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Metabolic and performance adaptations to interval training in endurance-trained cyclists

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Abstract This study examined the effects of sustained high-intensity interval training (HIT) on the athletic performances and fuel utilisation of eight male endurance-trained cyclists. Before HIT, each subject undertook three baseline peak power output \dot{W}_{peak} tests and two simulated 40-km time-trial cycling performance (TT₄₀) tests, of which the variabilities were 1.5 (1.3)% and 1.0 (0.5)%, respectively [mean (SD)]. Over 6 weeks, the cyclists then replaced 15 (2)% of their 300 (66) km · week⁻¹ endurance training with 12 HIT sessions, each consisting of six to nine 5-min rides at 80% of \dot{W}_{peak} , separated by a 1-min recovery. HIT increased \dot{W}_{peak} from 404 (40) to 424 (53) W ($P < 0.01$) and improved TT₄₀ speeds from 42.0 (3.6) to 43.0 (4.2) km · h⁻¹ ($P < 0.05$). Faster TT₄₀ performances were due to increases in both the absolute work rates from 291 (43) to 327 (51) W ($P < 0.05$) and the relative work rates from 72.6 (5.3)% of pre-HIT \dot{W}_{peak} to 78.1 (2.8)% of post-HIT \dot{W}_{peak} ($P < 0.05$). HIT decreased carbohydrate (CHO) oxidation, plasma lactate concentration and ventilation when the cyclists rode at the same absolute work rates of 60, 70 and 80% of pre-HIT \dot{W}_{peak} ($P < 0.05$), but not when they exercised at the same relative (% post-HIT \dot{W}_{peak}) work rates. Thus, the ability of the cyclists to sustain higher percentages of \dot{W}_{peak} in TT₄₀ performances after HIT was not due to lower rates of CHO oxidation. Higher relative work rates in the TT₄₀ rides following HIT increased the estimated rates of CHO oxidation from ≈ 4.3 to ≈ 5.1 g · min⁻¹.

Key words Exercise · Cycling · Performance · Carbohydrate · Ventilation

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Introduction

Acevedo and Goldfarb (1989) were perhaps the first to show that increased training intensity improved athletic performance in previously trained runners. We have found that when eight well-trained cyclists replaced a portion [15 (2)%] of their approximately 300 km · week⁻¹ base endurance training with sustained high-intensity interval training (HIT), they improved their laboratory measures of peak sustained power output (\dot{W}_{peak}), exercise time to fatigue at 150% of \dot{W}_{peak} and 40-km time-trial performances TT₄₀ (Lindsay et al. 1996). That study showed that only six HIT sessions (six to eight repetitions of 5 min duration at 80% of \dot{W}_{peak}) completed during a 4-week period was sufficient training stimulus to improve significantly the athletic performances of highly trained cyclists in laboratory tests ranging in duration from ≈ 60 s to ≈ 1 h. The improvements in TT₄₀ performances were due to an increase in both the absolute and the relative power outputs that the cyclists could sustain after the HIT programme.

Conventional studies of the effects of endurance training in previously untrained subjects have shown that improvements in maximal performance are associated with lower rates of carbohydrate oxidation (CHO_{ox}) at the same absolute exercise intensities after endurance training (Henriksson 1977; Hurley et al. 1986; MacRae et al. 1995). Such decreases in CHO_{ox} have been attributed mainly to the improved respiratory control sensitivity (Dudley et al. 1987) that results from the increases in mitochondrial density occurring in muscles after ≈ 12 weeks of endurance training (Henriksson 1977; Holloszy and Coyle 1984). With lower cytosolic ADP concentrations needed for given rates of oxidative phosphorylation, activation of glycogenolysis by displacements of the creatine kinase and adenylate kinase equilibria may be more closely matched to the demands of the mitochondria for pyruvate⁻ + H⁺. An increased quantity of mitochondria should also decrease

the cytosolic NADH and H^+ concentrations required for given rates of transport of reducing equivalents into the mitochondria. Less NADH + H^+ and pyruvate $^-$ + H^+ accumulation would decrease the cytosolic pyruvate $^-$ + NADH + H^+ \rightleftharpoons lactate $^-$ + NAD $^+$ and trans-sarcolemmal lactate $^-$ _{in} + H^+ _{in} \rightleftharpoons lactate $^-$ _{out} + H^+ _{out} equilibria displacements towards lactate production and H^+ + lactate $^-$ co-efflux at high rates of CHO_{ox} (Dennis et al. 1991).

Hurley et al. (1984) and MacRae et al. (1995) have reported lower rises in circulating lactate concentrations with increasing CHO_{ox} after endurance training. However, despite much lower "arterialised" venous blood lactate concentrations after training, MacRae et al. (1995) found that subjects still fatigued at similar relative rates of CHO_{ox} . Both before and after 9 weeks of endurance training, subjects were unable to complete a final 6-min progressive exercise stage when 82–83% of their energy was from CHO_{ox} .

In contrast, a reduced reliance on CHO_{ox} with increases in mitochondrial density may not explain the improvements in performance after six HIT sessions in already highly trained cyclists, as observed by Lindsay et al. (1996). In that study, skeletal muscle biopsy samples were taken from six of the eight subjects and subsequently analysed for physiochemical H^+ -buffering capacity and several glycolytic and mitochondrial enzyme activities. The results showed that, while HIT significantly increased the acid load required to produce a fall of 1 pH unit from ≈ 50 to 60 mEq H^+ \cdot l^{-1} of muscle water, it had no influence on the muscle glycolytic or oxidative capacities (Weston et al. 1996).

Accordingly, the primary aim of the present investigation was to examine whether HIT influences fuel utilisation at different comparable absolute and relative power outputs in already well-trained subjects. A second purpose of this study was to determine whether any changes in the relative rates of CHO_{ox} and fat oxidation FAT_{ox} might explain the improvements in TT_{40} performances after HIT.

Methods

Subjects

The subjects were eight male endurance-trained competitive cyclists who had not performed any interval training for a minimum of 3 months before this investigation. Each subject gave his written informed consent before participating in the study, which was approved by the Research and Ethics Committee of the Faculty of Medicine of the University of Cape Town.

Peak sustained power output \dot{W}_{peak}

Values of \dot{W}_{peak} were measured during a sustained incremental exercise test to fatigue on an electronically braked cycle ergometer (Lode, Groningen, The Netherlands), as described in detail previously (Hawley and Noakes 1992). Briefly, this test commenced at a work rate of 3.33 W \cdot kg^{-1} body mass for 150 s, after which the work rate was increased first by 50 W, and then 25 W every 150 s

until the subject fatigued. Fatigue coincided with a drop in the pedalling rate from ≈ 90 to < 70 revolutions \cdot min^{-1} . \dot{W}_{peak} was defined as the last completed work rate (in W) plus the fraction of time spent in the final non-completed work rate $\times 25$ W.

Simulated TT_{40}

The day after the incremental exercise test to exhaustion, subjects returned to the laboratory to perform a TT_{40} on their own bicycles which were mounted on a Kingcycle air-braked cycle ergometer (Kingcycle High Wycombe, Buckinghamshire, UK). This ergometry system has been described in detail previously, and has a coefficient of variation of 1.0 (0.5%) for a 40-km cycling time (Palmer et al. 1996). After the Kingcycle had been calibrated, subjects warmed up for 10 min at a self-selected sub-maximal exercise intensity. The exercise intensity and duration were kept constant before each subsequent TT_{40} performance ride. During the performance rides the only feed-back to the cyclists was their elapsed "distance".

Baseline testing

Before the start of the study, each subject performed three \dot{W}_{peak} and two TT_{40} tests to ensure that his cycling performances were stable prior to a HIT intervention. These tests were conducted on separate days and were preceded by 1 day of light training or rest.

Following these tests, and on a separate day, the cyclists performed successive 10-min rides at 50, 60, 70 and 80% of their best pre-HIT \dot{W}_{peak} . During this exercise test, the subjects wore a nose-clip and breathed through a mouthpiece attached to an Oxycon Alpha automated gas analyser (Mijnhardt, The Netherlands). Before each test, the gas analyser was calibrated with a Hans Rudolph 5530 3-l syringe and a 5% CO_2 :95% N_2 gas mixture. Analyser outputs were processed by an IBM computer which calculated $l \cdot min^{-1}$ ventilation (\dot{V}_E), oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) values using conventional equations.

After 3–4 min at each (higher) exercise intensity, \dot{V}_E , $\dot{V}O_2$ and $\dot{V}CO_2$ values were constant, suggesting that any respiratory compensation for metabolic acidosis was negligible compared to the overall $\dot{V}CO_2$. As explained in the Discussion, loss of HCO_3^- to CO_2 was calculated to increase $\dot{V}CO_2$ by $< 2\%$. Accordingly, it was felt that rates CHO_{ox} and FAT_{ox} could be calculated from the equations of Frayn (1983), assuming a non-protein respiratory exchange ratio.

Before the 10-min rides at 50, 60, 70 and 80% of \dot{W}_{peak} , an 18-gauge cannula was placed in a forearm vein and attached to a three-way stop-cock for the sampling of venous blood (3 ml) at rest and at the end of each work rate. Blood samples were collected into ice-cold tubes containing sodium fluoride and potassium ethylenediaminetetra-acetate and centrifuged at $500 \times g$ for 6 min at 4°C. The supernatant was then stored at $-20^\circ C$ for later analyses of plasma lactate and glucose concentrations. Plasma lactate concentrations were determined in duplicate using a DU60 Beckman spectrophotometer (Beckman Instruments, Fullerton, Calif., USA) with a commercially available enzymatic assay (Boehringer Mannheim, Germany). Plasma glucose concentrations were measured in duplicate with an automated LM3 glucose analyser (Analox Instruments, London, UK).

High-intensity interval training (HIT)

HIT training took place, after a self-paced and standardised warm-up, every 3 days on 12 occasions over a 6- to 7-week period. Each HIT session consisted of six to nine 5-min cycling bouts at 80% of the subject's \dot{W}_{peak} ; 80% of \dot{W}_{peak} value corresponds to ≈ 85 –88% of $\dot{V}O_{2peak}$ (Hawley and Noakes 1992). Between each repetition, the subjects either rested completely or cycled at a work rate of ≤ 100 W for 1 min. After every four HIT sessions (≈ 2 weeks,) subjects performed a further \dot{W}_{peak} test and the intensity of the

subsequent four HIT sessions was then adjusted to each subject's new (higher) \dot{W}_{peak} value.

Throughout the study, the cyclists kept a log-book of their training volume. That training volume was used to determine the cyclists' average weekly training distance and the proportion of that distance that was replaced by HIT. Distances covered during the laboratory HIT sessions were estimated using the following formula:

$$\text{km} = \text{power} \times \text{time} \times 0.1 [(\text{km} \cdot \text{h}^{-1}) \cdot \text{W}^{-1}] \quad (1)$$

where power is the power output (in W) during the HIT; time is the number of hours spent performing HIT and 0.1 is the linear $[(\text{km} \cdot \text{h}^{-1}) \cdot \text{W}^{-1}]$ relationship between speed and W in the simulated TT_{40} (described subsequently). The estimated "distances" covered during each interval training session in the laboratory were given to the subjects so that they could reduce their baseline, outdoor cycling distances in order to keep their total, weekly training volume constant.

Post-HIT testing

After the 12 HIT sessions, the subjects repeated the \dot{W}_{peak} and TT_{40} tests and the "steady-state" 10-min rides at 50, 60, 70 and 80% of their best pre-HIT \dot{W}_{peak} . In addition, 2 days later they repeated the rides at 50, 60, 70 and 80% of their higher post-HIT \dot{W}_{peak} so that comparisons could be made at both the same absolute and the same relative work rates before and after HIT.

Statistical analyses

All results are presented as the means (SD) on $n = 8$ subjects. The statistical significance of differences over time or with increasing work rates before and after HIT were assessed with a two-way analysis of variance for repeated measures, followed by a Tukey's post hoc test if a significant F -ratio was observed. Differences in performance before and after HIT were assessed with a paired Student's t -test. A Pearson product moment correlation coefficient (r) was used to examine the linear regressions between baseline \dot{W}_{peak} and TT_{40} cycling speeds, and between the increases in \dot{W}_{peak} and improvements in TT_{40} cycling speeds after HIT. Results were considered significant when P was < 0.05 .

Results

The mean ages, heights and masses of the eight endurance-trained cyclists were 25 (4) years, 190 (10) cm and 80 (13) kg, respectively, and their average weekly (baseline) training distances were 300 (86) $\text{km} \cdot \text{week}^{-1}$. During the HIT programme, the subjects replaced a total of 15 (2)% of their baseline training with HIT.

Table 1 shows the test-to-retest reliability of the measurements of the subjects' baseline \dot{W}_{peak} and simulated TT_{40} performances. The variabilities of the baseline \dot{W}_{peak} and TT_{40} tests were 1.5 (1.3)% and 1.0 (0.5)%, respectively.

As expected, there was a significantly ($r = 0.83$; $P < 0.01$) linear relationship between the pre-HIT (baseline) \dot{W}_{peak} and 40-km cycling speed ($\text{km} \cdot \text{h}^{-1}$). As reported previously by Lindsay et al. (1996), this relationship was described by a $\text{km} \cdot \text{h}^{-1} = (0.01 \pm 0.03) \dot{W}_{\text{peak}}$ equation (data not shown).

Twelve HIT sessions significantly increased both \dot{W}_{peak} and TT_{40} performances (Fig. 1). The \dot{W}_{peak} values

Table 1 The variability of the baseline measurements of peak power output \dot{W}_{peak} and simulated 40-km time-trial performance (TT_{40}). Results are the mean (SD) of three \dot{W}_{peak} and two TT_{40} measurements from eight subjects. (%V Percent variability)

\dot{W}_{peak} (W)	\dot{W}_{peak} (%V)	TT_{40} (min)	TT_{40} (%V)
404 (40)	1.5 (1.3)	57.2 (4.9)	1.0 (0.5)

increased from 404 (40) W to 419 (48), 421 (46) and 424 (53) W after 4, 8 and 12 HIT sessions, respectively ($P < 0.01$), and average TT_{40} cycling speeds increased from 42.0 (3.6) $\text{km} \cdot \text{h}^{-1}$ to 43.0 (4.2) $\text{km} \cdot \text{h}^{-1}$ after 12 HIT sessions ($P < 0.05$). The marked (≈ 1.5 min) improvements in TT_{40} performances from 57.2 (4.9) min to 55.8 (5.4) min after HIT were associated with the subjects being able to sustain significantly higher absolute and relative work rates during the time-trials (Fig. 2). HIT increased the absolute work rate during the time-trials from 291 (43) W to 327 (51) W ($P < 0.05$), and the relative work rates from 72.6 (5.3)% of pre-HIT (baseline) \dot{W}_{peak} to 78.1 (2.8)% of post-HIT \dot{W}_{peak} ($P < 0.05$). Although \dot{W}_{peak} was related to TT_{40} cycling speeds both before and after HIT, there was no significant correlation in the cyclists' increase in PPO and their improvement in TT_{40} performance (data not shown).

Figure 3 shows that HIT decreased the rate of CHO_{ox} during the 10-min cycling bouts at work rates of 60, 70 and 80% of pre-HIT \dot{W}_{peak} . Before HIT, the rates of CHO_{ox} were 3.2 (0.5), 4.3 (0.6) and 5.4 (0.8) $\text{g} \cdot \text{min}^{-1}$ and, after HIT, they were reduced to 2.9 (0.4), 3.6 (0.4), 4.8 (0.5) $\text{g} \cdot \text{min}^{-1}$, respectively ($P < 0.05$). In contrast, the rates of CHO_{ox} were similar before and after HIT when the subjects exercised at 50, 60, 70 and 80% of their post-HIT ($\approx 5\%$ higher) \dot{W}_{peak} (Fig. 3). Reductions in the rates of CHO_{ox} at the same absolute, but not at the same

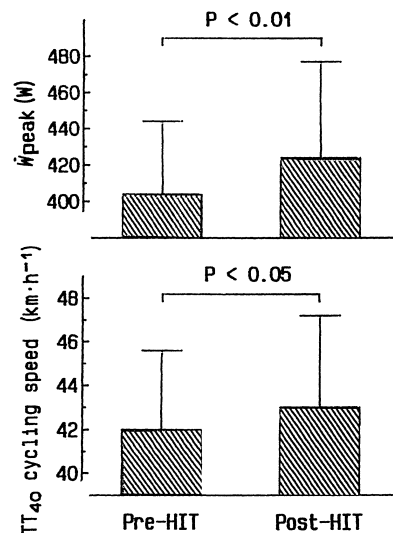


Fig. 1 Effects of sustained high-intensity interval training (HIT) on peak power outputs \dot{W}_{peak} and 40-km time-trial (TT_{40}) cycling speeds. Values are means (SD) ($n = 8$)

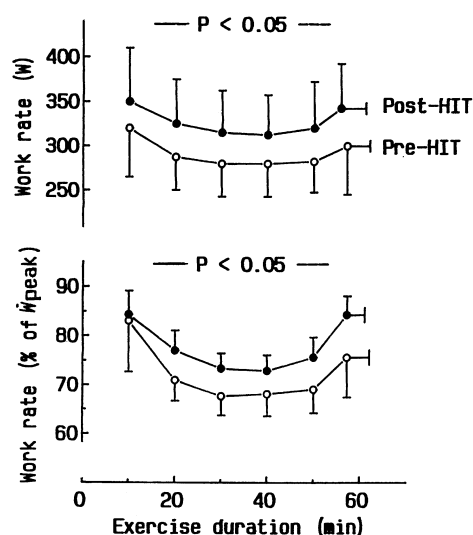


Fig. 2 Effects of HIT on the absolute work rates and percentages of \dot{W}_{peak} during the simulated TT₄₀. Values are means (SD) ($n = 8$)

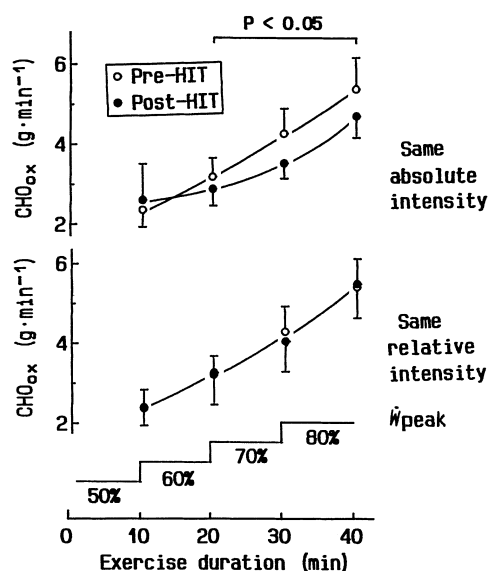


Fig. 3 Effects of HIT on rises in carbohydrate oxidation (CHO_{ox}) at increasing percentages of \dot{W}_{peak} . Values are means SD ($n = 8$). Post-HIT exercises at the same absolute and relative intensities were performed at percentages of pre-HIT and post-HIT \dot{W}_{peak} respectively. Post-HIT \dot{W}_{peak} values were $\approx 5\%$ higher than pre-HIT \dot{W}_{peak} values (Fig. 1)

relative, exercise intensities were associated with increased rates of FAT_{ox} at work rates of 60, 70 and 80% of pre-HIT \dot{W}_{peak} (Table 2). Before HIT, the contributions to energy production from FAT_{ox} at these work rates were 23 (7), 16 (10) and 6 (5)%, respectively, and after HIT they were increased to 29 (3), 26 (7) and 13 (7)%, respectively ($P < 0.05$).

Higher rates of FAT_{ox} and lower rates of CHO_{ox} at the same absolute work rates after HIT were not associated with any measurable differences in plasma glucose concentrations (data not shown). Both before and after

Table 2 Effect of sustained high-intensity interval training (HIT) on fat oxidation (FAT_{ox}) during exercise at either the same absolute intensities or the same relative intensities. Results are the means (SD) from eight subjects

	% \dot{W}_{peak}^a		
	Pre-HIT	Post-HIT	
		Absolute ^a	Relative ^a
	FAT_{ox} ($\text{g} \cdot \text{min}^{-1}$)		
50	0.44 (0.10)	0.49 (0.10)	0.48 (0.10)
60	0.39 (0.12)	0.50 (0.05)*	0.56 (0.13)
70	0.33 (0.21)	0.53 (0.21)*	0.40 (0.15)
80	0.15 (0.14)	0.30 (0.14)*	0.16 (0.15)
	Energy from FAT_{ox} (% mJ)		
50	29 (7)	32 (7)	32 (7)
60	23 (7)	29 (3)*	29 (7)
70	16 (10)	26 (7)*	19 (7)
80	6 (5)	13 (7)*	6 (5)

^a Post-HIT exercises at the same absolute and at the same relative intensities are described in the text and in the legend to Fig. 3.

* Indicates a significant ($P < 0.05$) effect of HIT

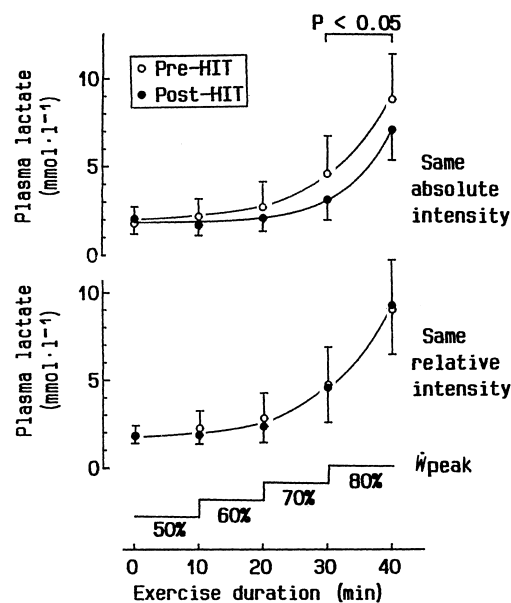


Fig. 4 Effects of HIT on rises in plasma lactate concentration at increasing percentages of \dot{W}_{peak} . Values are means (SD) ($n = 8$). Data are from the studies described in the legend to Fig. 3

HIT, mean plasma glucose concentrations rose from $\approx 4.5 \text{ mmol} \cdot \text{l}^{-1}$ at 50% of pre- and post-HIT \dot{W}_{peak} to $\approx 5.5 \text{ mmol} \cdot \text{l}^{-1}$ at 80% of pre- and post-HIT \dot{W}_{peak} . In contrast, the rises in plasma lactate concentrations during the rides followed a similar pattern to that of CHO_{ox} (Fig. 4). Although HIT did not influence plasma lactate concentrations when the cyclists rode at 50, 60, 70 and 80% of their higher post-HIT \dot{W}_{peak} (i.e. at the same relative exercise intensity), it decreased plasma lactate concentrations at work rates of 70 and 80% of pre-HIT \dot{W}_{peak} . At these same absolute work rates,

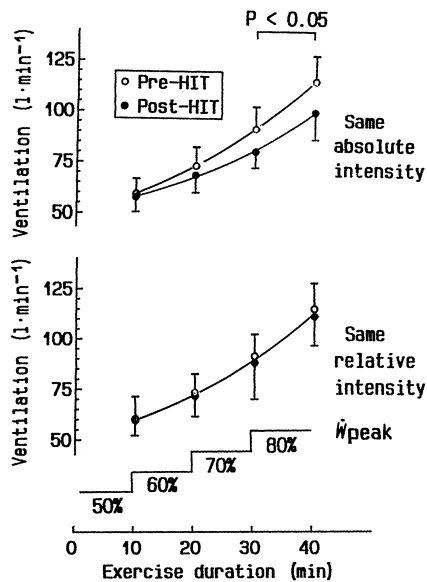


Fig. 5 Effects of sustained HIT on rises in ventilation (\dot{V}_E) at increasing percentages of \dot{W}_{peak} . Values are means (SD) ($n = 8$). Results are from the studies described in Fig. 3

plasma lactate concentrations were reduced from 4.7 (2.1) and 9.0 (2.5) $\text{mmol}\cdot\text{l}^{-1}$ to 3.3 (1.1) and 7.3 (1.7) $\text{mmol}\cdot\text{l}^{-1}$, respectively ($P < 0.05$).

HIT also decreased \dot{V}_E during the same absolute exercise intensities (Fig. 5). At 70% of pre-HIT \dot{W}_{peak} , \dot{V}_E was reduced from 92 (11) $\text{l}\cdot\text{min}^{-1}$ to 81 (8) $\text{l}\cdot\text{min}^{-1}$ ($P < 0.05$) and, at 80% of pre-HIT \dot{W}_{peak} , \dot{V}_E was reduced from 116 (13) $\text{l}\cdot\text{min}^{-1}$ to 100 (13) $\text{l}\cdot\text{min}^{-1}$ ($P < 0.05$). In contrast, there were no significant differences in \dot{V}_E values when the subjects rode at the same relative exercise intensities after HIT.

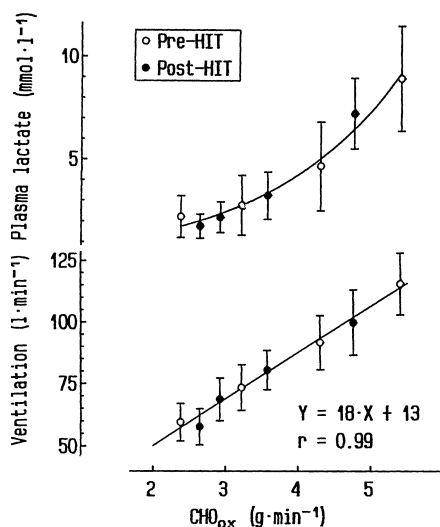


Fig. 6 Rises in plasma lactate concentration and \dot{V}_E with increasing rates of CHO_{ox} before and after HIT. Values are means (SD) ($n = 8$). Lactate concentration and \dot{V}_E data are from Figs. 4 and 5, respectively, and the CHO_{ox} data are from Fig. 3

Lower plasma lactate concentrations and \dot{V}_E values at the same absolute exercise intensities after HIT were associated with a reduced reliance on CHO_{ox} at those work rates. The curvilinear relationship between plasma lactate concentration and CHO_{ox} , and the linear relationship between \dot{V}_E and CHO_{ox} were similar before and after HIT (Fig. 6). Both before and after training \dot{V}_E rose by 18.2 (0.9) $\text{l}\cdot\text{min}^{-1}$ with every 1.0 $\text{g}\cdot\text{min}^{-1}$ increase in CHO_{ox} , irrespective of the changing rates of plasma lactate accumulation.

Discussion

The first finding from this study was that 12 sustained HIT sessions, undertaken over a 6-week period, significantly improved the \dot{W}_{peak} and simulated TT_{40} performances (Fig. 1) of well-trained endurance cyclists whose performances were stable prior to HIT (Table 1). Whereas the pre-HIT \dot{W}_{peak} and TT_{40} tests varied by ≈ 6 W and ≈ 30 s, respectively, the post-HIT \dot{W}_{peak} and TT_{40} tests improved by ≈ 20 W and ≈ 90 s, respectively. These improvements in exercise performance are similar to the ≈ 18 -W increases in \dot{W}_{peak} and the ≈ 120 -s decreases in TT_{40} cycling times observed to occur in highly trained cyclists after six HIT sessions over 4 weeks by Lindsay et al. (1996).

In agreement with the findings of Lindsay et al. (1996), there were close relationships between the cyclists' pre- and post-HIT \dot{W}_{peak} values and their TT_{40} performances. However, although \dot{W}_{peak} accounted for 70–90% of the variation in time-trial performances (Hawley and Noakes 1992; Lindsay et al. 1996), there was no significant relationship in this study between the $\approx 5\%$ increase in the cyclists' \dot{W}_{peak} values and their $\approx 2.5\%$ improvements in TT_{40} cycling performances. One possible reason for the poor correlation between increases in \dot{W}_{peak} and improvements in TT_{40} is that HIT not only increased the absolute work rates during the TT_{40} but also the relative work rates (Fig. 2). These data indicate that TT_{40} cycling performances are determined by a combination of the cyclists' absolute \dot{W}_{peak} , and their ability to sustain a high percentage of that \dot{W}_{peak} during endurance exercise, as has also been noted by Coyle et al. (1991).

Part of the higher work rates in the TT_{40} rides after HIT may have been associated with a decreased reliance on CHO_{ox} . In the sub-maximal rides at 60, 70 and 80% of pre-HIT \dot{W}_{peak} , rates of CHO_{ox} were decreased (Fig. 3) and rates of FAT_{ox} were increased (Table 2). Less energy from CHO_{ox} at the same absolute exercise intensities has been commonly observed after ≈ 9 –12 weeks of endurance training in previously less fit subjects (Henriksson 1977; Hurley et al. 1986; MacRae et al. 1992; 1995). As noted earlier, lower rates of CHO_{ox} at the same absolute work rates after endurance training in previously sedentary subjects have been attributed mainly to the improved respiratory control sensitivity (Dudley et al. 1987) that results from an increased

muscle mitochondrial density (Henriksson 1977; Holloszy and Coyle 1984). In the present studies, however, the lower rates of CHO_{ox} at the same absolute work rates after HIT in already well-trained subjects (Fig. 3) were unlikely to have been due to a measurable increase in the mitochondrial content of the working muscles. Previously, we found that six HIT sessions had no influence on the oxidative capacity of working muscle samples taken from already well-trained endurance cyclists (Weston et al. 1966). Instead, the decreases in CHO_{ox} at the same absolute exercise intensities after HIT appeared to be the result of the cyclists exercising at lower relative exercise intensities following training. When the subjects exercised at 50, 60, 70 and 80% of their $\approx 5\%$ higher post-HIT \dot{W}_{peak} , the rates of CHO_{ox} and FAT_{ox} were similar before and after HIT (Fig. 3; Table 2).

Rises in plasma lactate concentrations (Fig. 4) and \dot{V}_{E} (Fig. 5) during the sub-maximal rides at 50, 60, 70 and 80% of pre- and post-HIT \dot{W}_{peak} followed a similar pattern to that of CHO_{ox} . Lower venous plasma lactate concentrations and \dot{V}_{E} values at the same absolute, but not at the same relative, exercise intensities after HIT were associated with the reduced reliance on CHO_{ox} at those work rates. When plotted against increasing $\text{g}\cdot\text{min}^{-1}$ rates of CHO_{ox} , rises in venous $\text{mmol}\cdot\text{l}^{-1}$ plasma lactate concentrations and $\text{l}\cdot\text{min}^{-1}$ \dot{V}_{E} values were similar before and after HIT (Fig. 6). On both occasions, \dot{V}_{E} rose as a linear function of CHO_{ox} ($\dot{V}_{\text{E}} = 18\cdot\text{CHO}_{\text{ox}} + 13$), despite curvilinear rises in plasma lactate concentrations (Fig. 6). These data suggest that the respiratory compensation for "lactic acidosis" was negligible compared to the overall $\dot{V}\text{CO}_2$ in this study. Assuming that the maximum, $\approx 4 \text{ mmol}\cdot\text{l}^{-1}$, rise in plasma lactate concentration (Fig. 4) occurred in a non-steady-state distribution volume of $\approx 8 \text{ l}$ (Stanley et al. 1985), the buffering of $\approx 32 \text{ mmol}$ of "lactic acid" by the $\text{H}^+ + \text{HCO}_3^- \rightleftharpoons \text{H}_2\text{O} + \text{CO}_2$ equilibrium over 10 min would have increased the $\approx 4.3 \text{ l}\cdot\text{min}^{-1}$ $\dot{V}\text{CO}_2$ values by $\approx 0.07 \text{ l}\cdot\text{min}^{-1}$, or by $< 2\%$.

The lower \dot{V}_{E} values observed during steady-state exercise after endurance training (Byrne-Quinn et al. 1971; Martin et al. 1979) are therefore unlikely to be due to decreased blood lactate concentrations, as has been proposed by Casaburi et al. (1987). Instead, the decreased steady-state \dot{V}_{E} values at the same absolute work rate after training are more likely to be a result of a reduced relative exercise intensity and a lower rate of CO_2 production from CHO_{ox} (Brooks and Mercier 1994).

Decreases in CHO_{ox} at the same absolute work rates after HIT (Fig. 3) probably helped to improve athletic performances by reducing the accumulation of H^+ and lactate $^-$ (Fig. 4). The fraction of peak $\dot{V}\text{O}_2$ that an athlete can sustain appears to be related to the accumulation of lactate in the blood and, presumably, in the working muscles (Costill et al. 1973; Farrell et al. 1979; LaFontaine et al. 1981). However, a reduction in CHO metabolism did not explain the ability of the cyclists to sustain higher (78% vs 73% of \dot{W}_{peak}) relative work rates

during the TT_{40} performance rides after HIT (Fig. 2). Rates of CHO_{ox} and plasma lactate accumulation were similar at the same relative exercise intensities before and after HIT (Fig. 3 and 4). From these data, we estimated that the higher relative work rates observed during the TT_{40} rides after HIT would have increased the rates of CHO_{ox} from ≈ 4.3 to $5.1 \text{ g}\cdot\text{min}^{-1}$, and the plasma lactate concentrations from ≈ 5.1 to $7.5 \text{ mmol}\cdot\text{l}^{-1}$. Thus, HIT appeared to improve the ability of cyclists to sustain higher work rates through mechanism(s) that are unrelated to rates of CHO_{ox} .

Improvements in performance after HIT may occur through an additional recruitment of the motor units needed to sustain exercise at, or close to, race pace. Increases in strength in the first 2–8 weeks weight training are thought to be due largely to increased neuromuscular activation (for review see Kraemer et al. 1996), but whether that is the case with HIT remains to be determined.

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