Journal of Sports Sciences, 2004, **22**, 1065–1071

Taylor & Francis healthsciences

Skeletal muscle glycogen concentration and metabolic responses following a high glycaemic carbohydrate breakfast

COSTAS CHRYSSANTHOPOULOS*, CLYDE WILLIAMS, ANDREA NOWITZ and GREGORY BOGDANIS

Human Muscle Metabolism Research Group, School of Sport and Exercise Sciences, Loughborough University, Loughborough LE11 3TU, UK

Accepted 19 December 2003

The purpose of this study was to examine the influence of a carbohydrate-rich meal on post-prandial metabolic responses and skeletal muscle glycogen concentration. After an overnight fast, eight male recreational/club endurance runners ingested a carbohydrate (CHO) meal (2.5 g $CHO \cdot kg^{-1}$ body mass) and biopsies were obtained from the vastus lateralis muscle before and 3 h after the meal. Ingestion of the meal resulted in a $10.6 \pm 2.5\%$ ($P < 0.05$) increase in muscle glycogen concentration (pre-meal *vs* post-meal: 314.0 ± 33.9 *vs* 347.3 ± 31.3 $mmol \cdot kg^{-1}$ dry weight). Three hours after ingestion, mean serum insulin concentrations had not returned to pre-feeding values (0 min *vs* 180 min: 45 ± 4 *vs* 143 ± 21 $pmol \cdot l^{-1}$). On a separate occasion, six similar individuals ingested the meal or fasted for a further 3 h during which time expired air samples were collected to estimate the amount of carbohydrate oxidized over the 3 h post-prandial period. It was estimated that about 20% of the carbohydrate consumed was converted into muscle glycogen, and about 12 % was oxidized. We conclude that a meal providing 2.5 g $CHO \cdot kg^{-1}$ body mass can increase muscle glycogen stores 3 h after ingestion. However, an estimated 67% of the carbohydrate ingested was unaccounted for and this may have been stored as liver glycogen and/or still be in the gastrointestinal tract.

Keywords: carbohydrate meal, muscle glycogen, post-prandial metabolism.

Introduction

During exercise, liver glycogen plays an important role in maintaining blood glucose concentrations within normal values (Coggan, 1991). On the other hand, fasting overnight reduces the liver glycogen stores and so decreases the availability of blood glucose for muscle metabolism (Nilsson and Hultman, 1973). Therefore, endurance athletes who compete after an overnight fast may fatigue prematurely.

Ingesting high glycaemic index (HGI) foods increases insulin concentrations before exercise and reduces fatty acid mobilization during exercise (Sherman, 1991). The perceived risk of experiencing a hypoglycaemic rebound, in blood glucose, and a decrease in fat metabolism following the ingestion of large amounts of HGI food before training or competition has led some athletes to avoid these foods. Therefore, eating low glycaemic index (LGI) food may be a logical alternative. Although some researchers have reported less metabolic perturbations during cycling with LGI food (Thomas *et al.*, 1991), ingesting a LGI meal before running does not provide an advantage over a HGI meal (Wee *et al.*, 1999b).

The ingestion of HGI carbohydrate (CHO) before cycling delays the onset of fatigue and improves endurance capacity (Schabort *et al.*, 1999; Wright *et al.*, 1991). In contrast to cycling, running after heavy meals can cause gastrointestinal distress (Brouns and Beckers, 1993). In a recent study, runners tolerated the ingestion of a HGI meal that provided 2.5 g $CHO \cdot kg^{-1}$ body mass (about 180 g in total) 3 h before exercise and their onset of fatigue was delayed compared with no-meal conditions (Chryssanthopoulos *et al.*, 2002). A possible mechanism for this improvement may be an increase in pre-exercise muscle glycogen concentrations. However, on this matter conflicting results have appeared in the literature. Some

* Address all correspondence to Costas Chryssanthopoulos, Faculty of Physical Education and Sport Science, Department of Sport Medicine and Biology of Exercise, National and Kapodistrian University of Athens, 41 Ethnikis Antistassis Street, 172 37 Dafne, Athens, Greece. e-mail: chryssan@phed.uoa.gr

Journal of Sports Sciences ISSN 0264-0414 print/ISSN 1466-447X online © 2004 Taylor & Francis Ltd
DOI: 10.1080/02640410410001730007

investigators have reported that the ingestion of 140 g of CHO results in an increase in muscle glycogen 4 h after ingestion (Coyle *et al.*, 1985), whereas others have reported no significant changes after ingestion of 100–200 g of CHO (Neufer *et al.*, 1987; Schabort *et al.*, 1999). In these studies, no pre-meal muscle biopsies were obtained for the determination of muscle glycogen concentrations and the post-meal muscle glycogen concentrations were compared with values observed in a separate experimental trial in which the participants fasted overnight.

The purpose of this study was to determine the influence of a HGI meal on skeletal muscle glycogen concentration 3 h after ingestion. In addition, we examined the post-prandial metabolic responses of the meal on blood glucose, serum insulin and free fatty acids concentrations. Furthermore, the amount of the ingested CHO oxidized over the 3 h period was also estimated in a subsequent study.

Methods

Participants

Eight male recreational/club endurance runners volunteered for the study. Their age, body mass, height and body mass index were 36.4 ± 5.2 years, 69.9 ± 3.9 kg, 1.76 ± 0.03 m and 22.2 ± 0.6 $kg \cdot m^{-2}$, respectively (mean $\pm s_{\bar{x}}$). All participants were fully informed verbally and in writing about the nature of the experiments, any known risks, and the right to terminate participation at will before signing a formal consent statement. A health history questionnaire was also completed and any individuals with medical problems were excluded. The study had the approval of the Ethical Advisory Committee of Loughborough University.

Experimental design

Muscle and blood samples

The participants were instructed to consume their normal diet on the 3 days before the experiment, and to refrain from any form of training on the day before the experiment.

After a 12 h overnight fast, the participants arrived at the laboratory at 08:00 h, voided and then their nude body mass was obtained. A cannula was inserted in an antecubital vein (Venflon, 16–18 G, Ohmeda, Hatfield, UK) using a local anaesthetic while the participants lay on an examination couch. In addition, a biopsy site was prepared on the participant's thigh, using a local anaesthetic (2 ml of 1% lignocaine), to obtain a sample from the vastus lateralis muscle using a percutaneous needle biopsy technique (Bergstrom, 1962) with suction being applied. After 10 ml of venous blood and a muscle sample were obtained, the participants consumed a high glycaemic CHO meal designed to provide 2.5 g $CHO \cdot kg^{-1}$ body mass.

The meal consisted of white bread, jam, corn flakes, skimmed milk and orange squash, which provided 86% of energy intake from CHO, 11% from protein and 3% from fat (Table 1). The estimated glycaemic index of the meal was 70 (Wolever *et al.*, 1991; Foster-Powell *et al.*, 2002), and the total amount of CHO consumed was 175 ± 10 g. The participants were instructed to consume the meal within 15 min.

Three hours after ingestion of the meal, a second muscle sample was obtained from the vastus lateralis at a site 5 cm distal to the site of the first biopsy. Venous blood samples were also taken, while the participants lay on an examination couch, at 15, 30, 60, 120 and 180 min after the ingestion of the meal. During the 3 h post-prandial period, the participants remained quietly in the laboratory, and lay on the examination couch for at least 10 min before blood sampling. The laboratory temperature was 20.5 ± 0.1°C and the relative humidity was 56.4 ± 1.2%.

Post-prandial oxidation of carbohydrate

In a subsequent study, the amount of CHO oxidized during the 3 h post-prandial period was determined using six recreational/club endurance runners, four of whom had taken part in the biopsy study. Their age, body mass, height and body mass index were 37.3 ± 5.6 years, 74.1 ± 4.1 kg, 1.76 ± 0.03 m and 23.9 ± 0.5 $kg \cdot m^{-2}$, respectively. This part of the study consisted of two trials. Therefore, the participants recorded their normal food intake the day before the first trial and replicated the same diet the day before the second trial. Also, the participants did not exercise the day before each trial, and followed the same training schedule for 3 days before each trial. The two trials were separated by 3–7 days and the order of the two trials was randomized.

After a 12 h overnight fast, the participants arrived at the laboratory at 08:00 h, voided and then nude body mass was obtained. A 5 min expired air sample was obtained, after which the participants consumed either the same high CHO meal (2.5 g of $CHO \cdot kg^{-1}$ body mass; Table 1) or fasted for a further 3 h. In the carbohydrate meal trial, about 185 ± 10 g of carbohydrate were consumed. The participants were instructed to consume the meal within 15 min. During the 3 h post-prandial (carbohydrate) or post-absorptive (fasted) period, 5 min expired air samples were collected at 30 min intervals using the Douglas bag method while the participants were sitting on a chair. During the

Table 1. Composition of the high carbohydrate meal

Type of food	Quantity	Carbohydrate	Fat	Protein	Fibre
White bread	105 g	48.8	2.2	8.6	1.9
Jam	70 g	48.6	—	0.2	0.8
Corn flakes	56 g	47.0	0.6	4.5	0.9
Skimmed milk	280 ml	14.0	0.3	9.5	—
Orange squash	147 ml	16.6	—	0.2	—
Water	315 ml	—	—	—	—
Total		175.0	3.1	23.0	3.6

Note: Values (g) calculated from the food labels are for a 70 kg man.

collection of air samples, the participants remained seated quietly in the laboratory for at least 15 min before each expired air sample was collected. Both trials were conducted under similar laboratory conditions of temperature (carbohydrate meal: $20.1 \pm 0.4°C$; fasted: $20.8 \pm 0.3°C$) and relative humidity (carbohydrate meal: $55.9 \pm 3.1\%$; fasted: $52.8 \pm 2.8\%$).

Analyses

The apparatus used and the methods of collection and analysis of expired air and venous blood samples were as described previously (Williams *et al*., 1990). The proportions of energy derived from carbohydrate and fat were estimated by indirect calorimetry (Consolazio *et al*., 1963) assuming no contribution of proteins. Venous blood samples were analysed for blood glucose, blood lactate and plasma free fatty acids (FFA) as previously described (Williams *et al*., 1990). Blood samples were also analysed for serum insulin (I^{125} radioimunoassay; Coat-A-Count Insulin, DPC kit) using a gamma counter (Packard, Cobra 5000). Muscle samples were analysed for glycogen, glucose, glucose-6-phosphate (G-6-P), adenosine triphosphate (ATP), phosphocreatine, creatine and lactate concentrations. The treatment of muscle samples and the analyses of the muscle metabolites were conducted as previously described (Harris *et al*., 1974).

Statistical analysis

A one-way analysis of variance (ANOVA) for repeated measures on one factor (i.e. across time) was used to examine the metabolic responses of the meal in the blood during the post-prandial period. A two-tailed Student's *t*-test for dependent samples was used to assess the effect of the meal on the various muscle metabolites measured. A two-way ANOVA for repeated measures on two factors (treatment × time) was used to compare respiratory responses between the carbohydrate and fasted trials. When significant differences were revealed using ANOVA, a Tukey *post hoc* test was performed. Finally, a Pearson correlation coefficient was used to assess relationships between variables. The level of significance was set at $P < 0.05$. The results are reported as the mean ± standard error of the mean ($s_{\bar{x}}$).

Results

Blood metabolites

Blood metabolites during the 3 h post-prandial period are shown in Table 2. The highest blood glucose concentrations were observed 15 min after the ingestion of the meal, and gradually decreased with time. The highest serum insulin concentrations were also measured at 15 min post-prandially; they remained elevated for 2 h and then decreased during the third hour. Three hours after the ingestion of the meal, serum insulin concentrations had not returned to pre-feeding values (0 min *vs* 180 min: 45 ± 4 *vs* 143 ± 21 $pmol \cdot l^{-1}$). However, due to the high variance in the post-prandial insulin values (Table 2), this difference was not statistically significant ($P > 0.05$).

After the ingestion of the carbohydrate meal, plasma FFA concentrations decreased with time and after 3 h they were six-fold lower ($P < 0.01$) than pre-feeding values. Blood lactate concentrations were 75% higher at 30 and 60 min compared with pre-feeding values ($P < 0.01$), but returned to pre-ingestion values by the end of the 3 h post-prandial period.

Muscle metabolites

The muscle metabolite concentrations before and 3 h after ingestion of the carbohydrate meal are shown in Table 3. Muscle glycogen concentration was 10.6 ± 2.5 % higher ($P < 0.05$) 3 h after ingestion of the meal (pre-meal *vs* post-meal: 314.0 ± 33.9 *vs* 347.3 ± 31.3 $mmol \cdot kg^{-1}$ dry weight; range: 227–437 and 243–452 $mmol \cdot kg^{-1}$ dry weight, respectively). However, the increase in muscle glycogen concentration varied between 5% and 31% (Fig. 1). Furthermore, there was a modest ($r = -0.55$) non-significant correlation between the relative increase (%) and the pre-meal muscle glycogen concentration. The muscle glucose, G-6-P, ATP, phosphocreatine, creatine and lactate concentrations were not different 3 h after ingestion of the meal.

Oxygen uptake and respiratory exchange ratio

Oxygen uptake and respiratory exchange ratios in the carbohydrate meal and fasted conditions are shown in Table 4. Oxygen consumption was higher ($P < 0.01$) during the first 2 h in the carbohydrate than in the

Table 2. Blood glucose, serum insulin, plasma free fatty acids and blood lactate concentrations during the 3 h post-prandial period (mean $\pm s_{\bar{x}}$)

	Time (min)					
Variable	0	15	30	60	120	180
Glucose ($mmol \cdot l^{-1}$)	4.2 ± 0.1^a	5.9 ± 0.4	5.1 ± 0.4	4.3 ± 0.4^a	4.1 ± 0.2^a	3.5 ± 0.3^{ac}
Insulin ($pmol \cdot l^{-1}$)	45 ± 4	426 ± 102^b	340 ± 74^b	338 ± 43^b	325 ± 55^b	143 ± 21^a
Free fatty acids	0.49 ± 0.13	0.31 ± 0.08	0.23 ± 0.07^b	0.13 ± 0.03^b	0.10 ± 0.02^{bd}	0.08 ± 0.02^{ab}
Lactate ($mmol \cdot l^{-1}$)	0.8 ± 0.1	1.2 ± 0.1^b	1.4 ± 0.1^{be}	1.4 ± 0.1^{be}	1.0 ± 0.1	0.9 ± 0.1

[a] $P < 0.01$ *vs* 15 min; [b] $P < 0.01$ *vs* 0 min; [c] $P < 0.01$ *vs* 30 min; [d] $P < 0.05$ *vs* 15 min; [e] $P < 0.01$ *vs* 180 min.

Table 3. Muscle glycogen, glucose, glucose-6-phosphate (G-6-P), lactate, adenosine triphosphate (ATP), phosphocreatine and creatine concentrations ($mmol \cdot kg^{-1}$ dry weight) in vastus lateralis muscle before and 3 h after the ingestion of the carbohydrate meal (mean $\pm s_{\bar{x}}$; $n = 6$)

Variable	Before meal	After meal
Glycogen	314.0 ± 33.9	347.3 ± 31.3*
Glucose	2.5 ± 0.5	1.6 ± 0.2
G-6-P	1.1 ± 0.3	1.2 ± 0.3
Lactate	3.4 ± 0.3	3.5 ± 0.2
ATP	22.8 ± 1.5	23.7 ± 0.6
Phosphocreatine	77.7 ± 3.9	80.7 ± 2.7
Creatine	43.8 ± 5.7	40.7 ± 3.6

* $P < 0.05$ *vs* before the meal.

fasted trial. Although the diet, training, time of day, temperature, humidity and laboratory procedures were similar in the two trials, surprisingly mean oxygen consumption ($\dot{V}O_2$) in the carbohydrate trial at time 0 was higher ($P < 0.05$) by 50 ml of O_2 (Table 4), something that was not a consequence of the ingested meal. This difference was mainly down to one participant who had almost 1.7-fold higher oxygen uptake in the carbohydrate than in the fasted trial (320 *vs* 190 ml O_2). When this participant was excluded, mean $\dot{V}O_2$ in the carbohydrate trial at time 0 was similar to that in the fasted trial (220 *vs* 180 ml O_2).

The respiratory exchange ratios were higher ($P < 0.01$) at 120 min and during the third hour of the carbohydrate trial than in the fasted trial (Table 4). However, the estimated carbohydrate oxidation rates were higher throughout the 3 h experimental period in the carbohydrate than in the fasted trial, whereas the estimated fat oxidation rates were different only at 150 and 180 min between conditions (Table 4).

The estimated resting energy expenditure over 3 h during the carbohydrate trial was higher than during the fasted trial (carbohydrate meal: 237 ± 14 kcal; fasted: 187 ± 15 kcal; $P < 0.05$). Also, the estimated total amount of carbohydrates oxidized during the 3 h experimental period was greater in the carbohydrate than in the fasted trial (carbohydrate meal: 40 ± 3 g; fasted: 19 ± 3 g; $P < 0.01$). Finally, although the estimated total amount of fat oxidized in the fasted trial was 13 ± 2 g and that in the carbohydrate trial was 9 ± 1 g, this difference did not reach statistical significance ($P = 0.07$).

Discussion

The main finding of this study was that the ingestion of the high glycaemic index (HGI) carbohydrate meal (2.5 g $CHO \cdot kg^{-1}$ body mass; estimated glycaemic index = 70; 175 g carbohydrate) resulted in an 11% increase in the glycogen concentration of the vastus lateralis muscle. Neufer *et al.* (1987) also reported a similar increase in muscle glycogen concentration (15%) following a carbohydrate-rich meal (200 g), but this increase did not reach statistical significance. On the other hand, Coyle *et al.* (1985) found a 42% increase in muscle glycogen concentrations after their participants ingested 140 g of carbohydrate. In contrast, Schabort *et al.* (1999) reported that a HGI meal, providing 100 g of carbohydrate, did not increase muscle glycogen concentration in fasting individuals. Nevertheless, in a recent study from our laboratory, in which the same amount and type of CHO was ingested (2.5 g $CHO \cdot kg^{-1}$ body mass), we found that muscle glycogen concentration increased by 15% during a post-prandial period of 3 h. However, when the same amount of carbohydrate was provided in the form of low glycaemic index (LGI) foods, there was no increase in muscle glycogen concentration 3 h after consuming the LGI meal (Wee *et al.*, 1999a). Furthermore, in a study that employed the use of ^{13}C-nuclear magnetic resonance spectroscopy, a considerable amount of the dietary CHO (20%) was estimated to have been converted into glycogen in the skeletal muscle 5 h after the ingestion of

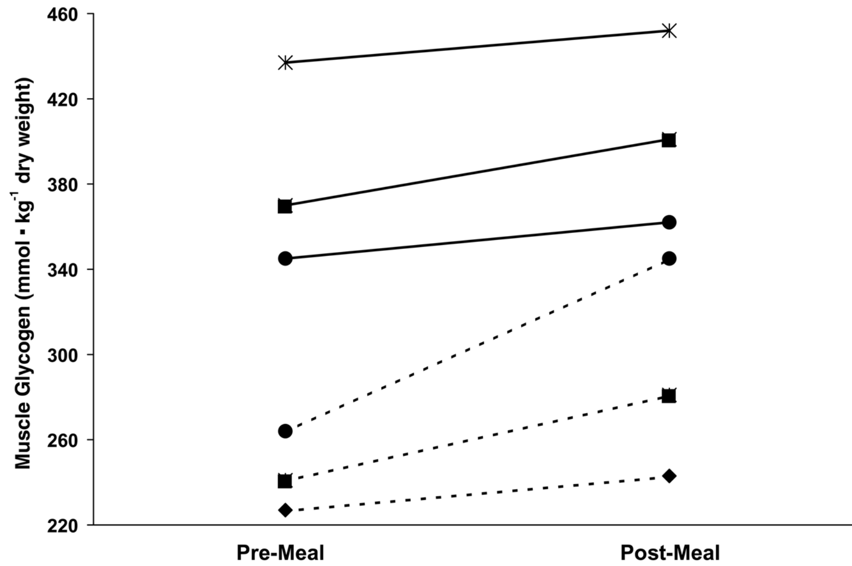

Fig. 1. Individual skeletal muscle glycogen concentrations before (Pre-Meal) and 3 h after (Post-Meal) ingestion of the carbohydrate meal.

Table 4. Oxygen consumption ($\dot{V}O_2$), respiratory exchange ratio (RER), carbohydrate (CHO) and fat oxidation rates (mean $\pm s_{\bar{x}}$)

	Time (min)						
Variable	0	30	60	90	120	150	180
$\dot{V}O_2$ (ml·min^{-1})							
Fasted	190 ± 20	200 ± 20	220 ± 20	220 ± 20	210 ± 20	230 ± 20	240 ± 20^{c}
Carbohydrate	240 ± 20^{b}	280 ± 10^{b}	290 ± 10^{ac}	280 ± 20^{a}	290 ± 20^{ac}	260 ± 20	260 ± 30
RER							
Fasted	0.84 ± 0.04	0.87 ± 0.04	0.83 ± 0.03	0.83 ± 0.03	0.80 ± 0.04	0.80 ± 0.03	0.80 ± 0.02
Carbohydrate	0.83 ± 0.03	0.89 ± 0.02	0.88 ± 0.02	0.89 ± 0.01	0.90 ± 0.01^{ac}	0.93 ± 0.02^{ac}	0.95 ± 0.01^{ac}
CHO oxidation (g·min^{-1})							
Fasted	0.10 ± 0.03	0.12 ± 0.03	0.10 ± 0.01	0.13 ± 0.02	0.09 ± 0.02	0.09 ± 0.02	0.09 ± 0.02
Carbohydrate	0.11 ± 0.02	0.20 ± 0.02^{bc}	0.20 ± 0.02^{ac}	0.22 ± 0.02^{ac}	0.23 ± 0.02^{ac}	0.23 ± 0.01^{ac}	0.25 ± 0.02^{ac}
Fat oxidation(g·min^{-1})							
Fasted	0.05 ± 0.02	0.05 ± 0.01	0.07 ± 0.01	0.07 ± 0.01	0.07 ± 0.01	0.08 ± 0.01^{a}	0.09 ± 0.01^{a}
Carbohydrate	0.07 ± 0.01	0.06 ± 0.01	0.06 ± 0.01	0.05 ± 0.00	0.05 ± 0.00	0.04 ± 0.01^{ac}	0.03 ± 0.01^{ac}

[a] $P < 0.01$ *vs* fasted; [b] $P < 0.05$ *vs* fasted; [c] $P < 0.01$ *vs* 0 min.

a meal containing about 290 g of CHO (Taylor *et al.*, 1993).

During the post-prandial phase, the metabolic disposal of the ingested CHO follows four main pathways: (a) uptake and glycogen synthesis in the liver, (b) uptake and glycogen synthesis in the muscles, (c) uptake and direct oxidation in various tissues, and (d) uptake and lipid synthesis in the liver and fat tissue (Jequier, 1994). In the present study, liver glycogen was not assessed. However, it is reasonable to suggest that after 12 h of fasting a considerable amount of the ingested CHO will have been synthesized as liver glycogen over the 3 h post-prandial period (Nilsson and Hultman, 1973; Nilsson and Hultman, 1974; Bjorntorp and Sjostrom, 1978; Flatt, 1995). Direct measurements of liver glycogen content have shown that a considerable amount of carbohydrate given orally (about 440 g daily; Nilsson and Hultman, 1973) or intravenously (about 270 g within 4 h; Nilsson and Hultman, 1974) is synthesized to liver glycogen.

In the present study, oxidation of the ingested carbohydrate load was estimated from the total carbohydrate oxidation in the carbohydrate trial after subtracting the carbohydrate oxidation in the fasted trial. Assuming that the excess carbohydrate oxidized in the carbohydrate trial was derived from the ingested meal (Acheson *et al.*, 1982), the excess 21 g of carbohydrates oxidized in the carbohydrate trial represented 11.6% of the ingested load (i.e. [21.4/185] $\times$ 100). This would also represent about 20 g of carbohydrates being oxidized in the first part of the study (i.e. [11.6 $\times$ 175] / 100), if expired air had been measured. This leaves about 155 g from the total 175 g to be accounted for. Assuming a molecular weight of 165 for the stored glycogen expressed in glucosyl units, a muscle mass of 40% of total body weight, a dry/wet weight ratio of muscle tissue of 4.3 (Nilsson and Hultman, 1974), and a similar degree of glycogen synthesis by the various muscles, the increase in muscle glycogen content found can account for about 36 g of the CHO ingested. This represents about 21% of the total amount of CHO consumed. Therefore, about 120 g of CHO, or 67% of the total CHO load, remains to be accounted for.

Considering the last of the four possible pathways for the disposal of CHO, then the amount of lipid formation from the ingested CHO was probably minimal. Although liver and adipose tissue contain the enzymes necessary for the conversion of CHO into fat, dietary carbohydrates do not easily increase an individual's fat content by *de novo* lipogenesis (Jequier, 1994). Even larger amounts of dietary CHO (500 g) than the amount ingested in the present study (175–185 g) do not produce a substantial degree of lipogenesis in healthy non-obese individuals (Acheson *et al.*, 1982, 1988). Nevertheless, the possibility that a small amount of lipid was formed cannot be excluded because the presence of elevated insulin concentrations during the post-prandial period may have promoted this process (Newsholme and Leech, 1983).

These arguments are based on the assumptions that the CHO meal was completely digested and absorbed during the 3 h post-prandial period. Even assuming that liver glycogen content after the overnight fast was 80 g and 3 h after the meal increased by 100 g, reaching a high value of 180 g (Nilsson and Hultman, 1973), there is still about 10% (18 g) of the ingested carbohydrate to be accounted for. Therefore, the possibility that some portion of the meal was still in the stomach or small intestine 3 h after ingestion should also be considered. This is supported by the fact that 3 h after the ingestion of the meal, serum insulin concentrations, although not statistically significant, had not returned to pre-feeding values (0 min *vs* 180 min: 45 ± 4 *vs* 143 ± 21 pmol $\cdot$ l^{-1}) (Table 2). This may indicate that the digestion and absorption processes were still in progress. However, these are only estimates, because gastric emptying and intestinal absorption were not determined in this study.

In theory, if a significant portion of the meal is still in the stomach 3 h after ingestion – that is, at the beginning of exercise – then gastrointestinal distress may be experienced, especially if running rather than cycling is the mode of exercise (Brouns and Beckers, 1993). However, the same type of meal used in the present study was also used in our two previous running studies and was well tolerated by all the runners (Chryssanthopoulos and Williams, 1997, Chryssanthopoulos *et al.*, 2002). Nevertheless, if larger meals are prescribed, then it is advisable that they are consumed more than 3 h before exercise to avoid possible gastrointestinal discomfort.

The elevated blood lactate concentrations observed during the first hour after the meal (Table 2) are consistent with other studies that showed that blood lactate concentration increases after the consumption of high carbohydrate (Chryssanthopoulos *et al.*, 1994) or mixed meals (Segal *et al.*, 1990). This post-prandial lactate may reflect a conversion of dietary glucose to glycogen via an indirect pathway in which the glucose is taken up by peripheral tissues and degraded to lactate, which, in turn, is transported to the liver to serve as gluconeogenic substrate (Katz and McGarry, 1984).

Both the present study and the studies reported by Sherman (1991) show that ingesting large amounts (> 170 g) of HGI carbohydrates before exercise cause an increase in serum insulin concentrations, even 3 h after ingestion, and reduced FFA concentrations during exercise. Nevertheless, there are performance benefits from ingesting a high CHO meal 3 h before cycling (Wright *et al.*, 1991) and running (Chryssanthopoulos *et al.*, 2002).

In summary, the ingestion of a HGI carbohydrate meal, providing 2.5 g CHO $\cdot$ kg^{-1} body mass, produced an 11% increase in the glycogen concentration of the vastus lateralis muscle. However, 3 h after ingestion of the meal the majority of the CHO ingested was probably in the liver and some still in the gastrointestinal tract.

References

Acheson, K.J., Flatt, J.P. and Jequier, E. (1982). Glycogen synthesis versus lipogenesis after a 500 gram carbohydrate meal in man. *Metabolism*, **31**, 1234–1240.

Acheson, K.J., Schutz, Y., Bessard, T. *et al.* (1988). Glycogen storage capacity and *de novo* lipogenesis during massive carbohydrate overfeeding in man. *American Journal of Clinical Nutrition,* **48**, 240–247.

Bergstrom, J. (1962). Muscle electrolytes in man. Determined by neutron activation analysis on needle biopsy specimens. A study on normal subjects, kidney patients, and patients with chronic diarrhoea. *Scandinavian Journal of Clinical and Laboratory Investigations,* **68**(suppl.), 1 – 110.

Bjorntorp, P. and Sjostrom, L. (1978). Carbohydrate storage in man: speculations and some quantitative considerations. *Metabolism*, **27**(suppl. 2), 1853 – 1865.

Brouns, F. and Beckers, E. (1993). Is the gut an athletic organ? Digestion, absorption and exercise. *Sports Medicine*, **15**, 242 – 257.

Chryssanthopoulos, C. and Williams, C. (1997). Pre-exercise carbohydrate meal and endurance running capacity when carbohydrates are ingested during exercise. *International Journal of Sports Medicine*, **18**, 543 – 548.

Chryssanthopoulos, C., Williams, C., Wilson, W., Asher, L. and Hearne, L. (1994). Comparison between carbohydrate feedings before and during exercise on running performance during a 30-km treadmill time trial. *International Journal of Sports Nutrition*, **4**, 374 – 386.

Chryssanthopoulos, C., Williams, C., Nowitz, A., Kotsiopoulou, C. and Vleck, V. (2002). The effect of a high carbohydrate meal on endurance running capacity. *International Journal of Sport Nutrition and Exercise Metabolism,* **12**, 157 – 171.

Coggan, A.R. (1991). Plasma glucose metabolism during exercise in humans. *Sports Medicine*, **11**, 102 – 124.

Consolazio, C.J., Johnson, R.E. and Pecora, L.J. (1963). *Physiological Measurements of Metabolic Function in Man.* New York: McGraw-Hill.

Coyle, E.F., Coggan, A.R., Hemmert, M.K., Lowe, R.C. and Walters, T.J. (1985). Substrate usage during prolonged exercise following a pre-exercise meal. *Journal of Applied Physiology*, **59**, 429 – 433.

Flatt, J. (1995). Use and storage of carbohydrate and fat. *American Journal of Clinical Nutrition,* **61**, 952S – 959S.

Foster-Powell, K., Holt, S.H.A. and Brand-Miller, J.C.B. (2002). International table of glycemic index and glycemic load values: 2002. *American Journal of Clinical Nutrition,* **76**, 5 – 56.

Harris, R.C., Hultman, E. and Nordesjo, L.O. (1974). Glycogen, glycolytic intermediates and high energy phosphates determined in biopsy samples of m. quadriceps femoris of man at rest: methods and variance of values. *Scandinavian Journal of Clinical and Laboratory Investigation*, **33**, 109 – 120.

Jequier, E. (1994). Carbohydrate as a source of energy. *American Journal of Clinical Nutrition,* **59**(suppl.), 682S – 685S.

Katz, J. and McGarry, J.D. (1984). The glucose paradox: is glucose a substrate for liver metabolism? *Journal of Clinical Investigation*, **74**, 1901 – 1909.

Neufer, P.D., Costill, D.L., Flynn, M.G., Kirwan, J.P., Mitchell, J.B. and Houmard, J. (1987). Improvements in exercise performance: effects of carbohydrate feedings and diet. *Journal of Applied Physiology*, **62**, 983 – 988.

Newsholme, E.A. and Leech, A.R. (1983). *Biochemistry for the Medical Sciences*. New York: Wiley.

Nilsson, L.H. and Hultman, E. (1973). Liver glycogen in man: the effect of total starvation or a carbohydrate-poor diet followed by carbohydrate refeeding. *Scandinavian Journal of Clinical and Laboratory Investigation*, **32**, 325 – 330.

Nilsson, L.H. and Hultman, E. (1974). Liver and muscle glycogen in man after glucose and fructose infusion. *Scandinavian Journal of Clinical and Laboratory Investigation*, **33**, 5 – 10.

Schabort, E.J., Bosch, A.N., Weltan, S.M. and Noakes, T. (1999). The effects of a pre-exercise meal on time to fatigue during prolonged cycling exercise. *Medicine and Science in Sports and Exercise*, **31**, 464 – 471.

Segal, K.R., Edano, A. and Thomas, M.B. (1990). Thermic effect of a meal over 3 and 6 hours in lean and obese men. *Metabolism*, **39**, 958 – 992.

Sherman, W.M. (1991). Carbohydrate feedings before and after exercise. In *Perspectives in Exercise Science and Sports Medicine, Vol. 4: Ergogenics. Enhancement of Performance in Exercise and Sport* (edited by D.R. Lamb and M.H. Williams), pp. 1 – 34. Brown & Benchmark.

Taylor, R., Price, T.B., Katz, L.D., Shulman, R.G. and Shulman, G.I. (1993). Direct measurement of change in muscle glycogen concentration after a mixed meal in normal subjects. *American Journal of Physiology*, **265**, E224 – E229.

Thomas, D.E., Brotherhood, J. and Brand, J.C. (1991). Carbohydrate feeding before exercise: effect of glycemic index. *International Journal of Sports Medicine*, **12**, 180 – 186.

Wee, S.L., Williams, C., Tsintzas, K. and Boobis, L. (1999a). Effect of high and low glycaemic index pre-exercise meals on muscle glycogen and exercise metabolism. In *Proceedings of the 4th Annual Congress of the European College of Sports Science* (edited by P. Parisi, F. Pigozzi and G. Prinzi), p. 44. Rome: University Institute of Motor Sciences.

Wee, S.L., Williams, C., Gray, S. and Horabin, J. (1999b). Influence of high and low glycemic index meals on endurance running capacity. *Medicine and Science in Sports and Exercise*, **31**, 393 – 399.

Williams, C., Nute, M.G., Broadbank, L. and Vinall, S. (1990). Influence of fluid intake on endurance running performance: a comparison between water, glucose and fructose solutions. *European Journal of Applied Physiology*, **60**, 112 – 119.

Wolever, T.M.S., Jenkins, D.J.A., Jenkins, A.L. and Josse, R.G. (1991). The glycemic index: methodology and clinical implications. *American Journal of Clinical Nutrition,* **54**, 846 – 854.

Wright, D.A., Sherman, W.M. and Dernbach, A.R. (1991). Carbohydrate feedings before, during or in combination improve cycling endurance performance. *Journal of Applied Physiology,* **71**, 1082 – 1088.