Diet and Health: The Nutritional Treatment of Diabetes

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Diabetes is a major public health problem. In the United States it has been estimated that the prevalence of diagnosed and undiagnosed diabetes in people aged 20 to 74 years is 6.6% (1). Diabetes prevalence increases with age, rising from 2% in the 20 to 44 year age-range to 18% in the 65 to 74 year age-range. There are two main types of diabetes (2). Non-insulin-dependent diabetes mellitus (NIDDM) accounts for 90% of diagnosed diabetes in the United States, while insulin-dependent diabetes mellitus (IDDM) accounts for the remainder. Diabetes is the underlying cause of 36,000 deaths every year, making it the seventh leading cause of death. It also contributes to nearly 95,000 additional deaths per year. The economic cost of diabetes is rising. In 1969 it stood at $2.6 billion, increasing to $9.7 billion in 1980 and $19.8 billion in 1986 (3).

Diabetes is a major risk factor for morbidity and mortality due to coronary heart disease, cerebrovascular disease, and peripheral vascular disease (4). Diabetics are two to four times more likely to have heart disease than nondiabetics and two to six times more likely to have a stroke. About 45% of all nontraumatic leg and foot amputations are carried out on diabetics. Hypertension is present in about 50% of diabetics. Diabetes is also strongly associated with microvascular disease, which causes progressive damage to the eyes and kidneys (4). Diabetes is the major cause of new cases of blindness in people aged 20 to 74 years. Nearly one quarter of all new patients with end-stage renal disease have diabetes.

The strong association of diabetes with macrovascular and microvascular disease has led many people to suppose that improved glycemic control will slow down, stop, or even reverse these long-term complications. It should be emphasized that while the duration and degree of hyperglycemia are associated with microvascular disease risk, no such relationship has been established for macrovascular disease risk (5). Indeed Jarrett has suggested that coronary heart disease, far from being caused by NIDDM, develops in parallel with it and that the two conditions may have a common cause (6). Reaven has suggested that insulin resistance is at the root
of glucose intolerance, hyperinsulinemia, increased VLDL-triglyceride, decreased HDL-cholesterol, and hypertension—all of which have been shown to increase cardiovascular risk—and has coined the term Syndrome X to describe their association (7). If this idea is correct, any factor that improves insulin sensitivity would play a major role in the management of NIDDM and its associated macrovascular risk.

There is evidence that nutrition plays a major role in the prevention and management of diabetes. This article will concentrate on the dietary management of NIDDM. There is a growing consensus that this approach has enormous potential. In 1983 Nuttall began an article on diet and the diabetic patient by saying, “In nearly every textbook or treatise on diabetes mellitus, diet is stated to be the cornerstone of treatment” (8). In 1986 Cahill, the chairman of a consensus development panel convened by the National Institutes of Health to examine diet and exercise in NIDDM, was quoted as saying that “diet is the hallmark” of diabetes therapy (9). More recently Alberti stated that “my package for NIDDM is diet, diet, diet and whatever exercise is appropriate for a particular person” (10). However, enthusiasm for the dietary approach toward management of NIDDM should be tempered by the realization that dietary change is very far from being acceptable to all non-insulin-dependent diabetics. In 1973 West wrote a review entitled “Diet Therapy of Diabetes: An Analysis of Failure” (11). This should serve as a reminder of the difficulties that need to be overcome to realize the potential of diet in the management of NIDDM.

ENERGY RESTRICTION

There is a strong association between NIDDM and obesity. Joslin studied a group of 4,596 diabetics who were at least 20 years old at the onset of the disease (12). He found that 78.5% of the men and 83.3% of the women were at least 5% overweight and 51.0% of the men and 59.3% of the women were at least 20% overweight at onset. It is clear, therefore, that a sizable proportion of diabetics are overweight.

Energy restriction has a long history in the management of NIDDM. Thomas Willis (1621–75), who was responsible for the rediscovery of the sweet taste of diabetic urine, was the first to write a case report on the dietary management of diabetes (13). This concerned “a certain noble Earl” whose diabetes Willis managed partly with herbal remedies and partly by diet:

His diet was only of milk, which he took sometimes raw and diluted with either distilled or Barley water and sometimes boiled with white bread, or Barley, severa. times in a day. By the use of these things growing daily better and better he seemed within a Moneth to be almost quite well: when he began to be very hearty, his Urin which was insipid, did not much exceed the quantity of that liquid matter which he took in: then afterward growing somewhat saltish it was less than what he drank till at last by degrees recovering his usual tenure of spirits and strength he returned to his former diet. But yet the disposition of this Distemper did not so totally leave him but that afterward, oftentimes, through disorders in his Diet and perhaps irregularities in the seasons of the year, being inclined to a relapse he made water at first in great quantities.
and then clear and sweet with the thirstiness feverishness and languishment of his spirits... 

This is the first recorded case of effective management of diabetes by a low energy, high carbohydrate diet. But the description also underlines the consequences of a failure to keep to the dietary prescription. The acceptability of low energy diets was already a problem.

John Rollo (d.1809) is usually credited with being the first physician to prescribe a systematic diet for the management of diabetes. In 1797 he published An Account of Two Cases of the Diabetes Mellitus (14). Much of the book is devoted to a case report on a Captain David Meredith who, when he was age 34, developed the classic symptoms of diabetes mellitus. From Rollo’s notes it can be calculated that the captain had a body mass index of 31.7 and he would, therefore, be described today as an obese non-insulin-dependent diabetic. His diet was prescribed as follows:

The diet to consist of animal food principally, and to be thus regulated:
- Breakfast. One and a half pint of milk and half a pint of lime-water, mixed together, and bread and butter.
- Noon. Plain blood-puddings, made of blood and suet only.
- Dinner. Game, or old meats, which have been long kept, and as far as the stomach may bear, fat and rancid old meats, as pork. To eat in moderation.
- Supper. The same as breakfast.

The dietary treatment began on October 19, 1796. Two days later the volume of urine passed in 24 hours had fallen from seven or eight quarts to six quarts. By November 1 the urinary volume was no more than four quarts, and on November 4 the volume had dropped to two quarts and no longer tasted sweet. By December 30 polyuria and polydipsia had disappeared. His diet was subsequently liberalized by the “use of cabbage, or greens of a similar nature, boiled onions, or salad without acid sauce, also mustard, horseradish, and common radish when in season.”

Rollo prescribed a low energy, high fat diet. The key words in his prescription are “to eat in moderation” and no doubt compliance with this instruction was helped by the disgusting nature of some of the foods recommended. Rollo summarized his dietary approach as follows: “it becomes, therefore, a principal object in the treatment to vary the articles of diet so as to gratify the earnest desire of the patient without bringing on a return of the complaint.” Striking a balance between these two requirements is still a problem today.

Apollinaire Bouchardat (1806–86) introduced the idea of the personal responsibility of the diabetic for his own treatment (15). He encouraged his diabetic patients to find out for themselves about the sugar-producing capacity of a food by eating it and testing their own urine. He recognized the sugar-producing capacity of foods containing carbohydrate such as bread and milk, which he advised against. He realized that dietary protein also gives rise to sugar, but in much lower quantities, so meats, for example, were allowed. He recommended green vegetables provided they had been boiled first to remove the starch. During the siege of Paris in the Franco-Prussian war of 1870–71, when the supply of food to the city was limited, Bouchardat observed that the urinary sugar levels of his diabetic patients fell. This experience
led him to propose that his diabetics eat as little as possible. He occasionally fasted his patients. Once again energy restriction emerged as an effective way of managing diabetes and keeping the urine free of glucose.

In December 1986 a Consensus Development Conference on Diet and Exercise in NIDDM was convened by the National Institutes of Health (16). In response to the question, "What are the appropriate components of the dietary prescription for patients with NIDDM?" the consensus development panel wrote, "Because most people with NIDDM have excess body fat, the primary dietary treatment is reduction of weight through caloric restriction." Energy restriction leading to loss of body weight is very effective in improving glycemic control and the mechanism of this effect has recently been investigated (17). Fasting plasma glucose levels fell in response to energy restriction due to a fall in hepatic glucose output. Improved insulin secretion and enhanced peripheral insulin sensitivity also contributed. Hence it is likely that energy restriction is effective in lowering the blood glucose concentration because it improves insulin sensitivity.

It is one thing to advise non-insulin-dependent diabetics to restrict their energy intake in order to lose body weight but quite another to motivate them to do so. Several programs have been developed to accomplish this. One of the most successful is the Düsseldorf treatment and teaching program (DTTP) for NIDDM (18). The program consists of teaching sessions of 90–120 minutes every week for four weeks for groups of 4-6 patients. The non-insulin-dependent diabetics are taught to distinguish between nutrients on the basis of their energy content. They are encouraged to find out for themselves what happens to their glycosuria when they lose a modest amount of body weight and what happens when they eat a meal rich in simple carbohydrates. The success of this approach has been evaluated over 12 months in a general practice setting. Diabetics participating in the program lost 2.7 kg body weight and their triglycerides fell by 20%. Although glycemic control was unchanged no diabetic began insulin treatment, whereas 10% of the control group did. The proportion taking oral hypoglycemic drugs fell from 68% to 38%. This program is currently being used by over 2,000 general practitioners in Germany and has been introduced into other countries such as Argentina. Its success is based on involving non-insulin-dependent diabetics in the management of their own disease and in this respect continues the tradition begun by Bouchardat.

THE PROPORTION OF CARBOHYDRATE AND FAT

The value of energy restriction in the management of NIDDM is a lesson from the past that has been learned. By contrast, the correct proportion of carbohydrate and fat in the diet of a non-insulin-dependent diabetic is still being debated despite the fact that this information has been available since the 1930s.

A series of dietary recommendations from the American Diabetes Association (ADA) has provoked this debate. In 1971 it was stated that "There no longer appears to be any need to restrict disproportionately the intake of carbohydrates in the diet
of most diabetic patients" (19). In 1979 it was stated that for insulin-dependent diabetics, "Carbohydrate should usually account for 50–60 percent of total energy intake." No recommendations were made for non-insulin-dependent diabetics (20). In 1986 it was recommended that "liberalization of the carbohydrate intake, of the unrefined variety, may also be beneficial in the management of hyperlipidemia" (21). In 1988 the European Association for the Study of Diabetes (EASD) stated that, "The most important general recommendation is a decrease in intake of saturated fatty acids. This may be compensated for by an increased intake of complex carbohydrate..." (22). The idea behind these recommendations is that a reduction in saturated fat intake should have favorable effects on blood lipid levels and hence on cardiovascular risk. Hence it is proposed to reduce saturated fat intake and, to compensate, increase complex carbohydrate intake. However, as Reaven has repeatedly pointed out (23,24) in non-insulin-dependent diabetics, who in the United States make up 90% of the diabetic population, high carbohydrate diets increase triglyceride levels, which is a risk factor for cardiovascular disease in this population (25). Reaven has also reminded us that this question was already addressed in the 1930s by Himsworth.

Himsworth was the first to propose a classification of diabetics based on their response to insulin (26). He distinguished between insulin-sensitive and insulin-insensitive types of diabetes mellitus corresponding to our modern classification of insulin-dependent and non-insulin-dependent diabetes. He then went on to show that the response of the two types to dietary carbohydrate was quite different:

Insulin sensitive diabetics react favourably to increase of dietary carbohydrate. Glycosuria does not increase, the fasting blood sugar level does not rise and the sugar tolerance and the sensitivity to insulin improve. In the case of the insulin-insensitive diabetic, on the other hand, increase of dietary carbohydrate causes increase in glycosuria, a tendency to higher fasting blood sugar levels, impairment of sugar tolerance and little or possibly no increase in sensitivity to insulin.

Thus in the case of high carbohydrate diets, as in all else, one cannot extrapolate from insulin-dependent to non-insulin-dependent diabetes.

More recently, Reaven's group has provided confirmation of Himsworth's findings. Non-insulin-dependent diabetics were given 40% carbohydrate/40% fat and 60% carbohydrate/20% fat isocaloric diets in random order for six weeks (27). On the high carbohydrate diet, fasting and postprandial glucose and insulin were higher, 24-hour urinary glucose doubled, total and VLDL-triglycerides increased by 30%, and HDL-cholesterol decreased. The high carbohydrate diet was quite clearly deleterious for metabolic control.

Despite the above results, increasing the carbohydrate content of a diet for non-insulin-dependent diabetics is not necessarily deleterious for metabolic control. It all depends on what is replaced with what. A high carbohydrate diet was recently compared with a high monounsaturated fat diet in non-insulin-dependent diabetics (28). A 35% carbohydrate/50% fat diet was compared with a 60% carbohydrate/25% fat diet. Complex carbohydrates in the latter diet were replaced with monounsaturated fat in the former. The diets were isocaloric and were given to the patients for
four weeks. Plasma glucose, triglycerides, and VLDL-cholesterol were lower and HDL-cholesterol was higher on the high monounsaturated fat diet. Thus monounsaturated fat is better than complex carbohydrate for metabolic control.

By contrast, when complex carbohydrates are replaced with saturated fat different metabolic effects are seen (29). A 43% carbohydrate/42% fat diet was compared with a 65% carbohydrate/21% fat diet. The diets were isocaloric and were given to non-insulin-dependent diabetics for five weeks. Total and LDL-cholesterol were lower on the high carbohydrate diet. There were no deleterious effects of this diet on HDL-cholesterol, triglycerides, or glucose.

These two reports serve to direct attention to two nutritional issues. First, what is the best type of dietary carbohydrate for non-insulin-dependent diabetics? And second, what is the best type of dietary fat?

THE TYPE OF CARBOHYDRATE

Dietary sucrose is generally believed to be deleterious for metabolic control in NIDDM but what evidence is there that it is any worse than any other carbohydrate? In order to demonstrate that sucrose itself, rather than the energy or carbohydrate it brings, is deleterious for metabolic control in NIDDM, it is necessary to study it in the context of isocaloric diets of constant carbohydrate composition. It is also preferable to study its metabolic effects over several weeks or months. One of the few papers that complies with these requirements was published in 1988 (30). Non-insulin-dependent diabetics were given two isocaloric diets, both of which contained 50% carbohydrate, for four weeks. One diet provided 2 g and the other 220 g sucrose per day. There were no differences in the fasting glucose concentration, 24-hour glycosuria, glycosylated hemoglobin concentration, triglyceride levels, or HDL-cholesterol. The case against sucrose in diets for non-insulin-dependent diabetics remains to be proven.

This result suggests that sucrose and carbohydrates such as starch are metabolically equivalent. However, not all starches are digested at the same rate and as a result some give lower peak values of postprandial glycemia than others. White beans can be processed in two ways, one of which ruptures the cells liberating the starch granules, while the other keeps the cells intact. Non-insulin-dependent diabetics were given a single meal containing beans with either intact cells or ruptured cells. Postprandial glycemia and insulinenia were significantly lower after eating beans containing intact cells (31). Such slowly digested starches, which lower postprandial glycemia, have obvious applications in the management of NIDDM. What is needed now, however, is studies of their metabolic effects over weeks or months. From the point of view of diets for non-insulin-dependent diabetics, it would be more useful to draw a distinction between fast and slowly digested carbohydrates, rather than between sucrose and complex carbohydrates.
THE TYPE OF FAT

Replacing complex carbohydrates with monounsaturated fat is beneficial for metabolic control in NIDDM, whereas replacing complex carbohydrates with saturated fat is detrimental. The nature of dietary fat can, therefore, play a key role in diets for non-insulin-dependent diabetics. Generally speaking unsaturated fat is recommended in preference to saturated fat but there are different types of unsaturated fat. Some information on this in relation to NIDDM is beginning to appear in published reports.

The most characteristic lipid abnormalities of NIDDM are a raised triglyceride level and a depressed HDL-cholesterol level. One way of lowering triglyceride levels which has proved effective in non-diabetic populations in the use of fish oils that contain ω3 fatty acids. Eight full papers have appeared on the metabolic effects of fish oil in NIDDM (see Table 6 in reference 32 for a summary). Doses of ω3 fatty acids varying between 2.8 and 7.5 g per day have been tested. While six of these studies have demonstrated a decrease in triglyceride levels of the order of 40%, four of them also found an increase in glycemia. For this reason encapsulated fish oils are not recommended in NIDDM.

A recent long-term study tested the metabolic effects of a linoleic-acid-enriched diet in NIDDM (33). Two isocaloric diets, both containing 38% fat, were given for 30 weeks in a crossover design. One diet had a polyunsaturated to saturated fat ratio of 0.3 and the other of 1.0, formulated by replacing products rich in saturated fatty acids with linoleic-acid-rich oils. Both total and LDL-cholesterol levels were lower on the linoleic acid diet, but glycemic control was unaffected. Unfortunately there were no effects on triglyceride and HDL-cholesterol levels, which are the characteristic lipid abnormalities of NIDDM.

CONCLUSION

For the overweight non-insulin-dependent diabetic, the primary dietary treatment is energy restriction leading to loss of body weight. However, because this is difficult to achieve and because not all such diabetics are overweight, other dietary approaches are needed. High carbohydrate diets are not appropriate in NIDDM, particularly in patients managed by diet alone or by diet plus hypoglycemic drugs. Slowly digested starches look promising in the short-term but more long-term information is needed. A major effort is needed to define which classes of unsaturated fatty acids are most beneficial in NIDDM.

REFERENCES

14. Rollo J. An account of two cases of the diabetes mellitus, with remarks as they arose during the progress of the cure. London: Dilly; 1797.
DISCUSSION

*Dr. James:* In manipulating the diet of diabetics you are presumably trying to maintain them in a state of optimum health for as long as possible. Have we the right criteria for deciding which diet might be best?

*Dr. Stanley:* Diet contributes to the quality of life. Obviously if people don’t like eating their diet, this is detracting from the quality of their lives. Metabolic control is aimed at preventing both acute and long-term complications. It is therefore very important to determine if there is a relationship between control of glycemia and the long-term micro- or macro-vascular complications. In non-insulin-dependent diabetics the main problem is macrovascular disease, with a doubling of the risk for coronary heart disease, a 4-fold increase in risk for cerebrovascular disease, and a 15-fold increase in risk for peripheral vascular disease.

*Dr. James:* What about microvascular disease, particularly renal disease?

*Dr. Stanley:* The best evidence we have at present is that control of glycemia contributes to delaying the onset of microvascular but not macrovascular complications.

*Dr. Ashwell:* In relation to this, and considering what we know about the genetics of diabetes, do you think that one of the dietary approaches might be to look at the progenitors of diabetics to find out which dietary treatments seem to have the greatest effects in the family and possibly to plan dietary management accordingly?

*Dr. Stanley:* The only thing we have to go on currently is the family history but at present this is simply a motivating factor, not a discriminant for treatment.

*Dr. El-Mahroub:* Professor Berger from Düsseldorf University considers that exercise is very important.

*Dr. Stanley:* Exercise increases insulin sensitivity and this is a major argument in its favor. However, some of the complications, such as retinopathy, do not respond well to excessive exercise.

*Dr. James:* Do diabetics have a particular problem in the metabolism of their essential fatty acids?

*Dr. Stanley:* It is certainly true that insulin-dependent diabetics have problems with essential fatty acid desaturases. Whether this is also true of non-insulin-dependent diabetics is an open question. The desaturases are insulin-dependent enzymes, insulin being required for the synthesis of the enzyme protein. Thus in its absence synthesis fails unless you treat with insulin. It is possible that with insulin resistance there may also be insufficient desaturase activity.

*Dr. James:* Could you comment on the theory that in insulin-resistant diabetics there is a fundamental abnormality of the second messenger in muscle involving a defect in the ability to generate the appropriate signaling system, thus explaining the well recognized postreceptor insulin resistance defect.

*Dr. Stanley:* I think the argument has moved away from receptor effects to postreceptor effects. I believe this to be correct because while there is only one receptor event there must be at least a dozen postreceptor events. There may be a constellation of explanations for insulin resistance and I feel that looking at these postreceptor effects is going to be very profitable.