Do Glycemic Index, Glycemic Load, and Fiber Play a Role in Insulin Sensitivity, Disposition Index, and Type 2 Diabetes?

In this issue of Diabetes Care, Liese et al. (1) report on the relation of dietary glycemic index, glycemic load, carbohydrate, and fiber intake to measures of insulin sensitivity, insulin secretion, and adiposity in the Insulin Resistance Atherosclerosis Study (IRAS). The evaluation was confined to those with normal or impaired glucose tolerance and did not include diabetic individuals. As the authors state, there has been no previous large epidemiologic study relating glycemic index and glycemic load to direct measures of insulin sensitivity and insulin secretion, whose dysfunction are the two hallmarks leading to type 2 diabetes.

There are some caveats to this study that must be pointed out. First, it is a cross-sectional study looking at one point in time. Longitudinal studies are certainly preferable. Second, it is an observational study and interventional studies are more valuable, though much more difficult and expensive to carry out. Third, the food frequency questionnaire used in this study was not specifically designed to test for glycemic index and glycemic load. While it has been validated as an overall instrument, it has not been validated for reproducibility and reliability as an appropriate glycemic index instrument, and this needs to be done by the IRAS group. This field has been dogged by the inaccuracy of dietary records and the difficulty in calculating dietary glycemic index and glycemic load levels of individuals from their reported intake of foods. Validation of experimental instruments is crucial. Fourth, the minimal model was instituted using 30 plasma samples to calculate insulin sensitivity (2). This study used 12. While this seems reasonable in a large epidemiological study, the reliability is likely to not be as high as using the originally described method.

Having said the above, the study reports very interesting results. IRAS showed a lack of association of glycemic index, glycemic load, and carbohydrate intake with measures of insulin sensitivity, insulin secretion, and adiposity, adjusting for energy intake. It also showed fiber to have a positive association with insulin sensitivity and an inverse association with adiposity and disposition index.

Insulin sensitivity is a very important component of carbohydrate homeostasis. Individuals with insulin resistance are more likely to eventually develop type 2 diabetes. The potential effect of diet on this physiological state is thus important in gauging risk. In short-term interventional metabolic studies in small numbers of people, the best trial to date, e.g., the longest and most comprehensive, has shown an improvement in insulin sensitivity with a high versus a low glycemic load diet (3). Other less rigorous studies have shown mixed results.

Let me deal with fiber first. The benefit of fiber in the diet on a number of chronic diseases has been documented repeatedly in epidemiological studies. An increased consumption has been associated with lower incidence of diabetes, coronary artery disease, and obesity in observational studies. With regard to diabetes, most studies have singled out cereal fiber as the important component, with other types of fiber giving much lower or no association. However, as stated in a report of the National Academy of Sciences “there is no conclusive evidence that it is dietary fiber rather than the other components of vegetables, fruits, and cereal products that reduces the risk of those diseases” (4). The present study breaks no new ground here, it just bolsters previous data impressively, suggesting that the effect on lowering risk of type 2 diabetes may work through enhancing insulin sensitivity. It supports the recommendation in the 2005 dietary guidelines for Americans (5) to increase their fiber intake. The present U.S. fiber intake is very low and an increase undoubtedly would improve health.

The question of glycemic index and glycemic load is more contentious. As the authors state, high glycemic index diets have been linked to an elevated risk of developing diabetes. There are two reports, one of the Nurses’ Health Study (6) and the other of the Health Professionals’ Study (7). However, reading these reports carefully, neither is significant for a glycemic load effect and only one for glycemic index effect at P < 0.04. Other epidemiological, observational, longitudinal studies have shown no significant effect. These include the Iowa Women’s study (8), the San Luis Valley Study (9), and the Atherosclerosis Risk in Communities study (10). The present IRAS report bolsters the negative data. A final study, the Nurses Study II, showed a significant effect of glycemic index, but both glycemic load and total carbohydrates were inversely associated with diabetes risk (11). These studies tried to relate glycemic index and glycemic load to risk of diabetes, they did not measure insulin sensitivity. Insulin sensitivity generally has been measured in metabolic ward studies with interventional trials of short duration, such as the Kiens and Richter (3) study mentioned earlier.

The Liese et al. study is the first to try to document the impact of a higher versus a lower–glycemic index and/or glycemic load diet on insulin sensitivity in a large epidemiological study. They were unable to document a relationship between either glycemic index or glycemic load and insulin sensitivity. A larger observational study in Denmark (12) also could not document an association of glycemic index with insulin resistance using a homeostasis model assessment of insulin resistance and found an inverse association between glycemic load and insulin resistance.

The IRAS investigators were also unable to find an association of glycemic index and glycemic load with disposition index. The disposition index measures the ability of the pancreas to respond to an increase in insulin resistance with an increased secretion of insulin, thereby maintaining normal blood glucose. An abnormal disposition index suggests β-cell strain and can lead to eventual β-cell failure. Thus, the inability to observe an inappropriately low pancreatic response associated with higher glycemic
index and glycemic load suggests normal pancreatic functioning on such diets.

In the last few years, there has been a very strong push by some investigators to declare a high–glycemic index and a high–glycemic load diet detrimental to health, particularly in relation to the development of obesity and type 2 diabetes. They have pressured public health authorities to recommend that such diets should be restricted for the population at large. But it must be remembered that the concept of the glycemic index was first proposed as a tool to try to improve glucose control in diabetic patients, where it may be of some help to patients with poor postprandial glucose control. But there is a great deal of confusion in interpreting the database available on glycemic index because data from studies in diabetic patients are often quoted to bolster policy suggestions for normal populations. This confusion is not conducive to a clear analysis of the issue. A pro and con discussion of the use of the glycemic index in normal population groups that is relevant in this context was published recently (13,14).

It is important to note that the window of glycemic index variability in a free-living population is quite narrow. This can be seen by the small SD of 4.0 around a mean of 58 in the Liese et al. study. It can also be seen that the glycemic index is actually already quite low. With such a low level of glycemic index and such a small variation around it, what determines the level of the glycemic load is the total amount of carbohydrate that an individual eats. Thus, with a small glycemic index window, the glycemic load primarily reflects the carbohydrate intake. And we know from repeated studies (in fact, all the epidemiological studies mentioned above plus the present IRAS study, plus many others) that no one to date has found that the amount of carbohydrate eaten per day is significantly associated with the development of type 2 diabetes. This then, greatly diminishes the importance of high glycemic load as an important risk.

My suggestion then, looking at the present study and others, is that until further evidence is available, we should concentrate on educating the public to opt for higher-fiber foods (especially cereal fiber) and downplay the glycemic index and glycemic load. There is excellent evidence that the higher-fiber foods, made up of whole grains, fruits, and vegetables, will do people good.

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References


