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Original Research

Pre-Exercise Maltodextrin Ingestion and Transient Hypoglycemia in Cycling and Running

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ABSTRACT

International Journal of Exercise Science 13(2): 1691-1704, 2020. This study examined the phenomenon of transient hypoglycemia and metabolic responses to pre-exercise carbohydrate (CHO) maltodextrin ingestion in cycling and running on the same individuals. Eleven active males cycled or ran for 30 min at 80% maximal heart rate (HRmax) after ingestion of either 1g/kg body mass maltodextrin (CHO-Cycle and CHO-Run respectively) or placebo (PL-Cycle and PL-Run) solutions. Fluids were ingested 30min before exercise in a double-blind and random manner. Blood glucose and serum insulin were higher before exercise in CHO (mean CHO-Cycle+CHO-Run) (Glucose: 7.4 ± 0.3 mmol·l^{-1}; Insulin: 59 ± 10 mU·l^{-1}) compared to placebo (mean PL-Cycle+PL-Run) (Glucose: 4.7 ± 0.1 mmol·l^{-1}; Insulin: 8 ± 1 mU·l^{-1}) (p<0.01), but no differences were observed during exercise among the 4 conditions. Mean blood glucose did not drop below 4.1 mmol·l^{-1} in any trial. However, six volunteers in CHO-Cycle and seven in CHO-Run experienced blood glucose concentration $\leq$ 3.5 mmol·l^{-1} at 20min of exercise and similar degree of transient hypoglycemia in both exercise modes. No association was found between insulin response to maltodextrin ingestion and drop in blood glucose during exercise. Blood lactate increased with exercise more in cycling compared to running, and plasma free fatty acids (FFA) concentrations were higher in placebo compared to CHO irrespective of exercise mode (p<0.01). The ingestion of maltodextrin 30min before exercise at about 80% HRmax produced similar glucose and insulin responses in cycling and running in active males. Lactate was higher in cycling, whereas maltodextrin reduced FFA concentrations independently of exercise mode.

KEY WORDS: Mode of exercise, supplementation, carbohydrate, insulin, glucose

INTRODUCTION

It has long been recognized that during prolonged strenuous exercise the body's carbohydrate (CHO) stores are seriously challenged and depletion of this important substrate is associated with fatigue (2). Therefore, sportsmen are advised to consume adequate amounts of CHO before an endurance event in order to optimize muscle and especially after an overnight fast that reduces liver glycogen stores (39). However, many investigations have shown that when CHO are consumed within the hour before cycling hyperinsulinemia and hyperglycemia occurs at the onset of exercise accompanied by a rapid fall of blood glucose to hypoglycemic levels ($\leq$3.5 mmol·l^{-1}) (9, 10, 12, 15, 18, 23, 24, 26). This phenomenon has been termed "rebound", "reactive" or "transient" hypoglycemia (20, 26). On the other hand, other studies also in cycling have not reported transient hypoglycemia (1, 13, 14, 25, 27, 36, 38). Fewer studies have examined transient hypoglycemia in running, with some finding evidence of the phenomenon and some have not (7, 28, 40). It seems that some individuals are prone to more sever blood glucose perturbations during cycling or running as a result of pre-exercise CHO ingestion (18, 19, 25, 26, 28, 30).

Investigations in cycling have reported a correlation between pre-exercise insulin concentrations and the fall of blood glucose during exercise (23, 24). Recently it was reported that subjects who developed transient hypoglycemia had an enhanced insulin response to pre-exercise CHO intake (25). Furthermore, some investigators have suggested that higher insulin sensitivity may contribute to transient hypoglycemia, whereas others have not reported such a relationship (19, 26). No such correlations or observations, however, have been reported in running. To the best of the authors' knowledge the phenomenon of transient hypoglycemia has not been studied directly in cycling and running on the same individuals. The sudden drop of blood glucose may be the result of an enhanced muscle glucose uptake due to the synergistic action of muscle contraction and hyperinsulinemia before exercise and a reduced hepatic glucose production (27). When the exercising muscle mass is increased glucose uptake by the working muscles is reduced, possibly in order to protect against premature hypoglycemia (35). Also, when a large muscle mass is active mobilization of blood glucose exceeds peripheral glucose uptake due to a sharp increase of plasma catecholamines (22). Bearing in mind that in running a higher muscle mass is involved compared to cycling, one may hypothesize that in running transient hypoglycemia might be less compared to cycling (29).

Furthermore, the pre-exercise hyperinsulinemia and the accompanied drop of blood glucose have also been associated with an increased muscle glycogenolysis (9, 15). This is accompanied by a depression of free fatty acids (FFA) (10, 12, 13, 23, 24, 27, 28, 36, 38) and lipolysis (17), and a premature onset of fatigue (12). On the other hand, the majority of studies have reported either no effect or an improvement in endurance performance as a result of pre-exercise CHO feeding (33). However, FFA, which are important substrates to the working muscle during prolonged exercise, and glycerol, that is a good indicator of lipolysis, have not been studied after pre-exercise CHO ingestion in cycling and running (16, 32). In addition, various forms of CHO have been employed as pre-exercise feedings (33). Nevertheless, maltodextrin, that has a high glycemic index and expected to produce hyperinsulinemia before exercise and transient hypoglycemia, has not been used (10, 14). This nutrient is commonly included in combination

with other CHO in sports drink formulations taken during exercise or used in mouth rinsing experiments during exercise to stimulate oral cavity receptors and central drive reward in an attempt to improve performance (4, 8).

The purpose of this study was to examine the prevalence of transient hypoglycemia and the insulin, FFA and glycerol responses of pre-exercise maltodextrin ingestion in cycling and running on the same individuals.

METHODS

After an overnight fast, volunteers cycled or ran for 30-min at 80% maximal heart rate (HRmax) after ingestion of either 1g/kg body mass (BM) maltodextrin (CHO-Cycle and CHO-Run respectively) or placebo (PL-Cycle and PL-Run) solutions. Trials were separated by five-seven days. Fluids were ingested 30-min before exercise in a double-blind and random way. The CHO-Cycle, CHO-Run, PL-Cycle and PL-Run trials were assigned the numbers 1-4 respectively. For every participant a computer randomized number sequence 1-4 was allocated. Diet and physical activity before each condition were also controlled.

Participants

Initially, 14 active males were recruited, however, three dropped out during the preliminary testing due to time availability. Eleven (age: 25.5 ± 3.2 years, height: 175.7 ± 2.0 cm, body mass: 76.5 ± 1.9 kg) completed all four trials. Participants were healthy, took no supplements and engaged in aerobic exercise at least three times a week for a minimum of 30-min. Volunteers were fully informed about the experimental procedures and possible risks involved before signing a written consent form and the study had the approval of the Ethical Committee of the University. This research was carried out fully in accordance to the ethical standards of the International Journal of Exercise Science (31). The sample size was estimated using G*Power software v. 3.1.9.7 based on blood glucose responses after administration of CHO before cycling and running, an α of 0.05 and statistical power $\beta = 0.80$ (7, 15).

Protocol

To identify the exercise intensity corresponding at 80% HRmax volunteers performed an incremental cycling test (Technogym, Artis, Cesena, Italy) to fatigue and also an incremental running test (ProForm 650, Ossett, UK) to fatigue. In cycling, the initial workload was 60-watt for 3-min and the workload was increased by 30-watt every 3-min until volunteers were unable to maintain a pedal speed of 50 revolutions/min. In running, the initial speed was 8-10 $km \cdot h^{-1}$ for 3-min on a level treadmill, depending on the running ability of the participant. Every 3-min, speed was increased by 1 $km \cdot h^{-1}$ until volitional fatigue. During incremental tests, heart rate was measured by telemetry (Polar T31, Kempele, Finland). Using regression analysis, the exercise intensity corresponding at 80% HRmax was estimated for both exercise modes. On separate visits participants exercised for 15-min on cycling and treadmill ergometers at the predicted 80% HRmax intensity, in order to verify the estimated intensity.

Participants weighed (Kenwood kitchen scale, UK) their food the day before the first main trial, and were asked to replicate this diet for the same time period before the subsequent three trials. No alcohol was allowed the day before each trial. Dietary records were analyzed based on published data and food labels (11). Furthermore, volunteers did not train two days before each trial.

Participants arrived at the laboratory at 08:00 hours after a 10-12 hour fast. A catheter was inserted in an antecubital vein and 10-ml venous blood sample and duplicated 25 µl of capillary blood samples were obtained. Afterwards, volunteers ingested either 1 g/kg BM maltodextrin (My Protein, The Hut.com Ltd., Northwich, UK) dissolved in water to produce a 15% CHO solution, or an equivalent volume of a placebo solution made of aspartame and sugar free orange juice. Maltodextrin is colorless and tasteless, but has a high glycemic index (> 90). To make the two solutions indistinguishable, aspartame and sugar free orange juice were also added in the maltodextrin solution. The fluids were prepared and provided by an independent researcher randomly to ensure double-blind conditions.

Thirty minutes after ingestion a further 10-ml venous and duplicated 25 µl of capillary blood samples were obtained. Then, participants cycled or ran for 5-min at 60% HRmax and then the exercise intensity was set at 80% HRmax for 30-min. Every 5-min during exercise, heart rate (HR) was measured and volunteers indicated their rating of perceived exertion (RPE) (3). Abdominal discomfort (AD) was also assessed on a scale ranging from 0 (completely comfortable) to 10 (unbearable pain). At 10-min, 20-min, and at the end of exercise 10-ml venous and duplicated 25 µl of capillary blood samples were obtained. Before and at the end of exercise, 0.7µl of capillary blood samples were also taken to measure lactate concentration. All trials were conducted under similar laboratory conditions regarding temperature (23-24 ^{o}C) and relative humidity (43-46 %).

Serum insulin was determined using an electrochemiluminescence immunoassay kit (Elecsys Insulin, Roche Diagnostics, Indianapolis, USA) on an automatic analyzer (Cobas e411, ROCHE Diagnostics GMBH, Mannheim, Germany). Plasma glycerol and FFA were measured photometrically only before and after exercise using commercially available kits (Randox Laboratories Ltd, Crumlin, UK). Capillary blood samples were dispensed into Eppendorf vials containing 250 µl of 2% Perchloric acid, mixed well, centrifuged and stored at -80 ^{o}C. The two deproteinized supernatants obtained for each time point were analyzed on an microplate reader (Molecular Devices, Versa max, San Jose, CA, USA) for glucose (Randox Laboratories Ltd, Crumlin, UK). Lactate was determined using an automated lactate analyzer (Lactate Plus, Nova Biomedical, Waltham, Massachusetts, USA). Using the fasting blood glucose and insulin data, the Quantitative Insulin Sensitivity Check Index (QUICKI), an indirect index of insulin sensitivity, was also calculated (21).

Statistical Analysis

Data were analyzed using SPSS Version 23. Three-way analysis of variance (ANOVA) (Exercise Mode x Fluid x Time) with repeated measures on one factor (time) were used to analyze differences in glucose, lactate, FFA, glycerol, insulin, HR, RPE, and AD. To examine post-pre

exercise changes in lactate, FFA, and glycerol, two-way ANOVA (Exercise Mode x Fluid) was used. Environmental conditions and diet were compared between trials by one-way ANOVA. Bonferroni adjustment for multiple comparisons was used to locate significant differences between mean values. Association between insulin and glucose concentrations was examined using Pearson correlation coefficient (r). Effect size was estimated by calculating partial eta squared (η^2). Also, 95% confidence intervals (95%CI) of the difference between means in the different conditions are reported. Data are reported as mean ± SE, and statistical significance was set at $p < 0.05$.

RESULTS

The HRmax in the incremental cycling test was 187 ± 3 b·min^{-1} and was lower ($p<0.001$) compared to that achieved in running (197 ± 3 b·min^{-1}). The maximal workload achieved in cycling was 284 ± 13 watt and in running the highest speed averaged 14.8 ± 0.4 km·h^{-1}. The intensity corresponding to 80% HRmax was 200 ± 10 watt and 10.5 ± 0.3 km·h^{-1} in cycling and running respectively.

Dietary record analysis showed no differences between conditions in energy ($p = 0.42$, $\eta^2 = 0.08$), carbohydrate ($p = 0.47$, $\eta^2 = 0.07$), fat ($p = 0.30$, $\eta^2 = 0.12$), or protein intake ($p = 0.65$, $\eta^2 = 0.04$) the day before each trial.

A mode X fluid effect was found in HR responses ($p=0.03$, $\eta^2=0.22$). Mean HR during exercise was higher in CHO-Run (161 ± 3 b·min^{-1}) and corresponded to 82 ± 1 %HRmax compared to PL-Run (156 ± 3 b·min^{-1}; 79 ± 1%). Similarly, averaged HR in CHO-Cycle (155 ± 3 b·min^{-1}) that corresponded to 83 ± 1 %HRmax was higher to PL-Cycle (145 ± 3 b·min^{-1}; 78 ± 1%).

Average RPE was similar between conditions (CHO-Cycle: 12 ± 1, CHO-Run: 11 ± 1, PL-Cycle: 11 ± 1, PL-Run: 11 ± 1). Average AD was not different between conditions and volunteers felt almost "completely comfortable" as indicated by the low AD responses (CHO-Cycle: 1.0 ± 0.0, CHO-Run: 1.4 ± 0.2, PL-Cycle: 1.1 ± 0.1, PL-Run: 1.1 ± 0.1).

Blood glucose responses are presented in Figure 1. The 3-way ANOVA revealed significant changes at Fluid ($p = 0.012$, $\eta^2 = 0.28$), Time ($p < 0.001$, $\eta^2 = 0.59$), and Fluid X Time ($p < 0.001$, $\eta^2 = 0.65$) levels, whereas no difference was observed at 3-way interaction ($p = 0.76$, $\eta^2 = 0.023$) or exercise mode (mean blood glucose for both cycling and running: 4.9 ± 0.2 mmol·l^{-1}, 95%CI: 4.4–5.4). Blood glucose was higher before exercise ($p < 0.01$) in both CHO (mean CHO-Cycle+CHO-Run: 7.4 ± 0.3 mmol·l^{-1}) compared to placebo trials (mean PL-Cycle+PL-Run: 4.7 ± 0.1 mmol·l^{-1}) (95%CI: 2.1–3.3). When maltodextrin was provided, mean blood glucose decreased markedly ($p < 0.001$) during exercise compared to the start (0-min), but did not significantly decrease below pre-ingestion levels or below 4 mmol·l^{-1} at any sampling point between trials. However, six volunteers in CHO-Cycle and seven in CHO-Run experienced transient hypoglycemia (≤ 3.5 mmol·l^{-1}) during exercise. The individual blood glucose concentrations during CHO-Cycle and CHO-Run are presented in Figure 2. With the exception of one person, the same six individuals who developed transient hypoglycemia in CHO-Run, also developed transient hypoglycemia

in CHO-Cycle. At 20-min of exercise blood glucose reached its nadir and for these individuals was 3.3 ± 0.1 mmol·l^{-1} in both CHO-Cycle and CHO-Run, whereas in those who experienced no blood glucose reduction was 5.4 ± 0.7 and 5.5 ± 1.0 mmol·l^{-1} respectively.

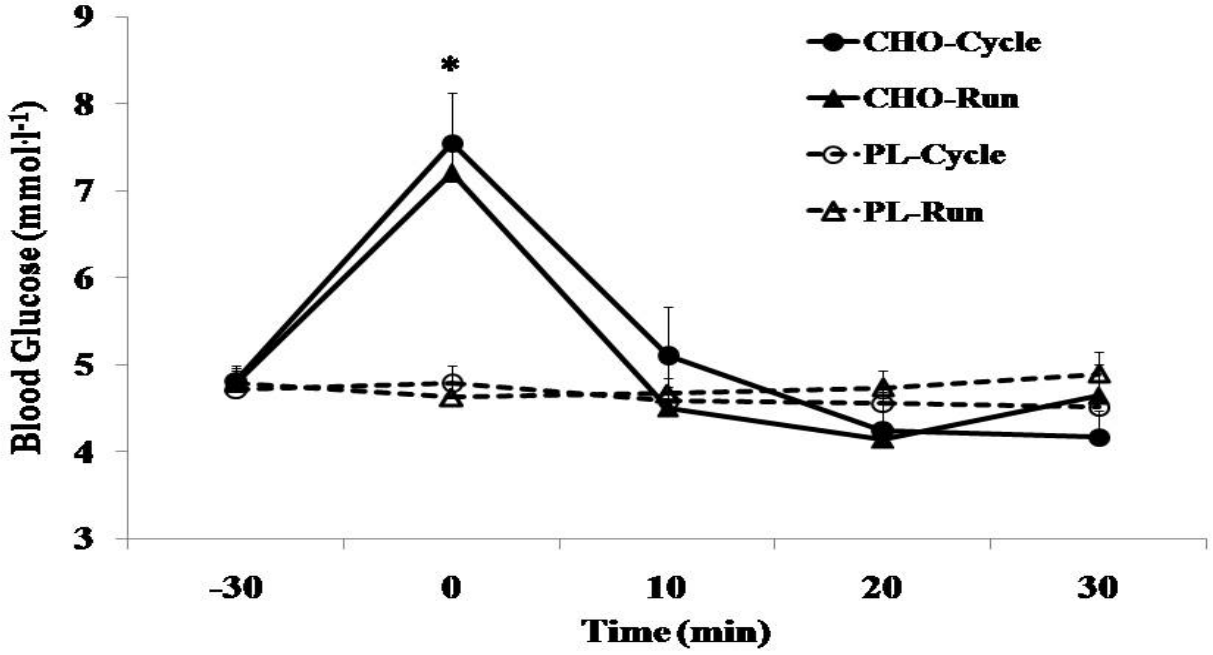

Figure 1. Blood glucose responses during the four trials (mean ± SE). *p< 0.001: Mean of both CHO (CHO-Cycle+CHO-Run) trials compared to mean of both Placebo (PL-Cycle+PL-Run) trials.

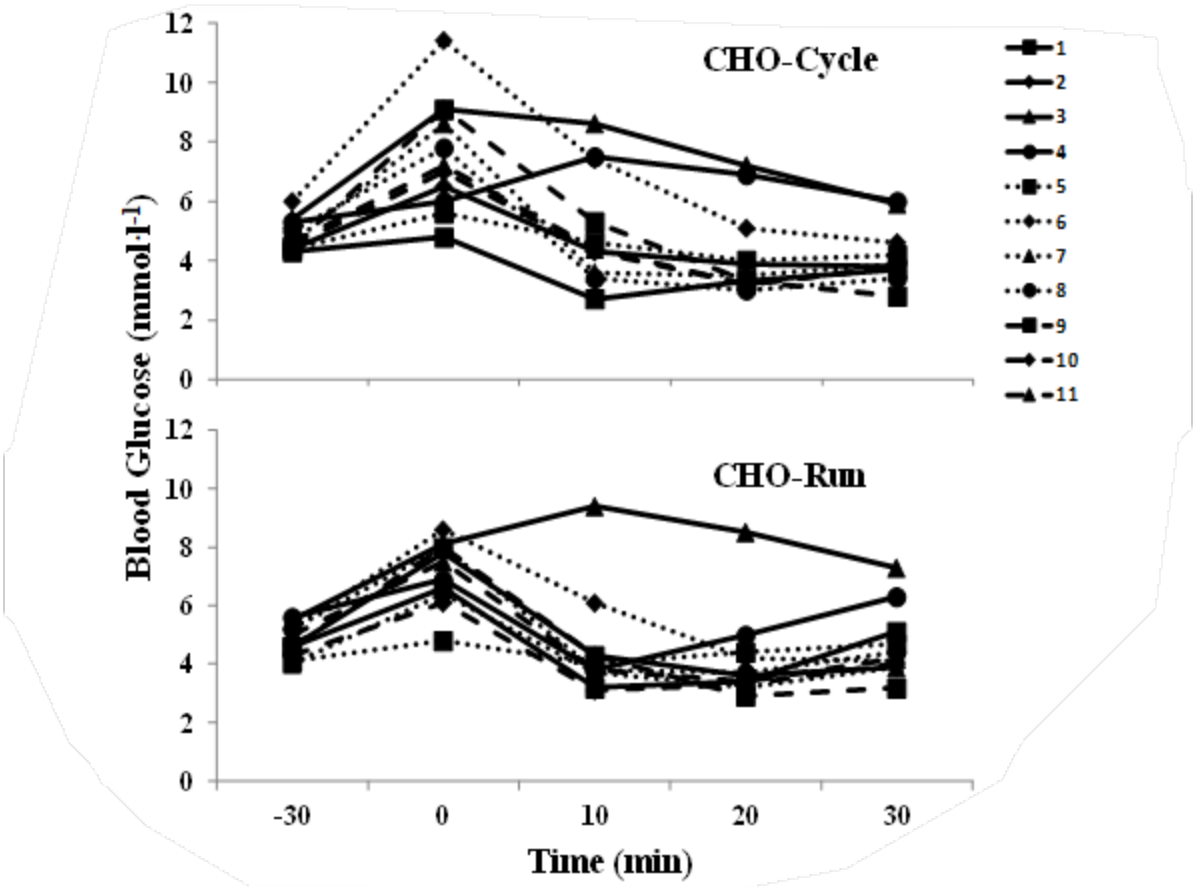

Figure 2. Individual blood glucose concentrations in the CHO-Cycle (above) and CHO-Run (below) trials.

Serum insulin responses are presented in Figure 3. Serum insulin was different at Fluid (p < 0.001, η^2=0.65) and Time (p < 0.001, η^2 = 0.52) levels, whereas no difference was observed at 3-way interaction (p = 0.45, η^2=0.05) or exercise mode (cycling: 18 ± 3 vs. running: 11 ± 3 mU·l^{-1}, 95%CI: -2–15, p = 0.11). Also, there was a Fluid x Time interaction and insulin was higher (p<0.01) in CHO trials compared to placebo at the initiation (mean CHO-Cycle+CHO-Run: 59 ± 10 vs. mean PL-Cycle+PL-Run: 8 ± 1 mU·l^{-1}, 95%CI: 30-71) as well as during exercise. For participants who experienced a drop in blood glucose, serum insulin at the start of exercise was 77 ± 25 and 54 ± 21 mU·l^{-1} in CHO-Cycle and CHO-Run respectively, whereas for those who did not develop transient hypoglycemia was 64 ± 19 and 33 ± 9 mU·l^{-1}. No statistical comparison was made for these values due to relatively small number in the two subgroups (transient hypoglycemia: 6-7 subjects and no transient hypoglycemia: 4-5 subjects). However, no correlations were observed between blood glucose decrease from the initiation (0-min) to 20-min of exercise and the increase in serum insulin concentration as a result of maltodextrin ingestion (-30-min to 0-min) in CHO-Cycle (r = 0.49, p = 0.13) and CHO-Run (r = 0.36, p = 0.28), or between insulin concentrations at the start of exercise with blood glucose at 20-min of exercise (CHO-Cycle: r = -0.22, p = 0.52; CHO-Run: r = -0.06, p = 0.87).

The QUICKI values were similar in individuals who developed transient hypoglycemia (CHO-Cycle: 0.37 ± 0.01 and CHO-Run: 0.37 ± 0.16) with the subjects who did not develop transient

hypoglycemia (CHO-Cycle: 0.34 ± 0.02 and CHO-Run: 0.36 ± 0.01). Also, no correlations were found between blood glucose drop at 20 min of exercise and QUICKI in CHO-Cycle (r = 0.05, p = 0.89) and CHO-Run (r = -0.28, p = 0.41).

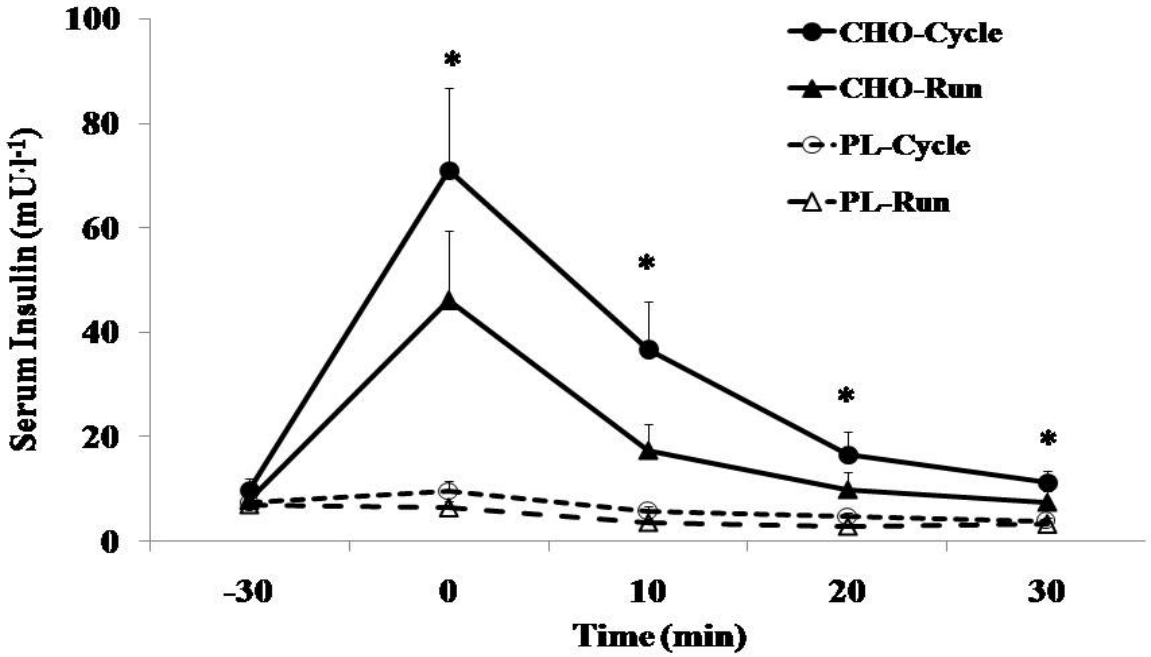

Figure 3. Serum insulin responses during the four trials (mean ± SE). *p < 0.01: Mean of both CHO (CHO-Cycle+CHO-Run) trials compared to mean of both Placebo (PL-Cycle+PL-Run) trials.

Plasma FFA, glycerol and lactate responses before and immediately after exercise are presented in Table 1. For FFA significant differences were found at fluid (p = 0.001, η^2 = 0.41) and fluid X time interaction (p < 0.001, η^2 = 0.63). At the end of exercise, plasma FFA concentrations were higher in placebo compared to CHO conditions irrespective of exercise mode (PL-Cycle+PL-Run: 356 ± 27 vs. CHO-Cycle+CHO-Run: 144 ± 26 µmol·l^{-1}, 95%CI: 142-283, p < 0.001). Also, when the post-pre exercise data were examined, when maltodextrin was provided FFA decreased, whereas in placebo FFA were increased.

For plasma glycerol differences were found at mode (running: 84 ± 5 vs. cycling: 65 ± 5 µmol·l^{-1}, 95%CI: 6–33, p=0.007), time (p < 0.001, η^2 = 0.80), fluid (Placebo: 84 ± 4 vs. CHO: 65 ± 5 µmol.l^{-1}, 95%CI: 5–33, p = 0.01, η^2=0.28) and 3-way interaction (p = 0.01, η^2 = 0.29). Post-exercise glycerol was higher in running compared to cycling in placebo (PL-Run > PL-Cycle), and at the same time point in running glycerol was higher in placebo compared to CHO (PL-Run > CHO-Run). When post-pre exercise values were compared, the increase in glycerol levels due to running was higher in placebo compared to CHO (PL-Run > CHO-Run).

Blood lactate responses were different at mode (cycling: 3.2 ± 0.2 vs. running: 1.6 ± 0.2 mmol·l^{-1}, 95%CI: 1.0-2.2, p < 0.001), time (p < 0.001, η^2 = 0.78) and mode X time (p < 0.001, η^2 = 0.62). At the end of exercise and irrespective of fluid, blood lactate was higher in cycling compared to running (CHO-Cycle+PL-Cycle: 4.9 ± 0.4 vs. CHO-Run+PL-Run: 1.9 ± 0.4 mmol·l^{-1}, 95%CI: 1.9-

4.0, $p < 0.001$). When post-pre exercise lactate concentrations were compared no difference was denoted between fluids in both cycling and running conditions. However, lactate increase with exercise was higher in cycling compared to running irrespective of fluid.

Table 1. Plasma FFA, glycerol and blood lactate concentrations before and after exercise in the four conditions (mean ± SE).

	Pre-Exercise				Post-Exercise				Post – Pre Exercise			
	CHO Cycle	CHO Run	PL Cycle	PL Run	CHO Cycle	CHO Run	PL Cycle	PL Run	CHO Cycle	CHO Run	PL Cycle	PL Run
Plasma FFA ($\mu mol.l^{-1}$)	239 ± 34	220 ± 34	237 ± 37	260 ± 37	140 ± 36	147 ± 36	320 ± 38	393 ± 38	-99 ± 35	-73 ± 51	83 ± 20[1]	133 ± 41[1]
Plasma Glycerol ($\mu mol.l^{-1}$)	40 ± 8[2]	59 ± 8[2]	52 ± 8[2]	60 ± 8[2]	80 ± 9	80 ± 9[3]	87 ± 8[3]	137 ± 8	40 ± 9	21 ± 7[3]	35 ± 8	77 ± 15
Blood Lactate ($mmol.l^{-1}$)	1.6 ± 0.2	1.2 ± 0.2	1.4 ± 0.2	1.3 ± 0.4	4.9 ± 0.4	2.0 ± 0.4	4.8 ± 0.4	1.9 ± 0.4	3.3 ± 0.6[4]	0.8 ± 0.2	3.4 ± 0.5[4]	0.6 ± 0.3

Note: 1. $p = 0.002$, compared to the corresponding CHO trial; 2: $p < 0.01$, compared to post-exercise; 3: $p < 0.01$, compared to PL-Run; 4: $p < 0.001$, compared to the corresponding Run trial.

DISCUSSION

The present data did not show any difference between the two exercise modes in the glycemic or insulinemic responses following pre-exercise CHO intake. Within 10 min of exercise in the maltodextrin trial blood glucose decreased to a similar degree in cycling (33%) and running (38%), whereas in both placebo conditions (PL-Cycle and PL-Run) blood glucose remained stable throughout the trial. Blood glucose decreased even further at 20-min of exercise when CHO was ingested, but the mean value was above 4.1 $mmol.l^{-1}$ in both CHO trials. The sudden drop of blood glucose levels in CHO trials may be the consequence of an increased glucose uptake by the working muscles through an insulin-independent GLUT-4 translocation to the muscle cell membrane (41). This increased muscle glucose uptake was further exacerbated by the elevated insulin levels at the initiation of exercise, and a possible concomitant depression of hepatic glucose production, leading to an inability of liver glycogenolysis to replace glucose substrate at the same rate as muscles use it (27).

Several studies have shown that, when CHO are ingested 30-45 min before exercise, a rapid transient decrease in blood glucose is observed within 30 min of exercise, in both cycling and running (7, 9, 10, 12, 15, 18, 23, 24, 26, 40). On the other hand, there are studies in cycling or running where no transient decrease in blood glucose was observed (1, 13, 14, 25, 27, 28, 36, 38).

In the present study six and seven participants developed reactive hypoglycemia in CHO-Cycle and CHO-Run, respectively. These volunteers, however, did not report or present any symptoms of hypoglycemia or discomfort. This observation is consistent with other studies and indicates that some individuals are prone to more severe blood glucose rebounds than others, as a result of pre-exercise CHO intake (18, 19, 25, 26, 28, 30). Also, the higher muscle mass involved in running did not affect blood glucose response during exercise since, with the exception of one person, the same individuals who developed rebound hypoglycemia in CHO-Run, also developed rebound hypoglycemia in CHO-Cycle. Consistency about the phenomenon of transient hypoglycemia has been reported when more than one CHO trial is performed by the same subject in cycling (20). However, to the best of the authors' knowledge the reproducibility of this metabolic phenomenon has not been studied under identical experimental conditions where the type and amount of CHO intake, feeding time before exercise, exercise intensity and mode are kept the same.

It has been suggested that, since insulin enhances glucose uptake in skeletal muscle, individuals who experience a high insulin response as a result of pre-exercise feeding, may be prone to transient hypoglycemia. In a recent study conducted by Kondo and colleagues, it was observed that after an overnight fast followed by the administration of glucose 30-min before cycling, the individuals who developed rebound hypoglycemia had about 114% higher insulin concentrations at the start of exercise compared to the participants who did not develop transient hypoglycemia (25). In the present study this difference, however, was much lower. Serum insulin concentration at the start of exercise was 17% and 39% higher in CHO-Cycle and CHO-Run respectively in the individuals who experienced transient hypoglycemia compared to those who did not. Neither was there any association in any of the two CHO conditions between insulin levels at the beginning of exercise and glucose concentrations at 20 min of exercise, or between insulin response to maltodextrin feeding and the decrement of blood glucose during exercise. This is opposite to earlier studies which reported a correlation between pre-exercise insulin concentrations and the fall of blood glucose during exercise (23, 24). If an association between insulin concentration and the degree of rebound hypoglycemia exists, then one would expect a different glucose response when pre-exercise insulin levels were different. However, different insulinemic responses before exercise have resulted to similar glycemic responses during exercise (18, 36). On the other hand, ingesting low glycemic index CHO produces a lower insulinemic response before cycling exercise and no rebound hypoglycemia compared to a high glycemic index CHO, which is associated with transient hypoglycemia and high pre-exercise insulin levels (10). Therefore, based on the available literature, it seems that the pre-exercise insulin levels are not always related to the incidence of rebound hypoglycemia observed when CHO are ingested before exercise.

Kuipers and colleagues have suggested higher insulin sensitivity as a contributing factor of transient hypoglycemia (26). However, in the present study, the QUICKI, an indirect index of insulin sensitivity, was similar in the group of participants who developed hypoglycemia and those who did not. Also, no correlation was found between QUICKI values and the blood glucose decrease during exercise. In addition, no association was reported between the prevalence of transient hypoglycemia and insulin sensitivity, as judged by an oral glucose

tolerance test (19). Therefore, the factors that contribute to an individual's susceptibility to transient hypoglycemia during exercise after pre-exercise CHO intake remain to be determined.

Plasma FFA concentrations were higher at the end of exercise in placebo compared to CHO trials irrespective of exercise mode. Similarly, the post-pre exercise FFA concentrations were also higher in PL-Cycle and PL-Run compared to CHO-Cycle and CHO-Run, respectively. It seems that, when maltodextrin was administered, FFA mobilization was suppressed compared to placebo in both modes of exercise, an observation usually reported in cycling and running when CHO are ingested before exercise (9, 10, 12, 13, 28, 36). The reduction of FFA levels was probably due to elevated insulin concentration before exercise as a result of maltodextrin ingestion. Insulin is an antilipolytic hormone that exerts its effect possibly through a reduction in cyclic monophosphate levels which inhibits protein kinase A activity, leading to a decrease in hormone-sensitive lipase and lipolysis (5).

At the end of exercise glycerol levels were higher in PL-Run compared to CHO-Run, as expected. However, this was not the case in cycling where glycerol concentrations were not different between CHO-Cycle and PL-Cycle. This has also been reported in other studies, where the ingestion of about 1 g/kg BM CHO 30-45 min before cycling reduced FFA concentrations compared to placebo, while no difference in glycerol concentrations was observed during exercise (12, 13). It can be speculated that the higher muscle mass recruited during running might have produced a higher catecholamine response, and a greater degree of lipolysis (22). However, since no metabolic cart, tracers, or other hormonal measurements were made in the present study, this glycerol response cannot be explained by the available data.

A clear exercise mode effect was found in the blood lactate responses as indicated by the higher lactate levels at the end of exercise in both cycling trials irrespective of the fluid ingested. This has also been reported by other investigators between cycling and running (34). The higher lactate concentration might be the result of different fiber type recruitment in cycling where the smaller muscle mass involved activates more the glycolytic type II muscle fibers (6).

Maltodextrin increased HR during exercise in both CHO-Run and CHO-Cycle trials, whereas the relative exercise intensities in placebo were similar and close to the initially planned 80% HRmax (PL-Cycle: 78% and PL-Run: 79%). However, the subjective effort of participants was not affected as indicated by the similar RPE values. It can be speculated that the hyperinsulinemia in CHO trials may have influenced HR response. Insulin increase elevates HR at rest in healthy individuals as a result of enhanced cardiac sympathetic activity (37). Nevertheless, although maltodextrin intake has not been used before exercise, elevated insulin as a result of pre-exercise CHO ingestion has not be reported to influence HR during exercise (9, 10, 14, 15, 18, 24, 27, 38). More studies are necessary to examine whether pre-exercise maltodextrin intake elevates HR during exercise.

In conclusion, ingestion of 1 g/kg BM maltodextrin 30-min before cycling or running at 80% HRmax produced similar glucose and insulin responses during exercise in active males. Maltodextrin reduced FFA concentrations in both types of exercise compared to placebo,

whereas lactate concentration was higher in cycling irrespective of the type of fluid ingested. Individuals, who experience transient hypoglycemia as a result of pre-exercise CHO intake, will probably develop this metabolic disturbance to the same extent either during cycling or running. However, this perturbation is not associated with a detrimental effect during subsequent endurance exercise, since many studies have reported an improvement in endurance capacity and performance following pre-exercise CHO intake, despite an observed transient hypoglycemia early in exercise (20, 33).

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