Original research

High versus low glycemic index 3-h recovery diets following glycogen-depleting exercise has no effect on subsequent 5-km cycling time trial performance

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ABSTRACT

Objectives: Some athletes train/compete multiple times in a single day and rapid restoration of muscle and hepatic glycogen stores is therefore important for athletic performance.

Design: Randomised, counterbalanced, crossover, single blinded study investigated the effects of low/high glycaemic index (GI) meals on the physiological responses to a 3-h recovery period and subsequent 5-km cycling time trial (TT).

Methods: Seven male cyclists completed glycogen-depleting exercise followed by a 3-h recovery period, when participants consumed either a high or low GI meal providing 2 g kg\(^{-1}\)BM of carbohydrate. Participants then performed a 5-km cycling TT. Blood samples were analysed for glucose insulin, free fatty acid (FFA) and triglyceride.

Results: There was no significant difference between the median (IQR) cycling TT time of 8.5 (3.0) min in the LGI condition and 8.4 (1.8) min in the HGI condition (\(p = 0.45\)). Serum insulin was significantly higher in the HGI condition throughout the 3-h recovery period (\(p = 0.025\)), FFA concentrations were higher in the HGI condition only at 30 min into recovery (\(p = 0.008\)). The respiratory exchange ratio (\(p = 0.028\)) and carbohydrate oxidation rate (\(p = 0.015\)) increased over time in the HGI condition, whereas the rate of fat oxidation demonstrated the opposite response (\(p = 0.001\)). No significant differences between conditions were observed for any physiological variables at the end of the 5-km TT.

Conclusions: Although the GI of the two meals indicated important metabolic differences during the recovery period, there was no evidence suggesting these differences influenced subsequent 5-km TT performance.

1. Introduction

Performance of high-intensity exercise is related to muscle glycogen availability at the onset.\(^1,2\) When athletes perform multiple training or compete within a single day, the provision of extra carbohydrate is essential to optimise muscle glycogen resynthesis between bouts. The restoration during the post-exercise period is therefore a major challenge for athletes who have busy training and/or competition bouts within schedules.\(^3\)

Muscle glycogen resynthesis following exercise depends on the timing of carbohydrate intake.\(^4\) The rate of muscle glycogen storage in glycogen-depleted muscle has been shown to be higher during the first 2-h period when the participants ingested carbohydrate immediately post-exercise, compared to when carbohydrate consumption was delayed for 2-h after the cessation of exercise.\(^5\) This is thought to be due to increased exercise-induced permeability of the sarcolemma to glucose and increases in muscle insulin sensitivity in early post-exercise recovery.\(^6\) Immediate post-exercise carbohydrate ingestion is most important when the interval between exercise sessions is short and the rate of refuelling must be maximised.\(^7\)

While the benefits of the timing of carbohydrate ingestion are well recognised, little attention has been given to the influences of different types of carbohydrates. Since glycogen storage is influenced by both insulin concentration and a rapid supply of glucose substrate, research has investigated the effects of the glycaemic index (GI) of carbohydrates on the restoration of muscle glycogen during post-exercise.\(^3,7\) Due to the large glycemc and insulineic responses following the ingestion of high GI (HGI) foods which favour muscle glycogen resynthesis, these types of foods are generally recommended to athletes during recovery. In contrast, consumption of low GI (LGI) foods has repeatedly been shown to produce lower glycemc and insulineic responses during rest in the postprandial period compared to HGI foods.\(^7\) However, research into the effects of the glycaemic index of foods on recovery and performance is equivocal.

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Kiens et al., found that HGI recovery meals were associated with greater muscle glycogen resynthesis and improved performance compared to the consumption of LGI meals. Burke et al., showed that HGI meals during a 24-h recovery period from prolonged heavy exercise resulted in higher muscle glycogen resynthesis than following the consumption of an iso-caloric LGI diet, although the effects on exercise performance were not investigated. According to these studies, HGI foods are desirable during the post-exercise period as they elicit greater substrate availability for glycogen resynthesis. In contrast, Stevenson et al., provided participants with LGI and HGI 24-h recovery diets prior to performing exercise to exhaustion. Their main finding was that the LGI diet resulted in a significantly increased endurance capacity the following day above that which was achieved following consumption of the HGI CHO recovery diet.

Research has investigated the ingestion of foods with different GI before exercise and during 24-h recovery following glycogen-depleting exercise. Little research has been conducted into the effects of the GI of meals on shorter recoveries between multiple bouts of exercise. Many of the previous studies provided single foods in the hour before exercise, which is unlikely to reflect normal dietary behaviour. The aim of this study was to investigate the influence of ingesting LGI and HGI recovery meals on the metabolic responses during a short-term (3–h) recovery period from glycogen-depleting exercise and subsequent 5-km cycling TT performance.

2. Methods

Seven cyclists participated in the study. Their mean (SD) age, height, body mass (BM), and incremental test maximal work rate \( \text{WR}_{\text{max}} \) were 29 (9) yr, 175.8 (8.9) cm, 75.2 (10.2) kg, and 310 (48) W, respectively. Participants cycled approximately 150 km per week ± 20 km per week. The study was approved by the Institutional Ethics Committee and participants provided written informed consent.

Each participant visited the laboratory on three separate occasions separated by 7 d. On visit, one participant performed a cycling test for maximal work rate. On visits two and three, participants completed glycogen-depleting exercise followed by a 3-h recovery period. At the start of the recovery period participants immediately consumed either a LGI or HGI carbohydrate meals providing 2 g–1 BM of carbohydrate. Participants then performed, in a randomised, counterbalanced, crossover, single blinded manner, a 5-km cycling TT. Participants followed the same diet and training schedule during the 2 d before visits two and three, verified by a food intake and training diary analysis. Participants refrained from strenuous exercise, caffeine and alcohol during the 24 h before each visit and signed a checklist to confirm they had followed all pre-test procedures. All testing took place at the same time of day, in the same laboratory under the same environmental conditions. Participants’ \( \text{WR}_{\text{max}} \) was determined using an electro-magnetically braked SRM© cycle ergometer (Schoberer Rad Messtechnik Konigskamp, Germany). Each participant cycled at 100 W for 5 min, followed by work rate increments of 50 W every 2.5 min. Work rate increments of 25 W were used once a heart rate of 150 bpm was exceeded. A cycling cadence between 75 and 90 rev min \(^{-1} \) was maintained throughout the test. The \( \text{WR}_{\text{max}} \) was calculated as follows: \( \text{WR}_{\text{max}} = \frac{\text{W}_{\text{out}} \times (t/150)}{\text{AW}} \), where \( \text{W}_{\text{out}} \) is the highest 2.5 min work rate stage that the subject completed, \( t \) is the number of seconds that the final uncompleted work rate stage was sustained, and \( \text{AW} \) is the work rate increment. The test was terminated when the subject could not maintain a cadence of at least 75 rev min \(^{-1} \), despite verbal encouragement. Pulmonary gas exchanges were determined throughout the test via standard open circuit spirometry (Quark b, Cosmed Srl, Rome, Italy).

Each participant reported to the laboratory 3-h post-prandial. Following a standardised warm-up consisting of cycling for 5 min at 50 W, the participants performed a glycogen-depleting exercise protocol on the SRM ergometer. This consisted of 2 min bouts of cycling at 90% \( \text{WR}_{\text{max}} \), interspersed with 2 min at 50% \( \text{WR}_{\text{max}} \). When participants were unable to maintain 90% \( \text{WR}_{\text{max}} \) for 2 min, the work rate was lowered to 80% and then 70% \( \text{WR}_{\text{max}} \). The exercise was terminated when 70% \( \text{WR}_{\text{max}} \) could not be maintained for 2 min. This protocol has been described in detail elsewhere and has been used extensively to induce muscle glycogen depletion. The glycogen-depleting exercise was followed by a 3-h recovery period. During the first 15 min of the recovery period, the participants ingested either a LGI or HGI carbohydrate meal and then sat quietly in the laboratory for the remainder of the 3-h. The HGI test meal (774 kcal, 140 g carbohydrate, 12 g fat, 31 g protein, GI = 72) and LGI test meal (672 kcal, 140 g carbohydrate, 13 g fat, 27 g protein, GI = 40) were similar in macronutrient content. The high GI meal consisted of Cornflakes (Kellogg Co., Manchester, UK) and semi-skimmed milk (GI: 72) whereas the low GI meal consisted of Muesli (Kelloggs Co., Manchester, UK) and semi-skimmed milk (GI: 40). The GI values of each test food were taken from Foster-Powell et al., and the GI of the total meal was calculated from the weighted means of the GI values for the component foods.

Venous samples were collected at 30, 120 and 180 min post-exercise for the determination of the concentrations of serum free fatty acids, triglycerides, and insulin. Capillary samples were collected every 30 min for the determination of blood glucose concentration. Gas exchange was determined for the calculation of the respiratory exchange ratio (RER), and estimated carbohydrate/fat oxidation rates were calculated using standard equations. Following recovery, participants cycled for 5 min at 50 W, then performed a 5-km TT on the SRM ergometer. Throughout the TT, pulmonary gas exchange was collected continuously. A venous and capillary blood sample was collected immediately following termination of the TT for measurement of the aforementioned blood variables. Participants were not aware of their performance times.

Fingertip blood samples were immediately analysed for glucose using a YSI 2700 Stat (Yellow Springs Instrument, Yellow Springs, USA). Venous blood was taken aseptically from the antecubital vein and dispensed into a serum separator, clot-activator Vacutette tube (Vacutette®, Greiner BIO-one, UK) and immediately centrifuged for 10 min at 13,000 × g. The serum was then stored at −80 °C and analysed for serum insulin (Immulite 1000 Insulin Kit and Immulite 1000 analyser; Siemens DPC, USA), free fatty acid (NEFA C, Wako Chemicals, kit number 999-75406, Germany) and triglyceride (Thermo Electron, #7500-023, Melbourne, Australia) concentrations according to manufacturers’ instructions.

Statistical analyses were conducted using IBM Statistics 19 (SPSS Inc., Chicago, IL). Time trial performance between the two conditions was analysed using the nonparametric sign test for two related samples, with the 95% confidence interval for the median difference estimated using the Hodges–Lehmann procedure. Two-way linear mixed models were used to explore the effects of the glycemic index and time on the insulin, glucose, free fatty acid, triglyceride, RER, carbohydrate oxidation, and fat oxidation responses during the 3-h recovery period following the glycogen-depleting exercise bout. Time was modelled as a continuous variable where a linear or quadratic response was evident and as a categorical variable otherwise. Paired t tests are reported for tests of the significance of linear slopes and quadratic effects, and omnibus F tests are reported for all other effects in the linear mixed models. The triglyceride data were log transformed to correct right skewed residuals. Post hoc pairwise comparisons with
Sidak adjusted $p$ values were performed as appropriate. The RER at the end of the 5-km time trial and the glucose, insulin, free fatty acid and triglyceride concentrations immediately post 5-km time trial were analysed using paired $t$ tests. Oxidation rates were not compared post TT because RER values at the end of the time trial were above 1.00 and the calculation of oxidation rates was therefore not appropriate. Two-tailed statistical significance was accepted as $p < 0.05$.

3. Results

Fig. 1 shows the 5-km TT times of each subject for the LGI and HGI conditions. There was no significant difference between the median (IQR) 5-km time trial time of 8.5 (3.0) min in the LGI condition and 8.4 (1.8) min in the HGI condition (median difference = 0.1 min, 95% CI = −0.05 to 1.0, $p = 0.45$).

The resting values of insulin, FFA and triglyceride concentrations were all within the normal range and were not different between the two trials (LGI/HGI). Serum insulin concentrations (Fig. 2) were significantly higher at 30-min in the HGI condition compared to the LGI condition during the 3-h recovery period (mean difference = 15.4 pmol l$^{-1}$, 95% CI = 2.1–28.7, $t = 2.4$, $p = 0.025$). Serum insulin significantly decreased over time in both conditions (mean slope = 0.14 pmol l$^{-1}$ per minute, 95% CI = 0.069–0.21, $t = 4.1$, $p < 0.001$); however, the difference in slopes between conditions was not statistically significant (mean slope difference = 0.095 pmol l$^{-1}$ per minute, 95% CI = −0.037 to 0.23, $t = 1.5$, $p = 0.15$). Post 5-km cycling time trial serum insulin concentration was not significantly different between conditions (mean difference = 7.8 pmol l$^{-1}$, 95% CI = −8.0 to 23.7, $t = 1.2$, $p = 0.27$).

Blood glucose, FFA and triglyceride concentrations during the 3-h recovery period and immediately after the 5-km time trial for the LGI and HGI conditions are shown in Fig. 3A–C. Although there was a transient increase in blood glucose concentration after the ingestion of the test meals, overall, blood glucose concentration showed a significant mean decrease of 0.0043 mmol l$^{-1}$.
Mean (SD) respiratory exchange ratio (RER), carbohydrate oxidation rate (CHOox) and fat oxidation rate (FAOx) during the first 150 min of the 3-h recovery period in the low glycemic index (LGI) and high glycemic index (HGI) conditions.

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>RER</th>
<th>CHOox (g min⁻¹)</th>
<th>FAOx (g min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>0.85 (0.06)</td>
<td>0.29 (0.12)</td>
<td>0.11 (0.05)</td>
</tr>
<tr>
<td>60</td>
<td>0.89 (0.04)</td>
<td>0.37 (0.09)</td>
<td>0.08 (0.03)</td>
</tr>
<tr>
<td>90</td>
<td>0.89 (0.04)</td>
<td>0.37 (0.08)</td>
<td>0.08 (0.03)</td>
</tr>
<tr>
<td>120</td>
<td>0.89 (0.02)</td>
<td>0.35 (0.05)</td>
<td>0.08 (0.02)</td>
</tr>
<tr>
<td>150</td>
<td>0.87 (0.04)</td>
<td>0.40 (0.06)</td>
<td>0.06 (0.02)</td>
</tr>
</tbody>
</table>

per min over the 3-h recovery period (95% CI = 0.0016–0.0071, t = 3.2, p = 0.002). The rate of decrease was greater in the HGI condition, however, the difference did not reach statistical significance (mean slope difference = 0.0049 mmol l⁻¹ per min, 95% CI = -0.00053 to 0.010, t = 1.8, p = 0.076). No significant main effects for condition was observed for blood glucose (mean difference = 0.29 mmol l⁻¹, 95% CI = -0.20 to 0.78, t = 1.2, p = 0.24). No significant main effects for condition (F = 0.6, p = 0.45) or time (F = 1.1, p = 0.35) were observed for the free fatty acid response during the 3-h recovery period, however, the condition × time interaction was significant (F = 4.9, p = 0.013). Mean free fatty acid concentration was significantly lower in the LGI condition at 30 min into recovery (mean difference = 0.062 mmol l⁻¹, 95% CI = 0.017–0.11, p = 0.008), but was not significantly different at 120 min (mean difference = 0.033 mmol l⁻¹, 95% CI = 0.010 to 0.076, p = 0.12) and 180 min (mean difference = 0 mmol l⁻¹, 95% CI = -0.043 to 0.043, p = 0.98). Regarding the triglyceride response during the 3-h recovery period, there were no significant effects for condition (F = 1.8, p = 0.19), time (F = 2.8, p = 0.08), or the condition × time interaction (F = 2.7, p = 0.096). Post-5-km cycling time trial blood glucose (mean difference = 0.1 mmol l⁻¹, 95% CI = -1.0 to 1.2 mmol l⁻¹, t = 0.2, p = 0.84), free fatty acids (mean difference = 0.0020 mmol l⁻¹, 95% CI = -0.076 to 0.080 mmol l⁻¹, t = 0.06, p = 0.95), and triglycerides (mean difference = 0.011 mmol l⁻¹, 95% CI = -0.30 to 0.75 mmol l⁻¹, t = 1.5, p = 0.19) were not significantly different between conditions.

Table 1 shows the RER, estimated CHO and fat oxidation rates for the first 150 min of the recovery period during the two conditions. The rate of CHO oxidation during the recovery demonstrated a quadratic trend over time, increasing over the first 90 min and 120 min in the LGI and HGI trials, respectively, and decreasing thereafter (t = 3.5, p = 0.001). A condition × time interaction was observed, where the increase in CHO oxidation rate over time was greater in the HGI condition (mean slope difference = 0.0011 g min⁻¹, 95% CI = 0.00022–0.0020 g min⁻¹, t = 2.5, p = 0.015). A similar response was observed for the RER in terms of the change over time and the relative difference between conditions. Fat oxidation showed an inverse quadratic trend compared to CHO oxidation, where the rate of fat oxidation decreased over the first 90 min and 120 min in the LGI and HGI conditions, respectively, and increased thereafter (t = 4.2, p < 0.001). The fat oxidation rate also demonstrated a condition × time interaction, with a greater overall decrease in the HGI condition (mean slope difference = -0.00052 g min⁻¹ per min, 95% CI = -0.00023–0.00080, t = 3.6, p = 0.001).

4. Discussion

This study aimed to investigate whether consuming an LGI or HGI meal immediately after a bout of glycogen-depleting exercise, altered metabolic responses during a 3-h recovery period and performance during a subsequent 5-km cycling TT. Typically, HGI meals, due to their large glycemic and insulinemic responses are associated with greater rates of muscle glycogen resynthesis and a subsequent improvement in performance. The present study, however, failed to confirm any difference in TT performance between LGI and HGI recovery meals, despite significant differences in metabolic responses during the preceding 3-h recovery period.

During the early postprandial period, there was no difference in substrate oxidation, which supports previous research. However over time, estimated CHO oxidation was greater with the HGI compared to the LGI meal, whereas fat oxidation exhibited the opposite response. Previous studies using foods with a GI similar to the HGI meal, providing 2–2.5 kg kg⁻¹ BM, have reported an increase in muscle glycogen content of between 10 and 40% over a 3–4 h postprandial period. Therefore, the increased CHO oxidation in the present study may provide some support for this speculation. Coyle et al. reported that compared with the fasted state, increased CHO oxidation during exercise paralleled the elevated pre-exercise muscle glycogen concentration after a HGI pre-exercise carbohydrate meal. It could be that, although not assessed in the present study, the HGI recovery meal was associated with greater rates of muscle glycogen synthesis as indicated by the increased carbohydrate oxidation rates. Additionally, it has previously been reported that when a LGI breakfast is consumed 3 h before exercise, less carbohydrate is stored as muscle glycogen than when a HGI breakfast is consumed. This 15% increase in muscle glycogen concentration was reported at the end of a 3-h postprandial period after the HGI breakfast. Only a small non-significant increase in muscle glycogen was reported after the LGI breakfast. This was accounted for primarily by the low glycemic and insulinemic responses to the LGI meal, followed by the slow digestion and absorption of the ingested foods. Similarly, in the present study, the LGI trial was associated with significantly higher fat oxidation rates throughout the recovery period. Fat oxidation rates may have been higher in the LGI trial as pre-exercise substrate availability is recognised as an important regulator of the patterns of fuel oxidation during exercise. Therefore, it could be that the higher rate of fat oxidation and consequently lower carbohydrate oxidation rate in the LGI trial might be a result of lower pre-exercise muscle glycogen concentrations compared to the HGI trial.

This increased estimated fat oxidation observed in the LGI trial may be beneficial for both recovery and subsequent performance. Although it is widely accepted that muscle glycogen is the primary fuel source during prolonged exercise, intramuscular triglyceride concentration is recognised as an important substrate source during prolonged exercise in healthy participants. Studies have reported a reduction in intramuscular triglyceride concentrations during prolonged exercise. Recent research reported that a high CHO diet consumed post-exercise inhibits post-exercise resynthesis of intramuscular triglycerides because increased FFA concentrations are required for the replenishment of intramuscular triglyceride content. Kiens and Richter reported a decrease in intramuscular triglyceride concentration during recovery from glycogen depleting exercise despite a large intake of carbohydrate. These studies reported that muscle glycogen repletion has such high metabolic priority during recovery that utilisation of lipids is essential to cover the energy expenditure in muscle. Therefore, it could be that in the present study the higher estimated fat oxidation and FFA concentrations during the postprandial period allowed for the resynthesis of some intramuscular triglycerides as well as the replenishment of muscle glycogen because of the carbohydrate intake.

Previously, improvements in performance have been reported when plasma FFA concentrations and fat oxidation have been elevated and investigators have suggested glycogen sparing as the...
reason for this improvement. Therefore, one may have expected to see an improvement in performance in this study. Additionally, given the observed differences in substrate utilisation and oxidation between the trials, differences in TT performance between the trials may have been expected. However, no difference in performance was observed between the LGI and HGI recovery diets.

Research investigating CHO intake on recovery and performance has typically employed performance measures lasting ≥ 1 h. However, in this study the 5-km TT employed may not have sufficiently challenged the glycogen stores to see a performance effect between diets. Possibly the higher estimated fat oxidation observed in the LGI trial may have spared muscle glycogen during exercise, which, had the TT been of a longer duration, might have resulted in an improved endurance performance by delaying depletion of muscle glycogen. The results of this study, indicate that if the recovery between exercise and training is short and subsequent exercise does not challenge glycogen stores, then the GI of the recovery meal has no impact on performance provided sufficient CHO is available during recovery from the last exercise bout.

5. Conclusion

Consumption of a HGI recovery meal was associated with greater estimated carbohydrate oxidation rates throughout the recovery period. The LGI recovery meal was associated with less estimated carbohydrate oxidation but greater estimated fat oxidation throughout recovery. This increased rate of fat oxidation may be a result of lower pre-exercise muscle glycogen compared to the HGI trial, but no difference in TT performance was observed between the two trials. This study indicates that following 3 h recovery, LGI and HGI recovery meals have greater estimated fat and carbohydrate oxidation rates, respectively, but if the subsequent exercise duration is short, the GI of the recovery diets has no influence on recovery and subsequent performance.

Practical implications

- When undertaking subsequent exercise the GI status of the meal is of little importance.
- Participants should continue to ingest food in the post exercise recovery period to replenish glycogen stores.
- LGI and HGI have different fat and carbohydrate oxidation rates and this may be a factor when considering post exercise meals.

Acknowledgements

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References