Title: The metabolic responses to high carbohydrate meals with different glycemic indices consumed during recovery from prolonged strenuous exercise

Authors: Emma Stevenson, Clyde Williams and Helen Biscoe

Institute: Sport and Exercise Nutrition Research Group, School of Sport and Exercise Sciences, Loughborough University

Address for correspondence: Professor Clyde Williams
Sport and Exercise Nutrition Group
School of Sport and Exercise Sciences
Loughborough University
Loughborough
Leicestershire
LE11 3TU, UK

Telephone number: +44 1509 226309

Fax number: +44 1509 226300

E-mail address: **C.Williams@lboro.ac.uk**

Abstract

The aim of the present study was to investigate the metabolic responses to high glycemic index (HGI) or low glycemic index (LGI) meals consumed during the recovery period following prolonged exercise. Eight male, well-trained recreational athletes participated in two trials separated by a week. In each trial, participants arrived in the lab after an overnight fast and completed a 90 min run at 70% VO_2max. During the recovery period, two meals were provided. The first meal was provided 30 min after the cessation of exercise and the second meal was provided 2 h later. The plasma glucose responses to both breakfast and lunch were greater in the HGI trial compared to the LGI trial ($p<0.05$). Following ingestion of breakfast, there were no differences in the serum insulin concentrations between the trials. Conversely, following lunch, serum insulin concentrations were higher in the HGI trial compared to the LGI trial ($p<0.05$). The results of the present study suggest that the glycemic index of the carbohydrates consumed during the immediate post-exercise period may not be important as long as sufficient carbohydrate is consumed. The high insulin concentrations following a HGI meal later in the recovery period may facilitate further muscle glycogen resynthesis.

Key Words: post-exercise, substrate utilization, hyperinsulinemia, insulin sensitivity, mixed meals

Introduction

It is well established that the onset of fatigue during prolonged strenuous exercise is associated with muscle glycogen depletion [21]. During the post-exercise recovery period, muscle glycogen repletion has high metabolic priority [12]. Nutritional interventions during this time therefore focus on methods of enhancing this process so that performance in a subsequent exercise bout is not impaired.

When no carbohydrate (CHO) is consumed during recovery from prolonged exercise, very little muscle glycogen resynthesis will occur [11]. Research has therefore focussed on the amount, type and timing of carbohydrate supplementation required to optimise muscle glycogen resynthesis. Athletes are usually encouraged to ingest carbohydrates immediately after exercise rather than hours later. A major reason for this is so that they take advantage of the acute effects of exercise *per se* to stimulate both glucose transport and glycogen synthase activity, promoting faster muscle glycogen resynthesis [26]. High glycemic index (HGI) carbohydrates are recommended due to the high insulinemic and glycemic responses that occur following their consumption. The early research in this area focused on single foods or drinks only. In reality, athletes are more likely to consume mixed meals, especially if the recovery time is greater than a few hours. Few studies have investigated the effects of carbohydrate meals with different glycemic indices on recovery from prolonged exercise. Burke et al [5] reported that the ingestion of HGI CHO meals resulted in higher muscle glycogen resynthesis compared to the resynthesis following the ingestion of LGI meals during a 24h recovery period. Interestingly, the author suggested that the differences in the glycemic and insulinemic responses to the meals were not sufficient to explain the differences in muscle glycogen resynthesis in the

two trials. It was later hypothesised that the LGI CHO may have been mal - absorbed thus providing a lower net amount of CHO for muscle glycogen resynthesis [4] however this hypothesis was not directly measured. It would be expected that endurance performance during subsequent exercise would be greater following the HGI diet but no assessment of endurance performance was carried out in this study. In contrast, a previous study from our laboratory investigating the effects of the glycemic index of recovery diets over 24 h reported increased endurance performance following a LGI diet [20].

The purpose of the present study was therefore to further investigate the metabolic responses to HGI and LGI CHO meals consumed during the early period of recovery from prolonged strenuous exercise.

Methods

Subjects

Eight male, well-trained recreational athletes participated in this study. Their mean (± SD) age, height, weight, $\dot{V}O_2$ max were 22.5 ± 2.3 years, 180 ± 1.0cm, 72.4 ± 9.7 kg and 64.5 ± 9.3 ml kg^{-1} min^{-1} respectively. A criteria 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. Loughborough University Ethical Advisory Committee approved the protocol and all subjects gave their written informed consent.

Preliminary tests

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 [25]. 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.

Experimental design

Each subject participated in two experimental trials separated by at least 7 days. On each occasion, subjects 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 [22]. This was followed by a 5 h recovery period in which subjects were provided with two recovery meals consisting of either high glycemic index (HGI) or low glycemic index (LGI) carbohydrates. The first meal was provided 30 min after the cessation of R1 and the second meal was provided 2 h later. A balanced randomisation of the trials was applied for the subjects.

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.) 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. 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 instructed to avoid alcohol, caffeine and smoking.

Protocol

On the day of the experiment each subject 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). A cannula (Venflon 18G, Becton Dickinson Ltd, Helsingborg Sweden) was then inserted into an antecubital vein for blood sampling. The cannula was kept patent by flushing with sterile 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). One-minute expired air samples and venous blood samples were collected every 30 min throughout the 90 min run. Heart rate was closely monitored and Rating of Perceived Exertion (RPE), using the Borg 15-point scale [2], was also recorded every 30 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 asked to consume the meal within 15 min and then a second meal was consumed 2 h later.

During the recovery period, subjects remained in the laboratory at rest. Ten ml venous blood samples and 5 min expired air samples were taken 15, 30, 60, 90 and 120 min after each meal. During each collection of expired air, ratings of gut fullness, hunger and thirst were recorded using 6-20 scales. Subjects were instructed not to eat anything other than the food provided for them. No extra drinks, apart from water, were permitted.

Ambient temperature and relative humidity were recorded every 30 min using a hygrometer (Zeal, London, UK) during the main trials. Temperature was maintained between 21-23°C and humidity was between 54-59%.

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 2 h later. Again, this meal was prepared and consumed in the lab. The amount of CHO provided in each meal was 2g 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 [28]. The calculated GI for the high and low diets was 75 and 38 respectively.

Sample Collection and Analysis

Expired air samples were collected and analysed as previously described [25] . The subjects remained seated for all expired air samples throughout the postprandial period. Substrate oxidation rates were calculated from $\dot{V}O_2$ and $\dot{V}CO_2$ values using stoichiometric equations [9].

Throughout the recovery, subjects remained seated at all times. Pre and post exercise blood samples were obtained from the subject 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) (2x20µl) 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 [8]. 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℃. The aliquoted plasma was then stored at -85°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 45min. Serum samples were then obtained after centrifugation at 4000rpm for 10 min at 4°C. 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). Pre-trial urine samples were measured for osmolality using a cryoscopic osmometer (Gonometer 030, Gonotec, Germany) and adequate hydration was assumed for osmolality values below 900 mosmol.kg^{-1} [18].

The incremental area under the curve for plasma glucose and serum insulin was estimated using the methodology described by Wolever et al [29]. The blood sample collected at 30min post-exercise was used as the baseline concentration.

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 Holm-Bonnferoni step-wise post-hoc test was utilised to determine the location of the variance. Differences were considered significant at $p<0.05$. All results are presented as mean ± SEM.

Results

Heart rate and rating of perceived exertion

There were no significant differences in heart rate (HR) or rating of perceived exertion (RPE) during R1 between trials.

Body Mass and Hydration Status

There were no significant differences in urine osmolality before R1 and none of the values suggested that any of the participants began exercise in a dehydrated state (632 ± 116 mosmol.kg-1 and 598 ± 113 mosmol.kg-1 in the HGI and LGI trials respectively). At the end of R1 subjects had lost 0.13 ± 0.38 and 0.18 ± 0.59 % of their pre-exercise body mass in the HGI and LGI trials respectively (NS).

Plasma volume

There was a significant decrease in plasma volume by the end of the exercise bout in both trials (4.8 ± 1.9% and 5.1 ± 2.2% in the HGI and LGI trials respectively) ($p<0.05$) however there were no differences between the trials. During the postprandial periods, plasma volume was restored to pre-exercise levels.

Plasma glucose and serum insulin

Following ingestion of the HGI and LGI breakfasts, plasma glucose concentrations increased rapidly and peaked at 15min during the postprandial period in both conditions. The peak concentration following the HGI breakfast was greater than following the LGI breakfast (7.8 ± 0.3 $mmol.l^{-1}$ and 6.0 ± 0.4 $mmol.l^{-1}$ respectively) ($p<0.01$) (Fig. 1). Thereafter, plasma glucose concentrations decreased in both trials until lunch nevertheless, values remained higher in the HGI trial than the LGI trial until 90 min into the postprandial period ($p<0.05$). Plasma glucose concentrations did not fall below pre-exercise fasting values in either trial. Following lunch, the increase in plasma glucose concentrations was considerably less than that seen following breakfast. Again, the peak plasma glucose concentrations occurred at 15 min after the

meal in both trials and was larger in the HGI trial compared to the LGI trial (5.9 ± 0.3 $mmol.l^{-1}$ and 5.9 ± 0.3 $mmol.l^{-1}$ respectively) ($p<0.05$). Following this peak, plasma glucose concentrations remained stable in the LGI trial however in the HGI trial, a decline was again seen with concentrations falling below fasting concentrations by 120 min after the meal.

In both trials, serum insulin concentrations peaked 30 min after breakfast (66.9 ± 7 $\mu lU.ml^{-1}$ and 55.1 ± 5.9 $\mu lU.ml^{-1}$ in the HGI and LGI trials respectively) (NS) and then declined for the rest of the postprandial period (Fig. 2). Throughout the last hour of the postprandial period following breakfast, serum insulin concentrations were higher in the HGI trial compared to the LGI trial ($p<0.01$). In comparison to the response to breakfast, the serum insulin response to the lunch was considerably greater in the HGI trial and considerably lower in the LGI trial. Peak insulin concentrations occurred 15 min after the meal in both trials, however the peak was considerably greater in the HGI trial than the LGI trial (113.8 ± 13.7 $\mu lU.ml^{-1}$ and 38.4 ± 2.9 $\mu lU.ml^{-1}$ respectively.) ($p<0.005$) Following the peak, serum insulin concentrations declined rapidly in the HGI trial but remained fairly constant in the LGI trial. Despite this, concentrations remained significantly higher throughout the postprandial period following lunch in the HGI trial compared to the LGI trial ($p<0.01$).

The incremental area under the curve (IAUC) for both glucose (235.9$mmol.l^{-1}.120min^{-1}$ vs. 115.6$mmol.l^{-1}.120min^{-1}$) and insulin (5435$\mu lU.ml^{-1}.120min^{-1}$ vs. 3440 $\mu lU.ml^{-1}.120min^{-1}$) over the postprandial period following breakfast was significantly greater in the HGI trial compared to the LGI trial ($p< 0.005$). Following lunch, the IAUC for insulin was significantly greater in the HGI trial compared to the LGI trial

(3483 µlU.ml^{-1}.120min^{-1} vs. 1250 µlU.ml^{-1}.120min^{-1}) ($p<0.005$) however no differences in the IAUC for plasma glucose were seen (29.42 mmol.l^{-1}.120min^{-1} vs. 23.01 mmol.l^{-1}.120min^{-1}) (NS).

Serum Cortisol

Throughout the postprandial period following breakfast there were no differences in serum cortisol concentrations between trials however there was a trend for concentrations to be higher in the HGI trial. Following lunch, serum cortisol concentrations were significantly higher at 15, 30 and 60 min during the postprandial period in the LGI trial ($p<0.05$) (Fig.. 3).

Plasma free fatty acids (FFA) and glycerol

In both trials, plasma FFA concentrations peaked 30 min after exercise (i.e. just before breakfast was consumed). Following both the HGI and LGI meals FFA concentrations were reduced however there was a trend for concentrations to be higher in the LGI trial compared to the HGI trial ($p=0.06$). This trend was observed throughout the whole recovery period (Fig. 4). Plasma glycerol concentrations peaked at 90 min of exercise and again were suppressed following consumption of both the HGI and LGI meals. Once more, there was a trend for plasma glycerol concentrations to be higher throughout the recovery period in the LGI trial compared to the HGI trial ($p=0.08$) (Fig. 5).

Blood Lactate

At the end of R1, blood lactate concentrations were 1.6 ± 0.2 mmol.l^{-1} and 1.5 ± 0.2 mmol.l^{-1} in the HGI and LGI trials respectively (NS). At 15 min and 30 min during the

postprandial period following breakfast, blood lactate concentrations were significantly higher in the LGI trial compared to the HGI trial ($p<0.05$). Following lunch, blood lactate concentrations increased slightly however there were no differences between trials (Fig. 6).

Estimated carbohydrate and fat oxidation rates

Throughout the recovery period there were no significant differences in the estimated rate of fat (Fig. 7) and carbohydrate (Fig. 8) oxidation between trials. During the first 30 min of the postprandial period following breakfast, there was a trend for fat oxidation to be higher and carbohydrate oxidation to be lower in the HGI trial than the LGI trial but this did not persist throughout the remainder of the postprandial period.

Following lunch, there was a trend for fat oxidation to be higher and carbohydrate oxidation to be lower in the LGI trial. This is reflected in the slightly lower respiratory exchange ratio (RER) values throughout this time (0.90 ± 0.10 and 0.87 ± 0.10 in the HGI trial and LGI trial respectively) (NS) (Table 2).

Gut fullness, hunger and thirst scales

Subjects reported significantly higher ratings of gut fullness ($p<0.05$) and significantly lower ratings of hunger ($p<0.05$) throughout the recovery period in the LGI trial compared to the HGI trial. There was no significant difference in the ratings of perceived thirst between the two trials (Table 3).

Discussion

The recovery of skeletal muscle from exercise is critical so that performance during subsequent exercise is not impaired. Central to this recovery process is the resynthesis of muscle glycogen stores. To facilitate muscle glycogen resynthesis, it is recommended that high glycemic index (HGI) carbohydrates are consumed immediately after exercise as the ingestion of HGI foods is associated with high blood glucose and insulin concentrations [6]. In the present study, participants were fed a high carbohydrate HGI or LGI breakfast 30 min after the cessation of prolonged strenuous exercise. Following ingestion of the two meals there were no differences in the insulinaemic responses during the first hour of the postprandial period. This finding is in agreement with Burke et al. [5] who also reported that a HGI and a LGI meal consumed immediately post exercise produced a insulin response that was independent of the GI of the foods.

When muscle glycogen is severely reduced following prolonged strenuous exercise, resynthesis becomes a metabolic priority in the recovery period. Two phases of muscle glycogen resynthesis have been observed during the post-exercise period [17]. On completion of exercise an initial, rapid insulin-independent phase of glycogen repletion occurs followed by a more prolonged insulin-dependent phase [15]. During the insulin-dependent phase, the muscle demonstrates a marked increase in the sensitivity and responsiveness of glucose transport and glycogen resynthesis to insulin [15]. This is made possible by an increase in GLUT-4 transporter proteins and an increase in the activation of the enzyme glycogen synthase [26]. The similar insulin responses to the HGI and LGI breakfasts observed in this study may therefore be

explained by an increase in insulin sensitivity following the exercise bout. Acute exercise has previously been shown to decrease the insulin response to an oral glucose tolerance test suggesting that peripheral insulin sensitivity is increased [31]. Indeed, the insulin concentrations observed in this study were considerably lower than concentrations observed when the same meals were consumed without previous exercise (unpublished observation).

In the present study, plasma glucose concentrations were significantly higher 15 min after breakfast in the HGI trial compared to the LGI trial. This is in contrast to the results from the study by Burke et al. [5] who reported that the meal provided immediately after exercise produced a large glycemic response that was independent of the GI of the foods eaten. The author suggested that this effect be due to a selective hepatic insulin insensitivity.

The second meal consumed during the recovery period resulted in very different plasma glucose and serum insulin responses compared to those following breakfast. The incremental area under the curve (IAUC) for serum insulin following the HGI lunch was significantly greater than the response to the LGI lunch however there were no differences in the IAUC for plasma glucose. The exercise-induced changes in insulin sensitivity of muscle glucose transport are linked to carbohydrate availability in the post-exercise period [27]. Muscle glycogen itself is thought to be an important regulator of enhanced insulin action on glucose metabolism following exercise [26]. Therefore a high carbohydrate intake early in the post-exercise recovery period increases muscle glycogen resynthesis and may reduce insulin sensitivity. Despite this, it has been reported that muscle glycogen concentrations alone cannot entirely

explain the changes in insulin sensitivity after exercise [7]. This has been clearly demonstrated by the fact that rodents continue to show enhanced insulin sensitivity beyond the point of full glycogen resynthesis [7]. Although the insulin response to the HGI lunch was significantly greater than the LGI lunch, the responses to both meals were still lower than the responses to the same meals observed when no exercise was carried out (unpublished observation). This suggests that despite a large intake of carbohydrate during the recovery period, enhanced insulin sensitivity may persist but to a lesser extent than when no carbohydrate has been consumed.

The frequency of carbohydrate feedings during the first 4-6h following prolonged exercise has been shown to influence the rate of muscle glycogen resynthesis. Several studies have reported that carbohydrate feedings provided at 15-30min intervals result in higher rates of muscle glycogen resynthesis compared to less frequent feedings [23, 24]. This has been attributed to the higher sustained insulin and glucose profiles achieved by such a feeding protocol. In the present study, the meals were provided 2h apart and a reduction in plasma glucose and serum insulin concentrations were observed between the meals. As discussed however, the second HGI meal resulted in a larger insulin response than the LGI meal. The GI of the pre-exercise feedings may therefore not be so important if the carbohydrate is given in small frequent feedings over a short recovery period. However when meals are provided at longer time intervals, the GI of the carbohydrates consumed greatly effects the metabolic responses to the meals and perhaps therefore muscle glycogen resynthesis.

Following R1, FFA and glycerol concentrations increased during the first 30 min of recovery however the ingestion of breakfast suppressed these metabolites in both

trials. High insulin concentrations suppress FFA mobilisation [10]. In the present study, the reduction in the concentration of FFA was not as marked as may be expected. This is probably due to the fact that the approximate energy expenditure of the 90 min run was 1300kcal and the test breakfasts provided only approximately 730 kcal (70kg person). Participants were therefore in a negative energy balance throughout the first part of the recovery period. Other studies have reported high FFA concentrations throughout the post-exercise recovery period despite a large intake of carbohydrate [12, 13]. The depletion of muscle glycogen from the previous exercise creates a fuel deficit in the muscle. It is hypothesised by the authors that the exogenous glucose is directed to the muscle for glycogenolysis and FFA are therefore required for oxidative muscle metabolism [12]. Following lunch, there was a trend for free fatty acid and glycerol concentrations to be higher in the LGI trial than the HGI trial. The higher insulin concentrations in the HGI trial may have suppressed free fatty acid and glycerol concentrations to a greater extent than in the LGI trial. There was also a trend for the estimated fat oxidation rate to be higher and estimated carbohydrate oxidation rate to be lower in the LGI trial. This is also reflected in slightly lower respiratory exchange ratio values in the LGI trial.

Interestingly, blood lactate concentrations were elevated during the postprandial period following ingestion of the HGI and LGI breakfasts but were significantly higher in the LGI trial. Several studies have reported elevated blood lactate concentrations following the ingestion of LGI CHO-containing foods [19, 30]. Although the mechanism is still unclear, studies have reported increased lactate concentrations following the ingestion of fructose [14, 16] . In the present study, the LGI breakfast contained more fructose (25g/70kg man) than the HGI breakfast

(11g/70kg man) and therefore this may explain the significantly higher blood lactate concentrations in the postprandial period. No differences in blood lactate concentrations were seen following the ingestion of lunch however very little fructose was present in these meals.

In both trials, serum cortisol concentrations were highest at the end of the exercise period but remained high throughout the first two hours of recovery. As previously mentioned, the energy content of the first meal was insufficient to replace the energy expended during the exercise bout. All the subjects were therefore in a negative energy balance throughout the first part of the recovery period and therefore this is likely to have caused high cortisol concentrations. During the first hour following lunch, serum cortisol concentrations were higher in the LGI trial than the HGI trial. The fibre content of the LGI lunch was higher than the HGI lunch, therefore it is possible that it was more difficult to digest causing a larger stress on the body.

Throughout the postprandial period following both meals, the sensation of gut fullness was reported to be higher in the LGI trial and ratings of hunger were lower. This was despite the fact that the meals were isocaloric and contained the same nutrient composition. Several studies have reported higher satiety ratings following the ingestion of LGI foods compared to HGI foods [1, 3]. This is of importance to those who want to lose weight as maintaining a sense of satiety for a longer period may help to restrict food intake.

In conclusion, the results of the present study provide further evidence that insulin sensitivity is increased following an acute bout of exercise. Additionally, the results suggest that the glycemic index of the carbohydrates consumed immediately after exercise may not be important as long as sufficient carbohydrate is consumed and perhaps provided as small frequent feedings. However, the GI of the carbohydrates consumed later in the post-exercise period may be important due to their influence on substrate oxidation. The results of this study suggest that a LGI diet may be more beneficial for continued utilisation of fat during the recovery period. Although muscle glycogen was not measured in the current study, it would be reasonable to speculate that the high insulin concentrations following a HGI meal may facilitate further muscle glycogen resynthesis later in the recovery period.

References

1. Ball, S.D., K.R. Keller, L.J. Moyer-Mileur, Y.W. Ding, D. Donaldson, and W.D. Jackson. Prolongation of satiety after low versus moderately high glycemic index meals in obese adolescents. *Pediatrics.*111(3):488-94,2003.
2. Borg, G.A. Perceived exertion: a note on "history" and methods. *Med Sci Sports Exerc*.5:90-93,1973.
3. Brand-Miller, J.C., S.H. Holt, D.B. Pawlak, and J. McMillan. Glycemic index and obesity. *Am J Clin Nutr*.76:281S-5S,2002.
4. Burke, L.M., G.R. Collier, P.G. Davis, P.A. Fricker, A.J. Sanigorski, and M. Hargreaves. Muscle glycogen storage after prolonged exercise: effect of the frequency of carbohydrate feedings. *American Journal of Clinical Nutrition*.64(1):115-119,1996.
5. Burke, L.M., G.R. Collier, and M. Hargreaves. Muscle glycogen storage after prolonged exercise: effect of the glycemic index of carbohydrate feedings. *J Appl Physiol*.75(2):1019-23,1993.
6. Burke, L.M., B. Kiens, and J.L. Ivy. Carbohydrates and fat for training and recovery. *J Sports Sci*.22:15-30,2004.
7. Cartee, G.D., D.A. Young, M.D. Sleeper, J. Zierath, H. Wallberg-Henriksson, and J.O. Holloszy. Prolonged increase in insulin-stimulated glucose transport in muscle after exercise. *Am J Physiol.*256(4 Pt 1):E494-9,1989.
8. Dill, D.B. and D.L. Costill. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol.*37:247-248,1974.
9. Frayn, K.N. Calculation of substrate oxidation rates in vivo from gaseous exchange. *J Appl Physiol*.55(2):628-634,1983.

10. Horowitz, J.F., R. Mora-Rodriguez, L.O. Byerley, and E.F. Coyle. Lipolytic suppression following carbohydrate ingestion limits fat oxidation during exercise. *Am J Physiol*.273(4 Pt 1):E768-75,1997.

11. Ivy, J.L., A.L. Katz, C.L. Cutler, W.M. Sherman, and E.F. Coyle. Muscle glycogen synthesis after exercise: effect of time of carbohydrate ingestion. *J Appl Physiol*.64(4):1480-5,1988.

12. Kiens, B. and E.A. Richter. Utilization of skeletal muscle triacylglycerol during postexercise recovery in humans. *Am J Physiol*.275(2 Pt 1):E332-7,1998.

13. Kimber, N.E., G.J. Heigenhauser, L.L. Spriet, and D.J. Dyck. Skeletal muscle fat and carbohydrate metabolism during recovery from glycogen-depleting exercise in humans. *J Physiol*.548(Pt 3):919-27,2003.

14. Koivisto, V.A., S.L. Karonen, and E.A. Nikkila. Carbohydrate ingestion before exercise: comparison of glucose, fructose, and sweet placebo. *J Appl Physiol*.51(4):783-7,1981.

15. Kuo, C.H., H. Hwang, M.C. Lee, A.L. Castle, and J.L. Ivy. Role of insulin on exercise-induced GLUT-4 protein expression and glycogen supercompensation in rat skeletal muscle. *J Appl Physiol*.96(2):621-7,2004.

16. Moore, M.C., A.D. Cherrington, S.L. Mann, and S.N. Davis. Acute fructose administration decreases the glycemic response to an oral glucose tolerance test in normal adults. *J Clin Endocrinol Metab*.85(12):4515-9,2000.

17. Price, T.B., D.L. Rothman, R. Taylor, M.J. Avison, G.I. Shulman, and R.G. Shulman. Human muscle glycogen resynthesis after exercise: insulin-dependent and -independent phases. *J Appl Physiol*.76(1):104-11,1994.

18. Shirreffs, S.M. and R.J. Maughan. Urine osmolality and conductivity as indices of hydration status in athletes in the heat. *Med Sci Sports Exerc*.30(11):1598-602,1998.

19. Stannard, S.R., N.W. Constantini, and J.C. Miller. The effect of glycemic index on plasma glucose and lactate levels during incremental exercise. *Int J Sport Nutr Exerc Metab*.10(1):51-61,2000.

20. Stevenson, E.J., C. Williams, G. McComb, and C. Oram. Improved recovery from prolonged exercise following the consumption of low glycaemic index carbohydrate meals. *Med Sci Sports Exerc*.36(5):S21,2004.

21. Tsintzas, K. and C. Williams. Human Muscle Glycogen Metabolism During Exercise: Effect of Carbohydrate Supplementation. *Sports Med*.25(1):7-23,1998.

22. Tsintzas, O.K., C. Williams, L. Boobis, and P. Greenhaff. Carbohydrate ingestion and single muscle fiber glycogen metabolism during prolonged running in men. *Journal of Applied Physiology*.81(2):801-809,1996.

23. Van Hall, G., S.M. Shirreffs, and J.A. Calbet. Muscle glycogen resynthesis during recovery from cycle exercise: no effect of additional protein ingestion. *J Appl Physiol*.88(5):1631-1636,2000.

24. van Loon, L.J., W.H. Saris, M. Kruijshoop, and A.J. Wagenmakers. Maximizing postexercise muscle glycogen synthesis: carbohydrate supplementation and the application of amino acid or protein hydrolysate mixtures. *Am J Clin Nutr*.72(1):106-11,2000.

25. Williams, C., M.G. Nute, L. Broadbank, and S. Vinall. Influence of fluid intake on endurance running performance. A comparison between water,

glucose and fructose solutions. *Eur J Appl Physiol Occup Physiol*.60(2):112-9,1990.

26. Wojtaszewski, J., S. Jorgensen, C. Frosig, C. MacDonald, J.B. Birk, and E.A. Richter. Insulin signalling: effects of prior exercise. *Acta Physiol Scand*.178:321-328,2003.

27. Wojtaszewski, J.F., J.N. Nielsen, and E.A. Richter. Invited review: effect of acute exercise on insulin signaling and action in humans. *J Appl Physiol*.93(1):384-92,2002.

28. Wolever, T.M. and D.J. Jenkins. The use of glycemic index in predicting the blood glucose response to mixed meals. *Am J Clin Nutr*.43:167-172,1986.

29. Wolever, T.M., D.J. Jenkins, A.L. Jenkins, and R.G. Josse. The glycemic index: methodology and clinical implications. *Am J Clin Nutr*.54(5):846-54,1991.

30. Wu, C.L., C. Nicholas, C. Williams, A. Took, and L. Hardy. The influence of high-carbohydrate meals with different glycaemic indices on substrate utilisation during subsequent exercise. *Br J Nutr*.90(6):1049-56,2003.

31. Young, J.C., J. Enslin, and B. Kuca. Exercise intensity and glucose tolerance in trained and nontrained subjects. *J Appl Physiol*.67(1):39-43,1989.

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

Meal	Description	Macronutrient Content
HGI breakfast	62g cornflakes[Ψ] + 257ml skimmed milk	730kcal, 139gCHO, 9.9g
	80g white bread + 10g flora +20g jam	fat, 20g protein
	155ml lucozade original[Ψ]	
LGI breakfast	86g muesli + 257ml skimmed milk	732 kcal, 139g CHO, 9g
	67g apple, 103g tinned peaches, 128g	fat, 23g protein
	yoghurt 257ml apple juice	
HGI lunch	158g white bread, 154g turkey breast,	1076kcal, 148g CHO, 24g
	50g cheese, 40g lettuce, 180g banana	fat, 63g protein
	200ml lucozade original[Ψ]	
LGI lunch	154g wholewheat pasta, 150g turkey	1075 kcal, 149g CHO, 25g
	breast, 50g cheese, 40g lettuce, 185g	fat, 60g protein
	pasta sauce, 150g pear, 150 ml apple	
	juice	
HGI total		1806kcal, 287g CHO, 33.9g fat
		83g protein (72%CHO, 11%
		fat, 17% protein)
		GI = 70*
LGI total		1807kcal,288gCHO, 34g fat
		83g 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).

[Ψ] Corn Flakes: Kellogg's (UK) Ltd. Manchester UK; Lucozade Original drink: GlaxoSmithKline (UK).

Table 2. Oxygen uptake (VO_2), carbon dioxide expired (VCO_2), and the respiratory exchange ratio (RER) during the high glycemic index (HGI) and low glycemic index (LGI) CHO trials (mean ± SEM).

Variable	Trial	Resting	Exercise Period	Postprandial Period 1	Postprandial Period 2
VO_2	**HGI**	0.36 ± 0.02	3.26 ± 0.1	0.39 ± 0.01	0.41± 0.02
	LGI	0.39 ± 0.02	3.28 ± 0.11	0.4 ± 0.01	0.38 ± 0.01
VCO_2	**HGI**	0.31 ± 0.01	2.91 ± 0.09	0.33 ± 0.01	0.36 ± 0.02
	LGI	0.32 ± 0.01	2.89 ± 0.11	0.34 ± 0.01	0.34 ± 0.11
RER	**HGI**	0.86 ± 0.02	0.89 ± 0.01	0.87 ± 0.01	0.90 ± 0.01
	LGI	0.82 ± 0.01	0.89 ± 0.01	0.87 ± 0.01	0.87 ± 0.01

Table 3. Gut fullness (GF), hunger and thirst scale ratings during the HGI and LGI CHO trials (mean ± SEM)

		Postprandial Period Meal 1					Postprandial Period Meal 2					
Variable	**Trial**	**FAST**	**15**	**30**	**60**	**90**	**120**	**15**	**30**	**60**	**90**	**120**
Gut fullness	**HGI**	7 ± 0	11 ± 1	11 ± 1	9 ± 1	9 ± 0	8 ± 0	14 ± 1	13 ± 1	11 ± 1	10 ± 1	10 ± 1
	LGI	7 ± 0	12 ± 1	11 ± 1	11 ± 1	9 ± 0	9 ± 1	14 ± 1	14 ± 1	13 ± 1	12 ± 1	11 ± 1
Hunger	**HGI**	17 ± 1	12 ± 1	12 ± 1	12 ± 1	15 ± 1.0	15 ± 1	10 ± 1	10 ± 1	11 ± 0	12 ± 1	14 ± 1
	LGI	17 ± 1	10 ± 1	10 ± 0	10 ± 1	12 ± 0	15 ± 1	9 ± 1	10 ± 1	10 ± 1	11 ± 1	11 ± 1
Thirst	**HGI**	14 ± 1	10 ± 1	11 ± 1	10 ± 1	10 ± 0	11 ± 1	9 ± 1	10 ± 1	10 ± 1	10 ± 1	11 ± 1
	LGI	12 ± 1	11 ± 1	10 ± 1	10 ± 1	10 ± 0	11 ± 1	10 ± 1	11 ± 1	11 ± 0	10 ± 0	11 ± 1

Gut Fullness and hunger significantly higher in the LGI trial compared to the HGI trial ($p<0.05$)

Figure Captions

Fig. 1. Plasma glucose concentrations ($mmol \cdot l^{-1}$) in the HGI and LGI trials (mean ± SEM). * HGI trial significantly higher than LGI trial.

Fig. 2. Serum insulin concentrations ($\mu lU.ml^{-1}$) in the HGI and LGI trials (mean ± SEM). * HGI trial significantly higher than LGI trial.

Fig. 3. Serum cortisol concentrations ($\mu g.dL^{-1}$) in the HGI and LGI trials (mean ± SEM). * LGI trial significantly higher than HGI trial.

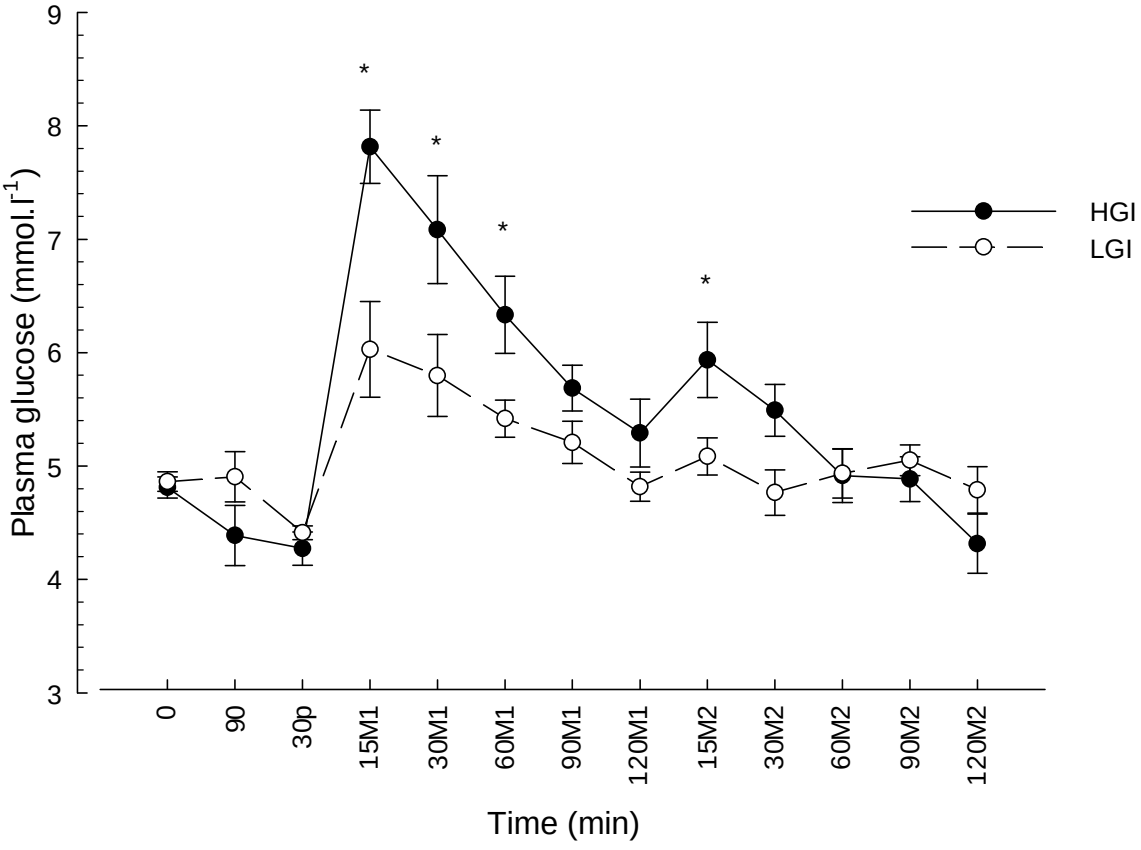
Fig. 4. Plasma FFA concentrations ($mmol \cdot l^{-1}$) during the recovery period in the HGI and LGI trials (mean ± SEM).

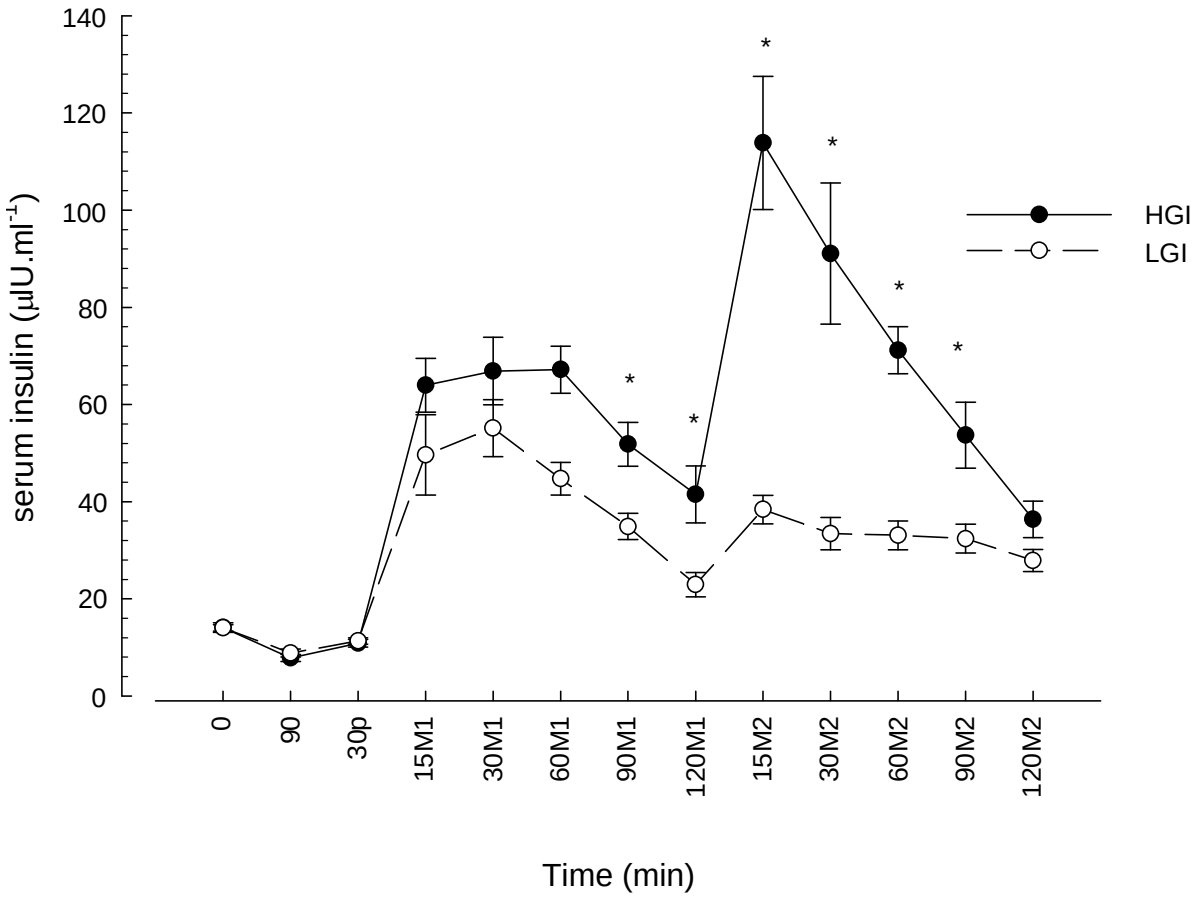
Fig. 5. Plasma glycerol concentrations ($\mu mol \cdot l^{-1}$) during the recovery period in the HGI and LGI trials (mean ± SEM).

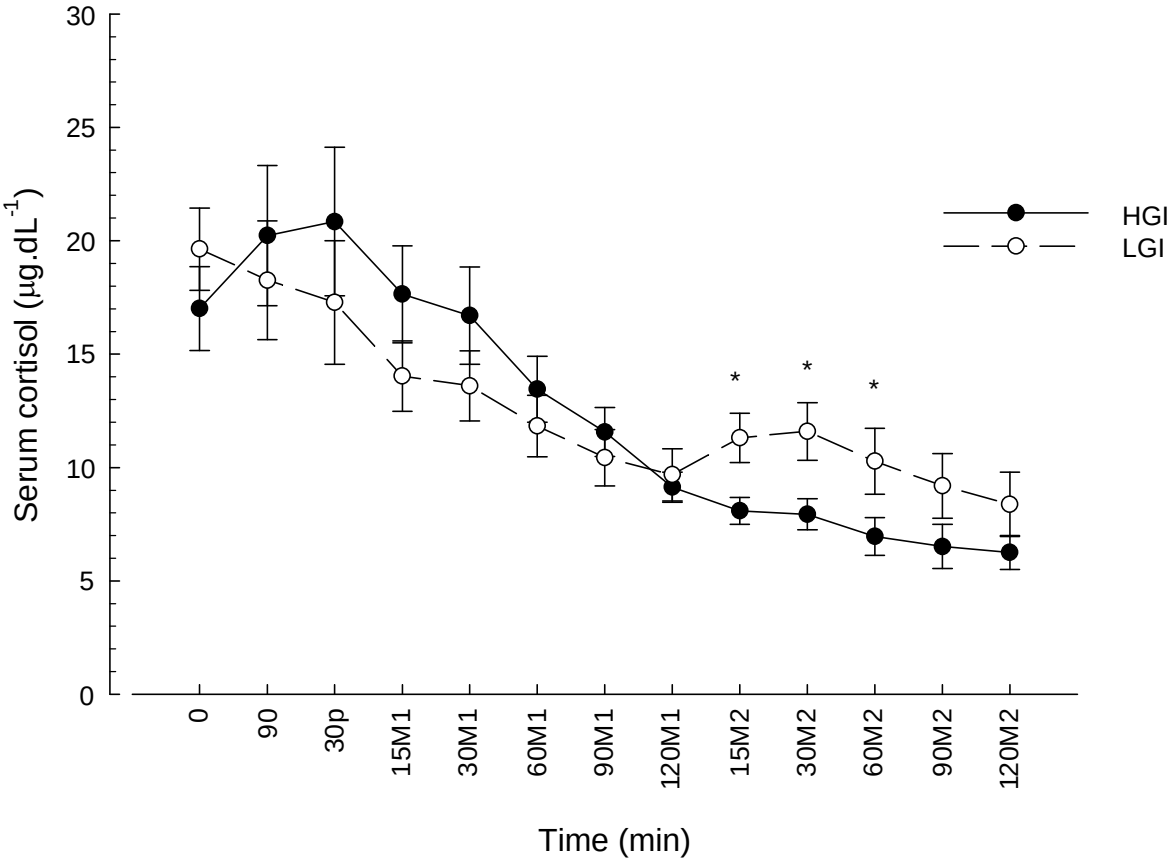
Fig. 6. Blood lactate concentrations ($mmol.l^{-1}$) in the HGI and LGI trials (mean ± SEM). * LGI trial significantly higher than HGI trial.

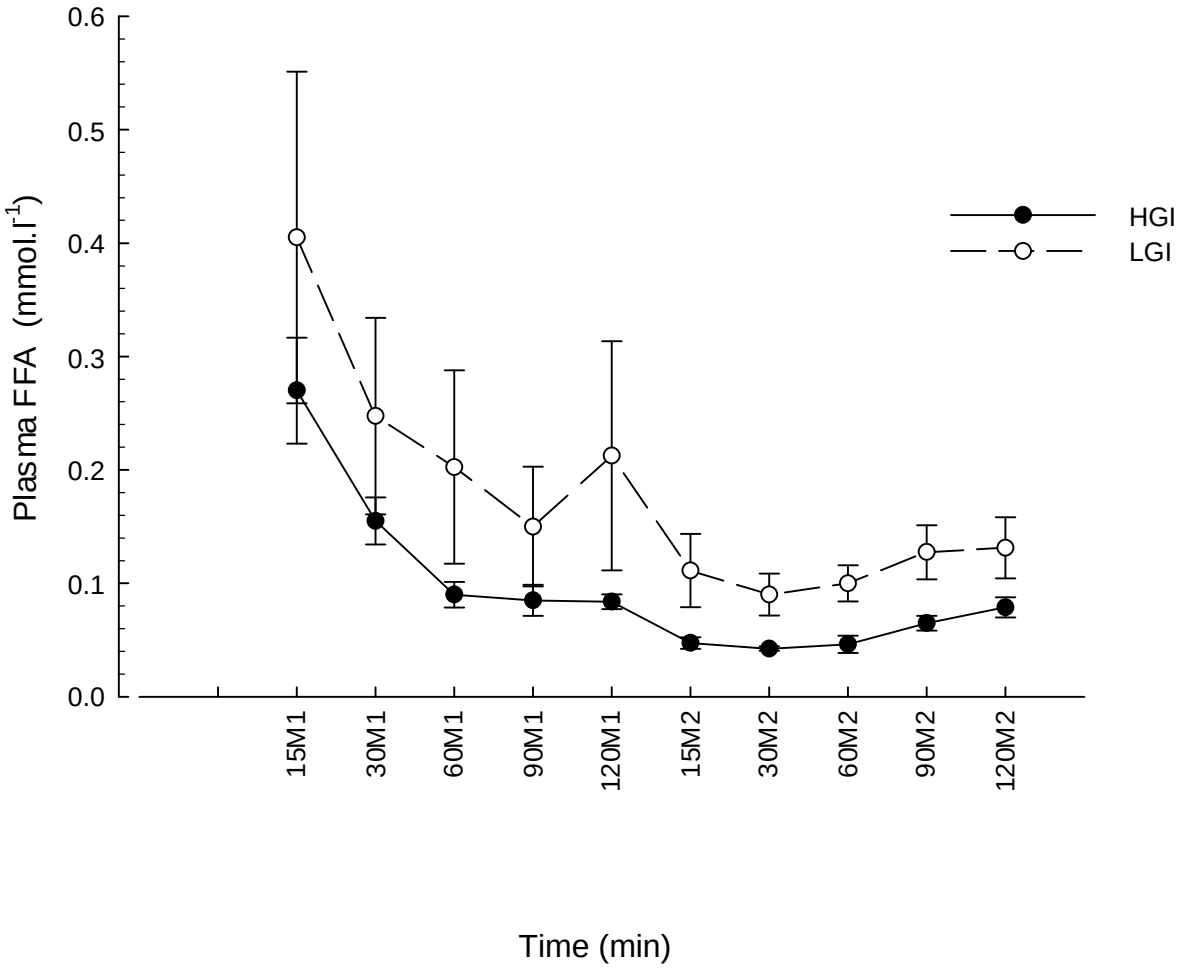
Fig. 7. Estimated rate of fat oxidation ($g.min^{-1}$) during the recovery period in the HGI and LGI trials (mean ± SEM).

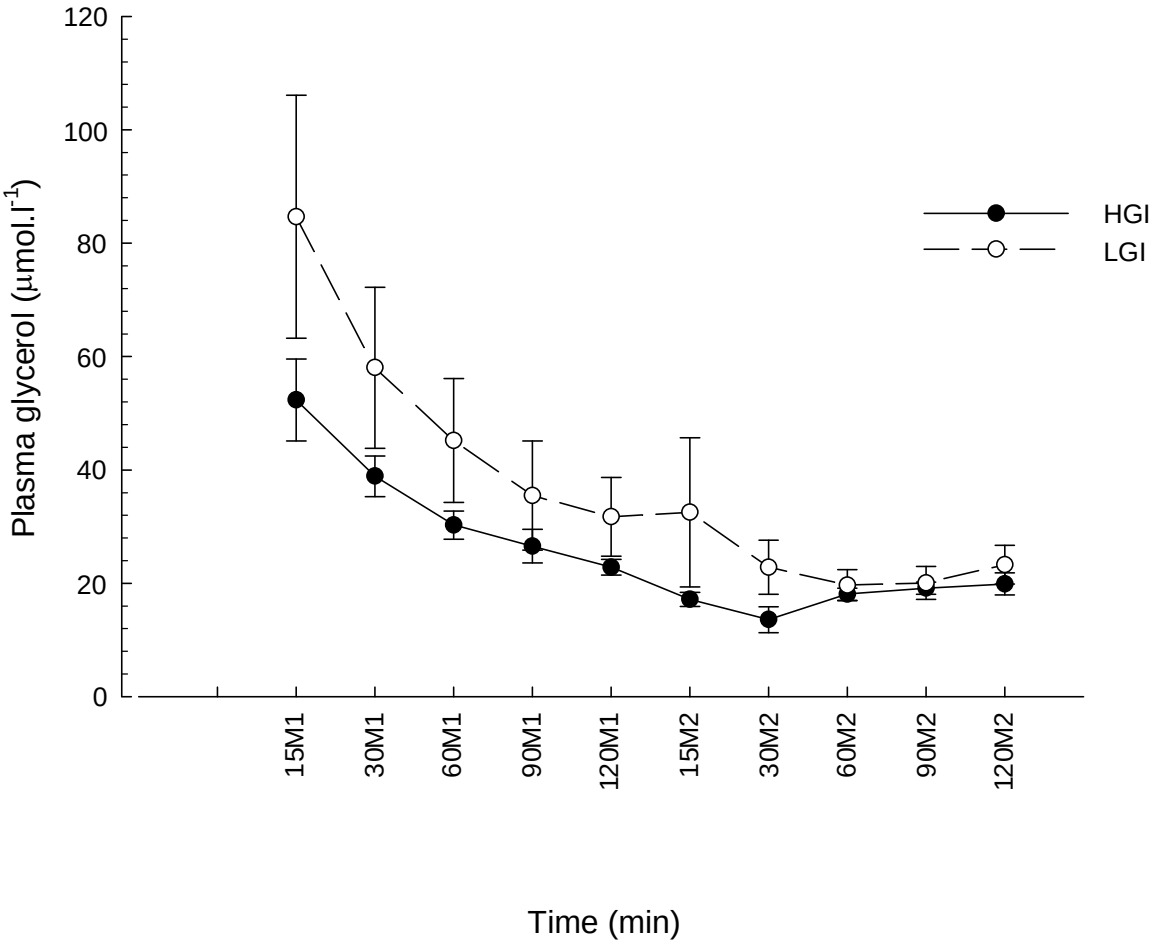
Fig. 8. Estimated rate of carbohydrate oxidation ($g.min^{-1}$) during the recovery period in the HGI and LGI trials (mean ± SEM).

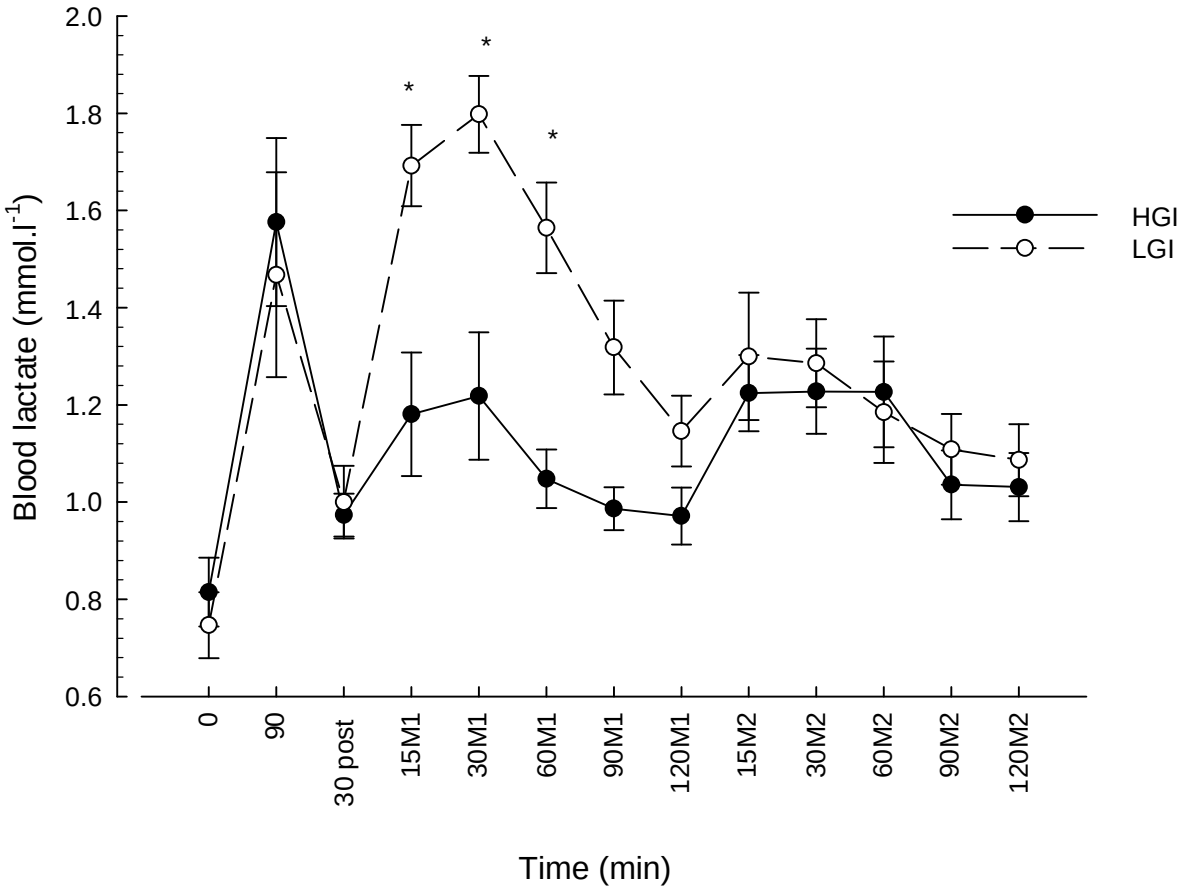

Plasma glucose (mmol.l-1)
9
8
7
6
5
4
3
*
*
*
*
HGI
LGI
0
90
30p
15M1
30M1
60M1
90M1
120M1
15M2
30M2
60M2
90M2
120M2
Time (min)

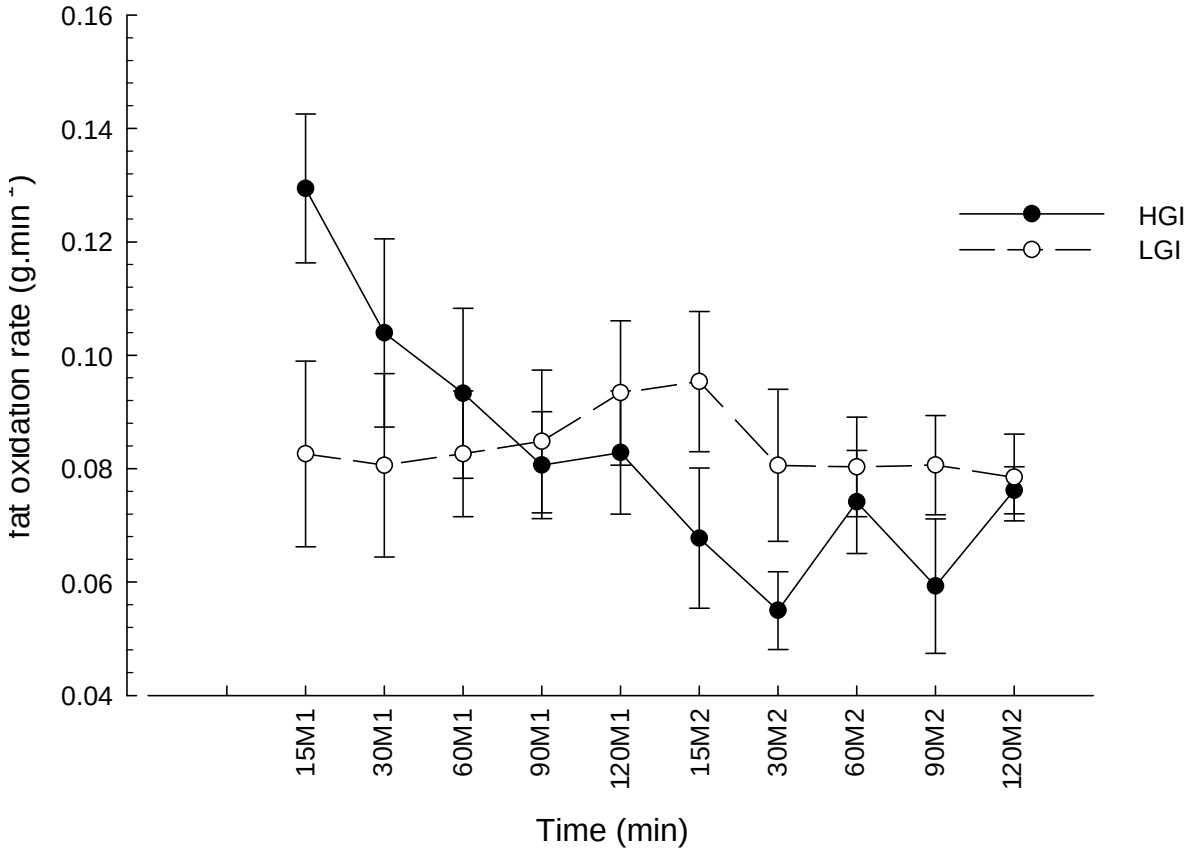

140
120
100
80
60
40
20
0
serum insulin (µIU.ml-1)
HGI
LGI
*
*
*
*
*
*
0
90
30p
15M1
30M1
60M1
90M1
120M1
15M2
30M2
60M2
90M2
120M2
Time (min)

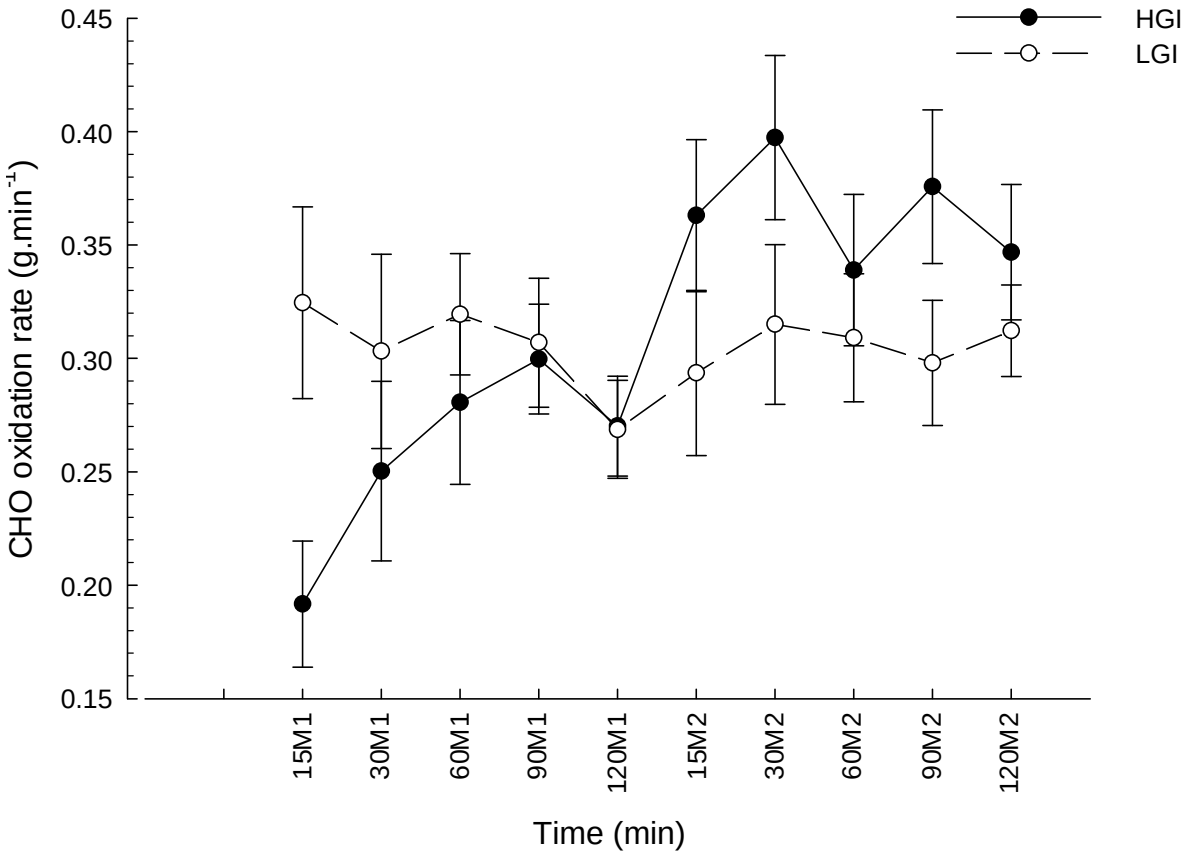

Serum cortisol (μg.dL-1)
30
25
20
15
10
5
0
0
90
30p
15M1
30M1
60M1
90M1
120M1
15M2
30M2
60M2
90M2
120M2
Time (min)
HGI
LGI
*
*
*


Plasma FFA (mmol.l-1)
0.0
0.1
0.2
0.3
0.4
0.5
0.6
HGI
LGI
15M1
30M1
60M1
90M1
120M1
15M2
30M2
60M2
90M2
120M2
Time (min)


120
100
80
60
40
20
0
Plasma glycerol (µmol.l-1)
HGI
LGI
15M1
30M1
60M1
90M1
120M1
15M2
30M2
60M2
90M2
120M2
Time (min)


Blood lactate (mmol.l-1)
2.0
1.8
1.6
1.4
1.2
1.0
0.8
0.6
*
*
*
HGI
LGI
0
90
30 post
15M1
30M1
60M1
90M1
120M1
15M2
30M2
60M2
90M2
120M2
Time (min)


0.16
0.14
0.12
0.10
0.08
0.06
0.04
fat oxidation rate (g.min⁻¹)
HGI
LGI
15M1
30M1
60M1
90M1
120M1
15M2
30M2
60M2
90M2
120M2
Time (min)


HGI
LGI
CHO oxidation rate (g.min-1)
0.45
0.40
0.35
0.30
0.25
0.20
0.15
15M1
30M1
60M1
90M1
120M1
15M2
30M2
60M2
90M2
120M2
Time (min)