Carbohydrate bioavailability

Klaus N. Englyst and Hans N. Englyst

Horizons in Nutritional Science

Carbohydrate bioavailability

Klaus N. Englyst* and Hans N. Englyst

Englyst Carbohydrates, 2 Venture Road, Chilworth Science Park, Southampton, UK

There is consensus that carbohydrate foods, in the form of fruit, vegetables and whole-grain products, are beneficial to health. However, there are strong indications that highly processed, fibre-depleted, and consequently rapidly digestible, energy-dense carbohydrate food products can lead to over-consumption and obesity-related diseases. Greater attention needs to be given to carbohydrate bioavailability, which is determined by the chemical identity and physical form of food. The objective of the present concept article is to provide a rational basis for the nutritional characterisation of dietary carbohydrates. Based on the properties of carbohydrate foods identified to be of specific relevance to health, we propose a classification and measurement scheme that divides dietary carbohydrates into glycaemic carbohydrates (digested and absorbed in the small intestine) and non-glycaemic carbohydrates (enter the large intestine). The glycaemic carbohydrates are characterised by sugar type, and by the likely rate of digestion described by *in vitro* measurements for rapidly available glucose and slowly available glucose. The main type of non-glycaemic carbohydrates is the plant cell-wall NSP, which is a marker of the natural fibre-rich diet recognised as beneficial to health. Other non-glycaemic carbohydrates include resistant starch and the resistant short-chain carbohydrates (non-digestible oligosaccharides), which should be measured and researched in their own right. The proposed classification and measurement scheme is complementary to the dietary fibre and glycaemic index concepts in the promotion of healthy diets with low energy density required for combating obesity-related diseases.
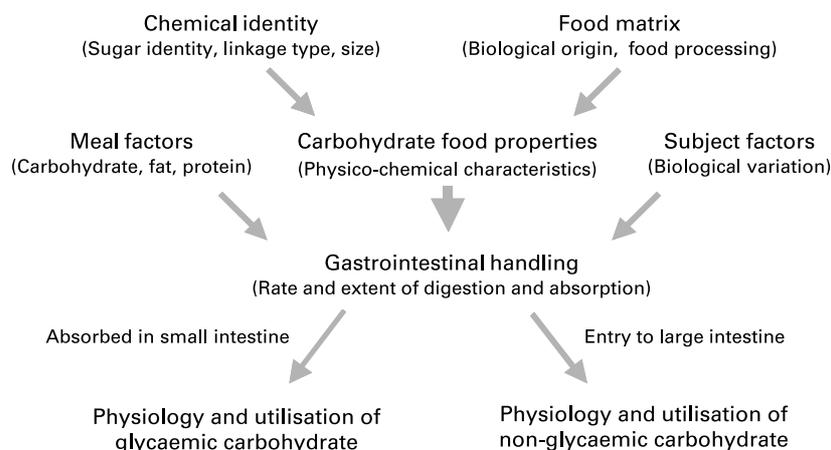
Carbohydrate classification: Starch: Non-starch polysaccharides: Glycaemic index: Dietary fibre

There are concerns about the prevalence of highly processed and energy-dense carbohydrate foods that predominate in many diets and their contribution to the rise in obesity-related diseases (World Health Organization, 2003; UK Parliament Health Committee, 2004). The disruption and removal of the plant cell-wall material (dietary fibre), as occurs with many types of bakery products and breakfast cereals, results in the loss of their original complement of micronutrients, and the starch and sugar components are often made easily digestible. Although rapid digestion and absorption of carbohydrate has benefits for some aspects of sports nutrition, this is not generally considered desirable due to the elevated glycaemic responses that result, especially in those with diabetes or with features of the metabolic syndrome. In contrast, there is evidence that a greater consumption of slow-release carbohydrates is likely to be associated with health benefits (Jenkins *et al.* 2002). Food processing techniques that retain or introduce characteristics that slow carbohydrate digestion should therefore generally be encouraged.

The effects of food processing on the rate and extent of carbohydrate digestion cannot be described by a solely chemical approach to the classification, measurement and food labelling for dietary carbohydrates. The complexities involved in describing carbohydrates in a nutritionally informative fashion may explain why this has lagged behind the description of fat, where specific physiological properties depend on differences in chain length and the number and position of unsaturated bonds. The aim of the present article is to provide a rational basis for the nutritional characterisation of dietary carbohydrates, through a structured exploration of the interrelating factors that determine the bioavailability of carbohydrates.

Carbohydrate bioavailability describes the utilisation and biological effect of dietary carbohydrates; its determinants are reviewed in the first sections of the paper and are summarised in Fig. 1. A clarification of this relationship between the food and the biological function of the carbohydrates is essential in establishing suitable measurements that describe the inherent food properties of nutritional interest. The final sections describe how analytical measures have been developed with consideration to carbohydrate bioavailability, and how these form the basis for a nutritional classification scheme for dietary carbohydrates. The rational for the glycaemic index (GI) and dietary fibre concepts is explored and discussed in the context of carbohydrate bioavailability.

Chemical identity
(Sugar identity, linkage type, size)

Food matrix
(Biological origin, food processing)

Meal factors
(Carbohydrate, fat, protein)

Carbohydrate food properties
(Physico-chemical characteristics)

Subject factors
(Biological variation)

Gastrointestinal handling
(Rate and extent of digestion and absorption)

Absorbed in small intestine

Entry to large intestine

Physiology and utilisation of
glycaemic carbohydrate

Physiology and utilisation of
non-glycaemic carbohydrate

Fig. 1. Summary of the determinants of carbohydrate bioavailability (utilisation and biological function) as described by the interaction between food and meal properties and gastrointestinal handling (for details, see p. 5).

Carbohydrate food properties

The carbohydrate components present in a food can be characterised by: (i) their chemical identities, which are determined by botanical origin and in the case of composite foods the mix of ingredients used; (ii) the food matrix, which in addition to botanical origin is determined by the degree of processing during food manufacture and during food preparation. Together, these physico-chemical properties of the food largely determine the gastrointestinal handling and utilisation of dietary carbohydrates.

The chemical identity of dietary carbohydrates is defined by sugar type, the linkages between sugars and the degree of polymerisation, which determines at the chemical level whether the endogenous digestive enzymes can hydrolyse the carbohydrate

and in what form they are presented for metabolism. The chemical classification of dietary carbohydrates is summarised in the chemical components column of Table 1.

The food matrix has a central position in the concept of carbohydrate bioavailability. In unrefined plant foods the cell-wall NSP have a structural role in maintaining the integrity of the cells, which in turn form the building blocks of the plant tissue. This produces an encapsulation effect, which restricts the rate at which starch and sugars are digested and absorbed in the small intestine. However, excessive food processing destroys the encapsulation effect: for example, the food matrix properties are largely lost when whole grains are milled (Heaton *et al.* 1988; Bjorck & Liljeberg-Elmstahl, 2003) and when fruit such as apples are stewed or juiced (Haber *et al.* 1977). It should be recognised that these unique

Table 1. Carbohydrate bioavailability classification

Main category	Chemical components	Nutritional grouping	Main biological function
Glycaemic carbohydrates	Free sugars	Fructose from free sugars	Largely metabolised by liver. Possible detrimental effect on lipid metabolism
	Maltodextrins	RAG and SAG	RAG and SAG reflect the rate of glucose release from food, which is an important determinant of the glycaemic index
	Starch		Evidence to suggest that metabolic response associated with slow-release carbohydrates (SAG) are most conductive to optimal health
Non-glycaemic carbohydrates		Resistant starch	Varied rate and extent of fermentation
	NSP	Intrinsic NSP: naturally occurring as cell-wall material in plant foods	Food matrix moderates carbohydrate release Marker for high-fibre diet, rich in micronutrients for which benefit to health has been shown
		Added NSP	Varied rate and extent of fermentation
	RSCC	Intrinsic RSCC: natural	Varied rate and extent of fermentation
		Added RSCC	
	Sugar alcohols	Present naturally and added	Absorbed, but not metabolisd. Fermented

RSCC, resistant short-chain carbohydrates; RAG, rapidly available glucose; SAG, slowly available glucose.

structural food matrix properties found in largely unprocessed plant foods cannot be reintroduced by the addition of 'fibre supplements'.

In the plant, starch exists in the form of starch granules, which due to their comparatively small size survive milling relatively intact. This means that despite the disruption of the plant cell-wall structures in raw flour preparations, the access of digestive enzymes is still restricted by the starch granule. The form of the crystalline structure of starch granules is an important determinant of their digestibility, and is determined by botanical origin. The starch granules in potato and plantain (and green banana) are very resistant to pancreatic amylase, the digestibility of legumes is intermediary and cereal starch granules are typically more susceptible to digestion (Gallant et al. 1992). The ratio of amylose:amylopectin can have an effect on starch digestibility, as amylose tends to form secondary structures that are hard to disperse, both in the starch granule and after food processing. However, for most foods the influence of amylose:amylopectin is overshadowed by the much greater effect of food processing. The exceptions are the very high-amylose starches, which can be very difficult to disperse and largely resist digestion in the small intestine (Gallant et al. 1992).

When heated in the presence of water, starch granules disrupt and gelatinise into a form easily available to pancreatic amylase (Colonna et al. 1992). While cooling does not reverse this process, some starch, especially high-amylose starches, may retrograde into forms less susceptible to digestion. Examples are bread and extruded breakfast cereals, which are digested rapidly apart from a small proportion of retrograded starch, which is more resistant to hydrolysis. Heating in the absence of water does not result in gelatinisation, and starch granules remain intact in some types of dry baked foods, as with some biscuits, and the starch in such products is therefore digested slowly (Englyst et al. 2003).

Some types of food processing result in dense secondary structures, which will slow the rate of enzymatic hydrolysis. For example, although pasta products are made from flour and are moist cooked so that the starch granules are gelatinised, they are still digested slowly due to the presence of the dense food matrix (Thomsen et al. 1994; Englyst et al. 1999).

Gastrointestinal handling

The gastrointestinal fate of carbohydrates defines whether they are glycaemic, i.e. they are absorbed in the small intestine and available for metabolism, or non-glycaemic, i.e. they enter the large intestine as substrates for fermentation (Fig. 1). The main determinants of the gastrointestinal handling in the small intestine are the carbohydrate food properties, which are the focus of this paper, but the overall meal composition and subject biological variation will have an influence as well.

Due to the nature of their glycosidic bonds, NSP and resistant short-chain carbohydrates (RSCC) cannot be hydrolysed by the endogenous enzymes of the small intestine and are therefore non-glycaemic (see Table 1). The sugar alcohols are non-glycaemic; even though some are absorbed sparingly in the small intestine, they are not metabolised and are excreted in the urine, and the unabsorbed sugar alcohols are fermentation substrates in the large intestine (Livesey, 2001).

On the basis of chemical identity alone, starch and sugar have the potential to be digested and absorbed in the small intestine.

In practice, this is a continuous process during passage through the small intestine, which, for a number of different reasons, may not go to completion. Sugar may escape digestion through encapsulation in the food matrix but the amounts would be small. In some individuals sugar may enter the large intestine due to genetic variation in the expression of the brush border enzymes required to hydrolyse disaccharides, the commonest cause being lactase deficiency (Gudmand-Hoyer, 1994).

The fact that, in addition to NSP, some starch may escape digestion was established by several studies that used human ileostomy subjects as a model to investigate the digestive physiology of the small intestine (Englyst & Cummings, 1985, 1986, 1987). Analysis of the effluents led to the definition of resistant starch (RS) as 'the starch and starch degradation products that on average reach the large intestine'. This definition recognises that the amount of starch reaching the large intestine will vary between individuals and may be affected by other factors that influence gastrointestinal transit time or inhibit starch hydrolysis. Gastrointestinal transit time is related inversely to the amount of starch escaping digestion (Silvester et al. 1995), which is not unexpected, as transit time will relate to the time of exposure to hydrolytic enzymes. The interaction effects of other meal components such as fat, protein and NSP on the extent of starch digestion have not been established conclusively due to the difficulties involved in achieving quantitative measures of starch entry into the large intestine.

The rate at which starch and sugars are digested and absorbed during transit of the small intestine has received considerable interest due to the association with the glycaemic response and postprandial substrate metabolism. Carbohydrate foods that contain free sugars, gelatinised starch and an easily dispersed food matrix will be digested and absorbed rapidly. Carbohydrate food properties that restrict enzyme access to starch and otherwise slow carbohydrate release from the food matrix will prolong the digestion process (Englyst et al. 2003).

Biological variations in gastrointestinal function and the interactive effect of meal components will influence the rate that carbohydrates are digested and absorbed. The rate that food is released from the stomach is regulated by the process of gastric emptying, which is affected by many factors, including the amount and type of macronutrients in a meal, the meal volume, food particle size, viscosity and pH (Rayner et al. 2001). These exert their effects by their physical presence in the stomach, and via a range of gut hormones as the incretin response. Once in the small intestine, physical properties such as viscosity will influence the stirred layer that determines the extent of enzyme access to, and nutrient release from, the alimentary food bolus (Cherbut, 1995).

Utilisation and biological function of dietary carbohydrates

Glycaemic carbohydrates

For these, bioavailability relates to the transport of the absorbed sugars to the tissues where they can be utilised, and the physiological mechanisms that regulate the body's overall substrate partitioning. The utilisation and physiology of glycaemic carbohydrates should therefore be explored at several levels: (i) their rate of appearance in the portal vein; (ii) the glycaemic response elicited; (iii) their uptake and utilisation by tissues;

(iv) the overall impact on carbohydrate and fat metabolism (substrate partitioning).

The rate of carbohydrate appearance in the portal vein is a direct function of absorption from the small intestine. To overcome the problems of direct measurements from the portal vein, the appearance of a labelled dietary carbohydrate can be followed in the peripheral blood, although tissue uptake and oxidation need to be taken into account in such studies (Robertson *et al.* 2002).

The commoner approach is to measure the overall glycaemic response in the peripheral circulation, which is the sum of net entry of exogenous glucose from the portal vein and endogenous glucose from hepatic output, and net removal of glucose by tissues. The actual size of the postprandial glycaemic response can vary greatly between subjects due to biological variation in hepatic glucose output and tissue glucose uptake, which means that nutritional investigations need to express relative glycaemic responses within individual subjects. Other factors such as time of day, previous meals and physical activity can influence blood glucose levels. If all these factors are controlled for during testing conditions, then glycaemic response represents a useful tool for investigating carbohydrate digestion and absorption. This approach has been applied successfully in the form of the GI measure, which ranks the foods by the extent that blood glucose is elevated in response to a portion of food containing 50 g carbohydrate (Jenkins *et al.* 1981).

However, there are some situations where the glycaemic response does not reflect carbohydrate absorption from the small intestine. For instance, a lower glycaemic response can result from an increased clearance of glucose from the circulation due to elevated levels of insulin. The insulin secretagogue effect of dietary protein is the best known example of this (Westphal *et al.* 1990), but may be due to any dietary factor that stimulates insulin secretion either directly or indirectly through the incretin response.

The other situation that has an impact on glycaemic response is the type of sugar consumed. Fructose and galactose elicit incremental glycaemic responses that are about 20 % that of the same amount of glucose. This is not due to a slower absorption, but rather reflects the fact that these glycaemic sugars are removed rapidly from the portal blood by the liver and enter the peripheral circulation in only small amounts (Nuttall *et al.* 2000; Gannon *et al.* 2001). Although small amounts of fructose and galactose are transformed to glucose and return to the circulation, the majority enters the carbohydrate and fat metabolic pathways of the liver (Mayes, 1993; Frayn & Kingman, 1995).

At the level of utilisation, the bioavailability of glycaemic carbohydrates relates to the rate that they are oxidised. Indirect calorimetry studies have been used predominantly to investigate how different ratios of macronutrient have been utilised, with few studies investigating within the context of carbohydrate bioavailability. However, it has recently been confirmed that fructose is oxidised more rapidly than glucose (Daly *et al.* 2000). Furthermore, a slower rate of starch digestion and absorption reduces the extent of the switch from fat to carbohydrate oxidation in the postprandial period, particularly in diabetic subjects (Seal *et al.* 2003). Still unresolved is the effect of an increased clearance of blood glucose due to protein-stimulated insulin secretion.

Investigations of the role of dietary carbohydrates in lipid metabolism have mostly concentrated on the effects of altering the amount, more than the type, of carbohydrate (Parks & Hellerstein, 2000). However, there is evidence from animal studies suggesting that high fructose intakes elevate triacylglycerols and decrease insulin sensitivity, but the situation in man is less clear (Daly *et al.* 1997). Human studies have shown that, compared with a high-starch diet, a high-sucrose diet results in an elevated late postprandial rise in triacylglycerols (Daly *et al.* 1998). The beneficial effect on lipid metabolism of slowing the rate of carbohydrate release in the small intestine has been demonstrated in numerous studies with low-GI diets (Jenkins *et al.* 2002). Further support for the importance of carbohydrate type comes from epidemiological investigations that have found lower HDL-cholesterol with low-GI diets (Frost *et al.* 1999; Liu *et al.* 2001). Critically, it appears that the extent of the effect of the amount and type of carbohydrate on lipid metabolism is linked closely to the underlying insulin sensitivity of the subjects (Jeppesen *et al.* 1997).

Non-glycaemic carbohydrates

These are not absorbed and utilised directly, which distinguishes them from most other nutrients. Instead, their utilisation and biological functions relate to their diverse physical and chemical properties in the gastrointestinal tract and their role as substrates for the gut microflora.

In the stomach and small intestine, the intrinsic plant cell-wall NSP (dietary fibre) have, due to the encapsulation of other nutrients, a unique role in slowing carbohydrate digestion and absorption. When added to foods in high amounts, soluble 'fibre preparations' can increase viscosity and slow absorption (Cherbut, 1995). Some NSP promote bile acid secretion, which has been proposed as a mechanism for lowering cholesterol (Truswell, 1994). Another functional role is the lectin-binding capabilities of specific species of non-glycaemic carbohydrates, which imparts anti-adhesive properties against pathogenic bacteria and mitogenic dietary lectins (Steer *et al.* 2000). In a case–control study of colon cancer and dietary intake (with values for individual NSP constituent sugars), NSP galactose showed a dose-related protective effect against colon cancer and it was suggested that this was due in part to the anti-adhesive properties against galactose-binding lectins (Evans *et al.* 2002).

In the large intestine, the non-glycaemic carbohydrates are all potential substrates for fermentation, a process that results in SCFA that can be absorbed and utilised by the host as an energy source. To describe non-glycaemic carbohydrates as non-digestible would therefore be potentially misleading. The chemical identity and physico-chemical properties of the non-glycaemic carbohydrates determine the rate (site) and extent of fermentation, and therefore the physiological effects. The less-fermentable carbohydrates, which tend to be the insoluble species such as wheat bran, have an important role in absorbing water and providing faecal bulk (Cummings, 1993). Many studies have investigated the effects of isolated preparations of non-glycaemic carbohydrates, with some types stimulating growth of specific bacteria (prebiotic effect), e.g. fructo-oligosaccharides linked with elevation of the beneficial bifidobacteria (Van Loo *et al.* 1999).

The consequence to health of potentially large amounts of RS reaching the large intestine is unknown. There have been some claims for the health benefits of RS; e.g. fermentation of RS in the human large intestine has been shown to reduce faecal ammonia (Birkett *et al.* 1996). The SCFA produced by fermentation of carbohydrates, including RS, have been implicated as a protective

factor against colon cancer (Van Munster *et al.* 1994). However, Young *et al.* (1996) have shown that RS can enhance tumour formation in rats and Burn *et al.* (1996) have shown significant enhancement of cancer formation by RS in a mouse model.

At this stage, not enough is known about the biological effects of the different amounts and fermentabilities of non-glycaemic carbohydrates. It would be inappropriate to conclude that all non-glycaemic carbohydrates are beneficial to health, just because they can be fermented (Wasan & Goodlad, 1996). Detailed chemical characterisations are essential for further elucidating the biological function of different amounts and types of non-glycaemic carbohydrates.

Measurement of glycaemic carbohydrates

By combining what is known about food properties, gastrointestinal handling and utilisation, the important issues relating to the bioavailability of glycaemic carbohydrates are identified as: (i) the type of sugar presented for metabolism; (ii) the rate of carbohydrate release from foods (see Table 1).

After digestion and absorption from the small intestine, the three types of monosaccharide that are presented for metabolism are glucose, fructose and galactose. Distinguishing between these glycaemic carbohydrates is of nutritional relevance due to their varied metabolic fates. For the purposes of characterising the plant-derived carbohydrates, the glycaemic glucose fraction is that derived from free glucose, maltose, starch digested in the small intestine and the glucose portion of sucrose. The total fructose fraction is the sum of free fructose and the fructose portion of sucrose. Lactose is derived from dairy products and therefore its glucose portion is not included in the glycaemic glucose fraction of plant origin, rather separate values for lactose should be included alongside the other carbohydrate fractions where appropriate.

The food properties that determine the digestibility of carbohydrates can be described by measuring the carbohydrate release characteristics under controlled *in vitro* conditions. The methodology for the determination of rapidly available glucose (RAG), slowly available glucose (SAG) and the non-glycaemic RS fraction has been developed to describe the likely rate and extent of glucose release from starch and sugars in the small intestine (Englyst *et al.* 1992, 2000b; Englyst & Englyst, 2004). The profile of RAG, SAG and RS values is determined by the physico-chemical characteristics of the food, which relates to the structural properties of the food matrix, the presence of intact plant cell walls, and the type and integrity of starch granules. It is these physico-chemical characteristics of foods that are the main determinants of the gastrointestinal fate and bioavailability of the carbohydrate component (Fig. 1). The *in vitro* bioavailability characteristics for a range of foods are shown in Fig. 2. There is variation in the SAG content within each product group, but, in general, breakfast cereals and bakery products have low SAG values, whereas whole grains, pasta and some types of biscuits have higher SAG values.

The physiological relevance of the division between RAG and SAG has been demonstrated in a series of studies investigating their relationship with glycaemic response and GI values. A study with thirty-nine starchy foods showed that RAG values were correlated strongly to published GI values (Englyst *et al.* 1996). This relationship was further confirmed in a study investigating the determinants of GI and insulinaemic index values of cereal products (Englyst *et al.* 2003). The carbohydrate model including the RAG and SAG fractions described 68 % of the variance in GI between products compared with 33 % described by the model with only starch and sugar. Another study investigated four foods with differing proportions of RAG and SAG consumed as either 25 g or 50 g carbohydrate portions. This demonstrated that it was RAG, by virtue of its rapid digestion and absorption in the small intestine, that was the main determinant of the postprandial
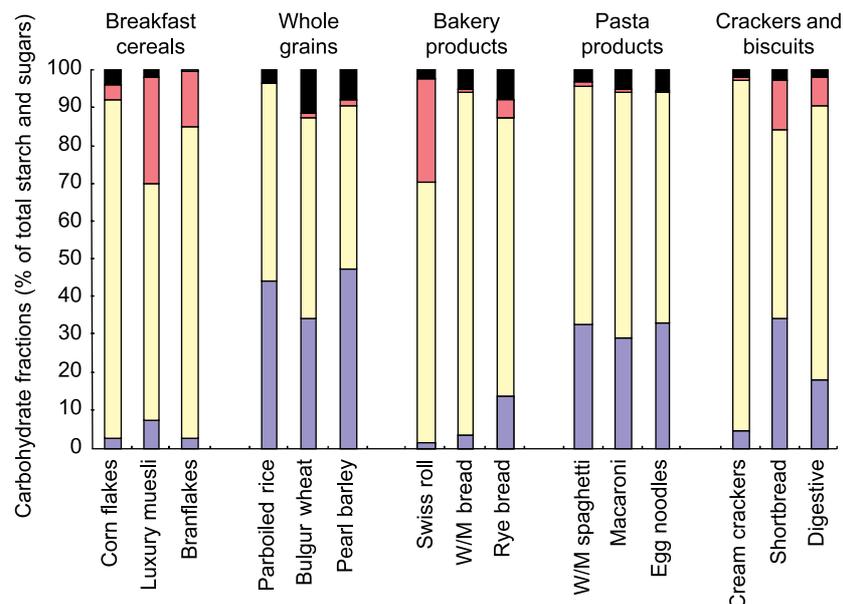

Fig. 2. Rapidly (□) and slowly (■) available glucose, fructose (■, including that derived from sucrose) and resistant starch (■) in some cereal products (W/M, wholemeal). Carbohydrate fractions are expressed as a percentage of the total starch and sugars.

rise in blood glucose concentrations (Englyst *et al.* 1999) and, furthermore, that SAG exerted its lowering effect on glycaemic response by replacing RAG in the test meal.

Due to the variation in both the rate and extent of starch digestion that is seen both within and between individuals in human studies, *in vitro* measurements are based on the average of measurements observed *in vivo*. Inevitably, the rate and extent of carbohydrate digestion of a food or meal will be subject to the effects of biological variation associated with physiological differences between individuals and within the same individual in different circumstances. This variance does not diminish the value of the RAG, SAG and RS values as measures of carbohydrate bioavailability, as what is required and provided is a consistent means of characterising these specific properties of foods, allowing a comparison between different products.

The *in vitro* bioavailability values for individual food items can be added to provide a definitive description of the carbohydrate component of the meal or diet. It is this unambiguous description of the carbohydrate release characteristics that provides the power of the RAG and SAG measures. It is important to remember that this approach to characterising carbohydrate bioavailability focuses solely on the *in vitro* rate of carbohydrate release from foods and the demonstrated relationship with glycaemic response. It does not describe the other dietary factors that can affect the glycaemic response.

Glycaemic index concept

The GI is an *in vivo* measure that takes into account the complex interaction of factors that determine the glycaemic response. In addition to the rate of carbohydrate digestion, food-mediated effects on both gastrointestinal events and post-absorptive metabolism can influence the GI. This emphasises the fact that GI values do not represent a direct measure of carbohydrate absorption from the small intestine. Rather, the GI values are determined by the combined effect of all the properties of a food or meal that influence the rate of influx and removal of glucose from the circulation.

However, it is not possible to identify which food factors are responsible for the GI of any specific food or meal. This is an important point, as the different food factors that lower the glycaemic response do not have the same overall health benefits. For example, low GI values can be achieved by:

(1) The presence of intact plant cell-wall structures, dense food matrices or ungelatinised starch granules that are digested slowly (Englyst *et al.* 2003). Such slow-release carbohydrate foods are recognised to be beneficial to health;

(2) A high fructose content will not elevate glycaemic response due to its rapid metabolism by the liver. But, there are concerns about the effects of this rapid carbohydrate utilisation on overall substrate partitioning (Daly *et al.* 1997);

(3) Adding guar gum or other viscous polysaccharide preparations to foods can slow gastric emptying as well as limit enzymatic hydrolysis in the small intestine by restricting access to the food bolus. But the benefits or otherwise of these supplements in the large intestine are unknown (Wasan & Goodlad, 1996);

(4) A high fat content can slow gastric emptying, thereby slowing entry of the carbohydrate into the small intestine (Collier *et al.* 1984). However, such lowering of GI values cannot counteract the detrimental effect of increased energy intake from the fat;

(5) A high protein content can stimulate insulin secretion, promoting blood glucose uptake and utilisation by tissues (Westphal *et al.* 1990). But high levels of insulin may have detrimental effects, and the overall implications for substrate metabolism are unknown.

Based on GI values alone, these examples would be indistinguishable from one another, which highlights the limitations of focusing on a single physiological parameter. The glycaemic response is only one of the physiological effects of carbohydrate foods that must be taken into account.

The diversity of factors that can influence the glycaemic response has implications for the utility of the GI in mixed-meal situations. One concern that has been raised is that the overall macronutrient composition of mixed meals will influence the glycaemic response to the meal in a way that is not reflected adequately by the combination of individual food GI values, especially when the meal includes added fat or protein components. This issue is demonstrated by the study of Flint *et al.* (2004), where meals with added macronutrient components (fat and cheese) did not result in the glycaemic responses predicted by literature GI values. It should be noted that in this study most of the variation in the predicted GI values was achieved by modifying the amount of milk (lactose) added, rather than selecting foods with different rates of starch digestibility.

In order to be a functional tool, measures of nutritional characteristics need to provide a clear description of the inherent properties of the carbohydrate food. The influence of fat and protein on the glycaemic response must be considered as a separate issue from these inherent properties of carbohydrate foods. For example, white bread has a GI of 70 when tested on its own, but a ham and cheese sandwich made with the same amount of white bread could have a GI of 50 due to the effects of the added meal components. While it may be argued that the sandwich represents a more realistic meal, the carbohydrate properties of bread can clearly not be improved by adding cheese and ham, although this issue can be potentially misleading for the consumer. Instead, pasta with its similar macronutrient profile provides a fairer comparison and with its lower GI of 40 would be a good substitute for white bread.

As the GI is a qualitative ranking of foods, it requires additional knowledge of carbohydrate content for it to be used quantitatively. This has the potential for confusion if foods with different carbohydrate contents are compared. For example, carrots and white bread have similar GI values, but have different carbohydrate contents, with a given weight of carrots containing much less carbohydrate than the same weight of bread. It is therefore essential that, in selecting a low-GI diet, appropriate consideration be given to each food according to the amount of carbohydrate that it contributes to the diet. Carrots should not be excluded from the diet because of their high GI, as they provide only a small proportion of total carbohydrate intake and, more importantly, represent a good source of micronutrients.

The glycaemic response to a meal is influenced by other factors, such as the amount of carbohydrate, the meal volume, previous meals and the recent physical activity of the subject. Overall, the acute (physical activity and second meal effects) or intermediate factors (weight change) that influence insulin sensitivity will affect the absolute physiological responses to diets with

different GI values. Therefore, more so than most other metabolic studies, those investigating the GI are likely to be confounded by other lifestyle factors. Nevertheless, the majority of the evidence suggests that, over an extended period of time, the accumulated effect of modest reductions in glucose and insulin excursions with low-GI diets will result in improvements in a range of metabolic risk factors (Jenkins *et al.* 2002; Wolever, 2003; Opperman *et al.* 2004). Subjects with diabetes or with features of the metabolic syndrome are likely to achieve the greatest improvements with the low-GI diet. In contrast there can be situations, such as in sports nutrition, where high-GI meals may improve physical performance and recovery (Burke *et al.* 2004).

Another potentially very important line of research with the GI has been its influence on satiety, hunger, energy intake and ultimately obesity. The evidence from short-term studies suggests that low-GI diets increase satiety and decrease hunger compared with high-GI diets (Ludwig, 2000; Brand-Miller *et al.* 2002). The longer-term benefits for energy balance with low-GI diets has been demonstrated in children as part of an obesity programme (Spieth *et al.* 2000). This is of considerable importance for the population as a whole, as weight loss improves insulin sensitivity and other features of the metabolic syndrome.

A fairly recent application of the GI has been in epidemiological studies as an assessment of the carbohydrate quality (diet GI) or the global insulin demand (glycaemic load) of the diet. Such approaches have indicated that the source of carbohydrate in the diet influences disease markers and disease incidence (Salmeron *et al.* 1997; Liu *et al.* 1998; Frost *et al.* 1999), although this has not been consistent (Meyers *et al.* 2000; Van Dam *et al.* 2000). The lack of clarity as to which factors are involved in the determination of diet GI values and glycaemic load scores has the potential to confound the interpretation of any observed links with health-related outcome measures.

Despite the continued accumulation of scientific support for the GI concept, its utility as a practical nutritional tool is still debated. The main issues can be summarised thus:

(1) GI values provide only a qualitative ranking expressed in terms of the glycaemic carbohydrate component of a food and require additional quantitative information in order to be applied in practice;
(2) Due to its basis as a physiological measurement, there has been an unrealistic expectation that the GI will always predict the glycaemic response, when in fact this relationship will be confounded by other meal factors and subject variation;
(3) The role of GI as a nutritional ranking of foods will be diminished if this terminology is used to describe dietary effects on glycaemic response other than those that are due to inherent carbohydrate food properties;
(4) GI values alone cannot distinguish between different types of low-GI foods or diets that can have varied effects on overall physiology.

If addressed, these potential limitations should not present a barrier to the wide-scale acceptance of the GI concept. If these issues are not resolved, the long-term consequence would be that the GI is left open to misinterpretation and will not provide the clear consistent message that is essential for the concept to succeed.

Many of these issues for the GI concept can be resolved by consideration of the physico-chemical properties of the foods being investigated, thereby providing information about the mechanisms responsible for the observed GI value. Such information can be provided conveniently by the *in vitro* bioavailability measures describing the sugar type and rate of carbohydrate release characteristics. The additional benefits of the *in vitro* bioavailability measures are:

(1) To provide the direct determination of the glycaemic carbohydrate composition that is essential in calculating the 50 g carbohydrate portion used in GI testing;
(2) Their expression in g/100 g food, thereby providing direct quantitative information on the carbohydrate content;
(3) To provide information on the inherent food properties that relate to their likely physiological properties, without giving the unjustified impression that they will predict glycaemic response in all situations;
(4) To provide the means with which to distinguish between different types of low-GI foods that may not all be beneficial, and to promote those containing the beneficial sustained-release carbohydrates.

The GI and the *in vitro* carbohydrate bioavailability measures are complementary, and only together do they provide a comprehensive picture.

Measurement of non-glycaemic carbohydrates

The non-glycaemic carbohydrates have a range of biological functions. Based on the measurement of chemically identified components, but taking into account known biological functions, a classification and measurement scheme (summarised in Table 1) has been developed for quantifying the three main types of non-glycaemic carbohydrates: NSP; RSCC; RS.

Non-starch polysaccharides

This fraction is divided into the naturally occurring intrinsic NSP that impart rigidity to the plant structure, and encapsulate and thus control the release of other nutrients, and added NSP, which include gums and refined preparations of cell-wall material that occur in foods mainly as additives. It is the intrinsic NSP that provide a good marker for the naturally fibre-rich diet for which health benefits have been shown.

Detailed working procedures are described elsewhere (Englyst *et al.* 1994, 2000a). Total, soluble and insoluble NSP with values for the constituent sugars have been published for a wide range of plant foods (Englyst *et al.* 1988, 1989). Fig. 3 illustrates the amount and type of NSP for a selection of cereal, fruit and vegetable products. Although cereals have the highest NSP content expressed 'as eaten' (Fig. 3(A)), the fruit and vegetables have a much higher NSP as a proportion of DM, reflecting their low energy density (Fig. 3(B)). The spectrum of constituent sugars, which through glycosidic linkages form the various types of NSP, is characteristic for different plant foods (Fig. 3(C)). Xylose is found predominantly as arabinoxylans in cereal products, whereas uronic acids from pectin are present only in fruit and vegetables. Glucose is present in all food types, as cellulose in fruit and vegetables, and as both cellulose and β-glucans in cereals. Arabinose, mannose and galactose are present in the NSP of all food types, with the minor constituents rhamnose and fucose being present in only some fruit and vegetables.
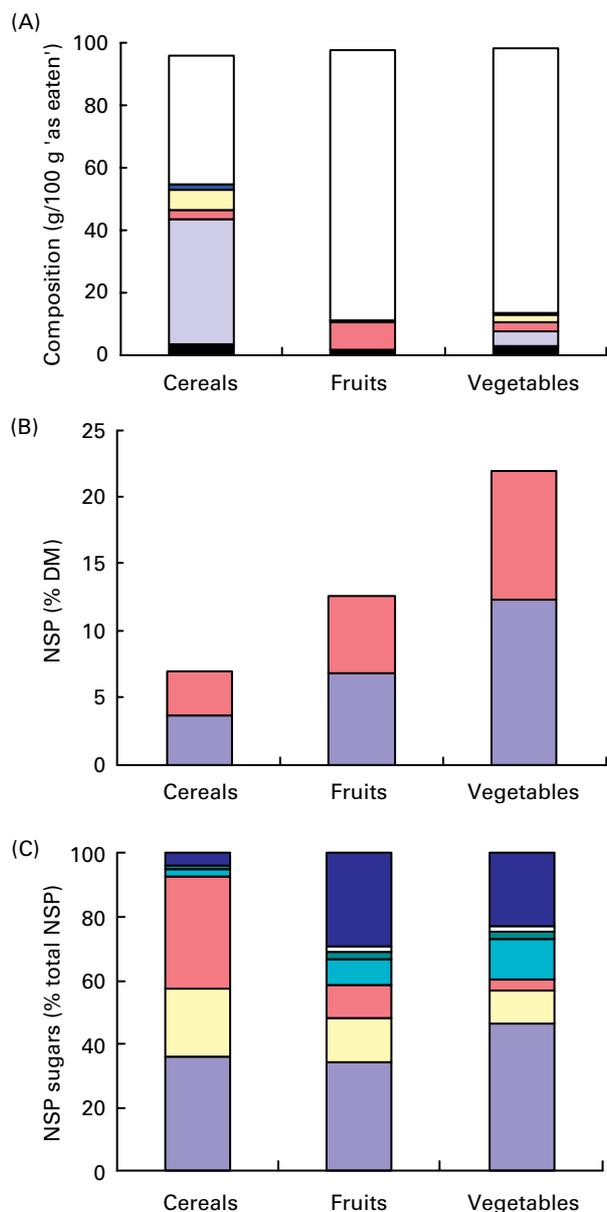
Fig. 3. NSP in different food groups. (A) Expressed as g/100 g 'as eaten' and including values for other macronutrients (■, fat; □, protein; ■, sugar; □, starch; ■, NSP) and water content (□). (B) NSP expressed as a percentage of DM and including values for soluble (■)/insoluble (■) NSP fractions. (C) NSP constituent sugars (■, uronic acids; □, rhamnose; ■, mannose; ■, galactose; ■, xylose; □, arabinose; ■, glucose) expressed as a percentage of total NSP. The foods in each group (NSP in g/100 g DM and water content of products 'as eaten') are: for cereals, wholemeal bread (9·2, 41·2), rye bread (13·3, 37·4), white bread (2·7, 37·4), corn flakes (0·9, 3), porridge (8·5, 87·3); for fruits, apple (13·3, 84·5), orange (15·0, 86·1), peach (13·5, 88·9), pineapple (9·1, 86·5), strawberry (11·9, 89·5); and for vegetables, cabbage (37·4, 90·1), carrot (26·0, 89·8), pea (20·9,74·6), potato (6·7, 81·7), tomato (22·7, 93·1). The composite values for each food group, in the three parts respectively, are calculated based on equal amounts of (A) g/100 g food 'as eaten', (B) g/100 g food 'DM' and (C) NSP content.

The solubility of NSP (Fig. 3(B)) is dependent on chemical structure (chain length and branching) and although cereals tend to have lower proportions of soluble NSP than fruit and vegetables, in the present example the cereal group includes barley and oat products, which contain high amounts of soluble β-glucans. The value of a solubility division of dietary fibre has been questioned (Food and

Agriculture Organization of the United Nations/World Health Organization Expert Consultation, 1998), in part due to the variation in values obtained by different methods using different pH extractions. While *in vitro* solubility is a somewhat arbitrary division, when extraction conditions are standardised, it can provide a simple indicator of physico-chemical parameters of NSP including viscosity, water holding and in some cases fermentability. Detailed information on NSP composition can be applied in metabolic and epidemiological studies investigating the link between the type of carbohydrate and health.

Resistant short-chain carbohydrates

This fraction encompasses all the non-glycaemic carbohydrates that are soluble in 80 % ethanol, other than the sugar alcohols, which are analysed separately. This includes carbohydrate species with a degree of polymerisation up to about 50, depending on sugar identity and branching. The RSCC fraction includes the species often described as the non-digestible oligosaccharides.

Although only a few foods contain RSCC in significant amounts, it is important to identify them to achieve a comprehensive determination of carbohydrate composition. In addition, these carbohydrates can be isolated or manufactured and used as food ingredients, and it is essential that these fractions can be characterised so that their biological functions can be established (Cummings *et al.* 2001).

Resistant starch

In the early development of the NSP procedure, a fraction of starch was identified that could not be hydrolysed without prior chemical dispersion (Englyst *et al.* 1982). This starch fraction, identified subsequently as retrograded starch, was termed RS. Later, in conjunction with ileostomy studies (Englyst & Cummings, 1985, 1986, 1987), other forms of RS were identified. RS_1 is physically inaccessible starch present in foods having a dense or rigid structure, e.g. whole-grain cereals and legumes; RS_2 is RS granules present in raw foods, e.g. bananas; RS_3 is retrograded starch present in foods that have been cooked and then cooled, e.g. bread, breakfast cereals and cold potatoes.

The RS content of food is very dependent on the degree of food processing, which can result in an increase or a decrease in the RS values from those found in the raw natural product. Therefore, RS needs to be measured in foods as they would normally be eaten and, for food labelling purposes, values cannot be derived by summing the RS contents of raw ingredients or indeed be measured in samples that have undergone laboratory preparation (freeze drying/milling) before analysis, as this can influence the RS content. The measurement of RS is part of the starch digestibility (RAG/SAG) procedure described earlier. Although other procedures for the determination of RS have been proposed (recently reviewed by Champ *et al.* 2003), these have seldom been validated by *in vivo* studies, and do not always incorporate the analysis of samples 'as eaten'. As a consequence such data do not reflect the actual amount of RS.

The dietary fibre concept

The dietary fibre hypothesis (Trowell, 1972, 1985), that a diet of unrefined plant foods is protective against Western diseases of affluence, has gained considerable support. There is convincing

evidence and a general consensus that: (i) naturally fibre-rich diets, i.e. those rich in fruit, vegetables and whole grains, are beneficial to health; (ii) some of the benefits to health are not due to fibre or carbohydrates directly, but reflect the fact that such diets tend to be rich in vitamins, minerals, antioxidants and other phytochemicals, and are often low in fat. It is essential that any measure of dietary fibre is a true marker of this naturally fibre-rich diet.

The term 'dietary fibre' was coined to describe the plant cell walls seen to be characteristic of unrefined plant foods. Consequently, dietary fibre was defined in terms of the plant cell walls, as 'the skeletal remains that are resistant to digestion by the enzymes of man' (Trowell, 1972). The principal components (approximately 90 %) of plant cell walls are polysaccharides that do not have α-glucosidic linkages and therefore collectively are termed NSP (Trowell 1985; Englyst et al. 1987). It is on these grounds that the naturally occurring (intrinsic) plant cell-wall NSP content of foods is a good marker for the unrefined plant foods embodied in the dietary fibre hypothesis. This was recognised by the recent joint WHO/FAO Expert Consultation on Diet, Nutrition and the Prevention of Chronic Diseases, which identified 'NSP (dietary fibre)' as one of the strongest dietary factors in the prevention of obesity, diabetes and CVD (World Health Organization, 2003).

The issue of defining and measuring dietary fibre for food labelling purposes has been the subject of considerable debate. It has been suggested that other types of non-glycaemic carbohydrates than plant cell-wall NSP should be included within the dietary fibre concept because, like the cell-wall NSP, they enter the large intestine undigested. However, the inclusion of material other than plant cell walls as dietary fibre has the potential to mislead the consumer, who has the expectation that dietary fibre labelling provides guidance towards the largely unrefined plant foods shown to be associated with health. The lack of evidence for consistent beneficial effects of fibre supplements or RS on colorectal cancer suggests that indigestibility alone does not automatically equate to health benefits and that large amounts of non-glycaemic carbohydrates may even be detrimental (Wasan & Goodlad, 1996; Food and Drug Administration, 2000; Goodlad & Englyst, 2001). Together, this suggests that the measure of dietary fibre should be limited to plant cell-wall material or NSP as a marker of this.

Nevertheless, specific health benefits have been associated with some individual non-glycaemic carbohydrate species; for example, the bifidogenic properties of fructans and the moderation of glycaemic response by certain viscous soluble NSP fractions. The potential health benefits of such non-glycaemic carbohydrate 'functional food' preparations should be recognised, researched and promoted in their own right. If, however, such material were to be grouped as dietary fibre, then the term would lose its status as a marker and measure of the largely unrefined plant-food diet that, with its content of cell-wall material and associated vitamins, minerals and antioxidants, is recognised as providing unique health benefits.

Irrespective of the debate on the definition of dietary fibre, it is essential to have an unambiguous classification and measurement scheme that includes all non-glycaemic carbohydrates.

Concluding remarks

With some types of dietary carbohydrates protecting against and others increasing the risk of obesity-related diseases, it is essential to have bioavailability measurements for carbohydrates that reflect the utilisation and biological functions of relevance to health. The specific biological function of dietary carbohydrates is closely linked to the gastrointestinal handling, which is determined mainly by the carbohydrate food properties (Fig. 1). The fact that the fate of dietary carbohydrates is also influenced by other meal components and subject biological variation does not alter or diminish the value in measuring the inherent properties of carbohydrate foods. It is not necessary for the GI measurement to predict the glycaemic response in all mixed-meal situations, as its purpose is to provide a ranking that will guide the consumer in selecting the slow-release carbohydrate foods with demonstrated longer-term benefits. However, the consumer would not be led towards a healthy diet if low GI values are achieved with a high content of fructose or fat, and consideration must therefore be given to overall nutritional composition.

The carbohydrate food properties of relevance to bioavailability and health are determined by the chemical identity and the food matrix, and both of these aspects must be taken into account. As it is difficult to measure directly the biological function associated with different carbohydrate food properties, measures of biological function are used to validate in vitro methods that then provide rapid and reproducible measures that relate to biological function. The classification and measurement scheme presented in Table 1 is based on these principles. Dietary carbohydrate fractions are ordered primarily into glycaemic and non-glycaemic carbohydrates, which can be subdivided to describe features of specific relevance to their biological functionality. The scheme incorporates information on carbohydrate bioavailability, including rate of release, whilst remaining faithful to the ideal of the measurement of chemically defined components, as proposed by the Food and Agriculture Organization of the United Nations/ World Health Organization Expert Consultation (1998).

The scheme can be applied in the development of healthier carbohydrate foods and in food labelling that will guide the consumer towards carbohydrate foods that are less energy-dense and more compatible with maintaining good health. This carbohydrate bioavailability approach is complementary to the dietary fibre and GI concepts in the promotion of diets containing largely unrefined plant foods that are rich in slow-release carbohydrates. The detailed values obtained by the presented scheme are valuable for epidemiological and mechanistic studies seeking to further establish the link between dietary carbohydrates and health.

References

Birkett A, Muir A, Phillips J, Jones G & O'Dea K (1996) Resistant starch lowers fecal concentrations of ammonia and phenols in humans. Am J Clin Nutr 63, 766–772.

Bjorck I & Liljeberg-Elmstahl H (2003) The glycaemic index: importance of dietary fibre and other food properties. Proc Nutr Soc 62, 201–206.

Brand-Miller JC, Holt SHA, Pawlak DB & McMillan J (2002) Glycemic index and obesity. Am J Clin Nutr 76, Suppl., 281S–285S.

Burke LM, Kiens B & Ivy JL (2004) Carbohydrates and fat for training and recovery. J Sports Sci 22, 15–30.

Burn J, Katheuser A, Fodde R, Coaker J, Chapman PD & Mathers JC (1996) Intestinal tumours in the Apc 1638N mouse: aspirin not

protective and resistant starch increases small bowel tumours. *Eur J Hum Genet* **4**, Suppl. 1, 13.

Champ M, Langkilde AM, Brouns F, Kettlitz B & Bail-Collet Y (2003) Advances in dietary fibre characterisation. 2. Consumption, chemistry, physiology and measurement of resistant starch; implications for health and food labelling. *Nutr Res Rev* **16**, 143–161.

Cherbut C (1995) Role of gastrointestinal motility in the delay of absorption by dietary fibre. *Eur J Clin Nutr* **49**, Suppl., S74–S80.

Collier G, McLean A & O'Dea K (1984) Effect of co-ingestion of fat on the metabolic responses to rapidly absorbed carbohydrates. *Diabetologia* **26**, 50–54.

Colonna P, LeLoup V & Buleon A (1992) Limiting factors of starch hydrolysis. *Eur J Clin Nutr* **46**, Suppl. 2, S17–S32.

Cummings JH (1993) The effect of dietary fiber on fecal weight and constipation. In *CRC Handbook of Dietary Fiber in Human Nutrition*, pp. 263–349 [GA Spiller, editor]. Boca Raton, FL: CRC Press.

Cummings JH, Macfarlane GT & Englyst HN (2001) Digestion of prebiotics and fermentation by intestinal microflora. *Am J Clin Nutr* **73**, 415–420.

Daly ME, Vale C, Walker M, Alberti KGMM & Mathers JC (1997) Dietary carbohydrates and insulin sensitivity: a review of the evidence and clinical implications. *Am J Clin Nutr* **66**, 1072–1085.

Daly ME, Vale C, Walker M, Littlefield A, Alberti KGMM & Mathers JC (1998) Acute effects on insulin sensitivity and diurnal metabolic profiles of high-sucrose compared with a high-starch diet. *Am J Clin Nutr* **67**, 1186–1196.

Daly ME, Vale C, Walker M, Littlefield A, Alberti KGMM & Mathers JC (2000) Acute fuel selection in response to high-sucrose and high-starch meals in healthy men. *Am J Clin Nutr* **71**, 1516–1524.

Englyst HN & Cummings JH (1985) Digestion of the polysaccharides of some cereal foods in the human small intestine. *Am J Clin Nutr* **42**, 778–787.

Englyst HN & Cummings JH (1986) Digestion of the carbohydrates of banana (*Musa paradisiaca sapientum*) in the human small intestine. *Am J Clin Nutr* **44**, 42–50.

Englyst HN & Cummings JH (1987) Digestion of the polysaccharides of potato in the small intestine of man. *Am J Clin Nutr* **45**, 423–431.

Englyst HN, Bingham SA, Runswick SA, Collinson E & Cummings JH (1988) Dietary fibre (non-starch polysaccharides) in fruit, vegetables and nuts. *J Hum Nutr Diet* **1**, 247–286.

Englyst HN, Bingham SA, Runswick SA, Collinson E & Cummings JH (1989) Dietary fibre (non-starch polysaccharides) in cereal products. *J Hum Nutr Diet* **2**, 253–271.

Englyst HN, Kingman SM & Cummings JH (1992) Classification and measurement of nutritionally important starch fractions. *Eur J Clin Nutr* **46**, Suppl., S33–S50.

Englyst HN, Quigley ME & Hudson GJ (1994) Determination of dietary fiber as non-starch polysaccharides with gas–liquid chromatographic, high-performance liquid chromatographic or spectrophotometric measurement of constituent sugars. *Analyst* **119**, 1497–1509.

Englyst HN, Quigley ME & Hudson GJ (2000) Dietary fibre analysis as non-starch polysaccharides. In *Encyclopaedia of Analytical Chemistry*, pp. 3912–3929 [RA Meyers, editor]. Chichester: John Wiley & Sons.

Englyst HN, Trowell H, Southgate DAT & Cummings JH (1987) Dietary fiber and resistant starch. *Am J Clin Nutr* **46**, 873–874.

Englyst HN, Veenstra J & Hudson GJ (1996) Measurement of rapidly available glucose (RAG) in plant foods: a potential *in vitro* predictor of the glycaemic response. *Br J Nutr* **75**, 327–337.

Englyst HN, Wiggins HS & Cummings JH (1982) Determination of the non-starch polysaccharides in plant foods by gas–liquid chromatography of constituent sugars as alditol acetates. *Analyst* **107**, 307–318.

Englyst KN & Englyst HN (2004) Detecting nutritional starch fractions. In *Starch in Food: Structure, Function and Applications*, pp. 541–559 [A-C Eliasson, editor]. Cambridge, UK: Woodhead Publishing.

Englyst KN, Englyst HN, Hudson GJ, Cole TJ & Cummings JH (1999) Rapidly available glucose in foods. An *in vitro* measurement that reflects the glycemic response. *Am J Clin Nutr* **69**, 448–454.

Englyst KN, Hudson GJ & Englyst HN (2000) Starch analysis in food. In *Encyclopaedia of Analytical Chemistry*, pp. 4246–4262 [RA Meyers, editor]. Chichester: John Wiley & Sons.

Englyst KN, Vinoy S, Englyst HN & Lang V (2003) Glycaemic index of cereal products explained by their content of rapidly and slowly available glucose. *Br J Nutr* **89**, 329–339.

Evans RC, Fear S, Ashby D, Hackett A, Williams E, Van Der Vliet M, Dunstan FD & Rhodes JM (2002) Diet and colorectal cancer: an investigation of the lectin/galactose hypothesis. *Gastroenterology* **122**, 1784–1792.

Flint A, Moller BK, Raben A, Pedersen D, Tetens I, Holst JJ & Astrup A (2004) The use of glycaemic index tables to predict glycaemic index of composite breakfast meals. *Br J Nutr* **91**, 979–989.

Food and Agriculture Organization of the United Nations/World Health Organization Expert Consultation (1998) *Carbohydrates in Human Nutrition. FAO Food and Nutrition Paper* no. 66. Geneva: FAO/WHO.

Food and Drug Administration (2000) Letter regarding supplement health claim for dietary fiber with respect to colon cancer. Document no. 91N-0098. http://www.cfsan.fda.gov/~dms/ds-ltr8.html

Frayn KN & Kingman SM (1995) Dietary sugars and lipid metabolism in humans. *Am J Clin Nutr* **62**, Suppl., 250S–261S.

Frost G, Leeds A, Dore J, Madeiros S, Brading S & Dornhorst A (1999) Glycaemic index as a determinant of serum HDL-cholesterol concentration. *Lancet* **353**, 1045–1048.

Gallant DJ, Bouchet B, Buleon A & Perez S (1992) Physical characteristics of starch granules and susceptibility to enzymatic degradation. *Eur J Clin Nutr* **46**, Suppl. 2, S3–S16.

Gannon MC, Khan MA & Nuttall FQ (2001) Glucose appearance rate after the ingestion of galactose. *Metabolism* **50**, 93–98.

Goodlad RA & Englyst HN (2001) Redefining dietary fibre: potentially a recipe for disaster. *Lancet* **358**, 1833–1834.

Gudmand-Hoyer E (1994) The clinical significance of disaccharide maldigestion. *Am J Clin Nutr* **59**, Suppl., 735S–741S.

Haber GB, Heaton KW, Murphy D & Burroughs LD (1977) Depletion and disruption of dietary fibre. *Lancet* **ii**, 679–682.

Heaton KW, Marcus SN, Emmett PM & Bolton CH (1988) Particle size of wheat, maize, and oat test meals: effects on plasma glucose and insulin responses and on the rate of starch digestion *in vitro*. *Am J Clin Nutr* **47**, 675–682.

Jenkins DJA, Kendall CWC, Augustin LSA, Franceschi S, Hamidi M, Marchie A, Jenkins DJA, Wolever TMS & Taylor RH (1981) Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* **34**, 362–366.

Jenkins DJA, Kendall CWC, Augustin LSA, Franceschi S, Hamidi M, Marchie A, Jenkins AL & Axelsen M (2002) Glycemic index: overview of implications in health and disease. *Am J Clin Nutr* **76**, Suppl., 266S–273S.

Jeppesen J, Schaaf P, Jones C, Zhou MY, Chen YDI & Reaven GM (1997) Effects of low-fat, high-carbohydrate diets on risk factors for ischemic heart disease in postmenopausal women. *Am J Clin Nutr* **65**, 1027–1033.

Liu S, Manson JE, Stampfer MJ, Hu FB, Holmes MD, Franz M, Sampson L, Hankinson SE & Willett WC (2001) Dietary glycemic load assessed by food frequency questionnaire in relation to plasma HDL-cholesterol and fasting triglycerides in postmenopausal women. *Am J Clin Nutr* **71**, 1455–1461.

Liu S, Stampfer MJ & Manson JE (1998) A prospective study of glycemic load and risk of myocardial infarction in women. *FASEB J* **12**, A260.

Livesey G (2001) Tolerance of low digestible carbohydrates: a general review. *Br J Nutr* **85**, Suppl., S7–S16.

Ludwig DS (2000) Dietary glycemic index and obesity. *J Nutr* **130**, Suppl., 280S–283S.

Mayes PA (1993) Intermediary metabolism of fructose. *Am J Clin Nutr* **58**, Suppl., 754S–765S.

Meyers KA, Kushi LH, Jacobs DR, Slavin J, Sellers TA & Folsom AR (2000) Carbohydrates, dietary fiber and incidence of type 2 diabetes in older women. *Am J Clin Nutr* **71**, 921–930.

Nuttall FQ, Khan MA & Gannon MC (2000) Peripheral glucose appearance rate following fructose ingestion in normal subjects. *Metabolism* **49**, 1565–1571.

Opperman AM, Venter CS, Oosthuizen W, Thompson RL & Vorster HH (2004) Meta-analysis of the health effects of using the glycaemic index in meal planning. *Br J Nutr* **92**, 367–381.

Parks EJ & Hellerstein MK (2000) Carbohydrate-induced hypertriacylglycerolemia: historical perspective and review of biological mechanisms. *Am J Clin Nutr* **71**, 412–433.

Rayner CK, Jones KL, Samson M & Horowitz M (2001) Relationships of upper gastrointestinal motor and sensory function with glycemic control. *Diabetes Care* **24**, 371–381.

Robertson MD, Livesey G & Mathers JC (2002) Quantitative kinetics of glucose appearance and disposal following a ^{13}C-labelled starch-rich meal: comparison of male and female subjects. *Br J Nutr* **87**, 569–577.

Salmeron J, Manson J, Stampfer M, Colditz G, Wing A & Willett W (1997) Dietary fiber, glycemic load, and risk of non insulin dependent diabetes mellitus in women. *JAMA* **277**, 472–477.

Seal CJ, Daly ME, Thomas LC, Bal W, Birkett AM, Jeffcoat R & Mathers JC (2003) Postprandial carbohydrate metabolism in healthy subjects and those with type 2 diabetes fed starches with slow and rapid hydrolysis rates determined *in vitro*. *Br J Nutr* **90**, 853–864.

Silvester KR, Englyst HN & Cummings JH (1995) Ileal recovery of starch from whole diets containing resistant starch measured *in vitro* and fermentation of ileal effluent. *Am J Clin Nutr* **62**, 403–411.

Spieth LE, Harnish JD, Lenders CM, Raezer LB, Pereira MA, Hangen J & Ludwig DS (2000) A low glycemic index diet in the treatment of pediatric obesity. *Arch Pediatr Adolesc Med* **154**, 947–951.

Steer T, Carpenter H, Tuohy K & Gibson GR (2000) Perspectives on the role of the human gut microbiota and its modulation by pro- and prebiotics. *Nutr Res Rev* **13**, 229–254.

Thomsen C, Rasmussen OW, Chriastiansen C, Andreasen F, Poulsen PL & Hermansen K (1994) The glycaemic index of spaghetti and gastric emptying in non-insulin-dependent diabetic patients. *Eur J Clin Nutr* **48**, 776–780.

Trowell H (1972) Crude fibre, dietary fibre and atherosclerosis. *Atherosclerosis* **16**, 138–140.

Trowell H (1985) Dietary fibre; a paradigm. In *Dietary Fibre, Fibre-Depleted Foods and Disease*, pp. 1–20 [HC Trowell, D Burkitt and KW Heaton, editors]. London: Academic Press.

Truswell AS (1994) Food carbohydrates and plasma lipids – an update. *Am J Clin Nutr* **59**, Suppl., S710–S718.

UK Parliament Health Committee (2004) *Health – Third Report, Session 2003–04*. London: The Stationery Office, http://www.publications.parliament.uk/pa/cm200304/cmselect/cmhealth/23/2302.htm

Van Dam RM, Visscher AWJ, Feskens EJM, Verhoef P & Kromhout D (2000) Dietary glycemic index in relation to metabolic risk factors and incidence of coronary heart disease: the Zutphen Elderly Study. *Eur J Clin Nutr* **54**, 726–731.

Van Loo J, Cummings J, Delzenne N, *et al.* (1999) Functional food properties of non-digestible oligosaccharides: a consensus report from the ENDO project (DGXII AIRII-CT94-1095). *Br J Nutr* **81**, 121–132.

Van Munster IP, Tangerman A & Nagengast FM (1994) Effect of resistant starch on colonic fermentation, bile-acid metabolism, and mucosal proliferation. *Dig Dis Sci* **39**, 834–842.

Wasan HS & Goodlad RA (1996) Fibre-supplemented foods may damage your health. *Lancet* **348**, 319–320.

Westphal SA, Gannon MC & Nuttall FQ (1990) Metabolic response to glucose ingested with various amounts of protein. *Am J Clin Nutr* **52**, 267–272.

Wolever TMS (2003) Carbohydrate and the regulation of blood glucose and metabolism. *Nutr Rev* **61**, Suppl., S40–S48.

World Health Organization (2003) *Diet, Nutrition and the Prevention of Chronic Disease*. Technical Report Series no. 916. Geneva: WHO, http://www.who.int/hpr/NPH/docs/who_fao_expert_report.pdf

Young GP, McIntyre A, Albert V, Folino M, Muir JG & Gibson PR (1996) Wheat bran suppresses potato starch-potentiated colorectal tumorigenesis at the aberrant crypt stage in a rat model. *Gastroenterology* **110**, 508–514.