Symposium: Dietary Composition and Obesity: Do We Need to Look beyond Dietary Fat?

Dietary Glycemic Index and Obesity[1,2]

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ABSTRACT Obesity is among the most important medical problems in America today. Currently, ~1 in 4 children and 1 in 2 adults are overweight, prevalence rates that have increased by 50% since the 1960s. In an attempt to combat this problem, the Federal government and various official medical agencies have advocated decreasing intake of total fat and sugar, while increasing consumption of "complex carbohydrate." Despite a recent reduction in fat consumption to near the recommended 30% of total energy, rates of obesity have continued to rise, suggesting that other dietary factors may play a critical role in body weight regulation. One such factor may be glycemic index. This review examines the physiologic effects of glycemic index and argues for the need for controlled clinical trials of a low glycemic index diet in the treatment of obesity. J. Nutr. 130: 280S–283S, 2000.

KEY WORDS: • *glycemic index* • *obesity* • *diet* • *dietary carbohydrate* • *blood glucose*

Obesity, a condition characterized by excessive body fat, is commonly believed to result in part from excessive fat consumption. Partly for this reason, the U.S. government (U.S. Department of Health and Human Services 1988), the American Heart Association (1996) and the American Diabetes Association (1997) recommend a low fat diet to prevent and treat obesity. However, the effect of dietary fat on body fat has been questioned in recent years (Katan et al. 1997, Larson et al. 1996, Willett 1998). Epidemiologic studies do not show consistently that high levels of dietary fat promote weight gain (Kant et al. 1995, Larson et al. 1996, Ludwig et al 1999a, Nicklas 1995), and weight loss with consumption of low fat diets is characteristically modest and transient (Katan et al. 1997, Lissner and Heitmann 1995). Moreover, mean fat intake in the United States has decreased since the 1960s, from 42% to ~34% of dietary energy (Lenfant and Ernst 1994, Nicklas 1995, Stephen and Wald 1990), whereas the prevalence of overweight has risen to 1 in 4 children and 1 in 2 adults (Flegal et al. 1998, Troiano and Flegal 1998). These observations suggest that dietary factors other than fat play an important role in body weight regulation.

The glycemic index

The concept of glycemic index (GI) was proposed by Jenkins and colleagues in 1981 to characterize the rate of carbohydrate absorption after a meal (Jenkins et al. 1981). GI is defined as the area under the glucose response curve after consumption of 50 g carbohydrate from a test food divided by the area under the curve after consumption of 50 g carbohydrate from a control food, either white bread or glucose (Wolever et al. 1991). Over the past two decades, the GI of most commonly consumed carbohydrate-containing foods has been measured (Foster-Powell and Miller 1995).

Many factors together, including carbohydrate type, fiber, protein, fat, food form and method of preparation, determine the GI of a particular food (Bjorck et al. 1994, Estrich et al. 1967, Welch et al. 1987, Wolever et al. 1991). Contrary to common belief, carbohydrate digestion rate, and therefore glycemic response, is not related to saccharide chain length. For example, Wahlqvist and colleagues demonstrated similar changes in blood glucose, plasma insulin and plasma fatty acid concentrations after consumption of glucose as a monosaccharide, disaccharide, oligosaccharide or polysaccharide (starch) (Wahlqvist et al. 1978). In fact, sugar may have a lower GI than some "complex carbohydrates" (Foster-Powell and Miller 1995) as demonstrated by an improvement in glycemic control among subjects with Type 1 diabetes mellitus after isoenergetic substitution of sucrose for starch (Rickard et al. 1998). In general, refined grain products and potato have a high GI, exceeding that of table sugar by up to 50%, whereas most vegetables, fruits and legumes have a low GI.

Low fat vs. low GI

Because protein intake for most individuals remains within a fairly narrow range, reductions in dietary fat tend to cause a compensatory rise in carbohydrate consumption. An increase in carbohydrate intake has in fact been observed in the U.S. since the 1970s (Nicklas 1995, Popkin et al. 1992, Stephen et al. 1995). The carbohydrate that replaces fat in low fat diets is

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TABLE 1

Studies comparing glycemic response with changes in hunger, satiety or energy intake

Reference	Modified dietary factor	Effect of low GI food
Haber et al. 1977[1]	Apple, whole or processed	Increased satiety
Krotkiewski 1984	Guar gum	Decreased hunger
Spitzer and Rodin 1987	Fructose or glucose	Lower voluntary energy intake
Rodin et al. 1988	Fructose or glucose	Lower voluntary energy intake
Leathwood and Pollet 1988	Bean or potato	Decreased hunger
Rodin 1991	Fructose or glucose	Lower voluntary energy intake
Holt et al. 1992	Breakfast cereal	Increased satiety
van Amelsvoort and Westrate 1992	Amylose or amylopectin	Increased satiety
Benini et al. 1995	Fiber added to meal	Decreased hunger
Gustafsson et al. 1995a	Vegetable type	Increased satiety
Gustafsson et al. 1995b	Raw or cooked carrots	Increased satiety
Holt and Miller 1995	Rice type	Lower voluntary energy intake
Lavin and Read 1995	Guar gum	Decreased hunger
Holt et al. 1996	38 individual foods	No change in satiety
Rigaud et al. 1998	Psyllium fiber	Lower voluntary energy intake
Ludwig et al. 1999b	Oatmeal type	Lower voluntary energy intake

[1] Haber et al. demonstrated differences in insulinemic, but not glycemic response.

typically high in GI. According to data from the Department of Agriculture, >80% of the carbohydrate consumed by children ages 2–18 y would have a GI equal to or greater than that of table sugar (Subar et al. 1998). Moreover, carbohydrate absorption rate (and therefore GI) is increased after a low fat meal because fat acts to delay gastric emptying (Estrich et al. 1967, Welch et al. 1987). Thus, the GI of the American diet has probably increased in recent years. Might this increase have contributed to the rising prevalence of obesity?

Effects of GI on appetite

To date, at least 16 studies have examined the effects of GI on appetite in humans (**Table 1**). For example, Leathwood and Pollet (1988) found lower blood glucose levels and slower return of hunger after meals with bean puree (a low GI starch) compared with meals with potato (a high GI starch). Holt and colleagues showed that glycemic and insulinemic responses to various breakfast cereals are inversely related to satiety score (Holt et al. 1992). In fact, all but one of these 16 studies demonstrated increased satiety, delayed return of hunger or decreased ad libitum food intake after low compared with high GI foods.

To explore the physiologic events that might relate GI to appetite, we compared the effects of three isocaloric test meals differing in GI during three separate 24-h admissions (Ludwig et al. 1999b). The low GI meal was a vegetable omelet with fruit, the medium GI meal was "steel-cut" oatmeal (a preparatory method that slows digestion rate) and the high GI meal was "instant" oatmeal. The medium and high GI meals were composed of similar foods to control for the effects of other potentially confounding dietary factors, whereas the low GI meal was designed to increase the range of GI in the study beyond that that could be achieved by manipulating food structure alone. Subjects included 12 obese teenage boys, at least 120% of ideal body weight, but otherwise in good health.

After the test breakfasts, area under the blood glucose curve differed between the high, medium and low GI meals as expected [284, 141 and 76.6 (mmol·min)/L, respectively]. The rapid absorption of glucose from the high GI meal resulted in relatively high insulin and low glucagon concentrations. These hormonal changes would be expected to promote uptake of glucose in muscle, liver and fat tissue, restrain hepatic release of glucose and inhibit lipolysis. As a consequence, access to the two major metabolic fuels was effectively impaired in the postabsorptive period, as shown by a "reactive hypoglycemia" (difference in glucose nadir of −0.5 mmol/L, $P = 0.02$) and lower free fatty acid concentrations 3 to 5 h after the high compared with the low GI meals. Subjects also consumed significantly more energy after the high GI (5.8 MJ) compared with the medium GI (3.8 MJ, $P < 0.05$) or the low GI (3.2 MJ, $P = 0.01$) test lunches.

Obesity

High GI foods elicit, calorie for calorie, higher insulin levels and c-peptide excretion than low GI foods (Haber et al. 1977, Jenkins et al. 1987, Wolever and Bolognesi 1996). The functional hyperinsulinemia associated with high GI diets may promote weight gain by preferentially directing nutrients away from oxidation in muscle and toward storage in fat. Cusin and colleagues reported that rats pretreated with insulin showed increased glucose utilization in white adipose tissue, but decreased utilization in muscle, changes that were associated with increased food intake and weight gain (Cusin et al. 1992). In humans, high acute insulin secretion after intravenous glucose tolerance tests predicts weight gain among glucose-tolerant offspring of parents with diabetes mellitus (Sigal et al. 1997). Pima Indian children with elevated fasting insulin levels gain prospectively more weight than those children with normal insulin levels (Odeleye et al. 1997). Indeed, excessive weight gain is recognized to be a complication of insulin treatment in Type 2 diabetes mellitus (UK Prospective Diabetes Study Group 1998) and intensive insulin treatment in Type I diabetes mellitus (Diabetes Control and Complication Trial Group 1988). Thus, hormonal responses to a high GI diet appear to lower circulating levels of metabolic fuels, stimulate hunger and favor storage of fat, events that may promote excessive weight gain.

SUMMARY

The concept that "a calorie is a calorie" underlies most conventional weight loss strategies. According to this principle, obesity results from an imbalance between energy intake and expenditure. The proposed cure is to eat less and exercise

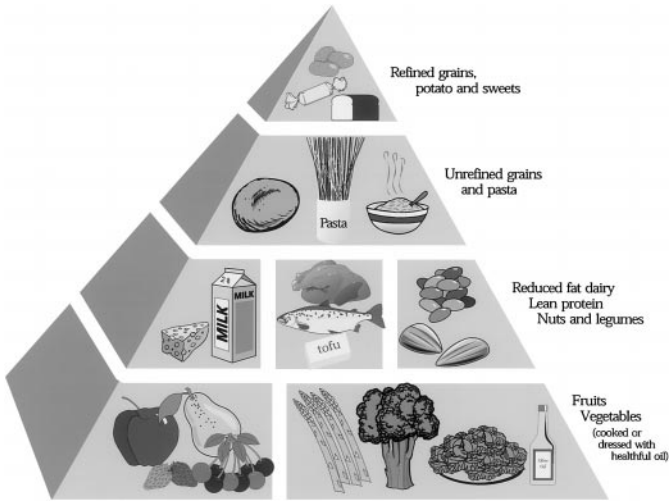

FIGURE 1 A low glycemic index "pyramid."

more. However, calorie-restricted, low fat diets have poor long-term effectiveness in the outpatient setting. In a sense, these diets may constitute symptomatic treatment that does not address the physiologic drives to overeat. From a hormonal standpoint, all calories are not alike.

The optimal diet for the prevention and treatment of obesity, if one exists, remains to be determined. In particular, the effects of GI on body weight regulation must be explored in long-term clinical trials. Nevertheless, a growing body of theoretical and experimental work suggests that diets designed to lower the insulin response to ingested carbohydrate (e.g., low GI) may improve access to stored metabolic fuels, decrease hunger, and promote weight loss. Such a diet would contain abundant quantities of vegetables, fruits and legumes, moderate amounts of protein and healthful fats, and decreased intake of refined grain products, potato and concentrated sugars (**Fig. 1**). Indeed, this diet bears a close resemblance to that consumed by human ancestors over the last several hundred thousand years (Eaton and Konner 1985). Finally, reductions in dietary GI may also lower the risks for various conditions associated with hyperinsulinemia, such as diabetes mellitus (Salmeron et al. 1997) and cardiovascular disease (Frost et al. 1999, Jenkins et al. 1985, Lamarche et al. 1998).

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