The Role Of Carbohydrate In Diabetic Nutrition: A Review

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Abstract

This review looks at the definition of carbohydrate and its classification; the type and effect of food processing that affect blood glucose and insulin response to carbohydrate. The glycaemic index of carbohydrate food and its role in nutritional management of diabetes was emphasized. The goals of MNT in diabetes management were briefly mentioned.

INTRODUCTION

Carbohydrates play a major role in human diets, comprising some 40-75% of energy intake. Their most important nutritional property is digestibility. In an attempt to prepare food tables for diabetic diet, McCance and Lawrence in 1929 [1] divided dietary carbohydrates into available and unavailable carbohydrates. Available carbohydrates are those that are hydrolysed by enzymes of the human gastrointestinal system to monosaccharides that are absorbed in the small intestine and enter the pathways of carbohydrate metabolism, while unavailable carbohydrates are not hydrolysed by endogenous human enzymes, but may be fermented in the large intestines to varying extents.

Carbohydrate may be define as "polyhydroxy aldehydes, ketones, alcohols, acids, their simple derivatives and polymers having polymeric linkages of the acetal type". [2] Carbohydrates are further classified according to the degree of polymerization as sugars (mono- and disaccharides); oligosaccharides (three to nine monosaccharide units); and polysaccharides (ten or more monosaccharide units). [3] Each of these three groups may be subdivided on the basis of the monosaccharide compositions. Sugars comprise monosaccharide, disaccharides and polyols (sugar alcohols). Oligosaccharides include malto-oligosaccharides, principally those occurring from the hydrolysis of starch, and other oligosaccharides, e.g. α-galactosides (raffinose, stachyose etc.) and fructo-oligosaccharides. The polysaccharides may be divided into starch (α-glucans) and non-starch polysaccharides of which the major components are the polysaccharides of the plant cell wall such as cellulose, hemicelluloses and pectin.

A number of carbohydrates are only partly or not at all digested in the small intestine and are fermented in the large bowel to short chain fatty acids. These include the non-digestible oligosaccharides, resistant starch and non-starch polysaccharides. The process of fermentation is metabolically less efficient than absorption in the small intestine and these carbohydrates provide the body with less energy.

METABOLISM OF CARBOHYDRATE MEALS

The digestion of dietary carbohydrates starts in the mouth, where salivary α-amylase initiates starch degradation. The starch fragments thus formed include maltose, some glucose and dextrins containing the 1,6-α -glycosidic branching points of amylopectin. The α-amylase degradation of starch is completed by the pancreatic amylase active in the small intestine. Dietary disaccharides, as well as degradation products of starch, need to be broken down to monosaccharides in order to be absorbed. This final hydrolysis is accomplished by hydrolases attached to the intestinal brush-border membrane, referred to as "disaccharidases". Glucose and galactose are transported actively against a concentration gradient into the intestinal mucosal cells by a sodium dependent transporter (SGLT 1). Glucose is pumped out of the enterocyte into the extracellular space by the glucose transporter 2 (GLUT 2). Fructose undergoes facilitated transport by another mechanism (GLUT 5).

When delivered to the circulation, the absorbed carbohydrates cause an elevation of the blood glucose concentration. Fructose and galactose have to be converted to glucose mainly in the liver and therefore produce less pronounced blood glucose elevation. The extent and duration of the blood glucose rise after a meal is dependent upon the rate of absorption, which in turn depends upon factors such as gastric emptying, as well as the rate of hydrolysis and diffusion of products of hydrolysis in the small intestine.

Insulin is secreted as a response to blood glucose elevation but is modified by many neural and endocrine stimuli. Insulin secretion is also influenced by food related factors, especially by the amount and the amino acid composition of dietary proteins, particularly arginine, lysine, leucine and phenylalanine. Insulin has important regulatory functions in both carbohydrate and lipid metabolism and is necessary for glucose uptake by most body cells.

Fermentation is the colonic phase of the digestive process and describes the breakdown in the large intestine of carbohydrates not digested and absorbed in the upper gut. This process involves gut microflora and is unique to the colon of humans because it occurs without the availability of oxygen. It thus results in the formation of gases e.g. hydrogen, methane and carbon dioxide; as well as short chain fatty acids (SCFA) (acetate, propionate and butyrate) which are rapidly absorbed and metabolized by the body. The gases are either absorbed and excreted in breath, or passed out via the rectum. [4]

DIETARY CARBOHYDRATES AND FOOD PROCESSING

Gelatinization: - Gelatinization refers to the irreversible loss of the crystalline regions in starch granules that occur upon heating in the presence of water. Gelatinization increases the availability of starch for digestion by amylolytic enzymes. Starch granules are not completely dissolved during food processing, thus food can be regarded as dispersion in which starch granules and/or granular remnants constitute the dispersed phase. The degree of gelatinization achieved by most commonly used food processes, however, is sufficient to permit the starch to be rapidly digested. Consequently even food processes, which result in a low degree of gelatinization (e.g. steaming and flaking of cereals), produce postprandial blood glucose and insulin increment similar to that with completely gelatinized foods. [56]

Retrogradation: - Gelatinized starch is not in thermodynamic equilibrium. There is, therefore, a progressive re-association of the starch molecules upon ageing of the starch molecules [7] . This recrystallisation is referred to as retro gradation, and may reduce the digestibility of the starch. The retro gradation of the amylopectin component is a long-term phenomenon occurring gradually upon storage of starchy foods. Amylose, however, re-associates more quickly. The crystallinity of retrograded amylopectin is lost following re-heating to approximately 70°C, whereas temperatures above 145°C are required to remove crystallinity of retrograded amylose. This is a temperature well above the range used for processing of starchy foods. This implies that retrograded amylose, once formed, will retain its crystallinity following re-heating of the food. [8]

Par-boiling: - During par-boiling of rice, the kernels are subjected to a pre-treatment involving heating and drying. This process reduces the stickiness of the rice, possibly by allowing leached amylose to retrograde and/or form inclusion complexes with polar lipids on the kernel surface. Parboiling also affects the final cooking properties of the rice. [8]

GLYCAEMIC INDEX OF FOOD

Epidemiological studies [9] have reported that as nations become more affluent, the nature of the people's carbohydrate consumption changes such that the ratio of complex (starches) to simple carbohydrates decreases. It has been suggested that this change in dietary pattern is responsible for the occurrence of various diseases, such as atherosclerosis, diabetes and hyperlipidaemia. One proposed physiologic basis underlying such suggestions is a traditionally held tenet that simple carbohydrates are more readily available for immediate absorption by the gut than are more complex carbohydrates and that they therefore produce a greater and faster rise in postprandial plasma glucose and insulin responses than do the supposedly more gradually digested and absorbed complex carbohydrate. Consequently, diets restricted in simple carbohydrates have been recommended in disease states in which control of plasma glucose and/or insulin is felt to be important. Dahiqvist and Borghstrom [10] as well as Fogel and Gray [11] have challenged this concept of carbohydrate digestion and absorption. These workers demonstrated that after test meals, more than enough intraluminal amylase is present to rapidly hydrolyze ingested starch. They concluded that absorption, not intraluminal digestion, was the rate-limiting step in over-all starch assimilation.

It has also been shown [12] that complex carbohydrates resulted in lower glucose and insulin responses than equivalent amounts of glucose or sucrose, indicating that despite adequate amylase, the process of starch digestion proceeds more slowly. It was also found that glucose and insulin responses to cooked potato were significantly higher than the glucose and insulin responses to rice. These latter findings suggest that not all starches are treated identically by gastrointestinal digestive and absorptive processes.

Evidence favouring the active reduction of blood lipids continues to accumulate, and several major diabetes associations now recommend that diabetic patients should reduce fat intake and increase carbohydrate intake to approximately 50% of total calories. Since one aim of diabetic therapy is to prevent large fluctuations in blood glucose throughout the day diabetics are advised to select carbohydrate foods that minimize the postprandial blood glucose excursions. In the absence of adequate information specific advice on food selection is not given, although high-fibre foods have been advocated [13] . Before detailed advice can be given, comparative data on the physiological effects of carbohydrate foods are required.

Otto et al recorded large differences in glycaemic response between carbohydrate foods in diabetic patients. [14] One of the aims of such studies was to develop tables from which diabetic diets could be constructed based on the biological equivalence of the foods prescribed. A similar method that permits such comparisons involves classification of foods in terms of their glycaemic indices (GI) [1516]

Figure 1

$$GI = \frac{\text{Blood glucose area under curve of test food}}{\text{Blood glucose area under curve of standard food}} \times 100$$

where the valuable carbohydrate content of the test and reference foods is the same. This method allows results of food testing in individuals with different glucose tolerance to be pooled and comparison made between findings of different investigators. In general, results of studies in young adult, normal weight, non-diabetic volunteers agree well with those in middle-aged and elderly, overweight, diabetic patients. [14]

DEFINITION OF GLYCAEMIC INDEX

The blood glucose responses of carbohydrate foods can be classified by the glycaemic index (GI). The GI is considered to be a valid index of the biological value of dietary carbohydrates. It is defined as the glycaemic response elicited by a 50g carbohydrate portion of a food expressed as a per cent of that elicited by a 50g carbohydrate portion of a standard food. [17] The glycaemic response is defined as the incremental area under the blood glucose response curve, ignoring the area beneath the fasting concentration (i.e. the area beneath the curve). [91819] The standard food has been glucose or white bread. If glucose is the standard, (i.e. GI of glucose = 100) the GI values of foods are lower than if white bread is the standard by a factor of 1.38 because the glycaemic response of glucose is 1.38 times that of white bread. GI values for several hundreds foods have been published. [2021]

FACTORS INFLUENCING GLYCAEMIC RESPONSE

Type of carbohydrate: Crapo et al studied the effects of four different kinds of dietary starch (potato, rice, corn, and wheat) on postprandial plasma glucose and insulin responses and showed that there is a range of plasma glucose and insulin responses to different starches, with rice and corn producing the lowest response curves. [9] In an earlier study, [12] Crapo proposed that the rate of digestion and absorption is not the same for all orally ingested starch molecules. The mechanism(s) for the differences between starches is not clear, although gastric emptying time, physical availability of starch to hydrolytic enzymes, and differences in the stimulation of gastrointestinal insulinogenic hormones may be factors.

Physical form of carbohydrates: Altering the physical form of a complex carbohydrate changes the postprandial glucose and insulin responses to it. [19] It has been demonstrated that a close correlation exists between the rates at which the starch in ground rice and unground rice was hydrolyzed in vitro and the glucose and insulin responses to equal loads of these different physical forms of rice in vivo. [20] Evaluation of brown rice and white rice demonstrated that there was no significant difference in glycaemic response between whole white rice and brown rice but glycaemic response to both were significantly and dramatically higher when the rice was ground into flours. [19] Similar results were seen with whole and ground lentils [21] . Pureed apples and apple and orange juice elicit higher blood insulin responses than do whole apples or oranges. [2223] Thus on the basis of these and other results it has been suggested that the rate of digestion and absorption of complex carbohydrates are critical factors in determining the metabolic response to dietary carbohydrates, and that this principle could be applied to the design of diets for the treatment of diabetes. Choosing foods that are most slowly digested and absorbed should flatten the postprandial glucose response curve and thereby reduce the insulin requirement. O’Dea and Collier showed that the flattening of the postprandial glucose curve after rice relative to ground rice or glucose was more striking in diabetic than in normal subjects. [24] This underlines the importance of not simply recommending that diabetics increase their consumption of complex carbohydrates to improve metabolic control. The actual form of the complex carbohydrates is critical in determining the metabolic response to it. Booher et al [25] in 1951 stated that conditions which increase the digestibility of starches include those modifications which produce obvious hydration of the granules, distinct from changes in chemical nature, or disruption of the organized structure. In general, it appears that the more change in physical form a food is, the higher the glycaemic response it will produce.

Viscosity: Certain gelling fibers (guar gum, pectin, tragacanth) when mixed with glucose during a glucose tolerance test result in a dose dependent flattening of postprandial glucose and insulin responses which has been attributed to the viscosity of the fiber. In vitro studies demonstrating a reduced rate of passage of glucose out of a dialysis sac when these gelling fibers were present support this suggestion. Similarly, it has been shown that carbohydrates of leguminous origin such as lentils and red kidney beans result in flattened postprandial glycaemic responses reminiscent of the guar-glucose mixture. [21]

Cooking: Cooking not only increases the viscosity but also splits the starch granules, thereby increasing the availability of the starch to amylase. The responses of serum glucose and insulin to both cooked and raw starch were studied by Collings et al. [26] It was observed that the response of serum glucose to glucose monohydrate and cooked starch were closely similar, while that to raw starch was significantly less. The serum insulin response was greatest with glucose monohydrate meal, and the area under this response curve was significantly greater than that after the cooked starch meal, which in turn was significantly greater than that after starch meal. The effect of moist and dry heat on in vivo and in vitro legume starch digestibility showed that boiling and pressure-cooking resulted in faster rates of digestion than roasting [27] . In addition, Jenkins et al [28] found that drying cooked red lentils in a warm oven for 12h resulted in a significantly enhanced glycaemic response and rate of in vitro starch digestion compared with lentils boiled for 20 minutes. Therefore, the type and time of cooking may influence the in vivo and in vitro digestibility of carbohydrate foods.

Enzyme Inhibitors and Anti-nutrients: Enzyme inhibitors and lectins have been shown to produce hypoglycemia and decreased growth rate in rats. [27] Furthermore, it has been shown that starch digestion may be inhibited in the gastrointestinal tract by anti-nutrients. Certain amylase inhibitors are known to cause decreased glucose absorption in dogs, rats, and humans as judged by peripheral blood glucose response. [29] Amylase and sucrase inhibitors, which have been shown to reduce the rate of carbohydrate digestion and absorption, have been used in the treatment of diabetes. Inhibition of intestinal α-glucosidases delays the digestion of starch and sucrose and flattens postprandial blood glucose excursions in type 2 diabetes; the α-glucosidase inhibitor, acarbose, is used widely in the management of type 2 diabetic patients. Furthermore, phytic acid supplements added to unleavened white bread decreased rates of release of starch digestion products in vitro, and decreased blood glucose responses compared with plain unleavened white bread. [27]

CLINICAL SIGNIFICANCE OF GLYCAEMIC INDEX

More than twenty years ago the first index of the relative glycaemic effects of carbohydrate exchanges from 51 foods was published by Jenkins et al. [15] Despite controversial beginnings, the GI is now widely recognized as a reliable, physiologically based classification of foods according to their postprandial glycaemic effects.

In 1997 the Food and Agriculture Organization (FAO) of the United Nations and the World Health Organization (WHO) reviewed available research evidence regarding the importance of carbohydrates in human nutrition and health, [8] and endorsed the use of the GI method for classifying carbohydrate-rich foods and recommended that the GI values of foods be used in conjunction with information about food composition to guide food choices.

Reliable tables of GI compiled from the scientific literature are instrumental in improving the quality of research examining the relation between the dietary glycaemic effect and health. Several studies from Harvard University indicate that the long-term consumption of a diet with a high glycaemic load (GL) (GL= GI x dietary carbohydrate content) is a significant independent predictor of the risk of developing type 2 diabetes and cardiovascular disease. More recently, evidence has been accumulating that a low-GI diet might also protect against the development of obesity, colon cancer, and breast cancer. [30]

The EURODIAB (Europe and Diabetes) study, involving >3000 subjects with type 1 diabetes in 31 clinics throughout Europe, showed that the GI rating of self-selected diets was independently related to blood concentrations of glycated haemoglobin in men and women [31] and to waist circumference in men. [32] In addition, higher blood HDL-cholesterol concentrations were observed in patients consuming low-GI diets from the northern, eastern, and western European centers participating in the study. [32] In parallel with these advances there have been studies documenting the importance of postprandial glycaemia per se for all-cause mortality and cardiovascular disease mortality in healthy populations. [33] In the Hoorn study there was a significant association between the 8year risk of cardiovascular death and 2hour postload blood glucose concentrations in subjects with normal fasting glucose concentrations, even after adjustment for known risk factors. [34] Multiple mechanisms are probably involved. Recurring, excessive postprandial glycaemia could decrease blood HDL-cholesterol concentrations, increase triglyceridaemia, and also be directly toxic by increasing protein glycation, generating oxidative stress, and causing transient hypercoagulation and impaired endothelial function. [30] If postprandial glycaemia is indeed important, then dietary treatment for the prevention or management of chronic diseases must consider both the amount and type of carbohydrate consumed. In the USA three intervention studies in adults and children with type 1diabetes showed that low-GI diets improve glycated haemoglobin concentrations. [30] In subjects with cardiovascular disease, low-GI diets were shown to be associated with improvements in insulin sensitivity and blood lipid concentrations. [30]

In addition, evidence from both short-term and long-term studies in animals and humans indicate that low-GI foods may be useful for weight control. Laboratory studies examining the short-term satiating effects of foods have shown that low-GI foods are relatively more satiating than are their high-GI counterparts. [35] Compared with low-GI meals, high-GI meals induce a greater rise and fall in blood glucose and a greater rise in blood insulin, leading to lower concentrations of the body's 2 main fuels (blood glucose and fatty acids) in the immediate postabsorptive period. The reduced availability of metabolic fuels may act as a signal to stimulate eating.

It is also important to emphasize that many low-GI foods are relatively less refined than are their high-GI counterparts and are more difficult to consume. The lower energy density and palatability of these foods are important determinants of their greater satiating capacity. In obese children, the ad libitum consumption of a low-GI diet has been associated with greater reductions in body mass indexes. [36] However, some experts have raised concerns about the difficulties of putting advice about GI values into practice and of the potentially adverse effects on food choice and fat intake. For this reason, the American Diabetes Association does not recommend the use of GI values for dietary counseling. However, the European Association for the Study of Diabetes, [37] the Canadian Diabetes Association, [38] and the Dietitians Association of Australia [39] all recommend high-fiber, low-GI foods for individuals with diabetes as a means of improving postprandial glycaemia and weight control.

In a meta-analysis it was concluded that choosing low-GI foods in place of conventional or high-GI foods has a small but clinically useful effect on medium-term glycemic control in patients with diabetes. [40]

NUTRITIONAL MANAGEMENT OF DIABETES MELLITUS

The prevalence of diabetes mellitus (DM) is increasing around the world and at a rate that appears as dramatic as to have been characterized as an epidemic. 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. 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). [41] Before the advent of insulin therapy in the early 20th century, MNT was the only form of therapy for DM. [42]

The goals of MNT for diabetes include:

1. Achieve and maintain near normal blood glucose goals
2. Achieve and/ or maintain optimal blood lipid levels
3. Achieve and/ or maintain normal blood pressure
4. Prevent, delay or treat nutrition related complications
5. Provide adequate kcalories for achievement of reasonable body weight
6. Provide optimal nutrition for maximizing health and for growth, development, pregnancy, and lactation

DIABETES MELLITUS AND SUGARS

The ADA recommends that the classifications sugars, starch, and fiber be used as the functional definitions of carbohydrates for MNT. Previously used terms such as simple sugars, complex carbohydrates, and fast-acting carbohydrates are now discouraged from further usage. With respect to carbohydrates, the key emphasis of MNT for DM is on the total amount of carbohydrate in terms of energy intake. [43] 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. [41]

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. [41] This perception of the sensitivity of metabolic control in DM to energy balance underlies the recommendations that emphasise carbohydrate content as a key point for patient education. The 3 points stressed are:

1. Knowledge concerning which foods contains 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 is appropriate within a meal or snack.

Negative energy balance can promptly induce reductions in hyperglycaemia and hypertriglyceridaemia, even before the achievement of substantial weight loss, whereas consumption of excess energy has the opposite effect. Increased energy consumption regardless of source, but certainly including carbohydrate, directly induces insulin resistance. [43]

Metabolic studies have been used to compare the glycaemic response to sugars consumption in persons with DM with isocaloric consumption of other sources of carbohydrates. Bantle et al [44] compared the postprandial glycaemic response to various forms of carbohydrates (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 glycaemic response in those with DM, but the other forms of carbohydrate had nearly identical responses. The recommendation is that if sucrose is consumed, it should be substituted for other carbohydrates.

DIABETES MELLITUS AND DIETARY FIBRE

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 glycaemic control, HDL and LDL cholesterol, and triacylglycerols. [45] However, a 3-month study by Jenkins et al [46] did not find a metabolic advantage of high-fiber over low fiber cereals. Also Erasmus et al [47] in a 3week study showed that treatment with guar gum does not lower the postprandial glucose level in both non-diabetic and diabetic Nigerian subjects

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. [48]

DIABETES MELLITUS AND THE DIETARY GENOME

The progression from a healthy state to a chronic disease state such as diabetes mellitus must occur by changes in gene expression or by differences in activities of proteins and enzymes. Since dietary chemicals are regularly ingested and participate indirectly and directly in regulating gene expression, it follows that a subset of genes regulated by diet must be involved in disease initiation, progression, and severity (79, 113). The clearest example of genotype-diet interactions in chronic disease is type 2 diabetes; once diagnosed with type 2 diabetes, some individuals can control symptoms by increasing physical activities and by reducing caloric intake, thus the expression of genomic information is changed by changing environmental (dietary) variables.

The link between high GI diets and diabetes may relate to glucose peaks and increased insulin demand. High GI foods lead to rapid rises in blood glucose and insulin levels. Hyper-insulinemia, in turn, may down-regulate insulin receptors and therefore reduce insulin efficiency, resulting in insulin resistance a known risk factor for type 2 diabetes. Low-GI foods tend to delay glucose absorption thereby resulting in reduced peak insulin concentrations and overall insulin demand. Several studies have found improvements in glycemic control with low-GI diets.

A recent study by Uyeda et al showed that feeding on a high carbohydrate diet induces transcription of more than 15 genes involved in the metabolic conversion of glucose to fat.

Carbohydrate responsive element-binding protein (ChREBP), the newly discovered transcription protein is activated in response to high glucose and up-regulates these genes. Cyclic AMP and a high fat diet inhibit ChREBP and slow down glucose utilization. ChREBP is able to control transcription of lipogenic enzyme genes in response to nutritional and hormonal inputs, and may play an important role in disease states such as diabetes, obesity, and hypertension.

CONCLUSION

Carbohydrate provides the majority of energy in the diets of most people. In addition to providing easily available energy for oxidative metabolism, carbohydrate-containing foods are vehicles for important micronutrients and phytochemicals. Dietary carbohydrate is important to maintain glycemic homeostasis and for gastrointestinal integrity and function. Unlike fat and protein, high levels of dietary carbohydrate, provided it is obtained from a variety of sources, is not associated with adverse health effects. An optimum diet should consist of at least 55% of total energy coming from carbohydrate obtained from a variety of food sources. Diabetes mellitus remains a universal health problem, and its management requires a multidisciplinary healthcare approach which includes a combination of diet, drug or insulin therapy, exercise, and behavioural modifications to ensure long-term compliance. However whether the disease is managed by insulin or oral drugs, dietary measures are necessary and inevitable to uphold the control of blood glucose, improve metabolic control and prevent complications. Despites its limitation glycemic index still has a major role to play in diabetic nutritional management. Further studies on the role of diet on gene modification in causation and management of chronic diseases will shed more light on the role of carbohydrate on diabetic nutrition.

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