Effect of different carbohydrate drinks on whole body carbohydrate storage after exhaustive exercise

J. L. BOWTELL,1 K. GELLY,1 M. L. JACKMAN,1 A. PATEL,1 M. SIMEONI,2 AND M. J. RENNIE1
1Department of Anatomy and Physiology, University of Dundee, Dundee, Scotland, DD1 4HN; 2Department of Electronics and Informatics, University of Padua, 35131 Padua, Italy

Bowtell, J. L., K. Gelly, M. L. Jackman, A. Patel, M. Simeoni, and M. J. Rennie. Effect of different carbohydrate drinks on whole body carbohydrate storage after exhaustive exercise. J Appl Physiol 88: 1529–1536, 2000.—Seven untrained male subjects participated in a double-blind, crossover study conducted to determine the efficacy of different carbohydrate drinks in promoting carbohydrate storage in the whole body and skeletal muscle during recovery from exhaustive exercise. The postabsorptive subjects first completed an exercise protocol designed to deplete muscle fibers of glycogen, then consumed 330 ml of one of three carbohydrate drinks (18.5% glucose polymer, 18.5% sucrose, or 12% sucrose; wt/vol) and also received a primed constant infusion of [1-13C]glucose for 2 h. Nonoxidative glucose disposal (3.51 ± 0.28, 18.5% glucose polymer; 2.96 ± 0.32, 18.5% sucrose; 2.97 ± 0.16, 12% sucrose; all mmol · kg–1 · h–1) and storage of muscle glycogen (5.31 ± 1.11, 18.5% glucose polymer; 4.07 ± 1.05, 18.5% sucrose; 3.45 ± 0.85, 12% sucrose; all mmol · kg–1 · h–1; P < 0.05) were greater after consumption of the glucose polymer drink than after either sucrose drink. The results suggest that the consumption of a glucose polymer drink (containing 61 g carbohydrate) promotes a more rapid storage of carbohydrate in the whole body, skeletal muscle in particular, than an isonenergetic sucrose drink.

FATIGUE DURING ENDURANCE EXERCISE is associated with a depletion of muscle glycogen (5). These stores must be replaced during recovery in order for performance to be reproducible in a subsequent exercise bout. Glycogen synthesis may be limited by blood glucose concentration, glucose transport, and the activity of the enzymes involved in the pathway, glycogen synthase (EC 2.4.1.11) in particular (for review, see Ref. 23). After fatiguing exercise, conditions within skeletal muscle favor the synthesis of glycogen. Glycogen synthase activity is inversely proportional to glycogen concentration (47); hence, in the glycogen-depleted state, postexercise, skeletal muscle (46) and most likely hepatic glycogen synthase activity are elevated (36). Basal glucose transport within skeletal muscle occurs via GLUT-1; however, the capacity of skeletal muscle to take up glucose is variable due to alterations in the GLUT-4 content of the sarcolemmal membrane (for review see Ref. 21). There are thought to be one or more intracellular pools of GLUT-4 proteins, which are translocated to the sarcolemma in response to both increased insulin concentration (45) and prior exercise (26); these effects are additive (19). In the postexercise period, therefore, muscle membrane permeability to glucose is high, thus favoring the accretion of glycogen. However, if carbohydrate is not provided during recovery, glucose availability will limit glycogen synthesis because the rate of endogenous glucose production from gluconeogenic precursors such as alanine and glycerol is inadequate to support maximal rates of glycogen synthesis (38).

Ingestion of carbohydrate increases glycogen synthesis in two ways. The first is increased substrate availability through the increased blood glucose concentration, which results in an increased glucose uptake (35) due to mass action. Also, the resultant increase in systemic insulin concentration stimulates the translocation of GLUT-4 transporters from an intracellular pool to the sarcolemmal membrane (20). Second, insulin is also a potent activator of glycogen synthase and inhibitor of glycogen phosphorylase (13). The efficacy of a particular carbohydrate in promoting resynthesis of the body's carbohydrate stores is dependent on the insulin and glucose response to the carbohydrate load (18). This is a function of gastric emptying and intestinal absorption rates as well as the insulinogetic potential of the carbohydrate, as indicated by the glycemic index of a carbohydrate (8). Leese et al. (28) found that the gastric emptying times for glucose polymer and sucrose drinks (18.5%, wt/vol), consumed after 1 h walking uphill on a treadmill at 70% maximum O2 uptake (V˙O2max), were not different. Sucrose is a disaccharide of glucose and fructose; because fructose is a weaker secretagogue of insulin (10), it might be expected that glycogen resynthesis rates would be greater after consumption of a glucose polymer rather than a sucrose drink.

The restoration of muscle rather than liver glycogen stores is prioritized during the early phase of recovery from exhaustive exercise (31). There is some evidence that fructose can be taken up into human skeletal muscle, where 70–80% is converted into lactate (49). However, fructose is largely metabolized in the liver, where it is converted to a substrate metabolically available to the rest of the body, either glucose or lactate (15). Consumption of fructose or sucrose during recovery may therefore increase the supply of glyco-
The use of [1-13C]glucose as a tracer.

**METHODS**

The aims of this study were twofold: first, to determine the efficacy of glucose polymer and sucrose drinks in promoting carbohydrate storage in the whole body and in skeletal muscle during the early phase of recovery from exhaustive exercise, and second, to attempt to determine the rate of muscle glycogen synthesis, rather than net storage, by measuring the incorporation of plasma [13C]glucose into muscle glycogen with the use of [1-13C]glucose as a tracer.

### METHODS

Seven men [mass 77.0 ± 1.6 kg, height 1.76 ± 0.03 m, body fat 14.6 ± 3.1%, and maximal oxygen uptake (VO2max) 42.3 ± 1.5 ml·kg⁻¹·min⁻¹] participated in three trials, receiving one of three different carbohydrate drinks by systematic rotation: 18.5% (wt/vol) glucose polymer solution (containing glucose, maltose, maltotriose, tetrasaccharide, pentasaccharide, and higher sugars), 18.5% sucrose, or 12% sucrose (all SmithKline Beecham Consumer Healthcare, Gloucestershire, UK). The trials were separated by at least 1 mo to allow subjects to recover fully from the biopsy procedures. Their VO2max values were determined in the week preceding each trial to control for any changes that may have occurred in the intervening month. An incremental exercise test on an electrically braked cycle ergometer (Monark), using the criteria of Taylor et al. (42), was adopted for the measurement of VO2max. Respiratory gases were measured by on-line gas analysis (Morgan Benchmate Exercise Test, PK Morgan, Gillingham, Kent, UK). The study was approved by the Tayside Ethics Committee, and all subjects gave their informed consent.

Subjects were instructed to refrain from exercise and consumption of alcohol on the day preceding each trial. After an overnight fast, the subjects completed an exercise protocol, validated by Vos et al. (44), designed to deplete both type I and type II muscle fibers of glycogen. Subjects cycled on the ergometer at 70% VO2max for 30 min; the work load was then doubled and they completed six 1-min bursts of activity separated by 2-min rests. This burst of high-intensity activity was designed to deplete type II muscle fibers of glycogen. Finally, they cycled for a further 45 min at 70% VO2max to ensure both a further depletion of glycogen in type I fibers and a low plasma lactate concentration at the end of exercise, to minimize glycogen resynthesis from lactate during recovery. Subjects were allowed to consume water ad libitum during the exercise period.

Immediately after exercise, cannulas were inserted into the antecubital vein of each forearm, one for blood sampling and the other for the infusion of [1-13C]glucose (MassTrace, Somerville, MA). Two basal blood samples were then taken, and expired air for [13CO2] analysis was collected into a 1-liter bag from which two aliquots were removed and stored in 10 ml evacuated glass tubes (Exetainers, Europa Scientific, Crewe, UK). A muscle biopsy was taken from vastus lateralis by conchoitome forceps under local anesthetic (1%, wt/vol, lignocaine, Phoenix Pharmaceuticals, Gloucester, UK) within 15 min of the end of exercise.

Once these postexercise samples had been collected, a 2-h, primed, constant [1-13C]glucose infusion was begun, always within 20 min of the cessation of exercise. The plasma glucose pool was primed with a 0.050 mmol·kg⁻¹ bolus of [1-13C]glucose to facilitate rapid attainment of a steady state of plasma glucose [13C]enrichment. The [1-13C]glucose was then infused at a rate of 0.047 mmol·kg⁻¹·h⁻¹ for the first 30 min. The subject consumed a carbohydrate drink (330 ml; 18.5% glucose polymer, 18.5% sucrose, or 12% sucrose) within 2 min of the start of the infusion. The rate of [1-13C]glucose infusion was increased to 0.056 mmol·kg⁻¹·h⁻¹ for the final 90 min to minimize the changes in tracer-to-tracer ratio.

Samples of blood and expired air were collected every 15 min during the tracer glucose infusion. Second and third vastus lateralis muscle biopsies were taken 1 and 2 h after the start of the [1-13C]glucose infusion. Respiratory gas exchange was measured for 10 min every 30 min with a Deltradic metabolic monitor (Datex, Helsinki, Finland). [13CO2] enrichment in expired air samples was determined by using an Automatic Nitrogen and Carbon Analyzer (ANCA; Europa Scientific) coupled to a Tracermass isotope ratio mass spectrometer (Europa Scientific); results were expressed as %[13C]PDB-1. In comparison to the International Standard Pee Dee Belemnite-1 (PDB-1). The enrichment (expressed as moles percent excess) was then calculated from the isotope ratios by using the isotope ratio of the sample taken immediately after exercise but before tracer infusion as the "baseline" value.

Blood was stored on ice until the end of the infusion period and then centrifuged for 20 min at 2,500 rpm at 4°C. The resultant plasma was stored at −20°C until the analyses could be performed, except for the plasma to be analyzed for insulin (radio-immunoassay kit, ICN Pharmaceuticals, Thame, Oxfordshire, UK), which was stored at −70°C. Plasma was analyzed for glucose and lactate concentration with a YSI 2300 STAT plus analyzer (Yellow Springs, OH). Plasma [1-13C]glucose enrichment was determined as follows: 0.5 ml plasma was passed down a Cl-Dowex column, and the columns were washed with 3 ml distilled water, thus removing all negatively charged ions. Glucose oxidase (EC 1.1.3.4) was added to the eluate, and the mixture was incubated for 30 min at 37°C, thus converting the glucose to gluconic acid. The solution was passed down another Cl-Dowex column, and the column was then washed with 15 ml distilled water. The gluconate was eluted from the column by using 2 ml of 1 M hydrochloric acid. The eluant was dried down by warming to 40°C and using a nitrogen gas stream (Turbovap LV, Zymark). The residue was dissolved in 100 µl distilled water and transferred to tin capsules. The samples were freeze dried, then the capsules were crimped closed and then combusted in an ANCA. [13C] to [12C] isotope ratio of the resultant CO2 was determined by isotope ratio mass spectrometry and expressed as %[13C]PDB-1 in comparison to the International Standard (PDB-1). The [13C] enrichment was calculated using the isotope ratios as described previously, and the resultant values for CO2 were multiplied by 6 to obtain [13C] enrichment of the glucose molecule to correct for dilution by the hexose carbons. The tracer-to-tracer mass ratios were then calculated from these values (12).

The muscle tissue was frozen immediately in liquid nitrogen and stored at −70°C until the analyses for measurement of muscle glycogen concentration and [13C] enrichment could be performed. Muscle glycogen concentration was determined by a method previously described by Varnier et al. (43). In brief, 30–40 mg muscle tissue cleaned of blood and connective tissue was homogenized in 250 µl distilled water by using a Teflon glass Potter-Elvehjem homogenizer on ice. The homogenate was transferred to screw-cap-topped glass hydrolysis tubes containing 50 µl of 6 M hydrochloric acid and incubated in a boiling water bath for 2 h. After cooling, the mixture was
neutralized with 2 M potassium hydroxide and then centrifuged, and the supernatant was assayed for glucose (Sigma Chemical). Muscle ([13C]glycogen) enrichment was determined by a method also previously described by Varnier et al. (43). Muscle (100 mg) was washed in 0.9 M saline and then homogenized on ice in 1 ml 10% TCA by a Polytron homogenizer (Kinematica, Littau, Switzerland). The homogenate was added to 5 ml 95% ethanol, vortex mixed, and left at −20°C overnight. The solution was centrifuged at 10,000 rpm for 30 min, and the resultant precipitate was washed with 5 ml 95% ethanol and centrifuged on two further occasions. The precipitate was then dissolved in 100 μl distilled water, transferred to tin capsules, and freeze-dried. Finally, the capsules were combusted in an ANCA and the 13C/12C ratio of the resultant CO2 was determined by isotope ratio mass spectrometry. 13C enrichment of the glycosyl units of the glycogen molecule was calculated as described for plasma glucose 13C enrichment.

The rates of glucose appearance (Ra) and disappearance (Rd) were estimated by using Steele’s equations (41)

\[
Ra(t) = \frac{V_a(t) - V_s(t) \cdot \frac{dz}{dt}}{z(t)}
\]

\[
Rd(t) = Ra(t) - V_s(t) \cdot \frac{dg}{dt}
\]

where \(V_a\) is Steele’s volume, \(g(t)\) is glucose concentration, \(z(t)\) is tracer-to-tracee ratio, and \(V_a\) is tracer infusion rate. \(V_s\) was assumed to be equal to 130 ml/kg (9). Note that, to reduce the effect of model error on Ra and Rd estimations (9, 11), variations of tracer-to-tracee ratio were minimized by adopting a two-step tracer administration format.

Plasma glucose oxidation was calculated

\[
O = \frac{V_{CO2} \cdot ECO2}{Epg \cdot C}
\]

where \(V_{CO2}\) is carbon dioxide production (µmol·kg−1·h−1), \(ECO2\) is breath 13CO2 enrichment, \(Epg\) is plasma glucose 13C enrichment, and \(C\) is a correction factor for the retention of 13C within the bicarbonate pool. A mean correction factor (46%) was used. This was derived from the pooling of data from two of our studies, in which the retention of 13C label within the bicarbonate pool during recovery from exercise (7, 29) was determined. This value is considerably lower than the 81% figure frequently used to calculate substrate oxidation in postabsorptive subjects in the resting state. However, during the postexercise period, sequestration of bicarbonate carbon for various metabolic processes such as ureagenesis and gluconeogenesis is increased, thus reducing 13C label recovery. Nonoxidative glucose disposal was calculated as the difference between Rd and plasma glucose oxidation.

The average net rate of muscle glycogen storage was calculated as the difference between muscle glycogen concentration in the biopsies obtained after 2 h of recovery and immediately after exercise. Total glycogen storage in the exercised leg muscle over the 2 h of recovery was estimated from the biopsy data, on the basis that 40% of body weight is composed of skeletal muscle and that leg muscle contributes 50% of total skeletal muscle mass (i.e., 15.4 ± 0.3 kg leg muscle).

The rate of muscle glycogen synthesis was calculated by using the traditional equation employed to determine fractional protein synthesis rate (4), where product enrichment (muscle glycogen; [13C]glycogen) is divided by the enrichment of the precursor pool (plasma glucose; [13C]glucose) over the same time period (both given as moles percent excess) and multiplied by average concentration of the product ([glycogen]average) and divided by time

\[
\text{Glycogen synthesis} = \frac{[13C]\text{glycogen} \times [\text{glycogen}]_{\text{average}}}{[13C]\text{glucose} \times t}
\]

Data were analyzed by repeated-measures two-way (trial vs. time) ANOVA to establish the presence of a significant difference between trials, and then a post hoc Tukey’s test was used to locate the site of the difference.

RESULTS

All subjects completed the exercise protocol, although in some cases the resistance had to be reduced for the final 10 min. Plasma glucose was increased by consumption of all carbohydrate drinks (P < 0.001, Fig. 1), attaining peak concentrations at 30 min (both sucrose drinks) and 45 min (glucose polymer drink) after ingestion of the drinks, respectively. Plasma glucose concentration was significantly greater after consumption of the glucose polymer than after consumption of the 12% sucrose drink at 45, 60, and 75 min of recovery and than after the 18.5% sucrose drink at 60 and 75 min of recovery (P < 0.05). The area under the plasma glucose concentration curve was calculated to provide a measure of the plasma glucose response to the oral glucose load. During the first hour of recovery, the area under the curve was significantly higher after consumption of the glucose polymer (6.52 ± 0.33 mM·h, P < 0.005) and the 18.5% sucrose (6.26 ± 0.25 mM·h, P < 0.05) drinks than after the 12% sucrose drink (5.59 ± 0.22 mM·h). During the second hour of recovery, the area under the curve was significantly higher after ingestion of the glucose polymer (5.39 ± 0.30 mM·h) rather than the sucrose drinks (4.51 ± 0.14, 18.5% sucrose; 4.34 ± 0.17, 12% sucrose; both mM·h).

![Fig. 1. Plasma glucose concentration during recovery from exhaustive exercise, means ± SE (error bars). Significantly different from values at end of exercise: *P < 0.05, glucose polymer trial; **P < 0.05, 18.5% sucrose trial, and ***P < 0.05, 12% sucrose trial. Significantly different from glucose polymer trial values (*P < 0.05) and from 18.5% sucrose trial values (†P < 0.05). Number of symbols indicates level of significance: 1 symbol P < 0.05, 2 symbols P < 0.005, and 3 symbols P < 0.0005.](http://jap.physiology.org/1531)
Plasma lactate concentrations at the end of exercise were only slightly elevated above normal resting values; however, the baseline lactate concentration was higher in the trial where 12% (2.50 ± 0.40 mM) rather than 18.5% sucrose solution (1.92 ± 0.24 mM, P < 0.05) was consumed (Fig. 2). After ingestion of the sucrose drinks, there was an increase in plasma lactate concentration during recovery, which attained statistical significance in the 18.5% sucrose trial (P < 0.005). Plasma lactate concentration during the 18.5% sucrose trial was significantly higher than during the glucose polymer trial between 30 and 90 min of recovery and than during the 12% sucrose trial between 45 and 90 min (P < 0.05).

Plasma insulin was increased by the consumption of the 18.5% glucose polymer and 18.5% sucrose drinks (Fig. 3, P < 0.005). Plasma insulin concentration was significantly higher in the glucose polymer trial than in the 12% sucrose trial between 30 and 60 min of recovery (P < 0.0005) and than in the 18.5% sucrose trial at 60 min of recovery (P < 0.05). There was a tendency for plasma insulin concentration to be higher throughout the second hour of recovery in the glucose polymer trial than in the sucrose trials, but this did not attain statistical significance. The area under the plasma insulin concentration curve was higher in the glucose polymer trial (112.96 ± 37.46 µU·ml⁻¹·h⁻¹) than in the 12% sucrose trial (49.73 ± 11.06 µU·ml⁻¹·h⁻¹, P < 0.05) and the 18.5% sucrose trial (76.83 ± 20.78 µU·ml⁻¹·h⁻¹), but this difference was not statistically significant.

Rd changed over time (P < 0.0005, Table 1) and tended to be higher during the second hour of recovery in the glucose polymer than in the sucrose trials, but this did not attain statistical significance. Rd changed over time (P < 0.0005, Table 1) and tended to be higher during the second hour of recovery in the glucose polymer than in the sucrose trials, but this did not attain statistical significance.

There was no significant effect of the drink on plasma glucose oxidation; however, the pattern of change over time was different in the three trials (Table 1, trial × time interaction effect, P < 0.005). Nonoxidative glucose disposal changed over time (Fig. 4, P < 0.0005). The area under the nonoxidative glucose disposal curve was calculated to provide an index of whole-body carbohydrate storage during the first and second hours of recovery. The area under the curve was significantly higher during the second hour of recovery in the glucose polymer (4.05 ± 0.43 mmol/kg) than in the 18.5% (3.12 ± 0.36 mmol/kg, P < 0.05) sucrose and 12% sucrose (2.89 ± 0.19 mmol/kg, P < 0.01) trials. Muscle glycogen stores were depleted at the end of exercise in all trials (down to 8 mmol glucosyl units/kg wet wt) compared with normal resting concentrations (110–170 mmol glucosyl units/kg wet wt, Ref. 23). There was a significant increase in muscle glycogen concentration during recovery in all trials (P < 0.01, Fig. 5). However, the average rate of muscle glycogen storage during the 2 h of recovery was greater after consumption of the glucose polymer (5.31 ± 1.11 mmol·kg⁻¹·h⁻¹) than after consumption of either of the sucrose drinks (4.07 ± 1.05 and 3.45 ± 0.85 mmol·kg⁻¹·h⁻¹, P < 0.05). The calculated rate of muscle glycogen synthesis was greater after consumption of the glucose polymer (3.56 ± 0.71 mmol·kg⁻¹·h⁻¹) than after consumption of either of the sucrose drinks (1.55 ± 0.35 and 1.55 ± 0.40 mmol·kg⁻¹·h⁻¹, P < 0.005). However, the observed rate of muscle glycogen storage was greater than the calculated rate of muscle glycogen synthesis for all trials (P < 0.05).

DISCUSSION

There was a significant resynthesis of muscle glycogen stores during the 2-h recovery period in all three trials. The process of glycogen synthesis is dependent on the availability of glycogenic substrate and the activity of the enzymes involved in glycogen synthesis,
which include hexokinase (EC 2.7.1.1) and glycogen synthase. Prior exercise enhances skeletal muscle glucose transport because of the translocation of GLUT-4 transporters from an intracellular pool to the sarcolemmal membrane (17). The capacity for skeletal muscle to extract blood glucose will thus be increased, and the glucose will tend to be directed toward glycogen synthesis in two ways, first by increasing GLUT-4 translocation (25), which will facilitate increased muscle glycogen synthesis because glycogen synthase is activated during recovery due to the low intramuscular glycogen concentration (47). These conditions favoring the resynthesis of glycogen can be exploited by the provision of carbohydrate supplements. The resultant increase in glucose availability and the insulin response to the glucose load would tend to stimulate a further increase in the GLUT-4 content of the sarcolemmal membrane (20). McCoy et al. (32) recently demonstrated that there is a direct correlation between the rate of glycogen storage during recovery and total muscle GLUT-4 protein content.

The storage of muscle glycogen was higher during recovery after consumption of the glucose polymer drink than after either of the sucrose drinks. Doyle et al. (18), using multiple regression analysis, found that 94% of the variance in glycogen synthesis rates in the literature could be attributed to variation in plasma glucose and insulin concentrations. In the studies reported in this paper, the glucose response to the carbohydrate supplements was lower in the 12% sucrose trial than in the other two trials during the first hour of recovery. During the second hour of recovery, the plasma glucose response was higher in the glucose polymer trial than in either sucrose trial. The availability of glucose for incorporation into hepatic and skeletal muscle stores was therefore greater in the glucose polymer trial. The insulin response to the carbohydrate load was also higher in the glucose polymer trial than in either sucrose trial, but the difference only attained statistical significance relative to the 12% sucrose trial. This increased insulin response will stimulate glycogen synthesis in two ways, first by increasing GLUT-4 translocation (25), which will facilitate increased muscle glycogen synthesis during recovery from exhaustive exercise. 

Table 1. Plasma glucose kinetics during recovery from exhaustive exercise

<table>
<thead>
<tr>
<th>Time, min</th>
<th>7.5</th>
<th>22.5</th>
<th>37.5</th>
<th>52.5</th>
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<th>82.5</th>
<th>97.5</th>
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<tr>
<td>GP</td>
<td>2.28 ± 0.07</td>
<td>3.42 ± 0.13</td>
<td>4.14 ± 0.19</td>
<td>4.42 ± 0.59</td>
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<td>Rd, mmol·kg⁻¹·h⁻¹</td>
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<td>GP</td>
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<td>O, mmol·kg⁻¹·h⁻¹</td>
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<tr>
<td>GP</td>
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<tr>
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<td>19.6 ± 1.5</td>
<td>19.0 ± 1.1</td>
<td>17.2 ± 2.9</td>
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Values are means ± SE. Ra, rate of appearance; Rd, rate of disappearance; O, plasma glucose oxidation; Epg, plasma glucose ¹³C enrichment; GP, glucose polymer drink; SH, 18.5% sucrose drink; SL, 12% sucrose drink. Values are significantly different from 7.5-min value (P < 0.05, glucose polymer trial; P < 0.005, sucrose trials); significantly different from 67.5-min value (P < 0.05); significantly different from 30-min value (P < 0.05); and significantly different from glucose polymer trial values (P < 0.05).
glucose uptake and thus glycogenic substrate availability, and second by activating glycogen synthase (13).

Fructose is less insulinogenic than glucose; the lower insulin response to the sucrose than to the glucose polymer drinks may be due to the fructose component of sucrose or may be related to a more rapid gastric emptying of the glucose polymer drink. The former explanation is the most feasible, because the calculated gastric emptying times for 330 ml of 18.5% (wt/vol) glucose polymer and sucrose drinks were 69.8 ± 2.9 and 66.5 ± 2.5 min respectively (28).

The glucose units supplied by the glucose polymer drink (358 mmol) could provide 73% of those stored in the whole body during the 2-h recovery period (calculated from nonoxidative glucose disposal). However, in the sucrose drink trials, only 42% (18.5% sucrose drink, 178 mmol) and 28% (12% sucrose drink, 116 mmol) of the glucose units stored in the whole body could directly be provided by the drink. This suggests that the availability of glucose units was higher after consumption of the glucose polymer drinks than after consumption of the sucrose drinks when subjects were more dependent on endogenous glycogenic substrate supplies and the conversion of fructose to a metabolically available substrate for skeletal muscle. There was a significant increase in plasma lactate in the recovery period after consumption of the sucrose drinks, especially the 18.5% sucrose drink, which probably reflects the hepatic export of lactate formed from the fructose portion of the sucrose disaccharide and the production of lactate from fructose in skeletal muscle (49). Lactate, glycerol, and glucogenic amino acids such as alanine, as well as glucose, can be used for hepatic glycogen synthesis (27, 40). This "indirect" pathway can account for up to 50% of hepatic glycogen synthesis in postabsorptive people (39). Lactate is a glycogenic substrate for skeletal muscle, especially in type II fibers (33). Astrand et al. (3) suggest that, after exercise, 50% of the lactate pool will be used for glycogen resynthesis, the remainder being oxidized. The increased muscle glycogen storage after consumption of the glucose polymer rather than sucrose drinks (61 g CHO) may, therefore, simply reflect the greater metabolic availability of the ingested carbohydrate.

Blom et al. (6) examined the effect of supplementation with different carbohydrate types on glycogen resynthesis after exhaustive exercise. Subjects received 0.7 g of glucose or sucrose per kilogram body weight every 2 h during 6 h of recovery from exhaustive cycling exercise. There was no difference between trials in the rate of muscle glycogen storage despite greater plasma glucose (6.31 ± 0.64 vs 5.23 ± 0.11, mM) and insulin (21 ± 3 vs 13 ± 3, μU/ml) concentrations during the glucose than during the sucrose trials, respectively. The muscle glycogen storage rates achieved by subjects in the first 2 h of recovery tended to be higher in the Blom et al. study than in the present study (7.5 ± 1.0 mmol·kg wet wt⁻¹·h⁻¹ after consumption of 0.7 g glucose/kg (Ref. 6), compared with only 5.3 ± 1.1 mmol·kg wet wt⁻¹·h⁻¹ after consumption of 61 g (0.8 g/kg body wt) glucose polymer). Our data are comparable to those of Ivy et al. (24): subjects consumed 1.5 g of glucose per kilogram body weight after exhaustive cycling exercise, and muscle glycogen resynthesis rate was 5.2 ± 0.9 mmol·kg wet wt⁻¹·h⁻¹. The discrepancy between the findings of the present study and that of Blom et al. (6) may be related to the carbohydrate concentration of the drinks. In the present study, the glucose polymer and sucrose were provided as an 18.5% solution, whereas in the Blom et al. study a 30% solution was consumed. Mitchell et al. (34) found that consumption of a more concentrated CHO solution enhanced CHO delivery, despite impaired gastric emptying and fluid replacement. It is likely, therefore, that the carbohydrate was absorbed into the blood stream more rapidly during the Blom et al. study (36). Thus the rate of delivery of glucose units derived from the sucrose disaccharide after consumption of the sucrose drink may also have been sufficient to achieve maximal rates of skeletal muscle glucose uptake and glycogen synthesis, and thus no difference was observed between glucose and sucrose trials. This is consistent with our hypothesis that the differences between trials in the present study were related to the greater metabolic availability of the ingested glucose polymer.

The exogenous glucose polymer has four possible fates: 1) oxidation, 2) storage as glycogen, 3) storage as fat after glycolysis to acetyl-CoA, or 4) formation of alpha-glycerophosphate and storage as glycerol in triglyceride. The first two and the fourth are likely to be the most important quantitatively in adult human beings (for review, see Ref. 22). Thus we have calculated whole body net carbohydrate storage as Rd minus oxidation. The nonoxidative disposal of glucose could account for 92% of Rd in all trials. Maelhum et al. (31) suggested that the resynthesis of muscle rather than hepatic glycogen was prioritized during the early phase of recovery from exercise. During the sucrose drink trials, the supply of glycogenic substrate to skeletal muscle appears to be more dependent on the conversion of the fructose moiety of the sucrose dimer and on gluconeogenesis from endogenous precursors such as lactate and alanine. One might therefore expect a redirection of carbohydrate storage away from the exercised muscle and toward the liver in the sucrose drink trials. Conlee et al. (14) investigated the effect of glucose or fructose feeding on hepatic and skeletal muscle glycogen resynthesis after exhaustive swim exercise in rats. Hepatic glycogen resynthesis was similar for both feeding regimes; however, fructose was a poor substrate for skeletal muscle glycogen resynthesis. In the present study, total muscle glycogen storage in the exercised leg muscle over the 2 h of recovery was estimated on the basis that 40% of body weight comprises skeletal muscle and that leg muscle contributes 50% of total skeletal muscle mass (i.e., 15.4 ± 0.3 kg leg muscle). It appears that there was no difference between trials in the compartmentalization of carbohydrate storage between the exercised muscle and the rest of the body; 21–28% of whole body carbohydrate storage can be attributed to glycogen storage in the exercised muscles, irrespective of the exogenous carbo-
hydrate source. In reality, total glycogen stored in muscle was probably greater than this (exercised + nonexercised); nonetheless, this estimation does not appear to support the hypothesis that skeletal muscle glycogen synthesis is prioritized during the early stages of recovery, because less than one-third of total carbohydrate storage can be attributed to muscle glycogen synthesis.

The calculated rate of muscle glycogen synthesis was greater after consumption of the glucose polymer than after consumption of an isoenergetic sucrose drink, this being qualitatively similar to the muscle glycogen storage data. However, the calculated rate of glycogen synthesis was less than the observed rate of glycogen storage. This brings into question the validity of the model, because even assuming that there was no glycogen breakdown during this period, synthesis must at least match the rate of storage. There are a number of factors that may contribute to the inadequacy of the model.

First, the assumption of plasma glucose as the immediate precursor to glycogen is problematic. Muscle glucose-6-phosphate (G-6-P) would have been a more biochemically accurate choice of precursor; however, we were not able to reproducibly isolate G-6-P from all other carbon sources. It is likely that G-6-P labeling was lower than the labeling of plasma glucose because of its dilution from glycogenic substrates other than glucose, e.g., pyruvate, lactate, and possibly alanine (33). This would lead to an underestimation of the glycogen synthesis rate.

Secondly, Adamo and Graham (1) have established that, as in rodent skeletal muscle (30), there are two pools of glycogen in human skeletal muscle: macroglycogen, termed “classic” glycogen, which is acid soluble, and proglycogen, which has a smaller molecular weight and high protein content and is acid insoluble. During the first 4 h of recovery after exhaustive exercise, Adamo et al. (2) found that there was no change in macroglycogen concentration, but that proglycogen synthesis was rapid and highly sensitive to carbohydrate availability. In the present study, an acid extraction method was used to isolate muscle glycogen carbon before combustion on the ANCA to determine 13C enrichment. It is therefore likely that the data obtained reflect rates of synthesis only for macroglycogen, which, during the immediate postexercise period, is slower than proglycogen synthesis. The underestimation of glycogen synthesis rate by the present method presumably reflects both the use of plasma glucose as the glycogen precursor in the kinetic model and the fact that only macroglycogen synthesis rate was determined. At present, the proposed method is not a viable means of measuring the rate of glycogen synthesis.

In conclusion, consumption of 61 g glucose polymer drink after exhaustive exercise promoted a more rapid storage of carbohydrate during the first 2 h of recovery than did consumption of an isoenergetic sucrose drink, both in the whole body and in skeletal muscle. This may simply be a consequence of the increased availability of glycogenic substrate after consumption of the glucose polymer drink, as indicated by the greater increase in plasma glucose and insulin concentrations and whole body glucose flux than after consumption of the sucrose drinks. This explanation is supported by the finding that 21–28% nonoxidative glucose disposal can be accounted for by carbohydrate storage within the exercised muscle, irrespective of nutritional intervention.

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Address for reprint requests and other correspondence: J. L. Bowtell, Sport and Exercise Science Research Centre, School of Applied Science, South Bank Univ., 103 Borough Rd., London SE1 0AA, United Kingdom (E-mail: bowtelljl@sbu.ac.uk).

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REFERENCES


17. Douen AG, Ramial T, Rastogi S, Bilan PJ, Cartee GD, Vrankic M, Holloszy JO, and Klip A. Exercise induces recruit-
GLYCOGEN POSTEXERCISE: EFFECT OF CARBOHYDRATE TYPE


