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# Nutrition therapy in the treatment of diabetes

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The optimal nutrition therapy for diabetes, like that of obesity, remains controversial because of the lack of high quality studies. There is increasing evidence that the conventional low-fat, high-carbohydrate diet is not ideal. Recent randomised controlled trials suggest that alternative dietary strategies with moderately lower carbohydrate content, including those with a lower glycaemic index, or higher fat (Mediterranean-style) or higher protein content are equally or more effective for managing diabetes. Observational studies suggest that such dietary approaches will also reduce the risk of complications. In Australia, consideration of both quantity and type of carbohydrate (carbohydrate counting, glycaemic index and glycaemic load) is an established part of diabetes management.

There is universal support for dietary therapy as an integral part of the treatment of both type 1 and type 2 diabetes. Although there are aspects of management unique to type 1 diabetes, much is similar because both types share the need to manage hyperglycaemia and hypoglycaemia on a day-to-day basis and to manage weight effectively. They both give rise to similar microvascular and macrovascular complications through common mechanisms arising from suboptimal glycaemic and metabolic control.

There is controversy surrounding the optimal diet or dietary pattern for diabetes because of lack of high quality studies. Current dietary recommendations are largely based on historical consensus and include limiting total fat, saturated fat and trans fatty acids, choosing high fibre and wholegrain foods, and monitoring and regulating carbohydrate intake to assist with glycaemic control. Moderate weight loss achieved through energy restriction and regular physical activity markedly improves glycaemia and cardiovascular risk factors in those who are overweight [1]. Unfortunately, on their own, these recommendations have been largely unsuccessful in practice in achieving good blood glucose control or maintenance of weight loss over the long term. For this reason, studies employing alternate strategies (eg high protein, low carbohydrate, vegetarian, Mediterranean-style, low glycaemic index/glycaemic load) have been explored.

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## The rationale

While there is now unequivocal evidence that dietary advice in conjunction with exercise can prevent or delay the development of type 2 diabetes in at-risk individuals (ie those with prediabetes) [2, 3], the role and type of dietary advice following the diagnosis is much less clear. In this chapter, we argue that the optimal diet composition for managing diabetes is not one judged simply by improvements in glycaemic control. Rather we contend that diets for diabetes should have proven efficacy in *all* of the following parameters:

- Optimising glucose metabolism (improving HbA1c, reducing postprandial glycaemia, reducing frequency and risk of hypoglycaemia, reducing glucose variability)
- Achieving appropriate weight loss in overweight or obese individuals or healthy weight maintenance in those not overweight
- Maintaining weight targets over the long term (years)
- In children and adolescents who are growing, providing appropriate energy intake and nutrients for optimal growth and development, while preventing or treating excessive weight gain
- Improving insulin sensitivity, even in the absence of weight loss
- Improving markers of the metabolic syndrome
- Improving markers of inflammation
- Reducing the future risk of cardiovascular disease and other complications linked to diabetes
- Being enjoyable and sustainable over the long term
- Maintaining healthy attitudes to food and avoiding eating disorders
- Being compatible with sustainable agricultural practices.

On this basis, there is evidence that some diets fare better than others. In the remainder of this review, we describe the best evidence available for different dietary approaches. We use randomised controlled trials in individuals with diabetes as our gold standard, and where not available, long-term prospective observational studies in large cohorts. Not all studies have been undertaken in individuals with diabetes, but the findings in overweight and obese individuals can be assumed to be indicative. It is also important to recognise that large-scale, long-term quality dietary composition studies are difficult to achieve in diabetes and obesity because of cost, patient-adherence factors, confounding concomitant therapies and other practical issues.

## Improving HbA1c and glucose metabolism

A 2007 Cochrane systematic review [4] concluded that there were no high quality dietary studies that demonstrated the efficacy of any form of dietary treatment in type 2 diabetes. Indeed, in their view, there was no good evidence that diet alone (usually low-fat, high-carbohydrate advice) improved glycated haemoglobin, a long-term measure of blood glucose control, at one year. They found that very low-calorie diets were associated with a

rise in glycated haemoglobin at 12 months. In contrast, adoption of exercise (± diet) was highly effective, reducing glycated haemoglobin by a significant 1%. That review considered there was insufficient data to compare one form of dietary advice with another, although their criteria excluded studies shorter than six months. In a more recent Cochrane review [5], Thomas and Elliott identified 11 high quality studies of low glycaemic index (GI) or low glycaemic load (GL) diets lasting one to 12 months. They concluded that on average, glycated haemoglobin decreased by 0.5% and hypoglycaemic episodes declined more on the low GI/GL diets compared to the conventional low-fat advice. Since then, Jenkins et al. have published the largest study to date, showing that a low GI diet was more effective at improving glycated haemoglobin than a high cereal fibre diet in 210 individuals with type 2 diabetes. While some small-scale studies [6, 7] suggest that high-protein, low-carbohydrate diets are more effective than other diets in improving glucose metabolism, there is a lack of high quality data. In a two-year trial, Shai found that changes in fasting glucose and insulin levels were more favourable among the diabetic participants assigned to the Mediterranean diet than those assigned to the low fat.

In addition to long-term goals, the optimisation of day-to-day blood glucose levels is also an important consideration, particularly the control of postprandial hyperglycaemia and avoidance of hypoglycaemia. Type 2 diabetes subjects (and type 1 subjects, discussed later) treated with insulin or other hypoglycaemic agents are at risk of hypoglycaemia which poses significant health and social risks, and these risks need to be minimised. There is also some evidence from observational data for adverse effects of blood glucose fluctuations (independent of overall glycaemic control) on diabetes complications [8]. For these reasons, monitoring and regulating the amount and type of carbohydrate on a daily basis (or carbohydrate counting) is recommended in diabetes management [9]. Such approaches have been more intensively applied to type 1 diabetes and are discussed below, but are also widely used in type 2 diabetes.

### Reduce weight

In adults with type 2 diabetes, weight loss and weight-loss maintenance are essential components of management yet only in the last decade have diets of different composition been given the degree of scientific study and scrutiny they deserve. Ideally, the management goal would be normalisation and maintenance of weight, BMI and abdominal circumference to within the normal healthy range; however it is recognised that this is currently a rarely achievable goal and therefore modest weight loss of 5% to 10% is an initial target [10]. In overweight children and adolescents who are still growing, weight maintenance is usually the initial goal, which translates into improved BMI as they grow, although modest weight loss that still allows normal linear growth is desirable in the obese [11]. Prevention of progression to overweight or obesity is also a primary goal at all ages in type 2 and type 1 diabetes.

Several meta-analyses and reviews have concluded that low-carbohydrate, high-protein diets [12–15] and low GI or low glycaemic load diets [5, 16, 17] may be more successful for weight loss than traditional low-fat, high-carbohydrate diets. Similar benefits have recently

been suggested for Mediterranean-style diets [18, 19]. Indeed, in our view, a conventional low-fat, high-carbohydrate diet appears to be one of the slowest ways to lose weight. However, one high-quality, long-term study found no difference in weight-loss outcomes between high vs low protein, high vs low fat, or high carbohydrate vs low carbohydrate diets at two years [16]. Only ~15% of subjects maintained a weight loss of 10% at the two-year mark. Notably, carbohydrate quality (high vs low GI) was not a variable.

### Maintain weight loss over the longer term

The ability of different diets to maintain weight loss is arguably the most important attribute of a diet but there is little research to date to guide recommendations. Some evidence suggests that alternative dietary approaches may also be more successful for maintenance of the achieved weight loss than low-fat, high-carbohydrate diets [10–14]. In the Diogenes Study [17], ~800 overweight and obese individuals were randomised to one of five *ad libitum* diets after 8% of body weight loss had been achieved by means of a very low-energy diet. Two levels of protein and two levels of GI were studied. After six months, the high-protein and low GI diets were shown to be equally successful, but the combination of both high protein *and* low GI, produced the greatest absolute weight-loss maintenance and the lowest study drop-out rate. Importantly, this large well-designed study found that the conventional low-fat diet with average protein and GI was associated with the fastest rate of weight re-gain.

### Improving insulin sensitivity

Improving insulin sensitivity is arguably the defining attribute of a good diet because insulin resistance is a fundamental contributor to the pathogenesis of type 2 diabetes. Indeed, insulin resistance combined with a defect in pancreatic insulin secretion, cause a relative insulin deficiency that occurs in most people with type 2 diabetes. The modifiable factors that worsen insulin resistance are excessive body weight, physical inactivity and smoking. Increasing muscle mass and lowering abdominal fat both markedly improve insulin sensitivity. Hence a combination of weight loss and physical activity, particularly resistance exercise, is the ideal lifestyle intervention to reduce the risk of type 2 diabetes.

However, diet composition has been shown to have a separate additional effect on insulin sensitivity. Observational studies and intervention trials have shown the macronutrient distribution (ie the ratio of fat:carbohydrate:protein energy) and the quality of individual macronutrients directly influence insulin sensitivity.

The effect of dietary fat and carbohydrate on insulin sensitivity have been debated for decades. Some of the controversy stems from divergent findings in animals versus humans, and in differing study designs. Insulin resistance can be induced in animal models by diets high in fat, sucrose or fructose. However, a single bout of exercise or high starch meal can completely reverse the defect. In humans, some studies suggest that a high intake of fat is associated with impaired insulin sensitivity but this may be modified by the type of fat and by the type of subject. Several studies indicate that a high-saturated-fat diet may be

especially deleterious in physically inactive, sedentary individuals, while short (three to four weeks), studies in lean, healthy subjects have shown no effects on insulin sensitivity [18]. The KANWU study included 162 healthy subjects who received isoenergetic diets for three months containing either a high proportion of saturated fatty acids (SAFA) or mono-unsaturated (MUFA) acids [19]. Within each group there was a second assignment to fish oil supplements or placebo. Insulin sensitivity was significantly impaired by the SAFA diet (–10%) but did not change on the MUFA diet. However, the beneficial effects of MUFA were *not* seen when total fat intake exceeded 37%E (the median level of participants). Addition of n-3 fatty acids did not influence insulin sensitivity, and neither diet altered insulin secretion. Taken together, these and other findings suggest that at fat intake close to average in industrialised nations (ie ~35% E), it is preferable to maintain the *higher* fat intake but to reduce relative saturated fat intake and thus increase the proportion of MUFA or PUFA fatty acids, rather than increase the percentage energy derived from carbohydrate.

The quantity and quality of carbohydrate can also influence insulin sensitivity. In a cross-sectional analysis of ~3000 individuals in the Framingham Offspring Study, wholegrains, total fibre from all sources, as well as fibre from cereals and fruit, were inversely related to insulin resistance [20], but there was no relationship with total carbohydrate intake. Dietary glycaemic index (GI) and glycaemic load (GL) were also directly related to insulin resistance with approximately 10% more insulin resistance in the highest quintile of GI than in the lowest.

*Some* high-carbohydrate diets appear to have beneficial effects on insulin sensitivity. In healthy, young persons, isoenergetic substitution of *high fibre* carbohydrate foods for saturated fatty acids improves insulin sensitivity within four weeks [21]. Indeed, carbohydrates consumed without fibre may produce detrimental effects [22]. In individuals with diabetes, higher-carbohydrate intake has the potential to raise postprandial glucose and increase insulin demand, an effect that might worsen insulin resistance. Low GI diets, however, in which the carbohydrates are more slowly digested and absorbed, resulting in lower postprandial glycaemia, have improved insulin sensitivity in some studies. Insulin sensitivity was 45% higher as judged by euglycaemic clamp procedure in type 2 diabetes patients who ate a low GI diet for four weeks compared with a macronutrient-matched high GI diet [23]. The alpha-glucosidase inhibitor, ecarbose, which slows carbohydrate digestion but is not absorbed into the systemic circulation, also produces improvements in insulin sensitivity [24]. Low GI diets also improve insulin sensitivity in overweight women with polycystic ovarian syndrome [25].

The evidence for wholegrains vs refined grains to improve insulin sensitivity is inconsistent. A well-designed but small intervention study, compared six to ten servings of breakfast cereal, bread, rice, pasta, muffins, cookies and snacks from either whole or refined grains (in both cases mostly ground to flour) in a conventional high-carbohydrate, low-fat diet. Using the glucose clamp, insulin sensitivity was higher after six weeks on the wholegrain diet compared with a similar period on the refined grain diet [26]. Unfortunately, this finding was not confirmed in the larger WHOLEheart Study in which 60 g to 120 g per day wholegrain foods were ingested for up to 16 weeks by overweight individuals [27].

The effect of fructose and sucrose on insulin sensitivity also remains controversial. Studies in animals, often fed extremely high intakes (eg 70% of total calories), have shown a detrimental effect of fructose and sucrose compared with starch or glucose [28]. When fructose and glucose were compared directly, fructose was found to be the culpable moiety.

The evidence in humans, however, suggests that fructose and sucrose in *realistic* amounts have beneficial effects on insulin sensitivity. In lean, young healthy males, a diet containing 25% sucrose produced higher insulin sensitivity as assessed in a two-step clamp procedure than a diet containing 1% sucrose [29]. Similarly, a study in patients with type 2 diabetes showed that a diet with 10% fructose produced a 34% improvement in insulin sensitivity measured by the glucose clamp [30]. In this study, patients lived in a hospital environment and all food was provided. Finally, using the glucose clamp, no effects on insulin sensitivity were noted after three months of a 13% fructose vs sucrose diet in type 2 diabetes [31]. It is conceivable, however, that at very high intakes (>30% E), sucrose and fructose have adverse effects.

### Improving markers of the metabolic syndrome

The best evidence that dietary changes can improve the metabolic syndrome comes from landmark studies in which intensive lifestyle interventions prevented or delayed progression from impaired glucose tolerance to type 2 diabetes mellitus [2, 3, 36]. Both studies employed low-fat, high-carbohydrate diets (30% of energy from fat, 10% from saturated fat) in combination with physical activity to achieve the goal of weight loss. Mistakenly, these findings have since been perceived as a rational basis for recommending low-fat, high-carbohydrate diets. Unfortunately, weight loss *per se* likely played the most important role, such that the superiority of low-fat diets for people with diabetes and the metabolic syndrome is questionable.

In intervention trials, increased carbohydrate intake is well known to increase serum triglycerides and lower HDL, two markers of the metabolic syndrome [32]. Indeed, the similarity implies that high carbohydrate diets of a certain nature play an etiological role in the metabolic syndrome. Several meta-analyses and reviews have concluded that low-carbohydrate, high-protein diets [12], low-GI or low-GL diets [5, 33, 34] and Mediterranean-style diets [35] may be more effective (or just as effective) for improving markers of the metabolic syndrome as traditional low-fat, high-carbohydrate diets. This higher effectiveness holds true over both the shorter and the longer term.

### Improving inflammatory markers

Chronic low-grade inflammation plays a recognised role in the development and the progression of both type 2 diabetes and vascular disease [36]. Inflammation is also the likely intermediary between aspects of carbohydrate nutrition and chronic disease. A single glucose challenge has been shown to increase the production of reactive oxygen molecules with mitochondria and activation of pro-inflammatory transcription factors, such as nuclear factor-kappaB (NF-kB). In individuals with impaired glucose tolerance, obese or

persons with type 2 diabetes, the result is pronounced and lasts longer (>2–3 h) than in non-diabetic persons [37]. Indeed, in individuals with type 2 diabetes, glucose fluctuations during postprandial periods have a more specific triggering effect on oxidative stress than chronic sustained hyperglycaemia [38]. High GI/GL diets that are associated with greater postprandial glucose excursions may therefore promote low-grade inflammation.

In short-term metabolic ward studies in healthy individuals, the consumption of high GI foods has been directly linked to the creation of oxidative stress, as judged by higher activation of NF-kB and increased generation of nitrotyrosine. The presence of oxidative stress can also be detected by an acute decline in antioxidant concentrations in plasma following a meal. Botero et al. [39] observed differences in fasting and postprandial total antioxidant capacity over the course of a five-hour observation period following one week on a low or high GI diet in overweight men. Plasma total antioxidant capacity in response to diet may therefore be the first metabolic adaptation linking carbohydrate nutrition to type 2 diabetes.

### Reducing future risk of microvascular and macrovascular complications

There are no intervention studies examining the risk of diabetes complications for diets of differing composition. In their absence, large, long-term prospective observational studies in healthy individuals can be regarded as indicative. In meta-analyses, replacing saturated fat with polyunsaturated fat is associated with a 26% reduction in risk of cardiovascular disease [40]. In contrast, replacing saturated fat with carbohydrate is linked to a non-significant increase in risk. Carbohydrate intake (whether high or low) is not usually an independent predictor of the development of type 2 diabetes mellitus. In meta-analyses, however, quality of carbohydrate intake as assessed as the GI, GL and dietary fibre shows a consistent positive relationship to the risk of type 2 diabetes mellitus and cardiovascular disease (CVD), despite non-significant findings in some individual prospective studies [41]. The highest relative risks (>2) are observed among those with both a higher dietary GI or GL and lower (cereal) fibre intake. Recently, some prospective cohort studies have demonstrated that higher intake of high GI carbohydrates, but not low GI carbohydrates, is associated with greater risk of developing CVD [42]. Similarly, there is increased risk of type 2 diabetes mellitus and overweight associated with dietary patterns that are characterised by higher intakes of refined grains or white bread, ready-to-eat breakfast cereals, sugar-sweetened beverages, potatoes or French fries, sweets or sweet bakery products [43]. In contrast, a protective pattern commonly included carbohydrate choices such as fruits, vegetables, legumes, wholemeal or wholegrain bread and high-fibre breakfast cereals.

The dietary approaches that reduce the risk of developing type 2 diabetes, obesity or CVD are likely to be the same as those that reduce the risk of complications in individuals with diabetes. Interestingly, the apparently protective diets share a unifying mechanism of reducing postprandial glycaemia and insulinaemia, despite variable macronutrient distribution. Thus it is possible but remains unproven at present whether any diet that facilitates a reduction in postprandial glycaemia without worsening dyslipidemia, is likely to improve insulin sensitivity and relieve the burden on the beta cell, thereby reducing

the risk of complications. The least effective diet will be one that increases postprandial glycaemia and places extra demands on beta-cell function. These adverse effects will be most detrimental for individuals with severe insulin resistance, that is, many individuals with diabetes.

### Enjoyable and sustainable diets

In the short term, a wide variety of diets will reduce weight and improve cardiovascular risk factors under realistic clinical conditions. But only a minority of individuals have been found to sustain dietary adherence over the longer term. There is no single diet that is associated with satisfactory dietary adherence, although a high protein–low GI diet was associated with significantly higher completion rates in the Diogenes Study of weight-loss maintenance [17]. More extreme diets, such as very low carbohydrate diets (Atkins) or very low-fat diets (eg Ornish) are more likely to be discontinued [44]. To manage the epidemic of obesity and diabetes, practical techniques to increase dietary adherence are urgently needed.

One way to do this is to offer a broad range of healthy diet options, to better match individual patient preferences, lifestyles and cultural backgrounds. Dansinger et al. [44] found that only one in four individuals were adherent one year after counselling, yet those individuals who sustained the greatest weight loss and risk reduction were those who were able to comply, no matter what the diet composition. Thus a diet's ease of adoption, rather than diet composition per se, is an important attribute of an effective diet. These findings challenge the assumption that one type of diet is appropriate for everyone and that popular diets can be ignored.

Another way to increase adherence is to offer intensive, systematic and individualised dietary counselling. In the Diabetes Prevention Program, freely available intensive dietary counselling was effective in sustaining weight loss, and cardiovascular and diabetes risk reduction over a three-year period, irrespective of ethnicity, socioeconomic group or cultural background [2]. Only 7.5% of individuals dropped out of the study despite the large number of face-to-face visits over a long period. While dietary counselling requires more resources from the health system, cost-effectiveness analyses show that the intensive lifestyle interventions cost no more than drug interventions [45]. Moreover, over the long term, we can expect fewer side effects and more benefits (eg reduced risk of other lifestyle diseases) associated with adherence to a healthy diet.

### Vegetarian diets for diabetes

A vegetarian or semi-vegetarian diet, with emphasis on plant foods such as wholegrains, legumes, nuts, fruits, vegetables, is widely believed to have a number of *nutritional* benefits over a meat-based diet for the management of diabetes. Vegetarian diets can be (but not necessarily) lower in saturated fat, higher in dietary fibre and richer in micronutrients such as magnesium, ie factors that are associated with higher insulin sensitivity. Observational studies show that a vegetarian or vegan diet is associated with reduced risk of development of

type 2 diabetes and lower risk of complications in those with existing diabetes. The European Prospective Investigation into Cancer and Nutrition study found that among participants with diabetes, there was a significant inverse association between cardiovascular mortality and intake of total vegetables, legumes and fruit [46]. In women with type 2 diabetes, frequent consumption of nuts was associated with a 50% reduction in the risk of CVD and a more favourable lipid profile [47]. One long-term intervention study, in which animal protein was partly replaced with soy protein, reported significant improvements in total cholesterol, LDL cholesterol, triglycerides and CRP levels in individuals with diabetes [48]. Nonetheless, it is difficult to separate vegetarianism from other healthy lifestyle behaviours, which also improve risk factors. Some evidence suggests it is the absence of processed meat products from vegetarian diets rather than meat per se that offers benefits.

### Compatibility with sustainable agricultural practices

Some health authorities have recommended vegetarian diets over meat-based diets on the grounds of environmental sustainability. However, the issue is debated even among the experts. Much of Australia is classed as arid or semi-arid, with vast areas that are unsuitable for crop agriculture, but suitable for grazing. In the Australian context, most agriculturalists and those who are knowledgeable consider extensive, free-range grazing systems with relatively low stocking rates and a rotation of cropping and pastures as sustainable. Such relatively low input–low output systems have been sustained for decades, eg the sheep-wheat-legume-based pasture systems in south-eastern Australia. The animal/pasture component in the rotation actively promotes sustainability. In Australia, Good Agricultural Practice and Best Management Practice production systems are widely recognised and used.

The notion that we should not eat meat or dairy for environmental reasons may in itself be a flawed argument because avoidance of meat requires the production of more grain to obtain the same nutrition. Animal source foods are nutrient dense and generally are able to nourish the human population more effectively than plants. Overconsumption and waste are profoundly more important for both sustainability and health than simply considering plant vs animal consumption. At present, over a quarter of all foods consumed are 'non-core foods', ie foods that are not essential to good health, yet demand a disproportionately large amount of scarce resources such as water, and generate large amounts of greenhouse gases.

### Particular aspects of nutrition therapy for type 1 diabetes

Although the underlying pathogenesis is initially different in type 1 vs type 2 diabetes (absolute insulin deficiency vs relatively greater insulin resistance, respectively) and mean age of onset differ, the subsequent metabolic derangements that stem from chronic hyperglycaemia and that are involved in the pathogenesis of diabetes complications are common to both. The principles discussed above for the nutritional management of type 2 diabetes therefore also apply to the management of type 1 diabetes, particularly as they relate to weight control, avoidance of hyperglycaemia and hypoglycaemia and optimisation

of metabolic control and insulin sensitivity. There are some particular considerations for type 1 diabetes, mainly relating to carbohydrate counting and type of carbohydrate.

Type 1 diabetes has an acute and earlier age of onset than type 2 diabetes, with the majority of cases diagnosed in children and adolescents after a short duration of symptoms. Most people diagnosed with type 1 diabetes are not obese, even prior to the weight loss that often occurs before the diagnosis. Because all type 1 subjects require insulin therapy, nutritional management commences with a different focus. A major priority is to have a nutritional and insulin plan that meets short- and long-term glycaemic goals, including control of hyperglycaemia, and minimises hypoglycaemia and glucose fluctuations. Additional goals are long-term maintenance of a healthy weight and prevention or reduction in risk of long-term complications that are not usually present at diagnosis (but can often be in type 2 diabetes). Not surprisingly, in line with trends to increasing prevalence of overweight and obesity in society, children diagnosed with type 1 diabetes are heavier and taller than their peers [49] and this has been postulated to be associated with the earlier onset of type 1 diabetes seen in various registries and termed the 'accelerator hypothesis'. This hypothesis suggests that the relative insulin resistance associated with being overweight or obese accelerates the progression to clinical onset of type 1 diabetes in those that are genetically predisposed [50]. There is also evidence that in the longer term, those with type 1 diabetes are heavier than their non-diabetic peers [51]. There is also a subgroup with type 1 diabetes who have significant inherent insulin resistance in addition to that associated with obesity; the term 'double diabetes' has been used for this group and they are often considered for co-treatment with insulin sensitising agents. Therefore the management of overweight and obesity in young people with type 1 diabetes is a frequent necessity.

Carbohydrate counting has become an established part of the nutritional management in type 1 diabetes and is a necessary component of flexible multiple daily injection plans and insulin pump therapy. There has been a movement away from recommending relatively fixed carbohydrate intake (or carbohydrate prescription) to more flexible and physiological approaches in which subjects are taught to match their insulin doses with their desired and counted carbohydrate intake. There is reasonable evidence to conclude that the use of carbohydrate counting with flexible insulin to carbohydrate ratio is associated with improved HbA1c and quality of life, but no conclusions regarding effects on body weight or severe hypoglycaemia [52, 53]. The DAFNE study demonstrated that adult type 1 diabetes subjects using such an approach achieved a significant improvement in HbA1c and quality of life over six months, without worsening severe hypoglycaemia or cardiovascular risk. However, there are not yet comparable data in children and adolescents. Despite such studies, there are limited data on the best methods and teaching to employ or the degree of accuracy required in carbohydrate counting. While trends have been to aim for more accurate matching of insulin to carbohydrate, one study demonstrated that postprandial BGL control was not affected by variations of up to 17% in the carbohydrate amount covered by the same insulin dose [54]. With insulin pump therapy, the only available randomised prospective study recently demonstrated that in adults, accurate carbohydrate counting had advantages over empirical estimation of doses in a number of parameters over 24 weeks, including HBA1c, BMI, waist circumference and quality of life [55].

There is also evidence that the type of carbohydrate as well as the quantity is important in minimisimg glucose fluctuations and hypoglycaemia; these are the concepts of glycaemic index and glycaemic load [4]. While there are no data in children and adolescents, the conclusions from the adult studies cited above should also be applicable to young people and their diabetes nutritional plans should incorporate these concepts.

## Conclusions

The findings of recent, high quality studies have been surprising and yet remarkably consistent. The conventional low-fat diet is probably *not* the optimal diet for managing diabetes, weight or risk factors for cardiovascular disease. Rather, there appear to be superior dietary patterns that offer good diabetes control, more flexibility and greater ability for individuals to choose a diet that they enjoy and can sustain over the longer term. These alternative nutritional strategies include those with a moderately lower carbohydrate content, those with a lower glycaemic index, or higher fat (Mediterranean-style), or higher protein content. Very low-carbohydrate diets (with higher protein and fat) cannot be recommended to people with type 1 diabetes because of adverse effects on renal function. In individuals with type 2 diabetes, there is a lack of long-term dietary studies that go beyond the measurement of risk factors (eg glycated haemoglobin) to assess hard outcomes such as death and diabetes-related complications. Unfortunately, such studies are difficult and expensive to conduct, not amenable to patent generation and unlikely to eventuate. Long-term prospective observational studies in healthy individuals remain our best guide. At the present time, diets low in saturated fat and trans fats, with a lower GI and GL, and higher fibre content, represent our best advice for increasing life expectancy and quality of life in individuals with diabetes. With current insulin delivery methods in type 1 diabetes, carbohydrate counting and consideration of glycaemic index and glycaemic load remain an important component of the nutritional plan.

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