Effect of a Reduced-CHO Diet on the Rate of Perceived Exertion Curve During an Incremental Test

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The objective of this study was to evaluate the effect of a reduced-carbohydrate (reduced-CHO) diet on the rate of perceived exertion (RPE) curve during an incremental test. Nine physically active men performed a progressive incremental test on a cycle ergometer (25 W·2 min⁻¹) after 72 hr on either a control diet (60% CHO) or a reduced-CHO diet (30% CHO). Lactate and RPE thresholds were identified using the Dmax method (DmaxLa and DmaxRPE, respectively). Power output, heart rate and RPE scores in DmaxLa and DmaxRPE were similar between the diets and were not different from each other, regardless of the diet. Lactate values were consistently higher (p < .05) in the control diet compared with the reduced-CHO diet during power output after the lactate breakpoint; however, they were not accompanied by a proportional increase in RPE scores. These results suggest that DmaxRPE and DmaxLa are not dissociated after a short-period reduced-CHO diet, whereas the lactate values after the lactate threshold are reduced with a reduced-CHO diet, although they are not accompanied by alterations in RPE.

Keywords: lactate threshold, RPE threshold, diet, exercise

Introduction

The lactate threshold (LT) is considered to be one of the major parameters in evaluating and monitoring exercise intensity (Cheng et al., 1992; Sjodin & Jacobs, 1981), but its application is expensive and invasive. The estimation of LT using a fixed rate of perceived exertion (RPE) score has been proposed as an alternative because it is highly correlated with the LT identified by blood lactate measurement (Eston, 2009). The RPE is typically assessed using 10- or 15-point Borg scales (Borg, 1998; Borg, 1982), both of which quantify the intensity of effort during exercise. A fixed RPE score of 5 on Borg’s category-ratio (CR-10) scale (Zamunér et al., 2011) or from 9 to 12 on a 15-point scale (Rynders et al., 2011) has been recommended for estimating the LT. When a fixed value is used in this manner, however, biological variations are not taken into account (Fabre et al., 2012). In this regard, Fabre et al. (2012) proposed a new mathematical model in which a third degree polynomial fit is applied to adjust RPE scores during the test, with a linear fit subsequently being obtained by connecting the extremes of the curve (first and last points); the largest distance between the two curves (Dmax) is called the RPE threshold (DmaxRPE). In that study, blood lactate curves were adjusted with a similar model (DmaxLT), producing a mathematical estimation of the LT. The authors reported that DmaxRPE...
Reduced-CHO Diet on the RPE and DmaxLT were found at similar intensities and were strongly correlated, suggesting that LT can be precisely estimated from an individually adjusted RPE curve during an incremental exercise (Fabre et al., 2012).

Some authors have suggested that the identification of the LT in an incremental test may be related to the amount of carbohydrates (CHO) consumed 2 to 3 days before the test (Langfort et al., 1996; Langfort et al., 2001). In those studies, the LT was shifted to the right and observed at a higher power output after a short period (3 days) on a reduced-CHO diet, suggesting that reduced CHO availability delays blood lactate accumulation. The mechanism by which this occurs is not fully understood, but it has been suggested that it may be due an inhibition of the enzyme phosphofructokinase 1 (PFK1) caused by reduced CHO availability to the muscle cell, which reduces glycolysis and blood lactate accumulation (Langfort et al., 1996). Notably, although LT is identified at higher power outputs after a reduced-CHO diet (Langfort et al., 2004), maximal power output is reduced, suggesting that the reduced glycolysis resulting from reduced CHO availability is detrimental to performance (Sabapathy et al., 2006).

The effect of a reduced-CHO diet on the RPE threshold has never been measured during an incremental test; however, results from constant-load exercises at high intensity (~90% of VO2max) indicate that RPE increases for a given power output after a 2-day reduced-CHO diet (Lima-Silva et al., 2010b). This increased RPE for a given power output during a constant-load exercise suggests that RPE might be systematically increased during an incremental test. It could be expected, therefore, that the RPE threshold would be shifted to the left, while the LT would be moved to the right after a short period on a reduced-CHO diet. While earlier studies suggested that a reduced-CHO diet affects blood lactate and exercise performance during an incremental test (Langfort et al., 1996; Langfort et al., 2001), whether any alteration in LT due to a reduced-CHO diet is accompanied by a similar alteration in RPE is still unknown. Therefore, because the RPE threshold has been used as a simple and precise indicator of LT (Fabre et al., 2012) and diet manipulations have been routinely applied during training and testing interventions (Cox et al., 2010), it would be useful to determine whether the RPE threshold remains a robust estimator of the LT when a diet manipulation precedes its determination. In addition, it would be interesting to investigate whether RPE could be strictly associated with blood lactate because blood lactate has been suggested to be a signal that influences the RPE response during exercise (Borg et al., 1987; Noble et al., 1983).

Thus, the objective of this study was to evaluate the effects of a short-term reduced-CHO diet on RPE and LT thresholds. The RPE and LT curves were adjusted using the Dmax model to provide objective, noninvestigator interferences, RPE and LT thresholds identification. We hypothesized that the DmaxRPE might be shifted to the left and the DmaxLT to the right after a 3-day reduced-CHO diet.

**Methods**

**Participants**

Nine adult male university students (age 26.0 ± 4.1 years, 175.9 ± 8.5 cm, 76.2 ± 15.1 kg, 12.9 ± 4.1% of body fat, 1,792 ± 218 kcal basal metabolic rate), all of them physically active (at least 1 year), participated in this study. All participants signed an informed consent form after verbal and written explanations of the study. This study was approved by the Ethics Committee of the Pontificie Catolica University.

**Experimental Design**

The subjects completed two incremental exercise tests on a cycle ergometer (Ergo Fit 167, Ergo-Fit GmbH& Co., Pirmasens, Germany) after either a 72-hr reduced-CHO diet or a control diet (control), using a randomized, counterbalanced design. The tests were performed with an initial power output of 25 W, and the power input was incrementally increased by 25 W every 2 min until exhaustion. The individuals maintained 60 to 70 rpm during the entire test and continued until voluntary exhaustion. The participants were instructed to not consume any caffeinated foods or beverages or to participate in any rigorous physical activity on the day before the test. All tests were performed in the morning after a standardized breakfast consumed 2 hr before the test. The breakfast composition followed the diet schedules for a given diet condition (see below), ie, either with 60% CHO, 20% protein, 20% fat for the control diet or containing a low CHO content (30% CHO, 35% protein, 35% fat) for reduced-CHO diet.

**Diet Protocol**

The diets consisted either of a reduced-CHO diet (30% CHO, 35% protein, 35% fat) or a control diet (60% CHO, 20% protein, 20% fat). The diets were isoenergetic, corresponding to 229 g of CHO, 267 g protein, and 118 g of fat for the reduced-CHO diet (~3000 kcal) and 457 g of CHO, 152 g protein, and 68 g of fat for the control diet (~3000 kcal). The glycemic index was similar in both diets, and the CHO sources in each were bread, fruits (apple and papaya), white rice, and juice. A dietician created all diets using food plans for each subject and took each individual’s daily energy expenditure (DEE) and food preferences into account. Food was not supplied by the investigators, but all subjects received a food option list to describe the allowed content for each food group and provide the recommended daily energy intake. All subjects recorded their daily food intake; analysis of the recorded food intake confirmed that subjects conformed to the recommended diets.
The DEE was estimated using the following equation (Cunningham, 1991):

\[
\text{Basal Metabolic Rate} = 370 + (21.6 \times \text{Fat Free Mass})
\]

(1)

Subjects’ lean mass was estimated using a bioelectric analyzer (Quantum II © RJL systems, Michigan USA) with the following equation (Carvalho & Pires Neto, 1998):

\[
\text{Lean mass} = 10.97556 - (0.03187 \times \text{resistance Ohms}) + (0.17576 \times \text{stature}) + (0.50702 \times \text{Body Weight}).
\]

(2)

For estimating the DEE in the first 2 days of the diet period, the individuals were considered to have a moderate level of physical activity (~2 hr of moderate exercise); therefore, a correction factor of 1.7 was used. For the third day, no physical activity was performed; thus, a correction factor of 1.3 was used (ACSM, 2009). To avoid any discomfort related to overnight fasting, all participants were instructed to have a standardized snack 2 hr before the incremental test; however, the CHO content was still reduced in the reduced-CHO diet (35% of CHO) and was normal in the control diet (50% of CHO).

Measurements

Heart Rate (HR) response was obtained using a heart rate monitor (Polar Electro Oy, FI-90440, Kempele, Finland). Blood lactate was measured with a validated portable lactometer (Accutrend Plus®, Roche Diagnostics GmbH, Germany) using a 25 µL blood sample extracted from the right forefinger (Perez et al., 2008). RPE was measured using Borg’s category-ratio scale (CR-10), with corresponding descriptors of nothing at all (0) and maximal effort (10). Subjects were familiarized with the RPE scale and were instructed to incorporate both muscular and central cardiorespiratory feelings into an overall perception of effort, and they indicated how hard, heavy and strenuous the exercise was in a given stage (Borg & Kajiser, 2006; Borg, 1998; Borg, 1982). Blood samples and RPE scores were measured 15 second before the end of each stage.

The maximal power output (Wmax) was considered the highest power output maintained during a complete stage. When the last stage was not completed, the last complete stage was considered the Wmax.

Determination of Lactate and RPE Thresholds

The LT was individually identified by the power output at which the largest difference between two lines occurred, i.e., the 3rd-order polynomial fit of the lactate values and the 1st-degree linear fit between the two extreme values for the lactate curve (DmaxLa; Figure 1A; Cheng et al., 1992). The RPE threshold was identified by the power output at which the largest difference between two lines occurred, i.e., the 3rd-order polynomial fit of the RPE values and the 1st-degree linear fit between the two extreme values for the RPE curve (DmaxRPE; Figure 1B; Fabre et al., 2012).

Statistical Analysis

The Shapiro-Wilk test was applied to verify distribution. Because normality was identified, the comparisons between variables were performed using a two-factor ANOVA with diets (control and reduced-CHO) and thresholds (DmaxLa and DmaxRPE) as factors. Duncan’s post hoc analysis was performed when necessary. The Wmax, HRpeak, time of exercise and lactate peak were compared between the control and reduced-CHO diets using a paired t test, whereas the maximal RPE values, which were nonnormally distributed, were compared using a Wilcoxon test. The Cohen’s effect size was calculated and interpreted as proposed by Hopkins (2013; http://www.sportsci.org/resource/stats/): ≤ 0.2 trivial; > 0.2 and ≤ 0.6 small; > 0.6 and ≤ 1.2 moderate; and > 1.2 large. The Pearson correlation coefficient was used to determine associations between variables. The Bland and Altman plot was used to check agreement between thresholds. The significance level of p < .05 was assumed, and all analyses were carried out in the OriginPro 8.5 statistical program (OriginLab Corporation, Northampton, Massachusetts, USA). Data are reported as the mean ± standard deviation (SD).

Results

The Wmax, HRpeak and time of test were significantly lower (p < .05) after the reduced-CHO diet than after the control diet (247.2 ± 53.7 vs. 263.9 ± 70.8 W; 179 ± 13 vs. 184 ± 8 bpm, and 1187 ± 258 and 1273 ± 336 second, respectively). The Wmax and time of test were reduced in 8 participants (88%) after the reduced-CHO diet in comparison with control diet. In addition, the lactate peak was significantly lower in the reduced-CHO diet than in the control diet (5.9 ± 2.1 and 9.0 ± 3.1 mmol·L–1, respectively, p < .05). However, the maximal RPE values were similar between the diets (8.9 ± 1.8 and 9.3 ± 1.0 units, respectively, p > .05).

The mean RPE scores at DmaxRPE were similar between the diets (reduced-CHO diet: 3.7 ± 1.5 units vs. control diet: 3.4 ± 1.9 units; p > .05). Likewise, the RPE scores at DmaxLa were similar between the diets (3.4 ± 1.2 and 3.9 ± 1.1 units, respectively, p > .05). The RPE scores were also similar between DmaxRPE and DmaxLa for both diets (p > .05). The power output and HR measured at DmaxLa and DmaxRPE for both absolute (watts and beats per minute, respectively) and relative (%Wmax and %HRpeak, respectively) terms are described in Table 1. Similar to the RPE scores, no significant differences were found between the diets or between thresholds for any of these parameters.

The power output values were significantly correlated between the two thresholds for the control diet
Figure 1 — Identification of lactate (Figure 1A) and rating of perceived effort (Figure 1B) thresholds using the Dmax method in a representative individual. Dmax: Largest perpendicular distance between the third-order polynomial and linear (adjusting the first and last points) curves for lactate (DmaxLa) and rating of perceived effort (DmaxRPE).

Table 1 Absolute and Relative Power Output and Heart Rate at RPE and Lactate Thresholds

<table>
<thead>
<tr>
<th></th>
<th>DmaxLa</th>
<th>DmaxRPE</th>
<th>DmaxLa</th>
<th>DmaxRPE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Reduced CHO</td>
<td>Control</td>
<td>Reduced CHO</td>
</tr>
<tr>
<td>Power output</td>
<td>158.3 ± 45.1</td>
<td>144.4 ± 57.0</td>
<td>147.2 ± 27.4</td>
<td>0.40</td>
</tr>
<tr>
<td>(W)</td>
<td></td>
<td></td>
<td></td>
<td>0.05</td>
</tr>
<tr>
<td>Heart rate</td>
<td>139 ± 13</td>
<td>133 ± 24</td>
<td>134 ± 24</td>
<td>0.80†</td>
</tr>
<tr>
<td>(bpm)</td>
<td></td>
<td></td>
<td></td>
<td>0.05</td>
</tr>
<tr>
<td>%W&lt;sub&gt;max&lt;/sub&gt;</td>
<td>60.1 ± 8.0</td>
<td>54.6 ± 17.2</td>
<td>61.5 ± 14.2</td>
<td>0.50</td>
</tr>
<tr>
<td>%HR&lt;sub&gt;peak&lt;/sub&gt;</td>
<td>75.4 ± 6.3</td>
<td>72.1 ± 11.9</td>
<td>74.7 ± 10.1</td>
<td>0.60</td>
</tr>
</tbody>
</table>

Note. D<sub>maxLa</sub> = lactate threshold identified by D<sub>max</sub> method, D<sub>maxRPE</sub> = rating of perceived effort threshold identified by D<sub>max</sub> method, ES = effect size. No significant statistical differences were found for any of the listed variables (p > 0.05), but a moderate effect size was found to heart rate (†).
(r = .75, p < .05), but not for the reduced-CHO diet (r = .04, p > .05). The heart rate values were not significantly correlated between the two thresholds for both diets (r = .61 control diet and r = .14 reduced-CHO diet, both p > .05). The Bland-Altman plot showed a small bias for power output between the thresholds for both diets. However, it was found a difference between the thresholds equal or superior than 50 W (> 30% from average) for three and four participants (for control and reduced-CHO diets, respectively, Figure 2A and B).

The lactate values up to 60% Wmax were not significantly different between the control and reduced-CHO diets (Figure 3A). However, the values were consistently higher (p < .05) in the control diet compared with the reduced-CHO diet beyond 60% Wmax (Figure 3A). When the lactate values were plotted against absolute power output, significant differences were found at 125 W and Wmax (p < .05, Figure 3B). The RPE values were similar between the diets throughout the test (Figure 4A and B).

Figure 2 — Comparison of the power output between DmaxLa and DmaxRPE (WDmaxLa and WDmaxRPE, respectively) for the control diet (Figure 2A) and reduced-CHO diet (Figure 2B). Thin line represents the mean difference between the thresholds, and the two extreme lines represent limits of agreement (1.96 SD around the mean difference). It is possible to observe only eight points because data from two individuals are overlapped.
Discussion

The main aim of this study was to investigate RPE and lactate behaviors during an incremental test after a short period on a reduced-CHO diet. To the best of our knowledge, this is the first study to investigate the effects of a reduced-CHO diet on both DmaxRPE and DmaxLa thresholds. We did not find significant differences between the diets for both thresholds, and the values identified by DmaxRPE were similar to DmaxLa regardless of the diet condition (Table 1). The lactate values above LT were significantly reduced after the reduced-CHO diet, but they were not accompanied by a reduction in RPE values. These results suggest, for the first time, that the relationship between DmaxRPE and DmaxLa thresholds are not influenced by a preceding reduced-CHO diet, but rather that the cause-effect relationship between blood lactate and RPE is questionable, especially during the highest power outputs of an incremental exercise.

Although Wmax and HR were lower with the reduced-CHO diet than the control diet, the RPE values
Figure 4 — Rate of perceived effort (RPE) as a function of relative (Figure 4A) and absolute (Figure 4B) power output during the progressive incremental test. No significant differences were found, $p > .05$.

at the end of the test were similar between the diets. It has been demonstrated that a reduction in CHO availability results in alternative metabolic pathways for the maintenance of muscular contraction (Bangsbo et al., 1991; Langfort et al., 1996). Specifically, lipid and protein oxidation are increased after a reduced-CHO diet (Greenhaff et al., 1987), impacting ventilatory (Lima-Silva et al., 2010b), hormonal (Langfort et al., 1996), and blood pH (Maughan et al., 1997; Sabapathy et al., 2006) systems. The products of fat oxidation, including NADH, acetyl-CoA and ATP are allosteric effectors that negatively regulate the pyruvate dehydrogenase complex. In particular, the acetyl group accumulation caused by a reduced-CHO diet can reduce the flow of the pyruvate dehydrogenase complex (Maughan et al., 1997). Alternatively, the reduction of CHO in the muscle may act as a signal to the brain to inhibit muscle contraction (Noakes & St Clair Gibson, 2004).

Several studies lead us to the notion that diet manipulation would dissociate RPE and lactate thresholds (Langfort et al., 2004; Langfort et al., 2001; Prusaczyk et al., 1992), although the effect of CHO availability on RPE and lactate thresholds has never been investigated. It has been suggested that the anaerobic metabolic pathway increases its contribution after 80% of Wmax (Zagatto et al., 2011), but reduced CHO availability from the diet
reduces the use of CHO during exercise performed at intensities above 70% of VO2max (Maughan et al., 1997; Pizza et al., 1995). Glycolysis and glycogenolysis are inhibited during incremental exercise performed in a setting of reduced CHO availability, mainly due to increased phosphokinase-2 (PFK-2) and reduced phosphokinase-1 (PFK-1) activities (Langfort et al., 1997). This increase in PFK-2 and reduction in PFK-1 may affect muscle capacity to support higher, but not lower, workloads. This may explain why the reduced-CHO diet reduced blood lactate concentration only after LT, i.e., ~80% of the maximal power output. However, note that the LT was not modified by diet manipulation, suggesting that the break point in the lactate-power output curve is not altered, although a reduced-CHO diet reduces blood lactate concentration at higher exercise intensities.

In the current study, both DmaxRPE and DmaxLa occurred at similar intensity regardless of the previous diet. It has been suggested that DmaxRPE takes into account individual variation in lactate concentrations and that any change in blood lactate would be accompanied by changes in RPE scores (Fabre et al., 2012). Zamunér et al. (2011) found a significant correlation between RPE threshold (identified at score 5 in 10-point CR Borg scale) and lactate threshold (identified at 4 mmol·L⁻¹), suggesting that RPE could be used to estimate LT. Fabre et al. (2012) also compared DmaxRPE with 1) lactate thresholds identified using fixed lactate values (2 and 4 mmol·L⁻¹); 2) Dmax method; 3) respiratory compensation point (RCP); and 4) intensity corresponding to a respiratory exchange ratio equal to 1.00. Oxygen uptake, HR and power output measured at DmaxRPE and DmaxLa were not different, but oxygen uptake measured at DmaxRPE was higher than at LT identified at 2 mmol·L⁻¹ and lower than LT identified at 4 mmol·L⁻¹. Nevertheless, DmaxRPE was significantly correlated with all the other thresholds.

Although we did not find differences between DmaxRPE and DmaxLa for both diets, the correlation between these methods was significant only in the control diet, but Bland and Altman plots showed a small bias and a large difference (> 30%) between the thresholds for some participants. In addition, the lactate concentration above the LT was reduced after the reduced-CHO diet (Figure 3A), but this was not accompanied by proportional increase in RPE (Figure 4A). Several studies have shown that blood lactate is reduced during exercise following a reduced-CHO diet (Lima-Silva et al., 2013; Maughan & Poole, 1981) but that RPE is increased (Lima-Silva et al., 2010b). Similar results were found by Johnson et al. (2006), who did not find an association between RPE and blood lactate. These results suggest that changes in blood lactate levels alone may be not able to alter the RPE response. It has been suggested that lactate may act as a signal to the CNS and influence the RPE response to exercise (Hampson et al., 2001). In this model, lactate and other peripheral signals would be perceived by group III and IV muscle afferents, stimulating sensorial areas in the brain (e.g., mid/anterior insula), which in turn could increase RPE and influence the motor cortex (Hilty et al., 2011). However, fatigue does not appear to be associated with lactate accumulation, but rather with changes in pH (Gladden, 2004). Because pH decreases with an increase in strong organic acids derived from an increased and incomplete lipid and amino acid metabolism after a reduced-CHO diet (Greenhaff et al., 1987; Maughan et al., 1997), pH may be a more important signal the CNS than lactate is. In addition, the RPE appears to be associated with ventilation (Prusaczyk et al., 1992), and reduced CHO availability induces an enhanced ventilatory response (Lima-Silva et al., 2010a; Lima-Silva et al., 2010b). Therefore, caution is recommended when using RPE to estimate the lactate threshold because other variables, such as cardiorespiratory and metabolic alterations, may influence the RPE response to exercise (Lima-Silva et al., 2010b).

On the other hand, the dissociation between RPE and lactate indicates that RPE may be generated by an alternative mechanism. It has been demonstrated that the recruitment of motor neurons by the central nervous system is associated with an elevated stress level (Dalsgaard & Secher, 2007). It was recently shown that RPE is associated with motor command center activity during exercise (de Morree et al., 2012). In that study, an increase in RPE with increasing load and fatigue was accompanied by movement-related cortical potential, suggesting that RPE may be related with corollary discharge of central motor commands instead of feedback from exercised muscles. However, because we have not measured other variables that could act as signals to the CNS (e.g., ventilation, blood pH and glucose, and hormones), we are not able to determine which model governs RPE during the exercise. Nevertheless, our results suggest that lactate is not an important signal during exercise.

In addition, we observed that the rate of increase in RPE, when expressed in % Wmax, was similar between the diets. This observation is similar to submaximal exercise following a reduced-CHO diet (Lima-Silva et al., 2010b). The mechanism by which this occurs has not yet been clarified in the literature, but it has been postulated that the RPE appears to be set at the beginning of the exercise regimen as part of a feed-forward/feedback mechanism and presents a scalar behavior as exercise progresses, suggesting that RPE is set as a function of remaining exercise time (Crewe et al., 2008; Lima-Silva et al., 2010b; Noakes, 2004). Our findings support this theory because there were no differences between the diets in the rate of RPE increment when the values were expressed as % Wmax.

A limitation of the current study is the small sample size. However, in an attempt to minimize possible bias, we calculated the Cohen’s ES between diets for both DmaxRPE and DmaxLa and observed trivial and small effects, respectively. The estimated sample size required to achieve α = .05 and β = 0.80 was > 52 participants, an unrealistic sample size for studies involving diet manipulation. In fact, several studies of dietary manipulation have been developed using relatively small sample
sizes because investigations of this nature involve many
days of testing and a high level of diet control (Lang-
fort et al., 2004; Lima-Silva et al., 2009; Lima-Silva et
al., 2013; Sabapathy et al., 2006). Although we cannot
ensure that Type II error did not occur, the ES values
indicate that diet had a small effect on the thresholds.
In addition, the validity of our results depends on the
effectiveness of and compliance with the diet protocols.
All participants in our study reported that they followed
the diet recommendations. A reduction in the maximum
power output reached during the incremental test after
a reduced-CHO diet reinforces that the diet protocol
was effective. In addition, a large body of evidence has
demonstrated that 2 to 4 days on a reduced-CHO diet is
effective to alter the glycogen levels in skeletal muscle
and physiological responses during exercise (Langfort
et al., 2004; Langfort et al., 1996; Langfort et al., 1997;
Langfort et al., 2001; Lima-Silva et al., 2010b; Maughan
& Poole, 1981; Prusaczyk et al., 1992; Ratz et al., 1989;
Sadamoto et al., 2000).

Practical Applications and Conclusions

A reduced-CHO diet decreases maximal power output
and lactate concentrations above the LT, while the rate of
RPE increases, but when expressed as %Wmax, remains
unaltered. DmaxRPE and DmaxLa were not dissociated
with each other regardless of the diet consumed before
the test, although they were slightly biased. These results
suggest, for the first time, that the relationship between
DmaxRPE and DmaxLa thresholds are not influenced by
a preceding short-period reduced-CHO diet. This study
revealed that blood lactate and RPE are not directly
associated during the highest exercise intensities, sug-

Recognition that other signals may influence RPE during
the last stages of the incremental test. Further studies should
clarify the influence of other signals (eg, pH and ventilation)
on the RPE response during incremental exercise.

From a practical viewpoint, these results may enable
to establish the importance of prior CHO control in the
diet before an incremental performance test to determine
LT and/or RPE thresholds. Although we did not find a
significant difference between them regardless prettest
diet, the slightly biased difference between them sug-
gests that diet recommendations before an incremental
test should be addressed in a clinical routine.

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