

# Glucose Polymer Molecular Weight Does Not Affect Exogenous Carbohydrate Oxidation

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## ABSTRACT

ROWLANDS, D. S., G. A. WALLIS, C. SHAW, R. L. P. G. JENTJENS, A. E. JEUKENDRUP. Glucose Polymer Molecular Weight Does Not Affect Exogenous Carbohydrate Oxidation. *Med. Sci. Sports Exerc.*, Vol. 37, No. 9, pp. 1510–1516, 2005. **Purpose:** To compare the effects of high (HMW) versus low molecular weight (LMW) glucose polymer solutions on the pattern of substrate oxidation during exercise. **Methods:** Eight cyclists ( $\dot{V}O_{2\max}$ :  $63 \pm 8$  mL·kg<sup>-1</sup>·min<sup>-1</sup>) performed three 150-min cycling trials at  $64 \pm 5\%$   $\dot{V}O_{2\max}$  while ingesting 11.25% HMW (500–750 kg·mol<sup>-1</sup>, 21 mOsm·kg<sup>-1</sup>) or LMW (8 kg·mol<sup>-1</sup>, 110 mOsm·kg<sup>-1</sup>) solutions providing 1.8 g of carbohydrate per minute, or plain water. Substrate oxidation was determined using stable-isotope methods and indirect calorimetry. **Results:** Exogenous carbohydrate oxidation rate was not affected by carbohydrate molecular weight ( $P = 0.89$ , peak rate:  $0.93 \times / \div 1.37$  g·min<sup>-1</sup>). There was no effect of carbohydrate molecular weight on endogenous carbohydrate or fat oxidation rates ( $P = 0.30$ ), plasma free fatty acid ( $P = 0.14$ ), lactate ( $P = 0.38$ ), or glucose concentrations ( $P = 0.98$ ), nor were there any serious gastrointestinal complaints reported for either of the two solutions during exercise. **Conclusions:** Despite previous reports of faster gastric emptying and glycogen resynthesis suggesting enhanced glucose delivery, a markedly hypotonic HMW glucose polymer solution had no effect on exogenous and endogenous substrate oxidation rates during exercise, relative to a LMW glucose polymer solution. These data are consistent with there being no effect of carbohydrate structure or solution osmolality or viscosity on exogenous glucose oxidation and that ingested glucose polymers can only be oxidized on average up to 1.0 g·min<sup>-1</sup> during exercise. **Key Words:** STABLE ISOTOPE, GLUCOSE OXIDATION, ENDURANCE EXERCISE, MALTODEXTRINS, GASTRIC EMPTYING, OSMOLALITY

It is well established that carbohydrate feedings during moderate to high-intensity exercise can enhance performance or exercise capacity. Most of the effect of carbohydrate feedings on performance has been ascribed to maintenance of euglycemia and high rates of carbohydrate oxidation in the contracting skeletal muscle late in exercise when endogenous carbohydrate stores are low (reviewed by Coggan and Coyle (2) and Tsintzas and Williams (22)). In addition, carbohydrate feedings will suppress hepatic glucose output and thus spare this carbohydrate source (14), and in some circumstances can spare muscle glycogen use (22). It is generally believed that high rates of exogenous carbohydrate oxidation are beneficial because of the relationship with reduced oxidation of endogenous carbohydrate stores (13).

Using stable and radioactive isotope methodology, researchers have found that ingested glucose, maltose, or polysaccharide can only be oxidized at rates up to a population average of 1.0 g·min<sup>-1</sup>; even ingestion of very large amounts (up to 3.0 g·min<sup>-1</sup>) of these carbohydrates will not

further increase exogenous carbohydrate oxidation (11,12,14,21,24). The possible sites for physiological limitations of exogenous carbohydrate oxidation are muscle uptake and oxidation, release of glucose from the liver, uptake of glucose from the duodenum at the epithelial brush border membrane, enzymatic hydrolysis of di- and polysaccharides, and gastric emptying. Among these, events at the duodenal epithelia or liver most likely restrict the exogenous glucose oxidation to 1.0–1.1 g·min<sup>-1</sup> (13).

Recently, Aulin et al. (1) reported that muscle glycogen resynthesis after exhaustive exercise was 1.7-fold faster when cyclists ingested a high molecular weight carbohydrate solution compared to an isoenergetic low molecular weight carbohydrate solution in the first 2 h following glycogen-depleting exercise. Indeed, using the same solutions, Leiper et al. (15) reported an 80% faster gastric emptying rate in the first 10 min following the ingestion of the high molecular weight carbohydrate compared with the control. It was suggested by Leiper et al. (15) and Aulin et al. (1) that the marked hypotonic nature of the high molecular weight carbohydrate solution (62–84 mOsm·kg<sup>-1</sup>) facilitated more rapid gastric emptying of the carbohydrate and faster delivery of carbohydrate to the duodenum than the control solution (336–350 mOsm·kg<sup>-1</sup>), and subsequently more substrate was available in the systemic circulation for muscle glycogen resynthesis (1).

Accentuated gastric emptying of an ingested carbohydrate may enhance the delivery of exogenous carbohydrate to the small intestine and circulation, which might lead to greater exogenous carbohydrate oxidation during exercise.

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Although most studies indicate similar rates of exogenous carbohydrate oxidation irrespective of the form of the ingested glucose or glucose polymer (9,16,19), the evidence of a greater postexercise glycogen resynthesis rate with the ingestion of markedly hypotonic very high molecular weight glucose polymer led us to suppose that such a carbohydrate may also accentuate exogenous carbohydrate oxidation during exercise. Therefore, the purpose of the present study was to quantify the oxidation of glucose derived from a high molecular weight carbohydrate during 150 min of cycling exercise and compare this to the oxidation of glucose derived from a lower molecular weight carbohydrate typically used in commercial sports drinks. We hypothesized that the high molecular weight carbohydrate with low osmolality would deliver carbohydrate faster and would result in higher exogenous carbohydrate oxidation rates during exercise compared with a mixture of glucose, maltose, and higher polysaccharides (maltodextrin).

## METHODS

**Subjects.** Eight endurance-trained male cyclists and triathletes aged  $27 \pm 7$  yr and with a body mass of  $79 \pm 12$  kg and a height of  $180 \pm 5$  cm volunteered to participate in this study. The cyclists had been training for  $10.6 \pm 8.2$  yr, and in the 3-month period before the study had been training  $4.3 \pm 1.6$  d $\cdot$ wk $^{-1}$  and  $9.5 \pm 4.7$  h $\cdot$ wk $^{-1}$ . All subjects were informed of the purpose, practical details, and risks associated with the procedures before giving their written informed consent to participate. All subjects were healthy as assessed by a General Health Questionnaire. The study was approved by the University of Birmingham School of Sport and Exercise Sciences ethics committee (Birmingham, UK).

**Preliminary testing.** One week before the start of the experimental trials, each cyclist performed a 3-min stage, 35-W step incremental test on a cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) to determine maximum power output ( $W_{\max}$ ) and maximal oxygen uptake ( $\dot{V}O_{2\max}$ ). After arriving at the lab, cyclists voided, and then body mass and height were recorded.  $W_{\max}$  was calculated from the last completed work rate plus the fraction of time spent in the final noncompleted work rate multiplied by the work-rate increment. Expired air was analyzed for minute volume and oxygen and carbon dioxide fractions in an on-line automated gas analysis system in breath-by-breath mode (Oxycon Pro, Jaeger, Wuerzberg, Germany). The air flow volume sensors were calibrated using a 3-L calibration syringe and the gas analyzers calibrated using room air and a 5.03% CO<sub>2</sub>:94.97% N<sub>2</sub> gas mixture (Alpha Standard).  $\dot{V}O_{2\max}$  was determined as the peak 20-s average breath-by-breath oxygen consumption ( $\dot{V}O_2$ ) that occurred near or at the point of exhaustion. The resulting  $W_{\max}$  and relative  $\dot{V}O_{2\max}$  outputs were  $360 \pm 45$  W and  $63 \pm 8$  mL $\cdot$ kg $^{-1}\cdot$ min $^{-1}$ , respectively.

**Experimental design.** Each cyclist completed three order-randomized exercise trials consisting of 150 min of cycling at 55%  $W_{\max}$  ( $63.9 \pm 5.9\%$   $\dot{V}O_{2\max}$ ). During exercise, cyclists ingested plain water (WAT) or isoenergetic

11.25% solutions containing low molecular weight maltodextrin (LMW; Glucidex-19, Roquette, Lestrem, France) or very high molecular weight glucose polymer derived from 98 to 99% amylopectin waxy maize starch (HMW; Vitargo, Carbamyl, Karlshamn, Sweden). The respective molecular weights of the carbohydrates were approximately 8,000 and 500,000–750,000 g $\cdot$ mol $^{-1}$ . The osmolality of the carbohydrate solutions were 110 and 21 mOsm $\cdot$ kg $^{-1}$  for LMW and HMW, respectively. The exercise trials were separated by 4–7 d. In order to quantify exogenous carbohydrate oxidation, carbohydrate solutions were prepared from corn-derived glucose polymers, which have a high natural abundance of  $^{13}\text{C}$  (LMW  $-11.01$  and HMW  $-12.17$  ‰ vs Pee Dee Bellemnitella (PDB), respectively).

**Diet and activity before testing.** Cyclists recorded their food intake and activity pattern 2 d before the first exercise trial, a pattern that was repeated before the following two trials. To prevent carryover of the  $^{13}\text{C}$  label following a carbohydrate trial and to clear any initial  $^{13}\text{C}$ -glycogen enrichment, a prolonged intense training session (glycogen-depletion ride) performed in the lab or field was undertaken 3–4 d before each experimental trial. Cyclists were further instructed not to consume foods with a high natural abundance of  $^{13}\text{C}$  (carbohydrates derived from C4 plants such as maize and sugar cane) following the initial glycogen-depletion ride and throughout the entire experimental period. These procedures have been shown previously to reduce the background (change in  $^{13}\text{C}$ ) from endogenous substrate stores (23).

**Protocol.** The cyclists arrived at the laboratory between 7:00 and 9:00 a.m. after an overnight fast. After voiding, the cyclists were weighed before a 20-gauge Teflon catheter (Quickcath, Baxter BV, Norfolk, UK) was inserted into an antecubital vein of an arm and a three-way stopcock attached (Sims Portex, Kingsmead, UK) for repeated blood sampling during exercise. The catheter was kept patent by flushing with 1–2 mL isotonic saline (0.9%, Baxter).

Following set-up, the cyclists then mounted the cycle ergometer and a resting breath sample was collected in duplicate from a mixing chamber into 10-mL exetainer tubes (Exetainer, Labco, High Wycombe, UK) for determination of the  $^{13}\text{C}/^{12}\text{C}$  ratio of the expired air. A resting 7–8 mL blood sample was also collected and stored on ice until centrifugation. Next, the cyclists started the 150-min exercise bout. Additional blood samples were drawn at 15-min intervals until the cessation of exercise. Expiratory breath samples were collected every 15 min until the end of exercise for breath  $^{13}\text{C}/^{12}\text{C}$  ratio as described above. The  $\dot{V}O_2$  and  $\dot{V}CO_2$  (carbon dioxide production) were measured every 15 min for 4 min using online automated gas analysis as described above. All exercise tests were performed under normal and standard environmental conditions (18–22°C dry bulb temperature and 50–60% relative humidity), and cyclists were cooled with fans to minimize thermal stress.

The total fluid intake during the exercise bout was 2.4 L, ingested as an initial bolus (600 mL) during the first 0–3 min of exercise, followed by 200 mL every 15 min following the 4-min respiratory gas collection. The average car-

bohydrate ingestion rate in the test solutions was  $1.8 \text{ g}\cdot\text{min}^{-1}$ .

Every 30 min, the cyclists rated their RPE for whole body and legs using a 6–20 Borg scale and (possible) stomach and/or gut problems using a 10-point Likert scale (1 = not at all, 10 = very, very much). The later assessment included questions on stomach problems, gastrointestinal cramping, bloated feeling, diarrhea, nausea, dizziness, headache, and belching, vomiting, urge to urinate/defecate. Responses were divided into severe and nonsevere. Severe complaints included nausea, stomach problems, bloated feeling, diarrhea, urge to vomit, and stomach and intestinal cramps because these are symptoms that commonly impair performance and may bring with them health risks. The above symptoms were only registered as severe symptoms when a score of  $\geq 5$  out of 10 was reported. When a score  $< 5$  was given, they were registered as nonsevere. All other symptoms were registered as nonsevere regardless of the score reported.

**Analyses.** All blood samples were collected into pre-chilled potassium-EDTA Vacutainer (Beckton Dickinson, Plymouth, UK) and centrifuged at 3000 rpm and  $2^\circ\text{C}$  for 10 min. Aliquots of the plasma were transferred into microtubes and frozen in liquid nitrogen and stored at  $-25^\circ\text{C}$  until analyzed. Plasma samples were analyzed enzymatically for glucose (Glucose HK, ABX Diagnostics, UK), lactate (Lactic Acid, ABX Diagnostics) and free fatty acid (NEFA-C Kit, Alpha Laboratories, UK) concentration on a semiautomatic analyser (Cobas Mira S-Plus, ABX, UK). Breath samples were analyzed for  $^{13}\text{C}/^{12}\text{C}$  ratio by gas chromatography continuous flow isotope ratio mass spectrometry (GC-IRMS; Europa Scientific, Crewe, UK).

**Calculations.** From nonprotein  $\dot{V}\text{O}_2$  and  $\dot{V}\text{CO}_2$ , total fat and carbohydrate oxidation rates ( $\text{g}\cdot\text{min}^{-1}$ ) were calculated using the equation of Frayn (6):

$$\text{Carbohydrate oxidation} = 4.55 \dot{V}\text{CO}_2 - 3.21 \dot{V}\text{O}_2 \quad [1]$$

$$\text{Fat oxidation} = 1.67 \dot{V}\text{O}_2 - 1.67 \dot{V}\text{CO}_2 \quad [2]$$

In the HMW and LMW trials, the rate of exogenous carbohydrate oxidation (CHO<sub>exo</sub>) was calculated from the  $\dot{V}\text{CO}_2$  and the stable isotope measurements (breath  $^{13}\text{C}/^{12}\text{C}$  ratio). The isotope enrichment was expressed as  $\delta\%$  difference between the  $^{13}\text{C}/^{12}\text{C}$  ratio of the sample and the international standard PDB according to the formula of Craig (3):

$$\delta^{13}\text{C} = [({}^{13}\text{C}/{}^{12}\text{C} \text{ sample}/{}^{13}\text{C}/{}^{12}\text{C} \text{ standard}) - 1] \cdot 10^3 \text{ per mil} \quad [3]$$

CHO<sub>exo</sub> was then calculated using the formula (18):

$$\text{CHO}_{\text{exo}} = \dot{V}\text{CO}_2 \cdot [(\delta \text{Exp} - \delta \text{Exp}_{\text{bkg}})/(\delta \text{Ing} - \delta \text{Exp}_{\text{bkg}})] \cdot 1/k \quad [4]$$

where  $\delta \text{Exp}$  is the breath  $^{13}\text{C}$  enrichment during the exercise trials with carbohydrate ingestion,  $\delta \text{Ing}$  is the  $^{13}\text{C}$  enrichment of the carbohydrate sources in solution,  $\delta \text{Exp}_{\text{bkg}}$  is the  $^{13}\text{C}$  enrichment of expired air in the water (WAT) trial (background), and  $k$  is the volume of  $\text{CO}_2$  produced by the oxidation of 1 g glucose ( $0.7467 \text{ L CO}_2\cdot\text{g}^{-1}$  glucose). Endogenous carbohydrate oxidation rate was calculated by

subtracting exogenous carbohydrate oxidation from total carbohydrate oxidation rate. Energy equivalents ( $\text{kJ}\cdot\text{g}^{-1}$ ) for substrate oxidation-rate calculations were 16.18 and 40.78 for carbohydrate and fat, respectively (6).

A methodological consideration when using  $^{13}\text{CO}_2$  in expired air to calculate exogenous substrate oxidation is the delayed equilibration of  $^{13}\text{CO}_2$  originating from the tissues with the large endogenous  $\text{HCO}_3^-$  pool. However, during exercise,  $\text{CO}_2$  production increases severalfold so that a physiological steady-state condition occurs relatively rapidly, and  $^{13}\text{CO}_2$  in the expired air will be equilibrated with the  $^{13}\text{CO}_2/\text{H}^{13}\text{CO}_2$  pool from around 60 min of exercise (17,20). As a consequence of this, calculations on substrate oxidation were reported for the last 90 min of exercise only (60–150 min of exercise).

**Statistical analyses.** The effects of carbohydrate type on blood parameters and substrate utilization during exercise were estimated with mixed modeling using Proc Mixed in the Statistical Analysis System (SAS Institute, Cary, NC). With the exception of substrate utilization expressed as a percentage of total energy, all metabolic variables were log-transformed before modeling to reduce nonuniformity of error (8). The modeled fixed effects were treatment and the interaction between treatment and sample time. Cyclist and cyclist \* treatment were the specified random effects. In addition, the residual or within-athlete variance was generated within the mixed procedure.

Measures of centrality and spread for subject descriptive variables, breath  $^{13}\text{C}$  enrichments, and perception ratings are raw means and SD. Spread for log-transformed variables were represented by factor ( $\times/\div$ ) SD generated from the analyses. For example, for a plasma glucose concentration of  $2 \text{ mmol}\cdot\text{L}^{-1}$  with a between-subject SD of 20%, the typical variation is  $2 \times 1.20$  to  $2 \div 1.20$ , or 2.40 to  $1.67 \text{ mmol}\cdot\text{L}^{-1}$ . Experimental outcome data presented in graphs, tables, and text that was subject to log-transformation are shown as back log-transformed least-squares means and between-subject SD for each time point derived from the repeated-measures analysis. Data for the 60- to 150-min period of exercise represent the overall effect derived from the statistical analysis. Precision of the estimates are shown as 95% confidence limits (CL), with corresponding  $P$  values accepted as statistically significant at a probability level of  $\leq 0.05$ .

## RESULTS

**Stable isotope measurements.** Expired breath  $^{13}\text{C}$  enrichment during each trial is shown in Figure 1. Resting breath  $^{13}\text{C}$  enrichment was similar at the beginning of all three exercise trials, averaging  $-26.67 \pm 0.67$ ,  $-26.72 \pm 0.54$ , and  $-26.56 \pm 0.59 \delta\%$  versus PDB for WAT, LMW, and HMW, respectively. In the LMW and HMW trials, expired breath  $^{13}\text{C}$  enrichment increased to  $-21.54 \pm 0.56$  and  $-21.99 \pm 0.94 \delta\%$  versus PDB, respectively, at 150 min of exercise. Breath  $^{13}\text{C}$  enrichment in the carbohydrate trials were greater than in the WAT trial from 45 min onward ( $P < 0.01$ ). During the  $^{13}\text{C}$  background WAT trial,

### Breath <sup>13</sup>CO<sub>2</sub> enrichment (‰ vs. PDB)

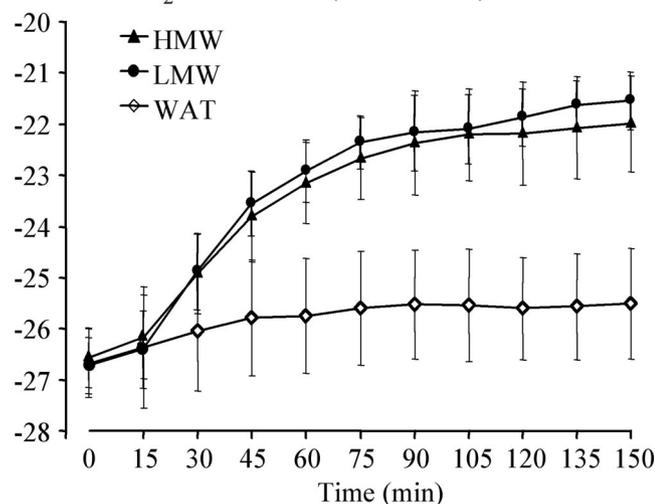


FIGURE 1—Breath <sup>13</sup>CO<sub>2</sub> enrichment during exercise with ingestion of HMW and LMW carbohydrates and or plain water (WAT; background). Values are raw mean ± SD.

expired breath <sup>13</sup>C enrichment increased slightly, but significantly from resting values to a maximum of  $-25.51 \pm 1.08$  ‰ versus PDB at 150 min of exercise ( $P < 0.01$ ). However, for the calculation of exogenous carbohydrate oxidation, the increase in breath <sup>13</sup>C enrichment at specific time points during the WAT trial was subtracted from the increase in the trials with carbohydrate ingestion in order to correct for the change in background enrichment.

#### VO<sub>2</sub>, RER, total carbohydrate and fat oxidation.

Summary data for  $\dot{V}O_2$ , RER, and carbohydrate and fat oxidation over the 60- to 150-min exercise period are shown in Table 1.  $\dot{V}O_2$  was similar in all conditions and at all time points. RER was 0.03–0.04 lower in the WAT trial than in the carbohydrate ingestion trials, but there was no clear difference between the carbohydrate conditions. Correspondingly, overall total carbohydrate and fat oxidation was lower and higher, respectively, in the WAT trial relative to the carbohydrate conditions.

Figure 2 shows the relative contributions of the substrates to total energy expenditure over the final 90 min of exercise expressed as a percentage contributions. Relative to the WAT condition, the percentage of contribution of fat was 9.24% ( $\pm$  95% CL: 5.54%,  $P$  value: 0.006) and 11.53% (5.54%, 0.001) lower in the LMW and HMW conditions, respectively (respective total carbohydrate contributions

### Contribution of Substrates to Energy Expenditure (%)

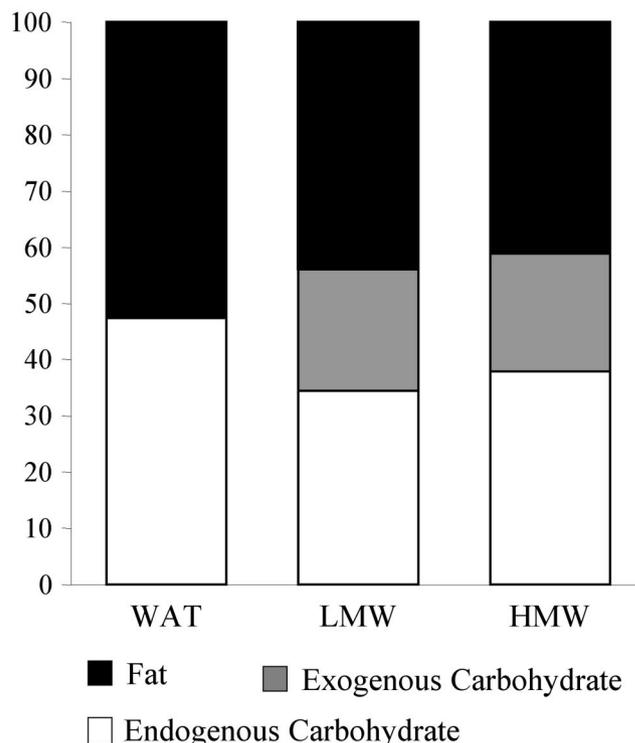


FIGURE 2—Overall relative contributions of substrates to total energy expenditure calculated for the 60- to 150-min period of exercise without ingestion of carbohydrate (WAT) and with ingestion of LMW or HMW glucose polymer solutions. Between-subject SD ( $\pm$ ): fat, 8.3%; carbohydrate exogenous, 4.9%; endogenous, 7.5%.

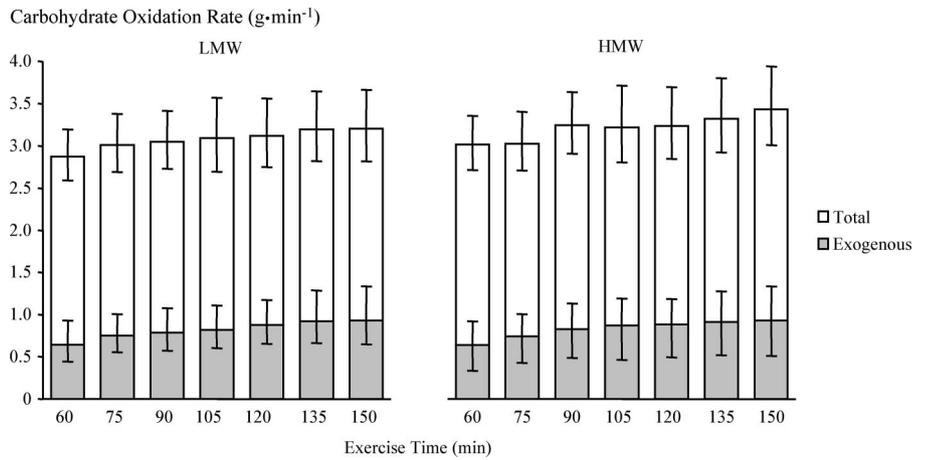
were correspondingly greater; see also Fig. 3). There was no difference in fat oxidation (2.29%,  $P = 0.488$ ), or endogenous (2.05%,  $P = 0.532$ ) or exogenous carbohydrate oxidation (2.05%,  $P = 0.532$ ) between the two carbohydrate conditions.

**Exogenous and endogenous carbohydrate oxidation rates.** Both total and exogenous carbohydrate oxidation rates in grams per minute increased over the duration of exercise in both drink conditions (Fig. 3). The average exogenous carbohydrate oxidation rate over the final 30-min exercise period was  $0.91 \times / \div 1.37$  for both LMW and HMW, whereas the peak rate reached at the end of exercise (150 min) was  $0.93 \times / \div 1.37$  g·min<sup>-1</sup> for both carbohydrates. There were no statistically significant differ-

TABLE 1.  $\dot{V}O_2$ , RER, and the overall average contribution of energy substrates during the 60- to 150-min period of cycling exercise.

	Drink condition			SD		Comparisons		
	WAT	LMW	HMW	Within	Between	LMW/WAT	HMW/WAT	LMW/HMW
$\dot{V}O_2$ (L·min <sup>-1</sup> )	3.15	3.10	3.16	1.02	1.13	0.983 (1.035, 0.412)	1.0 (1.035, 0.926)	0.981 (1.035, 0.352)
RER	0.837	0.866	0.873	1.014	1.026	1.033 (1.019, <0.0001)	1.040 (1.019, 0.0001)	0.992 (1.019, 0.499)
Substrate oxidation (kJ·min <sup>-1</sup> )								
FATtot	34.3	28.9	26.6	1.16	1.27	0.814 (1.155, 0.019)	0.774 (1.155, 0.004)	1.052 (1.155, 0.559)
CHOTot	30.8	36.0	38.5	1.09	1.19	1.185 (1.110, 0.008)	1.250 (1.110, 0.001)	0.948 (1.11, 0.401)
CHOendo	30.8	21.9	24.6	1.13	1.23	0.729 (1.159, 0.001)	0.798 (1.159, 0.013)	0.913 (1.159, 0.309)
CHOexo	—	13.4	13.4	1.13	1.39	—	—	0.984 (1.224, 0.896)

Data are least-squares means with corresponding within- and between-subject factor ( $\times / \div$ ) SD for the relevant outcome variable. Difference (or effect) comparisons are expressed as multiples or fractions of the reference condition, with the corresponding 95% confidence interval ( $\times / \div$  the estimate) and  $P$  value for the comparison in parentheses. Total carbohydrate (CHOTot), total fat (FATtot), endogenous carbohydrate (CHOendo), exogenous carbohydrate (CHOexo).



**FIGURE 3**—Total and exogenous carbohydrate oxidation rates from 60 to 150 min of exercise with the ingestion of LMW or HMW glucose polymer solutions. The first 60 min of exercise is not shown because of measurement uncertainty. Data are least-squares means; error bars are the between-subject SD derived from the square root of the variance for the back log-transformed least-squares mean.

ences in exogenous carbohydrate oxidation overall (Table 1) or for any of the individual 15-min sampling points during the exercise ( $P$  value range: 0.210–0.876). From 60 to 150 min of exercise, the peak exogenous carbohydrate oxidation rates ranged from 0.54 to 1.40  $\text{g}\cdot\text{min}^{-1}$  in the LMW condition and 0.62 to 1.81  $\text{g}\cdot\text{min}^{-1}$  in the HMW condition. Of the total 270 g of ingested carbohydrate,  $38 \pm 13$  and  $38 \pm 16\%$  was oxidized during exercise in the LMW and HMW carbohydrate conditions, respectively. Overall (60–150 min), there was no difference in exogenous (1.6%,  $P = 0.896$ ) or endogenous carbohydrate oxidation rates (9.6%,  $P = 0.309$ ) between the carbohydrate conditions.

**Plasma metabolites.** Plasma glucose, lactate, and free fatty acid concentrations during the 150-min exercise are shown in Figure 4; summary of the overall response for the 60- to 150-min period is shown in Table 2. Overall, both glucose and lactate were significantly elevated during the carbohydrate conditions relative to WAT. There was no difference in plasma glucose ( $P = 0.98$ ) or lactate ( $P = 0.38$ ) between the HMW and LMW conditions. Carbohydrate reduced plasma free fatty acid concentrations to around half that during the WAT condition. Overall free fatty acids were 70  $\text{mmol}\cdot\text{L}^{-1}$  (34%) higher in the LMW compared to the HMW condition, but the difference was not statistically significant ( $P = 0.14$ ).

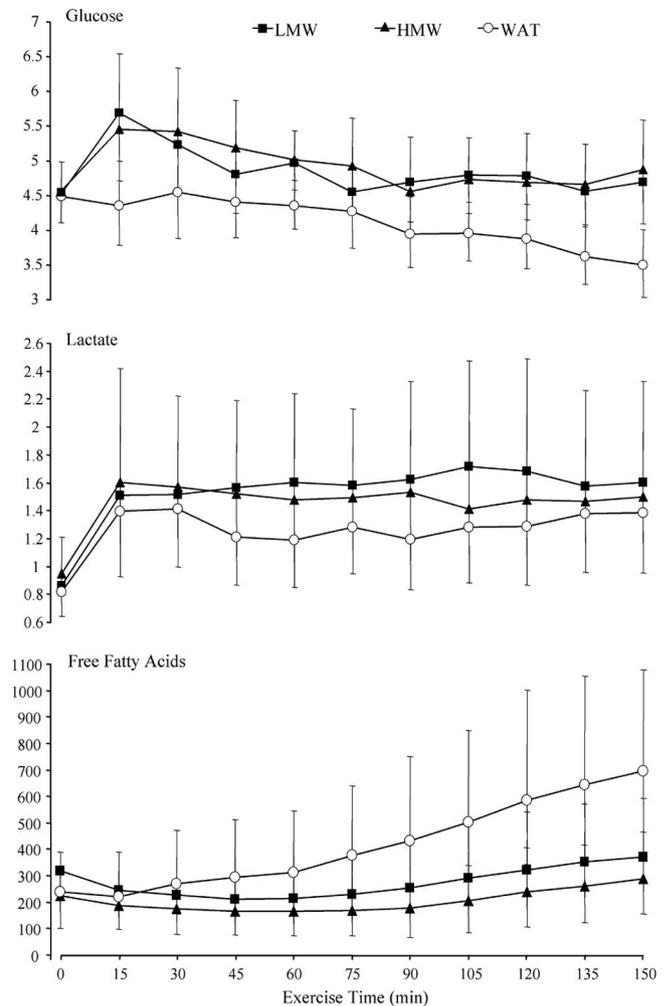
**Gastrointestinal discomfort and ratings of perceived exertion.** Gastrointestinal and related complaints were registered by a questionnaire, and the results are presented in Table 3. The most frequently reported problem was urge to urinate.

Overall ratings of perceived exertion were (mean  $\pm$  s)  $11.4 \pm 1.7$ ,  $11.9 \pm 1.4$ , and  $10.7 \pm 2.8$  for WAT, LMW, and HMW, respectively. Ratings of perceived exertion for the legs were  $11.7 \pm 2.0$ ,  $12.1 \pm 1.1$ , and  $11.3 \pm 2.5$  for WAT, LMW, and HMW, respectively.

## DISCUSSION

In this study, we investigated the effect of a carbohydrate solution made with a high molecular weight glucose polymer on exogenous carbohydrate and endogenous substrate oxidation during 150 min of endurance exercise, relative to

a solution containing a maltodextrin typical of that used in commercial carbohydrate sports drinks. The present investigation focused on the possibility that exogenous carbohydrate oxidation rates might be enhanced with HMW carbohydrate ingestion due to more rapid appearance of glucose



**FIGURE 4**—Plasma glucose, lactate, and free fatty acid concentrations ( $\text{mmol}\cdot\text{L}^{-1}$ ) during the 150 min of exercise. Bars are  $\pm$  between-subject SD derived from the square root of the variance for the back log-transformed least-squares mean; some horizontal bars have been omitted for clarity.

TABLE 2. Plasma glucose, lactate, and free fatty acid concentrations averaged over the 60–150 min of exercise.

	Drink condition			SD		Comparisons		
	WAT	LMW	HMW	Within	Between	LMW/WAT	HMW/WAT	LMW/HMW
Glucose	3.92	4.74	4.74	1.13	1.07	1.209 (1.084, 0.0002)	1.207 (1.084, 0.0002)	1.00 (1.084, 0.979)
Lactate	1.28	1.63	1.48	1.12	1.43	1.268 (1.195, 0.030)	1.153 (1.195, 0.189)	1.099 (1.195, 0.379)
Free fatty acids	489	280	210	1.19	1.75	0.576 (1.388, 0.006)	0.429 (1.388, <0.0001)	1.341 (1.388, 0.14)

Data are least-squares means (mmol·L<sup>-1</sup>) with corresponding within- and between-subject factor (×/÷) SD. See Table 1 for description of statistical comparisons.

in the blood secondary to faster delivery of the carbohydrate to the duodenum relative to the LMW solution. A faster delivery of carbohydrate to the duodenum was anticipated because Leiper et al. (15) found HMW carbohydrate solutions emptied from the stomach faster than LMW carbohydrate solutions, and Aulin et al. (1) found a 1.7-fold faster glycogen resynthesis rate during the first 2 h following glycogen-depleting exercise, an unknown effect possibly relating to the marked hypotonicity or to some other physical property of HMW carbohydrate solutions. Despite the possibility of faster gastric emptying, at the high ingestion rates used in this study (1.8 g·min<sup>-1</sup>), there was no effect of carbohydrate molecular weight on exogenous carbohydrate oxidation. Furthermore, endogenous fat and carbohydrate oxidation rates were not affected by carbohydrate molecular weight, and, on average, the peak exogenous carbohydrate oxidation rate did not increase higher than 0.93 g·min<sup>-1</sup> or 52% of the ingestion rate.

There are two likely explanations for our observations. First, although faster gastric emptying of the HMW carbohydrate solution might have occurred, the rate of emptying of the control solution may have been sufficiently fast and the quantity ingested great enough to provide carbohydrate to the duodenojejunal epithelia at saturation rates for glucose uptake. Furthermore, unlike in the study by Leiper et al. (15) in which one large 500-mL bolus containing 75 g of carbohydrate was ingested followed by measurement of gastric emptying using a double-sampling gastric aspiration technique, we provided a 600-mL bolus containing 67.5 g carbohydrate at the start of exercise followed by 200 mL top ups containing 22.5 g carbohydrate every 15 min. Leiper et al. (15) found that the major difference in gastric emptying between HMW and LMW carbohydrate solutions occurred during the first 10 min following ingestion; afterward, rates

were similar. During the first 10 min, a median of approximately 32 g of carbohydrate had been delivered to the small intestine following HMW carbohydrate solution ingestion, whereas only approximately 15 g had been delivered following ingestion of the control drink. Median carbohydrate delivery rates of both solutions ranged between 4 and 13 g·10 min<sup>-1</sup> for the remaining 50 min of sampling. The cumulative delivery over the hour was markedly greater with the HMW carbohydrate solution (15). In the present study, the top-up drinks every 15 min would presumably have increased the delivery rates further, so it is likely that both solutions provided carbohydrate to the duodenum at or above 15 g·10 min<sup>-1</sup>, which is within the range estimated for maximal glucose absorption rate across the total duodenojejunal epithelial (i.e., 1.3–1.7 g·min<sup>-1</sup>) based on extrapolation from the triple lumen tube technique (4).

Limitations in the rate of release of glucose from the liver into the systemic circulation provide the second explanation for the present observations. The present and previous reports show that when large amounts (≥1.8 g·min<sup>-1</sup>) of glucose or glucose polymers are ingested, exogenous carbohydrate oxidation rates, on average, do not exceed 1.0–1.1 g·min<sup>-1</sup> (12,14,16,19,23,24). As described above, the maximal intestinal absorption rate might be greater than the maximal average exogenous carbohydrate oxidation rate, suggesting that the liver then limits glucose release to the systemic circulation to the contracting muscle tissue and other tissues for oxidation (13). Be that as it may, the results from a number of more recent studies point circumstantially to the intestine rather than the liver as the site of limitation for exogenous carbohydrate absorption and appearance in the circulation. For example, the simultaneous feeding during 150 min of exercise at 63%  $\dot{V}O_{2max}$  of the multiple transportable carbohydrates glucose and fructose at an average of 1.2 and 0.6 g·min<sup>-1</sup>, respectively, resulted in an average peak exogenous carbohydrate oxidation rate of 1.26 g·min<sup>-1</sup>, compared with 0.83 g·min<sup>-1</sup> with 1.8 g·min<sup>-1</sup> of glucose-only feeding (11). Fructose is absorbed from the intestine by GLUT-5-mediated transport, whereas a different mechanism is responsible for glucose absorption (sodium dependent glucose transporters, SGLT1) (5). In a follow-up study, the ingestion of a solution delivering 1.2 g·min<sup>-1</sup> glucose and 1.2 g·min<sup>-1</sup> of fructose to the stomach during exercise at 63%  $\dot{V}O_{2max}$  resulted in a further elevation of the peak exogenous carbohydrate oxidation rate to 1.75 g·min<sup>-1</sup> (10), which is very close to the findings of Hawley et al. (7), who bypassed the splanchnic bed via hyperglycemic glucose infusion to maintain glucose concentration at 10 mmol·L<sup>-1</sup> and observed a peak plasma glucose oxidation rate of around 1.8 g·min<sup>-1</sup>. Therefore, the

TABLE 3. Gastrointestinal and related complaints during the 150 min of exercise.

Complaint	WAT	LMW	HMW
Nonsevere			
Dizziness	0	0	0
Flatulence	0	0	0
Urge to urinate	4	4	1
Urge to defecate	0	0	0
Belching	0	0	0
Stomach burn	0	0	0
Side aches left	0	0	0
Side aches right	0	0	0
Severe			
Stomach problems	0	1	0
Nausea	0	0	0
Urge to vomit	0	0	0
Bloated feeling	0	2	1
Stomach cramps	0	1	0
Intestinal cramps	0	0	0
Diarrhea	0	0	0

ingestion of glucose and fructose carbohydrate solutions at very high rates might provide exogenous carbohydrate to closely match the capacity for plasma glucose oxidation.

In summary, when a markedly hypotonic, high molecular weight carbohydrate solution was ingested at an average rate of  $1.8 \text{ g}\cdot\text{min}^{-1}$  during cycling exercise, average exogenous carbohydrate oxidation rates did not increase above  $1.0 \text{ g}\cdot\text{min}^{-1}$ ,

and this was not different from the oxidation observed with a low molecular weight carbohydrate solution.

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