

# Preexercise carbohydrate ingestion, glucose kinetics, and muscle glycogen use: effect of the glycemic index

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**Febbraio, Mark A., Justin Keenan, Damien J. Angus, Shannon E. Campbell, and Andrew P. Garnham.** Preexercise carbohydrate ingestion, glucose kinetics, and muscle glycogen use: effect of the glycemic index. *J Appl Physiol* 89: 1845–1851, 2000.—Eight trained men cycled at 70% peak oxygen uptake for 120 min followed by a 30-min performance cycle after ingesting either a high-glycemic index (HGI), low-glycemic index (LGI), or placebo (Con) meal 30 min before exercise. Ingestion of HGI resulted in an elevated ( $P < 0.01$ ) blood glucose concentration compared with LGI and Con. At the onset of exercise, blood glucose fell ( $P < 0.05$ ) such that it was lower ( $P < 0.05$ ) in HGI compared with LGI and Con at 15 and 30 min during exercise. Plasma insulin concentration was higher ( $P < 0.01$ ) throughout the rest period after ingestion of HGI compared with LGI and Con. Plasma free fatty acid concentrations were lower ( $P < 0.05$ ) throughout exercise in HGI compared with LGI and Con. The rates of [6,6-<sup>2</sup>H]glucose appearance and disappearance were higher ( $P < 0.05$ ) at rest after ingestion and throughout exercise in HGI compared with LGI and Con. Carbohydrate oxidation was higher ( $P < 0.05$ ) throughout exercise, whereas glycogen use tended ( $P = 0.07$ ) to be higher in HGI compared with LGI and Con. No differences were observed in work output during the performance cycle when comparing the three trials. These results demonstrate that preexercise carbohydrate feeding with a HGI, but not a LGI, meal augments carbohydrate utilization during exercise but does not effect exercise performance.

glucose uptake; glucose production; preexercise feeding; glycogenolysis

IN CONTRAST WITH THE CONSISTENTLY observed beneficial effect of carbohydrate (CHO) ingestion during prolonged exercise, the efficacy of preexercise CHO ingestion is unclear. This practice has been demonstrated to increase (4, 14), decrease (20), or have no effect (6, 7, 8, 17) on muscle glycogen use and to increase (11, 17, 26), decrease (4, 9, 29), or have no effect (5, 7, 13, 27, 31) on exercise performance. The conflict in the literature is probably due to the timing of the meal ingestion, the quantity of CHO ingested, or the degree to which the

preexercise meal alters the plasma glucose and insulin response. Both CHO nutrients and foods, which contain a combination of nutrients, are oxidized and absorbed at different rates (16) and, therefore, have different effects on glycemia and insulinemia. Because the glycemic index (GI) provides a method to rank foods rich in CHO according to their glycemic response, the use of GI has become a recent interest area in preexercise nutrition (3, 7, 17, 27, 29–31). However, even when the preexercise meal is quantified according to the GI, the literature is inconsistent. Some (27, 29–31) but not all (7, 16, 17) studies observed the ingestion of a preexercise low-GI (LGI) meal to reduce CHO oxidation when compared with the ingestion of a high-GI (HGI) meal. Of note, only two of these studies (7, 17) sampled muscle tissue and found the rate of glycogenolysis to be unaffected by GI, which is not surprising because these same studies observed similar rates of CHO oxidation during exercise. Clearly, the hypothesis that preexercise ingestion of LGI food would be advantageous during exercise by reducing CHO oxidation requires further examination.

Whether muscle glycogen utilization is affected by preexercise ingestion of CHO may be related to glucose availability, uptake by tissues, and subsequent oxidation. It has been suggested that muscle glycogenolysis is not affected by preexercise CHO ingestion if the reduction in fat oxidation due to hyperinsulinemia is offset by the increase in muscle glucose uptake and subsequent oxidation (7). If, however, the increase in muscle glucose uptake is not as large as the insulin-mediated decline in fat oxidation, muscle glycogen utilization may be augmented. It has been demonstrated that preexercise glucose ingestion results in increased muscle glucose uptake during 60 min of exercise when compared with the ingestion of a sweet placebo (22). No studies, however, have examined glucose kinetics during exercise preceded by ingestion of foods on the basis of GI.

Previous studies that have examined the effect of preexercise CHO ingestion of differing GI on muscle

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glycogen use have sampled muscle before and after exercise (7, 17). It has been suggested that, after the ingestion of CHO, glycogenolysis is slowed early in exercise when muscle glucose uptake is augmented but is accelerated when hypoglycemia develops later during exercise (2). Whether the pattern glycogen utilization is biphasic with preexercise CHO ingestion has not been investigated.

The present study was undertaken, therefore, to further examine the influence of preexercise ingestion of CHO with differing GI on substrate metabolism. We conducted a comprehensive study by obtaining multiple biopsy samples and glucose kinetics data throughout exercise and sought to better understand the effect of preexercise CHO ingestion on exercise. We hypothesized that ingestion of the HGI food would result in a more perturbed glycemic and insulinemic response. We hypothesized that this would result in an augmented rate of glucose disappearance ( $R_d$ ) and oxidation and a reduced rate of glycogen use early in exercise but a reduced rate of glucose disappearance and oxidation and an augmented rate of glycogen use late in exercise compared with the ingestion of the LGI food.

## METHOD

**Subjects.** Eight endurance-trained men [ $26 \pm 6$  (SD) yr;  $72.9 \pm 5.2$  kg; peak oxygen uptake ( $\dot{V}O_{2\text{ peak}}$ ) =  $60.5 \pm 5$  ml·kg<sup>-1</sup>·min<sup>-1</sup>] 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 University of Melbourne Human Research Ethics Committee.

**$\dot{V}O_{2\text{ peak}}$  and experimental protocol.**  $\dot{V}O_{2\text{ peak}}$  was determined during incremental cycling exercise to volitional fatigue on an electrically braked cycle ergometer (Lode Groningen, The Netherlands). Expired air was directed via a Hans Rudolf valve and plastic tubing into Douglas bags. Oxygen and carbon dioxide content of the Douglas bags were analyzed by using Applied Electrochemistry S-3A/II and CD-3A gas analyzers (Ametek, Pittsburgh, PA), respectively, calibrated before each test with a commercially prepared gas mixture of known composition. Volume of expired gases were determined by using a gas meter (Parkinson-Cowan, Manchester, UK). The subjects reported to the laboratory on three subsequent occasions, separated by at least 7 days, after an overnight fast and having abstained from alcohol, caffeine, tobacco, and strenuous exercise for at least 24 h. In an attempt to minimize differences in resting muscle glycogen concentration, subjects were provided with food (~14 MJ, 80% CHO) for 24 h before each trial. On arrival in the laboratory, the subjects voided, and catheters (20 gauge, Terumo, Tokyo, Japan) were inserted into a vein in the antecubital space of each arm for blood sampling and tracer infusion. After a basal blood sample was collected, the catheter was flushed with 0.5 ml of saline containing heparin (10 IU/ml). A primed (3.3 mmol) continuous (~44  $\mu\text{mol}/\text{min}$ ) infusion of [6,6-<sup>2</sup>H]glucose (Cambridge Isotope Laboratories, Cambridge, MA) was then commenced and maintained during 2 h of rest, 30 min between ingestion of the test meal and exercise, and 120 min of exercise. After the 2-h rest period, which allowed for mixing of the isotope with endogenous glucose, the subjects were provided with one of three test meals. These were muesli (LGI), instant mashed potatoes (HGI), and diet jelly (Con). The muesli consisted of oats and

wheat with a GI of 52. It was prepared by Nestec (Nestlé Research Center, Lausanne, Switzerland). The latter two test meals were identical to those reported previously (7). The meals were provided in random order, and there were no order effects on any measured parameter when analyzed statistically ( $P > 0.05$ ). The amount of CHO ingested in LGI and HGI was equal to 1 g/kg body wt and provided 20.3 and 22.4 kJ/kg of energy, respectively. Each test meal was made up to a volume of 600 ml with distilled water. Subjects had 10 min to eat the test meal and rested for the next 30 min before they commenced cycling on the previously mentioned ergometer at a workload corresponding to 70%  $\dot{V}O_{2\text{ peak}}$  for 120 min. During exercise, subjects were provided 250 ml of distilled water every 15 min, air was circulated by use of an electric fan, and the laboratory was maintained at 20–22°C. At the completion of the submaximal exercise, subjects rested for 1 min before they commenced the performance cycle. Subjects were instructed to perform as much work as possible for 30 min. Work (kJ) was recorded at the completion of this period.

Heart rate, oxygen uptake ( $\dot{V}O_2$ ), and respiratory exchange ratio were measured at 15-min intervals during the submaximal cycle by using the previously mentioned metabolic system. Substrate oxidation was calculated from these parameters (10). In addition to the basal blood sample, further samples were obtained 10, 20, and 30 min postprandial and at 15-min intervals during the submaximal exercise. Muscle samples were obtained from the vastus lateralis by using the percutaneous needle-biopsy technique modified to include suction immediately before, at 20 min during, and after submaximal exercise in all trials. These samples were rapidly (<20 s) frozen in liquid nitrogen.

**Analytic techniques.** Ten milliliters of blood were collected at each sampling time, some of which was placed in a tube containing fluoride heparin and spun in a centrifuge. The plasma was stored at -80°C and later analyzed for plasma glucose and lactate by using an automated method (EML-105, Electrolyte Metabolite Laboratory, Radiometer, Copenhagen, Denmark) and insulin by radioimmunoassay (Incstar, Stillwater, MN). A further aliquot of blood was mixed in a tube containing lithium heparin and spun in a centrifuge. The resultant plasma was stored for measurement of [6,6-<sup>2</sup>H]glucose enrichment as previously described (12). Briefly, 500  $\mu\text{l}$  of plasma were mixed with 500  $\mu\text{l}$  of 0.3 M Ba(OH)<sub>2</sub> and 500  $\mu\text{l}$  of ZnSO<sub>4</sub> and spun. The supernatant was passed down an ion-exchange column, washed with distilled water, and dried. The samples were then resuspended with distilled water, placed in glass vials, dehydrated overnight, and derivatized with the addition of pyridine and acetic anhydride. The derivatized samples were then measured by using a gas chromatograph-mass spectrometer (5890 series 2 gas chromatograph, 5971 mass spectrometer detector, Hewlett Packard, Avondale, PA). Rate of glucose appearance ( $R_a$ ) and  $R_d$  were determined from changes in the percent enrichment in the plasma of [6,6-<sup>2</sup>H]glucose, calculated by using the one-pool non-steady-state model (28), assuming a pool fraction of 0.65 and estimating the apparent glucose space as 25% of body weight. The remaining blood was added to an aliquot of preservative consisting of EGTA and reduced glutathione in normal saline, mixed gently, and spun in a centrifuge. Plasma was decanted, placed in vials, and stored at -80°C. These samples were later analyzed for plasma free fatty acids (FFAs) by using an enzymatic colorimetric method (23). Muscle samples were freeze-dried and divided into two portions. One portion was extracted according to the procedure of Harris et al. (15) and analyzed for lactate by using a standard enzymatic, fluorometric technique (21). Muscle glycogen con-

centrations were determined on the second freeze-dried portion extracted, neutralized, and analyzed according to the procedure of Passonneau and Lauderdale (24).

**Statistical analyses.** The data from the three trials were compared by using two-factor (time and treatment) ANOVA with repeated measures. A one-way ANOVA was used to compare work output during the performance cycle. Newman-Keuls post hoc tests were used to locate differences when ANOVA revealed a significant interaction. A Statistica computer software program was used to compute these statistics. All comparative data are reported as means  $\pm$  SE.

## RESULTS

Neither  $\dot{V}O_2$  nor heart rate throughout submaximal exercise was different when comparing the three trials (data not shown).

Ingestion of the meal during HGI resulted in a higher ( $P < 0.01$ ) plasma glucose concentration at 10, 20, and 30 min post-ingestion compared with LGI and Con. At the onset of exercise, plasma glucose fell ( $P < 0.05$ ) in HGI, such that it was lower ( $P < 0.05$ ) when compared with LGI and Con at 15 and 30 min during exercise (Fig. 1). Plasma FFA concentration was lower ( $P < 0.05$ ) in HGI compared with both LGI and Con at 20 and 30 min post-ingestion and throughout the exercise period. Of note, no differences in plasma FFA were observed when comparing LGI with Con (Fig. 1). Plasma insulin concentration was higher ( $P < 0.05$ ) in HGI, 10, 20, and 30 min after the ingestion of the test meal, when compared with both LGI and Con. The concentration of this hormone was not different, however, when comparing the latter two trials (Fig. 1).

The total glucose  $R_a$  and  $R_d$  were elevated immediately after meal ingestion and throughout exercise in HGI compared with LGI and Con (Fig. 2). In addition, at the onset and throughout the first 90 min of exercise, glucose  $R_a$  was higher in LGI compared with Con (Fig. 2). There were no differences in glucose  $R_a$  when comparing these two trials subsequent to this time (Fig. 2). Similarly, glucose  $R_d$  was higher ( $P < 0.05$ ) between 30 and 75 min when comparing LGI with Con (Fig. 2).

Both muscle ( $P < 0.05$ ) and plasma lactate ( $P < 0.05$ ) were increased during submaximal exercise when compared with rest. There were, however, no differences ( $P > 0.3$ ) in the concentration of these metabolites during rest or submaximal exercise when comparing the three trials (Fig. 3). Muscle glycogen concentration was not statistically different at rest when comparing the three trials. However, there was a tendency ( $P = 0.07$  for the interaction between time and treatment) for glycogen to be lower at 120 min in HGI compared with LGI and Con (Fig. 4).

Both the rate and total amount of CHO oxidized were higher ( $P < 0.05$ ) during HGI compared with LGI and Con, but neither was different when comparing these latter trials (Fig. 5). Conversely, the total amount of fat oxidized tended ( $P = 0.08$ ) to be lower in HGI compared with LGI and Con. Because we assumed that glucose  $R_d$  was approximately equal to the amount of glucose oxidized, we were able to obtain an indirect measure of glucose oxidation and carbohydrate oxida-

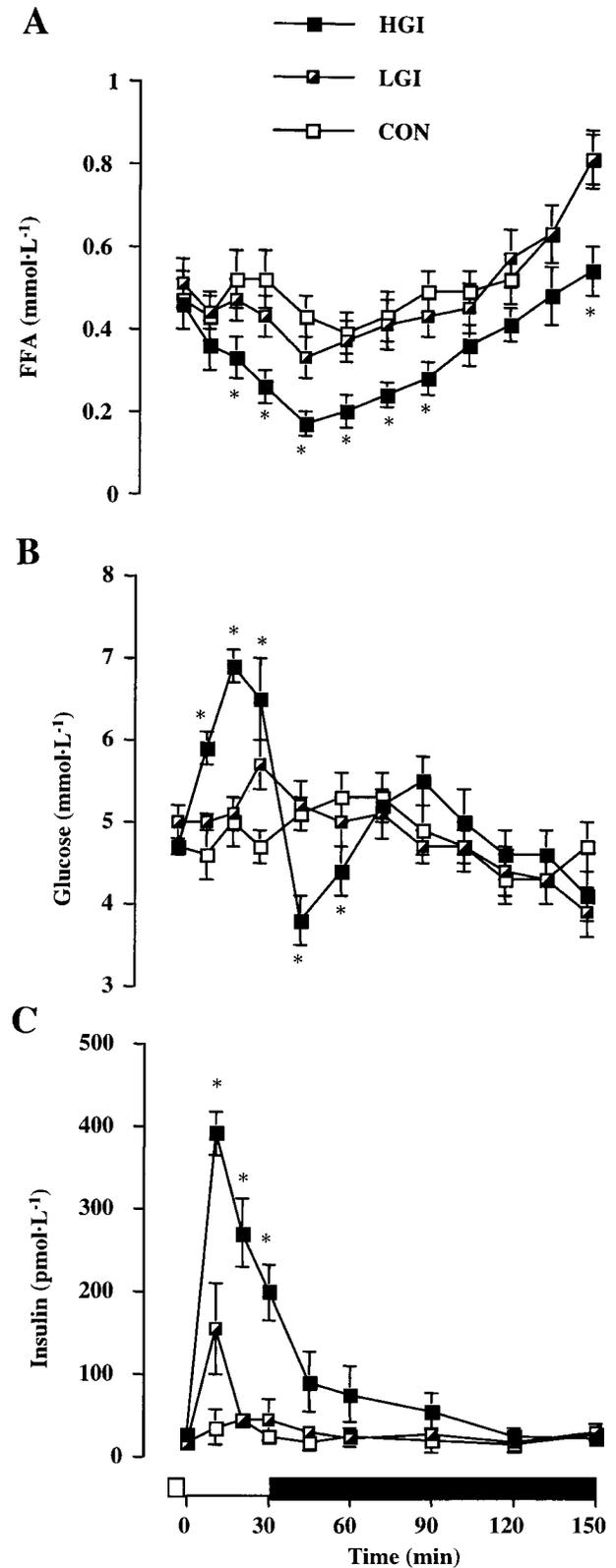


Fig. 1. Plasma free fatty acid (FFA; A), glucose (B), and insulin (C) concentrations at rest and during submaximal exercise with the ingestion of a high-glycemic index (HGI), low-glycemic index (LGI), and placebo (Con) meal. Values are means  $\pm$  SE;  $n = 8$  subjects. Open bar, ingestion; solid bar, exercise. \*Difference in HGI compared with LGI and Con,  $P < 0.05$ .

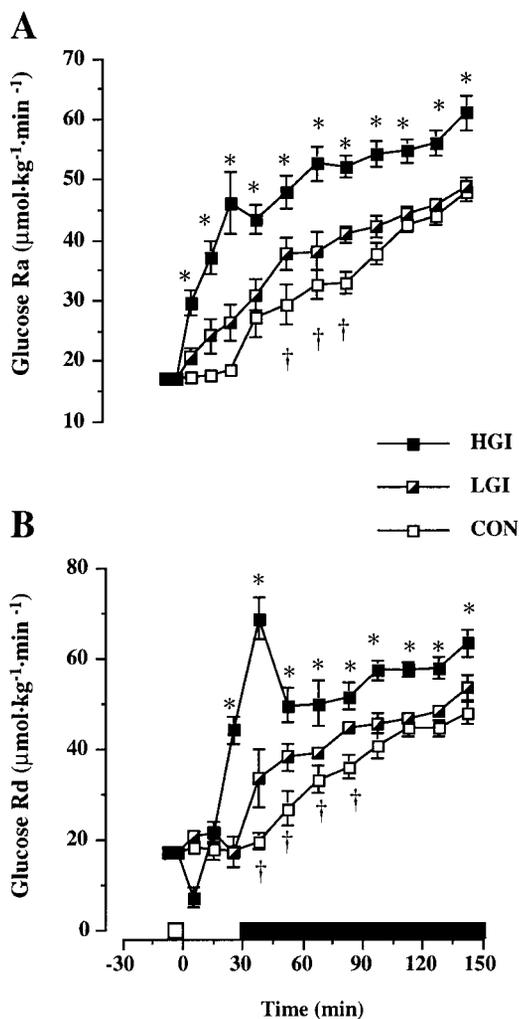


Fig. 2. Rates of plasma glucose appearance ( $R_a$ ; A) and disappearance ( $R_d$ ; B) at rest and during submaximal exercise with the ingestion of a HGI, LGI, and Con meal. Values are means  $\pm$  SE;  $n = 7$  subjects. Open bar, ingestion; solid bar, exercise \*Difference in HGI compared with LGI and Con,  $P < 0.05$ . †Difference in Con compared with LGI,  $P < 0.05$ .

tion derived from other sources (i.e., glycogen). These calculations demonstrated that the augmented CHO oxidation in HGI was largely due to an increased glucose oxidation, because this measure was higher ( $P < 0.05$ ) in HGI compared with LGI and Con. Of note, however, CHO oxidation derived from other sources also tended ( $P = 0.09$ ) to be higher in HGI compared with other trials (Fig. 5), with a  $P$  value consistent with our direct measure of muscle glycogenolysis. Although total CHO oxidation was not different when comparing LGI with Con, glucose oxidation was higher ( $P < 0.05$ ) in LGI (Fig. 5).

The total work performed during the performance cycle was not different when comparing the three trials (Fig. 6).

## DISCUSSION

The data from the present study indicate that pre-exercise CHO ingestion with different GI has altered

glycemic and insulinemic responses during exercise. The ingestion of HGI resulted in hyperinsulinemia, which increased glucose uptake and decreased FFA availability. This resulted in an augmented rate of CHO oxidation. In contrast, ingestion of LGI resulted in a more stable glycemic and insulinemic response. However, preexercise CHO ingestion irrespective of GI had no effect on exercise performance after 120 min of submaximal exercise.

It has been well established that the preexercise ingestion of a HGI CHO produces an immediate hyperglycemic response, followed by a rapid decline in plasma glucose concentration (4, 7, 14, 27, 31). The results from the present study support this observation. Whether a preexercise HGI CHO ingestion affects glycogen use and/or CHO oxidation during exercise is the subject of some controversy. The preexercise ingestion of such a CHO either augments (4, 14) or does not affect (7, 8, 17) muscle glycogenolysis. In addition, our

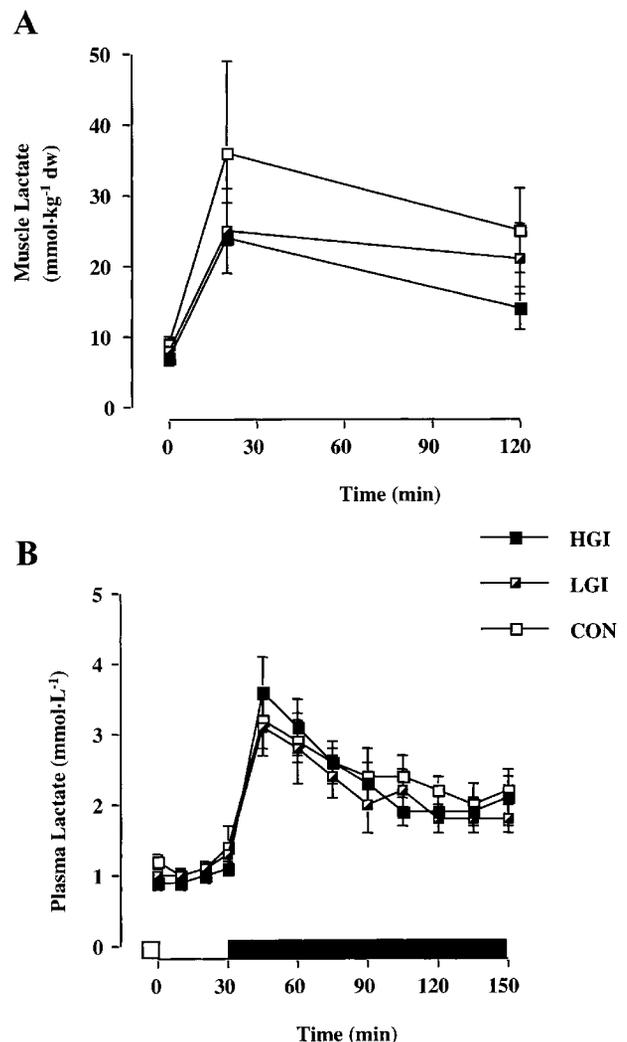


Fig. 3. Muscle (A) and plasma (B) lactate concentrations at rest and during submaximal exercise with the ingestion of a HGI, LGI, and Con meal. Values are means  $\pm$  SE;  $n = 8$  subjects for plasma, and  $n = 7$  subjects for muscle. dw, Dry wt. Open bar, ingestion, solid bar denotes exercise.

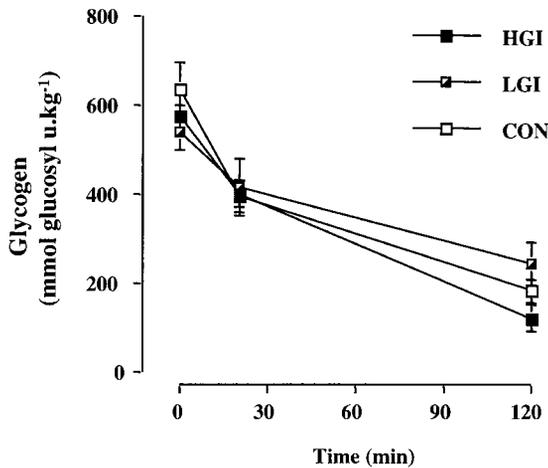


Fig. 4. Muscle glycogen before (0 min), during (20 min), and after (120 min) submaximal exercise with the ingestion of a HGI, LGI, and Con meal. Values are means  $\pm$  SE;  $n = 7$  subjects. Glucosyl u, glucosyl units.

laboratory previously observed CHO oxidation to be augmented (27) or unchanged (7) when comparing the preexercise ingestion of HGI with LGI food. In the latter study (27), it was speculated that the augmented CHO oxidation may be due to an increase in muscle glycogenolysis. In the present study, we measured both glucose kinetics and muscle glycogenolysis and were able to determine the source of the increase in CHO oxidation. Our present data support previous work demonstrating that the ingestion of HGI augments CHO oxidation and reduces fat oxidation during exercise (Fig. 5). Our data also suggest that an increase in glucose oxidation and possibly an increase in glycogenolysis may both contribute to increased CHO oxidation. If one assumes that glucose  $R_d$  is representative of glucose oxidation, it is possible to estimate the total amount of glucose oxidized throughout the exercise period. Such a calculation revealed that the total amount of glucose oxidized was higher in HGI compared with LGI and Con (Fig. 5). The rapid rise in glucose  $R_d$  in HGI was likely due to the synergistic effect of hyperglycemia, hyperinsulinemia, and contractile activity on muscle glucose uptake. It is important to note, however, that even when glucose and insulin levels returned to values similar to other trials, glucose  $R_d$  remained elevated (Fig. 2) and FFAs depressed (Fig. 1) in HGI. These data suggest that the persistent effect of hyperinsulinemia after insulin returns to normal levels during exercise. In addition to the augmented glucose oxidation, our data also suggest that ingestion of HGI could result in increased glycogen use. Although not statistically significant, both direct ( $P = 0.07$ ) and indirect ( $P = 0.09$ ) measures of muscle glycogen use tended to be higher in HGI. Although we hypothesized that glycogen use may be spared in the initial phase of exercise due to a rapid increase in glucose uptake, this was not the case (Fig. 4). In fact, in the present study, ingestion of HGI decreased fat utilization rather than muscle glycogen early in exercise.

Interestingly, ingestion of LGI did not affect the amount of CHO or fat utilized during exercise (Fig. 5). However, because glucose  $R_d$  was higher in LGI compared with Con at times during exercise, the calculated amount of glucose oxidized for the 120 min was greater ( $P < 0.05$ ) in LGI compared with Con. Although this suggests that other stores of CHO within the body were spared with the ingestion of LGI, results were not statistically significant.

It is apparent that the effect of preexercise CHO ingestion with different GI is a complex phenomenon because, as previously alluded to, the data from the present study support some (27) but not all (7) of previous work by our laboratory. Although, in the present study, the LGI meal had a GI of 52 compared with 29 in our previous studies, this fact is not likely to account for any discrepancies, because the glycemic, insulinemic, and lipolytic responses to LGI meal ingestion were very similar when comparing the present with our previous studies. The discrepancy in the lit-

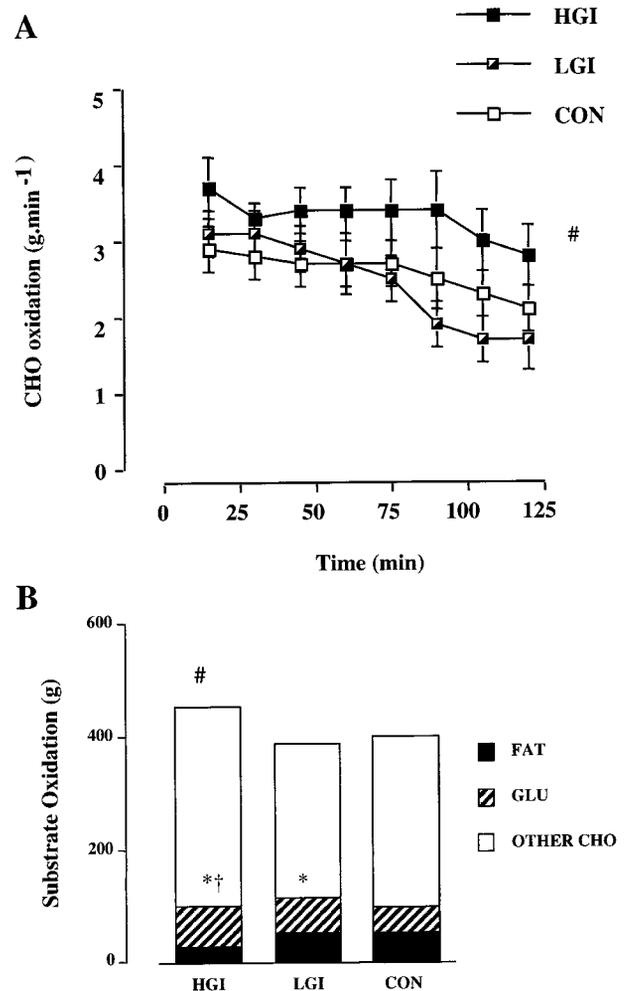


Fig. 5. Rate of carbohydrate (CHO) oxidation (A) and total substrate oxidation (B) with the ingestion of a HGI, LGI, and Con meal. Values are means  $\pm$  SE;  $n = 7$  subjects. Glu, glucose. #Difference in total CHO oxidation in HGI compared with LGI and Con,  $P < 0.05$ . \*Difference in glucose oxidation compared with Con,  $P < 0.05$ . †Difference in glucose oxidation compared with LGI,  $P < 0.05$ .

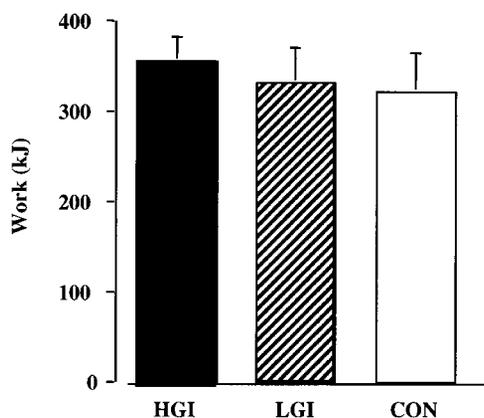


Fig. 6. Total work performed during a 30-min time trial cycle after submaximal exercise with the ingestion of a HGI, LGI, and Con meal. Values are means  $\pm$  SE;  $n = 8$  subjects.

erature appears, however, to be related to the extent to which the CHO ingested affects circulating glucose and FFA. During the studies of Costill et al. (4) and Hargreaves et al. (14), muscle glycogenolysis was augmented when glucose fell to lower than 3.5 mmol/l and remained low during exercise. In contrast, in our previous study in which glycogen use was not affected by preexercise carbohydrate ingestion (7), plasma glucose reached a nadir of 4.3 mmol/l before exercise with preexercise CHO ingestion. In addition, plasma glucose was not different during exercise when comparing the trials at any point during exercise (7). Although the subjects in the present study were similar in fitness to those in the previous study by our laboratory (7), and the HGI test meals were identical, the plasma glucose response to HGI ingestion was adversely affected during the present study. Apart from the effect of circulating glucose on glycogenolysis, it is apparent that FFA availability is also a critical determinant of CHO oxidation and glycogen use. It has been demonstrated that reduced FFA availability enhances muscle glycogenolysis during exercise (1). In the present study and in one of the previous studies from our laboratory (27), FFAs were lower in HGI compared with Con and LGI throughout exercise. As a consequence, in the present study glycogen use tended to be augmented in HGI, whereas CHO oxidation was higher in HGI relative to Con and LGI in both the present and previous (27) studies. In contrast, in the study of Febbraio and Stewart (7), FFAs were largely similar when comparing LGI with HGI, and concentrations of this metabolite were lower in both CHO trials when compared with a placebo in the second hour of exercise. Consequently, CHO oxidation was not different when comparing HGI with LGI. It is unclear why the FFA response would differ when comparing the previous (7) with the present study. It is possible, however, that in the previous study the subjects were more sensitive to small changes in insulin and, therefore, even the small rise in insulin with LGI ingestion was enough to blunt lipolysis to the same extent as a large rise in insulin associated with HGI ingestion. Indeed, it has been

previously demonstrated that in a relatively homogeneous group of cyclists insulin sensitivity varied in response to an oral glucose load, resulting in "rebound hypoglycemia" in some, but not all, subjects (19). Hence, the work from our laboratory and that of others (17, 19, 29–31) highlight the fact that the metabolic response to preexercise ingestion of carbohydrate with differing GI is complex and responses are largely individual.

Previous studies have shown plasma lactate to be either elevated (29) or unchanged (7, 8, 26, 27) when comparing the preexercise ingestion of HGI with a placebo. The data from the present study confirm these latter findings. Because  $\dot{V}O_2$  was not different when comparing the three trials, it is likely that the similar muscle and plasma lactate concentrations reflect a similar rate of anaerobic glycolysis.

A major finding of the present study was that preexercise CHO ingestion had no effect on endurance performance despite differences in CHO oxidation and GI of the ingested meals. These data support previous studies from our laboratory (7, 27) but are in contrast to those of others (17, 29), who observed an increase in exercise performance after the ingestion of a low- or moderate-GI meal. It is important to note, however, that in the studies that we have conducted performance was evaluated by measuring work in a given time period. In contrast, studies that have shown performance differences employed a time-to-exhaustion protocol (7, 27). A time-to-exhaustion measure of exercise performance is not practical because endurance events often, if not always, require athletes to complete a set amount of work in the quickest time possible. Therefore, the present results, along with those adopted in previous studies from our laboratory (7, 27), clearly demonstrate that the preexercise ingestion of 1 g/kg CHO within 60 min of the commencement of exercise does not affect exercise performance irrespective of the GI of the CHO. From another practical perspective, it is common for athletes to consume CHO during an endurance event, even if they have ingested a preevent meal. In these circumstances, the effect of the GI of the preevent meal is minimal (3).

There are circumstances in which preexercise CHO ingestion may affect performance. In the present study, although muscle glycogen content was reduced during the 120 min of submaximal exercise, it was not reduced to very low levels (Fig. 4). Although speculative, differences in performance may have been observed if the submaximal exercise period was extended. In addition, preexercise ingestion of either larger quantities of CHO (26) or ingestion of CHO 2–4 h before exercise (25) may also increase exercise performance. Therefore, the results from the present study cannot be generalized to all situations.

In summary, these data demonstrate that preexercise ingestion of CHO foods with different GI causes an alteration in glycemic, insulinemic, and lipolytic responses during exercise. As a result, the rate of CHO oxidation after preexercise CHO ingestion is influenced

by the GI, with the ingestion of LGI food providing a more stable metabolic response.

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