Carbohydrate Dependence During Hard-Intensity Exercise in Trained Cyclists in the Competitive Season: Importance of Training Status

Abstract

To test the hypothesis that intensive endurance training increases CHO utilisation during hard-intensity exercise, seven competitive road cyclists (Cy) performed three 50-min steady-state exercise tests on a cycle ergometer above their ventilatory threshold (+15%) over the course of a cycling season (January [ET1], May [ET2] and September [ET3]). We compared the data with the baseline values of seven sedentary controls (Sed). CHO oxidation in Cy was higher in ET2 and ET3 than in ET1 (p < 0.05), was lower in ET3 than in ET2 (p < 0.05) and was higher in Cy than in Sed only in ET2 (p < 0.05). Lactate kinematics were lower in Cy than in Sed in all conditions (p < 0.05), but in Cy they were lower in ET2 than in ET1 and higher in ET3 than in ET2 (p < 0.05). Race performance was impaired and the overtraining score was increased at ET3 in comparison with ET2 (p < 0.05). We conclude that competitive cyclists increase CHO oxidation during hard-intensity exercise over the course of a season, but show a decline by the end of the season in association with the appearance of an overtraining state. Thus, well-trained cyclists develop a CHO dependence, which is modified with training status.

Key words

Indirect calorimetry · performance · overtraining · substrate oxidation

Introduction

Endurance training is widely acknowledged to provoke a shift toward lipid dependence during submaximal exercise [6,9,18]. The factors that influence the patterns of substrate utilisation during exercise of different intensities are numerous, however, and controversy continues over the effects of exercise intensity and prior endurance training on these patterns. Some authors have shown that regular exercise decreases carbohydrate (CHO) utilisation and increases lipid utilisation, as indicated by a decreased respiratory exchange ratio (RER) [18,21,24] and a slower rate of muscle glucose breakdown [23] during moderate-intensity exercise. Others have shown a higher ability to utilise glucose [29,30] and a CHO preference during hard-intensity exercise [31,36,40]. Training thus appears to increase the ability to oxidise both lipids and CHO. The pattern of substrate utilisation in an individual at any point in time depends on the crossover between the exercise intensity-induced responses, which increase CHO utilisation, and the endurance training-induced responses, which promote lipid oxidation [6]. Although the effects of endurance training on substrate oxidation during exercise have been widely reported in the current literature, few studies have investigated these effects during hard-intensity exercise [10,36,40]. We recently reported that endurance training results in an increase in CHO oxidation during hard-intensity exercise in trained cyclists [31]. Most data come from cross-sectional studies, however, and no study has yet clearly elucidated the metabolic adaptations in substrate oxidation. Decreased CHO utilisation, expressed by a reduction in RER, has been evidenced during overtraining in endurance athletes [43]. Further, Petitbois et al. recently showed

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Accepted after revision: February 10, 2002

Bibliography

that carbohydrate is the first substrate involved in the overtraining process [38]. However, the respective influences of training and overtraining on carbohydrate metabolism during prolonged exercise seem to be incompletely understood.

Since most athletes (e.g., cyclists) perform at intensities that elicit >70–75% of maximum aerobic power during training and competition, our cyclists underwent exercise testing at this intensity three times over the course of a cycling season. To our knowledge, substrate oxidation has never before been investigated in such a longitudinal design. We hypothesised a) an increase in CHO oxidation during hard-intensity exercise over the course of a cycling season because of the ongoing intensive training, and b) a relationship between this CHO dependency and the physical performance achieved during cycling races. To this purpose, we examined CHO oxidation using indirect calorimetry [33,37] in seven competitive cyclists in a nine-month longitudinal study covering an entire cycling season. We also compared the data with the baseline values of seven sedentary controls.

Material and Methods

Subjects
Seven competitive male road cyclists and seven age-matched sedentary male controls participated in this study. None were on medication and none had a family history of diabetes or hypertension. Smokers or those currently using medication for the control of blood arterial pressure or lipid or carbohydrate metabolism were excluded. No subject exhibited electrocardiogram abnormalities at rest or during the maximal cycle ergometer test. The physical characteristics of the subjects are shown in Table 1. Body fat was assessed from the sum of four skinfold measurements (biceps, triceps, subscapular and suprailiac) [13]. None of the sedentary subjects participated in competitive sports or in organised leisure time activities.

Bicycle training program
The cyclists performed 14 hours of cycling (i.e., about 450 km) per week during the nine-month training period. This training protocol involved the same race team in the following weekly schedule: on Monday, 45 km (recovery); on Wednesday, 100–140 km (endurance); on Thursday, 30 km (recovery); on Friday, 45 km (interval training; only after the second month); on Saturday, 100–140 km (endurance or competition [beginning in March]); and on Sunday, 80–100 km (endurance or competition [beginning in March]). During the first month, training sessions were performed at low intensity with a specific target (below their ventilatory threshold: VT2). During the other months, they added interval-training sessions to their endurance training, wherein they performed at high intensity with a specific target heart above their VT2. The entire training program was carried out under cardio-tester control to monitor the hear rate and to set it at the target value. See Table 2 for more information.

After receiving a complete and accurate verbal description of the procedure, risks and benefits associated with the study, the subjects provided written consent. The experimental protocol was approved by the Committee on Research for the Medical Sciences.

Protocol
The subjects came to the laboratory on two separate days for (1) an incremental maximal exercise test to determine maximal oxygen uptake (VO2max) and ventilatory threshold (VT) (day 1), and (2) a 50-min steady-state exercise test 15% above their individual VT (day 2). The athletes underwent testing three times, once when training began (January: ET1), once in mid-season (May: ET2), and then at the end of the season (September: ET3). The two different tests were separated by at least four days and never more than seven days. Testing days were chosen with respect to cycling races to avoid excessive fatigue before metabolic exercises. The sedentary controls were tested in January. All subjects were requested to refrain from exercise performance and to take no cola drinks or coffee for the three days before testing. Prior to study enrolment, a brief interview was conducted to ascertain that all subjects had approximately the same dietary habits. For this purpose, we employed a simplified dietary questionnaire with a semi-quantitative evaluation of nutritional habits [28].

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Characteristics and ergonomic parameters of sedentary controls and cyclists during a cycling season</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedentary subjects (control values)</td>
<td>January (ET1)</td>
</tr>
<tr>
<td>Age, yr</td>
<td>24.5 ± 1.3</td>
</tr>
<tr>
<td>Height, cm</td>
<td>176 ± 1.4</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>73.4 ± 2.1</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>17.3 ± 1.2</td>
</tr>
<tr>
<td>VO2max, ml.kg⁻¹.min⁻¹</td>
<td>44.7 ± 1.5</td>
</tr>
<tr>
<td>Wmax, W</td>
<td>289 ± 1.2</td>
</tr>
<tr>
<td>[lact]max, mmol.l⁻¹</td>
<td>8.1 ± 1.2</td>
</tr>
<tr>
<td>HRmax, bpm</td>
<td>188 ± 5.1</td>
</tr>
<tr>
<td>RERmax</td>
<td>1.17 ± 0.02</td>
</tr>
</tbody>
</table>

Values are means ± SE. VO2max, maximal O2 uptake; Wmax, maximum workload; [lact]max, maximal blood lactate; HRmax, heart rate maximal; RERmax, respiratory exchange ratio maximal; VT, ventilatory threshold. Significant difference between cyclists and sedentary subjects, *p < 0.05; significant difference between cyclists before (ET1) and after training (ET2 and/or ET3), #p < 0.05.
Table 2  Detailed information on the cycling training program

<table>
<thead>
<tr>
<th>Season period</th>
<th>Jan</th>
<th>Feb</th>
<th>Mar</th>
<th>Apr</th>
<th>May</th>
<th>Jun</th>
<th>Jul</th>
<th>Aug</th>
<th>Sept</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endurance training (km)</td>
<td>1000</td>
<td>1400</td>
<td>1000</td>
<td>800</td>
<td>600</td>
<td>500</td>
<td>500</td>
<td>500</td>
<td>700</td>
</tr>
<tr>
<td>Interval training (km)</td>
<td>0</td>
<td>120</td>
<td>180</td>
<td>180</td>
<td>160</td>
<td>120</td>
<td>60</td>
<td>60</td>
<td>120</td>
</tr>
<tr>
<td>Active recovery (km)</td>
<td>150</td>
<td>200</td>
<td>240</td>
<td>240</td>
<td>240</td>
<td>240</td>
<td>240</td>
<td>400</td>
<td>240</td>
</tr>
<tr>
<td>Cycling race (km/month)</td>
<td>0</td>
<td>0</td>
<td>680</td>
<td>980</td>
<td>1140</td>
<td>1290</td>
<td>1400</td>
<td>1550</td>
<td>900</td>
</tr>
<tr>
<td>Total km</td>
<td>1170</td>
<td>1720</td>
<td>2100</td>
<td>2200</td>
<td>2140</td>
<td>2150</td>
<td>2360</td>
<td>2510</td>
<td>1960</td>
</tr>
<tr>
<td>Number of competitions</td>
<td>0</td>
<td>0</td>
<td>6</td>
<td>9</td>
<td>10</td>
<td>13</td>
<td>16</td>
<td>17</td>
<td>8</td>
</tr>
<tr>
<td>Number of stage-races</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Training was individually monitored with a cardio-tester. Intensity level for endurance training was below VT2, intensity level for interval training was above VT2 and active recovery level was below VT1.

To evaluate the physical performance of the cyclists over the course of their competitive season, we noted the number of times they ranked in the top ten places during competition, expressed as a percentage of cycling races performed. To complement this data, we also assessed their perceptions regarding their physical fitness and training conditions during the season with a self-administered psychological questionnaire of over-training that included 54 basic items [26], as previously reported [1,4]. Further, a subjective rating of perceived exertion was obtained using the Borg scale from 6 to 20 [3], after 5, 10, 20, 30, 40 and 50 min of exercise during all trials in cyclists.

Incremental maximal exercise test

The subject's VO₂ max was measured during 8–12 min of exercise performed on an electronically-braked cycle ergometer (Orion, France). During exercise, gas exchanges were measured breath-by-breath and averaged every 30 seconds with an automatic gas analyser (CPX analyser, Medical Graphics Corporation, USA). A nose clip and mouthpiece system was used to measure VO₂ and VCO₂. A 3-liter syringe was used to calibrate the volume pneumotachograph using flow rates similar to subject ventilation. The gas analysers were calibrated before each test with standard gases of known concentration using a certified commercial gas preparation. Heart rate was monitored throughout testing. The test started with a 3-min warm-up at 40 W. The workload was increased by steps of 20 W for the sedentary group and 30 W for the trained group every minute until maximal exercise was reached, which was evaluated in terms of maximal heart rate, respiratory exchange ratio (RER) (>1.15) and O₂ consumption (VO₂) stability. The estimation of the ventilatory threshold was performed by analysis of the behaviour of VCO₂ versus VO₂ following the V-slope according to the method of Beaver et al. [5]. We used this criterion with a test-to-retest reliability of r = 0.97 (Pearson product moment correlation analysis). The curves were analysed by two independent observers. If they did not agree, the opinion of a third investigator was included.

For this purpose, the profiles of both the breath-by-breath and 30-s mean values were analysed independently by each investigator and the average of the two independent determinations was taken to be VT. The agreement of VT for 25 exercise tests was as follows: 1) in 14 cases the difference was < 50 ml; 2) in 8 cases, between 51 to 100 ml; and 3) in 3 cases, between 101 and 135 ml.

Constant-load exercise test

Subjects arrived at the laboratory at 8:00 a.m. after a 12-hr overnight fast. At 8:30 a.m., a resting blood sample was drawn for lactate analysis and subjects then performed hard exercise for 50 min on an electronically-braked cycle ergometer (Orion, France). Water was given ad libitum during the tests. During the 50 min of exercise, the subjects were instructed to maintain a pedaling rate of 75 rpm. Ventilation (VE), RER, VO₂ and carbon dioxide production (VCO₂) were measured continuously, as described above. The measuring instruments were calibrated before each test and the necessary environmental adjustments were made. During this period, VO₂ and VCO₂ varied by less than 0.11 × min⁻¹ and VE varied by less than 0.51 × min⁻¹. We assessed test-to-retest reliability for RER and VO₂ by Pearson product-moment correlation analysis (respectively, r = 0.96 and r = 0.97).

Indirect calorimetry and carbohydrate oxidation measurements

The percentages of carbohydrate were calculated with the following equation [33]:

\[
\text{% carbohydrate} = \left( \frac{\text{[RER} - 0.71]}{0.29} \right) 
\times 100
\]

The RER value was the average of every 5-min period throughout the entire 50 min of exercise, as previously published [31].

The rates of substrate oxidation of carbohydrate were calculated from gas exchange measurements according to the table of non-protein respiratory quotients [37]:

\[
\text{carbohydrate} = 4.585 \times \text{VCO}_2 - 3.226 \times \text{VO}_2
\]

with mass expressed in grams per minute and gas flow in litres per minute.

VO₂ and VCO₂ values were the averages of every 5-min period throughout the entire 50 min of exercise, as previously published [31].

Blood lactate analysis

Capillary whole blood samples were drawn from fingertips at rest (t₀) and during the maximal test, at rest (t₀) and 5, 10, 30, 40, 50 min during prolonged exercise, and then after 2, 5, 10 and 15 min of recovery for both exercise tests. Blood samples were
immediately analysed using an enzymatic method [20] by an electro-analyser (Microzym SGI Analyser, Toulouse), as previously reported [34]. Quality control in our laboratory is performed three times a year by the centralised quality control group of the "Société Française de Médecine du Sport (SFMS)". The within-assay coefficient of variation (CV) for lactate as determined by repetitive measurements of the same sample was 5.2% and the between-assay CV was 6.1%. The sensitivity for enzyme (lowest detectable value) was <0.1 mmol × l⁻¹. 

Statistical analysis
The significance of differences between the cyclists (Cy) and the sedentary controls (Sed) during exercise was determined using an analysis of variance (ANOVA) with repeated measures. If the ANOVA indicated significant differences, these were located by a pair-wise multiple comparison procedure (Student-Newman-Keuls). To detect differences between parameters represented by a single measurement, non-parametric tests for unpaired (Mann-Whitney) and paired (Wilcoxon) data were used as appropriate. Significance was defined as p < 0.05. Data are presented separately as mean ± SE.

Results
Physical characteristics, ergometric parameters and VO₂ measurement
The Cy and Sed groups did not differ with respect to age, height, and weight; however, body fat was lower in Cy than in Sed (Table 1, p < 0.01). VO₂max, maximal power (Wmax) and power at VT (WVT) was higher in Cy in all conditions compared with Sed (Table 1, p < 0.01). However, in Cy, Wmax and WVT were higher in ET2 than ET1 (Table 1). When exercise levels were expressed as percentages of VO₂max, these percentages were quite similar for Sed and Cy at the three test periods (ET1, ET2, ET3; 76.2 ± 2.3 for Sed and 73.1 ± 2.7, 71.6 ± 2.4, 73.9 ± 3.1, respectively for Cy).

The RER, VO₂ and heart rate values in all subjects are presented in Fig. 1. VO₂ values during exercise were lower in Sed than in Cy at all periods (p < 0.05). RER values were higher in Cy at ET2 than in all other conditions (p < 0.05).

Carbohydrate oxidation during constant load
During exercise, the percentage of CHO oxidation was higher in Cy at ET2 than at ET1 and ET3 (p < 0.05, Fig. 2). However, the percentage of CHO oxidation was higher in Cy than in Sed only at ET2 (p < 0.05, Fig. 2).

Total CHO oxidation (g) was increased during training at ET2 and ET3 in comparison with beginning state (ET1) (respectively, + 40% and + 18%; p < 0.05, Fig. 3). Nevertheless, we showed a decrease between ET2 and ET3 (-15%, p < 0.05, Fig. 3). Total CHO oxidation was higher in Cy than in Sed only at ET2 (p < 0.05, Fig. 3).

Lactate concentrations at rest and during exercise
Baseline lactate values between Sed and Cy and during the course of the cycling season for Cy were not different. At the end of the maximal exercise test, the lactate concentration in Cy
Fig. 3 Comparison of total CHO oxidation expressed in grams during 50 min of cycle ergometer exercise performed above (+15%) the ventilatory threshold in cyclists (Cy, n = 7) at three periods (January [ET1], May [ET2] and September [ET3]) of the cycling season and between Cy and sedentary controls (Sed, n = 7). *p < 0.05, ET1 vs ET2. #p < 0.05, ET1 vs ET3. &p < 0.05, ET2 vs ET3. **p < 0.05, Sed vs ET2.

Fig. 4 Lactate concentration during 50 min of cycle ergometer exercise performed above (+15%) the ventilatory threshold and during recovery (5, 10, 15 and 30 min) in cyclists (Cy, n = 7) at different periods of the cycling season (January [ET1; ✔], May [ET2; ●] and September [ET3; ▲]) and between Cy and sedentary controls (Sed, n = 7; ○). See text, Results section, for statistical comparisons.

Fig. 5 Cycling performance and number of abandons (expressed in percentage of races performed) during the entire cycling season, & p < 0.05, ET2 vs ET3.

was higher at ET2 than at ET1 (p < 0.05, Table 1), whereas during recovery the values did not differ. However, lactate kinematics were lower in Cy than in Sed during prolonged exercise at 10, 20, 30 min and during recovery at 5, 10, 15 min in all conditions (ET1-ET3) (p < 0.05, Fig. 4). In addition, the lactate kinematics were lower in Cy than in Sed at all times of exercise and recovery during ET1 and ET2 (p < 0.05, Fig. 4). When we compared Cy at different periods of the training season, we found that lactate kinematics were lower at ET2 than at ET1 during exercise at 10, 20, 30, 50 min and during recovery at 5 and 15 min (p < 0.05, Fig. 4). They were higher at ET3 than at ET1 during exercise at 30, 40, 50 min and during the entire recovery (p < 0.05, Fig. 4), and they were higher at ET3 than at ET2 during exercise at 10, 20, 30, 40, 50 min and at 2, 5 and 10 min of recovery (p < 0.05, Fig. 4).

Physical performance during the cycling season

Race performance was markedly decreased between ET2 and ET3 (p < 0.01, Fig. 5).

In accordance, the number of abandons during races increased significantly during the same period (p < 0.01, Fig. 5). When we looked at individual data, all cyclists showed drops in performance and increased their abandon rates between these two periods.

Overtraining questionnaire

The first part of the questionnaire, which consists of an enumeration of symptoms of overtraining, evidenced an increase in the score of affirmative responses between ET2 and ET3 (p < 0.01, Fig. 6). More specifically, we noted an increase in the occurrence of symptoms related to appetite, motivation for training, quality of sleep and sociability (p < 0.05).

The second part of the questionnaire, which consists of analogic-numerical scales, evidenced an increase in arbitrary units of anxiety and a decrease in the subjective feeling of fitness between ET2 and ET3 (respectively, 2.74 ± 0.19 vs -1.86 ± 0.5 and 1.77 ± 0.41 vs -2.46 ± 0.24 [arbitrary units], p < 0.05).

In addition, the rating of perceived exertion for Cy from Borg-scale values increased gradually during exercise (p < 0.05, Fig. 6). However, this rating of exertion was lower in ET2 than in ET1 and was higher in ET3 than in ET2 (p < 0.05, Fig. 6).

Discussion

The main finding of the present study is that endurance training increased CHO utilisation during hard-intensity exercise over the course of a competitive cycling season. This result confirms our recent cross-sectional study that showed that endurance training promotes CHO oxidation during hard-intensity exercise [31]. Furthermore, in the present study, the effect of endurance training-stimulated CHO oxidation was clearly lower at the end as compared the middle of the season. This decline seemed to coin-
As shown in Fig. 1, both VO₂ and RER appear to be at steady state between 10 and 50 minutes of constant-load exercise. Interestingly, we are unable to observe any VO₂ drift (slow component of O₂ uptake) as described by many authors during heavy exercise [2, 44]. The reason for this apparent lack of VO₂ drift could be that in these endurance-trained cyclists this level of exercise was not high enough to induce such a drift. Moreover, training has been reported to reduce this phenomenon [16]. This is consistent with a previous study performed in our laboratory [31] in which we studied subjects exercising with a similar protocol (70% of VO₂ max for one hour) and found a remarkable stability of VO₂ without any VO₂ drift. Therefore, we consider that there was a satisfactory stability of VO₂ and RER during our constant-load protocol, allowing us to employ calorimetric calculation in conditions that have been fully validated by comparison with the stable isotope method [39].

Our results therefore show that intensive training increased the ability to oxidize CHO at high-intensity exercise. This finding is consistent with current studies that indicate that there is a shift towards a predominance of CHO-based fuels at high power outputs in order to meet the energy requirements of the exercising body [6]. According to these studies, intensive training appears to increase this phenomenon, perhaps because of the greater abundance of glycolytic as opposed to lipolytic enzyme systems in skeletal muscle working at this level of effort [9]. In addition, the higher CHO utilisation is also promoted by changes in the pattern of fibre recruitment to involve fast glycolytic motor units [17]. Thus, athletes train to compete at high power outputs and intensities where CHO-derived fuels, not lipids, predominate [36].

However, we showed a decrement in CHO utilisation during hard-intensity exercise at the end of a long training period (i.e., 9 months). The interpretation of this finding is difficult. A previous study from our laboratory may suggest an initial explanation. We reported that a training-induced increase in insulin sensitivity cannot further augment above an optimal value [29]. We suggested that there may be a physiological boundary of carbohydrate disposal after intensive training, perhaps to avoid exercise hypoglycaemia [29]. On the other hand, several lines of evidence indicate that there was some degree of overtraining in these subjects. It has been suggested that reduced muscle glycogen level may play a role in the pathophysiology of overtraining [15, 27], and that overtraining decreases CHO utilisation [43]. This conception has been challenged by Snyder et al. [42], who reported that a short period of overtraining (i.e., 15 days) may occur even when resting muscle glycogen levels are maintained. Therefore glycogen depletion is not an obligatory feature of the overtraining syndrome. However, it has been shown that an insufficient supply of carbohydrate during intense training impairs the ability to exercise at high intensity [11]. In our study, despite a brief dietary interview before each testing period, we did not determine the exact CHO intake in the days prior to the laboratory visit. It is thus possible that the cyclists had an insufficient carbohydrate intake just before ET3, resulting in a relative lack of carbohydrate that may have compromised performance.

This seems to be consistent with a recent study by Petibois et al. [38] using the spectral analytical technique (FT-IR spectroscopy), which evidenced that the first substrate lacking during over-
training was actually CHO. Obviously, these studies do not rule out the possibility of multiple metabolic and hormonal disturbances that may be involved in the mechanism of overtraining. Although the importance of carbohydrate status in the pathophysiology of overtraining remains controversial, our results are to some extent in agreement with the hypothesis.

Presumably, the differences observed between the different testing periods can to some extent be accounted for by differences in plasma hormone concentrations and more particularly in catecholamine kinematics [25] during exercise. Unfortunately, plasma epinephrine was not measured in this study, although it is possible that a very high concentration of this hormone associated with intense exercise [32] could explain the increase in CHO oxidation, as reported by previous studies [14,31]. The lack of plasma epinephrine measurements limits our ability to assess whether the differences in CHO utilisation between periods might have been adrenergically mediated.

In addition, our cyclists presented a higher CHO oxidation than our sedentary subjects only at the second period of testing (ET2, mid-season). This finding may explain the discrepancies regarding substrate oxidation measured during exercise by cross-sectional studies. On the whole, the present study confirms the hypothesis that training status modifies the pattern of substrate utilisation during exercise [6,7].

Specifically, "CHOs" refer to endogenous energy sources that include muscle and liver glycogen, blood glucose, and blood, muscle and liver lactate [6]. During hard-intensity exercise, lactate and muscle glycogen, but not blood glucose, are the major CHOs for oxidation [7]. In accordance, the lactate concentrations during exercise were higher in our sedentary subjects than in the trained cyclists. This is in agreement with the finding that endurance training increases lactate oxidation rather than lactate production [12]. More recently, Brooks et al. [8] demonstrated that lactate is the predominant monocarboxylate oxidised by the mitochondrial lactate shuttle. Lactate concentrations during exercise, however, did not decrease over the course of the cycling season. Indeed, we showed a decrease in lactate concentrations between the beginning (ET1) and the middle (ET2), but not at the end (ET3) of the season. In this case, these data lead us to suggest that intense endurance training enhances the ability to utilise CHO, including lactate [31], but only up to an optimal point.

Finally, this "CHO oxidation peak" observed at ET2 appears to correspond with the period in which the cyclists’ best competitive results were obtained. We then noted a decrease in cycling performance (expressed by the decline in competitive results and the increase in abandon rates during competitions) during the following period (i.e., the end of the season), which was matched by a decline in CHO oxidation measured during hard-intensity exercise at ET3. In accordance, we also showed an increase in the rating of perceived exertion (measured by a Borg scale) during exercise at ET3 and an impairment in psychological data (determined by a self-administered questionnaire) in the same testing period. These last results have already been observed in an overtraining state [4,43]. However, we did not show a decrease in V̇O₂max at ET3. This finding does not rule out the diagnosis of overtraining, since higher V̇O₂max despite decreased performance has already been reported in overtrained athletes [43]. Thus, we hypothesise that our cyclists were in an early stage of overtraining at the end of their cycling season, which represented an accumulation of several months of intensive training, including cycling races. This stage should be seen as a normal part of athletic training, since our cyclists had been high-performing athletes before this end-period. In view of these observations, we could suggest that CHO utilisation during high-intensity exercise is a potent determinant of physical performance in well-trained competitive athletes. To support this hypothesis, it should be noted that Urhausen et al. [43] showed that an overtraining state decreases CHO utilisation, as expressed by the decrement in RER, and that CHO is the first substrate implicated in the overtraining process [38]. We thus suggest that a decrease in CHO utilisation during high intensity exercise may be at least partly involved in the overtraining and/or overreaching syndrome. Nevertheless, further investigations are necessary to clarify this mechanism.

In summary, CHO oxidation during hard-intensity exercise increases with intense training in competitive cyclists. However, this CHO dependency is lessened by the end of a cycling season and this decline in dependency seems to be associated with a decrease in competitive results and increased ratings in a questionnaire on overtraining. This phenomenon may represent a "metabolic adaptation" to the state of overtraining from which most endurance athletes suffer at the end of a competitive season. Finally, cyclists who train to compete at high levels of effort develop a dependence on CHO as an energy source, but this phenomenon itself depends on the training status.

Acknowledgements

The authors are grateful to the subjects for their contribution of time and effort. Special thanks go to the cyclists of the Mauguière-Carbon Cycling Team.

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