Consumption of sugars and the regulation of short-term satiety and food intake[1–4]

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ABSTRACT This review examines the relation between the consumption of sugars and their effects on short-term (ie, to 2 h) satiety and food intake in humans. Many factors need to be considered in the evaluation of reported studies and the conclusions derived from this body of literature. These factors include evaluation of the dose and form (solid or liquid) of the treatments, time of day administered, characteristics of the subjects, sample size, and approaches used to measure satiety and food intake. Mechanisms by which sugars may signal regulatory systems for food intake need to be considered when evaluating both study designs and conclusions. For this reason, the relation between the blood glucose response to sugar consumption and subsequent feeding behavior is also examined. It is concluded that sugars stimulate satiety mechanisms and reduce food intake in the short term and that the mechanisms by which this response occurs cannot be attributed solely to their effect on blood glucose. *Am J Clin Nutr* 2003;78(suppl):843S–9S.

KEY WORDS Sucrose, sugars, food intake, satiety, preloads, young men, blood glucose

INTRODUCTION

Carbohydrates are the primary macronutrient source of energy in most diets and average between 45% and 60% of total energy intake (1). Sugars from all sources made up ≈50% of carbohydrates, or 22%, of the total energy intake in 1987–1988 (2). Sugars are either monosaccharides or disaccharides and include sucrose, lactose, maltose, glucose, and fructose.

Carbohydrate ingestion promotes satiety (3, 4). However, in recent years, the prevalence of obesity has increased despite reported declines in fat intake (5) and a subsequent rise in carbohydrate consumption. Thus, it has been suggested by some that because sugars and high–glycemic index (GI) carbohydrates have contributed to this increase, they are the cause of overeating and obesity (6–8).

The relation between the consumption of sugars and their immediate (up to 2 h) effects on satiety and food intake are explored here. Also examined is the hypothesis that the glycemic response to sugars predicts their effects on satiety and food intake. An evaluation of the effect of the consumption of sugars over the longer term, that is over a day or several days, on energy metabolism and body weight control is provided in a separate review (9).

SUGARS, APPETITE, AND FOOD INTAKE

Sugars that have received considerable investigation for their effects on appetite and food intake include sucrose and its components, glucose and fructose. Lactose and maltose have received much less attention. One factor driving the interest in the effect of some sugars on food intake is their sweet taste. Many have assumed that this is a cause of excessive consumption. Sweetness per se, however, does not stimulate food intake (10, 11) and may even have a weak effect on satiety. Thus, in studies of the effect of sucrose drinks on satiety and food intake, whether the control treatment is sweetened may influence interpretation of the data.

Sweet taste alone has been proposed to contribute to the reduction of hunger and increased feelings of fullness (12). Some studies support this hypothesis. For example, in the studies by Woodend and Anderson (13) (**Table 1**), the effect on food intake of drinks containing 25 g (418 kJ) and 50 g (836 kJ) sucrose was not different from that of the noncaloric sweetened control but was different from the water control. In another study of adult subjects, noncaloric sweetened beverages reduced hunger ratings to an amount intermediate between sucrose (20 g) and the water control (8). Similarly, an aspartame-sweetened beverage led to suppression of food intake in children compared with the effects of a water control (14).

The satiating effect of sweetness has been found not only in the absence of but also in the presence of energy. When given in lemon-flavored solutions, sucrose preloads increased fullness and decreased prospective consumption more than did maltose, which is less sweet (15). Prolonged orosensory stimulation by sucrose (15 g), consumed over 10 min as candies, compared with a drink consumed in 2 min, suppressed food intake immediately afterward (12). The authors conclude that this is further evidence of the effect of prolonged stimulation by sweetness and not calories. In

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[2] Presented at the Sugars and Health Workshop, held in Washington, DC, September 18–20, 2002. Published proceedings edited by David R Lineback (University of Maryland, College Park) and Julie Miller Jones (College of St Catherine, St Paul).

[3] Manuscript preparation supported by ILSI NA; research supported by ILSI Japan.

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TABLE 1
Food intake after sucrose[1]

Treatment	Energy intake[2]	Compensation, water control[3]	Compensation, sweet control[3]
	kJ	*%*	*%*
Water	4606 ± 376[a]	0	NA
Sweet control	4456 ± 380[a,b]	NA	0
418 kJ sucrose	4088 ± 347[b]	123	87
836 kJ sucrose	4088 ± 255[b]	62	44
1254 kJ sucrose	3477 ± 322[c]	90	79

[1] $\bar{x}$ ± SEM; n = 14 young men. From reference 13. Means with different superscript letters, within a column, are significantly different, $P < 0.05$ (one-way ANOVA followed by post hoc Duncan's test).

[2] Energy consumed at a pizza test meal served 60 min after ingestion of the preloads, which were provided as isovolumetric (300 mL) beverages, equalized for sweetness with addition of the noncaloric sweetener sucralose.

[3] Energy compensation at the test meal for energy in the preloads, compared with the water or sweet (sucralose) control. Compensation = [(kJ intake after control − kJ intake after treatment)/kJ in preload] × 100.

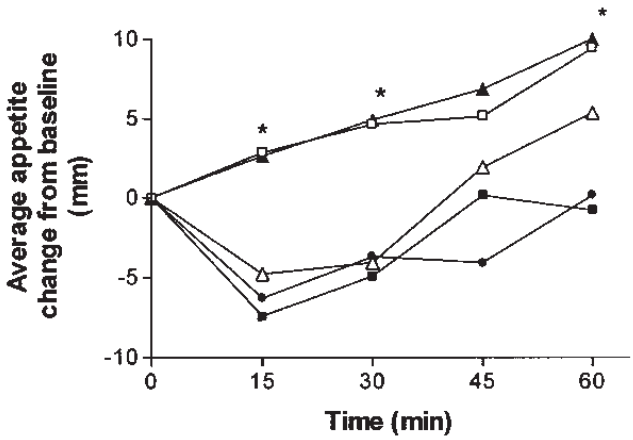

FIGURE 1. Change in average appetite scores, measured by visual analogue scales, over 1 h after treatment with a water control (□), sweet control (sucralose + water; ▲), 25 g sucrose (△), 50 g sucrose (■), and 75 g sucrose (●). The treatments, except for the water control, were equalized for sweetness with the addition of the noncaloric sweetener sucralose, and all treatments were provided as isovolumetric (300 mL) beverages. Within-subject design; n = 14 young males. *An overall treatment effect ($P < 0.05$) was observed at 15, 30, and 60 min. The calculated incremental areas under the curves were different between treatments as follows: □[a], ▲[a], △[a,b], ■[b], ●[b]. Treatments with different letter superscript are significantly different, $P < 0.05$. From reference 13.

contrast, sweetened noncaloric soft drinks sipped for 10 min had no greater effect on subjective measures of satiety than when consumed in 2 min (10). Thus, the role of sweetness in contributing to satiety requires further examination.

Evidence for an association between sugars and overweight has been derived from epidemiologic, observational, and experimental studies. Although epidemiologic studies consistently report an inverse association between body mass index and sugar intake in adult populations (16–18), a relation between the form of sugars consumed and overweight in children has been identified. Consumption of sweet beverages, especially soft drinks, was found to be associated with overweight in youths in one study (19) and with reported higher energy intakes in children in another study (20). Similarly, a prospective observational study reported an association between increased soft drink consumption and the development of obesity in children over a 19-mo period (6). Because one report suggested that the sugars consumed in liquid rather than in solid form are less likely to be compensated for during the day (21), it has been hypothesized that sugars, especially those used as caloric sweeteners, when consumed in drinks, contribute to excess energy intake through bypassing regulatory systems and by exacerbating hunger (6–8, 21).

The literature, however, provides no evidence that sugars bypass regulatory systems and, for this reason, create excess energy intakes. Experimental studies that have been designed to test the effect of quantity and the time interval between the dose and the test meal, and to compare sugars with other carbohydrates, show remarkably precise compensation in a subsequent meal (the short-term response) for the energy contained in sugars consumed 30–60 min before the meal. Furthermore, the compensation is better in the short term for high-glycemic than for low-glycemic carbohydrates.

Sucrose

Both adults and children are satiated by sucrose and reduce food intake if the time intervals between the preload and the test meal are appropriate for the dose consumed. Most of the literature shows that food intake is reduced after ingestion of ≥50 g sucrose in drinks presented 20–60 min before a meal (22). Larger amounts would be expected to prolong satiety, and this was shown recently. A beverage containing 135 g sucrose caused a stronger feeling of fullness and reduced ratings of prospective consumption and hunger compared with the water control for 2–3 h (15).

Even small amounts of sucrose decrease food intake. For example, when young men were given drinks (300 mL) containing 25, 50, or 75 g sucrose, the lowest dose of 25 g (418 kJ) increased subjective satiety, as assessed by visual analogue scales (**Figure 1**) and suppressed food intake from a pizza meal 1 h later (Table 1) (13). There was 123% compensation, compared with the water control, at 1 h for the 418 kJ (100 kcal) provided in the 25-g sucrose beverage. Compensation for the 50- and 75-g doses was 62% and 90%, respectively. Clearly the compensatory responses under similar conditions within an experiment are not precisely related to the energy contained in the drinks. However, there is no evidence that the variability in compensation is less and the response is more precise for other carbohydrates. Indeed, when comparisons were made among drinks equally sweetened with a noncaloric sweetener and containing 75 g sucrose, polycose (a linear oligosaccharide of glucose), or glucose, the energy compensation—in relation to the noncaloric sweet control, sucralose (Splenda; Tate & Lyle, Reading, United Kingdom)—was not different at a test meal consumed 1 h later (42 ± 14%, 36 ± 16%, and 48 ± 25%, respectively) (23). In another experiment, equally sweetened preload drinks containing 75 g sucrose and polycose led to significant compensation in a meal 1 h later (44 ± 13% and 65 ± 19%, respectively), whereas amylopectin and amylose did not (0 ± 22% and 23 ± 16%, respectively) (23). Again the comparison is with a noncaloric sweet control. Thus, the response to sucrose in pure form and not mixed with foods is at least as precise as for other sugars and better in the short term than for carbohydrates with a lower GI.

It might be argued that the compensation in a test meal for the energy content of the sucrose-containing preload was not perfect

and therefore would lead to a gradual accumulation of body fat. However, this needs to be put in perspective. By comparison, correction for the energy content of safflower oil preloads of 100, 200, and 300 kcal given 60 min before the meal averaged <40% and there was a statistically significant reduction only after the 300-kcal preload (13). Thus, if there is an error in intake correction for sugar preloads, the error is much less than that for fat.

The 25-g dose of sucrose may be at the low end of the detection limit for an energy preload to have an effect at a meal 1 h later in adults. Drinks containing 20 g sucrose (76 kcal) did not show a statistically significant suppression of food intake 1 h later in 20 subjects (18 females, 2 males) (8). However the difference in the mean intakes between the sucrose and the water treatment was 85 kcal. Possibly, if only the 2 treatments had been given in a paired design rather than a comparison of 4 treatments in a repeated-measures design, statistical significance could have been achieved.

Young children also compensate for sugar consumed as a beverage. In children aged 2–5 y (n = 24), 90 kcal from a sucrose drink was sufficient to suppress intake at test meals 0, 30, or 60 min after the preload (14). The compensation observed at 30 min was 100%, which the authors attributed to the ability of young children to rely solely on internal hunger cues. Although not found to be as precise, older children also compensate for energy derived from sucrose. When children aged 9–10 y consumed a cherry-flavored drink containing either 45 or 90 g sucrose, lunchtime food intake was reduced 30 min later (24). Compensation for the 45- and 90-g sucrose beverages was 68% and 63%, respectively.

The importance of timing of the test meals in relation to the size of the treatment dose is illustrated by the failure of 50–60 g sucrose to suppress food intake of children aged 9–10 y when the test meal was given 90 min later. However, hunger and desire-to-eat ratings were lower after the drinks containing sucrose than after the drinks containing aspartame at 85 min (25).

It is clear therefore, that under laboratory conditions, sucrose contributes to satiety and suppresses subsequent food intake. Thus, the data refute the hypothesis that sucrose leads to obesity by bypassing regulatory systems (6–8).

The literature does not report comparisons of the effects of corn syrups or of high-fructose corn syrups given in similar quantities with the effects of sucrose on short-term food intake. Some of the corn syrups are a source of sugars in the diet, although many used by the food industry are composed of long chains of glucose and are used for functional, not sweetening, purposes. The high-fructose corn syrups commonly used as sweeteners in foods and beverages have a monosaccharide composition similar to that of sucrose, ie, they contain a mixture of ≈55% fructose and 45% glucose. It seems unlikely that there would be a difference in satiety between a beverage containing sucrose and one containing high-fructose corn syrup.

Glucose and fructose

Because sucrose and high-fructose corn syrups are composed of glucose and fructose, one or both of these monosaccharides may explain their effect on food intake 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.

In young men, the consumption of 75 g (23) or 50 g (26) glucose in drinks or 50 g in yogurt (27) reduces food intake 1 h later. Fructose consumed alone also suppresses food intake. In many studies, 50 g fructose in a drink suppressed energy intake and did so to a greater extent than did glucose at test meals from 38 min (28) to 2.25 h later (29–31).

TABLE 2
Food intake after a carbohydrate preload of 1379 kJ[1]

Treatment	Energy intake[2]	Compensation[3]
	kJ	*%*
30 min after carbohydrate		
SW + fructose	3578 ± 355[a]	85.3 ± 21.2
SW + glucose	3486 ± 293[a]	91.9 ± 15.9
Water	4757 ± 385[b]	—
2 h after carbohydrate		
SW + fructose	4431 ± 364[a]	70.2 ± 16.8
SW + glucose	4811 ± 380[a]	42.5 ± 17.2
Water	5396 ± 359[b]	—

[1] $\bar{x}$ ± SEM; n = 13 young males. From reference 32. Means with different superscript letters, within a column, are significantly different, $P < 0.05$ (one-way ANOVA followed by post hoc Duncan's test).

[2] Energy consumed at a pizza test meal served 60 min after 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 2 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 cereal before serving; 150 mL water for drinking was also served.

[3] Compensation =[(kJ intake after control – kJ intake after treatment)/kJ in preload] × 100.

When fructose is consumed with another carbohydrate, its advantage over glucose disappears. For example, equicaloric cereal preloads containing additions of fructose (30 g) or glucose (33.5 g) reduced energy intake in meals consumed either 30 or 120 min later, but there were no differences between the treatments (32; **Table 2**). Similarly, no differences in food intake were observed between 50 g fructose and 50 g glucose at 2.25 h, when they were given in a mixed nutrient meal containing starch (31). As little as 15 g starch or glucose added to 50 g fructose prevents the decrease in food intake 2.25 h after a 50-g fructose preload (31).

Factors that might account for the relatively strong effects of fructose on satiety, when given alone, include its absorption characteristics and gastrointestinal effects. Fructose is absorbed slowly (33), which allows prolonged contact time with gastrointestinal receptors that produce satiety signals (34, 35). In addition, fructose is incompletely absorbed and as a result produces a hyperosmolar environment in the large intestine (36). A high concentration of solute within the gut lumen draws fluid into the intestine. This fluid shift can produce feelings of malaise or diarrhea (36), which can decrease food intake. When fructose is consumed with even a small amount of glucose or starch, these aspects are eliminated because these carbohydrates facilitate a more rapid and complete absorption of fructose (33).

At this time there is no evidence that the effect of sucrose or high-fructose corn syrups of similar composition on food intake can be attributed solely to one 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 time between consumption and eating is short. The duration of the effect has not been fully defined but is dose dependent.

Maltose and lactose

Only one study examined the effects of maltose using the preload paradigm, and it suggests that maltose can suppress appetite

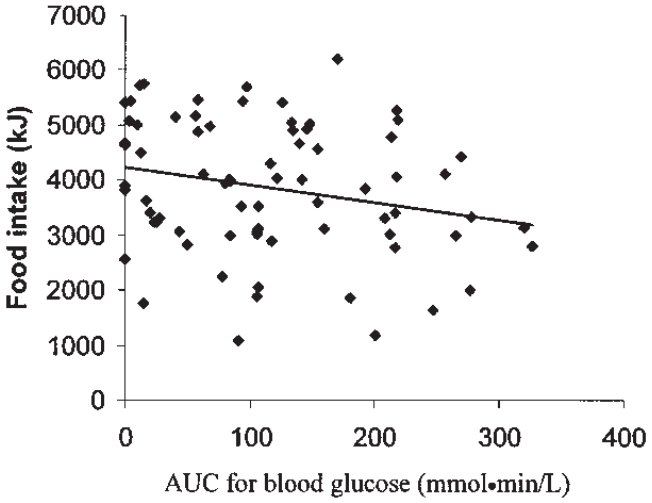

FIGURE 2. Correlation between food intake and area under the curve (AUC) for blood glucose in young males ($n = 15$). The subjects consumed drinks consisting of a sweet control (sucralose + water) or 75-g preloads of sucrose, glucose, a fructose-glucose mixture (80% fructose, 20% glucose), or polycose in a within-subject design. Caloric intake from a pizza test meal was measured 1 h later. The blood glucose response to treatments was calculated as the incremental AUC based on measurements taken at baseline and 20, 37, and 65 min after the treatments. The AUC was negatively correlated with total food intake at the 1-h test meal ($r = -0.24$, $P < 0.05$). From reference 23.

(15). An oral preload of 135 g maltose in a lemon-flavored solution decreased hunger compared with water 150–180 min later but did not increase fullness or decrease prospective consumption ratings as did sucrose (15). No reports of the effect of lactose preloads on food intake in humans were found in the published literature.

SUGARS, GLYCEMIC RESPONSE, AND FOOD INTAKE

In addition to the caloric bypass notion that assumes sugars ingestion does not lead to suppression of food intake, it has been suggested that consumption of higher-GI carbohydrates, including some sugars, stimulate food intake (7, 37). This hypothesis recognizes that there is a rapid increase in blood glucose concentrations after the consumption of rapidly digested starch or sugars. However, it also assumes that the associated insulin response not only regulates and returns blood glucose to baseline but also results in the glucose concentration falling below baseline, which in turn stimulates hunger and leads to an excess of energy intake. A direct test of this hypothesis has not been reported.

The effect of sugars on blood glucose can be described by measuring the total glycemic response over time. The GI was developed to provide a basis for comparing glycemic responses to carbohydrates in foods (38, 39). This index compares the incremental area under the blood glucose response curve of 50 g glycemic carbohydrate in a test food relative to 50 g carbohydrate of a standard food such as white bread, when ingested by the same subject (38). 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 after ingestion of sugars (40). Glucose produces a more rapid and higher increase in postprandial blood glucose and insulin than does fructose (30, 31). Sucrose tends to elicit a postprandial blood glucose concentration that is intermediate between glucose and fructose (39, 40). The GIs of glucose, fructose, and sucrose—expressed relative to 100 for white bread—are 149, 32, and 87, respectively (40, 41). Contrary to the belief that sugars produce higher blood glucose concentrations than does an equivalent amount of starch, fructose and sucrose have lower GI values by up to 50% compared with most common starchy foods (41, 42). For this reason, replacing a portion of the starch with sucrose in a high-GI breakfast cereal lowers the glycemic and insulin responses (43).

In the short term, high but not low glycemic responses are associated with satiety and reduced food intake (13, 44). 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 glycemic response, calculated as blood glucose area under the curve from time 0 to 60 min, was inversely related to food intake 1 h after treatment (23) (**Figure 2**). Glucose and sucrose decreased food intake compared with a control, but food intake after the fructose-glucose and polycose treatments was not different from that after all other treatments. Polycose, glucose, and sucrose produced a rapid increase in blood glucose between baseline and 20 min, which remained elevated above baseline at 65 min. The combined fructose-glucose treatment elicited a smaller increase in blood glucose than did all other carbohydrate treatments, and blood glucose returned to baseline by 65 min. Overall, there was a weak inverse relation between food intake from a pizza meal 1 h after treatments and both the incremental area under the curve for blood glucose and blood glucose concentrations at 37 min ($r = -0.24$, $P < 0.05$) and 60 min ($r = 0.23$, $P = 0.06$). Consistent with this observation is a study of the effect of 38 common foods on food intake 2 h later (44). Insulin area under the curve was inversely related to food intake. Thus, these studies suggest that the greater the response in blood glucose and in insulin, the greater the satiety after carbohydrate consumption, at least to 2 h.

It contrast, one review concludes that 5 reports of data from crossover studies show that meals or preloads, described by the author as having a high GI, are associated with overeating, whereas those described as having a low GI result in lower food intake at test meals consumed later (37). The results are attributed to the presumed effects of the treatments on blood glucose and assume that the GI of a food can be used to predict satiety after its consumption. This conclusion can be challenged, however, for 4 reasons. First, the relation of the glycemic response to food intake was not measured in these studies. Second, as pointed out earlier, the GI is a unit specifically designed to describe the glycemic response to a test food containing 50 g carbohydrate in comparison with a standard of 50 g carbohydrate in white bread or 50 g glucose (38). It is not a term to apply to a meal containing very low amounts of carbohydrate and primarily containing protein and fat as used in one (7) of the 5 studies. Because protein is more satiating than is carbohydrate, it would be expected that the high-protein meal (described as low GI) would be expected to suppress food intake at the next meal compared with the meal relatively high in carbohydrate (52). Third, the low glycemic response to fructose preloads as an explanation for reduced food intake after its consumption compared with food intake after a glucose preload as reported in 3 of the 5 studies can be questioned. Fructose

malabsorption and its effects in the gut and not its glycemic effect may account for the results (36). Finally, the role of blood glucose in determining the effect of carbohydrate on satiety and subsequent food intake remains uncertain and casts doubt on the use of the GI of a food to predict its effects on food intake, at least in the short term.

SUGARS AND FOOD INTAKE REGULATION

To explain the mechanisms by which carbohydrates regulate food intake, Mayer (45) proposed the glucostatic theory in 1953. The glucostatic theory proposes that low blood glucose concentrations trigger the onset of feeding, and high blood glucose concentrations signal satiety and the termination of feeding. In support of the hypothesis, transient declines in blood glucose of the correct magnitude and time course are believed to induce meal initiation as they are detected by peripheral and central glucoreceptive elements and mapped into feeding behavior (46). Indeed, a decrease in blood glucose is associated with the initiation of feeding in both animals and humans (46–48), but the initiator of this sudden drop in blood glucose is unknown. There is no evidence, however, that it arises from the same physiologic events that cause glucose to fall below baseline after consumption of a large quantity of rapidly absorbed carbohydrate or that both events similarly trigger hunger and eating.

Consistent with the glucostatic hypothesis are the observations that carbohydrate consumption and the resulting increase in blood glucose are associated with satiation (23, 49, 50). Also consistent with the hypothesis are the correlations observed between the duration of a rise in blood glucose and the intermeal interval (48). A rapid increase and then decline in blood glucose after sucrose (1000 kJ) ingestion was found to correspond to a shortened intermeal interval, whereas a small but sustained rise in blood glucose was found to be associated with a longer intermeal interval after a low glycemic preload. The results of this study were interpreted to support the view that a sustained elevation in postprandial blood glucose concentrations is the mechanism by which satiety is maintained. However, the low glycemic food in this study was one high in fat, leading to a possible alternative explanation. Because fat produces weaker but sustained satiety compared with carbohydrates (51), the delayed intermeal interval may reflect a satiety mechanism unrelated to blood glucose. For instance, fat stimulates the release of cholecystokinin, which can act peripherally to signal satiety (52).

Although the data provide indirect support for the hypothesis that satiety is associated with the effects of carbohydrates on blood glucose, a primary role for blood glucose in determining satiety remains uncertain (53). A lack of association between the blood glucose response and food intake is also easily shown. For example, the glycemic response as measured by the blood glucose incremental area under the curve to 2 h was 85 ± 10 and 175 ± 20 $mmol \cdot L^{-1} \cdot min^{-1}$ after breakfast cereals containing either 30 g fructose or 33.5 g glucose, respectively, but food intake was not significantly different 30 or 120 min after consumption (32) (Table 2).

Further evidence that blood glucose is neither the only nor the best predictor of satiety is provided by studies in which blood glucose concentrations have been altered through intravenous administration of glucose. Early studies showed either no effect (54) or an increase in hunger and food intake under hyperinsulinemic and hyperglycemic (10 mmol/L) conditions (55). In contrast, more recent studies have found that acute hyperglycemia (15 mmol/L) induces satiety over 240 min (50) and decreases food intake at 140 min (49).

From the foregoing studies it cannot be concluded that the effects of low- and high-GI carbohydrates on satiety are mediated by mechanisms sensitive to their effect on blood glucose concentrations. It is more likely that the glycemic response to carbohydrates serves to depict their absorption characteristics and not necessarily the specific mechanism by which they provide satiety signals (53). Many other mechanisms, including those based on the rate of gastric emptying and gut hormones, may account for the effects of carbohydrates on food intake. For example, contact of nutrients with the small intestine is postulated to be a major source of postgastric satiety (34, 35). Many peptides with the potential to influence satiety are released in response to the presence of food in the small intestine. These include, but are not limited to, cholecystokinin, glucagon, bombesin, gastrin, somatostatin, neurotensin, and glucagon-like peptide 1 (56)

Sugars also mediate satiety through mechanisms not directly involving their effect on blood glucose. For example, a rapid increase in the occupancy of glucoreceptors would be expected after ingestion of glucose, sucrose, or high-fructose corn syrups. 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 more extended effect of sugars on satiety might arise from the slowing of gastric emptying and the release of glucagon-like peptide 1. Glucagon-like peptide 1 has received considerable attention as a putative satiety peptide involved in regulating carbohydrate-induced satiety (57–59) and is released when glucose comes into contact with the L cells of the lower small intestine (60). A rise in blood glucose concentrations has been associated with a slowing of gastric emptying (61), which would also contribute to fullness and short-term satiety (62).

FUTURE RESEARCH

The emphasis of this review has been on the short-term food intake response to sugars. Although it is clear that the ingestion of sugars suppresses food intake, the effects of their chronic ingestion on energy balance cannot be predicted by these studies. The determinants of energy balance are many, and there appears to be much redundancy in control mechanisms (52). How and when corrections are made in food intake through intermeal intervals and meal-to-meal or day-to-day adjustments remain unknown and require investigation. On the basis of the short-term studies reported herein, it is reasonable to hypothesize that such events are not less sensitive to the ingestion of sugars than to fats or to other carbohydrates in the diet.

Further research is required to answer several questions that are unresolved on the topic of sugars and appetite control: *1*) Is there a difference between solid and liquid forms of the different sugars? Is it due to a caloric or sweetness response? *2*) What is the duration of the satiety response to sugars, and how does this compare with the duration of effect of other carbohydrates? *3*) How do sugars added to food affect satiety and food intake compared with the addition of polysaccharides? *4*) What is the effect of daily consumption of drinks containing sugars compared with the chronic consumption of drinks containing low-GI carbohydrates on body weight and energy intake? *5*) Does a glycemic rebound

occur after usual servings of sugars in beverages, and does this cause increased hunger? *6*) What is the role of satiety signals from the gut in controlling food intake and satiety after sugars compared with other carbohydrates?

In conclusion, sugars produce satiety and decrease food intake in the short term similar to other high-GI carbohydrates but to a greater extent than low-GI carbohydrates. Their effect is also greater than that of fat. Although the effect on food intake of high-GI sugars and carbohydrates often is associated with their effects on blood glucose, the mechanism by which sugars 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 the mechanisms by which they initiate and sustain satiety.

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REFERENCES

1. Asp N. Nutritional classification and analysis of food carbohydrates. Am J Clin Nutr 1994;59(suppl):679S–81S.
2. Gibney M, Sigman-Grant M, Stanton JLJ, Keast DR. Consumption of sugars. Am J Clin Nutr 1995;62(suppl):178S–94S.
3. Anderson GH, Stewart S, Kaplan R. Carbohydrate, behavior and health. Bahrain Med Bull 1998;20:69–76.
4. Li ETS, Anderson GH. Dietary carbohydrates and the nervous system. Nutr Res 1987;7:1329–39.
5. Willet WC. Is dietary fat a major determinant of body fat. Am J Clin Nutr 1998;67(suppl):556S–62S.
6. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. Lancet 2001;357:505–8.
7. Ludwig DS, Majzoub JA, Al-Zahrani A, Dallal GE, Blanco I, Roberts SB. High glycemic index foods, overeating and obesity. Pediatrics 1999;103:E261–6.
8. Canty DJ, Chan MM. Effects of consumption of caloric vs noncaloric sweet drinks on indices of hunger and food consumption in normal adults. Am J Clin Nutr 1991;53:1159–64.
9. Saris WHM. Sugar, energy metabolism and body weight control. Am J Clin Nutr 2003;78(suppl):850S–7S.
10. Black RM, Tanaka P, Leiter LA, Anderson GH. Soft drinks with aspartame: effect on subjective hunger, food selection, and food intake of young adult males. Physiol Behav 1991;49:803–10.
11. Rolls BJ, Kim S, Federoff IC. Effects of drinks sweetened with sucrose or aspartame on hunger, thirst, and food intake in men. Physiol Behav 1990;48:19–26.
12. Lavin JH, French SJ, Ruxton CH, Read NW. An investigation of the role of oro-sensory stimulation in sugar satiety. Int J Obes Relat Metab Disord 2002;26:384–8.
13. Woodend DW, Anderson GH. Effect of sucrose and safflower oil preloads on short term appetite and food intake. Appetite 2001;37:185–95.
14. Birch LL, Mcphee L, Sullivan S. Children's food intake following drinks sweetened with sucrose or aspartame: time course effects. Physiol Behav 1989;45:387–95.
15. Lavin JH, French SJ, Read NW. Comparison of oral and gastric administration of sucrose and maltose on gastric emptying rate and appetite. Int J Obes Relat Metab Disord 2002;26:80–6.
16. Bowman SA. Diets of individuals based on energy intakes from added sugars. Fam Econ Nutr Rev 1999;12:31–8.
17. Hill JO, Prentice AM. Sugar and body weight regulation. Am J Clin Nutr 1995;62(suppl):264S–74S.
18. Bolton-Smith C, Woodward M. Dietary composition and fat to sugar ratios in relation to obesity. Int J Obes Relat Metab Disord 1994;18:820–8.
19. Troiano RP, Briefel RR, Carroll MD, Bialostosky K. Energy and fat intakes of children and adolescents in the United States; data from the national health and nutrition examination surveys. Am J Clin Nutr 2000;72(suppl):1343S–53S.
20. Harnack L, Stang J, Story M. Soft drink consumption among US children and adolescents: nutritional consequences. J Am Diet Assoc 1999;99:436–41.
21. DiMeglio D, Mattes R. Liquid versus solid carbohydrate: effects on food intake and body weight. Int J Obes Relat Metab Disord 2000;24:794–800.
22. Anderson GH. Sugars, sweetness and food intake. Am J Clin Nutr 1995;62(suppl):195S–202S.
23. Anderson GH, Catherine NLA, Woodend D, Wolever TMS. Inverse association between the effect of carbohydrates on blood glucose and subsequent short-term food intake in young men. Am J Clin Nutr 2002;76:1023–30
24. Black RM, Anderson GH. Sweeteners, food intake and selection. In: Fernstom JD, Miller GD, eds. Appetite and body weight regulation: sugar, fat and macronutrient substitutes. Boca Raton, FL: CRC Press, 1994:125–36.
25. Anderson GH, Saravis S, Schacher R, Zlotkin S, Leiter L. Aspartame: effect on lunch-time food intake, appetite and hedonic response in children. Appetite 1989;13:93–103.
26. Rogers PJ, Carlyle JA, Hill AJ, Blundell JE. Uncoupling sweet taste and calories: comparison of the effect of glucose and three intense sweeteners on hunger and food intake. Physiol Behav 1988;43:547–52.
27. Rogers PJ, Blundell JE. Separating the actions of sweetness and calories: the effects of saccharin and carbohydrate on hunger and food intake in humans. Physiol Behav 1989;45:1093–9.
28. Rodin J. Comparative effects of fructose, aspartame, glucose and water preloads on calorie and macronutrient intake. Am J Clin Nutr 1990;51:428–35.
29. Spitzer L, Rodin J. Effects of fructose and glucose preloads on subsequent food intake. Appetite 1987;8:135–45.
30. Rodin J, Reed D, Jamner L. Metabolic effects of fructose and glucose: implications for food intake. Am J Clin Nutr 1988;47:683–9.
31. Rodin J. Effects of pure sugar vs. mixed starch fructose loads on food intake. Appetite 1991;17:213–9.
32. Stewart SL, Black R, Wolever TMS, Anderson GH. The relationship between the glycaemic response to breakfast cereals and subjective appetite and food intake. Nutr Res 1997;17:1249–60.
33. Riby JE, Fujisawa T, Kretchmer N. Fructose absorption. Am J Clin Nutr 1993;58(suppl):748S–53S.
34. Read N, French S, Cunningham K. The role of the gut in regulating food intake in man. Nutr Rev 1994;52:1–10.
35. Lavin JH, Wittert GA, Andrews J. Interaction of insulin, glucagon-like peptide-1, gastric inhibitory polypeptide and appetite in response to intraduodenal carbohydrate. Am J Clin Nutr 1998;68:591–8.
36. Ravich WJ, Theodore MB, Thomas M. Fructose: incomplete intestinal absorption in humans. Gastroenterology 1983;84:26–9.
37. Roberts SB. High-glycemic index foods, hunger, and obesity: is there a connection? Nutr Rev 2000;58:163–9.
38. Wolever TMS, Jenkins JJA, Jenkins AL, Josse RG. The glycemic index: methodology and clinical implications. Am J Clin Nutr 1991;54:846–54.
39. Jenkins DJA, Wolever TMS, Taylor RH. Glycemic index of foods: a physiological basis for carbohydrate exchange. Am J Clin Nutr 1981;34:362–6.
40. Lee BM, Wolever TMS. Effects of glucose, sucrose and fructose on

plasma glucose and insulin responses in normal humans: comparison with white bread. Eur J Clin Nutr 1998;52:924–8.
41. Foster-Powell K, Brand Miller J. International tables of glycemic index. Am J Clin Nutr 1995;62(suppl):871S–93S.
42. Ludwig D. Dietary glycemic index and obesity. J Nutr 2000;130: 280S–3S.
43. Brand-Miller JC, Lobbezoo I. Replacing starch with sucrose in a high glycaemic index breakfast cereal lowers glycemia and insulin responses. Eur J Clin Nutr 1994;48:749–52.
44. Holt SHA, Brand-Miller JC, Petocz P, Farmakalidis E. A satiety index of common foods. Eur J Clin Nutr 1995;49:675–90.
45. Mayer J. Glucostatic mechanism of the regulation of food intake. N Engl J Med 1953;249:13–6.
46. Campfield LA, Smith FJ, Rosenbaum M, Hirsch J. Human eating: evidence for a physiological basis using a modified paradigm. Neurosci Biobehav Rev 1996;20:133–7.
47. Campfield LA, Smith FJ. Transient declines in blood glucose signal meal initiation. Int J Obes 1990;14:15–33.
48. Melanson KJ, Westerterp-Plantenga MS, Campfield LA, Saris WHM. Blood glucose and meal patterns in time-blinded males, after aspartame, carbohydrate, and fat consumption, in relation to sweetness perception. Br J Nutr 1999;82:437–46.
49. Chapman IM, Goble EA, Wittert GA, Morley JE, Horowitz M. Effect of intravenous glucose and euglycemic insulin infusions on short term appetite and food intake. Am J Physiol 1998;274:R596–603.
50. Gielkens HA, Verkijk M. Effects of hyperglycemic and hyperinsulinemia on satiety in humans. Metabolism 1998;47:321–4.
51. Reid M, Hammersley R. The effects of sucrose and maize oil on subsequent food intake and mood. Br J Nutr 1999;82:447–55.
52. Anderson GH. Hunger, appetite, and food intake. In: Ziegler EE, Filer LJ, eds. Present knowledge in nutrition. 7th ed. Washington, DC: ILSI Press, 1996:13–8.
53. Feinle C, Horowitz M. Carbohydrate and satiety. Nutr Rev 2002;60: 155–69.
54. Woo R, Kisseleff HR, Pi Sunyer FX. Elevated postprandial insulin levels do not induce satiety in normal weight humans. Am J Physiol 1984;247:R745–9.
55. Rodin J, Wack J, Ferrannini E, DeFronzo RA. Effect of insulin and glucose on feeding behavior. Metabolism 1985;34:826–31.
56. Bray GA. Afferent signals regulating food intake. Proc Nutr Soc 2000;59:373–84.
57. Gutzwiller JP, Goke B, Drewe J, et al. Glucagon-like peptide-1: a potent regulator of food intake in humans. Gut 1999;44: 81–6.
58. Flint A, Raben A, Astrup A, Holst JJ. Glucagon-like peptide 1 promotes satiety and suppresses energy intake in humans. J Clin Invest 1998;101:515–20.
59. Peters CT, Choi YH, Brubaker PL, Anderson GH. A glucagon-like peptide-1 receptor agonist and an antagonist modify macronutrient selection by rats. J Nutr 2001;131:2164–70.
60. Holst JJ. Glucagon-like peptide 1:a newly discovered gastrointestinal hormone. Gastroenterology 1994;107:1848–55.
61. Schvarcz E, Palmer M, Aman J, Horowitz M, Stridsberg M, Berne C. Physiological hyperglycemia slows gastric emptying in normal subjects and patients with insulin-dependent diabetes mellitus. Gastroenterology 1997;113:60–6.
62. Bergmann JF, Chassany O, Petit A, Triki R, Caulin C, Segrestaa JM. Correlation between echographic gastric emptying and appetite: influence of psyllium. Gut 1992;33:1042–3.

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