Glycemic Index: The State of the Science, Part 2:
Glycemic Index and Glycemic Load:
Roles in Weight, Weight Loss and Satiety

PART 2
Introduction

The worldwide ‘diabesity’ epidemic has renewed discussions about what is the optimal diet composition for promotion of weight maintenance and weight loss. Such discussions inevitably lead not only to calories, but also to the kind of calories. Specifically, the dietary ‘holy grail’ quest is delineating the ideal combination of fat, protein and carbohydrate (CHO) to prevent overweight, to promote weight loss and to help with weight maintenance. Most recently there has been a shift in emphasis of these discussions from being mostly about fat and fat quality to CHO and CHO quality.

The 2010 Dietary Guidelines Advisory Committee (DGAC) Report addresses both the obesity and carbohydrate issues with calls for decreased ‘consumption of energy-dense carbohydrates, especially refined, sugar-dense sources, to balance energy needs.’ (1) In addition, the popular press, as well as professional authors, have called for attention to CHO by suggesting that CHO quality be assessed by its glycemic response with either the glycemic index (GI) or glycemic load (GL) as the measurement. The basics about these measures and their use have been described recently. (2, 3)

The manipulation of dietary GI or GL for weight loss and maintenance has been the source of vigorous debate with strident proponents both for and against its use. Both sides are able to produce scientific studies in support of their position. For example, The European DioGENES (The Diet, Obesity and Genes) project in a recent review did not give the concepts glowing endorsements. (4) Here is a concluding statement from the report “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.” (4) Another review states that the use of table values to predict the glycemic index of meals is of little–if any–value. (5) Similarly evidence-based reviews supporting the work of the 2010 DGAC found little or no association between GI and GL and measures of body weight. (1)

The lack of association noted by the 2010 DGAC evidence-based review and report spawned sharp criticism by some bloggers and proponents of the GI/GL concept. They felt that evidence supporting its use such as that from Jenny Brand Miller, nutrition professor at University of Sydney in Australia, was not properly considered. Dr. Brand Miller and her group have published many papers suggesting that low GI

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diets help in weight loss and maintenance, especially in certain subpopulations. A recent review conducted by her research group (6) concluded that there is 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.” (6)

Since debate remains, this paper will look at GI and GL evidence and evaluate the state of the evidence with respect to preventing overweight and obesity, maintaining weight and losing weight. These data will be put into the context of carbohydrate in the diet and its role in weight control.

**CHOs, GI/GL and Body Weight**

**CHO and Body Weight** - A comprehensive review of carbohydrate intakes and body weight was published in 2007. This analysis showed that individuals with the highest carbohydrate intakes, as measured in large population databases throughout North America (Table 1), are associated with having the lowest BMIs. Further, the inverse association between carbohydrate intake and BMI is graded across quintiles, e.g. the greater the CHO intake, the lower the mean BMI. (7) Such intake data can be criticized as there are studies showing that those who are overweight underreport their food intake. (8)

**GI and Body Weight** - The data with respect to total CHO intake and BMI are consistent across the various cohorts, but those with respect to GI and GL are anything but consistent. (7) In three large cohorts of women and in a large and small cohort of men, counter to dogma of some popular press diets, as GI increased BMI decreased. In a multiracial cohort of both genders, BMIs were unrelated to dietary GI.

**GL and Body Weight** - For GL and BMI, the results were not consistent. Either the GL of the diet is unrelated to BMI or inversely related. In other words as GL went up, BMI went down. For those ingesting diets with the highest GL compared to those ingesting those with the lowest, the BMI was between 0.3 to 2.1 BMI units lower. In no case did the data support the prevailing belief that with an increase in GL there is an attendant increase in BMI. (7) These cohort findings fail to support the thesis that higher GI or GL is associated with higher BMIs. In fact, it appears that analysis of associational data in cohort studies indicate that the converse is true.

Studies published after the 2007 comprehensive review of the cohort data continue to show great variability. A study in Italian men and women also showed an inverse association between GI and GL and BMI. (9) In other words as GI or GL increased, BMI decreased. However, data for subpopulations

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**Table 1: Databases Showing Inverse Associations Between Carbohydrate Intake and BMIs**

<table>
<thead>
<tr>
<th>Cohort</th>
<th>n</th>
<th>Quintile (Quartile) of Carbohydrate Intake</th>
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<tbody>
<tr>
<td>Nurses’ Health Study I</td>
<td>71,919 women</td>
<td>25.2 25.2 25.1 24.9 24.7</td>
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<tr>
<td>Nurses’ Health Study II</td>
<td>90,655 women</td>
<td>26 25</td>
</tr>
<tr>
<td>NHANES</td>
<td>6,125 women</td>
<td>26.3 26.4 26.1 25.9 25.5</td>
</tr>
<tr>
<td>Nurses’ Health Study II</td>
<td>91,249 women</td>
<td>24.7 24.6 25.2</td>
</tr>
<tr>
<td>Women’s Health Study</td>
<td>38,446 women</td>
<td>26.7 26.3 26.1 25.7 25.2</td>
</tr>
<tr>
<td>Health Professional’s Follow-Up Study</td>
<td>39,926 men</td>
<td>26.1 25.9 25.6 25.3 24.8</td>
</tr>
</tbody>
</table>

Ref. 7 [U.S. National Health and Examination Survey (NHANES)](Ref. 7) [The Consumer Survey of Food Intake of Individuals (CSFII)](Ref. 7) [The Nurses’ Health Study, I & II (NHS)](Ref. 7) [The Women’s Health Study (WHS)](Ref. 7) [Health Professionals Follow-Up Study (HPFS)](Ref. 7) [Prostate, Lung, Colorectal, and Ovarian Cancer Screening Trial (PLOC)](Ref. 7) [The Canadian National Breast Screening Study](Ref. 7)
of a Danish cohort gave a different picture from that observed in the Italian study. (10) There was a positive association between GI (but not GL) and BMI in Danish women (not men) with the association becoming more pronounced if the women were sedentary. (10)

**GI and GL and Weight in Youth** - Associations for children and teenagers also show inconsistency both within and across populations. Most studies in the recent review fail to show significant associations between GI and BMI or other measures of excess weight. This was the case in school-age children in Hong Kong, Latino teens, Danish girls under 16 years old and Danish boys under 10 years old (3). However, in Danish boys 10-16 years old, there was a positive association between GI and skinfold measures of fatness. (3) Also in 18- to 20-year-old Japanese dietetics students, both GI and GL were positively related to BMI.

**Potential Reasons for Inconsistency Across Studies**

Thus, the bulk, but not all, of the findings fail to support the thesis that high GI or GL is associated with higher body weights. There are several potential reasons for the inconsistency in the data. First, the data may accurately reflect the situation, e.g. neither GI nor GL of the diet is an important factor in determining BMI. Second, the arbitrary assignment of a food or a diet into a low, medium or high category certainly must affect the results. For example, for the cohorts in the 2007 review (7), 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 the GI categories is especially troubling in light of lack of precision and accuracy of the measure and errors associated with using table values to assign GI or GL to diets and foods from food frequencies. (2,3) These food frequencies often fail to have information of ripeness or cooking methods—just a few of many factors that affect GI and GL. (2, 3, 11) One review stated that the use of values from the tables is so variable as to render them of little use. (5) Fourth, underreporting of food intake (8, 12) by those who are overweight certainly affects the accuracy of the intake data, and would certainly impact GI and GL values assigned to foods and diets. Fifth, inadequate cohort segmentation can also mask an effect that is occurring in one segment. Subdivision of the population by age, gender or activity level may be required. Sixth, the measure of overweight may be important. In some studies BMI was unrelated but other measures such as skinfold and adiposity showed an association. Seventh, 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 one of these components alone and in combination has been associated with improved nutrient profiles and lower body weight measures. (13)

In contrast, some low GI or GL diets may be very low in CHO, fruit, whole grains and dietary fiber. Such diets may be high in meat and saturated fat. The latter foods alone and as part of the dietary patterns have been shown to increase risk of obesity and chronic disease. Further, 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 astonishing that the relationship between GI and GL and body weight is quite variable. In summary, the weight of existing evidence indicates that dietary GI and GL are not related to BMI or other measures of body weight. Further current data fail to support the thesis that higher GI or GL of the diet is related to increased body weight.
GI/GL and Weight Loss

GI/GL and Short-term Weight Loss
Weight maintenance and weight loss may require different dietary strategies. Some weight loss regimens have low- or no-CHO \(^1\) as a critical diet pillar. Others allow CHOs only if the CHOs are low GI or GL. Proponents of this latter strategy cite testimonials from mainstream authors as well as data from well-controlled, short-term studies. Many studies verify that greater weight loss occurs in the short term (less than 6 months) when dieters employ a low GI and GL strategy compared to other diet strategies. However, when calories are strictly controlled, the GI or GL of the diet makes no difference \((14)\). Such findings were affirmed in a Cochrane review (the gold-standard for quality associated with the review process) \((15)\). This review concluded that low GI/GL diets were shown to promote a small (~1 kg), but statistically significant, greater weight loss than other diets. Accompanying the slightly greater weight loss is a greater decrease in total and LDL cholesterol than seen with other diets. (This will be discussed in a subsequent paper that addresses GI/GL, coronary disease and blood lipids.)

GI/GL and Long-term Weight Loss and Weight Maintenance
Low CHO and GI and GL diets appear to fare well in the short term, but there are few studies that follow the dieter beyond 6 months. Of those that do, results do not provide an overwhelming endorsement, but show that there might be a small advantage not with respect to weight but with respect to blood lipid profiles. One recent randomized trial of 330 obese individuals (mean BMI=36) in a dieting situation with intensive behavioral interventions \((16)\) showed that a reduced CHO, low GI diet caused the same weight loss over the 2-year period as a traditional low fat diet.

However, there was slightly better lipid profile with the low GI diet. Both diet groups had participants who did not stick to the diet for the full 2 years. Sticking to the diet is important as was shown in a 1-year long randomized trial \((n=160)\). Those who continued for one year with their assigned diet – no matter which diet – lost weight. Actual pounds shed were not significantly different in the various diets, but diets most variant from mainstream diets such as the low-CHO (low GI and GL) Atkins diet had the most dropouts. \((17)\) Similar results both in weight loss and tendency to drop out were observed in an intervention comparing Mediterranean and low fat diets to a low-CHO diet. \((18)\) A much larger number on the Atkins-type diet (43%) compared to other diets reported themselves as non-compliant after 24 months. Data from the National Weight Control Registry (NWCR) of Successful Losers (persons who have lost 30 pounds or more and kept it off for more than 1 year) have only 10% of the participants reporting the use of a low–CHO diet. However, those reporting this diet strategy were equally likely to keep the weight off as those who used other diet methods. \((19)\) A 3-year study suggests that little, if any, advantage of low GI/GL diets in terms of weight loss. While weight loss on the low GI/GL was greater at 6 months, weight loss plateaued at 6 months. At the end of 3 years, there was greater tendency to regain weight with the low-CHO diet than with the low-fat diet. The net result is that over the 3-year period, loss was greater with the low-fat diet than with the low GI/GL diet. \((20)\)

Potential Mechanisms for Low GI/GL and Short-Term Weight Loss
Numerous reasons as to why low GI/GL diets increase weight loss have been put forward. These include effects on satiety, hormone changes with GI or GL, and psychosocial aspects of eating and behavior associated with dieting.

\(^1\) Low- and no-CHO diets are inherently low GL because GL always considers the GI of the CHO and the total amount of CHO.
**GI/GL and Satiety** - Increased satiety is one proposed way that low GI/GL foods and diets promote weight loss. A systematic review of 32 human intervention studies showed low GI/GL foods or meals to be more satiating in the short term than high GI/GL foods or meals. (21) Studies from this review were packaged as a dossier and submitted to the European Food Safety Authority (EFSA) to substantiate claims such as 'low-GI foods help one to feel fuller for longer than equivalent high-GI foods.' Despite the seeming concordance presented in the EFSA dossier, not all studies show low GI foods are associated with satiety. In some instances, high GI foods offered greater satiety. Boiled potatoes, a high GI food, were given the highest satiety index among foods. (22) One study with 22 overweight women showed that high GI meals compared with low GI meals better suppressed hunger and subsequent food intake better and caused greater satiety. (23)

Since satiety is impacted by many things including sensory aspects of the food, it is often difficult to attribute that one specific aspect of the food such as the GI or GL caused greater satiety. In a study designed to address this, GL was altered by feeding different amounts of white bread, a high GI food, to control for sensory aspects of satiety. (23) The increased GL changed in this way had no effect on satiety or hunger in the 2-hour period after eating.

Another difficulty with satiety studies is translating increased satiety into reduced food intake. One study using low GI foods with the WeightWatchers® POINTS program showed an initial effect during the early weeks of the study. Both ratings of hunger – especially in the afternoon – as well as desire to eat were consistently lower for those eating the low GI modification. However, after 12 weeks there was no difference between the groups in weight loss or other measures (24).

**GI/GL and Hormones** - Low GI/GL diets are touted as helping dieters by controlling the release of glucose and insulin and other hormones that affect appetite (Table 2).

**GI/GL and Glucose and Insulin** - It is true that low GI foods cause a lower rise in both glucose and insulin, (26) but the differences are not consistently associated with satiety. (27) In fact, the nearly 50-year-old glucostatic theory of Jean Mayer suggests that increased levels of blood glucose promote satiety.

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Where Released</th>
<th>Effect on Feeding</th>
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<tr>
<td>Insulin</td>
<td>Pancreas</td>
<td>Decrease</td>
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<tr>
<td>PYY</td>
<td>Intestine</td>
<td>Decrease</td>
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<td>Leptin</td>
<td>Fat Cells</td>
<td>Decrease</td>
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<tr>
<td>Cholecystokinin</td>
<td>Stomach</td>
<td>Decrease</td>
</tr>
<tr>
<td>Ghrelin</td>
<td>Stomach</td>
<td>Increase</td>
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<tr>
<td>Neuropeptide Y</td>
<td>Mucosa of Illeum and Colon</td>
<td>Increase</td>
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Despite variability in the literature, a group of experts convened to assess the data by the Carbohydrate Task Force of the International Life Sciences Institute, Japan (25) concluded that ingestion of high-GI foods increased hunger and lowered satiety in short-term human intervention studies, but hunger and satiety after the ingestion of foods with varying GIs were inconsistent in long-term human intervention studies.

Variability is a watchword in satiety studies. Different protocols including both time of measure and actual measurement can yield different outcomes. Further, satiety is not simply a biological factor but has a sensory component. Diners still want to have pumpkin pie after a holiday meal, even though from a caloric and other standpoint they are sated. Thus, it is difficult to conclude that the GI values of foods or mixed meals are a valid long-term predictor for appetite, hunger and satiety. (25)
This theory is consistent with recent findings that low glycemia is linked to weight gain and is a strong predictor of the amount of weight regained after weight loss. (28) The ability of blood glucose to signal hunger may depend on a person’s metabolic state. Some overweight individuals were only able to respond to low blood sugar if they were also exercising. (29) One study in normal men suggested that the glycemic response had little effect on short-term appetite sensations, but a low-glycemic index meal reduced subsequent energy intake. In normal men, only postprandial insulin was shown to affect short-term appetite sensations. (27)

So is the insulin response the answer since insulin release in response to high GI or GL of foods causes release of hormones that can affect hunger? Once again the answer is not clear-cut but appears to depend on the metabolic state of the eater. Those carrying excess weight may have reduced insulin sensitivity, which not only blunts response to insulin, but also blunts the body’s ability to respond to many other hormones and signals affecting appetite and satiety. (30, 31)

GI and GL and Other Hormones that Affect Eating - Leptin and gherlin are some of a number of hormones that affect hunger and satiety (Table 2). In normal weight subjects, leptin is released from fat tissue. Once in the blood stream the hormone travels to the hypothalamus and a signal to stop eating is released. Insulin levels and rate of glucose uptake affect the amount of leptin released, but their effectiveness may vary depending on insulin resistance. (30, 31)

Gherlin is a hormone that promotes eating. CHO–rich meals suppress gherlin. The theory is that low GI-CHOs cause adipose tissue to release leptin and the stomach to inhibit gherlin release. Thus both hormones would reduce appetite. However, actual effects appear to vary with baseline insulin levels of the subject. (32)

Other hormones such as cholecystokinin (CCK) can also inhibit eating behavior (Table 2). Some studies show that high GI meals were more sating because they caused greater CCK release. (23)

Sensory and Other Non-Physiological 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.

One factor is ease of following the diet. Low GI or GL diets appear simple. Calorie counting is unnecessary. Users need to consider only one aspect of the food. They can consult published tables and categorize foods as low, medium or high GI or GL. They then are able to decide if a food is allowed or not.

Food palatability is another aspect that affects satiety. Humans and rats alike will seek energy dense foods and over-consume highly palatable foods. A recent study showed that people on a ‘diet’ craved foods, which delivered a high energy density and fat content and were low in protein and
fiber, regardless of whether the diet had a high or low GL. (33) Further, the ability to compensate for calories is compromised by foods high in sugar and fat. Since high GI/GL foods and diets may be both highly palatable and energy dense, their lack of impact on satiety and subsequent caloric intake might be erroneously attributed to GI/GL when it should be attributed to other aspects. In other cases 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 to the same diet formulated with a higher GI food such as wheat. 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 this experimental condition the lowered glycemic and insulinemic response did not translate into lower caloric ingestion.

GI/GL Diets and Psychological Factors - Another important factor for some dieters is the seeming freedom offered by the diet. Statements such as “You can eat as much as you want as long as it is of allowed foods” or “you can eat until sated and do not need to feel hunger” are appealing to some dieters. Thus low GI or GL diets may appeal to those who are at one end of the scale for either the ‘restraint’ or ‘disinhibition’ scales. (34)

Table 3: Several Low GI Diets – Three Low CHO Meal Plans. They show the very different diet quality of three diets considered low CHO.

| Breakfast | 2 Eggs  
1 Apple sliced very thin  
½ Banana  
Ezekial bread  
1 Orange | Egg recipe*  
2 Eggs  
1/3 c. heavy cream  
1/3 c. shredded cheese  
1/3 c. cooked meat or veg.  
Buttered spinach | Hot and creamy barley with toasted almonds  
Grapefruit |
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<tbody>
<tr>
<td>Snack</td>
<td>Nuts, 1 oz</td>
<td>Bacon rinds</td>
</tr>
</tbody>
</table>
| Lunch | Tuna fish stuffed tomato with hard boiled eggs  
Berries, 1 c | | Greek salad with feta |
| Snack | Toasted garbonzo beans | Snickers Bar | Cherries |
| Dinner | Grilled chicken  
Tomato, arugula, pinenut salad  
Broccoli  
Apple custard | Triple cheeseburger without the bun  
Lettuce and tomato  
Broccoli  
Cheesecake | Filet of sole with butter sauce  
Collard greens with garlic |

Ref. 34
GI/GL and Nutritional Quality of the Diet

Diets that deliver adequate nutrition may reduce the desire to eat and impact satiety. (33) Low GI/GL diets may be constituted so they are replete with fruits, vegetables, and whole grains. These foods are rich in nutrients and low in calories. Also, they tend to have high volume per calorie and offer dietary fiber, resistant starch, and slowly available CHO, all of which have been shown to offer satiation and are associated with reduced body weight. Strict low CHO diets are also low GI and GL because of the restriction of all CHO foods. Such a diet plan may reduce overall food intake because choice of foods is limited and the diet can become boring. Three potential eating plans, which are constituted with low GI foods, show their great difference in diet quality (Table 3).

GI/GL and Satiety - The 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 as factors from baseline hunger to palatability and psychological state affect the measurement. 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 promise of greater weight loss promised in popular low CHO and low GI diets books often fails to materialize. When compared to a low fat diet, in the initial phase a slightly greater weight loss (~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 picture with the low CHO, low GI diet. (This will be addressed in an upcoming article.) While 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 may be more difficult than reduced calorie plans that require less extreme modifications. Some suggest that there is a role for low-GI diet in the management of very overweight children and adults. (36, 37) 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, high in low-energy density carbohydrates (fruit, vegetables, and whole grain products).” (34) Such diets haven’t been studied for longer periods of time.

In terms of weight loss a recent review (34) stated that reduction in dietary fat and increase in fiber were the strongest predictors of weight loss and diabetes-protective effects. Further the authors suggested that it remains to be shown whether a low-GI diet provides any benefit to weight control beyond a possible short-term impact.
References

2. Jones JM Nutrition Today