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Technical Review
Recommendations for the nutritional management of patients with diabetes mellitus

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Aims and implementation

The quality of life of the individual person must be considered when defining nutritional objectives. Health care providers must achieve a balance among the demands of metabolic control, risk factor management, patient well-being and safety. All nutritional programmes should be adapted to the specific needs of the individual person within the context of his or her own culture and lifestyle, which may change with time.

Over the last twenty years, as part of general moves towards evidence-based medicine, accountability and improved quality of care, diabetes associations have sought to question dietetic principles and practice. Greater emphasis on the scientific evidence has led to standardising recommendations on diet composition, and more recently on methods of implementation. In making such recommendations, a logical succession of underpinning questions needs to be answered. Firstly, is any difference required from food eaten in the normal general population? Secondly, are current recommendations for health in the general population applicable to patients with diabetes? Thirdly, are there reasons for recommending special foods for patients with diabetes? Fourthly, is evidence from controlled experiments of diabetic versus control subjects applicable to a free-living population, particularly a population other than that which was used for the specific experiment?

To answer these questions, the goals of dietary advice for patients with diabetes need to be made explicit: the general goal is maintenance of health and quality of life, for as long as possible, and freedom from vascular complications of diabetes. This will include contributing through diet to avoidance of short-term symptoms from hypoglycaemia (for example, insulin and sulphonamide treated patients) and from hyperglycaemia, and also to avoiding long term complications. Increasingly, evidence suggests that normalisation of blood glucose and lipid profiles are valid aims.

Recommendations will change over time as new data becomes available. The need for recommendations emerges when there is a clearly documented case such that there is cast-iron evidence of benefit, but importantly also where the evidence is absent or incomplete and there is a need for safe practical guidance pending new information. It is this latter situation, where issues are contentious and any guidelines will be debated, that a professional body most needs to exercise judgement in assessing the evidence, to identify research needs and to take responsibility for recommending changes. Dietary recommendations are therefore derived from evidence in the published literature, but often supported where necessary by consensus, or common sense views.

Dietary recommendations are made in terms of nutrients and their biochemical effects, in terms of physiological effects of foods such as glycaemic index, and in terms of the foods themselves. They are used for general patient education materials, but also have to be transformed into advice to individual patients and their families. Considerable interpretation may be involved. Advice to individual patients may be modified through negotiation with the carer and supplemented or rationalised by education from other appropriate sources. Advice may be qualitative or quantitative, but must be in terms of foods, patterns of eating, relation of food and eating to adjunctive therapy, for example, exercise, drug therapy, insulin replacement, and the context of diet in terms of age, lifestyle, cultural setting.

Energy balance and body weight

Detailed recommendations concerning energy intakes are not required for those diabetic subjects with a body mass index in the acceptable range (BMI: 19–25 kg/m^2) for adults. Those who are overweight should be encouraged to reduce their caloric intake and to enhance their energy expenditure so that BMI moves towards this range. Similarly, advice should be given for caloric restriction to avoid weight gain in those with this tendency, even while within the acceptable weight range. Advice concerning the reduction of energy dense foods and in particular those high in fat, will usually help to achieve weight loss without the need for precise energy prescription. If these measures do not achieve the desired weight reduction, it may be necessary to offer more precise advice concerning the dietary modifications needed to produce an energy deficit of at least 500 kcal/d. The amount of physical activity needs to be taken into account when considering total energy requirements.

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Metabolic rate and energy requirements

The importance of considering the energy requirement of an individual in the clinical situation would be when prescribing dietary components in absolute amounts. This is seldom desirable in clinical practice, but controlling energy balance is often desirable in metabolic research studies. The biggest determinant of energy requirement is body weight, more specifically the lean body mass, including muscle and the more metabolically active organs which determine basal metabolic rate. Basal metabolic rate (BMR) is measured by direct calorimetry, but can be predicted with considerable accuracy for an individual from published regression equations based upon body weight (WHO, 1985).

Total 24 h metabolic rate, which must be equalised by energy intake to maintain energy stores and body weight, is made up from the BMR, plus the energy costs of eating and digesting food (diet induced thermogenesis) and the more variable energy costs of physical activity and thermogenic stimuli such as cold-exposure, caffeine and smoking. Physical exertion accounts for between 10 and 50% of total energy requirements. The energy cost of physical exertion for any individual is itself closely related to body weight. Total energy expenditure and therefore total energy requirement is expressed as a function of basal metabolic rate. In most populations total energy expenditure, and therefore total energy requirement for weight stability, lies between BMR × 1.3 and BMR × 1.8 (WHO, 1985). The 'activity factor' of 1.5 proposed to promote cardiovascular health, can reasonably be applied to people with diabetes. This includes an amount of 'cardioprotective exercise' equivalent to about 20 mins per day moderate exertion. A lower activity factor of BMR × 1.3 is appropriate to the sedentary.

Physical exertion

Evidence from longitudinal cohort studies is convincing that physical activity protects against development of NIDDM in middle-aged women (Manson *et al*, 1991) and men (Helmrich *et al*, 1991). Regular moderately strenuous exercise is associated with a reduced relative risk of developing diabetes of about 0.6–0.7 and this figure still applies in overweight subjects. The benefit seems most marked in men at high risk of NIDDM (overweight, hypertensive or family history of NIDDM), and is restricted to intensity of activity greater than just walking (Helmrich *et al*, 1991). The benefit applies irrespective of family history in women. Other research points to several mechanisms for this benefit from exercise (Manson *et al*, 1991).

In patients with IGT, insulin action and glucose transporter GLUT-4 in muscle are increased by exercise training in the absence of change in diet, with improvement in glucose tolerance (Hughes *et al*, 1993). The benefit of training in NIDDM was questioned until recently, perhaps because early studies used inadequate exercise intensity. Work loads of 60–70% VO_2 max have now been shown to improve glucose tolerance and HbA1. Each bout of exercise depletes muscle glycogen stores, thus allowing more prolonged glucose disposal while these stores are replenished, and the size of glycogen stores is increased by training (Devlin, 1992). The benefit of training in NIDDM is lost within 3–10 d of stopping training (Heath *et al*, 1983; Mikines *et al*, 1988). With particular relevance to NIDDM and risks of cardiovascular disease, physical exercise leads to reduction in plasma triglyceride and increases in HDL and HDL2 subfraction (Schwartz, 1990). Blood pressure is elevated physiologically or excessively in NIDDM during exertion (Blake *et al*, 1990) but is lowered afterwards. The net effect on average blood pressure of diet and exercise is beneficial (Barnard *et al*, 1992). There may be other benefits of exercise in NIDDM on plasminogen activator activity, and as an adjunct to diet for weight loss (Schwartz, 1990).

The principal problem with the use of exercise to improve metabolic control in IDDM is that the net effect of exercise on blood glucose is unpredictable because of the many variables influencing glucose supply and utilisation, including state of nutrition and metabolic control, before and at the onset of exercise, the time of day that exercise is begun, duration and intensity of exercise, the state of physical training of the patient, type, dose and site of insulin injection. Exercise regimens for an individual with IDDM must be undertaken only after adequate medical examination and comprehensive teaching for self-management of their metabolic control (Wasserman *et al*, 1994). The evidence is persuasive for recommending regular moderately strenuous exercise for IDDM based on data from Moy *et al* (1993), showing a 3-fold reduction in mortality over 7 y in diabetic patients who were physically active (>2000 kcal/week) compared to sedentary (<1000 kcal/week) patients.

Walking at a normal pace expends energy at three times the BMR. Walking for 4 h per week will achieve an extra energy expenditure of 2000 kcal/week. This form of low intensity aerobic exercise can be easily sustained on a regular basis to improve insulin sensitivity as well as reduce body weight (Yamanouchi *et al*, 1995). For those who are otherwise fit, more strenous exertion may be taken, if blood glucose levels are properly monitored. A medical examination and exercise tolerance test may be prudent for older patients before starting, as CVD may be present (Nesto *et al*, 1988; Uusitupa *et al*, 1985). People's appetites automatically adjust, so it is unnecessary to make quantitative increases in diet advice. Typically, blood glucose falls more after exercise than during it, and late hypoglycaemia may relate to glycogen depletion. Extra food (including carbohydrate) is usually needed after exercise, eating before exercise impairs performance, so insulin reduction is preferred if exercise is planned.

Body mass index (BMI)

The 1988 DNSG of the EASD recommendation, that BMI should lie within the range 20–25 kg/m^2 (DNSG, 1988) was drawn from evidence in the normal non-diabetic population. There are small differences between men and women regarding optimum range for body mass index, 20–25 for men, 18.7–23.6 for women (RCP, 1983). Amongst the overweight, insulin sensitivity is decreased, and with it most aspects of diabetic control. Even modest weight loss of under 10% body weight improves insulin sensitivity and glucose tolerance and reduces serum cholesterol and blood pressure (Goldstein, 1992). It is recognised that overweight patients seldom lose weight to achieve BMI under 25 kg/m^2 but that much more modest degrees of weight loss may be sufficient to normalise glucose tolerance (Eriksson *et al*, 1991). The reduced life expectancy of overweight people with diabetes is improved in those who lose more weight and is probably normalised without achieving BMI under 25 kg/m^2 (Lean *et al*, 1990;

Williamson *et al*, 1995).

Longitudinal and actuarial evidence from diabetic individuals is not available to indicate an acceptable weight range different from the general population. The mortality risk of diabetic individuals in the American Cancer Association Study (Lew *et al*, 1979) was dramatically increased when BMI exceeded 25 kg/m^2. Data from the Nurses Health Study indicates a graded augmentation of risk, as BMI rises above 22 kg/m^2, which is exaggerated in those with diabetes (Manson *et al*, 1991).

Body shape and fat distribution

Diabetic or IGT patients, whether overweight or not, have a high proportion of intra-abdominal fat and associated increased health risks related to more marked insulin resistance, associated dyslipidaemia and hypertension (Van Gaal *et al*, 1988). There is evidence that weight loss leads to greater improvements in cardiac risk factors in individuals with higher WHR (Lean *et al*, 1995). It is possible that patients with central fat distribution would benefit from lower BMI, namely, more weight loss, and from other lifestyle advice than individuals with peripheral fat distribution simply on the basis of their increased risk (Yudkin, 1993). Because of the enlarged intra-abdominal fat mass, diabetic patients have more body fat than non-diabetic individuals of the same height and weight. Skin fold methods underestimate body fat in people with IGT or diabetes, and this is a reason to prefer methods based on waist circumference to estimate body fat (Lean *et al*, 1996)

Weight loss

Many overweight patients are frustrated by slow rates of weight loss using conventional dietary means. This results largely from unrealistic expectations of weight loss during dieting. It should be emphasised that sustained weight loss of 1–2 kg per month (that is, a reduction of 200–500 kcal/d below the requirement for average weight maintenance) should be considered satisfactory and a 2–4 kg per month reduction, excellent. Given that people tend to increase in weight up to the 6th decade, simply avoiding weight gain may be considered a success. Using conventional dietetic and behavioural methods, weight loss of 5–10 kg can be achieved in newly diagnosed NIDDM patients. Short term use of appetite-modifying drugs is unhelpful.

Diet for insulin treated overweight patients

Some patients with type 1 diabetes are overweight and weight gain in IDDM increases insulin requirement. The DCCT trial has shown how intensifying insulin treatment leads to a 12 month weight gain of about 3 kg (DCCT Research Group, 1988). This gain results from reducing glucose loss (70%) and decreasing metabolic rate (30%) (Carlson *et al*, 1993). Overweight patients treated with insulin should be encouraged to lose weight with a goal of body mass index of 18.5–25 kg/m^2. Insulin requirement can be titrated against blood glucose by home monitoring, making dosage changes in steps of approximately 10% per day. It is commonly found that insulin requirement drops dramatically during active weight loss but may restabilise at a higher level after weight restabilisation. Type 2 diabetic patients who are obese and treated with insulin may be able to withdraw insulin completely following weight loss without any deterioration in diabetic control if pancreatic response is adequate (Turkington *et al*, 1982).

It is also recognised that many patients, particularly young women, use reduction in insulin dose alone as a means of achieving weight loss. Reducing insulin dose leads to deterioration in diabetic control with consequent elevation of metabolic rate and glycosuria. This behaviour, which may be accompanied by bulimia and anorexia nervosa is likely to have an adverse effect on diabetic complications.

VLCD and NIDDM

There have now been a large number of published studies using very low calorie diets (VLCD) in patients with diabetes. It is clear that insulin sensitivity and indices of diabetic control can be improved rapidly using VLCD. The most marked plasma lipid changes are falls in triglyceride and rises in HDL cholesterol (Uusitupa *et al*, 1990). Most of the benefits are very rapid and relate to energy restriction, not weight loss (Henry *et al*, 1991).

Using VLCD, more weight is lost (10–15 kg) over a 3–6 month period than using conventional diets (5–10 kg). Even though longer term assessment over 1–2 y does not show continued benefit from the earlier use of VLCD, this approach has a place for some patients (Henry *et al*, 1991), if properly supervised. The use of VLCD in a cyclical on/off manner (as is used in an unsupervised way in the general public) has not been formally studied in diabetic patients. Anxieties remain about alteration of body composition and perhaps bone loss after repeated weight cycling or cyclical VLCD use (Avenell *et al*, 1994).

Diet macronutrient composition for weight loss

Fat is the most energy-rich of all energy-providing nutrients, therefore reducing fat intake is important in reducing total energy intake. Experimental data has suggested that to achieve satiety a minimal carbohydrate intake has to be maintained (Tremblay *et al*, 1991; Flatt, 1987), independent of the dietary fat content. Tremblay *et al* (1989) have shown that ingestion of a high fat diet is associated with an increase in total energy intake. Overweight people may, for genetic reasons, have impaired tissue oxidation of fat and a resultant increase in absolute need for carbohydrate (Tremblay, 1992). If the prevailing diet is low in carbohydrate and high in fat, these people may consume larger quantities of food with resultant weight gain. Change to a higher carbohydrate, lower fat diet allows the carbohydrate requirement to be satisfied without the high intake of fat, and low energy intake.

Macronutrient composition of the diet plays a role in weight maintenance (Westerterp, 1993). Adiposity is positively correlated with dietary fat content and negatively correlated with dietary carbohydrate (Miller *et al*, 1990; Dreon *et al*, 1988). Several studies (Sheppard *et al*, 1991; Lissner *et al*, 1991; Kendell *et al*, 1987) have shown that low fat diets (15–20% E) are associated with a 10–25% decrease in total energy intake, and simple shift to a low fat diet alone may result in weight loss. In addition, Prewitt *et al* (1991) have shown that long term consumption of a low fat, high carbohydrate diet resulted in significant reductions in body weight and fat in both obese and non-obese people, despite increased total energy intake. To achieve optimal weight loss through diet, advice to restrict both total energy as well as dietary fat is needed (Schlundt *et al*, 1993).

Maintaining weight loss

In most hands weight regain is usual after slimming and is of the order of 60–70% of that lost at 1 y. In general, radical interventions such as VLCD have rather poor long term maintenance, although intermittent VLCD can be effective (Wing *et al*, 1994). There are suggestions that cognitive and behavioural methods can improve long term maintenance of weight loss but the evidence is weak (Perri *et al*, 1993). Gastric restriction surgery for overweight NIDDM patients usually leads to massive and maintained weight loss (40–80 kg) and normalised glucose tolerance (Pories *et al*, 1992). Long term results are not available, but in some cases when risks are high (for example, young obese NIDDM), when dietary methods have failed, and where there is a good multidisciplinary team for lifelong review, the contentious assertion 'Diabetes is a Surgical Disease' (Pories *et al*, 1992) may be justified. Drug therapy with the newer agents in weight management may be more appropriate for long term suppression of weight gain rather than to achieve major weight loss, but such use will require evidence of safety and efficacy, and regulatory approval.

Components of dietary energy

Saturated fatty acids should provide under 10% total energy. A lower intake may be beneficial if LDL-cholesterol is elevated. Polyunsaturated fatty acids should not exceed 10% of dietary total energy. Protein intake should range between 10 and 20% total energy. Thus most dietary intake should come from a combination of carbohydrates and monounsaturated fatty acids with a *cis*-configuration. Carbohydrate containing foods which are rich in soluble fibre or have a low glycaemic index are especially recommended.

Dietary fats

Saturated fat

Patients with diabetes mellitus have increased risk of coronary heart disease, cerebrovascular disease and peripheral vascular disease of a magnitude of two to four times greater than those without diabetes mellitus but with other comparable risk factors (Stamler *et al*, 1993). In addition, the syndrome of low HDL-with high triglycerides, seen even in well treated NIDDM (Manzato *et al*, 1993), contributes to the risk of mortality (Fontbonne *et al*, 1991; Uusitupa *et al*, 1993).

A decrease in saturated fat intake can lower levels of total and LDL-cholesterol, an important predictor of CHD in people with diabetes (Laker, 1987). Epidemiological evidence suggests that people with diabetes in populations consuming low fat diets have a reduced morbidity and mortality (Uusitupa *et al*, 1993). In Europe, the average person with IDDM diabetes has a saturated fatty acid intake of 14–17% dietary energy (Toeller *et al*, 1996; Toeller, 1993).

Even though there are no large definitive studies demonstrating that the reduction of well established risk factors prevents coronary artery disease in diabetic patients, there is no reason to believe that diabetics will not be responsive to such treatment as is recommended to the general population (Yudkin, 1993). In keeping with this, it is generally accepted that saturated fat intake should constitute less than 10% dietary energy.

There is some evidence relating saturated fat to insulin sensitivity. In NIDDM subjects a stepwise increase in saturated fat from 5–45 g had no impact on the glycaemic response to carbohydrate (Gannon *et al*, 1993), although insulin increased after consumption of 15 g. Rasmussen *et al* (1996) found that butter increases the insulin response more than does olive oil (mono-unsaturated rich), and large amounts also increase fatty acid and triglyceride concentrations which may lead to hyperlipaemia and reduced insulin sensitivity in the long term. One cross-sectional survey found an association between saturated fat intake assessed from a food frequency questionnaire and microalbuminuria in Type I diabetic patients.

Individual fatty acids have distinct properties, with evidence suggesting that most of the deleterious effects of saturated fatty acids are due to lauric (C 12:0) acid and myristic (C 14:0) acid and palmitic (C 16:0) acid, while stearic acid (C 18:0), unlike other long chain fatty acids, may have no hypercholesterolaemic effect (Bonanome *et al*, 1988; Katan *et al*, 1994; Kris-Etherton *et al*, 1994). With the exception of a recent study (Storm *et al*, 1995) demonstrating a neutral impact of stearic (C 18:0) acid as compared to a cholesterol elevating influence of palmitic (C 16:0) acid in NIDDM, results obtained so far apply only in healthy subjects. For practical purposes it is reasonable to consider saturated fatty acids as a single group.

Polyunsaturated fatty acids

Increased intakes of PUFA help to lower LDL-cholesterol. However when large quantities are taken, a reduction in HDL-cholesterol may occur (Mattson *et al*, 1985; Schaefer *et al*, 1981). *In-vitro* experiments have also shown that polyunsaturated fatty acids are more susceptible to oxidative modification and therefore possibly more atherogenic. However, high-PUFA diets given to a group with NIDDM have been reported to lead to similar levels of lipid peroxidation as those given a high MUFA diet and significantly lower when compared to a group given a high saturated fat diet (Parfitt *et al*, 1994). The WHO recommendation for the general population is of 3–7% dietary energy (WHO, 1990).

Trans-isomer fatty acids

Some *trans*-isomers of PUFA occur naturally. Most are formed during partial hydrogenation of vegetable oils to produce margarine and vegetable shortening and can comprise up to 40% of fat in some foods, particularly baked goods and pastries (Enig *et al*, 1990). *Trans*-fatty acid isomers have biological effects similar to saturated fatty acids, and replacement of naturally occurring *cis*-forms with *trans*-isomers in body fat has been associated with increases in LDL cholesterol (Mensink *et al*, 1990; Zock *et al*, 1992). *Trans*-fatty acid intake has also been linked with increases in lipoprotein-(a) (Mensink *et al*, 1992; Nestel *et al*, 1992). Decreases in HDL cholesterol by 0.1 mmol/l were seen when 7.7% of dietary energy was obtained from *trans*-fatty acids (Zock *et al*, 1992).

The Nurses Health Study (Willett *et al*, 1993) has subsequently shown that high intakes of foods that are a major source of *trans*-isomers may be associated with risk of coronary heart disease. Against this however is the recent EURAMIC study (Aro *et al*, 1995) which found no major effect of dietary C 18:1 *trans*-fatty acid, as determined by analysis of subcutaneous fat, on risk of acute myocardial infarction. Again specific information in people with diabetes is lacking, but it would seem prudent

to consider the metabolic effects of *trans*-fatty acids and avoid high intake. Information about *trans*-fatty acid contents in foods is increasing. For practical purposes *trans*-fatty acid can be classified together with saturated fatty acid in view of their similarity.

Fish oils and n-3 fatty acids

Fish oils or n-3 fatty acids have been found able to reduce plasma triglycerides and VLDL concentrations in the diabetic population (Mori *et al*, 1990; Rillaerts *et al*, 1989; Popp-Snijders *et al*, 1987). One study also observed improved blood rheology when 2.7 g of sardine oil was consumed daily (Kamada *et al*, 1986). However, others have failed to show any change in membrane fluidity or whole blood viscosity in diabetic patients (Rillaerts et al, 1989; Popp-Snijders *et al*, 1987). Platelet aggregation is increased in diabetic patients (Jones *et al*, 1986). While bleeding time has been prolonged in healthy people (Lorenz *et al*, 1983) consuming fish oil, beneficial effects of fish oils on platelet function have been difficult to confirm in diabetic patients (Popp-Snijders *et al*, 1987; Glauber *et al*, 1988). In subjects with untreated hypertension, fish oils have anti-hypertensive effects (Appel *et al*, 1993). In diabetic patients on large doses of fish oil however, Landgraf-Leurs *et al* (Landgraf-Leurs *et al*, 1990) failed to demonstrate any anti-hypertensive effect. Feskens *et al*, (1993) studied fish intake in 272 normoglycaemic and glucose-intolerant elderly. Among normoglycaemic people, increased fish intake was found to be universally associated with reduced mortality from CHD. The effects of fish intake among the glucose intolerant population was smaller, suggesting that the beneficial effects of fish on coronary heart disease may be less in glucose intolerant people.

The major concern during fish oil supplementation in diabetes mellitus have been their potential deleterious effect on circulating LDL cholesterol and on glycaemic control. In IDDM, dietary supplementation of 7.5 g of fish oils per day led to increased total cholesterol and LDL cholesterol (Mori *et al*, 1989; Ascherio *et al*, 1995). Intakes of n-3 fatty acids above 3–4 g/d have been found to increase fasting plasma glucose and HbA1c in NIDDM patients (Ascherio *et al*, 1995; Schectman *et al*, 1988). Even in patients benefiting initially from the hypotriglyceridemic effect of fish oils, the efficacy of long term treatment is in doubt (Vessby *et al*, 1992). Indeed, a 6 y follow-up study of over 40 000 men was unable to observe any association between dietary intake of fish oil or fish intake and the risk of heart disease (Schectmann *et al*, 1989). Possible deleterious effects of fish oils on arterial compliance in diabetic patients as noted by Bonnema *et al* (1995) may further negate any beneficial changes seen in plasma lipids. While supplementation with fish oils is not to be generally recommended in diabetes as the evidence is conflicting on effects of fish oils on LDL cholesterol and glycaemic control, moderate intakes of oily fish in the diet of people with diabetes can be encouraged to reduce saturated fat intake.

Monounsaturated fat and carbohydrate proportions

Although low total fat intakes of well under 30% of energy have some documented health benefits, the constraints of prevailing European diet make it difficult for people to achieve fat intakes of <30% (Milne *et al*, 1994; Toeller *et al*, 1996). *Cis*-monounsaturated fatty acids (MUFA) diet contents of 10–18% have been tried as dietary enrichment for non diabetic patients following the American Heart Association Step 1 diet with significant resultant falls in plasma total cholesterol and no change in plasma triglycerides or HDL cholesterol (Ginsbert *et al*, 1990), while another study in NIDDM demonstrated improved levels of plasma triglycerides and HDL (Garg *et al*, 1994). Peripheral insulin sensitivity has also been reported to improve on a high MUFA diet (Parillo *et al*, 1992) and high monounsaturated fat diets may improve glycaemic control (Perrotti *et al*, 1984; Garg *et al*, 1988). Increased MUFA intakes have been associated with lowering of daytime blood pressure (Rasmussen *et al*, 1993) and lower the level of von Willebrand factor (Rasmussen *et al*, 1994; Thomsen *et al*, 1994), an endothelially synthesized plasma protein which reflects the presence of vascular damage possibly involved in hypertension and atherosclerosis. Dietary intake of MUFA may be an important factor in the proposed LDL-oxidation atherosclerosis hypothesis (Berry *et al*, 1992; Lorgeril *et al*, 1994) as MUFAs are more resistant to lipid peroxidation. A high MUFA diet is traditionally eaten in the Mediterranean regions, where much olive oil is used and where CHD is less prevalent (Keys *et al*, 1986). The major advantage of a higher MUFA diet is palatability, potentially aiding in compliance which has often been problematic with low fat diets.

For a patient with diabetes mellitus therefore, most dietary energy should be derived from a combination of *cis*-MUFA and high soluble-fibre carbohydrate. The actual proportion of *cis*-MUFA and high soluble fibre carbohydrate can depend on local preferences and the individual patient. Whether increased fat intake in the form of MUFA will inevitably lead to weight gain needs to be studied. Short term studies have shown no weight change on *iso*-caloric high MUFA diets (Garg *et al*, 1994).

Carbohydrate, dietary fibre and glycaemic index

Hypertriglyceridaemia, with associated low serum HDL cholesterol, is associated with increased mortality from heart disease in diabetics. A diet high in fibre-depleted carbohydrate can elevate serum triglycerides, particularly when accompanied by high fructose or sucrose intake and when islet cell function is poor. However, if the high carbohydrate intake includes soluble fibre then this effect is attenuated or abolished (Riccarrdi *et al*, 1984). Carbohydrate intake >50% is compatible with good diabetic control provided that the carbohydrate comes largely from complex sources and is associated with high intake of soluble fibre and resistant starch (Howard *et al*, 1991). The failure of some researchers to confirm the clinical benefits of a high carbohydrate diet in diabetes research has been partly due to a failure to take into account the fibre content of carbohydrate and the fact that not all dietary fibres are equally effective, and exaggerated claims for high carbohydrate diets may have resulted from studying relatively mildly diabetic subjects.

On the other hand, it is not necessary to consume more than 50% energy from carbohydrate to achieve excellent diabetic control. Previous dietary recommendations have tended to give rather precise, narrow prescriptions for fat and CHO. It has increasingly been recognised that if saturated fat is restricted, and PUFA limited for practical reasons, then there is a fairly wide range of dietary CHO and MUFA compatible with good diabetic control. Increasing dietary carbohydrate without a parallel increase in soluble dietary fibre gives no overall benefit and may be detrimental to serum lipid profiles.

Dietary fibre

Dietary fibre or non-starch polysaccharides may be classified into two broad classes: soluble fibre including gums, gels and pectins, and insoluble fibre, for example, cellulose and lignin. Both escape digestion in the small bowel, and provide an energy source for colonic bacteria. Epidemiological evidence suggests an association between low cereal fibre intakes and risk of NIDDM (Salmeron *et al*, 1997) but in experimental work insoluble, cereal-derived types of dietary fibre have minimal effects on plasma lipids or glucose. Soluble fibre, however, benefits both glycaemic and lipid metabolism. Soluble fibre appears to reduce fasting or basal glucose rather than simply acting to reduce the rate of post prandial glucose absorption (Aro *et al*, 1981). Improved insulin sensitivity is postulated to be the mechanisms by which soluble fibres improves hyperglycaemia. Insulin clamp studies are however conflicting (Ebeling *et al*, 1988; Fukagawa *et al*, 1990). Other mechanisms of possible action by which soluble fibre may modulate glycaemia include modifying the actions of gut hormones or the action of the metabolites produced from its fermentation in the colon to produce gas and short chain fatty acids, mainly acetate, butyrate and propionate (Akanji *et al*, 1989). Propionate can reduce cholesterol formation in *in vitro* and animal model experiments (Chen *et al*, 1986). However, in human feeding experiments, propionate has not demonstrated a clear effect in reducing LDL cholesterol (Venter *et al*, 1990; Todesco *et al*, 1991). Acetate, has been shown by Wolever *et al* (1988) to increase serum cholesterol when given by rectal infusion to achieve high portal concentrations.

Regular supplementation of meals with guar (Wolever *et al*, 1988), pectin, and other soluble fibres improve glycaemic control and in addition lower cholesterol. This should be considered as pharmacological therapy. It seems clear that the benefits of dietary fibre are most marked when soluble fibre is included with, or incorporated in, foods, rather than taken as an isolated supplement.

Glycaemic index

The glycaemic index (GI) was proposed by Jenkins and coworkers in 1981 as a method to guide food selection by assessing and classifying the glycaemic responses of the amount containing a standard carbohydrate load (for example 25 g) of foods, in relation to the glycaemic effect of the same amount of bread (Jenkins *et al*, 1981). The use of low GI staple foods to form the basic and major component of each meal offers benefits with lipid reduction (Wolever *et al*, 1992) and improvements in glycaemic control (Frost *et al*, 1994; Fontvieille *et al*, 1992; Brand *et al*, 1991). The GI of a food is affected by the rate of digestion and absorption of the carbohydrate present in the food (Hermansen, 1994). This is influenced in turn by factors such as amount of food eaten, presence of fat, protein, type of starch present, manner of processing and cooking (Hermansen, 1994). Provided that acceptability of a food should not be determined on the basis of having a low GI alone (many high fat foods have low GI and some foods with high GI such as bread, and potatoes are desirable for other reasons), it seems reasonable to use GI as a means of ranking starchy foods with equivalent uses to assist the dietitian in determining the likely effect if consumed (Ha *et al*, 1992; Bantle, 1989). The last decade has seen a vast number of publications on the glycaemic responses of different foods with different methods of preparation in different contexts and combinations, which is of some value to dietitians when dealing with different ethnic groups and diet.

Sweeteners

Non-alcoholic beverages sweetened by non-nutritive sweeteners may be used for people with diabetes. There are no known grounds for encouraging specially formulated 'diabetic' or 'dietetic' foods.

Sucrose

It has been long assumed that sucrose consumption by itself, or as part of a meal, aggravates hyperglycaemia, but many studies have recently shown that sucrose does not result in a greater rise in plasma glucose than *iso*-caloric amounts of other carbohydrates (Slama *et al*, 1984; Mann *et al*, 1971). However, sucrose, probably the fructose component, given in a large oral dose of 1–1.5 g/kg may inhibit the clearance of triglycerides after a high fat meal and contribute to abnormal lipid profiles (Mann *et al*, 1971). In addition, simple sugar intake of greater than 50 g per day may be associated with low nutrient density of the overall diet (Gerrits *et al*, 1993).

There are no healthful reasons for eating sucrose, apart from treating hypoglycaemia, so it seems prudent for people with diabetes to follow recommendations which exist for the general population in many countries to consume less than 10% total energy as sucrose. Sucrose therefore should be considered as part of the carbohydrate intake when planning meals provided that the energy content is taken into account and that it does not replace foods high in fibre. The use of sucrose is not recommended in those who are overweight and/or hypertriglyceridaemic.

Fructose

Dietary fructose in comparison with other carbohydrates, specifically sucrose, elicits a lower glucose and insulin response in healthy individuals and in individuals with diabetes mellitus (Anderson *et al*, 1989). Fructose added to a high fibre–high carbohydrate and low fat diet in patients with NIDDM led to improved fasting glucose levels but patients gained weight (Crapo *et al*, 1986). Chronic ingestion has been found to reduce plasma glucose and reduce glycosuria (Mann *et al*, 1971; Hollenbeck, 1993).

Dietary fructose in amounts comparable to those of sugars consumed in Western diets (7.5–20% daily dietary energy) can lead to increased fasting triglycerides and LDL concentration (Jenkins *et al*, 1982; Born *et al*, 1987). There may be gastrointestinal disturbance with large doses (Dills, 1993) and concerns about the possible role of dietary fructose on cataractogenesis and neuropathy exist (Grigoresco *et al*, 1988). On the available data however, there is no concern about diabetic people consuming foods that contain fructose naturally, for example, fruits (Woraich *et al*, 1994), but fructose does not offer proven advantages over sucrose as a sweetener. Fructose requires special techniques and recipes for cooking.

Nutritive sweeteners

These include sugar alcohols (for example, sorbitol and xylitol), honey, maltose as well as fructose, glucose and sucrose. All nutritive sweeteners contain calories and therefore must be accounted for in meal planning. Sugar alcohols like sorbitol and xylitol have a lower glycaemic response than sucrose and have a slightly lower caloric

value than sucrose as they are not completely digested and absorbed. Osmotic diarrhoea may be a problem with large amounts of sugar alcohols of 30–49 g. For people with diabetes there is probably little to gain from using nutritive sweeteners compared to sucrose. A major advantage of alcohol sugars is their low cariogenic effect in normal subjects. There is no reason to believe that this does not apply to diabetic people.

Non-nutritive sweeteners

These include saccharin, cyclamate, aspartame, alitame and sucralose. They have a useful role in some foods especially beverages, and are potentially useful in the meal planning for the overweight. The safety of these agents have been questioned, for example, saccharin as a risk factor for bladder cancer, and aspartame in precipitating seizures, producing hyperactivity and other behavioural problems in children (Walton *et al*, 1993) and altering mood in susceptible patients (Renwick, 1993). Well designed studies have not supported any of these claims (Tollefson *et al*, 1992; Toeller *et al*, 1997).

Protein and renal disease

Protein intake should range between 10 and 20% total energy. For those with incipient (abnormal microalbuminuria) or established nephropathy, protein intake should be at the lower end of the range (between 0.7 and 0.9 g/kg b.w./d).

The lower end of the safe range of daily protein intake for adults is 0.8 g/kg body weight/day (WHO, 1985) and most European populations eat considerably more than this. Individuals with diabetes mellitus often consume higher amounts of dietary protein than the general population, around 1.5 g/kg/d, or 10–20% of dietary energy, as both sugar restricted and fat restricted diets tend to increase the proportion of energy supplied as protein.

There have been concerns that diets rich in protein contribute to the pathogenesis of early diabetic nephropathy supported by epidemiological evidence (Kupin *et al*, 1987). Protein ingestion acutely raises GFR and changes in dietary content of protein alter GFR in both normal and diabetic patients (Kalk *et al*, 1992). In IDDM and NIDDM populations, Kalk *et al* (1992) and Jameel *et al* (1992) found that protein intake was similar in those with and without clinical proteinuria. However, in patients with evidence of preclinical diabetic nephropathy, dietary changes including protein restriction to <0.6 g/kg/d can reduce the elevated GFR and albuminuria (Viberti, 1988) independent of any effect of improved blood glucose control or blood pressure. In patients with persistent proteinuria, the use of low protein diets (0.6 g/kg/d) may modify the progression of nephropathy (Freidman, 1982; Zeller, 1991). These studies suggest that protein intakes close to the RDA may have advantages in people with diabetes (Holm *et al*, 1996).

A diet inadequate in protein (that is <0.6 g/kg/d) can lead in adults to loss of lean body mass, even though adequate calories are given (Barac-Nieto *et al*, 1979; Miller *et al*, 1966). Protein metabolism may be altered early in diabetes mellitus and obligatory minimum nitrogen loss is greater in diabetic patients than in control subjects, even under tight insulin control (Lariere *et al*, 1992; Gougeon *et al*, 1994). Non-diabetic people adapt to low protein intake with time, but in people with IDDM and early nephropathy, there appeared to be no adaptation to a low protein intake over a twelve week study period, and protein undernutrition occurred on a protein intake of 0.6 g/kg body weight/d (Brodsky *et al*, 1992).

Evidence exists that proteins from differing sources may have unique properties and different effects on the progression of renal disease, with benefits claimed for vegetable proteins (Quinn, 1993; Kontessis *et al*, 1990). It is still not clear whether the protein quality or some other related factor, such as dietary fibre or reduced intake of saturated fat, may be responsible for such effects. Riley and Dwyer found association between microalbuminuria and saturated fat intake, but an inverse relationship with protein consumption, as indicated from a food frequency questionnaire (Riley *et al*, 1998). Quinn (1993) recently recommended the use of essential amino acid supplements for patients following a very strict, low protein diet: evidence to support this advice is lacking.

Recommendations about protein intake for people with diabetes are based on incomplete evidence, and there is a lack of long term studies with clinical end points. Long term studies are required on diabetic subjects with different stages of renal disease.

Alcohol (ethanol)

Precautions regarding alcohol intake which apply to the general public, also apply to people with diabetes. For those who choose to drink alcohol, an amount equivalent to one or two glasses of wine per day is acceptable. When alcohol is taken by those on insulin or sulphonylureas, it should be consumed with carbohydrate-containing foods because of the risk of potentially profound and prolonged hypoglycaemia.

Alcohol effects of special note include danger of hypoglycaemia in fasting individuals primarily due to suppression of gluconeogenesis (Lieber, 1994). Sulphonylurea usage compounds the danger in those with NIDDM. Alcohol can also interfere with glucagon action and delay recovery in cases of insulin induced hypoglycaemia (Arky *et al*, 1968). Daily alcohol intake has also been associated with an increased risk of cataract independent of the effects of diabetes mellitus (Manson *et al*, 1994). Alcohol contributes 7 kcal/kg, so alcohol intake may impair glycaemic and weight control and aggravate hypertriglyceridaemia.

Recent studies have looked at the use of alcohol in NIDDM (Christiansen *et al*, 1993; Christiansen *et al*, 1993; Koivisto *et al*, 1993), and results suggest that patients with well-controlled diabetes are able to consume 21–28 g of alcohol with carbohydrate-containing meals with no alterations in glycaemic control (Christiansen *et al*, 1993). There are theoretical beneficial effects of alcohol use on blood lipids and coagulability which needs to be further studied in people with diabetes. Moderate intake (one to two glasses of wine per day) of wine, especially red wine, which contains compounds such as flavonoids and phenolics with antioxidant capability, may confer greater benefit than consumption of spirits or beer (Gronbaek *et al*, 1995).

Vitamins and antioxidant micronutrients

Foods naturally rich in dietary antioxidants (tocopherols, carotenoids, vitamin C and possibly such as flavonoids) should be encouraged.

Highly reactive oxygen free-radicals are increasingly implicated in the pathogenesis of many important diseases including atherosclerosis and cancer, by increasing the

atherogenicity of lipoproteins by the process of peroxidation, and altering DNA bases. They have also been implicated in causing direct pancreatic β-cell damage, thereby inducing or aggravating diabetes (Oberley, 1988), and may also contribute to the late diabetic complications (Wolff, 1993; Gazis *et al*, 1997). Transition metals (for example, iron and copper) may contribute to oxidative stress by catalysing the oxidation of glucose, ascorbate and PUFA, and generate a steady supply of reactive species, for example, hydrogen peroxide and lipid peroxides. The body has several antioxidant defence systems, based on endogonous compounds such as uric acid, trace element containing enzymes, for example, glutathione peroxidase, superoxide dismutases, and specific antioxidant micronutrients, for example, beta carotene, vitamin E and vitamin C.

The body of scientific literature concerning oxidative damage in the aetiology of diabetes mellitus and its complications, the role of diet and the many possibly protective antioxidant compounds like carotenoids and flavonoids is rapidly expanding. However, most of the findings are preliminary and firm scientific evidence from which to make definitive recommendations about specific compounds are sparse, especially when one endeavors to make firm recommendations, for example, when dealing with the controversial area of vitamin supplementation.

Previous recommendations have suggested that people with diabetes should have the same intake of vitamins as the normal population. In the light of current knowledge, the best advice is to encourage the population, and especially those with diabetes mellitus to consume a diet rich in foods which naturally contain significant quantities of dietary antioxidants, especially fresh fruit and vegetables. Routine or indiscrimate pharmocological vitamin or mineral supplementation is not justified on present evidence.

Carotenoids and vitamin A

Dietary carotenoids provide about 50% of the total vitamin A intake in the United States. Plasma levels reflect current intake of dietary sources, predominantly coloured fruit and vegetables. Only certain carotenoids can act as vitamin A precursors, for example, α-carotene and β-carotene. Other carotenoids which have previously been discounted may still have important antioxidant and immunomodulatory functions (Thurnham, 1994). High dietary intakes of carotene containing foods and high plasma levels of carotenes are associated with lower rates of cardiovascular disease ((Rieversma *et al*, 1991) in non-diabetic populations.

Diabetic populations may have differing intakes of carotenes, with diabetics in urban India having been found to consume less β-carotene containing foods and have lower plasma β-carotene (Singh *et al*, 1994), while a German study reported normal levels of β-carotene (Straub *et al*, 1993). Whether these differences contribute to different CVD risk in these diabetic populations has not yet been demonstrated.

Serum retinol levels can reflect acute disease states. Zinc deficiency, infections and liver disease are among the factors that may reduce retinol concentration (Gibson, 1990). Plasma retinol and retinol-binding protein has been reported to be reduced in young people with IDDM (Souissi *et al*, 1993). Others have found normal serum retinol (Ramachandran, 1981; Straub *et al*, 1993). Animal studies have suggested that vitamin A deficiency may be important in lens cellular damage (Linkater *et al*, 1992). Epidemiological studies have identified a reduced risk of cataract associated with a high vitamin A intake (Leske *et al*, 1991) and high levels of carotenoids in plasma (Knekt *et al*, 1992; Jacques *et al*, 1988). On the basis of such preliminary findings, supplementation has been suggested as having a role in prevention of disease. Evidence of benefit is lacking, and supplementation in pharmacological doses may be dangerous (Gerrits *et al*, 1993). The increase in cancers and CHD seen with β-carotene supplementation in smokers is a warning not to embark on indiscriminate high dose vitamin supplementation (ATBC Group, 1988).

B vitamins

The water-soluble B vitamins, thiamine, pyridoxine, riboflavin, and niacin, participate as coenzymes in a variety of reactions including the catabolism of carbohydrates, fats, and proteins. The body has limited stores and these vitamins are rapidly catabolised. Poor control of diabetes mellitus has been reported to result in excessive excretion of B vitamins in the urine (Kodentsova *et al*, 1993).

Daily requirement of thiamine is dependent on the amount of carbohydrate eaten. There is conflicting evidence of altered thiamine status with reports of lowered, normal (Marks, 1978) and elevated thiamine levels (Haugen, 1964) in diabetic populations. Transketolase enzyme activity has also been reported to be reduced in diabetic individuals independent of thiamine status (Saito *et al*, 1987). There have been anecdotal reports of patients with diabetic nephropathy responding to supplementation with thiamine, but no controlled studies.

Two compounds have niacin activity: nicotinic acid and nicotinamide. Niacin is a component of two coenzymes serving in oxidation and reduction reactions. Nicotinic acid used pharmacologically in the treatment of hyperlipidaemia can result in deterioration of carbohydrate tolerance. Several uncontrolled studies have suggested that nicotinamide may protect B cell function in animals (Kjosen, 1977) and humans (Reddy *et al*, 1990), but a controlled trial of nicotinic acid has shown no benefit in inducing remission of early IDDM (Pozzilli *et al*, 1989).

Low pyridoxine and pyridoxal 5′-phosphate levels have been seen in some patients with diabetes (Lewis *et al*, 1992). Pyridoxine supplementation has not been found to be beneficial in a population with diabetes (Davis *et al*, 1977) and toxicity can occur. Riboflavin is a component of two coenzymes. Low concentrations of erythrocyte riboflavin have been reported in diabetes (Rao *et al*, 1980) and diabetes mellitus is well recognised as a condition which can precipitate or exacerbate riboflavin deficiency (Kodentsova *et al*, 1993).

Low folic acid consumption and blood levels are associated with high circulating homocysteine, a risk factor for CHD. This does not appear to be a special problem in diabetes, but provides another reason to maximise fruit and vegetable consumption.

Polyglandular syndromes can present with IDDM and pernicious anaemia. With increasing age NIDDM and pernicious anaemia are increasingly seen and may occur together (Nichoalds, 1981). Vitamin B12 has been reported to improve the somatic and autonomic symptoms of diabetic neuropathy but again controlled studies are lacking (Straub *et al*, 1993).

Ascorbic acid

Plasma levels of ascorbic acid (AA) are decreased and its primary oxidation (DHAA) product is increased in experimental diabetes (Reed *et al*, 1990; McLennan *et al*, 1988). This has been observed in several diabetic populations (Yue *et al*, 1989; Som *et al*, 1981; Stankova *et al*, 1984) and appears to be a result of altered vitamin C metabolism in diabetes rather than reduced dietary intake (Sinclair *et al*, 1995).

There is epidemiological evidence that low ascorbic acid intake and low plasma Vit C levels were associated with increased risk for mixed and cortical cataracts which may be seen in diabetic subjects (Leske *et al*, 1991). The eye contains high concentrations of ascorbic acid, with a report by Taylor *et al* (1991) that the human eye contains 60-times the concentration of ascorbic acid than found in plasma. The relationship between cataracts and ascorbic acid is still unclear with some work supporting an antioxidant role (Schorah *et al*, 1988) while others suggest ascorbic acid may participate in the oxidative modification of lens protein that is seen in ageing (Slight *et al*, 1990; Russell *et al*, 1987).

There is need for further studies of vitamin C in diabetes mellitus, especially studies designed to address the issue of supplementation. Paolisso *et al* (1995), reported improved lipids, insulin levels and indices of oxidative stress in elderly diabetics given longterm vitamin C (1 g/d) supplements.

Vitamin D and calcium

In recently diagnosed IDDM, markedly reduced vitamin D3 levels have been found. There is also loss of the normal seasonal rhythm of increased vitamin D3 levels during summer (Baumgartl *et al*, 1991). Calcium is essential in normalising glucose intolerance that accompanies vitamin D depletion (Beaulieu *et al*, 1993). Supplementation with vitamin D has been found to enhance insulin secretion in patients with mild NIDDM (Inomata *et al*, 1986), but vitamin D had no effect on insulin secretion and no effect on glucose homeostasis in a group of patients with established NIDDM. This study by Orwoll *et al* (1994) raises the possible role of inadequate vitamin D resulting in worsened insulin secretion during early NIDDM. Vitamin D and calcium replacement in a group of patients with diabetes and osteomalacia was associated with a decline in fasting blood glucose levels (Kocian, 1992).

There is evidence of increased bone turnover in type 1 diabetes mellitus (Gallacher *et al*, 1993; Wakasugi *et al*, 1993). Some studies have shown that people with IDDM have an approximate 10% decrease in bone mineral content when studied a few years after clinical onset of diabetes (Mathiassen *et al*, 1990). It is suggested that metabolic abnormalities associated with poor control and longer duration of disease may be risk factors for decreased bone mineral density (Wakasugi *et al*, 1993). Hypomagnesaemia may have a pivotal role (Mathiassen *et al*, 1990). Despite these findings, there have been no studies showing an increase in incidence of fractures (Melchior *et al*, 1994). In overweight NIDDM, decreased bone mineral content is not a feature (Mooradian *et al*, 1987).

In view of the current findings it is advisable to follow the recommendations for calcium intake as for the general population, and certainly to guard against low intakes which may occur from ill-guided efforts to reduce fat intake. There is insufficient evidence for recommending routine vitamin D supplementation, but vitamin D deficiency when present may contribute to IGT, and this should be considered in South Asian migrant groups prone to vitamin D deficiency and in the elderly or institutionalised.

Vitamin E

Vitamin E is a potent fat soluble antioxidant capable of preventing the peroxidation of vitamin A and unsaturated fatty acids. Vitamin E has been shown to reduce protein glycation in some diabetic subjects (Ceriello *et al*, 1991) but other evidence is conflicting. Recent epidemiological studies have also associated high vitamin E intake with low incidence of cataract in the non diabetic population (Jacques *et al*, 1988; Knekt *et al*, 1992; Roberston *et al*, 1989). The dosage of vitamin E associated with reduced cataract risk is yet to be defined, however multivitamin supplementation once per week is associated with protection against cataractogenesis (Leske *et al*, 1991).

Vitamin E supplementation at enormous doses of 900 mg/d of alpha-tocopherol has been found in one study to improve metabolic control in NIDDM, possibly via improved insulin action (Paolisso *et al*, 1993), but other studies have been negative. Supplementation has been associated with a reduction in the risk of coronary heart disease in the non-diabetic male population (Rimm *et al*, 1993), but whether such benefits also apply to the diabetic population remains to be seen as studies have shown tocopherol levels to be already increased in diabetes (Vatassery *et al*, 1983) and the Finnish supplementation in smokers found no effect (ATBC GROUP, 1994). No clear recommendation can be made at present.

Flavonoids

These are naturally occuring water soluble substances with antioxidant activity found widely in vegetables, fruit, tea and red wine. Higher intakes of these phenolic compounds have been associated with reduced risk of CVD in Dutch men (Hertog *et al*, 1993) and in Finns. The action by which these compounds work is still to be determined. They may act as direct antioxidants or may act to modulate antioxidant enzymes. Synthetic flavonoids can reduce protein glycation (Vertommen *et al*, 1994).

Capillary abnormalities play a role in the development of diabetic microangiopathy. Mixtures of semisynthetic flavonoids, which may reduce capillary hyperpermeability, have been shown to reduce retinal vascular permeability in patients with diabetic retinopathy, but did not affect retinal haemorrhage (Engelen *et al*, 1989).

Minerals

Salt

In a trial of diet in hyperglycaemic obese patients, goals for weight reduction were more easily achieved than the goal for low sodium, high potassium and phosphate modification (Wadworth *et al*, 1992). The general population has been recommended to restrict the intake of sodium to less than 6 g salt daily. This would entail halving of the usual North European intake and would be associated at a population level with a reduction in heart disease and stroke (Marmot, 1982). Salt reduction is difficult in many European countries where salt is added during the processing of large

numbers of important foods, including staples such as breads and cereals, only about 20% of salt intake is added in the kitchen or at table.

Dietary sodium may have a role in the development of insulin resistance (Draznin, 1993). However concerns about dietary sodium arise more with respect to its effects on blood pressure. Moderate dietary sodium restriction is reported to reduce systolic blood pressure by 20 mmHg (Dodson *et al*, 1989) in a mildly hypertensive population with NIDDM. Consuming less than 1 g per day of salt resulted in 8 out of 12 individuals with diabetes and hypertension showing a sodium sensitive BP response (Tuck *et al*, 1990). Such severe restriction is extremely difficult to achieve, and its less than universal effect makes such restriction useful only for individuals who are motivated, and are clearly 'salt responders' (ADA, 1993). The interaction with weight loss in treating overweight and hypertension has made it difficult to separate the therapeutic benefits of salt restriction from energy restriction.

Magnesium

Diabetes mellitus is one of the chronic diseases most frequent associated with magnesium deficiency (Walter *et al*, 1991). Magnesium occurs widely in foods and dietary deficiency *per se* does not occur without starvation, a specific renal tubular defect may exist in diabetes mellitus which may act together with osmotic diuresis to result in large magmesium urinary losses (Garland, 1992; Ponder *et al*, 1990). Dietary induced magnesium deficiency has been shown to increase thromboxane synthesis and reduce insulin sensitivity (Nadler *et al*, 1993). Oral magnesium hydroxide replacement in patients with documented magnesium deficiency can reduce insulin requirements in patients with IDDM (Sjogren *et al*, 1988). Magnesium deficiency impairs parathyroid hormone secretion to reduce calcium levels, and reduce serum 1.25 vitamin D with possible detrimental effects on bone in diabetes mellitus (Fatemi *et al*, 1991). Magnesium deficiency can also lead to increased platelet aggregation (Hwang *et al*, 1993). It has also been linked epidemiologically with increased risk for diabetic retinopathy (McNair *et al*, 1978) and ischaemic heart disease (Seelig *et al*, 1974). Choice of oral hypoglycaemic agents may affect magnesium handling: biguanide usage is linked with hypomagnesaemia, whereas glipizide raises serum magnesium (McBain *et al*, 1988). Thiazide diuretics increase magnesium losses.

Studies demonstrating benefit from long-term magnesium supplementation are lacking. The use of magnesium supplementation should be considered in situations where its deficit may have a detrimental effect, for example, acute myocardial infarction and arrythmias, patients on diuretics, diabetic ketoacidosis (ADA, 1992).

Zinc

Patients with diabetes mellitus have altered zinc handling with low serum zinc and increased urinary zinc excretion (Walter *et al*, 1991). Serum zinc levels need to be kept constant to allow normal insulin secretion: very high or low levels may impair insulin secretion (Schroeder, 1974). Supplementation of zinc to diabetic patients has been advocated, but Raz *et al* (1989) found that zinc supplementation of 660 mg/d in deficient NIDDM can aggravate glucose intolerance. Studies are needed to investigate the role of zinc in the treatment of diabetic ulcers, as zinc supplementation has been shown to improve healing of leg ulcers (Hallbook *et al*, 1972). There are no good tests for zinc deficiency.

Copper

High plasma copper concentrations are found in people with diabetes mellitus (Walter *et al*, 1991) and are associated with increased risk of atherosclerosis (Schroeder, 1974). Mantarri *et al* (1994) also found a continuous and graded increment in coronary risk with rising caeruloplasmin (the storage form of copper) levels in dyslipidaemic men, and postulated the possible role of high copper levels as a promoter of lipid peroxidation. Others have found no evidence of altered copper status in people with diabetes (Rohn *et al*, 1993).

Chromium

'Glucose tolerance factor' was described as a chromium-containing compound which appears to be necessary for glucose transport, possibly by enhancing insulin binding to its receptors (Mertz *et al*, 1974). Dietary deficiency of chromium is associated with elevated blood glucose in animals (Schroeder, 1966), and in patients on prolonged TPN (Freund *et al*, 1979), and may possibly cause disturbances in lipid metabolism. Marginal deficiency states have been described in malnourished children, diabetics, and the elderly.

Studies have been performed to study the effects of chromium supplementation in populations with diabetes mellitus. No improvement in blood glucose control was seen (Rabinowitz *et al*, 1983; Uusitupa *et al*, 1983) in the majority of cases. Glucose intolerance from chromium deficiency is currently perceived unlikely to be a major issue in the pathogenesis of diabetes mellitus but again, lack of sensitive tests accurately to assess chromium status limits progress.

Health promotion and families of people with diabetes

To improve compliance, general aspects of dietary advice should be acceptable and applicable with potential benefit to the whole family. Given the strong genetic influence on responses to lifestyle factors as components of NIDDM, there are sufficient reasons to urge the children of people with NIDDM to avoid becoming overweight, to take regular exercise and to follow carefully the dietary recommendations for health in the general population.

The trend in most European populations is towards more prevalent obesity, and with it more prevalent NIDDM. Strategies for primary prevention of obesity are not yet established, but targeting of family members of people with NIDDM would be expected to increase the health benefits of weight management programmes for Health Promotion. Offspring of NIDDM should be identified from primary care records, and at opportunistic screening, for priority recruitment into local weight management programmes. Women found to have gestational diabetes have a very high risk of diabetes and could also be specifically targetted.

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