European Journal of Clinical Nutrition (2007) 61, 590–596
 0954-3007/07 $30.00
www.nature.com/ejcn

ORIGINAL ARTICLE

Effects of the glycemic index of breakfast on metabolic responses to brisk walking in females

SH Backhouse[1], C Williams[2], E Stevenson[3] and M Nute[2]

[1]*Carnegie Research Institute, Leeds Metropolitan University, Leeds, UK;* [2]*School of Sport and Exercise Sciences, Loughborough University, Loughborough, UK and* [3]*School of Psychology and Sports Sciences, Northumbria University, Newcastle Upon Tyne, UK*

Objective: The aim of this study was to examine the metabolic responses during 1 h of brisk walking, 3 h after ingesting high glycemic index (HGI) and moderate glycemic index (MGI) breakfasts.
Design and subjects: Six females completed three treadmill walking trials ($\sim$50% $\dot{V}O_2$ max), separated by at least 1 month. Three hours before walking, they ingested either water or a HGI or MGI breakfast. The MGI breakfast consisted of a mixture of an HGI breakfast cereal and low GI carbohydrate (CHO) foods. The GI values of the HGI and MGI meals were 77 and 51, respectively.
Setting: The study took place in the School of Sport and Exercise Sciences at Loughborough University, Loughborough, UK.
Results: In the HGI and MGI trials, plasma glucose and serum insulin concentrations peaked 15 min into the postprandial period. At the onset of exercise, plasma insulin concentrations were twofold higher in the HGI ($31.5 \pm 7.7\ \mu l\,U\,l^{-1}$) than in the MGI trial ($15.2 \pm 1.9\ \mu l\,U\,l^{-1}$) ($P<0.05$). However, there were no differences in substrate utilization between the two CHO trials.
Conclusion: These results suggest that although the addition of LGI CHO foods to an HGI breakfast cereal reduces the overall GI of the meal, the metabolic response to exercise is similar to that following a breakfast comprised entirely of HGI foods.
European Journal of Clinical Nutrition (2007) **61,** 590–596. doi:10.1038/sj.ejcn.1602566; published online 29 November 2006

Keywords: pre-exercise meal; CHO; glycemic index; walking; women

Introduction

Recently, there has been a great deal of interest in the potential of low glycemic index (LGI) carbohydrate (CHO) diets to aid weight loss (Diaz *et al*., 2005). The ingestion of a LGI CHO meal results in lower plasma glucose and insulin concentrations than occur after eating high glycemic index (HGI) meals. Furthermore, the rapid decline in blood glucose concentration during the initial stages of exercise is less after a LGI pre-exercise meal than after an HGI CHO meal (Wee *et al*., 1999). In addition, several studies have reported an increased rate of fat oxidation during exercise when participants have consumed LGI CHO in the pre-exercise period in comparison to HGI CHO (Sparks *et al*., 1998; Wu *et al*., 2003; Wee *et al*., 2005). This greater rate of fat oxidation is of relevance to individuals who are combining a diet and exercise regime, in an attempt to lose fat mass (Ludwig *et al*., 1999).

The acute metabolic responses to consuming single foods containing LGI (GI = $\sim$23–45) or HGI (GI = $\sim$68–100) CHO before exercise are well known (Thomas *et al*., 1991; Febbraio and Stewart, 1996; Febbraio *et al*., 2000). There is also an increase in information available on the acute metabolic responses to LGI and HGI pre-exercise mixed meals (Wu *et al*., 2003; Stevenson *et al*., 2005; Wee *et al*., 2005). The glycemic index of a mixed meal can be calculated by summing the weighted means of the GI values of the component foods (Wolever and Jenkins, 1986). This suggests that the GI of an HGI CHO food might be reduced by adding a LGI CHO resulting in a mixed meal of medium GI (MGI). This may occur frequently in reality when individuals are attempting to apply the GI concept to their daily diet. However, there is little information on the metabolic

Correspondence: Dr SH Backhouse, Carnegie Research Institute, Leeds Metropolitan University, Fairfax Hall, Headingley Campus, Leeds LS6 3QS, UK. E-mail: S.Backhouse@leedsmet.ac.uk
Contributors: SHB was involved in all parts of the study from conception to preparation of the final manuscript. CW was involved in the project design, discussion and interpretation of results and manuscript preparation. ES was involved in data analysis, discussion and interpretation of results and manuscript preparation. MN was involved in the experimental aspects of the study and sample collection, analysis and interpretation.
Received 28 April 2006; revised 12 October 2006; accepted 17 October 2006; published online 29 November 2006

consequences of eating a MGI mixed meal before low intensity exercise, i.e. walking. Furthermore, the majority of studies have used male participants even though women are more likely to consider including a low GI diet in a weight loss program.

Therefore, the purpose of the present study was to examine the influence of ingesting MGI and HGI CHO breakfasts, using foods that the general public regularly consume, on the metabolic responses to walking exercise, performed 3 h later in females.

Methods

Participants
Six healthy females volunteered to participate in this study. Their mean ($\pm$s.e.m.) age, height, weight and $\dot{V}O_2$ max were 21.0 ± 0.3 years, 170.0 ± 0.0 cm, 66.0 ± 2.7 kg and 42.5 ± 2.1 ml kg^{-1} min^{-1}, respectively. All participants were eumenorrheic and completed each trial in the follicular phase of the menstrual cycle. The protocol had the approval of the University Ethical Advisory Committee and all participants gave their written informed consent. All participants were non-smokers, without a history of cardiovascular or metabolic disease and were not taking the oral contraceptive pill.

Preliminary measurements
Participants completed two preliminary exercise tests before the first main trial. The first test consisted of 16 min of continuous walking on a treadmill (Technogym, Italy) in order to establish the steady-state relationship between submaximal oxygen uptake and treadmill walking speed and gradient. The walking speed remained constant throughout the test (range 4.8–5.5 km h^{-1}); however, the gradient was increased at the end of each 4 min stage by 3%, from an initial 4%. During the final minute of each 4 min stage, expired air samples were collected and rate of perceived exertion (RPE) and heart rate were recorded. The peak oxygen uptake ($\dot{V}O_2$ peak) of each participant was determined during a second uphill walking test. The speed was constant throughout the test (range 5.5–6.8 km h^{-1}) and was dependent upon the participant's performance during the first preliminary test. The treadmill gradients at each stage were 5, 10, 12.5, 15 and 17.5%, respectively. The participants had to progress as far as possible through each of the 3 min stages. Expired air samples, RPE and heart rate were collected during the 1:45–2:45 min of each stage. The final expired air sample was taken during the last minute of the test immediately after the participant signalled they could continue walking for only a further minute. This final $\dot{V}O_2$ was considered to be the $\dot{V}O_2$ peak of the participant. The results obtained from the two preliminary tests were used to predict the walking speeds required to elicit 50% of each participant's $\dot{V}O_2$ peak.

Familiarization
Before the three main trials, participants undertook a 60-min walk at a speed predicted to be equivalent to 50% $\dot{V}O_2$ max. This allowed confirmation that the individual relative exercise intensity (%$\dot{V}O_2$ max) was correct and allowed participants to become familiar with the experimental protocol.

Nutritional status
Each participant completed a 3-day weighed food intake diary before their first main trial, which was then analysed to provide a quantitative description of the composition of each participant's habitual diet (COMP-EAT). This nutritional information was used to prescribe a diet for the 2 days before each trial. All participants refrained from physical training, caffeine ingestion and alcohol intake for 48 h before each trial.

Menstrual cycle status
All main experimental trials were carried out during days 6–9 of the follicular phase of the menstrual cycle. None of the participants were taking the oral contraceptive pill.

Experimental protocol
Participants reported to the laboratory between 0800 and 0900 h following a 12-h overnight fast. An indwelling cannula (Venflon, 16 G) was inserted into an ante-cubital vein under local anesthesia (0.5 ml of 1% lignocaine) and was kept patent by frequent flushing with sterile saline immediately after blood sampling. Fasting blood (10 ml) and expired air samples were then collected before the test meal was consumed. The participants were allocated to one of the two CHO breakfasts or no-breakfast in a randomized crossover before the start of the three trials. They received, water (FAST), a moderate GI (MGI) or a high GI (HGI) breakfast (Table 1). The food and drinks were consumed within 15 min. During the 3-h postprandial period, the participants relaxed in the laboratory, and expired air samples and venous blood samples were collected at regular intervals. The final resting expired air and venous blood sample were collected while the participants stood on the treadmill. To standardize plasma volume changes, the participants stood for 15 min before the collection of and during all resting venous blood samples. The participants then walked for 60 min on a treadmill (Technogym, Italy) at a 5% gradient and at a speed that elicited 50% of their respective $\dot{V}O_2$ peak. One minute expired air samples were collected every 15 min followed by venous blood samples. During the expired air collections, RPE (Borg, 1982) and heart rate (Technogym, Italy) were recorded.

Sample collection and analysis

Samples of expired gas were collected using the Douglas bag method at the following times: pre-meal, during the postprandial period (15, 30, 60, 90, 120, 150 and 180 min) and every 15 min during exercise. Each expired gas sample was collected through a one-way low-resistance valve and a lightweight, wide-bore tubing (Falconia Baxter Woodhouse & Taylor Ltd, Macclesfield, Cheshire, UK) into a Douglas bag for 5 min during the resting collections and for 1 min during exercise. O_2 and CO_2 content were analysed using methods previously described (Williams *et al*., 1990). The energy expenditure, and total CHO and fat oxidation rates, were estimated from VO_2 and VCO_2 using stoichiometric equations (Frayn, 1983). Total CHO and fat oxidation was estimated from the area under the rate of oxidation vs time curve for each participant.

Venous blood samples were collected into lithium heparin and serum tubes after each expired air sample. Plasma glucose and lactate were determined as previously described (Williams *et al*., 1990). Plasma free fatty acids (FFA) (Wako Chemicals GmbH kit, Wako, Neuss, Germany) and serum insulin (Coat-A-Count Insulin, DPC kit, Diagnostic Products Corporation, Llanberis, UK) concentrations were determined using commercially available kits.

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 utilized to determine the location of the variance. Differences were considered significant at $P<0.05$. All results are presented as mean $\pm$ s.e.m.

Table 1 Characteristics of the test meals (for a 60 kg person)

Meal	*Description*	*Macronutrient content*
FAST	Tap water	820 ml water
MGI breakfast	80 g bran flakes[a] (74), 200 ml skimmed milk (32), 280 g canned peaches in own juice (38), 160 g apples (38), 400 ml unsweetened apple juice (40), 400 ml water	668 kcal 152 g CHO 3 g fat 18 g protein 914 g water 16 g fibre 114 g sugars, total GI = 51[b]
HGI breakfast	60 g corn flakes[a] (81), 240 ml skimmed milk (32), 75 g white bread (70), 20 g jam (80), 175 ml Lucozade Original drink[a] (95), 400 ml water	641 kcal 123 g CHO 2 g fat 16 g protein 796 g water 2 g fiber 57 g sugars, total GI = 77[b]

[a]Corn flakes and bran flakes: Kellogg's (UK) Ltd. Manchester, UK; Lucozade Original drink UK.
GI values of individual foods are given in parenthesis.
[b]Calculated by the method described in Wolever and Jenkins (1986) with GI values taken from Foster-Powell *et al*. (2002).

Results

Plasma glucose and serum insulin

Plasma glucose concentrations remained stable throughout the FAST trial (Figure 1), but rose rapidly after ingestion of the test meals, reaching a peak at 15 min in the HGI and MGI trials (6.6 ± 0.6 and 6.2 ± 0.2 mmol l^{-1}, respectively). Plasma glucose concentrations in the fed trials then declined during the remainder of the postprandial period. The incremental area under the curve (IAUC) during the postprandial period was not different between the HGI and MGI trial, but greater in the fed trials compared to the FAST trial ($P<0.05$) (HGI: 85 ± 30 vs MGI: 74 ± 26 vs FAST 8 ± 4 mmol.$l^{-1} \times 180$ min) during the postprandial period.

At 15 min during exercise, plasma glucose concentrations decreased markedly in the HGI trial, reaching a nadir (4.0 ± 0.2 mmol/l^{-1}) that was significantly lower (condition $\times$ time effect; $P<0.05$) than in the MGI and FAST trials. This difference persisted for 30 min into exercise ($P<0.05$). Thereafter, plasma glucose concentrations returned to pre-exercise values and at the end of exercise values were similar in all three trials (HGI: 4.7 ± 0.1 mmol l^{-1} vs MGI: 4.8 ± 0.1 mmol l^{-1} vs FAST: 4.9 ± 0.2 mmol l^{-1}).

Serum insulin concentrations (Figure 2) remained stable throughout the FAST trial. However, following the HGI and MGI breakfasts, serum insulin concentrations increased rapidly and peaked at 15 min during the postprandial period

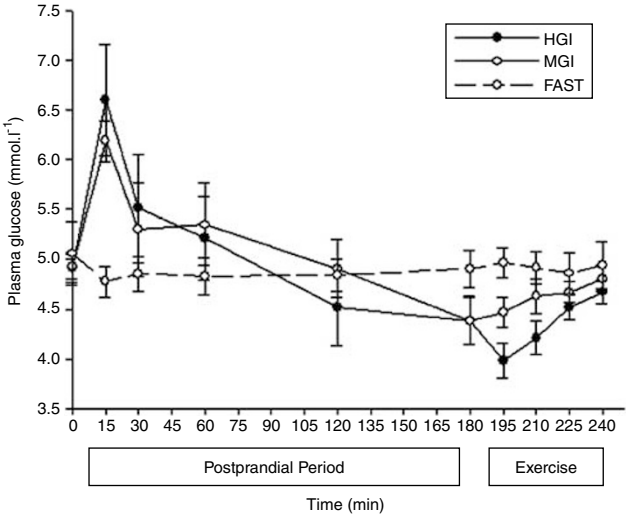

Figure 1 Plasma glucose concentration (mmol l^{-1}) during 3 h postprandial period and 1 h exercise period for the HGI, MGI and FAST trials (mean $\pm$ s.e.m.).

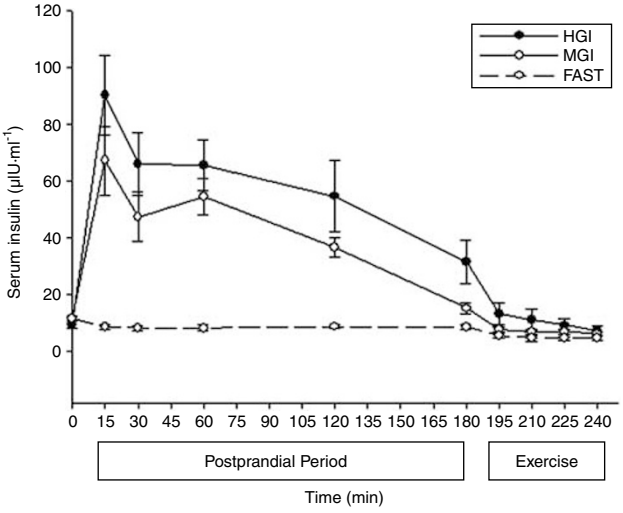

Figure 2 Serum insulin concentration (μlU l^{-1}) during 3 h postprandial period and 1 h exercise period, for the HGI, MGI and FAST trials (mean ± s.e.m.).

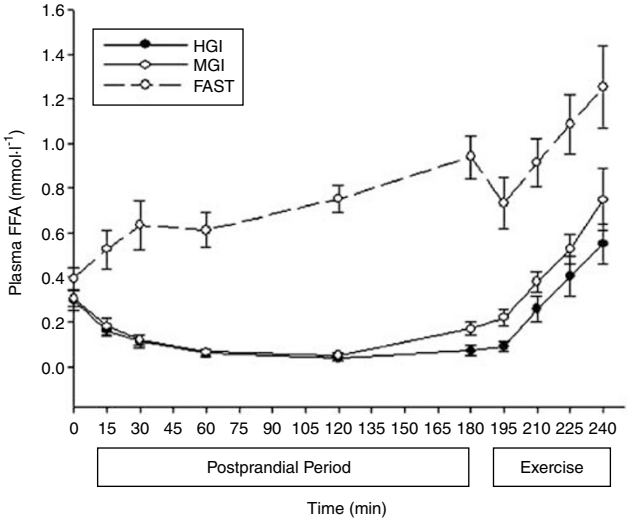

Figure 3 Plasma FFA concentration (mmol l^{-1}) during 3 h postprandial period and 1 h exercise period, for the HGI, MGI and FAST trials (mean ± s.e.m.).

(HGI: 90.1 ± 14.0 μl U l^{-1} vs MGI: 67.1 ± 12.2 μl U l^{-1}). This resulted in a significant ($P<0.01$) increase from the pre-meal baseline values of 9.4 ± 1.4 μlU l^{-1} and 11.7 ± 0.8 μl U l^{-1} for the HGI and MGI trials, respectively. Serum insulin concentrations then declined throughout the remainder of the postprandial period. The incremental area under the serum insulin response curve (IAUC) during the postprandial period was not different between the HGI and MGI trial, but greater in the fed trials compared to the FAST trial ($P<0.05$) (HGI: 8369 ± 1595 vs MGI: 5142 ± 707 vs FAST: 42 ± 42 μl U ml^{-1} × 180 min). Immediately before exercise, serum insulin concentrations were twofold higher in the HGI trial (31.5 ± 7.7 μl U l^{-1}) compared to the MGI trial (15.2 ± 1.9 μl U l^{-1}) ($P<0.05$). Thereafter, values were similar in each of the three trials (Figure 2).

Plasma free fatty acids

Following ingestion of the breakfasts, plasma FFA concentrations were reduced to below fasting values ($P<0.01$) in both fed trials and remained lower throughout the postprandial period (Figure 3). During exercise, FFA concentrations were higher in the FAST ($P<0.01$), compared to the HGI and MGI trials. In the HGI and MGI trials, FFA concentrations increased with time, but there was no difference between trials.

Plasma lactate

Following the ingestion of HGI and MGI breakfasts, plasma lactate concentrations increased significantly ($P<0.05$), peaking at 30 and 60 min during the postprandial period in the HGI and MGI trials, respectively. Plasma lactate concentrations were higher during the first 2 h of the postprandial period in the MGI (2.36 ± 0.22 mmol l^{-1}) than in the HGI (1.57 ± 0.12 mmol l^{-1}) and FAST (1.00 ± 0.12 mmol l^{-1}; $P<0.05$) trials. However, at the start of exercise, plasma lactate concentrations had returned to resting concentrations in the HGI and MGI trials. No differences were observed between trials throughout the 1 h walk.

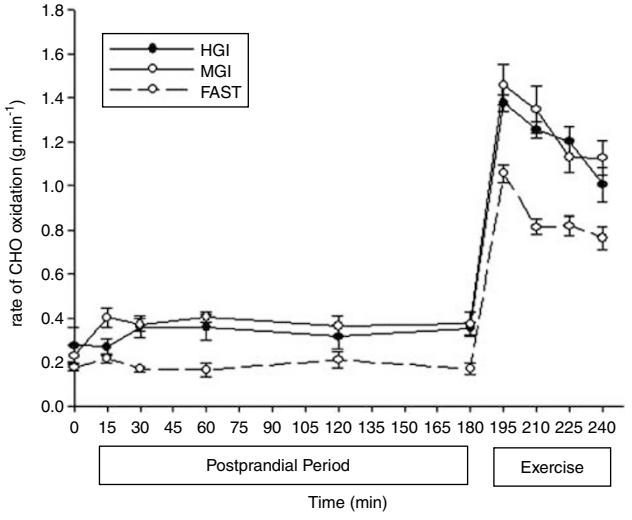

Figure 4 Carbohydrate oxidation (g min^{-1}) during 3 h postprandial period and 1 h exercise period, for the HGI, MGI and FAST trials (mean ± s.e.m.).

Estimated carbohydrate and fat oxidation rates

During the 1 h of brisk walking, the total carbohydrate oxidation (HGI: 1.2 ± 0.1. vs MGI: 1.3 ± 0.1 vs FAST: 0.9 ± 0.1 g min^{-1}) was higher (Figure 4) and total fat oxidation

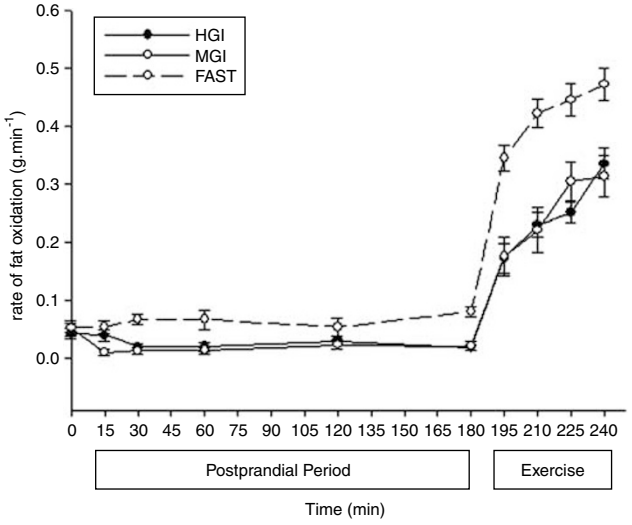

Figure 5 Fat oxidation ($g\,min^{-1}$) during 3 h postprandial period and 1 h exercise period, for the HGI, MGI and FAST trials (mean ± s.e.m.).

(HGI; 0.3 ± 0.01 vs MGI: 0.3 ± 0.1 vs FAST: 0.4 ± 0.1 $g\,min^{-1}$) was lower (Figure 5), in both the fed trials, when compared to the FAST trial ($P<0.05$). No differences were observed between the HGI and MGI trials.

Discussion

The main aim of the present study was to investigate the metabolic responses to a HGI breakfast cereal, when HGI and LGI foods were added to produce ecologically valid mixed meals. The main finding of the present study was that although the GI of the breakfast was lowered, this was not accompanied by significant differences in metabolic response. Indeed, the only notable difference was a trend for lower serum insulin concentrations during the MGI trial compared with the HGI trial that became significant at the end of the 3 h postprandial period.

The GI classification was developed by Jenkins *et al.* (1981), as a method of ranking CHO-containing foods based on their actual postprandial blood glucose response, compared to a reference food of white bread or glucose. However, it is not common practice to consume single foods alone and different CHO-containing foods are usually combined within a meal. Therefore, Wolever and Jenkins (1986) developed a method of calculating the glycemic response to a mixed meal composed of different carbohydrate-containing foods. The validity of this method has been questioned in the past (Coulston *et al.*, 1984; Hollenbeck *et al.*, 1988); however, there are several recent studies that have successfully applied the GI concept to mixed meals (Wu *et al.*, 2003; Stevenson *et al.*, 2005; Wee *et al.*, 2005)

Differences in postprandial glycemic and insulinemic responses to pre-exercise foods and/or meals with different GI values (LGI ~ 30 and HGI ~ 80) are consistently observed within 30 min of their ingestion (Sparks *et al.*, 1998; Wu *et al.*, 2003; Stevenson *et al.*, 2005). In the present study, the difference in the calculated GI of the HGI and MGI meal was 26, and therefore a reduced postprandial glucose and insulin response was expected following ingestion of the MGI meal. In contrast, the MGI breakfast gave rise to a response that was in fact remarkably similar to that observed in the HGI trial (Figure 1).

It was interesting to note that during the first 15 min of exercise, a sharp decline in plasma glucose concentrations was observed in the HGI trial (Figure 1). Indeed, plasma glucose concentration decreased to values that were close to being classified as hypoglycemic (plasma glucose $<3.5\,mmol\,l^{-1}$ (Costill *et al.*, 1977). The rapid decline in plasma glucose concentrations during the first 30 min of exercise in the HGI trial may be attributed to an increased insulin-mediated disposal of glucose (Costill *et al.*, 1977), owing to the significantly higher insulin concentration observed at the start of exercise. Wu *et al.* (2003) observed a similar sharp decline in plasma glucose concentrations in males, when they were provided with a similar HGI breakfast to that used in the present study. This is a relevant and potentially important finding because hypoglycemia is an episode characterized with symptoms such as hunger, nervousness and fatigue (Messer *et al.*, 1990). Such symptoms arise because of the counter regulatory hormones (e.g. adrenaline, cortisol) produced to prevent the decline in glucose concentrations (Messer *et al.*, 1990). In clinical practice, patients frequently report feeling miserable or tearful during or after an episode of acute hypoglycemia (Gold *et al.*, 1995).

Several studies investigating the effect of the GI of pre-exercise meals have reported a higher rate of fat oxidation and consequently a lower CHO oxidation rate during exercise following a LGI meal in comparison to an isocaloric, nutrient-matched HGI meal (DeMarco *et al.*, 1999; Wu *et al.*, 2003; Wee *et al.*, 2005). In a recent study by Wee *et al.* (2005), it was reported that the lower rate of CHO oxidation following a LGI breakfast could simply be explained by a lower rate of muscle glycogen utilization. A 15% increase in muscle glycogen concentration was reported at the end of a 3-h postprandial period following the HGI breakfast; however, only a small nonsignificant increase in muscle glycogen was reported following the LGI breakfast (Wee *et al.*, 2005). This was accounted for by the low glycemic and insulinemic responses to the LGI meal (secondary to slow digestion and absorption of the ingested foods). Although the LGI meal contributed less CHO to muscle glycogen synthesis in the postprandial period, a sparing of muscle glycogen during subsequent exercise was observed in the LGI trial, most likely as a result of better maintained fat oxidation.

In the present study, there were no differences in substrate oxidation during rest or exercise between the HGI and MGI

trials. The similar plasma glucose and insulin responses to the test meals probably resulted in similar muscle glycogen synthesis during the postprandial period and therefore participants commenced exercise in a similar metabolic state. Therefore, it appears that a pre-exercise meal must consist of solely LGI foods in order to increase exercise fat oxidation above that observed following a HGI pre-exercise meal.

It is important to note that although the two breakfasts used in the present study were matched for total carbohydrate, protein, fat and energy content, there were differences in the fiber and the total sugar content of the breakfasts (Table 1). It is therefore possible that any effect that may have resulted from differences in the GI of the two meals was masked by the large differences in fiber and sugar content of the meals. A previous study carried out by Paul *et al*. (1996) investigated the metabolic and physical performance responses to ingestion of pre-exercise meals with different macronutrient and fiber profiles. During exercise, no differences in substrate oxidation or in cycling performance times were reported between the trials (Paul *et al*., 1996). Previous studies that have observed a higher rate of fat oxidation during exercise following a LGI meal have reported much smaller differences in the fiber content of the pre-exercise meals (Stevenson *et al*., 2006) and have also had much larger differences in the GI of the meal. It is therefore evident that changing the fiber or sugar content of a pre-exercise meal is not sufficient to alter substrate oxidation during exercise but significantly lowering the GI of the carbohydrates provided in the meal is.

Interestingly, blood lactate concentrations were significantly elevated following the ingestion of the MGI breakfast, whereas the increase was minimal following the HGI breakfast. Several studies have reported that CHO that have high fructose concentrations result in higher blood lactate concentrations (Koivisto *et al*., 1981; Moore *et al*., 2000). Once fructose enters the cell, it is rapidly converted to fructose-1-phospate (F1P). The high concentrations of F1P inhibit the degradation of glycogen and facilitate the production of lactate (Henry *et al*., 1991). About two-thirds of fructose is converted to glucose and the rest of the metabolized fructose is released from the liver as lactate (Henry *et al*., 1991). In the present study, the MGI breakfast contained more fructose than the HGI breakfast owing to the fruit content of the MGI breakfast. This may therefore explain the higher blood lactate concentrations in the MGI trial during the postprandial period. Similar blood lactate results were also reported in recent studies from our laboratory who used similar breakfasts containing foods similar to those used in the present study (Wu *et al*., 2003; Stevenson *et al*., 2005).

In conclusion, the consumption of a mixed MGI breakfast elicited a metabolic response that was similar to that of a HGI breakfast. As the two breakfasts differed in fiber and sugar content, it can be concluded this is not an effective way to alter substrate oxidation during exercise. It is evident from this study and from previous research in this area that a large difference in the GI of a pre-exercise meal is necessary to achieve any differences in fuel oxidation during exercise. However, blood glucose concentrations were better maintained during exercise in the MGI trial. Further research is therefore warranted, because this might have an influence on the ability to continue exercise because participants may have felt better during the MGI trial.

Acknowledgements

The authors would like to thank Michelle Allmark and Claire Bennett for their help in the data collection process.

References

Borg GA (1982). Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* **14**, 377–381.
Costill DL, Coyle E, Dalsky G, Evans W, Fink W, Hoopes D (1977). Effects of elevated plasma FFA and insulin on muscle glycogen usage during exercise. *J Appl Physiol* **43**, 695–699.
Coulston AM, Hollenbeck CB, Liu GC, Williams RA, Starich GH, Mazzaferri EL *et al*. (1984). Effect of source of dietary carbohydrate on plasma glucose, insulin, and gastric inhibitory polypeptide responses to test meals in subjects with noninsulin-dependent diabetes mellitus. *Am J Clin Nutr* **40**, 965–970.
DeMarco HM, Sucher KP, Cisar CJ, Butterfield GE (1999). Pre-exercise carbohydrate meals: application of glycemic index. *Med Sci Sports Exerc* **31**, 164–170.
Diaz EO, Galgani JE, Aguirre CA, Atwater IJ, Burrows R (2005). Effect of glycemic index on whole-body substrate oxidation in obese women. *Int J Obes (London)* **29**, 108–114.
Febbraio MA, Keenan J, Angus DJ, Campbell SE, Garnham AP (2000). Preexercise carbohydrate ingestion, glucose kinetics, and muscle glycogen use: effect of the glycemic index. *J Appl Physiol* **89**, 1845–1851.
Febbraio MA, Stewart KL (1996). CHO feeding before prolonged exercise: effect of glycemic index on muscle glycogenolysis and exercise performance. *J Appl Physiol* **81**, 1115–1120.
Foster-Powell K, Holt SH, Brand-Miller JC (2002). International table of glycemic index and glycemic load values: 2002. *Am J Clin Nutr* **76**, 5–56.
Frayn KN (1983). Calculation of substrate oxidation rates *in vivo* from gaseous exchange. *J Appl Physiol* **55**, 628–634.
Gold AE, MacLeod KM, Frier BM, Deary IJ (1995). Changes in mood during acute hypoglycemia in healthy participants. *J Personality a Social Psychol* **68**, 498–504.
Henry RR, Crapo PA, Thorburn AW (1991). Current issues in fructose metabolism. *Annu Rev Nutr* **11**, 21–39.
Hollenbeck CB, Coulston AM, Reaven GM (1988). Comparison of plasma glucose and insulin responses to mixed meals of high-, intermediate-, and low-glycemic potential. *Diabetes Care* **11**, 323–329.
Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM *et al*. (1981). Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* **34**, 362–366.
Koivisto VA, Karonen SL, Nikkila EA (1981). Carbohydrate ingestion before exercise: comparison of glucose, fructose, and sweet placebo. *J Appl Physiol* **51**, 783–787.
Ludwig DS, Majzoub JA, Al-Zahrani A, Dallal GE, Blanco I, Roberts SB (1999). High glycemic index foods, overeating, and obesity. *Pediatrics* **103**, E26.
Messer SC, Morris RL, Gross AM (1990). Hypoglycemia and psychopathology: a short term memory, concentration, mood and blood glucose. *Clin Psychol Review* **10**, 631.

Moore MC, Cherrington AD, Mann SL, Davis SN (2000). Acute fructose administration decreases the glycemic response to an oral glucose tolerance test in normal adults. *J Clin Endocrinol Metab* **85**, 4515–4519.

Paul GL, Rokusek JT, Dykstra GL, Boileau RA, Layman DK (1996). Preexercise meal composition alters plasma large neutral amino acid responses during exercise and recovery. *Am J Clin Nutr* **64**, 778–786.

Sparks MJ, Selig SS, Febbraio MA (1998). Pre-exercise carbohydrate ingestion: effect of the glycemic index on endurance exercise performance. *Med Sci Sports Exerc* **30**, 844–849.

Stevenson E, Williams C, Mash L, Phillips B, Nute M (2006). Influence of high-carbohydrate mixed meals with different glycemic indexes on substrate utilization during subsequent exercise in women. *Am J Clin Nutr* **84**, 354–360.

Stevenson E, Williams C, Nute M (2005). The influence of the glycaemic index of breakfast and lunch on substrate utilisation during the postprandial periods and subsequent exercise. *Br J Nutr* **93**, 885–893.

Thomas DE, Brotherhood JR, Brand JC (1991). Carbohydrate feeding before exercise: effect of glycemic index. *Int J Sports Med* **12**, 180–186.

Wee SL, Williams C, Gray S, Horabin J (1999). Influence of high and low glycemic index meals on endurance running capacity. *Med Sci Sports Exerc* **31**, 393–399.

Wee SL, Williams C, Tsintzas K, Boobis L (2005). Ingestion of a high-glycemic index meal increases muscle glycogen storage at rest but augments its utilization during subsequent exercise. *J Appl Physiol* **99**, 707–714.

Williams C, Nute MG, Broadbank L, Vinall S (1990). Influence of fluid intake on endurance running performance. A comparison between water, glucose and fructose solutions. *Eur J Appl Physiol Occup Physiol* **60**, 112–119.

Wolever TM, Jenkins DJ (1986). The use of the glycemic index in predicting the blood glucose response to mixed meals. *Am J Clin Nutr* **43**, 167–172.

Wu CL, Nicholas C, Williams C, Took A, Hardy L (2003). The influence of high-carbohydrate meals with different glycaemic indices on substrate utilisation during subsequent exercise. *Br J Nutr* **90**, 1049–1056.