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Improved recovery from prolonged exercise following the consumption of low glycemic index carbohydrate meals

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Abstract

The present study examined the effects of the glycemic index (GI) of post-exercise carbohydrate (CHO) intake on endurance capacity and the metabolic responses during exercise the following day. Nine active males participated in two trials in a randomised cross-over design. The experimental protocol was completed over two days. On day 1, subjects completed a 90 min treadmill run at 70% $\dot{V}O_2$ max (R1). Thereafter, they were supplied with a diet consisting of either high glycemic index (HGI) or low glycemic index (LGI) CHO and provided 8g CHO·kg body mass $(BM)^{-1}$. On day 2, after an overnight fast, subjects ran to exhaustion at 70% $\dot{V}O_2$ max (R2). Eight subjects completed both performance runs (R2). Run time to exhaustion during R2 was longer in the LGI trial (108.9 ± 7.4 min) than in the HGI trial (96.9 ± 4.8 min) ($p<0.05$). Average RER values were higher in the HGI trial compared to the LGI trial ($p<0.05$). Fat oxidation rates and free fatty acid concentrations were higher in the LGI trial than the HGI trial ($p<0.05$). The results of the study suggest that the increased endurance capacity during R2 was largely a consequence of the greater fat oxidation following the consumption of the LGI meals.

Key Words: running, fat oxidation, muscle glycogen, endurance capacity

Introduction

It is well documented that the ability to perform prolonged exhaustive exercise is closely related to muscle glycogen stores [2, 8]. Many athletes train or compete on consecutive days and therefore the rapid restoration of muscle glycogen stores during the recovery period is essential. The intake of carbohydrate (CHO) after exercise has repeatedly been shown to increase muscle glycogen repletion [3, 13, 27]. Therefore the consumption of high carbohydrate food or drinks after exercise is now common practice.

Carbohydrate foods can be defined according to their postprandial glycemic responses [14]. The ingestion of high glycemic index (HGI) CHO is generally associated with high blood glucose and insulin concentrations. It is logical that carbohydrate sources with a HGI would enhance post-exercise glycogen resynthesis [7]. Several studies have examined the effect of HGI CHO drinks consumed during recovery from exercise [13, 16, 34] however few have examined the effects of carbohydrate meals. In practice, athletes are more likely to eat a combination of foods, especially if the recovery time is longer than a few hours. Burke et al.[6] has shown that consuming HGI CHO meals during a 24 h recovery period from prolonged heavy exercise resulted in higher muscle glycogen resynthesis than following the consumption of an isocaloric low glycemic index (LGI) diet. It would be reasonable to assume that endurance capacity would be greater during subsequent exercise, however this was not investigated.

Therefore, the aim of the present study is to investigate the effects of HGI and LGI CHO meals consumed during a 24 h recovery period (i.e. feeding during 12h post -exercise followed by a 12h fast) after glycogen depleting exercise on endurance capacity and the metabolic responses during exercise the following day.

Methods

Subjects

Nine recreational male athletes participated in this study. Their mean ($\pm$ SD) age, height, weight, $\dot{V}O_2$ max were 22.4 ± 1.5 years, 180 ± 1.0cm, 79.4 ± 10.9 kg and 61.0 ± 5.7 ml kg^{-1} min^{-1} respectively. A criterion for inclusion the study was that participants ran regularly and were able to run for at least one hour continuously at about 70% $\dot{V}O_2$ max. The protocol was approved by Loughborough University Ethical Advisory Committee and all subjects gave their written informed consent.

Preliminary measurements

Following familiarization with treadmill running and experimental procedures, subjects undertook two preliminary tests in order to determine: 1) the relationship between running speed and oxygen uptake using a 16 min incremental test and 2) their $\dot{V}O_2$ max using an uphill incremental treadmill test to exhaustion. All preliminary tests were conducted according to procedures previously described [32]. Based on the results of the two preliminary tests, the running speed equivalent to 70% of each subject's $\dot{V}O_2$ max was determined.

At least a week before the first main trial subjects undertook a 45 min treadmill run at 70% $\dot{V}O_2$ max in order to confirm the relative exercise intensity. The energy expended by each subject during the 45 min run was also calculated and used to estimate the energy expenditure for a 90 min run at the same speed.

Experimental Design

Each subject participated in two experimental trials separated by at least 7 days. The experimental testing protocol was completed over a 2-day period. For 2 days before the first trial, the subjects recorded their diet and exercise routine so that it could be repeated before trial 2 to minimise differences in pre-testing intramuscular substrate concentrations between experimental trials. Analysis of the pre-trial diets showed that the subjects consumed on average, a diet consisting of 60% carbohydrate, 25% fat and 15% protein. The average daily energy intake for each subject was also calculated from the dietary information. On Day 1 of the experimental trial, each subject completed a glycogen reduction protocol, which consisted of a 90 min constant pace treadmill run at 70% $\dot{V}O_2$ max (R1). This intensity and duration of exercise has previously been shown to significantly reduce muscle glycogen stores [28]. Thereafter, subjects were provided with a recovery diet that provided at least 8g CHO·kg body mass $(BM)^{-1}$ over the next 22 h and was composed of either high glycemic index (HGI) or low glycemic index (LGI) carbohydrates in a cross-over design. On Day 2, subjects were required to run to exhaustion at 70% $\dot{V}O_2$ max (R2). A balanced randomisation of the trials was applied for the subjects and they were not informed of their running times or the hypothesis being tested. Subjects were provided with water *ad libitum* during trial 1 and this was matched in trial 2. All trials were performed at the same time of day and under similar experimental and environmental conditions. The same treadmill was also used throughout the experiment (Technogym™ Run Race Treadmill, 47035, Gambettoio, Italy.) Subjects were advised to maintain their normal training schedule during the study but to abstain from any vigorous exercise in the 24 h period before the two experimental trials. During this period they were also required to avoid alcohol, caffeine and smoking.

Protocol

On Day 1 of the experiment subjects arrived in the laboratory at 0800 h following an overnight fast. On arrival, subjects completed the necessary health and consent forms and were then asked to void before nude mass was obtained (Avery, England). Urine samples were collected before and after each exercise bout. A cannula (Venflon 18G, Becton Dickinson Ltd, Helsingborg Sweden) was then inserted into an antecubital vein and connected to a 3-way stopcock (Connecta Ltd, Helsingborg, Sweden) with a 10cm extension tube for blood sampling. The cannula was kept patent by flushing with sterile isotonic saline (9g/l) immediately after the cannula was inserted and after blood sampling.

After the subjects had stood for 15 min, an 11ml pre-exercise venous blood sample was drawn from the cannula. A short-range telemeter (Technogym, Gambettoio, Italy) heart rate monitor was then attached to the subject to monitor heart rate (HR) and a 5 min resting expired air sample was also collected. Following a 5 min warm up at 60% $\dot{V}O_2$ max the treadmill speed was increased to the pace equivalent to 70% for $\dot{V}O_2$ max each subject (R1). Subjects then ran for 90 min or to volitional fatigue, whichever occurred first. Volitional fatigue was determined as the point at which subjects could no longer maintain the required running speed. One-minute expired air samples and venous blood samples were collected every 15 min throughout the run. Heart rate was closely monitored and Rating of Perceived Exertion (RPE), using the Borg 15-point scale [4], was also recorded every 15 min. After R1, subjects dried themselves to remove surface sweat before nude body mass was obtained. A final venous blood sample was collected 30 min after the cessation of R1 and then subjects ate their prescribed breakfast. Subjects were then free to leave the laboratory but returned for lunch and were given their evening meal and snacks to eat at home. Subjects were instructed

not to eat anything other than the food provided for them. No extra drinks, apart from water, were permitted.

On the morning of Day 2, subjects again arrived in the laboratory at 0800 h after a 10-12 h fast. Exactly the same procedures were followed as day 1 however subjects were required to run to exhaustion at 70% $\dot{V}O_2$ max. Exhaustion was defined as the time at which the subjects were no longer able to maintain the prescribed running speed. Near the end of the run, subjects had the option of slowing down the treadmill twice for 2 min each time to a walking speed of 4 kph. This was to ensure that subjects were truly fatigued. Each subject repeated the same procedure for the subsequent trial. As in R1, venous blood and expired air samples were collected every 15 min for 90 min. No further measurements were taken after this time until the last minute of running when an expired air sample was collected. The last venous blood sample was taken immediately after termination of the run while the subjects were standing on the treadmill. Following R2, subjects were asked to remove surface sweat before nude mass was obtained and then again, were asked to void. All subjects were given verbal encouragement throughout R2 however they were not aware of their performance times, heart rate or distance covered. Throughout the study, the investigators were aware of the trial order however no information on the purpose of the study was given to the subjects.

Ambient temperature and relative humidity were recorded every 30 min using a hydrometer (Zeal, London, UK) during the main trials. Temperature was maintained between 21-23°C and humidity was between 54-59%. Fans were used to cool the subjects and wet sponges were available *ad libitum* throughout all runs.

Test Meals

Isocaloric recovery meals consisting of HGI or LGI CHO foods were provided for each subject after R1 (Table 1). Breakfast was consumed in the lab 30 min after completion of R1 and lunch was provided 3 hours later. Again, this meal was prepared and consumed in the lab. Subjects were then provided with two snacks and an evening meal to be consumed at home. They were instructed to eat one snack between lunch and the evening meal and consume the second between 8pm and 9pm. The total energy intake for each subject was the sum of the calculated energy expenditure during the 90 min run (average energy cost of 1454 kcal) and an estimation of average daily energy intake from weighed food intake diaries completed before the first trial. The amount of CHO provided in the total recovery diet was 8g CHO·kg^{-1} BM. Other foods were added to the diet (e.g. margarine, cheese and milk) to make them more palatable however, the same quantity was used in both diets. The nutritional content of each meal was calculated from information provided by the manufacturer. Foods were carefully chosen so that each diet was matched for protein and fat therefore both diets consisted of 72% CHO, 11% fat and 17% protein. The GI of the total diets was calculated from the weighted means of the GI values for the component foods [33]. The calculated GI for the high and low diets was 70 and 35 respectively.

Sample Collection and Analysis

Expired air samples were collected and analysed as previously described [32]. Substrate oxidation rates and energy expenditure were calculated from $\dot{V}O_2$ and $\dot{V}CO_2$ values using stoichiometric equations [12].

All blood samples were obtained from the subjects whilst standing. At each sampling point, 11ml of blood was collected and 5ml of whole blood was immediately dispensed into an EDTA tube. Haemoglobin (Hb) concentration was determined using the cyanmethaemoglobin method (Boehringer Mannheim, Mannheim Germany) ($2\times20\mu l$) and hematocrit (Hct) values were determined in triplicate on samples of whole blood by microcentrifugation (Hawksley Ltd, Lancing, Sussex, UK). Changes in plasma volume were estimated from changes in Hb concentrations and Hct values, as described by Dill and Costill [11]. Blood lactate concentration was analysed by a photometric method using a spectrophotometer (Shimazu mini 1240, Japan). Plasma samples were obtained by centrifugation of the remaining whole blood for a period of 10 min at 4000rpm and 4°C. The aliquoted plasma was then stored at -20°C for later analysis of free fatty acids (FFA) (ASC-ACOD method, Wako NEFA C; Wako, Neuss, Germany), glucose (GOD-PAP method, Randox, Ireland) and glycerol (Randox, Ireland.) using an automatic photometric analyser (Cobas-Mira plus, Roche, Basel, Switzerland). The remaining whole blood sample was dispensed into a non-anticoagulant tube and left to clot for 45 min. Serum samples were then obtained after centrifugation at 4000rpm for 10 min at 4℃. The aliquoted serum was stored at -85°C and later analysed for insulin (Coat-A-Count Insulin ICN Ltd, Eschwege, Germany) and cortisol (Corti-Cote ICN Ltd, Eschwege, Germany) by radio immunoassay (RIA) using a gamma counter (Cobra 5000, Packard Ltd, Boston, MA, USA).

Statistical analysis

Analysis of variance (ANOVA) for repeated measures on two factors (experimental treatment and time) was used to analyse differences in the physiological and metabolic responses in both trials. If a significant interaction was obtained, a Tukey post-hoc test was applied to locate the differences between means. Statistical analysis was carried out on data for the first

90 min of R2. The point of exhaustion was analysed separately due to the large variation in the time that this occurred. A Student's paired t-test was used to analyse differences at this point and for non-time dependent variables. Differences were considered significant at $p<0.05$. All results are presented as mean $\pm$ SEM.

Results

Running Time and Subjective Observations

All subjects completed the 90 min glycogen reduction run on Day 1 of both trials. Seven out of the eight subjects who completed both experimental trials ran longer following the LGI carbohydrate recovery diet compared to the HGI recovery diet. The average running time in the LGI trial (108.9 ± 7.47 min) was significantly longer than the HGI trial (96.9 ± 4.83 min) ($p< 0.05$).

All subjects verbally reported feeling hungry before the start of the run to exhaustion following the consumption of the HGI recovery diet. This observation was not reported following the LGI diet despite the fact that both diets were matched for energy and nutrient content.

Heart Rate and RPE

There was a consistent tendency for heart rate to be 2-3 beats·min^{-1} lower during R2 in the LGI trial compared to the HGI trial however no statistically significant differences were reported. There was a trend for RPE to be higher in the HGI trial and subjects verbally reported that the LGI trial 'felt easier' (Table 2).

Substrate Utilisation

There were no significant differences in substrate utilisation during R1. During R2, the estimated fat oxidation rates, as calculated from the $\dot{V}O_2$ and $\dot{V}CO_2$ values, were significantly higher in the LGI trial compared to the HGI trial ($p<0.05$) (Fig 1). Carbohydrate oxidation rates were higher in the HGI trial compared to the LGI trial however this difference did not reach statistical significance (Fig 2). At the point of exhaustion, fat oxidation rates were significantly higher in the LGI trial compared to the HGI trial ($p<0.05$). However, there were no significant differences in carbohydrate oxidation rates.

$\dot{V}O_2, \dot{V}CO_2,$ *and Respiratory Exchange Ratio (RER)*

The indirect calorimetry data indicated a stable $\dot{V}O_2$ during both experimental trials (40.9 - 43.7 ml·kg^{-1} min^{-1}) and elicited ~ 71 ± 2%. $\dot{V}O_2$ max (Table 3). Average RER values were higher in the HGI trial compared to the LGI trial during the first 90 min of R2 ($p<0.05$) and at the point of exhaustion (Table 3).

Plasma Glucose, FFA and Glycerol

Plasma glucose concentrations were equally well maintained between 4 and 5 mmol·l^{-1} over both runs in both trials. At the point of exhaustion in R2, plasma glucose concentrations were higher in the HGI trial however there were no significant differences between the trials (Table 4). In both trials, plasma concentrations of FFA and glycerol rose progressively throughout exercise. During the first 30 min of recovery after R1, FFA concentrations continued to rise and increased from 0.84 ± 0.16 mmol·l^{-1} at the end of exercise to 1.19 ± 0.15 mmol·l^{-1} in the HGI trial and from 0.75 ± 0.08 mmol·l^{-1} to 1.16 ± 0.16 mmol·l^{-1} in the LGI trial. During the first 90 min of R2, FFA concentrations were significantly higher in the LGI compared to the HGI trial ($p<0.05$). At the point of exhaustion, FFA concentrations were also significantly higher in the LGI trial ($p<0.05$). (Table 4). There were no significant differences

in glycerol concentrations during R2 but a consistent trend for higher concentrations in the LGI trial (Table 4).

Serum Insulin and Cortisol

Serum insulin concentrations were similar between trials (Fig 3). In both trials, serum cortisol concentrations fell from pre-exercise values during the first 30 min of running and then increased gradually throughout the remainder of the run. At the point of exhaustion, cortisol concentrations were slightly higher in the LGI trial but this is probably due to the fact that most subjects ran for longer in this trial. There were however no significant differences between trials throughout the experimental protocol (Fig 4).

Blood Lactate

During R1 there were no differences in blood lactate concentrations between trials (average concentrations were 2.17 ± 0.18 mmol.l^{-1} in the HGI trial and 2.01 ± 0.12 mmol.l^{-1} in the LGI trial). Blood lactate concentrations were significantly higher throughout R2 in the HGI trial compared to the LGI trial ($p < 0.05$) (Fig 5). Pre-exercise values were similar between trials however in the HGI trial, blood lactate concentrations rose to approximately 1.9 mmol·l^{-1} and were maintained around this concentration throughout the run compared to the LGI trial in which concentrations were maintained around 1.7 mmol·l^{-1}. There were no differences between trials at the point of exhaustion.

Body Mass and Hydration Status

At the end of R1 subjects had lost $0.3 \pm 0.4\%$ and $0.3 \pm 0.3\%$ of their pre-exercise body mass in the HGI and LGI trials respectively (NS). At the end of R2, subjects had lost $0.3 \pm 0.4\%$ of

their pre-exercise body mass in the HGI trial and $0.1 \pm 0.7\%$ in the LGI trial (NS). Pre-exercise body mass was not different between trials. There were no significant differences in urine osmolality before or after R1 and R2 between trials. Only one subject started the run to exhaustion with a urine osmolality that suggested dehydration however, this was the same subject who failed to complete the experimental protocol due to injury.

Discussion

The main finding of the present study was that the ingestion of a LGI carbohydrate recovery diet consumed in the 24 h period following prolonged heavy exercise resulted in a greater endurance capacity during steady state exercise in the post-absorptive state the following day compared with a HGI carbohydrate diet.

During recovery from exercise, high glycemic index foods are generally recommended to athletes because the large glycemic and insulinemic response following their ingestion favours muscle glycogen resynthesis. Nevertheless, research on the effects of the glycemic index of carbohydrate feedings during 24 h recovery is limited and the results of studies investigating muscle glycogen resynthesis are equivocal. Burke et al. [6] reported that a HGI CHO diet resulted in greater muscle glycogen resynthesis than an isocaloric LGI CHO diet. Muscle biopsies were performed 24 h after exercise however no measure of performance was carried out at the end of the recovery period. Kiens et al.[20] attempted to study carbohydrate foods and muscle glycogen storage on the basis of actual glycemic responses to the foods. They reported that at 20 h of recovery there were no differences in muscle glycogen storage between the LGI and HGI diets. However, the diets are described interchangeably as simple / HGI CHO and complex / LGI CHO therefore the results can not be directly compared to the results reported by Burke et al [6].

The significantly lower RER values indicating a higher rate of fat oxidation during the run to exhaustion in the LGI trial may explain why subjects ran longer. Plasma FFA concentrations were also significantly higher in the LGI trial compared to the HGI trial during R2. An improvement in endurance performance has been reported when plasma FFA concentrations have been elevated [22, 23] and investigators have suggested glycogen sparing as the main reason for this improvement [17]. Despite this, the methods used to increase FFA concentrations have not involved normal dietary practice. Furthermore, although fat supplementation both before and during exercise has been shown to increase fat oxidation, this is usually regarded as undesirable as endogenous fat stores are adequate and make additional fat supplementation inappropriate [17].

It is unclear exactly why fat oxidation rates were higher during R2 in the LGI trial compared to the HGI trial. Pre-exercise substrate availability has emerged as an important regulator of the patterns of fuel oxidation during exercise [1] therefore this may provide a possible explanation. A recent study carried out by Arkinstall and co-workers [1] reported that there was a greater utilization of muscle glycogen during a 60 min run at 70% VO_2max when pre-exercise muscle glycogen concentrations were high. The authors also reported that there was a greater contribution from carbohydrate oxidation to total energy expenditure when commencing exercise with high glycogen concentrations. In the present study, the higher rate of fat oxidation and consequently lower carbohydrate oxidation rate in the LGI trial may therefore be a result of lower pre-exercise muscle glycogen concentrations compared to the HGI trial.

It is widely accepted that muscle glycogen is the primary fuel source during prolonged exercise [24.] There is now accumulating evidence to suggest that intramuscular

triacylglycerol (IMTG) also functions as an important substrate source during prolonged exercise in healthy subjects [29]. Several studies have reported the depletion of IMTG stores following prolonged exercise [5, 18, 21, 30] therefore, the repletion of both muscle glycogen and muscle fat stores during the recovery period needs to betaken into consideration. Recent research has reported that a high CHO diet consumed after exercise inhibits the post-exercise resynthesis of IMTG because increased FFA concentrations are required for the replenishment or elevation of IMTG content [19]. Decombaz et al. [9] reported that a high CHO, low fat diet (70% CHO, 14% fat and 14% protein) fed during the 30h preceding a 2h run at 50% VO_2max resulted in IMTG stores 5-17% lower than pre-exercise values. Similarly, Starling et al. [26] reported that a high CHO diet fed during the 24 h period following 120min cycling at 65% VO_2max resulted in significantly lower IMTG concentrations than when a high fat diet was consumed.

No studies to date have investigated whether changing the type of carbohydrate consumed during recovery from exercise can influence IMTG replenishment. Many studies have observed larger glycemic and insulinemia responses and a greater suppression of fat metabolism following a HGI CHO meal compared to a LGI CHO meal [10, 15, 31]. Therefore it is possible that FFA concentrations were higher throughout the recovery period in the LGI trial. This may have allowed some resynthesis of the IMTG stores as well as the replenishment of muscle glycogen as a result of the carbohydrate intake. This may help to explain the increased fat oxidation and endurance capacity in the LGI trial.

The effects of a number of other variables that may have influenced running performance in the HGI can be ruled out. For example, there were no significant differences in the physiological and metabolic responses to R1 between trials therefore the glycogen reduction

runs were similar. Furthermore, there were no differences in hydration status either at the end of R1 or at the start of R2 between trials. Plasma glucose, serum insulin and cortisol concentrations were also similar during R2 in both trials. Blood lactate concentrations were significantly higher during R2 in the HGI trial compared to the LGI trial. This would be expected as HGI CHO induce a higher rate of glycolysis than LGI CHO foods [25]. Blood lactate concentration did not, however, exceed 2.3mmol·l^{-1} in the HGI trial and so it is unlikely that this was a limiting factor to exercise performance.

In conclusion, the results of the present study show that the consumption of a LGI CHO diet in the 24 h following prolonged running increased endurance capacity the next day beyond that which was achieved following the consumption of a HGI CHO recovery diet. A higher rate of fat oxidation throughout the run to exhaustion in the LGI trial is a possible explanation for this increase in endurance capacity. The results of the present study also suggest that maximal muscle glycogen resynthesis should not be the only aim of post-exercise nutrition following prolonged exercise.

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Table 1. Characteristics of test meals (for a 70kg subject)

Meal	Description	Macronutrient Content
HGI breakfast	62g Corn Flakes$^\psi$ + 257ml skimmed milk 80g white bread + 10g flora +20g jam 155ml Lucozade original$^\psi$	730kcal, 139gCHO, 9.9g fat, 20g protein
LGI breakfast	86g muesli + 257ml skimmed milk 67g apple, 103g tinned peaches, 128g yoghurt 257ml apple juice	732 kcal, 139g CHO, 9g fat, 23g protein
HGI lunch	158g white bread, 154g turkey breast, 50g cheese, 40g lettuce, 180g banana 200ml Lucozade original$^\psi$	1076kcal, 148g CHO, 24g fat, 63g protein
LGI lunch	154g whole wheat pasta, 150g turkey breast, 50g cheese, 40g lettuce, 185g pasta sauce, 150g pear, 150 ml apple juice	1075 kcal, 149g CHO, 25g fat, 60g protein
HGI dinner	255g baked potato, 410g tinned spaghetti, 50g cheese, 40g lettuce, 67g mars bar, 170ml Lucozade original$^\psi$	1100kcal, 176g CHO, 31g fat, 28g protein
LGI dinner	360g chilli beans, 200g wheat tortilla, 50g cheese, 40g lettuce, 260ml orange juice	1100kcal, 176g CHO, 29g fat, 39g protein
HGI snacks	2x 77g white bread + 2x 20g jam + 2x 10g flora	600kcal, 96g CHO, 17g fat, 15g protein
LGI snacks	170g yoghurt, 100g apple, 100g flapjack	625kcal, 97g CHO, 25g fat, 15g protein
HGI total		3520kcal, 560g CHO, 84g fat 126g protein (72%CHO, 11% fat, 17% protein) **GI = 70***
LGI total		3600kcal, 560gCHO, 88g fat 135g protein (72% CHO, 11% fat, 17% protein) **GI = 35***

*calculated by the method described in Wolever (1986) with GI values taken from Foster-Powell et al (2002).

$^\psi$ Corn Flakes: Kellogg's (UK) Ltd. Manchester UK; Lucozade original drink UK

Table 2. Heart rate (HR) and rate of perceived exertion (RPE) during the high glyceamic index (HGI) and the low glyceamic index (LGI) CHO trials. (mean ± SEM)

Variable	Trial	R1						R2					
		15	30	45	60	75	90	15	30	45	60	75	90
heart rate (bpm)	HGI	156 ± 3	161 ± 5	164 ± 4	165 ± 4	165 ± 7	171 ± 3	157 ± 2	161 ± 2	162 ± 2	164 ± 3	164 ± 3	167 ± 3
	LGI	154 ± 2	161 ± 2	163 ± 3	162 ± 4	167 ± 3	167 ± 3	155 ± 2	159 ± 3	162 ± 2	163 ± 3	163 ± 3	162 ± 3
RPE	HGI	13 ± 0	13± 1	14 ± 1	15 ± 1	15 ± 1	15 ± 1	14 ± 0	15 ± 1	15 ± 1	16 ± 1	17 ± 1	17 ± 1
	LGI	13 ± 0	14 ± 0	14 ± 1	15 ± 1	16 ± 1	15 ± 1	13 ± 0	14 ± 1	14 ± 1	16 ± 1	16 ± 1	17 ± 1

Table 3. Oxygen Uptake (VO₂) carbon dioxide expired (VCO₂) and the respiratory exchange ratio during the high glyceamic index (HGI) and the low glyceamic index (LGI) CHO trials. (mean ± SEM)

		R1							R2							
Variable	Trial	Resting	15	30	45	60	75	90	Resting	15	30	45	60	75	90	Exh
VO₂ (l/min)	HGI	0.38 ± 0.03	3.29 ± 0.09	3.36 ± 0.07	3.36 ± 0.08	3.39 ± 0.1	3.37 ± 0.09	3.34 ± 0.09	0.40 ± 0.03	3.30 ± 0.08	3.29± 0.07	3.35 ± 0.09	3.37± 0.1	3.32 ± 0.09	3.31 ± 0.09	3.31 ± 0.11
	LGI	0.44 ± 0.05	3.28 ± 0.09	3.32 ± 0.09	3.29 ± 0.08	3.33 ± 0.08	3.34 ± 0.09	3.35 ± 0.09	0.41 ± 0.03	3.34 ± 0.07	3.38 ± 0.08	3.44 ± 0.10	3.38 ± 0.08	3.38 ± 0.08	3.30 ± 0.08	3.34 ± 0.11
VCO2 (l/min)	HGI	0.35 ± 0.03	3.05 ± 0.11	3.10 ± 0.11	3.05 ± 0.10	3.09 ± 0.11	3.04 ± 0.11	3.04 ± 0.11	0.36 ± 0.03	3.03 ± 0.10	3.00 ± 0.10	3.02 ± 0.11	3.04 ± 0.11	2.99 ± 0.09	2.95 ± 0.10	2.97 ± 0.13
	LGI	0.38 ± 0.05	3.01 ± 0.10	3.00 ± 0.10	2.98 ± 0.10	2.97 ± 0.09	2.99 ± 0.10	2.93 ± 0.10	0.36 ± 0.03	2.99 ± 0.09	2.99 ± 0.10	3.04 ± 0.12	2.98 ± 0.10	2.98 ± 0.08	2.89 ± 0.09	2.91 ± 0.14
RER	HGI	0.93 ± 0.04	0.93 ± 0.04	0.92 ± 0.02	0.91 ± 0.01	0.91 ± 0.01	0.90 ± 0.02	0.90 ± 0.01	0.88 ± 0.04	0.92 ± 0.01	0.91 ± 0.01	0.90 ± 0.01	0.90 ± 0.01	0.90 ± 0.01	0.90 ± 0.01	0.90 ± 0.01
	LGI	0.87 ± 0.03	0.92 ± 0.01	0.90 ± 0.01	0.90 ± 0.01	0.89 ± 0.01	0.89 ± 0.01	0.87 ± 0.01	0.86 ± 0.02	0.90* ± 0.01	0.88* ± 0.01	0.88* ± 0.01	0.88* ± 0.01	0.88* ± 0.01	0.88* ± 0.01	0.87* ± 0.01

*Mean values were significantly different from those in the HGI trial ($p < 0.05$)

Table 4. Plasma glucose, free fatty acid and glycerol concentrations during Run 1 and Run 2 in the high glyceamic index (HGI) and the low glyceamic index (LGI) CHO trials (mean ± SEM).

		R1 (min)							
Variable	Trial	Resting	15	30	45	60	75	90	30 REC
plasma glucose	HGI	4.79 ± 0.11	4.77 ± 0.15	5.03 ± 0.17	5.06 ± 0.09	4.88 ± 0.13	4.83 ± 0.12	5.12 ± 0.15	4.34 ± 0.12
(mmol.l⁻¹)	LGI	4.74 ± 0.14	4.76 ± 0.10	5.18 ± 0.15	5.28 ± 0.13	5.00 ± 0.08	4.73 ± 0.09	4.67 ± 0.13	4.29 ± 0.13
plasma FFA	HGI	0.29 ± 0.06	0.21 ± 0.04	0.29 ± 0.06	0.35 ± 0.07	0.43 ± 0.09	0.51 ± 0.1	0.84 ± 0.16	1.19 ± 0.15
(mmol.l⁻¹)	LGI	0.23 ± 0.04	0.20 ± 0.03	0.28 ± 0.04	0.34 ± 0.05	0.41 ± 0.05	0.50 ± 0.07	0.75 ± 0.08	1.16 ± 0.16
plasma glycerol	HGI	61.4± 19.8	126.2 ± 28.5	197.0 ± 40.4	238.1 ± 44.3	297.3 ± 52.5	361.1 ± 58.6	415.2 ± 58.3	191.7 ± 25.3
(µmol.l⁻¹)	LGI	33.7 ± 5.8	109.2 ± 18.1	176.4 ± 28.7	236.8 ± 34.6	279.8 ± 37.7	338.2 ± 37.5	399.9 ± 38.7	210.6 ± 41.1

		R2 (min)							
Variable	Trial	Resting	15	30	45	60	75	90	Exh
plasma glucose	HGI	4.65 ± 0.09	4.50 ± 0.11	4.81 ± 0.10	4.91 ± 0.09	4.66 ± 0.12	4.33 ± 0.12	4.15 ± 0.10	4.36 ± 0.11
(mmol.l⁻¹)	LGI	4.79 ± 0.10	4.53 ± 0.18	4.82 ± 0.17	4.88 ± 0.13	4.67 ± 0.09	4.51 ± 0.11	4.45 ± 0.17	4.26 ± 0.13
plasma FFA	HGI	0.32 ± 0.06	0.26 ± 0.03	0.36 ± 0.04	0.44 ± 0.05	0.46 ± 0.04	0.55 ± 0.06	0.80 ± 0.08	1.17 ± 0.19
(mmol.l⁻¹)	LGI	0.34 ± 0.07	0.35 ± 0.06*	0.46 ± 0.07*	0.55 ± 0.08*	0.58 ± 0.07*	0.68 ± 0.06*	0.75 ± 0.12	1.36 ± 0.20*
plasma glycerol	HGI	48.5 ± 6.4	116.9 ± 11.4	184.9 ± 18.8	239.1 ± 20.9	284.4 ± 25.1	327.1 ± 26.6	376.4 ± 26.7	409.2 ± 36.3
(µmol.l⁻¹)	LGI	42.9 ± 7.5	149.1 ± 23.4	224.6 ± 21.4	274.5 ± 26.4	321.1 ± 29.6	376.7 ± 36.6	426.6 ± 38.7	444.4 ± 57.1

*Mean values were significantly different from those in the HGI trial (p <0.05)

Figure Captions

Fig 1. The rate of fat oxidation (g·min^{-1}) during the HGI and LGI trials (R2) (mean ± SEM). *LGI trial significantly higher than HGI trial ($p < 0.05$). φ Point of exhaustion

Fig 2. The rate of CHO oxidation (g·min^{-1}) during the HGI and LGI trials (R2) (mean ± SEM). φ Point of exhaustion

Fig 3. Serum insulin concentrations (μlU·ml^{-1}) during the HGI and LGI trials (R2) (mean ± SEM). φ Point of exhaustion

Fig 4. Serum cortisol concentrations (μg·dl^{-1}) during the HGI and LGI trials (R2) (mean ± SEM). φ Point of exhaustion

Fig 5. Blood lactate concentrations (mmol·l^{-1}) during the HGI and LGI trials (R2) (mean ± SEM). *HGI significantly higher than LGI ($p < 0.05$). φ Point of exhaustion