

Diabetes Mellitus

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Meal planning is a cornerstone of diabetes management, and nutrition education is an essential component of a comprehensive program of diabetes education for patients and their families.^{1,2} Recommendations for medical nutrition therapy (MNT) of diabetes mellitus have changed repeatedly since the advent of insulin therapy in 1922. In this chapter, we briefly review the history of MNT, the scientific basis upon which the general principles are founded, and recent investigations that implicate dietary factors in the etiology of immune-mediated or type 1a diabetes mellitus. Finally, we discuss the goals of treatment, principles of meal planning, and formulation of an individualized meal plan based on current evidence as recommended by the Food and Nutrition Committee of the American Diabetes Association (ADA).³

PREVALENCE, DEFINITION, CLINICAL DESCRIPTION OF DIABETES MELLITUS

Diabetes mellitus is not a single entity but a group of metabolic diseases characterized by hyperglycemia that is caused by defects in insulin secretion, insulin action, or both.^{4,5} Most cases of diabetes fall into two broad etiologic categories: Type 1 diabetes, caused by an absolute deficiency of insulin secretion, is the most common form in children and adolescents,⁶ whereas type 2 diabetes is the most common form of diabetes worldwide, comprising 80 to 90% of the overall diabetic population, and is caused by a combination of resistance to insulin action and impaired insulin secretion.^{7,8} Type 2 diabetes usually has its onset in middle life and is often associated with abdominal (visceral) obesity and hypertension. Until relatively recently, immune-mediated or type 1a diabetes was the only type of diabetes prevalent among children in the United States, and 1 to 2% of children had type 2 or other rare forms of diabetes. Throughout the developed world, however, the prevalence of type 2 diabetes in youth has increased at an alarming rate^{9–13} and recent reports indicate that 8 to 45% of children with newly diagnosed diabetes have nonimmune-mediated diabetes.¹⁴ The variation in incidence appears to depend on race/ethnicity and sampling strategy. Most of these children have type 2 diabetes, but other types, especially maturity-onset

diabetes of youth (MODY) or monogenic diabetes, are being increasingly identified in adolescents and young adults.¹⁵

Changes in energy intake and expenditure have resulted in an epidemic of obesity in children and adolescents in the United States,¹⁶ which has been temporally associated with an alarming increase in the prevalence of type 2 diabetes in children and adolescents, especially in minority populations, in the past decade. Almost 95% of patients are either overweight or frankly obese.¹⁴

Simple obesity, even in the absence of glucose intolerance, is associated with impaired insulin action on target tissues.^{17–19} In children, body composition is also a major determinant of insulin sensitivity. Adiposity accounts for 55% of the variance in insulin sensitivity in white children.²⁰ An inverse relationship between increasing adiposity and insulin sensitivity also has been demonstrated in black children. As body mass index (BMI) and percent adiposity increase, insulin sensitivity decreases and serum insulin levels increase.²¹

The incidence of type 1 diabetes varies widely among geographic regions, from 0.1 per 100,000 per year in China and Venezuela to 54 per 100,000 per year in Finland.^{22,23} In 2001, there were approximately 155,000 youth with diagnosed diabetes in the United States.⁶ The estimated crude prevalence rate was 1.82 cases per 1,000 youth, being lower for children 0 to 9 years of age (0.79 cases per 1,000 youth) than for those 10 to 19 years of age (2.80 cases per 1,000 youth). Among younger children, type 1 diabetes accounted for at least 80% of diabetes, whereas among older youth the proportion of type 2 diabetes ranged from 6% for non-Hispanic white youth to 76% for American Indian youth.⁶

Type 1a diabetes occurs in genetically predisposed individuals as a consequence of immune-mediated destruction of the insulin secreting β cells of the pancreatic islet.²⁴ The onset of clinically overt diabetes represents a late stage in the process of chronic progressive immune-mediated β -cell destruction and occurs when the majority of β -cells have been destroyed.²⁵ The disease typically presents with the rapid onset of polyuria, polydipsia, and weight loss. The cardinal features are attributable to an absolute deficiency of insulin, which leads to hyperglycemia (the result of decreased uptake and utilization of glucose by

liver, muscle, and adipose tissue and uncontrolled hepatic gluconeogenesis), unregulated release of free fatty acids from adipose tissue, and increased ketogenesis.

Patients with type 1 diabetes depend on exogenous insulin to survive. Insulin normally is secreted in an oscillatory fashion at a low basal rate upon which are superimposed periodic increases in secretion in response to ingestion of food. In contrast, when insulin is injected subcutaneously, it is absorbed from the injection site in a somewhat predictable fashion depending on the specific type and combination of insulins used. Each type of insulin has a characteristic time of onset of action and maximum effect, duration of peak effect, and total duration of action (Table 1). When short- or intermediate-acting insulins are used, meals and snacks have to be timed to match the pharmacodynamics of insulin. Rapid-acting insulin analogs, delivered by injection or via continuous subcutaneous insulin infusion (CSII, insulin pump therapy), significantly reduce the variation in absorption

Table 1 Insulin Preparations Classified According to their Pharmacodynamic Profiles

	Onset of Action (h)	Peak Action (h)	Duration of Action (h)
Rapid-acting*			
Insulin lispro (Humalog [®]) [†]	0.25–0.5	0.5–2.5	≤ 5
Insulin aspart (Novolog [®]) [†]	<0.25	1–3	3–5
Insulin glulisine (Apidra [®]) [†]	<0.25	0.5–1.5	3–4
Short-acting*			
Regular (soluble)	0.5–1	2–4	5–8
Intermediate-acting*			
NPH (isophane)	1–2	2–8	14–24
Long-acting*			
Insulin glargine (Lantus [®]) [†]	2–4	Peakless	20–24
Insulin detemir (Levemir [®]) [†]	2–4	6–8	6–23

*Premixed combinations of intermediate-acting and either rapid- or short-acting insulins are available whose pharmacodynamic profiles have a bimodal pattern reflecting the two insulin components.

[†]Insulin analog developed by modifying the amino acid sequence of the human insulin molecule.

of insulin and permits greater flexibility in matching insulin delivery to glucose absorption after meals and snacks.

Children with idiopathic or nonimmune-mediated type 1 diabetes may be difficult to distinguish from those with immune-mediated diabetes. In the United States, most patients with idiopathic type 1 diabetes are African-American and have so called atypical diabetes mellitus or Flatbush diabetes.^{26,27} They typically have a positive family history of early-onset diabetes in several relatives and in multiple generations. Although insulin may not be required for survival after the episode of acute metabolic deterioration has resolved, blood glucose control usually is poor without insulin therapy and ketoacidosis may recur.

MODY is a form of diabetes caused by monogenic defects in β -cell function inherited in an autosomal dominant pattern.¹⁵ The clinical spectrum of MODY is broad ranging from asymptomatic hyperglycemia to a severe acute presentation. MODY occurs in all racial and ethnic groups, and molecular diagnostic testing is required for specific classification.¹⁵

Individuals with nonimmune-mediated diabetes may have clinical presentations indistinguishable from those of patients with immune-mediated type 1 diabetes. This is especially relevant because as the number of children with type 2 diabetes increases, it is important to classify correctly the cause of the child's diabetes to be able to institute appropriate therapy.

Acute, life-threatening consequences of diabetes are hyperglycemia with ketoacidosis or the hyperosmolar nonketotic syndrome.²⁸ Long-term complications of chronic hyperglycemia include: retinopathy with potential loss of vision, nephropathy leading to hypertension and renal failure, and peripheral and autonomic neuropathy with associated risk of foot ulcers and limb amputation, gastrointestinal, genitourinary, and cardiovascular symptoms.²⁹ Glycation of tissue proteins and other macromolecules and excess production of polyol compounds from glucose are among the mechanisms thought to produce tissue damage from chronic hyperglycemia.³⁰

The initial classification usually is based on the clinical picture at presentation. Children with immune-mediated type 1 diabetes are typically not overweight, have a short duration of symptoms, frequently have ketosis, and approximately 15 to 70% of new onset patients in North America and Europe present with ketoacidosis.³¹ As the childhood population in the United States has become increasingly overweight, the percentage of children with immune-mediated type 1 diabetes who are coincidentally overweight, if not obese, has increased.³² After metabolic stabilization, they may have an initial period of diminished insulin requirement (the honeymoon period) after which they require daily insulin replacement for survival and are always at risk for ketoacidosis. In contrast, approximately 95% of children with type 2 diabetes are either overweight or obese at the time of diagnosis and typically present with glucosuria, absent or mild polyuria and polydipsia,

and a little or no weight loss. Up to 33% have ketonuria at diagnosis, and 5 to 25% of patients classified as having type 2 diabetes present with ketoacidosis. Children with type 2 diabetes usually are more than 10 years old and are in middle to late puberty, 45 to 80% have at least one parent with diabetes and 74 to 100% have a first- or second-degree relative with type 2 diabetes. Children and adolescents of African-American, Hispanic, Asian, American Indian, and Pacific Islander descent are disproportionately represented.^{6,33}

HISTORY OF MNT FOR TYPE 1 DIABETES MELLITUS

Until the advent of insulin therapy in 1922, the only available treatment was diet and exercise. Meal plans severely restricted the total intake of energy and were low in carbohydrate and high in fat.³⁴ Nutrition recommendations were often based on dogma and a limited understanding of the metabolic derangements. After the introduction of insulin, nutrition recommendations changed, but generally involved restriction of carbohydrate to a maximum of 40% of total energy because this was thought to reduce the severity of hyperglycemia.^{35,36} By early in the twentieth century, several studies had shown that a high-carbohydrate meal plan, provided it was restricted in total energy, had beneficial metabolic effects.³⁷⁻³⁹ Change was slow and despite evidence to the contrary, patients continued to be indoctrinated to use a low-carbohydrate, high-fat meal plan until 1971 when the Committee on Food and Nutrition of the ADA formally recommended liberalizing the consumption of carbohydrate.⁴⁰ Sucrose had not been permitted in the meal plans of people with diabetes mellitus; however, considerable scientific evidence has shown that sucrose as part of the meal plan does not impair blood glucose control in people with either type 1 or type 2 diabetes (vide infra).

In 1950, the ADA, the American Dietetic Association, and the United States Public Health Service introduced the exchange system for meal planning to assist patients to choose foods consistent with current nutrition recommendations.⁴¹ The exchange system established portion sizes and grouped foods with similar macronutrient contents. The goal of the system was to enable individuals with diabetes to exchange foods within a group and still receive comparable amounts of macro- and micronutrients. The exchange system was an important advance because it antedated the widespread availability of standardized food composition analysis. Legislation mandating food labeling was not introduced in the United States until 1974. The exchange system has undergone four revisions. The 1976 revision improved the accuracy of the calorie content of foods within the lists and stratified foods by their fat content. The 1986 revision developed a database for nutrient composition and increased the carbohydrate content of fruits from 10 to 15 g, which is closer to their actual carbohydrate content. The 1995

revision grouped carbohydrate-containing foods into a single category, which allowed for greater flexibility in meal planning. This revision also added many new food items (eg, fat-modified, vegetarian, and fast foods) to reflect the US population's changing dietary habits and food choices. The most recent revision, in 2003, liberalized the use of sugar in the diet by establishing a food list titled "Sweets, Desserts and Other Carbohydrates that could be exchanged for Grains, Fruits and Dairy." Another new category, "Combination foods," was added in response to the increased availability of processed foods.

Modifications to the exchange system reflect societal changes in dietary habits, including the increased marketing and consumption of prepared foods, frequency of eating outside the home, and the modification of natural foods to lower their fat and carbohydrate content. The 2003 revision of the exchange system also includes the nutrient profiles of food items reflecting their increasing use by the general public. It is important, however, to recognize that the portion size on food labels may differ from the portion size and carbohydrate content listed in the exchange system. Although some food manufacturers list diabetes exchanges on their products, most do not. Since the introduction of mandatory food labeling, the general public is becoming more accustomed to reading nutrition facts provided on food labels. It is no longer essential, therefore, for people with diabetes to rely on the exchange system as the sole source of information regarding macronutrients for dietary planning.⁴²

Increasingly widespread use of intensive insulin therapy (either multiple daily injections, referred to as basal-bolus or flexible insulin therapy, or CSII using an insulin pump) in combination with carbohydrate counting (utilizing item-specific nutrient content from food labels, readily available reference books and software) allows patients with type 1 diabetes to have considerably more dietary choices and to live more normal lifestyles without compromising glycaemic control. Carbohydrate counting is discussed in more detail later in this chapter.

DIETARY FACTORS IN THE PATHOGENESIS OF TYPE 1a DIABETES MELLITUS

The nature of the interaction among genetic, environmental, and immunologic factors in the etiology of type 1a diabetes remains poorly understood. The genetic predisposition to diabetes is associated with specific DQ and DR alleles in the HLA region of chromosome 6, but the observation of less than 50% concordance for type 1 diabetes in identical twins indicates that environmental factors are also involved. The role of diet, particularly cow's milk protein, in the pathogenesis of human type 1a diabetes is controversial.⁴³ Changes in food, particularly its protein content, influence the onset of diabetes in rodent models of autoimmune diabetes. These

observations have been invoked as evidence that complex natural ingredients in standard human and rodent diets may be involved in the autoimmune-mediated destruction of β cells. It has been suggested that a dietary antigen may play a role as the factor driving the disease process toward clinical type 1 diabetes, and bovine insulin is thought to be an attractive candidate because an immune response, initially induced by bovine insulin, cross-reacts with and may target human insulin in the β -cell.⁴³

Antibodies reactive against a peptide sequence contained within bovine serum albumin (BSA) occur in humans, BB rats, and NOD mice, which cross-react with an islet antigen designated p69. Together with suggestive epidemiological data, these observations have given rise to the controversial theory that dietary cow's milk may contribute to the pathogenesis of type 1a diabetes in susceptible individuals.⁴⁴ Removal of bovine proteins from the diet prevents diabetes in NOD mice.⁴⁵ Some investigators have shown that diets containing cow's milk are diabetogenic in the BB rat; however, this has not been confirmed by others.⁴⁶ Diet clearly has a major diabetogenic effect in BB rats^{47,48} and NOD mice,⁴⁸ but the exact chemical identity of the food diabetogen(s) is still not clear.

Insulin is a crucial autoantigen in type 1 diabetes and is the only β -cell-specific autoantigen in postnatal life.⁴⁹ Insulin autoantigens (IAAs) are the first or among the first autoantibodies to appear when the disease process leading to type 1 diabetes is initiated in young children.^{50,51} Because cow's milk formulas are generally the first and most common dietary source of foreign complex proteins to which infants are exposed in developed countries, the question has been asked whether early exposure to cow's milk formula results in an immune response to bovine insulin (which differs from human insulin in 3 amino acids) present in formulas. Infants who were exclusively breastfed, at least up to the age of 3 months, had substantially lower IgG class antibodies to bovine insulin than those who were exposed to formula before 3 months of age.⁵² Follow-up demonstrated that the IgG class insulin antibodies started to decrease slowly after the age of 3 months as a sign of the development of oral tolerance in those infants who were given formula before the age of 3 months, whereas in those exclusively breastfed, at least to the age of 3 months, the antibody levels increased up to the age of 12 months before titers leveled off. It has been suggested that young children who present with early signs of β -cell autoimmunity lack the capacity to develop oral tolerance to bovine insulin.⁴³ Accordingly, the initial immune response to bovine insulin may be diverted into a response targeting human insulin in such individuals.

Data from three population-based case-control studies on cow's milk intake before the diagnosis of type 1 diabetes have yielded conflicting results. In New South Wales, Australia, cow's milk intake was higher in prediabetic children than in control children,⁵³ and in Finland, a high consumption of

cow's milk in childhood was associated with a more frequent appearance of diabetes-associated autoantibodies and type 1 diabetes in a prospective cohort of initially unaffected siblings of children with type 1 diabetes.⁵⁴ In contrast, a Swedish retrospective survey indicated that the frequency of milk consumption had been lower among children who presented with type 1 diabetes than among unaffected children.⁵⁵

Gluten or other cereal-derived proteins have also been implicated as potential driving antigens in type 1 diabetes. In small intervention studies performed in family members positive for diabetes-associated pancreatic autoantibodies, elimination of gluten from the diet did not significantly affect autoantibody titers, nor did titers change when gluten was reintroduced into the diet.^{56,57} These trials indicated that a gluten-free diet has no effect on the signs of β -cell autoimmunity in first-degree relatives of affected patients.

Early exposure to cereals may increase the risk of seroconversion to positivity for diabetes-associated autoantibodies.^{58,59} Both early (before the age of 4 months) and late (at the age of ≥ 7 months) exposures to cereals (either gluten- or nongluten-containing) were associated with increased risk of β -cell autoimmunity,⁵⁸ whereas another study implied that an increased risk was related to exposure to cereals before the age of 3 months.⁵⁹ It has been speculated that dietary gliadin may trigger intestinal inflammation in subjects with type 1 diabetes (because of the HLA DQ2/DQ8 genotype capable of binding gliadin-derived peptides).⁶⁰ Wheat gliadin could thus affect the immune activation stage of the gut immune system in individuals at genetic risk for type 1 diabetes and modify the autoimmune process involved in β -cell destruction.⁴³

There is seasonal variation in the appearance of the first pancreatic autoantibodies in young children with increased HLA-conferred susceptibility to type 1 diabetes.⁵¹ In addition to seasonal variation in viral infections, there is a marked seasonal variation in the amount of daylight and sunshine hours, especially in Northern Europe, which has the highest incidence of type 1 diabetes in the world.²² Without oral supplementation, sunlight-dependent synthesis of vitamin D in the skin is the most important source of this immunologically active prohormone. The lack of oral vitamin D supplementation in infancy may increase the subsequent risk of type 1 diabetes⁶¹ and vitamin D supplementation during infancy confers partial protection against type 1 diabetes. There is countervailing evidence, however, arguing against the role of vitamin D deficiency as a trigger of β -cell autoimmunity.

A pilot intervention study conducted mainly in Finland has sought to determine whether weaning to hydrolyzed formula over the first 6 to 8 months of life decreases the cumulative incidence of diabetes-associated autoantibodies. Preliminary evidence indicates that such a dietary intervention results in a decreased frequency of positivity for islet cell antibodies and at least one diabetes-associated autoantibody by the age of 6 years.⁶² On the basis

of these pilot data, in 2002 a randomized double-blind trial (Trial to Reduce IDDM in the Genetically at Risk, TRIGR) was initiated to determine whether it is possible to reduce the frequency of diabetes-associated autoantibodies and/or clinical type 1 diabetes by the age of 6 years and the cumulative incidence of type 1 diabetes by the age of 10 years by weaning to a highly hydrolyzed formula over the first 6 to 8 months of life.

Increased weight gain in infancy is a risk factor for type 1 diabetes later in childhood.⁶³ Children with type 1 diabetes are both heavier and taller in infancy⁶⁴ and increased height and weight later in childhood are also a risk factor for type 1 diabetes.⁶⁵ Accelerated linear growth and weight gain result in an enhanced β -cell load and increasing insulin resistance. Rapid growth and weight gain may induce β -cell stress; and active β -cells, as compared to resting β -cells, are more prone to cytokine-induced damage.

At present, no definitive nutrition recommendations can be made for preventing type 1 diabetes; however, increasing BMI is associated with a younger age at diagnosis of type 1 diabetes among those US youth with reduced β -cell function.⁶⁶ Avoiding excessive weight gain during childhood is clearly a desirable goal for many reasons.

GOALS OF MEDICAL NUTRITION THERAPY (MNT)

MNT is the process by which the nutrition prescription is individually tailored for the patient with diabetes based on medical, lifestyle, and personal factors and is an integral component of diabetes management and diabetes self-management education.³ The goals of MNT for children and adolescents are shown in Table 2.

The Diabetes Control and Complications Trial (DCCT) and the succeeding observational follow-up of the DCCT cohort in the Epidemiology of Diabetes Interventions and Complications (EDIC) study confirmed that long-term maintenance of blood glucose levels near to normal delays or prevents the microvascular complications of type 1 diabetes,⁶⁷ and the benefits of intensive treatment extend well beyond the period of its most vigorous implementation.⁶⁸ The DCCT also demonstrated that intensive management of type 1 diabetes is associated with an increased risk of severe hypoglycemia⁶⁹ and a tendency to gain weight.⁷⁰ Obesity (greater than 120% of ideal body weight or BMI >95th percentile) was more common in intensively treated patients.⁷⁰ Weight gain in intensively treated patients with type 1 diabetes has been primarily attributed to the elimination of glucosuria (70% of the weight gain) and a 5% reduction in daily energy expenditure (30% of the weight gain).⁷¹ Recurrent symptomatic hypoglycemia requiring oral carbohydrate to restore euglycemia also contributes to the tendency to gain weight in some intensively treated patients.

Our ability to help patients achieve these objectives is still limited by the imperfection of insulin

Table 2 Goals of Medical Nutrition Therapy for Diabetes Mellitus

Goals that apply to all persons with diabetes	<ol style="list-style-type: none"> 1. Attain and maintain optimal metabolic outcomes including <ol style="list-style-type: none"> a. Blood glucose levels in the normal range or as close to normal as is safely possible. b. A lipid and lipoprotein profile that reduces the risk for vascular disease c. Blood pressure levels in the normal range or as close to normal as is safely possible 2. To prevent, or at least slow, the rate of development of the chronic complications of diabetes by modifying nutrient intake and lifestyle. 3. To address individual nutrition needs, taking into account personal and cultural preferences and willingness to change 4. To maintain the pleasure of eating by only limiting food choices when indicated by scientific evidence
Goals specific to type 1 diabetes	To provide adequate energy to ensure normal growth and development, integrate insulin regimens into usual eating and physical activity habits
Goals specific to type 2 diabetes	To facilitate changes in eating and physical activity habits that reduce insulin resistance and improve metabolic control
Goals specific to patients on insulin or insulin secretagogues	To provide self-management education for treatment and prevention of hypoglycemia, acute illnesses, and exercise-related blood glucose problems
Adapted from reference 3.	

Table 3 American Diabetes Association Recommended Glycemic Targets²

	Blood Glucose Goal Range (mg/dL)		HbA1c %
	Preprandial	Bedtime	
Toddlers and preschool age children (<6 yr)	100–180	110–200	<8.5
School age children (6–12 yr)	90–180	100–180	<8%
Adolescents and young adults	90–130	90–150	<7.5%*
*Ideal is <7% if this can be achieved safely without frequent or severe hypoglycemia.			

replacement, which does not precisely mimic normal physiology, the lack of methods to continuously monitor blood glucose concentrations, and the risk of severe hypoglycemia when glucose levels are near to normal but insulin delivery is not regulated by a closed-loop feedback system.⁷² Target blood glucose and hemoglobin A1c goals vary by age, primarily because young children are frequently unable to recognize hypoglycemia and are at greater risk for severe hypoglycemia (Table 3).²

The child with diabetes and his or her family must assume primary responsibility for daily management, whereas the role of the healthcare professional is that of educator, advisor, and motivator. Even modest objectives are not achievable in all patients because educational, psychosocial, and economic factors make it impossible for some children and their families to perform daily *all* the tasks that comprise a comprehensive treatment program.^{73,74} The goals of therapy can, however, be accomplished in most patients most of the time by the use of flexible insulin regimens and dosage algorithms, frequent self-monitoring of blood glucose, individualized meal planning, and by educating older children and adolescents leading to patient empowerment and independent decision-making.

GENERAL PRINCIPLES OF NUTRITION MANAGEMENT

There is a little or no research on the nutrient requirements of children and adolescents with either type 1 or type 2 diabetes nor is there

evidence that their nutritional needs differ from those of healthy children. Therefore, nutrient recommendations are based on the requirements of healthy children and adolescents.^{75–79} Meal plans for children with diabetes should begin with the key recommendations outlined in The Dietary Guidelines for Healthy Americans 2005.⁸⁰ This important document provides guidelines that should serve as the foundation for nutrition management within each of the following areas: adequate nutrients within calorie needs, weight management, physical activity, food groups that should be encouraged, fats, carbohydrates, sodium and potassium, alcoholic beverages, and food safety. The guidelines address issues specific to children and may require further definition as they relate to diabetes management. For example, the guidelines suggest that carbohydrate choices should include fiber-rich fruits, vegetables and whole grains, and added sugar should be minimized.⁸⁰ People with diabetes treated with insulin or insulin secretagogues must be aware of the total amount of carbohydrate eaten at a meal.

Energy

Clinical dietitians frequently use equations such as the Schofield, WHO, Harris–Benedict, or RDA for age to calculate energy requirements (see Appendix II, “Nutritional Requirements”). The following simpler equation can be used to provide a crude approximation for children up to 12 years of age:

Basal daily needs for all children = 1,000 kcal;
for boys add to the basal needs
125 kcal × age in years,
and for girls add 100 kcal × age in years.

Add up to 20% more kilocalories for very active children.

It is important to assess a child’s usual dietary intake before the onset of symptomatic hyperglycemia to assist with the formulation of the initial meal plan. Because energy requirements change with age, physical activity, and growth rate, an assessment of height, weight, and energy intake is recommended every 3 to 6 months.⁸¹ Good metabolic control is essential for normal growth and development.⁸²

Growth is an excellent indicator of the adequacy of energy intake, which should be evaluated by regularly plotting weight and stature on the Centers for Disease Control and Prevention (CDC) pediatric growth charts (<http://www.cdc.gov/growthcharts>). If growth is not optimal, the diet should be reviewed and, in patients with type 1 diabetes, screening for symptoms and signs of inadequate insulin delivery,⁸³ hypo- or hyperthyroidism, celiac disease, and adrenal insufficiency is warranted.

For overweight and obese children and adolescents with either type 1 or type 2 diabetes, energy is reduced to arrest weight gain while supporting normal linear growth and physical development.

Distribution of Macronutrients

Many studies have attempted to determine the optimal mix of macronutrients that should comprise total energy intake. The general consensus is that a single prescription does not exist.³ The Dietary Reference Intake (DRI) is cited as a guideline that should be used to formulate the initial prescription. The DRI reports suggest a mix of 45 to 65% of calories from carbohydrate, 20 to 35% from fat, and 10 to 35% from protein. Once the nutrition prescription has been determined, the proportion of macronutrients can be modified, as necessary, according to blood glucose and plasma lipid goals and requirements for growth and development.

Carbohydrate

Control of blood glucose in an effort to achieve normal or near-normal levels is the primary goal of diabetes management. Because dietary carbohydrate is the major determinant of postprandial glucose levels, food and nutrition interventions that reduce postprandial blood glucose excursions are important.⁸⁴ Blood glucose concentrations after a meal are primarily determined by the rate of appearance of glucose in the blood (digestion and absorption) and its disappearance from the circulation. The normal insulin secretory response maintains blood glucose in a narrow range; however, in patients with diabetes, defects in insulin secretion, insulin action, or both impair regulation of postprandial blood glucose concentrations in response to dietary carbohydrate. The

amount, type, and source of carbohydrates in foods, as well as the dose and time interval between administration of rapid- or short-acting insulin and consumption of the meal influence postprandial glucose levels.

The diet should include carbohydrates from fruits, vegetables, whole grains, legumes, and low-fat dairy products.³ Dietary dogma in diabetes had been to avoid simple sugars and replace them with complex carbohydrates. This belief was based on the assumption that simple sugars are more rapidly digested and absorbed than starches and would lead to more pronounced hyperglycemia. During the past two decades, the blood glucose responses to various carbohydrate-containing foods have been extensively investigated and there is little scientific evidence to support this notion. Although insulin and glucose-lowering medications for type 2 diabetes can adequately control the glycemic impact of added sucrose and other sugars, their impact on total caloric intake suggests that moderation in the use of added sugars is still warranted.

Numerous factors influence the glycemic response to food, including the amount of carbohydrate,⁸⁵ the type of sugar (glucose, fructose, sucrose, lactose),⁸⁶ the nature of the starch (amylose, amylopectin, resistant starch),⁸⁷ cooking and food processing (degree of starch gelatinization, particle size, cellular form),⁸⁸ and food structure,⁸⁹ as well as other food components (fat and natural substances that slow digestion—lectins, phytates, tannins, fiber, and starch-protein and starch-lipid combinations).^{90,91} Furthermore, the glycemic response to a particular carbohydrate food is dependent on whether it is eaten alone or as part of a mixed meal.⁹² Fasting and preprandial blood glucose concentrations,^{93–96} the severity of glucose intolerance,⁹⁷ and the second meal effect⁹⁸ are other factors that can affect the glycemic response to foods.

The glycemic index (GI), proposed in 1981 as an alternative system for classifying carbohydrate-containing food, measures the glycemic response after ingestion of carbohydrate. Carbohydrates were previously classified only on the basis of their chemical structure: sugars, starches, or fiber. GI is defined as the incremental area under the plasma glucose response curve over 2 hours after consumption of a standard amount (usually a 50 g portion) of carbohydrate from a test food relative to that of a control food, either white bread or glucose.⁹⁹ The glycemic and hormonal responses to a large number of ingested carbohydrates have been systematically examined and their GIs defined.^{99–105} As a result of these studies, the validity of the dogma that ingestion of simple sugars causes a rapid and pronounced rise in blood glucose, whereas complex carbohydrates produce only modest increments in blood glucose concentration began to be disputed.¹⁰⁶ It is now clear that there is a wide spectrum of biologic responses to different complex and simple carbohydrates, with so much overlap that they cannot be simply classified into two distinct groups. Even a single food produces a substantially different glycemic response when it is

prepared in different ways. For example, a pureed apple causes a greater increment in blood glucose than does an apple eaten whole.¹⁰⁷ Similarly, ground rice or rice flour causes a greater glycemic and insulinemic response than does whole cooked rice,¹⁰³ and cooking increases the glycemic response to starch.¹⁰⁸ Blood glucose levels after ingesting wheat flour in the form of pasta are lower than after an equal amount of wheat flour in bread.¹⁰⁹ Thus, the form of a carbohydrate-containing food, in addition to its chemical composition, influences its glycemic effect. The effect of food form alters the rate of digestion and absorption and, therefore, affects postprandial glycemia. Fruits (eg, apples GI 40, Sunkist oranges GI 48, peaches GI 42, pears GI 38 vs. watermelon GI 72) and milk (eg, whole milk GI 27, skim milk GI 32)^{110–112} cause a lower glycemic response than most starches, and sucrose causes a glycemic response similar to that of bread, rice, and potatoes. In general, most refined starchy foods eaten in the United States have a high GI, whereas nonstarchy vegetables, fruits, and legumes tend to have a low GI (Table 4).

Use of the GI of a food alone ignores the glycemic response if the portion is other than 50 g. The concept of glycemic load was introduced to describe more accurately the postprandial glycemic response to a food when eaten in a realistic portion size. The glycemic load is determined by multiplying the GI of a food by the amount (grams) of carbohydrate in the portion size actually consumed. For example, 12 medium carrots contain 50 g of carbohydrate. Whereas carrots have a high GI, a usual portion

would not significantly increase the glycemic load of the meal. The glycemic load of a meal reflects the sum of the contributions of each food that is eaten.

The practical usefulness of low-GI diets in type 1 diabetes is controversial and there are limited data available in children. A meta-analysis of randomized controlled trials (virtually all adult studies) shows that choosing low-GI foods in preference to conventional or high-GI foods has a small (0.4% decrement in HbA1c) but clinically beneficial effect on medium-term glycemic control in patients with diabetes.¹¹³

Using a low-GI diet as the only approach to nutrition management is not recommended, but substituting lower-GI foods of approximately equal carbohydrate and nutritional value is a useful approach to improve glycemic control, especially in patients with pronounced postprandial hyperglycemia. When making recommendations regarding GI and glycemic load, it is important to focus on encouraging low- and moderate-GI foods from the food groups recommended in the Dietary Guidelines for Americans 2005 report: grains, fruits, vegetables, and low-fat dairy products.⁸⁰ For example, a chocolate bar (moderate GI) would not be a suitable substitute for French fries (high GI); whereas oven “fried” sweet potato (low GI) would be an appropriate substitute. A food with a low GI and glycemic load that is high in fat and low in vitamins and minerals is not a healthy substitute unless it is replacing an item of equally poor nutritional value.

Concerns about the acceptance of lower-GI foods have been expressed as a reason not to introduce a low-GI diet. At a diabetes summer camp for 7- to 16-year-old children, satisfaction ratings were equal when dinner and snacks containing lower-GI foods replaced standard food choices; however, they were lower (although within the acceptable range) at breakfast and lunch.¹¹⁴ Children 8 to 13 years old who received low-GI dietary advice did not report more limited food choices or compromise their macronutrient intake as compared to children who continued to use a traditional carbohydrate-exchange diet.¹¹⁵

Once patients have mastered the concept of matching carbohydrate intake to insulin, or distributing carbohydrate throughout the day to optimize glycemic control, we recommend providing additional guidance on how to lower the GI of the diet.

Fiber

Fiber refers to the portion of a plant that is not digestible in the human small intestine. It was thought to have no nutritive value and in the past century, the quantity of fiber in the American diet has declined. During the last two decades, however, considerable attention has been focused on the various plant fibers because of their influence on gastrointestinal physiology. It is now known that fiber markedly influences the digestion, absorption, and metabolism of many nutrients.^{116–119} Cellulose, lignins, and certain hemicelluloses, the fibers present in vegetables and grains are usually insoluble in water. Many fruits and certain legumes are rich in water-soluble fibers such as

Table 4 Glycemic Indexes of Common Foods

Low GI (<55)	Moderate GI (55–70)	High GI (>70)
Apples, apple juice, bananas, grapefruit, kiwi, mango, oranges, peaches, pears, plums, grapes, strawberries	Pineapple, raisins, cantaloupe, cranberry juice cocktail	Watermelon
Whole-grain pumpernickel	Hamburger bun, whole wheat pita bread, stoned wheat thins, oatmeal cookies, plain	Bagel, white bread
Sourdough wheat bread, sourdough rye bread, 100% stone-ground wheat bread		Saltines, rice cakes
Sweet potato or yam Corn		Mashed potato, baked potato
Oatmeal, from steel-cut oats, milk, yogurt		Rice Krispies, Cheerios

pectins, guar, and storage polysaccharides. When carbohydrate is ingested together with soluble fiber, the resulting increase in blood glucose concentration is less than when carbohydrate is ingested alone.¹²⁰ A large test meal containing carbohydrate, protein, and fat normally causes significantly less postprandial glycemia when the meal is supplemented with guar.¹²¹ The attenuated increment in blood glucose concentration that occurs when guar is added to ingested carbohydrate is due to delayed rather than incomplete absorption of carbohydrate.¹²²

Short-term studies using large amounts of fiber (>30 g/d) in small numbers of suboptimally controlled type 1 diabetic subjects suggested a positive effect of fiber on glycemia.¹²³⁻¹²⁶ However, in subjects using intensive insulin therapy, 56 g of fiber had no beneficial effect on glycemic control.¹²⁷ Subjects receiving ≥ 2 injections per day and with baseline HbA1c levels of 7 to 10% were randomized to either a high-fiber (50 g/d) low-GI diet, or a low-fiber (15 g/d) high-GI diet for 24 weeks. The high-fiber diet significantly decreased mean daily blood glucose concentrations, number of hypoglycemic events, and, in the subgroup of patients compliant with diet, HbA1c levels, but had no beneficial effect on cholesterol, HDL cholesterol, or triglyceride concentrations.¹²⁸ Conversely, a cross-sectional analysis of dietary fiber in type 1 diabetes patients enrolled in EURODIAB IDDM showed that a higher intake of fiber was independently associated with higher levels of HDL cholesterol in both men and women, and lower LDL cholesterol levels in men but not in women.¹²⁹ No substantial differences were observed between soluble- and insoluble-fiber intakes.

These observations suggest that inclusion of plant fiber in the diet may benefit patients with diabetes by diminishing postprandial hyperglycemia. Furthermore, certain soluble plant fibers significantly reduce serum cholesterol concentrations and decrease fasting serum triglyceride levels in diabetics with hypertriglyceridemia.¹¹⁸ The mechanism responsible for the effects on cholesterol and triglycerides is not known.

People with diabetes are encouraged to choose a variety of fiber-containing foods such as legumes, fiber-rich cereals (≥ 5 g fiber per serving), fruits, vegetables, and whole grain products because they provide vitamins, minerals, and other substances important for good health.³ Patients should be encouraged to achieve the fiber intake goals set for the general population of 14 g per 1,000 kcal.⁷⁵ Dietary fiber recommendations for children with diabetes are the same as for nondiabetic children. Children older than 2 years should have an amount of dietary fiber equal to or greater than their age plus 5 g/d.¹³⁰

Protein

Protein requirements are not increased when diabetes is well controlled; therefore, children with diabetes should follow the RDA guidelines suggested for children without diabetes. The physiologic requirement, determined by the amount of

protein necessary to sustain normal growth, is based on ideal weight-for-height and varies with age, being highest in infancy and early childhood. The protein requirement (per kilogram body weight) of infants, children, and adolescents is higher than that of adults. It is recommended that for individuals with diabetes and normal renal function, the protein content of the diet should be the same as for nondiabetic children and adolescents. The 2005 DRIs for children 1 to 3 years old is 5 to 20% and for children 4 to 18 years of age 10 to 30%, respectively, of total energy intake. The recommendation for adults with diabetes is to maintain protein intake at less than 20% of energy needs. Any ingested protein that exceeds the body's requirement for new protein synthesis enters the carbon pool and is converted into either glucose or fat.

Dietary protein should be derived from both animal and vegetable sources; however, the source of protein is not especially important provided that all the essential amino acids are available in adequate amounts. Good quality protein sources include meat, poultry, fish, eggs, milk, cheese, and soy. To lower the consumption of saturated fat, less protein should be derived from red meat, whole milk, and high-fat dairy foods than is customary in the United States. With the onset of nephropathy, lower intakes of protein should be considered. A protein intake similar to the adult RDA (0.8 g/kg body weight per day), $\sim 10\%$ of daily energy, is sufficiently restrictive and is recommended for individuals with evidence of nephropathy. With protein intake of 0.6 g per kg per day, evidence of protein under-nutrition has been reported.¹³¹

Fat

Cardiovascular disease (CVD) is more prevalent among patients with type 1 and type 2 diabetes than among those without diabetes.^{132,133} Type 1 diabetes is associated with at least a 10-fold increase in CVD as compared with an age-matched nondiabetic population.^{133,134} Its major clinical consequences are atherosclerosis of the coronary, cerebral, and lower extremity large arteries. In Westernized populations, atherosclerotic vascular disease is the major cause of mortality and morbidity in patients with diabetes. About 75% of patients with diabetes in Europe and North America die of coronary heart disease. This terrible toll is not inevitable. In other parts of the world, patients with diabetes who escape atherosclerosis have low serum cholesterol levels and live on diets that are usually high in starch and have a low content of saturated fat. Cognizance of these facts compels us to focus attention on the amount and type of fat consumed by persons with diabetes. The recent DCCT/EDIC report on the effect of intensive diabetes therapy on CVD in type 1 diabetes showed that higher total and LDL cholesterol levels at baseline were significantly associated with the occurrence of CVD independent of treatment assignment.¹³⁵

To reduce the risk for CVD, the goal is to limit the consumption of saturated fatty acids,

Table 5 Practical Guidelines for Implementing the American Heart Association Dietary Guidelines

- Reduce added sugars, including sugar-sweetened drinks and juices
- Use canola, soybean, corn, safflower oil, or other unsaturated oil in place of solid fats during food preparation
- Use fresh, frozen, and canned vegetables and fruits and serve at every meal; be careful with added sauces and sugar
- Introduce and regularly serve fish as an entrée
- Remove the skin from poultry before eating
- Use only lean cuts of meat and reduced-fat meat products
- Limit high-calorie sauces such as Alfredo, cream sauces, cheese sauces, and hollandaise
- Eat whole-grain breads and cereals rather than refined products, read labels and ensure that "whole grain" is the first ingredient on the food label of these products
- Eat more legumes (beans) and tofu in place of meat for some entrees
- Breads, breakfast cereals, and prepared foods, including soups, may be high in salt and sodium and/or sugar; read food labels for content and choose high-fiber, low-salt/low-sugar alternatives

trans fatty acids, and cholesterol, and to increase intake of omega-3 fatty acids and unsaturated fat. These recommendations are consistent with those of the Dietary Guidelines for Americans 2005, the American Academy of Pediatrics, and the American Heart Association.¹³⁶ Saturated and *trans* fatty acids are the principal dietary determinants of plasma LDL cholesterol. In nondiabetic individuals, reducing the consumption of saturated and *trans* fatty acids, and cholesterol decreases plasma total and LDL cholesterol levels. Reducing saturated fatty acids may also reduce HDL cholesterol and, importantly, the ratio of LDL cholesterol to HDL cholesterol is not adversely affected (Table 5).

Children and adolescents with well-controlled type 1 diabetes are not at high risk for developing lipid abnormalities, but should be screened and monitored according to guidelines issued by the National Cholesterol Education Program (NCEP) and by the American Academy of Pediatrics.¹³⁷ For children and adolescents who are growing and developing normally and have normal plasma lipid levels, it is recommended that less than 10% of energy comes from saturated fat, the daily intake of cholesterol should be less than 300 mg per day, and consumption of *trans* fatty acids should be minimal. Total fat should be reduced in the obese child to reduce total energy consumption. The consumption of saturated fat can be reduced by drinking less whole milk and more low-fat milk and eating less red meat and high-fat dairy foods, and by eating more poultry, fish, and vegetable proteins.

The NCEP Step II diet guidelines should be implemented in the patient with elevated LDL cholesterol (>100 mg/dL).¹³⁸ The guidelines recommend that total fat represent no more than 30%

of total calories, less than 7% of calories are from saturated fat, and dietary cholesterol is 200 mg/d.

Recent survey data of a representative sample of US youth with diabetes (89% with type 1) show that percent consumption of energy from total fat is 37 to 38% across subgroups of age (10 to 14 years, >15 years). Only 6.5% of the cohort met ADA recommendations of <10% of energy from saturated fat and less than 50% met recommendations for total fat, vitamin E, fiber, fruits, vegetables, and grains. It is evident that there is a critical need for improvement in dietary intake in youth with diabetes.¹³⁹

Sodium

The average consumption of salt in populations correlates well with the prevalence of hypertension¹⁴⁰ and a high intake of sodium causes high blood pressure in susceptible individuals. Most processed foods contain considerably more sodium than similar foods prepared from raw ingredients. Sodium is present in the form of sodium chloride; it may also be present as sodium glutamate, added to enhance flavor, or as sodium nitrite included as a preservative. The leaders in sodium content are take-out or “fast foods,” which account for an ever-increasing proportion of the US diet. It has been estimated that 70% of the sodium in the US diet is derived from prepared foods.

Sodium consumption in persons with diabetes should be the same as for the general population, between 2,400 mg¹⁴¹ and 3,000 mg daily.¹⁴² A reduction in salt intake lowers blood pressure in both normotensive and hypertensive individuals. In nondiabetic adults, a reduction in salt consumption lowers systolic blood pressure by 4 to 5 mm Hg^{143,144}; therefore, for diabetic patients with hypertension (in children and adolescents defined as the average of three separate systolic and/or diastolic blood pressures >95th percentile for age, sex, and height), a reduction in salt intake to 2,400 mg daily is recommended. Note, however, that sodium restriction has not been tested in the diabetic population in controlled clinical trials.¹⁴⁵ To achieve these goals, patients should be taught to reduce their use of table salt and decrease their consumption of convenience foods.

MEDICAL NUTRITIONAL THERAPY

A registered dietitian skilled in MNT is the diabetes team member who plays the leading role in providing nutrition care; however, it is important that all members of the diabetes team, including physicians and diabetes nurse educators, be knowledgeable about MNT and help to support its implementation.³

MNT begins with a nutrition history and assessment, taking heed of the ethnic, religious, and economic factors that pertain to the individual patient and his or her family. Family eating patterns, meal preparation methods, and nutrition beliefs must be identified. The initial evaluation of eating styles and nutrition beliefs within the family assist the dietitian to determine how best

to begin educating the patient and family to achieve nutrition guidelines for optimal management of the disease. The first priority is to teach families how diet and insulin (or other medications) affect blood glucose levels. Thereafter, it is important to teach dietary habits that will help patients reduce their risk of CVD.

The way meals are planned should be understandable to the patient and family. A great deal of lay literature about diet is available to the general public; some accurately describes evidence-based research, but much does not. Families that have received and applied advice consistent with the recommendations of the American Heart Association, American Academy of Pediatrics, and Dietary guidelines for Americans 2005 will benefit by an approach that builds on their current practices.

Changing how we think about our food and beverage selections on a day-to-day basis is, perhaps, the most difficult aspect of diabetes management. Before discovering that a member of the family has diabetes, families may give significant thought to their food purchases, but planning when and the precise amount to be eaten at a meal are not common in the general population.

Patients with both type 1 and type 2 diabetes have the same goals; namely, to achieve and maintain target blood glucose and glycated hemoglobin levels (Table 2). The initial focus of MNT, however, differs between the two major types of diabetes.¹⁴ Children with type 2 diabetes typically are obese at presentation and there is great emphasis on weight loss, limiting caloric intake, and distributing meals evenly throughout the day. Even modest weight reduction alone increases sensitivity to insulin and improves fasting and postprandial plasma glucose levels.¹⁸ Similarly, moderate calorie reduction decreases plasma glucose levels in type 2 diabetes.^{146,147} Studies in adults provide evidence that structured, intensive lifestyle pro-

grams involving participant education, individualized counseling, reduced energy and fat intake (~30% of total energy), regular physical activity, and frequent participant contact are necessary to produce long-term weight loss of 5 to 7% of starting weight.¹⁴⁸ Accordingly, lifestyle changes that lead to weight loss are the cornerstone of therapy in patients with type 2 diabetes. In contrast, in the child with type 1 diabetes, the primary goal is to match insulin delivery and carbohydrate consumption to achieve blood glucose levels in the age-specific target range.

Considerations When Formulating a Meal Plan For Type 1 Diabetes

Newly diagnosed children usually present with weight loss and are hungry for the first few days to weeks after starting insulin therapy. Therefore, the child’s initial dietary allowance is determined by appetite. The meal plan should be formulated to restore and then maintain an appropriate body weight and allow for normal growth and development. Energy requirements vary with age, height, weight, and gender, as well as with physical activity, season of the year, and stage of puberty. If the child’s usual food intake cannot be adequately determined, the nutrition prescription can be based on a formula for estimating energy and protein allowances (see above under Energy).

The daily eating patterns of young children generally include three meals and two or three snacks, depending on the length of time between meals, the age of the child, and level of physical activity. Although their daily energy intake is relatively constant over time, young children adjust their energy intake at successive meals.^{149,150}

Dietary strategies principally are determined by the patient’s insulin replacement regimen (Table 6). Rapid-acting insulin combined with

Table 6 General Approaches to Meal Management

Type 1 diabetes Split-mixed insulin regimen	<ul style="list-style-type: none"> • Three meals and 2 or 3 snacks daily • Meals and snacks spaced 2–3 hours apart • Consistent carbohydrate intake • Snack before bed to decrease risk of overnight hypoglycemia • Meal times consistent from day-to-day • Continued education and assessment of readiness to change lifestyle to achieve Heart Healthy diet
Basal–bolus or insulin pump therapy	<ul style="list-style-type: none"> • Carbohydrate content can vary • Must accurately count carbohydrate and match insulin dose to predetermined insulin:carbohydrate ratio • Should eat at least 3 meals daily • Snack before bed to decrease risk of overnight hypoglycemia • Should not eat less than a predetermined amount of carbohydrate per day • Continued education and assessment of readiness to change lifestyle to achieve Heart Healthy diet
Type 2 diabetes	<ul style="list-style-type: none"> • Meal plan to assist with evenly spaced carbohydrate intake and increased emphasis on reducing calories to promote weight loss
<p>Age related issues: Children’s activities often differ on weekdays compared to weekends and holidays, and appropriate allowance must be made for these differences. The meal plan must take into account the child’s school schedule, early or late lunches, gym classes, and after-school physical activity.</p>	

intermediate-acting (neutral protamine Hagedorn, NPH) insulin is often used at the outset because glycemia can be controlled with two or three daily injections: typically at breakfast, dinner, and/or bedtime. Rapid-acting insulin and intermediate-acting insulin are released from the subcutaneous injection site into the circulation reaching peak effects 1 to 2 hours and 4 to 10 hours after injection, respectively. A meal plan is formulated to provide a consistent carbohydrate intake because the breakfast dose of NPH will have its peak effect between lunch and dinner and the dose is based on the glycemic load provided by lunch and a mid-afternoon snack. Therefore, over- or undereating carbohydrate will result in hyper- or hypoglycemia, respectively. The use of fixed doses of rapid- or short-acting together with intermediate-acting insulin is not ideal for families whose lifestyles do not allow for consistent meal times. Similarly, very young children whose appetites are highly variable present significant challenges when rigid meal timing and constant carbohydrate intake are required to match a fixed dose of insulin. Using an insulin regimen that includes administering rapid-acting insulin after the meal provides a practical solution to this problem.¹⁵¹

Because the energy needs of children continuously change, food intake should be re-evaluated at 3- to 6-month intervals depending on the age of the child.^{152,153} Whenever the quantity of carbohydrate in the meal plan is changed, the insulin dose must be reviewed. If a pattern of premeal hyperglycemia is identified, it should be treated by adjusting the insulin dose and not by withholding food. Trends in hunger and satiety should be tracked. Additional servings of protein- and fat-rich foods should not be routinely offered to satisfy hunger, and force-feeding carbohydrate to achieve meal plan goals is discouraged. Instead, the meal plan should be adjusted when an abnormal trend in growth is observed, when the child frequently complains of being too full to eat the prescribed meal, or frequently complains of being hungry after meals. Occasional hunger between meals need not lead to a major dietary change.

In general, more physiologic and flexible insulin-replacement strategies are preferred. These employ CSII using an insulin pump or multiple daily doses of rapid-acting insulin in conjunction with a long-acting (basal) insulin. Meal plans need not be rigid with respect to the time meals are eaten or the amount of carbohydrate that has to be consumed at each meal. Patients adjust the premeal insulin dose of rapid-acting insulin to match (“cover”) the carbohydrate content of the meal. There is a strong relationship between the premeal insulin dose and the postprandial response¹⁵⁴; therefore, patients who use these regimens are encouraged to perform pre- and postprandial blood glucose monitoring to optimize prandial insulin doses. Variation in the carbohydrate content of meals does not modify the patient’s basal insulin requirement. The patient’s blood glucose level prior to the meal and the carbohydrate content of

the meal are used to determine the mealtime insulin dose. In contrast to patients receiving intermediate-acting insulin, patients who use basal-bolus insulin therapy or an insulin pump should not have excessive insulin action between meals and, therefore, are not obliged to eat snacks, unless they engage in strenuous or prolonged physical activity. They must, however, inject rapid-acting insulin or give a bolus of insulin via the pump each time they consume carbohydrate.

The concept of total carbohydrate intake determining the premeal insulin dosage is supported by data from the DCCT. Individuals who adjusted their premeal insulin dosages based on the carbohydrate content of their meals had a 0.5% lower HbA1c level than those who did not adjust premeal insulin doses.¹⁵⁵ It should, also, be noted that day-to-day consistency in the amount and source of carbohydrate is associated with lower HbA1c levels in individuals who receive fixed doses of short-acting (or rapid-acting) and intermediate-acting insulin.¹⁵⁶

Formulation of a Meal Plan for Type 2 Diabetes

Treatment aims for type 2 diabetes are to normalize fasting and postprandial blood glucose and hemoglobin A1c values, to identify and treat associated comorbidities such as hypertension and hyperlipidemia,¹ and to minimize the risk of acute and chronic complications associated with diabetes. Early onset of type 2 diabetes is associated with a high incidence of diabetic nephropathy¹⁵⁷ and vascular disease may already be present at diagnosis.^{158,159} The early age of onset of type 2 diabetes in children can be expected to increase the risk of microvascular complications, which are directly related to the duration of diabetes and hyperglycemia.

MNT for patients with type 2 diabetes emphasizes healthy eating to support optimal growth and weight management. A careful history of an individual’s usual eating and physical exercise habits can assist in determining the best approach to formulating a meal plan. Patients and their family members should be encouraged to adopt healthy eating habits. Guidance should be offered on behavior-modification strategies to change their lifestyle, decrease their consumption of high-energy, high-fat foods,¹⁶⁰ and incorporation of daily aerobic physical activity in their lives. In order to increase children’s physical activity, the amount of time devoted to sedentary activities such as viewing television, playing video games, and sitting in front of a computer, must be strictly limited.^{161,162} Recommendations outlined by the American Heart Association (Table 5) that include but are not limited to simple suggestions such as eliminating sugar-containing sodas and juices, and decreasing consumption of calorie-dense “fast foods” and high-fat snacks, can improve blood glucose levels and achieve weight loss. Patients who have no regularly

scheduled meals and eat large amounts of food toward the end of the day can also see improvements in glycemic control when carbohydrate intake is more evenly distributed throughout the day. If weight loss is not achieved by targeting identified eating habits and food selections, a more structured approach such as a low-glycemic load diet or a diet with defined portions and calories is warranted. These diets are based on energy requirements calculated from resting energy expenditure equations and modified based on weight change. Younger children can achieve weight loss and better glycemic control when weight is maintained or the rate of weight gain is slowed so that over time with linear growth their weight-for-height gradually returns toward normal. A child whose BMI exceeds the 95th percentile, however, should be encouraged to lose weight. When the diet is implemented linear growth should be monitored to assure that the decrease in calories does not adversely affect linear growth velocity.

Individuals with type 2 diabetes who do not achieve satisfactory glycemic control with a combination of weight loss, portion control, and optimal meal spacing require drug therapy. The meal plan then needs to be formulated based on the prescribed medical regimen.

Teaching Individual Nutrition and Meal Plans

The dietitian has the important task of evaluating the patient’s and family’s understanding of basic principles of nutrition and then providing sufficient knowledge to help clients implement their individualized meal plan. The aim is to lay a foundation for lifelong, healthy eating habits for the child and family. Even the most intensive regimens of insulin administration are not successful without careful attention to meal planning.¹⁵⁵ Nutrition education, like all aspects of diabetes education, has to be an ongoing process, with periodic review and revision of the meal plan. The child’s and parents’ levels of comprehension, ability to solve problems, and adherence to the nutrition goals should be regularly reassessed. Teaching should be followed by systematic assessment of the patients’ ability to perform in an environment that approximates as closely as possible the behaviors expected in the natural environment.¹⁶³

Healthcare professionals need to communicate recommendations in practical terms and teach clients how to minimize the daily burden of food calculations, maintain the pleasure of eating, and enjoy a wide variety of food choices whose use is supported by scientific evidence. A successful nutrition plan is effective only when it enables patients to achieve their healthcare goals. When implementing a nutrition plan, the use of commonly used educational materials and approaches will help a client’s other healthcare providers assist in evaluating their patient’s understanding of and adherence to the plan. Appreciation of a patient’s or family’s learning

style, math and reading skills, and attention to detail is important when devising and implementing the plan.

Several commonly employed meal planning approaches are available. These include: exchange or carbohydrate choices, carbohydrate counting, experience-based estimation,¹⁶⁴ low-GI diet,¹¹⁵ and a method based on the food guide pyramid. Although it is well accepted that diet is integral to successful diabetes care, methods of dietary education remain controversial and poorly evaluated and there is little evidence to support the superiority of one method over another.^{164,165}

Exchange System and Carbohydrate Choices

The exchange system approach for meal planning has been most widely used. It was originally developed in 1950 by a joint committee of the ADA, the American Dietetic Association, and the Diabetes–Arthritis Program of the US Public Health Service, and was last updated in 2003.¹⁶⁶ The system was based on six exchange lists: milk, fruit, vegetable, starch, meat, and fat. Each list indicates the appropriate size or volume of each food exchange. Each portion of food within a group was exchangeable because it contained approximately the same nutritional value in terms of calories, carbohydrate, protein, and fat. The food groups have been more recently cate-

gorized into three groups to simplify the teaching of carbohydrate consistency concepts (Table 7). Thus, by prescribing the meal plan in terms of a number of exchanges for each meal, consistency of total calories and the proportions of nutrients can be maintained, while allowing the patient to choose among numerous foods (Table 8). Accurate measurement of portion sizes has to be learned, and weighing and measuring foods help to achieve familiarity with the sizes of food portions specified in the exchange list. Weighing food is an educational exercise to train the eye and need not be continued indefinitely. If blood glucose control appears inexplicably to deteriorate, however, it is useful to resume weighing and measuring food portions to ensure that amounts are not excessive. The exchange system should not be used in isolation; rather, it should be one component of a nutritional program directed by a trained dietitian.

An example of how this system is applied to an individual patient is illustrated below. An 11-year-old girl's height is 144 cm (50th percentile on the CDC growth chart) and weight is 37.4 kg (50th percentile). Her daily energy requirement based on the RDA to support growth in the 50th percentile is 1,756 kcal. An appropriate distribution of macronutrients could be 50% of the total calories from carbohydrate, 20% as protein, and 30% as fat.

	kcal	g
50% carbohydrate	878	219
20% protein	351	88
30% fat	527	59
Total	1,756	

Table 7 shows how this patient's daily food allowance is distributed among the six food groups.

Recognizing that added sugar in the diet is not deleterious to glycemic control, the food exchanges have been modified to include foods that contain simple sugars. Exchanges that provide 15 g of carbohydrate per serving have been combined and are currently referred to as Carbohydrate Choices (Table 7). This allows for more flexibility in the patient's food choices while maintaining a constant carbohydrate intake. For example, a patient who wishes to substitute a serving of milk for a cookie cannot do so, because while the carbohydrate content will remain constant, other nutrients will be affected (eg, calcium intake will decrease) (Table 7).

Dietitians face new challenges when instructing patients how to use the exchange system. Food selections have become more numerous, patients are eating more meals outside the home, and manufacturers are altering the usual composition of food items in response to the public's interest in low-fat and low-carbohydrate diets. Foods previously assumed to have a uniform composition now can markedly vary in their carbohydrate content. For example, according to the exchange system a 1 ounce slice of bread provides 15 g (1 exchange) of carbohydrate; however, a 1 ounce slice of "low carb bread" provides only 8 to 10 g of carbohydrate. Therefore, it would be incorrect to count a slice of "low carb bread" as 1 exchange. To avoid errors in calculating exchanges, patients should be instructed to use the nutrition facts on a product's food label. Taking the example above, two slices of "low carb bread" could be considered 1 exchange. Patients may find it easier to count grams of carbohydrate rather than exchanges, by looking at food labels and pocket reference books, since this allows for a wider selection of foods. Most food labels do not provide diabetic exchange values, but all provide carbohydrate values.

Carbohydrate Counting

Carbohydrate counting is a meal planning method based on determining the amount (grams) of carbohydrate eaten during a meal (Tables 9 and 10). When educating patients and their families about this approach, the process begins by describing the primary sources of carbohydrate in the diet. It is also important to describe foods that provide little carbohydrate. An introduction to carbohydrate counting utilizes the "carbohydrate choice" defined as 15 g of carbohydrate. The food exchanges groups, milk, starch, fruit, and starchy vegetables have been merged and are referred to as Carbohydrate Choices (Table 7). In addition to the basic exchange groups, desserts and sweets have been added. A typical breakfast of 2 starch

Table 7 Calorie and Macronutrient Content of Exchange Lists

Groups/Lists	Carbohydrate (g)	Protein (g)	Fat (g)	Calories
Carbohydrate choices				
Starch	15	3	0–1	80
Fruit	15	0	0	60
Milk				90–150
Fat-free, low fat	12	8	0–3	90
Reduced Fat	12	8	5	120
Whole	12	8	8	150
Other carbohydrates	15	Varies	Varies	Varies
Nonstarchy vegetable (when using carbohydrate choices, a single serving is "free"; 3 servings = 1 carbohydrate choice)	5	2	0	25
Meat and meat substitutes				
Very lean	0	7	0–1	35
Lean	0	7	3	55
Medium fat	0	7	5	75
High fat	0	7	8	100
Fats	0	0	5	45

Table 8 Distribution of Daily Food Allowance among Six Food Groups for the Child Described in Text

Group	Exchanges	Carbohydrate	Protein	Fat
Starch	8	120	16	
Fruit	4	60		
Milk	3 low-fat (1%)	36	24	9
Vegetables	1	5	2	
Meat	6 medium-fat		42	30
Fat	4			20
Grams		221	84	59
Calories (%)		884 (50.5)	336 (19.2)	531 (30.3)

Table 9 Sample Menu Based on the Daily Food Allowance*

Foods	Exchanges	Carbohydrate Choices	Carbohydrate (g)
Breakfast			
$\frac{3}{4}$ cup cornflakes + 1 slice toast	2 starches	$3\frac{1}{2}$	32
$\frac{1}{2}$ cup 1% fat milk	$\frac{1}{2}$ low-fat milk		6
1 small banana (6 inch)	1 fruit		23
1 tsp margarine	1 fat		
Snack			
1 oz. Granola bar	1 starch	1	20
Lunch			
2 slices bread	2 starches	4	30
1 cup 1% fat milk	1 low-fat milk		12
1 small apple ($2\frac{1}{2}$ inches diameter)	1 fruit		15
Lettuce + tomato	“free vegetable”		
Mustard	“free food”		
1 slice turkey, 1 slice cheese	2 meat		
Snack			
17 grapes	1 fruit	2	15
3 cups low-fat popcorn	1 starch		12
Supper			
$\frac{1}{2}$ cup mashed potatoes	1 starch	4	15
1 cup 1% low-fat milk	1 low-fat milk		12
1 small orange ($2\frac{3}{8}$ inch diameter)	1 fruit		11
$\frac{1}{2}$ cup % broccoloi	3 vegetable		15
3 oz chicken	3 meat		
1 teaspoon margarine	1 fat		
Snack			
3 graham cracker squares	1 starch	$1\frac{1}{2}$	15
$\frac{1}{2}$ cup 1% fat milk	$\frac{1}{2}$ cup low-fat milk		6
2 Tbsp. peanut butter	1 meat + 2 fats		6

*For child described in text.

exchanges, 1 fruit exchange, and 1 milk exchange is equal to 4 carbohydrate choices or 60 g of carbohydrate (Table 9). Patients are taught to evaluate food labels. The “Nutrition Facts” on food labels show the portion size and the total amount of carbohydrate measured in grams per serving. Foods not included in the exchange lists may be more easily incorporated into a meal plan. Table 9 shows a sample menu that includes exchange servings, carbohydrate choices, and grams of carbohydrate for the child described in the text.

Although food labels are available to determine the carbohydrate content of foods, it is important that sample food labels be reviewed and that patients demonstrate proficiency in utilizing the Nutrition Facts to determine portions per container and the amount of carbohydrate in the portion that they actually consume. Among 200 adult participants recruited from a primary care setting, 89% reported regularly using nutrition facts, but only 37% accurately calculated the carbohydrate content of a 20-fluid ounce soda when 2.5 servings was indicated as the number of servings per container. Ninth grade or greater reading skills were identified in 77% of the participants and ninth grade math skill or greater in 37%.¹⁶⁷

Advanced Carbohydrate Counting

Advanced carbohydrate counting is encouraged for patients on basal-bolus insulin regimens or those utilizing insulin pump therapy (Table 10).

This approach attempts to more precisely count carbohydrates, and insulin doses are titrated to match anticipated or consumed carbohydrate intake. Portion sizes should first be determined by weighing or measuring foods to help to develop excellent visual estimation skills. The Nutrition

Facts on food labels and nutrition composition reference guides are then used to determine the carbohydrate content of the portion eaten. The total carbohydrate should be adjusted for fiber and sugar alcohol content to account for their decreased impact on glycemic response. General guidelines are to subtract fiber if it accounts for more than 5 g of the total carbohydrate in the meal, and half that from sugar alcohols. For practical purposes, it may be easier and provide a clearer message to subtract the entire amount of fiber, especially if a patient usually maintains a high-fiber diet. Currently, insulin doses are not adjusted for GI or glycemic load. Although fat intake does not contribute to glycemic load, a high-fat diet prolongs hyperglycemia due to delayed digestion and absorption of the meal.

Patients should be encouraged to keep records of food, medication, physical activity, and blood sugar levels to facilitate recognition of glycemic patterns. In individuals using intensive insulin therapy, if the premeal insulin is correctly adjusted to the carbohydrate content of the meal, the glycemic response 2 to 4 hours after the meal should be within 40 to 80 mg/dL of the premeal blood glucose value. Advanced features available on insulin pumps (eg, dual wave and extended bolus) should be exploited to optimize postprandial blood glucose control when macronutrient distribution is skewed. Diets with limited food choices are inconvenient and difficult to maintain. The broader the understanding of the relationship between food and exercise-related glycemic responses and cardiovascular health, the greater the likelihood that preferred foods can be included in the diet. By measuring pre- and postprandial glucose levels, many individuals use experience to evaluate and achieve postprandial glucose goals with a variety of foods. Reviewing these data with the clinical

Table 10 Constant Carbohydrate Plan and Basal-Bolus Insulin Plan

(A) Constant Carbohydrate Plan						
	Day 1		Day 2			
Breakfast	Grams	Choices	Grams	Choices		
Time: 7:30 am	1 cup 1% milk	12	1	1 cup 1% milk	12	1
Carbohydrate	$\frac{1}{2}$ cup oatmeal	12	1	1 slice wheat bread	15	1
Grams 45 or	1 (6 inch) banana	23	1	1 egg	0	0
Choices 3		47	3	$\frac{1}{4}$ cup strawberries	15	1
					42	3
(B) Basal-Bolus Insulin Plan						
	Day 1		Day 2			
Insulin:carbohydrate ratio is 1:10	Grams	Choices	Grams	Choices		
	1 cup 1% milk	12	1	$\frac{1}{2}$ cup 1% milk	6	0.5
	$\frac{1}{2}$ cup oatmeal	12	1	1 slice wheat bread	15	1
	1 (6 inch) banana	23	1	1 egg	0	0
		47	3		21	1.5
	The patient would take 5 units of rapid-acting insulin				The patient would take 2 units of rapid-acting insulin	

A. Foods are selected in amounts that provide approximately equal amounts of carbohydrate from day-to-day.

B. An insulin:carbohydrate ratio is individually determined for each patient and evaluated by measurement of premeal and 2–3 hour postprandial blood glucose levels.

dietitian experienced in MNT of diabetes can assist patients to optimize glycemic control and consume preferred foods.

Nutrition education is an ongoing process with periodic review of the meal plan and assessment of accuracy of carbohydrate counting. The patient with newly diagnosed diabetes and his or her parents should meet a dietitian several times during the first several weeks after diagnosis. Within a few weeks of the child resuming his or her usual schedule, the patient and family should review the meal plan with a dietitian who should also be available to patients and their families for telephone consultation. Because children's energy needs change with growth and variations in physical activity, food intake should be re-assessed every 3 to 6 months in young children.¹⁵³

Once the family is able to accurately apply the principles of nutrition management directed toward glycemic control, focus should turn to identifying food choices and cooking methods that will be consistent with guidelines aimed at lowering the risk of CVD. If the patient's glycemic control is poor, growth is failing, weight gain is excessive, or the insulin regimen is changed, the dietitian should be reconsulted to review the meal plan and to make any necessary dietary recommendations.

The use of individually applied imaginative teaching methods and materials will engage the attention and co-operation of children and parents more effectively than abstract concepts of nutritional principles and detailed diet sheets that list the composition of individual foodstuffs. Incorporating visual demonstrations with exercises such as sorting pictures, first for their carbohydrate content and then according to healthier carbohydrate choices, will keep the patient and family engaged. This approach also provides the dietitian with an opportunity to assess the patient's understanding of the topic.

Exercise

Regular exercise improves blood glucose control, reduces cardiovascular risk factors, and helps to reach and maintain a healthy weight.¹⁶⁸ School age children should participate daily in at least 60 minutes of moderate to vigorous physical activity that is developmentally appropriate, enjoyable, and involves a variety of activities.¹⁶⁹

Patients with type 1 diabetes should measure their blood glucose levels before exercise to determine whether additional carbohydrate is needed to prevent exercise-induced hypoglycemia. The amount of supplemental carbohydrate depends on the intensity and duration of exercise. If blood glucose is <100 mg/dL before exercise, added carbohydrate is required. If exercise is planned and the individual is familiar with his glycemic response to exercise, the insulin dose at the meal preceding exercise can be decreased in preference to increasing carbohydrate intake. This approach is especially appropriate for overweight patients, attempting to lose weight, who do not want added calories to offset the energy expenditure from exercise.

Adherence

Adherence to a meal plan is one of the behaviors patients must maintain for optimal management of type 1 diabetes and strongly correlates with blood glucose control in children less than 16 years old and in adults.¹⁵⁵ For many patients this is the most difficult aspect of diabetes management.^{170–173} Patients frequently do not adhere to their prescribed meal plan^{73,74,174,175} and parents often have difficulty helping their young child to follow a meal plan.¹⁷³ Furthermore, many children (and their parents) do not possess the knowledge and skill required for good dietary adherence regardless of motivation.^{163,176} Approximately 60% of Finnish adolescents reported at least a satisfactory level of adherence to their meal plans.¹⁷⁷ In the United States, a self-report survey of 144 adolescents who attended a tertiary care diabetes program showed that 15% took extra insulin to cover inappropriate food intake, 73% ate inappropriate foods, and 50% missed meals and snacks.¹⁷⁸ A recent survey of usual dietary intake among a large ethnically diverse cohort of youth with diabetes aged 10 years or older (89% type 1) showed that only 6.5% of the cohort met ADA recommendations of <10% of energy from saturated fat and 40% of youth with diabetes had a BMI of \geq 85th percentile. Less than 50% met recommendations for total fat, vitamin E, fiber, fruits, vegetables, and grains.¹³⁹ There clearly is a critical need for improvement in dietary intake in youth with diabetes in the United States. In contrast, in Northern Italy, where the national diet is already low in total fat, high in monounsaturated fat, and the intake of fruits and vegetables is consistent with recommended allowances, dietary recommendations and good glycemic control can be achieved.¹⁷⁹

Management of diabetes is difficult for children and adolescents and, as children progress through adolescence and into young adulthood, satisfactory adherence to therapy and optimal glycemic control become increasingly difficult to achieve and maintain.¹⁸⁰ To achieve better diabetes outcomes, families must remain actively involved in their youngster's diabetes management, regardless of the child's age, even as the primary responsibility for diabetes care shifts from parent to adolescent.^{181–183}

Eating disorders are a significant health problem among adolescents. Although there has been variation in the reported magnitude of the problem in type 1 diabetes, the combination of an eating disorder and type 1 diabetes has major adverse health consequences.¹⁸⁴ Adolescent girls and young women with type 1 diabetes frequently decrease or omit insulin doses to maintain or lose weight.^{185,186} Family meals are an important defense against disordered eating in adolescents with diabetes.¹⁸⁷

An important role of healthcare professionals involved in the care of children with diabetes is to motivate their patients to overcome the barriers to adherence so that they will be able to enjoy the benefits of good diabetes control.^{67,188} Empirical research shows that adherence to the meal plan can

be improved by psychosocial factors such as promoting a positive attitude toward the illness and its treatment, and fostering supportive relationships with family, peers, and the healthcare team.¹⁷⁷ Frequent follow-up and ongoing education aims to individualize the meal plan and set attainable goals are necessary to teach the patient and his or her family how to match insulin to exercise and food.^{189,190} Self-monitoring of blood glucose and carbohydrate counting provides patients with a greater degree of dietary flexibility and enables adolescents to take a more active and informed role in their diabetes nutritional management.^{183,191,192}

SUMMARY

Nutrition recommendations for treatment of diabetes have repeatedly changed since the preinsulin era, in which a starvation diet with negligible carbohydrate and 70% of calories from fat was the recommended treatment. Today, there is no one "diabetic diet." The recommended diet can only be defined as a meal plan based on a nutrition assessment and mutually accepted treatment goals and desired outcomes. Nutrition therapy should be individualized and consideration given to usual eating habits and other lifestyle factors. Monitoring clinical and metabolic parameters including blood glucose, glycated hemoglobin, lipids, blood pressure, and body weight, as well as quality of life, is crucial to ensure successful outcomes. Modern diabetes management, combining frequent self-monitoring of blood glucose with multiple-dose insulin regimens, or insulin pump therapy, and mastery of carbohydrate counting, has made it possible for children and adolescents with diabetes to have considerable dietary flexibility while maintaining near normal glycemic control.¹⁹³

Although, the introduction of insulin analogs, flexible insulin regimens, and insulin pump therapy has permitted greater flexibility in carbohydrate intake, timing of meals, variety of food choices, and overall quality of life, these therapeutic innovations may also have encouraged unhealthy food choices. Education must continue to emphasize dietary recommendations that maintain normal body weight and improve cardiovascular risk factors, including reduction of total fat, and especially saturated and *trans* fats, and promote consumption of cardioprotective foods such as those containing monounsaturated fats and antioxidants.¹⁶⁴

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