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Applied nutritional investigation

Ingestion of a high-molecular-weight hydrothermally modified waxy maize starch alters metabolic responses to prolonged exercise in trained cyclists

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ABSTRACT

Objective: We examined whether the ingestion of a hydrothermally modified starch (HMS) would alter metabolic and hormonal responses to prolonged cycling compared with maltodextrin (MAL).
Methods: Nine male cyclists (30 ± 2 y, 79.2 ± 2.1 kg, 4.7 ± 0.1 L of O_2/min, 7.5 ± 1.3 y training) fasted 10 h before cycling for 150 min at 70% peak oxygen consumption and completing a cycling-to-exhaustion trial at 100% peak oxygen consumption. Participants ingested 1g/kg of HMS or MAL 30 min before and within 10 min of completing the bout. Blood samples were provided every 15 min before, during, and 90 min after exercise. Expired gases were collected every 30 min during exercise. In a crossover, randomized, and double-blind fashion, identical testing was completed 1 wk later.
Results: Primary findings from this study were that 1) increases in serum glucose were greater during MAL (peak 9.5 mM) versus HMS (peak 7.4 mM, $P \leq 0.01$), 2) insulin levels were significantly lower during HMS (peak 2.5 μIU/mL) versus MAL (peak 20.3 μIU/mL, $P < 0.001$), and 3) HMS was associated with greater fat breakdown as indicated by the increased serum non-esterified fatty acids ($P < 0.01$) and glycerol levels ($P < 0.05$).
Conclusion: Ingestion of a low-glycemic HMS before prolonged cycling exercise blunted the initial spike in serum glucose and insulin and increased the breakdown in fat compared with MAL.

Introduction

Maintenance of normal blood glucose concentrations during long duration (>2-h) exercise is critical for supporting endurance performance [1–3]. Ingestion of carbohydrate (CHO) becomes an important source of glucose to prevent hypoglycemia and maintain high CHO oxidation rates later in exercise when muscle and liver glycogen stores are reduced. Although fast absorbing CHO (e.g., glucose, sucrose, maltodextrin) are commonly emphasized and facilitate CHO oxidation, they simultaneously inhibit mobilization and oxidation of fat due in part to their greater stimulation of insulin. Lipolysis is exquisitely sensitive to insulin, such that even small decreases in insulin within physiologic ranges are associated with marked increases in adipose tissue fatty acid release and oxidation [4]. Therefore, a CHO source that is digested and absorbed at a slower rate may have desirable effects on metabolic and hormonal responses to exercise.

Some controversy exists, however, with regard to different CHO sources and their effects on hormonal responses and subsequent exercise performance [5]. For instance, glucose consumed up to 1 h before exercise has been shown to maintain glucose availability during long duration bouts, but has also raised questions about its impact on decreasing performance and blood glucose in response to hyperinsulinemia. Commercial processing and preparation of a starch, however, have been shown to affect its glycemic and insulin response. For example, Johannsen and Sharp [2] reported elevated CHO oxidation rates throughout the first 90 min of exercise for endurance-trained men cycling at approximately 66.4% peak aerobic capacity (Vo_{2peak}) after ingesting 1 g/kg of dextrose or an acid-/alcohol-modified high-amylose cornstarch compared with a placebo or an unmodified high-amylose resistant cornstarch. Furthermore, CHO oxidation under the modified starch condition remained elevated for up to 120 min of exercise, whereas CHO oxidation

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rates were lowered under the dextrose condition in a manner similar to that of the other two trial conditions. Nonetheless, although other forms of starch have been used previously to determine their ability to affect performance [5,6], metabolic regulation [2], and recovery of glycogen [5,7] in response to endurance exercise, no studies exist investigating a hydrothermally modified starch (HMS).

Hydrothermal treatment of corn starch (*Zea mays*, or maize starch) is a novel processing technique that was developed as a therapeutic treatment for children with glycogen storage disease, a rare genetic disorder characterized by an inability to convert glycogen to glucose in the liver. Use of an HMS in this patient population has been shown to be superior to conventional treatments in preventing hypoglycemia over extended periods [8,9]. In addition, recent research has indicated that ingesting a formula containing slow-digesting CHOs can lower postprandial glucose responses, attenuate insulin changes, and promote higher levels of glucagon-like peptide-1 [10], whereas daily ingestion over a 6-mo period of a high sucrose (high-glycemic) diet can impair glucose uptake and increase enzymes related to lipogenesis [11]. Because limited data exist with HMS in endurance athletes, the purpose of this investigation was to compare the metabolic and hormonal effects of a novel HMS with maltodextrin (MAL) when consumed in two separate 1-g/kg doses before and after a prolonged cycling bout (i.e., 150-min submaximal cycling at ~70% Vo_{2peak}). We hypothesized the HMS would result in less of a spike in blood glucose and insulin and an increase in adipose lipolysis and fat oxidation during exercise and recovery. A secondary objective was to examine the effects on perceived exertion, heart rate, and exercise performance.

Materials and methods

Subjects

Nine healthy competitive male cyclists ($n = 9$, age 30 ± 2 y, height 179.6 ± 1.1 cm, weight 79.2 ± 2.1 kg, experience 7.5 ± 1.3 y, Vo_{2peak} 4.7 ± 0.2 L/min, ventilatory threshold [VT] 80.7 ± 1.4% Vo_{2peak}) volunteered to participate in a randomized, crossover, double-blind study investigating the effects of two CHO sources on metabolic and performance measurements when consumed before and immediately after long-duration cycling to exhaustion. All subjects completed comprehensive medical history questionnaires before being accepted into the test. Subjects were excluded if they were not able to achieve a Vo_{2peak} ≥4.00 L/min and a VT occurring at ≥75% of Vo_{2peak} or they consumed nutritional supplements affecting metabolism (e.g., >100 mg/d of caffeine, green tea catechins, ephedrine alkaloids, guggulsterone, etc.) and/or muscle mass (e.g., creatine, protein/amino acids, androstenedione, dehydroepiandrosterone, etc.) within 3 mo of starting the study. This study was approved by the University of Oklahoma (Norman, OK, USA) institutional review board for human subjects, and written informed consent was obtained from all participants before testing.

Peak oxygen consumption test

To ensure all subjects were highly trained, a preliminary cycling test was performed to assess Vo_{2peak} and VT. Failure to achieve an absolute Vo_{2peak} ≥4.00 L/min and a VT occurring at ≥75% of Vo_{2peak} resulted in exclusion from the study. Subjects' body weights were measured to the nearest 0.01 kg with the participants wearing only their cycling shorts; height was measured to the nearest 0.1 cm. Subjects were fitted with an electronic heart rate monitoring device and were then asked to complete a 30-W/min ramping exercise test to exhaustion on an electronically braked cycle ergometer (Corival 906900, Lode B.V. Medical Technology, Groningen, The Netherlands) to determine Vo_{2peak}, peak power output, and the percentage of Vo_{2peak} at which VT was reached [12]. Respiratory gases were monitored and continuously analyzed with open-circuit spirometry using a calibrated metabolic cart and the manufacturer's software (True One 2400 Metabolic Measurement System, Parvo-Medics, Inc., Provo, UT, USA). Data were averaged over 15-s intervals, with the highest 15-s oxygen consumption rate recorded as Vo_{2peak}. Power output during Vo_{2peak} testing began at 100 W for 3 min and increased 1 W every 2 s (30 W/min) until the cyclist could no longer maintain a pedaling cadence greater than 50 revolutions/min or until the subject signaled to end the test. Subjects included in the test were asked to keep a 2-d food log before the first long-duration exercise trial and to replicate the diet for subsequent tests.

Testing protocol

Approximately 1 wk after the inclusion/exclusion test, eligible subjects reported to the laboratory after a 10-h fast (i.e., caffeine, tea, coffee, soda, sports drinks, dietary supplements, over-the-counter medications, or anything other than water was to be avoided during that time). Briefly, a flexible catheter was inserted into an arm vein and a pre-exercise blood sample was obtained (−30 min). Participants then randomly ingested 1 g/kg of MAL or an HMS (UCAN Co., Woodbridge, CT, USA) dissolved in 350 mL of water. The HMS was derived from waxy maize with a high amylopectin content (>95%), high molecular weight (500 000 to 700 000 g/mol), and low osmolality. Subjects rested quietly for 30 min and additional blood samples were taken at −15 min and immediately before exercise (0 min). Participants then mounted their bicycles, which had been rear-wheel mounted to a Computrainer PRO indoor cycling trainer (RacerMate, Seattle, WA, USA), and began exercising at a workload prescribed to elicit 70% of their Vo_{2peak}. Respiratory gases were collected for up to 10 min after exercise initiation to ensure the proper intensity was achieved and stabilized. Maintenance of the exercise intensity was reassessed with subsequent expired gas analyses at 30-min intervals and continuous heart rate response monitoring. After 150 min of steady state exercise, the workload was increased to elicit 100% Vo_{2peak} until the subject could no longer maintain a minimum pedal cadence of 50 revolutions/min or the subject's power output decreased greater than 10% below the prescribed workload. Participants were allowed water ad libitum, and fans were provided within the climate-controlled laboratory (i.e., approximately 23°C, 40% humidity) to help maintain body temperature during exercise. After the long-duration exercise test, participants dismounted their bikes and immediately ingested a second bolus of their assigned supplement at a dose of 1 g/kg to assess the effects of CHO ingestion on recovery. Subjects rested in a seated position for a 90-min recovery period. Blood samples were collected every 15 min and expired gases were collected every 30 min during the exercise period. Approximately 1 wk after the testing condition, participants returned to the laboratory and completed an identical testing session when they ingested the alternate supplement in a crossover fashion.

Blood collection and analysis

Blood collection

Venous blood samples (~5 mL) were collected into an untreated plastic syringe from the right forearm of participants using standard catheterization techniques. After each blood collection, samples were injected into serum separator tubes and centrifuged at room temperature for 15 min at 1600 × *g*. The serum supernatant from each sample was subsequently transferred into a microcentrifuge tube and stored at −80°C for subsequent metabolite analyses.

Serum metabolite/hormone analyses

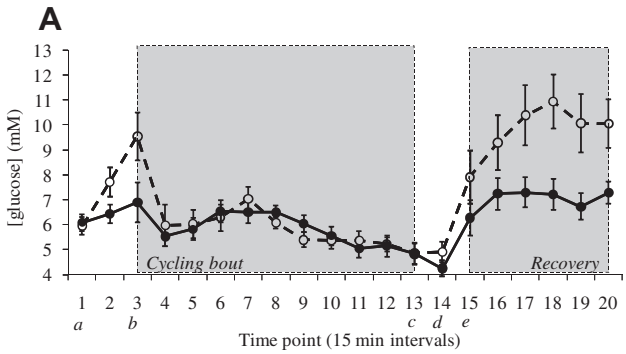
Stored serum samples were assayed in duplicate for glucose, glycerol, nonesterified fatty acids (NEFAs), insulin, and cortisol concentrations. Cortisol concentrations were analyzed because of their purported role as a counter-regulatory

Table 1
Subject demographics and dietary (energy and macronutrient) intake before each condition*

Demographics ($n = 9$)		Calorie/macronutrient intake	Before HMS	Before MAL	*P*
Age (y)	30 ± 2	24-h kcal/d	3446 ± 377	3472 ± 489	0.96
Weight (kg)	79.2 ± 2.1	24-h carbohydrate (g/d)	473 ± 45	478 ± 86	0.96
Training (y)	7.5 ± 1.3	24-h protein (g/d)	110 ± 12	107 ± 14	0.82
Vo_{2peak} (L/min)	4.7 ± 0.2	24-h fat (g/d)	127 ± 23	127 ± 23	0.96

HMS, hydrothermally modified starch; MAL, maltodextrin; Vo_{2peak}, peak oxygen consumption

* Values are expressed as mean ± SE. Between-trial means were compared using paired-samples *t* tests.



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Time (range)	Glucose (mM)		
	HMS	MAL	p-value
PreEx + Ex (*185 min)	1140 ± 60	1204 ± 68	0.22
Recovery (*75 min)	***608 ± 43***	***841 ± 80***	***0.001***
Total (*260 min)	***1747 ± 99***	***2045 ± 142***	***0.005***

Fig. 1. Serum glucose response to HMS (closed circles) versus MAL (open circles) before and after cycling bouts (A) and area under the curve analysis for different time ranges (B). Values are expressed as mean ± SE. (A) Intervals between time points spanned 15-min periods. a, Fasting blood before first drink ingestion (1 g/kg of body mass); b, onset of cycling bout; c, blood before time to exhaustion; d, blood immediately after time to exhaustion; e, blood 15 min after second drink ingestion (1 g/kg of body mass). (B) Between-trial values were compared using paired-samples *t* tests. Significant differences between trials ($P \leq 0.05$) are italicized. HMS, hydrothermally modified starch; MAL, maltodextrin; PreEx + Ex, before exercise plus exercise.

hormone in maintaining blood glucose concentrations over prolonged exercise bouts [13]. An enzymatic oxygen-rate analyzer (Analox GM7, Analox Instruments USA, Inc., Lunenburg, MA, USA) was used to analyze serum glycerol and glucose concentrations. Spectrophotometric assays were used to analyze serum NEFAs (Roche Diagnostics Corporation, Indianapolis, IN, USA), and enzyme immunoassays were used to analyze insulin (Diagnostic Systems Laboratories, Webster, TX, USA) and cortisol (DRG International, Inc., Mountainside, NJ, USA) concentrations. Assay precision (i.e., coefficient of variation [CV]) and accuracy (i.e., percentage of unrecovery, which is the percentage deviation from 100%) were calculated using glucose, glycerol, and NEFA control samples by eight replicate determinations per analyte. Likewise, CV and accuracy were examined for insulin analysis using a control serum provided by the manufacturer. The CV and percentage of unrecovery using an 8.0-mM glucose control serum were 2.0% and 0.2%, respectively. The CV and percentage of unrecovery using a 240-μM glycerol control serum were 2.5% and 8.9%, respectively. The CV and percentage of unrecovery using a 0.35-mM NEFA control serum were 1.2% and 3.6%, respectively. The intra-assay and interassay (i.e., plate-to-plate) CVs using a 25-μIU/mL insulin control serum were 5.9% and 6.4%, respectively. The intra-assay and inter-assay (i.e., plate-to-plate) CVs using a 100-ng/mL cortisol control serum were 2.3% and 10.5%, respectively. All precision and accuracy of these assays are in accordance with previously accepted guidelines [14].

Statistical analysis

Area under the curve (AUC) analysis was used to calculate the absolute concentrations for serum glucose, glycerol, NEFA, and insulin concentrations during the two long-duration exercise tests. Specifically, AUC for each of these parameters was calculated during three phases of time: 1) minutes −30 to 155 (i.e., before exercise plus exercise [PreEx + Ex] AUC), 2) minutes 155 to 245 (i.e., recovery AUC), and 3) minutes −30 to 245 (total AUC). Between-session differences in glucose, glycerol, NEFA, and insulin AUCs were compared using dependent *t* tests. Between-session metabolite and hormone AUC values were compared using dependent *t* tests because other investigators have allocated similar statistical approaches with crossover, counterbalanced studies [15]. All expiratory gas data (i.e., respiratory exchange ratios [RERs] and fuel oxidation rates) during exercise (i.e., minutes 0 to 155) were also compared using dependent *t* tests. All data in figures and tables are expressed as mean ± standard error of the mean, and the significance for all statistical analyses was determined using an α level of 0.05.

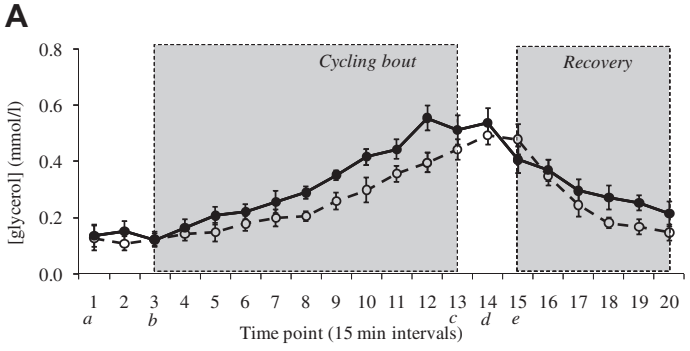
Results

Subject demographics and calorie intakes

Subject demographics and macronutrient profiles before each testing session are presented in Table 1. There were no significant differences in caloric/macronutrient intake 24 h before each condition ($P > 0.05$).

Serum metabolite and hormonal AUC responses

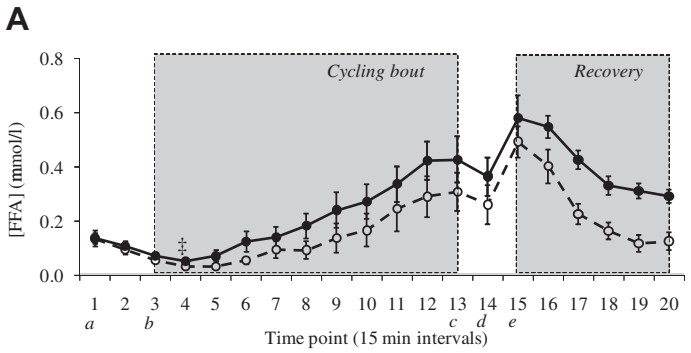
Between-condition comparisons of serum glucose, glycerol, NEFA, insulin, and cortisol concentrations are presented in Figures 1 to 5. Serum glucose concentrations were elevated during the MAL trial as evidenced through greater recovery



B

Time (range)	Glycerol (mM)		
	HMS	MAL	p-value
PreEx + Ex (*185 min)	***60 ± 4***	***47 ± 5***	***0.012***
Recovery (*75 min)	30 ± 3	26 ± 3	0.10
Total (*260 min)	***90 ± 7***	***74 ± 7***	***0.018***

Fig. 2. Serum glycerol response to HMS (closed circles) versus MAL (open circles) before and after cycling bouts (A) and area under the curve analysis for different time ranges (B). Values are expressed as mean ± SE. (A) Intervals between time points spanned 15-min periods. a, Fasting blood before first drink ingestion (1 g/kg of body mass); b, onset of cycling bout; c, blood before time to exhaustion; d, blood immediately after time to exhaustion; e, blood 15 min after second drink ingestion (1 g/kg of body mass). (B) Between-trial values were compared using paired-samples *t* tests. Significant differences between trials ($P \leq 0.05$) are italicized. HMS, hydrothermally modified starch; MAL, maltodextrin; PreEx + Ex, before exercise plus exercise.



B

Time (range)	NEFA (mM)		
	HMS	MAL	p-value
PreEx + Ex (185 min)	***40 ± 8***	***27 ± 7***	***0.022***
Recovery (*75 min)	***38 ± 4***	***24 ± 3***	***0.008***
Total (*260 min)	***78 ± 10***	***51 ± 10***	***0.006***

Fig. 3. Serum NEFA response to HMS (closed circles) versus MAL (open circles) before and after cycling bouts (A) and area under the curve analysis for different time ranges (B). Values are expressed as mean ± SE. (A) Intervals between time points spanned 15-min periods. a, Fasting blood before first drink ingestion (1 g/kg of body mass); b, onset of cycling bout; c, blood before time to exhaustion; d, blood immediately after time to exhaustion; e, blood 15 min after second drink ingestion (1 g/kg body mass). (B) Between-trial values were compared using paired-samples *t* tests. Significant differences between trials ($P \leq 0.05$) are italicized. FFA, free fatty acid; HMS, hydrothermally modified starch; MAL, maltodextrin; NEFA, non-essential fatty acid; PreEx + Ex, before exercise plus exercise.

AUC and total AUC values ($P < 0.05$; Fig. 1). Serum glycerol concentrations were elevated during the HMS trial as evidenced through greater PreEx + Ex AUC and total AUC values ($P < 0.05$; Fig. 2). Serum NEFA concentrations were elevated during the HMS trial as evidenced through greater PreEx + Ex AUC, recovery AUC, and total AUC values ($P < 0.05$; Fig. 3). Serum insulin concentrations were elevated during the MAL trial as evidenced through greater PreEx + Ex AUC, recovery AUC, and total AUC values ($P < 0.05$; Fig. 4). There were no differences in serum cortisol AUC values between trials (Fig. 5). Total metabolite and hormone AUC values between trials are summarized in Figure 6.

Fuel oxidation rates and RER during exercise

Table 2 presents estimates of CHO and fat oxidation rates in addition to RERs during exercise as assessed by indirect calorimetry. There were no significant differences between trials with regard to CHO oxidation rates, fat oxidation rates, and/or RER values during the 150-min exercise bout. RER values did, however, approach statistical significance ($P = 0.07$) in the HMS condition compared with MAL approximately 60 and 90 min into the exercise bout, respectively. These values are supported by near significance values for higher fat oxidation in the HMS condition compared with MAL at approximately 60 min ($P = 0.08$) and 90 min ($P = 0.07$), respectively.

Heart rate and perceived exertion during exercise

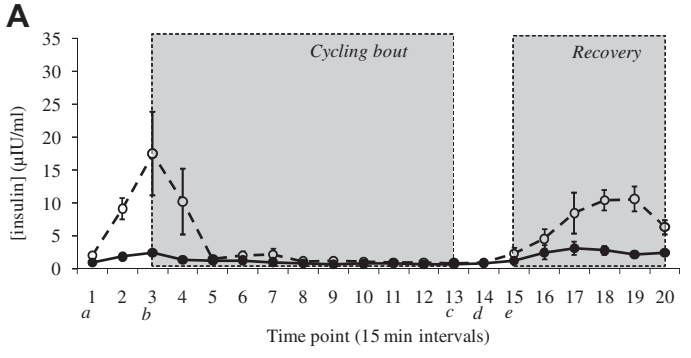
Heart rate and ratings of perceived exertion using a Borg scale were assessed at 30-min intervals during the 150-min prolonged cycling bout. Heart rate was significantly greater 30 min into the exercise bout during the MAL condition compared with HMS (HMS 150 ± 3 beats/min, MAL 154 ± 3 beats/min, $P = 0.047$). There were no significant differences between trials with regard to ratings of perceived exertion at each respective time point (data not shown).

Time trial performance

Upon completing the 150-min cycling bout, cyclists performed a time trial at 100% Vo_{2peak} to fatigue. Paired-samples *t* tests revealed that there was no difference between the HMS and MAL trials (HMS 125 ± 28 s, MAL 136 ± 27 s, $P = 0.66$).

Discussion

Nutrition provided before, during, and after exercise has profound effects on many physiologic processes and remains an active area of research owing to the myriad commercial products available and different dietary strategies used by athletes. This study compared two CHO sources with very different chemical structures on metabolic and hormonal responses to prolonged

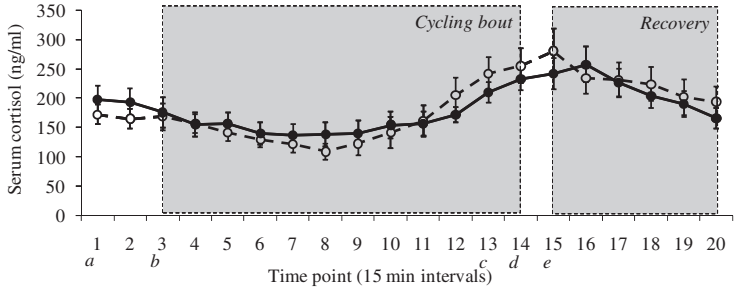


B

Time (range)	Insulin (μIU/ml)		
	HMS	MAL	p-value
PreEx + Ex (*185 min)	***286 ± 41***	***827 ± 183***	***0.012***
Recovery (*75 min)	***240 ± 45***	***636 ± 119***	***0.002***
Total (*260 min)	***525 ± 79***	***1463 ± 296***	***0.005***

Fig. 4. Serum insulin response to HMS (closed circles) versus MAL (open circles) before and after cycling bouts (A) and area under the curve analysis for different time ranges (B). Values are expressed as mean ± SE. (A) Intervals between time points spanned 15-min periods. a, Fasting blood before first drink ingestion (1 g/kg of body mass); b, onset of cycling bout; c, blood before time to exhaustion; d, blood immediately after time to exhaustion; e, blood 15 min after second drink ingestion (1 g/kg of body mass). (B) Between-trial values were compared using paired-samples *t* tests. Significant differences between trials ($P \leq 0.05$) are italicized. HMS, hydrothermally modified starch; MAL, maltodextrin; PreEx + Ex, before exercise plus exercise.

A



B

Time (range)	Cortisol (ng/ml)		
	HMS	MAL	p-value
PreEx + Ex (*185 min)	32,178 ± 3,396	31,146 ± 3,168	0.59
Recovery (*75 min)	12,478 ± 1,310	13,056 ± 1,711	0.57
Total (*260 min)	51,954 ± 5,117	52,075 ± 5,468	0.96

Fig. 5. Serum cortisol response to HMS (closed circles) versus MAL (open circles) before and after cycling bouts (A) and area under the curve analysis for different time ranges (B). Values are expressed as mean ± SE. (A) Intervals between time points spanned 15-min periods. a, Fasting blood before first drink ingestion (1 g/kg of body mass); b, onset of cycling bout; c, blood before time to exhaustion; d, blood immediately after time to exhaustion; e, blood 15 min after second drink ingestion (1 g/kg of body mass). (B) Between-trial values were compared using paired-samples *t* tests. Significant differences between trials ($P \leq 0.05$) are italicized. HMS, hydrothermally modified starch; MAL, maltodextrin; PreEx + Ex, before exercise plus exercise.

exercise in competitive cyclists. Compared with MAL, pre- and postexercise ingestion of a heat-moisture treated waxy maize starch significantly affected the physiologic response to prolonged cycling and recovery from exercise as evidenced by 1) more stable serum glucose levels, 2) markedly blunted insulin levels, and 3) increased serum NEFAs and glycerol levels. Serum cortisol appeared to be minimally different between conditions.

Similar to the present investigation, rapid elevations in serum glucose (i.e., ~7.5 mM) and insulin (i.e., ~80 μIU/mL) have been previously found immediately after ingesting a dextrose (high-glycemic) solution at a dosage of 1 g/kg 30 min before exercise [16]. These investigators also reported that when subjects ingested a waxy starch (100% amylopectin), serum glucose (i.e., ~6.5 mM) and insulin (i.e., ~35 μIU/mL) responses significantly increased in response to baseline values but were significantly lower when compared with ingestion of the high-glycemic (dextrose) beverage. The findings of Goodpaster et al. [16] and the present study potentially reflect the compositional differences between HMS and MAL, with the former being a high-molecular-weight CHO source consisting of approximately 95% amylopectin and 5% amylose and the latter being a small, linear, six to eight glucose residue structure formed from starch hydrolysis. In a somewhat counterintuitive fashion and under previous experimental scenarios, hydrothermal treatment of a waxy starch yields a starch with an extremely high amylopectin content but exhibits a lower glycemic response and attenuated postprandial release [8,9]. In this respect, the findings of the present study relative to changes in glucose and insulin values are supported by previous exercise and clinical research [8,9,16].

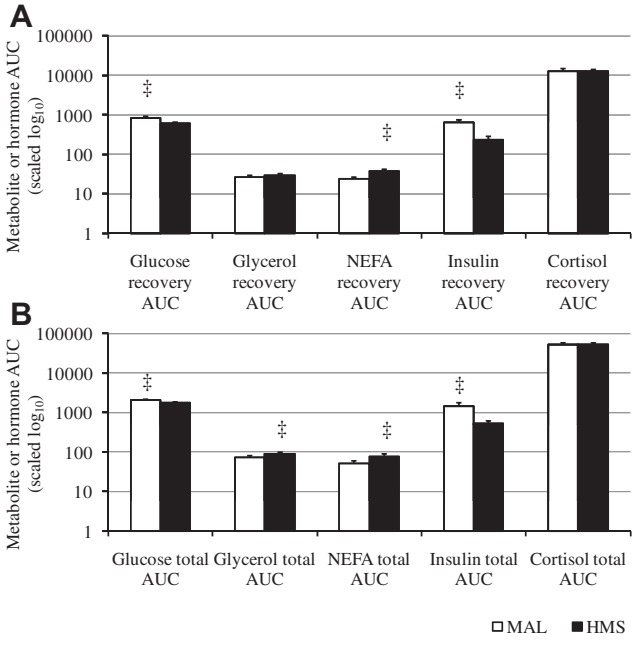


Fig. 6. Summaries of recovery (A) and total (B) metabolite and hormone AUC comparisons between trails. Values are expressed as mean ± SE and the *y*-axis is scaled to $\log_{10}$ values. Further, units for these values can be found in Figures 1 to 5. ‡ $P < 0.05$, significantly greater than other trial. AUC, area under the curve; HMS, hydrothermally modified starch; MAL, maltodextrin; NEFA, non-essential fatty acid.

Subjects in the present investigation also showed lower glycemic and insulinemic responses during the HMS trial versus MAL trial as assessed by AUC calculations over the respective 260-min testing sessions, although there were no statistical differences in the glucose and/or AUC values during the exercise bout (i.e., 0 to 155 min; data not shown). Although Goodpaster et al. [16] did not quantify blood glucose responses using AUC analysis, their findings parallel these results in that serum glucose and insulin concentrations exhibited differential responses immediately after ingestion and before exercise (i.e., MAL > HMS) and quickly normalized during the prolonged exercise bout (i.e., MAL = HMS). As mentioned previously, previous data have suggested that high amylopectin starches, especially those that were hydrothermally treated, are structurally resilient to hydrolysis and subsequent digestion [8,9,16]. Furthermore, visceral blood flow has been shown to be substantially decreased (~30%) in animals exercising at steady state compared with resting conditions [17]. Hence, although it is foreseeable that ingesting a "slowly digestible" starch before exercise may be disadvantageous for sustaining blood glucose concentrations during the later stages of exercise, data from the present study and other similar investigations [16,18] suggest that ingesting modified starches with different amylose and amylopectin contents before prolonged exercise elicits similar glycemic responses during exercise compared with higher-glycemic beverages.

Participants also ingested a second bolus (1 g/kg of body-weight) of each test drink immediately after the time-to-exhaustion bout at 100% Vo_{2peak}. In this instance, postprandial serum glucose and insulin concentrations peaked 45 to 60 min after ingestion of the postexercise bolus, an effect that was likely

Table 2
Fuel oxidation rates and RER comparisons between HMS and MAL trials*

Time range	Carbohydrate oxidation (g/min)			Fat oxidation (g/min)			RER (V_{CO_2}/V_{O_2})		
	HMS	MAL	*P*	HMS	MAL	*P*	HMS	MAL	*P*
0–5 min	0.32 ± 0.09	0.39 ± 0.12	0.60	0.04 ± 0.03	0.02 ± 0.05	0.70	0.93 ± 0.06	0.99 ± 0.09	0.56
25–30 min	2.94 ± 0.18	3.13 ± 0.13	0.35	0.36 ± 0.07	0.26 ± 0.04	0.27	0.93 ± 0.01	0.95 ± 0.01	0.29
55–60 min	2.73 ± 0.16	3.07 ± 0.16	0.11	0.45 ± 0.08	0.29 ± 0.05	0.08	0.92 ± 0.01	0.95 ± 0.01	0.07
85–90 min	2.64 ± 0.17	2.97 ± 0.16	0.15	0.51 ± 0.08	0.34 ± 0.06	0.07	0.91 ± 0.01	0.94 ± 0.01	0.07
115–120 min	2.39 ± 0.17	2.52 ± 0.16	0.52	0.58 ± 0.08	0.47 ± 0.07	0.20	0.89 ± 0.01	0.91 ± 0.01	0.23
145–150 min	2.40 ± 0.21	2.44 ± 0.18	0.84	0.56 ± 0.09	0.50 ± 0.08	0.55	0.89 ± 0.02	0.90 ± 0.01	0.52

HMS, hydrothermally modified starch; MAL, maltodextrin; RER, respiratory exchange ratio; V_{CO_2}, carbon dioxide production per unit time; V_{O_2}, oxygen consumption per unit time

* Values are expressed as mean ± SE. Between-trial means were compared using paired-samples *t* tests.

due to the gradual redistribution of blood to the viscera after prolonged, exhaustive exercise [17]. Nonetheless, MAL elicited a greater glycemic/insulinemic response during the recovery period as assessed by AUC analyses. Although some studies in the literature contend that ingesting high-glycemic CHOs after exercise is more beneficial for intramuscular glycogen resynthesis (reviewed by Kerksick et al. [19]), previous investigations that employed prolonged endurance exercise (~70–75% $V_{O_{2peak}}$) and provided 100 to 300 g of 100% amylopectin [5] or 78% amylopectin [7] during the postexercise period reported significantly greater resynthesis of lost muscle glycogen. Nonetheless, although the postexercise ingestion of HMS in the present study increased serum glucose and insulin concentrations, the present data do not provide any indication of an impact over resynthesis of muscle glycogen.

The HMS ingestion was also associated with greater fat breakdown during exercise and recovery as indicated by significantly increased serum NEFAs and glycerol AUC values. The present findings are identical to the results from Goodpaster et al. [16] who also demonstrated that serum glycerol levels were significantly greater during the later stages (i.e., 75–90 min) of steady-state exercise after ingesting a 100% amylopectin starch before exercise versus glucose ingestion. Although Goodpaster et al. did not assess NEFA concentrations during exercise, our findings suggest that elevations in NEFA concentrations also mirror glycerol patterns with between-condition differences (i.e., HMS > MAL) existing from 75 min until the cessation of exercise. Cyclists tended to oxidize more fat (55–60 min, HMS > MAL, $P = 0.08$; 85–90 min, HMS > MAL, $P = 0.07$) during the middle portion of each respective exercise bout when ingesting HMS versus MAL, although these differences were not statistically significant. This response occurred even though there were largely no differences in serum glucose concentrations during the exercise bout (glucose concentrations were significantly greater during the HMS condition 75 min into the cycling bout). Collectively, it is our contention that ingesting HMS may help to spare glycogen during prolonged endurance exercise. Nonetheless, a body of evidence exists suggesting that the consumption of different types and/or dosages of CHO do not alter glycogen utilization kinetics [1,20,21]. Thus, it cannot be definitely concluded that the modest, non-significant increase in fat oxidation that we observed during the HMS condition suggests that muscle glycogen oxidation was spared during the HMS trail. Therefore, future investigations that examine pre- to postexercise intramuscular glycogen concentrations and/or isotopically enriched CHO boluses (to assess exogenous CHO oxidation rates) are needed to confirm our hypothesis that HMS may affect this parameter.

Interestingly, circulating concentrations of glycerol and NEFAs during the recovery period were significantly greater during the HMS condition as assessed by AUC analyses. These findings are likely due to the attenuated insulinemic response occurring after the ingestion of the second postexercise beverage during the HMS trial. In this regard, Boden et al. [22] used the hyperinsulinemic-euglycemic clamp technique coupled with the infusion of isotopically labeled fatty acids to demonstrate that supraphysiologic concentrations of insulin decreased fatty acid release into the circulation by 71%. Although the present investigation did not use such tracer methodologies, NEFA levels followed a similar trend because they were 48% to 64% lower during the MAL versus HMS condition 45 to 90 min into the recovery period. Similarly, circulating insulin concentrations were 114% to 429% greater during the MAL versus HMS condition at these same respective time points.

Cyclists in the present investigation exhibited similar time-to-exhaustion values during both conditions during the 100% $V_{O_{2peak}}$ sprint that immediately followed the 150-min submaximal bout. Therefore, these data support the contention that high amylopectin starches may be just as effective as other high-glycemic beverages at sustaining short periods of maximal effort bursts after prolonged exercise.

Conclusions

In summary, our findings suggest that ingesting a low-glycemic HMS before prolonged cycling exercise blunted the initial spike in serum glucose and insulin and preserved a short-burst maximal performance measurement after a prolonged cycling bout. The differences in fatty acid breakdown patterns (assessed by circulating NEFA and glycerol) in HMS compared with MAL warrant future research to determine the extent to which altered fatty acid cycling may influence the usage of other fuel sources (e.g., glycogen, intramuscular triacylglycerols, other triacylglycerols, etc.) during prolonged exercise bouts. For example, HMS may differentially affect glycogen kinetics compared with MAL, with this suggestion being challenged in recent reports [1,20,21]. Similarly, potential interest exists regarding any role HMS ingestion may play as a component of weight loss or weight maintenance, but future research involving caloric manipulation with an HMS must first be completed.

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