Nutrition Principles for the Management of Diabetes and Related Complications

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Health professionals and people with diabetes recognize nutrition therapy as one of the most challenging aspects of diabetes care and education (1). Adherence to meal planning principles requires the person with diabetes to learn specific nutrition recommendations. It may require altering previous patterns of eating and implementing new eating behaviors, which requires motivation for a healthy lifestyle and may also require participation in exercise programs. Finally, individuals must be able to evaluate the effectiveness of these lifestyle changes. Despite these challenges, nutrition is an essential component of successful diabetes management. Effective nutrition self-management training requires an individualized approach that appropriate for the personal lifestyle and diabetes management goals of the individual with diabetes.

Achieving nutrition goals requires a coordinated team consisting of a registered dietitian, physician, nurse clinician/educator, and the person with diabetes. Access to behavioral and exercise specialists is also important. The Diabetes Control and Complications Trial reaffirmed the importance of health-care teams for successful diabetes management (2).

To ensure that nutrition goals are being met, monitoring of blood glucose, glycated hemoglobin, lipids, blood pressure, and weight is essential. If goals are not being met, changes should be made in the diabetes management plan.

This technical review discusses research related to nutrition and diabetes. Recommendations based on this review are found in an American Diabetes Association position statement (3). However, not all nutrition recommendations can be based on relevant published and definitive data. Data may be conflicting or not available. When this is the case, limitations are acknowledged and recommendations are based on clinical experiences and consensus. Additional research is needed to answer the many remaining questions.

GOALS OF MEDICAL NUTRITION THERAPY — The primary desired outcome of nutrition self-management training is to assist persons with diabetes in making changes in nutrition and exercise habits that will improve nutrition skills, status, and diabetes self-management. Optimal nutrition can lead to reduction of risk factors for chronic health problems and improved overall health.

Goals of medical nutrition therapy for persons with diabetes include the following:

1. Maintenance of near-normal blood glucose levels by balancing food intake with insulin (either exogenous or endogenous) or oral glucose-lowering medications and activity levels.
2. Achievement of optimal lipid levels (cholesterol, low-density lipoprotein [LDL cholesterol], triglycerides, very-low-density lipoproteins [VLDL cholesterol], and high-density lipoproteins [HDL cholesterol]).

The guidelines provided by the Adult Treatment Panel II (4) and The Expert Panel on Blood Cholesterol Levels in Children and Adolescents (5) of the National Cholesterol Education Program for classifications of desirable or acceptable, borderline-high, and high cholesterol levels also apply to persons with diabetes mellitus. Diabetes is a major risk factor for morbidity and mortality because of coronary heart disease, cerebrovascular disease, and peripheral vascular disease (6,7). Nutrition plays a key role in achieving recommended lipid levels.

3. Provision of adequate calories for maintaining or attaining reasonable weights for adults, for normal growth and development rates for children and adolescents, to meet increased metabolic needs during pregnancy and lactation or for recovery from catabolic illnesses.

Reasonable body weight is defined as that level of weight individuals and health care providers acknowledge as achievable and maintainable both short- and long-term. For some individuals this

LDL, low-density lipoprotein; VLDL, very-low-density lipoprotein; HDL, high-density lipoproteins; RDA, recommended dietary allowance; CVD, cardiovascular disease; IDL, intermediate-density lipoprotein; FDA, Food and Drug Administration; ADI, acceptable daily intake; BMI, body mass index; VLCD, very-low-calorie diet; RMR, resting metabolic rate; FFM, fat free mass; LPL, lipoprotein lipase; GDM, gestational diabetes mellitus; GFR, glomerular filtration rate.

may be their current weight, even if this weight is associated with excess body fat. For individuals who are overweight, it may preferably be a weight 5–10 kg (10–20 lbs) less than current weight. This is in contrast to desirable body weight (DBW), which is an ideal body weight for an individual based on height and frame size.

For obese persons with type II diabetes mellitus, moderate weight loss irrespective of starting weight has been shown to reduce hyperglycemia, dyslipidemia, and hypertension (8).

Weight management for people using exogenous insulin is also an important issue (9,10). Potential for weight gain can be minimized by focusing on food choices, portion control, and appropriate treatment of hypoglycemia (11).

Calories should be prescribed to provide for normal growth and development in children and adolescents. The meal plan is not a restriction of calories but is intended to ensure a reasonably consistent food intake and a nutritionally adequate diet. With appropriate caloric intake and metabolic control, children and adolescents with diabetes will grow and mature at normal rates (12).

During pregnancy and lactation women generally require increased calories. Monitoring blood glucose levels, urine ketones, appetite, and weight gain enables appropriate adjustments in calorie intake.

4. Prevention, delay, or treatment of nutrition-related risk factors and complications. Nutrition assessment and intervention are essential for risk factor reduction from obesity, dyslipidemias, and hypertension.

Medical nutrition therapy plays a role in the prevention and treatment of the acute complications of insulin-treated diabetes mellitus such as hypoglycemia, short-term illnesses, and exercise-related blood glucose problems. Nutrition therapy is also important in the treatment of long-term complications of diabetes mellitus such as renal disease, autonomic neuropathy, hypertension, and cardiovascular disease.

5. Improvement of overall health through optimal nutrition. *Nutrition and Your Health: Dietary Guidelines for Americans* (13) and *The Food Guide Pyramid* (14) summarize and illustrate nutritional guidelines and nutrient needs for all healthy Americans. They also serve as excellent guidelines for people with diabetes mellitus and their family members.

Family members and other support system individuals need to be an integral part of the education program and are encouraged to follow the same lifestyle recommendations as the person with diabetes. Sensitivity to ethnic and cultural food issues, as well as financial considerations, is essential for successful nutrition care.

Nutrition and Insulin Therapy

A meal plan based on the individual's usual food intake should be determined and insulin therapy integrated into the usual eating and exercise patterns. Day-to-day consistency in the timing and amount of food is important for people receiving conventional insulin therapy (one to two daily insulin injections). It is not desirable to divide meals and snacks into any artificial or unnatural pattern.

Individuals on insulin therapy need to eat at consistent times synchronized with the time-actions of insulin, monitor blood glucose levels, and adjust insulin doses for the amount of food usually eaten or required.

Intensive insulin therapy (multiple daily insulin injections or insulin infusion pumps) allows considerable flexibility in food and exercise regimens. People can be taught to adjust premeal insulin doses to compensate for departures from their meal plan, to delay premeal insulin for meals that are late, and to administer insulin for snacks that are not part of their meal plan. Insulin can also be adjusted for changes in physical activities.

Nutrition Therapy and Type II Diabetes

Although weight loss has been the primary focus for people with type II diabetes, current dietary strategies are often not effective in achieving long-term weight loss (15–18). Therefore, the emphasis for medical nutrition therapy should be placed on achieving glucose, lipid, and blood pressure goals. Although weight loss is desirable, several additional strategies can be employed to facilitate improved metabolic control (19). To determine individual success, monitoring of clinical outcomes is essential.

Making healthy food choices, especially reducing caloric intake, can be beneficial. Further, reduction of dietary fat may be associated with a reduction in energy intake and weight loss (20–24). A nutritionally adequate meal plan with a reduction in fat, especially saturated fats, should be implemented.

In some individuals, strategies such as spacing of meals (spreading nutrient intake throughout the day) instead of consuming only three meals, can be beneficial (25–27). Regular exercise facilitates weight management and metabolic control. Learning new behaviors and attitudes can promote long-term lifestyle changes (28–30).

A moderate caloric restriction (250–500 kcal less than the average daily intake) and an increase in physical activity may lead to improved diabetes and weight control. Mild to moderate weight loss has been shown to improve metabolic control even if desirable body weight is not achieved (19,31,32). Weight loss appears to increase insulin sensitivity and normalize hepatic glucose production (33).

Monitoring blood glucose, glycated hemoglobin, lipids, and blood pressure is essential to evaluate nutrition-related strategies. If metabolic parameters do not improve, oral glucose-lowering

medications, insulin, lipid lowering or antihypertensive drugs may be required.

PROTEIN — Adequate protein intake is required to ensure normal growth, development, and maintenance of body protein stores (34). In diabetes, variations in dietary protein may influence metabolic control by altering gluconeogenic substrate availability as well as insulin and counterregulatory hormone secretion (35). When protein is ingested with glucose, a synergistic effect on insulin secretion and a lower glycemic response than to glucose alone occurs in some individuals with type II diabetes mellitus (36).

Excessive protein intake has also been implicated in the pathogenesis of diabetic renal disease and restricting its intake may retard the progression of nephropathy (37). Nevertheless, there is limited scientific data upon which to establish firm nutrition recommendations for protein intake in diabetes (38).

Recent surveys indicate that the average protein intake in the U.S. for all age groups varies from 14 to 18% of the total daily caloric intake; ~65% of this protein is of animal origin (34,39). Individuals with diabetes mellitus also tend to have protein intakes in this range (40). For adults of both sexes, the current recommended dietary allowance (RDA) for protein is 0.8 g · kg body wt^{-1} · day^{-1}. This is ~10% of the total daily caloric intake (34). There is no evidence that individuals with diabetes have protein requirements greater than nondiabetic subjects. In general, individuals in the U.S. with and without diabetes consume more protein than necessary to meet known nutritional needs.

For people with diabetes mellitus, 10–20% of the total daily calories should come from protein. At the present time, there is insufficient scientific evidence to support either higher or lower protein intakes for people with diabetes than for the general population. Protein should be incorporated into the diabetic diet using a variety of foods and considering the content of other nutrients and the overall influence of diet in the management of diabetes (41,42).

Special Considerations for Protein

Obesity. Hypocaloric weight-reducing diets are frequently used in the treatment of diabetes with obesity and may result in accelerated protein breakdown. Increased protein intake is required to preserve lean body mass during periods of severe calorie restriction (43).

Exercise and sports nutrition. Protein requirements are determined by rates of protein turnover, which is an energy-dependent process. The enhanced energy expenditure resulting from exercise increases the need for additional protein, which should be met by increased consumption of a nutritionally balanced diet. A small amount of additional protein may be required for muscle growth resulting from chronic physical conditioning. This need is easily met by usual food intake (44).

Nephropathy. Diabetic nephropathy is the single most common cause of end-stage renal disease. High protein intake increases renal workload and glomerular filtration rate (GFR) and has been implicated in the pathogenesis of diabetic nephropathy (37). The Modification of Diet in Renal Disease Study (MDRD) did not show any significant effect of lowering protein intake on altering the rate of decline of the GFR in nondiabetic subjects with varying degrees of renal dysfunction (45). However, several studies in people with diabetes indicate that use of low protein diets in people with diabetes may modify the underlying glomerular injury and delay progression of nephropathy (46,47). Evidence also suggests that amino acids from meat proteins differ from vegetable proteins (48,49) and egg white, cheese, or cooked soybean protein (50). It may be that meat proteins have unique qualities that adversely influence GFR and the progression of renal disease. The stage at which diabetic nephropathy would respond to protein restriction remains to be determined. Most data supports some restriction or limitation of protein intake to prevent or delay development of diabetic renal disease, however, more research is necessary to determine the optimal protein content of the diet for individuals with diabetes. At the present time, a protein intake approximating the adult RDA of 0.8 g · kg body wt^{-1} · day^{-1} (~10% of daily calories) is sufficiently restricted and recommended for individuals with evidence of nephropathy. With protein intake of 0.6 g · kg body wt^{-1} · day^{-1} evidence of protein undernutrition has been reported (51).

Pregnancy, children and adolescents, and the elderly. There are a number of conditions in which protein requirements may vary from the adult RDA. In these situations the optimal protein intake for individuals with diabetes has not been specifically determined and thus the RDA for individuals without diabetes should be used. Protein per kilogram of body weight requirement is higher in infants, children and adolescents to support growth and development. The requirement progressively decreases toward adulthood with protein intakes ranging from 12 to 20% (0.9–2.2 g · kg body wt^{-1} · day^{-1}) of total daily caloric intake.

Protein requirements are also increased throughout pregnancy, and an additional daily allowance of 10 g of high quality proteins should be provided throughout pregnancy (52). An additional 15 g of protein/day should be provided during the first 6 months of lactation and 12 g daily thereafter (53).

In otherwise healthy elderly individuals with diabetes, protein requirements are assumed to be comparable to those of younger adults (0.8 g · kg body wt^{-1} · day^{-1}). Higher protein intakes are recommended to maintain nitrogen balance in individuals, which may be more common in the elderly, with concomitant chronic illness or acute catabolic stress (54).

FAT/CARBOHYDRATE — If dietary protein contributes 10–20% of the total caloric content of the diet, then 80–90% of calories remain to be distributed between dietary carbohydrate and fat. National food consumption surveys of the U.S. population conducted by the U.S. Department of Agriculture (USDA) reveal that ~43–47% of calories are contributed by dietary carbohydrate, whereas 36–37% of calories are contributed by dietary fat, with 13% from saturated fatty acids, 14% from monounsaturated, and 7% from polyunsaturated. Total dietary cholesterol is estimated at 180–187 mg/1,000 kcal (55,56).

A reduction in high dietary intakes of cholesterol-raising fatty acids, such as saturated fats, trans fatty acids, and cholesterol, is an important goal to reduce the risk of cardiovascular disease (CVD). The recommended distribution of dietary carbohydrate and unsaturated fat (monounsaturated and polyunsaturated) calories in the diets of people with diabetes is controversial. The nature of the controversy results from the potential contribution of both dietary carbohydrate and fat to alterations of glucose and lipid metabolism, body fat, and the acute and chronic complications associated with diabetes mellitus.

Although diabetes mellitus is usually categorized as a disease of carbohydrate metabolism, abnormalities of lipoprotein metabolism and adipose tissue distribution are also common. CVD accounts for the majority of deaths in people with diabetes (57). Analysis of the Multiple Risk Factor Intervention Trial data for men with diabetes matched with nondiabetic men reported the relative risk of death for men with diabetes was increased at a range from 2.83 to 4.46 depending on their level of serum cholesterol (58). These findings confirm that diabetes is a strong independent risk factor for CVD over and above the adverse effects of an elevated serum cholesterol.

The general recommendation for the U.S. population is to decrease total dietary fat to 30% or less of total calories, with emphasis on reduction in saturated fat, and to increase dietary carbohydrate, to decrease the incidence of CVD. When dietary saturated fats are decreased, polyunsaturated can be increased from current intake of 7% of calories to 10% of calories (4). Because diabetes is associated with a three- to fourfold increase in risk for CVD, nutrition recommendations for patients with diabetes have traditionally followed the same guidelines (59). Controversy arises, however, over whether saturated fat calories should be replaced as carbohydrate, unsaturated fat, or both.

Lipid Metabolism and Type I Diabetes

Uncontrolled type I diabetes is associated with elevated plasma lipids, but adequate insulin therapy usually restores lipid levels to normal. People with type I diabetes who are treated with insulin generally have plasma cholesterol, VLDL cholesterol, LDL cholesterol, and triglyceride concentrations similar to those of the general population of the same age and sex, but have higher than normal HDL cholesterol levels (60,61). Although not all studies agree, it appears that blood glucose control may directly influence the levels of several plasma lipid components (62). Finally, qualitative abnormalities such as size and density of lipoprotein composition may exist even when the usual clinical measurements of plasma lipids are normal.

Lipid Metabolism and Type II Diabetes

Compared with age- and sex-matched nondiabetic individuals, people with type II diabetes have a two- to threefold increase in the prevalence of dyslipidemia (63). Abnormalities of lipid metabolism have been identified in people with type II diabetes at diagnosis and later under reasonable control.

The most frequent lipid abnormalities in patients with type II diabetes mellitus are hypertriglyceridemia, increased VLDL cholesterol, and reduced HDL cholesterol. Although plasma levels of total and LDL cholesterol concentrations are similar to the general population, as many as 40% of the population with type II diabetes may have high-risk levels of LDL cholesterol that should not be neglected (63). The hypertriglyceridemia of type II diabetes is thought to be due, in part, to increased hepatic production of triglyceride-rich VLDL (64) and may also be associated with increased circulating levels of small VLDL and of intermediate-density lipoprotein (IDL) particles. Accumulation in the plasma of small, dense LDL particles in people with type II diabetes mellitus has also been reported (65). The presence of this subclass, referred to as LDL phenotype B, appears to follow familial distribution, be increased approximately twofold in individuals with type II diabetes mellitus, and may be associated with increased risk of CVD (64). Both type II diabetes and the LDL B phenotype are genetically determined and it is possible that they are linked. Alterations in the metabolism of these lipids may be related to the hyperinsulinemia, insulin resistance and intra-abdominal fat accumulation usually present in people with type II diabetes mellitus (66–68).

Whether hypertriglyceridemia itself leads to the formation of small, dense LDL or whether those who inherit the LDL B phenotype are more susceptible to the development of hypertriglyceridemia is unresolved. Studies suggest that both LDL composition and LDL particle number are important with respect to coronary atherosclerosis in populations where hypercholesterolemia is not the dominating lipoprotein disorder (69).

Studies of CVD risk factors conducted over the last 30 years have remained controversial with regard to the significance of elevated plasma triglyceride concentration. This is in contrast to the well-accepted role of LDL cholesterol as a significant risk factor for CVD (70) and an apparent protective effect of HDL cholesterol (71). In epidemiological studies of the incidence of CVD in men, hypertriglyceridemia and hyperinsulinemia

appeared as major risk factors associated with increased risk for CVD events and CVD death in subjects with diabetes (72,73).

These data point to the abnormalities in carbohydrate and lipid metabolism associated with CVD in people with diabetes that differ from lipid abnormalities attributable to familial or other primary forms of hypercholesterolemia. Thus, nutrition recommendations must include a mix of dietary carbohydrate and fats that does not aggravate the metabolic abnormalities seen in this population.

Nutrition Recommendation Dilemma

In an effort to lower dietary saturated fat intake, diets with no more than 30% of daily calories as fat have been recommended. Carbohydrate in these diets may comprise up to 60% of total daily calories (59). This approach focuses on plasma levels of total cholesterol and LDL cholesterol as primary risk factors for CVD. However, short-term controlled-diet studies have demonstrated that high-carbohydrate (60% of calories) low-fat (20–25% of calories) diets may aggravate hypertriglyceridemia, reduce HDL cholesterol, and increase postprandial glycemia and insulinemia in people with type II diabetes while LDL cholesterol levels either do not change (74,75) or decrease (76). Moreover, epidemiological investigations indicate that increased plasma triglyceride, glucose, and insulin concentrations are associated with increased risk for CVD in patients with diabetes (77,78). However, the same population studies suggest that relatively high-carbohydrate, low-fat diets are also associated with a reduced incidence of CVD.

Nevertheless, the potential adverse effects of high-carbohydrate diets on lipids in some patients have stimulated investigators to develop alternative dietary approaches. In one such approach, saturated fats are replaced with monounsaturated and polyunsaturated fatty acids. In short-term studies in which high-carbohydrate, low-fat diets have been compared with moderate-carbohydrate (~45% of calories), high-fat diets (40% of calories; saturated fats less than 10% of total fat), the adverse effects of high-carbohydrate diets have been avoided (75,79).

Few long-term studies have compared nutrition guidelines for people with type II diabetes mellitus in free-living situations. In a recent 18-month study (80), people with type II diabetes were randomly instructed on one of three diets: a weight-management diet, a high-carbohydrate/fiber diet, or a modified-lipid diet (36% fat, 45% carbohydrate). Focusing on increasing carbohydrate and fiber intake or replacing saturated fat with unsaturated fat in the diet both resulted in similar improvements in LDL cholesterol and glycated hemoglobin with no lasting differences in final nutrient intake; average carbohydrate intake being ~46% and fat ~32–34%.

Another area of concern regarding dietary fat is the biological importance of long-chain omega-3 fatty acids. Epidemiological data indicate that chronic consumption of fish and other omega-3 fatty acid-rich foods in amounts higher than typical in the U.S. diet are associated with reduced incidence of CVD. Studies using large doses of concentrated fish oil preparations suggest that their putative antiatherogenic properties may be due to the ability of omega-3 fatty acids to reduce circulating lipids, particularly triglycerides, decrease blood pressure, and exert inhibitory effects on specific aspects of inflammation and coagulation (81). In patients with type II diabetes and hyperlipidemia, fish oil supplements decrease circulating triglyceride levels but may increase LDL cholesterol concentrations and worsen glycemic control. These adverse effects of fish oils on blood glucose have been attributed to stimulation of hepatic glucose output and to inhibition of insulin secretion (82). The incorporation of fish per se in the diet of people with diabetes is not known to have deleterious effects. Supplementation with commercial fish oil preparations for the treatment of severe hypertriglyceridemia must only be undertaken with medical and dietary supervision and cannot be recommended for the diabetic population at large.

CARBOHYDRATES/ SWEETENERS — The relative glycemic potency of many carbohydrate-rich foods has been compared, and these data have been published in the form of a glycemic index (83). Although different responses have been reported, questions have arisen as to the clinical utility of these data (84–86). If people with diabetes are to be encouraged to eat a variety of foods to ensure a nutritionally balanced diet, recommending only foods with a low glycemic response severely limits food choices. Although the glycemic index may not be useful for individual meal planning, the research has challenged traditional dogma regarding food absorption rates and consequent glycemic effects. There appears to be a spectrum of glucose responses to different starches and sugars with much overlap. When equal grams of complex and simple naturally occurring carbohydrates are compared, no significant differences are noted in glycemic response (87). Many factors, such as how food is processed, prepared, and digested, affect a food's glycemic response. From a clinical perspective, first priority should be given to the total amount of carbohydrate consumed rather than the source of the carbohydrate.

Additional questions have arisen related to the type of carbohydrate to be recommended for people with diabetes. For most of this century, the most widely held belief about the dietary treatment of diabetes was that sugars should be avoided. Although the origin of this belief is uncertain, it appears to be based on the assumption that sucrose and other sugars are more rapidly digested and absorbed than starches and thereby aggravate hyperglycemia. However, little or no scientific evidence supports this assumption. When fed as a single nutrient, sucrose produces a glycemic response similar to

Table 1—*Studies comparing glycemic effects of isocaloric amounts of sucrose and starch in diabetic subjects*

Study	Number of diabetic subjects	Duration	Percent calories from sucrose	Sucrose found to have adverse effects on glycemia?
Bantle JP, Laine DC, Castle GW, Thomas JW, Hoogwerf BJ, Goetz FC (90)	22	Single meal	25	No
Slama G, Haardt MJ, Jean-Joseph P, Costagliola D, Goicolea I, Bornet F, Elbrably F, Tchobroutsky G (91)	18	Single meal	14	No
Bornet F, Haardt MJ, Costagliola D, Blayo A, Slama G (92)	21	Single meal	15	No
Forlani G, Galuppi V, Santacroce G, Braione AF, Gianguilio S, Ciavarella A, Vannini P (93)	6	Single meal	15	No
Peters AL, Davidson MB, Eisenberg, K (94)	18	Single meal	14	No
Bantle JP, Laine DC, Thomas JW (95)	24	8 days	23	No
Wise JE, Keim KS, Huisinga JL, Willmann BS (96)	16	5 days	7	No
Loghmani E, Rickard K, Washburne L, Vandagriff H, Fineberg N, Golden M (97)	10	2 days	10	No
Abraira C, Derler J (98)	18	4 weeks	38	No
Bantle JP, Swanson JE, Thomas W, Laine DC (99)	12	4 weeks	19	No

Meals were provided to subjects by the investigators.

that of bread, rice, and potatoes (88). Moreover, fruits and milk consistently are reported to have lower glycemic responses than many starches.

Sucrose

The per capita consumption of all sugars (naturally occurring and added) in the United States averages 95 g/day and accounts for ~21% of total calorie intake and 48% of total carbohydrate intake (89). Average daily per capita consumption of sucrose is 41 g, which represents 9% of total calorie intake.

Studies comparing the glycemic effects of meals containing sucrose or starch when given to subjects with diabetes are summarized in Table 1. Five studies assessing the effects of single meals containing from 12 to 25% of calories as sucrose all found no adverse effect of sucrose on glycemia (90–94). Other studies providing as much as 38% of calories as sucrose and lasting for as long as 4 weeks also found no adverse effect of sucrose on glycemia (95–99). Thus restriction of sucrose in the diabetic diet because of concern about adverse effects on glycemia cannot be justified.

It is less clear whether dietary sucrose has adverse effects on lipemia in people with diabetes. In one study, type II diabetic subjects fed a high sucrose diet (16% of total calories) and a sucrose-free reference diet for 15 days demonstrated an increase in fasting plasma cholesterol, fasting plasma triglycerides, and day-long plasma triglycerides during the sucrose diet (100). In contrast, two other studies reported that type II diabetic subjects fed high sucrose (220 g/day, 19% of total calories, respectively) or high starch diets (<3% of total calories from sucrose in both studies) for 4 weeks demonstrated no difference in fasting serum cholesterol, fasting serum triglycerides (98,99), or postprandial serum triglycerides (99). However, all three studies employed small numbers of subjects and therefore had limited statistical power to detect effects of dietary sucrose on serum lipids. Additional research will be necessary to determine with certainty whether dietary sucrose adversely affects serum lipids.

There is evidence that sucrose consumption contributes to the development of dental caries in the general population (89), and it is likely that sucrose will affect people with diabetes similarly. There is no convincing evidence that sucrose causes other adverse effects in the

general population or in people with diabetes (101).

If individuals with diabetes are to consume sucrose and sucrose-containing foods, such foods should be substituted for other foods in the meal plan. To make such substitutions, the nutritional value of sucrose-containing foods and the presence of other nutrients frequently ingested with sucrose, such as fat, must be considered.

Fructose

Fructose is a common natural monosaccharide that has a taste similar to that of sucrose. It provides ~4 kcal/g as do other carbohydrates. Dietary fructose produces a smaller rise in plasma glucose than isocaloric amounts of sucrose and most starchy carbohydrates (90), and therefore may have utility as a sweetening agent in the diabetic diet. Several studies reported that fructose, when it replaced other carbohydrates in the diets of subjects with diabetes, resulted in a reduction in postprandial glucose levels (95,102,103). However, this potential benefit must be tempered by concern that fructose may have an adverse effect on serum lipids, especially LDL cholesterol. Studies that have compared a fructose diet to a sucrose-containing baseline diet have reported no adverse effects of dietary fructose on serum triglycerides (95,103,104) and serum cholesterol (102,104). However, since sucrose is a disaccharide containing glucose and fructose, the design of these studies may not have been optimal for detecting the effects of fructose on serum lipids. In one study in subjects with diabetes, a high fructose diet (double usual intake or 20% of daily calories) was compared with a high starch diet nearly devoid of fructose. The fructose diet was reported to increase fasting serum total (6.9% higher) and LDL cholesterol (10.9% higher) (103). Moreover, at least three studies employing nondiabetic subjects have reported that a high fructose diet increased fasting serum cholesterol (105–107).

Based on the available data, there is no reason to recommend that people with diabetes avoid fruits, vegetables, and honey, in which fructose occurs naturally. However, because of concern about the potential adverse effect of fructose in large amounts on serum lipids, the use of added fructose as a sweetening agent in the diabetic diet may have no overall advantage over other nutritive sweeteners.

Nutritive Sweeteners

Nutritive (caloric) sweeteners, other than sucrose and fructose, include corn sweeteners such as corn syrup, fruit juice or fruit juice concentrate, honey, molasses, dextrose, and maltose. There is no evidence that these have any significant advantage over sucrose in terms of improvement in glycemic response or caloric content. All nutritive sweeteners contribute calories and like sucrose and fructose must be accounted for in meal planning.

Sorbitol, mannitol, and xylitol are common sugar alcohols (polyols) that have a lower glycemic response than sucrose and other carbohydrates. Because of potential gastrointestinal side effects when ingested in large amounts, the FDA requires the statement, "Excess consumption may have a laxative effect," if reasonable and foreseeable consumption may result in a daily ingestion of 50 g.

Starch hydrolysates are formed by partial hydrolysis of edible starches. Their reducing activity can then be eliminated by hydrogenation, and the product becomes a polyol. The exact caloric value of the sugar alcohols is difficult to determine and may vary depending upon the mode of ingestion and person-to-person factors such as gastrointestinal transit time. The European Economic Community recently accepted a Directive on Nutrition Labeling defining the caloric values of all sugar alcohols as 2.4 kcal/g (101). In another study, however, stool excretion of sorbitol, maltitol, and hydrogenated starch hydrolysate was reported to be negligible, indicating a high digestion and absorption rate from the small intestine. The energy value of three polyols studied was ~3.5 kcal/g, suggesting that little caloric savings can be expected from the consumption of these sugar alcohols (108). For people with diabetes, they also have no significant advantages over other nutritive sweeteners.

Nonnutritive Sweeteners

Currently, there are three nonnutritive sweeteners (also referred to as intense sweeteners, low calorie, or alternative sweeteners) approved for use in the United States: saccharin, aspartame, and acesulfame K (109–112). Food and Drug Administration (FDA) approval is being sought for three others: sucralose, alitame, and cyclamates (113–115). For all food additives, including nonnutritive sweeteners, the FDA determines an acceptable daily intake (ADI). The ADI is defined as the amount of a food additive that can be safely consumed on a daily basis over a person's lifetime without any adverse effects. It includes a 100-fold safety factor. Actual intake is much less than the ADI. For example, the average aspartame consumption in the general population who consume aspartame (including children) is 2–3 mg/kg per day or ~4% of the US ADI of 50 mg/kg (116). For people with diabetes, who theoretically would be frequent and consistent consumers of aspartame, average consumption ranges from 2 to 4 mg/kg/day, well below the ADI (116). Thus, it is essentially impossible, to consume the ADI. In addition, these products undergo rigorous scrutiny by the FDA and are not allowed on the market unless they are safe for the public, including people with diabetes, to consume.

FIBER — Dietary fiber is commonly categorized according to its solubility in water; insoluble components are noncarbohydrate components including cellulose, lignin, and many hemicelluloses. Soluble components include pectins, gums, mucilages, and some hemicelluloses. Total dietary fiber refers to the sum of insoluble and soluble plant polysacchar-

ides and lignin (117,118). Current estimates of total dietary fiber intake of American adults range from 10 to 30 g daily, with males and females averaging 10 and 13 g/day, respectively (119).

Soluble fiber is fermented in the colon to gases and short chain fatty acids and thus contributes little to fecal bulk. In contrast, insoluble fiber is poorly metabolized and increases stool volume. Soluble fiber prolongs the rates of gastric emptying and intestinal transit, while insoluble fiber has the opposite effect (119,120). These biological properties of fiber (particularly the insoluble components) provide a rationale for their beneficial effects in the treatment of constipation, hemorrhoids and diverticulitis and in the possible prevention of colon cancer (121). No data indicate that people with diabetes are less likely than others to reap these potential benefits of dietary fiber. Furthermore, there is no correlation between daily fiber intake and the risk of developing diabetes (122).

Although there is some evidence that large amounts of dietary fiber may inhibit the absorption of some ingested minerals, instances of clinically important deficiencies of vitamins or minerals (including calcium and iron) induced by dietary fiber have not been found (118,119,123). The assumption is that Western diets are sufficiently enriched in micronutrients to compensate for any inhibitory effect of fiber on the intestinal absorption of these substances (124). Likewise, it has not been demonstrated that chronic, high intake of dietary fiber increases the incidence of bezoar formation in people with or without diabetes (119).

Effects on Lipid Metabolism

Large amounts (up to 60 g/day) of "crude" (soluble and insoluble) fiber, or increased dietary intake of insoluble fiber, do not modify circulating levels of cholesterol or triglycerides in nondiabetic subjects consuming low or high amounts of dietary cholesterol (125). In contrast, many studies in diabetic and nondiabetic subjects have reported significant decrements in fasting total and LDL cholesterol levels, with maintenance of fasting HDL cholesterol concentrations after brief or chronic consumption of certain soluble fibers, usually in amounts exceeding 20 g/day (118,126). Oat bran (127–129), various gums (118) and psyllium (130) appear to be more effective than wheat bran in lowering cholesterol. The reduction in total and LDL cholesterol typically ranges from 7 to 15% and is accompanied by an increase in fecal bile acid excretion. However, a recent meta-analysis of 20 reasonably well controlled trials of oat bran indicate a more modest cholesterol-lowering effect of ~5 mg/dl compared with control diets (128). Nevertheless, the reported inverse association between daily fiber intake and coronary heart disease (reviewed in 118) is commonly attributed to these changes in blood cholesterol levels. Both fasting and postprandial triglyceride levels are reported to decrease after dietary supplementation with soluble fiber (119,120).

A problem in interpreting many dietary fiber trials is that body weight, total calories and caloric distribution have frequently not been adequately controlled. Most high fiber diets investigated have also been high in carbohydrates and low in total fats, making it difficult to determine the influence of fiber per se on lipid metabolism. An additional potential confounding variable in studies of people with type I diabetes mellitus has been the effect of changing insulin doses during the course of investigation. Despite these shortcomings, the cumulative evidence from well conducted clinical trials (131–133) indicates that chronic consumption of soluble fiber (20 g/day or more, a quantity which is difficult to consume by food alone) and a carbohydrate-enriched diet can marginally reduce blood total and LDL cholesterol, while maintaining HDL cholesterol levels, in people with type II diabetes mellitus beyond that achieved by only lowering dietary saturated fat and cholesterol.

Conclusions regarding the triglyceride-modifying effects of dietary fiber in people with type I or type II diabetes mellitus are even more difficult to interpret, owing to the frequent number of coexisting variables described above.

Effects on Carbohydrate Metabolism

In theory, soluble fibers might be expected to improve glycemic control in people with diabetes because *1*) they may reduce the rate of absorption of nutrients, including carbohydrates, from the bulk phase in the lumen of the small intestine (134–136), *2*) they may alter secretion of such gut hormones as gastric inhibitory polypeptide, glucagon and somatostatin, thereby indirectly affecting insulin secretion, counterregulatory hormone action and carbohydrate intestinal absorption (137–139), *3*) they may be associated with improved peripheral insulin action by increasing insulin receptor binding (140–142), and *4*) they may generate short chain fatty acids that inhibit fatty acid mobilization, thereby indirectly suppressing hepatic gluconeogenesis (135,143). Of these possible mechanisms, the one best substantiated is the first, delayed nutrient absorption. This relates to the ability of ingested fiber and food to form a viscous gel and thus potentially impair transfer of glucose to the absorptive surface of the intestine. Moreover, the biological significance of this effect may be limited to a few soluble fibers, since only guar and oat gums have been reported to reduce fasting hyperglycemia and blunt the postprandial rise in blood glucose (126).

The interpretation of results from studies of the glucose-modifying effects of dietary fiber are clouded by those same problems presented above for assessing the influence of fiber on lipid metabolism. Although selected soluble fibers are capable of inhibiting absorption of glucose from the small intestine, the effect of dietary fiber on glycemic control is probably insignificant. Furthermore, the supplemental addition to the normal diet of concentrated preparations of fiber cannot be recommended as a means of improv-

ing glycemic control in patients with diabetes.

Intake recommendations for people with type I or type II diabetes are the same as for people without diabetes. They should be advised to incorporate foods naturally rich in total fiber into their diet. The accepted nutritional guidance of increasing current intake to 20–35 g/day from a variety of food sources, including vegetables, legumes, grains, and fruits is prudent for the general public and for people with diabetes. (144–146).

SODIUM — In the United States, average sodium intake per day is reported to be between 4,000 and 5,800 mg (4–6 g). This is well above the amount needed for normal body function. In food, sodium is present mainly in table salt. A teaspoon of salt (5 g) contains ~2,300 mg of sodium.

Uncertainty exits as to the role of sodium in regulating blood pressure across the spectrum of normotensive to hypertensive. People appear to differ greatly in their sensitivity to sodium and as a result the effect of more or less sodium on blood pressure varies greatly. For instance, blacks may be more sensitive to sodium than other ethnic groups (147). Because sodium-sensitive individuals are not easily identified, it is usually recommended that the public reduce their sodium intake. The American Heart Association recommends that sodium intake for the general public be no more than 3,000 mg/day (148), while other authorities recommend not more than 2,400 mg/day (149). Total sodium intake of less than 2,400 mg of sodium (or less than 6 g/day of sodium chloride) is recommended for individuals with mild to moderate hypertension (7,147,150,151).

The effects of a low sodium diet (460 mg/day) compared with a high sodium diet (5,750 mg/day) in hypertensive and normotensive people with and without diabetes found that 8 of the 12 hypertensive subjects with diabetes showed a sodium sensitive blood pressure response with an augmented response to infused angiotensin II (152). In a study of mildly hypertensive subjects with diabetes on moderate dietary sodium restriction compared with subjects on their usual diabetic diet, an reduction of ~20 mmHg in systolic blood pressure was reported. A difference in diastolic blood pressure could not be demonstrated (153). Several studies have suggested that people with diabetes may have reduced sodium excretion, probably as a result of enhanced tubular reabsorption of sodium (154,155).

ALCOHOL — Alcohol in beverages is ethanol, the intoxicating molecule present in distilled spirits (hard liquor), wine, and beer. One drink is commonly defined as a 12-oz (360 ml) beer, 5-oz (150 ml) glass of wine, or 1.5-oz (45 ml) shot of ~80-proof spirits, each of which contains ~0.5 oz, or 12 g, of alcohol.

The liver is the major organ for alcohol metabolism. It metabolizes alcohol slowly, at an average rate of ~0.1 g/kg of body weight per hour (156). An average 70-kg man will require ~2 h to metabolize 1 oz (24 g) of alcohol.

Alcohol not immediately metabolized by the liver enters the general circulation, where it becomes a part of all body fluids. The total quantity and rate of alcohol ingestion determines its effect. The main pathway for initial oxidation of alcohol in the liver involves the enzyme alcohol dehydrogenase (ADH), which oxides alcohol to acetaldehyde and then to energy. The reaction generates excess of reducing equivalents in the form of NADH which affects many additional biochemical reactions (157).

Effects on Blood Glucose

Ingestion of alcoholic beverages in the fasting state may cause hypoglycemia or may mask the symptoms of hypoglycemia from other causes. Hypoglycemia can occur at blood alcohol levels that do not exceed the range of mild intoxication and may occur even in occasional drinkers who have missed or delayed food intake (158).

Alcohol can acutely interfere with counterregulation to insulin-induced hypoglycemia and usually occurs when plasma insulin levels are low and glucagon levels are elevated. The hypoglycemia induced by alcohol may not be ameliorated by glucagon as it appears to be related to depleted glycogen stores. Hypoglycemia is caused by an indirect impairment of gluconeogenesis and is not associated with excess insulin secretion (159).

Clinical manifestations of the effect of alcohol on glucose depend not only on the amount of alcohol ingested but also on its relationship to food intake. In the fasting state, alcohol may produce hypoglycemia, but in the fed state, it may produce hyperglycemia related to liver glycogenolysis and peripheral insulin resistance (160). This hyperglycemia may occur late in the course of alcohol intake and frequently when plasma concentrations of alcohol are declining. It is followed several hours by a fall in blood glucose to below the fasting level (161).

Chronic alcohol intake may impair glycemic control, may aggravate hypertriglyceridemia, and is also a risk factor for neuropathy (162).

Occasional Use of Alcoholic Beverage

Under normal circumstances, however, blood glucose levels will not be affected by the moderate use (~1 oz [2 drinks]) of alcohol when diabetes is well controlled (163–168). Nutritional guidelines published by the U.S. Department of Agriculture and the U.S. Department of Health and Human Services define moderate drinking as "no more than one drink a day for most women, and no more than two drinks a day for most men" (13).

Because alcohol may increase the risk for hypoglycemia in people treated with insulin or sulfonylureas, alcohol should only be ingested with a meal. The same precautions regarding the use of alcohol that apply to the general public also

apply to people with diabetes. Individuals who should be advised to abstain from alcohol use include alcohol abusers; individuals with other medical problems including pancreatitis, hypertriglyceridemia, gastritis, frequent hypoglycemic reactions, neuropathy, and certain types of kidney and heart disease; and women during pregnancy. Alcohol can interact with barbiturates, tranquilizers, and numerous other drugs.

MICRONUTRIENTS: VITAMINS AND MINERALS —

The role of noncaloric nutrients in the diet of people with diabetes must be considered in the context of total dietary intake. In general, when intake conforms to the overall recommendations for total nutrient content in terms of maintenance caloric needs and appropriate composition, then there is no need for additional vitamin and mineral supplementation. This is particularly true for the trace minerals copper, manganese and selenium. Similarly, there is no need in people with diabetes to supplement either water- or fat-soluble vitamins (169,170) The response to supplements is largely determined by nutritional state, so that only people with micronutrient deficiencies are likely to respond favorably.

People who are at greatest risk of deficiency and who need more comprehensive assessment regarding potential prescription of vitamin and mineral supplements include the following: people on weight-reducing diets, strict vegetarians, the elderly, pregnant or lactating women, those taking medications known to alter micronutrient metabolism, patients in poor metabolic control, or patients in critical care environments. Trace-metal urinary losses are accentuated during uncontrolled hyperglycemia and glycosuria.

There are special circumstances related to chromium, zinc, magnesium, vitamin E, vitamin B_{12} and iron that occur in people with diabetes that warrant mention.

Chromium deficiency in animal models is associated with elevated blood glucose, cholesterol, and triglyceride levels (171,172). However, it is unlikely that most individuals with diabetes are chromium deficient. In individuals with diabetes, three double blind crossover studies on chromium supplementation did not result in any improvement of blood glucose control (173–175). In individuals with impaired glucose tolerance who consumed a diet deficient in chromium for 4 weeks, chromium supplementation improved glucose tolerance (176). Glucose intolerance from chromium deficiency as a result of long-term parenteral nutrition, has been reportedly reversed with chromium supplementation (177). However, today total parenteral nutrition prescriptions routinely include the addition of trace minerals.

While serum zinc levels are generally lower in people with diabetes when compared to their nondiabetic counterparts, zinc replacement is only suggested to be of benefit in helping to heal venous leg ulcers (178). It is not clear whether zinc administration has efficacy under all such circumstances (179).

There may be a need for magnesium replacement in patients in poor glycemic control or who are on diuretics. Magnesium depletion has been associated with insulin insensitivity, which may improve with oral supplementation (180).

Vitamin E levels are variable in people with diabetes. Although there are hypothetical reasons to supplement vitamin E for the potential antioxidant benefits, there is little confirmatory evidence that such therapy has any benefit. Although observational data in both men and women (diabetic and nondiabetic) suggest benefit from vitamin E ingestion in reducing the risk for coronary heart disease (181–182), these observations must be interpreted with caution and must be supported by prospective studies before recommending routine use of vitamin E as a means to prevent atherosclerotic disease (183).

There is no evidence of benefit from routine supplementation of vitamin B_{12} in subjects with diabetes except under circumstances of documented deficiency such as may occur with polyglandular endocrinopathies associated with type 1 diabetes or elderly patients with type 2 diabetes (184). Similarly, iron replacement should be administered only in the face of documented iron deficiency (185).

OBESITY AND DIABETES —

Obesity, in particular central intra-abdominal obesity, is associated with hyperinsulinemia, insulin resistance and type II diabetes. Not all centrally obese people have type II diabetes, but most people with type II diabetes have central obesity and insulin resistance as well as defects in insulin secretion. Hypertension, hyperuricemia, and dyslipidemia are also more common with central obesity and type II diabetes.

Obesity, defined as an excess body fat can be expressed using body mass index (BMI) in kg/m^2. This numerical expression is limited, however, by the fact that BMI does not differentiate body fat mass from lean mass, centrally distributed body fat, or age, gender and ethnic differences in body fat content and distribution. Though the exact figure varies depending on the source, obesity generally is defined as a BMI of >28–30. This definition represents the segment of the adult population at increased risk because of their excess weight. In 1985, the National Institutes of Health Consensus Conference concluded that obesity (BMI >27.8 for males and >27.2 for females) had an adverse effect on health and longevity (186).

An increase in body fat occurs when the amount of calories consumed is not equivalent to the amount of calories expended. The cause of obesity is considered to be a complex interaction between genetic and environmental factors. Appetite regulation, food choices and physical activity are influenced by environmental and psychological factors and interact

with a genetic predisposition to increased body fat.

While caloric restriction resulting in a lower body weight has been shown to ameliorate the metabolic abnormalities associated with type II diabetes (19,187,188), the most successful approach to achieving and sustaining weight loss is unclear. Most individuals with a chronic tendency toward obesity can expect that successful weight control will be a lifelong effort. The majority of individuals who restrict calories to lose weight will regain lost weight and body fat. Frequent weight fluctuations are thought to be detrimental both psychologically and physiologically (189–191). Data from the Framingham Study suggest that total mortality, mortality from coronary heart disease and morbidity, increases with fluctuating body weights (189).

Wadden and colleagues (192) found no impaired ability to lose weight in weight cyclers. Cyclers were heavier and had more body fat than the less frequent weight cyclers. Other studies examining weight cycling identified increasing age (193) and decreasing dietary compliance (194) as the primary causes of failure to lose weight in frequent dieters.

Weight Reduction Methods

Successful weight reduction strategies most commonly employed include caloric restriction, regular exercise, behavior modification and prolonged and frequent peer or professional support. Pharmacological therapy and surgical procedures are less common and most often reserved for complex, difficult cases.

Calorie restriction. Caloric restriction, independent of weight loss, has the potential to improve blood glucose levels and insulin sensitivity (195).

Moderate restriction is defined as a reduction in caloric intake of 250–500 kcal/day below usual or maintenance intake or a caloric intake of 70% of maintenance energy requirements. Severe restriction, or very low calorie diets (VLCDs) are composed of less than 800 kcal/day either as a liquid supplement or high protein foods. They produce a more rapid weight loss initially and are appropriate only in individuals with a BMI >30 and with medical supervision.

Recent studies suggest that restricting fat calories can result in a decrease in body weight without restriction of calories from either carbohydrate or protein (22,23). These observations have sparked interest in restricting daily consumption of calories from fat alone, either by counting grams of fat or generally reducing fat intake. This diet modification may be useful in individuals with initially high fat intakes.

Controversy over the efficacy of using VLCDs has centered around concern that rapid weight loss produces a large decrease in resting metabolic rate (RMR) and thus contributes to the weight regain experienced more often than with modest caloric restriction. However, few studies have actually compared RMR during a VLCD and a modest restriction of calories (500 calories less than usual intake). In two separate studies (196–197), women consuming a VLCD were compared to those consuming 1,200–1,400 kcal/day. Resting energy expenditure decreased in proportion to decreased fat free mass (FFM). This confirmed previous findings that changes in FFM determine RMR and changes in FFM are inevitable during weight loss, accounting for nearly 25% of weight loss (198–199).

While initial weight loss is extremely good with VLCDs, long-term results are poor. Most subjects regain lost weight. The addition of behavior modification and exercise to a VLCD plan improves short-term and 1- to 2-year results as subjects maintain 48–63% of lost weight (200,201). However, the only study examining 3- to 5-year follow-up data showed no improvement in weight maintenance with the addition of behavior therapy (202).

VLCDs have been used safely and effectively in type II diabetes producing positive effects on blood glucose control, lipids and blood pressure (203,204). Adding a VLCD to behavior therapy did not improve weight loss at the end of one year, but did result in better glycemic control (205).

Exercise. Regular exercise (3–5 times/week) enhances weight loss in dieting individuals (206) and is identified as a predictor for successful weight maintenance (207–209). Individuals with diabetes also benefit from other metabolic improvements associated with exercise such as enhanced glucose uptake, improved insulin sensitivity (210), and increased lean body mass (associated with exercise) that increases daily energy expenditure (211). In general, men lose more weight and body fat with exercise than women (212). In one study, women with upper body obesity lose body fat more easily with exercise than do women with lower body obesity (213).

Studies in obese type II individuals suggest that exercise does not consistently (210) enhance weight loss efforts through diet and behavior modification. Success may be influenced by medication dosage and level of glucose control. Obese individuals cannot be expected to lose weight if food intake is increased with exercise to prevent hypoglycemia. The most significant effect of adding exercise to caloric restriction appears to occur during the weight maintenance and long term weight loss stage of managing obesity.

Behavior therapy. Behavior therapy in obesity is usually implemented in group programs and includes techniques such as self monitoring of food intake to identify problem foods and eating patterns as well as tracking food consumption and exercise. Other strategies such as stimulus control (i.e., eliminating environmental triggers for eating), preplanning, cognitive restructuring (i.e., changing self-perceptions and beliefs), and reinforcement techniques help patients change their environment.

Current behavior therapy programs result in a weight loss of 1–1.5 pounds/week during treatment (214). Total weight loss is proportional to the

length of time subjects are treated. The average program lasts 16–20 weeks with sessions held weekly or every two weeks. Follow up programs may enhance weight loss (215). The results of behavior therapy in studies of obese people with type II diabetes mellitus parallel the results of behavior therapy reported in nondiabetic obese people.

Pharmacological therapy. Despite somewhat disappointing long term effectiveness, a hypocaloric diet, exercise and behavior therapy are the most common treatment modalities for obesity currently available for people with and without diabetes. Most individuals with refractory obesity who have a BMI >30 and have struggled with weight loss efforts for years feel abandoned by this approach. Pharmacological therapy has been indicated as potentially beneficial in this group in whom the risk/reward ratio is favorable (216). Newer pharmacological agents, i.e., the serotonergic appetite suppressants and the thermogenic agents, lack potential addiction problems and the catecholamine-like side effects found in amphetamines.

Weight lowering effects have been demonstrated with both short and long term administration of fenfluramine (217). A 4-year study combining fenfluramine and phenteramine in a weight loss program that included diet, exercise and behavior therapy demonstrated that long term use can be safe and cause modest weight loss (218).

Studies with fenfluramine in obese individuals with type II diabetes mellitus show an effect on blood glucose that may or may not be in addition to weight loss or decreased food intake (219). This effect is thought to be related to reduced insulin resistance. More studies on the short and long term efficacy and safety of these drugs in people with diabetes mellitus are required.

Gastric reduction surgery. The 1991 NIH Consensus Development Panel on Gastrointestinal Surgery for Severe Obesity suggested that gastric reduction surgery be considered for patients with BMI >35 and a co-morbid condition such as diabetes mellitus (220). Two surgical procedures were recommended. They are vertical banded gastroplasty and gastric bypass. Gastric bypass produces greater weight loss, but also increases risk that nutritional deficiencies will develop (220). In a study of 515 morbidly obese patients who underwent gastric bypass, mean weight loss at five years was 95 pounds (221). Of 137 patients with NIDDM in this series, 107 (78%) experienced remission of diabetes. At present, there are no controlled clinical trials of gastric reduction surgery in people with type II diabetes mellitus on which to base an estimate of the relationship between benefit and risk.

HYPERTENSION AND DIABETES

— The relationship between diabetes and hypertension has been well described in a number of studies (222–229). In type II diabetes there is an association of hypertension with obesity, especially the central intra-abdominal type of obesity. However, the association of hypertension with diabetes exists even in the absence of obesity in both type I and type II diabetes. In people with diabetes the risk for macrovascular complications associated with hypertension and the increased risk with increasing blood pressure occurs to the same degree as in nondiabetic individuals. However, in people with diabetes the presence of any degree of hypertension doubles the risk for atherosclerotic events (147). These relationships are most evident in people with type II diabetes. In people with diabetic nephropathy, the presence of hypertension is associated with a more rapid progression of nephropathy (147).

Diet-Related Factors and Hypertension

Several diet-related interventions may be considered in the treatment of patients with diabetes and hypertension. The benefits of these interventions are due to the effects of nutrition therapy on hypertension in both nondiabetic and diabetic subjects. Interventions with evidence of benefit include weight reduction, sodium (salt) restriction, and restricted alcohol intake (230). These recommendations are made in conjunction with a recommendation for regular exercise. A number of other nutrients, i.e. potassium and calcium, have also been evaluated and may have benefit in some patients with diabetes and hypertension.

Weight reduction. While there is, general association in people with diabetes between weight reduction and a reduction in blood pressure, there is a great deal of variability in the response. The degree of blood pressure lowering to be expected from weight loss is not well quantified (149,231). Weight reduction results in diminished plasma volume and may also be associated with decreased sympathetic activity.

Sodium. By contrast there are much better data on the potential benefits of sodium restriction(232–234); however, the data also show a wide variety of responses suggesting that salt sensitive hypertension may be much more common in some patients with diabetes than others (152,153).

Alcohol. Information on alcohol intake and hypertension in people with diabetes is not available. However, in nondiabetic subjects regular alcohol ingestion (consumption >1–2 ounces of alcohol/day) is associated with a higher prevalence of hypertension with a pressor effect (149,235–237). Large scale studies do not show much difference in blood pressure among those people who consume <3 drinks/day (238).

Potassium. High potassium intake has also been associated with a reduction in blood pressure in epidemiological studies. Potassium administration is not uniformly associated with a reduction in blood pressure. However, potassium supplementation used with sodium restriction appears to offer little additional benefit over sodium restriction alone (239).

Special considerations for potas-

sium intake in people with diabetes include: *1*) the effects of antihypertensive therapy on serum potassium levels, 2) the effects of progressive renal dysfunction and 3) the presence of hyporeninemic hypoaldosteronism. In patients who take diuretics for hypertension, potassium loss may be sufficient to warrant supplementation. Doses should be adjusted based on serum potassium concentration to maintain normokalemia. In patients with progressive renal dysfunction potassium levels and intake need to be measured as hyperkalemia may accompany renal compromise. Dietary intake of potassium should be restricted in subjects who develop hyperkalemia in conjunction with progressive renal failure. Finally, in hyporeninemic hypoaldosteronism hyperkalemia may occur. This is usually managed with modest reduction in potassium intake and diuretic therapy.

Calcium. Interest in the possible beneficial effects of calcium ingestion has recently been reviewed (240). The blood pressure response to calcium ingestion is variable and unpredictable and may be related to salt sensitivity of subjects. The effect of calcium ingestion on blood pressure is not sufficiently consistent to recommend supplemental calcium intake to treat hypertension in people with diabetes.

NEPHROPATHY AND DIABETES

— The earliest clinical evidence of diabetic nephropathy is the presence of proteinuria (241–244). Studies show that the risk of proteinuria is related to glycemic control (245,246) and that the rate of progression of proteinuria is related to the presence of hypertension (247–249). The possibility that early changes in renal function, determined by changes in proteinuria, increased plasma creatinine levels and decreased creatinine clearance, may be modulated by restricting dietary protein intake (250–252) has been discussed in the section on protein.

As renal function deteriorates further, several other metabolic abnormalities occur that may be modified by dietary changes and administration of dietary supplements. Altered renal function is associated with impaired potassium excretion and may be associated with hyperkalemia. Reducing potassium intake helps control the severity of the hyperkalemia.

Renal failure is also characterized by impaired phosphorus excretion and an associated hyperphosphatemia. This is followed by relative hypocalcemia and, in turn, secondary hyperparathyroidism and metabolic bone disease. Increased calcium intake, especially in the form of calcium supplements is often necessary to correct these abnormalities.

Finally, metabolic products of protein breakdown, may contribute symptoms that characterize the uremic syndrome. This syndrome includes constitutional symptoms of itching, fatigue and nausea. Reducing protein intake and providing high biologically active protein will reduce the risk for these symptoms.

DYSLIPIDEMIA IN DIABETES

— The most common lipid abnormality in diabetes is an elevation of plasma triglyceride levels that occurs in ~20% of individuals with type II diabetes (253). In most cases, improved glycemic control with insulin or sulfonylurea treatment reduces triglyceride levels. However, patients with central obesity and the insulin resistance syndrome or those with a familial form of hypertriglyceridemia may require a lipid-lowering drug. The risks of elevated lipoprotein levels in diabetes fall into two categories: atherosclerosis and the chylomicronemia syndrome. Atherosclerosis is associated with elevated triglyceride levels, decreased HDL cholesterol levels, and abnormalities in the size and density of LDL cholesterol (254). Pancreatitis due to the marked hypertriglyceridemia of the chylomicronemia syndrome is less common.

The modest hypertriglyceridemia and lower HDL cholesterol levels seen in the majority of untreated patients with diabetes returns toward normal levels or to levels seen for their degree of adiposity after several months of insulin or oral sulfonylurea therapy, and correlates with changes in fasting glucose and glycated hemoglobin levels (255). Both the hypertriglyceridemia and the low HDL cholesterol in the untreated person with diabetes mellitus appear to be related to decreased lipoprotein lipase (LPL) in adipose tissue and in skeletal muscle. Abnormalities in LPL in skeletal muscle seem to correct rapidly after therapy for the hyperglycemia while the adipose tissue LPL defect corrects slowly, with the resultant return of the high triglyceride and low HDL cholesterol levels toward normal.

Pancreatitis due to severe hypertriglyceridemia in the untreated person with diabetes is less common. However, it is important to recognize, since it is a treatable and preventable condition. People with type I and type II diabetes who present with untreated hyperglycemia may have marked increases in triglyceride levels (from 2,000 to 20,000 mg/dl), and are at risk to develop acute pancreatitis. This unusual subset of individuals with diabetes have very high triglyceride levels due to the combination of diabetes and the independent inheritance of a familial form of hypertriglyceridemia. They frequently have nondiabetic hypertriglyceridemic relatives. After treatment of the hyperglycemia with insulin or oral sulfonylurea therapy, the triglyceride levels may decrease to levels similar to those in their nondiabetic relatives. After diagnosis, these people may need to be on very low fat diets for several months. With the maintenance of triglyceride levels below 1,000 mg/dl, the recurrence of pancreatitis in these individuals is uncommon (255).

The prevalence of atherosclerosis of the coronary, cerebral and peripheral vessels in type I and type II diabetes mellitus is in part related to the lipoprotein abnormalities seen in people with diabe-

tes. The plasma triglyceride level in these people is a marker for this predisposition and for the increase in risk of atherosclerosis at an early age. Abnormalities in the distribution of VLDL, IDL and LDL cholesterol, and a decrease in HDL_2 seem to be the mediators of this associated risk (256,257). Drug therapy aimed at correcting these abnormalities would seem to be indicated. The patient with marked hypertriglyceridemia and the chylomicronemia syndrome must be followed closely. If the hypertriglyceridemia does not respond to treatment with insulin or oral sulfonylureas, these patients are candidates for lipid-lowering drugs. This therapy, combined with the avoidance of diuretics and beta adrenergic blocking agents, should prevent recurrent pancreatitis in these individuals (255).

Rates of elevated total cholesterol in people with diabetes in the United States are only slightly higher than in those without diabetes after adjusting for age and sex. Nevertheless, high or borderline high total cholesterol is reported to be common in people with diabetes and may be present in as many as 70% of adults with diagnosed diabetes and 77% with undiagnosed diabetes in the U.S. population. Of these individuals, 95% have evidence of coronary heart disease or two or more risk factors for heart disease and should therefore have their LDL cholesterol measured. Based on national data, LDL cholesterol levels warranting nutrition treatment for hypercholesterolemia would be expected in 85% of these people (258). Furthermore, in the San Antonio Heart Study more than 40% of the subjects with diabetes were hyperlipidemic according to the criteria of the National Cholesterol Education Program, and an additional 23% had hypertriglyceridemia and/or low levels of HDL-cholesterol. By contrast, less than one-fourth of the subjects who did not have diabetes were hyperlipidemic (63).

Nutrition and physical activity changes remain the basis of treatment of lipid disorders in people with diabetes. Treatment should focus on weight reduction as indicated, increased physical activity, and a low-fat, low-saturated fat diet (7). Generally, the greater the weight loss, the greater the improvement in triglycerides, insulin sensitivity, glucose control, and reduction in total and LDL cholesterol, and increases in HDL cholesterol levels, however, even with weight loss of less than 5 kg (10 lbs) improvement in lipid patterns has been observed. Hypertriglyceridemia, hypercholesterolemia, and low HDL cholesterol may also improve greatly with aggressive treatment of hyperglycemia. If there is unacceptable response to an adequate therapeutic trial of diet, exercise, and improved glucose control, lipid-lowering pharmacological agents may need to be added to the treatment program (7).

CATABOLIC ILLNESS — The catabolic state induced by injury, inflammation or severe illness is associated with special nutrition considerations. Most studies of nutrient needs are in subjects who do not have diabetes but the results are likely applicable to people with diabetes mellitus. Many of the changes in nutrient requirements appear to be cytokine mediated events. Cytokines reduce albumin production, decrease plasma levels of some trace proteins through tissue redistribution, induce negative nitrogen balance and alter intermediary metabolism in such a way as to increase glucose levels through the actions of the counter regulatory hormones (259). These changes, as well as altered fluid balance, make nutritional assessment difficult during times of critical illnesses. Overall, catabolic disease states result in a change in body compartments that may be characterized by an increased extracellular fluid component (frequently with an actual increase in body weight) and an associated shrinkage of body fat and body cell mass. Body cell mass is defined as the actively functioning protein-rich tissue and associated intracellular fluid (260).

Antecedent weight loss is a predictor of the need for nutritional support. With less than 10% antecedent weight loss, fluid and electrolytes can be used for one week in most catabolic states. With mild illness only fluid and electrolyte support may be needed for people with an antecedent weight loss of up to 20%. However, with >20% weight loss in any catabolic illness or >10% weight loss in any severe catabolic illness, additional nutritional support is necessary. Enteral nutrition is preferred over parenteral nutrition as a method of nutrient delivery (259). When hyperglycemia occurs, continuous insulin therapy via an insulin drip or intermittent insulin therapy via the subcutaneous route can be given with either method of nutrient administration (261).

Nomograms that take into account body weight and body composition (based on age and gender) are used to calculate caloric intake. Overfeeding should be avoided because of effects on oxygen consumption and carbon dioxide production. Caloric needs for patients without significant weight loss are in the range of 30–35 kcal/kg every 24 h. Increased calories may be required for significant weight loss. Care must be taken not to exceed the overall nutrient disposal rate of ~50 kcal/kg every 24 h, or significant hyperglycemia will frequently ensue. Stressed patients should receive ~1.5 g of protein/kg of body weight per day. This will not decrease protein catabolism, but will result in an increase in protein synthesis. At least 30% of total calories should be given as lipids. Use of dextrose alone as an additional source of calories is associated with increased risk of hyperglycemia, hyperinsulinemia and hepatic dysfunction. Carefully done body composition studies have demonstrated that the net effect of dextrose alone as nutritional support will not prevent protein depletion and does increase body fat. Either endogenous hyperinsulinemia or use of high doses of exogenous insulin may be associated with decreased excretion of sodium (262). Careful and continuous monitoring of nutrition status is critical to

Table 2—*Recommended total weight gain ranges for pregnant women*

Weight-for-height category	Recommended total weight gain
Low (BMI <19.8)	12.5–18 kg (28–40 lb)
Normal (BMI 19.8–26)	11.5–16 kg (25–35 lb)
High (BMI 26–29)	7–11.5 kg (15–25 lb)
Obese (BMI >29)	<6 kg (15 lb)

Recommendations are from the National Academy of Science.

ensure that nutrient needs are being met and hyperglycemia is prevented.

IMPAIRED GLUCOSE TOLERANCE (IGT)— The prevalence of IGT in the U.S. population is reported to be 11.2%, compared to 6.6% for diabetes (diagnosed plus undiagnosed) (263). Thus, IGT comprises two-thirds of all glucose intolerance. The progression of IGT to overt diabetes is unclear, but there may be key factors occurring during the period of IGT that precede and are involved in the pathogenesis of type II (264–265).

Although IGT is not a major risk factor for microvascular complications, it is a major risk factor for macrovascular complications (6). Prevention of macrovascular disease (by management of associated known cardiovascular risk factors such as dyslipidemias and hypertension) and preventing the progression of IGT to overt diabetes should be a high priority for people identified with IGT. In terms of nutrition, a higher fat consumption in people with IGT was shown to predict progression from IGT to type II diabetes (266).

For people with IGT, lifestyle behaviors to promote a reasonable weight and physical exercise should be recommended (267). Nutrition guidelines and exercise recommendations for people with type II diabetes also apply to people with IGT. Furthermore, three recent studies have reported that physical activity has the potential to prevent or delay the onset of type II diabetes in individuals at risk (268–270).

PREGNANCY AND DIABETES— The goals of nutrition during pregnancy are to provide adequate maternal and fetal nutrition, to assist in appropriate maternal weight gain that is neither subnormal or excessive, and in pregnancy complicated by diabetes, to assist with normalization of maternal blood glucose levels. For women with diabetes before pregnancy, prepregnancy counseling, including nutrition assessment and therapy, is vital for the attainment of optimum diabetes control before pregnancy, essential for the prevention of congenital anomalies (271).

A favorable pregnancy outcome has been defined as a gestational duration of 39–41 weeks and a live birth weight of 6.6–8.8 lb (3–4 kg) (272). Prepregnancy weight, as well as weight gain during pregnancy, are known to influence birthweight, maternal morbidity, and perinatal mortality.

Weight Gain/Caloric Requirements

To promote a favorable outcome, the National Academy of Science (NAS) recommends the total weight gain ranges for pregnant women shown in Table 2, based on prepregnancy BMI (272). However, weight gain by women giving birth to healthy infants is highly variable. Approximately 70% of normal or underweight women gain 10–35 lbs. Weight gain in women >135% DBW has less of an effect on birthweights of infants. Obese women often have significantly heavier babies independent of weight gain from 0 to 30 lbs (273,274). Therefore, it is generally recommended that obese women gain a minimum equivalent to the products of conception (6.8 kg [15 lb]), although lower weight gains are often compatible with optimal birth weight.

Longitudinal studies of well-nourished pregnant women generally indicate a slight increase in energy intake during pregnancy, generally in the range of an additional 70–240 kcal/day. Women easily meet this by reducing intensity of physical work and by making small adjustments in food intake (275,276). The RDA suggests that unless women begin pregnancy with depleted body reserves, additional calories are probably not required during the first trimester, therefore, an additional 300 kcal/day is recommended during the second and third trimesters (277). The uncertainty of precise additional calories needed during pregnancy again emphasizes the need to individualize and monitor caloric requirements needed to meet desirable total weight gain for each woman.

Nutrition assessment, including a complete diet history, should be used to determine calories required during pregnancy. An individualized meal plan is then developed and followed up with monitoring of food records, weight gain, ketones, and blood glucose levels. Exogenous insulin can then be matched to food intake. Smaller frequent meals and snacks are helpful. A bedtime snack should be recommended to decrease the incidence of overnight hypoglycemia and fasting ketosis.

Maternal third trimester ketonemia has been associated with lower intelligence scores at 2–5 years in the offspring, so every attempt should be made to avoid ketonemia either from ketoacidosis or accelerated starvation ketosis in all pregnant women (278).

Gestational Diabetes

Gestational diabetes mellitus (GDM) is defined as carbohydrate intolerance of variable severity with onset of recognition during the present pregnancy (279). Treatment for GDM consists of improving metabolic abnormalities associated with

GDM through nutrition therapy, exercise, and, if needed, insulin therapy. Blood glucose monitoring is essential to determine if treatment goals are being met. The goal of nutrition therapy is to provide adequate calories and optimal nutrition during pregnancy without hyperglycemia or ketonemia.

For obese women with GDM a 30–33% calorie restriction (an intake of ~1,800 kcal/day) has been shown to reduce hyperglycemia and plasma triglycerides with no increase in ketonuria (280,281). Obese women (BMI >30) may do well with moderate calorie restriction as a goal.

Individualization of the meal plan is recommended, as the ideal percentage and type of carbohydrate is controversial. However, more important than percentages and type of carbohydrate may be the actual grams of carbohydrate. Breakfast meals often require less than 30 g of carbohydrate to prevent excessive elevations of postprandial blood glucose levels. Limited carbohydrate intake at breakfast and frequent small feedings throughout the day will often return blood glucose levels to normal.

Monitoring blood glucose levels, urine ketones, appetite, and weight gain can guide in developing an appropriate individualized meal plan and in making adjustments to the meal plan throughout pregnancy.

Exercise is a treatment modality that can assist in overcoming peripheral resistance to insulin and can lower postprandial glucose elevations. Exercise can be advocated as a treatment for GDM in women who do not have a medical or obstetric contraindication for an exercise program (282). The safest form of exercise is one that does not cause fetal distress, uterine contractions, or maternal hypertension. Appropriate exercises are those that use the upper body muscles or place little mechanical stress on leg and trunk regions during exercise (283).

After delivery, 97% of all women with GDM return to normal glycemia. However, women with GDM have up to a 60% chance of developing type II as they age (284). This prevalence rate can be reduced to 25% with maintenance of a desirable body weight after delivery (285). With an appropriate weight loss and exercise program, these women can improve their health and lower their risk of developing diabetes.

Nonnutritive Sweeteners

At intake levels of aspartame at least three times the Acceptable Daily Intake (ADI), there is no experimental evidence to suggest risk to the fetus from aspartame. Of the three components of aspartame, aspartate does not cross the placenta at any intake less than very enormous amounts (100 times normal). (286,287) Phenylalanine does cross the placenta, but it is highly unlikely that customary intake could raise fetal levels even close to a neurotoxic range. The maximum maternal plasma phenylalanine concentration occurring after ingestion of aspartame-sweetened foods (assuming normal use levels) would be less than that occurring after ingestion of protein meals and it is highly unlikely customary intakes could raise fetal levels close to a neurotoxic range (286). The amount of methanol ingested is very small (286).

In multigenerational studies, rats were fed diets containing 3% acesulfame K. Although acesulfame K crosses the placenta, reproduction studies, including teratogenicity, show reversibility and no adverse effects on fertility, number of animals per litter, birth weight, gross weight, and mortality. Thus it can be concluded that acesulfame K is also safe to consume during pregnancy (288). Saccharin can cross the placenta to the fetus but there is no current evidence that this compound is harmful.

In lactating women, aspartame administration at 34 mg/kg body weight did not elevate plasma and erythrocyte aspartate or phenylalanine concentrations above normal postprandial values (289).

Lactation

Breast feeding should be encouraged in women with preexisting diabetes or GDM; however, women with type I diabetes mellitus may experience an increased incidence of hypoglycemia within 20–40 min after breast feeding (290). Women with type I diabetes mellitus need to monitor blood glucose levels frequently for prevention of hypoglycemia. Caloric requirements may need to be increased or redistributed to late night snacks if necessary (291).

Although not all women lose weight while breast feeding, it is normal for weight loss to occur because of a greater energy expenditure during lactation. The RDA recommendations are for an additional average allowance of 500 kcal/day throughout lactation (291). Several studies of women without diabetes report adequate lactation on intakes less than the RDA (292) and suggest that food energy utilization and absorption may be more efficient in lactating women.

In one study, women with type I diabetes mellitus were able to establish lactation despite postpartum separation from their infants, delays in the start of breast-feeding, reduced frequency of feeding, increased use of supplemental feedings, and the high rate of cesarean section (290). However, clinicians must recognize the need to provide appropriate guidance and support so that women will not be discouraged by problems with breast feeding.

CHILDREN AND ADOLESCENTS AND DIABETES

— Nutrition recommendations for children with diabetes should be individualized making every effort to maintain health and a sense of normalcy for the child and his/her family.

Since newly diagnosed children may present with weight loss, the dietary prescription begins with estimating caloric requirements to restore and maintain an appropriate body weight and al-

low for normal growth and development. Caloric requirements vary with age, height, weight, and sex, as well as with physical activity, season of the year and growth spurts. Caloric needs change continuously in children and, therefore, food intake should be evaluated every 3–6 months (293). Some parents may want to withhold food, if a child's blood glucose levels are high, as an alternative to adjusting insulin doses. This should be discouraged.

A child's actual daily food intake should be used as the primary indicator of daily caloric needs. Actual intake can be estimated with a 24-h or 3-day diet history. This estimate of total calories can be modified as needed to maintain weight or stimulate appropriate weight gain. If the clinician is unsure of the caloric prescription, caloric requirements can be theoretically determined. Theoretical methods are not considered the methods of choice and should only be used to reinforce the actual estimate. Dietary adherence is thought to be compromised by the use of calculated estimates (293), yet adherence to a dietary plan is strongly correlated to blood glucose control in children under 16 years of age (294).

Once the caloric prescription is determined, the proportion of macronutrients can be modified as necessary. Special care should be taken to ensure that foods containing adequate amounts of protein, iron, calcium and vitamin C are included in the meal plan. Carbohydrate intake should be ample. While obvious sources of sucrose from items such as soft drinks and candy are generally avoided, sucrose-containing foods can be incorporated on a regular basis without detrimental effects if the child and the family so desire. Older children and teens can also be taught how to incorporate sucrose-containing foods into a meal plan without adversely affecting glycemic control. Insulin doses can be adjusted to carbohydrate intake as necessary. Most older children and teens can do well with this approach as it enhances their independence, helps them fit in with their peers, and develops a trusting and caring relationship with the health care team.

Recommendations to maintain a diet low in total fat, with saturated fat less than 10% of calories, are important in promoting overall health and reducing cardiovascular risk factors in children and adolescents. Childhood obesity can be minimized with attention to fat intake and exercise.

Daily eating patterns in children generally require three meals and two or three snacks depending on length of time between meals and physical activity level. The purpose of snacks is to prevent low blood glucose from occurring and to prevent hunger between meals. By using blood glucose records insulin doses can be adjusted to match food intake.

THE ELDERLY AND DIABETES — Aging is frequently associated with deterioration in glucose tolerance (295). This age-associated change in glucose tolerance is considered to be related to age or environmental factors such as increased adiposity, abdominal obesity, or decreased physical activity (296). A population-based study reported the prevalence of type II diabetes increased systematically with age and at each decade prevalence was more common among men than women with 16% of men and 13% of women between the ages of 50 and 89 years having diabetes (297).

In general, there are no differences in the nutrition guidelines for older adults with diabetes mellitus than those recommended for adult patients with diabetes mellitus. However, age-related changes in mental status, functional abilities and sensory function may interfere with the patient's ability to comprehend, understand, and comply with the treatment plans.

Patients with diabetes in poorer glucose control do not perform as well on tasks that involve learning, reasoning, and complex psychomotor performance (298). Cognitive function improves with improved glucose control.

Functional limitations related to the ability to eat, shop for food, and prepare meals must be evaluated when nutrient guidelines are interpreted into food choices. Increased functional limitations related to patient nutritional status are especially marked after 85 years of age. For example, limited ability to prepare meals is found in 4–9% of adults between 65 and 84 years of age and increases to 26% after age 85. The ability to shop for food is limited in 6–15% of adults between the ages of 65 and 84 years and reaches 34% after the age of 85 years (299).

There are limited data on the specific effects of nutrition intervention in the elderly population with diabetes. At present, the management of older adults with diabetes is largely based on clinical impressions and extrapolation of management strategies in younger patients. Patients with diabetes in a nursing home setting were found to be older (average age ~80 years) than an ambulatory diabetic population, had a decreased incidence of obesity (8.5% of nursing home patients were more than 20% over ideal body weight, whereas, 15% were overweight in the ambulatory population), and had glycated hemoglobin significantly lower than the ambulatory population (9% vs. 12%) (300).

In nursing home patients with diabetes, the metabolic impact of prescribed diabetic diets was compared to institutional standard diets (301). As a group, the nursing home residents with diabetes had good diabetic control with glycated hemoglobin levels being ~8%. When these patients consumed the standard institutional diet without restrictions, fasting plasma glucose was significantly elevated but remained clinically acceptable and glycated hemoglobin was not significantly altered. Body weight increased in some of the subjects, but the mean body mass index remained within the ideal range.

Patients with diabetes living in an institutional setting where food availabil-

ity is limited to specified meal times and the meals are planned in a pattern so that similar types and amounts of food are served at each meal, usually do not require dietary modification.

Body weight typically declines slowly after 60 years of age. Thus, many elderly patients are not obese when diabetes is diagnosed. Caloric restriction in these patients may result in malnutrition. Thus, weight reduction is not recommended in the elderly person unless they are more than 20% above desirable body weight (302). Potential changes in the eating habits of the elderly must be balanced against the potential impact on quality of life.

Although age, per se, should not be an excuse for suboptimal control of blood glucose, it is often cautioned that in the elderly the risk of acute complications of hypoglycemia may outweigh the benefits of tight plasma glucose control. Attention to establishing a consistent pattern of food intake and meal spacing may be sufficient to maintain glucose control.

EXERCISE AND DIABETES—

People with diabetes are encouraged to participate in either recreational or competitive physical activities because of potential to improve cardiovascular fitness and psychological well-being and for social interaction and recreation. However, physical activity is not without risks. Hypoglycemia, hyperglycemia, ketosis, cardiovascular ischemia and arrhythmia, exacerbation of proliferative retinopathy, and lower-extremity injury are potential complications of exercise (303–309).

Exercise and Insulin-Treated Diabetes

Although each person with insulin-treated diabetes responds differently to exercise, general guidelines are necessary so individuals can begin and continue to exercise safely. As individuals gain experience with exercise, they can adapt specific issues to meet their needs.

Blood glucose levels before an hour of unplanned exercise or increased physical activity can be used to decide on food or insulin adjustments. For example, if blood glucose is <5.5 mM (100 mg/dl) before exercise, a pre-exercise snack should be eaten. If blood glucose is 5.5–8.3 mM (100–150 mg/dl), exercise can proceed, and, if necessary, a snack eaten afterwards. If blood glucose is >14 mM (250 mg/dl), because of overall poor metabolic control and not because of excessive food intake before exercise, urine should be checked for ketones. If ketones are positive, insulin should be adjusted before exercising to achieve better control as exercise may increase glucose levels in an underinsulinized state.

Blood glucose monitoring before, during (if exercise is of long duration), and after exercise is essential if people with diabetes are to determine their response to exercise. Monitoring records are the most effective tool for determining a particular pattern of response to exercise. Patterns can then be used to adapt food or insulin to the time and amount of exercise planned.

For people exercising on a regular basis, the design of the meal plan and corresponding insulin dosage can be based on usual activity patterns. However, for people exercising more sporadically two strategies are available: anticipating exercise and adjusting insulin doses or increasing food intake.

Guidelines for increasing food intake for exercise should be based on blood glucose levels before and after exercise, the duration of the exercise, the proximity of unscheduled exercise to scheduled meals, the time of day, and the regularity of exercise sessions. If exercise is of a short duration, it often is best to delay consumption of extra food until after exercise, when testing of blood glucose can help determine how much (if any) extra carbohydrate is needed. In general, 10–15 g of carbohydrate should be eaten after or before one hour of moderate exercise. Vigorous exercise and/or exercise of a long duration may require an extra 10–15 g of carbohydrate every half hour. People with insulin-treated diabetes should be careful to not overeat with exercise (310). Along with ingestion of additional carbohydrate in events of long duration, athletes with diabetes may also have to decrease or omit their usual short-acting insulin.

Nutrition and Long-Duration Exercise

An exercising body initially uses carbohydrate as its main fuel source. In long-term exercise bouts, limited muscle and liver glycogen stores may be unable to sustain the rates of energy production needed with 2 h or more of activity (311). Therefore, an exogenous carbohydrate source is necessary to provide fuel during extended periods of activity.

Absorption and utilization of exogenous carbohydrate is determined to some extent by the osmolarity of the stomach's contents. Both glucose concentration of the solution and intensity of exercise retard gastric emptying. Drinks containing 5–10% carbohydrate are absorbed best. Concentrated drinks that exceed 10% carbohydrate can cause gastrointestinal upset (312). Fruit juices and regular soft drinks contain between 10% and 12% carbohydrate and need to be diluted for use. The advantage of diluted fruit juices or "sports drinks" is that they average 6–7% carbohydrate. In events lasting longer than 60 min, diluted fruit juices or "sports drinks" can be ingested, providing both a source of carbohydrate and fluids. In events lasting less than 60 min, plain water is usually the beverage of choice (313).

Nutrition before exercise/competition. The provision of adequate carbohydrate and fluid intake before exercise or competition is important. Two to six hours before an endurance activity (i.e., athletic events lasting longer than 1–2 h) athletes should eat a meal providing 85–200 g carbohydrate(314). The meal should provide mainly carbohydrate with smaller amounts of protein and fat. Five to ten

minutes before the event, fluids containing carbohydrate should be consumed.

Nutrition during exercise/competition. Small carbohydrate feedings during endurance events of more than one hour delay fatigue and prevent hypoglycemia. To prevent hypoglycemia carbohydrate must be ingested during as well as before exercise (315). This can result in continued use of blood glucose with a proportionate slowing of muscle glycogen use. For events of long duration, the limited stores of liver and muscle glycogen, even after supercompensation, are not sufficient to complete the event(316).

Nutrition after exercise/competition. In all athletes, carbohydrate consumption after exercise is required to ensure repletion of muscle glycogen. Muscles will replete glycogen stores to a higher degree when easily digested carbohydrates are consumed as soon as possible after exercise (317,318). For the athlete with diabetes mellitus, this may also prevent postexercise late-onset hypoglycemia.

Exercise and Type II Diabetes

Many individuals with type II diabetes may have been sedentary for many years and, as a result, are frequently deconditioned and unable to exercise continuously for any period of time. A program of gradually increasing exercise sessions, beginning with sessions of 5–10 min, is most successful and safest for this group (303). To improve insulin sensitivity and glycemic control individuals should exercise at least three days per week or every other day. When no exercise is performed for 24 h, glucose tolerance declines significantly (319). Muscle-strengthening exercises may also lead to improved glucose disposal and lipid levels (320).

Exercise appears to be most effective for normalizing blood glucose levels in people with IGT or mild-to-moderate diabetes (i.e., fasting glucose levels <11.1 mM [200 mg/dl]) (321–323). Improvement in glucose tolerance is related to a decrease in insulin resistance and can occur without changes in body weight, body fat content, or $VO_{2\ max}$.

People with type II diabetes usually do not need to eat before exercise. Because they are still producing endogenous insulin their blood glucose levels are not as unstable as the person who produces no endogenous insulin. However, in individuals treated with insulin or oral glucose-lowering medications, the same precautions that apply to people with type I diabetes mellitus should be followed. Blood glucose regulation during exercise in people with type II diabetes mellitus is not significantly different from that in people without diabetes. During mild to moderate exercise, elevated blood glucose levels fall toward normal, but generally do not reach hypoglycemic levels. Exceptions are individuals using insulin or oral glucose-lowering medications who have higher-than-normal insulin concentrations during exercise that may inhibit hepatic glucose production sufficiently and result in hypoglycemia. For people being treated by diet alone there is no need for supplementary food before, during, or after exercise, except when exercise is exceptionally vigorous or of long duration. In this case, extra food may be beneficial just as it is in people who do not have diabetes.

Every effort should be made to encourage people with type II to participate in an exercise program developed to meet their individualized needs and goals. Because of the potential risks for injury of exercise to people with type II diabetes, especially to elderly people, permission for exercise from the patient's physician is advisable.

NEEDS FOR FUTURE RESEARCH

— There are many unanswered important questions related to nutrition and diabetes. The following is a sample of such questions.

1. What is the relationship between plasma lipid levels, dietary lipid composition, or both to cardiovascular disease (CVD) in diabetes? What elements of the dyslipidemic profile are the principal risk factors for accelerated CVD in type I and type II diabetes and by what mechanisms do these factors exert their atherogenic effect?
2. What are the long-term effects of high-carbohydrate or high monounsaturated fat diets on glycemic and lipid control in people with diabetes? Will diets enriched in monounsaturated fats and restricted in carbohydrate promote weight gain and thereby worsen diabetes control in obese type II patients?
3. Will a low fat diet result in sustained weight loss in obese people with diabetes?
4. Do the types or amounts of dietary fat and carbohydrate alter LDL cholesterol composition and metabolism and thus the risk of CVD, independent of changes in the absolute level of circulating LDL cholesterol?
5. How does increasing dietary carbohydrate affect CVD risk in people with diabetes?
6. Do the benefits of protein restriction in people with nephropathy outweigh the adverse effects on body composition? Are the beneficial or adverse effects of protein restriction unique to any particular form or metabolic state of diabetes?
7. What level of protein restriction should be instituted in people with nephropathy and for what duration of time can this be tolerated without posing excessive risk?
8. Under what conditions might a liberal protein intake be beneficial to people with type II diabetes? Are there methods or criteria by which individuals with type II diabetes can be identified who might derive metabolic benefits from an increase in protein intake?
9. Do all forms of protein, animal or vegetable in origin, have adverse effects on diabetic nephropathy or are they unique to animal protein only?
10. What is the role of phosphorus, independent of protein intake, on

the progression of renal disease in diabetes? Should phosphorus be limited, when, and to what level?
11. Do dietary fructose or sucrose have adverse effects on serum lipids?
12. Is the relationship of hypertension in insulin resistance one that is a genetic association or one in which there is a causal effect of hyperinsulinemia?
13. Do dietary antioxidants play a role in preventing microvascular or macrovascular complications of diabetes mellitus?
14. How do meal composition and frequency relate to mean glucose concentrations, postprandial glycemic excursion, and the complications of diabetes mellitus?
15. How does pregnancy alter the glycemic response to macronutrients? How does this differ throughout the day?

It is clear much work remains before many of the above questions have answers. Until that time arrives, the best nutrition advice remains: begin with a nutrition assessment, modify lifestyle factors based on the primary problem and desired outcomes, implement nutrition therapy principles as outlined in the position paper, and monitor the metabolic consequences.

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