Acute High-Intensity Interval Training Improves $T_{vent}$ and Peak Power Output in Highly Trained Males

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Catalog Data

Key words: cycling, cyclists, heart rate, oxygen uptake, short-term training, ventilatory threshold
Mots-clés: cyclisme, cyclistes, fréquence cardiaque, consommation d’oxygène, entraînement à court terme, seuil ventilatoire

Abstract/Résumé
This study examined the effects of four high-intensity interval-training (HIT) sessions performed over 2 weeks on peak volume of oxygen uptake ($\text{VO}_{2\text{peak}}$), the first and second ventilatory thresholds ($VT_1$, $VT_2$) and peak power output (PPO) in highly trained cyclists. Fourteen highly trained male cyclists ($\text{VO}_{2\text{peak}} = 67.5 \pm 3.7 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) performed a ramped cycle test to determine $\text{VO}_{2\text{peak}}$, $VT_1$, $VT_2$, and PPO. Subjects were divided equally into a HIT group and a control group. The HIT group performed four HIT sessions (20 x 60 s at PPO, 120 s recovery); the $\text{VO}_{2\text{peak}}$ test was repeated <1 wk after the HIT program. Control subjects maintained their regular training program and were reassessed under the same timeline. There was no change in $\text{VO}_{2\text{peak}}$ for either group; however, the HIT group showed a significantly greater increase in $VT_1$ (+22% vs. -3%), $VT_2$ (+15% vs. -1%), and PPO (+4.3% vs. -4%) compared to controls (all P < .05). This study has demonstrated that HIT can improve $VT_1$, $VT_2$, and PPO, following only four HIT sessions in already highly trained cyclists.

Cettem étude analyse, chez des cyclistes très bien entraînés, les effets de quatre séances intenses d’entraînement par intervalle réparties sur deux semaines; l’évaluation porte sur la

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consommation d’oxygène de crête (\(\text{VO}_{2\text{crête}}\)), le premier et le deuxième seuils ventilatoires (\(\text{VT}_1, \text{VT}_2\)) et la puissance de pointe (PPO). Quatorze cyclistes très bien entraînés (\(\text{VO}_{2\text{crête}} = 67,5 \pm 3,7 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}\)) participent à une épreuve d’effort progressif pour établir leurs \(\text{VO}_{2\text{crête}}, \text{VT}_1, \text{VT}_2\), et PPO. Les sujets sont ensuite répartis également en deux groupes : expérimental (HIT) et témoin. Le groupe expérimental participe à quatre séances (20 x 60 s à la PPO, 120 s de récupération) et l’épreuve d’effort progressif est reprise moins d’une semaine après le programme d’entraînement. Le groupe témoin continue son programme d’entraînement habituel et il est réévalué selon le même scénario. Le \(\text{VO}_{2\text{crête}}\) des deux groupes ne change pas ; cependant, comparativement au groupe témoin, les valeurs de \(\text{VT}_1\) (+22 % vs –3 %), de \(\text{VT}_2\) (+15 % vs –1 %) et de PPO (+4,3 vs –0,4%) du groupe expérimental augmentent significativement (P < 0,05 partout). Ainsi, il est possible d’améliorer les valeurs de \(\text{VT}_1, \text{VT}_2\), et de PPO en seulement quatre séances d’entraînement chez des cyclistes très bien entraînés.

Introduction

It is accepted that short-term endurance training in sedentary or recreationally trained individuals elicits marked improvements in endurance performance and a number of cardiovascular and metabolic markers (Blomqvist and Saltin, 1983; Green et al., 1989, 1990). In highly trained subjects, however, endurance training at submaximal workloads does not appear to induce improvements in either endurance performance or corresponding physiological variables (Costill et al., 1988; Londeree, 1997). In these individuals, further improvements in endurance performance may require a different training stimulus.

Coaches and athletes have long used high-intensity interval training (HIT) to improve endurance performance (Faria, 1984). While HIT appears to be an effective training technique according to anecdotal evidence, scientific support for it remains sparse (Hawley et al., 1997). This may, in part, be due to the difficulty of convincing highly trained, competitive athletes to experiment with their normal training programs (Steptoe et al., 1998). Only in recent years has the influence of HIT on performance and associated physiological markers been examined in long-distance runners (Babineau and Leger, 1997; Billat et al., 1999; Collins et al., 2000), and endurance-trained cyclists (Steptoe et al., 1998; Westgarth-Taylor et al., 1997; Weston et al., 1997). Studies in runners have noted increases in 10-km run performance with reductions in lactate accumulation at submaximal workloads despite no improvement in the ventilatory threshold (\(T_{\text{vent}}\)), running economy, or \(\text{VO}_{2\text{peak}}\) (Acevedo and Goldfarb, 1989; Collins et al., 2000). Recent work with endurance-trained cyclists (Table 1) has found improvements in peak power output (PPO), time to fatigue at 150% of PPO, and 40-km time trial performance (Lindsay et al., 1996; Westgarth-Taylor et al., 1997; Weston et al., 1997). These performance improvements have been attributed to an enhanced fat oxidation at the same absolute exercise intensity (Westgarth-Taylor et al., 1997) and improvements in skeletal muscle buffering capacity without a change in glycolytic or oxidative enzyme activity (Weston et al., 1997).

While improvements in endurance performance and associated physiological variables in already highly trained cyclists have been observed to occur in response to as few as four HIT sessions, research has yet to describe the acute performance and associated physiological adjustments that may occur during
<table>
<thead>
<tr>
<th>Reference</th>
<th>N</th>
<th>HIT Sessions</th>
<th>Reps</th>
<th>Intensity (%PPO)</th>
<th>Work duration</th>
<th>Rest duration</th>
<th>HIT Duration</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Lindsay et al., 1996)</td>
<td>8</td>
<td>6</td>
<td>6-8</td>
<td>80</td>
<td>5 min</td>
<td>60 s</td>
<td>4 wk</td>
<td>↑ PPO, ↑TF&lt;sub&gt;150&lt;/sub&gt;, ↑TT&lt;sub&gt;40&lt;/sub&gt;</td>
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<td>(Weston et al., 1997)</td>
<td>6</td>
<td>6</td>
<td>6-8</td>
<td>80</td>
<td>5 min</td>
<td>60 s</td>
<td>4 wk</td>
<td>↑ PPO, ↑TF&lt;sub&gt;150&lt;/sub&gt;, ↑TT&lt;sub&gt;40&lt;/sub&gt;, ↑βm</td>
</tr>
<tr>
<td>(Westgarth-Taylor et al., 1997)</td>
<td>8</td>
<td>12</td>
<td>6-9</td>
<td>80</td>
<td>5 min</td>
<td>60 s</td>
<td>6 wk</td>
<td>↑ PPO, ↑TT&lt;sub&gt;40&lt;/sub&gt;, ↓CHO&lt;sub&gt;ox&lt;/sub&gt;</td>
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<tr>
<td>(Stepto et al., 1998)</td>
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<td>6</td>
<td>12</td>
<td>175</td>
<td>30 s</td>
<td>4.5 min</td>
<td>3 wk</td>
<td>↑ PPO, ↑TT&lt;sub&gt;40&lt;/sub&gt;</td>
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<tr>
<td>(Stepto et al., 1998)</td>
<td>3</td>
<td>6</td>
<td>12</td>
<td>100</td>
<td>1 min</td>
<td>4 min</td>
<td>3 wk</td>
<td>No change</td>
</tr>
<tr>
<td>(Stepto et al., 1998)</td>
<td>4</td>
<td>6</td>
<td>12</td>
<td>90</td>
<td>2 min</td>
<td>3 min</td>
<td>3 wk</td>
<td>↑ PPO, ↑TT&lt;sub&gt;40&lt;/sub&gt;</td>
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<tr>
<td>(Stepto et al., 1998)</td>
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<td>6</td>
<td>8</td>
<td>85</td>
<td>4 min</td>
<td>1.5 min</td>
<td>3 wk</td>
<td>↑ PPO, ↑TT&lt;sub&gt;40&lt;/sub&gt;</td>
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<tr>
<td>(Stepto et al., 1998)</td>
<td>4</td>
<td>6</td>
<td>4</td>
<td>80</td>
<td>8 min</td>
<td>1 min</td>
<td>3 wk</td>
<td>No change</td>
</tr>
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</table>

*Note:* Peak Power Output (PPO); time to fatigue at 150% PPO (TF<sub>150</sub>); 40-km time trial performance (TT<sub>40</sub>); skeletal muscle buffering capacity (βm); carbohydrate oxidation (CHO<sub>ox</sub>).
consecutive HIT sessions. Furthermore, while the influence of HIT on \( \dot{V}O_2 \text{peak} \) and \( T_{vent} \) have been reported in long-distance runners (Acevedo and Goldfarb, 1989), and recreationally trained cyclists (Norris and Petersen, 1998), the response of highly trained cyclists to HIT has yet to be described. Therefore, the purpose of the present study was to examine the influence of exhaustive HIT in highly trained cyclists on (a) \( \dot{V}O_2 \text{peak} \), PPO, and \( T_{vent} \) (both the first and second ventilatory thresholds [\( VT_1 \) and \( VT_2 \)]; Lucia et al., 2000), (b) cycling performance, and (c) changes in cardiovascular and metabolic markers measured throughout the HIT program.

**Methods**

**SUBJECTS**

Fourteen highly trained male cyclists (age = 23.5 ± 3.5 yr; height = 179.1 ± 2.6 cm; mass = 71.6 ± 5.8 kg) were recruited for this study; subjects had to have a minimum cycling \( \dot{V}O_2 \text{peak} \) value of 60 ml · kg\(^{-1} \) · min\(^{-1} \). Subjects had been training for and competing in cycling events on a regular basis for a minimum of 3 yr, but training completed by the subjects during the 2 months prior to testing was low-intensity in nature (289 ± 42 km · wk\(^{-1} \)). All subjects were undergraduate and postgraduate students, and/or university cycling team members, who were all familiar with the experimental setting and experienced with exercise testing procedures. After being fully informed of the risks and stresses associated with the study, subjects completed a medical history questionnaire and gave their written informed consent to participate. The experimental protocol was approved by the Medical Research Ethics Committee of The University of Queensland.

**TESTING REGIMEN**

Testing was conducted over a three-week period during the base period (low intensity in nature) of the participants’ training program. Subjects were divided equally into a HIT group (\( N = 7 \)) and a control group (\( N = 7 \)). All subjects were asked to keep a detailed training diary (of both distance cycled and time spent cycling). Due to the fact that one HIT session is completed in approximately 1 hr, the cyclists in the HIT group were asked to remove ~2 hr · wk\(^{-1} \) of training from their normal base training program during the HIT weeks. Control subjects were asked to maintain their current training status during this period. Subjects reported to a controlled environmental laboratory condition (~21 °C, 40-60%RH, 760–770 mmHg) at the same time of day for both \( \dot{V}O_2 \text{peak} \) tests, and HIT sessions. Before all \( \dot{V}O_2 \text{peak} \) and HIT sessions, athletes were asked to keep their eating habits constant, and to report to the laboratory at least 3-h post-parandial. During this time, they were encouraged to drink water to maintain euhydration. Subjects in the HIT group reported initially for their \( \dot{V}O_2 \text{peak} \) test, and upon meeting the inclusion requirements began the first of their HIT sessions within one week. The four HIT sessions were completed over a 2 wk period, and the subjects repeated the \( \dot{V}O_2 \text{peak} \) test within one week after completion of the final HIT session. Control subjects were reassessed over a similar time course (14–21 d between tests). The same encouragement was given during each \( \dot{V}O_2 \text{peak} \) test, and incremental power output (PO) was blinded to the athlete.
PEAK OXYGEN UPTAKE

$\dot{V}O_2$ was determined on an electronically braked cycle ergometer (Lode Excalibur Sport, Quinton) modified with clip-in pedals and low profile racing handlebars, using a continuous test as described previously (Bishop et al., 1998). Subjects were weighed beforehand for the calculation of $\dot{V}O_2$ in relative terms. The saddle and handle bar positions of the cycle ergometer were adjusted to resemble each subject's own bike, and subjects warmed-up at a self-selected pace for 5 min. The incremental test commenced at an initial workload of 100W; workload thereafter increased by 5 W every 10 sec (30 W · min$^{-1}$) until volitional fatigue. PPO was defined as the final completed work rate (e.g., maintained for 10 s). Expired air was continually analyzed for $F_\text{eO}_2$ and $F_\text{eCO}_2$ during exercise using Ametek gas analyzers (SOV S-3A11 and COV CD3A, Pittsburgh, PA). Minute ventilation ($V_e$) was recorded every 30 s using a turbine ventilometer (Morgan, Model 096, Kent, England). The gas analyzers were calibrated immediately before and after each test using a certified beta gas mixture (Commonwealth Industrial Gas Ltd., Brisbane, Australia); the ventilometer was calibrated pre- and post-exercise using a 1-L syringe in accordance with the manufacturer's instructions. $\dot{V}O_2$ was recorded as the highest $\dot{V}O_2$ value attained during the incremental test. In addition to calculating the subjects' $\dot{V}O_2$, PPO at the end of the incremental test was also recorded (Hawley and Noakes, 1992). Heart rate (HR) was measured using a Polar Sports Tester HR monitor (Polar Electro OY, Kempele, Finland). Volitional fatigue was defined by the following criteria: (a) the oxygen consumption ceased to increase linearly with a rising workload and approached a plateau or dropped slightly the last two values agreeing within ±2 ml·kg$^{-1}$·min$^{-1}$, (b) 90% of age predicted HR$\text{peak}$ was attained, and (c) respiratory exchange ratio (RER) was greater than 1.10.

$VT_1$ and $VT_2$ were measured by two independent reviewers according to methods that have been recently described (Lucia et al., 2000). Reviewers were blinded to each other's findings, and in the event of a disagreement, the opinion of a third investigator was sought. We have recently determined the coefficient of variation in our measures of $\dot{V}O_2$, PPO, $VT_1$, and $VT_2$ taken on 40 highly trained cyclists to be 1.8%, 2.1%, 6.1%, and 4.4%, respectively (P. Lausen, 2001, work in progress).

HIT SESSIONS

Subjects performed four HIT sessions over a 2-wk period. Each subject was weighed pre- and post-exercise wearing only his cycling shorts for the later estimation of fluid losses. For each of the four HIT sessions, subjects were required to complete 20 x 1-min bouts of cycling at their $\dot{V}O_2$ PPO; each bout was separated by 2-min recovery at 50 W. Exercise was performed on the same cycle ergometer as that used for the $\dot{V}O_2$ test. HR was measured at 1-min intervals throughout the HIT trials during both work and recovery stages, while $V_e$, $\dot{V}O_2$, and $\dot{V}CO_2$ were measured each 30 s and values averaged for a 3 min period during HIT bouts number 1, 5, 10, 15, and 20. If subjects could not complete a HIT bout, time performed at PPO was recorded. After the twentieth HIT bout, and another 2 min recovery stage, a final bout was completed at PPO until volitional fatigue (i.e., $T_{\text{lim}}$). Time to fatigue and HR$\text{peak}$ attained during this final bout were recorded. The total amount of work
performed at PPO ($W_{\text{PPO}}$) was defined as the sum of the total exercise time at PPO (i.e., total summed time of HIT bouts + $T_{\text{lim}}$). During all HIT sessions, subjects were permitted to drink water *ad libitum*; the volume of water consumed was recorded.

**STATISTICS**

A 2 x 2 (Group x Time) repeated measures analysis of variance (ANOVA) examined changes in the dependant measures over time between the experimental and control groups. For the experimental group, a 4 x 5 (Trial x Time) repeated measures ANOVA analyzed changes in the dependant measures during the HIT sessions. When a significant difference was revealed, Tukey’s *post hoc* test was used to specify where the difference occurred. Pearson’s product moment was used to establish relationships between variables. All statistics were run on SPSS 10.0 for Windows and the alpha level was set at .05. Results are expressed as $\bar{x} \pm SD$.

**Results**

**PRE AND POST $\dot{V}O_2\text{peak}$ MEASUREMENTS**

Table 2 shows data comparing the change in progressive exercise test variables. HIT resulted in a significantly greater increase in PPO compared to controls (+4.3% vs. -0.4%; $P < .05$), despite no change in $\dot{V}O_2\text{peak}$ for either group. Table 3 depicts the $T_{\text{rest}}$ data calculated from the $\dot{V}O_2\text{peak}$ tests. HIT resulted in a significantly greater increase in VT$_1$ (+22% vs. -3%) and VT$_2$ (+15% vs. -1%) as compared to the control group (both $P < 0.05$). As well, the power output corresponding to VT$_1$ (VT$_1\text{PO}$) and VT$_2$ (VT$_2\text{PO}$) were both significantly greater ($P < .05$) for the HIT group (+5.1 and +7.6%) as compared to the control group (+0.4% and -6.6%; Table 3). There were no significant changes in the HR corresponding to VT$_1$ and VT$_2$ for either group.

**HIT SESSIONS**

There were no significant differences throughout the HIT sessions in the volume of water consumed ($\bar{x} = +587 \pm 66$ ml) or change in body mass ($\bar{x} = -.3 \pm 0.1$ kg). However, there were significant increases throughout HIT sessions in $T_{\text{lim}}$ (+75.8%), $W_{\text{PPO}}$ (+12.4%), and total number of trials completed (+25.9%) (Figure 1; all $P < .05$). No significant differences in the peak HR ($P = 0.067$), $\dot{V}E$, or $\dot{V}CO_2$ obtained throughout the four HIT sessions were found. However, from the HIT session #1 to #4, $\dot{V}O_2$ was significantly increased and RER was significantly reduced at bouts #15 and #20 ($P < 0.05$). While there was no significant difference in both peak HR and 2-min recovery HR between HIT sessions, 1-min recovery HR readings were significantly reduced over the HIT sessions during the initial seven exercise bouts (Figure 2; $P < .05$).

**CORRELATIONS**

$\Delta W_{\text{PPO}}$ was significantly related to $\Delta \dot{V}O_2$ from Session #1 to #4 ($r = 0.804; P = .054$), while $\Delta T_{\text{lim}}$ was highly related to $\Delta \text{RER}$ ($r = -.942; P < .01$). Also, both
Table 2  $\bar{x} \pm SD$ Values for Pre- and Post-Data and the Percent Change ($\Delta$) in the Variable Derived From the Progressive Exercise Tests in the High-Intensity Interval Training Group (HIT) and Controls (CON)

<table>
<thead>
<tr>
<th></th>
<th>$\bar{V}O_{2\text{peak}}$ (ml · kg$^{-1}$ · min$^{-1}$)</th>
<th>$\bar{V}O_{2\text{peak}}$ (L · min$^{-1}$)</th>
<th>PPO (W)</th>
<th>HR$_{\text{peak}}$ (beats · min$^{-1}$)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>PRE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HIT</td>
<td>68.7 ± 3.6</td>
<td>4.90 ± 0.43</td>
<td>469 ± 38</td>
<td>193 ± 6</td>
</tr>
<tr>
<td>CON</td>
<td>66.3 ± 3.7</td>
<td>4.77 ± 0.44</td>
<td>490 ± 47</td>
<td>197 ± 12</td>
</tr>
<tr>
<td>POST</td>
<td>70.3 ± 5.7</td>
<td>5.02 ± 0.58</td>
<td>489 ± 45*</td>
<td>193 ± 4</td>
</tr>
<tr>
<td>CON</td>
<td>65.8 ± 4.3</td>
<td>4.72 ± 0.49</td>
<td>488 ± 45</td>
<td>196 ± 10</td>
</tr>
<tr>
<td>$\Delta$ (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HIT</td>
<td>3 ± 3%</td>
<td>2 ± 3%</td>
<td>4 ± 3%*</td>
<td>0 ± 2%</td>
</tr>
<tr>
<td>CON</td>
<td>-1 ± 3%</td>
<td>-1 ± 4%</td>
<td>0 ± 2%</td>
<td>-1 ± 2%</td>
</tr>
</tbody>
</table>

*Change in variable significantly greater in HIT versus CON ($P < .05$). Peak oxygen consumption ($\bar{V}O_{2\text{peak}}$), peak power output (PPO), and peak heart rate (HR$_{\text{peak}}$) are shown.

Table 3  $\bar{x} \pm SD$ Values and Percent Change ($\Delta$) for Pre- and Post-Ventilatory Threshold Data for the High-Intensity Interval Training (HIT) and Control (CON) Groups

<table>
<thead>
<tr>
<th></th>
<th>VT$_1$ (L · min$^{-1}$)</th>
<th>VT$_1$ PO (W)</th>
<th>VT$_1$ HR (beats · min$^{-1}$)</th>
<th>VT$_2$ (L · min$^{-1}$)</th>
<th>VT$_2$ PO (W)</th>
<th>VT$_2$ HR (beats · min$^{-1}$)</th>
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<tr>
<td></td>
<td>PRE</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HIT</td>
<td>2.31 ± 0.44</td>
<td>256 ± 30</td>
<td>138 ± 12</td>
<td>3.33 ± 0.44</td>
<td>340 ± 35</td>
<td>167 ± 11</td>
</tr>
<tr>
<td>CON</td>
<td>2.92 ± 0.41</td>
<td>263 ± 44</td>
<td>152 ± 14</td>
<td>3.70 ± 0.42</td>
<td>361 ± 17</td>
<td>174 ± 18</td>
</tr>
<tr>
<td>POST</td>
<td>2.81 ± 0.51*</td>
<td>269 ± 40*</td>
<td>142 ± 15</td>
<td>3.84 ± 0.63*</td>
<td>366 ± 44*</td>
<td>172 ± 10</td>
</tr>
<tr>
<td>CON</td>
<td>2.82 ± 0.39</td>
<td>264 ± 44</td>
<td>148 ± 19</td>
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<td>359 ± 21</td>
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<td>$\Delta$ (%)</td>
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<tr>
<td>HIT</td>
<td>23 ± 8%*</td>
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<td>3 ± 4%</td>
<td>15 ± 8%*</td>
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<td>-1 ± 3</td>
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*Change in variable significantly greater in HIT versus CON ($P < .05$). The first and second ventilatory thresholds (VT$_1$, VT$_2$), the power output corresponding to VT$_1$ and VT$_2$ (VT$_1$ PO, VT$_2$ PO), and the heart rate corresponding to VT$_1$ and VT$_2$ (VT$_1$ HR, VT$_2$ HR) are shown.
Figure 1. Time to fatigue ($T_{fat}$) and total number of high intensity interval training (HIT) bouts ($\#$ Bouts) completed during HIT Sessions 1–4.

* = Session #4 significantly greater than Session #1 ($P < 0.05$).

Figure 2. One-minute recovery heart rates (HR) during intervals in Session #1 and #4.

* = One-minute recovery HR significantly greater for Session #1 than for Session #4 ($P < 0.05$).
pre- and post-PPO were strongly related to their respective \( \text{VO}_{2\text{peak}} \) value in \( \text{L min}^{-1} \) \((r = 0.713 \text{ to } 0.916; \ P < .05)\). As well, \( \Delta\text{VT}_2 \) was strongly related to \( \Delta\text{PPO} \) \((r = 0.830; \ P < .05)\) in the HIT group.

**Discussion**

The first relevant finding of the present study was that when a group of already highly trained cyclists \((\text{VO}_{2\text{peak}} = 67.5 \pm 3.7 \text{ ml kg}^{-1} \cdot \text{min}^{-1})\) had brief HIT imposed on their normal moderate intensity base training, there was a small, yet significant improvement in PPO (+4.3%), as well as a more marked increase in \( \text{VT}_1 \) (+22%) and \( \text{VT}_2 \) (+15%) measured during the \( \text{VO}_{2\text{peak}} \) test (Tables 2 and 3; \( P < .05)\). A 19–27% increase in \( T_{\text{vent}} \) has been reported in recreationally active subjects after 7–8 wk HIT (Burke et al., 1994; Poole and Gaesser, 1985), but an increase has not been found in long-distance runners after 8 wk HIT (90-95% HR\(_{\text{max}}\), 3 d wk\(^{-1}\), 8 wk), despite improvements in both 10-km run performance and exercise time to fatigue at a supramaximal running speed (Acevedo and Goldfarb, 1989). In a heterogenous group of moderately-trained cyclists, Norris and Petersen (1998) reported increases in \( \text{VO}_{2\text{peak}} \), \( T_{\text{vent}} \), and 40-km time-trial performance after 8 wk training at \( T_{\text{vent}} \) HR. In highly trained cyclists, six HIT sessions over three weeks have shown significant improvements in 40-km time-trial performance, PPO, and time to fatigue at 150% PPO \((P < .05); \text{Table 1}; \text{Lindsay et al., 1996})\). However, \( T_{\text{vent}} \) and \( \text{VO}_{2\text{peak}} \) were not reported (Lindsay et al., 1996). Thus, despite the vast number of studies having shown strong relationships between \( T_{\text{vent}} \) and endurance performance (Laursen and Rhodes, 2001; Coyle et al., 1988; Rhodes and McKenzie, 1984), the present data are the first to demonstrate increases in \( T_{\text{vent}} \) coupled with improvements in PPO, \( W_{\text{PPO}} \), and \( T_{\text{lim}} \) in highly trained athletes.

The improvement in PPO demonstrated in the present study is important since others have found PPO is strongly related to 40-km time trial performance (Stepto et al., 1998; Westgarth-Taylor et al., 1997; Weston et al., 1997). In one study (Lindsay et al., 1996) involving eight endurance trained cyclists (Table 1), there was no change in PPO after 2 wk of HIT \((P = .08)\), but there was an increase after 4 wk (+4.3%; \( P = .01)\). The present changes in PPO (i.e., significant improvement after 2 wk) might be related to differences in intensity, duration, and recovery in the HIT session employed. Table 1 presents a summary of HIT studies in endurance-trained cyclists. While most HIT programs have been shown to elicit significant increases in PPO and endurance performance, it could be argued that a combination of high intensity work bouts, coupled with shorter recovery durations (therefore requiring a greater high-energy phosphate contribution; Hargreaves et al., 1998), collectively might bring about greater improvements in PPO and endurance performance (Harmer et al., 2000; MacDougall et al., 1998; Tabata et al., 1996). Thus, the present study, utilizing a maximal aerobic power HIT program, with 2-min recovery bouts, may have been a more taxing HIT program than that used in previous research in highly trained athletes (Table 1). Indeed, the HIT sessions were completed at near maximal efforts, represented by the fact that HR averaged 92 ± 1% of HR\(_{\text{peak}}\) and mean \( \text{VO}_2 \) consumption was 87 ± 3% of \( \text{VO}_{2\text{peak}} \) at peak measurement points during the HIT bouts. This may be one reason for the greater rate of improvement in PPO (i.e., significant improvement in PPO after only 2 wk; \( P < .05), \text{and } T_{\text{vent}} \) observed in the present study. While a number of
variables (i.e., recovery between exercise bouts, exercise intensity, etc.; see Table 1) may be responsible for the disparity in the time course of performance and physiological changes in response to HIT, it is important to note that highly trained athletes can gain improvements in PPO in as little as four HIT sessions over two weeks during the off-season.

Improvement in $W_{ppo}$ was significantly related to the increase in $\dot{V}O_2$ from HIT Session #1 to #4 ($r = .804; P = .054$), while $\Delta T_{lim}$ was highly related to the mean $\Delta RER$ from Session #1 to #4 ($r = -.942; P < .01$). These findings imply that the improvements in $T_{vent}$ and PPO may be related to alterations in substrate utilization. Most recently, MacDougall et al. (1998) found in untrained subjects that supramaximal HIT (4 x 30s all-out, 4 min recovery) significantly improved both muscle glycolytic and oxidative enzyme activity, as well as exercise performance and $\dot{V}O_2peak$. This, coupled with the findings of Westgarth-Taylor et al. (1997), who noted an increase in fat oxidation at submaximal workrates after 12 HIT sessions in highly trained cyclists (Table 1), are consistent with the reduction of RER and increase in $\dot{V}O_2$ found in the present study. In contrast to the work of these authors (MacDougall et al., 1998; Westgarth-Taylor et al., 1997), Weston et al. (1997) found no significant increase in oxidative enzyme activity after 4 wk of HIT (Table 1) in six endurance-trained cyclists. These workers did, however, find a significant increase in skeletal muscle buffering capacity, as well as a strong relationship ($r = -.82; P < .05$) between skeletal muscle buffering capacity and 40-km time trial performance (Weston et al., 1997). There was also a stated “trend” ($P$ value not reported) in the relationship between the change in skeletal muscle buffering capacity and the change in 40-km time trial performance, suggesting that improvements in performance in response to HIT could be related to an increased ability to buffer $H^+$ ions (Weston et al., 1997). Hence, the mechanisms responsible for the improved performances following HIT in already highly trained athletes remain unclear.

There were significant differences found in 1-min recovery HR data over the first seven HIT bouts between Session #1 and Session #4 (Figure 2; $P < .05$). The change in recovery HR could not be attributable to differences in fluid consumption or sweating rate, as fluid consumption and body weight loss were unaltered throughout the HIT sessions. Instead, this finding may reflect a plasma volume expansion following HIT, similar to that which has been observed in untrained subjects following acute submaximal training (Green et al., 1990) and HIT (Green et al., 1987; Hargreaves et al., 1998).

Certainly, the inherent mechanisms responsible for the improved performances following HIT in already highly trained athletes requires further investigation. An acute expansion of the plasma volume has been shown to be an important central (cardiovascular) adaptation to the improved physical work capacity found in previously untrained subjects following endurance training (Green et al., 1987, 1990, 1991). Our findings of a reduced recovery HR following HIT supports this mechanism in already highly trained athletes, but future examination of this premise using more direct measures is required. Peripheral adaptations, such as a concurrent up-regulation of aerobic and anaerobic respiration, appear to be responsible for the improvement in endurance performance and $\dot{V}O_2peak$ following HIT in previously untrained subjects (Harmer et al., 2000; MacDougall et al., 1998; Tabata et al., 1996). However, findings in highly trained athletes do not confirm this
assumption, and instead support an enhanced ability to buffer H+ ions (Weston et al., 1997). Our finding of an increase in the mean VO2 during the 15th and 20th HIT bouts support an enhanced capacity for oxidative phosphorylation. It should be emphasized, however, that 66% of this VO2 value was measured during the recovery phase of the HIT sessions. An enhanced capacity for aerobic respiration could contribute substantially during the recovery phase for the replenishment of phosphocreatine stores, and to aid in the removal of accumulated lactate (Linossier et al., 1993). One other possible mechanism that deserves future attention is whether the expression of Na+-K+-ATPase pumps, which is instrumental in maintaining muscle membrane potential (Green, 1998), are altered following HIT in the already highly trained athlete. Indeed, adjustments have been shown following exercise training in previously untrained subjects (Green et al., 1999a, 1999b) and well-trained mountain climbers following prolonged physical work at high-altitude (Green et al., 2000a, 2000b; MacDonald et al., 2001).

In summary, the present study has shown significant increases in Tvent and PPO as a result of four HIT sessions over two weeks in already highly trained athletes. The mechanisms responsible for these improvements require further research.

References


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