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Title: The effect of the glycemic index of an evening meal on the metabolic responses to a standard high glycemic index breakfast and subsequent exercise in men.

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Abstract

The present study was designed to investigate the effect of changing the glycemic index of an evening meal on responses to a standard high glycemic index (HGI) breakfast the following morning in men. The metabolic responses to an exercise bout 3 h after breakfast were also investigated. Seven recreationally active males completed two trials separated by at least a week in a randomised crossover design. In each trial, participants were provided with their evening meal on day 1, which was composed of either HGI or LGI (low glycemic index) carbohydrates. On day 2, participants were provided with a standard HGI breakfast and then performed a 60 min run at 65% VO_2max 3 h later. Plasma glucose concentrations following the HGI breakfast were higher when the HGI evening meal had been consumed compared to when the LGI evening meal was consumed ($p<0.05$). Similarly, serum insulin concentrations were higher in the HGI trial than in the LGI trial ($p<0.05$). During the subsequent sub-maximal run, there were no differences in substrate utilisation. The results of the study suggest that consuming a single LGI evening meal can improve glucose tolerance at breakfast but the metabolic responses to a subsequent exercise bout were not affected.

Key Words: hyperinsulinemia, glucose tolerance, second-meal effect, substrate utilisation

Introduction

A high carbohydrate meal is recommended 3-4 h before exercise to provide a sustainable source of carbohydrate during the exercise bout [12]. Despite this, ingestion of carbohydrate in the hours before exercise often results in a transient fall in glucose with the onset of exercise, increased carbohydrate oxidation and a blunting of free fatty acid (FFA) mobilisation [5, 16]. The glycemic index has been identified as a useful reference guide for the selection of the ideal CHO supplementation for exercise [18]. A number of studies have examined the effect of consuming carbohydrates with different glycemic indices before exercise [6, 10, 19, 20, 22, 26]. Studies investigating the effects on performance have yielded inconsistent results. Thomas et al [20] first reported an increased endurance capacity following ingestion of a LGI CHO 45 min before exercise compared to when a HGI CHO food was consumed. Subsequent studies have however reported no differences in endurance capacity [10, 19, 22] . Despite this discrepancy in the literature, all studies investigating the effects of the GI of a pre-exercise meal have demonstrated lower postprandial glycemia and insulinemia following a LGI meal or single food. This is accompanied by higher concentrations of plasma free fatty acids (FFA) and therefore higher rates of fat oxidation during exercise compared to the responses following ingestion of a HGI food [8-10, 26].

Low glycemic index pre-exercise meals are therefore recommended to athletes to maintain plasma glucose and free fatty acid concentrations during exercise without a large stimulation of insulin release in the period before exercise [20]. Despite this, athletes do not always adhere to the recommendations. Feelings of abdominal discomfort are often reported following the ingestion of LGI foods, which may be

explained by the high fibre nature of many LGI carbohydrates. From a practical perspective, this is not desirable before a training session or competition.

Studies carried out by Wolever et al [25], Jenkins et al [13] and more recently Liljeberg et al [14], have reported that a single LGI meal can improve glucose tolerance and therefore reduce hyperinsulinemia at a second meal. The study carried out by Wolever et al reported that this effect even occurred after an overnight fast. It was reported that consumption of a LGI evening meal improved glucose tolerance and therefore reduced the insulin responses to a standard HGI breakfast compared to when a HGI evening meal was consumed [25].

None of the previous studies investigating the second meal effect have considered whether the responses to exercise following the standard HGI breakfast would be different. If a LGI meal can reduce the hyperglycemia and hyperinsulinemia following a standard HGI breakfast, a similar effect may be achieved when a LGI pre-exercise meal is consumed and therefore there may be a higher rate of fat oxidation during the exercise bout.

Therefore the aim of the present study was to re-investigate whether a LGI meal consumed in the evening can reduce the glycemic and insulinemic response to a HGI standard breakfast compared to a HGI evening meal. The study also aimed to investigate whether the substrate utilisation during a subsequent exercise bout would be affected.

Methods

Subjects

Seven male recreational athletes participated in this study. Their mean (± SD) age, height, weight and $\dot{V}O_2$ max were 23.0 ± 2.8 years, 180 ± 1.0cm, 74.9 ± 7.4 kg and 61.1 ± 5ml kg^{-1} min^{-1} respectively. A criterion for inclusion into the study was that participants ran regularly and were able to run for one hour continuously at about 65% $\dot{V}O_2$max. Loughborough University Ethical Advisory Committee approved the protocol and all subjects gave their written informed consent.

Preliminary tests

Following familiarisation 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 [23]. Based on the results of the two preliminary tests, the running speed equivalent to 65% of each subject's $\dot{V}O_2$ max was determined.

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. On day 1, subjects were provided with an evening meal consisting of either high glycemic index (HGI) or low glycemic index (LGI) carbohydrates. On day 2, subjects arrived at the lab following a 13 h overnight fast and were provided with a standard HGI breakfast.

Following ingestion of breakfast, subjects remained in the lab at rest for 3 hours. At the end of this postprandial period, subjects completed a 60 min run at 65% $\dot{V}O_2$ max.

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 day 1 of the main trial, subjects were asked to record their food intake at breakfast and lunch. They were instructed not to consume any food after 4pm. At 7pm, subjects reported to the laboratory and after completing a health questionnaire, they were provided with their test meal. After ingestion of this meal, subjects were instructed not to consume any other food or beverages apart from water for the remainder of the evening.

On day 2 of the main trial, each subject arrived in the laboratory at 0800 h following a 13 h 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 saline solution (9g/l) immediately after the cannula was inserted and after blood sampling. A fasting 10ml venous blood sample and a 5 min resting expired air sample were collected and then the subjects were provided with the standard HGI breakfast. Subjects were asked to consume the breakfast within 15 min and then the 3 h postprandial period began.

During the postprandial 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, 120 and 180 min after breakfast. During each collection of expired air, ratings of gut fullness, hunger and thirst were recorded using 6-20 scales. The scales used were adapted from the Borg Scale [1] such that the anchor terms on each 6-20 scale ranged from 'not full' to 'very very full' and 'not thirsty' to 'very very thirsty' etc. No extra drinks, apart from water, were permitted throughout the trial. Water intake was monitored throughout trial one and matched in trial two.

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 50-60%.

Test Meals

Isocaloric evening meals consisting of HGI or LGI CHO foods were provided for each subject on day 1 of each trial (Table 1). On day 2, a standard HGI breakfast was 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. cheese and lettuce) 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 [24]. The calculated GI for the high and low GI evening meals were 72 and 34 respectively and the GI of the standard breakfast was calculated to be 79.

Sample Collection and Analysis

Expired air samples were collected and analysed as previously described [23]. 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 [11].

At each sampling point, 10ml 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 [7]. 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 -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 45 min. Serum samples were then obtained after centrifugation at 4000rpm for 10min at 4°C. The aliquoted serum was stored at -85°C and later analysed for insulin (Coat-A-Count Insulin 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} [17].

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

Plasma glucose and serum insulin

Following ingestion of the standard HGI breakfast, plasma glucose concentrations increased significantly above fasting concentrations ($p<0.001$) and peaked at 15 min during the postprandial period in both trials (Fig. 1). The peak concentration was

higher when the HGI evening meal had been consumed compared to when the LGI evening meal was consumed (8.1 ± 0.6 mmol.l^{-1} and 7.0 ± 0.38 mmol.l^{-1} respectively) ($p<0.05$). Following this peak, plasma glucose concentrations decreased in both trials however concentrations remained significantly higher in the HGI trial compared to the LGI trial at 60 min and 90 min during the postprandial period ($p<0.05$). No differences in plasma glucose concentrations were observed throughout the 60 min run.

In both trials, serum insulin concentrations increased significantly after ingestion of the standard HGI breakfast ($p<0.001$) and again, peaked at 15 min during the postprandial period (Fig. 2). The peak concentration was higher when the HGI evening meal had been consumed compared to when the LGI evening meal was consumed (181.6 ± 12.8 µlU.ml^{-1} and 144.4 ± 22.6 µlU.ml^{-1} respectively) ($p<0.05$). Throughout the rest of the postprandial period, serum insulin concentrations declined but remained higher in the HGI trial compared to the LGI trial. No differences in serum insulin concentrations were observed throughout the 60 min run.

The incremental area under the curve (IAUC) for plasma glucose during the postprandial period following breakfast was greater in the HGI trial than the LGI trial (175.2 ± 67.0 mmol.l^{-1} x 180 min and 78.8 ± 35.0 mmol.l^{-1} x 180 min respectively) ($p<0.01$). Similarly, the IAUC for serum insulin during the postprandial period was greater in the HGI trial than the LGI trial (15906 ± 1609 µlU.ml^{-1} x 180 min and 11592 ± 1234 µlU.ml^{-1} x 180 min respectively) ($p<0.05$).

Plasma free fatty acids (FFA) and glycerol

Following ingestion of the standard HGI breakfast, plasma FFA (Fig.3) and glycerol (Fig. 4) concentrations were significantly reduced in both trials ($p<0.01$) and concentrations remained below fasting concentrations throughout the postprandial period in both trials ($p<0.005$). No differences in plasma FFA or glycerol concentrations were reported between the trials during the postprandial period. During the exercise bout, plasma FFA and glycerol concentrations increased with time ($p<0.05$) however, there were no differences between the trials.

Blood lactate

In both trials, blood lactate concentrations increased significantly after ingestion of the carbohydrate breakfast and remained elevated above fasting concentrations throughout the remainder of both trials ($p<0.001$). No differences between the trials were observed throughout the experimental protocol . During the exercise bout, blood lactate concentrations were maintained at about 1.6 $mmol.l^{-1}$ in both trials (Fig 5).

Respiratory Exchange Ratio (RER) and estimated carbohydrate and fat oxidation rates

Following the ingestion of breakfast, there was no significant change in fat or carbohydrate oxidation and therefore RER from fasting values in either trial. Despite this, there was a trend for carbohydrate oxidation to increase and fat oxidation to decrease throughout the postprandial period in both trials. In both trials, the rate of fat and carbohydrate oxidation were only significantly different from fasting values during the 60 min run ($p<0.01$). There were no differences in the RER values between

trials (Table 2) and hence no differences were found for carbohydrate (Fig. 6) and fat (Fig. 7) oxidation rates during the postprandial period or the 60 min run.

Gut fullness, hunger and thirst scales

Before breakfast was consumed on the morning of day 2, subjects reported significantly higher ratings of gut fullness ($p<0.01$) and significantly lower ratings of hunger ($p<0.05$) in the LGI trial compared to the HGI trial. Following the ingestion of breakfast, ratings of gut fullness significantly increased ($p<0.01$) and hunger significantly decreased ($p<0.05$) from fasting values in both trials. Significantly higher ratings of gut fullness were reported in the LGI trial during the postprandial period following breakfast ($p<0.01$). No differences in gut fullness were observed during the subsequent 60 min run. There were no significant differences between trials in feelings of hunger but a trend for hunger ratings to be higher in the HGI trial. There were no differences in perceived thirst between the trials (Table 3).

Plasma volume changes and hydration status

There was minimal change in plasma volume throughout the postprandial period in both trials. Plasma volume was significantly decreased by the end of the exercise bout ($-3.82 \pm 2.56\%$ and $-2.48 \pm 2.96\%$ in the HGI and LGI trials respectively) ($p<0.05$) but there were no differences between the trials. At the start of each trial, all participants had a urine osmolality that suggested that they were well hydrated (747 ± 84 mosmol.kg-1 and 736 ± 88 mosmol.kg-1 in the HGI and LGI trials respectively) (NS).

Discussion

The main finding of the present study is that consumption of a LGI evening meal resulted in lower glycemic and insulinemic responses to a standard HGI breakfast compared to when a HGI evening meal was consumed. The results of the present study therefore support earlier findings that a single LGI meal can improve glucose tolerance after an overnight fast in healthy males [25]. Despite the reduced hyperglycemia and hyperinsulinemia in the postprandial period following breakfast in the LGI trial, there were no differences in substrate utilisation during the 60 min run between trials.

Jenkins et al [13] were the first to report that a single LGI CHO food (lentils) eaten at breakfast improved glucose tolerance at lunch compared to when a single HGI CHO food (wholemeal bread) was eaten. The authors reported that the slower rate of absorption of the lentils was responsible for the improved glucose tolerance at the subsequent meal. Evidence against malabsorption of the lentil meal was provided from breath hydrogen data and enterglucagon concentrations. The findings of this study were later extended by Wolever et al.[25] who reported that low glycemic index foods eaten at dinner improved the subsequent breakfast glycemic response. Both single foods and mixed meals were investigated in this study and similar results were reported. Therefore this also provides further evidence that the glycemic responses of mixed meals can be predicted from the GI of the individual foods consumed.

Although mixed meals were used in the study carried out by Wolever et al [25], they would not be realistic for an athletes diet. The meals used in the present study aimed to be a normal combination of foods that athletes may chose to eat nevertheless

differences in the glycemic response to breakfast were still observed. No blood samples were taken following the evening meal in the present study. The test meals used in the present study have however previously been used in investigations from our laboratory and result in significantly different glycemic and insulinemic responses during the postprandial period (the IAUC for plasma glucose during the postprandial period was 227.1 ± 46.2 mmol.l^{-1} x 180 min and 52.0 ± 19.6mmol.l^{-1} x 180 min in the HGI and LGI trials respectively) ($p<0.005$) (unpublished observation).

A LGI CHO-rich meal has been recommended as a suitable source of CHO before exercise [4]. The reduced hyperinsulinemia during the postprandial period following the LGI meal reduces the suppression of fat oxidation compared to when a HGI meal is consumed. This allows a shift in substrate utilisation toward fat oxidation during the subsequent exercise as well as providing a sustainable source of carbohydrate [26]. Nevertheless, in the study, despite reduced hyperglycemia and hyperinsulinemia in the postprandial period following the standard HGI breakfast in the LGI trial, no differences in substrate utilisation during the subsequent run were reported in the present study. Previous studies from our laboratory have observed insulin concentrations of approximately 90 µlU.ml^{-1} following a LGI breakfast. The insulin concentrations in the present study peaked at approximately 145 µlU.ml^{-1} following breakfast in the LGI trial. Therefore it is likely that the reduction in hyperinsulinemia observed in the present study following the LGI evening meal was insufficient to alter substrate oxidation during subsequent sub -maximal exercise.

Recently, there has been much interest in the use of LGI foods in weight management [21]. It has been hypothesised that LGI foods may be beneficial in 2 ways namely, by

promote feelings of satiety and promoting fat oxidation at the expense of carbohydrate oxidation [3]. Several studies have reported increased feelings of satiety in the hours after a LGI meal compared to an isocaloric HGI meal [15, 21, 26]. In the present study, higher ratings of gut fullness and lower hunger scores were reported on the morning of day 2 (whilst still in the fasted state) in the LGI trial compared to the HGI trial. Following the standard HGI breakfast, higher ratings of gut fullness continued to be reported in the LGI trial throughout the postprandial period. This is despite the fact that the breakfast was exactly the same in both trials and both evening meals were isocaloric and nutrient matched. The results therefore suggest that increased satiety following the LGI evening meal may have persisted even after an overnight fast. This has important implications in weight control because if food was available *ad libitum* then higher ratings of gut fullness may translate into reduced food intake.

Although no differences in substrate metabolism during exercise were reported in this study, the results from the postprandial data may have important health implications. The consumption of HGI foods is increasingly associated with increased risk of type 2 diabetes, coronary heart disease, obesity and cancer [2]. High glycemic index (HGI) meals are associated with rapid hyperglycemia and hyperinsulinemia which in many individuals, is followed by hypoglycemia and the secretion of counter regulatory hormones. The habitual intake of HGI carbohydrates is therefore associated with insulin resistance and decreased glucose tolerance. Although more longitudinal studies are required on the long-term effects of a LGI diet, the results of the present study show that improved glucose tolerance can be achieved in the short term by consuming a single LGI meal. It may be possible that only one meal a day needs to be composed of LGI carbohydrates to see an improvement in glucose tolerance. This

may increase adherence to diet programmes rather than asking individuals to consume only LGI carbohydrates in their diet.

In conclusion, the results of the present study show that ingestion of a LGI evening meal resulted in improved glucose tolerance at breakfast compared to when a HGI evening meal was consumed. Despite this, no differences in the metabolic responses to a subsequent exercise bout were observed.

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

Meal	Description	Macronutrient Content
HGI breakfast	62g Corn Flakes$^{\Psi}$ + 257ml skimmed milk 80g white bread + 10g flora +20g jam 155ml Lucozade$^{\Psi}$ original	730kcal, 139gCHO, 9.9g fat, 20g protein **GI = 79***
HGI dinner	158g white bread, 154g turkey breast, 50g cheese, 40g lettuce, 180g banana 200ml Lucozade$^{\Psi}$ original	1076kcal, 148g CHO, 24g fat, 63g protein **GI = 72***
LGI dinner	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 **GI = 34***

*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: 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).

		Postprandial Period (min)							Exercise Period (min)			
Variable	Trial	Resting	15	30	60	90	120	180	15	30	45	60
VO_2 (l/min)	HGI	0.35 ± 0.01	0.35 ± 0.01	0.35 ± 0.01	0.35 ± 0.01	0.34 ± 0.03	0.36 ± 0.01	0.34 ± 0.02	2.94 ± 0.07†	3.03 ± 0.07†	2.98 ± 0.07†	3.01 ± 0.07†
	LGI	0.32 ± 0.01	0.34 ± 0.02	0.36 ± 0.01	0.36 ± 0.01	0.35 ± 0.01	0.34 ± 0.01	0.35 ± 0.02	2.92 ± 0.08†	2.98 ± 0.09†	2.94 ± 0.1†	2.99 ± 0.08†
VCO2 (l/min)	HGI	0.30 ± 0.01	0.31 ± 0.01	0.32 ± 0.01	0.32 ± 0.01	0.31 ± 0.03	0.32 ± 0.02	0.32 ± 0.02	2.90 ± 0.09†	2.91± 0.03†	2.83 ± 0.07†	2.81 ± 0.07†
	LGI	0.29 ± 0.01	0.29 ± 0.01	0.32 ± 0.01	0.32 ± 0.01	0.32 ± 0.01	0.32 ± 0.01	0.33 ± 0.02	2.88 ± 0.08†	2.89 ± 0.08†	2.82 ± 0.10†	2.83 ± 0.08†
RER	HGI	0.85 ± 0.03	0.87 ± 0.03	0.89 ± 0.02	0.90 ± 0.02	0.92 ±0.03	0.91 ± 0.03	0.93 ± 0.02	0.99 ± 0.01†	0.97 ± 0.01†	0.95 ± 0.01†	0.93 ± 0.00†
	LGI	0.87 ± 0.04	0.84 ± 0.03	0.89 ± 0.01	0.90 ± 0.02	0.91 ± 0.02	0.92 ± 0.02	0.94 ± 0.01	0.99 ± 0.01†	0.97 ± 0.00†	0.96 ± 0.00†	0.95 ± 0.01†

† values significantly different from fasting in both the HGI and LGI trials ($p<0.01$).

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

		Postprandial Period (min)							Exercise Period (min)			
Variable	Trial	Fast	15	30	60	90	120	180	15r	30r	45r	60r
Gut Fullness	HGI	7 ± 0	12 ± 1†	12 ± 1†	11 ± 1†	10 ± 1†	10 ± 1†	9 ± 1	9 ± 1	10 ± 1	9 ± 1	8 ± 0
	LGI	8 ± 0 *	13 ± 1†	13 ± 1†	12 ± 1†	11 ± 1†	11 ± 1†	10 ± 1	9 ± 0	11 ± 1#	11 ± 2#	9 ± 1
Hunger	HGI	15 ± 1**	9 ± 0†	8 ± 0†	9 ± 1†	10 ± 1†	11 ± 1†	11 ± 1	11 ± 1	12 ± 1	12 ± 1	12 ± 1
	LGI	13 ± 1	8 ± 0†	8 ± 0†	9 ± 0†	9 ± 1†	10 ± 1†	11 ± 1	10 ± 1#	10 ± 1#	11 ± 1#	11 ± 2
Thirst	HGI	9 ± 0	9 ± 1	9 ± 0	9 ± 1	9 ± 0	10 ± 1	10 ± 0	12 ± 1	12 ± 1	12 ± 1	11 ± 1
	LGI	11 ± 1	9 ± 0	9 ± 0	10 ± 1	9 ± 1	9 ± 1	10 ± 1	12 ± 1	13 ± 1	13 ± 1	13 ± 1

Gut Fullness significantly higher throughout the postprandial period in the LGI trial compared to the HGI trial ($p<0.05$)
* values significantly higher in the LGI trial compared to the HGI trial ($p<0.01$).
** values significantly higher in the HGI trial compared to the LGI trial ($p<0.05$).
† values significantly different from fasting in both the HGI and LGI trials ($p<0.01$).
# values significantly different from fasting in the LGI 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.

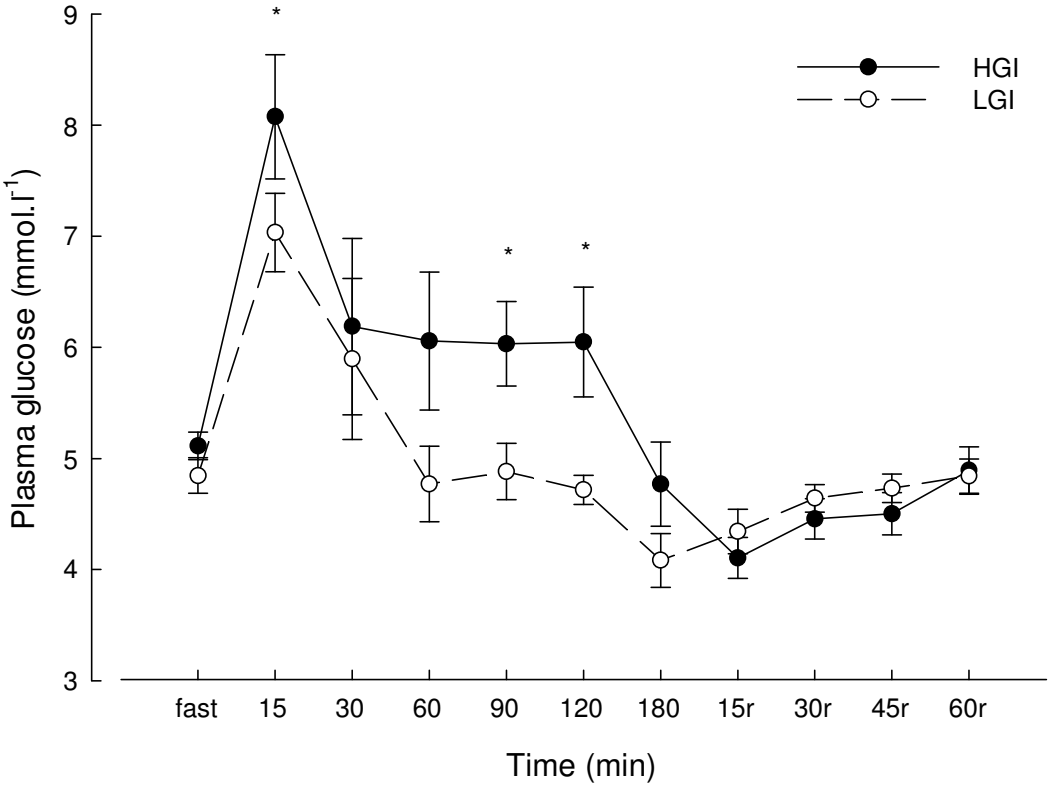
Fig 3. Plasma FFA concentrations ($mmol \cdot l^{-1}$) in the HGI and LGI trials (mean ± SEM).

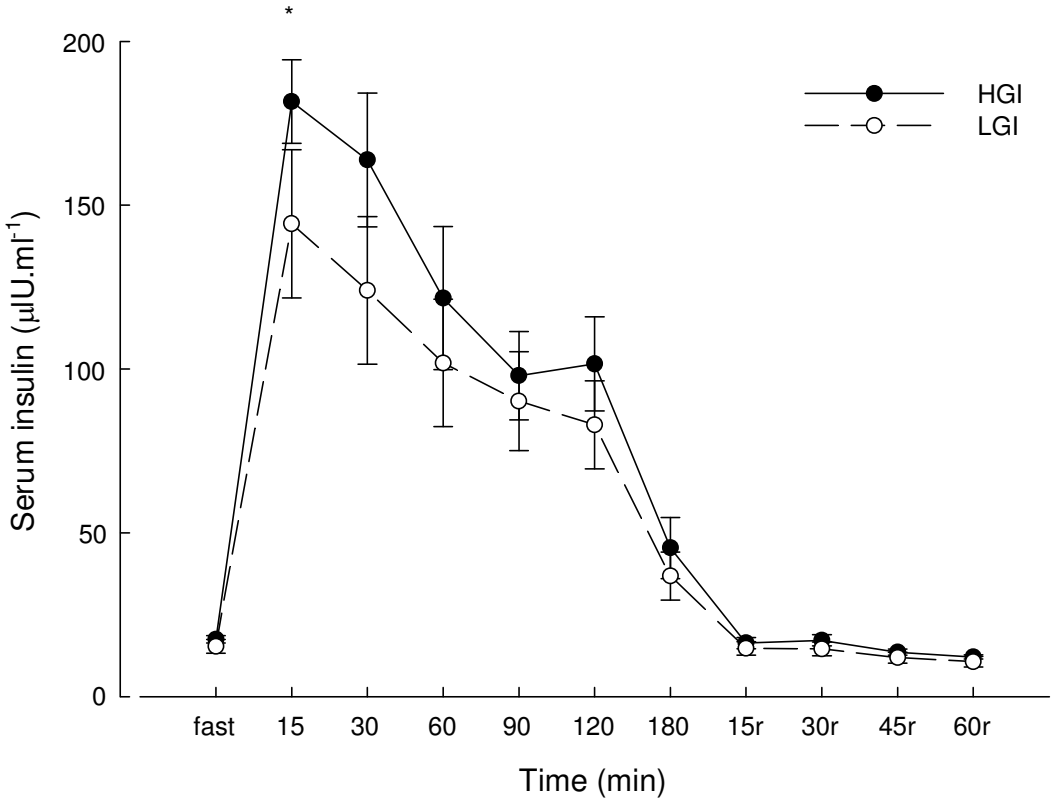
Fig 4. Plasma glycerol concentrations ($\mu mol \cdot l^{-1}$) in the HGI and LGI trials (mean ± SEM).

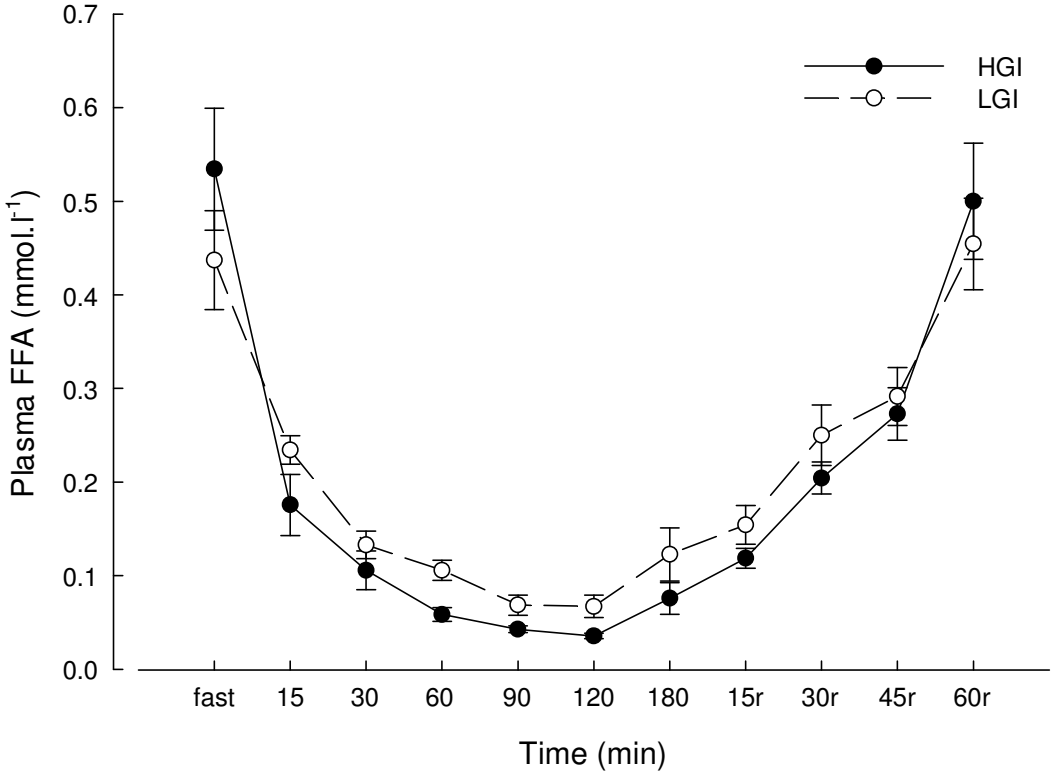
Fig 5. Blood lactate concentrations ($mmol.l^{-1}$) in the HGI and LGI trials (mean ± SEM).

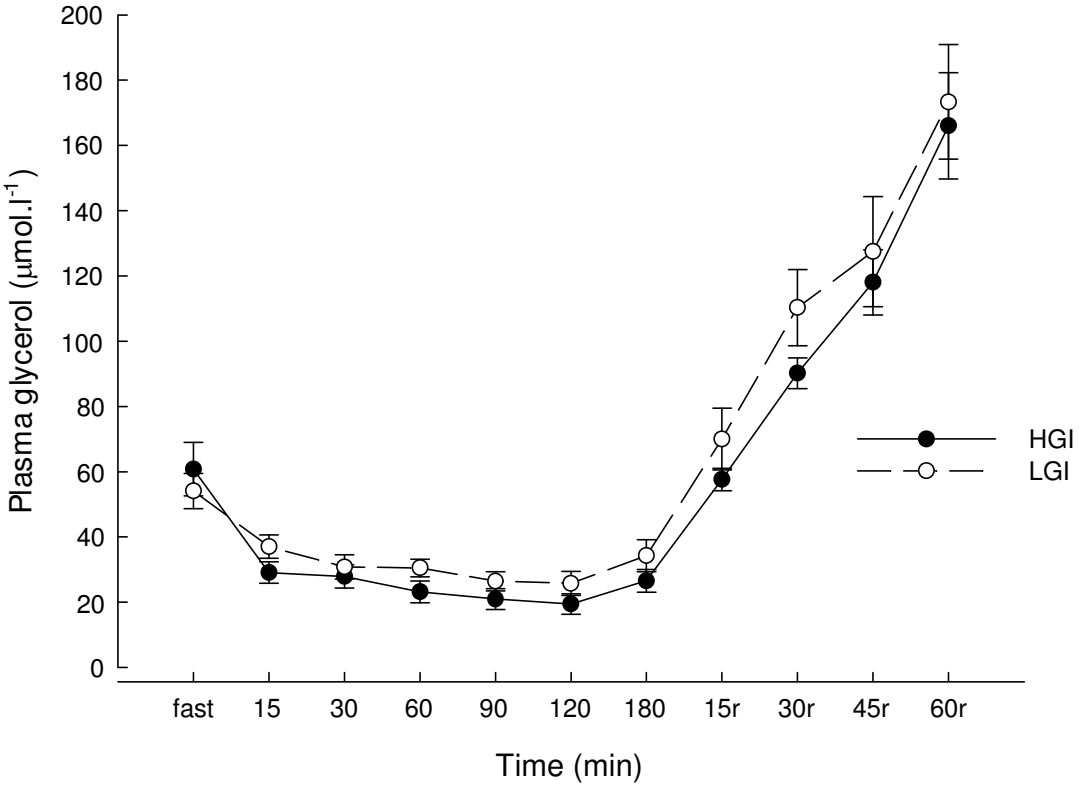
Fig 6..Estimated rate of carbohydrate oxidation ($g.min^{-1}$) in the HGI and LGI trials (mean ± SEM).

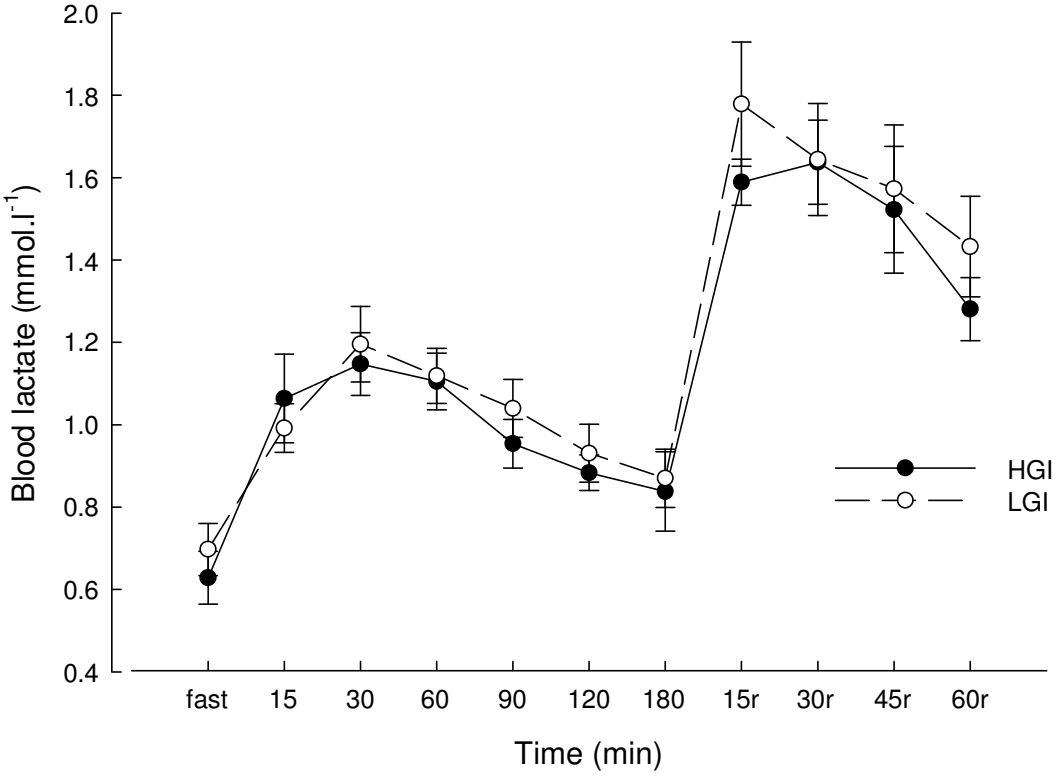
Fig 7. Estimated rate of fat oxidation ($g.min^{-1}$) in the HGI and LGI trials (mean ± SEM).

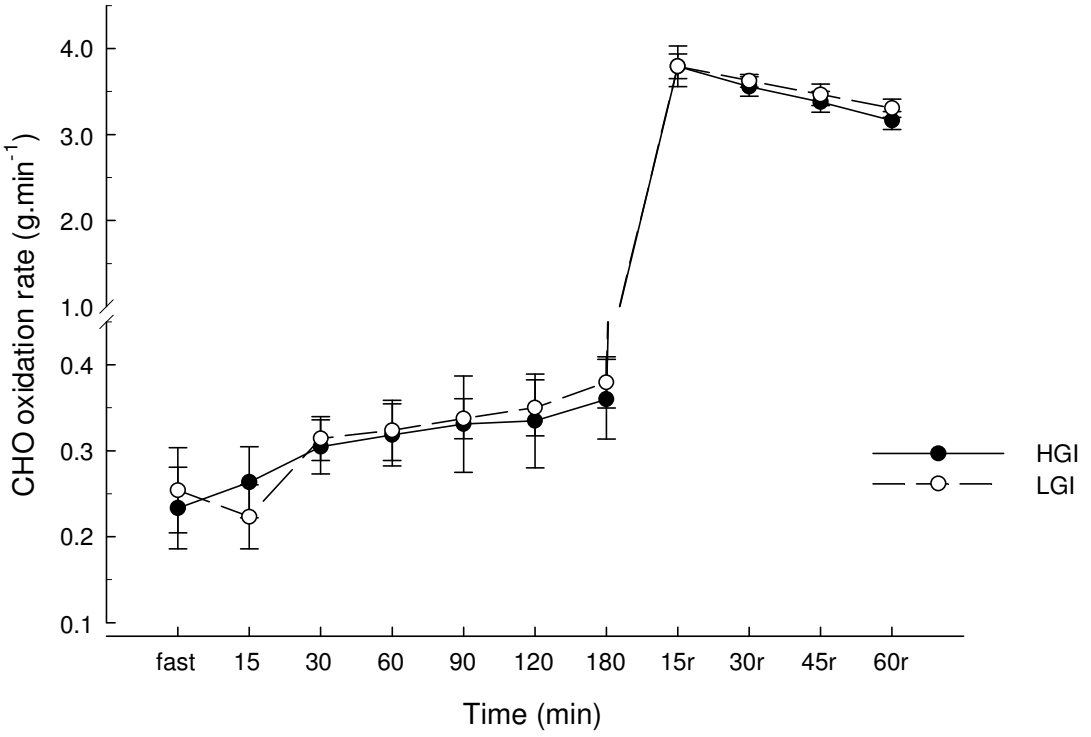

9
8
7
6
5
4
3
Plasma glucose (mmol.l-1)
*
*
*
HGI
LGI
fast
15
30
60
90
120
180
15r
30r
45r
60r
Time (min)

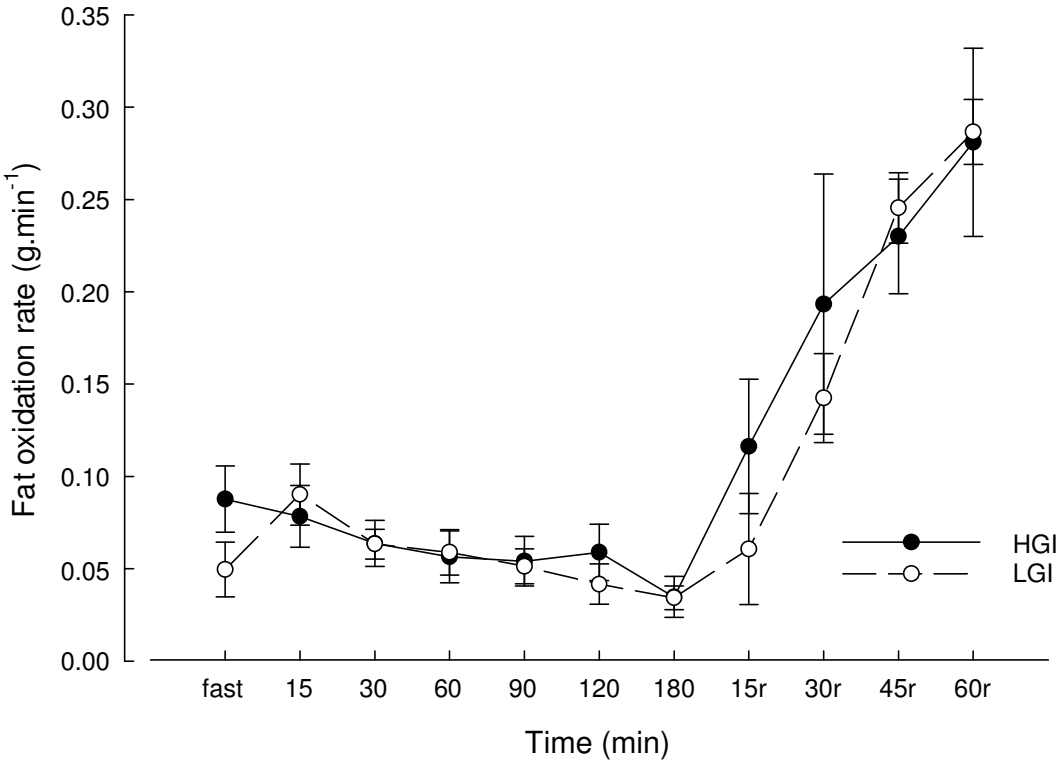

*
200
150
100
50
0
Serum insulin (µIU.ml-1)
HGI
LGI
fast
15
30
60
90
120
180
15r
30r
45r
60r
Time (min)


0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Plasma FFA (mmol.l-1)
HGI
LGI
fast
15
30
60
90
120
180
15r
30r
45r
60r
Time (min)


Plasma glycerol (µmol.l-1)
200
180
160
140
120
100
80
60
40
20
0
fast
15
30
60
90
120
180
15r
30r
45r
60r
Time (min)
HGI
LGI


2.0
1.8
1.6
1.4
1.2
1.0
0.8
0.6
0.4
Blood lactate (mmol.l-1)
fast
15
30
60
90
120
180
15r
30r
45r
60r
Time (min)
HGI
LGI


CHO oxidation rate (g.min-1)
4.0
3.0
2.0
1.0
0.4
0.3
0.2
0.1
fast
15
30
60
90
120
180
15r
30r
45r
60r
Time (min)
HGI
LGI


0.35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
Fat oxidation rate (g.min-1)
fast
15
30
60
90
120
180
15r
30r
45r
60r
Time (min)
HGI
LGI