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A role for the glycemic index in preventing or treating diabetes?[1,2]

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Because of dramatic increases in prevalence that are related to modernization, type 2 diabetes is now considered a high-priority public health problem in nearly every country in the world (1). Views on the role of diet in the management of diabetes have undergone an evolution over the past 30 years. For many years, a high-fat, low-carbohydrate diet was recommended (2); then, in 1979, the American Diabetes Association (ADA) issued a consensus statement suggesting that a high-carbohydrate, low-fat diet was best for most patients (3). This official position was subsequently modified to emphasize glucose and lipid control rather than weight loss; this change allowed the inclusion of more fat (primarily monounsaturated) in the diet (4). However, just as it is a topic for avid discussion in popular culture, diet remains a subject of lively debate with respect to diabetes treatment.

An aspect of the debate about diet in diabetes treatment that has received much attention since its introduction nearly 30 y ago (5) is the role of the glycemic index (GI). The GI is a system for ranking carbohydrates according to their effects on postprandial glucose concentrations. Although low-GI foods are known to produce less postprandial hyperglycemia and hyperinsulinemia than are high-GI foods (6), the role of low-GI foods in the prevention and treatment of diabetes remains unclear. Elsewhere in this issue of the Journal, Wolever et al (7) report the results of the Canadian Trial of Carbohydrates in Diabetes (CCD). Patients with well-controlled type 2 diabetes who were treated with diet alone were randomly assigned to receive either a high-GI diet, a low-GI diet, or a low-carbohydrate, high-monounsaturated fat diet for 1 y.

The study was carefully conducted and of longer duration than many earlier trials. The investigators found no weight loss and a small increase in glycated hemoglobin (HbA_{1c}) in all 3 groups. This increase in HbA_{1c} is what one would expect with no intervention (8). The fact that glucose concentrations 2 h after an oral glucose challenge were significantly lower in persons who had followed the low-GI diet for 1 y than in those who followed the other 2 diets for 1 y suggests improvement in either insulin sensitivity or insulin secretion (or improvements in both). A small study in which euglycemic clamps were conducted in healthy subjects after 30 d of a low-GI or a high-GI diet actually showed greater insulin sensitivity in the subjects following the high-GI diet; this improved insulin sensitivity was associated with lower fasting and postprandial free fatty acid concentrations (9). It is possible that these differences in insulin sensitivity and free fatty acid metabolism are transient and that they disappear when the diet is followed for a longer time.

In the report of Wolever et al, there were several intriguing findings that warrant attention and, perhaps, further investigation. High-sensitivity C-reactive protein (hs-CRP) was 30% lower in the low-GI group than in the high-GI group, despite no significant weight difference between the groups. Decreases in hs-CRP are known to accompany weight loss (10). The mechanism by which hs-CRP is decreased by a low-GI diet is not understood, but it may involve less postprandial hyperglycemia or hyperinsulinemia (or both).

The CCD investigators also reported an interesting observation regarding blood pressure. Diastolic blood pressure was highest in the high-GI diet group, and the difference was greatest between the high-GI diet group and the low-carbohydrate diet group. The clinical implications of this observation are uncertain but of potential importance. Diastolic blood pressure is generally thought to be less important than systolic blood pressure with respect to cardiovascular disease risk (11), but increases in diastolic blood pressure may reflect activation of the sympathetic nervous system (12). Although no significant differences in fasting insulin concentrations were observed in the CCD study, it is likely that the subjects following the high-GI diet had greater postprandial hyperinsulinemia than did those following the other 2 diets (6). Insulin is known to activate the sympathetic nervous system (13), and it is an underappreciated mediator of sodium retention and volume expansion (14, 15). Additional studies of blood pressure control, sympathetic activity, and sodium balance in relation to the GI are needed to determine the importance of this observation.

If there is ambivalence in the literature concerning the role of GI in diabetes management, there is even less agreement as to whether GI could influence the risk of diabetes. In a separate study in this issue, Sahyoun et al (16) found no association in older adults between GI or glycemic load (GL) and the risk of developing type 2 diabetes. Although this study had a somewhat shorter duration and smaller sample size than several earlier studies that found an association between GI and diabetes risk, it strengthens the argument that high-GI diets per se do not increase the risk of diabetes.

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The results of these studies will be disappointing to some advocates of low-GI diets. The ADA's position statement on nutrition concluded that the use of GI and GL may have a modestly greater benefit in controlling diabetes than is observed when total carbohydrate is considered alone (17). The ADA further stated that information is not sufficient to allow a conclusion that low-GL diets reduce diabetes risk; nevertheless, the consumption of low-GI foods that are rich in fiber is to be encouraged (17). The 2 studies reported in this issue of the Journal provide no compelling reason to modify those recommendations.

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