Nutritional Therapy for Type 2 Diabetes

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Abstract:

Objective: The purpose of this literature review is to compare and contrast recent literature regarding nutrition for the type II diabetic. This review will analyze how and why diet can be effective in both the prevention and care for diabetes. Special attention will be paid to the carbohydrate debate that is now raging among dieticians. Should the diabetic load carbohydrates or limit them? Also investigated will be the role of proteins, lipids, fiber, and antioxidants in the diet of the diabetic.

Data Collection: The resources utilized included indexed/referenced journal articles, text and reference books, and internet websites. Pubmed, Ebscohost, Chiroweb, Chiroaccess, and Mantis were databases used to find journal articles and publications related to the topic of diet therapy for the diabetic. The Mobius library catalog was utilized at the Learning Resource Center of Logan College of Chiropractic to find books relating to the topic of nutrition for the diabetic.

Results: The keyword search for type 2 diabetes turned up thousands of articles regarding diet therapy. Appropriate nutritional guidelines for type 2 diabetes have been under debate by leading researchers in the field. The following conclusions represent a consensus of research regarding good conservative management of type 2 diabetes.

Conclusion: Current literature emphasizes a practical dietary approach for type 2 diabetes that applies to anyone wishing to improve his/her health with diet. The diet therapy for the type II diabetic should include an adequate source of monounsaturated fatty acids, fiber, whole grains, fruits, and vegetables; and a limited number of refined sugars and saturated fatty acids. The diet does not have to be a low fat one. A variety of vegetable foods rich in monounsaturated fatty acids (MUFA's) are good sources of antioxidants and lipid control. Because the role of glycemic index has been challenged, the current carbohydrate recommendations follow that the intake of MUFA's and carbohydrates combined should compose 60-70% of total caloric intake. Consideration for supplementation must be given due to the role of magnesium, chromium, zinc, selenium, and vanadium on insulin resistance. It would be remiss for an individual to limit his diabetes management strategy to diet alone. A proper diet, exercise, and subsequent weight loss can essentially reverse the devastating effects of type 2 diabetes.

Key Words: Diabetes mellitus, non-insulin dependent/diet therapy, nutrition, glycemic index, cholesterol, triacylglycerol.

Introduction:

Most Americans hear the word diabetes and automatically think of managing a disease with the use of oral drugs and insulin injections. What if there was a better way? What if there was a way to reverse diabetes without the use of drugs or surgery? There is a great debate in America as to how to deal with the near epidemic rise in people with type II diabetes mellitus (DM). 16 million Americans have DM (1). DM is of particular concern because the incidence of the disease has tripled in the past 25 years (2). Historically, children were considered to be safe from the debilitating and deathly effects of DM. However, because obesity and inactivity has become a problem in today's children, DM is now striking several of America's kids (2). Before insulin was discovered in the early 19th century, DM was combated with diet and exercise alone. This literature review will discuss the potential effects of nutrition on DM.

Diabetes mellitus can be present in three major types: type I diabetes mellitus, type II diabetes mellitus, and gestational diabetes mellitus. Type I diabetes mellitus (insulin-dependent) develops at a young age and occurs when beta cells of the pancreas are no longer able to produce insulin. Insulin is responsible for glucose uptake by the cells of the body. In most cases, type I diabetes mellitus is caused by an autoimmune reaction in which the body makes antibodies against its own pancreas. Unless this condition is caught very early, it is essentially irreversible. Gestational Diabetes is a form of Diabetes that is first recognized, or develops, during pregnancy. In this disease, insulin is present at normal levels but does not work properly to get glucose into body cells. This is called glucose intolerance. Often times the glucose intolerance associated

with the condition disappears after pregnancy. However, a wide range of complications is associated with the disorder including risk of pre-eclampsia, cesarean delivery, and future type II diabetes mellitus (DM) (3). DM is the most common form, accounting for 90-95% of all cases of diabetes mellitus. DM also occurs when the body can no longer utilize glucose correctly. Insulin in the blood becomes increased, a condition known as hyperinsulinemia.

Two tests are commonly used to diagnose DM. The fasting blood glucose test is the most common and least expensive. It involves fasting over night and then having a small amount of blood drawn to evaluate glucose concentration. A reading of 126 mg/dl or higher is considered a positive value for DM. The glucose tolerance test involves drinking a sugary liquid, waiting two hours, and then having blood drawn. A glucose concentration reading of over 200 mg/dl is considered positive for DM. The ADA recommends the fasting blood glucose test over the glucose tolerance test (3). A simple C-peptide test will help determine type I from type II in the non-pregnant patient.

Symptoms for all three types of diabetes mellitus are the same and include: polyuria, polydipsia, polyphagia, and weight loss despite frequent eating. What is perhaps more serious, however, is the tremendous toll that Diabetes takes on the body in the long run. The number one cause of blindness in people age 20-74 is diabetic retinopathy. Glaucoma and cataracts are more frequent in the diabetic. 40% of kidney failure is due to diabetes. Nerve damage occurs in 60-70% of diabetics causing anything from sensory loss in the feet to generalized pain and the need for amputation. Cardiovascular disease is markedly increased in the diabetic. In fact, three-fourths of all

diabetics ultimately die from heart disease. Infections, memory loss, dementia, and periodontal or gum disease are also increased in the diabetic (2).

Many factors may contribute to the DM epidemic. Most prominent is the parallel increase in both obesity and DM. Obesity is thought to lead to DM because of increased insulin resistance. Insulin resistance leads to impaired glucose metabolism, hyperinsulinemia, hypertension, and dyslipidemia. In turn, insulin resistance can lead to risk of cardiovascular complications (4). Because DM is a disease directly related to carbohydrate, lipids, and protein metabolism, nutrition has always been thought to play an integral role in the management of DM. The contemporary term to describe the dietary restrictions and prescriptions for DM is Medical Nutrition Therapy (MNT) (5). The amount and type of lipid, carbohydrate, or protein to consume is questionable depending on what you read.

It might make sense for DM sufferers to lower carbohydrate intake, especially with regard to sugars since DM is actually defined by hyperglycemia. However, when you lower carbohydrates too much, often fat intake becomes too high which raises concerns with cardiovascular complications such as arteriosclerosis and plaquing. With regards to this issue, the ADA has most recently advised that each individual develop a diet based on his or her unique needs. The ADA no longer recommends a base line percentage of carbohydrates, lipids, or proteins to consume. It is now necessary for the DM patient to have a more in depth understanding of nutrition because the ADA now recommends that the DM sufferer actually follow a healthy diet appropriate for everyone, including both non-DM persons and DM persons (5).

Extensive literature on diabetes exists, but it does not take long to find conflicting information. Appropriate nutrition for the diabetic is possible, but finding agreement among authors is difficult for the average consumer. Even the American Diabetes Association (ADA) has not made definitive guidelines that leading experts can agree on (6). Where is the newly diagnosed diabetic to turn? This literature review attempts to define some current healthy diet recommendations and how these recommendations might apply to the DM patient.

Discussion:

Mechanism of DM:

The mechanism as to how hyperglycemia leads to loss of pancreatic beta cell function is not entirely clear. Whether the loss of pancreatic function results primarily from excessive secretion of insulin (beta cell exhaustion) or toxicity to beta cells because of hyperglycemia is uncertain (7). Either way, it would follow that a diet that produced higher blood glucose concentrations and a resulting greater demand for insulin would increase the risk of DM. By definition, high-glycemic index forms of carbohydrate are foods that produce a high concentration of blood glucose and increased insulin demand. In summary, the incidence of DM increases by either increasing insulin demand or decreasing insulin sensitivity. Both mechanisms describe what is known as insulin resistance syndrome (also called Syndrome X, Reaven's syndrome, and metabolic syndrome) (8).

The destruction of the body as a result of insulin resistance syndrome occurs in several ways. Elevated levels of glucose are harmful because they result in the accumulation of sorbitol in the cell, which can cause tissue damage. Elevated glucose will also accelerate the binding of sugar to proteins, also called glycosylation. Glycosylation is responsible for a number of the diseases that are a result of DM. The protein especially damages the kidney filtration system. When glycosylation occurs, the water goes with the protein into the blood. The loss of water leads to dehydration and the marked thirst that one feels when diabetic (2).

Hyperinsulinemia poses another problem in and of itself. Excess insulin can upset the normal metabolism of fat in the body. Elevated insulin levels cue the liver to make more very-low-density lipoproteins (VLDL). Elevated VLDL will actually lower the "good" cholesterol high density lipoprotein (HDL), which helps to escort cholesterol out of the body. VLDL also acts as a carrier of triglycerides in the blood and can actually be converted to low density lipoprotein (LDL). LDL is the "bad" cholesterol that gets deposited on artery walls. Hyperinsulinemia also raises levels of fibrinogen, which encourage the formation of blood clots that may contribute to heart attacks. Plasminogen activator inhibitor-1 (PAI-1) is also elevated as a result of hyperinsulinemia. PAI-1 normally slows down the breakup of blood clots. Lastly, hyperinsulinemia raised the inflammatory C-reactive protein, which contributes to cardiovascular risk (2).

DM onset is slow and not always obvious to the person with the disease. The average person has the disease 8 years before ever being diagnosed (9). Diabetes is fundamentally a condition of altered glucose metabolism. Therefore, some form of dietary or pharmacological management is crucial to preventing the onslaught of DM

symptoms and dangers. Major complications from DM are divided into two categories, medical emergencies and chronic diseases. Short term complications include Hypoglycemia, Hyperglycemia, and Ketoacidosis (9). Hypoglycemia usually occurs when a person's insulin therapy goes awry. Hyperglycemia occurs as a result of the insulin resistance syndrome discussed previously. Ketoacidosis occurs when the cells are so starved due to lack of glucose that the body converts fats into glucose, thereby releasing deadly toxins called ketones. All three of these medical emergencies can lead to coma or death (9). Chronic diseases such as neuropathy, retinopathy, nephropathy, and blood vessel disease all have a gradual onset and develop as a result of hyperglycemia.

Insulin and Drug therapy:

With regards to insulin and drug therapy for DM, both insulin resistance and faulty pancreatic beta cell function must be considered. In order to attain proper glycemic control with the use of direct acting agents, it is necessary to limit the unsatisfactory side effects of the insulin resistance syndrome. Because diabetes is a progressive disorder, a selection of different-acting agents is necessary to combat different features of the disease (10). It is now acknowledged that the majority of DM persons will not achieve satisfactory results with only one antidiabetic agent (11).

Insulin action enhancers help the DM person by assisting the glucose into cells that require it. A fungal metabolite, LY783281, has recently been shown to enhance insulin action by stimulating insulin receptor tyrosine kinase (12). Chiroinositol has been shown to fuel the kinase activity of insulin-signaling intermediates (13). Angiotensin

converting enzyme (ACE) inhibitors, lipoic acid, and bradykinins also improve insulin action (12). Potential insulin enhancing agents of the future might include insulin-like growth factor-1 and human growth hormone (14).

Other agents can directly effect glucose metabolism. However, they do not have the long-lasting effects of insulin-like agents (14). Dehydroepiandrosterone (DHEA), imidazole cabamide ribosides, and isoferulic acid inhibit hepatic gluconeogenesis. The phenacylimidazolium compound LY177507 works by suppressing glucose-6-phosphate translocase, which inhibits hepatic glucose output. Fatty acids are a source for gluconeogenesis and can be inhibited by substances such as oxirane caroxylate and alkylglycidates. Both agents work because they inhibit the enzyme carnitine palmitoyltransferase. Fibrates lower triglyceride concentrations, which reduces insulin resistance. Other lipid-lowering agents include nicotinic acid analogue acipimox (15).

Some other future pharmacological agents might include glucagon antagonists, anti-obesity agents, and insulin releasers. Glucagon antagonists have the potential to work because glucagon is a natural agent that suppresses the effect of insulin. Anti-obesity agents have the potential to be effective by reducing adipose tissue, which relieves insulin resistance. Current anti-obesity agents that have had some success include orlistat, pramlintide, and subutramine (16). Insulin releasers are designed to mimic the action of the pancreatic beta cell. Repaglinide and nateglinide have been introduced before meals to mimic the effects of beta cells (14). Perhaps the most promising would be the possible engineering of new pancreatic beta cells in the form of gene therapy to replace the old, worn down beta cells (14).

The Carbohydrate Debate:

The dietary recommendations for DM have evolved over the years. Before insulin could be administered as therapy, diet was the only therapy available. The old recommendations were simple; first you reduced carbohydrates (carbs). If this didn't work, you also reduced protein to take out the possibility of gluconeogenesis. If this didn't work, you reduced overall calorie intake. Besides the obvious problem of compliance, these recommendations were over-simplified. However, the basic premise was correct and just reducing major foods and stimulating weight loss worked for several individuals (17).

After the advent of insulin, the recommendations changed to allow DM persons to be more liberal with their food choices. Carbs were initially recommended to be 35%-40% of total food energy and patients were expected to weigh food. When this proved difficult, an exchange system consisting of 6 items (milk, vegetable, fruit, bread, meat and fat) was developed. Each portion of food in the exchange was to have the correct proportion of carbs, protein, and fat. Again, calculation was necessary and compliance with the exchange system was poor.

Soon, a concern over high-fat diets leading to atherosclerosis led the Food and Nutrition Committee of the American Diabetes Association (ADA) to recommend an increase in carbs to 45% in 1971, 50-60% in 1979, and 55-60% in 1994. Scientists were finding that more and more carbs could be introduced into the diet without causing insulin resistance. Thus, in 2003, the current recommendations were instated by the ADA. Now, an undetermined combination of monounsaturated fatty acids (MUFAs) and carbs is to constitute 60-70% of total caloric intake (18,19). This current

recommendation is vague. In fact, the national vice president for clinical affairs at the ADA, Nathanial Clark, has recently been quoted saying "There is no longer a diabetic diet. People with diabetes eat the exact same foods as anyone else... there is no harm in eating carbohydrates" (4). With the ADA's history in mind, it is clear that many issues with regards to carbs, fats, and proteins must be understood in order for the DM population to have a clue as to what constitutes a healthy diet for them.

The newest and perhaps most important revelation from the ADA with regards to carbs is that the diet must include the right type of carbs. Mainly, carbs should come from mostly vegetables, fruits, and whole grains to ensure adequate fiber and micronutrients (6). Metabolic studies compared the glycemic response in individuals with DM after consuming isocaloric sources of carbohydrate in the form of glucose, fructose, sucrose, potato starch, and wheat starch. Fructose ingestion led to a lower postprandial glycemic response, but other forms of carbs had nearly identical responses (20). The reason proposed for this is that fructose is primarily taken up by the liver with limited glucose release (21).

Glycemic Index:

Glycemic index (GI) is defined as the incremental rise in plasma glucose relative to that induced by a standard, usually 50 g glucose or a white bread challenge (22). Glycemic load is the amount of carbohydrate multiplied by its GI (23). GI is important because a diet with a high GI has been linked to heart disease while a diet with a low GI has been linked to an increase in HDLs, a decrease in LDLs, apolipoproteins, and PAI-1 (7). The GIs of foods are known, but many factors complicate the easy use of them for

monitoring blood sugar (18). Cooking gelatinizes starches, making them more apt to be hydrolyzed by pancreatic amylase, which raises the GI accordingly. Second, carbs with a dense food matrix, like spaghetti, hinder the action of amylase. Some carbs are digested slowly such as legumes and long-grained rice. Slower digestion results in lower GI. Perhaps the most complicating factor is that the presence of fat in combination with a starch can reduce its GI.

The positive effects of strict GI control on microvascular effects has been well established (18). However, a recent 2-year trial concerning intensive glycemic control on left ventricular function in DM persons revealed essentially no difference in the short term on macrovascular complications of DM (23, 24). In contrast, intense glycemic control for 2 years did make a difference with regards to microalbuminuria in DM persons but may not lessen the deterioration of glomerular function (25). It is clear that the amount and type of carb to consume is confusing. When assessing carb consumption, the DM patient must consider avoiding processed sugars and eating plenty of fruits, vegetables, and whole grains. When buying bread as a form of grain, the DM patient should take care to see that the label says either "whole grain" or "whole wheat" bread.

Fat and Protein Issues:

Those with DM need to have a particular concern over dyslipidemia and cholesterol. The issue over how much and what type of fat that ought to be consumed is under some debate. It was found that polyunsaturated fatty acids decrease serum LDLs and cholesterol while saturated fatty acids raise them. MUFAs did not affect LDLs and cholesterol at all (26). Polyunsaturated fatty acids have been associated with breast

carcinoma (27). Therefore, fat in the diet for DM persons should be primarily MUFAs such as olive or canola oil (28). Most sources agree that the saturated fatty acids in the diet should not exceed 10% of total calorie intake (26).

High MUFA diets have been tried in many studies as an alternative to a high-carb, low-fat diet for DM (29). In Garg's meta-analysis of 10 randomized crossover trials comparing isoenergetic high-MUFA and high-Carb diets in patients with DM, he concluded that consumption of high-MUFA diets improved fasting and post-prandial glucose and 24-h glucose and insulin profiles while having no effect on fasting insulin and glycated hemoglobin concentrations or insulin sensitivity (29). In another study, 2 low fat diets were compared with a high-MUFA diet and no difference was noted in the glycemic control (30). Similarly, Rodriguez-Villar et all reported no difference between the glycemic control in subjects consuming high-carb vs. high-MUFA (31). Thus, reducing carbs a bit and introducing MUFAs into the diet will not only improve satiety and compliance with a healthy diet, but the MUFAs might actually improve lipid profile.

Good sources of MUFA include olive oil, canola oil, sunflower oil, safflower oil, macadamia nuts, hazelnuts, pecans, almonds, cashews, peanuts, pistachios, avocados, and olives (32). Some processed animal products are also relevant sources of dietary MUFA, but must be consumed with caution because of containing a relatively high amount of saturated fat. High-MUFA animal products include ground beef, fried eggs, butter, and fried bacon. These items are considered better than items with only saturated fat content because of the high MUFA content (32). Many MUFA-rich foods of vegetable origin contain antioxidant properties as well. These properties may beneficially influence atherogenesis beyond the fatty acid composition. Extra-virgin olive oil is considered

better than processed olive oil because many of the pytochemical and antioxidant properties of olive oil are lost when it is processed. Also, heating olive oil at frying temperature in air may destroy antioxidant properties. Nuts may lose these antioxidant properties when roasted or peeled (33).

Many studies have reported that circulating Triacylglycerols (TAGs) after a meal may contribute to the development of arteriosclerosis, a primary concern for DM patients (34). In the comparison of high-MUFA and high-carb diets, no difference was found in the postprandial TAG component. However, it has been found that long-chain n-3 Polyunsaturated fatty acids (PUFAs) may lower TAG concentrations and have a beneficial effect on cardiac electrical conduction (35). Fish Oil is the chief source of n-3 PUFAs. The issue of fish oil causing a worsening of glycemic control has recently been investigated. It has been found that fish oil can have the favorable effect of lowering triglyceridemia without the adverse effects on hyperglycemia if intake does not exceed 3 g/d (5).

Another current issue concerns what type of protein a DM person should consume. The Brenner Hypothesis states that excessive protein intake causes hyperfiltration and glomerular hypertension resulting in progressive deterioration of kidney function, which causes nephropathy in some diabetes patients (36). The available evidence from studies shows that substituting soy protein for animal protein might have beneficial effects on diabetes patients. The soy protein is associated with less postprandial hyperfiltration and albuminuria (37). However, this finding is yet to be appropriately documented. What has been documented is the beneficial effect of a low protein diet verses a high protein diet for diabetic patients (37). A low protein diet does

not cause the hyperfiltration and glomerular hypertension that DM persons are prone to as a result of DM.

Fiber, Trace Elements, CLA, Biotin, and Homocysteine:

Fiber is an additional issue. Fiber is though to be beneficial in the prevention and treatment of chronic diseases including DM (38). In studies using high-fiber, high-carb diets with initial carbohydrate contents of 70% of total energy intake, glycemic control improved and cholesterol was improved (39). In other studies combined with exercise, the high-fiber diet also lowered triglycerides by 27% (40). Patient with DM who consumed a diet containing food naturally rich in fiber (50g fiber/day, 50% soluble) had significant improvements in glycemic control and lipid panels (40). The vegetarian diet involving plant foods over animal-based products has been studied with regards to the added fiber content in plant foods. However, it is difficult to isolate the effects of the added fiber as a scientific variable because weight loss usually occurs as a result of the diet. It is thought that weight loss may result from the vegetarian diet partially due to the added bulk (fiber). The bulk adds a component of satiety that may influence weight loss.

It has recently been shown that deficiencies in magnesium, chromium, and zinc may be associated with insulin resistance and that supplementation with these minerals may improve glucose oxidation and insulin resistance (14). Chromium may also reduce total cholesterol concentrations. Selenium and vanadium have been associated with improved glucose metabolism without increasing insulin secretion. Vanadium salts inhibit phosphatases that deactivate insulin receptors. Vanadium salts may also reduce appetite and decrease the rate of intestinal glucose absorption. Vanadium must be used at

low dosages to avoid toxicity. Other agents that might have similar effects include benzonaphthofurans and thiophenes (14).

Of note are conjugated linoleic acid (CLA) and Biotin. CLA is found in beef, lamb, and dairy. Diets containing CLA reduced adipose mass in various animal studies (41). It was thought that the t10c12-CLA isomer might be the bioactive isomer of CLA. Supplementation with this isomer might be of assistance for overweight DM sufferers. The joint administration of biotin and chromium picolinate has been said to combat insulin resistance, improve beta-cell function, enhance postprandial glucose uptake by both liver and skeletal muscle, and inhibit excessive hepatic glucose production. Glucokinase, expressed in hepatocyte and pancreatic beta cells, has a counter-regulatory role in glucose metabolism. Glucokinase is required for hepatic glucose output and gluconeogenesis. High-dose Biotin works by suppressing gluconeogenesis and promoting the transcription and translation of glucokinase getting hepatocytes. Chromium picolinate improves insulin sensitivity, which is unlikely to be directly influenced by Biotin (42).

Increased levels of homocysteine have been associated with increased cardiovascular risk (18). The most common causes of homocysteinemia are limited intake or availability of folate, vitamin B6, and vitamin B12. Doses of 200-400ugms of folate per day are necessary for a maximal reduction in homocysteine levels. In a study of 631 people in the Netherlands, a 1.6-fold stronger association with a cardiovascular risk due to homocysteinemia was found in DM persons vs. non-DM persons. Therefore, it may be more important for DM persons to monitor homocysteine levels and supplement with folate to reduce elevated levels. Deficiencies in B6 or B12 ought to be

ruled out before assuming that folate is the necessary supplement. Smokers are especially prone to deficiencies in B6 (18).

Antioxidants:

The literature contains many claims that free radicals or reactive species are of great importance in DM (43). Oxidative stress is defined as a serious imbalance between oxidative species and antioxidants. Some attention ought to be given to the antioxidant nutritional status of DM patients. For example, the antioxidants: vitamin E, vitamin C, and alph-tocopherol are shown to be reduced in DM patients with a fat-restricted diet (44). Oxidative Stress is a concern because it leads to the damage of all of our molecular structures including DNA, proteins, and lipids. As a result of oxidative stress, cells can die via necrosis or apoptosis.

A few important associations have been made in regards to dietary antioxidant treatment for the DM patient. Some clinical studies suggest a relationship between insulin-sensitivity and antioxidant treatment. It is unknown whether this relationship is modulated by the insulin receptor-signal transduction cascade or whether metabolism improves indirectly by an improvement of endothelial function (44). The metabolism of essential fatty acids is rate-limited by the desaturation steps that are inhibited by diabetes. Depressed hepatic desaturation results in lower plasma levels of the n-6 metabolites, gamma-linoleic acid (GLA) and arachnidonic acid. This reduces synthesis of the vasodilator, prostacyclin, by the vasa nervorum (45). This desaturation deficit may be bypassed by GLA or arachnidonic acid treatments, which improve nerve blood flow and NCV. Endothelium-derived hyperpolarizing factor has been shown to be decreased in

DM. This deficit was attenuated by antioxidant treatment with alpha-lipoic acid (44). As a general antioxidant recommendation, vitamins A, C, and E are shown to have positive consequences concerning metabolic control and late complications of DM.

Conclusion

Current literature emphasizes a practical dietary approach for DM that might apply to anyone wishing to improve his health with diet. A high intake of vegetables, legumes, fruit, and whole grains combined with a low intake of red meat, processed meat, high-fat dairy, and refined grains is recommended based on a variety of beneficial outcomes for the DM patient. Contrary to what was thought for a long time, the DM diet does not have to be a low fat one. A variety of vegetable foods rich in MUFAs are good sources of antioxidants and lipid control and will likely increase palatability and compliance. Vegetable sources of MUFAs such as high-oleic acid oils and nuts should be incorporated into the DM diet.

Current recommendations provide that the intake of MUFAs and carbs combined should compose 60-70% of total caloric intake. The relative allocation of intake between the two has not been specifically recommended. The clinical utility of the GI has been challenged. While it has been shown that lower GI foods are likely better for the DM patient, numerous variables can alter the GI, even for a given source of carbohydrate. This makes compliance to GI recommendations increasingly difficult. With regards to carbs and MUFAs, special care should be taken to ensure that an adequate amount of fiber and micronutrients are consumed. For this reason, the vegetarian diet might meet

the needs of DM individuals. Likewise, the vegetarian diet including fibers, vegetable proteins, plant sterols, and nuts may improve lipid abnormalities.

Consideration for supplementation must be given due to the role of magnesium, chromium, zinc, selenium, and vanadium on insulin resistance. Biotin and chromium picolinate when used in combination have also been shown to improve insulin sensitivity. An assessment of B6, B12, and folate ought to be done in order to ensure adequate control over homocysteine levels. CLA should be considered if an individual is having trouble losing weight. Further study must be done on the effects of soy protein vs. animal protein, and whether or not protein would still cause damage in a DM person with good glycemic control as a result of consuming the proper type and amount of carbs and fats.

No matter what dietary measures are taken, the DM person must consider his or her obesity and energy balance above all. Weight loss can be best achieved by a combination of diet and exercise. It would be remiss for an individual to limit his DM management strategy to diet alone. A need for new antidiabetic therapies is well recognized. In addition to diet therapy, numerous compounds with blood glucose-lowering activity are being evaluated, and considerable interest is focused on relieving insulin resistance and retrieving beta-cell function. In the longer-term, it is hoped that early intervention with gene therapy will be a feasible prospect. Despite medical advances, one would be foolish to ignore the evidence; a proper diet, exercise, and subsequent weight loss can essentially reverse the devastating effects of DM.

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