Effect of sucrose and safflower oil preloads on short term appetite and food intake of young men

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The effects of carbohydrate and fat on satiety have been examined primarily through meal composition studies. The purpose of this study was to compare the effects of pure sucrose and safflower oil, isovolumetric beverage preloads, on appetite (measured every 15 minutes by visual analogue scales) and food intake 60 minutes later. Young men consumed 0, 418, 836 and 1254 kJ of sucrose in the first two experiments and these same doses of safflower oil in the third. Finally, the largest doses of sucrose and safflower oil were compared. Sucrose, but not safflower oil, suppressed average appetite compared with control. In experiment 2, food intake was reduced \( (p < 0.05) \) by 518 kJ after the 418 and 836 kJ preloads and by 1129 kJ after the 1254 kJ sucrose preload. Only the 1254 kJ dose of safflower oil significantly suppressed food intake by 480 kJ in the third experiment. When the 1254 kJ doses were compared directly, sucrose suppressed food intake by 653 kJ compared with control whereas safflower oil did not. It is concluded that, in the short-term, sucrose produces a dose dependent reduction in appetite and food intake that is greater than that produced by safflower oil.

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Introduction

Although there is convincing evidence that protein is the most satiating macronutrient (DeCastro, 1987; Poppitt et al., 1998), the relative impact of fat and carbohydrate on satiety is less clear (Stubbs et al., 1996). Carbohydrate has been reported to be more satiating (Lissner, 1987; Rolls et al., 1990; Black et al., 1991; Blundell et al., 1993, 1994) or equally satiating to fat (Foltin et al., 1990; deGraaf et al., 1992; Rolls et al., 1994; Poppitt et al., 1998; Reid & Hammersley, 1999). This variation in results may be explained by choice of study population, experimental timing and test meal characteristics (Rolls et al., 1994). In addition, most studies have provided preloads that are only relatively high either in fat or carbohydrate. Thus the satiating power of the fat and carbohydrate components may be altered according to its association with other nutrients (Blundell & Macdiarmid, 1997).

The effect on satiety of individual pure carbohydrates has received some study, but primarily because of interest in the commonly used sweetener, sucrose. When provided to human subjects in the form of a sweet beverage, small preloads of 20 g (334 kJ) and 40 g (669 kJ) did not result in compensation at subsequent test meals (Rolls et al., 1988; Canty & Chan, 1991; Holt et al., 2000). These results could be interpreted to show that sugar calories are not detected and therefore contribute to obesity. However, the preloads used may have been below the energy threshold necessary for detection by food intake control mechanisms, and not necessarily a consequence of the source of calories. In general, the literature reports reduced mealtime intake results from ingestion of 50 g (836 kJ) or greater of sucrose, 20–60 min prior to a meal (Anderson, 1995). The threshold dose for detection of sucrose energy in comparison with fat or other carbohydrates has not been reported.

Only two studies have reported the effect of a pure lipid source, taken orally, on satiety and suggest that fat energy is either not detected or not different from other
sources of energy. In one, subjects fed a soup supplemented with or without 49 g (1880 kJ) of fat in the form of margarine ate similar amounts of food at a test meal presented 20 min later (Sepple & Read, 1990). In another, equicaloric (117 kJ) preloads of albumin, starch and corn oil fed as beverage drinks led to the consumption of equal amounts of a liquid test meal (Sustacal) 70 min later (Geliebter, 1979). These results are difficult to interpret because of the absence of a dose-response design and lack of monitoring for the time-course of satiety.

Thus the purpose of these four separate experiments was to define the short-term relationships among dose, subjective feelings of appetite and food intake after sucrose and safflower oil preloads.

**Methods**

**Subjects**

For Experiments 1 to 3, healthy, non-smoking males were recruited, aged 18 to 35 years with a body mass index (BMI) between 20 and 27 kg/m², the range generally accepted as ideal by Health and Welfare Canada (1990). For Experiment 4 the BMI upper cutoff was lowered to 25 in order to comply with the World Health Organization’s new stipulation of a healthy BMI range as 20 to 25 kg/m² (1998). Those who were diabetics, breakfast skippers, dieting or taking medication were excluded from all studies. Restrained eaters were also excluded upon their identification by a score of 11 or higher (Polivy et al., 1978; Herman & Polivy, 1980) on the administered Eating Habits Questionnaire. Subjects were recruited primarily by word of mouth and by posting signs around the University of Toronto St. George campus. The study was divided into four separate experiments, for which there were separate recruitments. However, subjects were allowed to participate in more than one experiment as they wished. The University of Toronto Human Subjects Review Committee approved this study.

Twelve subjects (mean age: 22.0, mean BMI: 22.4) completed Experiment 1 and 15 subjects (mean age: 24.0, mean BMI: 22.6) completed Experiment 2. One subject was dropped from the analysis of Experiment 2 data due to a breach of the 10-hour fast. Sixteen subjects (mean age: 22.6, mean BMI: 21.9) completed Experiment 3 and 18 subjects (mean age: 23.3, mean BMI 22.7) completed Experiment 4. One subject consumed all of the test meal after each preload in the fourth experiment. For this reason he was dropped from the food intake analysis as it cannot be assumed that he would not have consumed more food if it were presented in excess. Only one out of 60 subjects consumed the entire test meal, therefore the amount of food provided was in excess of what the majority of people could eat.

**Treatments**

The objective of the first experiment was to measure subjective appetite over 120 min post treatment to establish the most appropriate time for assessing the effect of treatments on food intake in the subsequent experiment. In this preliminary experiment subjects were provided in random order 0 g, 25 g, 50 g and 75 g of sucrose in 360 ml volumes of water. Sweetness was equalized for all beverages using additions of aspartame provided by the Nutrasweet company (Mississauga, ON, Canada). The 0 g, 25 g, 50 g and 75 g sucrose preloads were sweetened with 281 mg, 188 mg, 94 mg and 0 mg of aspartame respectively. The energy contributed by the aspartame additions was ≤ 4.18 kJ and therefore considered negligible. The Atwater energy value for sucrose is equivalent to 16.1 kJ (1 g = 16.1 kJ (4 kcal)), therefore the treatments provided 0 kJ, 418 kJ (100 kcal), 836 kJ (200 kcal) and 1254 kJ (300 kcal).

In Experiment 2, a water control, sweet control and three doses of sucrose (25 g, 50 g and 75 g) were served in a counterbalanced order. To establish the dose effect of sucrose on satiety, subjective appetite was measured every 15 min. At 60 min a pizza test meal was fed and food intake measured. The treatments, with the exception of the water control, were equalized in terms of sweetness with the addition of the non-caloric sweetener, sucralose. The 0 g, 25 g, 50 g and 75 g doses of sucrose were sweetened with 125 mg, 71 mg and 39 mg of sucralose respectively. McNeil Specialty Products Company (New Brunswick, NJ) provided the sucralose used in this study. To decrease excessive sweetness, 10 ml of lemon from concentrate was added to the chilled beverages the morning of the study. The treatment volume was reduced to 300 ml so that it could be delivered in one glass.

The choice of sweetener was changed from aspartame to sucralose to avoid the potential criticism that the sweetener was affecting the outcome. It has been suggested that aspartame (dipeptide of phenylalanine and aspartic acid) has the potential, when consumed in large amounts, to activate postigestive mechanisms (Black et al., 1993) and therefore influence appetite. However, sucralose has no effect on the carbohydrate metabolism, blood glucose, blood fructose, or insulin secretion (Mezitis et al., 1996). This sweetener passes through the body unmetabolized (Duffy & Anderson, 1998) and was therefore chosen as the sweetening agent for this and subsequent experiments. In Experiment 2, a water control was also included to determine the effect of sweetness alone on dependent measures.
In Experiment 3, the objective was to determine the effect of a pure lipid on subjective appetite and food intake to 60 minutes. Three doses of Microlipid (Mead Johnson Nutritionals, Ottawa, Ontario, Canada) a 50% safflower oil-water emulsion, and an energy free control were served in a counterbalanced order. The treatments consisted of 67 ml of water, 22 ml Microlipid mixed with 45 ml of water, 44 ml Microlipid mixed with 23 ml water and 67 ml Microlipid. The consumption of each treatment was followed immediately by 233 ml of water for a total volume intake of 300 ml. All treatments, including control, were flavored with 1/4 tsp. vanilla extract and sweetened with 10 mg of sucralose.

Microlipid is a product used clinically for oral and tube feedings and was chosen for this study because it is a more palatable vehicle for the provision of safflower oil than the oil in its natural form. The product was purchased from the Hospital for Sick Children Specialty Food Shop (Toronto, Ontario). Although, Microlipid does contain small traces of emulsifiers they are present in negligible amounts (1.3% of mixture). Therefore, the effects of Microlipid were assumed to reflect the actions of pure safflower oil. Microlipid provides 19 kJ/mL, thus the treatments contained 0 kJ, 418 kJ (100 kcal), 836 kJ (200 kcal) and 1254 kJ (300 kcal) respectively. It was refrigerated overnight and additions of water, sucralose and vanilla were added prior to serving the preload to the subjects.

In Experiment 4 the objective was to make a direct, within-experiment comparison of the short-term effects of sucrose and safflower oil on subjective measures of appetite up to 60 min and on food intake. Three treatments were served including 67 ml water, 75 g sucrose dissolved in 55 ml of water (total volume = 67 ml) and 67 ml Microlipid. Immediately after drinking the initial solution, the subjects consumed an additional 233 ml of water so that the total volume consumed was 300 ml. To equalize sweetness among treatments, 240 mg of sucralose was added to both the Microlipid and water. All preloads were flavored with 1/4 tsp. vanilla extract.

The sucrose and safflower oil preloads in the fourth experiment were equicaloric and equal volume. Energy density, using the convention of kJ/g, of the total sucrose and safflower oil preloads consumed were also approximately equivalent (± 1 kJ/g).

Subjects were required to fill out a Sleep Habits and Stress Factors Questionnaire, to provide information regarding any factors that could affect appetite (i.e. compliance with fast, unusual event, illness, sleep deprivation). The baseline Visual Analogue Scales (VAS), measuring Motivation to Eat (Motivation to Eat questionnaire) were filled out when subjects arrived at the Department of Nutritional Sciences. Upon completion of the questionnaires, the subjects proceeded across the hall into the taste panel room, where they were provided with the treatment. If more than one subject was scheduled on a particular day, they were seated in opposite cubicles facing away from one another.

In the Experiment 1 the subjects drank the treatments from a clear glass in which they poured the treatments from a pitcher. Similarly, in Experiment 2 the treatments were provided in a clear glass because the colour of the solutions was not distinguishable. In Experiments 3 and 4, subjects were presented with two styrofoam cups. The beverages with higher safflower oil content tended to adhere to the sides of glass but not styrofoam. Therefore the use of styrofoam cups in these experiments helped to limit observable differences between treatments. One cup contained the treatment (67 ml) and the other contained water (233 ml). Subjects were instructed to consume the treatment quickly and then drink all of the water. The beverages were all consumed in under 5 min. A timer was started upon the complete consumption of the entire preload.

Subjects returned to the original room after drinking the beverages and immediately filled out a questionnaire assessing the palatability, perceived sweetness and/or perceived fat content of each treatment. The Motivation to Eat VAS’s were completed at 15, 30, 45, 60, 90, 105, 120 min in Experiment 1. Based on the appetite findings from experiment 1, the VAS’s for subsequent experiments were completed every 15 min only up to 60 min. Each page of the questionnaire was not removed but was turned over once completed to prevent the subjects from basing their present feelings on their previous ratings. Subjects remained seated during the each experimental session and read or sat quietly when they were not completing the questionnaire.

Following the completion of the 60-min questionnaire, in Experiments 2 through 4, subjects returned to the taste panel room where they were served a pizza lunch and bottled spring water (1.5 L, Crystal Springs, Quebec). Four varieties of pizzas were available and subjects ranked the pizzas according to their preference prior to the sessions. Participants were served two pizzas of the variety they ranked first and one of each of the second and third choices per tray. The second served tray was identical to the tray presented first. Subjects were instructed to eat until they were “comfortably full”
and made aware that they would be presented with a second hot tray of pizzas in 5–10 min. Upon termination of the test meal, subjects rated the palatability of the pizza, and completed the post meal motivation to eat questionnaire.

Upon completion of each experimental session in Experiment 4 only, subjects were required to record their food and beverage intake for the remainder of the day. Each subject was asked to use household measures as the standard for recording the amount of food or drink consumed. They were instructed to include a detailed description of all food items including the ingredients of mixed dishes and brand names of whole foods. An example of the correct way to keep food records was reviewed and subjects were provided with written instructions and a sheet for record keeping.

The Motivation to Eat questionnaire tool, used to assess appetite, was composed of four individual VAS’s which measured: (1) desire to eat (“Very weak” to “Very strong”), (2) hunger (“Not hungry at all” to “As hungry as I’ve ever felt”), (3) fullness (“Not full at all” to “Very full”), (4) prospective consumption (“Nothing at all” to “A large amount”). Each VAS consisted of a 100 mm line anchored at either end with opposing statements. Subjects marked an “X” on the line to indicate their feelings at each measured time point. Scores were determined by measuring the distance in mm from the left most end of the line to the intersection of the “X”. The score for fullness was subtracted from 100 mm to account for the inverse nature of the question.

**Data analysis**

SAS version 7.1 (SAS Institute Inc., Carey, NC) was used to perform the statistical analyses. One-way repeated measure analyses of variance (ANOVA) were performed in order to test the effect of the treatments on the outcome variables: energy intake, palatability, perceived sweetness/fat content and physical comfort.

An average appetite score at each time point was calculated from the four individual questions according to the formula:

\[ \text{Average appetite} = \frac{\text{desire to eat} + \text{hunger} + (100 - \text{fullness}) + \text{prospective consumption}}{4} \]

Average appetite was reflective of the scores on the individual motivation to eat questions and therefore was used as a summary measure of subjective appetite for analyses.

A one way repeated measures ANOVA was completed on the average appetite scores in Experiment 1 to determine the effect of treatment at individual time points. The purpose of this analysis was to pinpoint the optimal time and dose of sucrose to observe significant effects. For all other experiments more in depth analysis was conducted. In Experiments 2 through 4 a two way repeated measures ANOVA was conducted on the change from baseline average appetite scores to detect main effects of time and treatment.

Items recorded in the food diaries by each subject in Experiment 3 were entered into the Nutripro GI dietary analysis program (University of Toronto, Canada; Wolever, 1997). This program, was used to calculate the total amount of energy provided by the foods recorded on each subject diet record. The sum of energy provided from both the test meal and foods consumed during the remainder of the day was then analyzed with a one way ANOVA.

Duncan’s post-hoc tests were performed where treatment effects were statistically significant. The General Linear Models (GLM) procedure was used to conduct ANOVA on all data sets. All values are presented as mean ± standard error of the mean (SEM). The p-value of less than 0.05 was considered to indicate statistical significance for all tests in the study.

**Results**

**Average appetite**

In Experiment 1 the 50 g and 75 g doses of sucrose significantly suppressed appetite more than the 25 g sucrose dose at 45 min, \( F(3,11) = 3.33, p < 0.05 \), and the 75 g dose suppressed appetite greater than both the 25 g and control treatment at 60 min, \( F(3,11) = 2.90, p = 0.05 \), (Table 1). There was no difference in appetite suppression at any of the measured time points through to 120 min. Therefore in subsequent experiments appetite was measured to 60 min.

In Experiment 2, a main effect of time on average appetite scores was detected (Table 2). Beginning at 15 minutes, appetite increased over time after all preloads, \( F(3,13) = 17.69, p < 0.0001 \). Treatment effects were observed 15, 30 and 60 min following preloads (Table 2, Figure 1). At 15 min, average appetite was lower after the 50 g and 75 g doses of sucrose relative to both the water and sweet controls, \( F(4,13) = 3.18, p < 0.05 \). Thirty minutes post preload a greater suppression \( F(4,13) = 2.93, p < 0.05 \) of appetite was observed after the 25 g and 50 g doses of sucrose than that observed after water and sweet controls. At 60 min, the 50 g and 75 g doses of sucrose prevented the increase in appetite from baseline values observed after the water and sweet controls, which were similar, \( F(4,13) = 2.62, p < 0.01 \).

In contrast to sucrose, safflower oil had no effect on average appetite scores in Experiment 3 (Table 3,
Figure 2). However, there was an effect of time, \( F(3,15) = 23.69, p < 0.0001 \). The scores for average appetite increased over time after a small decrease from baseline to 15 min.

When the largest dose of sucrose and safflower oil were compared directly in the fourth experiment, treatment and time effects, \( F(3,17) = 6.18, p < 0.01 \), were observed (Figure 3).Sucrose, but not safflower oil, caused a significant decline in average appetite scores compared to the flavored, sweetened water control at 15 min only, \( F(2,17) = 3.35, p < 0.05 \) (Table 4).

### Table 1. Experiment 1: average appetite scores

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Water 418 kJ sucrose</th>
<th>836 kJ sucrose</th>
<th>1254 kJ sucrose</th>
<th>( F, p^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>68.2 ± 3.8</td>
<td>74.5 ± 3.3</td>
<td>66.6 ± 5.5</td>
<td>67.9 ± 4.2</td>
</tr>
<tr>
<td>15</td>
<td>62.2 ± 4.3</td>
<td>59.4 ± 3.9</td>
<td>56.5 ± 2.4</td>
<td>56.9 ± 2.9</td>
</tr>
<tr>
<td>30</td>
<td>63.9 ± 4.9</td>
<td>66.0 ± 4.9</td>
<td>60.8 ± 3.3</td>
<td>56.8 ± 4.6</td>
</tr>
<tr>
<td>45</td>
<td>69.7 ± 3.5&lt;sup&gt;b&lt;/sup&gt;</td>
<td>72.1 ± 2.7&lt;sup&gt;a&lt;/sup&gt;</td>
<td>61.8 ± 4.6&lt;sup&gt;b&lt;/sup&gt;</td>
<td>64.0 ± 3.9&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>60</td>
<td>73.0 ± 4.0&lt;sup&gt;a&lt;/sup&gt;</td>
<td>73.3 ± 4.0&lt;sup&gt;a&lt;/sup&gt;</td>
<td>65.7 ± 3.9&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>63.7 ± 5.1&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>75</td>
<td>74.7 ± 3.8</td>
<td>75.5 ± 4.1</td>
<td>67.2 ± 4.1</td>
<td>68.8 ± 4.8</td>
</tr>
<tr>
<td>90</td>
<td>75.3 ± 4.2</td>
<td>76.7 ± 3.6</td>
<td>72.1 ± 4.6</td>
<td>73.2 ± 3.9</td>
</tr>
<tr>
<td>105</td>
<td>77.3 ± 3.2</td>
<td>81.5 ± 3.1</td>
<td>71.7 ± 4.5</td>
<td>75.4 ± 3.9</td>
</tr>
<tr>
<td>120</td>
<td>80.3 ± 3.2</td>
<td>82.0 ± 3.3</td>
<td>76.6 ± 4.5</td>
<td>76.0 ± 3.8</td>
</tr>
</tbody>
</table>

1 Mean ± SEM (mm); \( N = 14 \).

2 \( F \) and corresponding \( p \) values within each row.

* Means with different superscripts within a row are significantly different.

### Table 2. Experiment 2: change from baseline average appetite scores

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Water 418 kJ sucrose</th>
<th>836 kJ sucrose</th>
<th>1254 kJ sucrose</th>
<th>( F, p^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>2.9 ± 2.7&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.6 ± 2.5&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-4.8 ± 2.0&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>-7.4 ± 3.6&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>30</td>
<td>4.6 ± 3.0&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.9 ± 2.9&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-4.1 ± 2.8&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-4.9 ± 3.1&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>45</td>
<td>5.1 ± 3.7</td>
<td>6.9 ± 3.4</td>
<td>1.9 ± 1.6</td>
<td>0.1 ± 2.8</td>
</tr>
<tr>
<td>60</td>
<td>9.4 ± 3.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10.0 ± 3.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>5.4 ± 1.9&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.8 ± 2.6&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

1 Mean ± SEM (mm); \( N = 14 \).

2 \( F \) and corresponding \( p \) values within each row.

* Means with different superscripts within a row are significantly different.

One hour after the sucrose preloads tested in Experiment 2, there was an effect of treatment, \( F(4,13) = 8.63, p < 0.0001 \), on the amount of food consumed at the test meal (Table 5). There was no difference in the amount of food consumed after either the water or sweet control. Compared to the water preload the largest dose of sucrose, 75 g, resulted in lower food intake (1129 kJ) than that observed after all other treatments. Both the 50 g and 25 g doses decreased food intake by 518 kJ compared to the water preload.

A significant treatment effect, \( F(3,15) = 3.10, p < 0.05 \) on food intake was detected one hour following the safflower oil preloads administered in Experiment 3.
Table 3. Experiment 2: change from baseline average appetite scores

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Sweet control</th>
<th>418 kJ Safflower oil</th>
<th>836 kJ Safflower oil</th>
<th>1254 kJ Safflower oil</th>
<th>F; p²</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>−3.9 ± 2.1</td>
<td>−2.8 ± 2.7</td>
<td>−5.2 ± 3.3</td>
<td>−2.8 ± 2.5</td>
<td>0.32; 0.81</td>
</tr>
<tr>
<td>30</td>
<td>−0.5 ± 3.0</td>
<td>−3.6 ± 4.3</td>
<td>−2.1 ± 3.6</td>
<td>−0.9 ± 3.1</td>
<td>0.26; 0.86</td>
</tr>
<tr>
<td>45</td>
<td>5.2 ± 2.0</td>
<td>−0.2 ± 3.5</td>
<td>0.4 ± 2.7</td>
<td>2.5 ± 2.4</td>
<td>1.07; 0.37</td>
</tr>
<tr>
<td>60</td>
<td>6.8 ± 2.2</td>
<td>2.9 ± 3.0</td>
<td>3.6 ± 2.7</td>
<td>7.1 ± 2.1</td>
<td>1.03; 0.39</td>
</tr>
</tbody>
</table>

¹ Mean ± SEM (mm); N = 16.
² F and corresponding p values within each row.
³ Means with different superscripts within a row are significantly different; p < 0.05.

When the 1254 kJ doses of sucrose and safflower oil were compared in the final experiment a treatment effect on food intake was found, F(2,16) = 9.49, p < 0.001, (Table 7). The amount of food consumed after the sucrose preload was significantly less than that consumed after the control by 653 kJ and after the safflower oil by 443 kJ. Safflower oil did not significantly reduce energy intake at the test meal.

The total amount of energy consumed from the test meal and remainder of the day, as captured in the food diaries, was significantly lower after sucrose (10304 ± 874 kJ) than the control (12256 ± 677 kJ) F(2,14) = 3.78, p = 0.04. Total caloric intake following the safflower oil (11390 ± 740) was not different from control.

Discussion

These studies show that food intake regulatory centers in young men detect and compensate for as little as 25 g (418 kJ) of sucrose for at least 60 min after sucrose consumption. In contrast, the sensitivity to fat in the form of safflower oil is much less, with a statistically significant suppression in food intake occurring after the consumption of 1254 kJ in Experiment 2 but not in Experiment 4. Furthermore, compensation for the sucrose energy during the test meal was carried through the rest of the day, but there was no compensation for the energy from safflower oil either in the test meal or later in the day in Experiment 4.

The only other study that found a reduction in food intake after such a small dose of sucrose was conducted with children. Ninety kilojoules consumed in water was sufficient to suppress food intake by an equivalent amount at a test meal both 30 min and 90 min after the preload (Birch et al., 1989). The precise compensation was attributed to the ability of young children to rely solely on internal hunger cues. However, because of the much larger body size of the men in this study, the sensitivity shown to the 25 g dose is even more remarkable and questions whether physiological mechanisms compensating for energy intake are less sensitive in adults than in children.
The effect of sucrose on food intake one hour after administration (Table 5) is dose related and is consistent with other studies reported in the literature. High (≥50 g) carbohydrate drinks of both sucrose and glucose have been shown to reduce food intake one to one and a half hours post preload (Rogers et al., 1988; Green & Blundell, 1996; Rogers & Blundell, 1989).

Based on a literature review, it was predicted that the minimum dose of sucrose necessary to impact food intake 20–60 min later would be approximately 836 kJ (50 g) when a sample size of less than 20 subjects is used (Anderson, 1995). However, the smallest, 418 kJ (25 g) dose in this experiment decreased food intake relative to water. This may be the threshold dose for detection in short term studies because previous studies have not found doses of sucrose of 20 g or less to suppress test meal intake one hour later (Canty & Chan, 1991; Rolls et al., 1990).

These data refute the suggestion that sugar-sweetened drinks could lead to obesity by bypassing regulatory systems (Ludwig et al., 2001). The adjustment for the energy derived from sucrose preloads was remarkably precise at the 60-min pizza test meal. Average compensation for the energy in the three preloads in experiment two when compared with the water control averaged 92%, and when compared with the sweet control averaged 70%. A similar compensation of 65% for the calories in sucrose preloads compared with an aspartame sweetened control was found in children eating a free choice lunch 30 minutes later (Black & Anderson, 1994). Additional evidence that sucrose energy intake is compensated for is provided by the evidence that the 1254 kJ sucrose preload in Experiment 4 resulted in an average intake for the remainder of the day which was 2407 kJ (567 kcal) less than that observed following the control treatment.

An explanation for the sensitivity of regulatory systems to sucrose intake may reside in part in its effect on blood glucose (Mayer, 1953). Sucrose has a glycemic index (GI) of ~80 (Foster-Powell & Miller, 1995; Jenkins & Wolever, 1981). This GI is intermediate between glucose and fructose.

### Table 4. Experiment 3: change from baseline average appetite scores

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Safflower oil 1254 kJ</th>
<th>Sucrose 1254 kJ</th>
<th>Sweet control 0 kJ</th>
<th>F; p²</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>-5.9 ± 1.6abc</td>
<td>-10.3 ± 3.3b</td>
<td>-1.6 ± 3.4a</td>
<td>3.35; 0.049</td>
</tr>
<tr>
<td>30</td>
<td>-4.5 ± 2.4</td>
<td>-3.1 ± 3.5</td>
<td>2.5 ± 3.6</td>
<td>2.19; 0.13</td>
</tr>
<tr>
<td>45</td>
<td>-4.1 ± 2.4</td>
<td>-2.9 ± 3.5</td>
<td>5.5 ± 3.5</td>
<td>2.78; 0.08</td>
</tr>
<tr>
<td>60</td>
<td>-2.7 ± 2.4</td>
<td>-3.3 ± 4.3</td>
<td>7.0 ± 4.4</td>
<td>2.49; 0.10</td>
</tr>
</tbody>
</table>

1 Mean ± SEM (mm); N = 18.
2 F and corresponding p values within each row.
3 Means with different superscripts within a row are significantly different; p < 0.05.

### Table 5. Experiment 2: food intake after sucrose

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Energy intake (kJ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>4606 ± 376a</td>
</tr>
<tr>
<td>Sweet control</td>
<td>4456 ± 380ab</td>
</tr>
<tr>
<td>418 kJ sucrose</td>
<td>4088 ± 347b</td>
</tr>
<tr>
<td>836 kJ sucrose</td>
<td>4088 ± 255b</td>
</tr>
<tr>
<td>1254 kJ sucrose</td>
<td>3477 ± 322c</td>
</tr>
<tr>
<td>F</td>
<td>8.63</td>
</tr>
<tr>
<td>p</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

1 Mean ± SEM (kJ); N = 14.
2 Energy consumed at the test meal after the treatment relative to control.
3 Means with different superscripts are significantly different.

### Table 6. Experiment 3: food intake after safflower oil

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Energy intake (kJ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>4356 ± 360a</td>
</tr>
<tr>
<td>418 kJ safflower oil</td>
<td>4180 ± 305b</td>
</tr>
<tr>
<td>836 kJ safflower oil</td>
<td>3996 ± 372b</td>
</tr>
<tr>
<td>1254 kJ safflower oil</td>
<td>3876 ± 284b</td>
</tr>
<tr>
<td>F</td>
<td>3.10</td>
</tr>
<tr>
<td>p</td>
<td>0.04</td>
</tr>
</tbody>
</table>

1 Mean ± SEM (kJ); N = 16.
2 Energy consumed at the test meal after the treatment relative to control.
3 Means with different superscripts are significantly different.

### Table 7. Experiment 4: food intake after sucrose and safflower oil preloads

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Energy intake (kJ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sweet control (water)</td>
<td>3908 ± 206a</td>
</tr>
<tr>
<td>1254 kJ safflower oil</td>
<td>3696 ± 259a</td>
</tr>
<tr>
<td>1254 kJ sucrose</td>
<td>3255 ± 219b</td>
</tr>
<tr>
<td>F</td>
<td>9.49</td>
</tr>
<tr>
<td>p</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

1 Mean ± SEM (kJ); N = 17.
2 Energy consumed at the test meal after the treatment relative to control.
3 Means with different superscripts are significantly different.
between high glycemic foods (i.e. cornflakes, GI 122) and low glycemic foods (i.e. spaghetti, GI 59) (Foster-Powell & Miller, 1995).

It has been proposed that high-glycemic index foods promote excessive energy intake and that low glycemic foods suppress appetite, therefore preventing obesity (Roberts, 2000). If low glycemic foods do in fact produce greater suppression of appetite, it would be expected that sucrose would be more effective than many carbohydrate foods (including some breakfast cereals and breads) in producing satiety and less likely to produce obesity. On the other hand it would be predicted to be less satiating than other lower glycemic foods such as spaghetti or beans. To test this prediction will require comparison of the effects of sucrose with other pure carbohydrates such as a high GI carbohydrate (i.e. glucose, GI 138 [Foster-Powell & Miller, 1995; Jenkins & Wolever, 1981]) and low GI carbohydrates (i.e. amylose and amylopectin, GI ~59 and ~88 respectively [Foster-Powell & Miller, 1995]) on food intake in a within-subject design.

The hypothesis that foods with a lower glycemic index produce a greater suppression of appetite is based in part on the proposal that a small, sustained rise in blood glucose is more likely to promote satiety than a sharp initial peak in blood glucose. It is also assumed that the postprandial dip in blood glucose which occurs after a sharp rise in blood glucose stimulates hunger, and indeed, a dip in blood glucose has been shown to predict feeding in some animals (Campfield & Smith, 1990). In young men given beverages composed primarily of glucose syrup or cream, a positive correlation was found between the duration of the blood glucose response and meal interval (Melanson et al., 1999). Thus, the authors concluded that a rapid increase and then decline in blood glucose corresponded to lessened satiety, as indicated by shortened intermeal intervals after the carbohydrate treatment. However, total intake at the meals that followed was not different, leaving uncertain the relationship between glycemic response and subsequent food intake.

In contrast to the effects of sucrose, only the largest test dose (1254 kJ) of safflower oil was required to significantly suppress intake and only then in Experiment 3 (Table 5) but not 4 (Table 6). These results suggest that there is a higher caloric threshold for fat intake, at least in the form of safflower oil, which must be consumed before an effect on food intake is observed. There are no other reports of the relationship between the quantity of pure fat ingested and subsequent food intake. However, the literature generally supports the idea that fat is less satiating. Young males did not decrease food intake 70 min after consuming 240 kcal (1003 kJ) of corn oil (18) but a large dose of 500 kcal (2090 kJ) of Intralipid infused into the stomach of young males suppressed food intake 30 min later (Chapman, 1999). Furthermore mixed meal studies show that calorie for calorie, carbohydrate is more satiating than fat (Blundell et al., 1993; Rolls et al., 1994; Stubbs et al., 1996).

Several aspects of the design of these studies require comment. The palatability of the preloads, subsequent levels of comfort and the perceived characteristics of the solutions can not account for the observed treatment effects. There was no change in subjective ratings of comfort from baseline to 60 min, as assessed with a VAS question pertaining to wellness, after each of the preloads in Experiments 2 \([F = 0.37]\), 3 \([F = 2.09]\) and 4 \([F = 1.72]\). There were also no significant differences in subjective ratings of palatability between treatments in Experiments 2, 3 and 4 \([F = 1.26, F = 1.09, F = 0.17,\) respectively]. In addition, the three doses of sucrose and safflower oil received equivalent ratings of sweetness \([F = 1.03]/\)fat content \([F = 2.19]\). Similarly, in Experiment 4, the 1254 kJ preloads of safflower oil and sucrose were also rated to be equivalent with respect to both fat content \([F = 2.30]\) and sweetness \([F = 1.06]\).

For the sucrose solutions, it was easy to equalize the sweetness, but the level of sweetness used in Experiment 1 was reported to be somewhat unpleasant. As a result, in the second experiment lemon flavor was added to the drinks used in Experiment 2. It is uncertain whether this change or the change in sweetener from aspartame to sucralose accounted for the failure of the sweetened control to decrease average appetite in Experiment 2, as it did in Experiment 1. It seems more likely that the reduced sweetness was a factor because in previous studies aspartame added to similar volumes of water did not affect subjective ratings of appetite at 15 min (Black et al., 1991).

Within each experiment the volume of the drinks was constant but energy content was varied. This suggests that energy content and the macronutrients that provide it, not volume of the drinks, are the primary determinants of the physiological signals for satiety. In addition the energy density of preloads was constant in Experiment 4, therefore observed differences in food intake can not be attributed to energy density. The importance of the energy density of preloads on subsequent intake has not been specifically examined. It has been proposed that the energy density of a food or meal may be an important determinant of total daily energy intake (Stubbs et al., 1996; Poppitt & Prentice, 1996; Westerterp-Plantenga, 2001) based on the observation that individuals tend to consume a constant weight of food within a meal. This suggests the role of energy density is important to consider when examining ad libitum intake at a meal or throughout the day and less important when using the preload and fixed meal paradigm.
VAS’s for subjective appetite were used to track the effect of treatment with time on appetite. Safflower oil preloads did not produce a detectable suppression of appetite at any time point. In contrast, the effect of the sucrose treatments on average appetite with time was dose dependent, with the effect of the 50 and 75 g dose, but not the 25 g dose lasting to 60 minutes. This is consistent with the observation that \( \sim 70 \) g or 1/kg of sucrose is able to sustain its appetite suppressing properties long enough to cause a reduction in food intake relative to water 120 min post preload (Biernacka, 1995). There does seem to be a caloric threshold necessary to impact subjective satiety as studies using smaller doses of sucrose (20 g or less) failed to find an effect on subjective satiety (Rolls et al., 1988, 1990; Canty & Chan 1991) over time.

It should also be noted that the data derived from subjective appetite and food intake are dependent on the duration of the measurements made after the treatment. Based on Experiment 1, the time of measurement of 1 h was selected in order to test for the effect of small amounts of sucrose on food intake. Intervals between preload and food intake measured up to 1 h have previously been deemed adequate to detect compensation for a carbohydrate preload (Blundell et al., 1994; Anderson, 1995). It is also possible that the duration of sucrose’s effect on food intake is longer, at least for the larger doses. Sucrose (\( \sim 70 \) g or 1/kg) has been found to sustain its appetite suppressing properties causing a reduction in food intake relative to water 120 min post preload (Biernacka, 1995).

It is possible that the weak effect of safflower oil on appetite was a result of measurements made only to 1 h. A time course for the effect of long chain fat on satiety and food intake has not been described, but studies suggest that the effect of fat on satiety appears to be delayed compared to the action of carbohydrate. For example, subjects provided with two carbohydrate pasta lunches, containing either \( 50 \) g of a low-energy butter substitute (50 g of cooked starch) or \( 50 \) g of butter, requested dinner 38 min later following the fat compared with the starch supplemented meal despite no differences in appetite ratings (Himaya, 1997). Because the high fat meal was also 1580 kJ higher in energy it is difficult to attribute the response to the butterfat alone. In a more direct comparison, with time blinded subjects, 1000 kJ loads of a glucose syrup resulted in an intermeal interval of 80 min whereas equivalent energy loads from cream resulted in an intermeal interval of 120 min (Melanson et al., 1999). Thus the expression of the effect of fat vs. carbohydrate preload on food intake may depend on the time at which the test meal is fed.

Nevertheless, in the present study, food records maintained for the rest of the day (approximately 12 h) did not show any latent effect of the fat preload. Other studies have also failed to find compensation for a fat preload when food records for the entire day were examined (Cotton et al., 1994, 1996). Of course dietary records are fraught with inaccuracies. There may be problems with the subject’s estimation of portion sizes, detail, distortion of normal eating patterns (Hill & Prentice, 1995) and investigator’s interpretation of records. Even with subjects in a controlled environment over 8 h, compensation was not found for the fat energy consumed in preloads, despite a delayed intermeal interval (Melanson et al., 1999).

A more quantitative examination of the long-term effect of pure carbohydrates compared with fat would require the subjects to remain in the laboratory through out the day. This was done to test effects of a mixed diet and it was found that a diet with relatively more protein and carbohydrate was more satiating and resulted in a lower food intake over a 16 h period than a diet higher in fat (Westerp-Plantenga et al., 1999). Because their study did not dissociate the effects of protein from carbohydrate, we cannot conclude which component or macronutrients determined the outcome. A more precise evaluation of delayed compensation evoked by the macronutrients would require measurement of subjective satiety following pure preloads of fat and carbohydrate in a similarly controlled environment and over an appropriate duration.

Based on power analysis, a much larger sample would be required to capture an effect of safflower oil on satiety. Power analysis for a within subject design based on a previous study, examining sucrose and food intake (Biernacka, 1995), indicated a sample size of 8 would be adequate to detect treatment effects of sucrose preloads of 1254 kJ on satiety. Using the data collected in this experiment, power analysis shows that a minimum sample size of 38 would be required to detect an intake change of 20%, if it occurred following the 836 kJ preload of safflower oil. Even if statistical significance could be achieved with a large sample size, it remains evident that the effect of safflower oil on satiety and food intake is much less than for sucrose.

Although physiologic measurements were not made, it can be expected that sucrose produces greater satiety than safflower oil based on several control mechanisms contributing to the regulation of food intake. Sucrose is more rapidly absorbed, oxidized (Rolls & Shide, 1992; Stubbs, 1999) and causes a greater blood glucose and osmotic (Blundell & Stubbs, 1999) response. In addition, carbohydrate ingestion results in higher diet-induced thermogenesis (DIT) relative to fat (Doucet & Tremblay, 1997) and an inverse relationship between DIT and satiety has been established (Westerterp-Plantenga et al., 1999).
It must be noted, however, that fat-induced satiety is expected because at least in humans, fat ingestion releases the hormone cholecystokinin, which produces short-term satiety (Rolls & Shide, 1992). Moreover, the formation of chylomicrons results in formation of Apo IV, another possible satiety factor (Tso et al., 1999). It is possible that fat sources other than safflower oil more strongly stimulate these physiological mechanisms, producing greater satiety.

The results of this study do not directly provide an answer to the proposed role of sweeteners as determinants of overeating and obesity (Rogers et al., 1998). However, it is clear that, under laboratory conditions, sucrose energy suppresses subsequent food intake. Furthermore, there was no difference in food intake between the control preparation, prepared with sucralose, and water treatments suggesting that sweetness alone, in the absence of energy, neither stimulated nor suppressed food intake. This result is consistent with a previous report in which aspartame was the sweetener (Black et al., 1991).

It is concluded that, at least in the short term, sucrose consumption brings about a dose dependent reduction in appetite and food intake in young men that is greater than that produced by safflower oil.

References


