Sugars and starch in the nutritional management of diabetes mellitus

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ABSTRACT Nutritional recommendations, long recognized as an important aspect of diabetes mellitus treatment, have also been an area of persistent controversy, particularly regarding the proportions and types of carbohydrate and fat. This review addresses the role of sugars within medical nutrition therapy for diabetes mellitus. Nutritional recommendations for diabetes mellitus treatment were revised recently. The new guidelines do not specifically restrict intake of sugars, although general recommendations are made for including fiber, whole grains, vegetables, and fruits within dietary selections containing starches. For carbohydrates, the principle focus is on overall caloric amounts. In type 1 diabetes the most effective approach to the control of postprandial hyperglycemia continues to be adjustment of premeal doses of insulin on the basis of carbohydrate counting. In type 2 diabetes, in addition to a focus on caloric content of carbohydrate, consideration continues to be given to the role of the glycemic index as a determinant of postprandial hyperglycemia and overall metabolic control. Nevertheless, consensus recommendations do not support widespread use of the glycemic index. An area of some change is a more clear endorsement of including monounsaturated fatty acids. Current recommendations are that monounsaturated fatty acids and carbohydrates combined should provide 60–70% of daily energy intake, with individual flexibility in the respective proportions, whereas intake of saturated fats is limited to <10% of energy intake. This new emphasis reflects greater awareness of the importance of responding to individual and cultural dietary preferences and the need to address treatment of both hyperglycemia and dyslipidemia in diabetes mellitus. Am J Clin Nutr 2003;78(suppl):858S–64S.

KEY WORDS Diabetes mellitus, nutrition, diet, sugars, starch, glycemic index

INTRODUCTION

The prevalence of diabetes mellitus (DM) is increasing around the world and at a rate that appears so dramatic as to have been characterized as an epidemic (1, 2). This increase is nearly entirely due to an increased prevalence of type 2 DM, a disorder caused by a combination of insulin resistance and impaired insulin secretion. The incidence of type 1 DM, a serious metabolic disorder caused by failure of insulin secretion resulting from autoimmune destruction of pancreatic b cells, has remained relatively stable in comparison with that of type 2 DM. The increased prevalence of type 2 DM is being found in nearly all countries, virtually all racial and ethnic groups, and across the age range from childhood and adolescence to the elderly, although clear risk associated with aging persists. Many factors have been postulated to contribute to the DM epidemic. Environmental factors have drawn particular attention because of the rapidity of the increase in type 2 DM (3). Most notable is the increase in the prevalence of obesity that has paralleled the rise in type 2 DM and the interrelated aspect of sedentary lifestyles (4, 5). Not all persons with obesity or physical inactivity develop DM, but these factors constitute part of the risk for type 2 DM because of their strong effect to induce insulin resistance. Even among those who do not develop type 2 DM, the development of the insulin resistance syndrome, which is the clustering of impaired glucose metabolism, hyperinsulinemia, hypertension, and dyslipidemia as well as other metabolic perturbations, constitutes increased cardiovascular risk (6,7). Accordingly, there is renewed interest in prevention of insulin resistance and type 2 DM through both pharmacologic and nonpharmacologic or lifestyle interventions. The key lifestyle interventions are physical activity and a nutritional plan also entailing reduced caloric intake. In this article, the focus is on dietary factors in the management of DM.

Because DM is a disease directly related to carbohydrate, lipid, and protein metabolism, nutrition has always had an integral role in its management. The contemporary term used to describe the dietary prescriptions is medical nutrition therapy (MNT) (8). Before the advent of insulin therapy in the early 20th century, MNT was the only form of therapy for DM (9). The focus of nutritional recommendations at the turn of the last century was on type 1 DM: on control of the severe hyperglycemia to which these patients are vulnerable as well as on prevention of what was then, before the advent of insulin therapy, commonly a mortal episode of ketoacidosis. Dietary prescriptions in that period emphasized strict restriction of carbohydrate intake and, in particular, an avoidance of sugars (9). The restriction on intake of sugars was perhaps an intuitive concept in that period because there were few direct clinical trials using a randomized design; the literature from that time indicates that it was presumed that an illness defined by elevated blood sugar was almost certainly linked to the ingestion

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of sugars. Ironically, this is a concept that became deeply rooted and often persists today within the lay community and among some health professionals. To limit carbohydrate intake, physicians of that earlier period encouraged an increased intake of lipids. Later, as life expectancy for those with type 1 DM improved after the advent of insulin therapy, better recognition ensued of the serious cardiovascular complications of DM. By midcentury and later, nutritional recommendations for DM were being sharply revised to moderate consumption of fats and, on an isocaloric basis, to liberalize use of carbohydrates.

About 2 decades ago, questions arose again concerning the relative proportions of carbohydrates and fats that were most healthy for those with type 2 DM. Clinical investigations reported that when considered on an isocaloric basis, a high proportion of carbohydrates (relative to monounsaturated fats) causes plasma triacylglycerols to be elevated and therefore might lead to deterioration of the dyslipidemia characteristic of type 2 DM and insulin resistance (10, 11). The Seven Countries Study pointed to the health benefits of diets enriched in monounsaturated fatty acids (MUFAs) (12). Intervention trials in subjects without DM showed that in comparison with a high-carbohydrate diet, a diet with a high proportion of MUFAs led to lower concentrations of triacylglycerol (13). Similar findings were reported for type 2 DM (14), and glycemic control was reported to be improved as well. Other investigators reported that replacing saturated fat with complex carbohydrates achieved beneficial effects on dyslipidemia in type 2 DM, lowering LDL cholesterol independent of cholesterol intake without inducing adverse effects on HDL cholesterol and triacylglycerols (15).

Relatively few persons were studied in these investigations, but the findings stimulated vigorous debate concerning nutritional recommendations for type 2 DM; much of this debate centered on the risk of cardiovascular disease. Yet, for many patients, for the public at large, and even within the medical community, the notion persisted that persons with DM should specifically avoid the ingestion of sugars. The available data do not clearly implicate risk of type 2 DM in relation to consumption of carbohydrates in general or sugars specifically (16). Positive associations between consumption of fats and meat products and incidence of type 2 DM were found (17), as well as an association between an increased proportion of trans fatty acids in the diet and risk of type 2 DM (18).

The current recommendations for MNT of DM will be presented here as the background for more specific consideration of the role of sugars in the diet of those with DM. Related to this, the glycemic index (GI) in MNT of DM will be discussed and current controversies concerning the treatment of postprandial hyperglycemia in the management of DM will be considered.

CURRENT DIETARY RECOMMENDATIONS FOR DIABETES MELLITUS

Recommendations for MNT for DM are published annually by the American Diabetes Association (ADA) within an overall annual review of clinical care guidelines published in Diabetes Care (8). During the past year, however, in addition to this annual update, the findings of a more in-depth technical review on this topic, conducted by an expert panel, was completed and approved by the ADA (19). This technical review represented the most substantial revision of MNT by the ADA since its 1994 report (20). In the most recent technical review, recommendations for MNT were based on a review of the relevant literature by a group of selected experts in nutrition, and the strength of the evidence was appraised (19). The strongest levels of evidence were findings based on randomized clinical trials of sufficient size and duration that were consistent across at least several trials. A much weaker level of evidence was recommendations based on expert opinion but without the rigor of clinical trial testing. Epidemiologic findings that indicate associations between nutritional factors and health are of weaker significance than randomized clinical trials. Different recommendations were made for type 1 and type 2 DM; recommendations were made for macronutrients and micronutrients.

One of the most fundamental changes emphasized in the recent guidelines is the need for individualization of the nutrition prescription. Rather than prescribing a uniform proportion of fats and carbohydrates for all patients, as had been done previously (and as recently as the past few years), the current recommendations emphasize greater flexibility. Previous recommendations were that carbohydrates should compose ≈50% of caloric intake and fats should compose ≈30%. Current recommendations are that carbohydrates and MUFAs together should compose 60–70% of daily caloric intake. The exact proportions of carbohydrates and MUFAs are not specified and individualization is recommended.

One of the reasons for the move toward greater flexibility in the proportions of carbohydrates and MUFAs is the ethnic and cultural diversity of the large population of persons with DM; MNT must make realistic recommendations that can be adapted to the eating patterns that reflect this diversity and, of course, reflect individual preferences. The greater flexibility concerning carbohydrates and MUFAs also reflects the debates in prior decades over the potential effects of carbohydrates to aggravate dyslipidemia and the potential health benefits of MUFAs.

Considerable scientific progress has been made in delineating health effects of the fatty acid composition of the diet. A recent position statement from the American Heart Association reviews the potential health benefits and risks related to the fatty acid composition of the diet (21). Fatty acids differ in their effects on insulin sensitivity (22). However, the committee reviewing MNT for DM expressed concern that unrestricted use of MUFAs for type 2 DM might lead to greater weight gain than would occur with a diet higher in starches and fiber; obesity is the major risk factor for development of type 2 DM (3). Not only the severity of obesity but also recent weight gain increase the risk of type 2 DM (23) and, therefore, weight management is of crucial importance in MNT for type 2 DM. No randomized clinical trials directly address the issue of MUFAs. In general, higher intakes of fat dispose to weight gain. Some studies indicate that a diet with a relatively higher proportion of fiber and starches leads to weight stabilization or slight weight loss at least over the short term (24), a finding that was not replicated in another more recent study (25). We will return to the issue of fiber content in the diet later.

Although the recent MNT for DM liberalizes the guidelines with respect to MUFAs, the guidelines are clear in raising health concerns about intake of saturated fats. Recommendations from the ADA for MNT are that saturated fat should not exceed 10% of daily energy intake. There is strong evidence in populations with type 2 DM, as well as among those without DM, that saturated fatty acids can worsen dyslipidemia and insulin resistance, raise LDL cholesterol, and thereby increase the risk of cardiovascular disease (15, 22). Caution was also raised concerning
trans fatty acids. Several studies suggest adverse effects of trans fatty acids on insulin sensitivity and DM. The Nurses’ Health Study suggested that increased consumption of trans fatty acids was a risk factor for type 2 DM (18). The replacement of trans fatty acids by polyunsaturated fatty acids (PUFAs) may lessen or reverse this risk. A recent intervention study supports the link between consumption of trans fatty acids and induction of insulin resistance (26).

It is also recommended that fats high in PUFAs compose ≥10% of daily energy intake. Within the classes of PUFAs, particular attention has been given to n-3 PUFAs (found in fish oil). A recent review addressed many of the current issues and research related to ingestion of PUFAs with respect to DM (27). Long-chain n-3 PUFAs lower triacylglycerol concentrations and may have beneficial effects on cardiac electrical conduction that lessen the risk of arrhythmia and sudden death. However, despite these important potential benefits, there has been some concern that a relatively high intake of long-chain n-3 PUFAs could worsen hyperglycemia and aggravate insulin resistance. This issue has been investigated within clinical trials, and the findings provide a fairly clear basis for the current recommendations that the largely favorable effects of long-chain n-3 PUFAs to lower hyperglycemia can be attained without adverse effects on hyperglycemia if intake does not exceed 3 g/d (28–30).

CONSUMPTION OF SUGARS AND DIABETES MELLITUS

The ADA recommends that the classifications sugars, starch, and fiber be used as the functional definitions of carbohydrates for MNT (19). Previously used terms such as simple sugars, complex carbohydrates, and fast-acting carbohydrates are frequently found in the literature but are now discouraged from further usage and are no longer recommended for patient education. With respect to carbohydrates, the key emphasis of MNT for DM is on the total amount of carbohydrate in terms of energy intake (31). Regarding the type of carbohydrate ingested, the guidelines for MNT in DM clearly stress the value of selecting vegetables, fruit, and grains so that the starches consumed will include adequate amounts of both fiber and micronutrients (8).

The position on sugars follows directly from the principles presented above. Sucrose and other sugars can be consumed by those with DM and need to be considered primarily from the perspective of energy consumed and thus substituted for other sources of carbohydrate (8). One relative admonition concerning the consumption of sugars is that in sugar-based beverages and some prepared foods, micronutrient content will be low and a diet with an elevated proportion of sugars may be deficient in fiber and micronutrients (19). This perception of the sensitivity of metabolic control in DM to energy balance underlies the recommendations that emphasize carbohydrate content as a key point for patient education. The 3 points stressed are 1) knowledge concerning which foods contain carbohydrates, 2) recognition of the portion size for carbohydrate within a meal (with 15 g being the basis for estimating 1 carbohydrate serving), and 3) knowledge of how many carbohydrate servings are appropriate within a meal or snack.

This priority placed on total energy consumption and carbohydrate counting is fundamental in the overall emphasis that metabolic control, both in terms of hyperglycemia and in relation to plasma triacylglycerol concentrations in an overweight or obese person with type 2 DM, is highly sensitive to either positive or negative energy balance. Negative energy balance can promptly induce reductions in hyperglycemia and hypertriglyceridemia, even before the achievement of substantial weight loss, whereas consumption of excess energy has the opposite effect (32, 33). Of course, long-term weight loss also can improve metabolic control (34), and substantial weight loss as can occur with bariatric surgery can lead to remission of type 2 DM (35). Increased energy consumption regardless of source, but certainly including carbohydrate, directly induces insulin resistance (36).

Metabolic studies have been used to compare the glycemic response to sugars consumption in persons with DM with isocaloric consumption of other sources of carbohydrate. Bantle et al (37) compared the postprandial glycemic response to various forms of carbohydrate (42 g separately of glucose, fructose, sucrose, potato starch, and wheat starch) that composed 25% of total energy within a mixed meal also containing protein and fat. Fructose ingestion led to a lower postprandial glycemic response in those with DM, but the other forms of carbohydrate had nearly identical responses. In a short-term trial, the addition of sucrose did not adversely affect glucose control if accounted for on an isocaloric basis (38). The ADA expert panel analyzed 22 studies addressing this issue and concluded that when ingested in isocaloric quantities, sucrose does not affect glycemic control in DM significantly differently from other carbohydrates (19). The recommendation is that if sucrose is consumed, it should be substituted for other carbohydrates.

The reason that fructose substituted on a caloric basis for other carbohydrates has a lower glycemic response is attributable to a predominantly hepatic uptake of fructose with very limited release as endogenous glucose production. A recent physiologic study in dogs suggests that adding a small amount of fructose increases hepatic uptake of ingested glucose (39), a finding that is intriguing but that needs more study before being a basis for clinical recommendations. Although there is potentially a favorable metabolic postprandial pattern in relation to fructose absorption on hyperglycemia, fructose can elevate plasma lipids (40). For example, consumption of large amounts of fructose (15–20% of daily energy intake) increases LDL cholesterol, fasting cholesterol, and triacylglycerols in subjects without DM.

One additional issue is related to fiber intake. Current recommendations for the general population are that men have a fiber intake of ≥38 g/d and women have ≥25 g/d. There has been specific interest in the role that dietary fiber may have in the nutritional management of DM. Benefits of fiber were found with regard to glycemic control, HDL and LDL cholesterol, and triacylglycerols (25). However, a recent 3-mo study by Jenkins et al (41) did not find a metabolic advantage of high-fiber over low-fiber cereals. In consideration of the available data as a whole, the ADA expert committee did not perceive that there was value in recommending an increase in fiber intake above general recommendations for persons with DM (19).

THE GLYCEMIC INDEX

An area of persistent interest and controversy about carbohydrate content in MNT for DM is the GI of foods. The GI of a carbohydrate is defined as the incremental rise in plasma glucose (above baseline) relative to that induced by a standard, usually 50 g glucose or a white bread challenge (42). Lower GI values indicate a lesser rise in glucose. Another measure discussed in this regard is the glycemic load (43), which is the amount of carbohydrate multiplied by its GI. The glycemic load can be shaped by
the carbohydrate load, whereas the GI takes into account different plasma glucose responses against a background of equivalent available carbohydrate challenge.

The GI of various carbohydrates appears to be substantially determined by rates of intestinal hydrolysis (31, 32). Cooking gelatinizes starches, making them more accessible to pancreatic amylase, which raises rates of hydrolysis and accordingly raises the GI compared with the same food in its raw form. Similarly, carbohydrate with a dense food matrix, such as spaghetti, hinders the action of amylase. Foods such as legumes contain a resistant starch and are digested more slowly and correspondingly have a lower GI. Starch foods with amylose structures (ie, a long-chain starch molecule), such as long-grained rice, are more slowly hydrolyzed than are amylopectin foods (ie, a branched-chain structured starch). It might seem to follow from these principles on digestibility that sugars such as fructose or sucrose might have a high GI, yet this is not the case. Fructose is substantially taken up by the liver after ingestion and therefore only a small portion directly enters the peripheral circulation. Thus, fructose has a low GI; foods rich in galactose and fructose, such as many foods rich in sugars, have a lower GI than do starches, which are composed of a polymer of glucose.

Despite the accuracy and validity of the empiric findings under controlled conditions, the value of applying the GI to clinical care and nutritional therapy of DM has been met with some measure of skepticism as well as with strong enthusiasm. Perhaps representative of these attitudes and opinions is an article entitled “Is Diet the Cornerstone in Management of Diabetes?” by Wood and Bierman (44), which was published ≈2 decades ago, not long after the concept of the GI was initially developed. Woods and Bierman expressed interest in the concept of the GI but questioned whether these principles could be effectively taught to patients, given the multiple factors affecting the GI.

The debate about the GI remains at least as vibrant today as it was 2 decades ago (42, 43, 45–47). Arguing against broad incorporation of the GI into nutritional recommendations for treatment of DM, Pi-Sunyer (47) touched on several points similar to those raised by Wood and Bierman (44). Pi-Sunyer noted that the GI of foods is affected by the presence of other macronutrients in the meal. For example, the presence of fat within a meal alters (reduces) substantially the glycemic responses of foods such as potatoes, and therefore giving advice about foods in isolation is not likely to be informative. Another example is that cooking in general, and the types of cooking in some instances, can markedly affect the GI. Thus, as argued by Pi-Sunyer, even highly motivated patients would need to take into account a complex array of information, information that would seem at least on the surface to have internal inconsistencies, and this would render it difficult to use the GI, limiting the value of this information.

Arguments for advocating the use of the GI and incorporating it into MNT for DM include a pathophysiologic postulation that a higher prandial glycemic response will evoke more robust insulin secretion and hyperinsulinemia (48). This may have 2 deleterious effects: repeated strong stimulation of the pancreas could accelerate loss or exhaustion of pancreatic function, and hyperinsulinemia may further aggravate insulin resistance. Furthermore, it has been argued that a high GI and a high glycemic load would have greater adverse effects in persons with obesity, glucose intolerance, and insulin resistance, namely, those recognized to be at an increased risk for the development of type 2 DM (43). Luscombe et al (49) experimentally manipulated the GI within the diet of subjects with type 2 DM and did not find a clear effect on glycemic control, although some effects on HDL metabolism were noted. In reviewing 9 randomized clinical trials concerning implementation of a change in GI, the ADA expert panel did not find clear evidence in support of a beneficial effect of a low-GI diet on glycemic control in type 2 DM (19).

The physiologic rationale formulated in favor of considering the GI (43, 48) may lose significance in relation to overt type 2 DM as opposed to those with insulin resistance but normal glucose tolerance, because the multiple derangements of prandial metabolism that prevail in overt type 2 DM may overshadow the influence of the GI. Several metabolic factors contribute to postprandial hyperglycemia in type 2 DM (50–52), including impaired insulin secretion, impaired suppression of glucagon, reduced stimulation of glucose uptake by peripheral tissues, impaired suppression of lipolysis, and impaired suppression of endogenous glucose production. Of these, perhaps the physiologic process that has the greatest immediate effect on the severity of postprandial hyperglycemia is impaired suppression of endogenous glucose production (50, 51).

Normally, in those without type 2 DM (and even in those with insulin resistance but normal glucose tolerance), there is a prompt and substantial suppression of endogenous glucose production in response to the secretion of insulin and suppression of glucagon. The suppression of endogenous glucose production serves to substitute ingested glucose, thereby limiting postprandial hyperglycemia and promoting use of ingested glucose by both insulin-stimulated tissues (eg, muscle) and insulin-independent tissues (eg, brain) (50). In type 2 DM and in persons with impaired glucose tolerance, isotope tracers used to measure the systemic entry of ingested and endogenous glucose have shown that although patterns of absorption of ingested glucose are similar to those in persons with normal glucose tolerance, the severity of postprandial hyperglycemia is due to severe impairment in the suppression of endogenous glucose production (50–52). Additionally, the efficiency of the clearance of glucose by peripheral tissues is reduced. Against this background of relatively severe metabolic derangements, the isolated effect of the GI may be difficult to discern with any resolution, which may be the underlying issue with the relatively negative pattern of clinical trial data cited above. Perhaps if the postprandial kinetics of insulin secretion and impairment in the suppression of endogenous glucose production in type 2 DM were corrected, pharmacologically or otherwise, then a more distinguishable effect of the GI might emerge.

In type 1 DM, an often-cited clinical principal for the control of postprandial hyperglycemia is that the dose of premeal insulin and in turn the adjustment of this dose on the basis of the carbohydrate content of the meal is a key determinant of the glycemic response (53). Day-to-day variation in amounts of fat or protein have less effect on glycemic control. Because of this predominant relation of postprandial glycemia and insulin dose, the expert panel that reviewed MNT concluded that with respect to type 1 DM there is not clear evidence of benefit for incorporating the GI (9).

**ROLE OF POSTPRANDIAL HYPERGLYCEMIA AS A MANAGEMENT GOAL FOR DIABETES MELLITUS**

With the completion of the United Kingdom Progressive Diabetes Study, the Diabetes Control and Complications Trial, and other smaller trials, there is now universal acceptance of the importance of attaining normal or nearly normal glycemic control to lessen the incidence and severity of microvascular complications (54, 55). However, 2 important questions persist. The first is
whether glycemic control is equally important for the prevention of macrovascular complications of DM. Although neither of the studies mentioned above was designed to specifically address whether tight glycemic control lessens risk of macrovascular complications, the findings suggest that the effect is positive but not as robust as on microvascular complications. The importance of treating dyslipidemia and hypertension are strongly supported by clinical trial data, and in this context, rigorous control of hyperglycemia may be of additional benefit. The second question, one that has reemerged in recent years, is whether postprandial control of hyperglycemia per se, apart from control of fasting hyperglycemia and overall indexes of glycemic control (eg, as reflected in hemoglobin A1c) is an important metabolic risk factor for cardiovascular disease. Data from the DECODE study suggest that postprandial glycemia may be a determinant of risk for cardiovascular disease independent of fasting plasma glucose (56). A recent technical review of the topic by the ADA reached a more tentative conclusion (57) and neither recommends specific monitoring of postmeal glucose nor postulates target values but calls for more information in this area.

Several treatment options either currently available or under development primarily target postprandial hyperglycemia. These agents include several drugs within the class of α-glucosidase inhibitors; 2 agents within meglitinides that induce a more rapid insulin secretory response than do sulfonylureas; amylin, which slows gastric emptying; and several agents that are designed to increase glucagon-like peptide 1, a gastrointestinal incretin that also slows gastric emptying. Thus, we can anticipate that the issues of glycemic response to diet will remain an important area for continued research, and the nutritional determinants of this response will continue to warrant scrutiny as other components of treatment for DM develop and evolve.

RESEARCH NEEDS

Numerous potential areas of research concern the role of sugars and carbohydrate in the pathogenesis and treatment of DM. The data regarding the role of a high glycemic load as a risk factor for type 2 DM has generated considerable interest and also much debate as to the validity of this finding; this area warrants additional study, including study in diverse populations. Since the concept was developed in the 1980s, considerable research has been done on the GI as a dietary influence on metabolic control in DM. Perhaps one reason to further examine this issue is that with the advent of more specific pharmacologic therapies targeted at postprandial hyperglycemia, there may be renewed interest in reexamining whether combining such pharmacologic approaches with the nutritional principles of the GI might lead to further improvement in the control of postprandial hyperglycemia.

More research on the role of dietary carbohydrates in general and sugars more specifically in the pathogenesis of hypertriglycerideridemia would be of value for the general population, but there is particular need for these types of studies in persons with type 1 and type 2 DM. In this same regard, there is special interest in further understanding the role that triacylglycerol accumulation within skeletal muscle and liver has in the pathogenesis of insulin resistance and DM (58, 59). Although obesity is a strong risk factor for triacylglycerol accumulation within skeletal muscle and liver, little is known regarding the effect of nutritional patterns on these depots of lipid accumulation. Given that carbohydrates lead to increased plasma concentrations of triacylglycerols, the question of whether carbohydrates in general and starches and sugars, compared isocalorically, directly regulate tissue lipid not accounted for by energy intake should be examined.

CONCLUSIONS

During the past decade, several revisions in nutritional recommendations have occurred for persons with type 1 and type 2 DM. Several of these areas of revision have direct implications for the role of dietary sugars in the nutritional management of DM. The most recent set of recommendations for MNT for DM advocate flexibility in the distribution of caloric intake between MUFA’s and carbohydrates. Together, these 2 sources should compose 60–70% of caloric intake. The relative allocation of intake between carbohydrate and MUFA’s is not specifically delineated but instead it is recommended that this be individualized on the basis of eating preferences and other considerations. Among these are the consideration that MUFA’s can lower plasma triacylglycerol relative to isocaloric consumption of carbohydrates.

Current recommendations are that intake of sugars can be appropriate for those with DM, provided the consumption of sugars is taken into account on the basis of calorie consumption. A caveat in this regard is the recommendation for adequate intake of dietary fiber and micronutrients. This same emphasis on the primary of caloric intake as a determinant of metabolic control rather than distinguishing between sources of carbohydrate (or more generally, between MUFA’s and carbohydrates) has shaped the current consensus recommendations regarding the clinical utility of the GI. Although the nutritional merits of low-GI compared with higher-GI carbohydrates continue to be examined and debated, numerous variables can alter the GI, even for a given source of carbohydrate, and this consideration limits the practicality of implementing patient education on this topic. Moreover, whether the GI is a meaningful determinant of metabolic control in DM continues to be challenged on the basis of the collective data of prior clinical trials. Achieving good metabolic control of postprandial hyperglycemia remains one of the most challenging aspects of attaining good overall glycemic control. Perhaps not until several of the more specific pharmacologic approaches now under development prove to be effective will the appropriate circumstances arise for renewed consideration of the role for GI in optimizing nutritional approaches to metabolic control in DM.

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