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Dietary carbohydrate in the management of diabetes: importance of source and amount

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It is generally agreed that a cornerstone for managing diabetes is diet, but there is considerable controversy about what is the best kind of dietary advice. The central question is how much carbohydrate should be included in the diet. This is because the amount determines how much room is left for modifying protein and fat intake. With respect to dietary carbohydrates, current nutritional recommendations from the American Diabetes Association (ADA)[1] differ markedly from those of the Canadian Diabetes Association (CDA)[2] (Table 1). This issue of *Endocrinology Rounds*, reviews the classification of dietary carbohydrates, the acute effects of carbohydrates on glycemic responses, and the long-term effects of carbohydrates on glycemic control and blood lipids.

Classification of dietary carbohydrates

Dietary carbohydrates can be classified chemically or physiologically. The most common chemical classification is by the chain length of the carbohydrate molecule (measured as degree of polymerization DP): sugars (DP=1-2), oligosaccharides (DP=3-9), and polysaccharides (DP>9). Another chemical difference between carbohydrates is the nature of the monosaccharide subunit. The physiological classification is based on whether the carbohydrate is digested and absorbed in the small intestine or not. Carbohydrates that are absorbed from the small intestine are termed "glycemic carbohydrates," whereas, those that are not absorbed from the small intestine and enter the colon are called "non-glycemic carbohydrates."[3] As illustrated in Figure 1, chemical classification does not predict physiologic effect.

Dietary sugars include the monosaccharides, glucose and fructose, and the disaccharides, maltose (2 glucose units), sucrose (table sugar, glucose and fructose), and lactose (milk sugar, glucose and galactose). Lactose is malabsorbed by individuals with low intestinal lactase activity. Fructose absorption is facilitated by glucose, but its absorption is poor when it is the only carbohydrate consumed (occurs rarely in nature). Sugar alcohols, or polyols, are naturally present in some fruits, but are appearing more frequently in manufactured foods targeted for those with diabetes. Common polyols include the monosaccharides (sorbitol, mannitol and xylitol), and the disaccharides (lactitol, maltitol and isomalt). Approximately 50%-80% of most polyols are absorbed, except for lactitol that is almost completely malabsorbed.

There are 2 forms of starch, amylose and amylopectin; they are polysaccharides containing 10-3000 glucose units.

- Amylose consists of long unbranched chains of glucose units bound by $\alpha(1\rightarrow4)$ linkages.
- Amylopectin is a highly branched molecule consisting of a backbone of $\alpha(1\rightarrow4)$ glycosidic linkages with $\alpha(1\rightarrow6)$ branch points.

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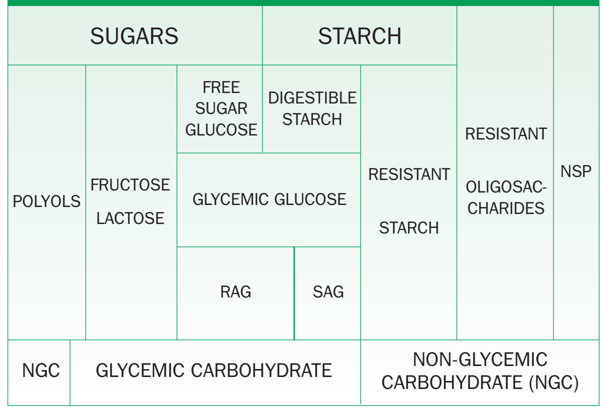
Table 1: Contrasting recommendations about dietary carbohydrates

	American Diabetes Association (2002)	Canadian Diabetes Association (1999)
Amount of carbohydrate	No recommendation	50%-60% energy
Sugars	No restriction; substitute for other CHO sources	Include added sugars up to 10% of energy
Glycemic effects of	The total amount of carbohydrate is more important than the source	The amount and source of carbohydrate in meal planning should be considered
Glycemic index	Not sufficient evidence to recommend as primary strategy	Including low GI foods may help to optimize glycemic control

Figure 1: Chemical and physiological classifications of dietary carbohydrates



Resistant starch is not digested in the small intestine. There are 4 types of resistant starch: physically inaccessible starch (protected with cell walls from attack by digestive enzymes), raw starch granules, retrograded amylose, and chemically modified starch. Hydration of starch by cooking is termed gelatinization and this makes starch more readily digestible. If gelatinized starch is allowed to cool or dehydrate, it undergoes a structural reconfiguration that is termed retrogradation, which reduces its digestibility. Digestible starch is classified into rapidly available glucose (RAG) and slowly available glucose (SAG) based on its rate of digestion in vitro.[4]

Non-starch polysaccharides (NSP) are not digested in the human small intestine. In North America, these compounds are referred to as dietary fibre.

Acute glycemic effects of carbohydrates

Nature of the monosaccharide absorbed

In both normal and diabetic subjects, fructose raises blood glucose by approximately 20% of the amount elicited by the same weight of glucose.[5,6] Presumably, this is because only a small proportion of ingested fructose is converted to glucose in the liver and released into the blood stream (Figure 2). A dose of 50 g of table sugar (sucrose, a disaccharide of glucose and fructose), elicits a glycemic response that is 60% of the response elicited by 50 g glucose; this value is equal to the consumption of 25 g of fructose plus 25 g of glucose.[5] This illustrates that the glycemic responses of combinations of carbohydrates can be predicted from their individual responses (in this case [20%+100%]/2 = 60%). Sorbitol and xylitol cause virtually no rise in blood glucose because they are only partly absorbed,[7,8] and 15%-50% of the amount absorbed is excreted in the urine.[9] Thus, only a fraction of ingested polyols is converted to glucose in the liver (Figure 2). Maltitol is a disaccharide of sorbitol and glucose. Since the glucose in maltitol is released and absorbed during digestion, chocolate sweetened with maltitol elicits virtually the same glycemic response as chocolate sweetened with sucrose.[10]

Amount of carbohydrate absorbed

As the amount of carbohydrate consumed increases, the blood glucose response increases linearly from 0 g to 50 g of carbohydrate. As intake of carbohydrate increases beyond 50 g, the dose-response curve flattens off, and the response after 100 g is only approximately 35% greater than that after 50 g carbohydrate in both normal and diabetic subjects.[5,11,12]

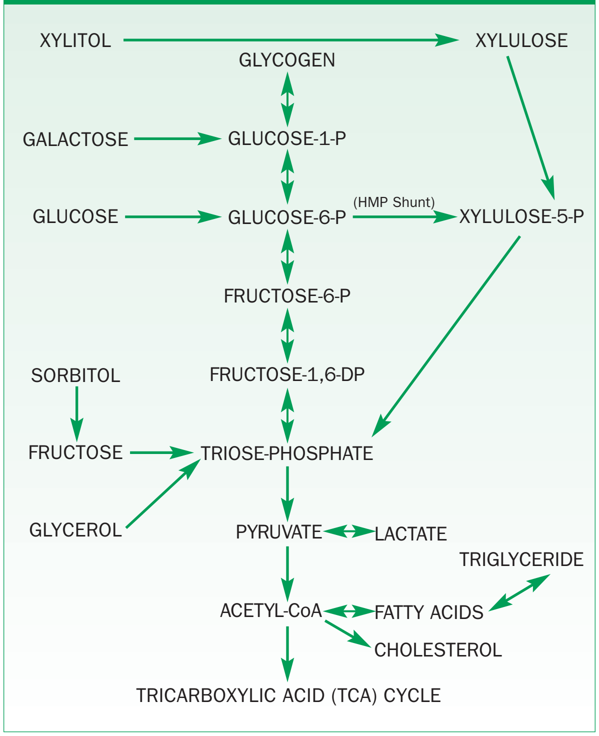
Rate of absorption of carbohydrate

The rate of carbohydrate absorption markedly influences the metabolic response elicited. The rate of absorption can be slowed by a reduced rate of intake (increased meal frequency), viscous dietary fibre, pharmacological enzyme inhibitors, and the use of slowly digested starchy foods.

Sipping 50 g of glucose evenly over 3.5 hours markedly flattened the blood glucose and insulin responses of normal subjects compared to consumption of 50 g of glucose in a bolus.[13] A similar effect has been shown in patients with type 2 diabetes.[14] The practical application of increased meal frequency is limited by the inconvenience of eating multiple meals and the tendency for increased energy intake that may offset the potential benefits.[15]

Viscous forms of dietary fibre such as guar and psyllium reduce glycemic responses and can improve glycemic control in diabetes, but fine mixing of fibre and food appears to be necessary.[16] Practical applications of this approach are currently limited by the lack of available, palatable food products.

Figure 2: Metabolism of carbohydrates in the liver



Alpha-glucosidase inhibitors reduce glycemic responses and improve glycemic control in diabetes[17] by competitive inhibition of the intestinal disaccharidases required for the digestion and absorption of most dietary carbohydrates.[18] These agents provide a useful model of the effects of reducing the rate of carbohydrate absorption, but they are not discussed further in this issue.

Many factors such as food form, particle size, cooking, processing, and starch structure affect the rate of digestion of the starch in foods, and the rate of digestion of starch is a key determinant of blood glucose and insulin responses in vivo.[19] The glycemic index (GI) is a biological classification of the potential for raising blood glucose by the carbohydrates in foods. The GI values of starchy foods are directly related to their rates of digestion in vitro.

Effects of carbohydrates on plasma free-fatty acids

The body uses as fuel whatever is consumed in the diet.[20] The two major fuels are carbohydrate and fat. Dietary carbohydrate is absorbed in the blood as glucose where it is immediately available to body tissues as a fuel. On the other hand, dietary fat is packaged into chylomicrons that transport the fat to adipose tissue primarily, where it is taken up and stored as triglycerides until needed. In the fasting state, when carbohydrate availability is low, fat is the primary fuel. The fat in adipose tissue becomes available for oxidation when the storage form, triglyceride, is broken down into fatty acids and released into the blood as free-fatty acids (FFA). High levels of FFA in the blood increase fat oxidation, inhibit the use of glucose as a fuel, and increase the output of glucose from the liver. After a carbohydrate containing meal, the rise in blood insulin shuts off the release of fat from adipose tissue reducing plasma FFA, and allowing glucose to be used by muscle. Plasma FFA concentrations are elevated in diabetes and may contribute to insulin resistance and the high blood glucose levels seen in the condition. Chronic elevations of FFA are also considered toxic to the ß-cell, and may contribute to the gradual deterioration in ß-cell function characterizing type 2 diabetes.

The extent and duration of the suppression in plasma FFA levels after eating depends on the nature and the amount of carbohydrate consumed.

Nature of the monosaccharide absorbed

Because plasma glucose and insulin concentrations tend to fall to baseline more quickly after the consumption of sucrose than after consuming starch, plasma FFA levels rebound more quickly after consuming sucrose-rich meals compared to starch-rich meals.[21] Plasma FFA concentrations also rebound more rapidly after fructose compared to starch.[22] Maltitol suppresses FFA to a similar extent as the same amount of sucrose.[23] The effect of other polyols on plasma FFA is not known.

Amount of carbohydrate absorbed

Reducing the amount of carbohydrate in a meal is associated with a more rapid rebound of plasma free-fatty acids (FFA) after eating, and an increased blood glucose concentration after a subsequent meal.[24]

Rate of absorption of carbohydrate

Sipping glucose for 3.5 h prevented blood glucose undershoot, the counter-regulatory response, and the rebound of plasma FFA seen after a glucose bolus. In addition, the disposal of an intravenous glucose load was significantly improved after glucose sipping compared to the glucose bolus.[13] High carbohydrate meals containing low GI foods are associated with a prolonged suppression of plasma FFA and improved carbohydrate tolerance to a subsequent meal.[24] Interestingly, however, after a low carbohydrate meal containing low GI foods, plasma FFA rebounded to a greater extent than after a low carbohydrate meal containing high GI foods.[24]

Effects of carbohydrates on glycemic control and blood lipids in diabetes

Nature of the monosaccharide absorbed

Since sucrose has a lower GI than many common starchy foods,[25] replacing moderate amounts of starch with sucrose causes no deterioration in glycemic control for patients with diabetes.[26] Fructose has a low GI and one small (n=13), but long-term (6 months) randomized trial showed that 60 g/d of fructose significantly improved glycemic control in poorly controlled diabetic patients (HbA_1 reduced by about 20%) with no deleterious effects on blood lipids.[27] Large amounts of fructose and sucrose are generally not recommended because of fears they may increase blood lipids. However, these effects are inconsistent and are usually not seen with moderate intakes (up to 10% energy).[28] The long-term effects of polyols on glucose and lipid control in diabetes are unknown.

Amount of carbohydrate absorbed

The effects of a low carbohydrate diet in diabetes depend on what is used to replace the carbohydrate. Low carbohydrate/high protein diets are not well studied in diabetes, though the increase in the glomerular filtration rate they elicit in non-diabetic subjects[29] is a cause for concern. However, low carbohydrate/high monounsaturated fat (MUFA) diets are well studied in diabetes and are recommended by some because of their effects on blood lipids and postprandial glucose. A meta-analysis of 10 studies of high MUFA diets in subjects with diabetes[30] showed that these diets significantly reduced serum triglycerides, significantly increased HDL cholesterol, and non-significantly reduced serum cholesterol. However, many of these studies employed larger dietary changes than are practical for most people. In addition, the studies only lasted from 2 to 6 weeks. Thus, the effects they demonstrated may only be temporary. As evidence for this, Tsihlias et al, demonstrated that a 10% increase in MUFA intake, at the expense of carbohydrate, significantly reduced the total:HDL cholesterol ratio after 3 months, but this difference had disappeared by 6 months.[31]

Most of the 10 studies with high MUFA diets demonstrate reduced postprandial blood glucose concentrations. This may simply reflect the acute effect of reduced carbohydrate intake on postprandial glucose because none of these studies revealed any significant improvement in overall glycemic control as assessed by glycated hemoglobin or glycated albumin (fructosamine).[32] The absence of an effect of high MUFA diets on overall glycemic control might be explained by increased plasma FFA that are thought to impair insulin action and insulin secretion. Tsihlias et al, showed that a 10% increase in MUFA intake resulted in a 33% increase in mean post-prandial FFA concentrations in subjects with type 2 diabetes.[31]

High carbohydrate, low GI diets

A low GI diet is one in which the source of carbohydrate is changed from high GI to low GI foods, but the total amount of carbohydrate in the diet is not altered. There have been 14 studies in the literature examining the effects of low GI diets in diabetes.[32] Eleven of these were similar in duration (2-6 weeks) to the studies of high MUFA diets, and 8 of them showed a significant improvement in overall glycemic control assessed by glycated hemoglobin or fructosamine. The other 3 studies lasted from 6 to 12 months, and in 2 of them, there was a statistically significant reduction in glycated hemoglobin. Combining the results of all 14 studies, there was a highly significant 6.5% reduction in glycated serum proteins.[32] Comparing the results of the low GI studies with those of the high MUFA studies suggests that for optimal glycemic control, the source of carbohydrate may be more important than the amount. This is not consistent with the Position of the American Diabetes Association (Table 1).

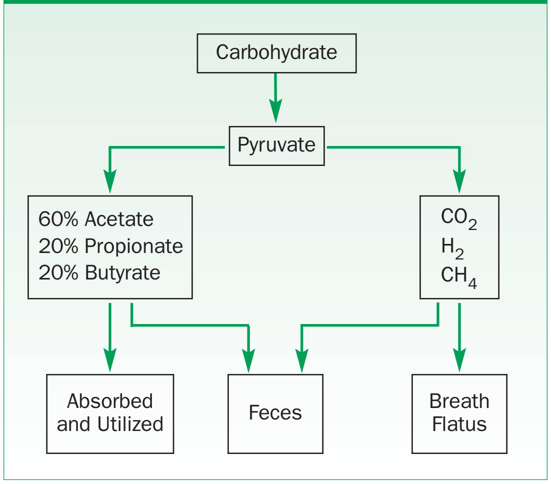
Fate of non-glycemic carbohydrate: colonic fermentation

It has been appreciated in the last 15 to 20 years that the colon plays a significant nutritional role by salvaging nutrients not absorbed in the small intestine. Carbohydrates not absorbed in the small intestine enter the colon where they are partly or completely fermented by colonic bacteria to produce gases and the short chain fatty acids (SCFA), acetic, propionic, and butyric acids (Figure 3) that are absorbed and contribute to systemic metabolism.[33] In a normal Western diet, it has been estimated that the SCFA provide 2%-4% of total energy requirements, but this may increase to 8% on a high carbohydrate, high fibre diet.[34]

The metabolic fate of the various SCFA differs:

- Butyrate is a major energy source for the colonic epithelium and appears in very low concentrations in the portal and peripheral circulations.[35]
- It is thought that most of the propionate in portal blood is taken up by the liver[35] where it acts as a gluconeogenic substrate[36] and inhibits the utilization of acetate for sterol and fatty acid synthesis.[37]
- Acetate is the only SCFA that appears in appreciable concentrations in peripheral blood.[35] Colonic acetate is a substrate for sterol and fatty acid synthesis,[37] and reduces plasma FFA concentrations.[36]

Figure 3: Simplified scheme of colonic fermentation



Colonic SCFA production may take months to adapt after a change in diet.[38] This is not surprising since the colon harbours 100s of species of bacteria and is a very complex ecosystem. It is possible that changes in colonic fermentation may, at least partly, mediate the metabolic adaptations that may occur when a high carbohydrate or high fibre diet is consumed for more than 3-6 months.[39] In addition to potential influences on carbohydrate and lipid metabolism, colonic fermentation may also enhance the absorption of certain vitamins and minerals from the colon, such as folate[40] and calcium.[41]

Further work needs to be done before the role of the colon in human nutrition is fully understood. In the future, however, it is possible that the colon will become a target for therapeutic diabetes interventions.

Summary

There is considerable controversy about how much and what kind of carbohydrate should be recommended for the nutritional management of diabetes. Dietary carbohydrates differ markedly in their chemical structures and the ways in which they are digested and metabolized. Dietary carbohydrates influence metabolism by at least 4 mechanisms: nature of the monosaccharides absorbed, amount of carbohydrate consumed, rate of absorption, and colonic fermentation. Caution is required before making long-term dietary recommendations based on dietary maneuvers that reduce postprandial blood glucose because different ways of reducing acute glycemic responses do not necessarily have the same long-term effects. For example, reducing diet the glycemic index reduces glycated hemoglobin concentrations, but there is no evidence that replacing carbohydrate with monounsaturated fat improves overall glycemic control in subjects with diabetes. Thus, current evidence supports the current recommendations from the Canadian Diabetes Association that both source and amount of dietary carbohydrate should be considered in meal planning for people with diabetes. For most people with diabetes, dietary carbohydrate should make up 50%-60% of energy. Naturally occurring and added sugars are part of a healthy diet and should be included as part of the daily carbohydrate allowance. Most people with diabetes can include added sugars for up to 10% of energy (about 50-60 g, or 10-12 teaspoonfuls per day). Choosing low GI foods more often may help to optimize glycemic control.

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