



Continuing Education

Glycemic Index

The State of the Science, Part 2—Roles in Weight, Weight Loss, and Satiety

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Lowering the glycemic index (GI) and glycemic load (GL) as a strategy to prevent weight gain, enable weight maintenance, and/or promote weight loss is a subject in the popular and scientific literature. Proponents both for and against such a dietary strategy can produce data from the scientific literature that support either position. This narrative review focuses on the role of GI or GL and weight and emanates from the white paper completed for the Wheat Foods Council. In addition, for the series in this publication, findings from relevant papers published since the completion of the white paper were added to the review. Overall, the findings are mixed. Studies in the aggregate fail to show a clear conclusion regarding the efficacy of adopting a low-GI or low-GL strategy for prevention of obesity or for any other aspect of weight control. Large cohort studies actually show that those whose diets are highest in GL tend to have lower body mass indexes. Intervention studies do not show an advantage of a low-GI or -GL diet for weight loss when calories are controlled. The impact of GI and GL on waist circumference, satiety, and hormones or other measures appears to be dependent on the characteristics of the participant, such as age and gender. Differences in diet composition to achieve lower GI or GL also impact outcomes. One large, recent study suggests that changes in GI alone may not matter, but that the interaction of high dietary protein and low-GI diets may help with prevent weight gain in children and aid weight loss and maintenance in adults, but more research is needed. Nutr Today. 2013;48(1):7-16

he worldwide "diabesity" epidemic has launched discussions not only about calories but also about the source of calories. Specifically, these discussions reinvigorate the dietary "holy grail" quest for delineation of the ideal combination of fat, protein, and carbohydrate (CHO) to prevent overweight, promote weight loss, and enable weight maintenance. Recently, the conversation has shifted from fat to CHO as the major concern, both in terms of quantity and quality. There is particular focus on CHO's role in controlling weight by affecting blood glucose and insulin responses as measured by the glycemic index (GI) and glycemic load (GL).

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Source of support: Wheat Foods Council.

The author has no conflicts of interest to disclose.

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DOI: 10.1097/NT.0b013e31827d8515

Both the obesity problem and CHO's potential role are specifically addressed in the 2010 Dietary Guidelines Advisory Committee (DGAC) report, which calls for decreased "consumption of energy-dense carbohydrates, especially refined, sugar-dense sources, to balance energy needs." ¹

Some popular and scientific literature suggests that both obesity and weight loss can be addressed by considering CHO quality and quantity and that lowering GI or GL or both is a viable strategy. Proponents, both for and against the use of these concepts, produce evidence in support of their position. For example, evidence-based reviews supporting the work of the 2010 DGAC found little or no association between GI and GL and measures of body weight. Another review concluded somewhat more positively with the following statement, "there is some, although not consistent, evidence for a lower body weight on diets with a lower GL, but the effect is likely to be small. There is currently no convincing evidence for a role of GI independent of GL."² The latter conclusion and those of the DGAC have spawned sharp criticism by proponents of this concept. Some suggest that either the evidence in support of GI and GL for weight maintenance, weight loss, and other health benefits was not given proper weight or that the conclusions may have been influenced by powerful food lobbies.^{3,4} Champions of the use of low-GI and -GL diets for obesity and weight control cite reviews with conclusions that read as follows, "a large body of evidence, which now comprises observational prospective cohort studies, randomized controlled trials, and mechanistic experiments in animal models, provides robust support for low- GI carbohydrate diets in the prevention of obesity."⁵

Such widely variant conclusions sparked the need for an update of a 2003 white paper,⁶ which is a narrative review conducted for the Wheat Foods Council. The 2010 white paper, also a narrative review, was published on the Web.⁷ The findings are being updated for a series of narrative reviews for *Nutrition Today*. This article is the second in the series. The first article introduces the measure and how it is calculated and discusses the many factors affecting its variability and other general aspects about GI and GL.⁸ Like the other articles in this series, this is a narrative review constructed from a MEDLINE search of articles published between 2003 and early 2010 (with some additional references added to the manuscript as it underwent review). The following search terms were used: *glycemic index* and

glycemic load were coupled with the terms obesity, BMI, weight, body composition, waist circumference, visceral adipose, and diet. The purpose of the review was to evaluate the evidence with respect to dietary GI and GL and their ability to prevent overweight and obesity or to aid weight loss and weight maintenance. These findings will be put into the context of total dietary CHO and its role in weight control.

CHO, GI/GL, AND BODY WEIGHT

CHO and Body Weight

The topic of GI and GL and diet must be placed in the framework of total CHO intake because both concepts describe the dietary CHO. A comprehensive review addressing the issue of CHO intake and body weight was published in 2007.9 The reviewer concluded that virtually every major North American epidemiologic study shows an inverse relationship between CHO intake and body mass index (BMI). Data from these studies are presented in Table 1. These show a trend toward decreasing BMI with increasing CHO intake for both genders. Furthermore, the effect was shown in both prospective (the Health Professionals Study I, the Nurses' Health Study I and II, the Women's Health Study, the Canadian National Breast Screening Study, and the Prostate, Lung and Ovarian Screening Study) and crosssectional studies (National Health and Nutrition Examination Study). However, some question the validity of such conclusions because overweight subjects have been shown to underreport their food intake. 10 Thus, conclusions linking intake of certain dietary components and BMI may be flawed.

GI and Body Weight

The same studies that looked at CHO intake also found no significant relationship between GI and BMI.⁹ In 2 prospective cohorts of women (the Nurses Health Study I and the Women's Health Study) and 1 of men (the Health Professionals Follow-up Study) and in 1 large and 1 small cohort of men, BMI decreased as GI increased. In the Atherosclerosis Risk in Community study, a multiracial cohort of both genders, BMIs were unrelated to dietary GI. In summary, data from the 9 cohort studies (n = 300 989) in the aggregate showed that the average BMI was 25.9 kg/m² for those in the quintile with the lowest dietary GI and 25.5 kg/m² for those in the quintile with the highest dietary GI.⁹

GL and Body Weight

For GL and BMI, the GL of the diet was either not significantly related to BMI or there was a trend for it to be inversely related. For the 11 cohort studies considered in the aggregate (n = 394 199), the average BMI was 26.1 kg/m² for those in the lowest quintile of GL intake and 25.2 kg/m² for those in the quintile with highest GL.9 Thus, results from these large cohorts do not support the tenet that an increase in dietary GI or GL or CHO intake is associated with an attendant increase in BMI. If anything, they suggest that BMI decreases as GL increases (Table 2).

Some studies on European populations give similar results to those observed in North American samples, but not all do. For example, in a case control study of Italian men and women (n=7724), there was an inverse association between both GI and GL and BMI. The mean BMI for men eating in the lowest tertile of GI was 26.59 kg/m² and for the highest tertile was 26.18 kg/m², and for women, 25.81

Populations Representation of Carbohydrate Intake in North American								
				BMI (kg/m²) by Increasing Quintiles (Tertiles) of Carbohydrate Intake			ertiles) of	
Cohort	Type of Study	Gender	n	I		Ш	IV	V
Nurses' Health Study I	Prospective	Women	71 919	25.2	25.2	25.1	24.9	24.7
National Health and Nutrition Examination Study	Cross-sectional	Women	6125	26.3	26.4	26.1	25.9	25.5
Women's Health Study	Prospective	Women	38 446	26.7	26.3	26.1	25.7	25.2
Canadian National Breast Screening Study	Prospective	Women	49 111	25.2	25.1	24.7	24.6	24.3
Health Professionals Follow-up Study	Prospective	Men	39 926	26.1	25.9	25.6	25.3	24.8
Prostate, Lung and Ovarian Screening Study	Prospective	Men	20 172	28.1		27.6		26.8

Adapted with permission from Gaesser GA. CHO quantity and quality in relation to body mass index. J Am Diet Assoc. 2007;107:1768–1780.

TABLE 2	Body Mass Index (BMI) From Large North American Cohorts by Quintile of
	Glycemic Load

				BMI (kg/m²) by Increasing Quintiles of Dietary Glycemic Load			Dietary	
Cohort	Type of Study	Gender	n		=	Ш	IV	V
Nurses' Health Study I	Prospective	Women	65 173	25.2	25.2	25.1	24.9	24.7
Women's Health Study	Prospective	Women	38 446	26.7	26.3	26.1	25.7	25.2
Health Professional's Follow-up Study	Prospective	Men	42 759	26.1	25.9	25.6	25.3	24.8
Health, Aging, and Body Composition (Health ABC) 70–80 y white and black	Prospective	Women	1169	27.4	26.7	27.8	26.2	27.1
Health, Aging, and Body Composition (Health ABC) 70–80 y white and black	Prospective	Men	1079	26.7	26.8	27.8	26.6	26.7

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and 25.09 kg/m², respectively.¹¹ These differences, albeit small, were highly significant. For GL the corresponding differences in BMI between the highest and lowest tertiles were 26.41 and 26.25 kg/m² in men and 26.01 and 24.93 kg/m² in women. The authors of the Italian study came to a similar conclusion to that of authors in the North American studies, "GI and GL were, if anything, inversely related to BMI and waist-hip ratio..."11 Findings from a crosssectional study on Spanish adults (n = 8195) also showed that GL was negatively associated with BMI. Unlike in the Italian study, GI was not associated with BMI even when underreporters were taken into account. 12 This study showed how different the food patterns of diets with the high GI were from those of high GL, noting that a diet rich in fruits, vegetables, and legumes was related positively to high GL but negatively to high GI.

In an elderly subset of a Danish cohort (n = \sim 400), the tendency to gain weight over time was studied. 13 This study in an older population gave a different picture from that observed in the Italian and Spanish studies that merely looked at weight at a point in time. Whereas GL was not related to a tendency to increase weight over time in either gender, GI was positively associated with increased weight gain and waist circumference in women (not men). The increases were more pronounced if the women were sedentary, causing the authors to suggest a potential interaction between gender and GI and obesity development. 13 In a healthy, elderly Mediterranean island population (n = \sim 1200), a 1% increase in dietary CHO predicted a 12% lower likelihood of central adiposity. In this population, a low-GI diet was not as effective as increases in total CHO for reducing the likelihood of central adiposity.¹⁴ These data indicate that higher total CHO, which also means lower fat, is more likely to prevent central adiposity than low GI.

Data from a prospective cohort study in 5 European countries—Denmark, Germany, Italy, the Netherlands, and the United Kingdom with 89 432 healthy adults—showed no consistent associations between GI and GL and subsequent changes in weight. However, the effect on waist circumference varied by country. The authors of this study concluded that dietary GI or GL did not impact weight change over time. However, because GI (not GL) was associated with increases in waist circumference in some countries, lower GI diets may be play a role in the prevention of abdominal obesity. The surface of th

GI and GL and Weight in Youth

Similar to the associations between BMI or measures of excess weight and GI or GL seen in adults, the associations in children and teenagers also lack consistency. No significant associations between GI and BMI or other measures of excess weight were observed either in a sample of 6- to 7-year-old normal and overweight children (n = 317) in Hong Kong 16 or in a sample of 10- to 17-year-old overweight Latino youth (n = 120). 17 However, studies of Danish youth and Japanese young women show a more complicated picture. For Danish 10-year-old (n = 485) and 16-year-old (n = 364) youth, dietary GI and GL were positively associated with markers of body fatness only in 16-year-old boys. 18 However, in a cross-sectional study of 18- to 20-year-old Japanese dietetics students (n = 3931), both GI and GL were positively related to BMI in young women. 19

In summary, a number of studies indicate that higher GL may be related to lower body weights. In most studies, GI

was not related to body weight but was related to increased tendency to gain weight over time. Effects of age, gender, or country need further study.

POTENTIAL REASONS FOR INCONSISTENCY ACROSS STUDIES

There are several potential explanations for the various observations. First, the data may accurately reflect the situation, for example, that neither the GI nor the GL of the diet is an important factor in determining BMI or that high GL actually is associated with lower body weights because of high intakes of fruits, vegetables, and grains. Second, the arbitrary assignment of a food or a diet into a low, medium, or high category can affect the results. Analysis of the different studies used in the 2007 review shows a wide variation in what was labeled as low and high in terms of GI and GL.⁹ Mean GI scores of the quintile (tertile) labeled as low ranged from 50 to 77, and those labeled as high ranged from 55 to 86.6. The large overlap of categories is especially troubling in light of lack of precision and accuracy of assigning GI to foods and diets from food frequency questionnaires using table values. 7,8,20 For example, data from these questionnaires often fail to give different values depending on the degree of ripeness or cooking methods—just a few of many factors that affect GI and GL.^{7,8} One review stated that the use of values from the tables is so variable as to render them of little use in studies.²¹ Third, underreporting of food intake by those who are overweight, 10 even when methods to more accurately capture dietary intake are used, 22 not only affects the accuracy of the intake data but also could certainly impact GI and GL values assigned to diets. Fourth, subject differences may affect the outcome so data sets may need to be subdivided by age, gender, ethnicity, dietary pattern, or activity level. Fifth, the actual measure of overweight may be important. In some studies, BMI was unrelated to GI or GL but was related to other measures such as skinfold and central adiposity. Sixth, markedly different foods and diets can yield the same GI and GL scores. For instance, low-GI and -GL diets may be constructed with fruits, vegetables, nuts, whole grains, and high-fiber cereals. Diets rich in any 1 of these components, alone or as part of the dietary pattern, are associated with improved nutrient profiles, better health profiles, and lower measures of body weight.²³ In contrast, low-GI or -GL diets may contain little CHO, fruit, whole grains, and dietary fiber. Such dietary patterns may be high in saturated fat²⁴ or other factors associated with increased risk of obesity and chronic disease. Furthermore, cohorts from different cultures may show different outcomes because eating patterns and other cultural and genetic aspects also affect the associations. Thus, it is perhaps not surprising that the relationship between GI and GL and body weight varies across studies. In summary, the weight of existing evidence indicates that dietary GI and GL are not related to BMI or GL may even be inversely related to body weight. A few studies indicate a possible relationship between GI or GL and increased visceral adipose or tendency to gain weight in certain groups. However, the variability among studies makes characterizing the precise relationship challenging.

GI/GL AND WEIGHT LOSS

GI/GL and **Short-term** Weight Loss

Weight loss and maintenance often require different dietary strategies than the prevention of weight gain. Some weight loss regimens use low or no CHO* as a critical diet pillar. Others allow CHOs only if they are low GI or the total diet is low GL. Proponents of such strategies cite testimonials from mainstream authors as well as data from well-controlled, short-term studies. Studies verify that greater weight loss occurs in the short-term (<6 months) when dieters select a low-GI or -GL food pattern than when they select other popular diet plans. However, in studies where calories were strictly controlled, the GI or GL of the diet made no difference in weight loss outcomes.²⁵

One review showed that low-GI and -GL diets caused significantly greater weight loss in some studies, but most only showed a nonsignificant trend favoring low-GI/GL diets. Thus, the reviewers suggested that other dietary factors beyond GI/GL play a role. Reviewers for a Cochrane review occurred that low-GI/GL diets promoted a small (~1 kg) but statistically significant greater weight loss than other diets did. Furthermore, these reviewers noted that the small weight loss was accompanied by other positive changes such as a greater decrease in coronary disease risk factors including blood lipids and markers of inflammation. (This will be discussed in subsequent articles that address GI/GL, coronary disease, and blood lipids and inflammation. ^{28,29})

GI/GL and Long-term Weight Loss and Weight Maintenance

Low-CHO and low-GI and -GL diets appear to outperform most other diets in the short-term (6–9 months). Only a few studies exist that monitor diet success for periods of a year or longer, and of the few that do, none show better weight loss or maintenance with low-GI or -GL diets. However, some show a small advantage with respect to blood lipid profiles. ^{30,31} For example, obese individuals (n = 330, average BMI, 36 kg/m²) were randomized either to a low-CHO diet, consisting of low-GI vegetables and unrestricted consumption of fat and protein, or to a low-fat diet. ²⁹ Weight loss over the 2-year period did not differ between the diets. Compared with the low-fat diet, lipid profiles were slightly better with the low-CHO, low-GI diet. It must

*Low- and no-CHO diets are inherently low GL because GL is calculated with the GI of the CHO multiplied by the total amount of CHO.

be pointed out that a larger number of those on the low-CHO, low-GI diet dropped out of the study.

Staying with a diet is very important to successful weight loss and weight maintenance. This was shown in a 1-year-long randomized trial (n = 160). Those who continued with their assigned diet—no matter which diet—lost weight.³⁰ Numbers of pounds shed were not significantly different for the various diets, but those diets that deviated most from mainstream ones such as the very low-CHO (low-Gl and -GL) Atkins diet had higher dropout rates than did the less extreme diets.³¹ Similar results both in weight loss and tendency to drop out were observed in an intervention trial with 322 moderately overweight subjects. The subjects were placed on either a Mediterranean, low-CHO, or low-fat diet.³² Dropout rates were higher for those assigned to the low-CHO diet (22%) than for those assigned to the other 2 diets (10%–15%).³³

Studies of weight loss for longer than 2 years are not common. One 3-year study suggests little, if any, advantage of low-GI/GL diets for weight loss.34 Although weight loss on the low-GI/ GL diet was greater at 6 months, weight loss plateaued after 6 months. At the end of 3 years, there was greater tendency to re-gain weight with the low-CHO diet than with the low-fat diet. The net result is that over the 3-year period, weight loss was greater with the low-fat diet than with the low- GI/GL diet.³⁴ Data from the National Weight Control Registry of Successful Losers (persons who have lost ≥30 lb and kept it off for >1 year) record that only 10% of the participants report following a low-CHO diet.³⁵ However, those reporting this diet strategy were equally likely to keep the weight off as those reporting other strategies.³⁵ Recently published results from a large, multicountry European study (the DIOGenes project†) with around 800 adults who had lost weight with a very-low-calorie diet (800 kcal) show that those assigned to a diet with modest increase in protein content and a modest reduction in GI maintained their weight loss better than those assigned to other dietary regimes.³⁶ Children in the DIOGenes study (381 boys and 446 girls, aged 5-18 years) randomized to the low-GI/high-protein diet were less likely to gain weight and had less adipose than did those assigned to other diets.³⁷ Two important findings from these studies are worth highlighting. First, these studies showed that low GI or GL alone was not effective in helping with weight maintenance in adults or preventing weight gain in children. Second, the papers noted that the dietary changes were moderate and not extreme making compliance more likely. 36,37

Potential Mechanisms for Low GI/GL and Weight Loss and Maintenance

Numerous reasons have been suggested as to why low-CHO and low-GI/GL diets increase weight loss in the initial

†DIOGenes stands for "Diet, Obesity and Genes" and is a pan-European program that targets the obesity problem from a dietary perspective.

phase of a diet. These include effects on satiety, hormones, and psychosocial and behavioral aspects associated with eating and dieting.

GI/GL and Satiety

Low-Gl and/or -GL foods and diets are theorized to promote weight loss because they induce satiety. For example, 1 study with 22 overweight women showed that low-Gl meals, compared with high-Gl meals, suppressed hunger and subsequent food intake and caused greater satiety. This type of study was indicative of human intervention studies included in systematic review. These reviews showed low-Gl/GL foods or meals to be more satiating in the short-term than high-Gl/GL foods or meals are. Studies such as these were packaged as a dossier and submitted to the European Food Safety Authority to substantiate claims such as "low-Gl foods help one to feel fuller for longer than equivalent high-Gl foods."

Despite the seeming concordance presented by studies in the European Food Safety Authority dossier, not all studies show that low-GI foods are associated with satiety. In some instances, the GI of a food neither predicts satiety⁴⁰ nor offers greater satiety. Boiled potatoes, a high-GI food, were given the highest satiety index among foods and were more satiating than lower GI french fries. 41 The lack of consistent effect of either GI or GL on satiety was reflected in a conclusion of a Japanese review on the subject. 42 A group of experts convened to assess the existing data by the CHO Task Force, the International Life Sciences Institute (Japan) concluded that ingestion of high-GI foods increased hunger and lowered satiety in short-term human intervention studies, but hunger and satiety ratings after the ingestion of foods with varying GIs were inconsistent in long-term human intervention studies.⁴³

Because many factors, including sensory ones, impact satiety, it is often difficult to deduce the particular attribute of the food responsible for greater satiety. One study designed to eliminate the effects of sensory differences on satiety changed dietary GL by using different amounts of white bread (a high-Gl food) to increase GL.⁴⁰ Increasing dietary GL by simply increasing the amount of a high-Gl food eaten had no effect on satiety or hunger in the 2-hour period after eating.

Furthermore, people ingest food even when from a caloric and other standpoints they are sated. Thus, it is difficult to conclude that the GI or GL values of foods or mixed meals are a valid long-term predictor for appetite, hunger, and satiety.⁴²

Another difficulty with satiety studies is translating the reported increased satiety into reduced food intake. One study using low-GI foods as part of the WeightWatchers POINTS program showed increased satiety in the early stages of the program.⁴⁴ Both ratings of hunger—especially in the

afternoon—and desire to eat were consistently lower for those ingesting the low-GI modification. However, after 12 weeks, there was no difference between the groups in weight loss or other measures.⁴⁴

Satiety and satiation can be examined through perceived hunger, feelings of fullness, and monitoring of food intake at various intervals after eating, just to name a few. Different measurement protocols and times at which measurement occurs can affect the outcome. These differences and the impact small changes in satiety on overall caloric intake could be some reasons for inconsistency among the studies. Adding to this is the fact that satiety is a function not just of physiological responses such as hunger but also to many food attributes, with GI or GL being only 1 among many.

GI/GL and Hormones

Low-GI and -GL diets are touted as helping dieters by controlling the release of glucose and insulin and other hormones that affect appetite (Table 3).

GL and Glucose and Insulin. Low-GI foods can result in a lower increase in both glucose and insulin, 45,46 but there is a question whether this is consistently associated with hunger or satiety. The nearly 50-year-old glucostatic theory of Jean Mayer suggests that increased levels of blood glucose promote satiety. 47 Data bolstering that theory are recent findings that indicate that low blood sugar levels are linked to weight gain and are a strong predictor of the amount of weight regained after weight loss.⁴⁷ These findings fail to give a clear role for low-GI or -GL diets in controlling hunger or weight gain. Further complicating the picture are findings indicating that the ability of blood glucose to signal hunger may depend on a person's metabolic or exercise state, with overweight individuals able to respond to low blood sugar if they were also exercising.48 Another study suggested that the glycemic response had little impact on short-term appetite sensations in normal men, but a low-GI meal reduced subsequent energy intake in those with insulin resistance.⁴⁹

TABLE 3 Some Internal Signals Associated With Satiety						
Hormone	Where Released	Effect on Feeding Behavior				
Insulin	Pancreas	Decrease				
PYY	Intestine	Decrease				
Leptin	Fat cells	Decrease				
Cholecystokinin	Stomach	Decrease				
Ghrelin	Stomach	Increase				
Abbreviation: PYY, peptide YY.						

Another postulate suggests that insulin release in response to high-GI or -GL foods might cause release of hormones that can affect hunger. The theory is interesting, but the measured outcomes appear to depend on the metabolic state of the eater. Excess weight may reduce insulin sensitivity, which not only blunts response to insulin but also may diminish the body's ability to respond to many other hormones and signals affecting appetite and satiety. 45,49,50 In summary, there is not strong agreement about the effects of blood glucose and insulin on the body's hunger and satiety responses. There may be a weak trend toward greater satiety and reduced hunger with a low-GI or-GL diet, but the differences in satiety often fail to translate into consistent, measurable decreases in caloric intake. GI and GL and Other Hormones That Affect Eating. Leptin and ghrelin are two of a number of hormones that affect hunger and satiety (Table 3). In normal-weight subjects, leptin is released from fat tissue. Once in the blood stream, the hormone travels to the hypothalamus where a signal to stop eating is released. Ghrelin is a hormone that promotes eating. Carbohydrate-rich meals suppress ghrelin. The theory is that low-GI CHOs cause adipose tissue to release leptin and the stomach to inhibit ghrelin release. Thus, the changes in both hormones would be expected to reduce appetite. The effectiveness of hormone release appears to depend on insulin sensitivity, and in some studies, subjects showed no change in hormone levels with a low-GI diet. 48-50 The actual effects may vary with baseline insulin levels of the subject.⁵¹ In rats, a high-GI diet caused a downregulation of ghrelin and increased fat deposition.⁵² However, in humans, both with and without insulin resistance, a low-GI diet and a normal American diet had the same impact on ghrelin release.⁵³ Other hormones such as cholecystokinin can also inhibit eating behavior (Table 3). Some studies show that high-GI meals were more sating because they caused greater cholecystokinin release.³⁸ Thus, the roles are all hormones on eating behavior need greater clarity.

Other Nonphysiological Impacts of Low-GI/GL Diets

Diets can be successful for a number of reasons beyond their physiological effects. This is true because so many factors influence eating behaviors.

Both ease of following a diet and the seeming freedom to eat unlimited amounts are important to some dieters. Low-Gl or -GL diets often have simple directions, and calorie counting is unnecessary. Dieters using published tables can categorize foods as low, medium, or high Gl or GL. Directions state that a dieter can either "eat as much as wanted as long as it is of allowed foods" or "eat until sated and never feel hungry." Thus, such diets may offer special attraction to those who are at 1 end of the scale for either the restraint or disinhibition scales. ⁵³

Food palatability is another aspect that needs to be considered. Humans and rats alike will seek energy dense

foods and overconsume highly palatable foods. A recent study showed that dieters craved foods with high energy density and fat and low in fiber. This was true regardless of whether the food had a high or low GL. 53 Furthermore, the

ability to compensate for calories is compromised by foods high in sugar and fat. Because high-GI/GL foods and diets may be both highly palatable and energy dense, their lack of impact on satiety and subsequent caloric

TABLE 4 3 Sample Low-Carbohydrate (CHO)/Low-Glycemic Index (GI) Meal Patterns With Fat, Fiber, and Fruit and Vegetable Servings							
Breakfast	2 eggs, soft boiled	Frittata-3 eggs	$\frac{1}{2}$ c hot and creamy barley, $\frac{1}{2}$ c protein skim milk and $\frac{1}{2}$ c toasted almonds				
	1 apple sl. very thin	⅓ c heavy cream, ⅓ c Colby cheese	½ grapefruit				
	½ large banana	1/3 c cooked bacon, 3 tbsp onions and peppers					
	1 orange	½ c buttered spinach					
	1 whole grain wrap						
Snack	1 oz walnuts	1 c bacon rinds	1 c plain yogurt with protein with $\frac{1}{2}$ c raspberries				
Lunch	$\frac{1}{2}$ c tuna salad in a medium tomato	Bologna, 2 slices	1½ c Greek salad with feta cheese				
	1 c strawberries	Cucumber, radishes	1 serving rye crisp bread				
	1 serving rye crisp bread	Lettuce with blue cheese dressing (1 tbsp)					
		1 serving rye Krisp with butter					
Snack	½ c toasted garbanzo beans	2 oz Snickers bar	³ / ₄ c fresh cherries				
Dinner	3 oz grilled chicken breast	Double cheeseburger (12 oz) with lettuce and tomato slices	6 oz poached filet of sole with butter sauce				
	1 c cucumber, arugula, pinenut salad with balsamic vinaigrette dressing (1 tbsp)	1 c mixed greens and pinenuts with ranch dressing (1 tbsp)	½ c broccoli spears, pinenuts and sautéed with garlic				
	2 slices sprouted bread	½ c broccoli spears with parmesan cheese	½ c brown rice				
	½ c broccoli spears	1 slice low-sugar ice cream					
	½ c apple crisp made with sugar substitute and 2 tbsp oat topping						
Fruit and vegetable servings	9	6	6				
Dietary fiber, g	33	16	25				
Total fat , g	50	180	15				
Total CHO, g	278	80	337				
Total kcal	1854	2200	1656				
Total GL	46	47	48				

intake might be erroneously attributed to GI/GL when it should be attributed to other aspects. Thus, the amount eaten may have more to do with what is being fed than on the GI of the food. For example, barley, a very low-GI food, did not cause diminished appetite in 14 healthy, normal-weight adults when compared with the same diet formulated with a higher GI food such as wheat.⁵⁴ Despite barley's 32% lower glycemic and insulinemic responses as well as much higher satiety index, food intake after subjects ate a barley meal was not diminished. In fact, calorie intake after the wheat meal was 23% less than after the barley meal. Thus, under these experimental conditions, the lowered glycemic and insulinemic response did not translate into lower caloric ingestion.

GI/GL AND NUTRITIONAL QUALITY OF THE DIET

One theory is that diets that deliver adequate nutrition may reduce the desire to eat and impact satiety. ⁵⁵ Low-GI/GL diets may be constituted so they are replete with fruits, vegetables, and whole grains. These foods tend to have high volume per calorie; contain dietary fiber, resistant starch, and slowly available CHO; and are rich in nutrients. All have been shown to be associated with reduced body weight. Strict low-CHO diets may also be low GI and GL because of the restriction of all CHO foods. Such diet plans may reduce overall food intake because food choice is limited and the diet can become boring.

Three potential eating plans, which are constituted with low-GI foods and give approximately the same dietary GL, are found in Table 4. These plans show their great difference in macronutrients and diet quality (Table 4). Menus 1 and 3 are high in fiber and fruits and vegetables and contain some cereal fiber, unlike menu 2. Menus 1 and 3 are also much lower in saturated fats than menu 2 is. Analysis of various micronutrients would show differences among the 3 menus. Thus, it can be seen that a diet that has a low GI or GL may not deliver the same health benefits depending on how the diet is constituted.

GI/GL and Satiety

The whole topic of GI/GL, satiety, and hormones affecting it needs much more study. Suggesting that a single property of a food or diet affects satiety is simplistic because so many factors from baseline hunger to palatability to physiological state are involved. Underlying this complex mix is the fact that research on many of the hormones affecting hunger and satiety is at an early stage of development. Thus, it is not surprising when studies state that no conclusions could be drawn about the long-term effect of GI/GL on satiety and subsequent body weight regulation.

In conclusion, the greater weight loss and satiety promised in books advocating low-CHO and low-GI or -GL diet are not consistently observed in scientific studies. When compared with a low-fat diet, a slightly greater weight loss (\sim 1 kg) occurs in the first 6 months on the diet. After 2 years, there is no difference in weight loss but slightly better blood lipid profiles and lower inflammation with the low-CHO, low-GI diet in some studies. (This will be addressed in an upcoming article.)²⁸ Although studies indicate that a low-CHO, low-GI diet can help with weight loss for those who choose it and stick with it, they also indicate that the ability to remain on the low-CHO, low-GI diet for long periods of time is more difficult for some than diet patterns that vary less from mainstream diets. Some suggest that there is a role for low-GI or -GL diets, especially when coupled with high protein, in the management of overweight children and adults. 36,37,55-57 However, there is still strong support that the "optimal diet for prevention of weight gain, obesity, metabolic syndrome, and type 2 diabetes is fat-reduced, fiber-rich, diet high in low-energy density carbohydrates (fruit, vegetables, and whole grain products)."56 Such diets have been studied for periods longer than a year and have been used by most people in the Weight Control Registry. 35,58 In terms of weight loss, a noted obesity researcher stated that reduction in dietary fat and an increase in fiber were the strongest predictors of weight loss and diabetes protection.⁵⁵ The role of dietary GI and GL in various aspects of weight needs more study and better control and characterization of the many variables that can impact the results. Finally, the results of the very large DIOGenes studies^{36,37} indicate that more than 1 aspect of diet may need to be studied together. In their studies, diets that helped adults lose and keep weight off and reduced chances of children gaining weight used a diet that was a combination of low GI with high protein. The other critical aspect of the DIO-Genes diet was that the dietary modifications were moderate, rather than extreme, making the long-term success of such a regimen more likely.

REFERENCES

- USDA Center for Nutrition Policy and Promotion. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010. http://www.cnpp.usda.gov/dgas2010dgacreport.htm. Accessed June 2010.
- 2. van Baak MA, Astrup A. Consumption of sugars and body weight. *Obes Rev.* 2009;10(Suppl 1):9–23.
- 3. Willett WC, Ludwig DS. The 2010 Dietary Guidelines—the best recipe for health? *N Engl J Med*. 2011;365:1563–1565.
- Katz S. Agri-industry vested interests exposed by low glycemic index pioneer. November 3, 2011. EDT Solo GI Nutrition Inc. http://www.prweb.com/releases/2011/11/prweb8926345.htm. Accessed December 2011.
- Brand-Miller J, McMillan-Price J, Steinbeck K, et al. Dietary glycemic index: health implications. J Am Coll Nutr. 2009;28(Suppl): 4465–449S.
- Jones JM. The Glycemic Index of Food. Ridgway, CO: Wheat Foods Council; 2002.
- Jones JM. The role of Glycemic Index & glycemic load on carbohydrate food quality: a status report, 2010. Wheat Foods Council. www.wheatfoodscouncil.org. Accessed October 1, 2001.

- Jones JM. Glycemic index: the state of the science, part 1—the measure and its variability. *Nutr Today*. 2012;47:207–213.
- Gaesser GA. CHO quantity and quality in relation to body mass index. J Am Diet Assoc. 2007;107:1768–1780.
- Pietiläinen KH, Korkeila M, Bogl LH, et al. Inaccuracies in food and physical activity diaries of obese subjects: complementary evidence from doubly labeled water and co-twin assessments. *Int* J Obes (Lond). 2010;34:437–435.
- Rossi M, Bosetti C, Talamini R, et al. Glycemic index and glycemic load in relation to body mass index and waist to hip ratio. Eur J Nutr. 2010 49:459

 –464.
- Mendez MA, Covas MI, Marrugat J, et al. Glycemic load, glycemic index, and body mass index in Spanish adults. Am J Clin Nutr. 2009; 89(1):316–322.
- Hare-Bruun H, Flint A, Heitmann BL. Glycemic index and glycemic load in relation to changes in body weight, body fat distribution, and body composition in adult Danes. Am J Clin Nutr. 2006;84:871–879.
- Tyrovolas S, Psaltopoulou T, Pounis G, et al. Nutrient intake in relation to central and overall obesity status among elderly people living in the Mediterranean islands: the MEDIS study. Nutr Metab Cardiovasc Dis. 2011;21:438–445.
- Du H, van der ADL, van Bakel MM, et al. Dietary glycaemic index, glycaemic load and subsequent changes of weight and waist circumference in European men and women. Int J Obes (Lond). 2009;33:1280–1288.
- Hui LL, Nelson EA. Meal glycaemic load of normal-weight and overweight Hong Kong children. Eur J Clin Nutr. 2006;60:220–227.
- Davis JN, Alexander KE, Ventura EE, et al. Associations of dietary sugar and glycemic index with adiposity and insulin dynamics in overweight Latino youth. Am J Clin Nutr. 2007;86:1331–1338.
- Nielsen BM, Bjørnsbo KS, Tetens I, et al. Dietary glycaemic index and glycaemic load in Danish children in relation to body fatness. Br J Nutr. 2005;94:992–997.
- Murakami K, Sasaki S, Okubo H, et al. Dietary fiber intake, dietary glycemic index and load, and body mass index: a cross-sectional study of 3931 Japanese women aged 18–20 years. Eur J Clin Nutr. 2007;61:986–995.
- van Bakel MM, Slimani N, Feskens EJ, et al. Methodological challenges in the application of the glycemic index in epidemiological studies using data from the European Prospective Investigation into Cancer and Nutrition. J Nutr. 2009;139:568–572.
- 21. Astrup A. How to maintain a healthy body weight. *Int J Vitam Nutr Res.* 2006;76:208–215.
- Moshfegh AJ, Rhodes DG, Baer DJ, et al. The US Department of Agriculture Automated Multiple-Pass Method reduces bias in the collection of energy intakes. Am J Clin Nutr. 2008;88: 324–332.
- 23. Davis MS, Miller CK, Mitchell DC. More favorable dietary patterns are associated with lower glycemic load in older adults. *J Am Diet Assoc*. 2004;104:1828–1835.
- Lv L, Yao Y, Wang L. Dietary glycaemic load and intakes of carbohydrates, fats and proteins in 1040 hospitalised adult Chinese subjects. Br J Nutr. 2011;106:1052–1057.
- Raatz SK, Torkelson CJ, Redmon JB, et al. Reduced glycemic index and glycemic load diets do not increase the effects of energy restriction on weight loss and insulin sensitivity in obese men and women. J Nutr. 2005;135:2387–2391.
- 26. Esfahani A, Wong JM, Mirrahimi A, et al. The application of the glycemic index and glycemic load in weight loss: a review of the clinical evidence. *IUBMB Life*. 2011;63:7–13.
- Thomas DE, Elliott E, Baur L. Low glycaemic index or low glycaemic load diets for overweight and obesity. Cochrane Database Syst Rev. 2007;18(3):CD005105.
- 28. Jones JM. Glycemic index: the state of the science, part 3: role in cardiovascular disease and blood lipids. *Nutr Today*. In press.
- Jones JM. Glycemic index: the state of the science, part 4: roles inflammation, insulin resistance and metabolic syndrome. Nutr Today. In press.

- Foster GD, Wyatt HR, Hill JO, et al. Weight and metabolic outcomes after 2 years on a low-CHO versus low-fat diet: a randomized trial. Ann Intern Med. 2010;153:147–157.
- Dansinger ML, Gleason JA, Griffith JL, et al. Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA*. 2005; 293:43–53.
- Shai I, Schwarzfuchs D, Henkin Y, et al. Weight loss with a lowcarbohydrate, Mediterranean, or low-fat diet. N Engl J Med. 2008; 359(3):229–241.
- Greenberg I, Stampfer MJ, Schwarzfuchs D, et al. Adherence and success in long-term weight loss diets: the Dietary Intervention Randomized Controlled Trial (DIRECT). J Am Coll Nutr. 2009;28:159–168.
- Vetter ML, Iqbal N, Dalton-Bakes C, et al. Long-term effects of low-CHO versus low-fat diets in obese persons. *Ann Intern Med.* 2010; 152:334–335.
- 35. Phelan S, Wyatt H, Nassery S, et al. Three-year weight change in successful weight losers who lost weight on a low-CHO diet. *Obesity (Silver Spring)*. 2007;15:2470–2477.
- 36. Larsen TM, Dalskov SM, van Baak M, et al. Diets with high or low protein content and glycemic index for weight-loss maintenance. *N Engl J Med.* 2010;363:2102–2113.
- 37. Papadaki A, Linardakis M, Larsen TM, et al. The effect of protein and glycemic index on children's body composition: the DiOGenes randomized study. *Pediatrics*. 2010;126:e1143–e1152.
- Burton-Freeman BM, Keim NL. Glycemic index, cholecystokinin, satiety and disinhibition: is there an unappreciated paradox for overweight women? *Int J Obes (Lond)*. 2008;32:1647–1654.
- Bornet FR, Jardy-Gennetier AE, Jacquet N, et al. Glycaemic response to foods: impact on satiety and long-term weight regulation. *Appetite*. 2007;49:535–553.
- 40. Wolever TM, Leung J, Vuksan V, et al. Day-to-day variation in glycemic response elicited by white bread is not related to variation in satiety in humans. *Appetite*. 2009;52:654–658.
- 41. Leeman M, Ostman E, Björck I. Glycaemic and satiating properties of potato products. *Eur J Clin Nutr.* 2008;62:87–95.
- 42. Niwano Y, Adachi T, Kashimura J, et al. Is glycemic index of food a feasible predictor of appetite, hunger, and satiety? *J Nutr Sci Vitaminol (Tokyo)*. 2009;55:201–207.
- 43. Saris WH. Glycemic carbohydrate and body weight regulation. *Nutr Rev.* 2003;61(5 Pt 2):S10–S16.
- 44. Bellisle F, Dalix AM, De Assis MA, et al. Motivational effects of 12-week moderately restrictive diets with or without special attention to the Glycaemic Index of foods. *Br J Nutr.* 2007;97:790–798.
- 45. Flint A, Gregersen NT, Gluud LL, et al. Associations between postprandial insulin and blood glucose responses, appetite sensations and energy intake in normal weight and overweight individuals: a meta-analysis of test meal studies. Br J Nutr. 2007; 98:17–25.
- 46. Trout DL, Hallfrisch J, Behall KM. Atypically high insulin responses to some foods relate to sugars and satiety. *Int J Food Sci Nutr.* 2004;55:577–888.
- Chaput JP, Tremblay A. The glucostatic theory of appetite control and the risk of obesity and diabetes. *Int J Obes (Lond)*. 2009;33:46–53.
- 48. Ciampolini M, Bianchi R. Training to estimate blood glucose and to form associations with initial hunger. *Nutr Metab (Lond)*. 2006;3:42.
- 49. Chaput JP, Tremblay A, Rimm EB, et al. A novel interaction between dietary composition and insulin secretion: effects on weight gain in the Quebec Family Study. *Am J Clin Nutr.* 2008;87:303–309.
- Mittelman SD, Klier K, Braun S, et al. Obese adolescents show impaired meal responses of the appetite-regulating hormones ghrelin and PYY. Obesity (Silver Spring). 2010;18:918–925.
- Zhang Z, Lanza E, Ross AC, et al. A high-legume low-glycemic index diet reduces fasting plasma leptin in middle-aged insulinresistant and -sensitive men. Eur J Clin Nutr. 2011;65:415

 –418.
- 52. Sculati M, Rossi F, Cena H, et al. Effect of dietary glycemic index on food intake, adiposity, and fasting plasma ghrelin levels in animals. *J Endocrinol Invest*. 2010;33:250–253.

- 53. Gilhooly CH, Das SK, Golden JK, et al. Food cravings and energy regulation: the characteristics of craved foods and their relationship with eating behaviors and weight change during 6 months of dietary energy restriction. *Int J Obes (Lond)*. 2007;31:1849–1858.
- 54. Keogh JB, Lau CW, Noakes M, et al. Effects of meals with high soluble fibre, high amylose barley variant on glucose, insulin, satiety and thermic effect of food in healthy lean women. Eur J Clin Nutr. 2007;61:597–604.
- 55. Lowe MR, Levine AS. Eating motives and the controversy over
- dieting: eating less than needed versus less than wanted. *Obes Res.* 2005;13:797–806.
- 56. Astrup A. Dietary management of obesity. *JPEN J Parenter Enteral Nutr.* 2008;32:575–577.
- Kong AP, Chan RS, Nelson EA, et al. Role of low-glycemic index diet in management of childhood obesity. Obes Rev. 2011;12:492–498.
- 58. Phelan S, Wyatt HR, Hill JO, Wing RR. Are the eating and exercise habits of successful weight losers changing? *Obesity (Silver Spring)*. 2006;14:710–716.

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