sectional experimental design, $\dot{V}O_2$ on-kinetics was speeded when the fitness level was increased from low to intermediate but not further improved as the aerobic fitness level increases from intermediate to a high level of elite athletes (Figueira et al. 2008). Since in highly trained athletes further enhancements in performance-related markers, such as $V_{O2max}$ and oxidative muscle enzymes, may not appear with an additional increase in submaximal exercise training (i.e. volume), but only with the insertion of HIT (Costill et al. 1988; Lake and Cavanagh 1996; Londere 1997; Laursen and Jenkins 2002), a group of highly trained endurance cyclists would be an interesting population to detect the potential influence of training intensity on $\dot{V}O_2$ on-kinetics. To date, the optimal training intervention to maximize such enhancements remains to be determined.

In conclusion, 4 weeks of two different HIT programs performed at the upper and lower boundaries of the severe exercise domain were similarly effective to enhance the pulmonary $\dot{V}O_2$ on-kinetics during moderate- and heavy-intensity cycling exercise. In addition, both interventions attenuate the amplitude of $\dot{V}O_2SC$, which were accompanied by reduced EMG signal and BLC accumulation. These data suggest that irrespective of the training intensity, $\dot{V}O_2$ response and, power output fluctuations during training sessions, HIT performed at the lower or at the upper intensities of the severe exercise domain are equally efficient to provoke adjustments linked to speeded $\dot{V}O_2$ on-kinetics in recreationally trained cyclists.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest. The results of the current study do not constitute endorsement of the product by the authors or the journal.

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