

Carbohydrate, Behaviour and Health

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The ingestion of all forms of dietary carbohydrate confers benefits to human behaviour and health. Dietary carbohydrate is an essential component of healthy diets. It is a macronutrient that not only provides energy, but also serves as an important regulator of the nervous system of human behaviours and of metabolism. Carbohydrate ingestion affects the nervous system through its provision of glucose and energy source and by its effect on neurotransmitter synthesis in the brain and by activation of the sympathetic nervous system. As a result, carbohydrate ingestion has a positive effect on several human behaviours, including appetite, sleep, activity, mood, cognition and physical performance. Through their effects on metabolism, high carbohydrate diets, compared with high fat diets, are generally associated with lower risk of chronic diseases, including obesity, diabetes, cancer and heart disease. An exception is dental caries which, in the absence of fluoridated water and attention to oral hygiene are increased by consumption of fermentable carbohydrate.

Dietary advice on carbohydrate should recognise the health benefits of all carbohydrate foods including those containing sugars, oligosaccharides and polysaccharides. An optimum diet contains at least 55% of total energy from carbohydrates, the sources of which may be selected, at least in part, on the basis of their glycemic index.

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Carbohydrate is the single most important source of food energy in the world, making up 40% to 80% of total food energy, depending on the cultural and economic status of the people. Carbohydrate includes, polyhydroxy aldehydes, ketones, alcohols, acids, their simple derivatives and their polymers having linkages of the acetal type. These are classified according to their degree of polymerization and may be divided into three principal groups, namely sugars, oligosaccharides and polysaccharides (Table 1).

Each of these three groups may be subdivided. Sugars may be monosaccharides, disaccharides, or polyols (sugar alcohols). Oligosaccharides include malto-oligosaccharides, principally those occurring from the hydrolysis of starch, and other oligosaccharides such as alpha-galactosides

(raffinose, stachyose, etc) and fructo-oligosaccharides. Polysaccharides are divided into starch (alpha-glucans) and non-starch polysaccharides (NSP), of which the major components are the polysaccharides of the plant cell wall such as cellulose, hemicellulose, and pectin.

The ingestion of all forms of dietary carbohydrate confers benefit to human behaviour and health. It is an essential component of healthy diets. It provides energy and is an important regulator of the nervous system, of behaviours, and of metabolism. Carbohydrate foods bring with them a wide array of essential nutrients and metabolically active substances important to the maintenance of health and prevention of diseases.

Table 1. The major dietary carbohydrates⁶⁶

Class	Subgroup	Components
Sugars (1-2)#	Monosaccharides	Glucose, Galactose, Fructose
	Disaccharides	Sucrose, Lactose, Trehalose
	Polyols	Sorbitol, Mannitol
Oligosaccharides (3-9)#	Malto-oligosaccharides	Maltodextrins
	Other oligosaccharides	Raffinose, Stachyose, Fructo-oligosaccharides
Polysaccharides (>9)#	Starch	Amylose, Amylopectin, Modified Starches
	Non-Starch Polysaccharides	Cellulose, Hemicellulose, Pectins, Hydrocolloids

Degree of polymerization

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Carbohydrate and the Nervous System

During digestion and absorption, dietary carbohydrate produces glucose, which is essential to the normal functioning of the nervous system¹. The nervous system depends on glucose as its major energy source. From the ingestion and metabolism of carbohydrate, the nervous system receives information that may influence brain neurotransmitter synthesis and function, and activate neuroregulatory process.

Carbohydrate ingestion has a positive effect on several human behaviours, including appetite, sleep, activity, mood and cognition and physical performance. Studies of carbohydrate and behaviour show that its effects are often subtle and hard to measure, but overall the conclusion is that its effect on behaviour is significant. Following is a review of the mechanisms by which carbohydrate affects the nervous system and behaviours.

Carbohydrate as Energy Substrate

The brain has a very high metabolic activity relative to most other body organs. Although it comprises only 2% of adult body weight, it receives 15% of cardiac output and accounts for 20-30% of whole body resting metabolic rate. In a premature infant, up to 60% of the whole body resting metabolic rate may be due to the metabolic activity of the brain². The respiratory quotient of the brain is 0.97, and since its glycogen store is only 0.1% of its weight, the brain depends on a continuous supply of oxygen and glucose to meet its high energy demands. The metabolic energy derived from glucose oxidation provides power for the brain's unceasing electrical activity, of which the main function is excitation and conduction.

In human infants, rates of cerebral energy metabolism and blood flow are associated during brain development, with the highest rates occurring during the period of active brain growth³. In five-week-old infants, cerebral glucose utilization is already at 71-93% of adult levels in most brain regions. Adult levels of cerebral glucose utilization are reached by two years of age. They continue to increase, however, until the child is three or four years old and are maintained until about nine years, at which time they begin to decline, returning to adult levels by 20 years of age. The high levels of brain energy metabolism in the first decade are due to the brain's basal energy needs as well as the biosynthetic requirements for the active maturational processes. The child's cognitive development is related to changes in blood flow in the different brain regions³.

Blood glucose in the normal individual may fluctuate over a relatively wide range without evidence of neurological consequences, because cerebral energy metabolism is not affected. When plasma arterial concentration falls from a normal level of 6-7 mmol/L to about 2.5-3 mmol/L, cerebral glucose content decreases in a directly proportional manner to whole blood glucose concentration; however cerebral energy metabolism remains the same. Below 2.5 mmol/L, glucose transport into the brain is diminished to a point where brain glucose no longer saturates hexokinase; it becomes rate-limiting and insufficient to support brain energy metabolism³.

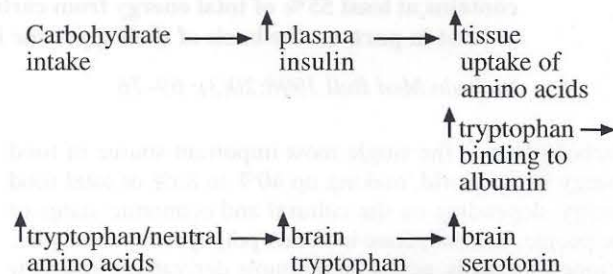
Under normal conditions, physiological and endocrine factors control blood glucose within narrow ranges in the normal

individual, both after a meal and in the absence of food, ensuring that the brain has available an excess of this critical nutrient. The availability of blood glucose may be modified, however, by endocrine disturbances (eg. diabetes) or with aging, when fluctuations in dietary carbohydrate intake and blood glucose levels affect the availability of glucose as an energy substrate.

Carbohydrate and Brain Neurochemistry

In the early 1970s, it was shown that carbohydrate consumption by rats resulted in an increase in the synthesis of the brain neurotransmitter 5-hydroxy-tryptamine, or serotonin⁴. This observation provided a physiological mechanism linking carbohydrate consumption to behaviour. Serotonin is a neurotransmitter known to regulate many behaviours, including appetite, sleep and mood.

The effect of carbohydrate consumption on brain serotonin is explained primarily by the digestion of carbohydrate to glucose and the resulting release of insulin as summarised below:



In animal species such as the rat and probably man, the transport Km of the blood-brain-barrier carrier system is approximately equal to the plasma amino acid concentration. Thus, competition among plasma amino acids is the primary determinant of the relative rate of uptake of each amino acid⁵. For this reason, a relative increase in transport of tryptophan into the brain will occur only if its concentration in plasma has increased relative to its competitors. Conversely, if there is a large increase in the concentration of other large neutral amino acids, as occurs when protein is consumed, the increase in plasma tryptophan is relatively small and the brain uptake of tryptophan decreases.

Carbohydrate consumption increases brain synthesis of serotonin, because it provides an increased brain uptake of tryptophan. Under usual brain concentrations of tryptophan, the enzyme converting tryptophan to serotonin is not fully saturated by the substrate. With diet-induced shifts in the availability of tryptophan, synthesis of serotonin is affected.

Carbohydrate and the Sympathetic Nervous System

The effect of carbohydrate on the brain is also reflected in the sympathetic nervous system (SNS)¹. This system regulates many vegetative processes, including blood flow, blood pressure, activity of visceral smooth muscle and glands, and metabolic reactions. Regulation achieved via the SNS occurs as a result of its direct innervation of most body tissues and its communication at these sites through the release of norepinephrine (NE), which exerts physiological and metabolic effects.

Of the three macronutrients, carbohydrate appears to have the strongest influence on SNS activity. Plasma NE levels increase following a meal of glucose but not after a meal of protein or fat⁶. In humans, about one-third of the observed rise in resting metabolic rate (RMR) during glucose infusion can be accounted for by an increase in SNS activity⁷.

Carbohydrate, Behaviour and Performance

Normal meals have definite effects on mood and performance in humans. The composition of a meal, for example, protein or carbohydrate content, influences these behaviours⁸. These effects have usually been attributed to altered serotonin function, because protein and carbohydrate have been found to affect brain serotonin in rats⁹. Although it is difficult to prove, whether serotonin accounts for the acute effects of these two macronutrients in humans⁸, the effects of carbohydrate consumption on behaviour are clear. They can be illustrated by examining its effect on appetite, sleep, mood and cognition, hyperactivity and physical performance.

Appetite Control

Carbohydrate foods, including those containing simple sugars, are easily recognised by the body as a source of calories and their ingestion leads to decreased appetite^{10,11}. In general, carbohydrates are more satiating than fats, but less so than protein¹².

Carbohydrates in the form of glucose, fructose, sucrose (table sugar), maltodextrins and starch, when given to humans in preloads or meals, suppress later food intake in amounts roughly equivalent to their energy value^{10,12}. Sugars in the amount of at least 50g (about the quantity in one and one-half soft drinks) given from 20 to 60 minutes before a meal, produce detectable decrease in meal-time food intake¹⁰. The monosaccharide fructose, however, is more effective than glucose in reducing meal-time intake and the desire to eat, when given as the only source of carbohydrate¹³. However, when consumed with other carbohydrate, no differences are seen between the different sugar treatments¹⁴. In meals eaten 30 minutes after preloads of cereal with either fructose or sucrose added, almost perfect compensation for the preload calories was seen when compared with a no-breakfast (water) preload.

In children, it is clear that both the energy content and sweetness of sugars decrease subsequent food (energy) intake and selection of sweet foods, respectively¹⁵.

Sleep

Studies in adult humans have indicated that more sleep occurs following a carbohydrate feeding, compared with water. In adult males, a liquid carbohydrate lunch has been reported to provide a longer postprandial sleeping time than no lunch¹⁶. Carbohydrate drinks taken before bed time by older adults enhance sleep duration and quality¹⁷.

In infants, consumption of carbohydrate alone can cause sleep. Based on the serotonin hypothesis, one study provided a 10% glucose feed containing added tryptophan (the serotonin precursor) and reported that newborns entered active and quiet sleep earlier than when receiving a balanced formula¹⁸. Less dramatic results were obtained in a more recent study in which a 6.7% lactose solution was fed¹⁹. The

failure of the lactose treatment to produce the effects observed when a 10% glucose solution with tryptophan was fed could be expected¹⁸. Lactose, composed of glucose and galactose, would stimulate much less tryptophan uptake by the brain than the glucose plus tryptophan mixture.

Mood and Cognition

Over the past two decades, there has been considerable effort made to define the effect of short-term nutritional intake on mood and cognition^{16,20,21}. It is clear that food intake affects mood, but it is less clear whether carbohydrate is the key component.

Breakfast intake is generally associated with an improvement in cognitive performance later in the morning. In contrast, lunch intake is associated with an impairment of mid-afternoon performance on mental tasks and more negative reports of mood. Intake of nutrients late in the afternoon appears to have a positive effect on subsequent performance on tasks involving sustained attention of memory²⁰. Carbohydrates may play a role in these responses.

The hypothesis that carbohydrate consumption will alter mood because of its presumed effect on brain serotonin has driven much of the research on the relationship between diet carbohydrate and mood. Evidence in support of the hypothesis has been gained from studies of both tryptophan supplementation and tryptophan depletion. Tryptophan supplementation has been used to treat many neuropsychiatric disorders, with variable success²². In normal subjects, supplementation results in a decrease in appetite, an increase in lethargy and sleepiness²³. Tryptophan depletion, obtained by feeding subjects an amino acid mixture free of tryptophan, has consistently resulted in a lowering of mood in normal and depressed subjects⁸. However, most studies, usually conducted at lunchtime, show either no specific effect of carbohydrate or no difference between high and low carbohydrate meals. Generally, mood alterations occur in early afternoon regardless of type of meal. Usually observed is a reduction in vigour, anger and depression scores^{21,24}.

In contrast to the inconsistent effect of carbohydrate on mood in healthy experimental subjects, there is strong evidence that individuals make use of carbohydrate to counteract negative mood states²⁵. Because a serotonin deficit enhance negative mood states⁸, the drive to consume carbohydrate to correct negative mood status is assumed to be linked to a reduction in serotonin synthesis. Although this link is not proven, it appears that carbohydrate intake is consumed as a self-administered mood regulator in:

- tobacco withdrawal symptoms²⁶
- alcohol withdrawal²⁷
- seasonal affective disorders²⁸
- obesity⁹
- premenstrual syndrome²⁹.

Consumption of carbohydrate has been shown to enhance cognitive function. The beneficial effect of carbohydrate is best shown after acute treatment with glucose drinks. The influence of glucose-containing drinks on cognitive function has been extensively examined in healthy adults. Acute doses of glucose improve cognitive performance in several situations, in comparison to a saccharin- or aspartame-

containing placebo drink³⁰. In general, the beneficial effects occur within one hour. Some have suggested that the effects may vary with task complexity^{31,32} and may be restricted to declarative memory³³. Declarative memory is defined as memory that is accessible to conscious awareness; a memory that can be declared or stated³⁴. This memory includes episodic memory (specific time and place events) and semantic memory (facts and general information) and is the memory expressed on tests of recall or recognition.

The strongest effect of glucose on memory may be in populations with pre-existing memory deficits or vulnerabilities to memory disorders³⁵. Pronounced improvements in memory occur after glucose drinks are consumed by early subjects³⁶⁻³⁹ and by those who are cognitively impaired⁴⁰. Abundant animal research, showing that glucose attenuates memory impairments caused by drugs and other amnesic agents, supports this notion⁴¹⁻⁴⁶.

Hyperactivity

A persistent myth about sugar is that it causes hyperactivity in children. The association to arise in the minds of parents who have observed that their children are excited at parties, and other celebrations, when sweet foods are served. Teachers have similar subjective opinions based on the classroom activity of some of their students, perhaps because they would like to have a simple explanation of children's behaviour. As well, a number of poorly designed studies in early literature have given some credence to this myth.

Numerous studies investigating the hypothesis that there is a relationship between sugar intake and children's behaviour have been reported in the peer-reviewed literature of the past 15 years. As summarised by White and Wolraich⁴⁷, results from 14 published studies, representing 16 controlled challenge studies and over 400 subjects do not support the hypothesis that refined sugar affects hyperactivity, attention span, or cognitive performance in children. To the contrary, a small but statistically significant decrease in motor activity has been observed in some children whose parents believed that they became hyperactive after consuming added sugar⁴⁸ and in children whose behaviour had not been considered hyperactive after consuming sugar⁴⁹.

Physical Performance

There is one aspect of human performance that clearly benefits from carbohydrate intake, and that is physical performance. At rest and during low intensity exercise, fat metabolism is the main provider of energy for resting metabolic processes and muscle contraction. However, at higher exercise intensities, the metabolism of carbohydrate reserves, such as blood glucose and liver and muscle glycogen, are the main providers of fuel for muscle contraction. In endurance activities, fatigue results when these substrata reach critically low amounts or are decreased, primarily because of reduced availability of substrate for the nervous system⁵⁰.

The optimization of carbohydrate availability to enhance performance has received much interest in the field of sports nutrition, and perspective have changed greatly in the past fifty years. The timing and frequency of carbohydrate intake at various stages are important determinants for optimizing fuel availability to maximise performance⁵¹. Pre-exercise

carbohydrate feedings are used to optimise substrate availability, whereas post-exercise carbohydrate ingestion promotes the re-synthesis of muscle and liver glycogen. Feedings during exercise, based on readily digested carbohydrates or sugars, provide a readily available source of exogenous fuel as stores deplete. Recognition of this essential role of carbohydrate has led to a marked change in advice over the past forty-five years to athletes performing prolonged exercise⁵¹. Forty-five years ago, long distance runners were advised to consume only water after every 5 km past 15 km. Now it is suggested that both water and carbohydrate may be appropriate every 3 km.

Carbohydrates and Maintenance of Health

Carbohydrate provides the majority of energy in the diet of most people. Carbohydrate-containing foods not only provide substrate for energy production, but also carry with them many important nutrients and phytochemicals. They are important in the maintenance of glycemic homeostasis and gastrointestinal integrity and function. High carbohydrate diets (55% or more of energy) are associated with reduced chronic disease, especially obesity and its co-morbid conditions.

Glycemia

Carbohydrates in food are digested to provide primarily glucose in the bloodstream. The effects of foods on blood glucose can be compared by means of the glycemic index.

Glycemic index (GI) is defined as the incremental area under the blood glucose response curve of a 50g carbohydrate portion of a test food expressed as a percent of the response to the same amount of carbohydrate from a standard food taken by the same subject. Using the GI makes it possible to select carbohydrate foods that have the most beneficial effect on metabolism. In general, low GI foods are preferred over high GI foods. The GI of some selected foods is given in Table 2.

Table 2. Glycemic index of selected foods⁵³

Food	Index(%)
Bread, white	100
Bread, whole meal	100
Rice, brown	81
Rice, pol., boiled 10-25 min.	81
Ice cream	69
Milk	45
Yoghurt	52
Sucrose	83
Fructose	26
Glucose	138
Beans, baked (canned)	70
Beans, soya (canned)	22
Peas, chick (canned)	60
Potato, new, white boiled	80
Potato, smashed	98
Cornflakes	121
Puffed Rice	132
All Bran	74
Potato chips	74

It is clear from Table 2 that the GI is affected by the composition of the carbohydrate, and by the presence of factor protein in the food. The glycemic response to common foods such as rice, whole-meal bread, potatoes, and many breakfast cereals is equal to or higher than the glycemic response to equivalent amounts of sucrose. This is because the starch in these foods as legumes, dairy products, and foods high in fat is lower than that of sucrose, because they contain components that slow the digestion and absorption of their carbohydrate. The GI of sucrose is only 83% of the GI of the standard, which is white bread⁵². Sucrose's lower glycemic response is explained by the effect of its components, glucose and fructose. Glucose alone has a GI of 138% relative to white bread, whereas the GI of fructose is only 26%⁵².

Because sugars create a lower insulin demand than do many starchy foods, there is no metabolic basis for the myth that sugars cause hypoglycemia⁵³. Experimental studies have shown, as well, caloric sweeteners do not cause hypoglycemia.

Obesity

In North America, obesity has been described as an epidemic. In the United States, prevalence of overweight is currently at a high of one-third of the adult population (based on body mass index of > 27.8 for men and > 27.3 for women)⁵⁴, and one-fifth of the children. For adults, the increase in prevalence was almost 8% (from 25.4% to 33.3%) between surveys conducted in 1976-80 and 1988-91. For children 12-19 years, the increase in prevalence was 5% (from 15% to 20% for males and from 17% to 22% for females). Severe obesity, defined as a BMI of 30 kg/m² or higher, is found in approximately 22% of American adults⁵⁵.

The main dietary factor contributing to obesity in North America has been judged to be its fat consumption and the resulting high energy-density of foods⁵⁶. Some studies show that the energy intake compensation is less precise at meals that follow the consumption of a preload high in fat, compared with one high in carbohydrate⁵⁷. Many high-fat foods such as ice-cream and chocolate contain sugar, which might suggest that sugar consumed in this combination is a factor encouraging excess fat intake¹¹. However, sugar-fat combinations alone are not likely the cause of excessive energy consumption. Furthermore, the role of fat per se is uncertain. Dietary surveys have shown that fat consumption has been decreasing gradually in the US during the past 15 years, yet the incidence of obesity continues to increase⁵⁸. So not only does sugar not appear to be the culprit, but fat intake alone is not likely to be the sole explanation for obesity.

Nevertheless, epidemiological studies show an inverse relationship between carbohydrate intake, including sugars and obesity¹¹, and a direct relationship between fat intake and obesity⁵⁶. For example, a study of 11,626 Scottish men and women aged 25-64 years reported the lowest prevalence of obesity in those consuming the highest total carbohydrate, total sugar, and added (extrinsic) sugars. The highest prevalence of obesity was among those with the highest consumption of fat relative to added sugar⁵⁹. Furthermore,

there is no relationship between the amount of sugar available in the food supply of a nation and the incidence of obesity in its population⁶⁰.

Given the complex aetiology of obesity, it is far too simplistic to attribute its origin to the composition of a particular food or of the food supply. Reviews of the literature have led to the conclusion that the prevalence of paediatric overweight is due to the dramatic decrease in physical activity^{61,62}. Fewer than 40% of high school students engage in vigorous physical activity more than three times per week⁶³.

The lower prevalence of obesity in Canada (14%) than that in the United States (22%)⁶⁴ has been attributed to the higher activity levels of Canadians, even though the food supply is similar. Therefore, in the presence of an abundant and palatable food supply, the most effective strategy in the prevention of obesity may be high-volume, low-intensity (prolonged endurance) exercise⁶⁵.

Excess body fat (obesity) arises very simply from the energy imbalance caused by eating too many calories and using too few of them. The sociological factors accounting for this problem vary from country to country and the understanding of these should be the basis of programmes and food-based dietary guidance aimed at the prevention of obesity.

Diabetes

One sequence of obesity is an increased risk of diabetes mellitus. The symptoms are caused by insulin deficiency that results either from decreased insulin production or from a diminished effect of insulin at the cellular level. The most prevalent form of diabetes is non-insulin-dependant diabetes mellitus (NIDDM). It starts most often in adulthood and is associated with obesity.

The major disease-management objectives for NIDDM focus on weight control through energy-reduced diets and increased physical activity. In the 1970s and 1980s, the diabetes associations of many countries began to recommend that fat in the diet should be reduced and the energy replaced by carbohydrate⁵³. The recent FAO/WHO report on carbohydrates in nutrition⁶⁶ suggests that consuming a wide variety of carbohydrate foods is now acceptable, and that 60% to 70% of total energy should be derived from a mix of monosaturated fats and carbohydrate. Diabetics should consume high-carbohydrate foods, emphasising fruits, vegetables and whole grains. These foods are recommended because of their slow release of glucose, which reduces the metabolic demands on insulin.

There has been a wide belief that simple sugars should be avoided in the dietary management of diabetes. However, there is no scientific evidence for this belief. Sugars are not more rapidly digested and absorbed than other starches⁶⁷. Thus, sugar in moderation is also now accepted as a component of diets for diabetics, because it has a low GI (Table 2). Sugars have not been directly implicated in the aetiology of diabetes, and recommendations allow intakes of sucrose and other added sugars, providing that: (a) they are not consumed in excess of the total energy allowance; (b) the nutrient-dense foods and non-starch polysaccharide (NSP)-rich foods are not displaced; and (c) they are consumed as part of a mixed meal⁶⁸.

Hyperlipidemia and Cardiovascular Disease

Dietary advice for people with coronary heart disease is to reduce the intake of fat and increase the intake of carbohydrate-rich foods, rich in NSP, especially cereals, vegetables and fruits. Certain NSPs, such as beta-glucans, have been shown to have a serum cholesterol-lowering effect when consumed in naturally-occurring foods, in enriched forms, or in dietary supplements⁶⁶.

Hyperlipidemia is the elevation of blood lipids and is believed to be an aetiologic factor in the process of atherosclerosis. There is no evidence that sucrose plays a role in the aetiology of coronary heart disease; however, it has been recognised for years that large amounts of dietary sugars, about two to three times the average consumption, can raise blood lipids.

The potential effect of sucrose consumption on blood lipids is explained partly by fructose's unique metabolic pathway in the liver. There is evidence that it is a better substrate than glucose for lipid synthesis. Therefore, it should not be a surprise to find an elevation of blood triacylglycerol concentrations in some subjects who are given excessive quantities of either fructose or sucrose. However, in studies in which amounts of sugars typical of the Western diet were provided, such responses were not observed, except in some carbohydrate-sensitive individuals⁶⁹. There is no evidence that this occurs when the increase in carbohydrate is due to increased consumption of vegetables, fruits and appropriately processed cereals⁶⁶.

Dental Caries

Caries prevalence has decreased markedly during the past 20 years in developed countries despite consistently high sugar consumption⁷⁰. The reason is that tooth decay is influenced by many factors in addition to diet, including oral hygiene procedures, fluoride delivery, bacterial components of plaque, the amount and composition of saliva, type of preventive and restorative care and immunological response of the individual. The consumption of sugar and other fermentable carbohydrates, in the absence of fluoridated water, toothpaste and attention to oral hygiene, does increase the incidence of dental caries. However, it is recommended that when such circumstances exist, the most effective approach is to improve oral hygiene and to use fluoride dentifrices. Dietary restrictions have not proven as effective^{70,71}.

Food Guides

Dietary advice must be transmitted to the public in the form of food-based dietary guidance in order to have an effect on the health status of the population. Food guides play this role in many countries.

The major nutrition education tool used in many countries is the national Food guide. Both Canada and the US have developed new Food Guides that incorporate their national dietary guidelines. In the past, the Food Guides concentrated on expressing a dietary pattern that would provide the individual with the essential nutrients in recommended amounts. The new Food Guides are a departure from the past as they recommend a dietary pattern that would not only meet the requirements for essential nutrients, but would also lead to a diet higher in carbohydrate and lower in fat

than currently consumed. Both the Canadian and American Food Guides are centres around food groups and both have a new category of foods, one that includes sugars, fats and oils.

The Canadian Food Guide⁷² incorporates the four food groups in a rainbow design. Meat and alternatives are arranged along the small, inner arc. Next come the milk products, then vegetables and fruits, and finally grain products along the large, outer arc. The design gives the visual cue that the greatest quantity of food should be selected from the grains group. A category of "Other Foods" is identified as not part of any food group, and includes foods that are mostly fats and oils, mostly sugar, high fat or high salt snack foods, beverages, herbs spices and condiments. It is acknowledged that "These foods can be used in making meals and snacks and are often eaten with foods from the four food groups", but it is recommended that they be consumed in moderation. Instructions on using the Food Guide include advice on reducing fat, but no mention is made of reducing sugar.

The US Food Guide⁷³ is in the form of a pyramid, showing five food groups, with the grain group at the base. The vegetable group and the fruit group share the next level of the pyramid and on top of these are the milk and meat groups, sharing equally the next level in the pyramid. The visual impression is that one should consume less of those foods higher up the pyramid. At the very top is the category of fats, oils and sweets. The consumer is advised that "These foods supply calories, but little or no vitamins and minerals".

Thus, both the Canadian and American Food Guides are designed to help the individual select carbohydrate food as the foundation for a healthy diet. Food selection based on the Food Guides will provide 55% of the energy, or greater from carbohydrate. Explicit in the Guides is also a recognition of the role of all forms of carbohydrates, including the sugars, oligosaccharides and polysaccharides and that food carbohydrates are more than an energy source. Food containing carbohydrate bring with them a wide array of nutrients and other metabolically and physiologically active components, such as fibre and phytoestrogens, which are beneficial to health. As well, the carbohydrate sweeteners are important not only for their contribution to hedonic value, but for their role in food preparation, such as bread making and preservation. As 10-12% of dietary energy, added sugars provide an important contribution to the benefit of carbohydrate consumption. Although the role of sugars in the diet and their effect on health has been controversial in the past, all recent evaluations^{70,74,75} have concluded that current consumption levels in the developed countries are consistent with healthy diets. With the exception of dental caries, sugars do not have any specific role in the aetiology of chronic disease.

The recent FAO/WHO report⁶⁶ recommends that in choosing carbohydrate foods, both glycemic index and food composition be considered. It will be some time before a complete knowledge of the GI of foods is available, although considerable information has been derived⁵². In the meantime, recognition of the determinants of the GI can help in perspective the metabolic, physiological and behavioural effects of consuming food carbohydrates.

CONCLUSION

Carbohydrate consumption has beneficial effects on human behaviour including appetite, sleep, mood, cognition and physical performance. The optimum intake of at least 55% of total energy from a variety of carbohydrate sources is associated with the maintenance of health and avoidance of chronic disease.

REFERENCES

- Li ETS, Anderson GH. Amino acids in food intake and selection. In: Kaufman S, ed. *Amino acids in health and disease: New perspective*. Alan R Liss Inc, 1987:345.
- Sokoloff L. Relation between physiological function and energy metabolism in the central nervous system. *J Neurochem* 1977;29:13.
- Nehlig A. Cerebral energy metabolism, glucose transport and blood flow: changes with maturation and adaptation to hypoglycemia. *Diab Metab* 1997;23:18.
- Frenstrom JD, Faller DV. Neutral amino acids in the brain: Changes in response to food ingestion. *J Neurochem* 1978;30:1531.
- Pardridge WM. Brain metabolism: a perspective from the blood-brain barrier. *Physiol Rev* 1983;63:1481.
- Welle S, Lilavivat U, Campbell RG. Thermic effect of feeding in man: increased plasma norepinephrine levels following glucose but not fat or protein composition. *Metab* 1981;30:953.
- Ravussin E, Bogardus C. thermogenic response to insulin and glucose infusions in man: a model to evaluate the different components of the thermic effect of carbohydrate. *Life Sci* 1982;31:2011.
- Young SN. The use of diet and dietary components in the study of factors controlling affect in humans: a review. *J Psychiatr Neurosci* 1993;18:235.
- Wurtman RJ, Wurtman JJ. The use of carbohydrate-rich snacks to modify mood state: a factor in the production of obesity. In: Anderson GH, Kennedy SH, eds. *The Biology of feast and famine: Relevance to eating disorders*. San Diego: Academic Press Inc, 1992:151.
- Anderson GH. Sugars, sweetness and food intake. *Am J Clin Nutr* 1995;56:2:195S-202S.
- Hill JO, Prentice AM. Sugar and body weight regulation. *Am J Clin Nutr* 1995;62:264S-74S.
- Blundell JE, Green S, Burley VJ. Carbohydrates and human appetite. *Am J Clin Nutr* 1994;59:728S.
- Rodin J, Reed D, Jamner L. Metabolic effects of fructose and glucose: implications for food intake. *Am J Clin Nutr* 1988;47:683.
- Stewart SL, Black RM, Wolever TMS, et al. The relationship between the glycaemic response to breakfast cereals and subjective appetite and food intake. *Nutr Res* 1997;17:1249.
- Black RM, Anderson GH. Sweeteners, food intake and selection. In: Frenstrom JD, Miller GD, eds. *Appetite and body weight regulation: Sugar, fat and Macronutrient substitutes*. Boca Raton: CRC, 1994:125-36.
- Zammit GK, Kolevzon A, Fauci M, et al. Postprandial sleep in healthy men. *Sleep* 1995;18:229.
- Southwell PR, Evans CR, Hunt JN. Effect of a hot milk drink on movement during sleep. *Br Med J* 1972;2:429.
- Yogman MW, Zeisel SH. Diet and sleep patterns in newborn infants. *N Engl J Med* 1983;309:1147.
- Oberlander TF, Barr RG, Young SN, et al. Short-term effects of feed composition on sleeping and crying in newborns. *Pediatrics* 1992;90:733.
- Kanarek R. Psychological effects of snacks and altered meal frequency. *Br J Nutr* 1997;77:S105.
- Kurzer MS. Women, food and mood. *Nutr Rev* 1997;55:268.
- Sandyk R. L-tryptophan in neuropsychiatric disorders; a review. *Int J Neurosci* 1992;67:127.
- Hrboticky N, Leiter LA, Anderson GH. Effects of L-tryptophan on short term food intake in lean men. *Nutr Res* 1985;5:595.
- Christensen L, Redig C. Effect of mean composition on mood. *Behv Neurosci* 1993;107:346.
- Christensen L. Effects of eating behaviour on mood: a review of the literature. *Int J Eating Disorders* 1993;14:171.
- Bowen DJ, Spring B, Fox E. Tryptophan and high-carbohydrate diets as adjuncts to smoking cessation therapy. *J Behav Med* 1991;14:97.
- Rosenfield SN, Stevenson JS. Perception of daily stress and oral coping behaviours in normal, overweight and recovering alcoholic women. *Res Nurs Health* 1988;11:165.
- Krauchi K, Wirz-Justice A. Seasonal patterns of nutrient intake in relation to mood. In: Anderson GH, Kennedy SH, eds. *The biology of feast and famine: Relevance to eating disorders*. San Diego: Academic Press Inc, 1992:157.
- Sayegh R, Schiff I, Wurtman J, et al. The effect of a carbohydrate-rich beverage on mood, appetite and cognitive function in women with premenstrual syndrome. *Obstet Gynecol* 1995;86:520.
- Rogers PJ, Lloyd HM. Nutrition and mental performance. *Proc Nutr Soc* 1994;53:443.
- Benton D. The impact of increasing blood glucose on psychological functioning. *Biolo Psychol* 1990;30:13.
- Holmes CS, Koepke KM, Thompson RG. Simple versus complex performance impairments at three blood glucose levels. *Psychoneuroendocrinol* 1986;11:353.
- Craft S, Murphy C, Wermstrom J. Glucose effects on complex memory and nonmemory tasks: The influence of age, sex and glucoregulatory response. *Psychobiol* 1994;22:95.
- Pinel JPJ. *Biopsychology*. 2nd ed. Boston: Allyn & Bacon, 1993.
- Azari NP. Effects of glucose on memory processes in young adults. *Psychopharmacol* 1991;105:521.
- Allen JB, Gross AM, Aloia MS, et al. The effects of glucose on nonmemory cognitive functioning in the elderly. *Neuropsychologia* 1996;34:459.
- Gonder-Frederick LA, Hall JL, Vogt J, et al. Memory enhancement in elderly humans: Effects of glucose ingestion. *Physio Behav* 1987;41:503.
- Manning CA, Hall JL, Gold PE. Glucose effects on memory and other neuropsychological tests in elderly humans. *Psychol Sci* 1990;1:307.
- Parsons MW, Gold PE. Glucose enhancement of memory in elderly humans: An inverted-U dose-response curve. *Neurobiol Aging* 1992;13:401.
- Manning CA, Parsons MW, Gold PE. Anterograde and retrograde enhancement of 24-h memory by glucose in elderly humans. *Behav Neural Biol* 1992;58:125.
- Ahlers ST, Shurtleff D, Schrot J, et al. Glucose attenuates cold-induced impairment of delayed matching-to-sample performance in rats. *Psychobiol* 1993;21:87.
- Messier C, Durkin T, Mrabet O, et al. memory-improving action of glucose: Indirect evidence for a facilitation of hippocampal acetylcholine synthesis. *Behav Brain Res* 1990;39:135.
- Ragozzino ME, Gold PE. Glucose effects on mecamylamine-induced memory deficits and decreases in locomotor activity in mice. *Behav Neural Biol* 1991;56:271.
- Ragozzino ME, Parker ME, Gold PE. Spontaneous alternation and inhibitory avoidance impairments with morphine injections into the medial septum. Attenuation by glucose administration. *Brain Res* 1992;59:241.
- Stone WS, Croul CE, Gold PE. Attenuation of scopolamine-induced amnesia in mice. *Psychopharmacol* 1988;96:417.
- Stone WS, Walser B, Gold SD, et al. Scopolamine- and morphine-induced impairments of spontaneous alternation performance in mice: reversal with glucose and with cholinergic and adrenergic agonists. *Behav Neurosci* 1991;105:264.
- White J, Wolraich M. Effect of sugar on behaviour and mental performance. *Am J Clin Nutr* 1995 62[suppl]:242S-9S.

48. Behar D, Rapoport J, Adams C. Sugar challenge testing with children considered behaviorally "sugar reactive". *Nutr Behav* 1984;1:277-88.
49. Saravis S, Schacher R, Zlotkin S, et al. Aspartame: effects on learning, behaviour and mood. *Physiol Behav* 1990;86:75-83.
50. Sherman WM. Metabolism of sugars and physical performance. *Am J Clin Nutr* 1995;62:228S.
51. Hawley JA, Burke LM. Effect of meal frequency and timing on physical performance. *Brit J Nutr* 1997;77:S91.
52. Wolever TMS. The glycemic index. In: Bourne GH, ed. *Aspects of some vitamins, minerals and enzymes in health and disease*. World Rev Nutr Diet Basel:Karger, 1990:120-85.
53. Wolever T, Brand J. Sugars and blood glucose control. *Am J Clin Nutr* 1995;62[suppl]:212S-27S.
54. Kuczmarski RJ, Flegal KM, Campbell SM, et al. Increasing prevalence of overweight among US adults. The National Health and Nutrition Examination Surveys 1960 to 1991. *J Am Med Ass* 1994;272:205-11.
55. Abu J, et al. Obesity solutions: report of a meeting. *Nutr Rev* 1997;55:150-6.
56. Prentice AM, Poppitt SD. Importance of energy density and macronutrients in the regulation of energy intake. *Int J Obes* 1996;20:S18-S23.
57. Hill JO, Peters JC. The impact of diet composition on energy and macronutrient balance. In: Angel A, Anderson H, Bouchard C, et al, eds. *Progress in Obesity Research: 7. 7th International Congress on Obesity*. London: John Libbey & Company Ltd, 1996:385-92.
58. Stephen AM, Wald NJ. Trends in the individual consumption of dietary fat in the United States, 1920-1984. *Am J Clin Nutr* 1990;52:465-9.
59. Bolton-Smith C, Woodward M. Dietary composition and fat to sugar ratios in relation to obesity. *Int J Obes* 1994;18:820-8.
60. Ottley C, Wall T, Cottrell R. *A quantitative dietary guideline for sugar ?* London: The Sugar Bureau, 1994.
61. Dietz WH. Prevention of childhood obesity. In: Angel A, anderson H, Bouchard C, et al, eds. *Progress in Obesity Research: 7. 7th International Congress on Obesity*. London: John Libbey & Company Ltd, 1996.
62. Schlicker SA, Borra ST, Regan C. The weight and fitness status of United States children. *Nutr Rev* 1994;52:11-17.
63. Heath GW, Pratt M, Warren CW, et al. Physical activity patterns in American high-school students: results from the 1990 youth risk behaviour studies. *Arch Pediatr Adolesc Med* 1994;148:1131-6.
64. Reeder BA, et al. Obesity and its relation to cardiovascular disease risk factors in Canadian adults. *Can Med Ass J* 1992;1[suppl]:37-47.
65. Bouchard C, Depris JP, Tremblay A. Exercise and obesity. *Obes Res* 1993;1:133-47.
66. FAO/WHO. *Carbohydrates in human nutrition*. Interim report of a joint FAO/WHO expert consultation. Italy: Rome, 1998.
67. American Diabetes Association. *Nutrition recommendations and principles for people with diabetes mellitus*. *Diabetes Care* 1997;20:514-7.
68. Canadian Diabetes Association (CDA). *The role of dietary sugars in diabetes mellitus*. *Can Diab* 1992;5:1-5.
69. Frayn K, Kingman S. Dietary sugars and lipid metabolism in humans. *Am J Clin Nutr* 1995;62[suppl]:250S-63S.
70. Konig KG, Navia JM. Nutritional role of sugars in oral health. *Am J Clin Nutr* 1995;62[suppl]:275S-83S.
71. Glinesman WH, Irausquin H, Park YK. Report from FDA's Sugars Task force: evaluation of health aspects of sugars contained in carbohydrate sweeteners. *J Nutr* 1986;116:S1-S216.
72. Health and Welfare Canada. *Canada's Food Guide to health eating for people four years and older*. Ottawa: Ministry of Supply and Services, 1992.
73. United States Department of Agriculture. *The Food guide pyramid*. Human Nutrition Information Services. Home and Garden Bulletin No. 252. Washington DC, 1992.
74. Anderson GH. Sugars and health: a review. *Nutr Res* 1997;17:1485-98.
75. Schneeman B. Summary, nutrition and health aspects of sugars. *Am J Clin Nutr* 1995;62:294S-96S.