Sugars, energy metabolism, and body weight control

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ABSTRACT Obesity represents a major threat to health and quality of life. Although obesity has strong genetic determinants, it is generally accepted that it results from an imbalance between food intake and daily physical activity. Health guidelines have been focused on 3 particular lifestyle factors: increased levels of physical activity and reductions in the intakes of fat and sugars. The dietary guidelines, especially, are under debate. This review covers evidence from carefully controlled laboratory studies, clinical trials, studies in populations at high risk of developing obesity, and epidemiologic studies on the role of sugars, particularly sucrose, in the development of obesity. Although many environmental factors promote a positive energy balance, it is clear that the consumption of a low-carbohydrate, high-fat diet increases the likelihood of weight gain. The evidence related to carbohydrate, particularly sugars, and the type of food (solid or liquid) is less clear because the number of long-term ad libitum dietary intervention trials is very small. Data on sucrose intake in relation to metabolism and weight gain do not associate high consumption of sucrose with the prevalence of obesity. The evidence supports the current dietary guidelines for reducing fat intake. However, the effect of the carbohydrate source and class and of the form in which carbohydrate is consumed (solid or liquid) on body weight control requires further consideration. *Am J Clin Nutr* 2003;78(suppl):850S–7S.

KEY WORDS Sucrose, energy metabolism, body weight control, obesity, glycemic index, carbohydrate, diet

INTRODUCTION

The prevalence of overweight and obesity has risen dramatically over the past 3 decades and is threatening to become a global epidemic (1). A substantial proportion of the population is at increased risk of morbidity and mortality as a result of increased body weight. In affluent countries, excess body fat accounts for ≈30–40% of coronary heart disease (2); cancers of the colon, breast, and endometrium; and most cases of type 2 diabetes (3). Genetic susceptibility predisposes people to the development of body fatness but cannot account for the exponential increase in obesity in nearly all Western countries. Obesity is generally accepted as resulting from an imbalance between food intake and daily physical activity. Obesity is thus the largest nutrition-related problem in the developed world. Despite the overwhelming amount of research and statistical analysis, no clear explanation can be given for the relation between changes in behavior and the rapid increase in obesity prevalence in the past 3 decades.

Health guidelines have been focused on 3 particular lifestyle factors: increased levels of daily physical activity and reductions in the intakes of fat and sugars, particularly added sugars. The urgency of taking public action regarding physical activity is generally accepted, but there is much debate about dietary factors, such as total fat intake, intake of sugars, and intake of rapidly digested carbohydrates. In the 1970s, some nutritionists considered sucrose, particularly added sucrose, as perhaps the most important dietary factor predisposing to weight gain (4). Since then, attention has shifted toward fat as the major nutritional component promoting excess energy intake and weight gain (5, 6). Evidence that the regulation of fat balance has a lower priority than the regulation of the intakes of carbohydrates, protein, and alcohol has contributed to the general knowledge that fat intake increases the risk of excess energy intake and the promotion of fat storage (7). Furthermore, data from national food surveys indicate a pronounced shift in the fat-carbohydrate ratio toward a fattier diet (8).

Despite the controversy about the particular role of sugars, the message that fat in the diet is responsible for excess energy intake and weight gain became stronger. As a consequence of recommendations to reduce fat intake, the market for low-fat food expanded rapidly in the 1990s (9). The actual intake of fat expressed as a percentage of energy, based on the subject's self-recordings, has decreased significantly over the past decade (10). The reduction in absolute fat intake was substantially less. Although a number of meta-analyses on the relation between ad libitum low-fat diets and body weight control showed that dietary fat intake is directly associated with obesity (11, 12), the scientific evidence for the relation between dietary fat content and the prevalence of obesity has also been challenged. For example, Katan et al (13) questioned the importance of low-fat, high-carbohydrate diets in the prevention and treatment of obesity. Reduction of fat intake resulted in a reduction of only a few kilograms of body weight.

Another important argument concerns the so-called fat paradox (14). With the increasing popularity of low-fat products, food intake statistics have shown a decrease in dietary fat intake,

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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).

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Am J Clin Nutr 2003;78(suppl):850S–7S. Printed in USA.

although the prevalence of obesity is rising. A direct relation between dietary fat and energy density was also questioned because of the observation that many currently available low-fat foods are based on sugars, and thus they have energy density values similar to those of their high-fat counterparts (14). This has renewed interest in sugars as the primary nutritional factor behind the increase in obesity. Many refined carbohydrate foods produce a high glycemic response, thereby promoting postprandial carbohydrate oxidation at the expense of fat oxidation and thus altering fuel partitioning in a way that may be conductive to body fat gain (15). This is in contrast to foods that produce a low glycemic response and lower postprandial insulin secretion.

This review examines the role of sugars, particularly sucrose, as dietary factors in relation to body weight control and compares the role of sugars with that of the other important dietary factor, fat.

MACRONUTRIENTS AND ENERGY BALANCE

An important issue to address first is whether the source of energy in the diet is important in relation to energy balance. Studies on diet-induced thermogenesis show that less energy is dissipated as heat after the digestion of fat (≈7%) than after that of carbohydrate (≈12%) and protein (≈22%). A study on the effects of different carbohydrates on postprandial thermogenesis over 6 h showed a lower level after the consumption of glucose (8%) and starch (8.2%) than after that of fructose (11.1%) and sucrose (11.4%) (16). These data are somewhat lower than earlier data of Macdonald (17), which ranged from 10% (glucose) to 17% (sucrose or fructose and glucose). Alcohol is of particular interest. Because alcohol cannot be stored in the body, it must be metabolized preferentially. However, the energy dissipated as heat is more or less similar to that from carbohydrate (14%) (18).

Alterations in the dietary macronutrient levels will change the mixture of metabolic fuels oxidized, although the effects on 24-h energy expenditure (EE) are small as long as the exchange in the diet is isoenergetic. Even when the carbohydrate content of the diet was varied from 9% to 79% with reciprocal changes in fat intake, 24-h EE measured by whole-body calorimetry was similar (19). Under conditions of overfeeding (50%), isoenergetic exchange did not make a substantial difference in 24-h EE when the excess was given as carbohydrate (≈215 kcal/d) or as fat (≈96 kcal/d) (20).

It is now widely accepted that macronutrients compete with each other in an oxidative hierarchy, with alcohol given first priority and fat the lowest. This is important, given the accumulating evidence that energy balance can be achieved only through macronutrient balance. The ability to achieve protein balance over a wide range of intakes is well documented in humans. Thus, the regulation of body weight is not primarily dependent on protein balance. For the maintenance of carbohydrate balance, one should know whether de novo synthesis of fatty acids from glucose is an active pathway. If carbohydrate could be converted to fat to a large extent, the concept of energy balance based on carbohydrate and lipid balance would be of little value.

DIETARY CARBOHYDRATES AND DE NOVO LIPOGENESIS

Because only a small reserve of 300–500 g carbohydrates can be stored as glycogen in the human body, any excess must be oxidized or converted to fat by de novo hepatic lipogenesis (21). Although this biochemical pathway is extensively used in animals, such as rats fed a low-fat food pellet diet, most experimental data in humans do not confirm this pathway as important for storage of energy. After a 480-g oral load of carbohydrate, fat synthesis did not exceed fat oxidation in young adult men (22). Isotope studies confirmed the absence of a quantitatively significant synthesis through de novo hepatic lipogenesis under most conditions of excess energy intake coming from carbohydrates (23). Recently, a combination of whole-body indirect calorimetry and isotope measurement of de novo hepatic lipogenesis showed that de novo hepatic lipogenesis of 3–8 g/d was stimulated by 4 d of excess 50% carbohydrate energy intake (24). This total de novo lipogenesis represents a small fraction of both the surplus carbohydrates ingested (360–390 g) and the total fat stored (60–75 g) in the body. Thus, the addition of excess carbohydrate in a mixed diet, leading to excess energy intake, results in the accumulation of body fat, but not by the conversion of carbohydrate to fat. The higher priority of carbohydrate over fat to be oxidized results in a suppression of dietary fat oxidation, which leads to fat storage. Although the de novo synthesis is relatively small compared with fat storage, the long-term metabolic consequences could be important. Further studies are required to provide a better understanding of this phenomenon. It is interesting that, in this well-designed study, a surplus of carbohydrate in the form of glucose or sucrose did not result in differences in de novo lipogenesis or fat storage. Also, no differences were found between the lean and the obese women.

In an acute study looking at the effect of a high-sucrose or high-starch meal in fuel selection, Daly et al (25) found that fructose is preferentially oxidized over glucose after a high-sucrose meal and that glucose is oxidized more slowly after a high-sucrose meal than after a high-starch meal.

So far, the indications for a substantial contribution of excess carbohydrate intake to de novo lipogenesis are weak, but some observations, as summarized by Hellerstein (26), do not exclude the possibility of a potential role. Carbohydrate intake massively exceeded 24-h EE in the Luru Walla overfeeding tradition in Cameroon, where adolescent boys gained 12 kg body fat over 10 wk while eating > 1750 g carbohydrate and only 60 g fat daily. This increase in fat showed the capacity of the human body to convert carbohydrate into fat in considerable amounts, but this was limited to extreme conditions. Direct measurement of VLDL-triacylglycerol production rates under conditions of overfeeding could reveal even higher levels of de novo lipogenesis. The role of de novo adipose lipogenesis cannot be excluded as important in triacylglycerol production from carbohydrate, as indicated in a glucose infusion study at rates substantially above 24-h EE for 4–7 d (27). Definitive answers await more appropriate methods for studying de novo synthesis in adipose tissue.

In contrast, fat oxidation is hardly affected at different levels of fat intake (28, 29). Under isoenergetic conditions, acute changes from a diet with 30% of energy from fat to a diet with 50% of energy from fat (decrease in food quotient) resulted in a slow increase in fat oxidation as indicated by a decrease in respiratory quotient over a 6-d period before a new fat balance was reached (30; **Figure 1**). In a follow-up study, lean subjects were able to rapidly (within 24 h) adjust fat oxidation to fat intake when glycogen stores were lowered by exhaustive exercise (31; **Figure 2**). A similar result was found in a group of obese subjects after a switch from a reduced-fat, high-carbohydrate diet to a high-fat, low-carbohydrate diet with or without initial glycogen-depletion exercise (32).

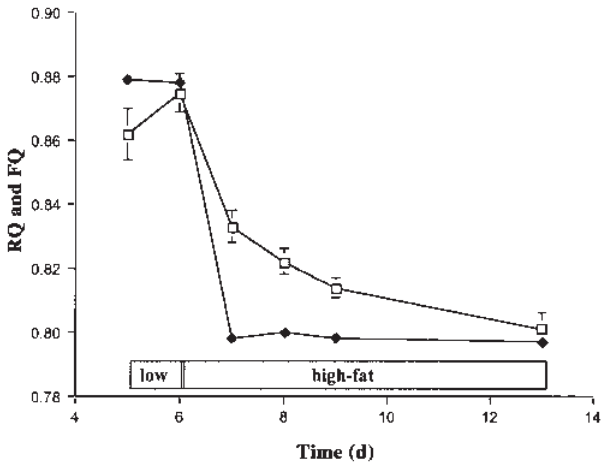

FIGURE 1. Mean (±SEM) 24-h respiratory quotient (RQ; □) and 24-h food quotient (FQ; ◆) as measured in a whole-body indirect calorimeter for days 5–9 and 13 in 12 lean men and women (BMI: 21.4) on a low-fat (30% of energy from fat), high-carbohydrate (55% of energy from carbohydrate) diet and a high-fat (60% of energy from fat), low-carbohydrate (25% of energy from carbohydrate) diet. Adapted from reference 30.

These results emphasize the importance of a high energy turnover by exercise to initiate lipolysis from fat stores. Of interest is the observation that, with increasing total EE, as can be observed in athletes, extra energy intake is selected exclusively from carbohydrates, with a preference for sugars (33).

GLUCOSE AND INSULIN RESPONSE

Carbohydrate ingestion raises blood glucose and insulin. Readily digestible carbohydrates give higher postprandial blood glucose and insulin concentrations than do less digestible carbohydrates. The glycemic index (GI) of foods based in this concept has been linked to weight gain (15). Many sugars-containing foods produce a high glycemic response, promoting postprandial carbohydrate oxidation at the expense of fat oxidation that may be conductive to body fat gain. Rapid changes in blood glucose concentration, in particular the so-called dynamic declines, were associated with spontaneous meal request in time-blinded subjects (34). High-GI foods were suggested to be less satiating than an isoenergetic portion of low-GI foods (15). In the 1950s, Mayer (35) proposed the glucostatic theory linking increased plasma glucose concentration to increased satiety; the meal request after a transient decline in blood glucose supports this theory. More recent studies support the role of glucose-induced plasma insulin concentrations as an important central satiety signal (36). Several studies examined the short-term effect of postprandial glucose concentration on satiety and food intake by using different foods and experimental approaches, but these differences make interpreting the results difficult. Anderson (37) did an extensive review on this topic and concluded that high-GI carbohydrates suppress short-term (1-h) intake more effectively than do low-GI carbohydrates, but the reverse occurs over the longer term. These short-term studies are less informative about the role of carbohydrates in long-term body weight control. A short-term increase or decrease in food intake can be compensated over days or weeks. Unfortunately, long-term intervention studies with diets containing different types of carbohydrates are few. Moreover, long-term dietary studies using the high-GI or low-GI of individual food products as intervention criteria do not always fit with the chemical classification of sugars or oligoaccharides and starches. The GI of sucrose is intermediate at 65 with glucose as standard (38). The GIs for monosaccharides such as glucose, fructose, and lactose are 97, 23, and 46, respectively. Jenkins et al (39) found no relation between sucrose content and GI in 62 foods, and Brand-Miller (40) had the same result in 44 sweetened and unsweetened products. Recently, Pi-Sunyer (41) reviewed the validity of GI in relation to the obesity problem and concluded that the concept, particularly with regard to calculated glycemic load as indicator for a total diet approach, is far from clear.

Many factors affect GI reproducibility, such as the physical form, processing, and preparation of the food and the combination of foods in a meal. The calculation of an average dietary GI or glycemic load from food intake measurements can be seriously questioned because of the uncertainties of both food intake and GI measurements (42).

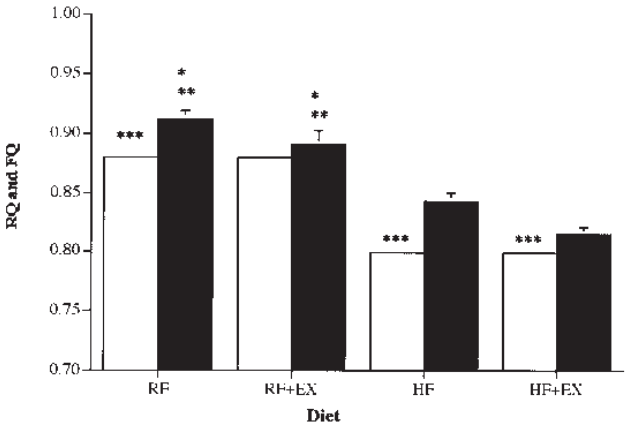

FIGURE 2. Mean (±SEM) 24-h respiratory quotient (RQ; ■) and 24-h food quotient (FQ; □) as measured in a whole-body indirect calorimeter at day 4 with a reduced-fat (RF) diet (30% of energy from fat) or high-fat (HF) diet (60% of energy from fat) with or without glycogen-depleted exercise (EX) in the evening of day 3 in 7 lean subjects (paired *t* test): $^{*}P < 0.05$ compared with HF+EX, $^{**}P < 0.05$ compared with HF, $^{***}P < 0.05$ compared with RQ. Adapted from reference 31.

FUEL PARTITIONING AND CARBOHYDRATE TYPE

The second important argument as to why high-GI food could facilitate weight gain is the alteration in fuel partitioning (15). Increased levels of glucose and insulin will inhibit lipolysis, reduce the amounts of circulating free fatty acids (FFAs), and consequently reduce fat oxidation. The reduced capacity of obese persons to mobilize and subsequently oxidize fat has been mentioned as a consequence of long-term hyperinsulinemia, but whether a direct link exists is questionable. Hyperglycemia and hyperinsulinemia are often accompanied by an increased circulation of FFAs, even though fat oxidation is diminished (43). Postprandial increases in glucose will indeed reduce blood FFAs and fat oxidation in the short term. The question that remains in relation to body weight control is whether these physiologic effects persist or whether a compensation occurs over 24 h.

The well-controlled study by Kiens and Richter (44), who provided high- or low-GI foods to lean volunteers ad libitum for 30 d

in a crossover design, could not find any differences in body weight between the 2 interventions. Sucrose was a considerable part of the high-GI diet. Nevertheless, significant differences in insulin profile during the day were observed between the 2 diets on days 3 and 30. An earlier study with healthy lean volunteers showed no significant effect on body weight over 2 wk (45). These daily excursions of glucose, insulin, FFAs, and substrate oxidation may be compensated during the night. This phenomenon was also seen in an intervention study with high- and low-intensity exercise at equal EE levels in moderately obese men kept for 36 h in a respiration chamber (46). Differences in fat and carbohydrate oxidation during the day (higher carbohydrate oxidation with high-intensity exercise) were completely compensated during the night. Long-term, well-controlled, randomized human intervention trials are needed before we really can confirm that high-GI diets or diets high in sucrose will shift metabolism to fat storage.

AD LIBITUM INTERVENTION TRIALS OF SUGARS, HIGH-GI FOOD, AND BODY WEIGHT CONTROL

Many animal studies have shown an overeating effect with glucose, sucrose, and high-GI diets (15, 47). As was shown decades ago with the cafeteria-food diets, palatability is a major determinant of feeding behavior in animals (48). This is of course true in humans also, but the availability of tasty food in the supermarket or elsewhere is not specifically restricted to high-fat and high-sucrose products. Most products with a broad variety of macronutrients are very palatable. Therefore, the outcome of animal ad libitum studies of the role of sucrose in the diet is not very indicative of the role of sucrose in the human diet.

Medium-term (≤3 mo) and long-term (≥6 mo) human intervention studies of the effect of the type of carbohydrate on body weight are very few. Because body weight changes are mostly related to differences in energy intake, one should study the relation between type of carbohydrate and body weight when subjects have free access to food. Studies comparing different diets under energy restriction or isoenergetic conditions are less valuable for providing information about the effects on body weight regulation than are overfeeding or ad libitum studies.

Raben et al (49) compared the effect of a high-sucrose diet, a high-starch diet, and a high-fat diet on 14-d ad libitum energy intake, body weight, and EE in normal-weight and formerly obese women. On average, energy intake was 13% and 12% lower with the starch diet than with the sucrose and fat diets, respectively. In both formerly obese and normal-weight subjects, body weight and fatness decreased significantly with the starch diet. No changes were observed during the fat and sucrose diets. After 14 d of the sucrose diet, 24-h EE was significantly greater than that with the starch and fat diets. The authors mentioned 3 reasons for the low energy intake with the high-starch diet: an increased satiating power because of the high fiber content and volume and less palatability than the sucrose and fat diets had. The higher energy intake with the sucrose diet was explained by the large amount of sucrose-containing drinks in this diet. Fluids in general are less efficient in increasing satiety and suppressing food intake than are solid foods (50). In a crossover study, subjects consumed dietary carbohydrate loads of 450 kcal/d as a liquid (soda) or solid (jelly beans) for 4 wk (51). Free-feeding energy intake during the solid period was significantly lower than the intake before this period compensating for the extra energy intake. No decrease in free-feeding energy intake occurred during the liquid period.

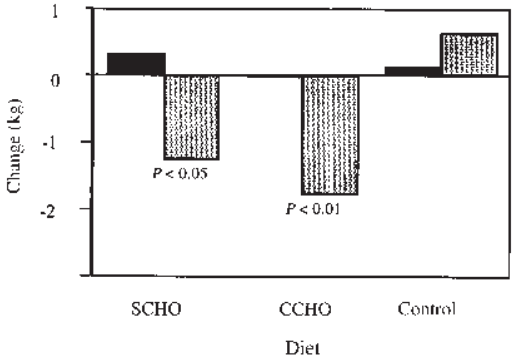

FIGURE 3. Changes (kg) in fat-free mass (■) and fat mass (■) during a 6-mo randomized controlled intervention trial with 398 moderately obese adults on a low-fat, high-sugars (SCHO) diet; a low-fat, high-oligosaccharide and high-starch (CCHO) diet; or a normal-fat, normal-carbohydrate (Control) diet (ANOVA with Bonferroni adjustment); SCHO and CCHO diets significantly different from control diet. Adapted from reference 54.

Consequently, body weight increased significantly (+0.5 kg) only during the liquid period.

This physiologic difference between solid and liquid food is one of the basic principles on which carbohydrate sports drinks were developed to provide maximal energy to the muscles of athletes. This weight-gaining effect of carbohydrate drinks was recently confirmed in a randomized 10-wk study in overweight subjects who used dietary supplements containing sucrose or artificial sweeteners (52). About 70% of the sucrose (28% of energy) was provided as drinks. Body weight increased in the sucrose group by 1.6 kg, whereas it decreased by 1.0 kg in the artificial sweetener group. This is in line with prospective observational data on the risk of weight gain in children and the use of sugar-sweetened drinks (53). For each additional serving of sugar-sweetened drink, the relative risk of obesity after 19 mo of follow-up, corrected for other dietary variables such as the percentage of energy as fat at baseline, physical activity, and television viewing, was a significant 1.44. The baseline consumption of sugar-sweetened drinks was independently associated with the change in body mass index for each daily serving. These studies indicate that the form of carbohydrate intake—ie, liquid or solid—may lead to extra energy intake before adequate feedback is received from satiety signals.

The only large-scale, long-term, randomized control trial on the role of sugars compared with oligosaccharides and starches is the Carbohydrate Ratio Management in European National diets (CARMEN) multicenter trial, which involved 398 moderately overweight subjects in 5 countries (54). This study investigated the effect on energy intake, body weight, and blood lipids of 6 mo of ad libitum intake of low-fat diets (reduction of ≈10% of energy from fat) rich in either sugars or oligosaccharides and starches. Commercially available food products or low-fat, high-sugars or high-oligosaccharide, and high-starch alternatives were provided by a validated laboratory "shop" system to increase compliance and food intake control. The results showed that both of the low-fat, high-carbohydrate diets reduced body weight significantly—by 1.6 kg (for high sugars) and 2.4 kg (for high polysaccharides)—compared with a control normal-fat, normal-carbohydrate diet. Mean (±SD) body fat loss over the 6 mo in the 2 low-fat groups was 1.3 ± 3.6 and 1.8 ± 3.9 kg, respectively, compared with a body fat gain of 0.6 ± 4.3 kg in the control group (**Figure 3**). The slightly lower weight loss and body fat loss in the sugars group

than in the high polysaccharides and starch group was not significantly different. Furthermore, no detrimental effects on blood lipids were observed during the consumption of both carbohydrate diets. The energy density of both carbohydrate diets was significantly reduced (by 0.10 and 0.18 kcal/g, respectively), although a large number of the low-fat alternatives contained higher levels of sugars, particularly sucrose.

The findings from the CARMEN study underline the importance of public measures to reduce fat intake. A decrease in body weight of 2–3 kg by means of a general reduction in fat intake of ≈10% of energy in the general population could reduce the prevalence of obesity from 25% to 15% (12). However, we need confirmation of these results in large-scale, randomized, controlled intervention trials using different types of carbohydrates.

SUCROSE AND WEIGHT-REDUCTION DIETARY INTERVENTION TRIALS

A few studies have been published on the effect on weight loss in an energy-restriction protocol that uses the type of carbohydrate as an intervention tool. Unfortunately, the number of studies using sucrose specifically as the carbohydrate source for the intervention is very small. Body weight loss was similar in both groups in a study with 95 moderately obese subjects randomly assigned to a −600 kcal/d energy-restricted diet with a low-fat, low-sucrose (< 5% of energy) or low-fat, high-sucrose (10% of energy) composition for 8 wk (2.2 and 3.0 kg, respectively) (55). In a similar type of weight-loss study for 6 wk with a relatively high-sucrose, energy-restricted group (43% of energy as sucrose) compared with a low-sucrose, energy-restricted group (4% of energy as sucrose), Surwit et al (56) found no differences in weight loss (7.0 and 7.4 kg, respectively). A number of other studies have been published comparing high-GI and low-GI foods in an energy-restricted diet. Slabbers et al (57) studied obese hyperinsulinemic females following an energy-restricted diet (1000–1200 kcal, 50% of energy from carbohydrate) with low-GI foods and the exclusion of high-GI products (Lo GI) or a conventionally balanced diet (Control) for 3 mo followed by a 3-mo washout period. Thereafter, about half of the subjects started with the alternative diet called "crossover" for 3 mo. In the parallel study, weight loss was not significantly different in the Lo GI and Control groups ($\bar{x} \pm$ SD: 7.4 ± 4.2 and 9.4 ± 2.5 kg, respectively). During the follow-up study, the Lo GI group had a significantly greater weight loss (7.4 compared with 4.5 kg; **Figure 4**). The reduction in fasting insulin concentrations was significantly greater in the Lo GI group during the parallel trial (−21 pmol/L) than in the Control group (−91 pmol/L).

In a randomized controlled trial with obese subjects with type 2 diabetes, Wolever et al (58) found no differences in weight loss with a low- or high-GI energy-restricted diet for 6 wk. A randomized crossover study compared the effects of a high- and low-GI, energy-restricted diet (−50% of predicted EE) in 10 overweight young men for 9 d (59). Unfortunately, high- and low-GI diets differed too much in macronutrient composition (protein:fat:carbohydrate for the high-GI diet: 15:18:67% of energy; for the low-GI diet: 27:30:43% of energy) to allow comparison of the type of carbohydrate. Low GI is confounded with a low carbohydrate intake in this study. Weight loss was 3.2 and 3.6 kg (NS), respectively, in the high- and low-GI diets. Serum leptin decreased to a lesser extent during the high-GI diet, whereas resting EE declined less (50%) in the low-GI diet. This thermogenic effect is most probably related to the high protein intake in the low-GI group.

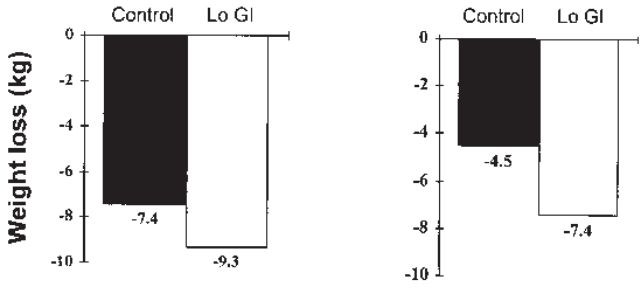

FIGURE 4. Weight loss in obese hyperinsulinemic women (n = 30) during a parallel (12-wk) between-groups (unpaired t test, NS) study (left) followed by a crossover (12-wk) (ANOVA, P = 0.04) study (right) of subjects (n = 16) randomly assigned to consume a conventional balanced diet (Control) or an energy-restricted diet with low-glycemic-index foods (Lo GI). Adapted from reference 57.

The same research group conducted another study in overweight children (60). Two weight-loss diets were tested, one in accordance with the US dietary recommendations (En%: 15–20% as protein, 25–30% as fat, and 55–60% as carbohydrate). The other diet was a nonenergy-restricted diet with a low GI but also low carbohydrate content (20–25% of energy as protein, 30–35% as fat, and 45–50% as carbohydrate). Weight loss was 1.2 kg in the low-GI group, whereas the recommended-diet group gained 1.4 kg. The results in this study regarding the type of carbohydrate are also difficult to interpret because a number of dietary factors apart from carbohydrate type, including the amount of carbohydrate, were different in the 2 groups. Lindroos et al (61) followed 409 gastroplasty patients for 2 y and found that patients who continued to select sweet foods appear to maintain lower energy intake and lose more weight.

These weight-loss studies with different types and amounts of carbohydrates including high and low sucrose do not indicate that weight loss is impaired by high-sucrose, energy-restricted diets.

EPIDEMIOLOGIC FINDINGS ON THE RELATION BETWEEN SUGAR INTAKE AND OBESITY

A substantial number of epidemiologic studies found a clear inverse relation between sucrose intake and body weight or body mass index, as well as sucrose intake and total fat intake. Most reports were reviewed in detail by Hill and Prentice (47) and Astrup and Raben (62), and the suggestion is that a high intake of sucrose may help to prevent weight gain. However, one should be very cautious with the interpretation of this type of data because of the enormous bias in the food intake records of overweight and obese people.

Few data are available about how much carbohydrate is ingested as a solid or a drink. This is important for many reasons. First, data are needed to validate the hypothesis that carbohydrates from fluids may promote excess energy intake and, consequently, weight gain. Second, but related, is the fact of the increase in the soft drink market in relation to the increase in prevalence of obesity. Third is the suggestion that the switch, particularly in the US beverage market in the 1980s, from sucrose to high-fructose corn

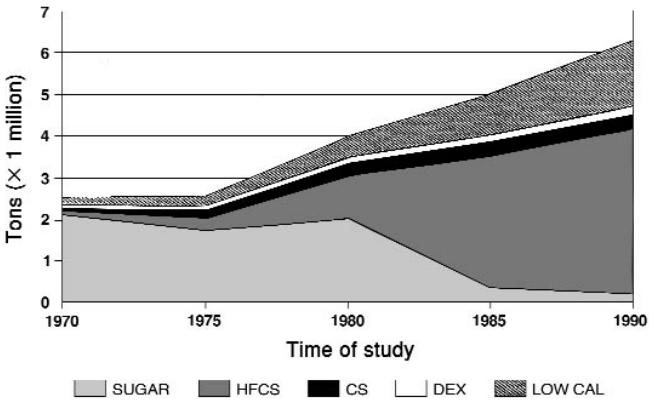

FIGURE 5. Use of sweeteners (million tons) in beverages in the United States over a 20-y period. HFCS, high-fructose corn syrup (predominantly with a fructose-to-glucose ratio of 55:45); CS, corn syrup (100% glucose); DEX, dextrose (glucose polymer); LOW CAL, artificial sweeteners. Adapted from reference 63.

syrup as the sweet carbohydrate source, is an explanation for the beginning of the exponential growth of obesity prevalence in the United States (**Figure 5**). Figure 5 is based on food availability statistics that must be interpreted with caution. Nevertheless, the shift in ingredient use is remarkable if the fact that it refers to only one food group is taken into account.

The fructose content of high-fructose corn syrup used in the beverage industry is only slightly (5%) higher than that of sucrose (64). With increased use of high-fructose corn syrup compared with sucrose, no large changes in metabolic response are expected; however, human data required to exclude this change in ingredient as a potential factor in the exponential increase in the prevalence of obesity in the 1980s in the United States are lacking. Other important changes, such as the introduction of the personal computer and the television remote control, also occurred in the same period. All could have contributed to an increased risk for weight gain.

FUTURE RESEARCH DIRECTIONS RELATED TO SUGARS AND BODY WEIGHT CONTROL

Although the relation between sucrose intake and body weight has been studied extensively in experimental and epidemiologic studies, unresolved issues remain as to the role of sugars compared with other dietary components in the prevention of weight gain:

- We need information from ad libitum, randomized, controlled, long-term dietary intervention studies on the effect of different types of carbohydrates on body weight control. Special attention should be given to the potential confounding effects of other macronutrient changes as well as the type of foods (fluid or solid).
- Most studies on sugars are related to the short-term (a few hours) effects on physiologic functions such as satiety and thermogenesis. Data are available for 2 wk or longer on the effects on body weight because, with the existing body- composition methods, we are unable to detect the small changes in body weight and body fat that occur over shorter periods. Few data are available in physiologic profiles for periods between 12 and 60 h. Information on compensatory effects over 24 or 48 h is of interest for predicting long-term weight control.
- A number of caloric sweeteners besides sucrose are used on a large scale in the food industry. However, no physiologic data, eg, on thermogenic effect, satiety, or blood glucose and insulin, are available.
- As stated in the introduction, there is general consensus about the need to increase the level of physical activity in the population. However, less is known about the interaction with food intake, in particular the ratio of carbohydrate to fat and the type of carbohydrate. The role of sugars in this interaction in relation to body weight control is of interest.

The increased prevalence of obesity is the result of the changes in several behaviorial and environmental factors over time. Large-scale computer modeling with input at a macro level, such as food availability and composition data and the use of cars and personal computers, as well as with input at a micro level, such as physiologic response on dietary and exercise intervention, could give us more information about the role of the different factors and their effect on measures to prevent weight gain.

CONCLUSION

Energy from different sources may be considered relatively equal as long as there is energy balance. The main differences between macronutrients are mostly mediated through the regulation of food intake. An overwhelming amount of evidence shows that the ratio of fat to carbohydrate in the diet is the primary factor in the macronutrient composition of the diet that easily causes passive overconsumption of energy and thus leads to weight gain. In contrast, high- carbohydrate diets seem relatively benign, regardless of the type of carbohydrate. There is little evidence that sugars have direct negative effects on body weight control. However, evaluating the different types of carbohydrates as part of a high-GI or low-GI diet gives conflicting results. Long-term studies are needed to delineate the independent effects of dietary sugars and glycemic load on body weight control. In particular, the frequent use of carbohydrate-sweetened beverages could play an important role in an increased postprandial insulin response leading to a reduction in lipolysis and fat oxidation. The combination of the frequent use of carbohydrate-sweetened beverages and an increasingly inactive lifestyle, which reduces the metabolic demand for fat as a fuel, considerably increases the risk of weight gain.

The evidence supports current dietary guidelines to reduce fat intake. However, the effects of carbohydrate source and class and of the form in which carbohydrate is consumed (solid or liquid) on body weight control require further consideration.

The author had no conflicts of interest.

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