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The Role of Carbohydrate in the Management of Diabetes

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INTRODUCTION

Diet is said to be the 'cornerstone' of management of diabetes, yet the recommended dietary guidelines remain controversial and relatively few patients succeed in being well controlled on diet alone (1). This may imply that dietary treatment is not sufficient in itself or the dietary changes are too difficult to comply with or even that the wrong type of diet is being recommended. Many experts argue against the current dietary recommendations for diabetes, with both the quantity and quality of carbohydrate being at the centre of the controversy. This chapter is designed to critically address the issues of how much and what type of carbohydrate should be recommended for people with diabetes. It takes an evidence-based approach, giving greater weight to the results obtained from randomised controlled intervention studies.

Important questions addressed in this chapter include:

- What is the scientific basis for recommending high-carbohydrate diets?
- What are the potential adverse effects of high-carbohydrate diets?
- What is the scientific basis for recommending diets high in monounsaturated fat (MUFA)?
- What is the scientific basis for recommending low glycaemic index diets?
- What is the optimal diet for improving insulin sensitivity?
- What is the optimal diet for weight loss?
- What is the evidence for a restricted versus liberal intake of sucrose?

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THE OBJECTIVES OF THE DIETARY MANAGEMENT OF DIABETES

The goals of dietary management are clear (beyond dispute) and they apply equally to both Type 1 and Type 2 diabetes. They should achieve:

- Near normal blood glucose with minimal risk of hypoglycaemia;
- Reduced risk of microvascular and macrovascular complications (as assessed by a variety of direct and surrogate measures, including blood lipids, clotting factors, blood pressure);
- Weight loss in overweight patients;
- Normal growth and development in children;
- Healthy outcomes for mother and child in diabetic pregnancy.

Dietitians have the enormous challenge of not only achieving all of the above, but tailoring each person's diet to suit their individual taste preferences and lifestyle.

GOOD GLYCAEMIC CONTROL IMPROVES PROGNOSIS

Good glycaemic control as indicated by near-normal HbA_{1c} levels has been shown to reduce the risk of developing microvascular complications in both Type 1 and Type 2 diabetes (2,3). In addition, there is increasing evidence that it also reduces the development and progression of macrovascular disease (1,4,5). People with diabetes are two to four times more likely to die of coronary heart disease than people without diabetes, even when total cholesterol level and blood pressure are the same. Thus, preventing the excess cardiovascular morbidity and mortality associated with diabetes is arguably the most pressing treatment goal. However, this does not mean that normalising blood lipid or clotting factors is more important than normalising blood glucose. Indeed, high blood glucose levels are now recognised to contribute directly to the pathogenesis of macrovascular disease in both diabetic and non-diabetic subjects (6,7).

Until recently, HbA_{1c} levels were thought to be mainly influenced by fasting and pre-prandial blood glucose levels. However, human beings spend much of their time in the post-prandial state and therapies which specifically reduce post-prandial glycaemia may be superior for improving overall glycaemic control and reducing the risk of complications (8,9). The degree of post-prandial glycaemia appears to adversely alter vascular function and directly contribute to thickening of the intima wall (10). For this reason, both the amount and type of carbohydrate are probably more important than presently recognised.

HISTORICAL PERSPECTIVES ON CARBOHYDRATE

Before the discovery of insulin in 1922, diets prescribed for diabetes were very low in carbohydrate, around 5% of energy, and very high in fat, around 75% [reviewed by Truswell (11)]. Even after the advent of insulin, doctors were cautious and very low carbohydrate diets continued. By 1930, diet prescriptions of carbohydrate had risen to 15% of energy. In the 1940s and 1950s, carbohydrate allowances had come up to 25–30% of energy and carbohydrate exchange lists came into use. There was little questioning of the principle that carbohydrates were bad for people with diabetes and focus was on the insulin treatment.

By the 1970s pharmaceutical treatments had expanded with the introduction of oral hypoglycaemic drugs and the average carbohydrate intake rose to about 40% energy. Prohibition of sucrose was now the main message. With extreme caution, several experimental studies compared higher carbohydrate diets ($>50\%$ energy) with the traditional diabetes diet and found improved glucose tolerance or insulin sensitivity (12–14). In the late 1970s, there was a revolution in thinking about diabetic diets and a spurt of experimental studies indicated that high-carbohydrate diets were no worse, if not better, for people with diabetes because they lowered blood cholesterol levels (see below). By then, low-fat, high-carbohydrate diets were being recommended for the prevention and treatment of cardiovascular disease in the general population.

Since 1980, dietary recommendations for people with diabetes have unanimously emphasised reducing saturated fat intake. However, if saturated fat intake is reduced, the energy has to be replaced by some other nutrient. Because there are concerns about potential adverse effects of high-protein diets on renal and bone health, the choice is either more carbohydrate or more unsaturated fat. And here lies the controversy. Since carbohydrate is the main glycaemic element in the diet (being the main precursor of blood glucose), an increase in dietary carbohydrate might be expected to result in greater post-prandial glycaemia and compromise diabetes control. An increase in fat, on the other hand, might promote weight gain and decrease insulin sensitivity.

CURRENT RECOMMENDATIONS FOR CARBOHYDRATE INTAKE

For the past 20 years, most diabetes associations around the world have recommended high-carbohydrate diets that are low in fat and high in fibre for people with diabetes (15,16). The British Diabetic Association's recommendations state that carbohydrate should provide 50–55% of the total energy content of the diet while fat should contribute 30–35% of energy intake, of which $<10\%$ should be saturated fat, $<10\%$ polyunsaturated fat (PUFA) and

10–15% monounsaturated fat (MUFA) (17). However, there is concern in some quarters that 50–55% of the total energy intake as carbohydrate may have adverse effects on blood triglyceride (TG), HDL-cholesterol and glucose levels compared with high-fat diets (>35% total energy) enriched with MUFA (18,19). During the 1990s, this issue has been the focus of much research. On the basis of the resulting evidence, the American Diabetes Association's guidelines now recommend that 60–70% of energy be divided between carbohydrate and monounsaturated fat, depending on patient preference and the appropriate nutritional goals for their medical status (20).

WHAT IS THE SCIENTIFIC BASIS FOR RECOMMENDING HIGH CARBOHYDRATE INTAKE?

There is no doubt that the goal of increasing carbohydrate intake was actually to reduce fat consumption, especially saturated fat. People with diabetes were no longer dying of diabetic ketoacidosis but coronary heart disease. In fact, some experts suspected that the prescribed high-fat (and high saturated fat) diabetic diets might actually be partly responsible for the heightened risk of cardiovascular disease among people with diabetes. Several well-designed intervention studies in diabetic subjects were undertaken and showed that high-carbohydrate diets (55–70% energy) could result in lower blood cholesterol and TG levels with no deterioration in glycaemic control compared to traditional 'diabetic' diets containing less carbohydrate and more saturated fat (12–14, 21–25). Indeed, much to their surprise, HbA_{1c}, glucose tolerance and fasting glucose were often improved following treatment with a high-carbohydrate diet. This implied that insulin sensitivity was improved on a higher carbohydrate intake as had been earlier demonstrated in non-diabetic subjects (26). Thus, in the 1980s, diabetes associations in the United States, Canada, Australia and Britain independently agreed that there was sufficient evidence to advocate an increase in the carbohydrate content of the diabetic diet.

It is important to note that these early studies used high-carbohydrate diets that were heavily based on wholegrain cereals, vegetables and legumes that concomitantly contained very large amounts of fibre (upwards of 75 g per day). This is more than three times that normally consumed and presented a very real challenge for the average person with diabetes. In addition, unless the fibre was of the soluble, viscous or leguminous type, then post-prandial blood glucose peaked at higher levels on the high-carbohydrate diet (27). Not surprisingly, some degree of weight loss was seen after subjects completed the high-fibre, high-carbohydrate dietary treatments, but not after the low-carbohydrate, high-fat diets. We now know that energy restriction *per se*, even before significant weight loss is evident, improves all aspects of diabetes control (28). A deficit of calories, rather than a high-carbohydrate intake, may

well have explained much of the improved profile. Even without weight loss, consistent benefits of high-carbohydrate diets have been reported only when the diets incorporate relatively unrefined, high-fibre foods (legumes, wholegrains, cruciferous vegetables, fruit) – resulting in meals that are somewhat different from those eaten by the general population.

Because very high fibre intakes are perceived as being unpalatable and hard to achieve, many health professionals took the view that simply increasing total carbohydrate intake was the main priority because this achieved the objective of lowering saturated fat. The American Diabetes Association, for example, presently recommends a moderate intake of dietary fibre of 20–35 g per day to help lower LDL-cholesterol and does not consider that dietary fibre offers significant benefits for glycaemic control (20).

WHAT ARE THE POTENTIAL ADVERSE EFFECTS OF HIGH-CARBOHYDRATE DIETS?

During the 1980s and 1990s, a number of controlled intervention studies in healthy individuals who maintained their body weight showed that high-carbohydrate diets often resulted in higher blood TG levels and lower HDL-cholesterol levels – changes that are atherogenic and increase the risk of coronary heart disease – despite improved total and LDL-cholesterol levels (29). These findings sparked particular concern for people with diabetes because their lipid abnormalities tended to be higher TG and lower HDL-cholesterol level rather than the high total and LDL-cholesterol typically observed in non-diabetic individuals (18). Hence the magnified risk of atherosclerosis in people with diabetes might be related to blood lipid risk factors that are specifically worsened by high-carbohydrate diets.

The biochemical mechanisms responsible for increased plasma TG levels following low-fat, high-carbohydrate diets remain uncertain but are clearly different to those responsible for elevated TG levels following increased fat intakes. Parks *et al*. (30) demonstrated that high-carbohydrate diets reduce the clearance of VLDL-TG from the plasma, but do not increase VLDL-TG secretion or *de novo* lipogenesis in the liver as had been postulated.

The mechanisms by which high-carbohydrate diets decrease HDL-cholesterol are also unknown and should be a priority in future research. In two recent cross-sectional studies of healthy adults, a significant inverse association was found between serum HDL-cholesterol concentration and dietary GI for both men and women (the higher the GI rating of the diet, the lower the HDL concentration) (31,32). In fact, the glycaemic index of the diet was the only dietary variable significantly related to serum HDL-cholesterol. These findings suggest that post-prandial glucose and insulin responses may directly influence HDL levels.

THE SCIENTIFIC BASIS FOR RECOMMENDING HIGH-MUFA DIETS FOR DIABETES

Many diabetes experts argue in favour of allowing a higher MUFA intake for people with diabetes, on the grounds that high-carbohydrate diets can increase blood glucose, insulin and TG levels and reduce HDL-cholesterol levels. A meta-analysis of nine studies with a total of 133 subjects comparing these two approaches to diet therapy in patients with diabetes revealed that high-MUFA diets (22–33% of energy intake; total fat = 37–50% energy) improved lipoprotein profiles as well as glycaemic control (19). Compared to high-carbohydrate diets (50–60% energy intake), high-MUFA diets reduced fasting TG and VLDL-cholesterol levels by about 20% and caused a modest increase in HDL-cholesterol (4%) but had no effect on LDL-cholesterol. There was no evidence that high-MUFA diets induced weight gain in these tightly controlled studies. However, there are several limitations that need to be raised before deciding whether they provide sufficient evidence to formulate recommendations for therapeutic diets:

- None of the studies controlled for/considered the confounding effects of the glycaemic index of the high-carbohydrate diets.
- The diets contained relatively small amounts of fibre (< 30 g/day, mostly in processed form).
- The studies were conducted under tightly controlled conditions, not allowing spontaneous weight loss/weight gain to occur.
- Most of the studies were of very short duration (two to four weeks), the longest being six weeks.
- A third of the studies were conducted by the same research group.
- Improvement in glycaemic control was assessed on the basis of urinary glucose and fasting, pre-prandial, post-prandial or 24-h blood glucose and insulin profiles.
- Notably, in the six studies that assessed HbA_{1c} or fructosamine (the best markers of long-term glycaemic control), none of the changes were significant.

Thus we lack the evidence that high-MUFA diets improve overall diabetes control by the most valid measure of disease risk (i.e. HbA_{1c}). This contrasts with the consistent effect of low-GI, high-fibre, carbohydrate-rich diets in lowering HbA_{1c} (see below). Furthermore, the positive effects of high-MUFA diets on blood lipids are often seen only when the high-MUFA diet is extremely high in fat (as much as 45–50% of energy) and very low in carbohydrate (about 35% of energy) (33). In studies with smaller and more realistic dietary changes, the effects of MUFA on blood lipids are more modest. One can question the effect of such a very high-fat diet on insulin sensitivity, weight control and ability of patients to comply. In fact, the largest study of this kind suggests that

the beneficial effects of MUFA on insulin sensitivity disappear when fat intake exceeds 38% total energy (33).

These studies confirm that there are definitive adverse risks associated with *low-fibre*, high-carbohydrate diets. However, they do not prove that the original recommendation to increase both carbohydrate and fibre was wrong, nor do they allow us to say whether a diet rich in monounsaturated fat is better than a high-fibre, high-carbohydrate diet.

THE EVIDENCE THAT LOW GLYCAEMIC INDEX, HIGH-CARBOHYDRATE DIETS ARE SUPERIOR

It is now well established that both the type and amount of carbohydrate influences the degree of post-prandial glycaemia (34). The type of carbohydrate is best described by its glycaemic index, a ranking of foods according to their immediate effect on blood glucose levels (Figure 11.1). Per gram of carbohydrate a food with a GI of 80 (e.g. potato) has twice the glycaemic impact of a food with a GI of 40 (e.g. pasta) and this applies even in mixed meals (35,36) (Figure 11.2). The proportions of starch, sugar, fat or fibre in foods are not a good guide to GI. Many common starchy foods (even wholemeal versions) such as bread, rice and breakfast cereals have surprisingly high GI values, while foods containing sugars often have a relatively low GI (37). Reducing the overall GI of the diet involves substitutions within those food groups that contribute most of the dietary carbohydrate (Table 11.1).

The GI of foods is highly relevant to the management of Type 1 and Type 2 diabetes. In nine well-designed long-term studies in diabetic subjects, low-GI diets (GI values $<55\%$) were shown to reduce glycosylated proteins (HbA_{1c} and/or fructosamine) by an average of almost 11% over periods ranging from two to 12 weeks (38). At the end of the low-GI, high-carbohydrate diet, urinary C-peptide levels (a measure of endogenous insulin demand) fell by an average of 20%, daytime blood glucose levels decreased by 16%, and total cholesterol and TG were reduced by 6% and 9%, respectively (39). Triglyceride levels fell to a much larger extent (by up to 20%) in patients with overt hypertriglyceridemia. In a recent, randomised, cross-over study, clotting factors were normalised in patients with Type 2 diabetes by a low-GI, high-carbohydrate diet, but unchanged by a high-GI diet containing similar amounts of energy, protein, fat, carbohydrate, starch and fibre (40).

Studies comparing the effects of high-GI versus low-GI carbohydrate-rich diets have been longer (four to 12 weeks) than the high-MUFA studies, and unlike the latter, have been able to document beneficial changes in HbA_{1c} and/or fructosamine levels. In the few studies that have directly compared high-carbohydrate, low-GI diets with high-MUFA diets, HDL levels were increased on both (compared to the

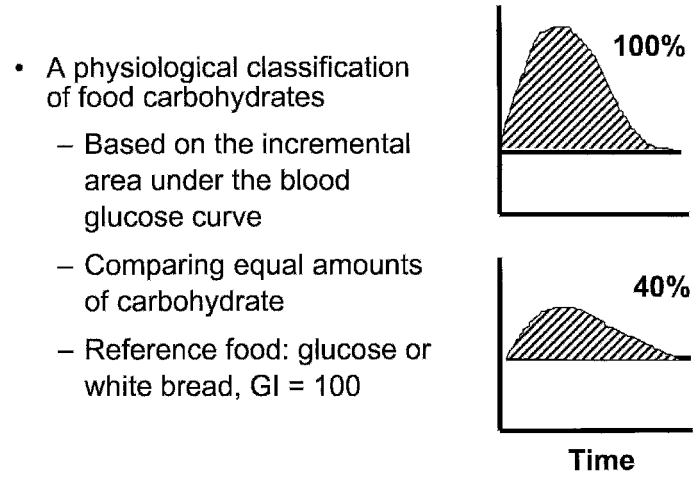

Figure 11.1 The derivation of the glycaemic index

high-carbohydrate, high-GI diet) but insulin secretory function and sensitivity appeared to be better only on the low-GI diet (41,42). Reductions in HbA_{1c} or fructosamine levels of 10% on the low-GI diets have been criticised as being 'modest', yet changes of this magnitude are commonly seen with oral hypoglycaemic drugs. Furthermore, these 'modest' changes were achieved in patients in free-living conditions, not in a controlled metabolic ward situation. Although the high- and

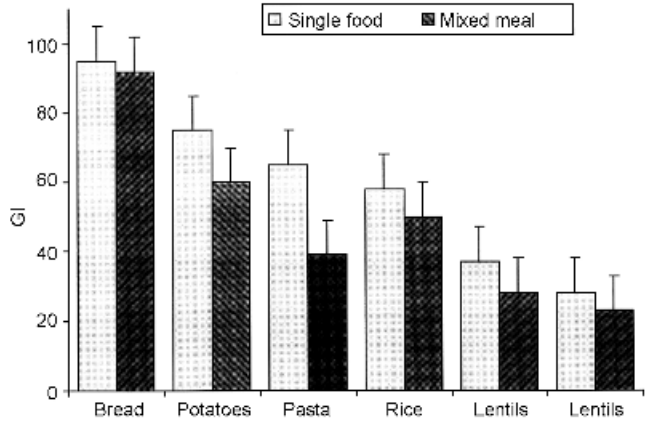

Figure 11.2 The GI of single foods predicts the GI of mixed meals in subjects with Type 2 diabetes
Source: Redrawn from Bornet *et al.* (35).

Table 11.1 A low-GI diet is achieved by substituting high-GI foods with GI alternatives. Breads, breakfast cereals and potatoes contribute the majority of carbohydrate in Western diets. Changes within these food groups have the biggest impact on the diet's overall GI

High-GI food	Low-GI alternative
Bread, ordinary wholemeal or white	Bread containing a high proportion of wholegrains ('granary' breads), sour dough breads, stone ground breads
Most breakfast cereals	Unrefined cereal such as oats (muesli or porridge). Some processed cereals (e.g. All-Bran)
Potato (all varieties)	Sweet potatoes, pasta, noodles, legumes
Most varieties of rice	Basmati or other high amylose rices
Cakes, biscuits and muffins	Versions made with fruit, oats, wholegrains
Tropical fruits such as bananas	Temperate climate fruits such as apples and stone fruit

low-GI diets were usually designed to be similar in macronutrient composition, in some studies the low-GI diet contained more fibre. Indeed in one outpatient study, dietary education with emphasis on low-GI foods resulted in higher carbohydrate and fibre intakes and less saturated fat intake than achieved by patients given 'traditional' dietary counselling (43). However, on the whole, studies comparing high- and low-GI diets have contained much less fibre (<50 g per day) than the earlier studies that provided the basis for recommending high-carbohydrate diets in diabetes (>75 g per day). Lastly, low-GI, high-fibre diets may be the *only* strategy (diet or drug) that enables HbA_{1c} to be improved while simultaneously reducing the incidence of hypoglycaemic episodes in Type 1 diabetes (44).

These findings suggest that any adverse effect of high-carbohydrate diets on blood lipids is almost certainly linked to the high GI of most such diets. Indeed, any strategy that slows down the rate of digestion and absorption of carbohydrate (e.g. nibbling versus gorging, alpha-glucosidase therapy or purified supplements of viscous fibre, as well as low-GI diets) has been shown to improve glucose and lipid metabolism in diabetes (45).

WHICH DIET IS BEST FOR IMPROVING INSULIN SENSITIVITY?

The body's sensitivity to the hormone insulin predicts how well it handles a meal containing carbohydrate, i.e. how easily and quickly it restores normal glucose levels after consumption. In insulin-resistant states, large amounts of insulin are needed to restore euglycaemia and glucose and/or insulin levels may still be high 2 h later. In Type 2 diabetes, insulin resistance is often severe and is combined with impairments in insulin secretory capacity. Obesity, particularly

abdominal obesity, is known to worsen insulin resistance and increase the risk of Type 2 diabetes (46).

The degree of insulin sensitivity is also affected by the energy content and macronutrient composition of the diet. Epidemiological and dietary intervention studies in humans indicate that a high-fat, energy-dense diet promotes weight gain and the development of obesity (47), impairs insulin sensitivity and increases the risk of developing Type 2 diabetes (48). Relatively high intakes of saturated fat appear to worsen insulin resistance and are also associated with higher blood levels of LDL-cholesterol and a greater risk of atherosclerosis (49,50).

Questions still remain about the optimal diet for improving insulin sensitivity in Type 2 diabetes. It is well recognised that higher carbohydrate intakes are related to improved insulin sensitivity in non-diabetic individuals (26). This is likely to be true in the early stages of Type 2 diabetes, but as pancreatic beta-cell function declines, higher carbohydrate intakes could compromise remaining insulin secretory capacity. There are few studies that document changes in insulin sensitivity directly in diabetic subjects and these tend to be inconclusive. Low-GI diets appear to improve insulin sensitivity in coronary heart disease patients (51,52) and animal models (53).

Indirect evidence suggests that the fibre content and GI of the diet may influence insulin sensitivity, weight gain and the risk of developing Type 2 diabetes. In the CARDIA study of young adults, low fibre consumption predicted 10-year weight gain and fasting insulin levels (a measure of insulin resistance) more strongly than did total or saturated fat consumption (54). Fibre but not amount and type of fat was associated with 2-h insulin levels. Two other large-scale prospective studies in healthy subjects showed that diets based on low-fibre, high-GI foods doubled the risk of developing Type 2 diabetes, after controlling for known risk factors such as age and body mass index (55,56). Importantly, the total carbohydrate and refined sugar content of the diet, and the amount and type of fat consumed, were *not* found to be independent risk factors in these studies.

DIETS FOR WEIGHT CONTROL: IS THE AMOUNT AND TYPE OF CARBOHYDRATE IMPORTANT?

Weight loss is usually a major treatment goal in Type 2 diabetes, but the ideal dietary composition for weight control is still the subject of debate. Many health professionals are concerned that high-fat diets, irrespective of the type of fat, might promote weight gain. The prevalence of obesity is often lower in people with high carbohydrate consumption (expressed as a percentage of energy) than in those with high fat intakes (but this is not always true). In animal studies, high-fat diets induce faster weight gain and greater insulin resistance compared with high-carbohydrate diets, whether fed *ad libitum* or isocalorically (50). In humans, several studies have shown that *ad libitum*

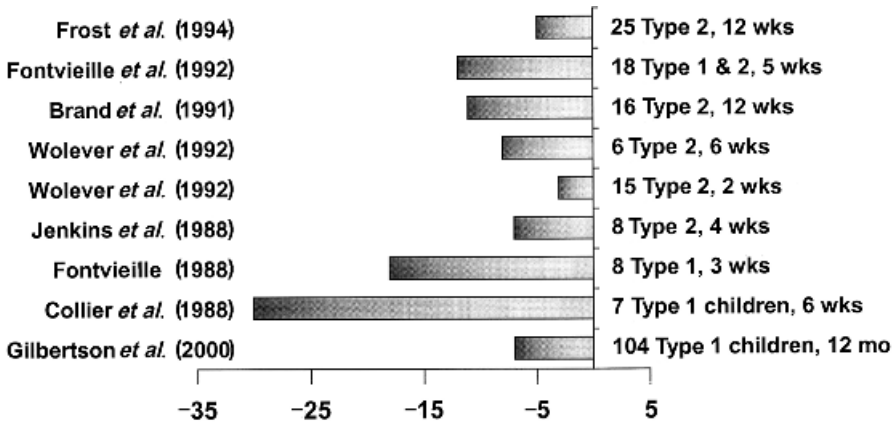

Figure 11.3 Reduction in glycosylated proteins (glycosylated haemoglobin or fructosamine) on low GI in nine studies comparing high-carbohydrate, low- versus high-GI diets
Source: Redrawn from Brand-Miller *et al.* (38).

consumption of high-carbohydrate diets, even one high in refined sugar, was more effective in promoting long-term weight loss than higher fat diets (47,57,58).

Despite this, results from several recent intervention trials have indicated that high-MUFA diets are just as effective as high-carbohydrate diets in producing weight loss in diabetic subjects (59,60). However, in these studies MUFA was used in the context of a strictly controlled low-energy diet. By directly controlling energy intake, any spontaneous reduction in energy intake and body weight associated with the high-carbohydrate or high-fibre diet was unlikely. The long-term effect of *ad libitum* consumption of Western diets enriched in MUFA is currently not known. There is concern that the promotion of energy-dense, high-MUFA foods to diabetic subjects [as recommended by the American Diabetes Association (20)] may lead to gradual weight gain.

THE SATIETY VALUE OF HIGH-CARBOHYDRATE DIETS

The satiating capacity of high-carbohydrate diets may be the major explanation for weight control benefits. The energy density of foods strongly influences the amount of food people consume and consequently influences body weight (61). High-fat foods are energy dense, very palatable and less satiating, a combination which makes them easy to 'passively overconsume'

(62). On the other hand, less refined, 'natural' high-carbohydrate foods (legumes, wholegrains, fruits and starchy vegetables) are more bulky and difficult to overeat. In laboratory studies comparing the short-term filling powers of equal-calorie portions of different foods, the weight of food per 1000 kJ was the strongest determinant of short-term satiety (63). However, many new reformulated low-fat foods on the market (e.g. snack products, biscuits, ice cream, yoghurt) are as energy dense as their full-fat counterparts and unlikely to offer weight control benefits.

Low-GI diets may be particularly beneficial for weight control in people with diabetes. Low-GI foods are more satiating, calorie for calorie, than their high-GI counterparts [reviewed by Ludwig (64)]. Low-fat, energy-restricted diets based on low-GI foods have been found to result in greater weight loss in overweight people than conventional reduced-fat diets with a higher GI rating (64,65). Higher satiety resulting from the prolonged digestion and absorption of carbohydrate in the small intestine and the reduced post-prandial insulin secretion may explain these findings. In animal studies, high-GI diets promoted faster weight gain, higher body fat, higher adipocyte volume and hyper-triglyceridaemia than low-GI diets providing similar amounts of energy and macronutrients (66,67). High-GI diets were also associated with increased myocardial infarction in the Nurses' Health Study (68).

EVIDENCE FOR SUCROSE RESTRICTION IN DIABETIC DIETS

Many randomised, controlled trials have shown that the isocaloric substitution of moderate amounts of refined sucrose for starch in diabetic diets has no adverse effects on blood glucose or lipid levels in people with diabetes (69–71). In fact, several studies show *improved* glycaemic control, especially in children with Type 1 diabetes (72). This makes sense when we consider that most foods containing sugar have a GI less than 60, while that of most modern starchy foods is over 70 (37,73). Many diabetes associations now officially recognise that sucrose restriction is not necessary in diabetic diets, although some put an upper limit of 30 g per day (the average intake in the non-diabetic population is about 60 g per day). Unfortunately, the dietary dogma of sucrose avoidance in diabetic diets is so well entrenched in the mind of the public and most health professionals that little change has occurred in practice. Intense sweeteners and low-joule soft drinks are almost universally recommended in diabetic diets in the belief that this will enhance both glycaemic control and weight loss. This often detracts from more important dietary messages for people with diabetes (e.g. reduced saturated fat, increased high-fibre and low-GI foods).

The belief that sucrose facilitates excessive energy intake is one reason for continued use of intense sweeteners. However, there is little evidence that the

long-term use of artificial sweeteners is particularly useful for weight loss – reducing sugar intake saves fewer calories than reducing dietary fat by the same amount (74). Some large-scale dietary surveys have shown that people who consume higher amounts of sugar and less fat tend to have lower body weights (75–77). Refined sucrose consumption correlates inversely with fat intake in both non-diabetic and diabetic populations. In addition, research shows that a moderate–high intake of sugar is not associated with a reduced intake of vitamins and minerals (78). One of the reasons for this is that sucrose increases the palatability and intake of nutritious foods such as cereals and dairy products. Sucrose also satisfies an instinctual desire for sweetness and has many functional roles in foods that extend beyond its sweetening power, including preservative, textural and flavour-modifying qualities.

Fructose has also been used as a sweetener in diabetic diets because it has a smaller blood glucose (GI = 20) and insulin-raising effect than isocaloric amounts of sucrose. Concerns about its potential to raise TG and LDL-cholesterol levels have limited its use (20), but in amounts up to 12% of energy, no untoward effects have been seen in subjects with diabetes (79).

Other nutritive/calorie-containing sweeteners such as maltodextrins, corn syrup, fruit juice/concentrate, honey, molasses, dextrose and maltose do not offer any advantage over sucrose in terms of energy content or glycaemic response. Indeed, post-prandial glycaemia is higher after maltodextrins and corn syrup than after sucrose. Sugar alcohols (sorbitol, mannitol, xylitol) and isomalt used as sweeteners in sugar-free confectionery produce a lower glycaemic response than sucrose and inhibit dental caries formation. Excessive consumption (> 20–30 g per day) should be avoided because of their laxative effect.

REALISTIC DIET PRESCRIPTIONS

Weight loss and weight control are arguably the most challenging aspects of managing diabetes, yet are likely to offer the most immediate and obvious benefit. People with diabetes find it more difficult to lose weight and maintain the loss compared with those without the disease. Fortunately, it is now clear that they do not need to reach their ideal body weight in order to improve their metabolic status; as little as a 5–10% reduction in body weight is sufficient to result in clinically relevant benefits (80).

Long-term weight control requires a comprehensive approach involving lifestyle changes, *not just food and energy restriction*. A modest reduction in energy intake (about 250 to 500 calories from the daily energy intake) and an increase in daily physical activity by 250 to 500 calories are realistic. A combination of strategies may help promote weight loss:

- Emphasis on low saturated fat, low-GI, high-carbohydrate foods – to promote satiety and reduce hyperinsulinaemia.
- Modest caloric restriction not extreme – to prevent excessive hunger.
- Distribution of carbohydrate intake throughout the day – smaller more frequent meals to reduce post-prandial hyperglycaemia.
- Increased physical activity – even incidental activity – to promote higher energy expenditure.
- Behaviour modification techniques and relaxing activities – to reduce stress-related eating.
- Support from family and other professionals – to increase compliance.

No single dietary approach will be suitable for all patients. Meal plans and dietary modifications need to be tailor-made to suit each patient's needs and lifestyle. Current medical status (HbA_{1c}, blood lipid levels, home blood glucose monitoring results, nutritional status, body weight, medication) needs to be assessed before any dietary modifications are recommended. Dietitians should reinforce that the dietary and exercise 'prescription' is an essential component of diabetes management, irrespective of medication.

THE FUTURE

Currently, many health professionals on both sides of the carbohydrate debate tend to believe that there is an ideal diet for everyone with diabetes – the 'one diet fits all' approach. But the future is likely to see the percentage of carbohydrate in the diabetic diet 'individualised' to increase compliance and take account of usual food habits. Emphasis on changes in the *types* of carbohydrate foods and *types* of oils and margarines may be more important to overall diabetes control than the amount of carbohydrate versus fat *per se*. While there is consensus that type of fat is important, there is less recognition of the major effects of fibre and rate of digestion of carbohydrate on glucose and lipid metabolism. There is sufficient evidence to say that a high-carbohydrate diet based on high-glycaemic index foods (even wholemeal versions) is probably not desirable in the management of diabetes. The *glycaemic load* (GI of the diet × carbohydrate content) of the diet needs to be considered and evaluated in intervention and observational studies, particularly in relation to insulin sensitivity, HbA_{1c} and risk of complications.

Post-prandial elevations in the level of blood glucose appear to be a major determinant of HbA_{1c} levels and therefore rates of complications of diabetes. But there is generally little recognition at present that post-prandial blood glucose values can be improved by diet, not just by drug therapy. If we are to recommend major changes to diet in the management of diabetes, then evidence-based medicine requires proof that they are safe and effective in the

long term. Randomised, controlled trials in free-living populations should be the standard of evidence and outcomes should be measured in terms of changes in HbA_{1c} and rates of complications rather than surrogate measures such as fasting and 2 h post-prandial blood glucose and insulin responses, lipid concentrations and blood pressure. Unfortunately, current dietary recommendations are often based on results from dietary intervention studies as short as two to four weeks, some better controlled than others, using surrogate measures of glucose and lipid metabolism. Taken as a whole, at the present time there is better evidence favouring high-carbohydrate, high-fibre, low-GI diets in the overall management of diabetes.

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