Pre-exercise feeding does not affect endurance cycle exercise but attenuates post-exercise starvation-like response

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ABSTRACT

CALLES-ESCANDÓN J., J. T. DEVLIN, W. WHITCOMB, and E. S. HORTON. Pre-exercise feeding does not affect endurance cycle exercise but attenuates post-exercise starvation-like response. *Med. Sci. Sports Exerc.*, Vol. 23, No. 7, pp. 818-824, 1991. The effects of ingesting a mixed-snack food (CB), fructose (FRU), or placebo (PBO) prior to exercise (70% peak VO2) on the metabolic response during and after cycle exercise were studied in eight normal healthy volunteers with a wide range of peak VO2 (30-70 cc·kg-1·min-1). The study was designed to minimize the impact of confounding factors by using various strategies. First, the volunteers were grouped in teams with stratification by peak VO2, and the tests were randomized by a Latin-square design. Second, subjects received two acclimation trials in the cycle ergometer to diminish the effect of learning experiences and allow them to get used to the room and equipment. In addition, financial incentives were offered for team and individual endurance times. The test meals were administered 30 min prior to the beginning of exercise, and the subjects exercised to exhaustion, which was defined with clear-cut endpoints. Gas and blood samples were taken at regular intervals before, during, and for 60 min after each exercise bout. CB and FRU induced higher pre-exercise glucose and insulin concentrations. Blood lactate increased 100% with FRU ingestion. Despite these differences; endurance time, substrate, and hormone concentrations as well as rates of substrate oxidation during exercise were identical among the three conditions. During the post-exercise recovery period, PBO was associated with a starvation-like pattern of substrate utilization in which lipid oxidation was 60% greater and carbohydrate oxidation 50% less than following either CB (75 ± 11, 248 ± 27 mg·min-1, P < 0.05) or F ingestion (93 ± 4, 221 ± 14 mg·min-1). Our results demonstrate that pre-exercise feeding does not affect endurance or the metabolic response during exercise, yet it may attenuate post-exercise starvation-like response.

ENDURANCE, FUEL METABOLISM, EXERCISE RECOVERY, PRE-EXERCISE MEAL

The effects of pre-exercise feeding on endurance exercise performance and metabolic response to exercise have received much attention recently (1,8-10,13,15,17,18,21-23,25,27). It has been proposed that glucose-induced hyperinsulinemia at the onset of exercise may adversely affect endurance by inhibiting lipolysis and enhancing muscle glycogen consumption (7, 18). On the other hand, pre-exercise fructose ingestion has been shown to increase endurance performance (27), an effect attributed to the reduced stimulation of insulin secretion induced by fructose (10). Recently we have shown that ingestion of a mixed snack food immediately prior to exercise, which increased blood glucose and insulin concentrations, did not have a deleterious effect on endurance performance (9). The reasons for these discrepant results are not clear. Different experimental designs and/or extraneous (thus confounding) factors (psychological drive, learning experiences, etc.) might explain some of these discrepancies. Thus, the project was designed to optimize the possibility of examining dietary effects per se and minimize the interference of other confounding variables.

Despite several studies dealing with the interaction of meal-induced thermogenesis and exercise (4,5, 20,29,30,33), the metabolic effects of pre-exercise feeding on early recovery from exercise has not been studied in detail. Bielinski et al. (4) concluded that an acute bout of exercise stimulates both energy expenditure and lipid oxidation for a prolonged period (>12 h). However, the design of that study precludes interpretation of pre-exercise feeding effects. Kaminsky et al. (20) found that pre-feeding exercise is associated with a preference for fat as a fuel after meal ingestion. Whether pre-exercise feeding may affect fuel utilization during the recovery phase from exercise is an open question. Thus, in addition to comparing the effects of differing pre-exercise meals on endurance performance and the metabolic responses during exercise, we also investigated the metabolic changes during early recovery (60 min) from exercise.

We compared the effects of ingesting a mixed snack food (candy bar), a fructose drink, and a placebo drink taken 30 min prior to starting high intensity (70% peak VO2) cycle exercise that continued until subjective exhaustion. Variable outcomes that were investigated

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were the endurance times, energy expenditure, carbohydrate, lipid and protein oxidation rates, and whole blood or plasma concentrations of metabolic substrates and hormones measured prior to, during, and for 60 min after exercise.

MATERIALS AND METHODS

Subjects. Nine healthy male volunteers were selected for participation in this study (height, 174 ± 3.2 cm; weight, 73 ± 5.4 kg); one volunteer did not complete the study, and his data were not included in the final analysis. All were within 15% of ideal body weight (IBW) and not taking any medication. Age range was 19–35 years and all were male. They were screened by medical history, physical examination, and routine laboratory chemistries. Before admission to the study, the nature and risks were explained in detail, and all subjects gave their written informed consent prior to participation. All tests were carried out on the General Clinical Research Center of the University of Vermont (CRC).

Study Design. Each subject had his maximum aerobic capacity (peak VO₂) determined on a Monark cycle ergometer (Stockholm, Sweden), using a continuous protocol with increments of 25 or 50 W every 2 min. Oxygen consumption (VO₂) and CO₂ production (VCO₂) were measured by indirect calorimetry (see below) during the last 30–60 s of each workload. Peak VO₂ was determined as the point where the subject was exercising at or above his predicted maximal heart rate and further increases in the workload did not raise VO₂ by >10% above the preceding workload. In all volunteers, subjective exhaustion was reached during the test, and the respiratory exchange ratio at peak VO₂ was >1.1.

The study design is shown in Figure 1. Two weeks after determination of peak VO₂, the subjects were allocated to three different teams (A, B, and C) with stratification by peak VO₂. High peak VO₂ (H) was considered >60 cc·kg⁻¹·min⁻¹, medium (M) VO₂ between 50 and 60 cc·kg⁻¹·min⁻¹, and low (L) VO₂ as <50 cc·kg⁻¹·min⁻¹. Initial screening and peak VO₂ determinations were done in a large pool of normal male candidates; nine of them were selected for participation in the study. One of these nine volunteers withdrew from the study prematurely (after the first test), and his data were not used in the final analysis. Thus, teams A and B had three subjects each (1 H, 1 L, and 3 L) while team C had only two (1 H and 1 L). To maximize the exercise endurance effort, the subjects were told that the study design included a competition among the different teams, with a monetary compensation for the members of the winning team, based on the team’s accumulated endurance time from all exercise tests. Furthermore, a monetary compensation was also offered for the particular individual with the longest endurance time in any of the tests. The subjects were unaware of the content of the pre-exercise meal (see below) and were informed that the study was designed to test the influence of solid vs liquid meals of similar composition on endurance exercise.

Two training sessions were scheduled for all subjects with two goals in mind: 1) to accustom the volunteers to the equipment and testing procedures, and thus minimize the effects of learning on subsequent performance; and 2) to establish the individual workloads needed to achieve ≈70% peak VO₂ during the actual tests. During these training sessions each volunteer

Figure 1—Study design. The design was chosen to maximize the possibility that dietary effects would become evident. Differences in fitness levels were balanced by stratification among the teams by peak VO₂ with random allocation. To increase further psychological drive, a sense of competition was introduced and monetary compensation was offered for best times (team and subject).
underwent exercise on the cycle ergometer for 30–45 min while VO₂, VCO₂, and heart rate were measured every 5–10 min. The workload of the cycle ergometer was adjusted to elicit ≈70% of peak VO₂. Once established, this workload was used throughout the study.

Each volunteer underwent three tests: placebo (PBO), fructose (FRU), and mixed snack meal (CB). The PBO drink consisted of 250 cc of flavored water (orange) sweetened with aspartame (Nutrasweet, Searle, Chicago, IL), the FRU drink contained 65 g of crystalline fructose dissolved in 250 cc of flavored water (orange), and the CB meal was a candy bar, isocaloric to the FRU drink (260 Kcal: 43 g of carbohydrate, 9 g of fat, and 3 g of protein). The order of the tests was randomized with a Latin-square design (one subject withdrew after the first test).

The design of the tests is shown in Figure 2. The volunteer was admitted to the CRC the night before each test, and a timed overnight urine specimen was collected for determination of urinary urea nitrogen (UUN) excretion. The volunteer was awakened at 6:30 a.m., and an intravenous catheter was placed in an antecubital vein for blood sampling and maintained open with a slow drip of isotonic saline solution. The volunteer remained in bed until between 7:00 and 8:00 a.m. while resting metabolic rate (RMR) was measured by indirect calorimetry (see below). After the RMR measurement, the volunteer was allowed to use the toilet and prepare for the exercise test. After a pre-meal blood sample at 8:00 a.m., the subject was asked to ingest one of the three experimental meals (see below) in <10 min.

Thirty minutes following the start of meal ingestion a second, post-meal, pre-exercise blood sample was taken, and the volunteer began exercising (≈8:30 a.m.) on the ergometer at the workload previously determined to elicit ≈70% of his peak VO₂. During exercise VO₂, VCO₂, and heart rate were measured every 10–15 min. Exhaustion was defined as the inability of the subject to maintain a pedalling frequency of 50 rpm despite the verbal encouragement of the investigator. Time was recorded and used as objective index of endurance. After the subject reached exhaustion the metabolic rate was then measured for the next 60 min using indirect calorimetry (see below). Throughout the test volunteers were allowed free access to water.

**Substrate and hormone measurements.** Blood samples for insulin (IRI), glucose (PG), acetoacetate and beta-hydroxybutyrate (KG), free fatty acids (FFAs), and glycerol (GLY) were taken prior to the meal, before exercise, every 15 min during the exercise, at the point of exhaustion, and at 15, 30, and 60 min during the post-exercise recovery period. Blood samples for lactate were taken prior to the meal, before exercise, at 40 and 50 min of exercise, at the point of exhaustion, and 30 and 60 min during recovery.

Serum IRI was measured by radioimmunoassay (32) and PG by using a glucose oxidase method (Beckman Glucose Analyzer II, Fullerton, CA). FFAs were determined by the method of Novak (26), and GLY, lactate, and KB were measured on protein-free filtrate of whole blood (30% perchloric acid). After separation the samples were stored at −70°C until assayed by the enzymatic methods described by Bergmeyer (2) using an American Instrument (Silver Spring, MD) fluorometer to record changes in NADH concentrations.

**Indirect calorimetry.** For determinations of energy expenditure during the basal state (prior to exercise) and during the recovery from exercise, the ventilated hood system was used as described in detail elsewhere (6). Briefly, the head of the subject is enclosed in a plastic, transparent hood that is ventilated with fresh air at a rate of 35–50 l·min⁻¹. Oxygen concentration is measured in expired air with a zirconium-fuel cell analyzer (Applied Electrochemistry, Sunnyvale, CA) and carbon dioxide with an infrared analyzer (Applied Electrochemistry). Flow through the system is measured with a pneumotachograph (Vertek, Burlington, VT). The voltage outputs of the instruments are converted to digital signals and read continuously in a

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**Figure 2—Test Design.** For details of the methodology used please see text. The order of tests was established with a Latin square design (three teams, three subjects/team⁻¹, three tests/subject⁻¹); however, one of the volunteers withdrew prematurely from the protocol.
Pre-exercise feeding

desktop personal computer (HP-85, Hewlett-Packard, Palo Alto, CA). VO₂ and VCO₂ are calculated by standard formulas (6). The contribution of protein oxidation to VO₂ and VCO₂ is calculated assuming that 1 g of UUN = 6.25 g of protein oxidized and subtracted from the original VO₂ and VCO₂ to yield non-protein VO₂ and VCO₂. A new respiratory exchange ratio (non-protein respiratory exchange ratio, NPRQ) is computed, and the relative contributions of carbohydrate and fat oxidation rates are calculated taking into consideration that at a NPRQ of 0.7, 100% of the NP-VO₂ is due to lipid oxidation, whereas at a NPRQ of 1.00 carbohydrate oxidation accounts for 100% of the NP-VO₂. Intermediate values are interpolated (31).

For exercise-related determinations we used a previously described mouthpiece system for collecting expired air (9), rather than the ventilated hood. Gas analyses were as described above.

Statistical analysis. All data are expressed as means ± SEM. Analysis was done using ANOVA. The design of the model included four ways (team, meal treatment, order, peak VO₂) and repeated measurements (substrate and hormones), deleting from the model team and peak VO₂ (two ways) as factors did not change the results. Post hoc separation of meal values was done with Scheffe test, and a P value < 0.05 was taken as significant (28).

RESULTS

All subjects were within 15% of IBW (105 ± 6%, Metropolitan Tables, 1979). The mean peak VO₂ was 54 ± 3.01 cc·kg⁻¹·min⁻¹. Of the eight subjects who completed the protocol, three were classified as having a high peak VO₂ (61, 63, and 66 cc·kg⁻¹·min⁻¹), three a medium (50, 53, and 55 cc·kg⁻¹·min⁻¹), and two a low peak VO₂ (41 and 43 cc·kg⁻¹·min⁻¹). The subject who dropped out had a low peak VO₂ (43 cc·kg⁻¹·min⁻¹).

The intensity of the workload was set for each subject in the training sessions, and as shown in Table 1, oxygen consumption and percent peak VO₂ during the exercise tests were similar among the three pre-exercise feeding conditions. Furthermore, there were no test order or team effects. Thus, in all three tests, each volunteer was exercising at the same relative intensity. Endurance time was not distinguishable statistically among the three conditions; indeed there was a wide individual variation in endurance times that was not correlated with test order, peak VO₂, team assignment, or feeding condition (PBO, FRU, CB).

As shown in Figure 3, there were no differences in pre-meal concentrations of serum IRI, plasma GLU, and blood lactate among the PBO, FRU, and CB tests. Both FRU and CB ingestion elicited a similar pre-exercise glycemic response that was significantly greater than the PG concentration observed in the PBO condition.

Ingestion of a CB was associated with a higher serum IRI concentration (201 ± 17.2 pmol/dl) than following FRU (152 ± 12.3 pmol/dl, P < 0.05) or PBO (102 ± 11.1 pmol/dl, P < 0.05 vs CB and FRU) ingestion. However, during the exercise period there was a prompt decline in IRI concentration in all three conditions to the extent that after 10 min of exercise similar values were measured during the remainder of the test. A

![Figure 3](image)

**TABLE 1.** Effect of preexercise feeding on endurance times.

<table>
<thead>
<tr>
<th>Oxygen Consumption During Exercise</th>
<th>Endurance Time (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1·min⁻¹</td>
</tr>
<tr>
<td>Placebo</td>
<td>2.76 ± 0.20</td>
</tr>
<tr>
<td>Fructose</td>
<td>2.94 ± 0.29</td>
</tr>
<tr>
<td>Candy Bar</td>
<td>2.85 ± 0.32</td>
</tr>
</tbody>
</table>

Figure 3—Serum insulin, plasma glucose and lactic acid responses. Panel A displays glucose response, panel B serum insulin, and panel C the lactic acid response. As described in detail in the text, the metabolic milieu was different among the three conditions, yet the metabolic impact of the exercise bout was such that the differences in the concentrations of glucose, insulin, and lactic acid during the exercise were remarkably similar.
transient increase in IRI concentration was evident in all three conditions during the recovery period, but concentrations were similar among the three dietary treatments.

FRU ingestion was associated with a doubling of blood lactate prior to exercise (from ≈1 mmol/l to ≈2 mmol/l), yet during the exercise and recovery periods the concentrations of blood lactate were similar in the three experimental conditions.

FFA and KB responses are displayed in Figure 4. Baseline and exercise-related concentrations of FFA and KB were similar among the three dietary conditions with large individual variations. During recovery from exercise there was a tendency for a lower FFA and KB response in association with ingestion of either the CB or FRU when compared with PBO. Owing to large individual variations, however, this pattern of response could not be demonstrated statistically.

Indirect calorimetry data are shown in Table 2. Baseline energy expenditure was similar in all tests. Energy expenditure rose tenfold during exercise to values that were similar in all three conditions. Energy expenditure during the early recovery period (60 min) was 20% higher when compared with the pre-exercise resting metabolic rate \((P < 0.05)\) and was not affected by test order, feeding condition, peak VO\(_2\), or endurance time. The rates of carbohydrate and fat oxidation were similar in the pre-exercise period among PBO, FRU, and CB conditions. There was a 12-fold increase in the rate of carbohydrate oxidation and a fivefold increase in fat oxidation during exercise. This metabolic response was similar among the three dietary conditions. The expected starvation-like metabolic pattern with higher rates of fat oxidation and lower rates of carbohydrate oxidation was observed in the post-exercise recovery period following PBO ingestion. This was markedly attenuated following FRU or CB ingestion. In both conditions, post-exercise carbohydrate oxidation was increased and fat oxidation decreased compared with PBO ingestion (Table 2).

**DISCUSSION**

Previous studies have demonstrated that the antecedent diet may affect the metabolic response during exercise and alter endurance performance at relatively high work intensities (3,14). However, whether acute pre-exercise ingestion of food might change the metabolic response and/or affect endurance is still an open question. In some studies it has been shown that an increase in IRI immediately prior to exercise may decrease endurance by inhibiting FFA supply to exercising muscles and accelerate muscle glycogen depletion (7,18). However, other investigators (15,19,21), including ourselves (9), have not been able to demonstrate this effect. It has also been postulated that an increase in the supply of carbohydrates to exercising muscles without a large increase in IRI (e.g., FRU ingestion) might enhance endurance by sparing muscle glycogen.

**Figure 4**—Free fatty acids and ketone bodies during and after exercise. Changes in the concentrations of free fatty acids and ketone bodies are depicted in panels A, B, and C, respectively.

**Table 2. Rates of energy expenditure and substrate oxidation.**

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy Expenditure</td>
<td>5.8 ± 0.59</td>
<td>5.5 ± 0.62</td>
<td>5.7 ± 0.54</td>
<td>210 ± 56.6</td>
<td>301 ± 19.9</td>
<td>226 ± 10.1</td>
</tr>
<tr>
<td>Carbohydrate Oxidation Rate</td>
<td>53 ± 1.6</td>
<td>57 ± 2.6</td>
<td>57 ± 2.3</td>
<td>2527 ± 116</td>
<td>2679 ± 90</td>
<td>2634 ± 103</td>
</tr>
<tr>
<td>Lipid Oxidation Rate</td>
<td>6.8 ± 0.69</td>
<td>6.9 ± 0.65</td>
<td>6.7 ± 0.49</td>
<td>126 ± 12*</td>
<td>248 ± 27</td>
<td>221 ± 14.2</td>
</tr>
<tr>
<td></td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>48 ± 4.5</td>
<td>25 ± 2.6</td>
<td>37 ± 4.1</td>
</tr>
<tr>
<td></td>
<td>257 ± 16</td>
<td>261 ± 22</td>
<td>266 ± 30</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>120 ± 7**</td>
<td>93 ± 3.5</td>
<td>75 ± 11</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

\(A = \) placebo; \(B = \) fructose; \(C = \) candy bar.

\(P < 0.05 (A < B, C).\)

\(P < 0.05 (A > B, C).\)
(21,27). However, data are conflicting, even in studies from the same laboratory (17,18), and it is not yet clear whether acute metabolic changes induced by ingestion of a meal immediately prior to exercise will affect endurance and/or change the metabolic response during exercise.

In this study, and in agreement with our previous findings (9), ingestion of a CB immediately prior to exercise did not change endurance compared with feeding a PBO meal.

There were also no effects of test order or peak $VO_2$. In this regard, it is important to point out that we designed our study to minimize the impact of learning experiences on endurance by acquainting our volunteers with the equipment prior to the actual tests. To increase subject cooperation even further, a sense of competition was created by the allocation of volunteers to different teams. To diminish the effect of peak $VO_2$, the allocation of subjects was stratified by peak $VO_2$. Furthermore, taking into consideration prior studies (11), a monetary compensation was offered to enhance further the psychological drive for endurance. Thus, we feel confident that our design allows for any effects of pre-exercise feeding on endurance to be demonstrated. This and our prior study (9) have led us to conclude that acute perturbations in the metabolic milieu induced by pre-exercise feeding (mixed-meal, fructose) have very little, if any, impact on endurance exercise performance at exercise intensities of approximately 70% of peak $VO_2$.

Pre-exercise ingestion of fructose increases serum insulin levels slightly (22) and presumably provides readily available fuel to exercising muscles, potentially sparing glycogen utilization during exercise (1,11,21). It has been proposed that these metabolic changes might increase endurance. In this study, FRU increased the PG concentration slightly and induced an IRI response that was intermediate between PBO and CB ingestion. As expected, it also doubled blood lactate concentration. However, we did not observe any enhancement of endurance by pre-exercise FRU ingestion compared with PBO. Recently it has been shown that exogenous fructose is not so available for oxidative purposes when compared with glucose or glucose polymer (16,24).

From the metabolic point of view, exercise has been considered a starvation-like state that is sometimes more evident during the post-exercise recovery period (12). From previous studies (4,5,20,29,30,33) it is clear that exercise induces metabolic changes that have carryover effects on subsequent fuel utilization (4) and possibly energy expenditure (4,5,20,29,30,33). However, whether the reverse is true, e.g.: “Is meal ingestion associated with ‘carryover effects’ on metabolic milieu, which may affect the pattern of fuel oxidation in the recovery from exercise?” is an open question. In association with PBO ingestion, we observed a metabolic pattern consistent with starvation-like response during the 60-min post-exercise recovery period. This was characterized by an increase in the oxidation rate of fat coupled with a decrease in the rate of carbohydrate oxidation. These results confirm previous observations (4). Strikingly, this starvation-like pattern was markedly attenuated by pre-exercise feeding of either the CB or FRU. Post-exercise carbohydrate oxidation rates were higher in association with either ingestion of the CB or FRU when compared with PBO ingestion, coupled with a reciprocal decrease in lipid oxidation rate. Thus, meal ingestion prior to exercise was associated with a blunting of the starvation-like response during the post-exercise recovery period, suggesting a more rapid restoration of the pre-exercise metabolic status and thus implying “carryover effects” of meal ingestion that persist after exercise and become evident in the early recovery period. The mechanism of this phenomenon is not evident from this study; however, it could be speculated that availability of fuel/substrates from dietary origin might spare endogenous fuel/substrate utilization. The role of hormonal-tissue interactions in these changes needs to be addressed. Whether pre-exercise feeding accelerates resynthesis of muscle glycogen and/or might improve endurance during a subsequent exercise bout are questions that deserve further exploration.

In summary, in this study we found that acute metabolic perturbations induced by pre-exercise feeding of a mixed meal snack or fructose did not change the metabolic response during exercise, but may have blunted the starvation-like response of the post-exercise recovery period. Acute perturbations of the metabolic milieu induced by pre-exercise feeding did not have any impact on endurance under the conditions of our study.

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