Effects of carbohydrate ingestion 15 min before exercise on endurance running capacity

Savvas P. Tokmakidis and Ioannis A. Karamanolis

Abstract: This study examined the effects of pre-exercise carbohydrate ingestion on exercise metabolism and endurance running capacity. Eleven active subjects ($VO_{2\ max}$ 49.0 ± 1.7 mL·kg^{-1}·min^{-1}, mean ± SE) performed two exercise trials 15 min after ingesting glucose (G; 1 g·kg body mass^{-1}) and placebo (CON). Each subject ran on a level treadmill for 5 min at 60%, 45 min at 70%, and then at 80% of $VO_{2\ max}$ until exhaustion. Serum glucose and plasma insulin reached their peak concentrations ($p < 0.01$) 15 min after glucose ingestion and declined at the onset of exercise. Serum glycerol concentrations were lower ($p < 0.01$) in the G trial than in the CON trial after 30 min of exercise to exhaustion. In addition, after 45 min of exercise to exhaustion, the levels of free fatty acids were lower in G than in CON ($p < 0.05$). No differences were observed in carbohydrate oxidation rates during exercise between treatments (G, 2.53 ± 0.08 g·min^{-1}; CON, 2.40 ± 0.09 g·min^{-1}). Time to exhaustion was 12.8% longer in G ($p < 0.01$) than in CON. These results suggest that glucose ingestion 15 min before prolonged exercise provides an additional carbohydrate source to the exercising muscle, thus improving endurance running capacity.

Key words: glucose, insulin, pre-exercise meal, metabolism, exhaustion.

Résumé : Dans cette étude, on analyse les effets de l'apport de sucre avant un exercice sur le métabolisme énergétique et l'endurance à la course. Onze sujets physiquement actifs ($VO_{2\ max}$, 49,0 ± 1,7 mL·kg^{-1}·min^{-1}, moyenne ± erreur-type) participent à deux séances d'exercice 15 min après avoir consommé du glucose (G; 1 g·kg de masse corporelle^{-1}) ou un placebo (CON). Tous les sujets courent durant 5 min sur un tapis roulant à une intensité correspondant à 60 % du $VO_{2\ max}$, durant 45 min à une intensité correspondant à 70 % du $VO_{2\ max}$, puis à 80 % du $VO_{2\ max}$ jusqu'à'épuisement. Quinze minutes après la consommation du glucose, les concentrations sériques de glucose et plasmatiques d'insuline passent par un pic ($p < 0,01$) puis diminuent au début de l'exercice. Trente minutes après le début de l'exercice jusqu'à épuisement, les concentrations sériques de glycérol sont moins importantes ($p < 0,01$) chez G que chez CON. De plus, 45 min après le début de l'exercice jusqu'à épuisement, les concentrations des acides gras libres sont plus faibles ($p < 0,05$) chez G que chez CON. D'un groupe à l'autre, on n'observe aucune différence du taux d'oxydation des sucres (G, 2,53 ± 0,08 g·min^{-1}; CON, 2,40 ± 0,09 g·min^{-1}). Le temps de performance jusqu'à épuisement est de 12,8 % plus long ($p < 0,01$) chez G que chez CON. D'après ces observations, l'apport de glucose 15 min avant le début d'un exercice procure une source additionnelle d'énergie pour les muscles sollicités et améliore l'endurance à l'effort.

Mots-clés : glucose, insuline, repas préexercice, métabolisme, épuisement.

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Introduction

Carbohydrate (CHO) ingestion before exercise has been shown to affect metabolic response, substrate utilization during performance, and exercise time to exhaustion. Consumption of glucose 30 to 60 min before strenuous exercise is associated with hyperinsulinaemia. In combination with exercise-induced muscle glucose uptake, it results in a rapid fall in blood glucose at the onset of exercise (Costill et al. 1977; Hargreaves et al. 1985). Under these conditions, glucose ingestion before exercise can induce an increase (Costill et al. 1977; Hargreaves et al. 1985), a decrease (Levine et al. 1983), or even no effect (Devlin et al. 1986; Fielding et al. 1987; Koivisto et al. 1985) on muscle glycogen utilization. Furthermore, it presents an increase (Gleeson et al. 1986; Sherman et al. 1991), a reduction (Costill et al. 1977; Foster et al. 1979), or has no effect (Chryssanthopoulos et al. 1994; Decombaz et al. 1985; Hargreaves et al. 1987; Tokmakidis and Volaklis 2000) on exercise time to exhaustion. These variable results may be explained, in part, by differences among subjects, such as hyperglycemia, hyperinsulinaemia, contractive activity on muscle glucose uptake, hepatic glucose output, and the ongoing absorption of the ingested glucose (Marmy-Conus et al. 1996).

More data is, therefore, required on the consumption of CHO shortly before the onset of running exercise. Although running is a common and popular activity, before now, cycling was the mode of exercise in which CHO ingestion has previously been studied. For example, Palmer et al. (1998) found that the ingestion of approximately 40 g of carbohydrate 10 min before a 20 km time-trial lasting approximately 30 min did not improve performance. Furthermore, 10 trained cyclists consumed 5.0 mL·kg^{-1} body mass of a 19.7% carbohydrate drink and immediately performed a

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Table 1. Subject characteristics (n = 11).

Age (y)	Body mass (kg)	Height (cm)	$VO_{2\ max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	Body fat (%)
25.3±2.4	74.2±1.8	174.1±2.5	49.0±1.7	14.4±1.5

Note: Values are presented as means ± SE; $VO_{2\ max}$, maximal oxygen consumption.

15 min controlled warm-up, followed by four 1.6 km repeated bouts of high-intensity cycling, and observed increases in blood glucose without any effects on performance time (Snyder et al. 1993). In addition, Nishibata et al. (1993) found that the ingestion of 45 g of glucose 15 min before the start and at 15 and 45 min of cycling exercise at an intensity of 75% $VO_{2\ max}$ until exhaustion did not improve endurance performance.

No study has yet reported the effects of glucose ingestion 15 min before prolonged treadmill running on exercise metabolism and endurance running capacity. Therefore, the aim of this study was to examine the effects of pre-exercise glucose ingestion on exercise metabolism and endurance running capacity during prolonged treadmill exercise in a manner that simulates endurance racing, where runners maintain a constant pace for most of the race and then increase their speed towards the finishing line. It should be noted that running involves greater muscle mass and has higher metabolic demands than cycling.

Materials and methods

Subjects

Eleven recreational runners (10 males and 1 female) volunteered to participate in this study (Table 1). The experimental procedures and risks were explained in detail and a written consent was obtained prior to the experiments. To participate in the study, insulin sensitivity and insulin secretion were assessed using the oral glucose tolerance test (OGTT), and all subjects had a normal response to the OGTT. This study was approved by the Ethical Committee of Democritus University of Thrace.

Measurement of maximal oxygen consumption ($VO_{2\ max}$)

$VO_{2\ max}$ was measured on a treadmill (Erich Jaeger, LE 6000, Wuerzburg, Germany) using an incremental running protocol to exhaustion. The starting treadmill speed was 11 $km \cdot h^{-1}$ and was increased 2 $km \cdot h^{-1}$ every 2 min until exhaustion. A stable inclination of 1% was used throughout the trial. Expired air was continuously analyzed for oxygen consumption and carbon dioxide production using an Oxycon Champion Analyzer (Erich Jaeger, IEC 601-1). The analyzer was calibrated before each test using commercially prepared gas mixtures and a standard air volume pump. The criteria used to determine the attainment of $VO_{2\ max}$ were (*i*) a levelling off of VO_2 (<1.8 $mL \cdot kg^{-1} \cdot min^{-1}$ increase) with an increase of work rate, (*ii*) a heart rate (HR) within 10 $beats \cdot min^{-1}$ of predicted maximum, and (*iii*) a respiratory exchange ratio (RER) > 1.1. All subjects met these criteria. In addition, body mass, height, and percent body fat were measured using the equation of Jackson and Pollock (1978).

Familiarization trial

The information obtained after the $VO_{2\ max}$ test was used to determine the intensity of exercise (60%, 70%, and 80% of $VO_{2\ max}$) for the experimental trials. Before the experimental trials, subjects performed a 45 min submaximal treadmill test (60% for 5 min, 70% for 30 min, and 80% of $VO_{2\ max}$ for 10 min) to determine the relationship between running speed and oxygen uptake, as well as to secure the chosen intensity used in the running protocol. This test also enabled the subjects to become familiar with the treadmill and the experimental procedure of taking VO_2 measurements while they were running. Blood samples were not obtained during this trial and no subject ran to exhaustion.

Table 2. Exercise time based on treatment and order (n = 11).

	Time (min)		
Trial	Mean ± SE	Minimum	Maximum
Glucose	83.0±4.4*	57	110
Placebo	73.6±5.2	59	105
1st run	78.9±4.7	57	105
2nd run	77.7±5.4	59	110

*Significantly different from placebo trial ($p < 0.01$).

Experimental trials

Each subject participated in two running trials on a motorized treadmill to exhaustion. The trials were given in random order at an interval of at least 1 week. Glucose (G; 1 g·kg body $mass^{-1}$) and placebo (CON; aspartame 0.05 g·kg of body $mass^{-1}$) solutions were ingested 15 min before the onset of exercise. An appropriate quantity of water was added so that the volume of each solution was 400 mL to minimize differences in gastric emptying. Subjects refrained from tobacco, alcohol, and caffeine for 24 h and from physical exercise for 48 h before the experimental trials. Instructions were given to the subjects to record food intake and activity during the 48 h period prior to the experimental trial.

Experimental protocol

Each subject arrived at the laboratory in the morning after an overnight fast. After resting for 15 min, a 5 min expired air sample was measured. A venous catheter was then inserted into an antecubital vein and a fasting blood sample was drawn. The subjects drank the solution (glucose or placebo in different sessions) and remained seated for 15 min. Resting expired air and a blood sample were obtained at the end of this period. Subjects then ran at 60% $VO_{2\ max}$ for 5 min, at 70% for 45 min, and at 80% $VO_{2\ max}$ until exhaustion. Blood samples were taken every 15 min with the subjects standing on the treadmill, and the final venous blood sample was taken at exhaustion. The interrupted time (~1 min for each blood sample) was excluded from the calculation of total running time. Expired air samples of 5 min

Table 3. Effects of exercise to exhaustion on physiological parameters.

	Time (min)						
Trial	–15	0	15	30	45	60	Exhaustion
VO_2 (mL·kg^{-1}·min^{-1})							
Glucose	3.3±0.1	3.5±0.1	34.2±1.2	34.9±1.3	35.4±1.3	38.7±1.2	40.1±1.3
Placebo	3.3±0.2	3.5±0.2	34.1±1.4	35.1±1.4	35.4±1.4	38.8±1.6	38.8±1.5
RER							
Glucose	0.86±0.02	0.88±0.02	0.93±0.01	0.92±0.01	0.92±0.01	0.94±0.01	0.94±0.01
Placebo	0.86±0.03	0.87±0.02	0.91±0.01	0.90±0.01	0.90±0.01	0.94±0.01	0.94±0.01
Heart rate (beats·min^{-1})							
Glucose	62±1.2	78±2.3	157±3.5	163±3.6	168±3.9	180±3.3	186±2.8
Placebo	61±0.9	68±6.4	153±3.3	160±3.6	167±4.0	179±3.2	183±2.6

Note: Values are means ± SE. VO_2, O_2 consumption; RER, respiratory exchange ratio.

were measured every 15 min during the run; the last expired air sample at exhaustion was measured for at least 3 min. Heart rate was continuously monitored in 5 s intervals using short-range telemetry (Polar, Finland). Carbohydrate and fat oxidation rates were calculated using VO_2, VCO_2, and RER values (Frayn 1983). During exercise, subjects were allowed to drink water to prevent the effects of dehydration. The subjects were encouraged to continue running for as long as possible without being shown the time. Exhaustion was defined as the time at which the volunteers were no longer able to maintain the proportional running speed.

Blood analyses

Eight millilitres of blood was obtained for each sample. The cannula was kept patent with a heparin–saline solution (5 IU·mL^{-1}), which was administered after each sample collection. A small amount of whole blood (0.2 mL) was immediately placed in a tube containing 0.4 mL trichloroacetic acid and centrifuged (UNIVERSAL 16) at 2500 r·min^{-1} (1475*g*) for 15 min at room temperature. The supernatant was transferred to another tube and frozen at –80 °C until it was analyzed later for lactate concentration using an enzymatic spectrophotometric technique (procedure No. 826-UV, Sigma Chemical Co., St Louis, Mo.). Three millilitres were drawn into an EDTA blood collection tube and centrifuged at 2500 r·min^{-1} (1475*g*) for 20 min at room temperature. Plasma was removed, divided into storage tubes, and frozen at –80 °C until it was analyzed later for insulin concentrations. Plasma insulin concentrations were measured using an enzyme immunoassay (EIA) technique (DRG Instruments GmbH, Insulin ELISA, Germany) by the spectrophotometric method (Reader 2001, Anthos, Salzburg, Austria). The remaining 5 mL of blood was centrifuged at 2500 r·min^{-1} (1475*g*) for 20 min at room temperature and the serum was drawn off, separated into 4 aliquots, and frozen at –80 °C until analyzed for glucose, glycerol, and free fatty acid (FFA) concentrations. Serum glucose concentrations were determined using enzymatic assays (procedure No. 510-UV, Sigma Chemical Co., St. Louis, Mo.). Serum glycerol concentrations were measured using enzymatic assays (procedure No. 337-UV, Sigma Chemical Co.), and serum FFAs were determined using an enzymatic colorimetric method (Wako Chemicals GmbH kit, Neuss, Germany). Lactate, glucose, glycerol, and FFA concentrations were determined by the spectrophotometric method (Hitachi U-2001).

Statistical analyses

Analysis of variance for repeated measures was used to evaluate changes over time for time-dependent variables. When analysis revealed significant main effects, Tukey's post-hoc test was used to locate any significant differences between means. In addition, data of time to exhaustion were analyzed using non-parametric paired *t* tests for the determination of differences of between-conditions measurements. The computer software program STATISTICA Software package (StatSoft Inc., Tulsa, Okla.) was used to complete these analyses. Statistical significance was set at $p < 0.05$. All data were expressed as mean ± SE.

Results

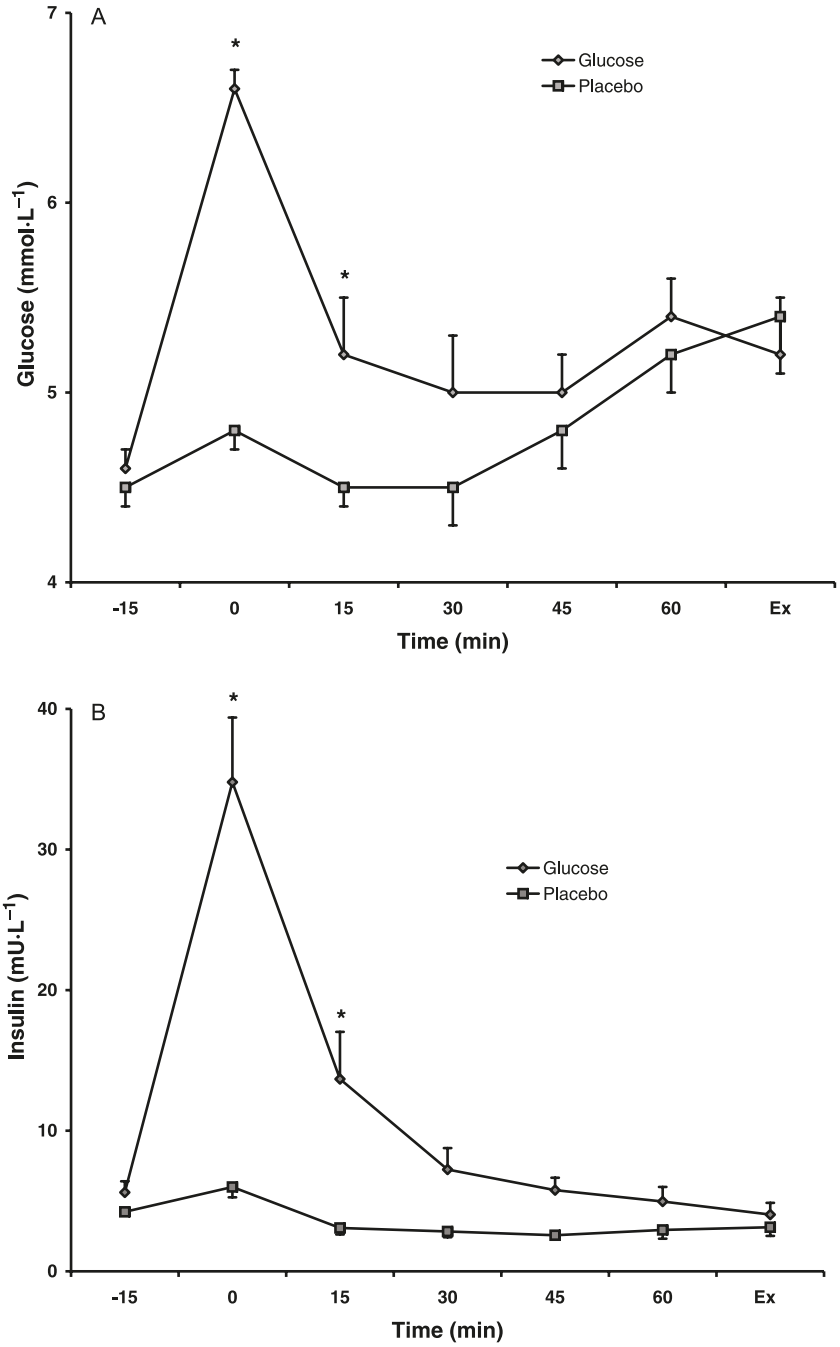
Endurance time, oxygen consumption, respiratory exchange ratio, and heart rate

The endurance time was higher in the G trial than in the CON trial ($p < 0.01$; Table 2). During the glucose trial, subjects ran 14.47 ± 0.98 km, but ran 12.9 ± 1.14 km during the placebo trial. Subjects were able to exercise for a longer period of time (12.8%) during the G trial. Three of them, however, ran for a longer time during the CON trial. Nevertheless, glucose and placebo trials were counterbalanced and there were no order effects between the first and the second running trials (Table 2). After 45 min of exercise, when the intensity was increased to 80% $VO_{2\ max}$, subjects ran 6.13 ± 0.89 km (33.0 ± 4.4 min) during the glucose trial but only ran 4.52 ± 1.07 km (23.6 ± 5.2 min) during the placebo trial. Oxygen consumption (VO_2) during exercise was not different between the two trials (Table 3). Furthermore, no differences were observed between trials for mean respiratory exchange ratio (RER) and heart rate values during exercise (Table 3).

Serum glucose and plasma insulin

Serum glucose levels were significantly higher ($p < 0.01$) during the G trial (6.6 ± 0.1 mmol·L^{-1}) than during the CON trial (4.8 ± 0.1 mmol·L^{-1}) at rest, 15 min after ingestion (Fig. 1). After a sharp post-prandial rise, a sharp decline of glucose was observed 15 min after the onset of exercise in the G trial (5.2 ± 0.3 mmol·L^{-1}); however, serum glucose concentrations were still higher in the G trial ($p < 0.01$) than in the CON trial (4.5 ± 0.1 mmol·L^{-1}). There were no significant differences in serum glucose levels between the

Fig. 1. Serum glucose and plasma insulin concentrations during rest and submaximal exercise for placebo and glucose trials. Values are means ± SE (n = 11). Time points at which values are significantly different from placebo ($p < 0.05$) are marked with an asterisk (*).

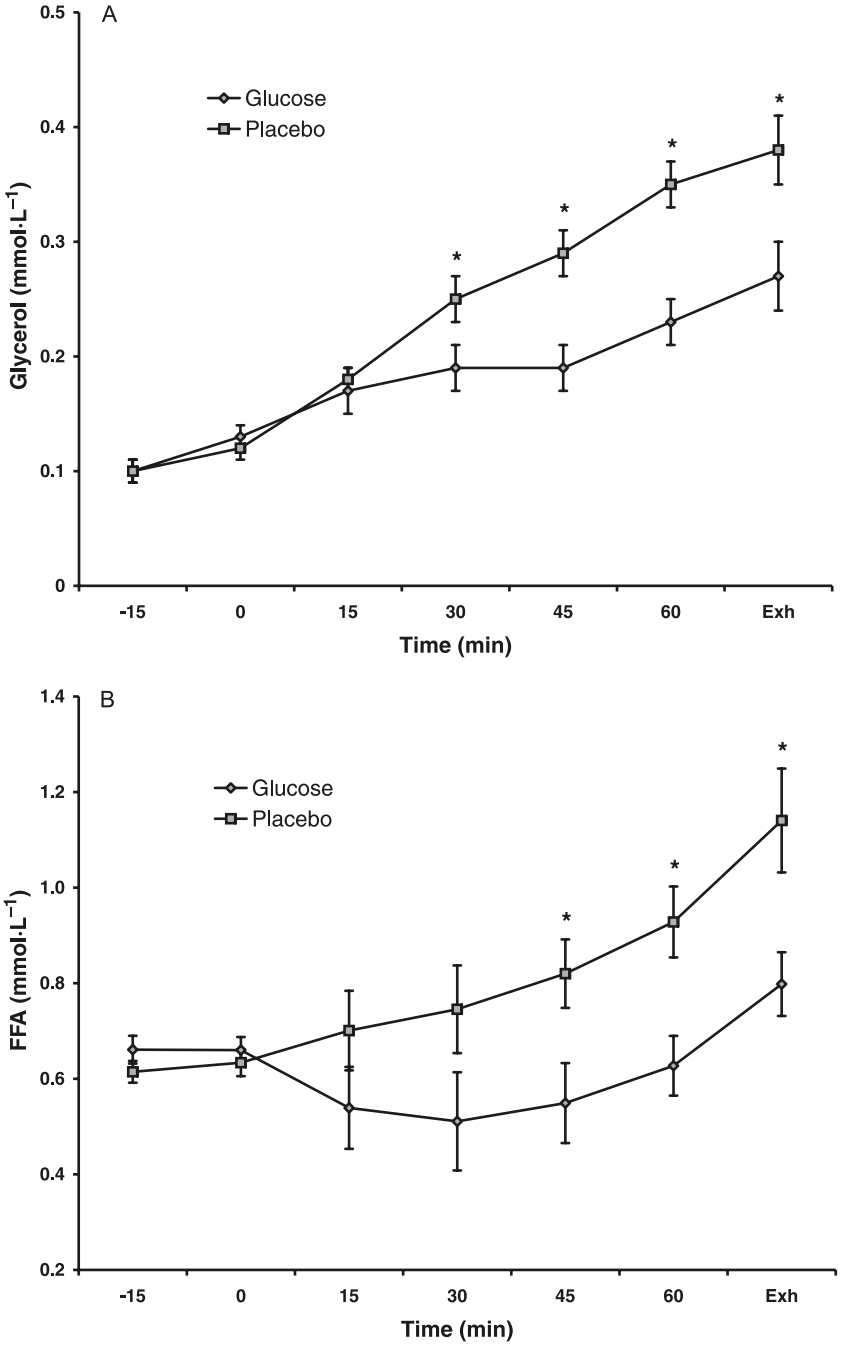


two trials from 30 min until exhaustion. Fifteen minutes after glucose ingestion, plasma insulin concentrations rose rapidly (34.8 ± 4.6 mU·L^{-1}), but 15 min after the onset of exercise, a sharp decline was observed (13.7 ± 3.4 mU·L^{-1}) (Fig. 1). Moreover, insulin levels were similar in the G and CON trials from 30 min until exhaustion (Fig. 1).

Serum glycerol and serum FFAs

Serum glycerol levels rose gradually during each trial throughout the exercise periods (Fig. 2). Glycerol levels were significantly higher ($p < 0.05$) from 30 min (0.25 ± 0.02 mmol·L^{-1}) until exhaustion (0.38 ± 0.03 mmol·L^{-1}) during the CON trial as compared with the G trial (0.19 ± 0.02 mmol·L^{-1} at 30 min, and 0.27 ± 0.03 mmol·L^{-1} at exhaustion, respectively). Similarly, serum FFA concentrations in the CON trial rose gradually throughout the exercise trial (Fig. 2). However, during the G trial, the levels of FFAs declined during the first minutes of exercise and rose gradually from 45 min (0.56 ± 0.08 mmol·L^{-1}) until exhaustion (0.80 ± 0.07 mmol·L^{-1}). Serum FFA concentrations in the G trial were significantly lower ($p < 0.05$) in comparison with the CON trial from 45 min until exhaustion (0.56 ± 0.08 vs. 0.86 ± 0.07 mmol·L^{-1} at 45 min, 0.61 ± 0.07 vs. 0.93 ± 0.07 mmol·L^{-1}

Fig. 2. Serum glycerol and serum free fatty acid (FFA) concentrations during rest and submaximal exercise for placebo and glucose trials. Values are means ± SE (n = 11). Time points at which values are significantly different from placebor ($p < 0.05$) are marked with an asterisk (*).



at 60 min, and 0.80 ± 0.09 vs. 1.15 ± 0.1 mmol·L^{-1} at exhaustion, for G and CON trials, respectively).

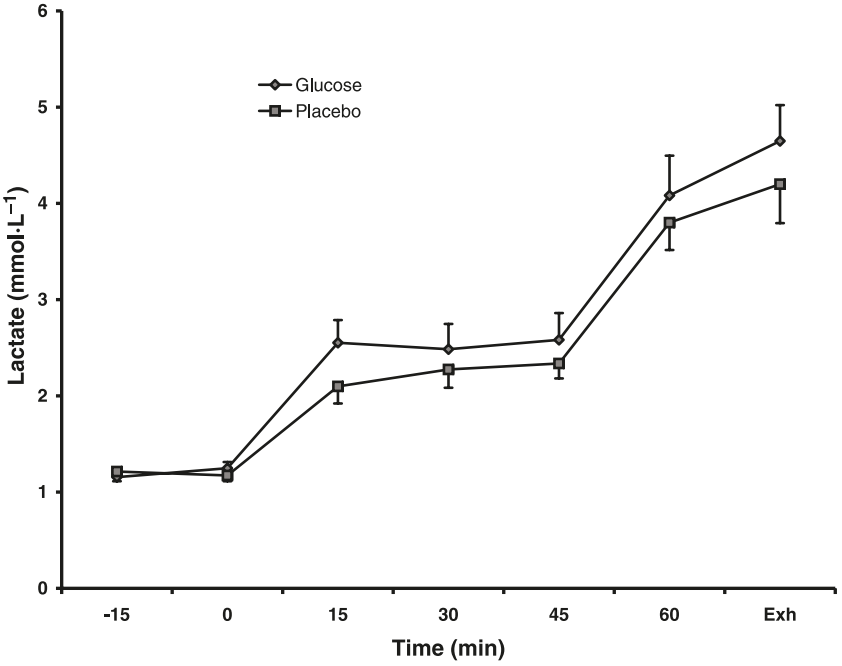
Blood lactate

Blood lactate concentrations increased in two different stages of exercise ($p < 0.05$): at the onset of exercise (intensity 70% $VO_{2\ max}$) and after 50 min of exercise when subjects ran at 80% $VO_{2\ max}$ (at 60 min of exercise: 4.08 ± 0.4 mmol·L^{-1} for G, and 3.80 ± 0.3 mmol·L^{-1} for CON) (Fig. 3). Throughout the exercise period, lactate levels tended to be slightly higher after glucose ingestion, but were never significantly different from the CON trial.

Carbohydrate and fat oxidation

The mean carbohydrate and fat oxidation rates increased significantly ($p < 0.05$) in two conditions at the onset of exercise (70% $VO_{2\ max}$), and were then stabilized. After 50 min, when the intensity of exercise was at 80% $VO_{2\ max}$, carbohydrate oxidation continued to increase ($p < 0.05$), but fat oxidation declined ($p < 0.05$) (Fig. 4). During exercise, the carbohydrate oxidation rates at the G trial were higher

Fig. 3. Lactate concentrations during rest and submaximal exercise for placebo and glucose trials. Values are means ± SE (n = 11).



($p < 0.05$) only at 45 min (2.34 ± 0.09 g·min^{-1}), compared with the CON trial (2.11 ± 0.09 g·min^{-1}) (Fig. 4). On the other hand, when the intensity of exercise was at 70% $VO_{2\ max}$, fat oxidation rates at the G trial were significantly lower ($p < 0.05$) only at 45 min (0.38 ± 0.02 g·min^{-1}), compared with the CON trial at the same timepoint (0.47 ± 0.04 g·min^{-1}) (Fig. 4). There was no difference in mean values throughout exercise between the G (2.53 ± 0.08 g·min^{-1}) and CON (2.40 ± 0.09 g·min^{-1}) trials in carbohydrate oxidation, but the mean values of fat oxidation were lower in the G trial (0.34 ± 0.03 g·min^{-1}) than in the CON trial (0.39 ± 0.03 g·min^{-1}) ($p < 0.05$).

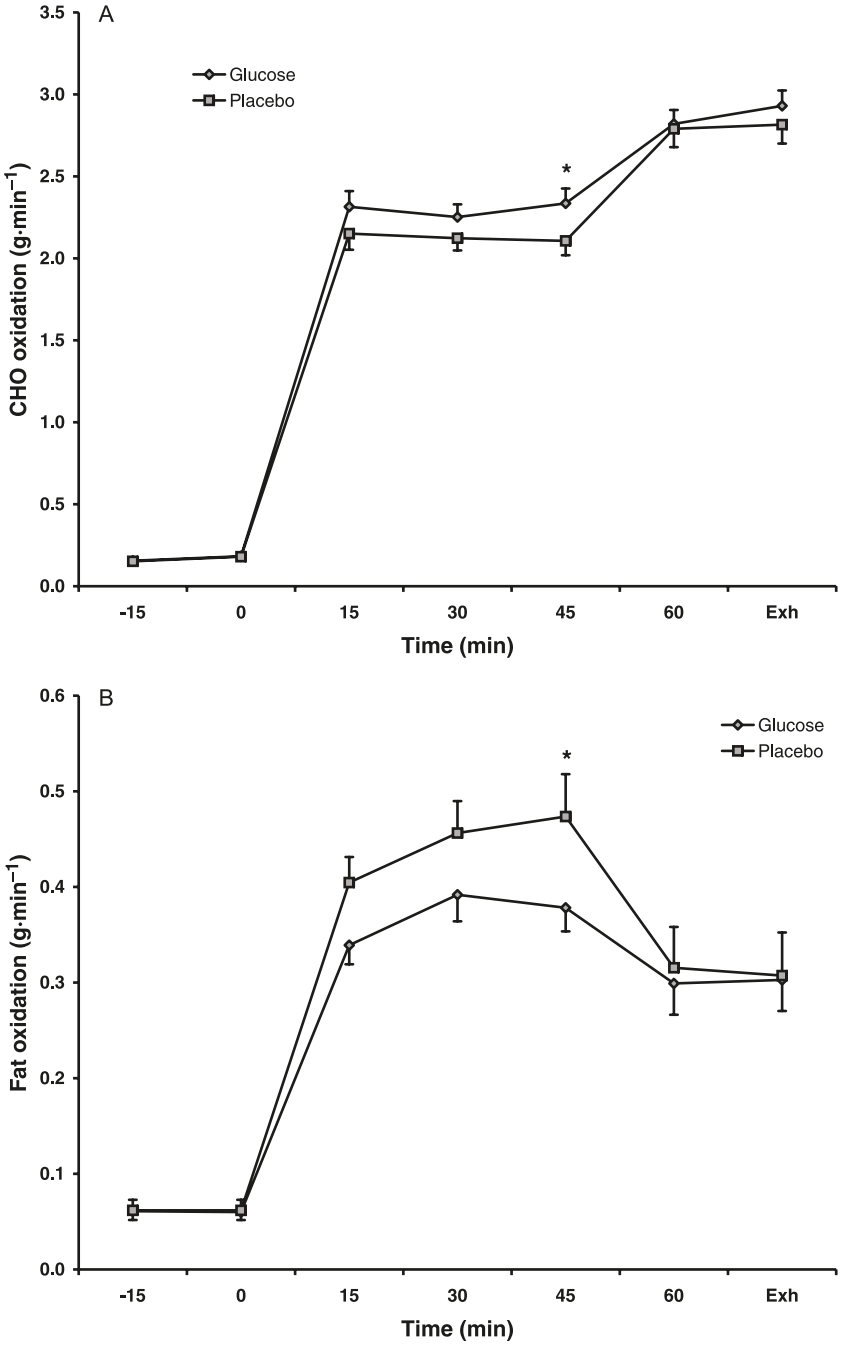
Discussion

The present study indicated the positive effects of CHO ingestion shortly (15 min) before exercise on endurance running capacity (Table 2). Our data are in disagreement with cycling studies that presented no differences in exercise performance after the pre-exercise ingestion of carbohydrates (Nishibata et al. 1993; Palmer et al. 1998; Snyder et al. 1993). It is important to note, however, that we examined endurance capacity, whereas in the study of Palmer et al. (1998), the performance time was very short (30 min). In addition, in the study of Snyder et al. (1993), the subjects performed repeated high-intensity, short-duration cycling bouts. Nevertheless, in agreement with our study, some investigators (Gleeson et al. 1986; Sherman et al. 1991) have observed better exercise performance time after CHO ingestion. The mode of exercise, running, along with glucose ingestion 15 min before exercise providing an additional source of energy for long-term exercising muscles, likely helped to increase endurance capacity. Running results in more marked changes in blood glucose and plasma insulin levels compared with cycling at the same relative intensity (Tsintzas and Williams 1998).

As it is known, consumption of CHO prior to exercise could result in elevated insulin concentrations at the beginning of exercise, followed by a rapid decline of serum glucose during the first minutes of exercise, a greater rate of muscle glycogen utilization, and lower FFA levels during exercise (Costill et al. 1977; Coyle et al. 1985). These effects, occurring when CHO was ingested 30–45 min before exercise, could contribute to the development of premature fatigue (Schabort et al. 1999). In our study, however, the 15 min pre-exercise glucose ingestion probably affected glycemic and insulinemic responses differently prior to the onset of and during exercise, simply because there was no time for insulin to reach its peak action. The sudden drop of serum glucose concentration during the first minutes of exercise has also been reported in other studies (Chryssanthopoulos et al. 1994; Costill et al. 1977; Foster et al. 1979; Goodpaster et al. 1996; Tokmakidis and Volaklis 2000) as a main characteristic of subjects who take glucose 30–90 min before exercise. Previous studies have also found that glucose ingestion before exercise caused hypoglycaemia during the first minutes of exercise (Costill et al. 1977; Foster et al. 1979; Kuipers et al. 1999). It is interesting to note, however, that serum glucose concentrations of all individuals in the present study did not fall below the level of hypoglycaemia throughout exercise. On the contrary, glucose levels during all periods of exercise in the G trial were higher than at rest, maintaining euglycemic levels until exhaustion (Fig. 1). This is likely why fatigue was postponed during the glucose trial. Probably, the availability of circulating serum glucose throughout running provided an additional source of energy and reserved muscle glycogen stores (Tsintzas and Williams 1998). As a result, the treadmill performance time was ameliorated.

Indeed, CHO ingestion during exercise in the form of CHO–electrolyte beverages has become a popular practice among recreational and endurance athletes and leads to per-

Fig. 4. Rates of carbohydrate (CHO) and fat oxidation during rest and submaximal exercise for placebo and glucose trials. Values are means ± SE (n = 11). Time points at which values are significantly different from placebo ($p < 0.05$) are marked with an asterisk (*).



formance benefits during prolonged exercise (Tsintzas and Williams 1998). These CHO solutions prevent hypoglycaemia, provide an immediate source of energy to working muscles, and delay the onset of fatigue during prolonged exercise. In addition, when CHO is ingested before exercise or when blood glucose levels are high at the beginning of exercise and the normal euglycaemia is maintained for a long period of time until the onset of fatigue, then the potential of sparing muscle glycogen stores plays a positive role on performance (Tsintzas and Williams 1998). In the present study, glucose was ingested 15 min before the onset of running exercise. Thus, at the beginning of exercise, serum glucose was significantly higher than placebo and this euglycaemic response was maintained until exhaustion (Fig. 1). It appears, therefore, that glucose ingestion shortly before running exercise helps to avoid the discomfort that occurs when fluid is ingested during exercise. It could result in reduced muscle glycogen utilization and thus improve endurance running capacity.

It is difficult to explain the main reasons for hypoglycaemia during the first minutes of exercise, after carbohydrate ingestion before exercise. The time of glucose supplementation (Tokmakidis and Volaklis 2000) or the amount of glucose ingested (Kuipers et al. 1999) prior to exercise may

indicate some reasons why an individual is susceptible to hypoglycaemia. In a previous study from our laboratory (Tokmakidis and Volaklis 2000), in which the subjects ingested a glucose solution (1 g·kg body mass^{-1} in 350 mL water), 30, 60, or 90 min before exercise, hypoglycaemia was not observed. Jentjens and Jeukendrup (2002) suggested that hypoglycaemia during exercise is not related to insulin sensitivity. The mechanisms that induce hypoglycaemia during exercise do not seem clear and may be viewed as an individual phenomenon (Kuipers et al. 1999).

Many studies investigated glucose ingestion at least 45 min prior to exercise and found that the culmination of glucose and insulin concentrations before the start of exercise takes place about 30 min after ingestion (Guezennec et al. 1989 and 1993; Koivisto et al. 1985; Thomas et al. 1991). The same studies refer to the synergistic effect of insulin on the exercise-induced muscle glucose uptake that results in a sudden decrease in blood glucose concentration during the first 30 min of exercise. It may be of interest to note, however, that glucose levels during exercise returned to the initial concentration at least 60 min after the onset of exercise. On the contrary, in the present study, it appears that the amount of glucose, as well as the time of ingestion 15 before exercise, was not sufficient to provide a full insulin effect. Adequate euglycaemia was maintained during the first minutes of exercise, and circulating glucose was available until exhaustion (Fig. 1). Moreover, this indicates that insulin concentrations, which declined back to basic levels just a few minutes after the onset of exercise (Fig. 1), could not have had a negative influence on performance.

Marmy-Conus et al. (1996) suggested that a 75 g glucose feeding 30 min before exercise provides additional supplies of energy for oxidation, and results in increased muscle glucose uptake and reduced liver glucose output during 60 min of cycling. The glucose absorbed from the gut increased after glucose ingestion, levelled off during the first 30 min of exercise, and then increased again towards the end of exercise (Marmy-Conus et al. 1996). Sherman et al. (1989) estimated the absorption rate of a 312 g carbohydrate meal using the results of Hunt et al. (1985), and calculated that only 63% of the carbohydrates would have been emptied from the stomach during the 4 h preceding exercise. Thus, it seems reasonable to suggest that in our study, the 75 g pre-exercise liquid was still being absorbed from the small intestine even at exhaustion. This can probably explain the higher endurance running capacity during the glucose trial. The availability of circulating blood glucose may preserve the muscle glycogen stores (Tsintzas and Williams 1998) and increase endurance capacity.

Pre-exercise glucose consumption did not result in a higher rate of CHO oxidation when compared with our placebo trial (Fig. 4). Nevertheless, an increase in the rate of CHO oxidation from 60 min until exhaustion, when the intensity of exercise was at 80% $\dot{V}O_{2\,max}$, was reflected by a reduction of fat oxidation (Fig. 4). Our data indicate that, due to the effect of insulin on lipolysis, FFAs in the glucose trial were lower after 45 min of exercise until exhaustion and tended to decline in the remaining time of the trial (Fig. 2). Moreover, glycerol levels were lower in the glucose than in the placebo trial after 30 min of exercise until exhaustion (Fig. 2). Thus, in the glucose trial, an improvement in running capacity was observed in our study, with lower FFA concentrations and without changes in the rate of CHO oxidation.

In conclusion, the data of the present study demonstrate that ingested glucose 15 min before running exercise provided an additional source of energy during exercise and increased endurance running capacity.

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