Effect of carbohydrate supplements and water on exercise metabolism in the heat

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YASPELKIS, BENEDICT B., III, AND JOHN L. IVY. *Effect of carbohydrate supplements and water on exercise metabolism in the heat.* J. Appl. Physiol. 71(2): 680–687, 1991.—Carbohydrate (CHO) supplements of different concentrations were compared with water to determine their effects on thermal regulation and plasma volume maintenance while subjects exercised for 2 h in the heat and to determine their impact on carbohydrate utilization. Trained cyclists (n = 12) rode at 48.8 ± 0.8% maximal O_2 consumption in an environmental chamber maintained at 33.0 ± 0.1°C and 51.7 ± 1.4% relative humidity on three separate occasions. During each exercise bout the subjects received 3 ml/kg body wt of H_2O, a 2.0% glucose polymer (LC) solution, or an 8.5% glucose polymer (HC) solution every 15 min. Muscle biopsies from the vastus lateralis were obtained before and after the H_2O and HC trials only. Rectal temperature and heart rate, but not O_2 consumption, rose from the 10- to 120-min period of exercise. No differences among treatments were found for these variables. There were also no significant differences among treatments for percent changes in plasma volume and blood volume. Plasma glucose and insulin were unchanged during the H_2O and LC trials but were significantly elevated during the HC trial. In addition, CHO oxidation was significantly greater during the HC trial than during the H_2O trial from 60 to 120 min of exercise. However, the reduction in muscle glycogen during the HC trial (206.5 ± 23.6 μmol/g protein) was significantly less ($P < 0.05$) than during the H_2O trial (342.3 ± 41.9 μmol/g protein). These results demonstrated that the 8.5% CHO supplement regulated body temperature and prevented disturbances in fluid homeostasis as effectively as water while maintaining CHO oxidation and reducing the rate of decline in muscle glycogen during low-intensity exercise in the heat.

fluid replacement; plasma volume; glucose; insulin; muscle glycogen

DURING SUSTAINED EXERCISE in the heat, large amounts of water may be lost as a result of sweating (1–2 l/h). Dehydration during exercise can result in an increased plasma osmolality, decreased sweat rate, and increased body core temperature (40). Additionally, an elevated body temperature has been reported to impair exercise performance and increase the risk of heat injury (22, 24, 31). However, water ingestion during exercise performed in the heat can attenuate a rise in body temperature (13, 22).

During prolonged exercise in the heat, it has been suggested that the ingestion of supplements containing >2.5% carbohydrate will inhibit fluid delivery (14, 17). Costill and Saltin (14) compared the rates of gastric emptying among water and beverages containing 2.5, 5.0, 10.0, and 15.0% carbohydrate. They observed that water and the 2.5% carbohydrate supplement resulted in similar rates of gastric emptying, but the beverages containing a greater concentration of carbohydrate delayed gastric emptying. On the basis of these findings, it has been recommended that the carbohydrate content of fluid replacement drinks be kept under 2.5% to optimize fluid delivery (2). However, more recent research has demonstrated that carbohydrate supplements containing as much as 15% carbohydrate do not differ from water in their ability to effectively support thermoregulation during prolonged exercise in a warm environment (9, 10, 32). In addition, a supplement containing 7.5% carbohydrate may result in a smaller percent change in plasma volume than water during exercise in the heat (10).

It has also been suggested that exercise performed in the heat accelerates fatigue because of an increased reliance on carbohydrate as substrate (21). It has been well documented that the ingestion of carbohydrate supplements during prolonged endurance exercise can compensate for the reduction of endogenous carbohydrate stores and delay the onset of fatigue (11, 15, 18, 27, 28).

Although it appears that carbohydrate supplements in the range of 7.0–10.0% can support thermoregulation as effectively as water during prolonged exercise in a warm environment, their effects on carbohydrate metabolism and the endogenous carbohydrate stores have not yet been addressed. Therefore the purpose of the present investigation was to compare carbohydrate supplements of 2.0 and 8.5% with water during low-intensity exercise in the heat to determine their impact on carbohydrate metabolism as well as their effect on thermoregulation and plasma volume changes.

METHODS

Subjects. The subjects were 12 male competitive cyclists between the ages of 18 and 24 yr who frequently cycled over prolonged periods (2–4 h). They weighed 73.1 ± 1.9 kg and had a mean maximal O_2 consumption ($\dot{V}O_{2\,max}$) of 65.8 ± 1.7 ml · kg^{-1} · min^{-1}. Before testing, all subjects were given a detailed explanation of the procedures to be used and the potential risks of the study and signed an informed consent form. The study was approved by the university's Institutional Review Board.

Experimental design. Each subject completed three randomly assigned treatments in which 3 ml/kg body weight of H_2O, a 2.0% (LC) carbohydrate, or an 8.5%

TABLE 1. *Beverage composition*

Beverage	CHO, %		Na^+, meq/l	K^+, meq/l	Osmolality, mosM
	Maltodextrin	Fructose			
H_2O	0	0	0	0	0
LC	2.0	0	3.48	1.53	54
HC	5.75	2.75	5.20	0.51	273

CHO, carbohydrate; H_2O, water; LC, 2.0% CHO; HC, 8.5% CHO.

(HC) carbohydrate supplement (Table 1) was provided immediately before exercise and every 15 min thereafter. The three trials were completed over a 2- to 3-wk period and were conducted in a Sherer environmental chamber, which was maintained at 33.0 ± 0.1°C and 51.7 ± 1.4% relative humidity.

Preliminary testing. $\dot{V}O_{2\,max}$ was determined during a continuous incremental cycling test performed on a Monark Ergomedic 818E cycle ergometer equipped with toe clips and straps. The incremental test protocol consisted of 2-min stages that were set to elicit 40, 60, 85, 95, and 110% of the subject's estimated $\dot{V}O_{2\,max}$. Subjects breathed through a Daniel's valve, with expired gases directed to a mixing chamber for analysis of O_2 (Applied Electrochemistry S-3A/1, Ametek, Pittsburgh, PA) and CO_2 (Beckman LB-2, Beckman Instruments, Palo Alto, CA). Inspired volumes were measured using a dry gas meter (Rayfield Equipment, Waitsfield, VT). Analog outputs from these instruments were directed to a laboratory computer for calculation of ventilation ($\dot{V}E$), O_2 consumption ($\dot{V}O_2$), CO_2 production ($\dot{V}CO_2$), and respiratory exchange ratio (R) every 30 s. The criteria used to establish the $\dot{V}O_{2\,max}$ were a plateau in $\dot{V}O_2$ with increasing exercise intensity and R > 1.10.

Protocol. The subjects reported to the laboratory after a 12-h fast and abstained from fluid consumption for the final 2 h of the fast. Food intake and activity levels were controlled for 2 days before the first trial and were duplicated for subsequent trials. On reporting to the laboratory, nude body weight was obtained, a rectal temperature probe was inserted to a depth of 10 cm beyond the anal sphincter, and a heart rate (HR) monitor (UNIQ Heartwatch model 8799, Computer Instruments, Hempstead, NY) was secured in place on the chest. A catheter was inserted into an antecubital vein and taped in place. The subjects then entered the chamber and sat quietly on the cycle ergometer for 5 min before the commencement of the test. The subjects performed 120 min of cycling at 48.8 ± 0.8% $\dot{V}O_{2\,max}$. On conclusion of the exercise bout, the subjects towel dried and nude body weight was recorded. Before and immediately after the H_2O and HC trials, a muscle biopsy was obtained from the vastus lateralis by the needle biopsy technique (4).

Sample collection and analyses. $\dot{V}E$, $\dot{V}O_2$, $\dot{V}CO_2$, and R were recorded for periods of 5 min by use of the respiratory gas analysis system previously described. Collection periods ended at 15, 30, 60, 90, and 120 min of exercise. Carbohydrate oxidation was calculated from the tables of Lusk (30), assuming a nonprotein R.

HR, by use of a radio telemetry unit, and body temperature [Yellow Springs Instruments] (YSI) series 400 temperature probe interfaced with a YSI 2100 Tele-thermometer, Yellow Springs, OH) were recorded immediately before the start of the exercise bout and every 15 min thereafter. Subjective ratings of perceived exertion (6) were obtained after 5 and 15 min of exercise and then at every 15 min of exercise.

Approximately 5 ml of venous blood were drawn while the subjects were seated on the ergometer immediately before the start of exercise and at 5, 30, 60, 90, and 120 min of exercise. Hematocrit was determined by microcentrifugation, and hemoglobin was analyzed by the cyanmethemoglobin method (Drabkin's reagent, Sigma Chemical, St. Louis, MO). Percent changes in blood volume, cell volume, and plasma volume were calculated as described by Dill and Costill (19). Four milliliters of each blood sample were anticoagulated with 250 μl of EDTA, and the plasma was separated by centrifugation at 4°C. The plasma was split into equal portions and stored at −80°C for subsequent measurement of glucose and insulin. Plasma was analyzed for glucose concentration with a glucose analyzer (model 23A, Yellow Springs Instruments). The insulin concentration was measured by radioimmunoassay (23) (Radioassay System Laboratories, Carson, CA). One-half milliliter of blood was deproteinized in 1 ml of cold 8.0% perchloric acid and centrifuged at 1,000 *g* (4°C) for 15 min. The acid extract was then stored at −80°C for subsequent enzymatic analysis of lactate (25).

Percutaneous muscle biopsies taken from the vastus lateralis were divided into two pieces. One portion was quickly frozen in isopentane cooled in liquid N_2 and stored at −80°C until analyzed for glycogen. For glycogen determination, the biopsies were weighed and homogenized in a 50% glycerol-20 mM Na_2HPO_4 buffer (50:1 wt/vol, pH 7.4) that contained 0.5 mM EDTA, 0.02% bovine serum albumin, and 5 mM β-mercaptoethanol. Homogenization was performed in a dry ice-acetone bath. Two hundred microliters of the homogenate were added to 140 μl of 2 N HCl and incubated at 100°C for 120 min. The homogenate was cooled to room temperature and neutralized with 1 N NaOH. The muscle glycogen concentration was determined enzymatically (34) and made relative to the protein concentration of the muscle (μmol/g protein). Muscle protein concentration was measured colorimetrically as described by Bradford (7).

The second piece of the biopsy sample was oriented in mounting media (OCT compound, Fisher Scientific, Pittsburgh, PA) and rapidly frozen in isopentane cooled to its freezing point in liquid N_2 and stored at −80°C. Serial sections (10 μm) were cut at −20°C in a cryostat and were stained for myosin adenosinetriphosphatase activity (pH 4.55) (8) and for glycogen via the periodic acid-Schiff (PAS) reaction (35). Sections from each biopsy sample were magnified, and the intensity of the PAS staining in the individual muscle fibers was rated visually on a scale of 0 (negative) to 4 (darkly stained).

Statistical analysis. The data were analyzed using a two-way analysis of variance (treatment × time) for repeated measures. The difference between the pre- and postexercise muscle glycogen concentrations (Δglycogen) was analyzed with Student's *t* test. Significant differences between means were determined using Tukey's

TABLE 2. *Heart rate, rectal temperature, and ratings of perceived exertion during 120 min of low-intensity exercise in the heat*

Beverage	Pre	Time, min 5	15	30	45	60	75	90	105	120
					HR, beats/min					
H_2O	76±3.0	125±3.9	130±4.6	134±4.5	137±4.6	137±4.5	142±4.4	142±4.6	146±5.2	147±4.9
LC	72±2.7	123±4.6	128±4.1	131±4.6	137±4.6	138±4.4	140±4.4	142±4.5	145±5.3	147±5.0
HC	70±2.9	124±4.7	128±4.9	131±4.5	135±4.6	136±4.5	140±4.4	140±4.7	145±5.1	145±5.2
					T_{re}, °C					
H_2O	37.1±0.07	37.2±0.07	37.5±0.07	37.8±0.06	38.0±0.07	38.1±0.08	38.2±0.08	38.2±0.09	38.2±0.10	38.4±0.17
LC	37.1±0.13	37.2±0.07	37.5±0.10	37.8±0.10	37.9±0.13	38.0±0.10	38.0±0.11	38.1±0.08	38.2±0.10	38.2±0.12
HC	37.1±0.08	37.2±0.07	37.5±0.07	37.8±0.08	38.0±0.08	38.0±0.10	38.1±0.09	38.2±0.10	38.2±0.10	38.2±0.09
					RPE, units					
H_2O		9.1±0.41	9.7±0.41	10.1±0.40	10.4±0.45	10.9±0.34	11.4±0.31	11.6±0.40	11.8±0.33	11.8±0.35
LC		8.6±0.51	9.5±0.38	10.3±0.36	10.7±0.33	11.1±0.34	11.3±0.38	12.0±0.43	12.1±0.45	12.3±0.57
HC		8.9±0.48	9.4±0.58	9.8±0.52	10.0±0.52	10.3±0.51	10.8±0.46	11.3±0.56	11.6±0.60	11.5±0.54

Values are means ± SE. Subjects were fed 3 ml/kg body wt of water (H_2O), 2.0% CHO (LC), and 8.5% CHO (HC) immediately before exercise and every 15 min during exercise. HR, heart rate; T_{re}, rectal temperature; RPE, rating of perceived exertion.

post hoc analysis. Differences were considered significant at $P \leq 0.05$.

RESULTS

Thermoregulatory and cardiovascular responses. The H_2O, LC, and HC treatments did not differ in their ability to effectively support thermoregulation as measured by rectal temperature (Table 2). Rectal temperature was 37.1°C at rest and then rose to a steady-state level of ~38.2°C by 90 min of exercise. This temperature was then maintained for the duration of the exercise bout.

HR was similar among all beverage treatments, although all treatments exhibited a cardiac drift of ~20 beats/min by the end of the exercise bout (Table 2). Subjects also did not perceive any difference in effort during exercise as a result of the treatments (Table 2). Although perceived exertion rose from ~9 units at 5 min of exercise to 11.9 units by 120 min, the subjects found the exercise intensity to be relatively easy.

Hematocrit and hemoglobin did not differ among treatments (Table 3). Within the first 5 min of exercise there was a significant increase in both hemoglobin and hematocrit, and over the following 115 min of exercise both gradually increased. The calculated changes for blood and plasma volume exhibited no difference among treatments throughout the exercise bouts (Table 3).

Plasma glucose, insulin, and blood lactate responses. As shown in Fig. 1, plasma glucose was not different among treatments before exercise. The HC treatment, however, rapidly elevated plasma glucose to 6.2 ± 0.4 mM during the first 30 min of exercise, which was significantly greater than the plasma glucose response of the H_2O and LC treatments ($P < 0.05$). Additionally, the HC treatment continued to maintain plasma glucose at ~5.5 mM during the remaining 90 min of exercise. The LC treatment resulted in a small increase in plasma glucose from 4.3 ± 0.15 to 4.9 ± 0.19 mM by 30 min of exercise. The plasma glucose then gradually declined during the LC treatment to 4.5 ± 0.20 mM by 120 min. Plasma glucose remained relatively stable at ~4.4 mM throughout the H_2O trial.

In response to the increased plasma glucose during the HC trial, the plasma insulin concentration increased from 9.4 ± 0.7 μU/ml at rest to 25.3 ± 3.9 μU/ml after 30 min of exercise (Fig. 2). The elevated insulin concentration at 30 min of exercise during the HC treatment was significantly greater than that during the H_2O and LC treatments ($P < 0.05$). The plasma insulin concentration during the HC trial remained significantly elevated ($P < 0.05$) above that of the H_2O and LC trials throughout the following 90 min of exercise. Plasma insulin concentrations did not differ during the H_2O and LC trials. They were ~10 μU/ml before exercise and declined to ~6 μU/ml by 120 min of exercise.

Blood lactate was elevated from ~0.9 mM at rest to 1.3 mM after 5 min of exercise (Fig. 3) and was not different among treatments. However, blood lactate levels during the HC treatment were significantly greater than those during the H_2O and LC treatments at 60 and 90 min of exercise ($P < 0.05$), although the differences were only 0.3–0.4 mM.

Respiratory exchange ratio and carbohydrate oxidation. R was 0.85 ± 0.01 for both the H_2O and LC trials after 30 min of exercise. During the LC trial, R did not change significantly (Table 4), but during the H_2O trial R had significantly declined by 120 min of exercise. The HC treatment resulted in a slightly elevated R by 30 min of exercise. By 60 min of exercise, R was significantly elevated ($P < 0.05$) above that of the H_2O trial, and by 90 min of exercise R was significantly elevated ($P < 0.05$) above those of the H_2O and LC trials. Because $\dot{V}O_2$ was not different between trials, the calculated carbohydrate oxidation paralleled the R responses (Table 4). During the H_2O and LC treatments, carbohydrate oxidation gradually declined. Carbohydrate oxidation was ~42.5 g/30 min for the first 30 min of exercise and declined to 32.39 ± 2.9 and 37.9 ± 3.5 g/30 min for the final 30 min of exercise during the H_2O and LC treatments, respectively. The HC treatment was able to maintain carbohydrate

TABLE 3. *Hematocrit, hemoglobin, and blood volume and plasma volume changes during 120 min of low-intensity exercise in the heat*

Beverage	Pre	Time, min 5	30	60	90	120
			Hct, %			
H_2O	44.9±0.5	46.8±0.5	46.5±0.5	46.5±0.4	46.8±0.4	46.9±0.5
LC	44.6±0.5	46.0±0.5	45.7±0.5	45.7±0.5	45.9±0.6	46.2±0.6
HC	44.9±0.4	46.9±0.3	46.3±0.4	46.2±0.5	46.6+0.5	46.7+0.4
			Hb, g/100 ml			
H_2O	15.2±0.2	15.9±0.2	15.9±0.2	15.9±0.2	16.0±0.2	16.1±0.2
LC	15.3±0.2	15.9±0.3	16.1±0.3	16.0±0.3	16.1±0.3	16.2±0.3
HC	15.4±0.2	15.9±0.2	15.9±0.2	15.9±0.2	16.0±0.2	16.2±0.3
			BV, % change			
H_2O		−5.0±0.6	−4.4±0.5	−5.1±0.7	−5.2±0.7	−5.9±0.8
LC		−4.0±0.4	−4.9±0.5	−4.7±0.6	−5.0±0.3	−5.4±0.6
HC		−3.9±0.5	−3.5±0.6	−3.7±0.7	−4.3±0.8	−5.0±0.9
			PV, % change			
H_2O		−8.2±0.7	−7.2±0.8	−7.8±0.9	−8.4±1.1	−9.3+1.4
LC		−6.3±0.5	−7.1±0.5	−6.9±0.8	−7.7±0.7	−8.5±1.1
HC		−6.7±0.8	−6.0±0.9	−6.2±1.3	−7.4±12	−7.4±1.4

Values are means ± SE. Hct, hematocrit; Hb, hemoglobin; BV, blood volume; PV, plasma volume. Details as in Table 2 footnote.

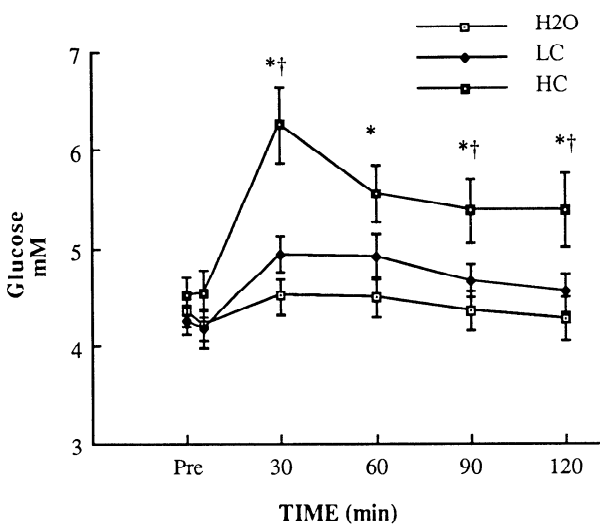

FIG. 1. Plasma glucose during 120 min of low-intensity cycling exercise in the heat. H_2O, water; LC, 2.0% carbohydrate (CHO); HC, 8.5% CHO. Values are means ± SE. *Significantly different from H_2O ($P < 0.05$). †Significantly different from LC ($P < 0.05$).

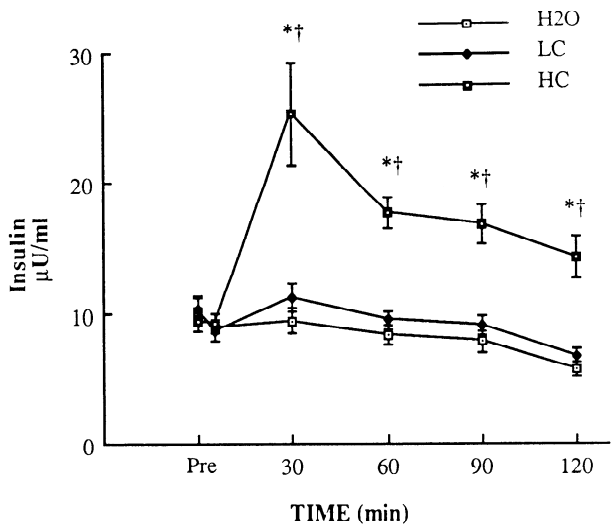

FIG. 2. Plasma insulin concentration during 120 min of low-intensity cycling exercise in the heat. See Fig. 1 legend for explanation of symbols. Values are means ± SE.

oxidation throughout the 120 min of exercise at ~48 g/30 min and was significantly different ($P < 0.05$) from the H_2O trial at 60, 90, and 120 min.

Glycogen concentration in the vastus lateralis muscle. Muscle glycogen concentrations for both the H_2O and HC trials were not statistically different before and after exercise when expressed relative to muscle protein concentration (Table 5). However, the difference between the pre- and postexercise glycogen concentrations (Δglycogen) was significantly different between treatments ($P < 0.05$). As shown in Fig. 4, 9 of the 12 subjects exhibited a reduced decline in muscle glycogen during the HC treatment while the other 3 subjects showed a reduced decline in muscle glycogen during the H_2O treatment.

Muscle sections were subjected to PAS staining to determine whether the distribution of glycogen between the type I and II fibers differed after the HC and H_2O treatments. Little difference was observed in the staining pattern in the type II fibers after exercise (Fig. 5). The primary differences in the glycogen-staining intensity occurred in the type I fibers. Approximately 14.0% more type I fibers stained heavily for glycogen (i.e., 3 and 4 on the 0–4 scale) after the HC treatment, and more type I fibers were lightly stained for glycogen (i.e., 0 and 1) after the H_2O treatment (H_2O 40.0% vs. HC 19.5%).

DISCUSSION

Initial research on the rates of gastric emptying of carbohydrate solutions indicated that emptying became progressively slower as the carbohydrate concentration was increased above 2.5% (14). Thus it was recommended that a fluid replacement drink not be ingested that con-

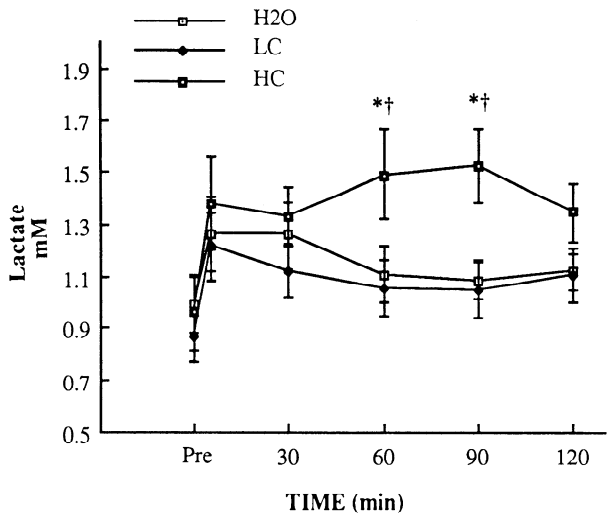

FIG. 3. Blood lactate during 120 min of low-intensity cycling exercise in the heat. See Fig. 1 legend for explanation of symbols. Values are means ± SE.

tained >2.5% carbohydrate. It was believed that if fluid delivery was inhibited because of an excessive carbohydrate concentration, the regulation of body temperature would be impaired when exercising in the heat. In contrast to this recommendation, we observed no difference among the H_2O, LC, and HC treatments on body temperature changes during exercise in the heat. Our present observation is in agreement with those of a number of other investigators who have recently found that fluid replacement drinks containing up to 15% carbohydrate can support thermoregulation as effectively as water when exercise is performed in a thermally stressful environment (9, 10, 13, 32, 33, 39). Candas et al. (9) observed that when subjects cycled for 4 h at 50% $\dot{V}O_{2\,max}$ in 34°C heat and ingested 100 ml every 10 min of either water or a 15% carbohydrate supplement, body temperature was similar between treatments throughout exercise. Additionally, carbohydrate supplements have been demonstrated to maintain thermoregulation during higher-intensity exercise in the heat. Murray et al. (32) had subjects perform 1.25 h of intermittent cycling at 65% $\dot{V}O_{2\,max}$ in an environment maintained at 33.4°C. The subjects ingested 2.5 ml/kg body wt of water, 6.0, 8.0, or 10.0% sucrose solutions every 20 min. It was found that the rise in rectal temperature during exercise was similar among all beverages. Therefore, on the basis of these results, it appears that a carbohydrate supplement containing as much as 15% carbohydrate can be ingested while subjects exercise in the heat without adversely affecting thermoregulation.

It has been observed that plasma volume shifts during exercise are not different whether water or carbohydrate supplements are ingested (9, 32, 36). In agreement with these investigations, we also observed that plasma volume shifts during exercise were similar among the H_2O, LC, and HC treatments. In contrast, Ryan et al. (39) observed that a 5.0% glucose polymer supplement minimized plasma volume shifts during exercise compared with water. Additionally, Carter and Gisolfi (10) demonstrated that a 7.5% glucose polymer supplement had a similar effect on plasma volume shifts. On the other hand, Owen et al. (33) reported that a greater decline in plasma volume resulted from ingestion of a 10.0% carbohydrate supplement than from water. The reason for the different plasma volume shifts in response to the ingestion of carbohydrate supplements noted by these investigators is not known but may be related to such differences as the types of carbohydrates used, the osmolality of the supplements, and their electrolyte concentration.

In addition to elevating body temperature, exercise that is performed in the heat appears to increase the rate of endogenous carbohydrate utilization (20, 42). Rowell et al. (38) observed that exercise at 50% $\dot{V}O_{2\,max}$ in 49°C heat increased hepatic glucose output compared with exercise in a thermoneutral environment. Therefore it may be advantageous for athletes performing prolonged aerobic exercise (i.e., >1.5 h) in the heat to ingest carbohydrate supplements to compensate for the increased utilization of carbohydrate stores. However, it has yet to be determined whether a sufficient amount of carbohydrate can be ingested during prolonged continuous exercise in

TABLE 4. *O_2 consumption, respiratory exchange ratio, and carbohydrate oxidation during 120 min of low-intensity exercise in the heat*

Beverage	Time, min				
	15	30	60	90	120
			$\dot{V}O_2$, l/min		
H_2O	2.36±0.09	2.34±0.08	2.35±0.08	2.36±0.07	2.46±0.08
LC	2.34±0.08	2.42±0.08	2.36±0.10	2.40±0.09	2.42±0.11
HC	2.34±0.09	2.33±0.07	2.34±0.08	2.31±0.08	2.36±0.08
			R		
H_2O		0.85±0.01	0.84±0.01	0.83±0.01	0.82±0.01
LC		0.85±0.01	0.85±0.01	0.85±0.01	0.84±0.01
HC		0.87±0.01	0.88±0.01*	0.88±0.01*†	0.87±0.01*†
			Carbohydrate oxidation, g/30 min		
H_2O		41.47±3.66	37.72±3.60	34.01±3.13	32.90±2.85
LC		43.58+2.54	41.06+2.92	40.34+2.65	37.91±3.52
HC		47.12±3.26	49.48±3.22*	49.57±3.45*	46.63±3.09*

Values are means ± SE. $\dot{V}O_2$, O_2 consumption; R, respiratory exchange ratio. Details as in Table 2 footnote. * Significantly different from H_2O ($P < 0.05$). † Significantly different from LC ($P < 0.05$).

TABLE 5. *Muscle glycogen and protein concentration before and after 120 min of low-intensity exercise in the heat*

Beverage	Pre	Post	Δ
	Glycogen, μmol/g protein		
H_2O	693.4±53.1	350.4±25.5	342.9±41.9
HC	608.2±31.9	401.7±33.3	206.5±23.6*
	Protein, mg/g wet wt		
H_2O	184.6±5.4	203.7±8.2	
HC	198.4±5.5	190.8±5.1	

Values are means ± SE. Muscle biopsy samples were obtained immediately before and after exercise. Δ, difference between pre- and post-exercise glycogen concentration. * Significantly different from H_2O ($P < 0.05$).

the heat to favorably enhance carbohydrate metabolism without adversely affecting thermoregulation. With regard to the effects of the carbohydrate supplements on metabolism, we found that the HC treatment increased plasma glucose above the preexercise resting level. The HC treatment also maintained carbohydrate oxidation throughout exercise, which was in contrast to the gradual decline in carbohydrate oxidation that occurred during the H_2O treatment. Most notably, the favorable impact on these variables occurred with no adverse effect on thermoregulation.

We were also interested in determining whether the ingestion of a carbohydrate beverage would affect muscle glycogen utilization when exercise was performed at a low intensity in an environment that could potentially accelerate the rate of endogenous carbohydrate utilization. We observed that the HC treatment reduced the decline in muscle glycogen by ~41% compared with the H_2O treatment. In support of our findings, Bagby et al. (3) found that glucose infusion resulted in muscle glycogen sparing in exercising rats. The rats were run for 1, 2, or 3 h and infused with glucose at a rate of 0.37 mmol/min. This rate of glucose infusion resulted in significantly elevated blood glucose and insulin concentrations, which may have aided in sparing muscle glycogen. Similarly, Bergström and Hultman (5) observed a sparing of

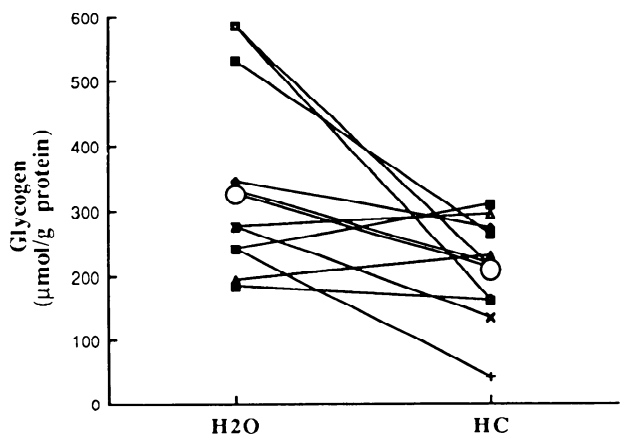

FIG. 4. Individual subject difference in muscle glycogen use relative to protein (μmol/g protein) during 120 min of low-intensity cycling exercise in the heat. See Fig. 1 legend for explanation of symbols. ○, Group means.

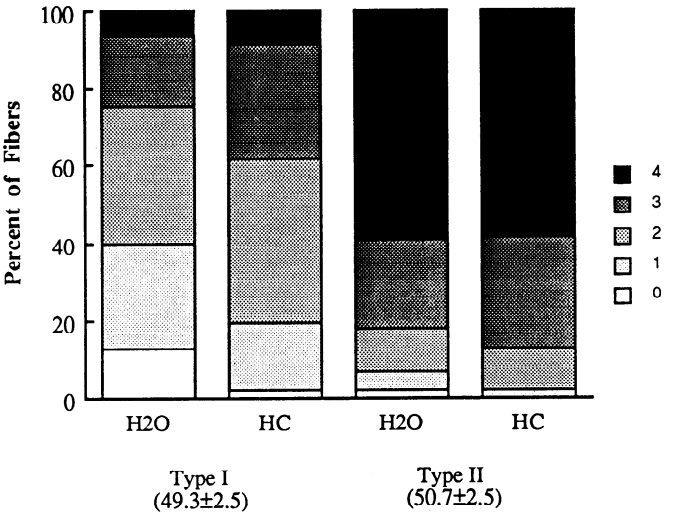

FIG. 5. Histochemical estimation of glycogen use in muscle sections from postexercise biopsies stained with periodic acid-Schiff (PAS) reagent. Pattern of staining is displayed for both type I and II muscle fibers. Fiber type percentage is shown in parentheses. Intensity of glycogen staining is rated on a scale of 0 (negative) to 4 (darkly stained).

muscle glycogen in humans who were infused with 18 mmol/min of glucose during 1 h of intermittent cycling exercise. Hargreaves et al. (27) also observed a reduced decline in muscle glycogen during intermittent exercise when subjects ingested 43 g of a solid carbohydrate supplement every hour. The exercise protocol for this 4-h trial was comprised of eight repeated 30-min sequences. The 30-min sequence consisted of 20 min of cycling at 50% $\dot{V}O_{2\,max}$ and then 10 min of four repeated bouts of cycling at 100% $\dot{V}O_{2\,max}$ for 30 s followed by 2 min of rest.

In contrast to our findings, Coyle et al. (15) and Hargreaves and Briggs (26) observed that carbohydrate supplements had no effect on the rate of muscle glycogen decline during continuous exercise. The difference among studies is most likely due to exercise intensity. The experimental protocol that was utilized by Coyle et al. (15) and Hargreaves and Briggs (26) consisted of having subjects cycle for 2 h at ~70% $\dot{V}O_{2\,max}$. In contrast, the present investigation required subjects to cycle for 2 h at 50% $\dot{V}O_{2\,max}$. Therefore it appears that the intensity at which exercise is performed may influence the effect of a carbohydrate supplement on the rate of decline in muscle glycogen.

Possibly accounting for this difference in muscle glycogen response are the differences in the plasma glucose and insulin responses elicited by a carbohydrate supplement during high- and low-intensity exercise. Ingestion of carbohydrate supplements during low-intensity exercise (i.e., <50% $\dot{V}O_{2\,max}$) increases plasma glucose and insulin concentrations and maintains these parameters at elevated levels throughout the exercise bout (1, 28). In contrast, carbohydrate supplementation during high-intensity exercise (i.e., 65–75% $\dot{V}O_{2\,max}$) only aids in the maintenance of plasma glucose, while the plasma insulin concentration actually declines (15).

Two possibilities can account for the reduced decline in muscle glycogen that occurred during the HC treatment: *1*) the HC treatment resulted in muscle glycogen synthesis during exercise as a result of the elevated

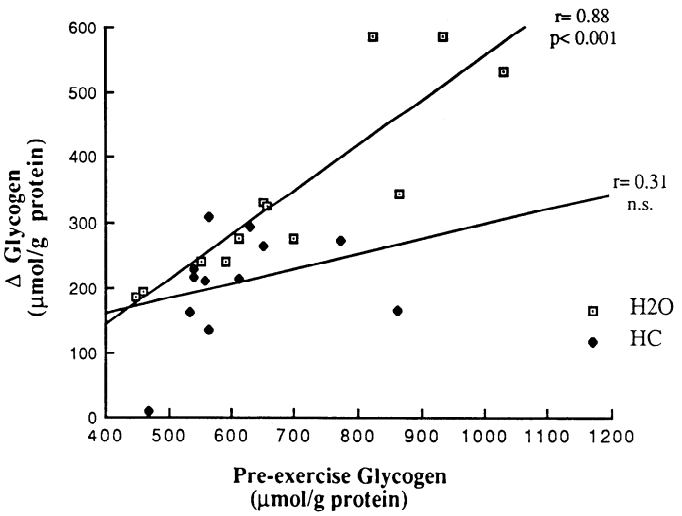

FIG. 6. Correlation between preexercise muscle glycogen concentration and muscle glycogen utilization for the H_2O and HC treatments.

plasma glucose and insulin levels, and 2) the HC treatment increased the reliance on blood glucose and slowed the rate of muscle glycogenolysis. Substantial evidence indicates that muscle glycogen synthesis can occur in glycogen-depleted muscle during low-intensity exercise if a carbohydrate supplement is supplied (12, 29). However, Kuipers et al. (29) found that the synthesis was restricted to type II fibers, indicating that only inactive fibers are capable of replenishing their glycogen stores. In the present study, histochemical analysis of the postexercise muscle biopsies indicated that the HC treatment, compared with the H_2O treatment, resulted in a substantial reduction in muscle glycogen utilization in the type I fibers but not in the type II fibers. Thus it appears that the HC treatment reduced the decline in muscle glycogen during low-intensity exercise by reducing the rate of muscle glycogen utilization rather than stimulating glycogen synthesis in inactive muscle fibers.

It was noted, however, that the average preexercise glycogen concentration was slightly higher during the H_2O trial than during the HC trial. It has been demonstrated that the rate of muscle glycogenolysis is directly related to the muscle glycogen concentration (16, 37, 41). Therefore the difference between the rates of decline in muscle glycogen during the H_2O and HC trials may have been a consequence of the difference in initial muscle glycogen levels. To address this concern, a Pearson product-moment correlation coefficient was conducted between the preexercise muscle glycogen concentrations and the Δglycogens for both the H_2O and HC trials. As shown in Fig. 6, a strong relationship existed between the initial muscle glycogen concentration and the rate of muscle glycogenolysis during the H_2O trial, which agrees with previous observations (16, 37, 41). In contrast, during the HC trial a nonsignificant relationship was found for these variables, suggesting that the HC treatment weakened the relationship between the preexercise muscle glycogen concentration and its rate of breakdown. Therefore this analysis further supports our contention that the HC treatment reduced the rate of decline in muscle glycogen and that the difference in rates of glycogenolysis between the HC and H_2O treatments was not due to differences in preexercise muscle glycogen concentrations.

In conclusion, the results of this study indicate that a fluid replacement drink containing 8.5% carbohydrate can regulate body temperature and maintain fluid homeostasis as effectively as H_2O during prolonged low-intensity exercise in the heat. In addition, an 8.5% carbohydrate supplement provides a sufficient amount of carbohydrate to prevent a decline in carbohydrate oxidation and possibly slow the rate of muscle glycogen depletion.

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