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Carbohydrate as a nutrient in adults: range of acceptable intakes

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This review considers the acute and chronic effects of different levels of carbohydrate (CHO) intakes. The type of CHO consumed, especially glucose *vs* fructose, affects the glycaemic, insulinaemic and thermogenic responses. In addition, other aspects of food (type of starch, method of processing or cooking, presence of other nutrients) affects the glycaemic response (glycaemic index). In general, the greatest benefit to health is derived from consuming foods with a low glycaemic index and a high non-starch polysaccharide (fibre) content. Healthy, moderately active adults require at least 200 g CHO per day to sustain normal brain metabolism and muscle function. Moreover, the CHO content should represent at least 50% of energy intake. Higher intakes of CHO can have deleterious effects on blood lipids (especially plasma triacylglycerol) in middle-aged and elderly subjects, and are really only appropriate for subjects with a high level of physical activity who need to maintain muscle glycogen content. Meals with a high carbohydrate content can lead to problems of postprandial hypotension in the elderly, and impaired exercise capacity in patients with angina.

Introduction

At the simplest level, carbohydrate (CHO) is needed in the diet for two purposes, as an immediate source of a substrate for energy metabolism and to enable replenishment of CHO stores for subsequent utilisation in the intervals between meals. CHO is also needed in numerous biochemical reactions not directly concerned with energy metabolism, but these are quantitatively of minor importance compared to the amount of CHO used in energy metabolism. Whilst the diet can contain a variety of different types of CHO, that is, simple sugars (mono- and disaccharides), oligosaccharides (maltodextrin) and polysaccharides (starch and non-starch polysaccharides), the major CHO of interest for human metabolism is glucose. Therefore, most dietary CHO is digested within the intestinal tract to form glucose which is absorbed, or if other monosaccharides are produced (galactose, fructose), they are either converted to glucose by intestinal or hepatic cells, or incorporated into intermediary stages of the glucose metabolic pathways.

After an overnight fast, the metabolic requirements for glucose are met by the release of glucose from the liver (and kidney). This endogenous glucose production (EGP) involves both breakdown of stored liver glycogen and gluconeogenesis from lactate and amino acids. In the healthy adult, after an overnight fast, EGP is approximately 2 mg per kg body weight per min, therefore in a 70 kg individual this would be 140 mg/min, or approximately 200 g CHO per day. Approximately half of this glucose is needed to satisfy the substrate requirements of the nervous system, being used in aerobic metabolism to produce ATP, therefore the minimum amount of CHO needed to sustain the nervous system could be as low as 100 g/d. The purpose of this review is to consider the effects of different levels of CHO intake, both in terms of acute responses to different amounts and types of CHO, and in terms of the consequences of diets with differing CHO contents. The principal focus will be in relation to healthy individuals, with references being made to diabetes or insulin resistant states where appropriate.

Dietary CHO

A recent FAO/WHO Expert Consultation on Carbohydrates in Human Nutrition (FAO/WHO, 1997) proposed the classification of dietary CHO into three sub-groups according to the degree of polymerization. Therefore the *sugars* would comprise monosaccharides (glucose, galactose, fructose), disaccharides (sucrose, lactose, trehalose) and polyols (sorbitol, marnitol); the *oligosaccharides* would contain 3–9 molecules and mainly represent intermediates in the digestion of starch (maltodextrins); the *polysaccharides* would consist of starch and non-starch polysaccharides. The majority of CHO in the human diet is made up of starch and sucrose, and an issue discussed later is the extent to which these CHO sources have different metabolic effects.

In recent years, a great deal of attention has focused on the need for an adequate intake of dietary fibre, both in relation to coronary heart disease and colonic function (Burkitt & Trowell, 1975). The details of the possible effects of fibre on human health are beyond the scope of this review, it is sufficient to acknowledge that this fibre is derived from components of CHO containing foods (mainly, non-starch polysaccharides) and also that this component of the diet may not be quite as inert as previously thought as far as whole body energy metabolism is concerned. By convention, dietary CHO which can be digested and absorbed into the circulation is given an energy value of 17 kJ (4 kcal) per gram. If one is considering monosaccharides, or the CHO content of the diet is expressed as monosaccharides, then the value should be 15.7 kJ (3.75 kcal) per gram. This conventional approach also assumes that indigestible CHO (non-starch polysaccharides and other sources of fibre) does not act as a source

of substrate for energy metabolism. However, this latter assumption is clearly invalid, as colonic fermentation of such CHO can occur, with the liberated short chain fatty acids being absorbed into the circulation and used as metabolic substrates by splanchnic and other tissues. Therefore, it has been suggested that such CHO reaching the colon has an energy value of approximately 8 kJ (2 kcal) per gram (Roberfroid *et al*, 1993; Livesey & Elia, 1995). As most diets only contain 10–30 g of fibre, this would make a minor contribution to total energy intake, and as the absorbed nutrients are short chain fatty acids, it is of minor significance in relation to CHO metabolism.

Acute responses to CHO ingestion

The consumption of CHO requires digestion of any oligo- and polysaccharides in the intestine before absorption of the monosaccharides. The liberated glucose is absorbed via a specific, intestinal, sodium linked glucose transporter (SGLT1) whereas any fructose is absorbed via a GLUT5 transporter (see Levin, 1994 for a review). The rate of monosaccharide absorption is not only determined by the processes of digestion and the activity of these transporters, but also by the rate at which food leaves the stomach. Therefore, any CHO-containing foods which empty from the stomach slowly, or which require an extended period in the intestine for digestion, will produce a less rapid rise in plasma glucose than foods which leave the stomach rapidly and whose CHO content is absorbed quickly. This forms the basis of the glycaemic index of foods, whereby CHO sources are characterised on the basis of the magnitude of the plasma glucose response produced.

The glycaemic response to carbohydrate containing foods is affected by a variety of factors including the nature of the carbohydrate, the method of cooking and the presence of other foods within a meal. Therefore, the different monosaccharides vary in their effects on blood glucose (glucose > fructose > galactose) as do the different types of starch (amylose/amylopectin *vs* resistant starch). The processing and cooking of food affects the glycaemic index, particularly when these have effects on the digestibility of starch. The other major factor affecting the glycaemic index is the presence of other food components, in particular fat, protein and fibre, which affect gastric emptying and/or intestinal transit. Minor components of foods can also affect gastric emptying and glycaemic index. For example, Liljeberg & Björck (1996) showed that sourdough bread (containing organic acids) produced a marked slowing of gastric emptying and reduced glycaemic response compared to wholemeal bread with the same carbohydrate content. High glycaemic index foods produce larger and more prolonged increases in plasma glucose and are discouraged for patients with diabetes. There is some evidence that patients with diabetes (Wolever *et al*, 1992) or with coronary artery disease (Frost *et al*, 1996) derive potential benefit from the consumption of foods with a low-glycaemic index.

In healthy subjects, the consumption of a CHO-containing meal provides an exogenous source of CHO which can replace the EGP which sustains glucose supply to the CNS in the fasting state. Therefore, as the exogenous CHO is absorbed, plasma glucose rises and insulin is released from the pancreas in larger amounts. This combination of a rise in plasma glucose and insulin concentration has three effects, to reduce EGP, to promote increased glucose oxidation, and to promote glucose storage as glycogen. In association with these changes in CHO metabolism, there is also a stimulation of thermogenesis which is partly a consequence of the energy costs of glycogen synthesis, but may also have a component due to activation of the sympathetic nervous system (Acheson *et al*, 1984).

There is clear evidence that the ingestion of glucose or fructose, either alone or as components of meals, produces different plasma glucose, CHO oxidation and thermogenic responses. Early studies by de Kalbermatten *et al* (1980) showed that infusion of fructose produced an increase in CHO oxidation without any increase in plasma glucose, and that this effect occurred independently of an insulin response. In the absence of insulin, it was clear that infused fructose was more effectively utilised as a metabolic substrate than infused glucose. A subsequent study compared the oral ingestion of glucose or fructose, measuring the plasma glucose and insulin, CHO oxidation and thermogenic responses (Tappy *et al*, 1986). This study showed that fructose was associated with a greater increase in CHO oxidation and thermogenesis (10% after fructose, 6.5% after glucose) than seen after oral glucose, even though there were much greater increases in plasma glucose and insulin after the glucose load. Similar, but less marked effects were observed by Schwartz *et al* (1989) when glucose or fructose were added to mixed nutrient test drinks. It should be recognised that not all studies have observed larger thermogenic responses to fructose than glucose. Fukagawa *et al* (1995) found no difference in the thermogenic responses to glucose and fructose in either young or elderly subjects, although this may in part be a consequence of a shorter period of measurement after CHO ingestion than in the previous studies.

Further evidence that fructose produces greater thermogenic responses than glucose is provided by Blaak & Saris (1996). They compared thermogenic and CHO oxidation responses to equal quantities of sucrose, fructose, glucose and corn starch, consumed in separate test drinks. Sucrose and fructose produced greater increases in CHO oxidation than the other CHO sources, and greater thermogenic responses, although the difference in the latter was only significant for sucrose.

It seems likely that a substantial part of any additional thermogenesis produced by fructose arises in the liver, as fructose uptake by liver cells occurs independently of insulin, and the low insulin responses to ingested fructose means that EGP is not reduced after fructose ingestion in the way that it is after glucose intake. However, Brundin & Wahren (1993) failed to observe any increase in splanchnic oxygen consumption after oral ingestion of either glucose or fructose, despite increases in whole body oxygen uptake, which were numerically greater after fructose than glucose. They did observe greater increases in whole body CHO oxidation after fructose, but concluded that with both monosaccharides, the thermogenic responses occurred in extra splanchnic tissues.

Therefore, the type of CHO consumed has an effect on the plasma glucose and insulin responses, and on the stimulation of CHO oxidation and thermogenesis. A greater stimulation of CHO oxidation leaves less CHO to be stored as glycogen, and may thus affect subsequent CHO availability when glycogen needs to be used. The differences in thermogenesis are intriguing, but their relevance for energy balance over a longer time period, and the identification of the sites of thermogenesis remain to be determined.

Cardiovascular effects of CHO ingestion

In healthy young subjects, the ingestion of food leads to an increase in gastrointestinal blood flow, with little disturbance of blood pressure due to an increase in cardiac output and a reduction in blood flow to the limbs (Sidery *et al*, 1991). Meals with high CHO content produce more marked increases in cardiac output and shorter periods of reduced limb blood flow than meals with a high fat content. The coordination of these vascular responses to food is dependent on normal functioning of the autonomic (especially the sympathetic) nervous system, and disruption of this can produce substantial reductions in blood pressure after a meal. Such an effect is particularly noticeable with elderly subjects consuming a high CHO meal, where postprandial hypotension can occur (Potter *et al*, 1989). This fall in blood pressure in the elderly is due to an inadequate cardiac output response, and a failure of vasoconstriction in the limbs, after a high CHO meal (Sidery *et al*, 1993). The absence of limb vasoconstriction is not seen after high fat meals, and seems to result from effects of the insulin secreted in response to CHO (Kearney *et al*, 1998).

In addition to the effect of CHO ingestion on postprandial blood pressure in the elderly, there are detrimental effects of high CHO meals in patients with chronic, stable angina. Such patients experience chest pain, and have characteristic ECG changes, during exercise. It is well known that such symptoms occur sooner if patients exercise immediately after eating, but it is now clear that high CHO meals have more marked effects than high fat meals (Kearney *et al*, 1997). In the first hour after a high CHO meal, the exercise time before the onset of chest pain was reduced by up to 25%, but no such changes occurred after a high fat meal. One needs to be careful in drawing up advice for patients on the basis of this observation, as a high fat diet would be accompanied by a serious risk of worsening the atherosclerosis underlying the angina. It would be more appropriate to advise such patients to try to avoid exercise for the first hour after eating.

Dietary CHO, insulin sensitivity, CHO oxidation and glycogen storage

Insulin released after food ingestion has three major effects, inhibition of EGP, stimulation of glucose uptake by tissues such as muscle and adipose tissue (but not liver, brain or erythrocytes), and stimulation of hepatic and skeletal muscle glycogen storage. An individual subject's sensitivity to these actions of insulin depends on a variety of factors including nutritional status and dietary CHO intake. Assessing the sensitivity to these actions of insulin after CHO ingestion is quite difficult, because measuring the rate of appearance into the circulation of exogenous glucose is rather complex. In addition, the plasma glucose and insulin responses to oral CHO (such as the glucose tolerance test) are dynamic, making it difficult to attribute the responses to specific insulin concentrations. A clearer understanding of the various effects of insulin on CHO metabolism has been obtained by using techniques such as the hyperinsulinaemic glucose clamp (De Fronzo *et al*, 1979) whereby steady state plasma insulin and glucose concentrations are obtained by intravenous infusion of glucose and insulin. This permits insulin sensitivity for total glucose disposal to be assessed, and combining the technique with isotope based estimates of EGP, and with indirect calorimetric estimates of CHO oxidation enables the three major components of insulin effects on CHO metabolism to be determined.

Dietary CHO and glucose tolerance

One of the earliest studies of the effects of diet composition on glucose tolerance and insulin sensitivity was conducted over 60 y ago by Himsworth (1935). These rigorous, elegant studies involved assessing the oral glucose tolerance and plasma glucose responses to exogenous insulin after 7 d on a series of different diets in healthy subjects. The diets were isoenergetic, but varied in total CHO content from 50 g (7.5% of energy) to 500 g (75% of energy) per day, with a reciprocal change in the fat content. Himsworth showed quite clearly that glucose tolerance improved, that is, the area under the plasma glucose curve fell, as the dietary CHO content increased. These studies preceded the availability of assays of plasma insulin, but the demonstration that the high CHO diets were also accompanied by greater reductions in plasma glucose after insulin injection, is consistent with an increase in insulin sensitivity with the high CHO diet. The major effects of dietary CHO occurred as the intake rose above 200 g/d, and has formed the basis of subsequent guidelines that assessments of insulin sensitivity and glucose tolerance should only be made when subjects consume at least 250 g CHO per day. For an individual with a total daily energy intake of 2–2500 kcal (8.4–10.5 MJ), this represents a CHO intake equivalent to 40–50% of total energy, and is well in excess of the minimum needed to match the basal rates of EGP discussed earlier.

Himsworth's observation that glucose tolerance was best with the highest CHO intake (75% of energy) was consistent with a subsequent observation by Thompson *et al* (1978), who fed liquid formula diets to healthy men for periods of 10 d. These diets contained either sucrose or corn syrup (oligosaccharides) as the sources of CHO, and for each of them the effects of diets containing either 45 or 65% of the energy as CHO were determined. With both sources of CHO, glucose tolerance tended to be better with the 65% of total energy as CHO than the 45% diet, and the difference between 65% as sucrose and 45% as corn syrup was statistically significant. It is interesting that the amount of CHO in the diet did not affect mean 24 h plasma glucose or insulin values, again suggesting greater insulin sensitivity with the higher CHO intake.

Since these studies, there has been some concern that very high CHO intakes may produce disturbances of lipid metabolism, which is discussed in more detail later.

Insulin, CHO oxidation and glycogen storage

As mentioned earlier, the ingestion of CHO-containing foods is accompanied by an insulin response which increases CHO oxidation and storage, while reducing EGP. The magnitude of these effects depends upon the amount of CHO ingested and the insulin sensitivity of the individual. An example of the relative distribution of CHO ulitization between oxidation and storage when consuming moderate amounts of CHO is provided by Whitley *et al* (1997). They studied CHO balance in healthy women over 5 h after ingestion of a meal providing 4 MJ (956 kcal) of which 49% was CHO. Over the 5 h period, approximately two-thirds of the ingested CHO was oxidised, and one-third was stored in the body, presumably as glycogen. Isoenergetic meals with smaller CHO contents produced less CHO

storage, such that there was no storage over 5 h when a meal with 20% of the energy as CHO was consumed.

Insulin sensitivity is not only affected by dietary CHO intake, but also by the size of the body's glycogen store. Depletion of muscle glycogen content by exhaustive exercise is followed by an increase in insulin sensitivity when assessed with the hyperinsulinaemic glucose clamp technique (Bogardus *et al*, 1983). Subjects exercised for approximately 45 min at a relatively high intensity, in order to reduce muscle glycogen content by approximately 40%. They then fasted overnight before insulin sensitivity was assessed the next morning, and found to be 17% above the initial values. There was a positive correlation between insulin stimulated glucose disposal and the degree of activation of glycogen synthase in muscle biopsy samples. Therefore, glycogen depleting exercise increases insulin sensitivity, which will ensure efficient replenishment of the glycogen stores when CHO is consumed.

When muscle glycogen concentrations are depleted to 25% or less of initial values (by prolonged, aerobic exercise), it can take in excess of 24 h to replenish the glycogen stores. Under these conditions, glycogen resynthesis is more rapid if the dietary CHO is of the high glycaemic index type, that is, it is rapidly digested and absorbed, producing higher plasma glucose responses. Burke *et al* (1993) showed that after glycogen depleting exercise on two separate occasions, with a subsequent CHO intake of 730 g in 24 h on both occasions, when high glycaemic index foods were consumed, there was almost 50% more glycogen synthesised than when low glycaemic index foods were eaten. Therefore, whilst in most circumstances, individuals should be advised to consume CHO-containing foods which have a low glycaemic index, recovery from glycogen depleting exercise requires a different strategy.

Another situation which affects insulin sensitivity and also the partitioning of glucose disposal between CHO oxidation and storage is undernutrition. It is well established that undernutrition and starvation are accompanied by insulin resistance and impaired glucose tolerance. This was demonstrated by Himsworth (1935) and was discussed in detail earlier. The availability of the glucose clamp technique in combination with indirect calorimetry to assess CHO oxidation has allowed this to be studied in more detail. It is now clear that the insulin resistance produced by acute starvation (up to 72 h) is entirely due to a decrease in the ability of insulin to stimulate CHO oxidation (Mansell & Macdonald, 1990; Webber *et al*, 1994). In healthy subjects who starve for up to 72 h, insulin stimulated glucose disposal is reduced by up to 50%. However, when non-oxidative disposal (storage) is estimated, this is unaffected by starvation. However, it is likely that the site of this glucose storage in the 48–72 h starved state is different from that with shorter periods of food deprivation. Such prolonged starvation will deplete liver glycogen stores more than those in skeletal muscle, thus making it likely that most of the subsequent glucose storage involves replenishment of liver glycogen.

Less severe undernutrition (underfeeding for 7 d) is also associated with reductions in insulin sensitivity, due to a decrease in glucose oxidation with no change in glucose storage (Gallen & Macdonald, 1990). In both starvation and underfeeding, it is likely that the increased availability and oxidation of fatty acids decreases CHO oxidation, due to the Randle Cycle (Randle *et al*, 1963). In undernourished individuals this phenomenon ensures that more of the ingested CHO can be stored as glycogen. However, operation of this cycle also seems to occur in non-insulin dependent diabetes (Henry *et al*, 1991), but in this context the accompanying insulin resistance contributes to the hyperglycaemia which characterises this disease.

Metabolic responses to overfeeding of fat or CHO

Although muscle glycogen content can be increased above normal levels by combinations of exercise and high CHO diets, there is a limit to the amount of glycogen which can be stored in the body. Once this limit is reached, further excess CHO intake should lead to the synthesis of fatty acids from glucose, that is, *de novo* lipogenesis. It might be expected that such excess CHO intake would lead to reduced insulin sensitivity, but in the short term this is not so. Welle & Campbell (1983) increased CHO intake of a group of healthy non-obese men from 250 g/d to 700 g/d for 17 d, such that total energy intake rose by 83%. This overfeeding led to a doubling of CHO oxidation, but CHO intake exceeded this such that *de novo* lipogenesis occurred. In addition, there was little oxidation of fat such that the dietary fat was deposited in adipose tissue. Standard oral glucose tolerance tests conducted throughout the overfeeding period showed improved glucose tolerance (lower plasma glucose concentrations) during CHO overfeeding. During the early stages of overfeeding this improved glucose tolerance was contributed to by increased insulin sensitivity, although eventually it was mainly due to an enhanced insulin secretory response.

Acheson *et al* (1988) made similar observations during short-term CHO overfeeding, showing that when glycogen stores were full, there was lipogenesis from the excess CHO intake. Acheson's subjects consumed approximately 900 g of CHO per day, oxidising between 400 and 500 g of this. The excess 450 g CHO was used for lipogenesis, producing approximately 150 g lipid per day. Therefore, the efficiency of fat synthesis from CHO is low, and is associated with substantial increases in energy expenditure.

Further evidence that excess CHO intake leads to an increase in CHO oxidation is provided by Schwarz *et al* (1995). They studied the effects of less marked increases in CHO intake, increasing total energy intake by 25–50% for 5 d, on CHO oxidation and *de novo* lipogenesis. Increasing CHO intake produced an increase in CHO oxidation and concomitant fall in fat oxidation, again allowing dietary fat to be stored in body tissues. Assessments of hepatic *de novo* lipogenesis showed relatively low absolute rates (approximately 5 g per day) suggesting that adipose tissue was also a likely site of fatty acid synthesis.

The proposition that excess CHO intake leads to a smaller increase in body fat content than an equivalent excess of dietary fat was confirmed by Horton *et al* (1995). They overfed non-obese subjects with a diet containing 50% excess energy for 14 d, with the excess as CHO on one occasion and fat on the other. With CHO overfeeding, there was a marked increase in CHO oxidation, and approximately 80% of the excess energy consumed was stored. By contrast, fat overfeeding did not increase fat oxidation, and almost 95% of the excess energy intake was stored in the body.

Further confirmation that excess CHO intake leads to increased CHO oxidation, but that excess fat intake does not increase fat oxidation, was provided by Jebb *et al* (1996), and Larson *et al* (1995a, b). Jebb's subjects

nearly doubled their habitual energy intake with a mixed diet, and within 5 d of consuming a diet providing 500 g CHO per day, CHO oxidation had increased to match the intake. The consequence was a drop in fat oxidation with a substantial amount of fat storage. The studies by Larson involved overweight subjects of Pima Indian origin. They were allowed to overeat spontaneously, selecting their meals from a cafeteria-style facility, and consumed 25–50% excess energy, with fat providing 40% and CHO 48% of total energy. In both studies, Larson observed increased CHO oxidation and decreased fat oxidation during overeating.

Therefore, there is clear evidence that increasing CHO intake leads to improved glucose tolerance and insulin sensitivity. An excessive CHO intake leads to increased CHO oxidation, and at least in the short-term to less body fat accumulation than seen when excessive amounts of dietary fat are consumed. It is clear that dietary CHO intake should be at least 250 g/d in healthy adults in order to have optimal glucose tolerance and insulin sensitivity. The demonstrations that very high CHO intakes lead to high rates of CHO oxidation may be interpreted as indicating that dietary CHO intake should be as high as possible, provided the total energy intake matches energy expenditure. However, there is some concern that very high intakes of CHO, except in individuals who need to replenish glycogen stores used in exhaustive exercise, may be detrimental and lead to increases in VLDL triglycerides, known risk factors for coronary heart disease.

Benefits and hazards of increasing dietary CHO intake

For more than 10 y, it has been recommended that people with non-insulin dependent diabetes consume a diet high in CHO (with a high fibre content) and low in fat (American Diabetes Association, 1987). It has recently been recognised that this may not be appropriate for patients with more severe diabetes, and an increased dietary monounsaturated fatty acid content is suggested for them. Nevertheless, the advice for most people with diabetes, and for the general population is to consume a high CHO diet, preferably mostly as complex CHO, whereby CHO contributes more than 50% of total energy. Whilst this will undoubtedly maximise glucose tolerance, there is some concern that it may worsen the blood lipid profile by raising plasma triglycerides and lowering HDL cholesterol more than total cholesterol. Smith (1994) suggested that such an effect may be overcome by consuming foods with a low glycaemic index and a high fibre content, although he acknowledges that this proposition has not been formally tested.

The possibility that high CHO diets may be detrimental as far as plasma lipid profile is concerned is reinforced by a recent study by Jeppesen *et al* (1997). They studied the effects of moderate and high CHO diets (40 or 60% of total energy) each for three weeks in a group of overweight (but not obese) post-menopausal women. There was no obvious improvement in glucose tolerance or insulin sensitivity on the higher carbohydrate intake, but there was a rise in total and VLDL triglycerides and a reduction in HDL, but not total, cholesterol. Therefore, in this older age group there were no obvious benefits of changing to a high CHO diet, despite the higher CHO diet containing approximately twice as much dietary fibre as the lower CHO diet.

Dietary CHO intake of populations and overall conclusions

The FAO/WHO (1997) report summarised worldwide population based surveys of dietary CHO intakes undertaken since 1980. CHO intakes as % of total energy were higher in Africa (45–79%) and Asia (57–89%) than in Europe (39–45%) or North America (37–54%). Mean total CHO intakes exceeded 200 g in all surveys, but only exceeded 400 g in Asia and some Pacific Islands. Less complete information was available on the proportions of the CHO derived from starch or simple sugars, but it was noticeable that in Europe, sugars contributed one-third to half of total CHO intake.

From the previous sections, it would appear that healthy, moderately active adults require at least 200 g of CHO per day, and that CHO should represent approximately 50% of energy intake. It would appear prudent to recommend that this CHO is consumed mainly in the form of foods with a low glycaemic index and high fibre content. It should be recognised that very high CHO intakes may produce undesirable effects on plasma lipids, although definitive studies have not been undertaken in healthy younger subjects. An exception to this are individuals with high levels of habitual physical activity, who require a high CHO diet in order to ensure adequate restoration of muscle glycogen stores between exercise bouts. Such individuals may actually benefit from high CHO foods with a high glycaemic index, that is, simple sugars, and oligosaccharides and some readily digestible starches. The potential hazards of a high CHO intake in the elderly, particularly in relation to maintenance of blood pressure after meals, need to be recognised.

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