Effect of Glycemic Carbohydrates on Short-term Satiety and Food Intake
G. Harvey Anderson, Ph.D., and Dianne Woodend, M.Sc.

We examine the relationships between glycemic carbohydrate and its effects on short-term satiety and food intake. Both high- and low-glycemic carbohydrates have an impact on satiety, but their effects have different time courses. High-glycemic carbohydrates are associated with a reduction in appetite and food intake in the short term (e.g., one hour), whereas the satiating effects of lower-glycemic carbohydrates appear to be delayed (e.g., 2 to 3 hours). There is no consistent evidence that an increase in blood glucose, either acute or sustained, is the primary determinant of their effects on food intake and satiety. Many other preabsorptive and postabsorptive signals for satiety exist and may be the determining factors. Further studies are needed to delineate the role of glycemic carbohydrates and their mechanisms of action in determining satiety.

Key words: sugars, glycemic carbohydrate, food intake
© 2003 International Life Sciences Institute

Introduction
Carbohydrates are the main source of energy in most diets. In addition to providing energy, carbohydrate ingestion affects many aspects of brain function, including the regulation of food intake. The concept that glucose derived from carbohydrate is central to the regulation of satiety and food intake is based on the glucostatic theory of food intake regulation. This theory proposes that blood glucose concentration is closely monitored and that food is ingested when the use of glucose by various organs is insufficient. Conversely, satiety and the termination of eating occurs after an increase in blood glucose concentration.

We explore the relationship between the consumption of glycemic carbohydrates and short-term satiety and food intake, as well as the hypothesis that the glycemic response to carbohydrates predicts their effects on satiety.

Carbohydrates, Appetite, and Food Intake
It is clear that carbohydrate ingestion promotes satiety. In recent years, however, the prevalence of obesity has increased despite decreasing fat intake and a subsequent rise in carbohydrate consumption. It has therefore been suggested that high-glycemic, rapidly digested carbohydrates are the cause of overeating and obesity because they fail to stimulate satiety mechanisms and exacerbate hunger.

The effect of carbohydrates on satiety is usually classified as intermediate between protein and fat. A hierarchy has been established whereby protein is considered to be more satiating than carbohydrate and carbohydrate more satiating than fat. Because this information is derived from studies in which humans were provided mixed diets that were high in one macronutrient compared with diets high in another macronutrient, the results are often equivocal. In addition to the response to a macronutrient being modified by the presence of other nutrients, and by the energy content of the preload, the source of the macronutrient selected within each macronutrient class is a factor.

We examine the importance of quantity, source, and composition of a macronutrient as illustrated by the effects of the different classes of carbohydrates, including monosaccharides, disaccharides, and polysaccharides, on satiety and food intake.

Sugars, Appetite, and Food Intake
Sugars receiving considerable investigation regarding effects on appetite and food intake include sucrose, glucose, and fructose.

Sucrose
Sugars, especially those used as caloric sweeteners, and when consumed in drinks, may contribute to excess
energy intake because they bypass regulatory systems. This explanation was offered after observing the association between soft drink consumption and the development of obesity in children. 12 Supporting this hypothesis are experimental data showing that when provided to human subjects in the form of a sweet beverage, small sucrose preloads of 83 kcal (20 g) and 166 kcal (40 g) did not result in compensation at subsequent test meals presented 30 or 60 minutes later. 13 However, the composition of the preloads in these studies may have been less important than other aspects of the study design. The majority of the literature suggests that caloric preloads of less than 200 kcal (in beverage form) do not consistently decrease food intake, perhaps because the quantity is below the threshold necessary for detection by hunger mechanisms. 14 An evaluation of the sensitivity of food intake regulatory mechanisms to sucrose or other sugars requires studies of both the quantity of the preload and the interval between treatment and measurement of the dependent variables. Furthermore, to fully evaluate the contribution of sugars to satiety, comparison should be made with other carbohydrates.

Even with similarly designed protocols, the outcome can vary between studies. In one study, 15 when the time interval between preload and test meal consumption was 60 minutes, the effect of sucrose was found to be dose dependent. When young men were given drinks (300 mL) containing 25, 50, or 75 g of sucrose, even the lowest dose of 418 kJ (100 kcal) increased subjective satiety, as assessed by visual analogue scales (Figure 1), and suppressed food intake from a pizza meal one hour later (Table 1). There was 87% compensation (compared with the sweet control) at the one-hour test meal for the 418 kJ (100 kcal) provided in the 25-g sucrose beverage. Compensation for the 50- and 75-g doses was 44% and 79%, respectively. By contrast, a similarly designed study found only 44% compensation for 75 g of sucrose, but the reduction of food intake was statistically significant (Table 2). 16

Young children also compensate for sugar consumed as a beverage. In 2- to 5-year-old children (n = 24), 90 calories derived from a sucrose drink were sufficient to suppress intake at test meals 30 or 90 minutes after the preload. 17 The compensation in the test meals for the calories in the preloads was close to 100%, which the authors attributed to the ability of young children to rely solely on internal hunger cues. Although less precise, older children also compensate for energy derived from sucrose. When 9- to 10-year-old children consumed a cherry-flavored drink containing either 45 or 90 g of sucrose, lunchtime food intake was reduced 30 minutes later. 18 Compensation for the 45-g and 90-g sucrose beverages was 68% and 63%, respectively.

Several other investigations have shown that ingestion of 50 g of sucrose or greater reduces intake at mealtime 20 to 60 minutes later. 14 Under laboratory conditions, therefore, the majority of studies show that the ingestion of sucrose energy suppresses food intake, refuting the suggestion that sucrose bypasses appetite regulatory systems. 7,12,19 Because sucrose is composed of glucose and fructose, one or both of these monosaccharides may explain the effect of sucrose on regulatory mechanisms. In general, when given as a beverage, the consumption of glucose alone decreases food intake, but the reduction is less than that produced by fructose.

**Glucose and Fructose**

In young men, either 75 g 16 or 50 g 20 of glucose in drinks, or 50 g in yogurt, 21 reduced food intake one hour later. Consumption of 50 g fructose in a drink suppresses energy intake to an even greater extent at test meals from 38 minutes 22 to 2.25 hours later. 23–25 This advantage of fructose over glucose disappears, however, if it is consumed with another carbohydrate. For example, no difference was observed between isocaloric cereal preloads containing additions of fructose (30 g) or glucose (33.5 g) on mealtime energy intake at 30 minutes or 120 minutes after consumption (Table 3). 26 Similarly, no differences in food intake were observed between 50 g fructose and 50 g glucose at 2.25 hours when given in a mixed-nutrient meal containing starch. 25 The presence of starch likely altered the effect of fructose on satiety, masking differences between the two sugars; investigators have demonstrated the addition of as little as 15 g of

---

**Figure 1.** Change in average appetite scores, measured by visual analogue scales, over one hour following treatment with a water control, sweet control (sucralose + water), and sucrose preloads (25, 50, and 75 g). The treatments, with the exception of the water control, were equalized for sweetness with additions of the noncaloric sweetener sucralose, and all treatments were provided as isovolumetric (300 mL) beverages. 15 Within-subject design, n = 14 young males, *F = 3.18, **F = 2.93, ***F = 3.71 for treatment. At 15, 30, and 60 minutes the differences among the means as indicated by the different superscripts (P < 0.05) were: water, a sweet control, 25 g sucrose, b 50 g sucrose, b and 75 g sucrose. b Reproduced with permission from Elsevier Science.
starch can prevent a decrease in food intake 2.25 hours after a 50-g fructose preload.25

Factors that might account for the effects of fructose on satiety, when given alone, include its absorption characteristics and gastrointestinal effects. Fructose is absorbed slowly,27 which allows prolonged contact time with gastrointestinal receptors that produce satiety signals.28,29 Fructose is also incompletely absorbed and, as a result, produces a hyperosmolar environment in the large intestine.30 A high concentration of solute within the gut lumen draws fluid into the intestine. This fluid shift can produce feelings of malaise or diarrhea,30 which can decrease the propensity of an individual to consume food. These factors are eliminated when fructose is consumed with even a small amount of glucose or starch because these carbohydrates facilitate more rapid and complete transport of fructose out of the intestine.27

There is presently no evidence that the effect of sucrose, or of high-fructose corn syrups of similar composition, can be attributed solely to one or the other of their monosaccharide components. It is clear, however, that sucrose and its component sugars suppress food intake even when consumed in small quantities if the duration between consumption and eating is brief. The duration of the effect has not been defined.

### Starch and Food Intake Regulation

Glycemic carbohydrates other than sugars also suppress food intake. Again their impact depends on quantity and composition.

The composition of starches is a determinant of satiety because it influences the metabolic response following ingestion. Starch is a polysaccharide comprising primarily amylose and amylopectin. Amylose is the minor component of starch; it is primarily a linear glucose polymer in which the individual monomers are connected solely by alpha (1–4) glycosidic linkages. Amylopectins are the major components of starch (~70%), containing alpha (1–4) and alpha (1–6) linkages to form

### Table 1. Food Intake After Sucrose

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Energy Intake (kJ)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>4606 ± 376†</td>
</tr>
<tr>
<td>Sweet Control</td>
<td>4456 ± 380†‡</td>
</tr>
<tr>
<td>418 kJ Sucrose</td>
<td>4088 ± 347§</td>
</tr>
<tr>
<td>836 kJ Sucrose</td>
<td>4088 ± 255§</td>
</tr>
<tr>
<td>1254 kJ Sucrose</td>
<td>3477 ± 322§</td>
</tr>
</tbody>
</table>

*Energy intake is given as mean ± SEM (kJ), n = 14 young men. Energy consumed at a pizza test meal served 60 minutes following ingestion of the preloads, which were provided as isovolumetric (300 mL) beverages, equalized for sweetness with additions of the noncaloric sweetener, sucralose.

†‡§Means with different superscripts are significantly different at P < 0.05 by one-way ANOVA followed by post-hoc Duncan’s. Data taken from reference 15.

### Table 2. Food Intake after 75 g Carbohydrate Treatments*

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Energy Intake (kJ)†</th>
<th>% Compensation‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sucralose</td>
<td>4251 ± 293§</td>
<td></td>
</tr>
<tr>
<td>Amylose</td>
<td>3954 ± 237§‡</td>
<td>23.7 ± 15.6§‡</td>
</tr>
<tr>
<td>Amylopectin</td>
<td>4255 ± 386§</td>
<td>−0.2 ± 21.7§</td>
</tr>
<tr>
<td>Polycose</td>
<td>3440 ± 364§</td>
<td>64.9 ± 19.4§</td>
</tr>
<tr>
<td>Sucrose</td>
<td>3695 ± 351§</td>
<td>44.4 ± 13.1§</td>
</tr>
</tbody>
</table>

* Food intake expressed as mean ± SEM (kJ), n = 14 young males, reproduced from reference 16.
† Energy consumed at a pizza test meal served 60 minutes following ingestion of the preloads, which were provided as isovolumetric (200 mL) beverages, equalized for sweetness with additions of the noncaloric sweetener, sucralose. Immediately following ingestion of each treatment, subjects consumed an additional 200 mL of spring water for a total volume intake of 400 mL.
‡ Compensation = (kJ intake after control – kJ intake after treatment/kJ in preload) × 100.
§ Means with different superscripts, within a column, are significantly different at P < 0.05 by one-way ANOVA followed by post-hoc Duncan’s.

### Table 3. Food Intake 30 Minutes after Carbohydrate Preloads (1379 kJ)*

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Energy Intake (kJ)†</th>
<th>Percent Compensation‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>SW + fructose</td>
<td>3578 ± 355§</td>
<td>85.3 ± 21.2</td>
</tr>
<tr>
<td>SW + glucose</td>
<td>3486 ± 293§</td>
<td>91.9 ± 15.9</td>
</tr>
<tr>
<td>Water</td>
<td>4757 ± 385§</td>
<td></td>
</tr>
</tbody>
</table>

* Mean ± SEM (kJ), n = 13 young males, from reference 15.
† Energy consumed at a pizza test meal served 60 minutes following ingestion of the preloads. Three preloads were served as follows: water (no breakfast control), Mini Shredded Wheat cereal (SW) + 30 g fructose (15.9 kJ/g), and SW + 33.5 g glucose (14.2 kJ/g). The two breakfast cereal treatments were of similar palatability and equal macronutrient composition. The fructose and glucose were dissolved in 100 mL 1% milk, and then added to 51 g of the cereal prior to serving. Also served was 150 mL of water for drinking.
‡ Compensation = (kJ intake after control – kJ intake after treatment/kJ in preload) × 100.
§ Means with different superscripts, within a column, are significantly different at P < 0.05 by one-way ANOVA followed by post-hoc Duncan’s.
a branched structure. The branched form of amylpectin allows greater gelatinization and a greater surface area for digestive enzymes, and is subsequently more rapidly absorbed. The ratio of amylose to amylpectin in starches influences the rate of starch digestion and the glucose and insulin response, which in turn may determine the capacity of starch to affect satiety and food intake.

High amylose-containing meals induce more prolonged satiety over six hours than high amylpectin-containing meals, suggesting that it is preferable to consume slowly absorbed carbohydrates if the goal is to control food intake. However, one cannot conclude that these results hold true in the short term, or indeed that polysaccharides are more effective than monosaccharides or disaccharides.

When comparisons are made, sugars are more effective than starches in reducing short-term appetite and food intake. Neither 75 g preloads of high-amylose cornstarch nor 75 g of high-amylpectin cornstarch in the form of sweetened drinks, affected food intake one hour later. Suppression of appetite, as assessed by visual analogue scales, was detected only at 30 minutes following the amylpectin preload (Figure 2). By contrast, polycose and sucrose, both of which are rapidly digested, decreased food intake compared with the control (Table 2).

In addition to the “caloric bypass” notion, which assumes sugar ingestion does not lead to suppression of food intake, some have suggested that consumption of high-glycemic carbohydrates stimulates food intake. This hypothesis acknowledges that there is a rapid increase in blood glucose concentration after the consumption of rapidly digested starch or sugars. It also assumes that within a short time, there is an insulin-induced decline in glucose concentration, such that a glucose concentration below baseline occurs, which in turn stimulates hunger and leads to excess energy intake.

### Carbohydrates and Blood Glucose

To explain the mechanisms by which carbohydrates may regulate food intake, Mayer proposed the glucostatic theory in 1953, which has been the basis of many studies. The glucostatic theory proposes that low blood glucose concentrations trigger the onset of feeding and that high blood glucose levels signal satiety and the termination of feeding. It now appears that decreased glucose utilization, or decreased intracellular glucose concentrations, rather than absolute concentration of blood glucose, is the stimulator for meal initiation.

Transient declines in blood glucose of the correct magnitude and time course are believed to induce meal initiation because they are detected by peripheral and central glucoreceptive elements and mapped into feeding behavior. Indeed, transient drops in blood glucose are associated with initiation of feeding in both animals and humans. The cause of a transient drop in blood glucose is unknown. There is no evidence, however, that transient decreases in blood glucose arise from the same physiologic events that cause glucose to fall below baseline after consumption of a large quantity of rapidly absorbed carbohydrate, or that the latter event initiates eating.

Dynamic postprandial declines in blood glucose concentration, defined as a decline in blood glucose from a peak induced by macronutrient ingestion, are also associated with spontaneous meal initiation. Thus a carbohydrate drink compared with a high-fat drink led to earlier spontaneous meal initiation, although the total amount of food consumed did not differ. These data support the notion changes in blood glucose are involved in the physiologic regulation of meal pattern, but do not prove causality.

Consistent with the glucostatic hypothesis, carbohydrate consumption and increased blood glucose concentration are associated with satiation. One might logically assume, therefore, that the effect of carbohydrates on satiety is predicted by the effects of carbohydrates on blood glucose.

### Glycemic Response and the Glycemic Index

The effect of carbohydrate ingestion on blood glucose is best described by measuring the total glycemic response over time. To provide a basis for comparing glycemic responses with foods, the glycemic index (GI) was developed. This index compares the incremental area
under the blood glucose response curve of a 50 g carbohydrate portion of a test food relative to 50 g of a standard food such as white bread, when ingested by the same subject. Because the GI standardizes the glycemic response to a test food, it corrects for between-subject variation, thereby allowing glycemic responses from different studies to be compared.

A range of glycemic responses is observed following ingestion of sugars. Glucose produces a more rapid and higher increase in postprandial blood glucose and insulin compared with fructose. Sucrose tends to elicit a postprandial blood glucose concentration that is intermediate between glucose and fructose. The glycemic indexes of glucose, fructose, and sucrose are 149, 32, and 87, respectively. Contrary to the belief that sugar produces higher blood glucose concentrations than an equivalent amount of starch, sucrose has a lower GI value, by up to 50%, compared with most common starchy foods. Replacing a portion of the starch with sucrose in a high-GI breakfast cereal lowers the glycemic and insulin responses.

Starches also display variable glycemic effects, determined in part by their amylopectin-to-amylose ratio. The open-branched structure of amylopectin starch makes it easier to digest than the linear amylose starch and, as a result, meals made with high-amylose starch induce a lower postprandial plasma glucose response than meals made from high-amyllopectin starches. For example, the average GI of high- and low-amylose rice is 59 and 88, respectively. Indeed, high-amylose compared with high-amyllopectin starch meals produce postprandial glucose and insulin responses that are lower initially to 2 hours but are sustained at higher concentrations to 6 hours. These metabolic responses have been correlated with greater satiety in these studies, but not in others.

**Glycemic Response, Satiety, and Obesity**

The relationship between glycemic responses to carbohydrates and satiety remains unclear for several reasons. First, only a few studies have made concurrent measurements of blood glucose, appetite, and food intake over extended periods of time. Second, the literature contains contradictory information on the relationship. Thirdly, the GI of a food is often assumed, incorrectly, to describe the mechanism by which satiety occurs.

In the few studies that have measured blood glucose and satiety simultaneously over extended periods of time, small and sustained changes in blood glucose were associated with satiety. Consumption of a high-amylose (low-glycemic) starch mixed meal (average of 864 kcal) produced a stronger decrease in hunger and increased feelings of fullness for up to 6 hours after consumption compared with a low-amylose (higher-glycemic) meal. Similarly, ingestion of high-amylose puffed rice (986 kJ or 235 kcal) resulted in greater satiety for up to two hours and decreased energy intake at meal 2 hours later compared with ingestion of low-amylose puffed rice (957 kJ or 229 kcal). Because blood glucose was lowest after consumption of the high-amylose rice, it was concluded that a small rather than a large response in blood glucose is associated with greater satiety, suggesting that even a short-term high glycemic response is counterproductive.

By contrast to the foregoing, high but not low glycemic responses are associated with greater satiety and reduced food intake in short-term studies of approximately one hour. For example, when pure isovolumetric (400 mL) preloads of 75 g polycose, sucrose, glucose, or a fructose/glucose mixture were consumed by young men, the greater the glycemic response, the greater the decrease in reported appetite and food intake after one hour. Glucose and sucrose decreased food intake compared with control, but food intake after the fructose/glucose and polycose treatments was not different from all other treatments (Table 4). Polycose, glucose, and sucrose produced a rapid increase in blood glucose between baseline and 20 minutes that remained elevated above baseline after 65 minutes (Figure 3). The combined fructose/glucose treatment elicited a smaller increase in blood glucose than all other carbohydrate treatments and returned to baseline by 65 minutes. Overall, there was an inverse relationship between both the area under the curve for blood glucose and food intake (Figure 4) and blood glucose at 37 minutes (Figure 5).

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Energy Intake (kJ)</th>
<th>% Compensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sucrose</td>
<td>4172 ± 292</td>
<td>11.5 ± 15.7</td>
</tr>
<tr>
<td>Fru/Glu</td>
<td>4030 ± 323</td>
<td>42.3 ± 14.3</td>
</tr>
<tr>
<td>Sucrose</td>
<td>3641 ± 330</td>
<td>36.4 ± 16.05</td>
</tr>
<tr>
<td>Polycose</td>
<td>3716 ± 302</td>
<td>48.1 ± 25.5</td>
</tr>
<tr>
<td>Glucose</td>
<td>3570 ± 341</td>
<td></td>
</tr>
</tbody>
</table>

* Mean ± SEM (kJ), n = 15, from reference 16.
1 Energy consumed at a pizza test meal served 60 minutes following ingestion of the preloads, which were provided as isovolumetric (200 mL) beverages, equalized for sweetness with additions of the noncaloric sweetener, sucralose. Immediately following ingestion of each treatment, subjects consumed an additional 200 mL of spring water for a total volume intake of 400 mL.
2 Compensation = (kJ intake after Control − kJ intake after Treatment/kJ in preload) × 100.
3 The fructose/glucose mixture (fru/glu) contained 80% fructose and 20% glucose (to decrease the extent of malabsorption).
4 Means with different superscripts, within a column, are significantly different at P < 0.05 by one-way ANOVA followed by post-hoc Duncan’s.
and food intake 60 minutes after the preloads. Similarly, in a study of the effect of 38 common foods, the higher insulin area under the curve was associated with lower food intake 2 hours later.45 This implies that the higher the glycemia, the greater the satiety.

The GI was originally developed to provide a physiologic basis for carbohydrate exchange in diets of diabetics.39 More recently it has received application in the prediction of satiety arising from foods and as a guide for weight management and meal planning. When used in this way, it is important to recognize that the GI reflects much more than the type of carbohydrate in the food or diet. The GI is affected not only by the class of carbohydrate in the food, but also by its processing, food form, and the presence of fat or protein. Therefore, it cannot be assumed that the outcome of studies of high- or low-GI diets or foods can be attributed to their effects on blood glucose. For example, when children were provided a low-glycemic, unrestricted diet or a low-fat, hypocaloric diet for a period of one month, the children in the low-glycemic group lost significantly more weight than those in the reduced-fat group. 46 On the surface, these results appear to support a role for low-glycemic foods in weight management efforts. However, children on the low-glycemic diet were instructed to consume protein with all meals and snacks to achieve a target protein intake of 20 to 25% compared with the target of 15 to 20% in the high-glycemic group. Similarly, Ludwig et al.7 concluded that a low-GI diet is preferable to a high-GI diet in promoting satiety and weight loss in children. The low-GI diet contained 30% protein and the high-GI diet contained only 16% protein.

The benefit of the low-GI, high-protein diets might more logically be attributed to their high protein content and not their GI. Protein is much more satiating than either fat or carbohydrate.47,48 Diets containing 24% protein compared with 12% protein lead to greater satiety and weight loss in obese adults.49 It is therefore unclear what characteristic of a food or diet is leading to satiety and being marked by the GI. It seems unlikely that it is only the carbohydrate component.
Glycemic Carbohydrates and Mechanisms of Food Intake Regulation

The blood glucose response to low- and high-glycemic carbohydrates is a possible component of mechanisms underlying their effect on satiety. There is, however, no consistent evidence that an increase in blood glucose is the primary determinant of satiety subsequent to carbohydrate consumption. Many other regulatory mechanisms may be involved.

Consistent with the glucostatic hypothesis of food intake regulation, the rapid increase in blood glucose following ingestion of rapidly digestible, high-glycemic index carbohydrates is associated with suppression of short-term (1 hour) satiety (Figure 4). Similarly, the higher sustained blood glucose with low-glycemic meals compared with high-glycemic meals is associated with greater satiety for up to 6 hours after consumption. However, interpretation of the role of blood glucose in determining satiety must be made with caution. For example, consistent with the hypothesis is the reported correlation between the duration of a rise in blood glucose and inter-meal interval. A rapid increase and then decline in blood glucose following sucrose (1000 kJ) corresponded to a shortened inter-meal interval compared with the small but sustained rise in blood glucose and longer inter-meal interval caused by a low-glycemic preload. This supports the view that sustained elevation in postprandial blood glucose concentrations is the mechanism by which satiety is maintained. Because the low-glycemic food in this study was one high in fat, fat may produces greater satiety than carbohydrate over longer periods of time, possibly reflecting a satiety mechanism unrelated to blood glucose.

A lack of association between blood glucose response and food intake is also easily demonstrated when foods or meals of mixed composition are fed and the carbohydrate content is kept constant. For example, the glycemic response to breakfast cereals containing either 30 g fructose or 33.5 g glucose differed greatly (Figure 6), but food intake was the same 30 minutes or 120 minutes after consumption. (Table 3). Similarly, no relationship was observed between glycemic response and appetite, as assessed with a seven-point hunger scale after 30 and 120 minutes.

Further evidence that blood glucose is not the only indicator of satiety is provided by studies in which blood glucose concentrations have been altered through intravenous administration of glucose. Early studies demonstrated either no effect or an increase in hunger and food intake under hyperinsulinemic and hyperglycemic (10 mmol/L) conditions. By contrast, more recent studies have found that acute hyperglycemia (15 mmol/L) induces satiety over 240 minutes and decreases food intake at 140 minutes. Therefore, it is unclear the extent to which the effects of low- and high-glycemic carbohydrates on satiety are mediated by mechanisms sensitive to their effect on blood glucose concentrations.

It is more likely that the glycemic effect of carbohydrates depicts their absorption characteristics and not the specific mechanism by which they provide satiety signals. Many other mechanisms, including those based on rate of gastric emptying and gut hormones, may

![Figure 6. In a within-subject design, young males consumed three treatments: water (no breakfast control), Mini Shredded Wheat cereal (SW) + 30 g fructose (fru), and SW + 33.5 g glucose (glu). The two breakfast cereal treatments were of similar palatability and equal macronutrient composition. The fru (15.9 kJ/g) and glu (14.2 kJ/g) were dissolved in 100 mL 1% milk, and then added to 51 g of the cereal prior to serving. Also served was 150 mL of water for drinking. Blood glucose concentrations were measured on two separate occasions: (A) 30 minutes following preloads and (B) 2 hours following preloads. Means with different letters, within the same time point are significantly different ($P < 0.05$, $n = 13$). Vertical bars on each point represent SEM. Reproduced with permission from Elsevier Science.](image)
into the bloodstream. A rise in blood glucose concentration would occur more quickly as the glucose is transported from the gut lumen because of the presence of food in the small intestine. These include cholecystokinin, glucagon, bombesin, gastrin, somatostatin, neurotensin, and glucagon-like peptide-1 (GLP-1). GLP-1 has received considerable attention as a putative satiety peptide involved in regulating carbohydrate-induced satiety and is released when glucose comes into contact with the L-cells of the lower small intestine.

Prolonged intestinal contact, as would be expected from slowly digested food, enhances satiety. When 5 g of guar gum was added to a 300-kcal glucose beverage, an increase in satiety and a decrease in hunger was observed. The effect of the guar gum was not mediated by a change in the rate of gastric emptying. It was proposed that the viscous properties of guar gum increased satiety through delayed absorption of glucose and increased contact of glucose with receptors in the small intestine and subsequent release of putative satiety peptides. High-glycemic carbohydrates might also mediate satiety through mechanisms independent of their effect on blood glucose. For example, a rapid increase in the occupancy of glucoreceptors would be expected following ingestion of high-glycemic carbohydrates. Thus, a surge of preabsorptive satiety signals would be produced, but they would be expected to dissipate relatively quickly as the glucose is transported from the gut lumen into the bloodstream. A rise in blood glucose concentrations has been associated with a slowing of gastric emptying, which would also contribute to fullness and short-term satiety.

Conclusion

Carbohydrates, including simple sugars, are recognized by food intake regulatory systems. The ability of carbohydrates to effect satiety varies according to their composition. Both high- and low-glycemic carbohydrates have been shown to suppress appetite and food intake, but they follow different time courses. High-glycemic carbohydrates suppress short-term (1 hour) intake more effectively than low-glycemic carbohydrates, but the reverse occurs over longer periods of time (6 hour).

Although the effect of glycemic carbohydrates on food intake appears to be related to their effects on blood glucose, the mechanism by which glycemic carbohydrates modulate food intake is unlikely to be solely based on this effect, as proposed by the glucostatic hypothesis of food intake regulation. The release of putative satiety peptides, mediated by the intensity and length of interaction of carbohydrates in the gastrointestinal tract, is no doubt a crucial component of mechanisms initiating and sustaining satiety.

In conclusion, both high- and low-glycemic foods promote appetite and food intake suppression in healthy individuals. It may be suggested, therefore, that a diet containing both high- and low-glycemic carbohydrates is appropriate for the maintenance of a healthy body weight. Further studies are required to delineate the effect of high- and low-glycemic carbohydrates on appetite and weight management, however, and on the mechanisms by which they bring about these effects.

15. Woodend DW, Anderson GHA. Effect of sucrose
50. Krishnamachar S, Mickelsen O. The influence of


