Carbohydrate ingestion does not influence the change in energy cost during a 2-h run in well-trained triathletes

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Abstract

The aim of this study was to examine whether the increase in the energy cost of running (C_r), previously reported to occur at the end of a prolonged run, could be influenced by the ingestion of either an artificially sweetened placebo (Pl) or a 5.5% carbohydrate (CHO) solution. Ten well-trained triathletes completed three testing sessions within a 3-week period. The aim of the first session was to determine maximal oxygen uptake (V^02max) and the velocity associated with ventilatory threshold (v_{VT}). The second and the third sessions were composed of two submaximal treadmill runs (20 min long, 0% grade, performed at v_{VT}), before and after an 80-min overground run, also conducted at v_{VT}. During these submaximal tests, the subjects ingested (in a random order) either a Pl or CHO solution prior to the first submaximal run and every 20 min after that. During the first session, Ventilatory threshold (VT) occurred at [mean (SD)] 81.2 (2.5)% $V0_{2max}$ and 16.5 (0.6) km • h^{-1}. A significant effect of exercise duration was found on C_r (ΔCr) at the end of the run, whatever the solution ingested (ΔC_r = 5.7% and 7.01% for CHO and Pl, respectively). A reduction in the respiratory ex-change ratio (from 0.98 to 0.90) was observed only at the end of the Pl trial. In this study, C_r seems to be affected only to a minor extent by substrate turnover. Moreover, the increase in the demand for oxygen, estimated from the increase in ventilation, accounted for only a minor proportion of the increase in C_r (11% and 17% for CHO and Pl, respectively). No correlation was found between the changes in C_r and the changes in the other physiological parameters recorded. These results suggest, indirectly, that C_r increases during a 2-h run at 80% $V0_{2max}$ in well-trained subjects can be explained mainly by alterations in neuromuscular performance, which lead to a decrease in muscle efficiency.

Key words Energy cost of running • Fatigue • Carbohydrate feeding • Triathlon

Introduction

Triathlon is an endurance activity in which athletes compete sequentially in swimming, cycling and running. In addition, the main constraint placed upon competitors during a triathlon race is the exercise duration, which ranges from 1 h to 10 h (Sprint to Ironman, respectively). Numerous studies have tried to identify those physiological Variables that are the most likely to result in successful triathlon performance. For exercise lasting more than 2 h, successful performances are dependent upon various physiological, biomechanical and psychological factors. Variables such as maximum oxygen uptake (V02 max), the maximal fraction of $VO2_{max}$ that can be sustained and the energy cost of locomotion are often cited (e.g., O'Toole and Douglas 1995). At the end of a triathlon, recent studies have observed a

significant increase in energy cost that is related to a variation in some physiological or biomechanical parameters (Guezennec et al. 1996; Hausswirth et al. 1997).

The energy cost of running (C_r), defined as the amount of energy spent per unit distance (di Prampero 1986), depends upon both physiological and biomechanical factors, and several non-exclusive mechanisms have been proposed to explain the increase in C_r (e.g., Martin and Morgan 1992). Therefore, the issue of the effect of exercise duration on the increase in energy cost remains to be established in this area of research, which is essentially interdisciplinary. On the one hand, factors such as changes in running pattern and neuromuscular performance have been suggested to influence C_r through an alteration in running mechanics (Brisswalter et al. 1998; Hausswirth et al. 1997; Nicol et al. 1991). On the other hand, previous studies on long-distance events have implicated the role of physiological factors such as thermoregulation and substrate turnover in this increase in C_r (Bosch et al. 1990; Brisswalter et al. 1998; Bruckner et al. 1991; Guezennec et al. 1996). It has been suggested that muscle glycogen depletion, which occurs in active muscle fibers during prolonged exercise, affects C_r (Amstrong et al. 1977; Guezennec et al. 1996). Earlier studies have clearly shown that the significant alteration in C_r observed after a race longer than 15 km is concomitant with a significant decrease in the respiratory exchange ratio *(R)* to values close to 0.85. (Bosch et al. 1990; Brisswalter et al. 1998; Bruckner et al. 1991). This decrease indicates that a long exercise duration leads to glycogen depletion and an increase in free fatty acid oxidation (e.g., Ahlborg et al. 1974). Thus, about half of the demand for energy is accounted for by the oxidative breakdown of lipid substrates, and the low efficiency of lipid oxidation can partly explain the increase in C_r (e.g., Sahlin 1986).

Classically, it has been reported that glycogen sparing during prolonged exercise is linked with the metabolism of the exogenous supply of carbohydrate (CHO: e.g., Tsintzas et al. 1996). Ingesting a CHO solution during prolonged submaximal exercise delays the onset of fatigue and improves endurance performance, especially in exercise lasting 2 h or more (e.g., Coyle 1991). In addition, in some studies it has been observed that running performance is higher at the end of a 30-km run when subjects ingested a CHO-electrolyte solution than when they drank water (Tsintzas et al. 1995; Williams et al. 1990). To the best of our knowledge, the effect of CHO ingestion during exercise lasting longer than 1 h has been studied mostly for exercise of moderate intensity (<75% $_{VO2max)}$. Nevertheless, endurance triathletes have been reported to be able to run at high submaximal intensities VO2max) corresponding to their ventilatory threshold (VT), at the end of a triathlon (Sleivert and Rowlands 1996). The aim of the current investigation was therefore to compare the change in C_r (ΔC_r) of well-trained triathletes during a 2-h run completed at VT, when ingesting a CHO solution or a sweetened placebo solution (Pl).

Methods

Subjects

The subjects were ten well-trained triathletes who were competing regularly at inter-regional or national level. All subjects were experienced treadmill runners and were injury free. The physical characteristics of the subjects are given in Table 1. Before participating in this study, the subjects were fully informed about the protocol, and informed consent was obtained prior to all testing, according to local ethical committee guidelines.

Protocol

Each subject completed three testing sessions in a 3-week period. Subjects were asked to stop their training program 24 h before each testing session. The first session was used to determine VO_{2max} and oxygen uptake (VO2) at the VT. The second and the third sessions were composed of two submaximal treadmill runs, which were performed before and after an 80-min overground run. All of these submaximal tests were conducted at the velocity that elicited VT (v_{VT}). According to previous studies (Coyle 1991; Gisolfi 1996; Tsintzas et al. 1995, 1996), during these two sessions subjects had to ingest (in a random order), either an artificially sweetened PI drink, or a 5.5% CHO solution (glucose, fructose, malto-dextrins). The P1 and CHO solutions used contained the same concentration of

electrolytes (sodium: 20 mEq, potassium: 5 mEq). Furthermore, subjects were required to record their normal diet and to consume exactly the same diet for 2 days before each of the submaximal sessions.

VO2max

Session 1: evaluation of VO2max

VO2max was determined in a first session during an incremental protocol on a treadmill, according to the criteria described by Howley et al. (1995). After familiarization on the treadmill at 10 km • 11^{-1} and 3% grade, the speed of walking was increased by 1 km • h^{-1} every 1 min until the subject reached volitional exhaustion. $V0_{2\ max}$ was deemed to have been reached when the following criteria had been achieved: a plateau in VO_2, an *R* value of 1.15 or greater, and a post-exercise blood lactate level above 8 mmol • l^{-1}. The VT was estimated from the non-linearity of a plot of carbon dioxide output against VO_2 (Sherill et al. 1990).

Sessions 2 and 3: overground run and submaximal tests

During the second and the third sessions, an overground run was undertaken on a 400-m track very close to the laboratory. The running speed of each subject was checked and regulated every 400 m, so that it was kept constant over the 80-min period. Before and after this overground test, each subject completed a submaximal test. Before the first submaximal test (Test 1), the subjects performed a 10-min treadmill familiarization run at 10 km • h^{-1}.

After a 4-min rest period, the first 20-min, 0% grade submaximal run was carried out. The same test was performed immediately after the overground run (Test 2; Fig. 1). Throughout the test, the treadmill speed was controlled continuously by a photoelectric cell.

According to the study of Tsintzas et al. (1996), after the accommodation run, 8 ml • kg^{-1} body weight of fluid had been ingested, and thereafter 2 ml • kg^{-1} body weight was ingested every 20 min.

Experimental measurements and calculations

At rest and during the treadmill exercise, respiratory gases were collected using a breath-by-breath system (CPX Medical Graphics). The interval between measurement of respiratory parameters was set at 10 s. Five periods were analyzed: the 3rd (T_3), the 10th (T10), and the 20th (T_{20}) min of Test 1, and at the beginning (T_{100}) and the end (T_{120}) of Test 2. The analyzers were calibrated before and after each subject's session by using gases of known concentration. Heart rate (HR) was recorded continuously (Baumann 5000). Fingertip samples of arterialized capillary blood were obtained before exercise and immediately at the end of each submaximal test (T_{20} and T_{120}). The samples were analyzed for blood glucose concentration with an automated glucose oxidase method, and for lactate concentration (YSI, Yellow Spring Instruments). At the end of each submaximal treadmill test, the subjects were asked to report their rating of perceived exertion (RPE) using the Borg 15-grade scale (Noble and Robertson 1996). Body mass (BM) was measured before and immediately after the submaximal run in order to observe a possible mass loss at the end of the prolonged run.

For each period, the C_r was calculated from the VO_2 values recorded, according to the equation (di Prampero 1986): C_r [(VO2 - V_{02rest})v^{-1}) • 60], where C_r is expressed in ml • k-g^{-1} $km^{1,}$ VO_2 in ml • kg^{-1} • min^{-1}, and v in km • h^{-1}. VO_{2rest} is resting VO_2 and was recorded during the 4 min preceding T1; the last 2-min values were used.

Statistical analysis

All data are expressed as the mean (SD). Two-way analyses of variance (CHO vs Pl) with repeated measures were performed using C_r, minute ventilation (V_E), frequency of breathing (f_b), *R* (five periods), lactate concentration, plasma glucose concentration (three periods), RPE and BM (two periods) as dependant variables. Newman-Keuls post hoc tests were used to compare specific means.

Relationships between variables were analyzed using Spearmanrank correlation coefficients. For all of the statistical analyses, the level of significance was set at $P < 0.05$.

Results

The mean data for the maximal parameters recorded during the first session are presented in Table 1. During this test, VT occurred at 81.2 (2.5)% V02max and 16.5 (0.6) km • h^{-1}. Table 2 shows the mean Values of C_r V_E, f_b, and R measured at T3 and T120 for both conditions (Pl vs CHO). In our study, the first period (T_3) was used to control intra-individual variability (i.e., coefficient of variation, cv) in physiological parameters among sessions. The results show no significant variation in C_r or the other variables between sessions [for C_r: cv = 2.5 (1.4)%, $P > 0.05$].

A significant effect of period was found for Cr, whereas no significant interaction effect of CHO or Pl ingestion was observed. Whatever the condition, we have found a slight but significant increase in C_r, where ΔC_r, = $[(T_{120}—T_3)/T_3] \bullet 100$, at the end of the run (ΔC_r, = 5.7% and 7.01% for CHO and Pl, respectively). The mean Values of $V0_2$ represented 81.4% and 86.8% V02max.for T3 and $T_{120,}$ respectively < 0.05). Conversely, a significant interaction effect was found for R Values. At the end of the run, R decreased significantly only during the Pl trial (0.98 and 0.90, for T_3 and T_{120}, respectively), whereas a non-significant trend was observed during the CHO trial (T_3 vs T_{120}: 0.97 vs 0.95, respectively, $P > 0.05$).

During the first submaximal test, no significant difference was found between the C_r values calculated at T_3, T_{10} and T_{20}, and a significant increase in plasma glucose concentration was observed during the CHO trial at T_{20}.

Significant variations in C_r and R were observed at the beginning of the second test [ΔC_r (T_3 vs T_{100}) = 4.6% vs 4.7%, respectively, and ΔR (T_3 vs T_{100}) = -6.8% Vs -1.05%, respectively: Fig. 2]. Furthermore, a significant increase in V_E, HR, and f_b, were found with exercise duration, without any interaction effect of the solution ingested.

No effect of CHO ingestion was observed for RPE. Between the two submaximal tests, the RPE increased significantly from "very light" [9.7 (0.8) Vs 10.1 (0.9) for the Pl and CHO conditions, respectively] to "somewhat hard" [13.3 (1.2) vs 12.6 (0.8) for Pl and CHO trials, respectively]. Moreover, a significant increase in lactate concentration was observed between T_{20} and T_{120} (Table 3). Finally, for both conditions no significant changes in BM or plasma glucose concentration were found after the 2-h run.

The mean significant Variations expressed as a percentage of Test 1 [$\Delta = RT_{120} - T_3)/T_3] \bullet 100$} observed between the two tests were: ΔVE= 15.2 (12)%, Δf_b = 10.3 (9.8)%; ΔHR = 6.2 (4.6)%; ΔRPE = 30.6 (8.4)%; and Δ lactate concentration = 18 (5)%. No significant correlations were found between these variations, and a wide inter-individual variability in these changes was observed for C_r (range: -2% to 12%), V_E (range: -8% to 38%), and RPE (range: 18% to 44%).

Discussion

The main finding of this study was that the significant increase in C_r observed at the end of a 2-h run in well-trained triathletes seemed to occur independently of whether the CHO or Pl solution was ingested during exercise. This result can be compared with those of previous studies indicating similar results for exercise of a shorter duration (i.e., <90 min; Below et al. 1995; Sproule 1998) or lower intensities (i.e., <75% $VO_{2max;}$ Blomstrand et al. 1995; Mitchell et al. 1989; Tsintzas et al. 1996).

Like previous studies conducted with triathletes (Guezennec et al. 1996; Hausswirth et al. 1997), the present study examined the effect of a 2-h run on the Variability in C_r. While for a shorter exercise duration an increase in C_r is not always reported to occur (e.g., Bruckner et al. 1991; Dressendorfer 1991; Sproule 1998), this increase seems to occur systematically when the exercise duration is longer than 90 min (e.g., Bruckner et al. 1991; Guezennec et al. 1996; Hausswirth et al. 1997; Tsintzas et al.

1996 Xu and Montgomery 1995). In our study, the mean increase in C_r increase ranged from 5.7% to 7.01%. These Values are in agreement with those reported previously. Bruckner et al. (1991) observed a 5% increase in C_r at the end of a marathon, and Guezennec et al. (1996) and Hausswirth et al. (1997) noted a 7% increase in C_r in triathletes after a 135-min run.

Most of these studies have associated the increase in C_r observed at the end of a long-duration running exercise with glycogen depletion and the progressive use of the fatty acids to replace CHO as an energy source. Classically, it has been suggested that glycogen depletion in working muscle is related to fatigue in athletes during exercise ranging in duration and intensity from 1 to 3 h and 65% to 85% V^02 max respectively (e.g., Coyle 1991). Within this framework, CHO feedings during exercise improved endurance performance and delay, but did not prevent fatigue (e.g., Coyle et al. 1986). It has been suggested that during prolonged cycling exercise, CHO ingestion maintains plasma glucose concentrations late in exercise. Subjects would therefore be able to maintain a higher exercise intensity at the end of exercise (Coggan and Coyle 1991). In the present study, no hypoglycemic state was observed at the end of exercise during any of the treatments, and no effect of treatment was observed on plasma glucose concentration after 2 h. This is in agreement with the work of Tsintzas et al. (1996), who found no significant difference between a P1 and a CHO trial from 40 min into a 2-h run [132 (12.3) min]. For running events, the effect of CHO ingestion on endurance response and/or glycogen sparing seems less clear, and is different than for cycling (e.g., Derman et al. 1996). For example, an improvement in endurance running performance is not systematically observed with CHO ingestion (Noakes et al. 1988). Furthermore, it has been suggested that glycogen depletion, and thus the importance of CHO availability, is less significant if the duration of the run is not longer than 2 h (e.g., Coggan and Coyle 1991). It is therefore possible that in well-trained athletes, a 2-h run is not long enough to observe, on the one hand, a significant decrease in whole-muscle glycogen levels and, on the other hand, a significant effect of CHO ingestion on glycogen sparing. This assumption may be illustrated by the higher, but not significant, variation in C_r observed during the P1 trial when compared to the CHO trial between T_{100} and T_{120} [ΔC_r, (T_{120}-T_{100}) = 2.5% and 1.2% for the Pl and CHO trials, respectively]. Furthermore, we observed a significant decrease in *R* from T_{100} to T_{120} during the P1 trial only. Classically, a decrease in *R* is reported to occur during prolonged exercise, leading to a lower energy equivalent of $V0_2$. This decrease is often used to explain increases in C_r that occur as a result of the effect of substrate turnover. When this effect is slight, earlier results have indicated that the variability in C_r could be independent of the reduction in *R.* For example, it has been reported that running economy could remain constant for 1 day after a 30-min run at 85% V02max despite a lower *R* (Morgan et al. 1990). In the study presented here, we could make the hypothesis that in well-trained athletes, during a 2-h run the decrease in *R* was not sufficient to be associated with a significant rise in C_r. These results seem, therefore, to minimize the role of metabolic changes in the increase in C_r that occurs during high-intensity submaximal runs that range in duration from 1 to 2 h (e.g., Sproule 1998).

In the present study, several other factors could be responsible for the increase in C_r. For one, we observed a systematic increase in the relative intensity of exercise with exercise duration, with a variation in the mean values of VO_2 that represented from 81.4% to 86.8% $VO_{2\ max}$ • Furthermore, we have found a significant increase in lactate concentration and in RPE between the submaximal tests. It is well known that when exercise is associated with a concomitant lactic acid accumulation (heavy intensity), a slow rise in VO_2, known as the VO_2 slow component, occurs during constant-intensity prolonged exercise (e.g., Barstow 1994; Poole and Richardson 1997). While no significant effect of exercise duration on C_r was found between the 3rd and the 20th min of exercise, the continuous additional rise in C_r observed from the 100th min could be related to the mechanism underlying the VO_2 slow component. Several factors, such as the work of breathing, thermal stress, or the role of the exercising muscle, could contribute to this phenomenon (Casabury et al. 1989; Poole 1994). On the one hand, thermal stress could be involved in the increase in V_E observed at the end of the 2-h run (Hanson et al. 1982). Therefore, an increase in the work of breathing could be responsible for one part of the VO_2 slow component. For mean group values, our results indicate a significant increase of V_E at the end of the test. Based on the equation proposed by Coast et al. (1993), the VO_2 demand estimated from the increase in the work of breathing represents, in our study, 39.5 (0.5) $m10_2$ • min^{-1} and, therefore, could only explain 11% and 17% of the increase in C_r observed in the Pl and CHO trials, respectively. When individual adaptations are considered, no significant correlations were found between increases in V_E and increases in C_r (Fig. 3).

On the other hand, biomechanical factors are often attributed to the VO_2 slow component. Poole (1994) suggested the important link between changes in the pattern of activity of the working muscle and the additional increase in V^02. Prolonged exercise is commonly associated with changes in electromyographic activity and/or changes in fiber recruitment. For instance, progressive increases in exercise intensity toward "heavy intensity" exercise would lead to a larger recruitment of type II fibers. Therefore, the resulting decrease in thermodynamic muscle efficiency (e.g., Woledge 1998) could explain, at least in part, the increase in the energy cost of locomotion. Furthermore, recent studies have observed that the reduction in neuromuscular performance previously reported to occur during prolonged cycling exercise (e.g. Capelli et al. 1993) is more relevant after a marathon than after a triathlon (Hausswirth et al. 1997; Lepers and Hausswirth 1998). While the relationship between increases in C_r and mechanical changes with fatigue remains unclear, a major portion of the alteration in C_r could be attributed to the increase in mechanical constraints (i.e., eccentric muscle work) that exists during prolonged running at high, but submaximal intensity.

In conclusion, the results of this study confirm that C_r increases during a 2-h run. The increase in C_r observed here was not influenced by CHO ingestion during exercise or by a reduction in *R*. These results appear to minimize the role of metabolic changes or increases in the work of breathing during prolonged exercise at 80% $V0_{2\ max}$ suggesting (indirectly) that the increase in C_r observed in our well-trained subjects can be explained mainly by a decrease in muscle efficiency.

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Figure 1 Experimental protocol. *(v_{VT}* - Ventilatory threshold)

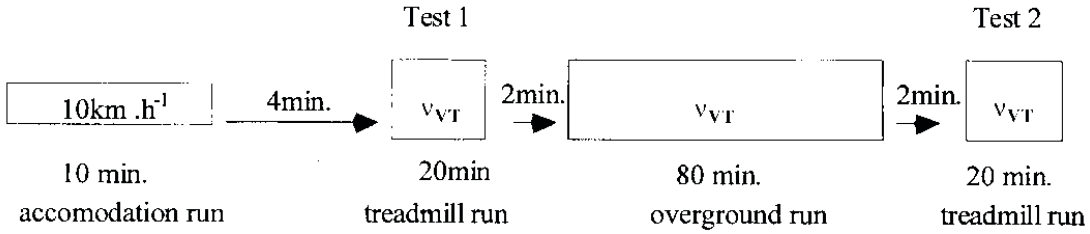

Figure 2 Mean group changes in the energy cost of running *(C_r,* left ordinate) and respiratory exchange ratio *(R,* right ordinate) during the carbohydrate ingestion *(CHO)* and the placebo ingestion *(Pl)* trials

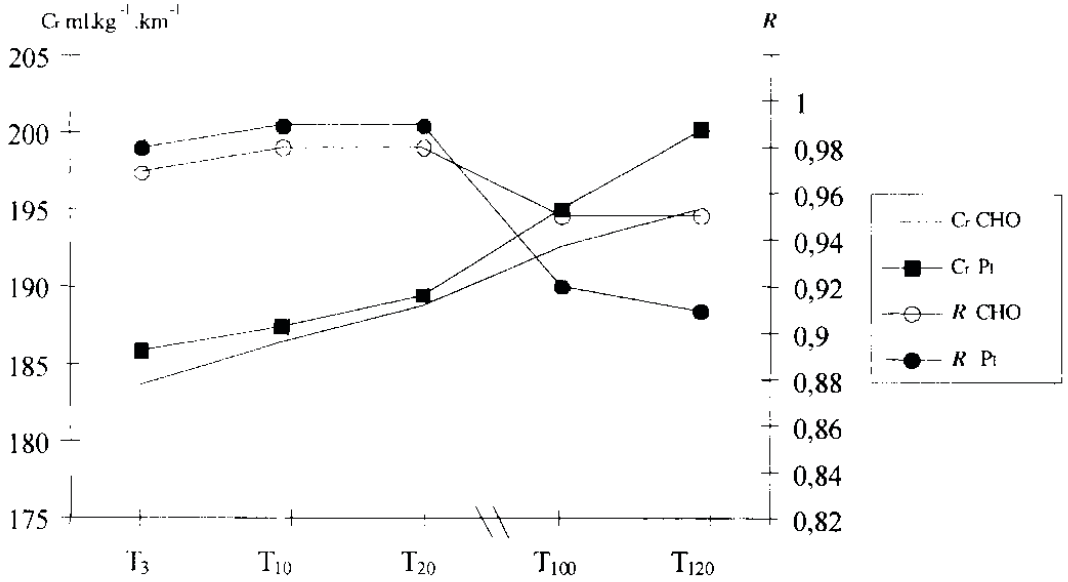

Figure 3 *Scatter plot* for change in C_r (ΔC_r) and change in minute ventilation (ΔV_E) between T_3 and T_{120}

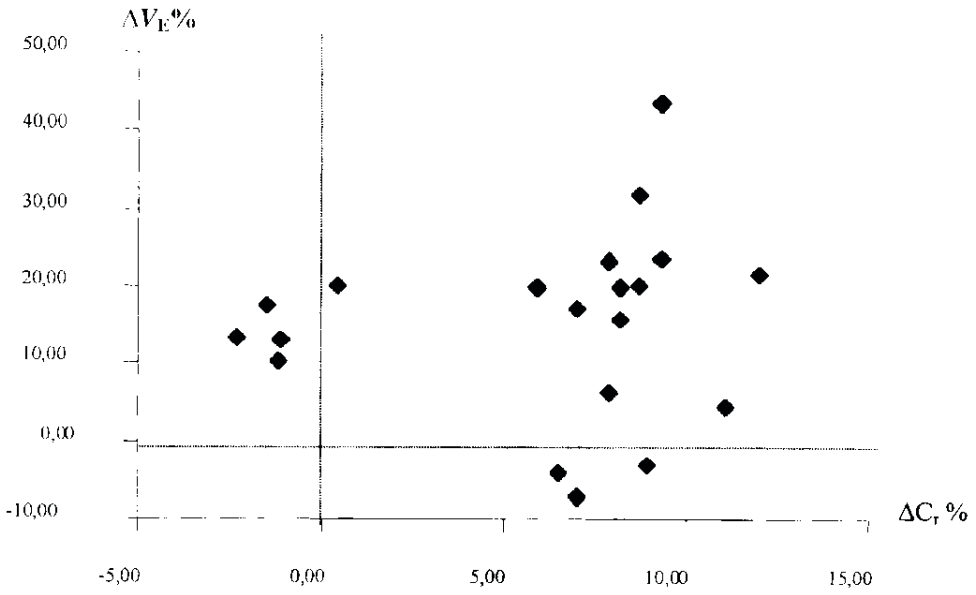

Table 1 Physical characteristics and maximal physiological data of the subjects *(n* = 10). *(V02ma$_x$* Maximum oxygen uptake, *H Rmax* maximum heart rate, v_{max} maximum velocity)

Parameter	Mean (SD)	Minimum	Maximum
Age (years)	24.5 (3.2)	21	31
Body mass (kg)	70.2 (7.9)	61	86
Height (cm)	179.7 (7.8)	173	195
VO2max (m1-kg^{-1} • min^{-1})	66.8 (3.9)	59.2	73.4
HR_{max} (beats • min^{-1})	183 (9.7)	177	201
v_{max} (km • h^{-1})[a]	20.1 (1.45)	18	22

[a] V_{max} represents the speed that elicits VO_{2max}.

Table 2 Mean group values of the energy cost of running (Cr), minute ventilation *(VE),* respiratory frequency (f_b) and respiratory exchange ratio *(R),* recorded at the 3rd min of Test 1 (T_3) and at the end of Test 2 (T_{120}), while ingesting either a placebo (Pl) or a carbohydrate (CHO) solution

Variable	Solution	T_3		T_{120}	
		Mean	SD	Mean	SD
Cr (ml • kg^{-1} • km^{-1})	Pl	185.9	8.7	200.6*	9.2
	CHO	183.5	9.7	195.1*	9.1
V_E, (1 • min^{-1})	P1	92.1	15.6	112.1*	17.7
	CHO	92.2	13.8	107.8*	14.8
f_b (cycles • min^{-1})	P1	42.4	3.5	46.9*	4.1
	CHO	42.1	3.7	46.1*	4.2
R	Pl	0.98	0.02	0.90*	0.04
	CHO	0.97	0.03	0.95**	0.03

* Significant difference between T_3 and T_{120}; ** significant difference between trials (Pl vs CHO)

Table 3 Parameters recorded at rest and at the end of each submaximal test exercise while ingesting either a Pl or a CHO solution

Parameters	Solution	Rest		T_{20}		T_{120}	
		Mean	SD	Mean	SD	Mean	SD
Body mass (kg)	Pl	70.2	5.9			69.5	5.4
	CHO	70.2	5.9			69.7	6.2
Lactate concentration (mmol • 1^{-1})	P1	1.1	0.1	1.8	0.2	4.2*	0.5
	CHO	0.9	0.1	1.6	0.2	3.9*	0.4
Plasma glucose (mmol • 1^{-1})	Pl	4.7	0.2	5.1	0.2	4.9	0.4
	CHO	4.9	0.3	5.8*,**	0.3	5.1*	0.5

* Significant difference between periods; ** significant difference between trials (Pl vs CHO)