

# Does Eating High-Glycemic-Index Foods Lead to Obesity, Diabetes, and Heart Disease?

By ACSH Staff — January 1, 2000

Does the relative ability of individual foods and diets to raise blood glucose have a unique or tremendous influence on human health?

According to four of the most popular diet books of recent years *The Zone* (1995), whose primary author is Barry Sears, Ph.D., *Protein Power* (1997), by medical doctors Michael R. Eades and Mary Dan Eades, Dr. Atkins' *New Diet Revolution* (1997), by Robert C. Atkins, M.D., and *Sugar Busters! Cut Sugar to Trim Fat* (1998), by H. Leighton Steward and associates diets high in carbohydrate (CHO) are responsible for insulin increases that inhibit fat-burning and lead to obesity, type 2 diabetes (non-insulin-dependent diabetes mellitus), and atherosclerosis (specifically, coronary heart disease, or CHD). More recently, actress Suzanne Somers has joined the anti-CHO bandwagon, with *Suzanne Somers' Get Skinny on Fabulous Food* (1999).

A glycemic index (GI) is the degree to which a food ingested alone increases the concentration of glucose in the blood comparative to a standard, such as white bread. The authors cited above profess that foods with high glycemic indexes i.e., foods whose ingestion rapidly results in high glucose concentrations are, primarily because of the insulin response their digestion elicits, the most dangerous foods. They further contend that diets relatively high in protein and/or fat are the key to reducing blood insulin, losing weight, and improving health.

**Last year the American Diabetes Association rescinded its advocacy of high-carbohydrate diets for all diabetics . . . .**

There is a grain of truth to what these authors say. The trend of scientific evidence from CHO research suggests that, at least for some individuals, both the total quantity and the proportion of the various carbohydrates in a diet may figure complicatedly in feeding behavior, metabolism, and health. But consumers who entertain the assertions of diet-book authors must consider those assertions with more than a grain of salt. For example, the authors named above affirm that beets and carrots are fattening which is absurd.

## **The Basis of the Glycemic Index**

Glycemic indexes are based on the human physiologic response, in terms of plasma glucose concentrations, to ingestion of a food specimen that contains 50 grams of digestible CHO, comparative to the response to an equivalent intake of either glucose or white bread. The larger the blood-glucose increase, the higher the GI. GIs have been assigned to more than 600 foods, but the typical American supermarket carries over 6,000 foods. So it is likely that many persons are regularly consuming many foods that have not been tested regarding GI. Moreover, cooking or otherwise preparing a food can alter its potential blood-glucose effects. As a rule, processing a

nonprocessed food amplifies its ability to increase plasma glucose concentrations. In any case, the relationship of high-GI foods and/or a high-CHO diet to the development of obesity, type 2 diabetes, and cardiovascular disease (CVD) remains to be puzzled out.

**Diminishing insulin responses does not, as some authors claim, prevent or diminish either conversion of excessive energy from dietary CHO to fat or storage of such fat.**

### **The Pima Paradox**

Epidemiological studies have not lent much credence to the notion that high-CHO diets in general, or those consisting mostly of high-GI foods, lead to obesity, diabetes, and CVD. The contention that the quantity or percentage of CHO in a diet, or the relative ability of individual foods or diets to increase plasma glucose concentrations, is the primary determinant in the development of obesity, insulin resistance,\* and type 2 diabetes is inconsistent with some observations of human populations. For example, although the diet of the Pima Indians living in northern Mexico consists largely of potatoes and corn tortillas both high-CHO, high-GI foods these Pimas weigh 60 to 65 lbs less than the Pima residents of Arizona, who consume much more protein and fat than do their Mexican cousins.

Foods high in protein and/or fat tend to have GIs below those of high-CHO foods. But by age 50 more than half of the Arizonan Pimas become obese and develop type 2 diabetes, whereas among the Mexican Pimas type 2 diabetes is rare and occurs mostly among the elderly. Because the Mexican Pimas and the Arizonan Pimas are of the same genetic stock, it is unlikely that genetic factors can account for this disparity. That the Mexican Pimas are much more physically active than their Arizonan counterparts is probably a crucial factor both in the diabetes disparity and in the body-weight disparity.

Scientific findings have positively associated (1) diets high in fat (especially saturated fat) and in refined CHOs (particularly sugars) and (2) the development of insulin resistance. On the other hand, high-complex-CHO diets and improvement of sensitivity to insulin appear associated.

### **Priorities**

On September 1, 1998, the American Heart Association (AHA) released a report stating that diets very low in fat (i.e., diets in which less than 15 percent of calories come from fat) may carry serious health risks for some persons and are not recommended for the public. The authors noted that, according to many research findings, very-high-CHO diets reduce blood HDL-cholesterol (a compound that tends to retard heart disease) and increase blood triglycerides (TGs) more than do diets relatively high in unsaturated fat.

But in every study that has shown adverse effects of high-CHO diets on blood lipid concentrations, the high-CHO diet and the diet higher in fat were isocaloric, that is, equally restricted calorically. In similar studies without calorie controls, adverse effects have been minimal because for most persons, calorie intakes on diets reduced in fat tend to be less than those on diets unmodified with respect to fat. And in one study in which isocaloric diets were used, following the high-CHO diet, which had a low GI, resulted neither in a decrease in blood HDL-cholesterol nor in an increase in blood TGs. Therefore, it is possible that high-CHO diets adversely affect blood lipid concentrations

only when they consist largely of processed high-CHO, high-GI foods and one's calorie intake is at least that which one would have on a diet relatively high in fat.

**It has been shown experimentally that humans consume high-carbohydrate foods at caloric levels below those at which they consume fat-enriched versions of those foods . . . .**

Last year the American Diabetes Association (ADA) rescinded its advocacy of high-CHO diets for all diabetics and took the stand that the composition of diabetic diets should be individualized. Because body weight, activity level, endogenous insulin output, degree of insulin resistance, and risk factors for other diet-related diseases vary widely among diabetics, that their diets should be individualized almost goes without saying. The ADA's rescindment stemmed largely from short-term studies that had associated high-CHO diets (relative to diets higher in unsaturated fat), potentially adverse blood-lipid changes, and little or no improvement in sensitivity to insulin and in blood-glucose concentrations. But it was expected in these studies that the high-CHO-diet intake would be calorically equal to the high-fat-diet intake. Furthermore, the high-CHO diets in these studies consisted largely of highly processed CHO-rich foods, most of them high-GI foods. It is too early to generalize from the findings of such studies applicably to all type 2 diabetics.

The ADA does not recommend taking GIs into account in diabetic meal planning. It also has maintained that the cumulative GIs of mixed meals cannot be extrapolated reliably. The scientific evidence, however, suggests that such GIs can be dependably extrapolated. In any case, the ADA advises giving top precedence in the planning of diabetic diets to CHO quantity rather than to CHO sources.

Diabetes mellitus increases the risk of developing CVD, and most type 2 diabetics in the United States are overweight. Thus, for most of them, top precedence should probably be given to the calorie content of the diet in conjunction with the diet's ability to satisfy the desire to eat. It appears that (a) reducing the intake of calories, saturated fat, trans fatty acids, \*\* salt, and cholesterol and (b) optimizing both fiber intake and exercising are each more important in type 2 diabetes than are the CHO quantity, CHO percentage, and cumulative GI of the diet. With type 1 diabetics\*\*\* it is likely that regularity in daily CHO consumption in terms of quantity and sources helps to limit blood glucose concentrations.

### **Insulin Scores and GIs**

The insulin score is a measure of bodily insulin output in response to ingestion of a food specimen of a specified caloric value (usually 250 kilocalories). The insulin scores and GIs of foods low in both fat and protein correspond. Because CHO is absorbed only from the small intestine and dietary fat in the stomach slows gastric emptying, a mixed meal high in fat will usually reduce the GIs of the high-CHO foods in it to which fat has been added. But while dietary fat tends to decrease the GIs of CHO-rich foods, it magnifies the insulin-output response to rises in plasma glucose. Therefore, the insulin scores of high-fat foods generally are much higher than those that their GIs might suggest. For example, the GI of ice cream, which is high in fat, is below that of carrots or potatoes, but the insulin scores of these three foods are similar.

Dietary protein attenuates but prolongs the glycemic response to a meal. This effect may be an advantage in cases of type 1 diabetes, in which large glycemic swings can make effective

treatment more difficult. In nondiabetics, ingestion of protein and/or fat normally induces pancreatic emission of insulin. Ingestion even of foods containing little or no CHO elicits a fairly substantial insulin response despite having little effect on plasma glucose concentrations. It is simplistic to say that increasing consumption of fat and protein and decreasing CHO intake will dramatically reduce the need for insulin and/or reduce insulin secretion.

In any event, diminishing insulin responses does not, as some authors claim, prevent or diminish either conversion of excessive energy from dietary CHO to fat or storage of such fat. Excessive dietary energy from any source can become stored fat. In a six-week study of overweight type 2 diabetics, published in a 1992 issue of *Diabetes Care*, T. Wolever and colleagues found that following a calorically restricted, high-GI diet had resulted in about as much weight loss as had following an equally restricted low-GI diet.

### **The Satiety Index and GIs**

In dietetics, "satiety" refers to the effects of a food or a meal on appetite after eating has concluded. One possible predictor of the calorie-intake and body-weight effects of the ingestion of various foods is how diminished one's desire for food is after consuming various foods at specific caloric levels. One such measure is the satiety index (SI), which is based on subjects' grading of postprandial appetite satisfaction and on their calorie intake two hours after a standardized meal. Theoretically, a high-SI food would be likelier than a low-SI food to satiate one's desire to eat, to slow the development of hunger, and to contribute to the prevention of excessive calorie intakes. It seems probable that a diet composed of higher-SI foods would limit hunger and thus facilitate limiting calorie intake and preventing weight gain.

**There is some evidence that, at the same caloric level, dietary carbohydrate, protein, and fat promote appetite satisfaction to different respective degrees.**

The notion that a high-CHO, high-GI diet leads to obesity rests ultimately on the assumption that SIs and GIs correlate. Whether or not following a calorically unrestricted diet composed of higher-SI foods would have a long-term effect in limiting calorie intake remains to be learned. But that the potato which is among the highest-GI foods has the highest SI so far assigned is incompatible with the claim that consuming high-GI foods causes insulin-output excesses and thus leads to overeating. Indeed, other high-GI foods have SIs higher than those of many low-GI, high-fat foods.

The insulin output in response to a meal corresponds much more with the caloric value of the meal than with its cumulative GI or its ratio of CHO to fat or protein. There are no credible-research findings suggesting, as some popular- diet-book authors claim, that the consumption of beets, carrots, potatoes, and other high-GI foods is conducive to obesity while the consumption of bacon and cheese omelets is not.

### **Energy Density, GIs, and Satiety**

In nutrition, the term "energy density" (ED) refers to the concentration of physiologically releasable energy in a food or group of foods of a specified weight. The ED of the Arizonan Pimas' diet is higher than that of the diet of their Mexican cousins. This ED difference is partly responsible for the difference in the prevalence of obesity and type 2 diabetes between the two groups. It has been

shown experimentally that humans consume high-CHO foods at caloric levels below those at which they consume fat-enriched versions of those foods, even when the high-CHO foods and their fat-enriched counterparts are equally palatable. But it has likewise been shown that, when the ED of a calorically unrestricted high-fat diet and that of a calorically unrestricted diet higher in CHO are equal, calorie intakes do not necessarily differ between them. Indeed, clinical research has shown that ED is primarily responsible for calorie-intake differences in calorically unrestricted diets both those low in fat (i.e., diets about 20 percent of whose calories come from fat) and those high in fat (i.e., diets about 40 percent of whose calories come from fat).

**Exercising even relatively briefly can moderate insulin resistance for about 24 hours.**

Of course, because adding fat to any food always increases the item's ED and most naturally occurring high-fat foods have high EDs, following a high-fat, low-ED diet would be very difficult. Such a diet would consist largely of stir-fried vegetables and tofu.

The EDs of fresh fruits and vegetables, nonfat dairy products, very lean animal-protein foods, and many whole-grain foods vary from very low (65 kcal/lb) to moderate (550 kcal/lb). It is precisely because the EDs of diets relatively high in fat are, in general, higher than those of diets relatively high in CHO that, irrespective of GIs, the former diets are greater contributors to calorie-intake excesses, weight gain, and metabolic problems.

Both the GIs and the EDs of most highly processed CHO-rich foods are high. Consuming CHO-rich foods whose EDs are high is largely responsible for lack of satisfaction of the desire to eat. In the U.S., high-GI foods for instance, "fat free" bakery products (most of whose EDs are high) tend to have low SIs. Madison Avenue has tried to make foods labeled "fat free" look nonfattening. It is reasonable to assume that experience with such products disillusioned many chronic dieters and disposed them toward low-CHO- diet gurus, who condemn high-CHO diets across the board but particularly damn high-GI foods most of which have high EDs. But the SI of a diet is a more important weight-control factor than is either GI or the percentage of calories from CHO.

There is some evidence that, at the same caloric level, dietary CHO, protein, and fat promote appetite satisfaction to different respective degrees. Of these macronutrients, dietary fat has the least satiety value. But in a recent randomized six-month study of obese subjects on calorically unrestricted, "fat reduced" diets comparable in ED, A. R. Skov and associates found that substituting protein for some of the CHO in a diet had led to significantly more weight loss than had nonsubstitution. This finding has not been replicated, however.

Substituting low-fat animal products or other foods relatively high in protein for some of the sugar and some of the highly processed CHO-rich foods in a diet might definitively turn out advantageous for some individuals. But considerably increasing dietary protein can contribute to osteoporosis and reductions in kidney function. Until scientific research uncovers substantially more about the long-term effects of very-high-protein diets (i.e., diets at least 25 percent of whose calories come from protein), it would be inappropriate to recommend such diets for long-term weight management.

In a study published in *The American Journal of Clinical Nutrition*, K. H. Duncan and colleagues compared the effects of two calorically unrestricted diets on obese and non-obese subjects: (1) a

low-ED (318 kcal/lb), high-fiber diet consisting primarily of minimally processed plant foods, and (2) a high-ED (681 kcal/lb), high-fat, low-fiber diet. The researchers found that, while both food acceptance and satiety ratings on the low-ED diet had been equivalent to such acceptance and ratings on the high-ED diet, calorie intake on the low-ED diet had been about half of that on the high-ED diet.

## **Exercise**

Exercising even relatively briefly can moderate insulin resistance for about 24 hours. Exercising reduces liver and muscle supplies of glycogen (the main form in which carbohydrates are stored in humans) and thus results in rapid uptake of blood sugar by these tissues. The muscles of sedentary persons are full of glycogen and thus resist insulin. Research has also shown that, on unrestricted diets, activity increases do not automatically lead to corresponding increases in calorie intake. This may be the main reason that relatively active persons are less likely to gain weight or to develop insulin resistance or type 2 diabetes than are relatively sedentary persons.

**. . . [R]esearch has shown that, comparative to starch . . . dietary fructose raises blood LDL-cholesterol . . . .**

## **High-CHO Diets, High-GI Diets, and CHD**

Solid scientific research has linked (1) hyperinsulinemia due to insulin resistance and (2) an increase in the risk of developing ischemic heart disease (IHD, characterized by atherosclerosis of the coronary arteries). Such research has led to speculation that high-GI foods may promote IHD. The Nurses' Health Study has uncovered a positive correlation of high GIs and women's risk of having a heart attack. Another study, published last year in *The Lancet*, associated a diet relatively high in high-GI foods and relatively low serum HDL-cholesterol concentrations.

As suggested above, however, it is simplistic to claim that consumption of high-GI foods results in hyperinsulinemia and thus leads to adverse changes in blood lipid concentrations and to atherosclerosis. For example, findings from a study published in *The Journal of the American College of Cardiology* in 1998 suggest that large insulin outputs promote coronary atherosclerosis only when they are very disproportionate to increases in blood glucose levels as occurs, largely because of insulin resistance, in type 2 diabetes and syndrome X (a combination of hyperlipidemia, hypertension, obesity, and type 2 diabetes). The researchers found that insulin resistance is a risk factor for IHD; they also found, however, that without insulin resistance, relatively high blood insulin concentrations were not associated with an increase in the risk of developing IHD.

In a study published in 1997 in a supplement to *The Canadian Journal of Cardiology*, researchers found that even without weight loss, following a calorie-controlled, high-CHO diet composed largely of minimally processed, high-fiber foods comparative to following a calorically equal diet characterized by foods that were more processed had resulted in a decrease in blood TG concentrations. They concluded: "[T]here appear to be important cardiovascular benefits from choosing a plant based diet over a convenience-food based diet for meeting national dietary guidelines . . . ."

Unlike most simple sugars, fructose (a very sweet sugar that occurs especially in fruit juices and honey) has a low GI, but research has shown that, comparative to starch (whose GI is high), dietary fructose raises blood LDL-cholesterol, an excess of which is a risk factor for CVD. There is also scientific evidence that sucrose (commonly used as table sugar), whose GI is lower than the GIs of many high-starch foods, increases blood concentrations of cholesterol and TGs when it replaces dietary starch; and that vegetables and whole grains, when they replace dietary sucrose, improve blood lipid concentrations.

Nutrition scientists recognize that GI is not the only characteristic of high-CHO foods responsible for how they affect blood lipid concentrations and CVD risk.

### **The Bottom Line**

Credible scientific evidence is absent for the claim that a diet relatively high in protein and fat and consisting only of low-glycemic-index foods is the key to preventing and treating obesity, type 2 diabetes, and ischemic heart disease. It has, however, increasingly become apparent that which types of carbohydrate are ample in one's diet can affect the desire to continue eating; calorie intake; body weight; and blood concentrations of glucose, insulin, and lipids and probably the risk of developing type 2 diabetes and IHD.

Taking GIs into account may be somewhat useful, particularly in planning meals for diabetics. But in general it appears that the best diet for preventing and treating obesity, type 2 diabetes, and cardiovascular disease is one that:

*\* is high in minimally processed whole-grain foods for example, brown rice, cooked whole-grain cereals, corn, and whole-wheat pasta;*

*\* is high in fruits, vegetables, and starchy foods such as beans, lentils, peas, and potatoes;*

*\* includes nonfat dairy products in moderate quantities; and*

*\* includes, in small quantities, seafood and/or very lean poultry or meat.*

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\*a state in which a given blood concentration of insulin has a biological effect less than that expected

\*\*A trans fatty acid is an unsaturated fatty acid (specifically, one with at least one double bond) having an unusual structure that renders it apt to raise plasma cholesterol.

\*\*\*Type 1 diabetes stems from the immune system's destruction of beta cells, usually develops in childhood or adolescence, and is usually treated with daily insulin dosing.

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