

# Effect of Preexercise Ingestion of Modified Cornstarch on Substrate Oxidation During Endurance Exercise

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The purpose of this study was to investigate differences in substrate oxidation between dextrose (DEX) and unmodified (UAMS) and acid/alcohol-modified (MAMS) cornstarches. Seven endurance-trained men ( $\text{VO}_{2\text{peak}} = 59.1 \pm 5.4 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) participated in 2 h of exercise ( $66.4\% \pm 3.3\% \text{ VO}_{2\text{peak}}$ ) 30 min after ingesting 1 g/kg body weight of the experimental carbohydrate or placebo (PLA). Plasma glucose and insulin were elevated after DEX ( $P < 0.05$ ) compared with UAMS, MAMS, and PLA. Although MAMS and DEX raised carbohydrate oxidation rate through 90 min of exercise, only MAMS persisted throughout 120 min ( $P < 0.05$  compared with all trials). Exogenous-carbohydrate oxidation rate was higher in DEX than in MAMS and UAMS until 90 min of exercise. Acid/alcohol modification resulted in augmented carbohydrate oxidation with a small, sustained increase in exogenous-carbohydrate oxidation rate. MAMS appears to be metabolizable and available for oxidation during exercise.

**Key Words:** carbohydrate metabolism, low glycemic index, isotopic ratio

Endurance performance is associated with the ability to maintain euglycemia and high rates of carbohydrate oxidation while exercising. Although high-glycemic-index carbohydrate feedings before exercise augment carbohydrate oxidation rates, the resultant insulin surge and rebound hypoglycemia during exercise have been implicated in performance detriments caused by elevated rates of muscle glycogenolysis (1, 4, 11) and marked reductions in hepatic glucose output (16) and free-fatty-acid availability (2, 4, 5, 6, 8, 10, 13, 14, 23, 24, 26, 28). Studies that fed slowly digestible carbohydrates before exercise demonstrated either negligible or slightly elevated increases in carbohydrate oxidation rates, depending on the rate of digestibility of the fed carbohydrate, without significant insulinemic responses (9, 21, 23, 24, 26). Studies that fed either moderately digestible carbohydrate sources or apparently low-glycemic-index meals, however, had similar carbohydrate oxidation rates and reductions in rebound hypoglycemia during exercise (4, 9, 10, 13, 14). These results imply that there might be an ideal glycemic index that provides adequate exogenous plasma glucose to maintain high rates of carbohydrate oxidation and improves plasma glucose homeostasis without influencing fatty-acid mobilization or hepatic glucose output.

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Types 2 (raw) and 3 (retrograde) resistant starches, classified by Englyst et al. (3), are reported to be approximately 60–70% digestible and slowly absorbed, resulting in only minimal plasma glucose and insulin disturbances after ingestion (25, 29). Isotopic-ratio studies using naturally labeled  $^{13}\text{C}$  (corn starch) have shown that Types 2 and 3 resistant starches are slowly digested and absorbed in the gastrointestinal tract and are oxidized during normal resting metabolism (25). Acid/alcohol modification of high-amylose, Type 2 resistant cornstarch (MAMS; US Patent # 5,695,803; amylose:amylopectin = 70%:30%) results in a carbohydrate source that is approximately 92% digestible in rats, with minimal insulin secretion (29). Because this particular starch is developed from an unmodified high-amylose cornstarch (Amylomaize VII; UAMS), MAMS contains glucose naturally enriched with the stable  $^{13}\text{C}$  isotope. Recent technological and methodological advances have enabled researchers to determine the contribution of exogenous glucose to carbohydrate oxidation during exercise (17, 19, 22).

The purpose of this study was to outline metabolic responses of corn-derived glucose (DEX) and unmodified (65% digestible) and modified (92% digestible) amylo maize cornstarch fed 30 min before 2 h of moderate-intensity exercise. Furthermore, we used isotope-ratio analysis to determine differences in absorption and oxidation between high- (glucose) and low- (MAMS and UAMS) glycemic-index carbohydrates and between UAMS and MAMS. We hypothesized that preexercise MAMS ingestion would provide additional exogenous glucose during exercise, resulting in carbohydrate oxidation rates similar to those with DEX ingestion; augment carbohydrate oxidation late in exercise; and preserve plasma glucose homeostasis late in exercise.

## Methods

### Subjects

Seven healthy elite cyclists volunteered to participate in a randomized, repeated-measures, double-blind study consisting of four 2-h intervention trials investigating the effects of carbohydrates of varying glycemic index ingested before exercise on glucose oxidation. Mean ( $\pm$  standard deviation) age, weight, and peak oxygen consumption ( $\text{VO}_{2\text{peak}}$ ) were  $24 \pm 4$  y,  $74.9 \pm 14.6$  kg, and  $59.1 \pm 5.4$  mL $\cdot$ kg $^{-1}\cdot$ min $^{-1}$ , respectively. This study was approved by the Iowa State University institutional review board, and written informed consent was obtained from all participants before testing. Medical-history questionnaires were completed and reviewed before any physical activity assessments were conducted.

### Preliminary Measurements

After height and weight were measured, participants completed a graded exercise test to exhaustion on an electronically braked cycle ergometer (Lode BV, Groningen, The Netherlands) to determine aerobic capacity ( $\text{VO}_{2\text{peak}}$ ). Respiratory gases were analyzed with computer-interfaced oxygen and carbon-dioxide analyzers calibrated with standardized gases (Physio-dyne Instrument Corp, Quogue, NY). Power output during the  $\text{VO}_{2\text{peak}}$  test began at 100 W and increased incrementally by 50 W every 3 min until the cyclists signaled they were approaching exhaustion,

at which time the power output increased 25 W until they could no longer maintain a comfortable pedaling rate. Initial measurements were completed at least 1 wk before the first of four 2-h cycling trials.

Each subject was instructed to keep a 3-d diet diary before the first trial and to replicate the diet in subsequent weeks. Because we used stable-isotope analysis for the exogenous-carbohydrate analyses, participants were required to follow a low-corn ( $^{13}\text{C}$  foods in general) diet to control endogenous  $^{13}\text{C}$  levels. A list of foods that should be avoided, with reasonable substitutions, was given to all participants. To allow for adequate rest, trials were separated by at least 1 wk, and participants were instructed to abstain from vigorous exercise for at least 24 h before each testing session.

## Experimental Protocol

Participants entered the lab on the morning of the trial after a 10-h overnight fast. After they rested for 10 min in a seated position, respiratory gases were analyzed and a preingestion breath sample was collected in standard gas-collection tubes (Labco Limited, High Wycombe, Buckinghamshire, UK). Fullness index (0 = *not full at all*, 10 = *very full*) and rating of perceived exertion (RPE) were recorded during the last minute of the 8-min resting, steady state. After the preingestion breath sample was collected, a flexible catheter (BD-Angiocath, Sandy, UT) was inserted into an antecubital vein and a preingestion blood sample was drawn, immediately injected into a heparinized tube (Monoject, 143 U.S.P. heparin), mixed, and placed on ice until centrifugation. The catheter was kept patent with sterile isotonic saline after each blood sample. Participants then ingested 1 of 3 solutions containing 1g/kg body weight of corn-derived dextrose (DEX), unmodified high-amylose cornstarch (UAMS: GI  $\approx$  25), or acid/alcohol-modified high-amylose cornstarch (MAMS: GI  $\approx$  29) in flavored, sugar-free media (mL of media = body weight/0.19 up to 350 mL) or a fourth beverage containing an equal volume of sugar-free media (PLA). Twenty-five minutes after the ingestion of the experimental beverage, participants mounted their cycle ergometers and rested for 5 more minutes. Preexercise breath and blood samples were collected during the last minute of this rest period. Exactly 30 min after ingesting the experimental beverage, the cyclists began to exercise at a power output prescribed to elicit 60% of their  $\text{VO}_{2\text{peak}}$ . A protocol similar to pre-exercise was followed every 25 min until conclusion of the exercise; subsequently, breath and blood samples were collected 30, 60, 90, and 120 min after the start of exercise. Fullness index and RPE were recorded each time a breath and blood sample were taken. Participants were allowed water ad libitum, and fans were provided to maintain body temperature during the entire exercise session.

## Breath-Gas and Blood Analyses

Breath-gas samples were sent to Metabolic Solutions, Inc (Nashua, NH), for analysis of  $^{13}\text{CO}_2$  isotopic ratio in the expired air. Rates of expired carbon-dioxide production ( $\text{VCO}_2$ ) and oxygen consumption ( $\text{VO}_2$ ) were used to calculate substrate oxidation rates by stoichiometric equations (15). The total amount of carbohydrate (TotCHO) and fat oxidized was calculated as area under the curve versus time.

The following equation shows Mosora's calculation for exogenous-carbohydrate oxidation rate ( $M_{\text{exo}}$ ) (17):

$$M_{\text{exo}} \text{ (g/min)} = V\text{CO}_2 \text{ (L/min)} \times (\delta^{13}\text{C}_{\text{PDB-EXP}} - \delta^{13}\text{C}_{\text{PDB-REF}}) / (\delta^{13}\text{C}_{\text{PDB-CHO}} - \delta^{13}\text{C}_{\text{PDB-REF}}) \times (1/k) \quad (1)$$

In this equation,  $\delta^{13}\text{C}_{\text{PDB}}$  represents isotopic ratio ( $^{13}\text{C}:^{12}\text{C}$ ), which is calculated for the background reference (REF), carbohydrate (CHO), and breath sample (EXP) compared with the Pee Dee Belemnite standard value (PDB);  $V\text{CO}_2$  is the average steady-state volume of expired carbon dioxide for the sample; and  $k$  is the amount of  $\text{CO}_2$  provided by the oxidation of glucose (0.7426 L  $\text{CO}_2/\text{g}$ ). The total quantity of exogenous carbohydrate oxidized ( $\text{Tot}M_{\text{exo}}$ ) was calculated as area under the curve for  $M_{\text{exo}}$  versus time. Mosora's calculations assume that the background reference enrichment of  $^{13}\text{C}$  is constant (17), but, as demonstrated by Peronnet (19), during exercise stored glycogen containing significant amounts of  $^{13}\text{CO}_2$  might contribute to an increase in  $^{13}\text{CO}_2:^{12}\text{CO}_2$  ratio, thus inflating the actual contribution of exogenous carbohydrate to energy production. To account for this, we considered the corresponding time point during the PLA trial to be  $\delta^{13}\text{C}_{\text{PDB-REF}}$ .

Blood samples were centrifuged at  $300 \times g$  for 10 min, and plasma was stored at  $-20^\circ\text{C}$  until analyzed. Plasma glucose (Sigma glucose dehydrogenase colorimetric assay/Beckman Spectrophotometer) and insulin concentrations (Sigma RIA/Packard Cobra-II automated gamma counter) were measured in duplicate.

Five grams of each experimental carbohydrate were sent to Metabolic Solutions, Inc, for carbohydrate isotopic-ratio ( $\delta^{13}\text{C}_{\text{PDB-CHO}}$ ) determination. Reference values for DEX, UAMS, and MAMS were  $-12.57$ ,  $-12.31$ , and  $-12.71$ , respectively.

## Statistical Analysis

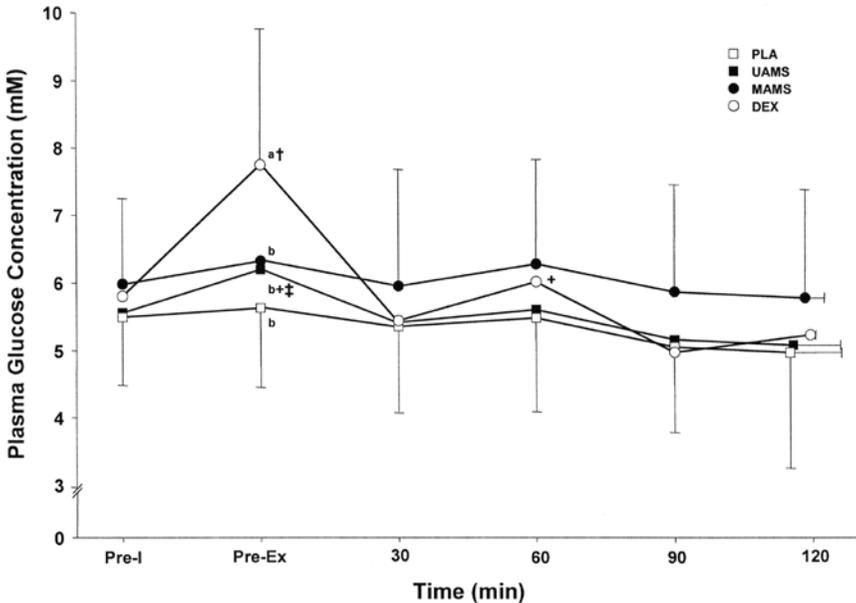
All data were analyzed using JMP (SAS Institute Inc, Cary, NC) statistical software. In 1 participant, total mass of exogenous carbohydrate oxidized was higher for the 3 carbohydrate-feeding trials than what was fed before exercise, so his data were removed from  $^{13}\text{C}$  analysis ( $n = 6$ ); however, all other data were acceptable and remained in the analyses ( $N = 7$ ). Substrate oxidation and plasma responses from the 4 trials were examined using 2-factor (trial and time) repeated-measures analysis of variance (ANOVA). Specific time and trial responses were investigated further using Tukey's HSD (honestly significant difference) post hoc analyses. Because of potentially large differences between rest and exercise values within a trial, substrate oxidation variables were divided into rest (preingestion and preexercise) and exercise (EX30, 60, 90, and 120) and analyzed with separate ANOVAs. Substrate oxidation during rest did not change and was omitted from the Results section; however, resting substrate oxidation was left in the figures to provide baseline information. Substrate oxidation variables include carbohydrate ( $\text{CHO}_{\text{ox}}$ ) and fat oxidation ( $\text{FAT}_{\text{ox}}$ ) rates,  $\delta^{13}\text{C}_{\text{PDB-EXP}}$  and exogenous-carbohydrate oxidation rate ( $M_{\text{exo}}$ ). Plasma variables include glucose and insulin concentrations. Anthropometric data, preingestion data, average exercising fullness index, RPE, oxygen-consumption rate ( $\text{VO}_2$ ), and respiratory-exchange ratio (RER), as well as total carbohydrate ( $\text{TotCHO}$ ), exogenous ( $\text{Tot}M_{\text{exo}}$ ) carbohydrate, and total fat

oxidized, were analyzed using 1-way repeated-measures ANOVAs with differences between trials subjected to Tukey's HSD post hoc analyses. Exercise data for RPE, fullness index,  $\text{VO}_2$ , and RER were averaged because no significant time effects were observed. All data are reported as mean  $\pm$  standard deviation, and significant differences were determined at  $P \leq 0.05$ .

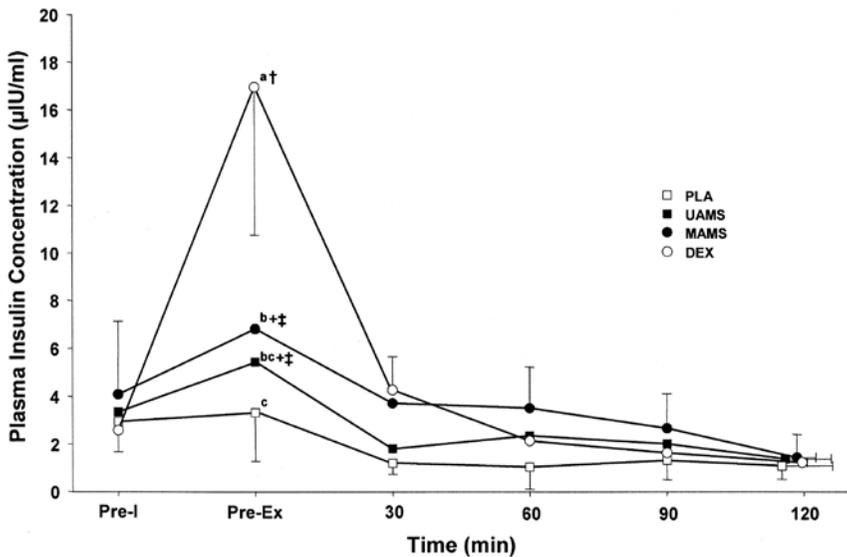
## Results

### Plasma Glucose and Insulin Responses

Plasma glucose showed a significant time-by-trial interaction ( $P < 0.001$ ) because of a normal plasma glucose-response curve after DEX ingestion (Figure 1). No other trials had significant changes in plasma glucose. Higher plasma glucose concentrations before exercise in DEX resulted in a significant rise in plasma insulin immediately before exercise compared with all other trials ( $17.0 \pm 6.2$  vs.  $3.3 \pm 2.1$ ,  $5.4 \pm 3.2$ , and  $6.8 \pm 2.4$   $\mu\text{IU/mL}$  for DEX vs. PLA, UAMS, and MAMS, respectively; time-by-trial interaction  $P < 0.001$ ; Figure 2). A small, significant rise in plasma insulin was also seen after ingestion of MAMS ( $P < 0.05$  compared with PLA). All plasma glucose and insulin concentrations had returned to baseline after 30 min of exercise. Plasma glucose during exercise in DEX further declined after



**Figure 1** — Change in plasma glucose concentration ( $n = 7$ ; mean  $\pm$  standard deviation) during rest and exercise. There was a significant effect of time ( $P < 0.001$ ) and a significant trial-by-time interaction ( $P < 0.001$ ) for plasma glucose concentration. Dissimilar letters indicate difference between trials; †, difference from all other time points; +, difference from 90 min; and ‡, difference from 120 min of exercise within a trial. Standard-deviation bars have been removed to prevent obscuring internal data points.



**Figure 2** — Change in plasma insulin concentration ( $n = 7$ ; mean  $\pm$  standard deviation) during rest and exercise. There were significant effects of trial ( $P = 0.002$ ) and time ( $P < 0.001$ ) and trial-by-time interaction ( $P < 0.001$ ) for plasma insulin concentration. Dissimilar letters indicate difference between trials; †, difference from all other time points; ‡, difference from 90 min; and ‡, difference from 120 min of exercise within a trial. Standard-deviation bars have been removed to prevent obscuring internal data points.

90 min of exercise compared with 60 min (Figure 1). In addition, plasma insulin concentrations fell after 90 min of exercise in both MAMS and UAMS compared with preexercise levels ( $P < 0.05$ ; Figure 2).

## Substrate Oxidation

The prescribed cycling workload elicited an average of  $66.4\% \pm 3.3\%$  of  $VO_{2peak}$  and was not different between trials ( $P = 0.56$ ). Although each exercise trial was set for 2 h, some participants stopped exercise early. Mean time at the end of exercise was similar for all 4 trials ( $115 \pm 11$ ,  $116 \pm 10$ ,  $118 \pm 4$ , and  $120 \pm 1$  min for PLA, UAMS, MAMS, and DEX, respectively;  $P = 0.71$ ). Trials that were not finished were halted by the participants. Four of the participants stopped exercise early, but each participant finished at least 2 of the 4 trials. Six of the total 28 trials (4 treatments with 7 participants) were not finished: 1 trial each for MAMS and DEX and 2 trials each for PLA and UAMS. No trial effects were found for mean  $VO_{2,}$  RPE, or fullness index during the 2-h exercise period ( $P = 0.56$ ,  $0.39$ , and  $0.13$ , respectively; Table 1), but mean exercise RER was different by trial ( $P = 0.04$ ). Post hoc analyses revealed no significant differences between trials for mean RER during exercise.

$CHO_{ox}$  during exercise was higher during the first hour after MAMS and DEX compared with both PLA and UAMS, but during the second hour of exercise,

**Table 1 Descriptive Variables During 2 h of Exercise at 66.4% ± 3.3% VO<sub>2peak</sub><sup>a</sup>**

	Placebo	UAMS	MAMS	Dextrose
VO <sub>2</sub> (L/min)	2.87 ± 0.31	2.93 ± 0.26	2.90 ± 0.31	2.89 ± 0.32
Respiratory-exchange ratio	0.83 ± 0.02	0.83 ± 0.04	0.87 ± 0.04	0.86 ± 0.02
Rating of perceived exertion	14 ± 2	15 ± 3	13 ± 3	14 ± 2
Fullness index	2 ± 1	1 ± 1	1 ± 1	1 ± 1

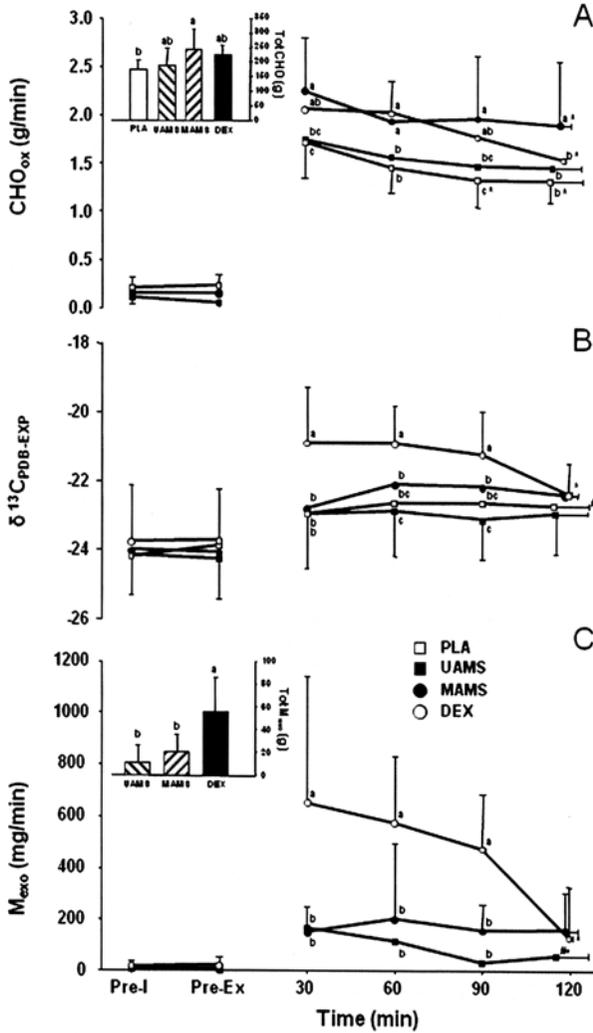
<sup>a</sup>Sample  $N = 7$ . Individual averages during exercise were used to calculate the mean for each trial. Measurements were taken 30 min after ingestion of 1 g/kg of unmodified (UAMS) or modified high-amylose cornstarch (MAMS), dextrose, or noncaloric, sweetened placebo. Responses were similar for all trials.

CHO<sub>ox</sub> declined rapidly in DEX (Figure 3A). As a result, CHO<sub>ox</sub> only remained elevated in MAMS during the second hour of exercise ( $P < 0.05$  compared with all trials). Because CHO<sub>ox</sub> was elevated during the entire 2 h of exercise in MAMS, TotCHO ( $P = 0.03$ ; Figure 3A inset) was also higher after MAMS compared with PLA. Although not significant, TotCHO was also 22% higher in MAMS compared with UAMS and 16% and 21% higher in DEX compared with UAMS and PLA, respectively. Conversely, FAT<sub>ox</sub> was lower in MAMS and DEX through 60 min of exercise compared with both PLA and UAMS and rose significantly in DEX by 120 min (trial-by-time interaction  $P = 0.05$ ). Unlike TotCHO, however, post hoc analysis showed only a trend for differences in total fat (trial effect  $P = 0.06$ ). In all trials, CHO<sub>ox</sub> decreased and FAT<sub>ox</sub> increased significantly during exercise (time effect;  $P < 0.001$  for both).

Experimental isotopic-ratio ( $\delta^{13}\text{C}_{\text{PDB-EXP}}$ ) responses during each of the 4 trials are shown in Figure 3B. Effects of trial ( $P < 0.001$ ), time ( $P = 0.01$ ), and time-by-trial interaction ( $P < 0.001$ ) were observed, caused primarily by higher  $^{13}\text{CO}_2$  concentrations ( $\delta^{13}\text{C}$  closer to 0) in DEX at the onset of exercise that subsided by 120 min. Further analysis of the time-by-trial interaction showed that MAMS  $^{13}\text{CO}_2$  concentrations were also higher after 60 and 90 min of exercise compared with UAMS but not PLA. The total mass of exogenous carbohydrate oxidized had trial effects ( $P < 0.001$ ) caused by higher rates of  $M_{\text{exo}}$  in DEX compared with both MAMS and UAMS for at least 90 min of exercise ( $55 \pm 31$  vs.  $20 \pm 15$  and  $11 \pm 15$  g, respectively; Figure 3C inset). Like CHO<sub>ox</sub>, however,  $M_{\text{exo}}$  showed a rapid decline between 90 and 120 min of exercise in DEX (Figures 3A and 3C).

## Discussion

Dextrose ingestion resulted in a typical glucose-response curve, specifically, marked hyperglycemia and hyperinsulinemia that reportedly result in suppressed hepatic glucose (16) output and peripheral lipolysis (27) during subsequent exercise. High glucose and insulin concentrations, as seen in the dextrose trial, together with muscle contractions at the onset of exercise, also led to high rates of carbohydrate oxidation, probably because of enhanced rates of glucose disposal (4, 16, 27). On the other hand, a similar bolus of acid/alcohol-modified resistant starch before exercise prevented large disturbances in glucose and insulin homeostasis while at the same



**Figure 3** — Change in the rate of carbohydrate oxidation (Graph A;  $CHO_{ox}$ ;  $N = 7$ ; mean  $\pm$  standard deviation),  $^{13}C$  isotopic ratio (Graph B;  $\Delta^{13}C_{PDB-EXP}$ ;  $n = 6$ ), and rate of exogenous-carbohydrate oxidation ( $M_{exo}$ ) after PLA subtraction (Graph C;  $n = 6$ ) during rest and exercise. Inset in Graph A represents total carbohydrate oxidized (TotCHO), and inset in Graph C represents total exogenous carbohydrate oxidized (Tot $M_{exo}$ ) during 120 min of exercise.  $CHO_{ox}$  during exercise differed by trial ( $P = 0.03$ ) and time ( $P < 0.001$ ) but showed only a trend for an interaction of trial and time ( $P = 0.08$ ). TotCHO also differed by trial ( $P = 0.03$ ). There were significant effects of trial ( $P < 0.001$ ) and time ( $P = 0.01$ ) and trial-by-time interaction ( $P < 0.001$ ) for  $^{13}C$  isotopic ratio during exercise. There were significant effects of trial ( $P < 0.001$ ) and time ( $P = 0.02$ ) and trial-by-time interaction ( $P < 0.001$ ) for  $M_{exo}$  during exercise. Tot $M_{exo}$  also differed by trial ( $P < 0.001$ ). Dissimilar letters indicate difference between trials; \*, difference from 30 min of exercise; #, difference from 60 min of exercise; and +, difference from 90 min of exercise within a trial. Standard-deviation bars have been removed to prevent obscuring internal data points.

time augmenting carbohydrate oxidation rate. This suggests that the modified starch provides a metabolizable glucose source and is consistent with results of Vonk et al. (25), who showed that, even under resting conditions, resistant starch is at least partially digested and contributes to total carbohydrate oxidation.

Additional evidence of a slow absorption profile is provided by measurement of exogenous-carbohydrate oxidation, which demonstrated a small but sustained increase after the modified-starch feeding. Exogenous-carbohydrate oxidation rate showed no decline toward the end of the exercise (120 min) after modified-starch feeding even though dextrose and unmodified-starch feedings had already returned to baseline ( $M_{\text{exo}}$  not significantly different from 0). This result, together with lower plasma glucose concentrations after 60 min of exercise and possible insulin-inhibited endogenous-fuel availability in DEX, could explain why total-carbohydrate oxidation rate decreased dramatically near cessation of exercise in DEX but remained elevated in MAMS. In addition, the absence of hyperinsulinemia with the modified-starch feeding could account for the apparently augmented endogenous-carbohydrate oxidation with the modified-starch feeding. Direct measurements of glucose kinetics and muscle-glycogen utilization in this study would have provided additional insight into the exact mechanism, but previous research in our lab has shown that this particular modified starch is able to raise total-carbohydrate oxidation without increasing muscle-glycogen utilization (C.J.L. Lockard, unpublished observations). In addition, data from other research groups using low-glycemic carbohydrates show either a glycogen-sparing effect (4, 12) or no effect (5, 11, 13) on glycogen use during endurance exercise, implicating greater hepatic glucose output and utilization after modified-starch ingestion.

Acid/alcohol modification of the high-amylose starch produces a smaller, partially hydrolyzed starch granule (7, 20) that is up to 92% digestible in rodents (29) yet retains its slow absorption profile as judged by the low glycemic and insulinemic responses after ingestion (Figures 1 and 2). Because total-carbohydrate oxidation (measured by indirect calorimetry) was increased by the same magnitude after the modified-starch trial as a preexercise dextrose feeding and knowing the low insulinemic properties of the modified starch, we expected that both exogenous- and endogenous-carbohydrate oxidation would be significantly augmented during exercise after MAMS ingestion. Peak exogenous-carbohydrate oxidation in this study was 0.65 g/min after 30 min of exercise with the ingestion of glucose but only rose to a peak of about 0.20 g/min after ingestion of a similar quantity of modified resistant starch. Because of the slow, but complete, digestibility of the modified starch, we expected lower, extended exogenous-carbohydrate oxidation rates that resembled relatively smaller, repeated glucose feedings. We think this might have been the case, but because of the natural variability in the measurement of  $^{13}\text{C}$  observed in all trials, actual exogenous-carbohydrate oxidation rates might have been obscured, especially in the modified-starch trial, in which the difference from the placebo is minimal.

The increased total-carbohydrate oxidation without a commensurately elevated exogenous-carbohydrate oxidation is not likely the result of systematic error. Each trial in this study was counterbalanced, appropriate calibration controls were used in all trials, and breath-collection and -analysis procedures were standardized. Nonetheless, it is possible that some subjects did not adequately replicate their pretrial diet, resulting in higher background  $^{13}\text{C}$  stores. Small elevations in background  $^{13}\text{C}$

can substantially affect calculated exogenous-carbohydrate oxidation rates, especially if they occur in a placebo trial. The increase in  $\delta^{13}\text{C}_{\text{PDB-EXP}}$  in PLA during exercise was greater than UAMS and rises almost as high as MAMS (Figure 3B). This effectively lowers all actual exogenous-carbohydrate oxidation rates because of higher concentrations of endogenous  $^{13}\text{C}$  in PLA compared with, at least, UAMS. Although we did not observe differences in resting  $^{13}\text{C}$ , indicating adequate dietary control immediately before the trial, and participant diet was similar 3 d before each trial, future studies should include an endogenous-carbohydrate-depleting bout of exercise before the trial followed by a standardized low-corn diet in order to reduce the possibility of endogenous- $^{13}\text{C}$  contamination.

A major concern with feeding large doses of slowly absorbed carbohydrate is the potential for gastric distress resulting from anaerobic-bacteria fermentation (18). As indicated by our fullness index, we found no evidence that would indicate gastric distress with either the modified or unmodified versions of the resistant starch during 2 h of exercise. We have also completed resting studies with the modified resistant starch in which gastric distress (feeling full or bloated, rumbling, abdominal pain, nausea, intestinal gas, or diarrhea) was evaluated 4 h postprandially and before bed. No adverse effects were noted except for small increases in intestinal rumbling and gas immediately before bed (unpublished observations). This indicates that the modified starch is well tolerated, especially within the time frame of normal postprandial exercise.

In conclusion, MAMS increased carbohydrate oxidation rate to the same extent as DEX without raising plasma glucose or insulin concentrations before exercise. More important, carbohydrate oxidation rate remained elevated after MAMS throughout the exercise, whereas after 120 min of exercise after DEX, carbohydrate oxidation rate had fallen to placebo levels. The additional carbohydrate oxidized in MAMS during the last 30 min of exercise appeared to have originated from both exogenous (Figure 3C) and endogenous sources. Methodological variability might have obscured differences in exogenous-carbohydrate oxidation with MAMS compared with UAMS. Future studies should include a glycogen-depleting bout of exercise and mandatory low- $^{13}\text{C}$  meals 3 d before trials to further reduce endogenous- $^{13}\text{C}$  concentrations, effectively improving the sensitivity for detection of small differences that might occur between modified and unmodified carbohydrates. In addition, because MAMS is slowly digested, a longer postprandial time before exercise is suggested. The potential glycogen-sparing effects or incorporation of exogenous glucose into muscle after ingestion of MAMS needs to be examined.

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