Ingestion of a high-glycemic index meal increases muscle glycogen storage at rest but augments its utilization during subsequent exercise

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Ingestion of a high-glycemic index meal increases muscle glycogen storage at rest but augments its utilization during subsequent exercise. Preexercise CHO ingestion may elicit metabolic responses to favor a more economical utilization of muscle glycogen during exercise after ingestion of a LGI than a HGI meal. However, there were no differences in endurance running times between trials.

Preexercise feeding; macroglycogen; proglycogen; fat metabolism

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preexercise meals are consistent with current recommendation for athletes (4, 6). It was hypothesized that, when compared with HGI, ingestion of a LGI meal would induce a smaller metabolic perturbation resulting in lower CHO oxidation and muscle glycogen utilization during exercise.

MATERIALS AND METHODS

Subjects. Seven male recreational runners [age 31 ± 4 yr, body height 174 ± 2 cm, body mass 71.7 ± 3.1 kg, VO2max 55.1 ± 2.3 ml·kg⁻¹·min⁻¹] volunteered as subjects for this study after being informed of the risks associated with the procedures and signing a letter of informed consent. The study was approved by the Loughborough University Ethical Committee.

Preliminary tests. All subjects performed three preliminary tests: 1) a 16-min incremental submaximal running test to determine the relationship between running speed and oxygen uptake (VO2), 2) an uphill treadmill running test to determine VO2max as described elsewhere (44), and 3) a 30-min run on the treadmill at 70% of their respective VO2max to familiarize themselves with the measurements to be taken during the two main experimental trials.

Dietary control. All subjects adhered to their normal diets throughout the study. Subjects were required to record their training and weighed food intake during the 2 days before the first trial and to replicate these before the second trial. The dietary information obtained was then analyzed by a registered dietician. Each subject refrained from physical training, caffeine, and alcohol intake the day before each trial. On the days of the main trials, the subjects arrived at the laboratory between 8:00 and 10:30 AM, after a 12-h overnight fast. After collection of baseline data, they received one of the test meals. The HGI and LGI meals were of identical macronutrient composition, energy content, and volume (Table 1).

Experimental protocol. The two experimental trials separated by 14 days. When they arrived at the laboratory, each subject rested quietly on an examination couch for at least 15 min. An indwelling cannula (Venflon, 16 gauge) was then placed in an antecubital forearm vein under local anesthesia (1% lignocaine). The cannula was kept patent (Venflon, 16 gauge) was then placed in an antecubital forearm vein under local anesthesia (1% lignocaine) while the subject was lying on an examination couch. After removal of the biopsy needle from the leg, the needle containing the muscle sample was immediately immersed in liquid nitrogen. Muscle samples were stored in liquid nitrogen until use. Muscle samples were stored in liquid nitrogen until they were freeze-dried, after which they were stored at −70°C. At a later date, the freeze-dried muscle was washed twice with petroleum ether (40%) to remove fat, dissected free of visible blood and connective tissue, and then powdered. Muscle was then extracted with PCA, and concentrations of free glucose, glucose-6-phosphate (G-6-P), lactate, ATP, creatine (Cr), and phosphocreatine (PCr) were determined enzymatically (23, 31). A fraction of the extract as well as the PCA-insoluble fraction were hydrolyzed in 1 M HCl and used for the determination of macroglycogen (MG; from PCA extract) and proglycogen (PG; from PCA-insoluble fraction) (1, 25). The total mixed muscle glycogen concentration (GLG) was calculated by adding the MG and PG concentrations. All muscle metabolites except lactate were adjusted to peak total Cr for each subject to correct for

<table>
<thead>
<tr>
<th>Meal</th>
<th>Description</th>
<th>Nutrient Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGI</td>
<td>60 g corn flakes + 240 ml skimmed milk + 100 g white bread + 30 g raspberry jam + 265 ml Lucozade original energy drink + 587 ml water</td>
<td>3.43 MJ, 175 g CHO, 21 g protein, 4 g fat, 1,070 ml water estimated GI = 80*</td>
</tr>
<tr>
<td>LGI</td>
<td>80 g bran flakes + 200 ml skinned milk + 360 g canned peach in own juice + 300 g apples + 500 ml unsweetened apple juice</td>
<td>3.43 MJ, 175 g CHO, 21 g protein, 4 g fat, 1,070 ml water estimated GI = 36*</td>
</tr>
</tbody>
</table>

GI, glycemic index; LGI, low GI; HGI, high GI; CHO, carbohydrate. *Calculated by a method described in Wolever (46) with GI values taken from Foster-Powell et al. (20). Information from manufacturer or food tables was used to calculate macronutrient energy contribution.
variability in blood, connective tissue, and other nonmuscle constituents between biopsies.

Statistics. The changes in physiological and biochemical variables were analyzed by a two-way ANOVA for repeated measures (meal × time). For values attaining this criterion, the Tukey’s post hoc test was used to locate the difference(s). The dietary data, incremental area under the curve, total substrate oxidation, and energy expenditure were analyzed by Student’s paired t-tests. Statistical significance was set at \( P < 0.05 \). All data are reported as means ± SE.

RESULTS

During the 2 days before each trial, there were no significant differences between the recorded average daily energy intake and amounts of CHO, fat, or protein consumed. The mean daily energy intake and macronutrient composition were 12.61 ± 1.12 MJ, 422 ± 8 g CHO, 97 ± 3 g fat, and 121 ± 7 g protein before the LGI trial and 12.16 ± 1.20 MJ, 414 ± 15 g CHO, 84 ± 3 g fat, and 124 ± 14 g protein before the HGI trial. All subjects replicated their training schedule on the second day before the second trial and refrained from exercise, caffeine, and alcohol 24 h before each trial.

Plasma glucose, glucagon, serum insulin concentrations, and the ratio of glucagon to insulin concentrations during the 3-h postprandial period and exercise are shown in Fig. 1. Plasma glucose concentrations peaked at 15 min after ingestion of both meals and returned to basal levels by the end of the 3-h postprandial period. The incremental areas under the plasma glucose response curve for 1, 2, and 3 h after the HGI meal were 1.9-, 3.2-, and 3.9-fold greater, respectively, than those after the LGI meal (\( P < 0.05 \) in all cases). At 10 min into exercise during the HGI trial, plasma glucose concentrations declined sharply to values (4.05 ± 0.18 mM; \( P < 0.05 \) from LGI) lower than that at the onset of exercise (4.75 ± 0.29 mM; \( P < 0.05 \)). However, by the end of exercise, plasma glucose concentrations in the HGI trial (5.16 ± 0.20 mM) had increased to values higher than those in the LGI trial (4.79 ± 0.24 mM; \( P < 0.05 \)).

Serum insulin concentrations peaked at 15–30 min after ingestion of both meals. The incremental area under the serum insulin response curve for 1, 2, and 3 h after the HGI meal were 1.0-, 1.2-, and 1.4-fold greater, respectively, than those after the LGI meal (\( P < 0.001 \) in all cases). Just before exercise, serum insulin concentrations during the HGI trial (24.0 ± 3.6 mIU/l) was twice that observed at the same time during the LGI trial (12.1 ± 0.6 mIU/l; \( P < 0.05 \)). At that time, serum insulin concentrations during both trials were still elevated (\( P < 0.01 \)) compared with fasting values (7.6 ± 0.2 mIU/l). After 10 min of exercise, serum insulin concentrations during the HGI trial (10.9 ± 1.4 mIU/l) were still slightly higher compared with the LGI trial (8.0 ± 0.3 mIU/l; \( P = 0.07 \)) but decreased to similar values thereafter.

After the LGI meal, plasma glucagon concentrations increased above fasting values and remained elevated for 2 h (\( P < 0.05 \)). Plasma glucagon concentrations were higher in the LGI than the HGI trial throughout the 3-h postprandial period (\( P < 0.05 \)). The differences in pancreatic hormonal responses after the HGI and LGI meals became clearer when the glucagon-to-insulin ratios were compared. These ratios were higher throughout the postprandial and exercise period after the LGI than the HGI meal (\( P < 0.05 \)).

Fig. 1. Plasma glucose (A), serum insulin (B), plasma glucagon (C), and ratio of glucagon to insulin concentrations (D) during the 3-h postprandial period and subsequent 30 min of exercise in the high-glycemic index (HGI) and low-glycemic index (LGI) trials. Values are means ± SE. Time is given as h:min. *\( P < 0.05 \) from LGI. a\( P < 0.05 \) from time 0:00. b\( P < 0.05 \) from time 3:00.
The plasma FFA and glycerol response curves are shown in Fig. 2. Plasma FFA and glycerol concentrations were lower than fasting values after both meals during the 3-h postprandial period (P < 0.05). However, this suppression of fatty acid mobilization was less after the LGI meal than after the HGI meal. Indeed, plasma FFA and glycerol concentrations were higher at the end of the postprandial period and during exercise in the LGI than the HGI trial (P < 0.05).

Mean values for VO₂ and HR during both trials are shown in Table 2. VO₂ and RER responses were higher during the postprandial period compared with the fasted state but were not different between trials. Blood lactate concentrations were higher during the 3-h postprandial period after the LGI meal (P < 0.05) than the HGI meal. During exercise, VO₂ and HR were not different between trials. The average intensities sustained during exercise were 71.0 ± 1.2% V̇O₂ max and 71.0 ± 1.3% V̇O₂ max during the LGI and HGI trials, respectively. The mean RER values and blood lactate concentrations during exercise were lower in the LGI than the HGI trial (P < 0.05). Overall, substrate oxidation during the postprandial period did not differ between trials (Fig. 3). During exercise, total CHO oxidation was 12% lower (P < 0.05) during the LGI trial with a compensatory increase in fat oxidation (P < 0.05) compared with the HGI trial, such that the overall energy expenditure was similar (Fig. 3).

Table 2. Mean oxygen uptake, RER, blood lactate concentrations, and heart rates during the LGI and HGI trials

<table>
<thead>
<tr>
<th></th>
<th>LGI</th>
<th>HGI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen uptake, l/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal</td>
<td>0.29±0.02</td>
<td>0.29±0.02</td>
</tr>
<tr>
<td>Postprandial</td>
<td>0.37±0.02</td>
<td>0.36±0.02</td>
</tr>
<tr>
<td>Exercise</td>
<td>2.85±0.18</td>
<td>2.82±0.21</td>
</tr>
<tr>
<td>RER values</td>
<td></td>
<td></td>
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<tr>
<td>Basal</td>
<td>0.81±0.03</td>
<td>0.82±0.03</td>
</tr>
<tr>
<td>Postprandial</td>
<td>0.91±0.02</td>
<td>0.94±0.02</td>
</tr>
<tr>
<td>Exercise</td>
<td>0.96±0.01*</td>
<td>0.99±0.01</td>
</tr>
<tr>
<td>Blood lactate concen.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal</td>
<td>0.8±0.1</td>
<td>0.7±0.1</td>
</tr>
<tr>
<td>Postprandial</td>
<td>1.8±0.1*</td>
<td>1.1±0.1</td>
</tr>
<tr>
<td>Exercise</td>
<td>2.7±0.5</td>
<td>3.2±0.5</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise</td>
<td>161±6</td>
<td>162±5</td>
</tr>
</tbody>
</table>

Values are means ± SE. RER, respiratory exchange ratio. *P < 0.05 from HGI.

Premeal, preexercise, and postexercise GLG concentrations are shown in Fig. 4. The exercise and dietary control before each trial resulted in similar GLG concentrations before the respective meals (LGI: 380 ± 26 mmol/kg dry mass; HGI: 407 ± 45 mmol/kg dry mass). Three hours after the HGI meal, muscle glycogen concentration was elevated by 15% (P < 0.05) but remained unaltered after the LGI meal. At the end of exercise, glycogen concentrations were reduced in both trials (P < 0.05) and values did not differ between trials (LGI: 315 ± 16 mmol/kg dry mass; HGI: 340 ± 46 mmol/kg dry mass). However, net muscle glycogen utilization was 46% greater (P < 0.01) during the HGI (129 ± 16 mmol/kg dry mass) compared with the LGI (88 ± 15 mmol/kg dry mass) trial.

Concentrations of the MG and PG pools are shown in Fig. 5. The PG fraction was always in excess of MG, which varied from 2 to 21% of GLG. Similar to the changes in GLG, MG (+32%; P < 0.05) and PG (+15%; P < 0.05) concentrations increased 3 h after the HGI meal but remained unaltered after the LGI meal. Although the fractional contribution of PG to the total amount of glycogen synthesized during the postprandial period was 76% as opposed to 24% for the MG pool (5 of the 7 subjects demonstrated higher storage of PG than MG), this difference failed to reach statistical significance (P = 0.14).

Fig. 2. Plasma free fatty acid (FFA; A) and glycerol (B) concentrations during the 3-h postprandial period and subsequent 30 min of exercise in the HGI and LGI trials. Values are means ± SE. Time is given as h:min. *P < 0.05 from HGI.

Fig. 3. Substrate oxidation during the 3-h postprandial rest period and subsequent 30 min of exercise in the HGI and LGI trials. Values are means ± SE. CHO, carbohydrate. *P < 0.05 from HGI.
After exercise in both trials, the fractional (% of preexercise value) reduction in the respective pools was similar. In the HGI trial, MG decreased by 59% (P < 0.05), whereas PG decreased by 23% (P < 0.05). In the LGI trial, MG decreased by 41% (P < 0.01), whereas PG decreased by 20% (P < 0.01). However, the rate of muscle PG utilization was higher than that of the MG pool in the LGI trial (2.4 ± 0.5 vs. 0.6 ± 0.1 mmol·kg⁻¹ dry mass·min⁻¹; P < 0.05) but did not reach statistical significance in the HGI trial (3.0 ± 0.6 vs. 1.4 ± 0.2 mmol·kg⁻¹·dry mass·min⁻¹; P = 0.16).

Changes in concentrations of other muscle metabolites are presented in Table 3. Where changes in ATP, PCR, and G-6-P did occur, they were similar between trials. In agreement with the blood lactate data during exercise, muscle lactate concentrations were higher in the HGI than the LGI trial after exercise (P < 0.05).

**DISCUSSION**

The major finding of this study was that when different preexercise breakfasts were consumed, a HGI meal contributed less CHO to the muscle glycogen stores during a 3-h postprandial period compared with a HGI meal. However, plasma FFA availability and fat oxidation were better maintained in the LGI trial, together with a sparing of muscle glycogen utilization and lower muscle lactate accumulation during 30 min of submaximal running. Furthermore, our results also suggest that when muscle glycogen concentration is normal, that is neither supercompensated nor depleted, changes in the PG fraction appear to occur more readily than the MG fraction in response to feeding and exercise.

A number of studies have quantified muscle glycogen storage 3 or 4 h after breakfast consisting of HGI or MGI CHO foods and reported glycogen accumulation ranging from 10 to 42% (9, 12, 34, 39). The 15% increase in GLG concentration 3 h after ingestion of the HGI meal is in close agreement with these studies. However, GLG concentration did not alter after the LGI meal of identical macronutrient content. In another study, muscle glycogen storage after 24 h of recovery after glycogen-depleting exercise was greater with a HGI than a LGI diet (7). The subjects in the present study were on a normal mixed diet and agreed not to exercise the day before the experiment. Their premeal GLG concentrations were normal and not supercompensated. It is most likely that the rapidly digested and absorbed foods in the HGI meal supplied the necessary glucose to the blood and muscle for glycolysis within the 3-h postprandial period. This is supported by the greater incremental area under the plasma glucose (3.9-fold) and serum insulin (1.4-fold) response curves after ingestion of the HGI compared with the LGI meal and the observation that postprandial CHO oxidation rates were similar between trials. Thus the low glycemic and insulinemic responses to the LGI meal (secondary to slow digestion and absorption of the ingested foods) may account for the small nonsignificant increase in muscle glycogen content in that trial. These results demonstrate that, even without stimulation of prior muscle contraction, HGI foods confer an advantage in terms of muscle glycogen storage compared with LGI foods.

Utilization of plasma FFA decreases after a CHO meal partly because the CHO-induced rise in insulin inhibits the mobilization and hence availability of circulating FFA and partly due to an increase in CHO oxidation, which may reduce fat oxidation by inhibiting the rate of long-chain fatty acid entrance into the mitochondria for β-oxidation (13, 37). Even during low-intensity exercise, preexercise glucose ingestion suppresses lipolysis to a point at which it limits fat oxidation (24). Preexercise ingestion of glucose either increases (11, 22) or does not influence (19) muscle glycogen utilization at 70% $\text{VO}_{2\text{max}}$. A number of investigators have applied the GI concept to address the still controversial question of the benefit of CHO intake within the hour before exercise (15–17, 27–29, 38, 40, 41). However, sportsmen and sportswomen normally eat a CHO-rich meal a few hours before, rather than eating shortly before, undertaking endurance exercise. Nevertheless, the suppression of fat oxidation during exercise persists for at least 4 h after a high CHO (2 g CHO/kg body mass) meal, even when insulin concentration had returned to premeal basal levels (33).

Two studies have examined the effect of GI of preexercise meals consumed in the hours before endurance exercise on metabolism and endurance performance (5, 43). Burke and coworkers (5) reported that the GI of preexercise meals does not influence exercise metabolism and performance when large amounts of CHO are ingested during cycling exercise. Because it is difficult to deliver as much fluid and CHO during running compared with cycling, the preexercise meal is likely to play a more important role in preparation for running. In our present and previous (43, 48) studies, eating a LGI meal (2–2.5 g

![Fig. 4. Muscle glycogen concentrations premeal, preexercise, and postexercise in the HGI and LGI trials. Values are means ± SE. dm, Dry mass. *P < 0.05 from premeal. **P < 0.05 from preexercise.](Image)

![Fig. 5. Muscle proglycogen (PG) and macroglycogen (MG) concentrations premeal, preexercise, and postexercise in the HGI and LGI trials. Values are means ± SE. *P < 0.05 from premeal. **P < 0.05 from preexercise.](Image)
CHO/kg body mass) 3 h before running at ~70% \( \dot{V}_{\text{O}_2} \text{max} \) resulted in reduced glycemic and insulinemic responses, increased plasma FFA availability, and a shift in substrate utilization from CHO to fat during exercise compared with a HGI meal. In the present study, the greater CHO metabolism during the HGI trial was accompanied by a greater degradation of muscle glycogen and accumulation of muscle and blood lactate during exercise. Because no relationship was found between the preexercise glycogen concentrations and the utilization of glycogen in that trial (\( r = -0.1 \)), the data from the present study support the notion that the increased utilization of glycogen in that trial was related to the metabolic environment provided by the HGI feeding.

The present study is the first report of reduced muscle glycogen utilization during exercise after a LGI compared with a HGI meal. Our finding is in contrast with that of Febbraio and Stewart (16), who reported that, irrespective of the GI of preexercise meals, preexercise CHO ingestion has no effect on muscle glycogen utilization. A number of factors could account for the discrepancy. In that study (16), subjects ingested 1 g CHO/kg body mass of HGI and LGI foods (instant mashed potato vs. lentils, respectively) 45 min before 2 h of cycling at 70% \( \dot{V}_{\text{O}_2} \text{max} \). In contrast to the present study, such a CHO load may not be sufficient to elicit large enough metabolic perturbation to be measured (as indicated by similar plasma glucose concentrations in the HGI and LGI trials and lower FFA levels during the first hour of exercise only, Ref. 16), especially in well-trained and insulin-sensitive subjects (33). However, the most important difference between the present study and that by Febbraio and Stewart (16) is the timing of postexercise muscle biopsy, which was obtained after 2 h of exercise, as opposed to 30 min in this study. Any increase in muscle glycogenolysis after preexercise ingestion of HGI CHO is likely to be more pronounced at the early stages of exercise (11), especially when muscle glucose uptake is not matched by glucose output from the liver (10).

In the present study, the lower glucagon-to-insulin ratio during exercise in the HGI than the LGI trial (Fig. 1D) suggests that hepatic glucose output was likely to be lower in the HGI trial. After 10 min of exercise, plasma glucose concentration in the HGI trial declined sharply to values lower than that at the onset of exercise in the LGI trial. Because the effect of insulin and contraction on muscle glucose uptake is synergistic, the sharp decline in plasma glucose concentration is probably a reflection of insufficient glucose provision in the face of increased muscle uptake in the HGI trial.

Interestingly, in a study using the same HGI but a different LGI meal (muesli instead of lentils) than their previous study (16), Febbraio et al. (17) observed no difference in the rate of muscle glycogen utilization after 20 min of exercise (contrast to their hypothesis that predicted that a HGI meal will cause muscle glycogen sparing) and a tendency \( P = 0.07 \) for higher muscle glycogen utilization after 120 min in the HGI trial compared with LGI and control. The rates of blood glucose disposal (assessed using stable isotopes) and total CHO oxidation were higher throughout exercise in the HGI compared with LGI (17). In contrast to the earlier study of Febbraio and Stewart (16), plasma glucose and FFA were lower in HGI than LGI throughout exercise. The authors suggested that the discrepancy in the results may be due to the extent to which the CHO ingested affects circulating glucose and FFA. Indeed, in a study that compared MGI with HGI meals providing a small amount of CHO (75 g) ingested 45 min before cycling at 70% \( \dot{V}_{\text{O}_2} \text{max} \) to exhaustion, plasma FFA levels and muscle glycogen use were not different between trials, whereas plasma glucose was lower after the first hour of exercise in the HGI trial (29). However, because subjects exercised longer in the LGI trials, it is difficult to assess whether the changes in muscle glycogen utilization occurred early in exercise. In contrast, in the present study, the increase in muscle glycogen utilization observed in the HGI trial was accompanied by suppressed plasma FFA levels and a sharp decline in plasma glucose early in exercise.

It is also possible the very high preexercise muscle glycogen concentrations (>600 mmol/kg dry mass) observed in the study by Febbraio et al. (17) may have acted to buffer any potential effect of the HGI meal on muscle glycogen utilization during the first 20 min of exercise, a period of time characterized by a very fast rate of muscle glycogen utilization (more than double the rate observed in our study). This, coupled with differences in the timing and amount of CHO ingested, may explain the discrepancy in the results between the present study and previous studies (16, 17).

During 30 min of exercise in the present study, total CHO oxidation during the two trials was 99 ± 7 g in the LGI trial and 112 ± 9 g in the HGI trial, and muscle glycogen utilization was 88 ± 15 mmol/kg dry mass in the LGI trial and 129 ± 16 mmol/kg dry mass in the HGI trial. Assuming that the dry mass of two legs is equal to ~5% of total body mass (26) and that all this muscle is involved in contraction during running, then the estimated amount of dry muscle mass engaged in exercise is ~3.7 kg. Compared with the HGI trial, 41 mmol/kg dry mass less muscle glycogen was used during the LGI trial, which translates to a total of 152 mmol or 27 g of CHO. Therefore, the lower CHO oxidation during the LGI trial could be entirely explained by the lower rate of muscle glycogen utilization.

As distinct from cycling, CHO ingestion during running (~70% \( \dot{V}_{\text{O}_2} \text{max} \)) delays the onset of fatigue by reducing the rate of muscle glycogen utilization (42). All being equal, it is reasonable to predict a greater endurance running capacity after

### Table 3. Muscle metabolite concentrations during the HGI and LGI trials

<table>
<thead>
<tr>
<th>Metabolite</th>
<th>LGI</th>
<th>Preexercise</th>
<th>Postexercise</th>
<th>HGI</th>
<th>Preexercise</th>
<th>Postexercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATP</td>
<td>25.7±0.6</td>
<td>25.9±0.8</td>
<td>25.8±0.8</td>
<td>25.9±1.1</td>
<td>26.0±0.6</td>
<td>25.2±0.7</td>
</tr>
<tr>
<td>PCr</td>
<td>83.2±2.1</td>
<td>83.5±2.1</td>
<td>69.9±2.0</td>
<td>84.2±1.9</td>
<td>83.6±2.4</td>
<td>71.9±2.0</td>
</tr>
<tr>
<td>G-6-P</td>
<td>0.9±0.2</td>
<td>0.8±0.1</td>
<td>2.0±0.3</td>
<td>0.7±0.1</td>
<td>0.8±0.1</td>
<td>2.2±0.3</td>
</tr>
<tr>
<td>Lactate</td>
<td>4.7±0.2</td>
<td>5.8±0.3</td>
<td>8.1±0.8*</td>
<td>5.0±0.3</td>
<td>5.7±0.6</td>
<td>10.8±1.6</td>
</tr>
</tbody>
</table>

Values are means ± SE given in mmol/kg dry mass. PCr, phosphocreatine; G-6-P, glucose-6-phosphate. *\( P < 0.05 \) from HGI.
the LGI than the HGI meal due to a reduction in muscle glycogen utilization during the earlier stages of exercise. However, our laboratory’s previous study showed no difference in endurance running capacity 3 h after ingestion of similar HGI and LGI meals (43), which is in agreement with some (16, 17, 38) but not all (15, 27, 29, 40) studies in the literature. In the present study, glycogen storage after the HGI meal within the 3-h postprandial period was 40 mmol/kg dry mass higher than after the LGI meal. This is similar to the difference in muscle glycogen utilization between the HGI and LGI trials (41 mmol/kg dry mass). Consequently, GLG concentration was similar in both trials at the end of 30 min of exercise. It is possible that muscle glycogen sparing may also occur after 30 min of exercise after ingestion of an LGI meal, possibly due to prolonged suppression of fat oxidation (48). In contrast, it is also possible that sparing of live glycogen may occur after ingestion of an HGI meal (as suggested by the lower glucagon-to-insulin ratio during exercise in the HGI than the LGI trial in the present study). The spared liver glycogen may become available late in exercise and offset any advantage conferred by an LGI meal or even lead to muscle glycogen sparing (17). Therefore, the extent to which the CHO-induced perturbations in postprandial and exercise glycemia and insulinemia affect the balance between the use of muscle and liver glycogen may explain the inconsistency in the results in the literature relating to the presence (15, 27, 29, 40) or absence (16, 17, 38, 43) of a performance advantage of LGI or MGI meals over HGI meals.

Glycogen exists in two forms in human muscle: one form is acid soluble (termed MG) and the other is acid insoluble (termed PG) (1, 25). In the present study, we measured the PG and MG fractions of muscle glycogen after PCA extraction of muscle samples (1, 25). Recent studies suggest that rates of accumulation and utilization of both forms are different, depending on GLG concentration and other metabolic factors (2, 3, 21). In the present study, the storage and depletion patterns of both pools were similar to that of GLG after the meals and exercise. However, five of the seven subjects demonstrated higher storage of PG than MG during the postprandial period. This is in agreement with the observations that the rate of PG synthesis is greater after ingestion of CHO during the first few hours of recovery from exercise than the MG synthesis rate (2) and that the glycogen synthase activity associated with PG has higher affinity for UDP-glucose than that linked to MG fraction (14). This is the first study in humans to show that the PG pool can change in response to CHO feeding without prior depleting exercise. During exercise, the rate of muscle PG utilization was higher than that of the MG pool in the LGI trial but did not reach significance in the HGI trial (P = 0.16). This is in contrast to findings from a previous study that showed that during cycling for 45 min at 70% V̇o2max net muscle glycogen breakdown was similar between the MG and PG pools (21). This discrepancy in the results may be due to the different modes of exercise employed and/or the fact that in the study by Graham et al. (21) subjects exercised a few hours after a light meal rather than a large CHO meal as used in the present study. This further supports the notion that the PG pool is more sensitive than MG in response to exercise, in particular when performed during the postprandial period.

In summary, our findings indicate that 1) a HGI meal results in greater muscle glycogen storage than a LGI meal in over-night fasted subjects with normal glycogen concentration; 2) the relative shift in substrate metabolism during 30 min of exercise from CHO to fat after ingestion of a LGI compared with a HGI meal was accompanied by a reduction in the rate of muscle glycogen utilization and not blood glucose oxidation; and 3) compared with MG, concentrations of PG appear to be more sensitive to feeding and exercise when total glycogen is not supercompensated or depleted. However, the greater fat oxidation and lower muscle glycogen utilization after the LGI meal may not always translate to a greater endurance capacity than after the HGI meal, as indicated by the inconsistency in the literature regarding the ergogenic effects of LGI or MGI meals over high GI meals. Further studies are required to investigate the relative roles of muscle and liver glycogen in energy metabolism and the onset of fatigue during exercise performed after ingestion of meals with different glycemic indexes.

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GRANTS

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