REVIEW

Carbohydrates and dietary fibre

J. Lunn and J. L. Buttriss
British Nutrition Foundation, London, UK

Summary

The health benefits of including sufficient dietary fibre in the diet have been well described and have formed the basis of dietary recommendations around the world. However, dietary fibre is a complex dietary entity, consisting of many non-digestible components of food. Debate surrounding the definition and measurement of dietary fibre has resulted in inconsistencies in labelling, description and recommendations set across the world. In the UK, dietary recommendations are made using the fraction of non-digestible material described as non-starch polysaccharide that is measured by the Englyst method. However, the Association of Official Analytical Chemists (AOAC) methods, used widely by the food industry, capture a much greater range of non-digestible material, that some suggest should be included in any definition of dietary fibre. An attempt to resolve such discrepancies, possibly by taking an approach that considers the health effects of fractions not captured in the Englyst method, is probably overdue.

Additionally, it is clear that the effects of these various non-digestible components of dietary fibre are not interchangeable, and it is important that fibre comes from a range of sources to ensure maximum health benefits from the fibre in the diet. Traditional 'insoluble' fibres are required to add bulk as well as rapidly fermentable, viscous fibres to bring about cholesterol lowering. There is also a convincing argument for including slowly fermented components, such as resistant starches, that are well tolerated in the digestive system and can bring about improvements in gut function. Currently there is insufficient data from well designed human intervention trials to make specific recommendations on the amounts of these fibre components in the diet, but it may be useful for health professionals to talk in terms of the different food sources of these types of fibre, as well as total fibre amounts.

Keywords: AOAC methods, carbohydrate, dietary fibre, dietary recommendation, dietary fibre

Correspondence: Dr Joanne Lunn, Nutrition Scientist, British Nutrition Foundation, High Holborn House, 52–54 High Holborn, London WC1V 6RQ, UK.
E-mail: j.lunn@nutrition.org.uk

Introduction

Foods containing carbohydrates and dietary fibre make up a major component of a healthy, balanced diet. The categorisation of compounds as carbohydrates is straightforward, as they are defined chemically, dependent on the presence of carbon, hydrogen and oxygen molecules in the correct ratios. However, there is much dispute over the components of dietary fibre, and there is not currently a single, worldwide definition for dietary fibre. As a result of this, there are some components which appear to have the health benefits traditionally ascribed to fibre, that are not included in analyses of fibre content. This report takes an in-depth analysis of the issues surrounding dietary fibre intake in the UK, and proposes some practical examples of how higher fibre intakes can be achieved in the context of a varied, balanced diet. After first looking at the controversies regarding the definition of dietary fibre, the report then considers how dietary recommendations are set in the UK and throughout the world, to make an assessment of whether the current recommendation is appropriate.

Description of carbohydrate

Chemically, carbohydrates are relatively stable organic molecules containing carbon, hydrogen and oxygen [Empirical formula $C_x(H_2O)_y$]. Carbohydrates are derived almost exclusively from food of plant origin, where they are synthesised from carbon dioxide and water using energy harnessed from sunlight. In the human diet, carbohydrates fall into three main groups: sugars, starch and non-starch polysaccharides, and are the major source of energy, providing 50–70% of energy intake.

The simplest form of carbohydrate is a monosaccharide sugar, such as glucose, fructose or galactose. Also included in the sugar classification are disaccharides and sugar alcohols. Disaccharides, such as sucrose or lactose, also occur naturally in foods and comprise two monosaccharide units (glucose + fructose = sucrose; glucose + galactose = lactose). Sucrose is by far the most common sugar in the human diet, accounting for approximately 14% of total energy intakes. The sugar alcohols are naturally occurring, but are also synthesised commercially for use as sweeteners (*e.g.* sorbitol or xylitol). These sugars are also not absorbed in the small intestine.

Oligosaccharides consist of 3–15 monosaccharide units and are not digested by enzymes in the human digestive tract, although they are broken down by bacterial enzymes in the large bowel. Such compounds, including raffinose, stachyose and verbascose, are found in plant seeds and are considered to be 'unavailable carbohydrate'. A group of physiologically relevant oligosaccharides are the inulins. Inulin is a type of carbohydrate known as a fructan, which is a chain of fructose molecules (fructo-oligosaccharide). Inulin is used by some plants as a means of storing energy and is typically found in roots or rhizomes. Most plants which synthesise and store inulin do not store energy in other carbohydrate forms.

The larger carbohydrate polymers are referred to as polysaccharides or, to use a popular lay term, 'complex' carbohydrates. Nutritionally, there are two classes: starches and non-starch polysaccharides. Starches are polymers of glucose, which are either straight chain (amylose) or branched (amylopectin). The non-starch polysaccharides are a varied group of compounds which are not digested by human digestive enzymes. The term complex carbohydrate was coined for use in a US Senate Select Committee report in 1977 and was used without a formal definition. In the context of the report, it was used to distinguish simple sugars from polysaccharides, and it is this definition that has been adopted for general use. There has been some debate about the validity of this term, as it is classifying a group of carbohydrates with very different physiological properties under one umbrella term. Indeed, some suggest that, as a fundamental difference between carbohydrates is their digestibility, it is this feature that should differentiate them, rather than their size. These so-called 'complex' carbohydrates can actually be as rapidly digested as simple sugars, and thus it could be considered misleading to use the term, as consumers might perceive complex carbohydrates to be more slowly broken down in the gastrointestinal tract. The Food and Agriculture Organisation/World Health Organization (FAO/WHO) Expert Consultation of Carbohydrates in Human Nutrition (1997) deemed the term 'complex' to have little merit and advised against its use.

Digestion and absorption

Carbohydrates are broken down throughout the gut. Salivary amylase is an enzyme in the mouth that initiates the digestion of carbohydrates in the form of starches by catalysing the hydrolysis of polysaccharides into disaccharides. Breakdown occurs to a small extent in the mouth and continues down the length of the oesophagus. Despite the low pH of the stomach contents, which inactivates salivary amylase, some

hydrolysis can also occur in the stomach in the centre of the food mass, where the stomach acid is unable to penetrate completely. However, the main site for carbohydrate digestion is the small intestine. Once in the duodenum, the alkaline secretions from the pancreas neutralise the acidic stomach contents and allow further digestion to occur. Amylase from the pancreas completes the breakdown of starch polysaccharides into disaccharides that are then hydrolysed into glucose, fructose and galactose by disaccharidases located in the small intestine's brush border. These monosaccharide units are then absorbed into the intestinal cell and eventually enter the bloodstream either by passive diffusion (fructose) or through a sodium and energy-dependent active transport mechanism (glucose and galactose).

Not all the carbohydrate in the diet is digested and absorbed in the small intestine, and a significant portion arrives in the large bowel, where it is fermented by bacteria naturally present.

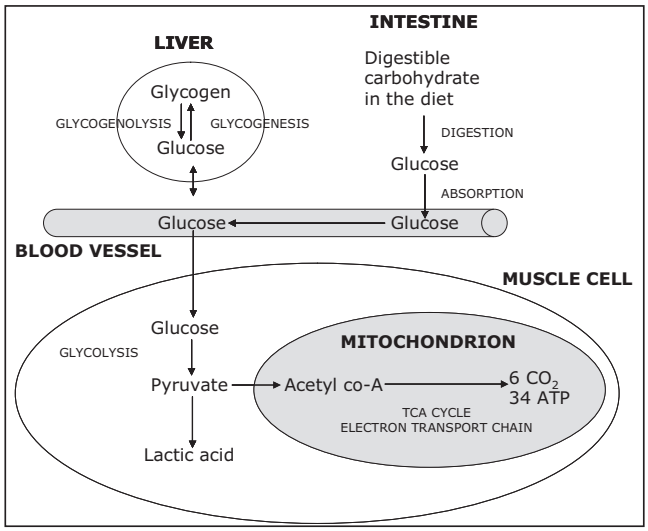

Figure 1 Schematic detailing the metabolism of glucose. ATP, adenosine triphosphate; co-A, co-enzyme A; TCA, tricarboxylic acid.

Metabolism of glucose

The majority of the carbohydrate in the diet enters the bloodstream as glucose, which is then transported around the body to the tissues (Fig. 1). Here it is either: used for energy, stored as glycogen in the liver and the muscles, or converted into fat. The fate of the glucose circulating in the bloodstream is determined by the relative concentrations of the hormone insulin. Insulin is released from the β-cells of the pancreas in response to glucose absorption, and triggers glucose uptake into the muscle and liver cells, where it is either utilised for energy or stored. Insulin also suppresses metabolic pathways in the liver which synthesise glucose from amino acids, lactic acid or glycerol. Thus, provided the body is sensitive to the actions of insulin, blood glucose concentrations will fall. Concurrently, insulin levels decrease, preventing glucose uptake into the muscle, liver and adipose tissue, and leaving a readily available source of fuel for the brain.

The metabolism of glucose yields energy in the form of adenosine triphosphate. Glucose is first broken down to pyruvate in the cell cytosol by an anaerobic process called glycolysis. Pyruvate then has one of two fates: it is either converted to lactic acid under anaerobic conditions to yield a small amount of energy, or transported into the mitochondrion where it is further broken down to carbon dioxide and water. This latter process requires the presence of oxygen (aerobic respiration). Human digestion and metabolism do not distinguish between sugars found naturally in foods and those added to foods. On average, 1 g of carbohydrate provides 17 kJ (4 kcal), although the different forms of carbohydrate have slightly different metabolic yields: monosaccharides = 15.7 kJ (3.74 kcal)/g, disaccharides = 16.6 kJ (3.95 kcal)/g, and starch = 17.6 kJ (4.18 kcal)/g.

Factors affecting carbohydrate digestion and absorption

Many factors influence the rate of carbohydrate digestion and absorption. This is of great physiological relevance, because it determines the overall effect that the carbohydrate has on blood glucose and insulin concentrations, and so ultimately the impact that the food has on the body. Carbohydrates in the form of rapidly digestible starches are those that have open, branched structures, such as amylopectin. The amylase has greater access to the carbon–carbon bonds it is targeting, and is able to break down the polymer faster than the tightly packed, unbranched linear amylose polymer. Yet, regardless of structure, there are other factors that influence carbohydrate digestion (Fig. 2). In this context, carbohydrates are categorised by the effect that they have on blood glucose concentrations (increase or no effect) although, regardless of the overall effect, they are still utilised by the body to some extent. Those carbohydrates that are not digested and absorbed (resistant starch, non-starch

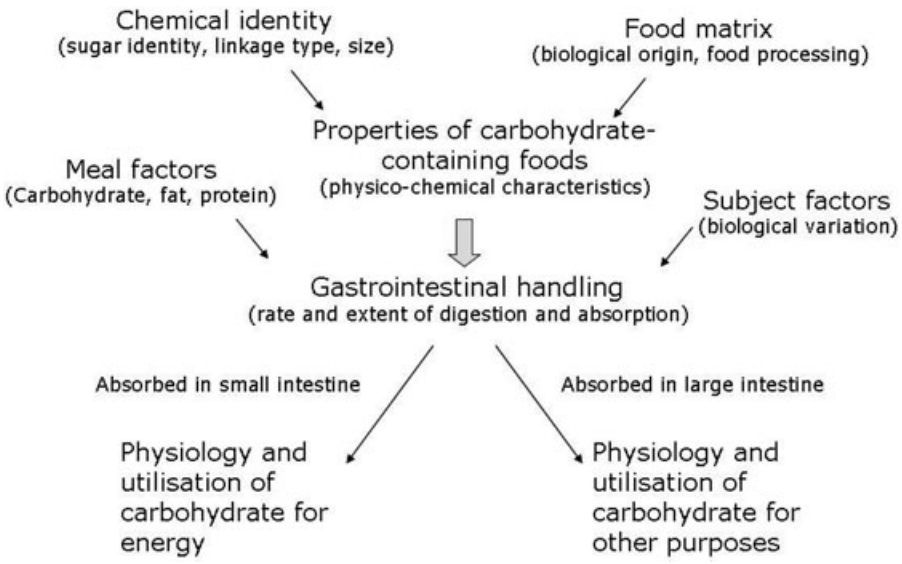

Figure 2 Factors influencing the digestibility of carbohydrate and dietary fibre in the gastrointestinal tract. Adapted from Englyst and Englyst (2005).

polysaccharide, oligosaccharides and sugar alcohols) are likely to be fermented in the large bowel. For a thorough review on the factors that affect carbohydrate bioavailability, see a recent paper by Englyst and Englyst (2005), which takes both a physiological and chemical approach to explain the variability in the way that different dietary carbohydrates are handled in the body.

Resistant starch

Starch is one of the main forms of carbohydrate in the diet. As described above, digestible starches are broken down by digestive enzymes in the small intestine into glucose molecules. The glucose is then absorbed into the blood and used to provide energy for the body. However, some starch in the diet is resistant to digestion and passes into the large intestine (colon). This may have a number of health benefits, which will be discussed in more detail. There are several reasons why resistant starch is not digested:

- The starch may be physically inaccessible to the digestive enzymes such as in grains, seeds or tubers.
- The starch granules themselves are structured in a way which prevents the digestive enzymes from breaking them down (*e.g.* raw potatoes, unripe bananas and high amylose maize starch) (Nugent 2005).
- When starches are heated, they gelatinise and become more easily digested. However, if these starch gels are then cooled, starch crystals that are resistant to enzyme digestion form in the food. This form of 'retrograded' starch is found in small quantities (approximately 5%) in foods such as cornflakes or cooked and cooled potatoes, as in a potato salad.
- Selected starches that have been chemically treated (etherisation, esterisation, cross-bonding) cannot be broken down by digestive enzymes.

Resistant starch has been classified into four general subtypes called RS1–RS4 (Englyst *et al.* 1992; Brown *et al.* 1995). Table 1 outlines a summary of the different types of resistant starch, their classification criteria and food sources. Resistant starch is found in a wide range of foods, including intact wholegrains, legumes, pasta, unripe bananas, raw potatoes, cooked and cooled potatoes, and foods containing commercial sources of modified starches (*e.g.* bread, breakfast cereals and nutrition bars). Modified resistant starch (RS4) is a novel food not yet approved by the European Union (EU).

Glycaemic response to a meal

Leaving aside the non-starch polysaccharides, resistant starches and oligosaccharides, which are not digested by enzymes in the human digestive tract, other carbohydrates do differ in the extent to which they bring about

Table 1 Classification of types of resistant starch, food sources and factors affecting their resistance to digestion in the colon

Type of resistant starch	Description	Food sources	Resistance reduced by
RS1	Physically protected	Whole- or partly milled grains & seeds, legumes, pasta	Milling, chewing
RS2	Ungelatinised resistant granules with B-type crystallinity	Raw potatoes, green bananas, some legumes, high-amyyose starches	Food processing & cooking
RS3	Retrograded starch (*i.e.* non-granular starch-derived materials)	Cooked & cooled potatoes, bread, cornflakes	Processing conditions
RS4	Selected chemically modified starches owing to cross-bonding with chemical reagents, ethers, esters, etc.	This type of modified resistant starch is a novel food, not yet approved by the EU	Less susceptible to digestibility *in vitro*

Source: Nugent (2005).
EU, European Union.

changes in the concentration of glucose circulating in the blood. As outlined above, when carbohydrates are broken down, glucose is absorbed into the bloodstream, resulting in an increased concentration in the blood. In response to this, insulin is released from the pancreas, sending a signal to the body tissues to increase their uptake of glucose, thus resulting in a fall in blood glucose concentration. A number of factors influence the rate and duration of the glycaemic response. These include: the type of sugar that forms the carbohydrate; the nature and the form of the starch, as some are more digestible than others (see above); the cooking and processing methods used; and the other nutrients in the food, such as fat or protein (see Alfenas & Mattes 2005).

The glycaemic index (GI) is a way of classifying carbohydrates in foods according to their effect on the rate at which glucose enters the bloodstream. It is determined by comparing the blood glucose response to 50 g available carbohydrate from a test food, with that of a reference food (either glucose or white bread, given a value of 100). A food that has a high GI (>70) causes a rapid, sharp rise in blood glucose concentrations, whereas a low-GI food (<55) brings about a slower and more sustained release of glucose into the blood. It is thought that diets rich in low-GI carbohydrates are associated with reduced risk of chronic diseases although, despite popular belief, there is little evidence that a low-GI diet is directly associated with weight reduction.

The glycaemic response to a meal is influenced by other factors, such as the amount of carbohydrate, the nature of the meal, previous meals and the recent physical activity of the subject. Additionally, the acute (physical activity and second-meal effects) or intermediate factors (bodyweight change) that influence insulin sensitivity will affect the absolute physiological responses to diets with different GI values; hence there is considerable individual variation.

Table 2 Glycaemic index (GI) and glycaemic load of a selection of carbohydrates in the UK diet

	GI	Serving size (g)	Glycaemic load
High GI (GI > 70)			
White baguette	95	30	15
Cornflakes	81	30	21
Baked potato	85	150	26
Medium GI (GI 56–69)			
Crisps, salted	57	24	26
Crumpets	69	20	13.4
Pizza, cheese	60	100	20.8
Low GI (GI < 55)			
Pineapple juice	46	250	16
Bran flakes	42	30	4
Sausages	28	100	1.5

Source: Theobald (2004).
GI compared with glucose = 100.

A more sensitive measure of the overall effect of dietary carbohydrate is the glycaemic load (GL). This considers the impact that a diet has on blood glucose concentrations, as it takes into account the total amount of carbohydrate in the food, as well as its GI. GL is calculated by multiplying the GI of a food by the percentage of carbohydrate in an average serving (Table 2). The presence of indigestible oligosaccharides or resistant starches in foods will therefore lower the overall GL, as these compounds do not affect blood glucose concentrations.

Carbohydrate quality

It is now appreciated that carbohydrates differ in ways other than their chemical formula or chain length. Indeed, a better understanding of the rate and extent of carbohydrate digestion and subsequent glucose absorption has led to the development of the term carbohydrate quality. In general, higher-quality carbohydrates are slowly digested and have physiological effects above simply providing the body with energy (Jenkins *et al*. 2002). Some indigestible carbohydrates, such as resistant starch, have been shown to behave more like compounds traditionally referred to as dietary fibre, and have been linked with improvements in glycaemic control, bowel health and cardiovascular disease risk factors (see Nugent 2005). This is not to say that carbohydrates considered to be of lower quality do not still have an important place in the diet. These carbohydrates tend to be more simple sugars (*e.g.* fructose) or refined carbohydrates, and so are more rapidly digested in the digestive system. In times when an immediate burst of energy is required, such as during an endurance sporting event, rapidly digestible carbohydrate is the best choice to make and thus is why glucose tablets and sports drinks are so popular.

Wholegrains

An extension to the carbohydrate quality concept is the growing interest in wholegrains. The commonly consumed cereals in the UK are wheat, corn, oats, barley, rye and rice. These grains can be eaten as both the intact grain or in more refined forms. The wholegrain includes all parts of the grain kernel: the fibre-rich bran, the nutrient-rich aleurone layer, the endosperm, and the nutrient-packed germ (Fig. 3). Wholegrains consumed in the diet have often undergone some degree of processing. However, the term wholegrain encompasses flours produced by different production processes. Flours which have been produced by traditional milling processes based on stone grinding, where the grain is crushed without the components being separated, are wholegrain flours. Yet so are flours that have been produced by roller milling practices, where the individual components are ground and separated by sieving but are then reconstituted to reform the wholegrain. (The differences in the processing method selected do determine the digestion rate of the grain in the gastrointestinal tract; for instance, grains in muesli are digested at a slower rate than the milled wholegrains present in extruded breakfast cereals.) The American Association of Cereal Chemists (AACC) produced a definition of wholegrain to assist food manufacturers and consumers, which was approved and adopted in 1999: 'Whole grains shall consist of the intact, ground, cracked or flaked caryopsis, whose principal anatomical components – the starchy endosperm, germ and bran – are present in the same relative proportions as they exist in the intact caryopsis' (AACC 2005). This definition encompasses cereal grains (*e.g.* barley, buckwheat, bulgar, corn, millet, rice, rye, oats, sorghum and wheat) (FDA 2006). Products derived from legumes, oilseeds and roots are not considered to be wholegrains.

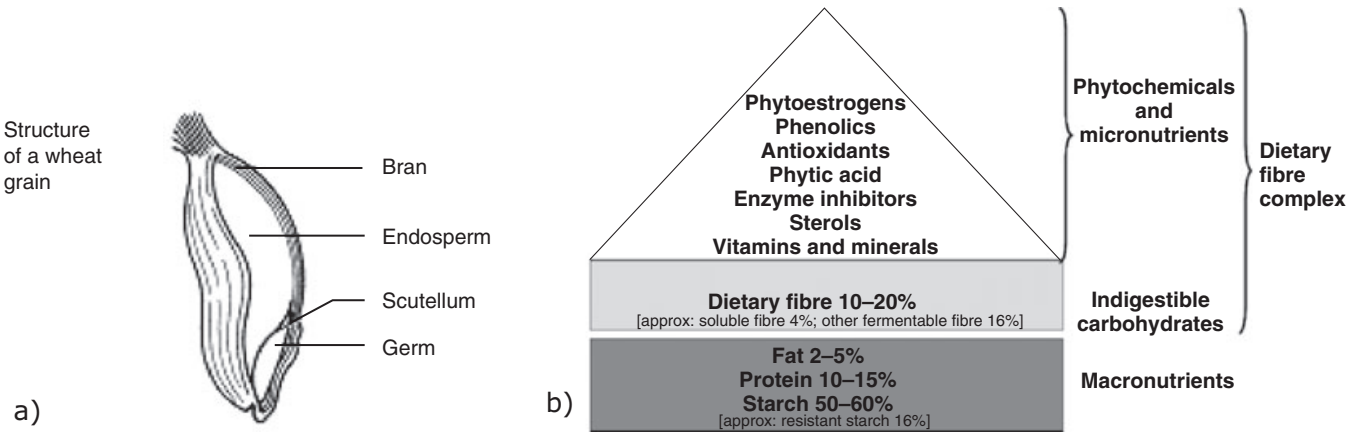

Figure 3 (a) Diagrammatic representation and (b) nutrient composition of a typical wheat grain (estimations of fibre components from Bednar *et al*. 2001).

Evidence is mounting, especially in the USA, where oats are the major wholegrain, that there are considerable health benefits to be gained from consuming cereals in the form of the wholegrain. For instance, consuming the intact grain has been associated with a reduced risk of weight gain compared with refined carbohydrates (Schulze *et al*. 2006). Other health effects of regular consumption of wholegrain include reduced total mortality, reduced cancer mortality at certain sites, and a reduced risk of coronary heart disease (CHD), ischaemic stroke and type 2 diabetes (Smith *et al*. 2003; Seal 2006; Seal *et al*. 2006). Wholegrains contain a number of components that may contribute to a reduced risk of heart disease, such as vitamin E and dietary fibre. They also contain resistant starch and oligosaccharides that are fermented by intestinal bacteria to short-chain fatty acids (SCFAs) that may help reduce blood cholesterol, as well as plant sterols that may also have cholesterol-lowering effects (see Seal *et al*. 2006). However, the resistant starch content of the wholegrain is reduced during milling and processing. A fuller examination of these health effects will be presented in this review. However, at this point it would be prudent to note that people who regularly consume wholegrain foods also tend to live healthier lifestyles, consuming more fruits and vegetables, and being more active.

Description of dietary fibre

The term dietary fibre was first used by Hipsley in 1953 to describe the non-digestible components of plants that make up the plant cell wall (*i.e.* cellulose, hemicellulose and lignin). At this time, it was purely a physiological-botanical description, and it was not until the 1970s that researchers started using the term in conjunction with health-related hypotheses (Burkitt *et al*. 1972; Trowell 1972). By the late 1970s, the definition of dietary fibre had been expanded to include other non-digestible polysaccharides, such as gums and mucilages (Trowell *et al*. 1976). Over time, further refinements of the definition have been published, gradually expanding the nature of the substances included and eventually including health effects (see DeVries 2004).

There are many 'official' definitions for fibre that are used by various authoritative bodies around the world. These include:

I. American Association of Cereal Chemists (AACC 2001)

> Dietary fibre is the remnants of the edible part of plants or analogous carbohydrates that are resistant to digestion and absorption in the human small intestine, with complete or partial fermentation in the large intestine. Dietary fibre includes polysaccharides, oligosaccharides, lignin and associated plant substances. Dietary fibres promote beneficial physiological effects, including laxation, and/or blood cholesterol attenuation, and/or blood glucose attenuation.

II. Food and Nutrition Board (FNB 2001)

> * *Dietary Fiber* consists of non-digestible carbohydrates and lignin that are intrinsic and intact in plants.
> * *Functional Fiber* consists of isolated, non-digestible carbohydrates that have beneficial physiological effects in humans.
> * *Total Fiber* is the sum of *Dietary Fiber* and *Functional Fiber*.

III. Food Standards Australia New Zealand (FSANZ 2001)

> Dietary fibre means that fraction of the edible parts of plants or their extracts, or synthetic analogues, that are resistant to the digestion and absorption in the small intestine, usually with complete or partial fermentation in the large intestine. Dietary fibre includes polysaccharides, oligosaccharides (degree of polymerisation >2) and lignins, and promotes one or more of the following beneficial physiological effects:
>
> (i) laxation
> (ii) reduction in blood cholesterol
> (iii) modulation of blood glucose.

IV. Codex Alimentarius Commission, FAO/WHO (2006) (proposed)

Definition:

Dietary fibre means carbohydrate polymers[1] with a DP not lower than 3, which are neither digested nor absorbed in the small intestine. A DP not lower than 3 is intended to exclude mono- and disaccharides. It is not intended to reflect the average DP of a mixture. Dietary fibre consists of one or more of:

- Edible carbohydrate polymers naturally occurring in the food as consumed,
- Carbohydrate polymers, which have been obtained from food raw material by physical, enzymatic or chemical means,
- Synthetic carbohydrate polymers.

Properties:

Dietary fibre generally has properties such as:

- Decreases intestinal transit time and increases stools bulk,
- Fermentable by colonic microflora,
- Reduces blood total and/or LDL cholesterol levels,
- Reduces postprandial blood glucose and/or insulin levels.

With the exception of non-digestible edible carbohydrate polymers naturally occurring in foods as consumed where a declaration or claim is made with respect to dietary fibre, a physiological effect should be scientifically demonstrated by clinical studies and other studies as appropriate. The establishment of criteria to quantify physiological effects is left to national authorities.

[1]When derived from a plant origin, dietary fibre may include fractions of lignin and/or other compounds when associated with polysaccharides in the plant cell walls and if these compounds are quantified by the AOAC gravimetric analytical method for dietary fibre analysis: Fractions of lignin and the other compounds (proteic fractions, phenolic compounds, waxes, saponins, phytates, cutin, phytosterols, etc.) intimately 'associated' with plant polysaccharides are often extracted with the polysaccharides in the AOAC 991.43 method. These substances are included in the definition of fibre insofar as they are actually associated with the poly- or oligosaccharidic fraction of fibre. However, when extracted or even re-introduced into a food containing non-digestible polysaccharides, they cannot be defined as dietary fibre. When combined with polysaccharides, these associated substances may provide additional beneficial effects.

AOAC, Association of Official Analytical Chemists; DP, degree of polymerisation; LDL, low density lipoprotein.

These definitions differ in their complexity, some choosing to define carbohydrate in a purely physiological way, with others also including chemical criteria. However, they all attempt to encompass the diversity of indigestible carbohydrates in the human food supply: plant cell wall and storage carbohydrates, carbohydrates contributed by animal foods, and those isolated and low-molecular-weight carbohydrates that either occur naturally or are synthesised (sometimes termed analogous carbohydrates).

It is essential that legislators and the food industry are provided with an accurate definition of dietary fibre to work with. Without such a definition, confusion arises as new compounds can be designed or isolated that have the chemical properties attributed to dietary fibre, but that lack the health benefits, or indeed vice versa. Up until now, definitions have been developed that can be used for a specific purpose. For instance, when the US and Canadian Dietary Reference Intakes were set, three new definitions of dietary fibre were developed to enable the National Academy of Science to formally set recommended intakes. At the time, there were discrepancies between the definitions used by the USA and Canada (the Canadian definition excluded non-digestible carbohydrate of animal origin). However, the new definition could also establish uniformity for nutrition labelling and set standards as new fibre sources are developed, if it were to be formally adopted in North America. As nutrition labelling becomes increasingly uniform around the world, what is now required is a more joined-up approach to set definitions that can be used internationally. Indeed, in

the EU's recent discussions surrounding the implementation of legislation to regulate nutrition and health claims, scientists have turned to the Codex/FAO-proposed definition as the basis for any claim relating to fibre content. The Regulations are expected to be introduced early in 2007, and there will be a need for a common fibre definition and methodology to be adopted across Europe and, along with this, it is likely that consideration will have to be given to redefining the dietary reference value (DRV) for fibre used in the UK.

Defining dietary fibre – first principles

In the context of this report, it is prudent to return to first principles when considering whether any or all of the four current definitions encompass all that is required of a definition. Dietary fibre is not an entity, but a collective term for a complex mixture of substances with different chemical and physical properties, which exert different types of physiological effects. As mentioned above, dietary fibre was first defined as non-digestible components of plants that make up the plant cell wall: cellulose, hemicellulose (both non-starch polysaccharides) and lignin. Thus, it was recognised that a proportion of the polysaccharide carbohydrate in the diet is indigestible and therefore contributes to the dietary fibre content of the diet. Such carbohydrates include the glucans in oats and barley, and pentoses in rye, and these can be distinguished by their solubility in water. 'Insoluble' fibre is said to constitute the tough, fibrous parts of the plants. Foods rich in insoluble fibre include wheat and rye, and a small amount is present in fruits and vegetables. 'Soluble' fibre is found mostly in vegetables, especially pulses and legumes, and is also in many fruits and some grains, such as oats and barley.

To some extent, the solubility of dietary fibre determines its physiological properties, so this is a useful way of differentiating the components of dietary fibre in the diet. Insoluble fibre has passive water-attracting properties that help increase bulk, soften stools and shorten transit time through the intestinal tract. Most insoluble fibres are also resistant to fermentation in the large bowel. Soluble indicates a fibre source that readily holds water, forming a viscous solution as it passes through the gastrointestinal tract, and is fermented in the large bowel (Stephen & Cummings 1980).

Yet some believe that these definitions are not accurate enough. First, there are many components, such as the resistant starches and the oligosaccharides, which, by their indigestible nature, could be considered to contribute to the total amount of dietary fibre in the diet. If such components were included in a definition of dietary fibre, they do not fit into this neat soluble/insoluble categorisation. Resistant starches and oligosaccharides have physiological characteristics that are more akin to other similar components than to 'soluble' or 'insoluble' components. As a result, were these compounds included, they would need to be considered separately. Second, to differentiate by solubility in water ignores the 'metabolism' that occurs in the large bowel. Here, fermentation of different types of fibre produces end-products with significant health effects, although effects are not predicted by solubility. It is now clear that some insoluble fibres are in fact fermented in the large bowel whereas some soluble fibres have no clear health effect. Indeed, the WHO expert consultation on carbohydrate nutrition recommended that soluble and insoluble are not useful terms to distinguish dietary fibre (WHO/FAO 1997). If these terms are used, it might be useful to include additional categories such as those suggested in Figure 4.

However, even when the resistant starches and oligosaccharides are considered separately, it is clear

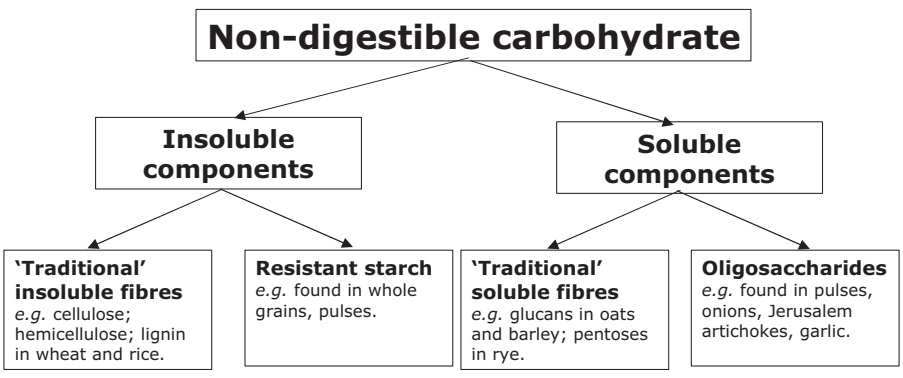

Figure 4 Possible method of classifying 'dietary fibre'.

that other compounds contribute to the dietary fibre content of foods when certain analytical methods are used. These include proteic fractions, phenolic compounds, waxes, saponins, phytates, cutin and phytosterols that exist within the plant cell structure and that are isolated along with the carbohydrate components of dietary fibre. These compounds are indigestible and contribute to the material that passes through the digestive tract, and are likely to have physiological benefits. Thus, it is essential that any working definition also takes into account the presence of these compounds. So an alternative way to classify fibre is as a group of compounds with different physiological characteristics, rather than to be constrained by defining it chemically. Yet, some differentiation has to be made between these indigestible plant components and other partially digested material, such as protein, that appears in the large bowel.

Diets naturally high in dietary fibre can be considered to bring about five main physiological consequences:

- improvements in gastrointestinal health;
- improvements in glucose tolerance and the insulin response;
- reduction of hyperlipidaemia, hypertension and other CHD risk factors;
- reduction in the risk of developing some cancers;
- increased satiety and hence some degree of weight management.

Therefore, it is not appropriate to state that 'dietary fibre' has a single all-encompassing physiological property, as these effects are dependent on the type of dietary fibre in the diet. The beneficial effects of high-fibre diets are the summation of the effects of the different types of fibre present in the diet and also other components of such diets. Thus, a high-fibre product containing a single source of a cellulosic fibre ingredient would also not confer all the health benefits observed when a varied, high-fibre diet is followed.

Defining dietary fibre physiologically allows recognition of the analogous carbohydrates. These are carbohydrates with structures and physiological properties similar to those of naturally occurring dietary fibres. These compounds are produced during food processing by chemical and/or physical processes affecting the digestibility of starches, or by purposeful synthesis (AACC 2001). Inclusion of such compounds ensures that the nutritional properties of various food ingredients, whether they are plant extracts, concentrates, modified carbohydrates or compounds produced by design, are not overlooked.

In summary, a workable definition of dietary fibre should: clarify the constituent makeup of dietary fibre; recognise that a primary characteristic is resistance to digestion and absorption in the small intestine and fermentation in the large intestine; and demonstrate that fibre has physiological properties.

The current working UK definition was derived in 1991 by the Committee on Medical Aspects of Food Policy (COMA) when the DRVs were set. At that time, the panel deemed the term 'dietary fibre' to be imprecise and therefore obsolete (DH 1991). Instead, the evidence was considered in relation to dietary non-starch polysaccharide. The justification for this approach was that non-starch polysaccharide makes up the major fraction of 'dietary fibre', is chemically identifiable, and can be measured with reasonable precision (DH 1991). The compounds that the term non-starch polysaccharide encompasses are listed in Table 3.

Table 3 Components of non-starch polysaccharides

	Description	Solubility at pH 7	Monomers	Occurrence
Cellulose	Unbranched 1–4 β-glucan	Insoluble	Glucose	Very widely distributed, especially beans, leafy vegetables, peas
Non-cellulose polysaccharides:				
	Pectins	Soluble	Galactauronic acid	Mainly fruits and vegetables
	Glucans	Soluble	Glucose	Oats, barley, rye
	Arabinogalactans	Partly soluble	Arabinose, xylose, galactose, glucose	Wheat, barley, rye
	Gums	Soluble	Galactose	Plant gums used as food additives
	Mucilages	Soluble	Arabinose, xylose	Seeds
	Inulin	Soluble	Fructose, galactose, mannose	Jerusalem artichokes
	Chitin	Insoluble	Amino sugars	Mushrooms and other fungi, exoskeleton of crustacean

Source: DH (1991).

Table 4 Constituents of dietary fibre included in the definition of the American Association of Cereal Chemists

Non-starch polysaccharides and resistant oligosaccharides	
Cellulose	Oligofructans
Hemicellulose	Galacto-oligosaccharides
Arabinoxylans	Gums
Arabinogalactans	Mucilages
Polyfructoses	Pectins
Inulin	
Analogous carbohydrates	
Indigestible dextrins	Polydextrose
Resistant maltodextrins (from maize and other sources)	Methyl cellulose
Resistant potato dextrins	Hydroxypropylmethyl cellulose
Synthesised carbohydrate compounds	Indigestible (resistant) starches
Lignin substances associated with the non-starch polysaccharides and lignin complex in plants	
Waxes	Saponins
Phytate	Suberin
Cutin	Tannins

Source: DeVries (2004).

This definition has been used to set the DRVs for the UK, and has also been the basis for the criteria for nutrition and health claims in the UK. When compared with the constituents of dietary fibre according to the AACC's definition, it is clear that there are substantial groups of compounds which are overlooked (Table 4). Given that these overlooked compounds can have health benefits, using non-starch polysaccharide-based methods for determining total dietary fibre content is increasingly being recognised as misleading. This is further complicated, in that traditional fibre fortification has been with added refined wheatfibre. While this ingredient is chemically analysed as non-starch polysaccharide, there are few clinical studies to substantiate that this inert material provides any of the health benefits previously described.

Measurement of the carbohydrate and dietary fibre content of food

A major factor that influences which compounds are included when we consider the dietary fibre content of food is the method selected to measure the non-digestible portion.

It is difficult to determine the exact amount of carbohydrate, both digestible and non-digestible, in individual foods, because the food composition tables use very different methods. However, a common approach is 'by difference'. In this case, an approximation is made by subtracting the measured protein, fat, ash and water from the total weight. Thus, the estimated carbohydrate content is the sum of nutritionally available carbohydrate (dextrins, starches and sugars), nutritionally unavailable carbohydrate (pentosans, pectins, hemicelluloses and cellulose) and non-carbohydrates (*e.g.* organic acids and lignins). This method is often inaccurate because of the summation of the errors in estimating the protein, fat, water and ash contents. This method also does not distinguish between those carbohydrates that are digested, absorbed and utilised by the body, and those that are unavailable for digestion in the small intestine and pass through to the colon. However, there are more time-consuming, but more accurate, methods that can be used to quantify the different types of carbohydrate present. Individual sugars can be extracted with alcohol and measured by high-performance liquid chromatography (Dean 1978; Southgate *et al*. 1978) or specific enzymatic colorimetric tests (Southgate 1976). Starches and dextrins are first hydrolysed, and the resultant glucose can then be measured in a similar way (Dean 1978).

Owing to the chemical diversity of dietary fibre, a number of different methods have evolved to estimate the quantity of indigestible material in foods. All methods use a dried, defatted food sample, but each measures a different chemical fraction. The methods available can be broadly divided into enzymatic-gravimetric and enzymatic-chemical methods.

Enzymatic-gravimetric methods attempt to isolate the fraction of the diet that resists digestion in the gastrointestinal tract and, in doing so, measures a variety of different components. This methodological approach complements definitions based on the indigestibility of dietary fibre; enzymes are used to mimic digestion of the non-fibre components in the gastrointestinal tract, and then the remaining fraction is weighed (hence 'gravimetric'). It is not possible to measure the quantities of the individual polysaccharides present, or assess the amount of soluble and insoluble fibre, unless modifications (solvent extractions) are made to the 'traditional' method (AOAC 985.29; Prosky *et al*. 1985).

Alternatively, the enzymatic-chemical methods identify the non-starch polysaccharides present in food (Englyst & Cummings 1988). The methods again digest the fibre using enzymes, and then solvents are used to extract the different fractions. However, instead of weighing the isolated fraction, the sugar units are determined using colorimetric or chromatographic methods. Alternative versions of this method also estimate the lignin fraction of dietary fibre (Southgate 1969).

Some believe non-starch polysaccharide to be the more physiologically relevant fraction and the best indicator of the functional, plant-derived dietary fibre in the diet (DH 1991). However, enzymatic-chemical methods exclude compounds such as inulin and resistant starch that have physiological effects but are not components of the cell wall. When the decision was made to use this method in the UK, the number of foods affected by this underestimation of dietary fibre was deemed to be small. However, now that the health effects of these compounds are better understood, and given that they are increasingly being used as functional food ingredients, this approach may have an adverse impact on the types of nutrition and health claims that such products can make. An overview of the different components of dietary fibre, measured by the various methods of fibre analysis that are currently in use around the world, is displayed in Table 5.

Unsurprisingly, the different methods used to assess the 'total' fibre content can produce substantial variations in the estimate of the dietary fibre content for the same food (Table 6). Thus, it is

Table 5 Components measured by the various methods of dietary fibre analysis, presented in alphabetical order by author

Method	Lignin	NSP	Resistant starch	Inulin	Oligo-saccharides	Poly-dextrose	Resistant maltodextrose	Chitin	Non-carbohydrate
Asp *et al.* (1983)	✓	✓	SOME	SOME	×	×	×	SOME	SOME
Craig *et al.* (2000) AOAC 2000.11	×	×	×	×	×	✓	×	×	×
Englyst and Cummings (1988) (GC)[2]	×	✓	×	×	×	×	×	SOME	×
Englyst and Hudson (1987) (C)	×	✓	×	×	×	×	×	SOME	×
Gordon and Ohkuma (2001) AOAC 2001.03	✓	✓	SOME	✓	✓	✓	✓	SOME	SOME
Hoebregs (1997) AOAC 997.08	×	×	×	✓	×	×	×	×	×
Lee *et al.* (1992) AOAC 991.43[1]	✓	✓	SOME	SOME	×	×	×	SOME	SOME
Li and Cardozo (1994) AOAC 993.21	✓	✓	SOME	SOME	×	×	×	SOME	SOME
McCleary *et al.* (2000) AOAC 999.03	×	×	×	✓	×	×	×	×	×
McCleary and Monaghan (2002) AOAC 2002.02	×	×	✓	×	×	×	×	×	×
Mongeau and Brassard (1993) AOAC 992.16	✓	✓	×	×	×	×	×	SOME	SOME
Prosky *et al.* (1985) AOAC 985.29[1]	✓	✓	SOME	SOME	×	×	×	SOME	SOME
Quigley and Englyst (1994) (HPLC)	×	✓	×	×	×	×	×	SOME	×
Schweizer and Wursch (1979)	✓	✓	SOME	SOME	×	×	×	SOME	SOME
Southgate (1969)	✓	✓	SOME	×	×	×	×	SOME	×
Theander and Aman (1979)	✓	✓	SOME	×	×	×	×	SOME	×
Theander and Westerlund (1986)	✓	✓	SOME	×	×	×	×	SOME	×
Theander *et al.* (1995) AOAC 994.13	✓	✓	SOME	×	×	×	×	SOME	×

[1]Methods that are most commonly used for labelling purposes globally; [2]method that has been in common use in the UK (although not for labelling purposes).
Source: FNB (2001).
AOAC, Association of Official Analytical Chemists; GC, enzymatic-gas chromatographic; C, colorimetric; HPLC, high-performance liquid chromatographic.

Table 6 Dietary fibre content of dried red kidney beans estimated by five different methods

Method	Fibre content (g/100 g)	Fibre components
Englyst	15.7	Soluble + insoluble (not lignin or resistant starch)
Southgate	23.4	Soluble + insoluble, lignin
AOAC	21.5	Soluble + insoluble, lignin, resistant starch
Neutral detergent fibre (Van Soest)	10.4	Insoluble fibre only
Crude fibre	6.2	Part of the insoluble fibre

Source: Lyons-Wall (2000).
AOAC, Association of Official Analytical Chemists.

essential that, when comparisons are made between carbohydrate contents of foods taken from different data sets, consideration is given to the method that has been used.

Issues relating to the determination of resistant starch

A further complication when analysing dietary fibre involves the fraction of the starch present in certain foods that is not completely digested, so is in part unavailable for absorption. The biological significance of this resistant starch fraction was previously not appreciated, so it was not an issue that any resistant starch present in the sample was digested by the enzymes in the assay used and, in effect, assumed to be digestible. However, now health benefits have been attributed to this fraction (see Nugent 2005), it is reasonable that any resistant starch present in a food is also accounted for in the estimate of dietary fibre content. Any method to measure the content of resistant starch in foods must first remove all of the digestible starch from the product using thermostable amylases (McCleary & Rossiter 2004). At present, the method of McCleary and Monaghan (2002; AOAC method 2002.02) is considered the most reproducible measurement of resistant starch in plant materials, but it has not been shown to analyse all resistant starch as defined (Champ *et al*. 2003). It is based on the principle of enzymic digestion, and measures the portions of starch resistant to digestion at 37°C that are typically not quantitated owing to the gelatisation at 100°C followed by digestion at 60°C.

The most commonly used methods internationally to determine total dietary fibre in foods for labelling purposes (AOAC 985.29; AOAC 991.43) account for some of the resistant starch present (*i.e.* RS3, the retrograded portion, and RS2 as found in high amylose maize) as part of the total dietary fibre value, although in some cases, only part of the true resistant starch portion is measured. Therefore, while it does measure some resistant starch as part of the total dietary fibre figure, additional methods are needed for quantification of the other categories of resistant starch (Champ *et al*. 2003). Again, this highlights the need for a universally agreed definition and method of analysis for all of the components of dietary fibre, including resistant starch. The Englyst method used in recent years to quantify non-starch polysaccharide in the UK does not measure the resistant starch content of food (see Table 5) and, hence, apparent total fibre values are lower than when the AOAC method is applied.

Food sources of carbohydrates and dietary fibre

Mono- and disaccharides

Simple sugars are abundant in the human diet. Glucose occurs naturally in fruit and plant juices, and can be manufactured from starch for use as a food ingredient (glucose syrups). Such syrups are produced by the acidic or enzymic hydrolysis of maize or wheat starch, and consist of mostly glucose, although other monosaccharides may also be present. Typically, these syrups are less sweet than glucose, and are an economical alternative to using sucrose derived from sugar cane and sugar beet. They are present in many manufactured foods, such as confectionery, soft drinks and preserves, providing foods with good eating quality, improved texture and longer shelf life, compared with similar foods that do not contain these sugar syrups (Hanover & White 1993). Fructose also occurs naturally in most fruits and vegetables, and honey, and it is the sweetest sugar known, so less can be used. Some of the glucose in glucose syrups can be converted to fructose to produce high-fructose syrups.

It has recently been suggested that the increase in consumption of glucose-fructose syrups specifically over the last 30 years, in the United States, is in part responsible for the increase in prevalence of obesity (Bray *et al*. 2004) and also type 2 diabetes (Gross *et al*. 2004). However, a similar increase in rates of both obesity and type 2 diabetes has been observed in the UK; yet here the use of glucose-fructose syrups is not widespread. At present, there is insufficient evidence to confirm or refute an association between glucose-fructose syrup consumption and obesity and type 2 diabetes in the USA. However, it is clearly an area of consumer, media and scientific interest that requires further research. In the meantime, it is prudent to consume foods high in sugar in moderation, in order to prevent the risk of too much weight gain and to limit frequency with regard to tooth decay. This applies to foods naturally high in sugar, as well as those containing these sugar syrups.

Sucrose, maltose and lactose are the most common disaccharides in the diet. They occur naturally in sugar cane (sucrose), sugar beet (sucrose), malt beverages such as beer (maltose), and milk (lactose).

In 1991, COMA took the decision to describe sugars as either intrinsic or extrinsic. Intrinsic sugars are those which are incorporated into the cellular structure of foods (*e.g.* sugars in whole fruits and vegetables), whereas extrinsic sugars are those that are not bound into a cellular structure [*e.g.* the lactose (milk sugar) in

dairy products]. Honey, fruit juices, table sugar and confectionery are also examples of foods containing extrinsic sugars, referred to as non-milk extrinsic sugars (NMES), and their occurrence in the diet should be limited. Once broken down, the body handles the glucose in exactly the same way, so the distinction is purely a means of categorising the dietary source of the sugar. However, from an analytical point of view, it is not possible to determine the amount of NMES in a product directly (instead, relatively complex estimates need to be made). As a result, the value for 'total sugars' is often quoted as an alternative, as this can be determined analytically. Furthermore, it is 'total sugars' that is required for UK (and EU) labelling purposes.

Polysaccharides

Plant foods provide almost all the polysaccharides in the human diet. These are present in a variety of forms. Very small amounts of glycogen are consumed in liver, as well as mucopolysaccharides, which are present in low levels in most animal tissues. However, most polysaccharides are broken down to glucose when the animal dies.

The major dietary polysaccharides are starches. These exist in granules of a size and shape characteristic for a plant. In this form, some starches are insoluble in water, and indigestible if eaten raw. This includes starches found in potatoes and in flour. When the foods are boiled in water, the starch granules swell and gelatinise. This processing makes the starch granules more easily digested. Starches are present in all cereal grains and flours, both wholegrain and white, as well as legumes and nuts, and very small amounts in fruits and vegetables. The forms of processing used to manufacture some breakfast cereals, such as dry heat, make some of the starch present non-digestible (resistant starch). Similarly, if gelatinised starches are cooled, as happens when cooked potatoes cool, the starch is once again resistant to digestion (see section on *Resistant starch*).

Dietary fibre

All plant foods that contain cell wall material contain substances that are indigestible in the human gastrointestinal tract. Cereals, especially wholegrain foods, are rich sources of dietary fibre. Typically, a cereal grain contains 10–15% non-digestible material. Wheat and maize contain a high proportion of insoluble polysaccharides, such as cellulose and hemicelluloses; oats and barley contain soluble gums, such a beta-glucans, and rye contains soluble pentoses. Cereals also contain a high proportion of lignin. Lignin is not a polysaccharide, but is a polymer of phenylpropane units. It is chemically linked to the hemicelluloses in plant cell walls, so is considered as a component of dietary fibre, although it is not captured by the Englyst method. Vegetables contain lower amounts of non-starch polysaccharides, as they have such a high water content. Of the non-starch polysaccharide fraction that is present, approximately 30–40% is cellulose. The remaining compounds are non-cellulosic polysaccharides, such as polymers of uronic acids and arabinogalactans. Some seed legumes, such as beans and peas, contain non-starch polysaccharides stored in the cell walls (*e.g.* guar gum, locust gum and galacto-oligosaccharides).

However, there are other non-digestible components of plants that contribute to the total dietary fibre intake in the diet. These include lignin in the outer coats of seeds, the waterproof waxy materials cutin, and suberin in the outer layers of leaves and fruits. Beans and pulses also contain thick cutinised seed coats, and fungi contain chitin, which is a polymer of amino sugars. Algae are not widely consumed in the UK, but they contain polysaccharides that are used as food additives, such as agar and carageenans.

Table 7 illustrates the wide variation in the carbohydrate and dietary fibre content of foods. The data presented are taken from the UK McCance and Widdowson food tables (FSA 2002) and, so the values for fibre were measured by the Englyst method (non-starch polysaccharide). Total dietary fibre content has been estimated using a conversion factor of 1.33.

Additional information on fibre subfractions has also been published in supplements to the McCance and Widdowson food tables: cereals (MAFF 1988a), vegetables (MAFF 1991), and fruits and nuts (MAFF 1992), which supply values for the various subfractions of non-starch polysaccharides, including values for cellulose, soluble and insoluble non-cellulosic polysaccharides, lignin and resistant starch, as well as dietary fibre measured using both the Englyst and Southgate methods. This more extensive data set has been included in the Appendix.

Analytical methods for measuring the components of dietary fibre have improved and, in many cases, the data available in national food composition tables lag behind that which is used in nutritional studies (*e.g.* Bednar *et al.* 2001; Liljeberg Elmstahl 2002; see Table 8, which illustrates the substantial contribution that resistant starch can make to the total amount of non-digestible material present).

Table 7 The carbohydrate content (g/100 g as eaten) of foods

Food	Sugars	Starch	NSP	AOAC	Food	Sugars	Starch	NSP	AOAC
Cereals					Courgette	1.9	0.1	1.2	1.6
All-bran	19.0	27.6	24.5	32.6	Cucumber	1.4	0.1	0.6	0.8
Barley, pearled	0	27.6	1.6	2.1	Kidney bean, red	3.6	12.8	6.2	8.2
Bran, wheat	3.8	23.0	36.8	48.9	Lentil, red	0.8	16.2	1.9	2.5
Brown bread	3.0	41.3	3.5	4.7	Lettuce	1.7	0	0.9	1.2
Brown rice	0.5	31.6	0.8	1.1	Marrow	1.4	0.2	0.6	0.8
Buckwheat	0.4	84.5	2.1	2.8	Mushroom, fried	0.1	0.2	1.5	2.0
Cornflakes	7.2	77.7	0.9	1.2	Okra	2.3	0.5	3.6	4.8
Cornflour	0	92.0	0.1	0.1	Old potato	1.0	14.5	1.1	1.5
Crispbread	3.2	67.4	11.7	15.6	Onion, fried	10.0	0.1	3.1	4.1
Croissants	1.0	37.2	1.6	2.1	Parsnip	5.9	6.4	4.7	6.3
Digestive biscuit	13.6	55.0	2.2	2.9	Potato crisps	0.7	52.6	5.3	7.0
Granary bread	2.2	44.1	4.3	5.7	Radish, red	1.9	0	0.9	1.2
Hot cross bun	23.4	35.1	1.8	2.4	Runner bean	2.0	0.3	1.9	2.5
Macaroni	0.3	18.2	0.9	1.2	Tomato	3.1	0	1.0	1.3
Naan bread	5.5	44.6	1.9	2.5	Turnip	1.9	0.1	1.9	2.5
Noodles, egg	0.2	12.8	0.7	0.9	Watercress	0.4	0	1.5	2.0
Oat & wheatbran	16.7	51.0	17.9	23.8	Yam	0.7	32.3	1.4	1.9
Oat bran flakes	16.8	57.2	10.0	13.3					
Pitta bread, white	2.4	55.5	1.6	2.1	*Fruits & Nuts*				
Porridge	0	9.0	0.8	1.1	Almond	4.2	2.7	7.4	9.8
Puffed wheat	0.3	67.0	5.6	7.4	Apple	11.8	0	1.8	2.4
Rice Krispies	10.6	79.1	0.7	0.9	Apricot	7.2	0	1.7	2.3
Rye bread	1.8	44.0	4.4	5.9	Avocado	0.5	0	3.4	4.5
Scones, plain	5.9	47.9	1.9	2.5	Blackberry	5.1	0	3.1	4.1
Shredded wheat	0.8	67.5	9.8	13.0	Brazil nut	2.4	0.7	4.3	5.7
Spaghetti, white	0.5	21.7	1.2	1.6	Cashew nut	5.6	13.2	3.2	4.3
Spaghetti, wholemeal	1.3	21.9	3.5	4.7	Cherry	11.5	0	0.9	1.2
Sweetcorn	9.6	16.6	1.4	1.9	Fig	48.6	0	6.9	9.2
Water biscuit	2.3	73.5	3.1	4.1	Gooseberry	18.5	0	1.7	2.3
Weetabix	5.2	70.5	9.7	12.9	Grapefruit	6.8	0	1.3	1.7
Wheat flour, white	1.5	76.2	3.1	4.1	Hazelnut	4.0	2.0	6.5	8.6
Wheat flour, wholemeal	2.1	61.8	9.0	12.0	Kiwi fruit	10.3	0.3	1.9	2.5
Wheatgerm	16.0	28.7	15.6	20.7	Lychee	14.3	0	0.7	0.9
White bread	2.6	46.7	1.5	2.0	Mango	13.8	0.3	2.6	3.5
White rice	0	30.9	0.1	0.1	Melon, cantaloupe	4.2	0	1.0	1.3
					Nectarine	9.0	0	1.2	1.6
Vegetables					Olive	0	0	2.9	3.9
Asparagus	1.4	0	1.4	1.9	Orange	8.5	0	1.7	2.3
Aubergine	2.6	0.2	2.3	3.1	Passion fruit	5.8	0	3.3	4.4
Baked bean	5.8	9.3	3.5	4.7	Peanut	6.2	6.3	6.2	8.2
Beetroot	8.8	0.7	1.9	2.5	Pear	10.0	0	2.2	2.9
Broad bean	0.9	4.3	5.4	7.2	Pineapple	10.1	0	1.2	1.6
Broccoli, green	0.9	0	2.3	3.1	Plum	8.3	0	1.5	2.0
Brussels sprouts	3.0	0.3	3.1	4.1	Prune, canned	19.7	0	2.4	3.2
Butter bean	1.5	15.6	5.2	6.9	Raspberry	4.6	0	2.5	3.3
Cabbage	2.0	0.1	1.8	2.4	Strawberry	6.0	0	1.1	1.5
Carrot	4.6	0.2	2.5	3.3	Sunflower seed	1.7	16.3	6.0	8.0
Chickpea	1.0	16.6	4.3	5.7	Tangerine	8.0	0	1.3	1.7
Chips, fried	0.6	29.5	2.2	2.9	Walnut	2.6	0.7	3.5	4.7

Source: FSA (2002).

AOAC, Association of Official Analytical Chemists; NSP, non-starch polysaccharide.

Table 8 Analyses of components of dietary fibre in starchy foods

Food	Resistant starch (g/100 g dry matter)	Soluble fibre (g/100 g dry matter)	Insoluble fibre (g/100 g dry matter)
Legumes†			
Red kidney beans	24.6	0.5	36.3
Lentils	25.4	0.1	33.0
Black-eyed peas	17.7	0.2	32.4
Cereal grains†			
Barley	18.2	5.0	12.0
Corn	25.2	3.6	16.0
White rice	14.1	0.3	1.2
Wheat	13.6	2.3	14.7
Oats	7.2	3.8	33.9
Flours†			
Corn	11.0	0.0	2.8
Wheat	1.7	3.6	8.5
Rice	1.6	1.7	3.4
Potato	1.7	1.0	1.1
Grain-based food products†			
Spaghetti	3.3	1.9	3.7
Rolled oats	8.5	3.4	6.6
Reference substrates†			
Corn starch	8.1	0.0	0.0
Potato starch	66.9	0.0	0.0
High-amylose maize starch	52.0	2.1	3.2
Cereal products‡			
Crisp bread	1.4	n/a	n/a
White bread	1.9	n/a	n/a
'Granary' bread	6.0	n/a	n/a
Extruded oat cereal	0.2	n/a	n/a
Puffed wheat cereal	1.2	n/a	n/a
Oat porridge	0.3	n/a	n/a
Cooked spaghetti	2.9	n/a	n/a
Cooked rice	3.7	n/a	n/a
Potato products‡			
Boiled potatoes	2.0	n/a	n/a
Chips	4.8	n/a	n/a
Mashed potatoes	2.4	n/a	n/a

Source: †Bednar *et al*. 2001) – quantification of resistant starch portion involved setting up an *in vitro* model of digestion using bacteria isolated from faeces. ‡Liljeberg Elmstahl (2002) – quantification of resistant starch involved chewing and then enzymic incubation according to the method of Akerberg *et al*. (1998).

Effects of dietary fibre in the gastrointestinal tract

Consumers, health professionals, researchers and manufacturers are interested in dietary fibre because of the physiological effects of fibre in the gastrointestinal tract. Indeed, the health benefits of consuming diets rich in non-digestible plant components were appreciated long before the term dietary fibre was even determined:

> "*And this I know, moreover, that to the human body it makes a great difference whether the bread be fine, or coarse; of wheat with or without the hull, whether mixed with much or little water, strongly wrought or scarcely at all, baked or raw and a multitude of similar differences; and so, in like manner, with the cake; the powers of each, too, are great, and the one nowise like the other. Whoever pays no attention to these things, or, paying attention, does not comprehend them, how can he understand the diseases which befall a man?*" (Hippocrates, 400 BC)

The current interest in dietary fibre stems from the 'dietary fibre hypothesis' put forward by Burkitt, Trowell, Walker, Painter and colleagues in the 1970s (Burkitt *et al*. 1972; Trowell 1972; Trowell 1974; Painter 1975).

Here the health effects observed in individuals on high-fibre diets were, for the first time, related to the proportion of the diet that resisted digestion in the human gut. Over time, the consumption of low amounts of fibre has been associated with numerous health problems, including constipation and diverticular disease, as well as colorectal cancer, hiatus hernia, appendicitis, obesity, CHD, duodenal ulcers, breast cancer and gallstones. For these latter diseases, there is no strong evidence to suggest that lack of fibre does affect an individual's risk of developing these diseases, but it is a clear demonstration of the wide range of body systems that fibre has been associated with. As was apparent earlier, dietary fibre is itself a complex dietary component, consisting of a number of different components that all have different biological functions. However, there are five main areas where there is sufficient evidence that can be assessed to generate a consensus opinion of the health effects of dietary fibre. These are:

- improvements in gastrointestinal health;
- improvements in glucose tolerance and the insulin response;
- reduction of hyperlipidaemia, hypertension and other CHD risk factors;
- reduction in the risk of developing some cancers;
- increased satiety and hence some degree of weight management.

Yet, the different methods used to determine the dietary fibre content of foods have slowed the speed of progression in this area. Some studies measure dietary fibre as non-starch polysaccharide, whereas others use the AOAC method and determine the total dietary fibre content (although the precision of the final value once again will depend on the method selected). Thus, the available studies are difficult to compare, as they utilise different definitions as well as different study methodologies. A further complication is that a high-fibre diet is often lower in fat and higher in antioxidant vitamins and minerals, which introduces potential confounding factors; that is, this represents a generally healthier dietary pattern (Davey *et al*. 2003). Nevertheless, it is prudent to consider how the health effects of dietary fibre in the body are thought to be brought about before considering the strength of evidence in each area.

Dietary fibre helps prevent constipation by providing bulk to the faeces. Bulky faeces move through the gut faster and result in an increased stool weight, and the increased faecal bulk 'dilutes' the effect of any genotoxic agents in the large intestine, thereby reducing the extent of DNA damage to the cells lining the colon. However, other mechanisms via which fibre can exert health effects in the body have been described (James *et al*. 2003).

Formation of gels in the stomach and small intestine

The traditional soluble fibres form gel-like substances when they are exposed to water in the stomach and small intestine. The presence of these gels slows gastric emptying, hastens small intestinal transit, and helps control the absorption of nutrients. This can have a major impact on the rate at which glucose appears in the bloodstream and thus on the GI of a food. It can also impact on an individual's feeling of fullness (satiety) and ultimately the total amount of food consumed over a period.

Fermentation of indigestible material by colonic bacteria

The various components of dietary fibre, especially those associated with resistant starch, reach the large intestine (*i.e*. the colon) virtually unchanged (Baghurst *et al*. 2001). However, these compounds (including polysaccharides, oligosaccharides) are fermented by the microbial flora naturally present in the colon, to produce low levels of the gases carbon dioxide, methane and hydrogen, as well as organic acids and SCFAs. The main SCFAs produced in the human gut are butyrate, propionate and acetate. The concentrations of SCFAs in the large intestine vary, depending on the types of polysaccharides fermented, although generally acetate is the most abundant and butyrate is the least abundant (MacFarlane & MacFarlane 2003). Fermentation of resistant starch appears to favour the production of butyrate (see Nugent 2005). Concentrations also vary in the different regions of the large intestine, with higher concentrations detected in the area nearest the junction with the small intestine (70–140 mM; Topping *et al*. 2003). The SCFAs lower the pH of the contents of the large intestine. This is beneficial to health, because the reduced pH creates an environment that prevents the growth of harmful bacteria (Topping & Clifton 2001). A lower pH is thought to aid the absorption of minerals, such as calcium and magnesium. SCFAs increase the blood flow to the colon and provide the cells in the wall of the intestine with a metabolic fuel (mainly in the form of butyrate) (Schwiertz *et al*. 2002). Additionally, butyrate has been shown to induce programmed cell death (apoptosis) and exert a level of control over the cell cycle (Mentschel & Claus 2003). This suggests that butyrate might play an important role in maintaining the integrity of the gut wall, by preventing the uncon-

trolled proliferation of abnormal cells that occurs in the early stages of colorectal cancer.

Additionally, by fibre providing a fuel for the anaerobic bacteria residing in the large bowel, the numbers of these bacteria increase. An increased bacterial population results in an increased use of sulphur and nitrogen substances, such as phenols and ammonia. Thus, the luminal levels of these potentially toxic compounds are reduced, which minimises the potential exposure to the colonic epithelium. It is believed that the fermentation of certain dietary components, such as inulin, selectively stimulates the growth of bifidobacteria which are thought to be associated with a healthy gut (see Roberfroid 2005). Such compounds have been termed prebiotics.

'Mop and sponge' effect in the colon

Insoluble components of dietary fibre, including the non-starch polysaccharides, trap water molecules within the branched structure. For example, wheatbran holds 2–6 g of water per gram, and fruits and vegetables hold 18–30 g per gram (BNF 1990). These fibres also bind water like a sponge during transit through the gastrointestinal tract, which has a number of biological effects, both beneficial and potentially adverse. Soluble polysaccharides also hold a substantial amount of water, which can cause intestinal distension and help move the luminal contents through the gastrointestinal tract. Insoluble fibres also bind molecules, such as bile acids and carcinogens, preventing them from damaging the colonic epithelium, hence acting like a 'sponge'. The 'mop' effect is not fully understood, but the physical presence of insoluble fibres in the colon in some way exerts a trophic effect on the epithelium, which appears to enhance barrier function and suppresses tumourigenesis (Sengupta *et al*. 2001), although this has not been confirmed (Wong & Gibson 2003).

Cholesterol-lowering effects

The presence of viscous fibres in the diet, such as beta-glucan in oats, effects a reduction in serum total and low density lipoprotein (LDL)-cholesterol concentrations (see Spiller 1999). This is brought about by a reduction in the amount of cholesterol absorbed from the small intestine (Lia *et al*. 1997) and the amount of bile acids absorbed in the ileum (Lia *et al*. 1995). Also, there is evidence that specific SCFAs produced as a result of bacterial fermentation in the colon, namely propionic acid, may inhibit cholesterol synthesis in the liver (Hara *et al*. 1999).

Concomitant changes in other aspects of the diet

Diets that are high in fibre (either from natural sources or added fibre) tend also to be lower in fat and higher in micronutrients, than low-fibre diets. They are also more satiating, often leading to reduced total food intake. As a result, such diets are more likely to meet national recommendations for a healthy diet. Owing to its inherent indigestibility, dietary fibre has a lower calorific value than that of digestible carbohydrate (16 kJ/g or 4 kcal/g). Therefore, foods high in any form of dietary fibre may provide less energy, weight for weight, although this will depend on the amounts of other macronutrients in the food. Currently, for labelling purposes in the EU, dietary fibre is assumed to have a zero energy value. However, discussion is ongoing to agree on a conversion factor to account for the metabolisable energy potential of dietary fibre – the EU is yet to decide what figure will be used, but it is likely that a value of 8 kJ/g or 2 kcal/g will be adopted.

Proposed adverse effects of high-fibre diets

Legitimate concerns have been raised that consuming large amounts of fibre may be harmful to health. The gaseous products of fermentation are hydrogen and carbon dioxide. The unwanted hydrogen gas has a number of different fates: it is exhaled in the breath; anaerobic bacteria in the gut convert it, along with carbon dioxide, to acetate (acetogenesis); it leads to the formation of methane in the lumen of the gastrointestinal tract; or it is used to chemically reduce luminal sulphates. The resultant sulphides are potentially toxic to the colonic epithelium, and it has been suggested that these compounds are involved in the pathogenesis of ulcerative colitis (Roediger *et al*. 1993). Also, the production of excess methane, along with the bulking effects of a high-fibre diet, can induce bloating and excess wind. Such symptoms are not harmful to health but are poorly tolerated by many individuals, and are problematic especially when a high-fibre diet is initially followed. The colon does eventually adapt to these dietary changes, albeit over a period of several weeks.

Fermentable fibres that lower luminal pH may enhance mineral absorption in the large intestine (Trinidad *et al*. 1996). However, the absorption of certain minerals in the duodenum and ileum may be compromised by the presence of high-fibre foods if they contain phytate (*e.g*. wheatbran). Phytate (inositol hexaphosphate) forms complexes with many minerals in the gastrointestinal tract and reduces their availability for absorption. This can have a significant effect on the

amounts of iron, calcium, magnesium and zinc absorbed and so, for individuals with inadequate intakes of these nutrients, this may have an impact on micronutrient status (Frolich 1990). However, as food sources of fibre introduce a mixture of fermentable and non-fermentable fibres into the gut, there may be opposite effects in different portions of the gut. Availability of certain micronutrients may be reduced in the small bowel, but may be enhanced by the lower pH of the large bowel. As a result, for the general population, fibre is considered to have a neutral effect on mineral bioavailability.

Finally, although for the majority of the population it is advantageous that high-fibre foods have a low energy density, for some, this can be an issue. Young children and elderly people who consume proportionally less food might find it difficult to meet their energy requirements if their diets are high in fibre. Consequently, care must be taken when dietary recommendations are set, to ensure that this will not have an adverse effect on health.

Recommendations for dietary fibre intake

Owing to the nature of nutrition research, many, often conflicting, results are generated. In an attempt to promote new research, and owing to the current media interest in nutrition, the general population are often exposed to confusing messages. It is therefore essential that governments, health professionals and consumers have a set of authoritative nutrition recommendations that represent the consensus opinion of nutrition experts. Recommendations can be set in one of two ways. First, there are recommended nutrient intakes, which state a desired level for population intakes of the major nutrients in the diet. Alternatively, dietary goals or guidelines which help consumers to choose foods that are most likely to bring about improvements in long-term health can be set. Such guidelines include advice to 'eat more whole grain foods' or to 'cut down on salt'.

Scientific studies are rarely designed specifically to identify or to support particular dietary guidelines, so information must be collated and interpreted from studies conducted for other purposes (McMurry 2003). Expert committees generally base their assessment on a number of sources of information, including evidence-based systematic reviews, consensus documents, research studies and expert presentations. Committees also consider comments from stakeholders, including the public, as draft recommendations or guidance are often released for consultation before the final report is published. The next section summarises current dietary advice on dietary fibre from a global perspective.

Current dietary advice

United Kingdom

The UK's DRVs were published in 1991 by the Department of Health following guidance from COMA. At this time, a DRV was set for non-starch polysaccharide primarily on the basis of improved bowel function in adults (DH 1991). The Committee observed that stool weights below 100 g/day were associated with an increased risk of bowel disease, and there was sufficient evidence to suggest that such weights occurred at intakes of non-starch polysaccharides less than 12 g per day. Increasing intake of non-starch polysaccharides brought about a concurrent increase in stool weight up until intakes of 32 g per day. There was insufficient evidence to suggest that intakes above this amount were associated with any further increase in stool weight. The current population average intake was 13 g per day, and the Committee concluded that increasing intakes to 18 g per day would increase stool weight by 25%. Thus, the DRV was set as 18 g of non-starch polysaccharides per day, with a range for individuals of 12–24 g per day.

The non-starch polysaccharides should come from a variety of foods whose constituents contain it as a naturally integrated component. This 18 g/day recommendation is not applicable for children, and no specific value for those under 18 years of age was set. However, it was assumed that the effects of non-starch polysaccharides were likely related to body size, so children should consume proportionally lower intakes. Additional advice was given for children under 2 years of age. In this case, it was not appropriate to consume foods rich in non-starch polysaccharides at the expense of energy-rich foods which are required for growth. Similarly, a caution was included to avoid high intakes in the elderly population, as phytate present in non-starch polysaccharides-rich foods may reduce mineral absorption.

Australia and New Zealand

Prior to 1997, dietary guidelines were set for Australia by an expert committee, and these guidelines were then adopted by the New Zealand Government. Thus, although the guidelines were used across Australasia, they were derived with an Australian population in mind. At a workshop in 1997, it was formally suggested that the nutrient recommendations should be derived by an expert committee made up of eminent scientists from Australia and New Zealand, to ensure that such values were appropriate for both countries. In 2006, the first

complete set of values was published by FSANZ (NHMRC 2006).

FSANZ defines dietary fibre as: 'that fraction of the edible parts of plants or their extracts, or synthetic analogues, that are resistant to the digestion and absorption in the small intestine, usually with complete or partial fermentation in the large intestine. Dietary fibre includes polysaccharides, oligosaccharides (degree of polymerisation >2) and lignins, and promotes one or more of the following beneficial physiological effects:

(i) laxation
(ii) reduction in blood cholesterol
(iii) modulation of blood glucose.

This definition is dependent on the use of an AOAC method to measure total dietary fibre that includes inulin, fructosaccharides and polydextrose (FSANZ 2001). The definition would encompass the resistant starch component in foods but, currently, FSANZ has no official method to measure this fraction. Whereas the recommendation made for the UK population is based solely on non-starch polysaccharide's effect on bowel health, the Australian and New Zealand committee considered other health outcomes:

(1) Cholesterol-lowering ability of soluble fibres. The panel concluded that, while wheatbran, wheat wholemeal products and cellulose have no effect on serum cholesterol, pectin, oat bran, psyllium and guar gum can effect a reduction in total and LDL-cholesterol. Consequently, soluble fibre can bring about a reduction in cardiovascular disease risk, and the levels at which a significant effect is seen is around the 70–80th centile of current intakes (Pietinen *et al*. 1996; Rimm *et al*. 1996; Wolk *et al*. 1999).
(2) Cancer prevention. The Panel concluded that results from well-designed prospective cohort studies are inconsistent and there is no clear evidence that high-fibre diets prevent breast or colorectal cancer. Correlations between intakes of fruits and vegetables and cancer outcomes are better than those considering fibre from cereal sources.

The DRVs are published as adequate intakes (AIs). This value corresponds to the average daily nutrient intake level based on observed or experimentally determined approximations, or estimates of nutrient intake that are assumed to be adequate by apparently healthy people. In the case of dietary fibre, the AI is set at the median dietary fibre intake in Australia and New Zealand based on the 1995 National Nutrition Survey of Australia and the 1997 National Nutrition Survey of New Zealand. For all ages, the values for males and females were set at the highest median of any age group, plus an allowance of slightly more than 4 g/day for men and slightly less than 3 g/day for women for the component of resistant starch not included in the data on dietary fibre. These are the first national dietary recommendations that have included special mention of the resistant starch component in the dietary fibre. AIs are displayed in Table 9 and can be seen to be considerably higher than the UK values, a situation that is only partially accounted for by the difference in methodology used (Englyst in the UK, compared with AOAC).

If these recommendations are adhered to, average intakes will have to move to what is currently the median of the highest quintile of population intake (*i.e.* the 90th centile). This may require a substantial shift in diet if the increase in intake is brought about by additional vegetables, legumes and fruits in the diet (although this would also increase intakes of antioxidant vitamins and folate). An alternative option is to encourage the incorporation of high-fibre food ingredients into manufactured foods, such as resistant starches. Indeed, Australia's Commonwealth Scientific and Industrial Research Organisation (CSIRO), which independently assesses the evidence relating to specific dietary components and health outcomes and publishes its own dietary advice, has recommended that intakes of resistant starch should be around 20 g per day, which is

Table 9 Nutrient reference values for Australia and New Zealand

	Adequate intake (g/day)		Adequate intake (g/day)
Males		Females	
0–6 months	n/a	0–6 months	n/a
7–12 months	n/a	7–12 months	n/a
1–3 years	14	1–3 years	14
4–8 years	18	4–8 years	18
9–13 years	24	9–13 years	20
14–18 years	28	14–18 years	22
19–30 years	30	19–30 years	25
31–50 years	30	31–50 years	25
51–70 years	30	51–70 years	25
71+ years	30	71+ year	25
		Pregnancy	
		14–18 years	25
		19–30 years	28
		31–50 years	28
		Lactation	
		14–18 years	27
		19–30 years	30
		31–50 years	30

Source: NHMRC (2006).

almost four times greater than that currently provided by a typical western diet (Baghurst *et al*. 1996).

United States of America and Canada

The process for the revision of the DRVs for the United States and Canada was initiated in 1993. After over a decade of discussion and analysis, a comprehensive set of reference values for nutrient intakes for healthy US and Canadian individuals and populations was published in 2005. The final report, 'Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients)', was a product of the FNB, working in cooperation with Canadian scientists.

During this process, it was acknowledged that a new definition of dietary fibre was required to set new recommended intakes. The Institute of Medicine in the United States convened a subcommittee to evaluate all existing definitions for fibre and propose a new definition to use. The new definition put forward by the subcommittee included carbohydrate components not captured in the AOAC analysis, such as some resistant starch fractions and oligosaccharides (FNB 2001). Thus, 'Dietary Fibre' consists of non-digestible carbohydrates and lignin that are intrinsic and intact in plants. 'Functional Fibre' consists of isolated, non-digestible carbohydrates that have beneficial physiological effects in humans (*i.e*. those that might be used as supplements or ingredients). 'Total Fibre' is the sum of 'Dietary Fibre' and 'Functional Fibre'.

A substantial amount of evidence was reviewed by the expert committee, and they concluded that the different physiological effects have substantial health benefits. For example, the soluble fibres may delay the gastric emptying of ingested foods into the small intestine, resulting in a sensation of fullness, which may contribute to weight control. Additionally, these fibres may reduce postprandial blood glucose concentrations and potentially have a beneficial effect on insulin sensitivity. Evidence on the relationship of fibre intake with colon cancer is not currently consistent enough to link high intakes with reduced cancer risk. However, the soluble fibres can interfere with the absorption of dietary fat and cholesterol, as well as with the enterohepatic recirculation of cholesterol and bile acids, which may result in reduced blood cholesterol concentrations. These observations, along with an acknowledgement that the inclusion of dietary and some functional fibres in the diet improves faecal bulk and laxation, led to the panel setting AI values for total fibre in foods (FNB 2005; see Table 10). For young men and women, the AI is set at 38 and 25 g/day, respectively, based on the intake level observed to protect against CHD (14 g per 1000 kcal). These recommendations are double the amount of fibre Americans currently consume; American men eat 16.5–17.9 g/day of fibre, and women consume 12.1–13.8 g/day of fibre (as determined by AOAC methodology).

Table 10 Dietary fibre recommendations in the USA and Canada

	Adequate intake (g/day)		Adequate intake (g/day)
Males		Females	
1–3 years	19	1–3 years	19
4–8 years	25	4–8 years	25
9–13 years	31	9–13 years	26
14–18 years	38	14–18 years	26
19–30 years	38	19–30 years	25
31–50 years	38	31–50 years	25
51–70 years	30	51–70 years	21
71+ years	30	71+ years	21

Source: FNB (2005).

It is worth noting that these figures are in some instances substantially higher than recommended intake levels in other developed countries, especially for children. Currently, there are no specific recommendations for children in the UK; while derivation of recommendations requires the existence of relevant data sets, absence of specific recommendations can hamper dietary assessment and the provision of dietary advice. Within the scientific press, there has been considerable debate about the new US/Canada recommendations. The general opinion is that they are rather high, although unlikely to be detrimental to health, provided that they are met via a varied and healthy diet. However, the especially high values for children may make it difficult for them to meet energy requirements.

Netherlands

At the time of completion of this report, the most recent country to adopt new guidelines on dietary fibre intake was The Netherlands (HCN 2006). The committee chose not to set a dietary reference intake for fibre, instead opting to issue a guideline based on the importance of fibre for intestinal function and its protective effect against CHD. The extent of the literature review was set very much in the context of the USA/Canada Institute of Medicine guidelines, assessing the relevance of the findings to the Dutch population. Despite a degree of uncertainty regarding a potential level of intake for optimal health, the committee agreed that the American

value of 3.4-g dietary fibre per megajoule (14 g per 1000 kcal) was also a suitable guideline for adults and children over the age of 1 year in The Netherlands, provided that this was achieved via a mixed diet. These recommendations are higher than the amount of fibre that the Dutch population currently consumes, on average 2.2 g dietary fibre per megajoule.

European Union

Despite increasing centralisation of European policies, dietary recommendations are still made by individual member states (Table 11). However, some dietary advice is given by European-wide information councils and by pan-European expert committees. The EURODIET project was initiated in 1998, with the aim to contribute towards a co-ordinated EU health-promotion programme on nutrition, diet and healthy lifestyles. The project was supported by the European Commission and entailed a 2-year process of scientific consultation, evaluation and debate. Using the evidence available at the time, the committee set a population target of at least 25 g of dietary fibre (specific type undefined) per day (Kafatos & Codrington 2000). This value is consistent with the dietary fibre (AOAC) recommendation in the FAO/WHO report from 1998, and is based on an association between high-fibre diets and the prevention and management of weight gain, obesity and diabetes. The committee also noted that high-fibre diets were likely to help prevent heart disease, stroke and some cancers.

Table 11 Recommendations for fibre intake around the world

Country	Recommendation
Worldwide (FAO/WHO)	>20 g (NSP); >25 g (AOAC)
Europe (EURODIET)	>25 g (Dietary fibre – unspecified)
Denmark	25–30 g (AOAC)
Finland/Sweden/Norway	25–35 g (AOAC)
France	25–30 g (Dietary fibre – unspecified)
Germany	30 g (Dietary fibre – unspecified)
Ireland	18 g (NSP)
Netherlands	30–40 g (Dietary fibre – unspecified)
Spain	30 g (AOAC)
Sweden	25–35 g (Dietary fibre – unspecified)
UK	18 g (NSP)
USA/Canada	38 g (men 19–50 years); 31 g (men 50+ years) (AOAC)
	25 g (women 19–50 years); 21 g (women 50+ years) (AOAC)
Australia/New Zealand	30 g (men); 25 g (women) (Dietary fibre – unspecified)
Japan	20–30 g (AOAC)
South Africa	30–40 g (AOAC)

AOAC 985.29/AOAC 911.43 – total dietary fibre in foods; NSP, non-starch polysaccharide (Englyst method).
AOAC, Association of Official Analytical Chemists; FAO/WHO, Food and Agricultural Organisation/World Health Organization.

Around the world

Recommendations vary around the world, in terms of both the absolute amount of dietary fibre and the way in which the dietary fibre is defined. Table 11 attempts to summarise the published recommendations in the developed world.

Summation of the available evidence on fibre and health

In light of the recently published guidelines in the USA/Canada, Australia/New Zealand, and The Netherlands, and the associated reviews of the latest evidence on fibre and health, some have questioned whether the UK recommendation for dietary fibre intake is due a revision. This section summarises the evidence associating dietary fibre, and its individual components, with specific health benefits, and draws conclusions in the context of future recommendations for fibre intake in the UK.

Methodological approach taken

There is an overwhelming amount of published literature on the health benefits of fibre in the human diet. In order to produce a summary of the evidence in a reasonable time frame, an approach was adopted that focused on ensuring that the most physiologically relevant studies were identified and assessed. The basis of this review was the documentation produced by the FNB (US/Canada), the Health Council of the Netherlands, and FSANZ (Australia/New Zealand) to accompany the publication of their dietary guidelines (FNB 2005; HCN 2006; NHMRC 2006). Also considered were clinical trials investigating the effect of fibre on health, which were identified by a computerised literature search of articles published between 1982 and 2006 and indexed on MEDLINE. As an important aspect of this review was to assess the impact of including dietary fibre components, such as resistant starch and non-digestible oligosaccharides, as well as the more traditionally considered fibre fractions, special attention was paid to ensuring that studies of the most relevance to human consumption patterns were considered. Two recent reviews were used for this purpose (Nugent 2005; Pool-Zobel 2005), along with the findings from studies

identified from additional MEDLINE searches to ensure that the data available were as up to date as possible.

Dietary fibre and hyperlipidaemia, hypertension and coronary heart disease

There are almost 100 published studies investigating the association between dietary fibre and cardiovascular disease risk. Such trials have included large-scale prospective cohorts that have observed the occurrence of cardiovascular events in populations all over the world, as well as smaller-scale intervention studies measuring cardiovascular disease risk factors. Individuals consuming high-fibre diets (13–14.7 g/1000 kcal) have a substantially (15–59%) reduced risk of developing CHD than those consuming the lowest amounts of fibre (3–6 g/1000 kcal). Several large-scale cohorts have estimated the effect of increasing fibre intake on CHD risk. An increase in intake of 6 g/day of dietary fibre is associated with a 33% and 24% reduction in risk of CHD in women and men, respectively (Khaw & Barrett-Connor 1987). A similar study reported a 19% decrease when fibre intake was increased by 10 g/day (Rimm *et al*. 1996). A pooled analysis of ten prospective cohort studies reported a 14% decrease in the risk of CHD for each 10 g/day increase in dietary fibre intake (Pereira *et al*. 2004). Intervention trials have also demonstrated the effectiveness of dietary fibre in modifying blood lipid concentrations. Trials investigating total dietary fibre have had inconclusive results, as many studies have been assessing the impact of a dietary pattern (*i.e*. low fat, high fibre), rather than purely assessing the effect of a high-fibre diet, which makes interpretation of the data more complicated. However, several trials using oat bran or oatmeal as an intervention have had results that are more consistent. In fact, it has been calculated that consumption of at least 3-g soluble fibre from oats per day lowers blood cholesterol levels by 0.13 mmol/L in people whose blood cholesterol levels were less than 5.9 mmol/L (*i.e*. only moderately elevated) (Ripsin *et al*. 1992). Similar cholesterol-lowering effects have been reported when other viscous fibres, such as guar gums, pectin and psyllium, are fed (Brown *et al*. 1999).

There is now emerging evidence that the type or source of dietary fibre is especially relevant in terms of CHD prevention. A recent review by Flight and Clifton (2006) has demonstrated the importance of cereal grains in reducing CHD risk. Of course, when the wholegrain is considered, mechanisms involving the other components of wholegrains (*i.e*. magnesium, folate or vitamin E) cannot be discounted and, indeed, the exact mechanism may never be fully understood.

Mechanisms Viscous, soluble fibres, such as gums, psyllium and beta-glucan, are believed to reduce cholesterol concentrations by altering cholesterol and bile acid absorption, and by effects on hepatic lipoprotein production and cholesterol synthesis. However, as suggested earlier, the reduction in CHD risk may be a complex interplay between other components present in plant foods.

Conclusions and recommendations There is a strong relationship between the amount, and type, of dietary fibre (notably soluble fibres in oats and legumes) in the diet and the risk of developing CHD. It thus seems prudent to set a dietary recommendation at an intake level that has been demonstrated to have a significant impact on risk of developing the disease in large cohort studies. On this basis, population recommendations should be in the range of 13–15 g/1000 kcal (*i.e*. 26–30 g of fibre per day for a woman consuming 2000 kcal per day, and more for men). This is the value used as the basis of the recommendations published in the USA/Canada and more recently in The Netherlands.

Dietary fibre and gastrointestinal health

Proper functioning of the gastrointestinal tract can be assessed by considering faecal weight and transit time (*i.e*. the amount of time it takes for material to pass through the gastrointestinal tract). A meta-analysis of 100 studies investigating the effect of different forms of fibre and changes in stool weight was conducted by Cummings in 1993. The findings of this meta-analysis have been used to assess the impact of increasing dietary fibre intakes on bowel function. An increase of 1-g faecal bulk can be achieved by 3-g cellulose, 5.4-g wheatbran, 1.3-g pectin, or 4.9-g fruits and vegetables (Cummings 1993). Cummings *et al*. (1992) have taken this approach one step further and derived an algorithm for calculating faecal weight: faecal weight = $4.9 \times$ dietary fibre intake + 35. It is known that, if faecal weight falls below 160–200 g/day, there is a strong association between weight of faeces and the time it takes to pass through the digestive tract. However, provided faecal weight exceeds 160–200 g/day, passage through the intestines will take less than 48 hours (Spiller 1993). Thus, using the algorithm of Cummings *et al*. (1992), 26–34 g of fibre would be required to produce a stool output of 160–200 g/day. Others have attempted to calculate the amount of fibre required in the diet to achieve optimal stool output and have published higher values: 32–40 g/day (Monro 2004) and 35–45 g/day (Spiller *et al*. 1986). However, not all forms of dietary fibre

bring about an increase faecal bulk – beta-glucan and gums are notable exceptions, and there are other factors which may affect faecal bulk or transit time that are independent of dietary fibre intake, such as physical activity and the amount of fluids consumed (Anti *et al.* 1998).

Conclusions and recommendations Certain types of dietary fibre increase the speed at which food passes through the gastrointestinal tract and reduce the risk of developing constipation and other gastrointestinal disorders. Although adequate fluid intake and an active lifestyle are also important, historically the effect of fibre on stool bulk and transit time has formed the basis of most dietary fibre recommendations around the world. Estimations to achieve an optimal faecal output are varied, although those published by Monro (2004) and Spiller *et al.* (1986) are in line with each other. Thus, on the basis of the evidence summarised here, an intake around 32–40 g/day of total dietary fibre would seem to convey gastrointestinal health benefits that may not be achievable with lower intakes.

Dietary fibre and glucose tolerance and insulin response

Dietary fibre may attenuate the insulin response to a meal by delaying glucose uptake. Viscous fibres, such as guar gum, beta-glucan and pectins, appear to significantly reduce the glucose response to a meal (Wolever & Jenkins 1993), so may be worthwhile including in the diets on individuals looking to moderate the glycaemic response. However, for the healthy population, this may not be so physiologically relevant. Resistant starch has similarly been shown to have a beneficial effect on the glycaemic response to a meal (Nugent 2005).

With respect to development of diabetes, individuals consuming diets with a high GL (165) and a low intake of cereal fibre (<2.5 g/day) are at a twofold increased risk of developing type 2 diabetes, compared with those consuming more than 5.8 g of cereal fibre per day and a low-GL (<143) diet (Salmerón *et al.* 1997a, 1997b). A similar association was reported in a large cohort of middle-aged women (Schulze *et al.* 2004).

Conclusion and recommendations It has been suggested that dietary fibre, especially when this includes fibre from wholegrain products, can protect against type 2 diabetes. However, despite the huge interest in this area, and the large number of studies available investigating this effect, there is no conclusive evidence to suggest that a dietary recommendation for total dietary fibre should be based on attenuated glucose response. It has also been noted that, even if the evidence were sufficient to warrant a recommendation based on prevention of type 2 diabetes, this would not result in a higher value being set than that derived from data associating fibre intake with CHD risk (FNB 2005; HCN 2006).

Dietary fibre and satiety and weight management

Overweight [body mass index (BMI) 25–30 kg/m^2] and obesity (BMI above 30 kg/m^2) are more commonly observed in individuals with low fibre intakes (20.9 ± 1.8 g), compared with those individuals with higher intakes of dietary fibre (26.9 ± 1.8 g; Miller *et al.* 1994). Similarly, an association has been reported between higher dietary fibre intakes and a lower body fat percentage (Smith 1987). However, the evidence to show a protective effect of fibre against weight gain is not as conclusive. After 8 years of follow-up, weight gain in individuals with the lowest quintile of fibre intake was 1.4 kg, compared with 0.39 kg in the highest quintile group (Koh-Banerjee *et al.* 2004). Thus, although it appears that a high-fibre diet is an important factor in preventing weight gain, it is not currently possible to specify an effective level (HCN 2006).

It has long been considered that dietary fibre has an effect on the modulation of appetite (see Levine & Billington 1994). The satiating effect of a diet high in dietary fibre is possibly owing to the attenuation of the blood glucose response and slower glucose absorption after a meal (Holt *et al.* 1992). A task force report produced by the British Nutrition Foundation (BNF 1990), entitled *Complex Carbohydrates in Food,* concluded that foods rich in non-starch polysaccharide are useful in weight reduction because of the satiating effect of fibre, but also because foods high in fibre tend to be lower in fat (and thus energy) and may also take longer to chew. Soluble fibres, such as those found in oats, delay gastric emptying, which results in a feeling of fullness and delays the absorption of glucose and other nutrients (Roberfroid 1993). Similarly, consumption of wholegrains has been associated with reduced weight gain, although this is questionable (Koh-Banerjee & Rimm 2003). There is no clear mechanism to explain this observation, and it may simply be the confounding effects of the healthier lifestyle pattern associated with diets rich in wholegrains.

Intervention studies investigating the effect of high-fibre meals on subsequent energy intake throughout the day have also had mixed findings. To have an effect, large amounts of fibre have to be included in the meal; for example, 29-g fibre from sugar beet included in the breakfast meal resulted in a 14% decrease in energy

intake at lunchtime (Burley *et al*. 1993). Despite this being a significant decrease in energy intake, this is not necessarily a practical eating pattern for the whole population; thus fibre consumed at current levels is unlikely to have an effect on overall energy intake. Indeed, it has also been demonstrated that weight loss resulting from a high-fibre diet is also rarely sustained (Heaton 1973).

Conclusions and recommendations Intakes of dietary fibre appear to be negatively related to BMI, suggestive of a role for fibre in weight management. However, very high amounts of fibre (approximately 30 g/meal) are required to reduce subsequent energy intake throughout the day. Thus, data are not currently consistent enough to set dietary recommendations for fibre intake based on weight management.

Although results look promising, no conclusions can be drawn about the specific types of dietary fibre. There are plausible mechanisms via which some soluble fibres might increase feelings of fullness, although it has still to be determined how relevant these effects are at a population level.

Dietary fibre and cancer

The possibility that dietary factors may modify the risk of developing cancers was first considered by early philosophers. However, it was not until the latter half of the 20th century that quantification of the contribution of potential factors was attempted. Observational studies comparing rates of cancers in different populations detected occupational and environmental factors associated with increased risk of developing cancer. Doll and Peto (1981) estimated that the involvement of dietary factors in the development of cancers at all sites was comparable to that of tobacco smoke. Colorectal cancer is the cancer for which the evidence of a dietary involvement is the strongest (Cummings & Bingham 1998). Cross-sectional epidemiological studies have attributed diet as a causal factor in approximately 80% of sporadic bowel cancers in the western world (Willett 1995).

Fruits and vegetables have been associated with a decreased risk of colorectal cancer. The odds ratio between the highest and lowest quintiles of vegetable consumption has been found to be 0.48 (95% CI 0.41–0.57) from a meta-analysis of six case–control studies (Trock *et al*. 1990). However, prospective studies have not confirmed an effect (Michels *et al*. 2000). Fruits and vegetables are a complex dietary exposure. They contain numerous compounds that have anticarcinogenic properties *in vitro* (*e.g.* carotenoids, folate, ascorbic acid, phenols, flavonoids, isothiocyanates and indoles) (WCRF 1997). They are also rich in fibre, which is believed to be one of the most important, yet also most controversial, factors in the prevention of colorectal cancer, and there are well-studied mechanisms that support this hypothesis.

A study identifying the effect of fibre from all food sources has reported that there is a 40% reduction in risk between the lowest (15 g/day) and the highest (35 g/day) quintiles of intake (Bingham *et al*. 2003). This finding was one of the results from the European Prospective Investigation into Cancer and Nutrition study, which is a large prospective cohort study of over half a million individuals across Europe with relatively heterogeneous dietary habits. However, a protective effect of a high-fibre diet has not been seen in cohorts in the USA, Finland and Sweden (Fuchs *et al*. 1999; Pietinen *et al*. 1999; Terry *et al*. 2001); indeed, some studies have even reported an increased risk on high-fibre diets (*e.g.* Potter & McMichael 1986). The subject is further complicated when trials looking at intermediate-stage biomarkers are considered. Large intervention trials have shown that supplements of soluble fibre, bran or vegetables have not reduced recurrence rates of adenomatous polyps, precursors to cancerous lesions, in patients (Alberts *et al*. 2000; Bonithon-Kopp *et al*. 2000; Schatzkin *et al*. 2000), and this has been confirmed by a Cochrane review (Asano & McLeod 2002).

Confusion may have arisen because the resistant starch content of the diet is often not taken into account. Although such international comparative studies are one of the weakest forms of epidemiological evidence, such studies show a greater correlation between colorectal cancer and starch intake than with total dietary fibre (Cassidy *et al*. 1994).

Mechanisms Fibre is thought to reduce the risk of colorectal cancer in a number of ways. It increases faecal bulk and reduces transit time through the large bowel, which has the effect of diluting genotoxins in the stool and reducing the amount of time that the contents of the faecal stream are in contact with the colonic mucosa. This limits the potential amount of DNA damage (Hu *et al*. 2002). Fibre, as non-starch polysaccharides and resistant starch, is also fermented by the anaerobic bacteria in the gut to SCFAs, including butyrate (Nordgaard *et al*. 1995). Butyrate is an anti-proliferative agent and has been shown to induce apoptosis (programmed cell death) in colon cell lines, so is therefore important for maintaining a healthy, functional epithelium (Blottiere *et al*. 2003; Pool-Zobel 2005). There is a substantial amount of animal data to suggest that fermentable fibres in the diet reduce cancer risk by reducing exposure to

genotoxic components in the gut, or by reducing their genotoxic impact. However, more trials are needed to determine whether lifelong supply of butyrogenic dietary fibres (including inulin-type fructans and resistant starch) may contribute substantially to colorectal cancer chemoprevention.

Conclusions and recommendations Detecting associations between diet and cancer is particularly difficult, owing to the amount of time between the dietary exposure and the development of the disease. Prospective cohort studies, where a group of individuals with known dietary patterns are followed until presentation of the disease, are exceptionally costly to conduct, and there are relatively few cohorts available to compare findings. Whenever associations between diet and cancer are reviewed, the evidence available is either too conflicting or inadequately understood to draw conclusions (FNB 2005; HCN 2006). Indeed, reports from both the WHO/FAO (2003) and the World Cancer Research Fund (WCRF 1997) concluded that, although an effect of fibre on cancer risk was possible, and that people with very low fibre intakes are at an increased risk of colorectal cancer, the data were insufficient to set a population recommendation. Instead, dietary advice to reduce the risk of developing cancer at all sites is based on following a varied, balanced diet with plenty of fruits and vegetables.

Adverse effects of dietary fibre

Several adverse health effects have been reported for individuals consuming very-high-fibre diets, and it is essential that these are considered when dietary recommendations are set. Of greatest concern is the potential for high-fibre diets to affect mineral bioavailability in the small intestine. Absorption of iron, calcium, magnesium and zinc has been identified as being especially susceptible to the presence of phytate in the bowel. However, the authors of a review of all available literature, conducted in 1995, stated that: 'there was no convincing scientific evidence that any dietary fibre, even when consumed in large amounts (*i.e.* over 50 g per day) has any effect on mineral absorption' (Gordon *et al.* 1995). Indeed, some have even suggested that the SCFAs, produced via the fermentation of non-digestible carbohydrates in the large bowel, lower the pH of the intestinal contents to such an extent that the absorption of these minerals is actually enhanced (see Nugent 2005).

Finally, many individuals who alter their diets to increase the amount of fibre experience gastrointestinal discomfort. This can present as flatulence, belching, bloating and stomach aches, brought about by the rapid fermentation of soluble fibres. Fibre components that are more slowly fermented produce gas at a rate which is absorbed into the bloodstream without discomfort and, in many cases, discomfort decreases as the microflora adapts to the dietary change.

Overall, a high intake of dietary fibre seems unlikely to produce substantial deleterious effects when part of a healthy diet. Indeed, it is worth bearing in mind that the health risks of a low-fibre diet are potentially much greater than those of a very-high-fibre diet, and the effects described above should not detract health professionals from promoting the importance of increasing the total fibre content of the diet.

Overall conclusions

Taking into account the evidence presented above, there is a persuasive argument for reassessing the recommendations for dietary fibre intakes in the UK, in particular taking into consideration the emerging evidence for non-digestible carbohydrates such as resistant starch and oligosaccharides. The current recommendation, to achieve an average population intake of 18 g non-starch polysaccharides per day, was set in 1991 and seems not to take account of developments in dietary fibre research over the past 15 years, which have been the basis of revisions to fibre recommendations in North America, Australia/New Zealand, and The Netherlands in recent years. Nevertheless, it is important to recognise that there is a limit to the effect of a high-fibre diet with respect to cholesterol-lowering properties, cancer protection, bowel health or glycaemic control, so that these health benefits alone cannot justify very high intakes. Other factors are known to affect an individual's risk of developing these diseases, some of which will not be ameliorated by a high-fibre diet.

Another aspect to take into consideration is that total dietary fibre intakes in the UK are already well below the 1991 recommended level; simply setting higher recommended levels is not going to solve the problem of these relatively low intakes. More emphasis needs to be given to the likely benefits of increasing dietary fibre intake achieved by including a greater number and range of fibre-rich (and essential nutrient dense) foods within dietary patterns adopted in the UK (see practical examples below).

Through innovation by the food industry, one option is to develop a wider range of foods that include non-digestible carbohydrates as ingredients. However, further research is needed to establish whether added fibre

ingredients are as effective as whole food. There is currently a lack of evidence for consistent beneficial effects of fibre supplements, which suggests that indigestibility alone does not automatically equate to health benefits (Wasan & Goodlad 1996; Goodlad & Englyst 2001).

Furthermore, with nutrition and health claim legislation about to be regulated at a European level, there will be a need for harmonisation across Europe regarding definitions of fibre and analysis methodology. The UK is likely to have to fall in line with other member states in adopting the AOAC method of analysis as the norm, and this will mean that the current DRV of 18 g (based on Englyst methodology) will not be synchronised with the information on fibre presented on food labels, which will be misleading for consumers. Given that, in COMA's view (DH 1991), the Englyst methodology is most closely aligned with evidence for health benefits, one approach would be simply to extrapolate from the current value of 18 g [as has already been conducted by the Government's Scientific Advisory Committee on Nutrition (SACN), see later] to bring the value roughly in line with AOAC methodology (*i.e.* raise the DRV to 24 g/day for adults). But this approach fails to take account the higher levels of dietary fibre required to bring about heart health protection that has been instrumental in the development of the reference values now in use elsewhere, as described earlier.

Table 12 illustrates the values that would be achieved if the North American approach based on a recommended daily intake of 14 g dietary fibre (AOAC) per 1000 kcal were to be applied using current UK energy recommendations for different adult age groups. Values have only been tabulated for adults, because the values derived using this approach for children published in the USA are now widely considered to be on the high side. Because active teenagers can have very high energy requirements, derivation of fibre requirements on a purely energy basis results in values as high as 39 g/day and 30 g/day, respectively, for boys and girls aged 15–18 years. Furthermore, application of the approach for children as young as 4–6 years yields a value that is consistent with current recommendations for adults in the UK, and would require a dietary pattern so bulky that it might be likely to have an adverse impact on a child's ability to acquire sufficient energy and micronutrients. The scientific basis of the approach shown in Table 12 would be to help ensure gastrointestinal health (increased stool weights and reduced transit times), as well as deliver cardiovascular benefits.

Table 12 An illustration featuring the application of the North American approach to derivation of fibre recommendations using UK energy recommendations

	Dietary reference value (g/day)		Dietary reference value (g/day)
Males		Females	
19–50 years	36	19–50 years	27
51–59 years	36	51–59 years	27
60–64 years	33	60–64 years	27
65–74 years	33	65–74 years	27
75+ years	29	75+ years	25

Figures derived from dietary reference values for energy (EAR; DH 1991). It should be noted that UK energy values are currently under review and revisions are expected in 2007, which are believed to be taking account of activity level.

EAR, estimated average requirement.

Use of the 14 g/1000 kcal value relates to inclusion of all fibre components measured using the AOAC 985.29/AOAC 991.43 methodology (see Table 5), which represents a diversity of compounds, but still excludes some non-digestible starch. Currently, no comprehensive attempts have been made to identify recommendations for individual fractions of non-digestible carbohydrates, although some have speculated about possible values for individual components. To do this would require more detailed knowledge of unique physiological effects of individual fractions. Although it is clear that effects of the different fractions are not interchangeable, currently we do not have sufficient data from well-designed human intervention trials to attempt to make specific recommendations. However, some more detailed information is beginning to emerge. For example, soluble fermentable fibre components, such as inulin, have been shown to afford additional protection against CHD by effecting a reduction in blood cholesterol concentrations, as well as providing a substrate for fermentation in the large bowel which is believed to be important for healthy bowel function (Pool-Zobel 2005).

Also, Australia's CSIRO has recommended that total intakes of resistant starch should be around 20 g of resistant starch a day based on a study by Baghurst *et al.* (2001). However, compared with current intakes of resistant starch in the UK population and elsewhere, to achieve intakes at this level would require substantial dietary changes and, indeed, may only be reached by the consumption of foods containing resistant starches as a food ingredient, rather than in the natural form. However, resistant starch could make a valuable contribution to dietary fibre intakes, as it is fermented slowly in the large bowel and is therefore tolerated better than other soluble fibres.

In summary, therefore, a scientific rationale exists for reviewing dietary fibre recommendations, and such an

approach has already been adopted elsewhere. Although relevant evidence is now emerging, it would be premature to identify specific recommendations for subfractions of non-digestible carbohydrates. But an approach that takes the health effects of fractions not captured in the Englyst method is probably overdue. However, it is important not to lose sight of the fact that average intakes in the UK remain well below the current recommendation of 18 g/day, and this in itself needs to be addressed through health-promotion activities.

With this in mind, there are some food-based dietary recommendations that could assist the population in making healthy choices to achieve a higher total fibre intake, as well as ensuring that different subfractions of fibre are included, so the population can benefit from the associated health attributes of such a diet.

The '5 a Day' message to promote fruit and vegetable consumption has had a favourable effect on fruit and vegetable intakes, although much of the increase has been seen in terms of more fruit portions (Defra 2006). Further clarification of whether there is a difference in the effect of fibre from fruit or vegetable sources may be required, as it is emerging that the insoluble fibre from cereals, legumes and vegetables may be of most relevance with respect to cardiovascular disease prevention (Flight & Clifton 2006). If '5 a Day' skews food choices towards increasing fibre from fruits, but not vegetables, perhaps a message more along the lines of that used in Australia (5 vegetables + 2 fruits) could be useful.

Wholegrain messages also have the potential to impact on the amount of fibre in the diet. Fibre provided by the wholegrain includes a substantial resistant starch component, as well as varying amounts of soluble and fermentable fibres, depending on the wholegrain source. Current advice from the Food Standards Agency (FSA) is to eat plenty of starchy foods and to choose wholegrain varieties, whereas in the USA, there is a specific recommendation to consume three 16 g servings of wholegrains each day. This provides 48 g wholegrains each day, which would contain approximately 8 g resistant starch, 2 g soluble fibre and 8 g of other fermentable fibres (using approximations from Bednar *et al*. 2001). However, the consumption patterns of wholegrains in the USA (mainly oats) are different from those in the UK (mainly wheat), so this will have implications for the amount of soluble and fermentable components present, as well as the proposed health benefits.

Finally, a food-based recommendation for beta-glucan soluble fibre in oats could also be of benefit in the UK population. There is an approved claim associating this type of soluble fibre with significant reductions in cholesterol concentrations that food manufacturers or retailers can use on products containing at least 0.75 g of beta-glucan soluble fibre per serving (see http://www.jhci.org.uk). This is based on a suggested intake of 3 g/day. As rolled oats contain at least 4% beta-glucan soluble fibre, a recommendation to consume two portions of oats/oat bran (roughly 75 g) per day would ensure an intake of beta-glucan of approximately 3 g per day; however, given current dietary patterns, few people are currently likely to consume this quantity of oats on a daily basis.

Practical advice for meeting the proposed recommendations

At a first glance, the prospect of increasing population intakes of dietary fibre from approximately 13 g/day of non-starch polysaccharide to these higher levels seems daunting. However, there are some useful suggestions that health professionals could take on board which could demonstrate that it is relatively easy to achieve an intake approaching 36 g total dietary fibre (AOAC) without making drastic changes to the diet. It is becoming increasingly clear that the various components of fibre contribute, to varying extents, to several health benefits. 'Traditional' insoluble fibres (*e.g.* cellulose found in wheatbran) help provide bulk to the contents of the gut, facilitating their passage through the gastrointestinal tract. Such fibre is resistant to fermentation by the bacteria in the large bowel and is not believed to effect any reduction in serum cholesterol concentrations. Conversely, the 'traditional' soluble fibres (*e.g.* beta-glucan found in oats) are potentially capable of holding more water than the insoluble components, and are rapidly fermented by the bacteria in the large bowel to produce SCFAs that are believed to be beneficial to health (*e.g.* propionic acid associated with blood cholesterol lowering). The gases produced during this rapid fermentation can cause gastrointestinal discomfort, especially if fibre intake is suddenly increased substantially over a short period, and so it is best to increase intake gradually. On the other hand, other 'insoluble' fibre components, such as resistant starches, are slowly fermented in the large bowel to produce SCFAs which can contribute to overall bowel health, and so carry less risk of gastrointestinal discomfort. Thus, it is clear that, in order to reap the most benefit to health, an individual's daily fibre intake should ideally include a variety of fibre types.

As yet, there is insufficient evidence to make specific recommendations for the exact amounts of these different fibre types, but the food combinations shown in

Table 13 Examples of food combinations that would result in total fibre intakes in the range 30-36 g/day

	Total AOAC Fibre (g)	Total NSP fibre	Insoluble NCP fibre	Soluble NCP fibre	Resistant starch
Muesli with milk (70 g)	3.9	3.9	1.8	0.9	2.2
Orange juice, tea or coffee (50 ml)	0.1	0.1	0.1	0.1	n/a
Baked beans (150 g)	7.0	5.3	0.9	3.2	2.9
on wholemeal toast (70 g) with grated cheese	5.4	4.1	2.2	1.1	1.0
Apple (112 g)	2.7	2.0	0.4	0.8	n/a
Mixed nuts (40 g)	3.2	2.4	1.1	0.7	n/a
Baked potato (180 g) with tuna mayonnaise	6.5	4.9	0.7	2.7	n/a
Broccoli (85 g)	2.6	2.0	0.3	0.9	n/a
Tomato (85 g)	1.1	0.9	0.2	0.3	n/a
	33.6	**25.3**	**7.7**	**10.6**	**6.1**
	Total AOAC Fibre (g)	Total NSP fibre	Insoluble NCP fibre	Soluble NCP fibre	Resistant starch
Porridge with milk (160 g) and	1.7	1.3	0.5	0.8	0.3
stewed rhubarb (140 g)	5.0	3.8	2.2	0.7	n/a
Orange juice, tea or coffee (50 ml)	0.1	0.1	0.1	0.1	n/a
Pea soup (220 g)	4.7	3.6	0.5	1.1	2.8
RS enriched bread (70 g)	9.2	6.9	0.4	0.6	3.9
Banana (120 g)	1.8	1.3	0.1	2.0	7.4
Mixed dried fruit (50 g)	1.5	1.1	0.2	0.6	n/a
Grilled chicken with cold potato salad (180 g)	6.5	4.9	0.7	2.7	9.0
Mushrooms (40 g)	0.8	0.6	0.2	0.1	n/a
Tomato (85 g)	1.1	0.9	0.2	0.3	n/a
RS enriched bread (35 g)	4.6	3.4	0.2	0.3	1.8
Low fat fruit yoghurt	0.1	0.1	0.1	0.1	n/a
	37.0	**27.9**	**5.5**	**9.5**	**25.2**

Table 13 demonstrate how intakes of the different fibre components can contribute to a total dietary fibre intake in the range of 30–36 g of total fibre (AOAC) per day.

Current intake levels

The amount of digestible carbohydrate in the diet is often measured in dietary surveys, although the detail supplied differs depending on the country of interest. Most quote the total carbohydrate consumed, as well as the percentage energy obtained from carbohydrate. Some more sophisticated national dietary assessment programmes also report on sugars and starch intake. Many countries in Africa, Asia and North America calculate carbohydrate 'by difference', which is subject to inherent inaccuracies as a result of the number of estimates that are required to calculate these values. The by-difference method also includes carbohydrates that are resistant to digestion in the gastrointestinal tract. Most countries in Europe and Oceania analyse carbohydrate directly and, therefore, values do not contain unavailable carbohydrate that is resistant to digestion (FAO 1997). Similar issues are at the forefront when intakes of dietary fibre are considered. The inconsistent approaches taken by governments and researchers around the world to define and measure dietary fibre has generated limited and often non-comparable data on global intake levels.

National dietary surveys

United Kingdom

The National Diet and Nutrition Survey (2000–2001) was conducted on a sample of adults aged 19 to 64 years, and was one of a programme of national surveys which aims to gather information about the dietary habits and nutritional status of the British population. This survey was published in 2002–2003, and it reported that the average daily intake of carbohydrate

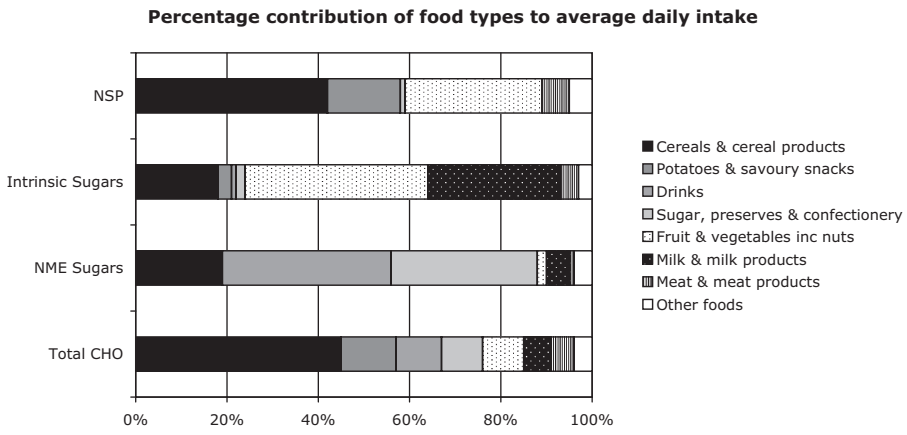

Figure 5 Contribution of the main food types to the average daily intake of carbohydrates in the adult's diet in the UK. CHO, carbohydrate; NME, non-milk extrinsic; NSP, non-starch polysaccharide.

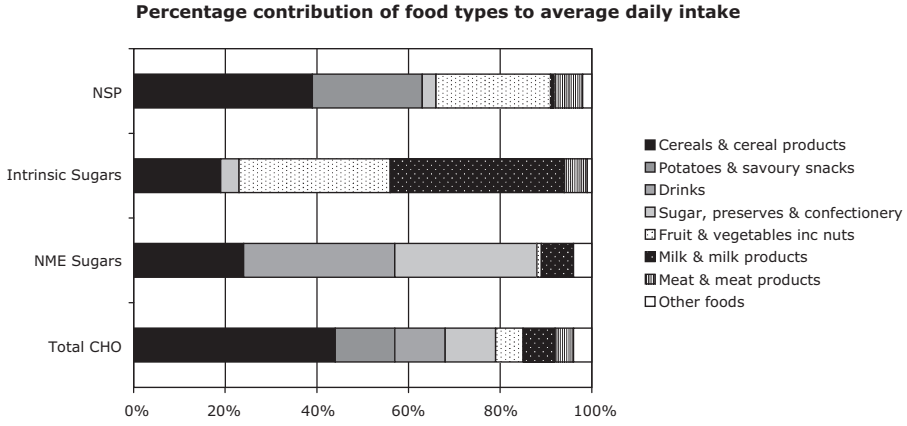

Figure 6 Contribution of the main food types to the average daily intake of carbohydrates in children's diets in the UK. CHO, carbohydrate; NME, non-milk extrinsic; NSP, non-starch polysaccharide.

was 275 g for men and 203 g for women (Henderson *et al.* 2002). This represented 47.7% of food energy intake for men and 48.5% of food energy for women.

As can be seen from Figure 5, cereal and cereal products were the main source of carbohydrate for adults in Britain. An earlier study carried out among children aged 4–18 years reported that boys were consuming 260 g of carbohydrate per day (51.6% energy) and girls 214 g (51.1%) (Gregory *et al.* 2000) (Fig. 6).

The average intake of NMES in men was 13.6% of food energy, while in women, it provided 11.9% of food energy. Bearing in mind that the population target is 11% (DH 1991), these values are slightly higher than desired. NMES provided, on average, 16.7% and 16.4% of food energy for boys and girls, respectively (Gregory *et al.* 2000). The main sources of NMES among children were carbonated soft drinks and chocolate confectionery, with the proportion provided by carbonated soft drinks increasing with age.

The mean daily intake of intrinsic and milk sugars was 39 g for men and 37 g for women, with fruits and nuts, and milk and milk products, accounting for the majority.

Intrinsic sugars, milk sugars and starch should provide 39% of food energy for the population (DH 1991). On average, men obtain 34.9% and women 37% of food energy from this group (Henderson *et al.* 2003).

In the context of the National Diet and Nutrition Survey (NDNS), dietary fibre is measured as non-starch polysaccharide (*i.e.* all non-alpha-glucan polysaccharides) present, as measured by the technique of Englyst and Cummings (1988). For reference, the Department of Health recommendation (DH 1991) is to consume 18 g non-starch polysaccharide per day (individual range 12–24 g). In 2003, it was reported that the mean daily intake was 15.2 g among men and 12.6 g among women (data collected in 2000–2001). All gender- and age-groups had a mean intake of less than 18 g per day, and a third of men and half of women had intakes below 12 g per day. Likewise, intakes of non-starch polysaccharide in children were also below the recommendations for adults – boys consumed on average 11.2 g per day and girls consumed 9.7 g. Intakes of subgroups are displayed in Table 14.

The NDNS also collects socio-economic data and is able to report on intakes in households in receipt of ben-

Table 14 Dietary fibre (NSP) intakes in the UK

	Mean intake (g/day)	DRV (g/day)		Mean intake (g/day)	DRV (g/day)
Males			Females		
1.5–2.5 years†	5.0	n/a	1.5–2.5 years†	5.0	n/a
2.5–3.5 years†	6.0	n/a	2.5–3.5 years†	6.0	n/a
3.5–4.5 years†	7.0	n/a	3.5–4.5 years†	6.0	n/a
4–6 years‡	9.1	n/a	4–6 years‡	8.0	n/a
7–10 years‡	10.3	n/a	7–10 years‡	9.8	n/a
11–14 years‡	11.6	n/a	11–14 years‡	10.2	n/a
15–18 years‡	13.3	n/a	15–18 years‡	10.6	n/a
19–24 years§	12.3	18*	19–24 years§	10.6	18*
25–34 years§	14.6	18*	25–34 years§	11.6	18*
35–49 years§	15.7	18*	35–49 years§	12.8	18*
50–64 years§	16.4	18*	50–64 years§	14.0	18*
65–74 years¶	13.8	18*	65–74 years¶	11.6	18*
75–84 years¶	13.1	18*	75–84 years¶	10.4	18*
85+ years¶	11.9	18*	85+ years¶	10.0	18*

Source: †Gregory *et al.* (1995); ‡Gregory *et al.* (2000); §Henderson *et al.* (2002); ¶Finch *et al.* (1998). NB: There is some overlap between the National Survey of Infants (1.5–4.5 years) and the National Survey for Children (4–18 years).

*Individual range recommended to be 12–24 g/day for adults. No official recommendation exists for children (DH 1991).

DRV, dietary reference value; NSP, non-starch polysaccharide.

efits. These households had significantly lower intakes of non-starch polysaccharide, although there were no differences in intakes of total carbohydrates or NMES, which again were higher than recommended.

A second method of collecting UK nutrient intake data is via the Expenditure and Food Survey (EFS), conducted by the Department of the Environment Food and Rural Affairs (Defra). This survey collects food diaries from households across the UK. The EFS began in April 2001, replacing the National Food Survey (which began more than 50 years ago). An advantage of the EFS is that it measures purchases of food at the household level, rather than consumption, which reduces the risk of under-reporting, a common problem in all surveys. Purchases of all food entering the home are recorded over a 2-week period, and all household members above the age of 7 years complete a 2-week expenditure diary. The EFS does not make allowances for wastage or spoilage of edible food and drink components in its estimate of purchases, but this is taken into account when average nutrient intakes are calculated and compared with reference nutrient intakes, using an approximate figure of 10% for wastage of all types of food and drink. The report 'Family Food' is the source of detailed statistical information on purchased quantities, expenditure and nutrient intakes, derived from both household and eating-out food and drink. Energy and nutrient intakes are calculated using standard profiles for each type of food.

The most recent Family Food analysis reported an average intake of carbohydrate from household food and drink of 257 g per person per day (Defra 2006). As the survey has been running in some form for more than 50 years, trends have been observed; since 1994, there has been an overall 4.7% fall in the average intake of carbohydrate (g/day), but there has been little change in the percentage of food energy intake derived from carbohydrate (MAFF 1990). The average intake of NMES from household food and drink is 80 g per person per day. This equates to 15% of food energy intake, which is above the 11% recommendation made by COMA in 1991 (DH 1991). However, compared with the survey carried out the previous year, there was a slight increase during 2004–2005 in the average intake of fibre to 13.2 g per person per day (expressed as non-starch polysaccharides) from total food, but there is no clear trend over time.

European Union

Intakes of dietary fibre across Europe were published in 2000 during the process of devising a framework for food-based dietary guidelines in the EU. Fibre intakes, as determined by the AOAC method, varied widely both within countries and between countries (Gibney 2000). Figure 7 displays the range of intakes for eight member states, alongside the nutrient-based recommendation for fibre (25–38 g of total fibre per day).

United States of America

The major tool for dietary assessment in the USA is the National Health and Nutrition Examination Survey (NHANES). NHANES is a programme of studies designed to assess the health and nutritional status of adults and children in the USA. Nutrient intake, compared with the recommended intake level, is published as a report entitled '*What we Eat in America*'. The most recent report (Moshfegh *et al*. 2005) analysed data collected in 2001–2002 and reported values for total dietary fibre displayed in Table 15. As is immediately apparent from these data, intakes of dietary fibre in the USA are inadequate. Intakes of total carbohydrates, starch and sugars show a similar trend to those in the UK.

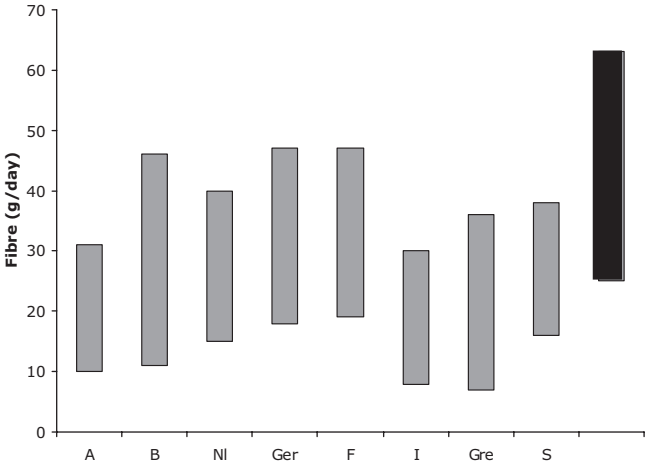

Figure 7 Range of intakes (mean of highest to mean of lowest quartiles/tertiles) of fibre (g/day) in relation to recommendation (black box). A, Austria; B, Belgium; Nl, Netherlands; Ger, Germany; F, Finland; I, Ireland; Gre, Greece; S, Spain. Source: Gibney (2000).

Components of dietary fibre

The degree of detail provided by national dietary surveys is constrained by the sheer quantity of analysis that is required. As a result, intakes of carbohydrates are expressed in a relatively simple format. Often, this is sufficient to give an insight into where effort needs to be focused to move closer to the population recommendations. However, often more information is required about the specific components of dietary fibre.

For the purpose of this report, an attempt has been made to estimate the amounts of the various components of dietary fibre in the UK diet. Using a model devised for the BNF Taskforce report (1990) entitled '*Complex Carbohydrates in Food*', household intakes of starch, resistant starch, cellulose, soluble non-cellulosic polysaccharides, insoluble non-cellulosic polysaccharides, non-starch polysaccharides and unavailable carbohydrate have been calculated (see Table 16 for explanation of these terms). This model used data from the National Food Survey (MAFF 1988b), combined with estimates of the various fractions of carbohydrates from Southgate *et al*. (1978) and Englyst and Cummings (1988). The intake data have been updated using the most recent EFS (Defra 2006), and the same composition values have been applied (Table 16).

Although not without inaccuracies, this method of estimating dietary fibre intakes produces values in line with estimations from smaller, experimental studies. Additionally, the estimated value for total non-starch polysaccharide intake (13.08 g) is in line with the figure quoted in the 2005 Family Food report (13.2 g) (Defra 2006).

Resistant starch

Several studies have attempted to quantify population dietary intakes of resistant starch. However, a number of

Table 15 Dietary fibre intakes in the USA

	Mean intake (g/day)	Adequate intake (g/day)		Mean intake (g/day)	Adequate intake (g/day)
Males			Females		
1–3 years	9.5	19	1–3 years	9.5	19
4–8 years	11.6	25	4–8 years	11.6	25
9–13 years	14.2	31	9–13 years	12.3	26
14–18 years	15.3	38	14–18 years	11.7	26
19–30 years	17.2	38	19–30 years	13.5	25
31–50 years	18.6	38	31–50 years	14.1	25
51–70 years	17.8	30	51–70 years	15.4	21
71+ years	16.9	30	71+ years	13.7	21

Source: Moshfegh *et al*. (2005) – see comments about level of adequate intake in recommendations section.

Table 16 Daily household intakes of major components of dietary fibre in the UK in 1988 and 2005

	Amount (g)	Starch (g)	RS (g)	Cellulose (g)	Insoluble NCP (g)	Soluble NCP (g)	NSP (g)	Unavailable CHO (g)
1988								
Dairy products	244	0.1	0	0	0.	0	0	0.1
Meat & meat products	131	4.5	n/a	0.1	0.1	0.1	0.2	0.7
Fish & fish products	18	0.8	n/a	0	0	0	0	0
Eggs, fats, sugars & preserves	86	0.2	0	0	0	0	0	0.1
Potatoes	130	18.6	n/a	0.5	0.1	0.7	1.3	1.8
Other fresh veg	60	0.4	n/a	0.5	0.2	0.8	1.2	1.9
Processed veg	69	10.2	n/a	0.9	0.4	1.0	2.1	4.0
Fruit & fruit products	114	0.4	0.2	0.4	0.3	0.6	1.3	1.8
Bread & flour	121	65.8	1.0	0.4	1.6	1.5	3.4	6.9
Cakes & biscuits	27	13.1	0.1	0.1	0.2	0.3	0.6	1.1
Other cereal products	45	22.9	0.3	0.2	0.8	0.5	1.5	2.3
Other foods	40	1.7	n/a	0.1	0	0.1	0.2	0.3
Total		138.5	1.5	3.2	3.8	5.7	12.5	21.0
2005								
Dairy products	299	0.1	0	0.1	0	0	0.1	0.2
Meat & meat products	150	5.2	n/a	0.1	0.1	0	0.2	0.8
Fish & fish products	23	1.0	n/a	0	0.1	0	0	0.1
Eggs, fats, sugars & preserves	56	0.1	0	0	0	0	0	0
Potatoes	117	16.8	n/a	0.4	0.1	0.7	1.2	1.6
Other fresh veg	109	0.6	n/a	1.0	0.4	1.4	2.6	3.5
Processed veg	14	2.1	n/a	0.2	0.1	0.2	0.5	0.8
Fruit & fruit products	167	0.6	0.2	0.6	0.5	0.9	2.0	2.6
Bread & flour	107	58.2	0.9	0.3	1.4	1.3	3.0	6.1
Cakes & biscuits	42	20.0	0.2	0.2	0.4	0.5	1.0	1.6
Other cereal products	77	38.9	0.4	0.4	1.3	0.9	2.6	3.9
Total		143.5	1.8	3.2	4.3	5.9	13.1	21.2

Intake data from the Expenditure and Food Survey 2004–2005; Household Food Consumption and Expenditure 1988; RS, resistant starch; NCP, non-cellulose polysaccharides, excluding starch; CHO, carbohydrate; NSP, non-starch polysaccharide (cellulose, soluble and insoluble NCP); unavailable carbohydrate also includes resistant starch, some other starch and lignin. Model based on published composition information (Southgate *et al*. 1978 and Englyst and Cummings 1988).

different methods to analyse resistant starch content were used in these studies, and this makes any detailed comparisons between countries and/or studies difficult. Dietary intakes of resistant starch in the UK are estimated at 2.76 g/day (Tomlin & Read 1990). This is somewhat higher than the 1.78 g estimated from the data from the EFS. However, this difference is unsurprising. Nutrient intake data are estimated from information on household purchases, and will not take into account the changes in resistant starch content that are brought about as the foods are prepared for consumption.

Worldwide, dietary intakes of resistant starch vary considerably. It is estimated that intakes of resistant starch in developing countries with high starch consumption rates range from approximately 30 to 40 g/day (Baghurst *et al*. 2001). Intakes in the EU are thought to lie from 3 to 6 g/day (Dyssler & Hoffmann 1994), and are believed to range from 5 to 7 g/day in Australia (Baghurst *et al*. 2001). It should be noted that intakes of resistant starch in Australia are likely to be higher than in Europe, owing to the commercial availability of top-selling breads, baked goods and cereals that contain ingredients high in resistant starch. These intakes sit in the context of an approximate recommendation made by CSIRO of 20 g of resistant starch per day for good health.

Wholegrain

In the UK, more than 90% of adults are considered to be not eating enough wholegrain foods (Lang & Jebb 2003). The average intake of wholegrain foods has been estimated as 7 g per day (Thane *et al*. 2005). There is no specific recommendation for wholegrain in Britain, other than to increase consumption; the FSA encourages consumers to select wholegrain varieties, but no amount is specified. However, nutritionists in America recommend that at least three of the seven servings of grains

that should be eaten each day should be wholegrain. In this context, a portion of wholegrain has been designated as 16 g and is defined as:

- 1 cup (16 table spoons) of wholegrain cereal;
- 1 slice/small roll of wholemeal bread;
- 1 cup of brown rice;
- 1 cup of whole-wheat pasta.

Yet, despite this clear advice, wholegrain consumption in the USA is still far short of the recommendation of 48 g for good health. It is estimated that the average wholegrain intake in the USA is less than one serving (<16 g) per day (Moshfegh *et al*. 2005).

Inulin

As a result of the interest in inulin as a functional food ingredient, several studies have attempted to quantify dietary intakes. American diets provided, on average, 2.6 g of inulin and 2.5 g of other fructo-oligosaccharides. Intakes of these components varied by gender and age, ranging from 1.3 g for young children to 3.5 g for teenage boys and male adults (Moshfegh *et al*. 1999). Estimates of consumption in Europe are somewhat higher, with intakes ranging from 3 to 11 g/day (Van Loo *et al*. 1995).

Issues surrounding increased intakes

Fibre intakes in the developed world are much lower than the levels recommended for good health. There is therefore much scope to increase intake. In an ideal world, sufficient fibre could be obtained by individuals consuming the recommended five portions of fruits and vegetables per day – alone equivalent to up to 24 g of non-starch polysaccharide (BHF 2006). However, with population intakes of fruits and vegetables hovering around 2.5 portions per day (Henderson *et al*. 2002), in the short-term, this is impractical as it would require a huge change in eating patterns. In the past, high-fibre diets have been encouraged by advising the inclusion of wholemeal bread, dried fruits, pulses and legumes in the daily diet, but again this often requires a major adaptation to an individual's diet. An alternative approach is to increase the fibre content of commonly consumed foods. This could be achieved by adding high-fibre ingredients (*e.g.* bran or oats) to composite dishes. Another option is to incorporate non-digestible carbohydrates into manufactured foods. There are two clear schools of thought on this matter: some believe that the inclusion of material other than plant cell walls as dietary fibre has the potential to mislead the consumer, whose expectations are likely to be that dietary fibre labelling provides guidance towards the largely unrefined plant foods shown to be associated with health (Englyst & Englyst 2005); whereas others accept that these fibre supplements are capable of contributing to some of the health effects associated with high fibre intakes (*i.e.* improved bowel function and glycaemic response), and can therefore make a valuable contribution to the fibre content of the diet (Brennan 2005).

Labelling issues

The inclusion of additional fibre in food products raises some issues with regard to labelling. Other than the inconsistencies in the way that the fibre content of foods is measured, there are also variations in the amount of fibre considered to be essential for good health, and as the food industry becomes more global, this can lead to increased costs, as different product specifications and packaging are required in different countries. Here we take a brief look at some of the issues in the UK, and consider how the food industry and regulators have attempted to work together to provide consistent and accurate consumer information.

Guideline daily amounts

In the UK, current dietary recommendations are based on the Englyst method of measuring dietary fibre and so are expressed as non-starch polysaccharide. However, across the EU, the standard method used to derive values for food labels is the AOAC method. Food manufacturers in the UK are increasingly using the AOAC value in line with advice issued by relevant UK departments (MAFF/JFSSG 1999) and to be consistent with fibre declarations globally. However, as current dietary recommendations for fibre in the UK are expressed in terms of non-starch polysaccharides comparisons of information on food packaging with the dietary guidelines can be misleading, if the relevant conversion factor is not applied. This discrepancy will need to be addressed for clarity.

For a typical UK diet where fibre is obtained from a variety of sources, the fibre content determined using the AOAC method is approximately one-third higher than that obtained using Englyst non-starch polysaccharide values. The FSA has further developed understanding in this area, as a result of decisions made during the development of a nutrient profiling model for use as a method to categorise foods high in fat, added sugar or salt for the purpose of restricting food advertising to children. Fibre content is one of the positive attributes of foods

used in the model (see Buttriss 2007 for a brief summary). The FSA had to grapple with whether to use values obtained using either the Englyst or AOAC methods to express its recommendations for fibre in the model. The FSA asked SACN for advice on how best to assess the fibre content of these foods. SACN recommended to the FSA: 'to avoid additional costs to industry because of the practical difficulties associated with measuring non-starch polysaccharide fibre, the final model should be adjusted to allow fibre levels to be determined using the AOAC method of determination of dietary fibre, subject to a conversion factor 1.33. This is because the mean ratio of non-starch polysaccharide to AOAC fibre in foods is approximately 1:1.33 according to a study funded by the former Ministry of Agriculture, Fisheries and Food'. The variations are illustrated in Table 17.

In 2005, the Institute of Grocery Distribution (IGD) decided to review and extend its guideline daily amounts (GDAs) for energy, fat and saturates, for men and women, which it originally published in 1998 (IGD 1998). The purpose of these GDAs was to support information provided in the nutrition panel on food packaging, and they were designed to help consumers put nutrition information into context. The GDAs are an estimate of typical appropriate intake levels for subgroups, which are derived from the DRVs published in 1991 (DH 1991). They provide consumers with a single figure which can be used to gain an improved understanding of their daily consumption of nutrients within the context of a healthy balanced diet. Following industry and consumer research, the GDAs developed in 1998 were reviewed and extended by IGD to include a set of GDAs for men, for women, for 'adults' and for children, for the following nutrients: energy, fat, saturates, protein, carbohydrate, total sugars, salt and fibre (IGD 2005).

The decision about the value for fibre was initially problematic. However, in line with the advice published by the FSA (referred to above), IGD published the values shown in Table 18, which not only propose a value for fibre using the AOAC method but also suggest values for children, which were derived using an 'Age + 7.5' approach. The basis for this came from a practical approach used by the American Health Foundation when setting guidelines in 1994 (Williams 1995). This suggested an AOAC value of 'Age + 5' as a minimum and 'Age + 10' as a maximum. Thus, a decision was made by IGD to set the GDA on the mid-point between these two figures (*i.e.* 'Age + 7.5'). This value was divided by the conversion factor (1.33) to derive the non-starch polysaccharide values.

This extended range of GDAs is being used increasingly by retailers and manufacturers (both front and back of pack) to help consumers interpret nutrition labelling information. Some manufacturers are also using a GDA-type value for wholegrain, devised as a means of comparing the wholegrain content of their product with a value recommended for health (the 'GDA'). The basis behind the 'GDA' is that one serving is equivalent to 16 g of wholegrain, and the US recommendation is to consume three servings a day (*i.e.* 48 g/day) (USDA 2005). However, the use of a GDA in this instance could be misleading to consumers and is not being introduced consistently across the board. Also, a study underway in the UK is using 20 g as a serving,

Table 17 Analysis of major food groups for fibre (% dry matter)

Food group	NSP	AOAC	Ratio AOAC/NSP
Bread	4.5	5.9	1.31
Other cereals	3.8	4.8	1.26
Meat products	1.1	1.1	1.00
Green vegetables	25.2	30.2	1.20
Potatoes	6.1	7.6	1.25
Other vegetables	10.0	17.7	1.77
Canned vegetables	11.3	16.9	1.50
Fresh fruit	8.7	12.1	1.39
Fruit products	3.1	4.0	1.29
Nuts	6.9	9.2	1.33
		Overall mean ratio	1.33

AOAC, Association of Official Analytical Chemists; NSP, non-starch polysaccharide.

Table 18 Guideline daily amounts for fibre intake in the UK

		Children			
	Adults	4–6 years	7–10 years	11–14 years	15–18 years
Dietary fibre (NSP) (g)	18	9	12	15	18
Dietary fibre (AOAC) (g)	24	12	16	20	24

Source: IGD (2005).

AOAC, Association of Official Analytical Chemists; NSP, non-starch polysaccharide.

based on a slightly different interpretation of the available data (Seal 2006).

Nutrient content claims

If high-fibre ingredients are introduced into some manufactured foods, it is logical that some form of nutrient content or health claim will be made to highlight the additional benefits of these products over other similar foods. Currently, under UK legislation, a reference to the relative or absolute amount of a nutrient, outside the declarations made within a nutrient panel on the food packaging, is considered to be a nutrient claim, but an EU regulation on nutrition and health claims was adopted in October 2006 and is expected to come into force during the early months of 2007, bringing with it new definitions and criteria (see Buttriss 2007).

Under the new legislation, a 'nutrition claim' will mean any claim that states, suggests or implies that a food has particular beneficial nutritional properties owing to the energy (calorific value) it provides, provides at a reduced or increased rate, or does not provide; and the nutrients or other substances it contains, contains in reduced or increased proportions, or does not contain. Examples include high fibre, source of fibre, and low fat. Definitions are also provided for the terms 'health claim' and 'reduction of disease risk claim' (see below).

Nutrition-related claims on food packaging are used by consumers as quick and easy markers of the nutrient features of a product to support decisions when comparing products. Currently and under the new legislation that will shortly come into force, if a nutrient content claim is made, the 'Big 8' nutrient labelling declaration (*i.e.* energy, protein, carbohydrate, fat, saturates, sugars, fibre and sodium) must be used on the pack. All claims relating to nutrient content made on food labelling and in advertising in the UK are subject to the general provisions of the Food Safety Act 1990, which makes it an offence to 'falsely describe a food or to mislead as to its nature, substance or quality' (see BNF 2002).

Table 19 outlines the FSA's existing guidance on making nutrient content claims in relation to fibre content. Provided that these criteria are met, foods can be designated as a 'source of', 'high in', or contain 'increased' fibre. But these criteria are set to change under the forthcoming legislation, as described below.

The issue is further complicated by the fact that the FSA guidance relates to the non-starch polysaccharide content of foods. As also mentioned previously, food manufacturers are increasingly using values obtained by the AOAC method for labelling purposes. Consequently, a conversion factor is required to 'translate' the non-starch polysaccharide guidelines into more practical AOAC values. This is of benefit, as it allows such claims to be used for foods containing components of dietary fibre that are not measured by the Englyst method (*e.g.* resistant starch). In light of the advice from SACN to the FSA in the context of the FSA's nutrient profiling model, a value of 1.33 has been suggested for this purpose (see earlier).

It is anticipated that many of these discrepancies will be addressed when the new EU regulation comes into force. Table 20 outlines the scheduled criteria for nutrition claims for fibre. Additionally, the regulation allows for development by the European Food Safety Authority (EFSA) of nutrient profiles, which will govern whether or not a food can carry a nutrition or health claim (a period of up to 2 years after the regulation comes into

Table 19 FSA guidance on nutrient content claims

	Source	Increased	High
Fibre	Either >3 g per 100 g or at least 3 g in the amount of food likely to be eaten each day	>25% more than a similar food for which no claim is made	Either >6 g per 100 g or at least 6 g in the amount of food likely to be eaten each day

NB: Values quoted are NSP fibre.
FSA, Food Standards Agency; NSP, non-starch polysaccharide.

Table 20 EU guidelines on nutrition claims

	Source	Increased	High
Fibre	Either >3 g per 100 g or >1.5 g of fibre per 100 kcal	>30% more than a similar food for which no claim is made	Either >6 g per 100 g or >3 g of fibre per 100 kcal

NB: Values quoted are assumed to be AOAC fibre. (http://ec.europa.eu/food/food/labellingnutrition/claims/index_en.htm)
AOAC, Association of Official Analytical Chemists; EU, European Union.

force has been scheduled for their introduction). These nutrient profile criteria are expected to take into account the quantities present in a food of those nutrients for which average intakes are currently too high, such as fat, saturated fatty acids, and salt; they may also take account of nutrients that have positive health benefits, such as fibre. However, nutrition claims, such as those relating to an increased fibre content, will be allowable on foods where a single nutrient exceeds the profile criteria, provided that a statement about the level present in the food of the specific nutrient appears in close proximity to the nutrient claim.

Health claims

A nutrient content claim requires the consumer to have an appreciation of why it is beneficial to increase consumption of the particular nutrient that is highlighted. An extension to the concept of nutrient content (or nutrition) claims is the health claim. In this case, not only is the amount of the nutrient highlighted, but also the effect on health that consuming more of the nutrient would have. The term 'health claim' is open to a variety of interpretations.

Under the new EU regulation, a 'health claim' means any claim that states, suggests or implies that a relationship exists between a food category, a food or one of its constituents and health (*e.g.* fibre and regular bowel habit). Another category has also been defined, a 'reduction of disease risk claim', which means any health claim that states, suggests or implies that the consumption of a food category, a food or one of its constituents significantly reduces a risk factor in the development of a human disease (*e.g.* helps reduce blood cholesterol). Again, particular criteria govern the use of these claims (see Buttriss 2007 and, for the full text of the regulation, http://ec.europa.eu/food/food/labellingnutrition/claims/index_en.htm in particular Articles 10–15). Medicinal claims for foods (*i.e.* those that claim to treat, cure or prevent disease) are illegal in the UK and will remain so under the new legislation.

Although legislation is now imminent, in the absence of any formal legislation in the UK, the Joint Health Claims Initiative (JHCI) was set up a few years ago to provide effective consumer protection and consistency in the use of health claims in the UK (see http://www.jhci.org.uk). The JHCI is a joint venture involving consumer organisations, enforcement authorities and industry trade associations in the UK, which established a voluntary Code of Practice for health claims on food. The JHCI has approved the use of two claims relating to dietary fibre intake. In 2002, use of the health claim: 'People with a healthy heart tend to eat more whole grain foods as part of a healthy lifestyle' on foods containing 51% or more wholegrain ingredients by weight per serving, was approved. In this case, the term 'whole grain' refers to the major cereal grains, including wheat, rice, maize and oats. The committee also noted that the health impact of a diet containing wholegrain foods depends on the rest of the diet, as well as other lifestyle factors, such as physical activity; thus, the claim must be set within this context. Other countries have instigated their own procedures for the voluntary substantiation of health claims, for example the Food and Drug Administration (FDA) in the USA. The claim currently allowed by the FDA goes further than the JHCI-permitted claim. In this case, products that contain 51% or more of wholegrain ingredients by weight may make the following FDA-approved health claim: 'Diets rich in whole grain foods and other plant foods, and low in total fat, saturated fat and cholesterol, may reduce the risk of heart disease and certain cancers'.

The JHCI has also endorsed a generic health claim for oats and reduction of blood cholesterol. The claim 'The inclusion of oats as part of a diet low in saturated fat and a healthy lifestyle can help reduce blood cholesterol' may be applied to foods containing at least 0.75 g per serving (or in an amount that is customarily consumed in a day that makes a reasonable contribution to a healthy diet). This is based on a recommended daily intake of 3 g beta-glucan soluble fibre, equivalent to roughly 20 g oats (assuming rolled oats provide at least 4% beta-glucan soluble fibre).

However, it is expected that, in the very near future, the opinions of the JHCI will be supplanted by formal legislation contained within the EU Regulation on Nutrition and Health Claims. Once in place, this will introduce Europe-wide standards and criteria for the use of nutrition and health claims. Before a claim can be used, evidence will be considered by EFSA, and guidelines will be set for the use of such statements.

The new EU regulations will have several categories for types of nutrition and health claims. Article 13 concerns well-established health claims linking a nutrient with a particular function ('functional claims'). Each member state will be responsible for collating possible nutrition claims, and the FSA has already begun this process for the UK, inviting industry and other interested parties to submit suggestions. Decisions about the acceptability of the evidence underpinning such claims will be undertaken by EFSA. EFSA will also be responsible for assessing dossiers submitted to underpin disease risk-reduction health claims, which are covered under Article 14, and for establishing whether (and in

what form) nutrient profiling is required to identify which foods are to be excluded from carrying claims (see Buttriss 2007).

Conclusion

Dietary fibre is an essential component of a healthy balanced diet, as there are undeniable benefits with regard to bowel health when sufficient fibre is included in the diet. However, it is clear that dietary fibre is not an entity, but a collective term for a complex mixture of substances with different chemical and physical properties which exert different types of physiological effects. These various non-digestible components of dietary fibre are not interchangeable, and it is important that fibre comes from a range of sources to ensure maximum health benefits from the fibre in the diet. Traditional 'insoluble' fibres are required to add bulk, as well as rapidly fermentable, viscous fibres, to bring about cholesterol lowering. There is also a convincing argument for including slowly fermented components, such as resistant starches, that are well tolerated in the digestive system and can bring about improvements in gut function. Currently, we do not have sufficient data from well-designed human intervention trials to attempt to make specific recommendations on the amounts of these fibre components in the diet, but it might be useful for health professionals to talk in terms of the different food sources of these types of fibre, as well as total fibre amounts.

The complex nature of dietary fibre has also led to confusion in the definition and measurement of fibre in the diet, and this has hampered advancements in this area. There is no single official definition of dietary fibre and, currently, different countries are setting dietary recommendations and regulating the use of nutrient content and health claims using various definitions of fibre. It is important that a workable definition of dietary fibre should: clarify the constituent makeup of dietary fibre; recognise that a primary characteristic is resistance to digestion and absorption in the small intestine and fermentation in the large intestine; and demonstrate that fibre has physiological properties. However, until definitions and methodologies are synchronised, there are great challenges for health professionals, regulators, industry and researchers.

Acknowledgements

The authors wish to thank National Starch and Chemical Limited for financial support in the publication of the finished report.

References

AACC (American Association of Cereal Chemists) (2001) AACC Dietary Fiber Technical Committee. The definition of dietary fiber. *Cereal Foods World* **46**: 112–26.

AACC (American Association of Cereal Chemists) (2005) AACC Definition/Reports. Available at: http://www.aaccnet.org/definitions/wholegrain.asp (accessed November 2006).

Akerberg AK, Liljeberg HG, Granfeldt YE *et al.* (1998) An in vitro method, based on chewing, to predict resistant starch content in foods allows parallel determination of potentially available starch and dietary fiber. *Journal of Nutrition* **128**: 651–60.

Alberts DS, Martinez ME, Roe DJ *et al.* (2000) Lack of effect of a high-fiber cereal supplement on the recurrence of colorectal adenomas. Phoenix Colon Cancer Prevention Physicians' Network. *New England Journal of Medicine* **342**: 1156–62.

Alfenas RC & Mattes RD (2005) Influence of glycemic index/load on glycemic response, appetite, and food intake in healthy humans. *Diabetes Care* **28**: 2123–9.

Anti M, Pignataro G, Armuzzi A *et al.* (1998) Water supplementation enhances the effect of high-fiber diet on stool frequency and laxative consumption in adult patients with functional constipation. *Hepatogastroenterology* **45**: 727–32.

Asano T & McLeod RS (2002) Dietary fibre for the prevention of colorectal adenomas and carcinomas. *Cochrane Database Systematic Reviews* (2): CD003430.

Asp N-G, Johansson C-G, Hallmer H *et al.* (1983) Rapid enzymatic assay of insoluble and soluble dietary fiber. *Journal of Agriculture and Food Chemistry* **31**: 476–82.

Baghurst KI, Baghurst PA & Record SJ (2001) Dietary fibre, non-starch polysaccharide and resistant starch intakes in Australia. In: *CRC Handbook of Dietary Fibre in Human Health* (GA Spiller ed.), pp. 583–91. CRC Press: Boca Raton, FL.

Baghurst PA, Baghurst KI & Record SJ (1996) Dietary fibre, non-starch polysaccharides and resistant starch – a review. *Supplement to Food Australia* **48**: S3–35.

Bednar GE, Patil AR, Murray SM *et al.* (2001) Starch and fiber fractions in selected food and feed ingredients affect their small intestinal digestibility and fermentability and their large bowel fermentability in vitro in a canine model. *Journal of Nutrition* **131**: 276–86.

BHF (British Heart Foundation) (2006) *Heart health*. Available at: http://www.bhf.org.uk (accessed November 2006).

Bingham SA, Day NE, Luben R *et al.* (2003) Dietary fibre in food and protection against colorectal cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC): an observational study. *Lancet* **361**: 1496–501.

Blottiere HM, Buecher B, Galmiche J-P *et al.* (2003) Molecular analysis of the effect of short-chain fatty acids on intestinal cell proliferation. *Proceedings of the Nutrition Society* **62**: 101–6.

BNF (British Nutrition Foundation) (1990) *Complex Carbohydrates in Foods*. Chapman & Hall: London.

BNF (British Nutrition Foundation) (2002) *Nutrition Labelling and Health Claims*. BNF: London.

Bonithon-Kopp C, Kronborg O, Giacosa A *et al.* (2000) Calcium and fibre supplementation in prevention of colorectal adenoma recurrence: a randomised intervention trial. European Cancer Prevention Organisation Study Group. *Lancet* **356**: 1300–6.

Bray GA, Nielsen SJ & Popkin BM (2004) Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *American Journal of Clinical Nutrition* **79**: 537–43.

Brennan CS (2005) Dietary fibre, glycaemic response, and diabetes. *Molecular Nutrition and Food Research* **49**: 560–70.

Brown IL, McNaught KJ & Moloney E (1995) Hi-maize™: new directions in starch technology and nutrition. *Food Australia* **47**: 272–5.

Brown L, Rosner B, Willett WW *et al.* (1999) Cholesterol-lowering effects of dietary fiber: a meta-analysis. *American Journal of Clinical Nutrition* **69**: 30–42.

Burkitt DP, Walker ARP & Painter NS (1972) Effect of dietary fibre on stools and transit times, and its role in the causation of disease. *Lancet* **2**: 1408–12.

Burley VJ, Paul AW & Blundell JE (1993) Sustained post-ingestive action of dietary fibre: effects of a sugar-beet-fibre-supplemented breakfast on satiety. *Journal of Human Nutrition and Dietetics* **6**: 43–50.

Buttriss JL (2007) Nutrition and health claims. *Nutrition Bulletin* **32**: 72–6.

Cassidy A, Bingham SA & Cummings JH (1994) Starch intake and colorectal cancer risk: an international comparison. *British Journal of Cancer* **69**: 937–42.

Champ M, Langkilde AM, Brouns F *et al.* (2003) Advances in dietary fibre characterisation. 1. Definition of dietary fibre, physiological relevance, health benefits and analytical benefits. *Nutrition Research Reviews* **16**: 71–82.

Craig SAS, Holden JF & Khaled MY (2000) Determination of polydextrose as dietary fiber in foods. *Journal of the Association of Official Analytical Chemists International* **83**: 1006–12.

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

Cummings JH & Bingham SA (1998) Diet and the prevention of cancer. *British Medical Journal* **317**: 1636–40.

Cummings JH, Bingham SA, Heaton KW *et al.* (1992) Fecal weight, colon cancer risk, and dietary intake of nonstarch polysaccharides (dietary fiber). *Gastroenterology* **103**: 1783–9.

Davey GK, Spencer EA, Appleby PN *et al.* (2003) EPIC-Oxford: lifestyle characteristics and nutrient intakes in a cohort of 33 883 meat-eaters and 31 546 non meat-eaters in the UK. *Public Health Nutrition* **6**: 259–69.

Dean AC (1978) Method for estimation of available carbohydrates in food. *Food Chemistry* **3**: 241–50.

Defra (Department of the Environment Food and Rural Affairs) (2006) *The Expenditure and Food Survey*. HMSO: London.

DeVries JW (2004) On defining dietary fibre. *Proceedings of the Nutrition Society* **62**: 37–43.

DH (Department of Health) (1991) *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom*. HMSO: London.

Doll R & Peto R (1981) The causes of cancer. *Journal of the National Cancer Institute* **66**: 1191–308.

Dyssler P & Hoffmann D (1994) Estimation of resistant starch intake in Europe. In: *Proceedings of the Concluding Plenary Meeting of EURESTA* (N-G Asp, JMM van Amelsvoort & JGAJ Hautvast eds), pp. 84–6. EURESTA: Wageningen, The Netherlands.

Englyst HN & Cummings JH (1988) Improved method for measurement of dietary fiber as non-starch polysaccharides in plant foods. *Journal of the Association of Official Analytical Chemists* **71**: 808–14.

Englyst HN & Hudson GJ (1987) Colorimetric method for routine measurement of dietary fibre as non-starch polysaccharides. A comparison with gas-liquid chromatography. *Food Chemistry* **24**: 63–76.

Englyst HN, Kingman SM & Cummings JH (1992) Classification and measurement of nutritionally important starch fractions. *European Journal of Clinical Nutrition* **46**: S33–50.

Englyst KN & Englyst HN (2005) Carbohydrate bioavailability. *British Journal of Nutrition* **94**: 1–11.

FAO/WHO (Food and Agriculture Organisation/World Health Organization) (1997) *Carbohydrates in Human Nutrition*. Report of a Joint FAO/WHO Expert Consultation. Rome.

FAO/WHO (Food and Agricultural Organisation/World Health Organization) (2006) *Report of the 27th Session of the Codex Committee on Nutrition and Foods for Special Dietary Uses*. Bon, Germany, 21–25 November 2005.

FDA (Food and Drug Administration) (2006) Health Claim Notification for Whole Grain Foods with Moderate Fat Content. Available at: http://www.cfsan.fda.gov/~dms/flgrain2.html (accessed November 2006).

Finch S, Doyle W, Lowe C *et al.* (1998) *National Diet and Nutrition Survey: people aged 65 years and over. Vol 1: Report of the Diet and Nutrition Survey*. TSO, London.

Flight I & Clifton P (2006) Cereal grains and legumes in the prevention of coronary heart disease and stroke: a review of the literature. *European Journal of Clinical Nutrition* **60**: 1145–59.

FNB (Food and Nutrition Board) (2001) *Dietary Reference Intakes: Proposed Definition of Dietary Fiber*. The National Academies Press: Washington, DC.

FNB (Food and Nutrition Board) (2005) *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. The National Academies Press: Washington, DC.

Frolich W (1990) Chelating properties of dietary fiber and phytate. The role for mineral availability. *Advances in Experimental Medicine and Biology* **270**: 83–93.

FSA (Food Standards Agency) (2002) *McCance and Widdowson's The Composition of Foods*, 6th summary edn. Royal Society of Chemistry: Cambridge.

FSANZ (Food Standards Australia New Zealand) (2001) Section 1.2.8 of the Food Standards Code. Available at: http://www.foodstandards.gov.au/thecode/foodstandardscode.cfm (accessed November 2006).

Fuchs CS, Giovannucci EL, Colditz GA *et al.* (1999) Dietary fiber and the risk of colorectal cancer and adenoma in women. *New England Journal of Medicine* **340**: 169–76.

Gibney M (2000) *EURODIET: A Framework for Food-Based Dietary Guidelines in the European Union*. Health & Consumer Protection: Brussels.

Goodlad RA & Englyst HN (2001) Redefining dietary fibre: potentially a recipe for disaster. *Lancet* **358**: 1833–4.

Gordon DT & Ohkuma K (2002) Determination of total dietary fiber in selected foods containing resistant maltodextrin by enzymatic-gravimetric method and liquid chromatography: collaborative study. *Journal of the Association of Official Analytical Chemists International* **85**: 435–44.

Gordon DT, Stoops D & Ratliff V (1995) Dietary fiber and mineral nutrition. In: *Dietary Fiber in Health and Disease* (D Kritchevsky & C Bonfield eds), pp. 267–93. Eagan Press: St Paul.

Gregory J & Lowe S (2000) *National Diet and Nutrition Survey: Young People Aged 4 to 18 Years. Volume 1: Report of the Diet and Nutrition Survey*. The Stationery Office: London.

Gregory JR, Collins DL, Davies PSW *et al.* (1995) *National Diet and Nutrition Survey: Children Aged 1.5 to 4.5 Years. Vol 1: Report of the Diet and Nutrition Survey*. London: HMSO.

Gregory J, Lowe S, Bates CJ *et al.* (2000) *National Diet and Nutrition Survey: Young People Aged 4 to 18 Years. Vol 1: Report of the Diet and Nutrition Survey*. London: The Stationery Office

Gross LS, Li L, Ford ES *et al.* (2004) Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the United States: an ecologic assessment. *American Journal of Clinical Nutrition* **79**: 774–9.

Hanover LM & White JS (1993) Manufacturing, composition, and applications of fructose. *American Journal of Clinical Nutrition* **58**: 724S–32S.

Hara H, Haga S, Aoyama Y *et al.* (1999) Short-chain fatty acids suppress cholesterol synthesis in rat liver and intestine. *Journal of Nutrition* **129**: 942–8.

HCN (Health Council of the Netherlands) (2006) *Guideline for Dietary Fibre Intake*. HCN: The Hague.

Heaton KW (1973) Food fibre as an obstacle to energy intake. *Lancet* **2**: 1418–21.

Henderson L, Gregory J & Swan G (2002) *The National Diet and Nutrition Survey: Adults Aged 19 to 64 Years. Volume 1: Types and Quantities of Foods Consumed*. The Stationery Office: London.

Henderson L, Gregory J, Irving K *et al.* (2003) *The National Diet and Nutrition Survey: Adults Aged 19 to 64 Years. Volume 2: Energy, Protein, Carbohydrate, Fat and Alcohol Intake*. The Stationery Office: London.

Hippocrates, On Ancient Medicine, 400BC.

Hipsley EH (1953) Dietary 'fibre' and pregnancy toxaemia. *British Medical Journal* **2**: 420–2.

Hoebregs H (1997) Fructans in foods and food products, ion-exchange chromatographic method: Collaborative study. *Journal of the Association of Official Analytical Chemists International* **80**: 1029–37.

Holt S, Brand J, Soveny C *et al.* (1992) Relationship of satiety to post-prandial glycaemic, insulin and cholecystokinin responses. *Appetite* **18**: 129–41.

Hu Y, Martin J, Le Leu R *et al.* (2002) The colonic response to genotoxic carcinogens in the rat: regulation by dietary fibre. *Carcinogenesis* **23**: 1131–7.

IGD (Institute of Grocery Distribution) (1998) *Voluntary Nutritional Labelling. Guidelines to Benefit the Consumer*. IGD: Watford, UK.

IGD (Institute of Grocery Distribution) (2005) *Report of the IGD/PIC Industry Nutrition Strategy Group Technical Working Group on Guideline Daily Amounts*. IGD: Watford, UK.

James SL, Muir JG, Curtis SL *et al.* (2003) Dietary fibre: a roughage guide. *International Medicine Journal* **33**: 291–6.

Jenkins DJ, Kendal CW, Augustin LS *et al.* (2002) High-complex carbohydrate or lente carbohydrate foods? *American Journal of Medicine* **113**: 30S–7S.

Kafatos A & Codrington CA (2000) *EURODIET: Nutrition & Diet for Healthy Lifestyles in Europe; Science & Policy Implications*. Health & Consumer Protection: Brussels.

Khaw KT & Barrett-Connor E (1987) Dietary fiber and reduced ischemic heart disease mortality rates in men and women: a 12-year prospective study. *American Journal of Epidemiology* **126**: 1093–102.

Koh-Banerjee P & Rimm EB (2003) Whole grain consumption and weight gain: a review of the epidemiological evidence, potential mechanisms and opportunities for future research. *Proceedings of the Nutrition Society* **62**: 25–9.

Koh-Banerjee P, Franz M, Sampson L *et al.* (2004) Changes in wholegrain, bran, and cereal fiber consumption in relation to 8-y weight gain among men. *American Journal of Clinical Nutrition* **80**: 1237–45.

Lang R & Jebb SA (2003) Who consumes whole grains, and how much? *Proceedings of the Nutrition Society* **62**: 123–7.

Lee S, Prosky L & DeVries JW (1992) Determination of total, soluble, and insoluble die-tary fiber in foods – enymatic-gravimetric method, MES-TRIS buffer: collaborative study. *Journal of the Association of Official Analytical Chemists International* **75**: 395–416.

Levine AS & Billington CJ (1994) Dietary fiber: does it affect food intake and body weight?. In: *Appetite and Body Weight Regulation: Sugar, Fat, and Macronutrient Substitutes* (JD Fernstrom & GD Miller eds), pp. 191–200. CRC Press: Boca Raton, FL.

Li BW & Cardozo MS (1994) Determination of total dietary fiber in foods and products with little or no starch, nonenzymatic-gravimetric method: collaborative study. *Journal of the Association of Official Analytical Chemists International* **77**: 687–9.

Lia A, Hallmans G, Sandberg AS *et al.* (1995) Oat beta-glucan increases bile acid excretion and a fiber-rich barley fraction increases cholesterol excretion in ileostomy subjects. *American Journal of Clinical Nutrition* **62**: 1245–51.

Lia A, Andersson H, Mekki N *et al.* (1997) Postprandial lipemia in relation to sterol and fat excretion in ileostomy subjects given oat-bran and wheat test meals. *American Journal of Clinical Nutrition* **66**: 357–65.

Liljeberg Elmstahl H (2002) Resistant starch content in a selection of starchy foods on the Swedish market. *European Journal of Clinical Nutrition* **56**: 500–5.

Lyons-Wall P (2000) Food analysis and food composition tables. In: *Essentials of Human Nutrition* (J Mann & S Truswell eds), pp. 397–407. Oxford University Press: Oxford, UK.

McCleary BV & Monaghan DA (2002) Measurement of resistant starch. *Journal of the Association of Official Analytical Chemists International* **85**: 665–75.

McCleary BV & Rossiter P (2004) Measurement of novel dietary fibres. *Journal of the Association of Official Analytical Chemists International* **87**: 707–17.

McCleary BV, Murphy A & Mugford DC (2000) Measurement of total fructan in foods by enzymatic/spectrophotometric method: collaborative study. *Journal of the Association of Official Analytical Chemists International* **83**: 356–64.

MacFarlane S & MacFarlane GT (2003) Regulation of short-chain fatty acid production. *Proceedings of the Nutrition Society* **62**: 67–72.

McMurry KY (2003) Setting dietary guidelines: the US process. *Journal of the American Diet Association* **103**: S10–16.

MAFF (Ministry of Agriculture, Fisheries and Food) (1988a) *Cereal and Cereal Products: Third Supplement to the Fifth Edition of McCance and Widdowson's The Composition of Foods*. Royal Society of Chemistry: Cambridge.

MAFF (Ministry of Agriculture, Fisheries and Food) (1988b) *Household Food Consumption and Expenditure*. Annual Report of the National Food Survey Committee. MAFF: London.

MAFF (Ministry of Agriculture, Fisheries and Food) (1990) *Fifty Years of the National Food Survey*. HMSO: London.

MAFF (Ministry of Agriculture, Fisheries and Food) (1991) *Vegetables, Herbs and Spices: Fifth Supplement to the Fifth Edition of McCance and Widdowson's The Composition of Foods*. Royal Society of Chemistry: Cambridge.

MAFF (Ministry of Agriculture, Fisheries and Food) (1992) *Fruit and Nuts: First Supplement to the Fifth Edition of McCance and Widdowson's The Composition of Foods*. Royal Society of Chemistry: Cambridge.

MAFF/JFSSG (Ministry of Agriculture, Fisheries and Food/Joint Food Safety and Standards Group) (1999) Letter from Rosemary Hignett to Industry entitled 'Definition and determination of dietary fibre for nutrition labelling'.

Mentschel J & Claus R (2003) Increased butyrate formation in the pig colon by feeding raw potato starch leads to a reduction of colonocyte apoptosis and a shift to the stem cell compartment. *Metabolism* **52**: 1400–5.

Michels KB, Giovannucci E, Joshipura KJ *et al.* (2000) Prospective study of fruit and vegetable consumption and incidence of colon and rectal cancers. *Journal of the National Cancer Institute* **92**: 1740–52.

Miller WC, Niederpruem MG, Wallace JP *et al.* (1994) Dietary fat, sugar, and fiber predict body fat content. *Journal of the American Dietetic Association* **94**: 612–15.

Mongeau R & Brassard R (1993) Enzymatic-gravimetric determination in foods of dietary fiber as sum of insoluble and soluble fiber fractions: summary of collaborative study. *Journal of the Association of Official Analytical Chemists International* **76**: 923–5.

Monro JA (2004) Adequate intake values for dietary fibre based on faecal bulking indexes of 66 foods. *European Journal of Clinical Nutrition* **58**: 32–9.

Moshfegh A, Goldman J & Cleveland L (2005) *What We Eat in America*, NHANES 2001–2001: Usual nutrient intakes from food compared to dietary reference intakes. US Department of Agriculture, Agricultural Research Service: Washington D.C.

Moshfegh AJ, Friday JE, Goldman JP *et al.* (1999) Presence of inulin and oligofructose in the diets of Americans. *Journal of Nutrition* **129**: 1407S–11S.

NHMRC (National Health and Medical Research Centre) (2006) *Nutrient Reference Values for Australia and New Zealand Including Recommended Dietary Intakes 2006 Commonwealth of Australia*. NHMRC Publications: Canberra.

Nordgaard I, Mortensen PB & Langkilde AM (1995) Small intestinal malabsorption and colonic fermentation of resistant starch and resistant peptides to short-chain fatty acids. *Nutrition* **11**: 129–37.

Nugent A (2005) Health properties of resistant starch. *Nutrition Bulletin* **30**: 27–54.

Painter NS (1975) *Diverticular Disease of the Colon: A Deficiency Disease of Western Civilization*. Heinemann: London.

Pereira MA, O'Reilly E & Augustsson K (2004) Dietary fiber and risk of coronary heart disease: a pooled analysis of cohort studies. *Archives of Internal Medicine* **164**: 370–6.

Pietinen P, Rimm EB, Korhonen P *et al.* (1996) Intake of dietary fiber and risk of coronary heart disease in a cohort of Finnish men. The Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study. *Circulation* **94**: 2720–7.

Pietinen P, Malila N, Virtanen M *et al.* (1999) Diet and risk of colorectal cancer in a cohort of Finnish men. *Cancer Causes Contrology* **10**: 387–96.

Pool-Zobel BL (2005) Inulin-type fructans and reduction in colon cancer risk: review of experimental and human data. *British Journal of Nutrition* **93**: S73–90.

Potter JD & McMichael AJ (1986) Diet and cancer of the colon and rectum: a case–control study. *Journal of the National Cancer Institute* **76**: 557–69.

Prosky L, Asp NG, Furada I *et al.* (1985) Determination of total dietary fiber in foods and food products: collaborative study. *Journal of the Association of Official Analytical Chemists International* **68**: 677–9.

Quigley ME & Englyst HN (1994) Determination of the uronic acid constituents of non-starch polysaccharides by high-performance liquid chromatography with pulsed amperometric detection. *Analyst* **119**: 1511–18.

Rimm EB, Ascherio A, Giovannucci E *et al.* (1996) Vegetable, fruit, and cereal fiber intake and risk of coronary heart disease among men. *Journal of the American Medical Association* **275**: 447–51.

Ripsin CM, Keenan JM, Jacobs DR Jr *et al.* (1992) Oat products and lipid lowering. A meta-analysis. *Journal of the American Medical Association* **267**: 3317–25.

Roberfroid M (1993) Dietary fiber, inulin, and oligofructose: a review comparing their physiological effects. *Critical Reviews in Food Science and Nutrition* **33**: 103–48.

Roberfroid MB (2005) Introducing inulin-type fructans. *British Journal of Nutrition* **93**: S13–25.

Roediger WE, Duncan A, Kapaniris O *et al.* (1993) Sulphide impairment of substrate oxidation in rat colonocytes: a biochemical basis for ulcerative colitis? *Clinical Science (London)* **85**: 623–7.

Salmerón J, Ascherio A, Rimm EB *et al.* (1997a) Dietary fiber, glycemic load, and risk of NIDDM in men. *Diabetes Care* **20**: 545–50.

Salmerón J, Manson JE, Stampfer MJ *et al.* (1997b) Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *Journal of the American Medical Association* **277**: 472–7.

Schatzkin A, Lanza E, Corle D *et al.* (2000) Lack of effect of a low-fat, high-fiber diet on the recurrence of colorectal adenomas. Polyp Prevention Trial Study Group. *New England Journal of Medicine* **342**: 1149–55.

Schulze MB, Liu S, Rimm EB *et al.* (2004) Glycemic index, glycemic load, and dietary fiber intake and incidence of type 2 diabetes in younger and middle-aged women. *American Journal of Clinical Nutrition* **80**: 348–56.

Schulze MB, Fung TT, Manson JE *et al.* (2006) Dietary patterns and changes in body weight in women. *Obesity* **14**: 1444–53.

Schweizer TF & Würsch P (1979) Analysis of dietary fibre. *Journal of the Science of Food and Agriculture* **30**: 613–19.

Schwiertz A, Lehmann U, Jacobasch G *et al.* (2002) Influence of resistant starch on the SCFA production and cell counts of butyrate-producing Eubacterium spp. in the human intestine. *Journal of Applied Microbiology* **93**: 157–62.

Seal CJ (2006) Whole grains and CVD risk. *Proceedings of the Nutrition Society* **65**: 24–34.

Seal CJ, Jones AR & Whitney AD (2006) Whole grains uncovered. *Nutrition Bulletin* **31**: 129–37.

Sengupta S, Tjandra JJ & Gibson PR (2001) Dietary fibre and colorectal neoplasia. *Diseases of the Colon and Rectum* **44**: 1016–33.

Smith AT, Kuznesof S, Richardson DP *et al.* (2003) Behavioural, attitudinal and dietary responses to the consumption of wholegrain foods. *Proceedings of the Nutrition Society* **62**: 455–67.

Smith U (1987) Dietary fibre, diabetes and obesity. *International Journal of Obesity* **11**: 27–31.

Southgate DA (1976) *Determination of Food Carbohydrates*. Applied Science Publishers: London.

Southgate DA, Paul AA, Dean AC *et al.* (1978) Free sugars in foods. *Journal of Human Nutrition* **32**: 335–47.

Southgate DAT (1969) Determination of carbohydrates in foods. II. Unavailable carbohydrates. *Journal of the Science of Food and Agriculture* **20**: 331–5.

Spiller GA (1993) Suggestions for a basis on which to determine a desirable intake of dietary fiber. In: *CRC Handbook of Dietary Fiber in Human Nutrition* (GA Spiller ed.), pp. 351–4. CRC Press: Boca Raton, FL.

Spiller GA, Story JA, Wong LG *et al.* (1986) Effect of increasing levels of hard wheat fiber on fecal weight, minerals and steroids and gastrointestinal transit time in healthy young women. *Journal of Nutrition* **116**: 778–85.

Spiller RC (1999) Impact of dietary fiber on absorption in the small intestine. *Current Opinions in Gastroenterology* **15**: 100–2.

Stephen AM & Cummings JH (1980) Mechanism of action of dietary fibre in the human colon. *Nature* **284**: 283–4.

Terry P, Giovannucci E, Michels KB *et al.* (2001) Fruit, vegetables, dietary fiber, and risk of colorectal cancer. *Journal of the National Cancer Institute* **93**: 525–33.

Thane CW, Jones AR, Stephen AM *et al.* (2005) Whole-grain intake of British young people aged 4–18 years. *British Journal of Nutrition* **94**: 825–31.

Theander O & Åman P (1979) Studies on dietary fibres. 1. Analysis and chemical characterization of water-soluble and water-insoluble dietary fibres. *Swedish Journal of Agricultural Research* **9**: 97–106.

Theander O & Westerlund E (1986) Determination of individual components of dietary fiber. In: *CRC Handbook of Dietary Fiber in Human Nutrition* (GA Spiller ed.), pp. 57–75. CRC Press: Boca Raton, FL.

Theander O, Åman P, Westerlund E *et al.* (1995) Total dietary fiber determined as neutral sugar residues, uronic acid residues, and Klason lignin (the Uppsala method): collaborative study. *Journal of the Association of Official Analytical Chemists International* **78**: 1030–44.

Theobald HE (2004) Glycaemic index: what's the story? *Nutrition Bulletin* **29**: 291–4.

Tomlin J & Read NW (1990) The effect of resistant starch on colon function in humans. *British Journal of Nutrition* **64**: 589–95.

Topping DL & Clifton PM (2001) Short-chain fatty acids and human colonic function: roles of resistant starch and nonstarch polysaccharides. *Physiological Reviews* **81**: 1031–64.

Topping DL, Fukushima M & Bird AR (2003) Resistant starch as a prebiotic and symbiotic: state of the art. *Proceedings of the Nutrition Society* **62**: 171–6.

Trinidad TP, Wolever TMS & Thompson LU (1996) Effect of acetate and propionate on calcium absorption from the rectum and distal colon of humans. *American Journal of Clinical Nutrition* **63**: 574–8.

Trock B, Lanza E & Greenwald P (1990) Dietary fibre, vegetables and colon cancer: critical review and meta-analyses of the epidemiological evidence. *Journal of the National Cancer Institute* **82**: 650–61.

Trowell H (1972) Ischemic heart disease and dietary fibre. *American Journal of Clinical Nutrition* **25**: 926–32.

Trowell HC (1974) Definition of dietary fibre. *Lancet* **1**: 503.

Trowell HC, Southgate DAT, Wolever TMS *et al.* (1976) Dietary fiber redefined. *Lancet* **1**: 967.

USDA (United States Department of Agriculture) (2005) Dietary Guidelines for Americans. Available at: http://www.healthierus.gov/dietaryguidelines (accessed November 2006).

Van Loo J, Coussement P, de Leenheer L *et al.* (1995) On the presence of inulin and oligofructose as natural ingredients in the western diet. *Critical Reviews in Food Science and Nutrition* **35**: 525–52.

Wasan HS & Goodlad RA (1996) Fibre-supplemented foods may damage your health. *Lancet* **348**: 319–20.

WCRF (World Cancer Research Fund) (1997) *Food, Nutrition and the Prevention of Cancer: A Global Perspective*. American Institute for Cancer Research: Washington, DC.

WHO/FAO (World Health Organization/Food and Agriculture Organisation) (2003) *Global Report on Diet, Nutrition and Prevention of Chronic Diseases*. Technical Report 916. WHO: Geneva.

Willett WC (1995) Diet, nutrition, and avoidable cancer. *Environmental Health Perspectives* **103**: 165–70.

Williams CL (1995) Importance of dietary fiber in childhood. *Journal of the American Dietetic Association* **95**: 1140–6.

Wolever TMS & Jenkins DJA (1993) Effect of dietary fiber and foods on carbohydrate metabolism. In: *CRC Handbook of Dietary Fiber in Human Nutrition*, 2nd edn (GA Spiller ed.), pp. 111–52. CRC Press: Boca Raton, FL.

Wolk A, Manson JE, Stampfer MJ *et al.* (1999) Long-term intake of dietary fiber and decreased risk of coronary heart disease among women. *Journal of the American Medical Association* **281**: 1998–2004.

Wong CSM & Gibson PR (2003) The trophic effect of dietary fibre is not associated with a change in total crypt number in the distal colon of rats. *Carcinogenesis* **24**: 343–8.

 Nutrition Bulletin, **32**, 21–64

Appendix

The carbohydrate content (g/100 g as eaten) of foods

Food	Sugars	Starch	Fibre – E	Fibre – S	RS	Lignin	Insoluble NCP	Soluble NCP
Cereals								
All-bran	15.4	27.6	4.5	30.0	0.1	1.5	15.9	4.1
Barley, pearled	tr	27.6	N	2.0	N	N	N	N
Bran, wheat	3.8	23.0	36.4	39.6	0	3.2	26.1	3.3
Brown bread	3.0	41.3	3.5	5.9	0.8	0.4	1.8	1.1
Brown rice	0.5	31.6	0.8	1.5	N	tr	0.5	tr
Buckwheat	0.4	84.4	2.1	N	0.3	N	0.6	1.0
Cornflakes	7.2	77.7	0.9	3.4	2.9	N	0.2	0.4
Cornflour	tr	92.0	0.1	N	0.1	0.0	tr	0.1
Crispbread	3.2	67.4	11.7	11.6	1.8	1.1	6.9	3.9
Croissants	1.0	37.2	1.6	2.5	N	tr	0.8	0.8
Digestive biscuit	13.6	55.0	2.2	4.6	0.3	0.5	0.9	1.1
Granary bread	2.2	44.1	4.3	6.5	0.9	0.6	1.5	2.1
Hot cross bun	23.4	35.1	1.7	2.2	N	N	0.7	0.8
Macaroni	0.3	18.2	0.9	1.5	0.6	0.1	0.3	0.5
Naan bread	5.5	44.6	1.9	2.2	N	N	0.9	0.9
Noodles, egg	0.2	12.8	0.6	1.0	0.5	0.1	0.2	0.3
Oat bran flakes	N	N	N	N	N	N	N	N
Pitta bread, white	2.4	55.5	2.2	3.9	N	N	N	N
Porridge	4.7	9.0	0.8	0.8	N	N	0.3	0.5
Puffed wheat	0.3	67.0	5.6	8.8	0.7	1.2	N	N
Rice Krispies	10.6	79.1	0.5	1.1	0.2	0.1	0.2	0.1
Rye bread	1.8	44.0	4.4	5.8	0.8	0.3	1.8	2.2
Scones, plain	5.9	47.9	1.9	2.2	N	tr	0.9	0.9
Shredded wheat	0.8	67.5	9.8	10.1	0.8	1.1	6.2	2.0
Spaghetti, white	0.5	21.7	1.2	1.8	0.5	0.2	0.5	0.6
Spaghetti, wholemeal	1.3	21.9	3.5	4.0	0.4	0.2	2.0	0.8
Sweetcorn	2.3	16.9	2.2	4.2	N	0.1	1.4	0.1
Water biscuit	2.3	73.5	3.1	6.1	0.5	1.3	1.1	1.8
Weetabix	6.4	68.5	9.7	11.6	0.2	1.1	5.1	3.1
Wheat flour, white	1.5	76.2	3.1	3.6	0.3	tr	1.5	1.5
Wheat flour, wholemeal	2.1	61.8	9.0	8.6	0.3	0.3	5.6	2.0
Wheatgerm	16.0	2837.0	15.6	N	0.2	N	9.7	3.2
White bread	2.6	46.7	1.5	3.8	0.8	0.4	0.5	0.9
White rice	tr	30.9	0.1	1.0	N	0.2	0.1	tr
Courgette	1.9	0.1	1.2	N	N	0.1	0.1	0.6
Cucumber	1.4	0.1	0.6	0.7	N	0.1	0.1	0.2
Kidney bean, red	1.0	14.5	6.7	9.0	N	N	1.8	3.2
Lentil, red	0.8	16.2	1.9	3.3	N	N	1.0	0.6
Lettuce	1.7	tr	0.9	1.3	N	tr	0.1	0.5
Marrow	1.4	0.2	0.6	1.0	N	tr	0.1	0.2
Mushroom, fried	0.1	0.2	1.5	3.0	N	tr	0.9	0.3
Okra	2.3	0.5	3.6	4.1	N	0.5	0.4	2.2
Old potato	0.7	16.3	1.2	1.4	N	tr	0.1	0.7
Onion, fried	2.0	0.3	1.9	3.1	N	0.2	0.4	0.7
Parsnip	5.9	6.4	4.7	4.4	N	tr	1.4	2.7
Potato crisps	1.0	62.0	6.3	13.7	N	0.4	0.9	3.5
Radish, red	1.9	tr	0.9	0.9	N	0.1	0.1	0.4
Runner bean	2.0	0.3	1.9	3.1	N	0.2	0.4	0.7
Tomato	3.1	tr	1.0	1.3	N	0.3	0.2	0.4
Turnip	1.9	0.1	1.9	2.0	N	tr	0.3	0.7
Watercress	0.4	tr	1.5	3.0	N	tr	0.2	0.7
Fruits & Nuts								
Almond	4.2	2.7	7.4	12.9	N	N	4.4	1.1
Apple	11.8	tr	1.8	2.0	N	0.1	0.4	0.7
Apricot	7.2	0	1.7	1.9	N	0.1	0.2	1.0
Avocado	0.5	tr	3.4	N	N	0.2	0.7	1.6
Blackberry	5.1	0	3.1	6.6	N	N	0.9	1.0
Brazil nut	2.4	0.7	4.3	8.1	N	N	1.4	1.3
Cashew nut	4.6	13.5	3.2	N	N	N	1.0	1.6
Cherry	11.5	0	0.9	1.5	N	tr	0.2	0.5
Fig	9.5	0	1.5	2.3	N	N	0.3	0.9
Gooseberry	3.0	0	2.4	2.9	N	N	0.9	0.9
Grapefruit	6.8	0	1.3	1.6	N	0.1	0.1	0.9
Hazelnut	4.0	2	6.5	8.9	N	N	1.8	2.5
Kiwi fruit	10.3	0.3	1.9	N	N	0.2	0.3	0.9
Lychee	14.3	0	0.7	1.5	N	tr	0.1	0.5
Mango	13.8	0.3	2.6	2.9	N	0.3	0.5	1.6
Melon, cantaloupe	4.2	0	1.0	0.9	N	tr	0.2	0.3
Nectarine	0.9	0	1.2	2.2	N	0.2	0.2	0.6

Appendix *Continued*

Food	Sugars	Starch	Fibre – E	Fibre – S	RS	Lignin	Insoluble NCP	Soluble NCP
Vegetables								
Asparagus	0.7	tr	0.7	0.7	N	tr	0.1	0.3
Aubergine	2.6	0.2	2.3	2.9	N	0.2	0.3	1.1
Baked bean	5.8	9.3	3.5	6.6	N	0.2	0.6	2.1
Beetroot	8.8	0.7	1.9	2.3	N	0.1	0.3	0.9
Broad bean	0.9	4.3	5.4	3.8	N	N	0.6	1.2
Broccoli, green	0.9	tr	2.3	N	N	tr	0.4	1.0
Brussels sprouts	3.0	0.3	3.1	2.6	N	0.1	0.5	1.6
Butter bean	1.6	15.6	5.2	4.6	N	N	1.5	2.2
Cabbage	2.0	0.1	1.8	2.3	N	0.3	0.4	0.8
Carrot	4.6	0.2	2.5	2.8	N	0.1	0.2	1.4
Chickpea	1.0	16.6	4.3	5.1	N	0.7	1.9	1.4
Chips, fried	1.7	28.8	2.2	3.0	N	tr	0.2	1.3
Olive	tr	0	2.9	4.0	N	tr	1.7	0.3
Orange	8.5	0	1.7	1.8	N	0.1	0.2	1.1
Passion fruit	5.8	0	3.3	N	N	N	1.4	0.5
Peanut	6.2	6.3	6.2	7.3	N	N	2.3	1.9
Pear	10.0	0	2.2	N	N	N	0.8	0.7
Pineapple	10.1	0	1.2	1.3	N	0.1	0.6	0.1
Plum	8.8	0	1.6	2.3	N	0.1	0.2	1.1
Prune, canned	19.7	0	2.4	N	N	tr	0.5	1.5
Raspberry	4.6	0	2.5	6.7	N	2.2	0.6	0.7
Strawberry	6.0	0	1.1	2.0	N	0.1	0.2	0.5
Sunflower seed	1.7	16.3	6.0	N	N	N	2.8	1.8
Tangerine	8.0	0	1.3	1.7	N	tr	0.1	0.9
Walnut	1.1	0.3	1.5	2.5	N	N	0.4	0.6

Source: MAFF (1988a, 1991, 1992).

Fibre E, Englyst method; Fibre S, Southgate method; RS, resistant starch; NCP, non-cellulosic polysaccharide; tr, trace; N, no data available.