Should the Bee Suck Honey or Lard?

That is the question

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Of all the nonpharmacologic endeavors available to diabetic patients to mitigate the complications of diabetes, none has as profound an effect as nutritional manipulation. There has, however, continued to be a great CHO versus fat controversy. The *Papyrus Ebers*, written around 1500 BC, advocated a high-CHO diet consisting of wheat grains, fruit, and sweet beer "to drive away the passing of too much urine." Araetus the Cappadocian coined the term diabetes (to flow through), and prescribed starches, fruit, and sweet wines (1). These sentiments were echoed by the ADA in their consensus statement *Nutritional Recommendations and Principles for Individuals with Diabetes Mellitus* (2), first published in 1986. This statement recommended liberalizing CHO intake, not so much to replace the lost CHO, but to fill the void derived from a restriction in protein intake, to protect the kidney, and to reduce total fat intake to reduce morbidity and mortality from macrovascular disease. The recommendations of ADA were extremely similar to those being promoted by the AHA and the American Cancer Association, which are concerned with the possible relationship between fat intake, heart disease, and bowel and breast cancer. Other researchers, however, disagree with the liberalization of CHO intake. They argue that a high-CHO intake might worsen glycemic control, and suggest that a high-fat diet is much more palatable to the American public than a high-CHO diet. There is no doubt that meals for "fat teeth" will always suit the hedonistic tendencies of our population. Nonetheless, considerable recent evidence strongly supports the 30% dietary-fat guideline recommended by ADA.

Evidence for the role of dietary fat in diabetic complications derives from numerous studies, both clinical and basic (3–5). These data clearly point to increased disease risk with increased dietary fat. Well-controlled human and animal studies have provided some insight into how dietary fat may alter the physiological response to insulin. Humans with normal body weight fed a diet consisting of 65% fat had measurably decreased insulin sensitivity within 3 days of treatment (6). This study strongly indicts dietary fat, rather than obesity per se, in the resistance to insulin that may herald the onset of diabetic complications.

Further evidence for this relationship has been provided in primate studies (7). Harris and Kor (8) recently provided even more substantial and relevant data as to the importance of dietary fat in insulin sensitivity. In this study, rats fed a 40% fat diet for 10 wk showed significant changes in insulin sensitivity within 3 days of being switched to a 30% fat diet. Total lipid synthesis was also greater in adipocytes from rats fed 40% fat compared with those fed the lower-fat diet. The improvement in insulin sensitivity of animals with the lower fat intake is remarkable because of the short duration of exposure to the 30% fat diet, as well as the fact that dietary fat was only reduced from 40 to 30%. Previous animal experiments in which fat was reduced by 50% (from 30 to 15% of energy) also demonstrated enhancement of the adipocyte response to insulin (9), but the Harris and Kor experiment is more representative of the situation of the typical American, whose diet consists of ≥38% fat. Our animal model of diet-induced obesity in Sprague-Dawley rats (10) provides further evidence for the role of dietary fat in diabetes. In this model, ~50% of the rats fed a 32.5% fat diet became obese and insulin resistant, while the others remained lean. The implication here is that even low levels of dietary fat by human standards can elicit obesity and insulin resistance in animals so genetically predisposed.

Although the mechanism of insulin resistance remains controversial, the association of insulin resistance with an increased predisposition to macrovascular events cannot be denied (11). Several studies have shown improvements in lipids and glycemic control when subjects have switched from an average American

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CHO, CARBOHYDRATE; ADA, AMERICAN DIABETES ASSOCIATION; AHA, AMERICAN HEART ASSOCIATION; LDL, LOW-DENSITY LIPOPROTEIN; VLDL, VERY LOW-DENSITY LIPOPROTEIN; IDDM, INSULIN-DEPENDENT DIABETES MELLITUS; NIDDM, NON-INSULIN-DEPENDENT DIABETES MELLITUS.

diet to a low-fat, high-fiber diet, or to a high–monounsaturated fat diet. For example, Ginsberg et al. (12) completed studies in 36 healthy young men being fed an average American diet comprising 38% fat. These young men were then placed on an AHA step-one diet, with 30% of calories from fat, (10% saturated, 10% monounsaturated, and 10% polyunsaturated), or a monounsaturated-supplemented AHA diet with 38% total fat calories (10% saturated, 10% polyunsaturated, and 18% monounsaturated). The results of these studies showed a significant reduction in total cholesterol on both diets, as well as a slight reduction in the total triglycerides and LDL cholesterol. An improvement in the lipid profile was associated with either a reduction in total fat or an 8% increase in monounsaturated fat intake. Garg et al. (13) compared the average American diet with a high-CHO diet (60% CHO, 15% protein, and 25% fat) and a high–monounsaturated fat diet (50% total fat, 35% CHO, and 15% protein). In the monounsaturated fat diet, 33% of fat calories were derived from olive oil, whereas, in the high-CHO diet, the fat was derived from 30% corn oil and 70% palm oil. When subjects were switched from their baseline average American diet to the high-CHO diet, fasting glucose levels fell from 129 ± 4 to 117 ± 5 mg/dl, HbA_1 from 11.3 ± 0.6 to 7.8 ± 0.7%, and triglycerides from 285 ± 62 to 218 ± 32 mg/dl with a fall in VLDL cholesterol from 58 ± 12 to 43 ± 7 mg/dl. These changes from the low-fat, high-CHO diet were similar to those found on the high–monounsaturated fat diet, and were achieved without providing foods of high caloric density for protracted periods, which may have other untoward consequences such as obesity, insulin resistance, dyslipidemia, and predisposition to macrovascular disease. Ireland et al. (14) fed 16 versus 53% fat to 10 IDDM patients, and showed deterioration of CHO tolerance with the high-fat diet. Fat restriction increased sensitivity to insulin, decreased insulin requirements, and improved glycemic control (14).

Diets rich in fat calories may even promote adiposity, irrespective of energy intake (15), and the benefit of calorie restriction is mitigated if a low-calorie diet is fat-rich (16). In both men (17) and women (18), obesity correlates directly with the fat content of the diet. If the primary objective is to reduce weight in NIDDM patients, the majority of whom are obese, liberalization of fat calories, irrespective of the total number of calories, seems to fly in the face of available information.

The current recommendation for diets consisting of no more than 30% fat seems quite conservative and is supported by the above data. Moreover, the type of dietary fat used in the diet may be less important than the level of fat in the diet (9). Obese NIDDM patients may benefit from even lower levels of dietary fat regardless of the composition of the diet. Diabetic individuals may find it easier and more straightforward to deal with the calculation of an amount of fat rather than trying to analyze the composition of the fat they consume. Overall, there appears to be no doubt that the health risk to these patients may be reduced by observing the 30% dietary fat goal. In this respect, we think that the general recommendations of the National Research Council (4) and the Surgeon General (5) (which were not made arbitrarily nor without considering all the evidence) that "the American public reduce their fat intake" should apply equally well to the subset of people with diabetes until incontrovertible evidence proves otherwise.

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