High-intensity Interval Training in the Boundaries of the Severe Domain: Effects on Sprint and Endurance Performance

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Abstract

In order to compare the effects of two 4-week interval training programs performed at the lower (Critical Power, CP) or at the higher (The highest intensity at which VO2max is attained, IHIGH) intensities of the severe exercise domain on sprint and endurance cycling performance, 21 recreationally trained cyclists performed the Wingate Anaerobic Test (WAnT) and a 250-kJ time trial. Accumulated oxygen deficit (AOD), surface electromyography (RMS), and blood lactate kinetics were measured during the WAnT. Subjects were assigned to 105 % CP or IHIGH groups. During the WAnT, significantly greater improvements in peak (Mean ± 95 %CI) (5.7 ± 2.3 % vs. 0.2 ± 2.2 %), mean power output (MPO) (3.7 ± 2.0 % vs. 0.5 ± 1.8 %), and RMS (17.8 ± 7.4 % vs. −15.7 ± 7.9 %) were observed in the IHIGH group (P < 0.05). Higher and lower AOD, respectively, at the start and during the second half of the WAnT were observed after IHIGH training. The changes in RMS and MPO induced by the training were significantly correlated (r = 0.584). The 2 interventions induced improvements in the 250-kJ time trial. In conclusion, although the improvements in endurance performance were similar, training at IHIGH led to higher gains in WAnT performance than training at 105 % CP.

Introduction

Competitive cycling is a sport that requires high levels of not only aerobic metabolism, but also of anaerobic metabolism, neuromuscular function and disposal of selected metabolic by-products [13]. The importance of this integrative physiology is evident in many aspects including breakaway attempts, hill climbing, and final sprints during competitive bicycle races [13]. In this regard, high-intensity interval training (HIT) has been identified as one of the most effective forms of exercise for improving performance in endurance athletes [7, 21], and longer times spent at or near maximal oxygen consumption (VO2max) have been suggested as optimal stimuli promoting greater aerobic adaptations [7, 21, 38]. Since the severe intensity domain is the only domain where VO2max can be reached during constant exercise, this important training zone is suitable for HIT implementation [10, 19].

In support of this notion, it has been shown that VO2max was not achieved during constant exercise performed until exhaustion at critical power (CP), but only at intensities slightly above it (i.e., ≥ 105 % of CP) [10, 12, 19, 32]. Therefore, CP demarcates the boundary between the heavy and severe exercise domains, and it has been considered an important performance-related marker strongly correlated with endurance performance [10, 31]. The upper boundary of the severe intensity domain is represented by the highest intensity at which VO2max is still attained during constant exercise (IHIGH). Hence, above IHIGH the exercise duration would be too short for VO2max to be reached [12, 19]. Since exercise duration during HIT has been suggested as the most important parameter in terms of improving aerobic metabolism [7, 21, 27], a comparison of the effects of HIT performed at the 2 boundaries of the severe intensity domain may be instrumental in understanding the influence of exercise intensity on endurance performance. In addition to adaptations related to aerobic metabolism, neural adaptations in response to HIT have been shown to affect sprint cycling performance [6, 34]. In support of this notion, Creer et al. [12] observed an improvement in the performance of 4 successive Wingate Anaerobic tests after 4 weeks of sprint interval training,
which was accompanied by higher surface electromyography (EMG) activity of the vastus lateralis muscle. Thus, the neuromuscular response combined with high cardiopulmonary and metabolic demand highlight the important role of HIT in stimulating a complex integrative physiology [6, 7].

The above consideration also emphasises the importance of HIT performed at higher intensities (e.g., supramaximal exercises) for promoting adjustments not only related to endurance performance but also to sprint performance [6]. This has been the case in a number of studies showing that improvements in sprint performance caused by various regimens of HIT were accompanied by enhancements in parameters pertaining to aerobic metabolism [8, 9, 22, 23, 33, 41]. However, while many studies demonstrated that HIT performed at higher intensities induced greater improvements in sprint performance than training at lower intensities [8, 9, 33, 36], other studies suggested that the 2 training modes resulted in similar improvements in endurance performance [9, 36]. Since shorter intervals and higher intensities seem to lead to a greater neuromuscular load [6, 33], an experiment conducted at the boundaries of the severe intensity domain during HIT may enable a comparison of the effects of different intensities within the same intensity domain on short- and long-duration cycling performance. Therefore, the purpose of the present study was to compare the effects of short-term HIT performed at the lower (i.e., 105 %CP) or upper (i.e., IHIGH) intensities of the severe domain, matched in terms of total work, on sprint and endurance performance. To identify the underlying mechanisms, we used surface EMG, and measured blood lactate concentration kinetics and cardiorespiratory responses during the Wingate Anaerobic Test (WAnT). In addition, metabolic and cardiorespiratory responses were also measured during a 250-kJ time trial. On the one hand, since higher exercise intensities seem to result in greater increases in neuromuscular, metabolic, and cardiovascular function [6, 7, 27], we expected larger improvements in WAnT results after training at IHIGH training than after training at 105 %CP. On the other hand, since gains in aerobic parameters have been observed for different regimens of HIT, the 2 training modes were expected to result in similar improvements during the 250-kJ time trial.

**Methods**

**Subjects**

21 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 and the ethical standards of the IJSM [18]. Subjects were regarded as recreationally trained cyclists according to their VO₂max [14], and were instructed to continue normal daily activities until the completion of the study. Inclusion criteria were: I) actively training for at least 3 days per week during the 3 months prior to the intervention, II) no musculoskeletal or cardiorespiratory disease and III) older than 18 years. The subjects were engaged in recreational cycling, but not for competitive purposes. Following the pre-training period, the subjects were assigned to a HIT group with training performed either at the lower (105 %CP; n = 11; 10 males) or at the upper (IHIGH; n = 10; 9 males) intensity of the severe-intensity exercise domain. Procedures

All participants performed a WAnT and a 250-kJ time trial before and after 4 weeks of training. Subjects first performed an incremental test. During their second, third, and fourth visits, subjects performed 2 constant time-to-exhaustion (Tlim) tests per day to determine IHIGH, TLOW, and CP and the finite work capacity above CP (Ẇ). IHIGH and CP were designed for training prescription [38]. The fifth and sixth visits consisted of a WAnT and a 250-kJ laboratory time trial, respectively. All tests were performed at a similar time of day (±2 h) and were separated by 48 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 caffeine for at least 2 h before each trial.

**Materials**

All exercise testing was conducted using an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). The pulmonary gas exchange was measured breath-by-breath using an automated open-circuit gas analysis system (Quark PFT, COSMED, Rome, Italy). Before each test, the turbine flow meter was calibrated with a 3 L calibration syringe (COSMED, Rome, Italy), and the analysers were calibrated with gases of known concentration. Heart rate (HR) was also monitored throughout the tests (Polar, Kempele, Finland). Body mass was measured to the nearest 0.05 kg on a digital scale (Toledo 2096PP, Brazil). Ear lobe capillary blood samples (25 μL) were taken for the determination of blood lactate concentration ([La]). Arterialised capillary blood was then stored in Eppendorf tubes containing 50 μL of 1 % NaF in a −30 °C environment. Later, samples were analysed using enzyme electrode technology (YSI 1500 Sport, Yellow Springs, Ohio, USA).

**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 [12]. VO₂max and lactate threshold (LT) were determined according to a previously published protocol [12, 39]. The intensity that would require 50 % of the difference between the LT and VO₂max (Δ50%) was calculated.

**Wingate anaerobic test**

After a standardized warming up of 3 min unloaded cycling (20 W) followed by 6 min cycling at Δ50% intensity and a 5-min passive rest, subjects performed the WAnT [4] with a non-inertial start. VO₂ was measured throughout the 30 s of the test. The mechanical power was analyzed for each 1-s. Peak power (PPo), mean power (MPO) and minimum power were defined as the highest mechanical power output, the average power sustained throughout the 30-s period, and the power output attained before the end of the test, respectively. The fatigue index was also calculated [4].

**Electromyography**

During the WAnT, quadriceps surface electromyograms (EMG) were recorded from the right vastus lateralis using bipolar 10 mm diameter Ag-AgCl surface electrodes (Tyco Healthcare Group LP, Massachusetts, USA). The raw data of the EMG were collected during the warm up and the WAnT. In the warm up, during the 3 min of unloaded cycling phase, EMG data were
recorded in order to permit EMG normalisation. The unloaded cycling phase corresponded to 20 W and subjects were instructed to reproduce the same cadence. The placement and location of the electrodes are according to 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. EMG was recorded with a common mode rejection ratio of 110 dB and an input impedance of 10 GΩ with a 4-channel EMG system (Miotool 400 USB, Miotec Ltda., Porto Alegre, Brazil). The EMG signals were amplified (×400), A/D converted (14 bits resolution) at a sampling rate of 2 kHz, and stored for later analysis. The root mean square (RMS) of the WAnT was calculated over 5-s intervals and for the 30-s exercise. The values were normalized to the average RMS measured during the 0–60 s interval of unloaded cycling during the warm-up.

Accumulated oxygen deficit
During the incremental test, the VO₂ values for the last 30 s of each 3-min step between 30% and 90% of VO₂max were recorded [28]. Then, the O₂ demand was estimated individually by extrapolating the linear relationship between VO₂ and power output. Finally, the accumulated oxygen deficit (AOD) was computed as the difference between the O₂ demand and the O₂ consumed during the WAnT [11]. The data were calculated over 5-s intervals and accumulated over time.

Blood lactate kinetics
During the WAnT, capillary blood samples were collected at rest, immediately before the WAnT and then for 45 min of passive recovery (every min from 0 to 10 min, 2 min from 10 to 20 min, and 5 min from 25 to 45 min). The [La] kinetics was analyzed by using the 3-parameter biexponential model proposed earlier [5]. A 3-parameter model was chosen over the traditional 4-parameter model because it provided more realistic parameter estimations with respect to the elimination of lactate from the blood compartment at relatively short periods of blood sampling [3]. The non-linear regression encompassed only post-exercise [La], as in the traditional 4-parameter model [15]. Therefore, this bicompartmental model requires samples of [La] at rest, immediately after exercise ([La]₀) and for at least 45-min post-exercise. This model approximates the vascular increase in lactate concentration after the end of exercise (AL). It furthermore estimates 2 velocity constants describing the corresponding kinetics of the appearance (K₁) and the disappearance (K₂) of lactate into and out of the blood compartment. The quantity of lactate accumulated in the whole-body during the WAnT (QLA) was calculated by summing the net increase in [La] during exercise ([La]₀ – pretest [La]) to the exponential term AL, and then multiplying this value for the total body water [40]. The body mass of pre- and post-training were utilized to estimate QLa in the respective training period.

\[
[La]_{tot} = ([La]₀ - [La]_{rest})e^{-K₂t} + \frac{[ALK]_1}{K₂ - K₁}[e^{K₁t} - e^{-K₂t}] + [La]_{rest}
\]

250-kJ time trial performance
After the standardized warming up, subjects were instructed to complete a 250-kJ self-paced laboratory time trial on the ergometer as quickly as possible [8]. VO₂ and HR were measured throughout the test. Capillary blood samples were collected at the end of the test and then at the third and fifth minute of recovery.

Exercise training protocols
Both HIT groups trained 3 times per week for 4 weeks. In order to devise HIT, CP and IHIGH were determined first [38]. CP and W’ were estimated from 3 predictive trials in a random order, at intensities yielding TIHM between 3 and 9 min, and then calculated according to the linear model \((P = W/TIHM + CP)\). This model exhibited the best fit \((R²\) values range: 0.984–1.000) and the lowest CP standard error of estimate (105 %CP: 3.5 ± 3.0%; IHIGH: 3.4 ± 3.4%). IHIGH was determined from 2–3 TIHM tests and was defined for each subject as the highest power output at which the VO₂max was attained [12, 38]. The lowest exercise duration at which VO₂max was attained \((TIHM)\) was the TIHM performed at IHIGH.

A summary of training sessions is presented in Table 1 and Fig. 1. The IHIGH training session was performed with a duration equal to 60 %TIHM and a work-to-rest ratio of 1:2 with an active recovery at 80 % lactate threshold [38]. The training was progressed by including a single extra exercise interval per series per week in both groups. The total work completed during the training period was calculated as a product of the amount of time completed at the assigned power output in each HIT session, including recovery bouts. During the first training session and the WAnT, the torque generated at the crank axle was measured by strain gauges developed and bonded on to the crank arm by the Lode cycle ergometer manufacturer. The peak torque (mean of the highest propulsive torque during the downstroke phase) was averaged from both legs. The torque values of the first training session were expressed as percentages of the peak torque of the WAnT. VO₂ and HR were measured in the first training session, and the peak of each interval was recorded to determine the training average. Blood lactate was collected in the end of training session.

### Table 1 Subject’s characteristics, training protocols, and acute responses during the first session.

<table>
<thead>
<tr>
<th>Subject’s characteristics</th>
<th>105 %CP</th>
<th>IHIGH</th>
</tr>
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<tbody>
<tr>
<td>Age (years)</td>
<td>22 ± 2</td>
<td>23 ± 3</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>76 ± 6</td>
<td>78 ± 8</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175 ± 6</td>
<td>174 ± 7</td>
</tr>
<tr>
<td>VO₂max (ml·kg·min⁻¹)</td>
<td>47.0 ± 5.4</td>
<td>48.5 ± 5.4</td>
</tr>
<tr>
<td>Training protocol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bouts/Session</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Work Intensity (W)</td>
<td>218 ± 39</td>
<td>355 ± 60 *</td>
</tr>
<tr>
<td>Work Duration (s)</td>
<td>300</td>
<td>79 ± 16</td>
</tr>
<tr>
<td>Rest intensity (W)</td>
<td>0</td>
<td>56 ± 20</td>
</tr>
<tr>
<td>Rest duration</td>
<td>60</td>
<td>158 ± 32</td>
</tr>
<tr>
<td>Acute responses</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VO₂peak (%max)</td>
<td>93 ± 7</td>
<td>96 ± 4</td>
</tr>
<tr>
<td>HRpeak (%max)</td>
<td>91 ± 4</td>
<td>98 ± 4 *</td>
</tr>
<tr>
<td>[La]₀ (mmol·l⁻¹)</td>
<td>10.9 ± 1.8</td>
<td>15.2 ± 1.9 *</td>
</tr>
<tr>
<td>Peak torque (%max)</td>
<td>50 ± 9</td>
<td>62 ± 7 *</td>
</tr>
<tr>
<td>Cadence (rpm)</td>
<td>81 ± 6</td>
<td>82 ± 7 *</td>
</tr>
</tbody>
</table>

Data are in Mean ± SD. CP: Critical power; IHIGH and TIHM: The highest intensity and the lowest exercise duration at which VO₂max is attained, respectively; HR: Heart rate; [La]: Blood lactate concentration. * Significant difference from 105 %CP (P < 0.05)
Table 2  Changes in performance measures in both training groups.

<table>
<thead>
<tr>
<th></th>
<th>Pre 105 %CP</th>
<th>Post 105 %CP</th>
<th>Δ (%) and 95 %CI&lt;sup&gt;†&lt;/sup&gt;</th>
<th>Pre IHIGH</th>
<th>Post IHIGH</th>
<th>Δ (%) and 95 %CI&lt;sup&gt;†&lt;/sup&gt;</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Wingate Anaerobic Test</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>PPO (W)</td>
<td>910 ± 125</td>
<td>909 ± 138</td>
<td>0.2 ± 2.2</td>
<td>906 ± 101</td>
<td>958 ± 110</td>
<td>5.7 ± 2.3</td>
<td>0.002</td>
</tr>
<tr>
<td>MPO (W)</td>
<td>728 ± 79</td>
<td>725 ± 85</td>
<td>0.5 ± 1.8</td>
<td>716 ± 73</td>
<td>742 ± 74</td>
<td>3.7 ± 2.0</td>
<td>0.025</td>
</tr>
<tr>
<td>Minimum PO (W)</td>
<td>488 ± 60</td>
<td>518 ± 48*</td>
<td>8.6 ± 3.9</td>
<td>520 ± 82</td>
<td>513 ± 84</td>
<td>−1.3 ± 4.3</td>
<td>0.002</td>
</tr>
<tr>
<td>Fatigue index (%)</td>
<td>45.6 ± 8.3</td>
<td>42.3 ± 8.3*</td>
<td>−4.5 ± 2.7</td>
<td>41.4 ± 8.8</td>
<td>45.3 ± 9.0*</td>
<td>3.9 ± 2.9</td>
<td>0.000</td>
</tr>
<tr>
<td><strong>250-kJ time trial</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time (s)</td>
<td>1 148 ± 217</td>
<td>1 040 ± 188*</td>
<td>−9.2 ± 3.1</td>
<td>1 137 ± 199</td>
<td>1 014 ± 208*</td>
<td>−8.7 ± 3.4</td>
<td>0.775</td>
</tr>
</tbody>
</table>

Data are in Mean ± SD. PPO, peak power output; MPO, mean power output; Minimum PO, minimum power output. Δ, relative change from pre-training values. † Changes and 95 %CI are expressed relative from log-transformed values. * Significant difference from pre-training period. 《 Significant difference from pre-training ($P < 0.05$).

**Statistical analysis**

Descriptive data are presented as arithmetic means and standard deviations (±SD). Comparisons of performance measures, EMG signal, [La] kinetics and AOD were performed with the mixed linear modeling procedure of the IBM SPSS statistics program (Version 19.0, IBM Corporation, New York, USA). The subject term was used as a random effect. Group (105 %CP and IHIGH), moment (pre- and post-training) and time (5-s intervals during WAnT, for EMG and AOD measures) were fixed effects. Analysis was performed in the log-transformed data and effects on performance measures and related physiological parameters were expressed as percent change of back-transformed values and 95 % confidence limits. 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 a Bonferroni correction. A comparison of variables during the first training session and total work performed between groups was made using unpaired t-test for unequal variances. Pearson’s product moment was used to examine relationships between variables. All tests were analyzed at an alpha level of 0.05.

**Results**

**Training sessions**

The acute responses during the first training session are presented in **Table 1**. The average HRPeak, final [La], and peak torque generated were significantly higher during the IHIGH session than during the 105 %CP session ($P < 0.01$). There were no differences between the groups with respect to total work completed during the total training period (105 %CP: 4 420 ± 653 kJ; IHIGH: 4 426 ± 744 kJ; $P = 0.984$). All the subjects adhered completely to the protocols of training sessions.

**Performance measures**

The training group at IHIGH was able to increase PPO and MPO during the WAnT, whereas the group training at 105 %CP improved the minimum power output. As a result, the fatigue index decreased in the 105 %CP group and increased in the IHIGH group. When the 2 groups were combined, the peak torque generated during the first training session was significantly correlated ($P < 0.05$) with the changes in PPO ($r = 0.559$) and MPO ($r = 0.502$) induced by training. The improvements in 250-kJ time trial performance (**Table 2**) and average power output (105 %CP: 226 ± 47 to 248 ± 47; IHIGH: 227 ± 45 to 252 ± 40 W; $P < 0.01$) after training were similar in the 2 groups (**Table 2**). Neither training mode affected the O2, HR and [La] responses during 250-kJ time trial.

**Electromyography**

The temporal profiles of the EMG signals throughout the WAnT for each training condition are illustrated in **Fig. 2a**. A significant interaction (group * moment) term was observed ($P < 0.01$). There was a significant decrease (15.7 %) in the overall RMS after training in the 105 %CP group (confidence limits, ± 7.9 %; $P < 0.01$), whereas a 17.8 % increase was found in the IHIGH group. When the 2 groups were merged, the relative changes in MPO caused by training were positively correlated with changes in overall RMS of the WAnT ($r = 0.584, P = 0.018$).
Accumulated oxygen deficit
The temporal profiles of the AOD throughout the WAnT for each training condition are illustrated in Fig. 3. There was a significant effect of time \((P<0.01)\) and interaction (group \(*\) moment \(*\) time) \((P<0.01)\). The AOD increased significantly during the 25–30 s interval after 105%CP training \((P=0.021)\). After \(I_{\text{HIGH}}\) training, the AOD increased during the 0–5 s interval \((P=0.020)\) and decreased during the 15–20, 20–25, and 25–30 s intervals \((P<0.05)\). There were no training effects on the overall AOD in either groups \((P>0.05)\). A significant effect of 105%CP training was observed in accumulated O\(_2\) uptake during the WAnT \((793\pm161\text{ vs. }854\pm182\text{ ml, }P=0.043)\), with no such effects in the \(I_{\text{HIGH}}\) group \((858\pm228\text{ vs. }901\pm325\text{ ml, }P=0.503)\).

Blood lactate kinetics
The parameters of blood lactate kinetics are presented in Table 3. No significant effects of training were observed in these parameters in either groups (Table 3). A significant negative correlation was found between changes in \(K_1\) and changes in 250-kJ time trial performance after training when all the participants were combined \((r=-0.553, P=0.011)\).


Discussion

The fact that athletes spend a smaller part of their annual training cycle for HIT highlights the importance of a well-designed HIT training session [35, 37]. The purpose of the present study was to determine the boundaries of the severe domain, which resulted in a very large difference in the intensity of the training protocols, and to investigate the influence of training at the lower and higher severe-intensity boundaries on sprint and endurance performance. The main finding of the present study is that, although both training programs improved the performance in a 250-kJ time trial to similar extents, only the training performed at higher intensities improved WAnT performance. The improvements in MPO following IHIGH training were moderately correlated with increases in vastus lateralis muscle activation and were accompanied by lower AOD in the second half of the WAnT. In addition, the significantly higher PPO was accompanied by higher AOD in the first 5 s of the test. On the other hand, the higher O2 consumption during the WAnT following HIT performed at 105 %CP may suggest an increased aerobic contribution that, in combination with an unaltered PPO, resulted in a lower fatigue index.

Since the neuromuscular load induced during an acute training session might modulate long-term adaptations, intensities ranging from 80–85 % to 120 % of VO2max have been suggested as optimal to promote positive neuromuscular loading [6]. However, shorter intervals at higher intensities may induce greater neuromuscular load than longer intervals at lower intensities during HIT [6]. In addition, while a large pool of muscle fibres might already be recruited during long intervals (considering a minimal recruitment threshold at >75–85 % of VO2max for type II fibres) [2, 6, 17], the firing rate and relative force developed per fibre are likely greater during short intervals at higher intensities [6]. Furthermore, the frequent accelerations and decelerations accompanying training using shorter intervals increase metabolic and muscle force demands [6]. As hypothesised, training performed at IHIGH induced higher vastus lateralis RMS during the WAnT in comparison with training at 105 %CP, and this was associated with greater improvements in MPO. Similar results were previously obtained after sprint training [13]. When combined with the positive correlation between peak torque during a training session and training-induced changes in MPO and PPO, the present results provide further evidence of the importance of higher severe exercise intensities, resulting in higher forces applied to the pedals, for neuromuscular adaptations during short-duration cycling performance.

In addition to the changes in neuromuscular function, the changes in physiological measures may also have contributed to the improved sprint performance. After IHIGH training, the improvements in WAnT performance were accompanied by alterations in AOD but no changes in peak [La] or quantity of [La] accumulated during the test. While these results could suggest no changes in the contribution of the glycolytic metabolism [4, 16], the improved PPO and the higher AOD in the first 5 s of the WAnT indicate that IHIGH training increased phosphocreatine hydrolysis. In addition, the reduced AOD in the second half of the WAnT indicates a higher aerobic contribution that could have increased MPO. Others researchers have observed that improvements in the WAnT results were accompanied by enhanced aerobic metabolism and unaltered [La] values [8, 23, 41]. However, it is important to note that although previous studies have observed that training performed at low intensities improved sprint performance [9, 41], 4 weeks of HIT at the lower boundary of the severe domain did not improve WAnT performance. Thus, high exercise intensities within the severe domain, which require a higher neuromuscular load, are recommended to enhance sprint performance.

In contrast to previous studies [24, 25], neither of the training interventions used in the present study changed the parameters of blood [La] kinetics. Although differences in the exercise protocols between the studies (i.e., relative intensity vs. all-out exercise) may have contributed to this disagreement in the results, improvements in lactate clearance in the present study are also possible. In addition, a negative correlation between changes in K1 and performance in the 250-kJ time trial was observed in the present study. High K1, which can prevent early lactate accumulation and a concomitant fall in intramuscular pH [20], is an important determinant of supramaximal performance [26]. Improvements in capillary supply and density [25], capacity of sarcolemmal carrier-mediated lactate transport [29], and synthesis of new transporters proteins such as MCT1 and MCT4 may contribute to lactate transport capacity improvements with training [25, 30]. Therefore, the significant correlation found in the present study further highlights the importance of higher lactate exchange ability for improving endurance performance in the severe domain [24, 25].

Given the wealth of literature on training adaptations following HIT, it is becoming extremely difficult to design further studies that can offer novel mechanistic insight beyond minor increments to existing knowledge. Although we are aware that an accurate estimation of the upper (İHIGH) and the lower (ÇP) boundaries of the severe exercise intensity domain are laborious and time consuming, which is appropriate in a scientific study but may be unsuitable for real-world training; such a high precision would not be necessary in a practical setting involving elite athletes. Although the present results demonstrate the general efficacy of training in the severe domain for improving endurance performance, the larger improvements in the WAnT results

<table>
<thead>
<tr>
<th>105%CP</th>
<th>Pre-training</th>
<th>Post-training</th>
<th>IHIGH</th>
<th>Pre-training</th>
<th>Post-training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest [La], mmol·L(^{-1})</td>
<td>1.4±0.3</td>
<td>1.2±0.4 *</td>
<td>1.5±0.4</td>
<td>1.4±0.5</td>
<td></td>
</tr>
<tr>
<td>Pretest [La], mmol·L(^{-1})</td>
<td>3.8±0.8</td>
<td>3.4±0.9</td>
<td>3.7±0.8</td>
<td>3.2±0.9</td>
<td></td>
</tr>
<tr>
<td>[La]0, mmol·L(^{-1})</td>
<td>6.6±1.3</td>
<td>5.9±1.5</td>
<td>7.1±1.6</td>
<td>6.4±2.0</td>
<td></td>
</tr>
<tr>
<td>Peak [La], mmol·L(^{-1})</td>
<td>14.2±1.0</td>
<td>14.2±1.6</td>
<td>14.3±1.8</td>
<td>14.2±2.4</td>
<td></td>
</tr>
<tr>
<td>AL, mmol·L(^{-1})</td>
<td>12.0±2.6</td>
<td>12.6±2.2</td>
<td>11.0±2.3</td>
<td>12.0±2.2</td>
<td></td>
</tr>
<tr>
<td>K1, min(^{-1})</td>
<td>0.32±0.11</td>
<td>0.34±0.09</td>
<td>0.37±0.06</td>
<td>0.37±0.16</td>
<td></td>
</tr>
<tr>
<td>K2, min(^{-1})</td>
<td>0.042±0.01</td>
<td>0.043±0.01</td>
<td>0.040±0.01</td>
<td>0.045±0.01</td>
<td></td>
</tr>
<tr>
<td>QLa, mmol</td>
<td>656±131</td>
<td>662±121</td>
<td>621±113</td>
<td>663±160</td>
<td></td>
</tr>
</tbody>
</table>

Data are in Mean±SD. * Significant difference from pre-training (P<0.05)
elicited by \textsuperscript{1}H\textsubscript{igh} training further highlights the importance of higher intensities in facilitating the integration of multiple physiologic systems in recreational cyclists. Although these findings may help coaches optimize training programs for recreational athletes, further research is needed for elite athletes. From a practical point of view, the combination between the highest exercise intensity and tolerable duration eliciting high neuro-muscular, metabolic, and cardiovascular loads seems to be a better way to promote simultaneous adjustments for both sprint and endurance exercise performance in recreationally trained cyclists.

One possible limitation of the present investigation is the use of EMG measurements in a longitudinal study. While care was taken to utilise the same sites for pre- and post-training measurements, training effects might very subtly alter the motor unit recruitment, resulting in sampling of different fibres. However, in addition to using similar cadence during the unloaded cycling phase between training periods, the normalisation procedures were performed according to the recommendations for improving EMG reliability and sensibility during dynamic exercise \cite{1}.

Another possible limitation of the present study is the lack of a 250-kJ familiarisation trial. This shortcoming, however, would not affect the comparisons between the groups, whereas the coefficient of variation in untrained subjects has been found to be small (2.6\%) \cite{8}. Nevertheless, the presence of a learning effect could have affected the ~9\% improvements observed in both groups, which are similar to the results reported in previous studies \cite{8}.

In conclusion, we found that \textsuperscript{1}H\textsubscript{igh} training leads to significantly better training adaptations and improvements in the \textit{WAnT} results after 4 weeks than 105\%CP training. Moreover, changes in EMG and MPO were correlated and followed by an improved interaction within the energy systems. Since both groups demonstrated positive adaptations in the 250-kJ time trial, the severe domain appears to be an effective range of exercise intensities for HIT, even though higher intensities improved sprint performance to a greater extent.

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Conflict of interest: The authors have no conflict of interest to declare.

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