Acute fuel selection in response to high-sucrose and high-starch meals in healthy men1–3

Mark E Daly, Catherine Vale, Mark Walker, Alison Littlefield, K George, MM Alberti, and John Mathers

ABSTRACT

Background: Despite considerable controversy over the inclusion of sucrose in the diets of people with diabetes, the acute metabolism of sucrose is not completely understood.

Objective: Our objective was to investigate the metabolism of the monomeric constituents of sucrose after a high-sucrose meal.

Design: Three test meals were consumed in a randomized, crossover design by 7 healthy male volunteers. Two of the meals were high in sucrose; one was supplemented with 200 mg uniformly labeled [13C]fructose and one was supplemented with 200 mg [13C]glucose. The other meal was high in starch, supplemented with 200 mg [13C]glucose. Fifty percent of energy was supplied as sucrose in the high-sucrose meals and as starch in the high-starch meal. Breath [13C]CO2 enrichment was measured at 15-min intervals and indirect calorimetry was performed for five 20-min sessions immediately before and during a 6-h postprandial period.

Results: Carbohydrate oxidation rates rose much faster after the high-sucrose meals than after the high-starch meal. Breath [13C]CO2 enrichment rose faster and peaked earlier and at a higher value when [13C]fructose rather than [13C]glucose was given with the high-sucrose test meal. Values for breath [13C]CO2 enrichment from [13C]glucose after the high-starch meal were intermediate.

Conclusions: These results show that fructose is preferentially oxidized compared with glucose after a high-sucrose meal.

INTRODUCTION

The metabolic effects of high-sucrose and high-fructose diets have been the subject of intense study for the past 25 y. The effects of such diets in laboratory animals are well characterized, but there is controversy about responses in humans. Diets that are very high in sucrose or fructose can induce insulin resistance or hypertriglyceridemia in animals (1–10), particularly rats. Evidence indicates that certain groups of humans, possibly those who are initially hypertriglyceridemic or insulin-resistant, are more susceptible to the effects of such diets (11, 12; see 13 for a recent review). These studies (11, 12) included individuals who were deemed to be insulin resistant on the basis of exaggerated postprandial hyperinsulinemia but who did not have diabetes. Other authors have argued that a review of the literature suggests that people with diabetes may be protected from this susceptibility to hyperlipidemia by chronic consumption of high-sucrose diets (14). The liver is the main site of fructose metabolism (15–17), and studies have shown that oral ingestion of fructose or sucrose increases the carbohydrate oxidation rate and thermogenesis more than does ingestion of glucose (18–20). However, it is less clear how the monosaccharides glucose and fructose are metabolized when consumed together as the disaccharide sucrose and also in comparison with other sources of commonly consumed carbohydrates, such as starch.

Understanding the metabolic consequences of sucrose intake is important, because this sugar makes a substantial contribution to habitual diets in many countries; eg, in the United Kingdom sucrose contributes ~16% of total dietary energy (21). Current recommendations to lower fat intake with the intention of reducing cardiovascular disease risk are matched by recommendations to increase total carbohydrate intake, especially intake of complex carbohydrates (22). Previous studies have shown a tendency for individuals to increase their intake of sugars when they reduce fat intake (23), which may lead to a further increase in sucrose consumption. In the present study, we investigated the acute metabolism of the monosaccharide components of sucrose in the resting postprandial state to test the hypothesis that there is differential fuel selection between glucose and fructose when they are consumed as part of a sucrose-rich meal. Comparisons were made with a high-starch test meal.

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SUBJECTS AND METHODS

Subjects

Seven healthy, weight-stable male volunteers were recruited from the students and staff of the University of Newcastle upon Tyne and the staff of the Royal Victoria Infirmary, Newcastle upon Tyne. None had diabetes mellitus (or a first-degree relative with diabetes), ischemic heart disease, hypertension, or any other disease associated with altered insulin sensitivity. None were taking any drugs known to alter insulin sensitivity or affect carbohydrate or lipid metabolism. All were nonsmokers and had a habitual alcohol intake of <21 units/wk (1 unit = 8 g alcohol; 24).

The experimental protocol was approved by the Joint Ethics Committees of the Newcastle and North Tyneside Health Authorities, the University of Newcastle upon Tyne, and the University of Northumbria at Newcastle. Each subject gave informed, written consent. All studies were conducted in the Human Diabetes and Metabolism Research Centre, Royal Victoria Infirmary, Newcastle upon Tyne, United Kingdom.

Experimental protocol

Each subject took part in 3 experimental sessions according to a randomized, crossover design. Subjects were admitted at 0700 after they had fasted since 2200 the previous evening. Alcohol and strenuous exercise were avoided for 24 h before each experimental session. Subjects consumed high-sucrose test meals on 2 occasions and a high-starch test meal on 1 occasion. A combination of techniques that involved stable isotopes and indirect calorimetry was used to determine fuel selection and substrate oxidation. Concentrations of blood glucose, serum insulin, plasma fatty acids, serum triacylglycerols, and other blood metabolites (pyruvate, lactate, glycerol, and hydroxybutyrate) were measured frequently. Subjects recorded their food intake for the 24-h period before the first experiment and were asked to repeat that food intake pattern for the 24 h before each subsequent experiment. Each study period was separated from the next by ≥1 wk of washout.

Anthropometry

Percentage body fat was calculated with the equations of Siri (25) by using estimates of body density. These estimates were derived from skinfold-thickness measurements (Holtain/Tanner-Whitehouse skinfold caliper; Holtain, Crosswell, United Kingdom) taken at 4 separate sites (26).

Assessment of fuel selection

Indirect calorimetry

Carbon dioxide production (\(\dot{V}_{\text{CO}_2}\)) and oxygen consumption (\(\dot{V}_{\text{O}_2}\)) were measured with a Deltatrac Indirect Calorimeter (Datex Instrumentarium Corporation, Helsinki) that had a ventilated, transparent hood system. Five measurements (1 basal and 4 postprandial), each of 20 min duration, were made during each experimental period. Urine was also collected during each 6-h experimental period, and urinary nitrogen excretion was measured by combustion and IRMS of the resulting carbon dioxide.

\(\dot{V}_{\text{CO}_2}\) enrichment represents the excess \(^1\text{C}\) present compared with background levels of the isotope. By using IRMS, the differences between measured \(^1\text{C}\) and the international standard for \(^1\text{C}\) abundance, Pee Dee Belemnite (PDB), were calculated and expressed as atoms percent:

\[
\text{Atoms percent} = 100 \times \frac{\dot{V}_{\text{CO}_2}}{\dot{V}_{\text{CO}_2} + \dot{V}_{\text{O}_2}} \times \frac{\text{APDB} + 1}{\text{PDB} + 1}
\]

where the International Pee Dee Belemmite Standard = 0.0112372. This was then transformed into atoms percent excess (levels of enrichment of the isotope above an individual’s norm) by calculating the changes from baseline for each individual value, where baseline was calculated as the mean of 2 samples taken 15 min apart before consumption of the test meal. This yielded the excess of \(^1\text{C}\) compared with \(^1\text{C}\) and, together with quantification of \(\dot{V}_{\text{CO}_2}\) by indirect calorimetry, may be used to calculate the rate of total \(^1\text{C}\) produced in mL/min. This was then converted into molar concentrations of \(^1\text{CO}_2\) according to the following equation:

\[
\text{\(^{1\text{CO}}_2\) (\mu\text{mol/min}) = \frac{1}{22260} \times \frac{\text{\(^{1\text{CO}}_2\) produced \times 1000000}}{22260}\}
\]

where 22260 is the constant for the volume (in mL) occupied by 1 mol CO\(_2\) under standard temperature and pressure conditions.

From this value, the quantity of labeled monosaccharide that was oxidized was calculated by dividing by 6 (based on the assumption that all carbon atoms were uniformly labeled in the tracer). The resulting value was then converted into the mass of tracer oxidized/min by multiplying by 186, the molecular weight of the uniformly labeled hexose. Finally, the total quantity of labeled \(^1\text{C}\) compared with baseline enrichment (the 200 mg added label) and the enrichment above the individual’s baseline measurements were used to derive the percentage of label oxidized/min, which also reflected the percentage of monosaccharide supplied by the meal that was oxidized/min. This was then divided by 100 and multiplied by the total amount of available monosaccharide in each test meal, whether in polysaccharide or disaccharide form, to calculate the quantity of monosaccharide oxidized/min.

Metabolic profiles

Arterialized blood samples were obtained via a retrograde intravenous cannula inserted into a dorsal vein of a hand kept in a
TABLE 1
Composition of the test meals

<table>
<thead>
<tr>
<th></th>
<th>High-starch meal</th>
<th>High-sucrose meal</th>
<th>% of energy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein</td>
<td>8.0</td>
<td>7.7</td>
<td></td>
</tr>
<tr>
<td>Total carbohydrate</td>
<td>64.8</td>
<td>65.0</td>
<td></td>
</tr>
<tr>
<td>Starch</td>
<td>50.1</td>
<td>8.2</td>
<td></td>
</tr>
<tr>
<td>Sucrose</td>
<td>7.9</td>
<td>50.2</td>
<td></td>
</tr>
<tr>
<td>Other sugars</td>
<td>6.7</td>
<td>6.6</td>
<td></td>
</tr>
<tr>
<td>Total fat</td>
<td>26.7</td>
<td>26.0</td>
<td></td>
</tr>
<tr>
<td>Saturated</td>
<td>8.0</td>
<td>8.3</td>
<td></td>
</tr>
<tr>
<td>Monounsaturated</td>
<td>8.2</td>
<td>8.5</td>
<td></td>
</tr>
<tr>
<td>Polyunsaturated</td>
<td>8.0</td>
<td>8.4</td>
<td></td>
</tr>
</tbody>
</table>

Heated box at 55 °C. Samples were taken at baseline, every 15 min for the first 2 h after the meal, and every 30 min thereafter. Blood glucose concentrations were measured with a glucose analyzer (Yellow Springs Instrument Co, Yellow Springs, OH) by using the glucose oxidase method (interassay CV: 1.7%). Serum insulin concentrations were estimated by using an enzyme-linked-imunosorbent–specific insulin assay with an intraassay CV of 3.2% (DAKO Diagnostics Ltd, Ely, United Kingdom). Plasma fatty acid concentrations were estimated by using an enzymatic colorimetric method with an intraassay CV of 3.2% (Wako Chemicals GmbH, Neuss, Germany). Blood concentrations of metabolites including lactate, hydroxybutyrate, and glycerol (intraassay CVs of 3.2%, 3.5%, and 4.3%, respectively) were measured with a centrifugal analyzer fitted with a fluorimetric attachment (28).

TABLE 2
Sample menus for the 2 test meals

<table>
<thead>
<tr>
<th>Meal component</th>
<th>Amount (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High-sucrose meal</td>
<td></td>
</tr>
<tr>
<td>Custard</td>
<td></td>
</tr>
<tr>
<td>Skim milk</td>
<td>270</td>
</tr>
<tr>
<td>Whipping cream</td>
<td>14</td>
</tr>
<tr>
<td>White sugar</td>
<td>34</td>
</tr>
<tr>
<td>Full-fat margarine</td>
<td>17</td>
</tr>
<tr>
<td>High-MUFA margarine</td>
<td>6.5</td>
</tr>
<tr>
<td>Custard powder</td>
<td>18.4</td>
</tr>
<tr>
<td>Jelly</td>
<td></td>
</tr>
<tr>
<td>Sugar-free jelly powder</td>
<td>10</td>
</tr>
<tr>
<td>White sugar</td>
<td>35</td>
</tr>
<tr>
<td>Carbonated sugar drink</td>
<td>320</td>
</tr>
<tr>
<td>High-starch meal</td>
<td></td>
</tr>
<tr>
<td>Milkshake</td>
<td>210</td>
</tr>
<tr>
<td>Skim milk</td>
<td>14</td>
</tr>
<tr>
<td>Whipping cream</td>
<td>16</td>
</tr>
<tr>
<td>Custard powder</td>
<td>63</td>
</tr>
<tr>
<td>White bread</td>
<td>110</td>
</tr>
<tr>
<td>Full-fat margarine</td>
<td>16</td>
</tr>
<tr>
<td>High-MUFA margarine</td>
<td>6</td>
</tr>
</tbody>
</table>

1Each meal provided 40% of a daily requirement of 8400 kJ (3360 kJ). Volunteers with higher energy requirements received greater amounts of foods but the same percentage of energy from each macronutrient. MUFA, monounsaturated fatty acid.

2 Contained corn flour, salt, flavoring, and color (annatto).

TABLE 3
Characteristics of the volunteers

<table>
<thead>
<tr>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
</tr>
<tr>
<td>BMI (in kg/m²)</td>
</tr>
<tr>
<td>Body fat (%)</td>
</tr>
<tr>
<td>Waist-to-hip ratio</td>
</tr>
</tbody>
</table>

1 ± SD; range in parentheses; n = 7.
2 Calculated from skinfold-thickness measurements (25).

Test meals and habitual dietary intakes of subjects

Test meals were planned with the aid of the computer program MICRODIET (29) and United Kingdom national food tables (30). Each test meal provided 40% of the estimated daily energy requirement, which was calculated by multiplying basal metabolic rate (determined from age, sex, and body mass; 31) by a physical activity factor of 1.5 (32). Quantities of all food items were adjusted according to each individual’s calculated energy requirement so that proportions of nutrients did not differ among the subjects.

The 2 test meals were similar in terms of the proportions of energy supplied by protein, carbohydrate, and fat; the macronutrient composition of the meals is summarized in Table 1. Both meals were high in carbohydrates (~65% of total energy) and they differed only in the source of the carbohydrate. Fifty percent of energy was supplied by sucrose for the high-sucrose meals and by starch for the high-starch meal. Readily available foods rather than formula meals were used; sample menus for the 2 test meals are shown in Table 2. The starch in the custard powder was provided by corn flour (maize starch), which we found to be 99% digested in the small bowel of an animal model (33). The subjects’ habitual food intakes were estimated by asking them to keep a 7-d food record. Subjects quantified their food portion sizes with the aid of a photographic food atlas (34).

Statistical analysis

Statistical analysis was carried out by using MICROSOFT OFFICE EXCEL (Microsoft Corp, Redmond, WA). When appropriate, paired data such as areas under the curve were compared by using a two-tailed paired t test. When data for the 2 high-sucrose meals appeared to simply represent repetition (eg, plasma glucose profiles), we conducted analyses to confirm that there were no significant differences between the 2 high-sucrose meals. Then the mean of the values for the 2 high-sucrose meals

TABLE 4
Habitual daily energy and nutrient intakes of the volunteers

<table>
<thead>
<tr>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (MJ)</td>
</tr>
<tr>
<td>Protein (% of energy)</td>
</tr>
<tr>
<td>Total carbohydrate (% of energy)</td>
</tr>
<tr>
<td>Sucrose (% of energy)</td>
</tr>
<tr>
<td>Total fat (% of energy)</td>
</tr>
<tr>
<td>Saturated</td>
</tr>
<tr>
<td>Monounsaturated</td>
</tr>
<tr>
<td>Polyunsaturated</td>
</tr>
<tr>
<td>Alcohol (% of energy)</td>
</tr>
</tbody>
</table>

1 ± SD; n = 7.

Alcohol (% of energy) | 12 ± 4.8  |
was compared with the data for the high-starch meal. Data are presented as means ± SEMs or SDs.

RESULTS

Subject characteristics

Our subjects were a group of healthy men with a wide age range (22–68 y). Their mean BMI (in kg/m²) was within the normal range, but their mean percentage body fat (21%) was relatively high (Table 3). The habitual dietary intake data suggested that mean consumption of carbohydrates was higher and that of fats was lower (Table 4) than intakes reported for the adult population of the United Kingdom (21).

Fuel selection

The carbohydrate and lipid oxidation rates estimated from indirect calorimetry after the test meals are summarized in Figures 1 and 2, respectively. To obtain the high-sucrose-meal values, we used the means of the values for the 2 high-sucrose meals. After the high-sucrose meals were consumed, there was a marked increase in car-
bohyrate oxidation, with a corresponding decline in lipid oxidation; the latter remained suppressed until 150 min after the meal. Thereafter, both the lipid and the carbohydrate oxidation rates returned to fasting values. After the high-starch meal, there was a smaller and more delayed increase in the carbohydrate oxidation rate, mirrored by a smaller and more delayed decrease in the lipid oxidation rate.

The enrichment of breath carbon dioxide increased much more rapidly and to a higher peak after the [13C]fructose-labeled high-sucrose meal than after the other test meals (Figure 3). 13C labeling of breath carbon dioxide was slower and peaked much later when the labeled glucose accompanied the sucrose-rich meal than when it accompanied the starch-rich meal. In all cases, breath carbon dioxide enrichment remained substantially elevated above background levels 6 h after the test meals, a time when rates of oxidation for carbohydrate and lipids had returned to fasting values. In comparisons of the enrichment of carbon dioxide from the labeled glucose after the high-starch and high-sucrose meals, note that the 200 mg of tracer was in a larger pool of glucose with the high-starch meal than with the high-sucrose meal. This effect is accounted for in Figure 4.
which we showed the rate of carbohydrate oxidation derived from the stable-isotope measurements. These values were calculated from the enrichment values of breath carbon dioxide in atoms percent excess, the VCO₂ data, the quantity of isotope administered, and the carbohydrate contents of the meals. Separate oxidation rates for the fructose and glucose moieties and for total carbohydrate (glucose plus fructose) in the high-sucrose test meals are shown in Figure 4, as is the glucose oxidation rate after the high-starch meal. Fructose was oxidized much more rapidly than glucose after the high-sucrose meals. The curve representing glucose oxidation after the high-starch meal was higher than that for the individual monosaccharides in the high-sucrose meals, which was expected given the greater quantity of glucose supplied by the high-starch meal. However, the overall carbohydrate oxidation rate after the high-sucrose meals was higher than the glucose oxidation rate after the high-starch meal, which is consistent with the indirect calorimetry data (Figure 1).

**Metabolic profiles**

The glycemic profiles for the 2 test meals were quite different (Figure 5). There was a more rapid rise in blood glucose concentration after the high-sucrose meal than after the high-starch meal, to a slightly higher (NS) peak value. Blood glucose concentrations dropped more rapidly to a lower value after the high-sucrose meal than after the high-starch meal. Post-

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**FIGURE 5.** Mean (±SEM) plasma glucose and serum insulin concentrations after consumption of the test meals. $n = 7$. 

![Graph showing plasma glucose and serum insulin concentrations over time after consumption of test meals.](https://academic.oup.com/ajcn/article-abstract/71/6/1516/4729491)
prandial blood glucose concentrations after the first 75 min were maintained at higher values after the high-starch meal than after the high-sucrose meal. Similarly, after the high-sucrose meal there was a higher (NS) peak in serum insulin concentration, which then tended to be lower during the final 3 h of the experimental period.

Plasma fatty acid concentrations fell rapidly after both meals (Figure 6) and remained below fasting concentrations throughout the postprandial period after the high-starch meal. In contrast, after the high-sucrose meal, plasma fatty acid concentrations began to rise at \(< 150\) min and returned to premeal values by the end of the measurement period. Pyruvate and lactate concentrations in whole blood rose more rapidly and to much higher peaks after the high-sucrose meal than after the high-starch meal (Figure 7). After both test meals, concentrations of both metabolites returned to fasting values by the end of the study.

**DISCUSSION**

The health effects of fructose and sucrose are of considerable interest for several reasons. It is thought that the fructose component causes most of the characteristic metabolic effects of high-sucrose diets (ie, those not shared by high-starch diets), whether these effects occur acutely after consumption of a meal or with longer-term exposure to a sucrose-rich diet (13). However, there has been a paucity of research on the effects of dietary sucrose on tissue fuel selection and on how the 2 constituent monosaccharides (glucose and fructose) are metabolized relative to each other. The main purpose of this study was to compare the metabolism of glucose and fructose after a high-sucrose meal, with the metabolism of glucose after a starch-rich meal as a reference point.

Clearly, under these conditions, fructose is oxidized more rapidly than is glucose (Figure 3). This is not unexpected given what is understood about its metabolic fate. Only small amounts of fructose are found in the peripheral blood after sucrose or fructose ingestion because absorbed fructose is predominantly taken up by the liver. The rapid metabolism of fructose may be explained in part by the fact that it bypasses one of the key regulatory enzymes in glycolysis, 6-phosphofructokinase. Fructose enters glycolysis after phosphorylation to fructose-1-phosphate, a reaction that is mediated by hepatic fructokinase. Fructose-1-phosphate stimulates pyruvate kinase, which explains the high concentrations of pyruvate and consequently lactate after fructose ingestion; research by Brundin and Wahren (17) supports this. They found much greater lactate and pyruvate concentrations after human volunteers consumed a 75-g fructose load than after they consumed a 75-g glucose load. Our use of \([13\text{C}]\)glucose and \([13\text{C}]\)fructose in separate but otherwise identical high-sucrose test meals enabled us to investigate both constituents of sucrose. Despite the rapid increase in carbohydrate oxidation rate after the high-sucrose test meals, glucose was oxidized much more slowly than was fructose and was also oxidized more slowly than was the labeled glucose in the high-starch test meal (Figures 3 and 4).

Quantitative interpretation of the isotopic-tracer studies is based on several assumptions. The first assumption is that the amount of \(13\text{C}\) in breath carbon dioxide above fasting amounts was derived from the oxidation of the administered dose of \([13\text{C}]\)glucose or \([13\text{C}]\)fructose. Our gas chromatography–IRMS measurements showed that the other constituents of the test meals contributed \(< 3\%\) of the excess \(13\text{C}\). The second assumption is that the enrichment of breath carbon dioxide in the fasting period (before the test meal) is a reliable estimate of the enrichment of carbon from oxidation of exogenous materials throughout the study. In theory, this assumption might present a problem if the enrichments of body stores of fat and carbohydrate were markedly different and if there was a major shift in the proportion of each that was oxidized in response to the test meal. However, background enrichments are relatively low in European subjects, so this potential problem is likely to have little or no effect (35).

**FIGURE 6.** Mean (±SEM) plasma fatty acid concentrations after consumption of the test meals. \(n = 7\).
The third assumption is that oxidation of $[^{13}\text{C}]$carbohydrate can be quantified on the basis of enrichment of breath $^{13}\text{CO}_2$. Carbon dioxide produced as the end product of oxidation reactions is in equilibrium with the body’s bicarbonate pool and a considerable proportion ($\geq 50\%$) of label administered as bicarbonate (36) can be sequestered in the body during the time course of a study. Provided that the size of the bicarbonate pool was similar for all treatments, such sequestration would not be expected to alter the conclusions drawn about differences among the treatments in our study. This assumption would be true for both sucrose-rich meals, and therefore the comparison of oxidation rates for the glucose and fructose moieties of this disaccharide is not affected.

The greater rise in blood lactate concentrations after the high-sucrose meal than after the high-starch meal requires further consideration. An increase in blood lactate concentration tends to lower the pH. This in turn tends to decrease both the size of the bicarbonate pool and the amount of $^{13}\text{CO}_2$ being sequestered. This would lead to a higher estimate of carbohydrate oxidation. If this was quantitatively important in the present study, it would mean that the actual rate of glucose oxidation for glucose derived from sucrose would be even lower with respect to the rate of glucose oxidation for glucose derived from starch than is apparent from Figures 3 and 4 for the early and mid-postprandial phases.

In conclusion, this study showed that the fructose component of sucrose is oxidized much more rapidly than is the glucose

\[\text{FIGURE 7. Mean (± SEM) pyruvate and lactate concentrations in whole blood after consumption of the test meals.} \, n = 7.\]
component after a high-sucrose meal and that glucose is oxidized more rapidly after a high-starch meal than after a high-sucrose meal. Thus, fructose may have a sparing effect on oxidation of exogenously supplied glucose. These differences in fuel selection help explain the metabolic changes that accompany consumption of these 2 major dietary carbohydrates.

We are grateful to Lesley Morrison and Linda Ashworth for their hard work in the laboratory and to Sister Mavis Brown for her nursing assistance.

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