Glycemic index and obesity[1–4]

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ABSTRACT Although weight loss can be achieved by any means of energy restriction, current dietary guidelines have not prevented weight regain or population-level increases in obesity and overweight. Many high-carbohydrate, low-fat diets may be counterproductive to weight control because they markedly increase postprandial hyperglycemia and hyperinsulinemia. Many high-carbohydrate foods common to Western diets produce a high glycemic response [high-glycemic-index (GI) foods], promoting postprandial carbohydrate oxidation at the expense of fat oxidation, thus altering fuel partitioning in a way that may be conducive to body fat gain. In contrast, diets based on low-fat foods that produce a low glycemic response (low-GI foods) may enhance weight control because they promote satiety, minimize postprandial insulin secretion, and maintain insulin sensitivity. This hypothesis is supported by several intervention studies in humans in which energy-restricted diets based on low-GI foods produced greater weight loss than did equivalent diets based on high-GI foods. Long-term studies in animal models have also shown that diets based on high-GI starches promote weight gain, visceral adiposity, and higher concentrations of lipogenic enzymes than do isoenergetic, macronutrient-controlled, low-GI-starch diets. In a study of healthy pregnant women, a high-GI diet was associated with greater weight at term than was a nutrient-balanced, low-GI diet. In a study of diet and complications of type 1 diabetes, the GI of the overall diet was an independent predictor of waist circumference in men. These findings provide the scientific rationale to justify randomized, controlled, multicenter intervention studies comparing the effects of conventional and low-GI diets on weight control. *Am J Clin Nutr* 2002;76(suppl):281S–5S.

KEY WORDS Glycemic index, obesity, overweight, insulin, glucose responses, carbohydrate

INTRODUCTION

Rates of obesity and overweight continue to increase in most Western countries despite the efforts of governments and health care providers to prevent and reduce this trend. In the United States, Europe, and Australia, >40% of adults are now overweight or obese, a rate almost double that of the early 1980s (1–3). The underlying reasons for this global pandemic are complex. Whereas genetic susceptibility plays a part, changes in the genetic makeup of the population cannot explain the dramatic rise in obesity rates over the past 10–15 y. Reduced physical activity; abundant, easily available, and affordable energy-dense, highly palatable foods; and social and economic influences are all likely contributors to the rising prevalence of overweight and obesity. In this report, we further suggest that current dietary recommendations to increase the consumption of carbohydrate-dense foods are counterproductive to weight control. Our hypothesis is that a high-carbohydrate diet based on foods that promote a high glycemic response [ie, high-glycemic-index (GI) foods] alters appetite and energy partitioning in a way that is conducive to body fat gain.

Reducing fat intake has been the primary focus of dietary prevention and treatment of overweight and obesity for >20 y. The most concentrated source of energy, fat, is efficiently stored as body fat. Furthermore, high-fat foods are relatively less satiating than are isoenergetic portions of high-carbohydrate or high-protein foods (4, 5). Intervention studies have shown that ad libitum, high-carbohydrate diets facilitate greater weight loss than do high-fat diets (6). Many epidemiologic studies have shown that relatively high dietary fat intakes correspond with increased obesity rates (7).

Whereas clinical studies have shown that a reduction in fat intake can produce clinically significant weight loss in overweight persons, the results are typically modest (6). A reduction of 10–20% of energy intake from fat produces, on average, weight losses of 5–7 kg in obese persons, but weight regain often occurs.

RATIONALE FOR HIGH-CARBOHYDRATE, LOW-GI DIETS

Adherence to standard dietary advice to reduce fat intake while increasing carbohydrate intake generally increases the glycemic effect of the diet. Both the quantity and quality of a carbohydrate influence postprandial glycemia, and the interaction between the 2 may be synergistic. A typical Western, high-carbohydrate diet based on high-GI foods such as potatoes, breads, and low-fat cereal products is digested and absorbed

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rapidly, resulting in a high glycemic load and increased demand for insulin secretion (8, 9). In insulin-resistant persons who consume high-GI foods, postprandial hyperglycemia and insulinemia are magnified, possibly contributing to β cell exhaustion and the development of type 2 diabetes (10, 11). On the other hand, low-GI, high-carbohydrate foods may maintain insulin sensitivity and increase the weight-loss potential of ad libitum, low-fat diets (12).

Low-GI foods may benefit weight control in 2 ways: *1*) by promoting satiety and *2*) by promoting fat oxidation at the expense of carbohydrate oxidation. These 2 qualities of low-GI foods stem from the slower rates at which they are digested and absorbed and the corresponding effects on postprandial glycemia and hyperinsulinemia. Even when appearance and nutrient content are matched, low-GI foods typically induce higher satiety than do their high-GI counterparts and are followed by less energy intake at subsequent meals (12). Corresponding with the progressive refinement of carbohydrate-rich foods, such as apples or wheat grains, is a step-by-step increase in the food's GI rating and, with it, a reciprocal decrease in satiety (13). Similarly, mixed meals with low GIs were found to induce greater cholecystokinin secretion and greater satiety over a 180-min period (14). These differences are likely to be clinically important. A 50% increase in a meal's GI (eg, from 50 to 75) resulted in a 50% decrease in satiety. Overall, 16 of 17 studies confirmed that low-GI meals increase fullness to a greater extent than do comparable high-GI meals (12).

To many, it may appear paradoxical that a food producing a low blood glucose response should be more satiating than an isoenergetic portion of food producing a high blood glucose response. However, because the former is characterized by slower rates of digestion and absorption in the small intestine, nutrient receptors in the gastrointestinal tract are stimulated for a longer period of time, resulting in prolonged feedback (through signals such as cholecystokinin and glucagon-like peptide-1) to the satiety center in the brain (15). Additional mechanisms may also account for the differences in the satiating effects of high- and low-GI foods. After the consumption of a high-GI meal, insulin concentrations rise dramatically, leading directly to a rapid reduction in both glucose and fatty acid concentrations, often below fasting concentrations. Hence, between 3 and 5 h postconsumption, concentrations of 2 major metabolic fuels circulating in the blood are low simultaneously, a situation that could be interpreted by the central nervous system as "low fuel status" (12). Spontaneous requests for meals by time-blinded human subjects were found to correspond with transient declines in blood glucose concentrations during the postabsorptive state (16). Meal requests were also associated with so-called dynamic declines in blood glucose concentrations immediately after the peak blood glucose concentration induced by carbohydrate consumption. Thus, the marked hyperglycemic and hypoglycemic effects of high-GI foods could partly explain the lower satiety observed in the postprandial period.

Differences in GI also dictate differences in fuel partitioning and oxidation. Postprandial rises in glucose and insulin concentrations increase carbohydrate oxidation acutely through the rapid activation of key rate-limiting enzymes. For example, malonyl-CoA, an intermediate of glucose oxidation, strongly inhibits fatty acid transport into mitochondria, resulting in decreased fatty acid oxidation (17). Longer exposure to chronic hyperglycemia and hyperinsulinemia results in decreased expression of the rate-limiting enzymes and alters the potential for fat oxidation. Reduced capacity to oxidize fatty acids is present in some obese human subjects (18) and obesity-prone rats (19). Reduced rates of fat oxidation were linked with greater weight gain in several prospective studies (20, 21). Whether high-GI diets, which induce chronic hyperglycemia and hyperinsulinemia, can reduce the body's capacity to oxidize fat and significantly increase body fat storage is still questionable, although some evidence supports the hypothesis.

DIETS BASED ON HIGH-GI CARBOHYDRATES ENHANCE FAT STORAGE AND WEIGHT GAIN

Acute short- and long-term studies in humans and animals provide evidence that a high-GI diet affects appetite and nutrient partitioning in a way that promotes body fat storage.

Acute studies

Obese teenage boys who consumed fixed high-GI breakfasts and lunches scored higher on hunger tests and overate a standard ad libitum diet during the remainder of the day compared with those who consumed lower-GI meals containing similar amounts of energy and nutrients (22). Voluntary energy intake after the high-GI meals was 53% greater than after the lower-GI meals and was associated with reduced concentrations of fatty acids and glucagon and higher concentrations of plasma epinephrine and growth hormone. Elevations in counterregulatory hormone concentrations suggest that the reduction in major metabolic fuels is biologically significant.

High-GI meals produced lower fatty acid concentrations throughout the day (23) and lower rates of fat oxidation than did low-GI meals of similar composition (24, 25). In young rats, high-GI starches produced not only higher respiratory quotients for several hours than did low-GI starches, but respiratory quotients reached >1.0, indicating that maximal rates of carbohydrate oxidation and glycogen synthesis were reached and de novo lipogenesis was taking place (26). In healthy subjects, when a high-GI meal was ingested 1–2 h before exercise, there was a greater shift in substrate utilization from fat to carbohydrate compared with when a low-GI meal was ingested (25). Thus, at least for several hours postprandially, both at rest and during exercise, fat utilization is depressed after high-GI meals compared with low-GI foods.

Short-term studies

Differences in the glycemic load (GI × carbohydrate content) of a diet were shown to affect energy expenditure when Agus et al (27) compared the effects of energy-restricted diets with high and low glycemic loads on hormonal and physiologic adaptations in a group of overweight young men for 9 d. They found that during the high-glycemic-load diet, energy expenditure and serum leptin concentrations declined more, nitrogen balance tended to be more negative, and voluntary food intake was greater. Limited availability of the major metabolic fuels after a high-GI meal elicits marked increases in the counterregulatory hormones, some of which have proteolytic actions. This in turn could favor the catabolism of lean body tissue. Howe et al (28) found that consumption of a low-GI diet based on high-amylose starch for 14 wk appeared to increase protein retention to a greater degree than did a diet based on high-GI starch in both normal and hyperinsulinemic men.

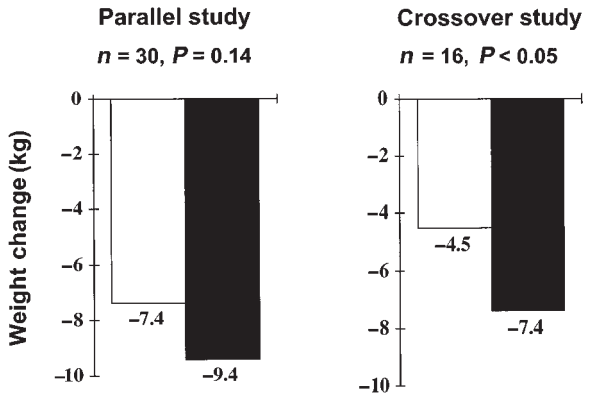

FIGURE 1. Weight loss during a parallel and crossover study of overweight women randomly assigned to consume high-glycemic-index (□) or low-glycemic-index (■) diets for 12 wk each (29).

Medium-term studies in humans

Several medium-term studies have compared the weight-loss potential of high- and low-GI diets. Slabber et al (29) studied obese females who consumed 2 energy-restricted diets, one high-GI and one low-GI, in a 12-wk parallel arm study (n = 30) that was followed by a 12-wk crossover study (n = 16). Both diets produced weight loss during the first 12 wk ($\bar{x}$: 9.4 compared with 7.4 kg), but there was no significant difference in the amount lost. During the follow-up crossover study, the low-GI diet produced greater weight loss (7.4 compared with 4.5 kg; P = 0.04) than did the high-GI diet (**Figure 1**). In both arms, the reduction in fasting insulin concentrations was greater with the low-GI diet than with the isoenergetic, macronutrient-balanced, high-GI diet. In slightly overweight men, Bouché et al (C Bouché, SW Rizkalla, J Luo, A Veronese, and G Slama, unpublished observations, 2000) showed that consumption of a low-GI diet for 5 wk, compared with a high-GI diet of equal energy and macronutrient content, decreased total fat mass by 500 g ($P < 0.05$, as measured by dual-energy X-ray absorptiometry), despite no difference in body weight. The decrease in fat mass was mostly abdominal and was associated with a decrease in *ob* gene expression in subcutaneous fat tissue. In a retrospective, nonrandomized study comparing the effects of a low-GI diet (n = 64) with those of a conventional reduced-fat diet (n = 43) in the management of pediatric obesity, body mass index (BMI; in kg/m^2) and body weight decreased more in the low-GI group than in the conventional diet group, even after adjustment for age, sex, ethnicity, baseline BMI, and baseline weight (30). Significantly more patients in the low-GI group achieved a decrease in BMI of at least 3 units (17% compared with 2%; $P < 0.001$). In contrast with the results of these studies, Wolever et al (31) reported similar weight loss in obese patients with type 2 diabetes who were randomly assigned to receive high- or low-GI hypoenergetic diets for 6 wk (2.5 compared with 1.8 kg; NS).

Apart from differences in weight loss, other intervention studies suggest that a diet's glycemic effect influences fuel storage within the body. In healthy, lean males, chronic consumption of high-GI diets, compared with nutrient-balanced low-GI diets, was associated with higher muscle glycogen (14%) and muscle triacylglycerol (22%) concentrations (both $P < 0.05$; 23). Compared with an equivalent low-GI meal, a high-GI meal consumed by well-trained athletes after prolonged exercise was associated with 48% more muscle glycogen (P = 0.02) after 24 h of recovery from the exercise (32). Finally, 12 healthy pregnant women who were randomly assigned to consume ad libitum high- and low-GI diets at 8 wk gestation gained significantly more weight by full term with the high-GI diet than with the low-GI diet (19.7 compared with 11.8 kg, respectively; $P < 0.05$; 33).

Long-term studies in animal models

Studies in animal models have the advantage of providing information on both the mechanisms and effects of longer lifetime feeding of high- and low-GI diets. Feeding rats high-GI starch over 5 wk resulted in higher epididymal fat weights (an index of visceral adiposity in rats) and larger adipocyte volume than did feeding low-GI starch ad libitum (34). Expression and activity of fatty acid synthase complex in white adipose tissue, and glucose uptake into adipocytes, were significantly higher in rats fed the high-GI starch diet ($P < 0.05$; 35, 36). For 32 wk, we fed 2 groups of young adult rats diets similar in energy distribution to modern Western diets, comprising 45% carbohydrate, 20% protein, and 35% fat as a proportion of energy (37). One group was fed a high-GI-starch diet (100% amylopectin, the most common form of starch in Western diets); the other was fed low-GI starch (60% amylose starch, the form of starch more common in traditional diets in nonindustrialized countries). The rats were fed 2 large isoenergetic meals per day to simulate meal feeding in humans and to maximize differences in postprandial hyperglycemia between the diets. A significantly different pattern of weight gain was observed during the 32 wk of dietary intervention (**Figure 2**). Whereas the low-GI group remained weight-stable, the high-GI group gradually gained weight and were 16% heavier at the end of the study. Total fat mass, assessed by measurements of total body water, was significantly (40%) higher in the high-GI group ($P < 0.05$). Each of the fat pads, but not the heart or liver, was heavier in the high-GI group. More importantly, the average weight of visceral fat in the high-GI group was twice that of the low-GI group and remained significantly higher when expressed as a proportion of total body fat ($P < 0.05$). In contrast, the subcutaneous fat pads, adjusted for total adiposity, did not differ between groups. We measured substrate utilization over 6 h postprandially and found different patterns both at the beginning and at the end of the study, with a marked decrease in whole-body fat oxidation in the high-GI group. Furthermore, the high-GI group had higher rates of hepatic lipogenesis and higher liver and red oxidative muscle glycogen stores. The high-GI group showed

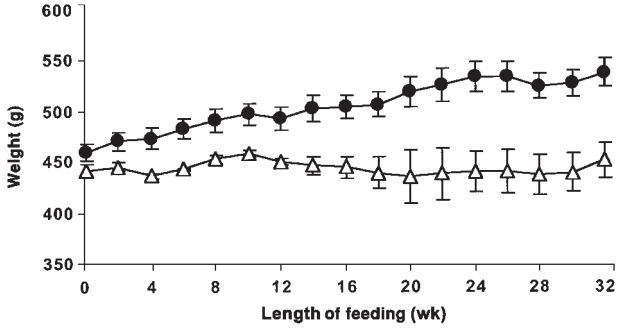

FIGURE 2. Weight changes in adult rats fed isoenergetic, nutrient-balanced diets based on high-glycemic-index (●) or low-glycemic-index (△) starch for 32 wk (37).

reductions in carnitine palmitoyltransferase 1 (CPT-1) messenger RNA (mRNA) in the liver, a key regulatory site of long-chain fatty acid flux through β oxidation. A concomitant increase in hepatic acetyl-CoA carboxylase (ACC) mRNA (ACC catalyzes the formation of malonyl-CoA) was observed. In the liver, malonyl-CoA is both an intermediate in de novo lipogenesis and a potent inhibitor of CPT-1. Thus, in addition to causing an acute effect on fuel oxidation, chronic high-GI feeding characteristically leads to changes in enzyme expression, thereby decreasing the potential for hepatic fat oxidation.

These findings in animal models have important implications because they challenge the assumption that "a calorie is a calorie." High-GI starch caused increased fat accumulation in visceral stores and reduced lipolytic capacity despite the fact that macronutrient and energy intake were precisely controlled.

Epidemiologic evidence

Cohort studies provide further evidence that the glycemic effect of the diet might influence weight control. In the EURODIAB Complications Study of nearly 3000 adults with type 1 diabetes, consumption of a lower-GI diet was found to predict lower waist-to-hip ratio and waist circumference independently of carbohydrate, fat, and fiber intakes (38). In the CARDIA study of young adults, low fiber consumption (GI was not assessed) predicted higher 10-y weight gain, waist-to-hip ratio, and 2-h postglucose insulin concentrations (a measure of insulin resistance) to a greater extent than did total or saturated fat consumption (39). Although fiber and GI are not precisely related, viscous dietary fibers and foods in which the natural cell wall architecture remains intact (eg, legumes) generally have lower GIs (40).

THE HYPOTHETICAL BIOCHEMICAL SCENARIO

The following scenario for high-GI compared with low-GI diets in weight regulation is hypothesized on the basis of factual but incomplete evidence from studies in animals and humans. Consumption of a high-carbohydrate, high-GI diet results in recurrent postprandial hyperglycemia and hyperinsulinemia that is accentuated in sedentary persons who are overweight, insulin-resistant, or both. As a consequence, carbohydrate oxidation is higher and fat oxidation lower throughout the postprandial period, whether the person is at rest or exercising. The expression of enzymes involved in lipid synthesis, such as ACC mRNA, is up-regulated, whereas the expression of those involved in lipid oxidation, such as CPT1 mRNA, are down-regulated. Glycogen stores in liver and muscle are maintained at higher concentrations with high-GI diets, but glycogen use and gluconeogenesis may also be higher. Counterregulatory hormonal responses (eg, of cortisol and noradrenaline) are also higher because of the hyperglycemic-hypoglycemic rebound after consumption. This stimulates gluconeogenesis from gluconeogenic amino acids as well as meal initiation in free-feeding individuals. The 0–6-h period following consumption of a high-GI diet is therefore characterized by a greater dependence on carbohydrate and protein as sources of fuel and less dependence on fat. Because carbohydrate and protein stores are limited, their higher rate of usage may stimulate appetite and encourage overconsumption. If energy intake and energy expenditure are matched over the long term, then body weight remains stable. However, even the small energy imbalances that are characteristic of modern lifestyles are more likely to promote gradual expansion of the fat stores (possibly at the expense of lean tissue) when the diet is based on high-GI foods.

CONCLUSIONS

The postprandial effects of carbohydrate-dense, high-GI foods described above may help to explain why low-fat diets have not lived up to their potential to inhibit weight gain when consumed ad libitum. Postprandial hyperglycemia and hyperinsulinemia are consequences of typical Western diets that are not seen in rural societies where high-carbohydrate, low-GI diets are typically consumed. This is especially true of persons who are sedentary, overweight, and genetically prone, in whom insulin resistance is common. Faster digestion and absorption and higher insulin responses after high-GI meals dictate differences in satiety and energy partitioning that, over the long term, favor expansion of the fat stores. Carefully designed multicenter studies to assess the efficacy of high-carbohydrate, low-GI diets in the treatment and prevention of obesity are clearly justifiable on scientific grounds.

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