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Effects of a low- or a high-carbohydrate diet on performance, energy system contribution, and metabolic responses during supramaximal exercise

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Abstract: The purpose of the present study was to examine the effects of a high- or low-carbohydrate (CHO) diet on performance, aerobic and anaerobic contribution, and metabolic responses during supramaximal exercise. Six physically-active men first performed a cycling exercise bout at 115% maximal oxygen uptake to exhaustion after following their normal diet for 48 h (control test). Seventy-two hours after, participants performed a muscle glycogen depletion exercise protocol, followed by either a high- or low-CHO diet (70% and 25% of CHO, respectively) for 48 h, in a random, counterbalanced order. After the assigned diet period (48 h), the supramaximal cycling exercise bout (115% maximal oxygen consumption) to exhaustion was repeated. The low-CHO diet reduced time to exhaustion when compared with both the control and the high-CHO diet (−19 and −32%, respectively, p < 0.05). The reduced time to exhaustion following the low-CHO diet was accompanied by a lower total aerobic energy contribution (−39%) compared with the high-CHO diet (p < 0.05). However, the aerobic and anaerobic energy contribution at the shortest time to exhaustion (isotime) was similar among conditions (p > 0.05). The low-CHO diet was associated with a lower blood lactate concentration (p < 0.05), with no effect on the plasma concentration of insulin, glucose and K⁺ (p > 0.05). In conclusion, a low-CHO diet reduces both performance and total aerobic energy provision during supramaximal exercise. As peak K⁺ concentration was similar, but time to exhaustion shorter, the low-CHO diet was associated with an earlier attainment of peak plasma K⁺ concentration.

Key words: carbohydrate availability, time to exhaustion, AO₂ deficit, metabolism, fatigue, potassium.

Introduction

Studies have shown that when compared with a control condition, glycogen-depleting exercise followed by 2–3 days on a high-carbohydrate (CHO) diet increases time to exhaustion (TTE) during submaximal exercise (70% of maximal oxygen uptake (VO₂max)); this is associated with an increase in the energy supply derived from CHO oxidation (Bosch et al. 1993; Wagenmakers et al. 1991). In contrast, a low-CHO diet seems to impair submaximal TTE because of a reduction in CHO oxidation (Baldwin et al. 2003; Bergstrom et al. 1967; Lima-Silva et al. 2009). Similar effects seem to occur during supramaximal exercise (i.e., above the power that elicits VO₂max) because of a reduction in CHO oxidation. For example, Maughan and Poole (1981) showed that following prolonged
exhausting exercise, a 2-day, low-CHO diet (3% CHO) reduces TTE during a supramaximal exercise bout at 105% V˙O₂max in comparison with a normal diet (42% CHO); this has been confirmed in several other investigations (Greenhaff et al. 1987a, 1987b, 1988). However, contrasting findings have been reported in studies investigating the effects of a high-CHO diet (Maughan and Poole 1981; Vandenbergehe et al. 1995). Vandenbergehe et al. (1995) conducted a study where participants consumed a high-CHO diet (70% CHO) for 3 days after a glycogen-depletion exercise. It was demonstrated that when compared with a normal diet (50% CHO), there was no effect on TTE during a subsequent supramaximal trial (125% V˙O₂max). In contrast, Maughan and Poole (1981) reported that TTE (105% V˙O₂max) was significantly increased after a high-CHO diet (84% CHO) versus a normal diet (42% CHO). However, it should be mentioned that the differences in CHO content in high-CHO diets (70% vs 84% of CHO), and the differences in percentage of CHO between the low- and high-CHO diets (~20% vs 42% CHO) may explain these conflicting results. Thus, the effects of glycogen-depleting exercise, followed by a low- or high-CHO diet, on performance during supramaximal exercise performance requires further research.

An important feature of supramaximal exercise is that both the aerobic and anaerobic systems contribute substantially to energy provision. For example, Bertuzzi et al. (2010) estimated the anaerobic contribution during a supramaximal exercise trial (110% V˙O₂max) by using the accumulated O₂ deficit (AO₂) method, and showed that approximately 35% of total energy expenditure was derived from anaerobic sources. Since CHO can be metabolized by both anaerobic and aerobic pathways, it is important to consider the contribution of both energy systems when investigating the effects of a low- or high-CHO diet on supramaximal exercise performance. Currently there are no studies that describe the contribution of aerobic and anaerobic energy systems during a supramaximal, TTE test after a low- or high-CHO diet. Yet, some data suggest that despite an altered TTE, blood lactate accumulation is not altered (as should occur if aerobic glycolysis is increased or reduced) after supramaximal exercise under different CHO diet compositions (Greenhaff et al. 1987a, 1987b, 1988; Maughan and Poole 1981). Although this finding needs to be interpreted with caution, since a number of factors other than the contribution of anaerobic glycolysis could influence blood lactate accumulation, these results suggest that changes in TTE during the low- or high-CHO diet may have been influenced by alterations in the aerobic energy contribution.

A few studies have suggested that CHO availability might influence the rate of aerobic metabolism by altering the time course and amplitude response of oxygen uptake (V˙O₂) at the onset of high-intensity, submaximal exercise (Carter et al. 2004; Lima-Silva et al. 2011). In a previous study, we have demonstrated that a glycogen-depleting exercise, followed by 2-day, low-CHO diet, reduced both the time course and the amplitude response of V˙O₂ during exercise at ~90% V˙O₂max when compared with normal- and high-CHO diets (Lima-Silva et al. 2011). Thus, if the V˙O₂ response during supramaximal exercise was to change following diet manipulation, exercise tolerance might be altered by modifying the reliance on aerobic energy production. However, while a reduced provision of energy from CHO as a result of a low-CHO diet may reduce the TTE during supramaximal exercise, it is not known whether this is caused by a reduction in the aerobic or anaerobic contribution. Similarly, while the extra energy obtained by a high-CHO diet may contribute to prolonging the TTE, it is also not known whether this extra energy is predominantly provided by aerobic or anaerobic sources.

Alternatively, CHO availability may influence TTE during supramaximal exercise by affecting skeletal muscle fatigue sites, rather than the aerobic or anaerobic contribution (Simmonds et al. 2010). In particular, disturbances to K⁺ homeostasis are important factors that may be involved in fatigue (McKenna et al. 2008). It has been demonstrated that a rise in plasma K⁺ concentration precedes muscle fatigue (Sjøgaard 1990) because of, at least in part, a reduction in Na⁺-K⁺-ATPase activity (Green 2004; Green et al. 2007). Since both muscle glycogen and blood glucose might be affected by dietary CHO manipulation, and the Na⁺-K⁺-ATPase pump appears to prefer CHO as a substrate for ATP resynthesis to meet its energy requirements (Okamoto et al. 2001), it is possible that plasma K⁺ concentration could be compromised by the CHO content of the diet. In addition, CHO availability seems to change insulin concentration (Weltan et al. 1998), which could reduce Na⁺-K⁺-ATPase activity and lead to a more rapid rise in plasma K⁺ concentration (Claussen 2003). However, the effect of CHO availability on plasma K⁺ concentration and insulin concentration during supramaximal exercise has not been investigated.

Therefore, the main purpose of this investigation was to examine the effects of a high- or a low-CHO diet on TTE, as well as on aerobic and anaerobic contribution during supramaximal exercise. We hypothesized that, when compared with a normal-CHO diet, a low-CHO diet would reduce performance and that this would be associated with a reduction in the aerobic energy contribution, while a high-CHO diet would have the opposite effect. Additionally, blood lactate, plasma glucose, insulin, and K⁺ were measured to provide some possible mechanistic insights into the altered performance during supramaximal exercise.

### Materials and methods

#### Participants
Six healthy, physically active men who were familiarized with supramaximal exercise (age, 29.7 ± 7.6 years; height, 179.3 ± 4.4 cm; mass, 75.8 ± 10.0 kg; body fat, 13.0% ± 2.7%; and V˙O₂max, 46.7 ± 10.9 mL·kg⁻¹·min⁻¹) volunteered to participate in this study. Participants were informed about the experimental risks and the protocol that would be undertaken, and signed an informed consent form before the investigation. The participants completed the Canadian Society for Exercise Physiology’s Physical Activity Readiness Questionnaire and a general medical questionnaire to be included in the study. The inclusion criteria included: previous experience with high-intensity exercise, to be a nonsmoker, to not be taking dietary supplements or medications, and to be free from neuromuscular disorders or cardiovascular dysfunctions. The study procedures were approved by the Ethics Committee of the School of Physical Education and Sport at the University of São Paulo, Brazil.

#### Experimental design
Participants reported to the laboratory on 6 different occasions. During the first visit, anthropometric measures were carried out and an incremental test was performed to obtain the V˙O₂max and the lactate threshold. During the second visit, performed 72 h after the first, participants performed a supramaximal trial after following a normal diet for 48 h (control test). On the third visit, performed 72 h after the second, the participants performed a muscle glycogen depletion protocol. After the muscle glycogen depletion protocol, participants were assigned to either a high- or a low-CHO diet for 48 h. The high or low CHO diets were applied in a random, counterbalanced order (Gollnick et al. 1973, 1974; Heigenhauser et al. 1983; Lima-Silva et al. 2009, 2011). At the fourth visit, immediately following 48 h on the assigned diet, the participants performed a second supramaximal trial. After 1 week to “wash out” any residual effects of fatigue (Piehl 1974; Vandenbergehe et al. 1995), the participants visited the laboratory for the fifth and sixth time and repeated the same experimental procedures described for visits 3 and 4, but using the alternative diet. All supramaximal trials (low-CHO, control and high-CHO) were performed in the morning after a 12-h overnight fast. The participants were recommended to not perform any exercise, or to not consume any alcohol, tobacco, and caffeine for 48 h before each supramaximal exercise.
trial or muscle glycogen depletion protocol. The abstention from alcohol, tobacco, and caffeine was checked by a clinical history questionnaire. The participants received no information related to the power output and TTE until all tests had been completed.

**Anthropometric measurements**

All anthropometric measurements were performed according to the procedures described by Norton and Olds (1996). Skinfold thickness was measured at pectoral, abdomen, and thigh sites (Harpenden caliper, West Sussex, UK) and the body density was predicted by the equation by Jackson and Pollock (1978). The body density was converted to body fat by using the equation by Brozek et al. (1963).

**Incremental test**

The incremental test was performed on a cycle ergometer (Ergo Fit 167, Ergo-Fit GmbH & Co., Pirmasens, Germany) and started at an initial work rate of 50 W with 20 W increases every 3 min until exhaustion. Exhaustion was assumed as the inability to maintain a pedal cadence above 60 r·min⁻¹, and verbal encouragement was provided to ensure maximal effort. Ventilation (VE), VO₂, and carbon dioxide production (VCO₂) were continuously sampled and averaged over 30-s intervals using an on-line breath-by-breath gas analyzer (Quark b2, Cosmed, Rome, Italy). The gas analyzer was calibrated according to the manufacturer's specifications before each test (Quark b2 instruction manual). At the end of each stage, 25 μL of blood was drawn from the ear lobe and immediately analyzed to determine the blood lactate concentration. Blood lactate concentrations were analyzed by using an automated analyzer (YSI 1500 Sport, Yellow Springs Instruments, Yellow Springs, Ohio, USA). The LT1 and LT2 lactate breakpoints were determined by linear regression analysis (Ribeiro et al. 1986). The LT1 was defined as the power output corresponding to an initial change in the rate of lactate accumulation in the blood, while LT2 was defined as the power output corresponding to the second change in the rate of lactate accumulation. VO₂max was determined when 2 or more of the following criteria were met: an increase in VO₂ of less than 2.1 mL·kg⁻¹·min⁻¹ on 2 consecutive stages, a respiratory exchange ratio greater than 1.1, a blood lactate concentration higher than 8.0 mmol·L⁻¹, and a heart rate ±10 beats·min⁻¹ of the maximal age-predicted heart rate (Howley et al. 1995). The maximal workload (Wₘₐₓ) was determined as the highest workload reached. When the last stage was not completed, the Wₘₐₓ was calculated from the following equation:

\[
W_{max} = W_{maxICS} + (t/180) \times 20
\]

where WmaxICS is the power output in the last complete stage performed, t is the time in seconds sustained in the last incomplete stage, 180 is the duration of each stage, and 20 is the increment of power output between the stages.

**Control test**

During the 5 days before the control test, time, type, and quantities of food consumed were assessed by a 24-h dietary recall. The dietary recall was used to determine habitual energy intake, diet composition, and the subjects' food preferences, and was later used by a dietician to create the low- and high-CHO diets. For the control test, participants reported to the laboratory between 0700 and 0900 h. Upon arrival, a 25-μL blood sample was drawn from each subject's ear lobe to obtain the resting blood lactate concentration. Thereafter, the cannula was flushed with 1 mL of a 0.9% saline solution to maintain patency, and participants were transferred to the cycle ergometer. Participants were asked to rest quietly on the cycle ergometer for 5 min to determine baseline VO₂. After a 5-min warm-up at 50 W, the participants performed a constant-workload test until exhaustion at a power output that corresponded to 115% VO₂max. The power output at 115% VO₂max was calculated from the linear VO₂-power relationship obtained in the incremental test (Bishop et al. 2002). The VE, VO₂, and VCO₂ were measured breath-by-breath throughout the trials. End-exercise VO₂ was defined as the average of the last 30 s of the supramaximal test. Heart rate was continuously measured using a heart rate monitor (Polar S810i, Polar Electro OY, Kempele, Finland). Blood samples from the ear lobe and from the antecubital vein were again obtained immediately after the participants had reached exhaustion. Participants were then asked to rest quietly on the cycle ergometer for a 5-min period. Additional blood samples were collected from the ear lobe at 1, 3, and 5 min during the recovery for the determination of peak blood lactate concentration.

**Muscle glycogen depletion protocol**

To reduce muscle glycogen stores, participants arrived at the laboratory between 0700 and 0900 h and cycled for 90 min at a power output corresponding to 50% of the difference between LT₁ and LT₂. This procedure was followed by six 1-min exercise bouts at 125% VO₂max interspersed with 1-min rest periods. This protocol was previously validated for reducing the muscle glycogen content by ~70% of pre-exercise values (Bergstrom et al. 1967; Gollnick et al. 1973, 1974; Heigenhauser et al. 1983).

**Diet procedures**

The low- and high-CHO diets consisted of 25% carbohydrate, 45% lipids, and 30% protein; and 70% carbohydrate, 20% lipids and 10% protein, respectively. The daily energy intake was estimated from the 24-h dietary recalls and maintained similar to the habitual daily energy intake. The participants received a list of food choices that contained descriptions regarding the allowed quantity to each food group as well as the recommended daily intake. Participants recorded all food intakes for the 48-h period following the exercise depletion protocol. Diet records were subsequently analyzed for caloric contributions of fats, proteins, and carbohydrates, using DietWin software (DietWin professional software, DietWin, Porto Alegre, Brazil).

**Low- and high-CHO exercise supramaximal tests**

After 48 h on the assigned diet (low- and high-CHO diets), the participants performed a constant workload test until exhaustion at 115% VO₂max (between 0700 and 0900 h) after a 12-h overnight fast under the same conditions and procedures previously described for the control test.

**Blood sample analysis**

The blood samples drawn from the antecubital vein were immediately transferred to tubes containing EDTA and then centrifuged for 10 min (3000 r·min⁻¹ (1800g) at 4 °C). The supernatant plasma was stored at −80 °C until subsequent analysis. Plasma glucose concentrations were analyzed by enzymatic colorimetric method (Biotecnica, São Paulo, Brazil), plasma insulin concentrations by commercial kits RIA (DPC, Los Angeles, Calif., USA), and plasma K⁺ concentrations by ion selective determination (AVL 9180, Roche, Basel, Switzerland).

**Calculations of aerobic and anaerobic energy system contribution**

First, breath-by-breath VO₂ data set for each test was edited to remove any outliers, i.e., 3 SDs around the local mean. After, the breath-by-breath responses of VO₂ were interpolated to 1 value per second and VO₂ data were fitted with a monoeponential model (Barstow and Molé 1991; Özyener et al. 2001) by using a nonlinear least squares fitting procedure (eq. (2)) (Origin, Microcal, Mass., USA).
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Fig. 1. Total energy expenditure (i.e., accumulated oxygen demand) and aerobic and anaerobic (accumulated oxygen deficit) contributions during supramaximal exercise following low-carbohydrate (CHO), control, and high-CHO diet conditions. Data are means ± SE. *Significantly lower than both the control and high-CHO diet (p < 0.05); †, significantly lower than high-CHO diet (p < 0.05). There was a tendency for aerobic contribution for the control diet to be lower than the high-CHO diet (p = 0.08) and higher than the low-CHO diet (p = 0.09).

Aerobic and anaerobic contribution

The parameters derived from the monoexponential eq. (1) were similar among the low-CHO, control, and high-CHO diets (baseline $\dot{V}O_2$: 0.90 ± 0.33, 0.71 ± 0.35, and 0.85 ± 0.48 L·min⁻¹; amplitude: 2.49 ± 0.44, 2.56 ± 0.30, and 2.51 ± 0.48 L·min⁻¹; and time constant: 39.7 ± 5.7, 40.6 ± 4.9, and 43.6 ± 7.6 s, respectively, p > 0.05). In addition, no difference in $\dot{V}O_2$ was detected at exhaustion (3.35 ± 0.45, 3.40 ± 0.52, and 3.49 ± 0.43 L·min⁻¹, respectively, p > 0.05). However, the accumulated $\dot{V}O_2$ demand was significantly higher in high-CHO diet and control than in the low-CHO diet (p < 0.05), but there was no difference between the high-CHO diet and control (p = 0.11) (Fig. 1). Similarly, the $W_{an}$ was significantly higher in the high-CHO diet than in the low-CHO diet (p < 0.05), and there was a tendency for high-CHO diet to be higher than the control (p = 0.08) and for the low-CHO diet to be lower than the control (p = 0.09). The absolute $W_{an}$ was similar across all conditions (p > 0.05). When the parameters were expressed in percentages of total energy, the percentage of energy expenditure attributed to the aerobic system was higher (75.3% ± 7.6%) and the anaerobic contribution lower (24.7% ± 7.6%) following the high-CHO diet than the low-CHO diet (69.1% ± 7.8% and 30.9% ± 7.8%, respectively, p < 0.05). No difference was found (p > 0.05) between high- or low-CHO diet and the control diet (control aerobic: 71.9% ± 10.9%; control anaerobic: 28.1% ± 10.9%). The values estimated using the isotime are shown in Table 1. There was no difference between values for accumulated $\dot{V}O_2$ demand or absolute aerobic and anaerobic expenditures (p > 0.05). Similarly, there was no difference in aerobic and anaerobic expenditures among the conditions when data were analyzed for each 15-s interval.

Blood and plasma metabolites and hormones concentration

The blood and plasma metabolites and insulin concentrations are shown in Table 2. Significant exercise effects on blood lactate concentrations were observed in all conditions (p < 0.05). The blood lactate concentration was higher in the high-CHO than in low-CHO diet (p < 0.05), but there were no differences between high-CHO diet and control or between low-CHO diet and control (p > 0.05). An interaction between time and condition was also found for plasma lactate concentration (p < 0.05). When compared with rest, plasma K⁺ increased at the end of exercise, but there was no difference between the 3 conditions or an interaction between

$$\dot{V}O_2(t) = \dot{V}O_2(baseline) + A \times [1 - e^{-(t-t_0)}]$$

where $\dot{V}O_2(t)$ is the $\dot{V}O_2$ at time $t$ in seconds, $\dot{V}O_2(baseline)$ is the $\dot{V}O_2$ at baseline in liters/min, $A$ is the amplitude in liters/min, $t_0$ is the time delay in seconds, and $r$ is the time constant in seconds.

The monoexponential model started 15 s after the onset of exercise (i.e., phase I was ignored). The $\dot{V}O_2$ estimated by eq. (1) was integrated over time to obtain the accumulated $\dot{V}O_2$ during the supramaximal test. Thereafter, the resting $\dot{V}O_2$ was integrated over the same interval of time. The integrated $\dot{V}O_2$ rest was subtracted from the integrated $\dot{V}O_2$ during the supramaximal test to estimate the work derived from the aerobic system ($W_{aer}$).

The anaerobic energy provision was obtained using the $\dot{V}O_2$ deficit method. Briefly, an individual relationship between the oxygen cost and power output was established during the last 1 min of each submaximal stage during the incremental test (Bickham et al. 2002; Bishop et al. 2002; Gastin et al. 1995; Russell et al. 2000). Then, the oxygen demand for the supramaximal trial was estimated by extrapolation (Bishop et al. 2002; Gastin et al. 1995) and multiplied by TTE. The work derived from the anaerobic system ($W_{an}$) was then calculated as the difference between the estimated oxygen demand and the accumulated $\dot{O}2$ uptake (i.e., $W_{aer}$), with a ratio of 10% to correct for the contribution of body oxygen stores to the energy supply (Medbo et al. 1998). An energy equivalent of 20.9 kJ·LO₂⁻¹ was used to convert oxygen equivalents to kilojoules for both the aerobic and anaerobic energy systems.

To investigate if CHO availability also alters the rate of aerobic and anaerobic energy production, the $\dot{V}O_2$ deficit for each 15-s interval during the first 2 min of exercise was calculated to give $W_{aer}$ and $W_{an}$ for each epoch. Additionally, to determine whether energy system differences among conditions were a result of their different times of exercise, the $W_{aer}$ and $W_{an}$ were also calculated at isotime. Isotime was defined as the shortest TTE recorded across the conditions (Simmonds et al. 2010).

Statistical analysis

The data are presented as means ± SD unless otherwise noted. The differences among the diet compositions, TTE, kinetics parameters, and aerobic and anaerobic contribution for the 3 experimental conditions were assessed by 1-way ANOVA with repeated measures. The plasma and blood parameters were compared using 2-way ANOVA with repeated measures to investigate the effect of time (rest vs end of exercise) and condition (control, high-, and low-CHO diets). A least significance difference test identified the means that were significantly different from p < 0.05. All analyses were performed using SPSS software (version 13.0; SPSS Inc., Chicago, Ill., USA).

Results

$\dot{V}O_2$–power relationship

The $\dot{V}O_2$–power relationship determined during the incremental test reflected a linear relationship ($r = 0.986 ± 0.010, p < 0.001$), and the corresponding standard error of the estimate for individual $\dot{V}O_2$–power regressions averaged 0.101 ± 0.045 L·min⁻¹. The mean power output corresponding to 115% $V_{O2max}$ was 289.0 ± 41.3 W.

TTE

The TTE was significantly (p < 0.05) lower following the low-CHO diet (3.0 ± 0.2 min) than following the control (3.7 ± 0.3 min) or high-CHO diet (4.4 ± 0.3 min), but there was no difference between the high-CHO diet and the control condition (p = 0.15). All 6 participants reduced their TTE with the low-CHO diet compared with the control, while only 4 of them increased with the high-CHO diet.

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TTE during a supramaximal trial at low-CHO diet (from 3% to 7% of CHO, during 2 to 4 days) reduced time and condition interaction effects were found for $V\dot{O}_2$max. It is interesting to note that differences in TTE between the control and high-CHO diets were maximized when subjects consumed a very high percentage of CHO in the high-CHO condition (~84% CHO) and there were large differences between the high-CHO and control diets (~42% CHO). In the present investigation, our intention was to provide ~70% of CHO in the high-CHO diet, since in practice it is extremely difficult to consume a diet with more than 70% of CHO. In fact, the participants’ diet recall resulted in significantly more CHO content than the control diet (~22% more CHO than control), similar to what had been reported in several studies (Greenhaff et al. 1987a, 1987b, 1988; Vandenbergh et al. 1995). Nonetheless, we successfully produced 3 representative diet conditions on a “continuum” of the CHO diet composition (70%, 48%, and 25% of CHO for high-CHO, control, and low-CHO diets, respectively, $p < 0.05$). Taken together, it appears that a low-CHO diet (~25% of CHO) reduces the performance during supramaximal exercise, but that a high-CHO diet may not confer any additional benefits to the performance. In addition, the effect of the low-CHO diet on performance seems to be more homogenous between the participants, while the high-CHO diet seems to have a more individualized effect.

It has been estimated that the anaerobic contribution to supramaximal exercise ranging from 110% to 120% $V\dot{O}_2$max is ~35% (Bertuzzi et al. 2010; Simmonds et al. 2010). Although our values, based on the $AO_2$ deficit method, were slightly lower (~28%), our results suggest that CHO diet composition has no effect on the anaerobic contribution, even though there was a reduced end-exercise blood lactate. This finding is compatible with the assumption that the amount of anaerobic energy that can be produced during a supramaximal exercise until the exhaustion is a constant value (Fukuba et al. 2003; Medbo et al. 1988). The maximum $AO_2$ deficit has been considered a valid and reliable method to measure anaerobic work capacity, and reflects a finite available energy store composed of a phosphagen pool, an anaerobic glycolytic component, and an $O_2$ store (Medbo et al. 1988). It has been reported that durations of 2 min or longer are required to maximize the $AO_2$ deficit during supramaximal exercise, while shorter performance times seem to be insufficient to exhaust the anaerobic capacity (Medbo et al. 1988). The TTE in the present study ranged from 2–4 min, but the $AO_2$ deficit was sufficient to allow complete exhaustion of the anaerobic capacity with no further effect of diet composition on anaerobic work capacity. In addition, our results are in accordance with Gastin et al. (1995), who found that the total amount of work was significantly greater the longer the test duration (ranged from 62 to 208 s), but there was no significant difference

| Table 1. Accumulated oxygen demand, absolute and relative aerobic, and anaerobic contribution in the low-carbohydrate (CHO), control, and high-CHO diet conditions, standardized by the shortest time to exhaustion (i.e., isotime). |
|----------------------------------|----------------|----------------|----------------|
| Low-CHO diet                     | Control        | High-CHO diet  | $p$            |
| Accumulated $O_2$ demand (kJ)    | 217.0±10.4     | 217.2±12.0     | 218.5±10.7     | 0.939 |
| Anaerobic (kJ)                   | 73.9±8.1       | 74.0±11.3      | 73.6±9.9       | 0.994 |
| Aerobic (kJ)                     | 143.1±7.3      | 143.3±9.8      | 144.8±8.3      | 0.988 |
| Anaerobic (%)                    | 33.8±2.7       | 33.7±4.3       | 33.4±2.7       | 0.970 |
| Aerobic (%)                      | 66.2±2.7       | 66.3±4.3       | 66.6±2.7       | 0.970 |

| Table 2. Blood and plasma metabolites and hormones at rest and at the end of exercise for the low-carbohydrate (CHO), control, and high-CHO diet conditions. |
|----------------------------------|----------------|----------------|----------------|
| Low-CHO diet                     | Control        | High-CHO diet  | $p$            |
| Lactate (mmol·L⁻¹)               | 0.43±0.08      | 0.57±0.07      | 0.48±0.05      | 9.38±1.27 |
| Glucose (mmol·L⁻¹)               | 4.49±0.66      | 4.44±1.77      | 4.56±1.30      | 4.57±1.35 |
| Potassium (mmol·L⁻¹)             | 4.07±0.09      | 4.05±0.29      | 3.92±0.15      | 4.48±0.99 |
| Insulin (μU·mL⁻¹)                | 4.19±0.08      | 3.89±1.28      | 4.26±1.07      | 3.89±0.52 |

Main effect of condition (high-CHO diet higher than low-CHO diet).
Main effect of time (end exercise higher than rest).
Interaction effect. Lactate values at the end of exercise correspond to the highest values identified from the samples obtained at exhaustion and 1, 3, and 5 min after exercise using capillary blood measurements.
for AO2 deficit; this suggests that the additional work is derived from aerobic processes.

The lower \( W_{\text{er}} \) in the low-CHO diet condition in the present study was accompanied by a significantly lower TTE when compared with the high-CHO diet (\( p < 0.05 \)). There was also a tendency for \( W_{\text{er}} \) during the low-CHO diet to be lower (−23.5%) than the control condition, but this did not reach statistical significance (\( p = 0.09 \)). The present study is the first to measure aerobic contribution during a supramaximal, TTE exercise after diets with different CHO contents. The low-CHO diet might reduce energy produced via the aerobic energy system by a reduced CHO oxidation. Carbohydrate is the fuel preferentially oxidized during high-intensity exercise (Romijn et al. 1993), and a 2-day, low-CHO diet following a muscle glycogen depletion protocol results in a loss of the ability to oxidize the available carbohydrate store at a rate sufficient to fuel high-intensity exercise (Lima-Silva et al. 2009). Havemann et al. (2006) have suggested that fat oxidation could satisfactorily replace muscle glycogen oxidation during low- and moderate-intensity exercise, but not during high-intensity exercise. Thus, low CHO availability could potentially reduce CHO oxidation during supramaximal exercise without an adequate replacement by fat oxidation. However, it seems less likely that the limit of tolerance in supramaximal exercise results from an available and balance energy resource limitation rather than from a build-up of fatigue metabolites such as hydrogen ions and K+ homeostasis disruption, which impairs muscle contractility and produces limiting symptoms (Fukuda et al. 2003).

In addition to a reduced total aerobic contribution, we had hypothesised that a low-CHO diet might reduce exercise tolerance by altering the VO2 kinetics. We have demonstrated in a previous study that glycogen-depleting exercise, followed by a 2-day, low-CHO diet, reduces both the time course and the amplitude response of VO2 during exercise at -90% \( VO_{2\max} \) when compared with normal- and high-CHO diets (Lima-Silva et al. 2011). In the present study, we found that neither a low- nor high-CHO diet altered the VO2 response during supramaximal exercise performed at 115% \( VO_{2\max} \). Also, energy provided by the aerobic and anaerobic pathways at isotime was remarkably similar between conditions. Carter et al. (2004) found that the VO2 primary component response during a heavy constant-load exercise (50% of the difference between ventilatory threshold and \( VO_{2\max} \)) can be increased, while the VO2 slow component can be reduced, by depletion of glycogen in type II muscle fibers. Additionally, in our previous study, we identified that a low-CHO diet reduced the amplitude response of VO2 by reducing the VO2 slow component during exercise at ~90% \( VO_{2\max} \) (Lima-Silva et al. 2011). However, during supramaximal exercise (110% \( VO_{2\max} \), the on-transient VO2 kinetics revert to a mono-exponential profile because of the lack of a VO2 slow component during the on-transient VO2 kinetics, reflecting the short tolerable duration of exercise (i.e., shorter than that required to induce the slow component), or the difficulty to discriminate the VO2 slow component in a region that is proportionally dominated by the primary component (Oyzenner et al. 2001). Thus, we suggest that it is unlikely that the reduced TTE in the low-CHO diet, compared with the control and high-CHO diet in the present study, would have been caused by an altered rate of reliance on aerobic or anaerobic energy production.

Since low CHO availability seems to reduce the total amount of energy provided by the aerobic pathway during supramaximal exercise, but has no systematic effect on the rate of reliance on aerobic and anaerobic energy production, TTE may be shortened following a low-CHO diet by an alternative physiological mechanism. For example, extracellular K+ accumulation during exercise depletes membrane excitability and has been proposed to contribute to skeletal muscle fatigue (Mckenna et al. 2008). An increase in sarcolemma Na+-K+-ATPase pumping reduces extracellular accumulation of K+ (Mckenna et al. 2008). Stewart et al. (2007) have suggested that the ergogenic effect of glucose supplementation occurs as a result of improved protection of muscle membrane excitability during prolonged exercise (−60% \( VO_{2\max} \)). Accordingly, Green et al. (2007) have demonstrated that glucose supplementation increases maximal Na+-K+-ATPase activity and TTE during prolonged exercise (−57% \( VO_{2\max} \)). These changes occurred in the face of a blunting of the normal exercise-induced decrease in serum insulin. Although the extrapolation of these results to supramaximal exercise should be done with caution, we observed that plasma K+ values measured at exhaustion following the low-CHO, control, and high-CHO diets were not significantly different, despite the reduced TTE following the low-CHO diet.

Our results suggest that CHO availability might regulate the TTE by altering the time taken to reach critical levels of K+ during supramaximal exercise. Vollestad et al. (1994) have demonstrated that K+ concentration in the femoral vein during exercise at 110% \( VO_{2\max} \) rises exponentially throughout the exercise period (−4 min) to reach a maximum value at exhaustion. Although we were unable to measure interstitial K+ concentration, it has been shown that interstitial K+ concentration is highly associated with plasma K+ concentration (Green et al. 2000). Thus, the findings of the present study support the hypothesis that reduced CHO availability may decrease TTE during supramaximal exercise by accelerating the rate of plasma K+ accumulation (Simmonds et al. 2010). Simmonds et al. (2010) also suggested that caffeine ingestion might attenuate fatigue during exercise by increasing sarclemma Na+-K+ pumping, thereby reducing extracellular accumulation of K+. Interestingly, Hallén (1996) has suggested that during dynamic exercise, the loss of K+ from the exercising muscles to the circulation is due to lack of stimulation of the Na+-K+ pump, but is not caused by insufficient Na+-K+ pump capacity for K+ reuptake. Thus, although some evidence suggests that glucose supplementation is correlated with an increase in the maximal Na+-K+-ATPase activity during prolonged exercise (Green et al. 2007), further studies should investigate if low-CHO availability can reduce Na+-K+ pump activity during supramaximal exercise.

In conclusion, we showed that a low-CHO diet significantly reduced TTE during supramaximal exercise, but that a high-CHO diet did not improve performance. The reduced performance after the low-CHO diet was also accompanied by a lower total aerobic, but not anaerobic energy contribution. However, the reduced TTE in the low-CHO diet occurs without altering the rate of aerobic or anaerobic energy production. Alternatively, similar plasma K+ concentration at the end of exercise was identified in all 3 diet conditions, even with a reduced TTE in the low-CHO diet, suggesting that CHO availability potentially accelerates the attainment of peak levels of extracellular K+ thereby altering fatigue. This regulation of plasma K+ concentration seems to occur independently of insulin changes in the plasma. However, studies measuring hormone response during the isotime are necessary to fully confirm this last hypothesis.

References


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