Glycemic index in early type 2 diabetes

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INTRODUCTION

In this issue of the Journal, Wolever et al (1) report the results of the Canadian Trial of Carbohydrates in Diabetics. This is an interesting report comparing the effects of high- and low-glycemic-index (GI) diets with those of a low-carbohydrate diet on various metabolic markers of disease. The bottom line is that following these 3 diets for 1 y produced essentially no difference in the subject groups with respect to glycated hemoglobin (HbA1c), lipids, or insulin. As reported in a recent Cochrane review (2), few to no high-quality long-term data are available on the dietary treatment of type 2 diabetes mellitus. Therefore, a well-carried-out, extended study such as that of Wolever et al is very welcome, and, from it, several interesting observations emerge.

First, the report by Wolever et al shows the difficulty of accurately measuring food intake in overweight persons. The average body mass index (BMI; in kg/m²) of the group was 31, and the range was 24–40. The average caloric intake reported with the 3 diets at baseline ranged from 1810 to 1930 with an average weight of 84 kg and at the end of the study ranged from 1800 to 2020 with a slightly higher average weight. Patients did not lose weight; they actually gained. But, even if he or she is very sedentary, a person weighing 84 kg requires more calories than are reported in the study to maintain weight. Thus, the reporting of caloric intake by diaries is shown clearly to be inaccurate, and there is significant underestimation of energy intake. Such underestimation has been reported previously from this laboratory (3) and by many others. The tools for measuring food intake in humans are very imprecise, as documented here. Yet investigators (and journals) persist in publishing such data as if they were accurate and persist in presenting percentages of macronutrients to one decimal place (see Table 3 in reference 1) as if there were any confidence in such decimals.

Second, compared with baseline data, these mildly diabetic type 2 patients actually did worse with regard to HbA1c and weight while following each of the 3 experimental diets (1). This finding suggests that we must be careful about disrupting subjects’ or patients’ diets with radical, doctrinaire changes that may actually be counterproductive. Furthermore, the diets had carbohydrate contents that varied from 39% to 52% of energy intake, and yet this variability had no effect on the subjects’ HbA1c. This finding confirms previous reports that the proportion of carbohydrate in the diet is not very important in determining the concentration of fasting blood glucose and that variations of 10% to 15% of total calories make little difference to overall control in patients with early type 2 diabetes.

This report is unique in having followed subjects for 1 y and in using careful monitoring of the subjects’ diet and providing continued professional nutritional advice. It is interesting that the long-term results show that the 3 diets had little ultimate effect on either triacylglycerol or HDL-cholesterol concentrations. Thus, the arguments of the champions of a low-GI or a low-carbohydrate diet—that these 2 types of diets will result in lower triacylglycerol and higher HDL concentrations—have not been upheld in this careful, year-long dietary study.

One of the few statistically significant results of the study by Wolever et al was that the cohort following the low-GI diet had a higher fasting glucose and a lower glucose concentration 2 h after an oral glucose challenge than did the subjects following the other 2 diets (1). The difference, however, is small, and its effect on HbA1c is clearly nil. A lower 2-h post-glucose challenge glucose concentration would be expected, but a higher fasting glucose is not. The cause of a higher fasting glucose is not immediately evident, because insulin did not change in these patients. The authors state that the concentrations of free fatty acids (FFAs) were 8% higher than at baseline but not significantly different (data not given), and they suggest that this may be the cause. If the FFA concentrations did not differ significantly between baseline and year 1, FFAs cannot be considered a cause of the higher fasting glucose concentration. In addition, when the results of the breakfast meal from the patients’ initial “regular diet” and those of the breakfast meal from their subsequent “test diet” (see Figure 6 in reference 1) were compared, no significant difference in postprandial glucose excursions was found between the high-GI and low-carbohydrate diets. A slight decrease in postprandial glucose was found in subjects in the low-GI diet group, but, to this observer, it seems biologically insignificant.

Wolever et al (1) also observed a drop of 20% from baseline in C-reactive protein (CRP) concentrations with the low-GI diet, but the diet × time interaction was not significant. Nevertheless, Wolever et al make much of this result. A few comments seem indicated with regard to this outcome. First, the CRP was significantly different at baseline in the 3 treatment groups (3.4, 2.6, 2.4)

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Received October 3, 2007.
Accepted for publication October 8, 2007.

and 1.94 mg/L). Second, the baseline CRP value—which dropped in the low-GI diet group by 20% at 1 y—was in the normal range in all subjects (95% confidence limits: 1.48, 2.55 mg/L). It is not known what effect a small change within the normal range, from an average of 1.92 to one of 1.55 mg/L, will have on subsequent risk of cardiovascular disease. Third, CRP was measured in this study by nephelometry with the use of a high-sensitivity commercial kit. The fluctuation of 20% within the normal range in the low-GI diet cohort could be a true effect or merely an artifact of measurement of low concentrations. The discussion regarding this finding—which suggests that the lowering of CRP is due to a change in postprandial glucose in the low-GI diet group—is misleading. It is difficult to square that suggestion with the actual increase in HbA1c in those following the low-GI diet, which indicates an overall increase in the blood glucose concentration. Wolever et al suggested that the effect may be due to fewer high peaks of glucose, but this interpretation is purely speculative. More studies with additional inflammatory markers (eg, white blood cells, interleukin-6 or -10, or tumor necrosis factor-α) will be required to confirm the observed modest decline in a single inflammatory marker before the findings of this study can be considered to represent a true lowering of the risk of cardiovascular disease.

Finally, for the proponents of a low-GI diet, the fact that these investigators, who are well known for their nutritional expertise, were able to provide a sustained difference in GI of only 8 units over 1 y attests to the difficulty of maintaining a low-GI diet over the long term. A realistic lower-GI diet that could be sustained in these patients with mild type 2 diabetes had no significant effect. Given the data from Wolever et al and the previous equivocal data with respect to this issue (4, 5), it seems unwise at this point to burden type 2 diabetes patients with trying to pick and choose among different high- and low-GI foods.

The author had no personal or financial conflict of interest.

REFERENCES