High-intensity cycling training: the effect of work-to-rest intervals on running performance measures

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Running Head: Cycling HIT for running performance

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ABSTRACT

The work-to-rest ratio during cycling based high-intensity training (HIT) could be important in regulating physiological and performance adaptations. We sought to determine the effectiveness of cycling based HIT with different work-to-rest ratios for long-distance running. 32 long-distance runners (age: 39 ± 8y; sex: 14M, 18F; average weekly running training volume: 25 miles) underwent baseline testing (3-km time-trial, \(\dot{V}O_2\)peak and time to exhaustion, Wingate test) prior to a 2-week matched-work cycling HIT of 6 x 10 sec sprints with different rest periods (30s (R30), 80s (R80), 120s (R120) or control). 3-km time trial was significantly improved in the R30 group only (3.1 ± 4.0%, \(p = 0.04\)), whereas time to exhaustion was significantly increased in the two groups with a lower work-to-rest ratio (R30 group 6.4 ± 6.3%, \(p = 0.03\) vs. R80 group 4.4 ± 2.7%, \(p = 0.03\) vs. R120 1.9 ± 5.0%, \(p = 0.2\)). However, improvements in average power production were significantly greater with a higher work-to-rest ratio (R30 group 0.3 ± 4.1%, \(p = 0.8\) vs. R80 group 4.6 ± 4.2%, \(p = 0.03\) vs. R120 group 5.3 ± 5.9%, \(p = 0.02\)), whereas peak power significantly increased only in the R80 group (8.5 ± 8.2%, \(p = 0.04\)) but not the R30 group (4.3 ± 6.1%, \(p = 0.3\)) or the R120 group (7.1 ± 7.9%, \(p = 0.09\)). Therefore cycling based HIT is an effective way to improve running performance and the type and magnitude of adaptation is dependent on the work-to-rest ratio.
Keywords: training adaptations; time-trial; time to exhaustion; $\dot{V}O_2$peak; Wingate test

INTRODUCTION

Training volume is an important variable in the prediction of running performance (36). However, a sudden increase in running volume, particularly with an absolute volume above 40 miles per week, is one of the strongest independent risk factors associated with running injuries (20). 70% of runners at different performance levels are reported to sustain overuse injuries yearly (26). Therefore, reducing weekly running volume may help prevent the risk of injury among runners by decreasing the training impact load. However, this cannot be done at the expense of losing important physiological adaptations. Maximal oxygen uptake ($\dot{V}O_2$max), lactate threshold (LT) and running economy are the main physiological determinants of long-distance running performance (36). It has also been suggested that anaerobic capacity and skeletal muscle power may influence endurance performance (7, 38). Therefore, introducing other forms of training that target these components may improve running performance and help reduce orthopaedic injuries (18, 19, 37). Further, cross-training has been found to be an effective method to maintain training adaptations in runners during recovery following injury (39).

Recent findings demonstrate that high-intensity Wingate-based interval training (HIT) offers a time-efficient training method for sport performance by inducing similar skeletal muscle and cardiovascular adaptations when compared to traditional endurance training (1, 10, 22, 25). 2 weeks of HIT has been shown to increase skeletal muscle lactate and $H^+$ transport capacity (40), increase aerobic and anaerobic enzyme activity (41), improve muscle oxidative capacity and mitochondrial content (8, 10). These adaptations have also been shown to result in functional improvements in moderately-trained male triathletes as reflected by increased
cycling VO2 peak, cycling time to exhaustion and reduced self-paced cycling and running time-trials (17, 27).

Although the HIT protocol appears to induce endurance adaptation, the most effective method remains unclear given that the work-to-rest ratios utilized by the aforementioned studies (1, 10, 22, 25) varied substantially. For example, Hazell et al. (2010) used three experimental groups with unmatched training load with work-to-rest ratios of 1:8, 1:12 and 1:24 over 2 weeks. Therefore an ideal work-to-rest stimulus for HIT is still unknown. More recently, a study by Edge et al. (2013) demonstrated no significant effect on muscle or endurance performance adaptations between the two intense interval training protocols of matched training volume and intensity but different rest periods (i.e. 1 min versus 3 min). However, they used 2 min HIT bouts and it is unclear if manipulation of the rest period would lead to the same results during shorter supramaximal HIT exercise bouts.

Therefore, the primary aim of the present study was to determine whether six sessions of high-intensity cycling training involving 6 x 10-s sprints over 2 weeks is an effective method to improve running performance measures. A secondary aim was to measure the magnitude of performance change in three different work-to-rest groups. It was hypothesized that the HIT would improve running performance without increasing running mileage and that the group using the shortest work-to-rest period would demonstrate the greatest change in physiological and performance outcomes.
METHODS

Experimental Approach to the Problem

A four group pre-post research test design was employed in the study. To assess responses to cycling HIT, the subjects were tested pre and post training with testing being performed over a seven day period and within 48 hours after completing training. Testing included body height and mass assessment and 3-km self-paced running time-trial (day 1), \( \dot{V}_{\text{O}_2}\text{peak} \) determination and time-to-exhaustion (day 2) and 30-s Wingate anaerobic test (day 3). Subsequently, subjects were randomised using stratified sampling based on their baseline \( \dot{V}_{\text{O}_2}\text{peak} \) into one of the three HIT groups or a control group. Over the next two weeks all training groups underwent six sessions of 6 x 10-s ‘all-out’ cycling efforts against a resistance equalling 7.5% of body weight with different recovery periods (R30 GROUP – 30-s rest, R80 GROUP - 80-s rest and R120 GROUP – 120-s rest). The remaining group acted as a control (CON) group which completed pre- and post-tests 2 weeks apart whilst maintaining their normal training regime but did not complete HIT protocol. Post-training tests were performed in the identical order. Furthermore, for a given subject, each training and testing protocol was performed within 2 hours of the same time of the day. The subjects were also asked to refrain from vigorous training for 24 hours before each test and to maintain their normal training and diet routine throughout the study period.

Subjects

Twenty four competitive runners (10 males and 14 females) from local running clubs volunteered to participate in this study. At the time of the study all subjects ran for at least 25 miles per week. In addition, eight subjects (4 males and 4 females) served as controls.

Physical characteristics of the subjects are shown in Table 1, with no statistical difference between groups for performance measures. Subjects were also familiar with exhaustive
treadmill running and could complete 3-km in less than 18 minutes. All provided written informed consent and completed a Physical Activity Readiness Questionnaire (PAR-Q) prior to participation in the procedures that were approved by the University Research Ethics Committee.

**TABLE 1.** Demographic characteristics of all subjects (mean ± SD).

<table>
<thead>
<tr>
<th></th>
<th>R30 Group</th>
<th>R80 Group</th>
<th>R120 Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>3M; 5F</td>
<td>3M; 5F</td>
<td>4M; 4F</td>
<td>4M; 4F</td>
</tr>
<tr>
<td>Age (y)</td>
<td>41 ± 12</td>
<td>38 ± 7</td>
<td>42 ± 6</td>
<td>34 ± 8</td>
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<tr>
<td>Height (cm)</td>
<td>168 ± 11</td>
<td>168 ± 9</td>
<td>168 ± 7</td>
<td>169 ± 9</td>
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<tr>
<td>Body Mass (kg)</td>
<td>69 ± 15</td>
<td>64 ± 10</td>
<td>71 ± 11</td>
<td>67 ± 7</td>
</tr>
<tr>
<td>$\dot{V}O_2$ peak (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>45 ± 8</td>
<td>43 ± 4</td>
<td>44 ± 7</td>
<td>46 ± 6</td>
</tr>
<tr>
<td>3km Time-Trial (s)</td>
<td>889 ± 83</td>
<td>858 ± 113</td>
<td>875 ± 71</td>
<td>814 ± 126</td>
</tr>
</tbody>
</table>

**Procedures**

**Baseline Testing**

Subjects were required to report to the Human Performance Laboratory for performance tests on three separate days with at least 24 hours between each testing session over the course of one week. During the initial visit the height and mass of each subject was measured to the nearest 0.1 cm (SECA 216 Mechanical Stadiometer, Hamburg, Germany) and 0.1 kg (SECA 796 Column Scale, Hamburg, Germany), respectively.

**3-km Time-Trial**

Participants performed a 3-km self-paced running time-trial on a treadmill (H/P/Cosmos Mercury, Germany) set at 1% gradient (30) and were given instructions to complete the test
in the fastest possible time. A coefficient of variation of the 3-km running time-trial on an outdoor 400-m track has been reported to be 1.2% (29). Before the time-trial, the participants did a 5 minute standardised warm-up which consisted of running for 3 minutes at a constant speed (7.5 km•h⁻¹ for women and 9.1 km•h⁻¹ for men) and 2 minutes of pre-race stretching routine. After setting initial speed at 11.2 km•h⁻¹ and 12.9 km•h⁻¹ for women and men, respectively, participants were free to manually control their own speed and were able to adjust it constantly throughout the time-trial. However there was no visual feedback apart from distance covered to ensure that subsequent trials are not affected by a pacing strategy (3). Standardized verbal encouragement for each participant was provided throughout the test.

**Assessment of \( \dot{V}O_2 \text{peak} \)**

Participants performed a continuous graded exercise test (GXT) on a motorised treadmill (H/P/Cosmos Mercury, Germany) to determine \( \dot{V}O_2 \text{peak} \) via breath by breath gas collection system (Metalyzer\textsuperscript{®} 3B gas analyser, Cortex, Leipzig, Germany). In addition, time-to-exhaustion was recorded using a Quantum 5500 stopwatch (EA Combs Ltd., UK) and subjects were not allowed to see the time throughout the test. Briefly, following a 5 minute standardised warm-up which consisted of running at a constant speed (9.6 km•h⁻¹ for women and 11.2 km•h⁻¹ for men) at 0% gradient the speed was increased (11.2 km•h⁻¹ and 12.9 km•h⁻¹ for women and men, respectively). After 2 minutes the gradient was increased by 2.5% and then every 2 minutes until volitional exhaustion (44). \( \dot{V}O_2 \text{peak} \) was taken as the highest value averaged over 30-s collection periods.

**Anaerobic Power Test**

Anaerobic power output was assessed by a 30-second Wingate Anaerobic Test (WAnT) using a Monark Ergomedic 894E cycle ergometer (Monark Exercise AB, Varberg, Sweden) against
a resistance equalling 7.5% of body weight. After the ergometer seat height had been adjusted, participants performed a 3-min warm-up cycling at 60 W subjects and were instructed to perform a 30-s ‘all-out’ sprint pedalling as fast as possible from the start with a predetermined resistance automatically applied to the flywheel upon reaching 100 r•min$^{-1}$.

Verbal encouragement was provided throughout the test. Relative values of peak power (the highest power output over any 5-s reached during the test) and mean power (over the entire test) were automatically calculated by computer (Monark Anaerobic Test Software version 2.24.2, Monark Exercise AB, Varberg, Sweden). In addition, blood lactate concentration was determined from a fingertip capillary blood sample (Lactate Pro, Arkay Inc., Kyoto, Japan) at rest, immediately after the test, 2 min and 5 min post-test. Briefly, the skin was punctured using an Accu-check single use lancet (Roche Diagnostics, UK) and pressure applied to the finger to draw the blood. The initial drop was discarded and the second drop was taken for lactate analysis.

**High-Intensity Training**

At least 48 hours after the last baseline test, subjects completed their first session of HIT consisting of 6 x 10-s ‘all-out’ cycling efforts against a resistance equalling 7.5% of body weight. Rest periods were prescribed based on the relevant groups (i.e. R30, R80 and R120) with active recovery in the form of unloaded pedalling at 60 r•min$^{-1}$ completed in between intervals. Subjects were weighed in before each session to ensure that accurate resistance on the flywheel was used throughout the duration of the training programme. The same training routine was repeated five more times over the next 2 weeks with at least 24 hours between the sessions. Heart rate (Polar Electro, Kempele, Finland) and peak and mean power output (Monark Anaerobic Test Software version 2.24.2, Monark Exercise AB, Varberg, Sweden) were recorded continuously throughout. All subjects were verbally encouraged during training.
Post-Training Testing

All subjects in the training groups repeated 3-km time-trail, \( \dot{V}O_2 \)peak and Wingate anaerobic tests in the same order within 48 hours after completing training. Subjects in the control group were retested 2 weeks after the completion of baseline testing.

Statistical Analyses

All performance data was normalised to baseline to determine the magnitude of change prior to statistical analysis. Data was checked for skewness and kurtosis and these values did not exceed twice the standard error, therefore the data was deemed to be normally distributed. A one-way ANOVA with Student Newman Keuls post hoc testing was used to compare the magnitude of change between groups and to analyse the average training heart rate response. A paired samples t-test was used to compare the pre- and post-outcomes in the control group. The null hypothesis was rejected at the 5% level \((p \leq 0.05)\). The Cohen’s \( d \) effect size was calculated to quantify the magnitude of difference between groups with effect size defined as trivial (0.0-0.2), small (0.2–0.5), moderate (0.6–1.1) and large (1.2–1.9) (11).

RESULTS

Time-Trial

At baseline time to complete 3-km was not significantly different between groups (R30 group 889 ± 83 sec; R80 group 858 ± 113 sec; R120 group 875 ± 71 sec; control group 814 ± 126 sec, \( p > 0.05 \) for all group comparisons) and the control group was unchanged after 2 weeks (pre: 814 ± 126 sec; post: 813 ± 114 sec, \( p = 0.7 \)). 2 weeks of HIT training resulted in a significant decrease in 3-km time-trial in the R30 group (3.1 ± 4.0 \%, \( p = 0.04 \), compared to control, \( d = 0.9 \); Figure 1), but a non-significant change in the R80 group (2.4 ± 2.7 \%, \( p = 0.1 \), compared to control, \( d = 0.9 \); Figure 1) and R120 group (2.4 ± 2.4 \%, \( p = 0.1 \), compared...
to control, $d = 0.9$; Figure 1). There was no significant difference in the magnitude of change between the HIT groups (R30 vs. R80, $p = 0.6$, $d = 0.2$; R30 vs. R120, $p = 0.6$, $d = 0.2$; R80 vs. R120, $p = 1.0$, $d = 0.0$).

Insert Figure 1 about here

**Time to Exhaustion**

At baseline time to exhaustion was not significantly different between groups (R30 group 703 ± 76 sec; R80 group 709 ± 84 sec; R120 group 692 ± 68 sec; control group 755 ± 89 sec; $p > 0.05$ for all group comparisons, Figure 1) and the control group was unchanged after 2 weeks (pre: 755 ± 89 sec; post: 750 ± 97 sec; $p = 0.5$). Following 2 weeks of HIT training time to exhaustion significantly improved in the R30 group (6.4 ± 6.3 %, $p = 0.003$, compared to control, $d = 1.4$; Figure 1) and the R80 group (4.4 ± 2.7 %, $p = 0.03$, compared to control, $d = 1.6$; Figure 1), but there was no significant increase in the R120 group (1.9 ± 5.0 %, $p = 0.2$, compared to control, $d = 0.6$; Figure 1). There was a significant difference in the magnitude of change between the R30 and R120 groups ($p = 0.05$, $d = 0.8$) but not between the R30 and R80 groups ($p = 0.4$, $d = 0.4$) or the R80 and R120 groups ($p = 0.3$, $d = 0.6$).

**$\dot{V}O_2$peak**

At baseline $\dot{V}O_2$peak was not significantly different between groups (R30 group 45.1 ± 8.4 ml.kg$^{-1}$.min$^{-1}$; R80 group 43.3 ± 3.9 ml.kg$^{-1}$.min$^{-1}$; R120 group 43.6 ± 7.2 ml.kg$^{-1}$.min$^{-1}$; control group 46.6 ± 6.2 ml.kg$^{-1}$.min$^{-1}$; $p > 0.05$ for all group comparisons; Figure 1) and the control group was unchanged after 2 weeks (pre: 46.6 ± 6.2 ml.kg$^{-1}$.min$^{-1}$; post: 46.9 ± 6.3 ml.kg$^{-1}$.min$^{-1}$; $p = 0.8$). 2 weeks of HIT training did not increase $\dot{V}O_2$peak significantly in any of the groups (R30 group 6.9 ± 14 %, $p = 0.2$, compared to control, $d = 0.5$; R80 group 4.7 ± 6.4 %, $p = 0.4$, compared to control, $d = 0.6$; R120 group 0.3 ± 4.4 %, $p = 0.8$, compared to
control, $d = 0.2$; Figure 1). There was no significant difference in the magnitude of change between groups (R30 vs. R80, $p = 0.6$, $d = 0.2$; R30 vs. R120, $p = 0.1$, $d = 0.7$; R80 vs. R120, $p = 0.3$, $d = 0.9$).

**Wingate Performance**

At baseline peak and average power was significantly different between R80 and R120 ($p > 0.05$) groups but not between other groups (Figure 2) and the control group was unchanged after 2 weeks (peak power - pre: $11.3 \pm 2.5$ W.kg$^{-1}$; post: $11.4 \pm 2.4$ W.kg$^{-1}$; $p = 0.9$; average power - pre: $7.5 \pm 0.8$ W.kg$^{-1}$; post: $7.5 \pm 0.9$ W.kg$^{-1}$; $p = 0.7$). Following 2 weeks of HIT training there was no significant change in average power in the R30 group ($0.3 \pm 4.1\%$, $p = 0.8$ compared to control, $d = 0.2$; Figure 2), but significant increases in the R80 group ($4.6 \pm 4.2\%$, $p = 0.03$, compared to control, $d = 1.4$; Figure 2) and the R120 group ($5.3 \pm 5.9\%$, $p = 0.02$, compared to control, $d = 1.2$; Figure 2). There was a significant difference in the magnitude of change between the R30 and R80 groups ($p = 0.05$, $d = 1.0$) and the R30 and R120 groups ($p = 0.03$, $d = 1.0$) but not the R80 and R120 groups ($p = 0.7$, $d = 0.1$). Peak power was not increased significantly in the R30 group ($4.3 \pm 6.1\%$, $p = 0.3$, compared to control, $d = 0.6$; Figure 2) or the R120 group ($7.1 \pm 7.9\%$, $p = 0.09$, compared to control, $d = 0.9$; Figure 2) but there was a significant in the R80 group ($8.5 \pm 8.2\%$, $p = 0.04$, compared to control, $d = 1.1$; Figure 2). There was no significant difference in the magnitude of change between the groups (R30 vs. R80, $p = 0.2$, $d = 0.6$; R30 vs. R120, $p = 0.4$, $d = 0.4$; R80 vs. R120, $p = 0.7$, $d = 0.2$).

*Insert Figure 2 about here*

Blood lactate concentrations after the Wingate were not changed following 2 weeks of HIT, regardless of recovery duration (Table 2).
Average Heart Rate During Training

Average heart rate was significantly greater in the R30 group compared to the R120 group for all training sessions ($p = 0.000$; Figure 3) and was significantly greater in the R80 group compared to the R120 group for training sessions 1 and 2 ($p = 0.004$; Figure 3). There was no significant difference between R30 and R80 for average training heart rate but there was a trend for it to be greater in the R30 group ($p = 0.268$; Figure 3).

DISCUSSION

The purpose of this study was to measure the magnitude of changes of running performance and aerobic capacity (as determined by $\dot{V}O_2$ peak) following a matched-work cycling HIT protocol with different rest periods. In accordance with our hypothesis, the main finding was that a short cycling HIT protocol, especially with lower work-to-rest ratio, provides sufficient training stimulus to have a carry-over effect into running performance (Figure 1). These findings demonstrate that a non-specific training may contribute to improved running performance in already trained runners. Recent studies using cycling HIT have reported varying degrees of health and performance benefits (17, 22, 33). Although the mechanisms contributing to HIT adaptation is dependent on the training statuses of individuals, performance improvement following cycling HIT have been attributed to alterations in central (i.e. cardiovascular) and peripheral (i.e. skeletal muscle) adaptations (22, 33). However, the extent of performance improvements and related physiological changes elicited by cycling HIT appears to depend on the duration and intensity of each bout, number of intervals completed as well as the type and duration of recovery used (32, 42). The major
findings of this study support the notion that manipulation of rest duration during a matched-work cycling HIT protocol results in different adaptations for long-distance runners.

**Time-trial**

Following 6 sessions of HIT over 2 weeks, the R30 group significantly \( p \leq 0.05 \) improved the 3-km time-trial (TT) performance, but there was no significant improvement in the R80 and R120 groups \( p > 0.05 \) (Figure 1). The magnitude of change was not significantly different between the HIT groups \( p > 0.05 \), with only a small effect size reported for the R30 group compared to the R80 \( d = 0.2 \) and R120 \( d = 0.2 \) groups (Figure 1). The R30 group had the greatest (3.1%) reduction in time taken to complete the TT compared to R80 (2.4%) and R120 (2.4%), and the magnitude of change is similar to that reported previously for a 5-km cycling TT (3 - 5.2%) (25). Even greater improvements were found by Jakeman et al. (2010) who demonstrated that six training sessions using 10 x 6-s Wingate-based sprints resulted in a 10% decrease in self-paced 10-km cycling TT in sub-elite triathletes. Similarly, Burgomaster et al. (2005, 2006) observed a 9.6% decrease in a 250-kJ self-paced TT (approx. 10km) and a 4% decrease in a 50-kJ self-paced TT (approx. 2km) in young active men after six sessions of 4-6 x 30-s cycling sprints. This suggests that despite different HIT volumes (duration x number of sprints) used in the above studies, the magnitude of performance improvements can be expected to be greater during a longer time-trial. It was suggested by Sloth et al. (2013) that aerobic gains may depend on the work-to-rest ratios used during HIT protocols. This may explain why the magnitude of change was greatest in the R30 group (Figure 1). Shorter recovery times between repeated high-intensity cycling sprints increase aerobic metabolism demand in subsequent sprints (21), mainly due to reduced muscle PCr availability (4). Although we did not measure oxygen uptake \( \dot{V}O_2 \) during recovery periods directly, average heart rate (HR) data from training sessions were significantly greater in the...
R30 group compared to the R120 group in all training sessions (\(p = 0.000\)) and in the R80 group compared to the R120 (\(p = 0.004\)) during the first two training sessions (Figure 3). This indicates that the overall cardiovascular demand of a training session was higher with shorter recovery, suggesting a greater aerobic demand. In other studies that have measured heart rate during a repeated sprint session, shorter work-to-rest periods produce a greater cardiovascular demand (6, 35). Likewise, levels of oxygen uptake during and following sprints are greater with shorter work-to-rest ratios (24).

**Time to exhaustion**

TT is a more ecologically valid test to measure endurance performance, whereas time to exhaustion is a test of exercise capacity (14). Only R30 and R80 groups significantly improved their time to exhaustion (\(p \leq 0.05\)), with no significant improvement in the R120 group (\(p > 0.05\)) (Figure 1). The magnitude of change was dependent on recovery duration as lower work-rest ratios (R30 +6.4\%, \(d = 0.8\) and R80 +4.4\%, \(d = 0.6\)) had a greater effect on time to exhaustion compared to a higher work-rest ratio (R120 +1.9\%). In one study (8) recreationally active subjects doubled their endurance time to fatigue following 2 weeks of HIT during cycling exercise at \(~80\% \dot{V}O_2\text{peak}\). In contrast, Jakeman et al. (2010) reported a non-significant change during an incremental ramp cycling test to volitional exhaustion after 2 weeks of cycling HIT. One of the underlying mechanisms responsible for a better exercise tolerance observed in two groups (i.e., R30 and R80) with less rest time may be a greater improvement in skeletal muscle oxidative capacity. Alterations in mitochondrial activity are reported after 2 weeks of cycling HIT (10) as well as a rightward shift in the blood lactate curve (27) seen with a 1:8 and 1:10 work-to-rest ratio, respectively. These muscle adaptations are important in explaining the improvement in endurance performance that occurs with training and the longer recovery may not provide a large enough stimulus to promote
sustained changes in these systems. Others have suggested improvements in time to
exhaustion are related to a smaller O\textsubscript{2} deficit at the start of the exercise resulting in a smaller
anaerobic contribution (15). However, based on the results of this study it is impossible to
determine whether central or peripheral physiological mechanisms are affecting the
improvement in running time to exhaustion after cycling HIT training with short recovery
periods.

\textit{\breve{V}O}_2 \text{peak}

Despite significant changes in TT in the R30 group and time to exhaustion in groups R30 and
R80, there were no significant changes in \textit{\breve{V}O}_2 peak in any of the training groups ($p > 0.05$)
(Figure 1). Although the magnitude of change between groups was non-significant ($p > 0.05$),
the R30 (+6.9%, $d = 0.7$) and R80 (+4.7%, $d = 0.9$) groups showed a trend for greater
adaptations compared to the R120 group (-0.3%). The magnitude of change in \textit{\breve{V}O}_2 peak in
the R30 and R80 groups is similar to that which has been reported previously for 30 second
sprint protocols over 2 weeks (1). Therefore, an increase in the length of sprint may not
result in greater adaptations of \textit{\breve{V}O}_2 peak. Hazell et al. (2010) also reported a smaller
adaptation in \textit{\breve{V}O}_2 peak with a 1:12 work-to-rest ratio compared to a 1:8 work-to-rest ratio. This
shows that the shorter recovery period places a greater demand on the body leading to greater
adaptations in aerobic capacity. Together with the heart rate data (Figure 3) these findings
suggest that cardiovascular training intensity may be important in regulating training
response of \textit{\breve{V}O}_2 peak.

\textbf{Wingate performance}

Peak power output (PPO) was significantly increased only in the R80 ($p \leq 0.05$) but not the
R30 and R120 groups ($p > 0.05$) (Figure 2). The R80 showed the greatest change in PPO
(+8.5%) in comparison to the R30 and R120 groups (+4.3%, \(d = 0.6\) and +7.1%, \(d = 0.2\), respectively). Mean power output (MPO) was significantly higher in the R80 and R120 groups \((p \leq 0.05)\) but not the R30 group \((p > 0.05)\) (Figure 2). The magnitude of change in MPO was highest in the R120 group (+5.3%) compared to the R30 group (0.3%, \(d = 1.0\)) and the R80 group (4.6%, \(d = 0.1\)). The magnitude of improvements is similar to that reported previously for 10 sec sprints and 1:8 work-to-rest ratios (1, 25). HIT has been shown to up-regulate a number of glycolytic enzymes (31, 34) and result in significant increases in PCr and glycogen concentrations after training (41). Two minutes of recovery after the 10-s cycling sprint has been demonstrated to be long enough for subjects to reproduce peak but not mean power in a subsequent sprint (5, 25). Therefore, lower than 1:8 ratios seem to have a negative effect on anaerobic adaptations due to the subsequent sprints relying more heavily on aerobic metabolism. Positive changes in power production following HIT may also occur due to the neuromuscular changes, such as increased motor unit recruitment, firing rate and synchronization (13). However, shorter recovery periods between sprint bouts may induce greater fatigue thereby limiting neural stimuli for optimal power development. The extent of blood lactate accumulation after the Wingate test was the same for each group before and after 2 weeks of cycling HIT (Table 2). This is in line with previous research, however skeletal muscle lactate accumulation is reported to be less following HIT (41). This may reflect an increased efficiency of lactate shuttling following HIT as it has been demonstrated that there is an increase in skeletal muscle and heart lactate transporters (2, 28, 40). This would allow for a greater transfer of lactate around the body and may reflect a better buffering capacity for the skeletal muscle (45).

In conclusion, the present data show that only six sessions of 6 x 10-s all-out cycling sprints over 2 weeks performed alongside normal training is an effective method for recreationally competitive runners to improve running performance. The differences in the magnitude of the
improvement in 3-km TT, time to exhaustion, \( \dot{V}O_2 \) peak, PPO and MPO between the three groups suggest that performance adaptations are dependent on the work-to-rest ratio. 30 seconds rest in between sprint bouts seems to have a detrimental effect on power production in subsequent sprints whereas 120 seconds appears to be too long to induce favourable endurance adaptations. Therefore, 80 seconds seems optimal when targeting both, power and endurance adaptations.

**PRACTICAL APPLICATIONS**

These data demonstrate that cycling HIT has the ‘transferability’ of training effect to running performance by providing sufficient training stimulus to improve a number of important variables. Most importantly, the non-weight-bearing nature and minimal eccentric contraction of leg muscles during stationary cycling (23) offers runners and other athletes a low-volume alternative modality which could minimise the risk of overuse injury. Further, evidence suggests that doubling of training workload does not lead to an increase in performance in well-trained participants (12). Therefore, a relatively fast performance gains with a reduced training volume makes it a time efficient method of training, which could be used during a taper period, a pre-season training and the times of traveling. Finally, this type of training could also be used by athletes during a rehabilitation period who are recovering from injuries that prevent them from running.

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We thank our participants for their time and effort.
REFERENCES


16. Edge, J, Eynon, N, McKenna, MJ, Goodman, CA, Harris, RC, and Bishop, DJ. 


Figure 1. Percentage change in performance indicators. * p ≤ 0.05 pre versus post

Figure 2. Percentage change in Wingate peak and average power.

Figure 3. Average heart rate for each training session. ** p < 0.01 compared to R120; * p ≤ 0.05 compared to R120

TABLE 2. Blood lactate concentration after the Wingate test pre- and post-training.
**TABLE 2.** Blood lactate concentration after the Wingate test pre- and post-training (mean ± SD).

<table>
<thead>
<tr>
<th></th>
<th>Blood Lactate (mmol.l⁻¹)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>baseline</td>
<td>0 min post</td>
<td>2 min post</td>
<td>5 min post</td>
</tr>
<tr>
<td><strong>R30</strong></td>
<td>Pre</td>
<td>1.6 ± 0.5</td>
<td>4.5 ± 1.7</td>
<td>9.6 ± 2.2</td>
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<tr>
<td></td>
<td>Post</td>
<td>1.4 ± 0.6</td>
<td>4.6 ± 3.2</td>
<td>9.6 ± 3.1</td>
</tr>
<tr>
<td><strong>R80</strong></td>
<td>Pre</td>
<td>1.9 ± 1.4</td>
<td>6.2 ± 3.7</td>
<td>10.8 ± 1.7</td>
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<tr>
<td></td>
<td>Post</td>
<td>1.6 ± 0.8</td>
<td>5.3 ± 3.2</td>
<td>11.0 ± 1.9</td>
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<tr>
<td><strong>R120</strong></td>
<td>Pre</td>
<td>1.3 ± 1.3</td>
<td>5.0 ± 3.3</td>
<td>8.1 ± 2.4</td>
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<tr>
<td></td>
<td>post</td>
<td>1.4 ± 0.8</td>
<td>5.7 ± 3.9</td>
<td>11.1 ± 3.4</td>
</tr>
<tr>
<td><strong>control</strong></td>
<td>Pre</td>
<td>2.1 ± 0.9</td>
<td>5.6 ± 2.6</td>
<td>10.2 ± 2.5</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>2.6 ± 1.6</td>
<td>5.6 ± 2.3</td>
<td>10.8 ± 2.1</td>
</tr>
</tbody>
</table>