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Abstract

Objective: To assess the effects of negative energy balance on the metabolic response of a meal containing either glucose or fructose as the primary source of carbohydrate after exercise in obese individuals in energy balance, or negative energy balance.

Research Methods and Procedures: Fourteen adults with mean body mass index (BMI) 30.3 ± 1 kg/m^2, age 26 ± 2 years, and weight 93.5 ± 5.4 kg, adhered to an energy-balanced (EB) or a negative energy-balanced (NEB) diet for 6 days. On Day 7, subjects exercised at 70% VO_{2peak} for 40 minutes then consumed either high glucose (50 g of glucose, HG) or high fructose (50 g of fructose, HF) liquid meal. Substrate utilization was measured by indirect calorimetry for 3 hours. Blood samples were collected before exercise and 0, 30, 60, 120, and 180 minutes after consuming the meal.

Results: The HG produced 15.9% greater glycemic ($p < 0.05$) and 30.9% larger insulinemic ($p < 0.05$) responses than the HF under both EB and NEB conditions. After the NEB diet, carbohydrate and fat oxidation did not differ for HG and HF. In contrast, carbohydrate oxidation increased 31%, and fat oxidation decreased 39% with HF compared with HG after the EB diet. Thus, HF and HG consumed after exercise produced marked differences in macronutrient oxidation when obese subjects followed an EB diet, but no difference when adhering to a NEB diet.

Discussion: The data suggest that the use of fructose in supplements/meals may provide no additional benefit in terms of substrate utilization during a weight loss program involving diet and exercise.

Introduction

Fructose is a naturally occurring carbohydrate found in fruit and honey and is also being increasingly used as a sweetener in processed foods and beverages ((**1**)). Schwarz et al. ((**2**)) suggests that using fructose as part of the carbohydrate source in obese and/or insulin-resistant individuals may be beneficial due to increased thermogenesis and carbohydrate oxidation. Additional possible benefits include low postprandial glycemic and insulinemic responses ((**2**)). Fructose may also hold benefits in low energy diets for obesity treatment, because, in addition to the above benefits, fructose reportedly suppresses food intake ((**3**), (**4**)).

Although the metabolic response to fructose has been well characterized in subjects consuming normal diets ((**5**), (**6**), (**7**)), few studies have investigated the effects of fructose consumption under conditions typically associated with weight loss. Most weight loss programs include a combination of reduced food intake and regular exercise to induce negative energy balance. This in turn reduces hepatic and muscle carbohydrate (glycogen) stores, which may alter the metabolic response to fructose. Fructose is primarily metabolized by the liver ((**8**)). When the amount of fructose metabolized in the liver exceeds the hepatic oxidation capacity, fructose has to be disposed of nonoxidatively. Lowering hepatic glycogen stores through negative energy balance and exercise may allow greater disposal of fructose as liver glycogen in response to consuming a meal and reduce substrate cycling through other pathways, which may increase thermogenesis and/or carbohydrate oxidation typically associated with fructose consumption. Thus, characteristic responses to different carbohydrates may be altered during negative energy balance, which could impact their utility in weight loss programs involving regular exercise.

The purpose of the present study was to assess the effects of negative energy balance on the metabolic response of a meal containing either glucose or fructose as the primary source of carbohydrate after exercise in obese individuals in energy balance, or negative energy balance. We hypothesized that placing subjects in a condition of negative energy balance would reduce or eliminate characteristic differences in substrate utilization in response to glucose and fructose after exercise.

Research Methods and Procedures

Subjects

Seven men and seven women were recruited through public advertisements. Criteria for participation were: a body weight 120% to 150% above ideal value ((**9**)), physical activity (moderate exercise ≥ 3×/wk), age of 18 to 40 years, non-smoker status, and lack of medication being taken that are known to affect resting energy expenditure (REE) or body weight. Except for obesity, participants were required to be in good health. Subject characteristics are presented in **Table 1**. Study procedures were approved by the Purdue University Committee on the Use of Human Research Subjects.

Table 1. Subject characteristics

Subject	**Age (years)**	**Height (cm)**	**Weight (kg)**	**BMI (kg/m^2)**	**Body fat (%)†**	**REE (kcal/d)**	***VO*$_{2peak}$ (mL/kg/min)**
Male (*n* = 7)	26.4 ± 2.3*	182.7 ± 3.5	107.9 ± 5.7	32.3 ± 1.3	31.6 ± 2.2	2021 ± 47	39.9 ± 2.6
Female (*n* = 7)	26.0 ± 1.6	166.6 ± 2.1	79.2 ± 5.0	28.4 ± 1.1	36.5 ± 1.6	1478 ± 55	34.0 ± 2.7

* Mean ± SE.

† Percentage body fat in Subjects 2 and 8 was not measured due to equipment failure.

Study Design

This protocol involved a within-subject 2×2 factorial design with diet (energy balance [EB] and negative energy balance [NEB]) and post-exercise liquid meal (PELM) (high glucose or high fructose) as independent factors (**Figure 1**). Pre-exercise and post-exercise energy expenditure and substrate oxidation were measured by indirect calorimetry after 6 days of the treatment diet. Subjects were involved in each of four treatments. 1) 6-day EB diet + post-exercise high glucose meal (EBHG), 2) 6-day EB diet + post-exercise high fructose meal (EBHF), 3) 6-day NEB diet + post-exercise high glucose meal (NEBHG), and 4) 6-day NEB diet + post-exercise high fructose meal (NEBHF). PELMs were randomized with a 1-week or greater washout period after energy-restricted treatments. The PELM contained either glucose (50 g of glucose) or fructose (50 g of fructose) mixed in skim milk immediately before consumption. The meal contained 371 kcal, 73.8 g (80%) of carbohydrate, 16.7 g (18%) of protein, and 0.9 g (2%) of fat. Post-exercise energy expenditure and substrate oxidation measures were preceded by 40 minutes of treadmill exercise at 70% to 75% VO_{2peak}. Energy expenditure during the exercise was approximately 603 kcal for males and 377 kcal for females. Heart rate was monitored to maintain proper exercise intensity (Polar Electro Inc., Port Washington, NY). During the treatment periods, participants were asked to maintain their normal exercise schedule but abstain from exercise 1 day before coming into the laboratory.

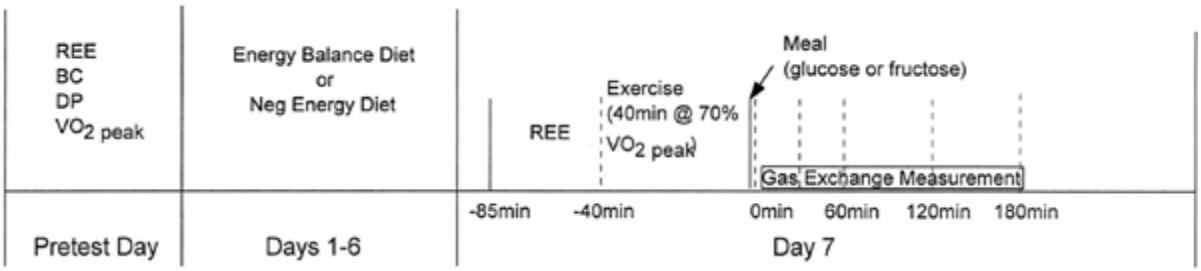

Figure 1 Flow chart of experimental design. Resting energy expenditure (REE), body composition (BC), diet prescription (DP), and VO_{2peak} occurred before each treatment. On Day 7, after 6 days of an EB or NEB diet, REE was measured and subjects exercised for 40 minutes at 70% to 75% VO_{2peak}. Immediately after exercise, a liquid meal containing glucose or fructose was administered (10 minutes was allowed for consumption) and gas exchange was measured for 3 hours. Blood and hunger questionnaires were obtained at minutes −40, 0, 30, 60, 120, and 180 (indicated by dotted lines).

Preliminary Testing

One week before initiating the testing protocols, each subject underwent measurement of REE, VO_{2peak}, and body composition. Subjects were instructed to drive to the laboratory immediately on waking and in a 12-hour-fasted state. REE was measured in the supine position for 45 minutes using a metabolic cart (SensorMedics, V_{max} 229n; SensorMedics Corp., Yorba Linda, CA) and a ventilated respiratory canopy. The analyzers were calibrated with fresh atmospheric air and standard calibration gas mixtures (4% CO_2, 24% O_2, balanced with N_2; 0% CO_2, 26% O_2, balanced with N_2). Raw data were continuously recorded online and averaged every 30 seconds. The values for oxygen consumption, carbon dioxide production, respiratory quotient (RQ) (VCO_2/VO_2), and rate of energy expenditure (in kcal/min) were calculated using V_{max} software (version 03-1). The last 10 minutes of data were used to determine REE.

VO_{2peak} was determined using a motorized treadmill (MedTrack ST55; Quinton Instrument Co.). Subjects first warmed up on the treadmill for 3 minutes at a rate of 1.2 mph and 0% grade. Exercise stages consisted of 3 minutes in duration, and the gradient increased 2% every stage. The first stage of the test started at 1.7 mph at a 10% grade. The second stage increased to 2.5 mph, and the third stage increased to 3.4 mph. Every stage thereafter was maintained at a rate of 3.4 mph and only the gradient increased. Testing continued until the subject reached the point of exhaustion. Expired gas was continuously analyzed for oxygen and carbon dioxide with a metabolic cart (SensorMedics Corp.). VO_{2peak} was determined as the largest value for oxygen consumption per minute.

Hydrostatic weighing was used to determine body composition. Residual lung volume was measured by oxygen dilution ((**10**)). Body density was calculated, and the percentage fat was derived according to the Siri equation ((**11**)).

Diet

Subjects followed the EB or NEB diets for 6 days before testing with each of the meals. The EB diet was designed to meet the energy requirements of the individual and was estimated using their REE multiplied by an activity factor based on their estimated level of daily activity ((**12**)). Level of daily activity was estimated based on the subject's frequency, duration, and intensity of weekly exercise. The NEB diet contained 75% of the kilocalories provided in the EB diet. Average energy intake was

2697 kcal/d for males and 1919 kcal/d for females on the EB diet and 1640 kcal/d for the males and 1431 kcal/d for the females on the NEB diet. The American Dietetic Association Exchange Lists for Weight Management ((**13**)) were used to develop individual meal plans. The macronutrient composition of the EB diet was approximately 56% carbohydrate, 17% protein, and 27% fat, and the NEB diet was 57% carbohydrate, 19% protein, and 23% fat. Both consisted of commonly available foods. Before engaging in the four testing protocols, a registered dietitian or trained nutritionist gave individual diet instructions regarding proper food selection and accurate recording of food intake. Subjects were required to keep a diet record during each treatment period. These food records, as well as body weight, were reviewed weekly by the investigators to assess dietary compliance. Diets were analyzed by the Nutritionist IV nutrient database, volume 4.1 (Hearst Corp., San Bruno, CA).

Measurement Procedure

Gas Exchange Measurements.

Oxygen consumption and carbon dioxide production were integrated over 30-minute periods. Protein oxidation was estimated by determining the corrected urinary urea nitrogen ((**14**)) and ammonia as described by Burge et al. ((**15**)) and were used to calculate the nonprotein respiratory quotient (NPRQ) during the test. The oxidation of carbohydrate and fat were calculated from nonprotein oxygen consumption ($NPVO_2$), and their relative oxidative proportions were calculated as indicated by the NPRQ as well as and the amount of oxygen consumed per gram of substrate oxidized. The calculations were performed as follows ((**16**)):

$$\text{Carbohydrate oxidation (g/min)} = \text{NPVO}_2 \times(\text{NPRQ} - 0.707)/(0.293\times0.746)$$

(1)

$$\text{Fat oxidation (g/min)} = \text{NPVO}_2\times(1 - \text{NPRQ})/(0.293\times0.746)$$

(2)

$$\text{Protein oxidation (g/min)} = \text{N}\times6.25\times0.966$$

(3)

Metabolic rate (MR) was calculated using the following equation:

$$\text{MR (kcal/min)} = ([4.686 + 1.096\,(\text{NPRQ} - 0.707)])\text{NPVO}_2 + 4.60(\text{PVO}_2)$$

(4)

Because urinary nitrogen excretion could not be accurately assessed during the initial RMR measurement, the formula described by Westrate ((**17**)) was used:

$$\text{MR (kcal/min)} = ([4.686 + 1.096(\text{RQ}) - 0.707]\text{VO}_2$$

(5)

This formula determines energy expenditure with <1% error compared with the metabolic rate determined by **Equation 4** ((**17**)).

Sample Analysis.
Six blood samples were drawn from an antecubital vein, immediately before exercise and at minutes 0, 30, 60, 120, and 180 post-exercise for each treatment. Serum was analyzed for glucose colorimetrically using an automated sample analyzer (COBAS MIRI Plus; Roche Diagnostic Systems, Branchburg, NJ). Urine was analyzed for preformed ammonia and urea nitrogen colorimetrically using a urea nitrogen test kit (kit 640; Sigma Diagnostics, St. Louis, MO). This was a colorimetric method were urea was hydrolyzed by urease to ammonia and carbon dioxide. Preformed ammonia was measured before treating the urine with urease. Serum insulin was analyzed by radioimmunoassay (kit 14K; Linco Research, St. Louis, MO).

Hunger Ratings.
During the 3-hour postprandial measurement period, subjects were asked every 30 minutes to rate the degree to which they felt hungry by placing a mark on a visual analogue scale numbered zero to eight. Zero indicating “not hungry at all” and eight indicating “as hungry as I have ever felt.” A pictorial measure of hunger sensation was also used. This measure is based on subjects outlining on drawings of the human body where they experienced hunger at the stipulated times ((**18**)). The area of sensation on the body associated with hunger was measured by photocopying the marked figure drawings and weighing each marked area.

Statistical Analysis
All data are expressed as means ± SE. The results of the four treatments were compared using ANOVA with repeated measures. Posthoc testing was performed by using the paired comparison *t* test with a two-sided rejection region and a confidence level of 95%. Tukey's Honestly Significantly Different procedure was used to adjust significant levels where applicable. Statistical analysis was performed with the SPSS software package release 7.5.2 (SPSS Inc. Chicago, IL).

Results

Body Weight
Body weight remained stable during the EB periods. However, 6 days on the NEB diets resulted in a significant ($p < 0.05$) loss of body weight, 1.09 ± 0.08 kg and 0.56 ± 0.11 kg for NEBHG and NEBHF, respectively.

Plasma Glucose
Response curves for plasma glucose concentrations are shown in **Figure 2**. Glucose concentrations at minutes 30 and 60 after the EBHG and NEBHG meals were significantly ($p < 0.05$) elevated from baseline (time 0) and significantly ($p < 0.01$) greater compared with EBHF and NEBHF. Peak glucose concentrations of the EBHF and NEBHF meals were also significantly higher than baseline (time 0). Glucose concentrations were not significantly different from baseline (before exercise) at minute 120 for the high glucose meals and minute 60 for the high fructose meals.

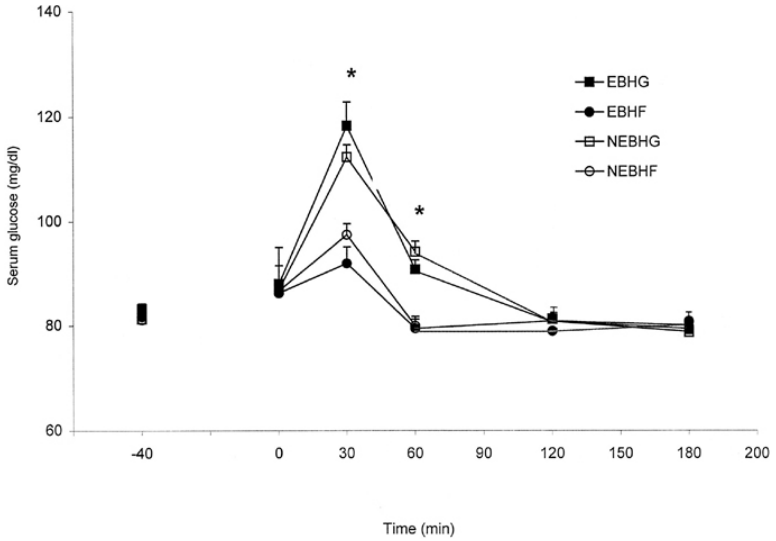

Figure 2 Plasma glucose concentration (mean ± SE) in obese subjects before 40 minutes of 70% VO_{2peak} exercise and after ingestion of a PELM. Meal was given at time 0. At time 30 and 60 minutes, EBHG and NEBHG were significantly greater than EBHF and NEBHF ($p < 0.01$, $p < 0.05$, respectively).

Serum Insulin

Response curves for serum insulin are shown in **Figure 3**. Insulin concentrations at 30 and 60 minutes were significantly ($p < 0.0001$) greater compared with baseline (time 0). Peak insulin concentration occurred at 30 minutes and was significantly ($p < 0.05$) higher for the EBHG than for the EBHF and NEBHF meals. At minute 60, the insulin concentration was significantly ($p < 0.05$) higher after the EBHG and NEBHG meals than after EBHF and NEBHF. At 180 minutes, serum insulin concentration was not significantly different from baseline (time 0).

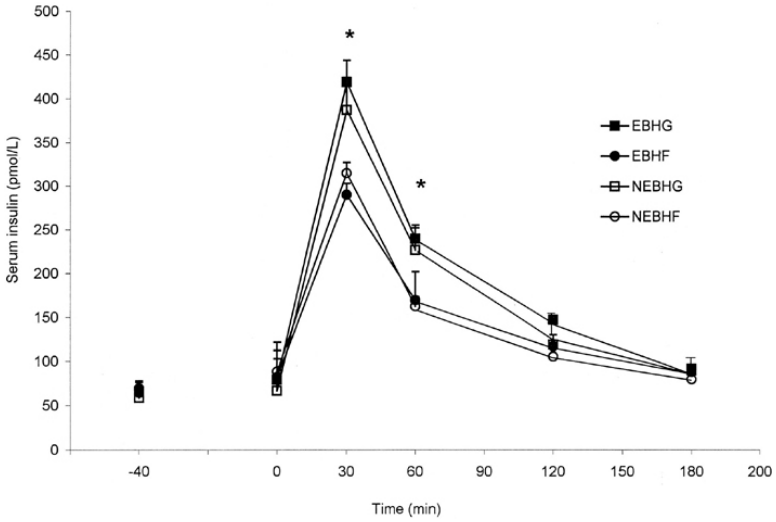

Figure 3 Serum insulin concentration (mean ± SE) in obese subjects before 40 minutes of 70% VO_{2peak} exercise and after ingestion of PELM. Meal was given at time 0. At time 30 minutes, EBHG was significantly greater than EBHF ($p < 0.05$). EBHG and NEBHG were significantly greater than EBHF and NEBHF ($p < 0.05$) at 60 minutes.

Energy Expenditure

Resting energy expenditure was similar pre-exercise in all four treatments (**Figure 4**). Post-exercise/post-meal energy expenditure peaked at 43.17, 41.35, 50.28, and 42.20 kcal/30 min after the EBHG, NEBHG, EBHF, and NEBHF meals, respectively. When summed over the 3-hour measurement period, energy expenditure was not significantly different between trials. Summed values over the 3-hour period were 239 ± 16.1, 229 ± 12.5, 264.0 ± 31.5, 63.1 ± 7.50, and 237 ± 14.0 kcal for EBHG, NEBHG, EBHF, and NEBHF, respectively.

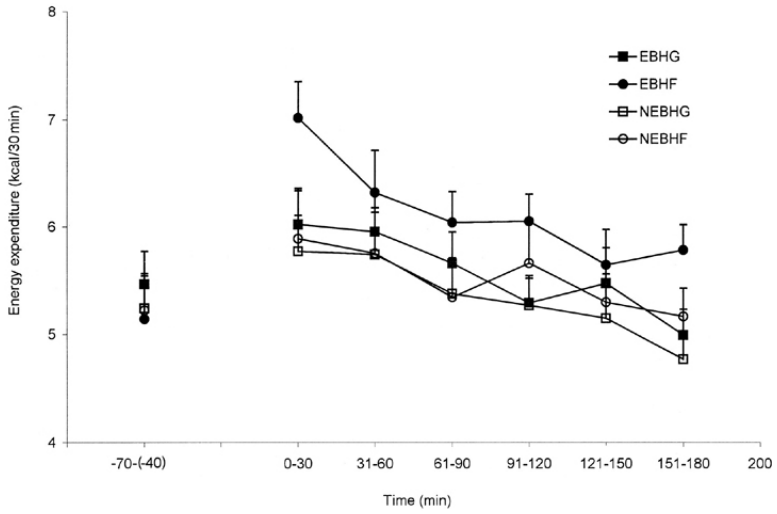

Figure 4 Integrated REE (mean ± SE) in obese subjects before 40 minutes of 70% VO_{2peak} and after ingestion of PELM. Meal given at time 0. REE was measured in 30-second intervals and then averaged over each 30-minute period.

Substrate Oxidation

The response curves for carbohydrate oxidation are shown in **Figure 5**. Pre-exercise carbohydrate oxidation rates were similar for all treatments. There was a significant ($F = 6.55$, $p < 0.001$) increase in carbohydrate oxidation with all PELM values during all periods of energy intake. Peak carbohydrate oxidation rates were 7.32 ± 0.89, 7.59 ± 0.88, 10.67 ± 1.60, and 9.24 ± 1.82 g/30 min for EBHG, NEBHG, EBHF, and NEBHF, respectively. At time intervals 0 to 30 and 31 to 60 minutes, carbohydrate oxidation after EBHF was significantly greater then EBHG and NEBHG ($p < 0.05$). Substrate oxidation summed over the 3-hour postprandial period led to significantly ($p < 0.05$) more carbohydrate being oxidized after EBHF (43.50 ± 6.96 g) than after EBHG (29.84 ± 4.57 g) and NEBHG (30.84 ± 3.34 g).

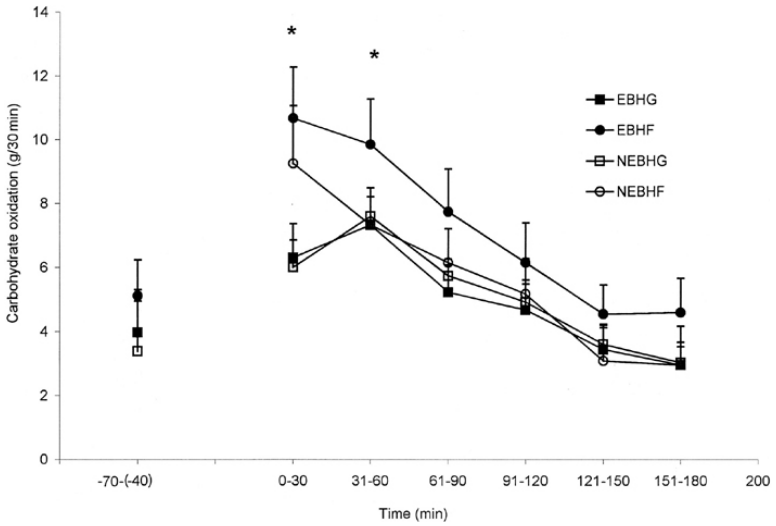

Figure 5 Integrated carbohydrate oxidation rates (mean ± SE) in obese subjects before 40 minutes of 70% VO_{2peak} exercise and after ingestion of PELM. Meal was given at time 0. EBHF was significantly greater between time intervals 0 to 30 and 31 to 60 minutes than were EBHG and NEBHG ($p < 0.05$).

Pre-exercise fat oxidation rates were similar for all treatments (**Figure 6**). Fat oxidation decreased significantly ($p < 0.05$) at time intervals 0 to 30 and 31 to 60 minutes for EBHF and NEBHF and at time interval 31 to 60 minutes for EBHG and NEBHG compared with REE. Fat oxidation increased significantly ($F = 3.27$, $p < 0.05$) over time subsequent to exercise and test meals for all treatments. When summed over the 3-hour postprandial period, fat oxidation was significantly ($p < 0.05$) lower after EBHF (17.63 ± 4.71 g) than after EBHG (29.17 ± 5.02 g) and NEBHF (27.79 ± 5.62 g). Summed substrate oxidation is depicted in **Figure 7**.

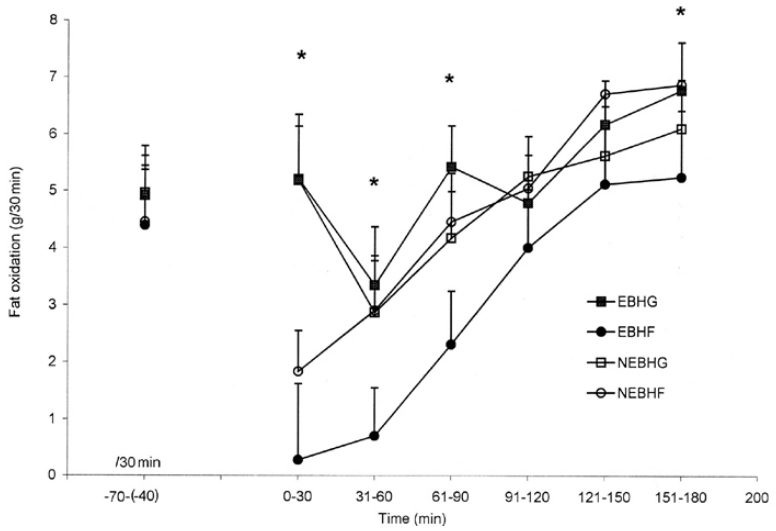

Figure 6 Integrated fat oxidation rates (mean ± SE) in obese subjects before 40 minutes of 70% VO_{2peak} exercise and after ingestion of PELM. Meal was given at time 0. At time interval 0 to 30 minutes, the fructose PELM values were significantly lower than the glucose PELM values. Significant differences occurred at time intervals 31 to 60 and 151 to 180 minutes between EBHF and NEBHF and at the 61- to 90-minute interval between EBHF and EBHG (*$p < 0.05$). Throughout the entire postprandial period, EBHF remained significantly lower than EBHG, NEBHG, and NEBHF ($F = 4.68$, $p < 0.01$).

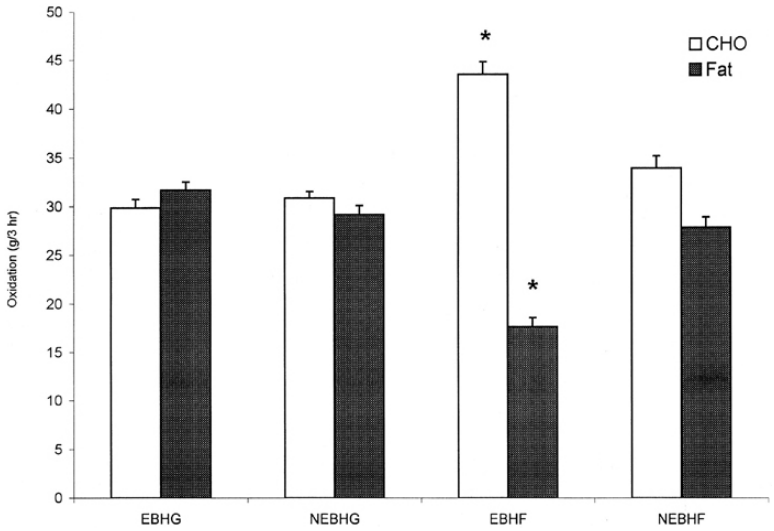

Figure 7 Postprandial carbohydrate and fat oxidation rates (mean ± SE) in obese subjects summed over 3 hours. The quantity of carbohydrate oxidized was significantly higher for the EBHF ($p < 0.05$) than for the EBHG and NEBHG meals. The quantity of fat oxidized over this period was significantly lower than the EBHG and NEBHF ($p < 0.05$, $p < 0.01$, respectively).

Hunger Ratings

There were significant ($p < 0.01$) increases in perceived hunger as time elapsed for all treatments measured by both the analog and pictorial scales. Ratings of perceived hunger were similar with all treatments.

Discussion

The purpose of this study was to assess the combined acute effects of exercise and NEB on the metabolic utilization of energy substrates by determining the metabolic consequences of consuming a high glucose vs. high fructose meal after exercise during EB or NEB in overweight individuals. High fructose foods are not potent insulin secretagogues. Thus, the actions of insulin on lipogenesis and lipolysis are negated, i.e., insulin-stimulated lipogenesis and insulin's effect on attenuating lipolysis are reduced. The use of fructose as a low glycemic index sweetener has also gained popularity due to its greater sweetening capacity per kilocalorie compared with glucose ((**19**)), and it has been recommended as a replacement for other sugars in the diets of diabetic and obese people. In addition, fructose promotes a greater thermogenic response than glucose ((**8**)). For these reasons, high fructose foods have been recommended during periods of intentional weight loss via energy restriction.

During the 3-hour postprandial period, blood glucose and insulin concentrations reached higher peak values and took longer to return to baseline after the EBHG and NEBHG meals than after the EBHF and NEBHF meals. Energy restriction did not affect these responses and can be explained by the well-documented glycemic properties of the carbohydrate used in each type of meal. Glucose is absorbed by active transport and is metabolized primarily in muscle tissue, whereas fructose is absorbed by carrier-mediated facilitated diffusion and is rapidly metabolized in the liver ((**20**)).

Fructose is rapidly removed from the portal circulation and subsequently phosphorylated to fructose 1-phosphate by fructokinase. The formation of the triose phosphates glyceraldehyde 3-phosphate and

dihydroxyacetone phosphate is the point were glucose and fructose metabolism converge, although, fructose arrives at this point by bypassing the main rate-controlling enzyme phosphofructokinase. This unique property of fructose explains, in part, the greater increase in carbohydrate oxidation with fructose consumption.

Unlike the EBHF, carbohydrate oxidation during the NEBHF was similar to both glucose trials. Although glycogen levels were not measured, this phenomenon may be attributed to the lower hepatic glycogen levels after the energy restricted diet. Fructose is rapidly phosphorylated to dihydroxyacetone phosphate and glyceraldehyde 3-phosphate providing substrate primarily for glycolysis and subsequent oxidative phosphorylation. We hypothesize that, during energy restriction when hepatic glycogen stores are low, the fructose-derived triose phosphate intermediates are shifted from glycolysis and oxidative phosphorylation to glycogenesis. Therefore, rather than being oxidized for energy, fructose is directed toward replenishing glycogen stores during energy restriction. The greater amount of carbohydrate oxidized during the EB high fructose treatment may also be explained by the formation of fructose 1-phosphate and its involvement in attenuating the inhibitory effects of glucokinase's regulatory protein. Fructose 1-phosphate binds to this regulatory protein and abolishes its inhibitory effect on glucokinase ((**21**)). Increased glucokinase activity is associated with stimulation of glucose phosphorylation, glycolysis, and glycogen synthesis ((**22**)). Elevated fructose 1-phosphate formation would most likely occur equally among the two fructose meals and is likely, in part, one of the explanations for the elevated carbohydrate oxidation after the EBHF meal and the speculated increase in glycogenesis in the NEBHF meal.

Oxidation rates for fat were opposite to those for carbohydrate oxidation. The mechanism that inhibits fatty acid oxidation during concurrent elevated carbohydrate oxidation rates in humans remains unclear ((**23**)). However, the regulatory properties of malonyl-coenzyme A seem to have a major role in fatty acid oxidation in liver and muscle tissue. During heightened carbohydrate flux, hepatic glycolysis and mitochondrial citrate synthesis are elevated. The consequent increased formation of malonyl-coenzyme A inhibits the activity of carnitine palmitoyltransferase 1, the rate-limiting enzyme responsible for transporting long-chain fatty acids into the mitochondria ((**24**)).

Fructose elicits a greater thermogenic response than glucose in normal weight humans ((**8**)). However, the extent to which obesity blunts the thermogenic effect of food remains a subject of controversy. Previous research comparing the thermic response between glucose and fructose in the obese indicates that fructose is more thermogenic ((**2**)). Although mean energy expenditure was higher after the EBHF, it was not significant. We suspect this lack of significance between glucose and fructose thermogenesis occurred due to the large interindividual variability (coefficient of variation = 25.5, 44.6, 20.5, and 22.2% for EBHG, EBHF, NEBHG, and NEBHF, respectively).

The potential role that fructose may have in the control of food intake has also provided rationale for its use in weight loss drinks. Some studies have suggested that fructose ingestion modulates subsequent food intake ((**4**), (**25**)). This study could not determine such a phenomenon, because food intake was not allowed during post-exercise measurements and we did not quantify food intake after the end of the experiment. Hunger ratings during the 3-hour postprandial period revealed increasing hunger over time, but no significant differences where observed between treatments. Although this

does not support the proposed satiety effect of fructose, the association between hunger and food intake is weak ((**26**)).

Studies in humans ((**27**), (**28**), (**29**)) have consistently demonstrated that fructose is more lipogenic then glucose. Zakim et al. ((**27**)) demonstrated that the conversion of [^{14}C]fructose to free fatty acids and carbon dioxide was between 3- and 8-fold greater than an equivalent amount of [^{14}C]glucose. Increasing dietary fructose consumption can also significantly increase fasting plasma triglyceride and cholesterol concentrations ((**30**), (**31**)). It is important to note that this study was performed using healthy obese subjects, and these results may not apply to individuals with altered carbohydrate metabolism such as impaired glucose tolerance and diabetes as well hypertensive, hyperinsulinemic, hypertriglyceridemic, and postmenopausal people.

In conclusion, this study demonstrates that consumption of a high fructose meal after exercise during NEB provokes a macronutrient utilization response similar to that of a high glucose meal. Future studies are necessary to determine whether these changes were a result of reduced energy intake, a bout of exercise, or a combination of both. The data suggest that the use of fructose in supplements/meals provides no additional benefit in terms of substrate utilization during a weight loss program involving diet and exercise. However, with adequate energy intake, a high glucose meal post-exercise seems to produce a more favorable metabolic response (greater carbohydrate storage and fat oxidation) than a high fructose meal.

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