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Scientific Advisory Committee on Nutrition

Draft Carbohydrates and Health report

Scientific consultation: 26 June to 1 September 2014

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Executive summary

To be included after the consultation process is complete.

Chapter 1. Introduction and methods

Background

1.1 Dietary carbohydrates and their role in health were last considered by the Committee on Medical Aspects of Food and Nutrition Policy (COMA, the predecessor of Scientific Advisory Committee on Nutrition) in the following reports: Sugars and Human Disease (COMA, 1989), Dietary Reference Values for Food Energy and Nutrients for the United Kingdom (COMA, 1991) and Nutritional Aspects of Cardiovascular Disease (COMA, 1991; COMA, 1994). Considerable evidence has emerged since these reports were published, particularly in the areas of cardio-metabolic, colo-rectal and oral health. It was considered important, therefore, to review the literature on carbohydrate intake in relation to these health outcomes and ensure that the dietary reference values reflect the current evidence base. In addition, it was necessary to review the classification and definition of carbohydrates, as these can vary between scientific studies, as well as between national and international organisations, which can lead to confusion and difficulties when assessing the evidence base and comparing intakes across countries.

Terms of Reference

1.2 The Scientific Advisory Committee on Nutrition (SACN) was requested by the Food Standards Agency and the Department of Health in 2008 to provide clarification of the relationship between dietary carbohydrate and health and make public health recommendations. To achieve this they were asked to review:

- The evidence for a role of dietary carbohydrate in colo-rectal health in adults (including colo-rectal cancer, irritable bowel syndrome, constipation) and in childhood and infancy.
- The evidence on dietary carbohydrate and cardio-metabolic health (including cardiovascular disease, insulin resistance, glycaemic response and obesity).
- The evidence in respect to dietary carbohydrates and oral health.
- The terminology, classification and definitions of types of carbohydrates in the diet.

Methodology

1.3 . Due to the wealth of data available and because of the concerns around their limitations, case-control, cross-sectional and ecological studies were not considered. Only prospective cohort studies and randomised controlled trials were considered for this report. Evidence on adverse effects of very high intakes of specific carbohydrates, e.g. gastrointestinal symptoms, was not part of the remit of this report.

1.4 Systematic reviews on cardio-metabolic, colo-rectal and oral health in relation to carbohydrates and food sources of carbohydrates were commissioned to inform this report (see Annex 1 for individual systematic reviews). These were based on literature published through December 2009, November 2010 and January 2012, respectively. The search strategy and inclusion and exclusion criteria are described in Annex 2.

1.5 Due to the timing of cut-off dates for literature identified in the systematic reviews, there was concern that the evidence base would be incomplete (out of date) when the report was published. Therefore an updated literature search was performed using the search terms provided in the individual reviews from January 2010 (cardio-metabolic health), December 2010 (colo-rectal health review) and February 2011 (oral health review) up to June 2012. The same inclusion and exclusion criteria detailed in the individual reviews were used to identify relevant articles; however, the update search was not a systematic review. After this cut-off date additional studies were considered only if they were thought potentially to impact on or inform the conclusions drawn in this report. This was particularly the case where there was limited evidence or when it was difficult to interpret how evidence from the update search affected the conclusion.

1.6 A total of 225 prospective cohort studies and 403 randomised controlled trials have been considered in this report. For the individual systematic reviews and update search see Annex 1.

1.7 The evidence was assessed using the SACN framework for the evaluation of evidence (SACN, 2012) and graded according to the system described in Annex 2. This system was devised specifically to consider the evidence included in the Carbohydrates and Health report.

Interpretation of studies

1.8 With cohort studies there is substantial potential for biases and the possibility of confounding by extraneous variables that correlate with both the dependent variable and the independent variable (residual confounding) and any associations must be interpreted with caution. As a range of dietary and lifestyle factors may be associated with the health outcome considered, it is possible that an indicated association could be due to an unidentified factor that correlates with the studied factor. The dietary assessment methods used in cohort studies are potentially subject to measurement error due to the inability to estimate portion sizes, under- and over-reporting of food items, and inherent limitations in the quality and completeness of food nutrient databases. A further issue is that the quality of the dietary assessment varies between studies, with some studies only making a single assessment before following up a health outcome many years later. Overall, this can affect the ability of studies to observe an association as well as affecting the magnitude of an indicated association with a dietary factor, e.g. under-reporting would diminish any indicated association with that dietary factor. The definitions used in cohort studies to characterise and quantify a specific dietary exposure, e.g. ‘whole grains’, may vary between studies.

1.9 A general limitation with the randomised controlled trials is that they investigate markers and risk factors, but not actual disease outcomes. Consideration of disease outcomes in relation to carbohydrate intake is, therefore, dependent on prospective cohort studies. Many of the trials included in this report involve mixed interventions that modify other dietary components, e.g. the proportion and type of fat or micronutrient content, and also involve energy restriction goals that result in weight loss, both of which could potentially affect the outcomes considered. This limits the conclusions that can be drawn; in particular this applies to the effect of variation in total carbohydrate intake and glycaemic index and load on cardio-metabolic risk factors. A further limitation of some trials is that the dietary interventions vary greatly between trials examining a specific outcome and it is often not possible to consider dose-response effects; this is especially so with regard to trials varying total carbohydrate intake.

1.10 Where possible, the dose-response relationship between carbohydrate intakes and health outcomes has been considered and used to inform the dietary reference values.

1.11 The subjective nature of self-reported dietary intake assessment methods presents numerous challenges to obtaining accurate dietary intake and nutritional status. This limitation could be overcome by the use of dietary biomarkers, which are able to objectively assess dietary consumption (or exposure) without the bias of self-reported dietary intake errors (Hedrick *et al.*, 2012). Biomarkers of sugars intake and dietary fibre intake, in particular, are needed to support existing dietary recommendations. In relation to carbohydrate exposures there are, as yet, no biomarkers of dietary exposure that are validated, reproducible, and able to detect changes in intake over time that are suitable for the general population. There are several potential biomarkers of carbohydrate exposure currently being investigated, such as: 24-hour urinary sucrose (Tasevska *et al.*, 2005; Joosen *et al.*, 2008; Tasevska *et al.*, 2009; Tasevska *et al.*, 2011); carbon stable isotope biomarkers of sugar intake from corn and sugar cane (Jahren *et al.*, 2014); and several studies have examined plasma alkylresorcinol and its metabolite concentrations as a possible whole grain wheat and rye intake biomarker (Landberg *et al.*, 2008; Söderholm *et al.*, 2009).

Chapter 2. Classification, biochemistry, absorption, metabolism and definitions of carbohydrates

2.1. The term 'carbohydrate' is defined as polyhydroxy aldehydes, ketones, alcohols, acids, their simple derivatives and their polymers having linkages of the acetal type. The primary classification of carbohydrate is based on chemistry, i.e. the character of individual monomers (e.g. monosaccharides), degree of polymerisation[1] (DP) and type of linkage (α or β[2]), as recommended at the Food and Agriculture Organization/World Health Organization Expert Consultation in 1997 (FAO/WHO, 1998). This divides carbohydrates into three main groups, sugars (mono- and di-saccharides, DP 1–2), oligosaccharides (DP 3–9) and polysaccharides (DP ≥10) (see table 2.1).

Table 2.1 Chemical classification of carbohydrates (FAO/WHO, 1998)

Class	Sub-group	Components
Sugars (DP 1-2)	Monosaccharides	Glucose, galactose, fructose
	Disaccharides	Sucrose, lactose, maltose
	Sugars alcohols/polyols	Sorbitol, mannitol
Oligosaccharides (DP 3-9)	Malto-oligosaccharides	Maltodextrins
	Non-digestible oligosaccharides	Raffinose, stachyose, fructo-oligosaccharides
Polysaccharides (DP >9)	Starch	Amylose, amylopectin, modified starches
	Non-starch polysaccharides	Cellulose, hemicellulose, pectins, hydrocolloids (gums)

2.2. In 2006, an FAO/ WHO update on some of the key issues relating to carbohydrates in human nutrition endorsed the primary classification recommended by the 1997 Expert Consultation, but acknowledged that a chemical classification, although providing a practical basis for measurement and labelling, did not allow a simple translation into nutritional effects (Mann *et al*., 2007). Each class of carbohydrate has overlapping physiological properties and effects on health.

2.3. Carbohydrates can be classified based on their digestion and absorption in the small intestine rather than their chemical characteristics. Digestible carbohydrates are absorbed and digested in the small intestine; non-digestible carbohydrates are resistant to hydrolysis in the small intestine and reach the human large intestine where they are at least partially fermented by the commensal bacteria present in the colon. The term 'dietary fibre' refers to either some or all of the constituents of non-digestible carbohydrates and may also include other quantitatively minor components, e.g. lignin, that are associated with non-digestible carbohydrates in the plant cell walls (see paragraphs 2.27 to 2.34 for a consideration of different dietary fibre definitions).

[1] The number of monomer units incorporated into polymer chains

[2] The linkages found in disaccharides, oligosaccharides and polysaccharides are classified as α or β. These glycosidic linkages are the bonds between two monosaccharides. The α-linkages are easily digested by the human body, but the β-linkages are stronger than α-linkages because they are more stable. Carbohydrates with β-linkages are not easily digested by the human body, except for lactose, because most humans have an enzyme which breaks down this disaccharide.

Chemical classification

Sugars

2.4. The term ‘sugars’ conventionally describes mono- and di-saccharides. Pentoses are five-carbon sugars that occur in polymeric forms and are collectively known as pentosans. Xylose and arabinose are the constituents of pentosans present in the non-starch polysaccharides hemicelluloses and pectin (McNaught, 1997). The three principal hexoses (six-carbon sugars) are glucose, fructose and galactose, which are the building blocks of naturally occurring di-, oligo- and poly-saccharides. The hexose mannose is present in some plant polysaccharides collectively termed mannans and is present in hemicelluloses. Glucose is widely distributed in small amounts in fruits, plant juices and honey. Fructose is found in the free state alongside glucose in ripening fruits and honey. Chemically combined with glucose it forms sucrose, which is the predominant disaccharide occurring in the free form. Galactose occurs in milk, in chemical combination with glucose as lactose, while the disaccharide maltose, derived from starch hydrolysis, comprises two glucose molecules (Cummings & Stephen, 2007).

Oligosaccharides

2.5. Oligosaccharides include maltodextrins, which principally occur from the hydrolysis of starch and are widely used in the food industry to modify the texture of food products. Maltodextrins are digested and absorbed in the small intestine. Oligosaccharides that are not digested and absorbed in the small intestine include raffinose, stachyose and verbascose, which are three, four and five sugar polymers respectively. In effect these are sucrose joined to varying numbers of galactose molecules and are found in a variety of plant seeds e.g. peas, beans and lentils. Other non-digestible oligosaccharides are inulin and fructo-oligosaccharides. These have saccharide backbones that are mainly composed of fructose (fructans) and are the storage carbohydrates in artichokes and chicory with small amounts of low molecular weight found in wheat, rye, asparagus and members of the onion, leek and garlic family (Cummings & Stephen, 2007). The DP of inulin varies from 2-60 sugar units (Roberfroid, 1993).

2.6. Human milk contains more than 100 different oligosaccharides of great diversity of structure that are predominantly galactose containing (Bode, 2006). The principal oligosaccharide in human milk is lacto-N-tetraose. Total oligosaccharides in human milk are in the range 5.0–8.0 g/l, but only trace amounts are present in cow’s milk and these differ from those found in human milk (Ward *et al.*, 2006). Human milk oligosaccharides, among other functions, may also serve as substrates for colonic fermentation (Kunz *et al.*, 1999; Bode, 2006). Small-chain non-digestible oligosaccharides, abundantly present in the early stage of lactation, are selectively fermented by specific strains of *Bifidobacterium longum biovar, infantis* (Ninonuevo & Lebrilla, 2009).

Polysaccharides

2.7. Starch is a high molecular weight polymer of glucose, and is the principal storage carbohydrate in plants, and the principal carbohydrate in most diets. Starch is defined as α1-4 linked glucan (a polysaccharide of glucose monomers), which can have straight (amylose) or branched (amylopectin) chains. Amylopectin also has α1-6 glycosidic bonds to branched chains. (Elia & Cummings, 2007). Enzymes capable of catalysing the hydrolysis of starch (α-amylases) are produced in the salivary gland and the pancreas.

2.8. Non-starch polysaccharides are plant cell wall constituents and comprise all other polysaccharides in the diet. They are not digested or absorbed in the small intestine. Non-starch polysaccharides are the most diverse of all the carbohydrate groups and comprise a mixture of many molecular forms, of which cellulose, a straight chain β1–4-linked glucan (DP 103–106) is the most widely distributed (McNaught, 1997).

2.9. Other non-starch polysaccharides in common occurrence are the hemicelluloses and pentosans. Hemicellulose (e.g. arabinoxylan) contains a mixture of hexose and pentose sugars, often in highly branched chains. Common to all cell walls is pectin, which is primarily a galacturonic acid polymer, although 10–25% other sugars such as rhamnose, galactose and arabinose, may also be present as side chains. β-glucans are a heterogeneous group of non-starch polysaccharides, consisting of D-glucose monomers linked by β-glycosidic bonds (Cummings & Stephen, 2007).

2.10. Plant gums and storage polysaccharides, e.g. gum Arabic, karaya (sterculia) and guar gum, plant mucilages, e.g. psyllium, and algal polysaccharides e.g. agar and carrageenan are all non-starch polysaccharides primarily found in manufactured foods, usually at low concentrations as modifiers of texture or viscosity (Cummings & Stephen, 2007). Each sub-type comprises a diversity of naturally occurring and chemically modified molecular and structures and weights, resulting in different properties.

Soluble and insoluble dietary fibre

2.11. The terms ‘soluble’ and ‘insoluble’ fibre arose out of the early chemistry of non-starch polysaccharides, which showed that the fractional extraction of non-starch polysaccharides could be controlled by changing the pH of solutions. This divides non-starch polysaccharides into those which may have effects on glucose and lipid absorption from the small intestine (soluble) and those which were slowly and incompletely fermented in the colon and have more pronounced effects on bowel habit (insoluble) (Cummings & Stephen, 2007). The separation of soluble and insoluble fractions is very pH dependent, making the link with specific physiological properties less certain. Much insoluble fibre is completely fermented and not all soluble fibre has effects on glucose and lipid absorption; also the various forms of fibre exist together mostly in intact plant cell walls.

2.12. As the differentiation is method-dependent, and solubility does not always predict physiological effects, it has been proposed that the distinction between soluble and insoluble fibre should be phased out (FAO/WHO, 1998). The terms are, however, still widely used. The soluble fibres include pectin and β-glucans and the insoluble fibres include cellulose and hemicelluloses.

Digestion and absorption

2.13. Only glucose and galactose are actively absorbed in the human small intestine via the sodium dependent transporter (SGLT1). Fructose is not actively absorbed, but is taken up via a specific facilitative transport pathway (Thorens & Mueckler, 2010).

2.14. Di-, oligo- and poly-saccharides must be hydrolysed to their component monosaccharides before being absorbed. Starch occurs in plants in the form of semi-crystalline granules that must be gelatinized by processing or cooking prior to digestion. The digestion of solubilised starch polysaccharides begins with salivary amylase, but this activity is much less important than that of pancreatic amylase in the small intestine. Amylase hydrolyses starch, with the primary end products being maltose, maltotriose, and α-dextrins, although some glucose is also produced. The products of α-amylase digestion are hydrolysed into their component monosaccharides by enzymes expressed on the brush border of the small intestinal cells, the most important of which are maltase, sucrase, isomaltase and lactase (FAO/WHO, 1998). Carbohydrates containing glycosidic linkages that are resistant to cleavage by the pancreatic and brush border enzymes, i.e. non-digestible carbohydrates, are not hydrolysed in the small intestine and reach the human large intestine where they may be fermented, to some degree, by the commensal bacteria present in the colon that contain enzymes capable of hydrolysing the glycosidic linkages (Hawksworth *et al.*, 1971).

2.15. Resistant starch is the sum of starch and products of starch digestion that are not absorbed in the small bowel (Englyst *et al.*, 1992; Champ *et al.*, 2003). While all unmodified starch, if solubilised, can be hydrolysed by pancreatic α-amylase, the rate and extent to which starch is broken down is altered by a number of physical and chemical properties of food. This has led to the classification of resistant starch into four types (Englyst *et al.*, 1992):

- Physically inaccessible starch (RS_1), such as occurs in whole, and partly milled grains, seeds, and legumes;
- Resistant starch granules (RS_2), such as raw potato, banana, and high amylose corn;
- Retrograded amylose (RS_3), formed in foods such as cooked, cooled potato, bread, and cornflakes; and
- Chemically modified starch (RS_4), which is commercially manufactured.

Metabolism

2.16. Carbohydrates are principally substrates for energy metabolism. The human body is able to capture some of the chemical energy from carbohydrates through cellular metabolism, resulting in the generation of an intermediary chemical form, adenosine triphosphate, which acts as an energy source for cellular processes mainly through phosphorylation of proteins and other intermediates. It is regenerated from adenosine diphosphate using the energy in food. The brain, nervous system and red blood cells have an obligatory requirement for glucose as an energy source.

2.17. Following absorption, monosaccharides are transported to the liver and from there to the systemic circulation. The plasma concentration of insulin increases immediately after the ingestion of glycaemic carbohydrate. Cellular uptake is via a family of glucose transporters (GLUT), and in some tissues (adipose tissue, skeletal muscle) is insulin-dependent. Fructose uptake into tissues is not insulin-dependent. Intracellular glucose is metabolised via glycolysis following phosphorylation by hexokinase (all cells) and glucokinase (liver only). Hexokinase and phosphofructokinase catalyse early regulatory steps in glycolysis. Galactose and mannose are metabolised to glycolytic intermediates (glucose 6 phosphate and fructose 6 phosphate, respectively) and progress through glycolysis is, therefore, subject to regulation by phosphofructokinase. In the adipose tissue and muscle, fructose can also be converted to the glycolytic intermediate fructose 6 phosphate, although probably not quantitatively very important; fructose metabolism in the liver is not directly under the regulation of hexokinase and phosphofructokinase (Feinman & Fine, 2013).

2.18. The amount of energy yielded from different carbohydrates in food, that are digested in the small intestine varies according to the molecular form i.e. glucose, disaccharides and starch, the actual available energy content per unit weight is 15kJ (3.6 kcal/g), 16kJ (3.8 kcal/g) and 17kJ (4.0 kcal/g), respectively (FAO, 2003). Carbohydrate that is not digested and absorbed in the small intestine may also provide energy, as fermentation in the colon results in the formation of short-chain fatty acids, some of which are absorbed into the blood stream and are used as energy. An available energy content per unit weight has been estimated as 8 kJ/g (1.9 kcal/g) for fermentable non-starch polysaccharide, 9 kJ/g (2.2 kcal/g) for resistant starch, 8-9 /g (1.9-2.2 kcal/g) for non-digestible oligosaccharides and 6-10 kJ/g (1.4-2.4 kcal/g) for non-digestible sugar alcohols (Elia & Cummings, 2007).

Glycaemic index and glycaemic load

2.19. Glycaemic index (GI) and glycaemic load (GL) are used as measures of the glycaemic characteristics of the diet. The GI is a relative measure of the plasma glucose response induced by a specific ingredient, food or meal, as compared with the response induced by the same amount (usually 50g) of available carbohydrate from a reference source, such as pure glucose or an alternative source (such as white bread) which has been calibrated against it (Brouns *et al.*, 2005). The GL is the product of a specific food's GI and its carbohydrate content (Brouns *et al.*, 2005), therefore taking into account both the quality and quantity of carbohydrate consumed. The GI (and thus also GL) is determined not only by the nature of the carbohydrate component of a food or diet, but also by the types and amounts of protein, fat and non-starch polysaccharide, resistant starch and oligosaccharide, energy density, protein or fat content, as well as food processing and storage (Brouns *et al.*, 2005; Venn & Green, 2007). Unless tightly controlled, in an experimental situation higher and lower GI/GL diets will, in most cases, differ in many ways other than the carbohydrate fraction. Variation in GI does not does just reflect variation in rates of carbohydrate influx, but can also reflect effects on rates of glucose production and disposal (Schenk *et al.*, 2003; Eelderink *et al.*, 2012a; Eelderink *et al.*, 2012b). As such, the majority of the literature on GI and GL does not allow for certainty that the carbohydrate content of an exposure is the sole influence on the GI or GL of a diet, nor that a similar GI or GL for different foods has the same physiological basis. This limits the confidence in assigning cause-effect relationships for outcomes based on variation in diet GI or GL.

Fermentation of sugars in the oral cavity

2.20. Dental caries is the result of demineralisation of enamel and dentine in the presence of acid. The pH associated with decay is close to the 'critical pH for demineralisation' of the tissue or about 5.5 for enamel and about 6.0 for dentine. The acids that induce demineralisation at this level are produced by the fermentation of dietary sugars (including sugars produced by hydrolysis of starch in mouth), particularly sucrose, by acidogenic bacteria from the oral microbiota. Lactose is fermented more slowly than sucrose (Birkhed *et al.*, 1993). Demineralisation from bacterially-derived acids (predominantly lactic acid) occurs during virtually every meal as natural sugars are present in virtually all foods. Saliva is, however, a very efficient remineralising solution allowing for repair of demineralised tissue. Saliva neutralises and buffers acids, contains calcium phosphate and is supersaturated with biological apatite. Tooth decay occurs when the frequency or extent of demineralisation in any one person's mouth exceeds the capacity of saliva to remineralise the tissue (García-Godoy & Hicks, 2008).

Definitions used in different dietary recommendations

2.21. The classification of carbohydrate components in dietary recommendations may vary between different authorities. The terms carbohydrate and starch have distinct chemical definitions that do not differ between different organisations. There is diversity of opinion regarding the measurement of dietary components defined on the basis of functionality rather than chemical composition (Englyst *et al*., 2007). The classification used to define sugars and dietary fibre in dietary recommendations varies between different authorities and these differences are considered below.

Sugars

2.22. While the term 'sugars' refers to monosaccharides and disaccharides, various terms are used to define the types of sugars described in dietary recommendations. A summary of the different definitions for sugars is given in table 2.2.

2.23. In the UK, dietary reference values have been provided for 'non-milk extrinsic sugars'. Intrinsic sugars are those naturally incorporated into the cellular structure of foods; extrinsic sugars are those sugars not contained within the cellular structure of a food. The extrinsic sugars in milk and milk products (e.g. lactose) were deemed to be exempt from the classification of sugars in relation to the dietary reference value (Committee on Medical Aspects of Food Policy, 1991). Non-milk extrinsic sugars include sugars added to foods, e.g. sucrose, glucose and fructose, and sugars naturally present in fruit juices, e.g. glucose and fructose.

2.24. The 2002 FAO/WHO Expert Consultation defined the term 'free sugars' as all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and fruit juices (WHO, 2003). This term describes sugars that may have physiological consequences different from sugars incorporated within intact plant cell walls.

2.25. In the United States, dietary reference intakes are set for 'added sugars', which are defined as sugars and syrups that are added to foods during processing and preparation. Added sugars do not include naturally occurring sugars such as lactose in milk or fructose in fruits (Institute of Medicine, 2005).

2.26. The European Food Safety Authority defines sugars as total sugars, including both indigenous (sugars naturally present in foods such as fruit, vegetables, cereals and lactose in milk products) and added sugars (EFSA, 2009). The term 'added sugars' refers to sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup, isoglucose) and other isolated sugar preparations used as such, or added during food preparation and manufacturing.

Table 2.2 Different definitions used for sugars in dietary recommendations

Term	Definition
Non-milk extrinsic sugars* – UK	Sugars not contained within the cellular structure of a food and sugars in milk and milk products.
Free sugars* – WHO	Sugars added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups fruit juices and fruit concentrates.
Added sugars – US	Sugars and syrups that are added to foods during processing and preparation.
Added sugars – EFSA	Sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup, isoglucose) and other isolated sugar preparations used as such or added during food preparation and manufacturing.

* The only difference between non-milk extrinsic sugars and free sugars is that non-milk extrinsic sugars includes 50% of the fruit sugars from stewed, dried or canned fruit (Bates et al., 2012), but free sugars includes none.

Dietary fibre

2.27. Differences in the definition for dietary fibre between dietary recommendations from different authorities to some extent reflect differences in the analytical methods used to determine dietary fibre, in conjunction with the degree to which indigestibility and non-absorbability in the small intestine are considered to be a satisfactory basis for the definition of dietary fibre. Dietary fibre analytical methodologies have adopted two general approaches: enzymic–gravimetric methods and enzymic–chemical methods. A summary of the different definitions for dietary fibre is given in table 2.3.

2.28. In the UK, dietary fibre has been defined as non-starch polysaccharide, comprising cellulose and non-cellulose polysaccharides (e.g. pectins, glucans, arabinogalactans, arabinoxylans, gums and mucilages) (COMA, 1991; COMA, 1994). This is analytically defined using an enzymic–chemical method (Englyst et al., 1994) and does not include resistant starches, non-digestible oligosaccharides or lignin.

2.29. In 2008, the SACN reviewed the available scientific evidence for components under consideration for inclusion in the Codex definition of dietary fibre for nutrition labelling purposes (SACN, 2008). The Committee defined dietary fibre as material that is resistant to digestion and absorption in the small intestine and that has a demonstrable

physiological effect potentially associated with health benefits in the body, such as increasing stool bulk, decreasing intestinal transit time, decreasing post-prandial glycaemia or the lowering of total cholesterol and LDL-cholesterol concentrations. This includes non-starch polysaccharides. Inclusion of other components in the definition would require beneficial physiological effects to be demonstrated. Evidence only of increased fermentation in the gut should not be included under this definition, since although this has a direct effect on the microbiota, it must also be shown to have a demonstrable benefit to the host to be considered as dietary fibre.

2.30. In the United States 'total dietary fibre' is defined as the sum of dietary fibre and functional fibre (Institute of Medicine, 2005). Dietary fibre is defined as non-digestible carbohydrates and lignin that are intrinsic and intact in plants. Functional fibre is defined as isolated, non-digestible carbohydrate components that have beneficial physiological effects in humans, with a DP of 3 or more, and which may be isolated or extracted using chemical, enzymatic, or aqueous steps. Excluded are non-digestible mono- and disaccharides and polyols, some resistant starch and non-digestible animal carbohydrates.

2.31. Total dietary fibre is analytically defined as the material isolated by enzymic–gravimetric methods approved by the Association of Official Analytical Chemists (AOAC), generally AOAC Method 985.29 and 991.43 (Prosky et al., 1988; Lee et al., 1992). These AOAC methods determine non-starch polysaccharides, some resistant starches, lignin and some inulin, plus lignin, but do not measure most non-digestible oligosaccharides. AOAC methods have subsequently been developed to measure all resistant starches and low molar weight dietary fibres such as non-digestible oligosaccharides and inulin, e.g. AOAC method 2009.01 (McCleary et al., 2010; McCleary et al., 2012).

2.32. The 2006 FAO/WHO scientific update on carbohydrates in human nutrition considered that the term 'dietary fibre' should be reserved for the cell wall polysaccharides of vegetables, fruits and whole grains, the health benefits of which have been clearly established, rather than synthetic, isolated or purified oligosaccharides and polysaccharides with diverse, and in some cases unique, physiological effects (Mann et al., 2007).

2.33. In 2008, the Codex Committee on Nutrition and Foods for Special Dietary Uses agreed a definition of dietary fibre, which was agreed by the Codex Alimentarius Committee in 2009 (Cummings et al., 2009; FAO/WHO, 2010). This defines dietary fibre as carbohydrate polymers with ten or more monomeric units, which are not hydrolysed by endogenous enzymes in small intestine of human beings plus lignin and/or other compounds when associated with polysaccharides in the plant cell walls. The decision on whether to include carbohydrates from three to nine monomeric units in the definition of dietary fibre was left to national authorities.

2.34. In 2010, the European Food Safety Authority established a dietary reference value for dietary fibre and concluded that dietary fibre should include all non-digestible carbohydrates and lignin (EFSA, 2010a). This includes non-starch polysaccharides, all resistant starches, all non-digestible oligosaccharides with three or more monomeric units and other non-digestible, but quantitatively minor components that are associated with the dietary fibre polysaccharides, especially lignin. As in the Codex definition, to be defined as dietary fibre natural carbohydrate polymers obtained from raw material in food by physical, enzymatic, chemical means, or synthetic polymers, must have beneficial

physiological effects demonstrated by accepted scientific evidence (EFSA, 2010a). The Codex and European Food Safety Authority dietary fibre definitions are chemically defined using the AOAC method 2009.01 (McCleary et al., 2010; McCleary et al., 2012).

Table 2.3 Different definitions used for dietary fibre in dietary recommendations

Term	Definition
Non-starch polysaccharides – UK	Non-α-glucan polysaccharides: cellulose and non-cellulose polysaccharides (e.g. pectins, glucans, arabinogalactans, arabinoxylans, gums, and mucilages).
Dietary fibre – WHO	Intrinsic plant cell wall polysaccharides, i.e. non-starch polysaccharides
Total dietary fibre – US	Non-digestible carbohydrates and lignin that are intrinsic and intact in plants, and isolated, non-digestible carbohydrate components that have beneficial physiological effects in humans, with a DP of three or more. It was noted that the methodologies used at that time chemically defined total dietary fibre as non-starch polysaccharides, some resistant starches, lignin and some inulin, but did not include non-digestible oligosaccharides.
Dietary fibre – Codex	Carbohydrate polymers with ten or more monomeric units, which are not hydrolysed by endogenous enzymes in small intestine of human beings plus lignin and/or other compounds when associated with polysaccharides in the plant cell walls. The decision on whether to include carbohydrates from three to nine monomeric units in the definition of dietary fibre was left to national authorities.
Dietary fibre – EFSA	Non-starch polysaccharides, all resistant starches, all non-digestible oligosaccharides with three or more monomeric units and other non-digestible, but quantitatively minor components that are associated with the dietary fibre polysaccharides, especially lignin.

A comparison of the previous UK dietary reference values for carbohydrate with other international authorities

2.35. A summary of the different dietary carbohydrate recommendations from different authorities is given in table 2.4. The UK carbohydrate dietary reference values were set for adults and children aged five years and older (COMA, 1991; COMA, 1994). For total carbohydrates a population average of approximately 50% dietary energy was recommended (COMA, 1994). This was derived from observations that diets high in fat, particularly saturated fatty acids, are associated with higher cardiovascular disease. The carbohydrate value was set to restore the energy deficit from reducing dietary fat intake. It was recommended that starches and intrinsic and milk sugars should provide the balance of dietary energy not provided by alcohol, protein fat and non-milk extrinsic sugars, that is, on average 37% total dietary energy or 39% food energy (excluding alcohol[3]) for the population (COMA, 1994).

[3] Energy intake from alcohol was assumed to average 5% approximating current intakes at the time and was factored into the total energy value, but excluded from the food energy value.

2.36. The dietary reference value for non-milk extrinsic sugars was for a population average of no more than 10% of total dietary energy or 11% of food energy (excluding alcohol) (COMA, 1991). This was based on evidence that sugar intake is associated with greater dental caries. The value was derived from the observation that dental caries is rare in countries which have a sugar supply approximately equating to 10% of dietary energy. The dietary reference value for non-starch polysaccharides was for a population average of 18g/day, with an individual range of 12 g to 24 g/day. This was based on the effect of non-starch polysaccharide on increasing faecal weight and the observation that higher faecal weights were observed in populations with a lower incidence of bowel disease (COMA, 1991).

Table 2.4 Comparison of dietary carbohydrate recommendations by different authorities

	Total carbohydrate	Sugars	Dietary fibre*
UK	50% dietary energy	non-milk extrinsic sugars no more than 10% of total dietary energy	18g/day non- starch polysaccharide
US	Recommended daily allowance 130g/day	No limit set for added sugars **	Adequate intake level of 38g/day for men and 25g/day for women of 'total dietary fibre'
WHO	Range of 55 to 75% dietary energy	less than 10% of dietary energy as free sugars, with a (proposed) conditional recommendation of less than 5% of dietary energy as free sugars	greater than 20g/day non-starch polysaccharide (Englyst *et al.*, 1994)or greater than 25g/day of 'total dietary fibre'
EU	Range of 45 to 60% dietary energy	No limit set for added sugars	25g/day of dietary fibre

* For definitions of dietary fibre see table 2.2; ** a maximum level of intake of 25% of energy or less due to decreased intakes of certain micronutrients observed at these consumption levels was suggested.

Whole grains

2.37. A central tenet of dietary recommendations in many countries is to eat a diet rich in whole grains, fruits and vegetables (Cummings & Stephen, 2007). Whole grains include whole wheat, whole wheat flour, wheat flakes, bulgar wheat, whole and rolled oats, oatmeal, oat flakes, brown rice, whole rye and rye flour and whole barley. A precise definition, however, is needed, and the role of intact versus milled grains needs to be established (Cummings & Stephen, 2007). An analysis of whole grains intake of adults in the 1986-7 and 2000-1 NDNS surveys, including foods with less than 51% whole grains content, estimated median intakes in 1986–7 and 2000–1 to be 16g/day (inter quartile range 0–45g/day) and 14g/day (inter quartile range 0–36 g/day), respectively (Thane et al., 2007).

2.38. There are discrepancies between definitions of a whole grain food (including whole grain flour) used for labelling purposes between countries and within and outside the EU. In the UK and the USA whole grain foods must contain 51 % or more whole grain ingredients by wet weight, whereas in Sweden and Denmark the requirement is 50 % or more whole grain ingredients on a dry matter basis. In Germany, whole grain bread must be 90 % whole grains (EFSA, 2010b).

2.39. An EU research project (The Healthgrain Consortium) developed a single definition of whole grains for use across Europe and where possible equivalent to definitions outside Europe, notably the American Association of Cereal Chemists International definition[4] (van der Kamp et al., 2014). A full definition describing the grains included and the processing aspects was developed and an overall definition of whole grains given as follows:

- Whole grains shall consist of the intact, ground, cracked or flaked kernel after the removal of inedible parts such as the hull and husk. The principal anatomical components – the starchy endosperm, germ and bran – are present in the same relative proportions as they exist in the intact kernel.
- Small losses of components – that is, less than 2% of the grain/10% of the bran – that occur through processing methods consistent with safety and quality are allowed.

[4] Whole grains consist of the 'intact, ground, cracked or flaked caryopsis (grain), whose principal components-the starchy endosperm, germ and bran-are present in the same relative proportions as they exist in the intact caryopsis' (American Association of Cereal Chemists International, 1999). To be considered whole grain, a food may contain intact grains, minimally processed grains, or milled grains from which no component has been removed during the milling process.

Chapter 3. Dietary sources and intakes of carbohydrates

3.1. Nationally representative data on carbohydrate intakes of the UK population was drawn from the National Diet and Nutrition Survey (NDNS) rolling programme, a continuous survey of diet and nutrition in adults and children aged 18 months upwards. Data presented in this chapter are based on 3450 adults aged 19 years and over and 3378 children aged 1½-18 years collected over years 1-4 combined (2008/09 to 2011/12) (Bates et al 2014).

3.2. Carbohydrate intake data are also available for infants and young children aged 4-18 months from the 2011 UK Diet and Nutrition Survey of Infants and Young Children (Lennox et al. 2013). Carbohydrate intakes for the UK low income / materially deprived population, collected in 2003-2005, are available from the Low Income Diet and Nutrition Survey (Nelson et al. 2007).

3.3. Dietary sources of carbohydrates are presented at a broad category level, for example: cereals and cereal products, milk and milk products (Annex 7 Table 3.16-3.18). The top twenty contributors are also presented at a more detailed food group level (Annex 7 Tables 3.19 – 3.22).

3.4. In the NDNS rolling programme, diet was assessed by the participant (or the parent/carer for children) keeping a diary of all foods and drinks consumed for four consecutive days, using household measures and other methods to estimate quantities consumed. The survey was designed to represent all days of the week equally in the final data but there is actually a slight over-representation of Fridays and weekend days in the dataset compared with other days.

3.5. Dietary surveys are reliant on self-reported measures of intake. Misreporting of food consumption in dietary surveys, generally under-reporting, is known to be a problem in NDNS as in dietary surveys worldwide. A doubly-labelled water sub-study carried out as part of the NDNS rolling programme found that reported energy intake in adults aged 16-64 years was, on average, 34% lower than total energy expenditure (TEE) measured by doubly labelled water. The difference for other age groups was similar except for children aged 4-10 years where reported energy intake was 12% lower than TEE. This discrepancy between reported energy intake and TEE is likely to be due to a combination of underreporting of actual consumption and changing the diet during recording period. It is not possible to extrapolate these estimates of underreporting of energy intake to individual foods or nutrients, nor is it possible to correct or adjust the intake estimates to take account of under-reporting.

3.6. The carbohydrate intakes of the UK population are tabulated (Annex 7 Tables 3.1 – 3.22). Intakes in this chapter are compared with the current Dietary Reference Values (DRVs) set by COMA in 1991. Carbohydrate intakes are presented as grams/day, as a percentage of total energy intake (that is including energy from alcohol) and as a percentage of food and drink energy intake (that is, excluding energy from alcohol).

Total carbohydrate intakes and dietary sources

3.7. Mean intakes of total carbohydrate in the UK in adults and children aged 4 years and over were 200-240 grams/ day, 150 g/day in children aged 1½- 4 years. Mean intakes of total carbohydrate as a percentage of total energy were 51-52% in children and 46% in adults. Mean intakes in children met the DRV and in adults were close to the DRV (Annex 7 Table 3.1).

3.8. Cereals and cereal products was the main source of carbohydrate intake in all age groups, providing around 45% of intake; bread was the largest single source. The other food groups that made a substantial contribution to intake were potatoes, drinks (soft and alcoholic), and table sugar, preserves and confectionery; each provided around 10% of intake (Annex 7 Table 3.16).

3.9. White bread was the top contributor to intake of total carbohydrate in adults and children aged 4 years and over, providing 10-12% of intake. In children aged 1½-4 years milk and fruit were the top contributors, each providing 10% of intake. Soft drinks provided 10% of total carbohydrate intake in children aged 11-18 years (Annex 7 Table 3.19a-e).

3.10. In children aged 4-18 months mean intakes of total carbohydrate ranged from 93-126 grams/day and contributed 49-52% of total energy intake. The major contributor to carbohydrate intake for children aged 4 to 11 months was infant formula which provided 46% of intake in the youngest children reducing to 28% in those aged 10-11 months. For children aged 12 to 18 months the major contributor to carbohydrate intake was cereals and cereal products (34%). Commercial infant foods were also major contributors to intakes at the younger end of the age range and milk and milk products at the older end.

Total and individual sugar intakes and dietary sources

3.11. Mean total sugar intakes were around 95-100 g/day in adults and children over 4 years, 76g/day in children aged 18 months to 3 years. This represented about a fifth of total energy intake in older children and adults, a quarter in children under 10 years. About half of total sugar intake was sucrose – mean intakes were 40-50g/day for adults and children over 4 years. Mean intakes of glucose and fructose were 15-18 g/day each; lactose 10-13g/day and maltose 5-8g/day. The proportion of total sugar intake from lactose was slightly higher in children under 3 years (18g/day) due to higher milk consumption (Annex 7 Tables 3.3-3.11).

3.12. In adults aged 19-64 years soft drinks and fruit were the top contributors to total sugar intakes, each providing 13% of intake. Table sugar provided 9% of intake. In older adults aged 65 years and over fruit was the top contributor, providing 18% of total sugar intake with table sugar and milk each providing 10%. Soft drinks were the main contributor in children aged 11-18 years, providing 24% of intake. In the 4-10 year age group fruit was the top contributor at 14% of intake, followed by soft drinks and milk and cream each at 11% of intake and fruit juice at 9%. In the youngest age group – 1½-3 years milk and cream, and fruit each provided a fifth of total sugar intake (Annex 7 Table 3.20a-e).

3.13. In children aged 4-18 months mean intakes of total sugar were 71g/day in the youngest group reducing to 66g/day in the oldest. Total sugars provided 38% of energy in the youngest group and 26% in the 12-18 month age group. The main contributor to total sugar intake for children aged 4 to 11 months was infant formula providing over 50% of intake in the youngest age group and 40% in older children. For children aged 12 to 18 months, milk and milk products was the main source of total sugars (33%) followed by fruit (19%).

Non-milk extrinsic sugar intakes and dietary sources

3.14. Mean intakes of non-milk extrinsic sugars exceeded the DRV (that the population average intake should not exceed 10% of total energy) in all age/sex groups. Young children under 4 years and older adults aged 65 years and over had mean intakes closest to the DRV (11.9% and 11.2% total energy respectively). The highest mean intakes were in children aged 4-10 years and 11-18 years (14.7% and 15.4% of total energy respectively). Intakes at the 97.5th percentile provided 25-30% of total energy intake (Annex 7 Table 3.5).

3.15. The main contributors to NMES intake differed for adults and children. For older children aged 11-18 years soft drinks was the largest single source, providing 30% of intake, with a further 10% of intake from fruit juice. In younger children aged 4-10 years soft drinks provided 17% of intake and fruit juice 13%. Cereals and cereal products provided a quarter of NMES intake in children, mainly from added sugars in biscuits, buns, cakes and pastries and breakfast cereals; table sugar, preserves and confectionery provided a fifth of intake. In adults (19-64 years) soft and alcoholic drinks and fruit juice combined provided a third of intake, table sugar, preserves and confectionery provided a quarter of intake (table sugar the largest source) and cereals and cereal products provided a fifth of intake. In older adults aged 65 years and over, the contribution from cereals and cereal products was higher and that from drinks was lower (Annex 7 Table 3.17).

3.16. In children aged 4-18 months mean intakes of NMES provided 4.3% of energy intake in the youngest group and 7.7% in the 12-18 month age group. The main contributor to NMES intake for children aged 4 to 6 months and 7 to 9 months was the food group commercial infant foods. For children aged 10 to 18 months, the main contributor to NMES was milk and milk products.

Intrinsic and milk sugars intakes and dietary sources

3.17. Mean intakes of intrinsic and milk sugars were 30-43g/day across the age groups and provided 6-10% of total energy intake in adults and children aged 4 years upwards, 13% of energy intake in children aged 1½-3 years. Main sources were milk and fruit (Annex 7 Table 3.4)

3.18. In children aged 4-18 months mean intake of intrinsic and milk sugars provided 34% of energy for children aged 4 to 6 months, decreasing to 18% for children aged 12 to 18 months. Infant formula was the largest contributor to intake of intrinsic and milk sugars intake for children aged 4 to 11 months. The second largest contributors were breast milk for those aged 4 to 6 months and 7 to 9 months and fruit for those aged 10 to 11 months. For the 12 to 18 month age group, the largest contributor was milk and milk products followed by fruit.

Starch intakes and dietary sources

3.19. Mean starch intakes were 110-135g/day in adults and children over 4 years; 76g/day in younger children aged 1½-3 years. This represented 25-29% of total energy (Annex 7 Table 3.2).

3.20. White bread was the top contributor to starch intake in all age groups, providing a fifth of intake in adults and children aged 4 years and over, 15% in children aged 1½-3 years. Chips, fried potatoes and potato products was the second largest contributor in adults and children from 4 years, boiled, mashed and baked potatoes in older adults and high fibre breakfast cereals in children aged 1½-3 years. Pasta and pasta-based dishes provided 6-8% of starch intake in children and adults aged 19-64 years (Annex 7 Table 3.21).

3.21. In children aged 4-18 months mean starch intakes were 22g/day in the youngest group (11% of energy), increasing to 60g/day in the 12-18 month group (23% of energy). For children aged 4 to 6 months, the main contributor to starch intake was commercial infant foods. For children aged 7 to 9 months, the main contributors were commercial infant foods and cereals and cereal products. For children aged 10 to 11 months and 12 to 18 months, the main source of starch was cereals and cereal products.

Intrinsic and milk sugars and starch intakes

3.22. The DRV is for intrinsic and milk sugars and starch combined to provide 37% of total energy (including alcohol) as a population average. Mean intakes provided 34-35% of total energy in adults and children aged 11 years and over and 37-39% in younger children (Annex 7 Table 3.6)

Non-starch polysaccharides intakes and dietary sources

3.23. Mean intakes of non-starch polysaccharides in adults were 14g/day, well below the population average DRV for adults of 18g/day but above the individual minimum of 12g/day. Intakes at the 97.5th percentile were 25-26g/day. Mean intakes for children were lower at 11-12g/day (8g/day in children under 4 years) (Annex 7, Table 3.12).

3.24. At a broad food category level, the main sources of non-starch polysaccharides were cereals and cereal products which provided about 40% of intake across age groups, and vegetables and potatoes (a quarter to a third of intake) (Annex 7, Table 3.18).

3.25. Vegetables and vegetable-based dishes was the top contributor to non-starch polysaccharides intake in all age groups, providing just under a fifth of intake in children and a quarter in adults. Fruit was the second largest contributor in all age groups except 11-18 years providing 13-16% of intake in children under ten years and 10-13% in adults. In the 11-18 year age group white bread and chips and fried potatoes were the second largest sources each providing about 10% of intake (Annex 7 Table 3.22).

3.26. In the 4-18 month age group mean non-starch polysaccharide intakes were 4-7 grams per day. The main contributors to non-starch polysaccharide intake for children aged 4 to 11 months were commercial infant foods and infant formula. For children aged 12 to 18 months the main contributor to non-starch polysaccharides intake was cereals and cereal products followed by vegetables and potatoes.

Consumption of selected carbohydrate-containing foods

3.27. Tables 3.13 to 3.15 in Annex 7 presented consumption of selected carbohydrate-containing foods, based on data from NDNS. Consumption data are presented as population averages including participants who did not consume the food over the four day diary and as averages for consumers only (excluding non-consumers) with the percentage who consumed over four days.

3.28. Consumption of sugary soft drinks was highest in the 11-18 year age group. Population mean consumption of sugary soft drinks in boys aged 11-18 years was 310g/day, 80% of boys in this age group consumed sugary soft drinks over the four day period. For girls in the same age group 75% consumed sugary soft drinks over the four days and mean population consumption was 210g/day. Mean consumption in the 4-10 year ago group was lower at 128g/day with 69% consumers over four days.

3.29. Consumption of fruit juice was highest in children aged 4-10 years, 93g/day with 62% consumers over 4 days. Mean consumption in other age groups was 30-50g/day with 40-50% consumers.

3.30. White bread was the most commonly consumed type and eaten in the greatest quantities in all age groups. Mean daily consumption of all bread was 80-90 grams in adults and older children.

3.31. Mean consumption of breakfast cereals was 22-28g/day across the age groups except in older adults aged 65 years and over for whom it was higher at 47g/day. Consumption of high fibre breakfast cereals was higher than that of other breakfast cereals in all age groups except for children aged 11-18 years.

3.32. Mean consumption of potatoes was about 90g/day for adults and older children. Children aged 11-18 years consumed 54g/day of chips and fried potatoes and 33g/day of boiled, mashed and baked potatoes.

3.33. Children aged 4-18 years consumed 18-19 g/day of sugar and chocolate confectionery. Adults consumed 11-14 grams of table sugar per day.

Socio-economic differences in carbohydrate intakes

3.34. Analysis by equivalised household income in NDNS showed that for adults 19-64 year the lowest income quintile for both men and women had a higher percentage of energy from total carbohydrate than did the highest quintile. No clear pattern was seen in other age groups.

3.35. Overall, mean intakes of non-milk extrinsic sugars for adults tended to be higher in the lowest income quintile compared with the highest. For example in women aged 19-64 years mean NMES intake was significantly higher in the lowest quintile (12.7% total energy) compared with the highest (10.0%). This pattern was not seen in children and in boys 4-10 years the lowest quintile had significantly lower % energy from NMES than did the highest.

3.36. There is a clear income gradient in non-starch polysaccharide intake with NDNS showing lower intakes in the lowest income quintile compared to the highest in most age groups. This partly reflects lower fruit and vegetable consumption in lower income groups. For example in women 19-64 years latest NDNS data shows a significantly lower non-starch polysaccharide intake in the lowest quintile (11.8g/day) compared with quintile 5 (13.7g/day). However mean intakes were below the DRV in all income quintiles.

3.37. Evidence from other surveys confirm the socio-economic gradient. The Family Food Survey, based on household food purchases, found that the proportion of energy coming from NMES in the household diet was higher in the lowest income quintile compared with the highest while the non-starch polysaccharide content of food purchased by households in the lowest income quintile was lower than that of the highest quintile.

3.38. The UK Low income diet and nutrition survey carried out in 2003-05 found that intakes of carbohydrates were generally similar to those in the general population although where intakes failed to meet DRVs this tended to be more marked in the low income/ materially deprived population. Mean intakes of total carbohydrate were similar to intakes in the current NDNS (2008-2012) but mean intakes of non-milk extrinsic sugars as a percentage of energy were higher than the current NDNS and also higher than previous NDNS carried out in the 1990s while mean intakes of non-starch polysaccharides were lower than current and previous intakes in NDNS.

Summary

3.39. UK NDNS data shows that mean intakes of total carbohydrate meet or are close to meeting recommendations in all age groups while intake of non-milk extrinsic sugars exceeded recommendations in all age groups and were particularly high in children. Soft drinks provided almost a third of the intake of non-milk extrinsic sugars in children aged 11-18 years. Biscuits, buns, cakes, and puddings, confectionery and fruit juice were also significant contributors. Non-starch polysaccharide intakes were below recommended levels for adults. There is evidence from NDNS and other surveys of a socio-economic gradient in intakes of both sugar and fibre, with higher intakes of non-milk extrinsic sugars as a percentage of energy for adults and lower intakes of non-starch polysaccharide for both adults and children in the lower income groups.

Chapter 4. Background on health outcomes (disease prevention)

4.1. The relationships between dietary carbohydrate intake and cardio-metabolic, colo-rectal and oral health outcomes have been considered in this report. Evidence has been evaluated to assess whether intakes of specific carbohydrates are a factor in the risk for cardiovascular disease, obesity, type 2 diabetes mellitus, and colo-rectal cancers. The relationship between dietary carbohydrate intake and risk factors and markers related to these diseases has also been considered. Table 4.1 lists the risk factors and measures with the references substantiating their use for a given disease or function.

Cardiovascular disease

4.2. Cardiovascular disease is a major cause of disability and premature death throughout the world, and contributes substantially to the escalating costs of health care. Coronary heart disease is the UK's single biggest killer and is responsible for almost 74,000 deaths each year, while stroke causes more than 41,000 deaths in the UK each year (British Heart Foundation, 2014). The underlying pathology is atherosclerosis (whereby arteries become stiffened and narrowed by lesions containing white blood cells and fatty substances, such as cholesterol, called plaques or atheromas), which develops over many years and is usually advanced by the time symptoms occur, generally in middle age (WHO, 2007a).

4.3. The rate of progression of atherosclerosis is influenced by cardiovascular risk factors: tobacco use, diet and physical inactivity (which together result in obesity), elevated blood pressure (hypertension), abnormal blood lipids (dyslipidaemia) and elevated blood glucose (diabetes). See the blood pressure, fasting blood lipid concentration, coronary and vascular factors, inflammatory markers and type 2 diabetes mellitus related risk factors sections in table 4.1. Continuing exposure to these risk factors leads to further progression of atherosclerosis, resulting in unstable atherosclerotic plaques, narrowing of blood vessels and obstruction of blood flow to vital organs, such as the heart and the brain. The clinical manifestations of these diseases include angina, myocardial infarction, transient cerebral ischaemic attacks and strokes (WHO, 2007a).

Obesity

4.4. The prevalence of obesity (BMI 30kg/m^2 or over) in the UK is high; for example within England there was an increase from 13.2% of men in 1993 to 24.4% in 2012 and from 16.4% of women in 1993 to 25.1% in 2012 (Health and Social Care Information Centre, 2013a). Obesity is associated with a range of health problems including type 2 diabetes mellitus, cardiovascular disease and cancer (see body weight and body composition section in table 4.1).

4.5. Obesity occurs when energy intake from food and drink consumption is greater than energy expenditure through the body's metabolism and physical activity over a prolonged period, resulting in the accumulation of excess body fat. There are, however, many complex behavioural and societal factors that combine to contribute to the causes of obesity, e.g. people's latent biological susceptibility interacting with a changing environment that includes more sedentary lifestyles and increased dietary abundance (Foresight, 2007). Dietary factors affecting energy intake and eating motivation are important factors in the aetiology of obesity (Blundell et al., 2010).

Type 2 diabetes mellitus

4.6. In 2013, 6% of the UK population, over 3.2 million people, had diagnosed diabetes, of which 90% had type 2 diabetes mellitus (Diabetes UK, 2014). Plasma glucose concentration is used to diagnose diabetes (WHO, 2006). A considerable body of research has indicated that diabetes is a strong independent risk factors for cardiovascular disease (Sarwar et al., 2010). Often, cardiovascular disease and type 2 diabetes mellitus exist together as they share common modifiable risk factors such as obesity, and in particular elevated central adiposity. Of all serious diseases, type 2 diabetes has the strongest association with obesity and body weight control is a key factor in the prevention of progression from impaired glycaemic control to type 2 diabetes mellitus (American Diabetes Association and National Institute of Diabetes Digestive and Kidney Diseases, 2002; Pi-Sunyer, 2007). See the type 2 diabetes mellitus related risk factors and body weight and body composition sections in table 4.1.

4.7. Diet and lifestyle management is of upmost importance in reducing the incidence of type 2 diabetes mellitus (Diabetes UK, 2013). It is important to identify which dietary aspects improve glycaemia, insulinaemia and insulin resistance in individuals with normal or moderately compromised glycaemic control to further elucidate the role of diet in the prevention of type 2 diabetes mellitus.

Colo-rectal health

4.8. Colo-rectal cancer is the third most common cancer in men and women in the UK, surpassed only by lung and breast cancers in women and lung and prostate cancers in men (Cancer Research UK, 2014b). While a small proportion (<5%) of colo-rectal cancers are attributable to familial cancer syndromes – familial adenomatous polyposis and hereditary non-polyposis colo-rectal cancer – the majority appear to arise sporadically. There is strong epidemiological evidence for environmental factors in the development of sporadic colo-rectal cancer.

4.9. A large body of evidence indicates that adenomatous polyps are the precursor for most colo-rectal cancers (Kinzler & Vogelstein, 1996; Leslie et al., 2002), but only a small proportion of adenomas progress to invasive cancer (Schatzkin & Gail, 2002). There are, however, no other validated risk factors for colo-rectal cancer, and the evidence for faecal parameters in relation to colo-rectal cancer is putative at best (see colo-rectal cancer risk factor and colo-rectal function sections in table 4.1).

4.10. The parameters faecal weight and moisture content and intestinal transit time are quantifiable aspects of colo-rectal function used as measures of laxation. They have, to a limited extent, been associated with different diseases, but these relationships are, as yet, not well defined (Cummings *et al.*, 1992; Lewis & Heaton, 1999). There is no single accepted definition of what constitutes normal laxation (Weaver, 1988). It has been suggested that an intestinal transit time of about two to three days and a defecation frequency of once a day and a faecal moisture of >70%, with a faecal weight of about 150g/day, may be considered adequate for normal laxation in adults and this requires an intake of about 25g/day dietary fibre (for definition see paragraph 2.34, page 17) (EFSA, 2010a).

4.11. Differences in faecal microbiota and short chain fatty acid content and pH have been associated with different diseases, but these relationships are, as yet, not well defined (Björkstén *et al.*, 2001; Kalliomaki *et al.*, 2001; Penders *et al.*, 2007; Takaishi *et al.*, 2008; Kalliomaki *et al.*, 2008; Packey & Sartor, 2009; Schwiertz *et al.*, 2010). In infants, comparisons between breast-feeding and breast milk substitute-feeding have shown differences in these faecal parameters, where breast-milk fed infant faecal microbiota and short chain fatty acid content and pH are defined as optimal or normal (Ogawa *et al.*, 1992; Penders *et al.*, 2006). In children and adults it is not possible to define optimal or normal faecal microbiota and short chain fatty acid content and pH, but they can act as indices for effects on colonic fermentation.

4.12. Constipation is defecation that is unsatisfactory because of infrequent stools, difficult stool passage, or seemingly incomplete defecation. Faeces are often dry and hard, and may be abnormally large or abnormally small. Functional constipation is chronic constipation without a known cause. Constipation is more common in women, the elderly, and during pregnancy (Higgins & Johanson, 2004; Cullen & O'Donoghue, 2007). Reported prevalence rates vary widely, at least partly because criteria for diagnosis vary.

4.13. Constipation was traditionally defined as less than three bowel movements per week (Connell et al., 1965), but many who fit this definition do not consider themselves constipated, while many who do consider themselves constipated also do not fit this definition. Subsequent evidence has suggested effort to defecate, and stool consistency, or form, to be more important (Spiller & Thompson, 2010). While hard stools correlate well with slow transit, and loose stools with fast transit through the colon, difficulty with defecation and stool frequency do not, as they are determined by factors other than colon transit (Spiller & Thompson, 2010).

Oral health

4.14. Humans have two separate dentitions, the deciduous (colloquially known as ‘milk teeth’) and the permanent dentitions. Deciduous teeth begin to erupt during the first year of life and the 20 teeth of the deciduous dentition are normally all present by the age of four. These are progressively shed during the ‘mixed dentition’ phase when some deciduous and some permanent teeth are present. The period characterised as the ‘mixed dentition’ occurs between about six and twelve years of age. The permanent dentition has 32 teeth with three molar teeth erupting behind the deciduous dentition as the jaws grow on each side and including 20 teeth (premolars, canines and incisors) that directly replace the deciduous teeth during the ‘mixed dentition’ phase.

4.15. The permanent dentition starts to erupt into the mouth at the age of six with the first permanent molars erupting behind the deciduous dentition and the central incisors replacing their deciduous predecessors at or about this age. The eruption of the permanent dentition is usually complete by the early twenties, although the presence and pattern of eruption of the third permanent molars (colloquially known as ‘wisdom teeth’) is variable within the population.

4.16. Carbohydrate in the diet exerts an effect after eruption when the teeth are exposed to the oral environment. There are three major disease processes that affect oral health, caries, periodontal disease and wear.

4.17. In the UK, there are many people whose oral health and function does not meet the best possible standards and variations with social class are very apparent (Steele & O’ Sullivan, 2011). In 2009 just under a third of adults (31 per cent) had obvious dental caries, with more men having dental caries than women (34 per cent compared to 28 per cent). Adults from routine and manual occupation households are more likely to have dental decay than those from managerial and professional occupational households (37 per cent compared with 26 per cent).

4.18. In 15-year old children, 13% had obvious dentinal caries in permanent teeth in 2003 (Pitts et al., 2006). In 5 year old children, the experience of obvious dentinal caries in deciduous teeth was 1 in 4 in 2003. As with adults, the prevalence of dental decay is associated with social factors, with children from more deprived backgrounds or from lower social status groups being substantially more likely to have decay in most age groups, with the differences most clear cut amongst younger children (Steele & Lader, 2004).

Markers and measures and their related health outcomes

Table 4.1 Risk factors and measures for cardio-metabolic and colo-rectal health

Risk factors and measures considered	Related outcomes	References
Blood pressure: Blood pressure Systolic blood pressure Diastolic blood pressure	cardio-vascular diseases and renal impairment	(MacMahon *et al.*, 1990; Whelton, 1994; Lewington *et al.*, 2002; Bidani & Griffin, 2002; Shammas, 2007)
Fasting blood lipid concentrations: Total cholesterol HDL cholesterol LDL cholesterol LDL cholesterol: HDL cholesterol Total cholesterol: HDL cholesterol Non-HDL cholesterol Triglycerides Non esterified fatty acids Apolipoproteins (A1 & B) Lipoprotein (a)	cardio-vascular diseases	(Gordon & Rifkind, 1989; Rader *et al.*, 2003; Lewington *et al.*, 2007; Shammas, 2007; Brainin & Heiss, 2009; Goldberg *et al.*, 2011; Miller *et al.*, 2011)
Coronary & vascular factors: Coronary calcification Aortic calcification Carotid plaque formation Carotid artery intimal medial thickness Carotid artery intimal medial thickness change Pulse wave volume Flow mediated dilation Vascular compliance Arterial compliance	cardio-vascular diseases	(Wilson *et al.*, 2001; Kelm, 2002; Witte *et al.*, 2005; Mattace-Raso *et al.*, 2006; Yeboah *et al.*, 2007; Finn *et al.*, 2010; Janner *et al.*, 2011)
Inflammatory markers: Acute phase protein (incl. CRP, serum amyloid A) Cytokines (incl. IL-6, tumour necrosis factor-α) Adhesion molecules Clotting cascade (incl. clotting associated factor PAI-1, fibrinogen, factor VII)	cardio-vascular diseases and type 2 diabetes mellitus	(Ross, 1999; Libby *et al.*, 2002; Kolb & Mandrup-Poulsen, 2005; Basu *et al.*, 2006; Wang *et al.*, 2013)

Risk factors and measures considered	Related outcomes	References
Body weight & body composition: Body weight BMI Fat distribution Fat free mass Total body fat Body fatness Waist circumference Hip circumference Waist to hip ratio	hypertension, cardio-vascular disease, type 2 diabetes mellitus, various cancers and dyslipidaemias	(World Cancer Research Fund & American Institute for Cancer Research, 2007; WHO, 2007b; Klop *et al.*, 2013)
Energy intake & eating motivation	overweight/obesity	(Blundell JE *et al.*, 2008; Hetherington *et al.*, 2013)
Type 2 diabetes mellitus related risk factors: Glycaemia; Insulinaemia; Insulin resistance ; Glycosylated blood proteins	type 2 diabetes mellitus and cardio-vascular disease	(Barr *et al.*, 2007; Shammas, 2007; Brainin & Heiss, 2009; Sarwar *et al.*, 2010; WHO, 2011)
Colo-rectal cancer risk factors: Colo-rectal adenomas	colo-rectal cancer	(Kinzler & Vogelstein, 1996; Leslie *et al.*, 2002; World Cancer Research Fund & American Institute for Cancer Research, 2007)
Colo-rectal function: Faecal weight Intestinal transit time Faecal moisture Faecal microbiota Faecal pH Faecal short chain fatty acid content	colo-rectal function	(Weaver, 1988; Cummings *et al.*, 1992; Ogawa *et al.*, 1992; Lewis & Heaton, 1999; Björkstén *et al.*, 2001; Kalliomaki *et al.*, 2001; Takaishi *et al.*, 2008; Packey & Sartor, 2009; Schwiertz *et al.*, 2010)

Chapter 5. Total carbohydrates

5.1. This assessment is based on 48 prospective cohort studies and 164 randomised controlled trials investigating the relationship between total carbohydrate intake and cardio-metabolic, colo-rectal and oral health outcomes. Four prospective cohort studies and four randomised controlled trials were conducted in children and adolescents and are considered separately at the end of the chapter. Links to the individual systematic reviews and update search are given in Annex 1 and the relevant page or table is given in the text below.

5.2. Evidence on health/disease outcomes have been discussed in detail only where there are sufficient data for a conclusion to be drawn, from studies meeting the pre-agreed inclusion criteria. Outcomes where there are too few such studies to reach a conclusion, are listed at the end of the chapter (see tables 5.1 and 5.2). Outcomes in which the evidence was considered too inconsistent to make a valid judgement are listed in tables 5.3 and 5.4. Therefore, not all 48 cohort studies and 164 randomised controlled trials are listed in the chapter.

5.3. Prospective cohort studies included in this chapter either provide data on total carbohydrate intake, as grams/day or percentage energy, or on dietary patterns. For dietary patterns, a low-carbohydrate-diet score based on the percentage of energy from carbohydrate, fat and/or protein in the diet has been constructed: the higher the score, the more closely the participant followed a low carbohydrate diet. Alternatively, cohort studies have used substitution models to observe the outcome of replacing fat with total carbohydrate or lower, medium or higher glycaemic index carbohydrates.

5.4. For the prospective cohort study meta-analyses the relative risks for total carbohydrate intake are presented for either each 8% energy or each 70 g/day increase, both equivalent to approximately one standard deviation in adult carbohydrate intake, as based on UK data from the National Diet and Nutrition Survey (Bates *et al.*, 2009).

5.5. The randomised controlled trials considered in this section involve manipulating the macronutrient composition of the diet so that subjects consume differing proportions of total carbohydrate. Where there are only a few studies, the evidence has been considered under a heading of higher carbohydrate diets. If there are enough trials, these have been grouped into three main categories according to whether the dominant dietary change was in total carbohydrate and fat, total carbohydrate and protein or whether the changes involved all three macronutrients. For inclusion in a meta-analysis, a 5% difference in energy from total carbohydrate and a 2% difference in fat and/or protein are considered as being meaningful. Where possible, actual consumption is used rather than the intended diet.

5.6. Nearly all trials are conducted in overweight or obese individuals and the diets involve energy restriction goals and most trials result in weight loss. Consideration has been given to whether an effect indicated on the studied parameters could be due to greater weight loss in one of the experimental groups. This is particularly important in trials assessing effects on cardio-metabolic risk markers, as weight loss and gain influence fasting insulin levels and insulin sensitivity (Weyer et al., 2000), blood pressure (Truesdale et al., 2008) and fasting blood lipids (Poobalan et al., 2004).

5.7. Nearly all the trials presenting evidence on diets differing in the proportion of carbohydrate to fat or fat and protein result in a reduction of saturated fatty acid intake in the higher carbohydrate group, as well as affecting the proportion of total fat in the diet. As variation in fat and fatty acid intake affects fasting blood lipid concentrations (Mensink *et al.*, 2003) consideration has been given to whether an effect indicated on the studied parameters could be due to greater fat or saturated fatty acid intake in one of the experimental groups. The degree by which saturated fatty acid intakes are affected differs greatly between trials. For example in some trials saturated fatty acids are reduced and replaced with carbohydrate, whereas in others unsaturated fatty acids are replaced with carbohydrate, leading to different effects.

Cardiovascular disease events

5.8. Four cohort studies were identified that presented evidence on total carbohydrate intake as % energy and incident cardiovascular disease events (Farchi *et al.*, 1995; Esrey *et al.*, 1996; Liu *et al.*, 2000c; Oh *et al.*, 2005), three of which were included in a meta-analysis. One cohort study could not be included in the meta-analysis and indicated no significant association between carbohydrate intake as % energy and incident cardiovascular disease events (Farchi *et al.*, 1995). Two cohort studies were subsequently identified in the update search (Nilsson *et al.*, 2012; Wallstrom *et al.*, 2012) (Cardio-metabolic review, cardiovascular disease chapter p 23-24, 29; Update search table 2).

5.9. No significant association is indicated between carbohydrate intake as % energy and incidence of stroke and coronary events combined (RR 1.00, 95% CI 0.89, 1.12, for each 8% energy increase; p=0.97). The meta-analysis combines different cardiovascular events including both ischaemic and haemorrhagic strokes. These events have different aetiologies and associated risks. The two cohort studies identified in the update search indicate no significant association between carbohydrate intake as % energy and incidence of stroke and coronary events.

5.10. Four cohort studies were identified that presented evidence on total carbohydrate intake as g/day and incident cardiovascular disease events (Fehily *et al.*, 1993; Esrey *et al.*, 1996; Beulens *et al.*, 2007; Drogan *et al.*, 2007). Three cohort studies could not be included in meta-analysis, which left an insufficient number of studies to provide a meta-analysis. No further studies were identified in the update search (Cardio-metabolic review, cardiovascular disease chapter p19-24, 30).

5.11. No significant association is indicated in any of the studies between total carbohydrate intake as g/day and cardiovascular disease events.

Total carbohydrate (g/day and % energy) and cardiovascular disease events
• No association • Moderate evidence

Coronary events

5.12. Three cohort studies were identified that presented evidence on total carbohydrate intake as % energy and incidence of coronary heart disease (Farchi *et al.*, 1995; Esrey *et al.*, 1996; Liu *et al.*, 2000c). One cohort study could not be included in meta-analysis (Farchi *et al.*, 1995), which left an insufficient number of studies to provide a meta-analysis. No further cohort studies were identified in the update search (Cardio-metabolic review, cardiovascular disease chapter p 21, 23-24).

5.13. There is no consistent direction of association between these studies and overall they indicate no significant association between total carbohydrate intake as % energy and incidence of coronary heart disease.

5.14. Three cohort studies were identified that presented evidence on total carbohydrate intake as g/day and incidence of coronary heart disease (Fehily *et al.*, 1993; Esrey *et al.*, 1996; Beulens *et al.*, 2007). Two cohort studies could not be included in meta-analysis, which left an insufficient number of studies to provide a meta-analysis. Two cohort studies were subsequently identified in the update search (Sieri *et al.*, 2010; Burger *et al.*, 2011) (Cardio-metabolic review, cardiovascular disease chapter p 21, 23-24; Update search table 3).

5.15. The three cohort studies identified in the original review indicate no significant association between carbohydrate intake as % energy and incidence of coronary events. Of the two cohort studies identified in the update search, one indicates higher carbohydrate intake as g/day is associated with a higher incidence of coronary events in men, but not in women (Burger *et al.*, 2011). The other study indicates higher incidence of coronary events in women, but not men, with higher carbohydrate intake as g/day (Sieri *et al.*, 2010).

Total carbohydrate (g/day and % energy) and coronary heart disease
• No association • Limited evidence

Stroke

5.16. One cohort study was identified that presented evidence on total carbohydrate intake as % energy and incident stroke events (Oh *et al.*, 2005), one cohort study was subsequently identified in the update search (Wallstrom *et al.*, 2012) (Cardio-metabolic review, cardiovascular disease chapter p 26-27; Update search table 4).

5.17. No significant association is indicated between total carbohydrate intake as % energy and incident stroke events in either study.

5.18. Two cohort studies were identified in the update search that presented evidence on total carbohydrate intake as g/day and incidence of stroke (Oba *et al.*, 2010a; Burger *et al.*, 2011) (Update search table 4).

5.19. No significant association is indicated between total carbohydrate intake as g/day and incident stroke events in either study. There is inadequate evidence to distinguish the impact of total carbohydrate consumption on ischaemic or haemorrhagic stroke separately.

Total carbohydrate (g/day and % energy) and stroke
• No association • Limited evidence

Vascular function

5.20. Three randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate in relation to flow mediated dilatation, all of which were included in a meta-analysis (Keogh *et al.*, 2007; Keogh *et al.*, 2008; Phillips *et al.*, 2008). One trial was subsequently identified in the update search (Wycherley *et al.*, 2010) (Cardio-metabolic review, vascular function chapter p 10; Update search table 115).

5.21. No significant effect is demonstrated for diets differing in the proportion of carbohydrate in relation to flow mediated dilatation (0.68% difference between groups, 95% CI -0.47, 1.83%; p=0.25). The trial identified in the update search reports a decrease in flow mediated dilatation in the lower carbohydrate group compared with the lower fat (higher carbohydrate) group. All trials employ energy restricted weight loss diets with wide ranges of both carbohydrate (between 5% and 60% energy) and fat intakes (between 18% and 37% energy) between groups.

Higher carbohydrate diets and vascular function
• No effect • Limited evidence

Blood pressure

5.22. Thirty one randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate in relation to blood pressure. Five trials were not included in any of the meta-analyses: one trial (Tinker *et al.*, 2008) duplicated the results of the study by Howard et al., (2006); three trials (Wolever & Mehling, 2002; O'Brien *et al.*, 2005; Noakes *et al.*, 2006) did not provide sufficient information and one trial was excluded because the differences in carbohydrate intake were less than 5% energy between groups (Dale *et al.*, 2009) (Cardio-metabolic review, incident hypertension and blood pressure chapter p 47-57). The trials have been stratified according to whether fat or protein, or both, were adjusted as a result of changes in carbohydrate intake.

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

5.23. Fourteen randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to fat on systolic and diastolic blood pressure, all of which were included in the meta-analysis (Golay *et al.*, 2000; Foster *et al.*, 2003; Lovejoy *et al.*, 2003; Clifton *et al.*, 2004; Ley *et al.*, 2004; Dansinger *et al.*, 2005; Ebbeling *et al.*, 2005; Howard *et al.*, 2006b; Ebbeling *et al.*, 2007; Gardner *et al.*, 2007; Maki *et al.*, 2007b; Phillips *et al.*, 2008; Frisch *et al.*, 2009; Sacks *et al.*, 2009). Six trials were subsequently identified in the update search (Foster *et al.*, 2010; Gulseth *et al.*, 2010; Jebb *et al.*, 2010; Howard *et al.*, 2010; Tierney *et al.*, 2011; Brooking *et al.*, 2012) (Cardio-metabolic review, incident hypertension and blood pressure chapter p 49, 53; Update search table 47).

5.24. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat on systolic blood pressure at the end of the intervention (0.71mmHg, 95% CI, -0.71, 2.14mmHg; p=0.33). Of the six trials identified in the update search, four report no significant effect of diets differing in the proportion of carbohydrate to total fat on systolic blood pressure (Gulseth *et al.*, 2010; Jebb *et al.*, 2010; Tierney *et al.*, 2011; Brooking *et al.*, 2012), while the other two provide follow-up measures from trials already included in the meta-analysis and report no significant effect on systolic blood pressure (Foster *et al.*, 2010; Howard *et al.*, 2010). Nearly all trials employ energy restricted weight loss diets that varied both carbohydrate (from 5% to 65% energy) and fat (from 20% to 40% energy) between groups.

Higher carbohydrate, lower fat diets and systolic blood pressure
• No effect • Adequate evidence

5.25. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat on diastolic blood pressure (0.02mmHg, 95% CI, -0.81, 0.86mmHg; p=0.96). Of the five trials identified in the update search which reported on diastolic blood pressure, four report no significant effect of diets differing in the proportion of carbohydrate to total fat on diastolic blood pressure (Gulseth *et al.*, 2010; Jebb *et al.*, 2010; Tierney *et al.*, 2011;

Brooking *et al.*, 2012), while the other provides follow-up measures from trials already included in the meta-analysis and reports no significant effect on diastolic blood pressure (Foster *et al.*, 2010). Nearly all trials employ energy restricted weight loss diets that varied both carbohydrate (from 5% to 65% energy) and fat (from 20% to 40% energy) between groups.

Higher carbohydrate, lower fat diets and diastolic blood pressure
• No effect • Adequate evidence

Higher carbohydrate, average protein diets compared with lower carbohydrate higher protein diets

5.26. Four randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to protein on systolic and diastolic blood pressure, all of which were included in the meta-analysis (Appel *et al.*, 2005; Leidy *et al.*, 2007; Claessens *et al.*, 2009; Delbridge *et al.*, 2009). Three trials were subsequently identified in the update search (Aldrich *et al.*, 2011; Gogebakan *et al.*, 2011; Toscani *et al.*, 2011) (Cardio-metabolic review, incident hypertension and blood pressure chapter p 51, 55 ; Update search table 48). The proportion of carbohydrate in the diets varies between 40-62% energy and protein between 15-30% energy.

5.27. An effect is demonstrated, with higher carbohydrate, average protein diets resulting in less of a reduction in systolic blood pressure (2.17mmHg, 95% CI, 0.08, 4.25mmHg; p=0.04) as compared with the lower carbohydrate, higher protein diets. Body weight is kept constant in one of the trials (Appel *et al.*, 2005), but the other three are weight loss trials. When the difference in weight loss between experimental groups in these trials is plotted on a forest plot it is proportional for each trial to the relative reduction in systolic blood pressure. It is not possible, therefore, to exclude confounding by concomitant weight loss on the effect on systolic blood pressure. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein on systolic blood pressure.

Higher carbohydrate, average protein diets and systolic blood pressure
• Effect • Limited evidence • A higher carbohydrate, average protein diet may result in less of a reduction in systolic blood pressure as compared with a lower carbohydrate, higher protein diet, but it is not possible to exclude confounding by other variables, e.g. less weight loss in one of the experimental groups • The effect is biologically relevant

5.28. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat on diastolic blood pressure (0.81mmHg, 95% CI, -0.83, 2.46mmHg; p=0.33). The three trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein on diastolic blood pressure.

Higher carbohydrate, average protein diets and diastolic blood pressure
• No effect • Limited evidence

Higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate, average or higher fat and higher protein diets

5.29. Seven randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to protein and fat on systolic and diastolic blood pressure, all of which were included in the meta-analysis (Brehm *et al.*, 2003; Meckling *et al.*, 2004; Brehm *et al.*, 2005; Keogh *et al.*, 2007; Meckling & Sherfey, 2007; Keogh *et al.*, 2008; de Luis *et al.*, 2009b). Three trials were subsequently identified in the update search (Klemsdal *et al.*, 2010; Lim *et al.*, 2010; Wood *et al.*, 2012) (Cardio-metabolic review, incident hypertension and blood pressure chapter p 52, 56; Update search table 49).

5.30. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein and fat on systolic blood pressure (0.55mmHg, 95% CI, -3.01, 1.91mmHg; p=0.44). Two of the trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein and fat on systolic blood pressure. One trial reported a significant decrease in systolic blood pressure with a very low carbohydrate, very low fat and high unsaturated fat diets compared to the control diet; however, there was no significant difference between the intervention groups (Lim *et al.*, 2010). All trials employ energy restricted weight loss diets that varied carbohydrate (from 12% to 57% energy), fat (from 54% to 20% energy) and protein (from 18% to 37% energy) between groups.

Higher carbohydrate, lower fat, average protein diets and systolic blood pressure
• No effect • Adequate evidence

5.31. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein and fat on diastolic blood pressure (1.16mmHg, 95% CI, -0.96, 3.27mmHg; p=0.29). Two of the trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein and fat on diastolic blood pressure. One trial reported a significant decrease in diastolic blood pressure with a very low carbohydrate, very low fat and high unsaturated fat diets compared to the control diet; however, there was no significant difference between the intervention groups (Lim *et al.*, 2010). All trials employ energy restricted weight loss diets that varied carbohydrate (from 12% to 57% energy), fat (from 54% to 20% energy) and protein (from 18% to 37% energy) between groups.

Higher carbohydrate, lower fat, average protein diets and diastolic blood pressure
• No effect • Adequate evidence

Fasting blood lipids

5.32. Fifty eight randomised trials were identified that presented evidence on diets differing in the proportion of carbohydrate on fasting blood lipid concentration and were considered for inclusion into meta-analyses. The outcomes examined were fasting total cholesterol, LDL-cholesterol, HDL- cholesterol, triacylglycerol, total cholesterol: HDL- cholesterol ratio, LDL-cholesterol: HDL-cholesterol ratio. For fasting total cholesterol concentration, ten trials could not be included in a meta-analysis (Peterson & Jovanovic-Peterson, 1995; Wolever & Mehling, 2002; Drummond *et al.*, 2003; Foster *et al.*, 2003; O'Brien *et al.*, 2005; Johnston *et al.*, 2006; Dyson *et al.*, 2007; Kirkwood *et al.*, 2007; Dale *et al.*, 2009; Layman *et al.*, 2009) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 65-70). For fasting LDL-cholesterol concentration, six trials could not be included in a meta-analysis (Wolever & Mehling, 2002; Johnston *et al.*, 2006; Kirkwood *et al.*, 2007; Dyson *et al.*, 2007; Lasker *et al.*, 2008; Layman *et al.*, 2009) and for triacylglycerol concentration, eight trials could not be included in a meta-analysis (Peterson & Jovanovic-Peterson, 1995; Wolever & Mehling, 2002; Johnston *et al.*, 2006; Kirkwood *et al.*, 2007; Dyson *et al.*, 2007; Lasker *et al.*, 2008; Dale *et al.*, 2009; Layman *et al.*, 2009) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 122-162). The trials have been stratified according to whether fat or protein, or both, were adjusted as a result of changes in carbohydrate intake.

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

Fasting total cholesterol

5.33. Nineteen randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat on fasting total cholesterol were included in the meta-analysis (Campos *et al.*, 1995; Nelson *et al.*, 1995; Ginsberg *et al.*, 1998; Turley *et al.*, 1998; Zambon *et al.*, 1999; Golay *et al.*, 2000; Colette *et al.*, 2003; Couture *et al.*, 2003; Lovejoy *et al.*, 2003; Clifton *et al.*, 2004; Ley *et al.*, 2004; Pelkman *et al.*, 2004; Segal-Isaacson *et al.*, 2004; Cornier *et al.*, 2005; Lofgren *et al.*, 2005; Petersen *et al.*, 2006; Howard *et al.*, 2006b; Due *et al.*, 2008b; Frisch *et al.*, 2009). Five trials were subsequently identified in the update search (Jebb *et al.*, 2010; Howard *et al.*, 2010; Haufe *et al.*, 2011; Tierney *et al.*, 2011; Brooking *et al.*, 2012) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 65-72; Update search table 62).

5.34. An effect is demonstrated, with diets higher in carbohydrate and lower in fat decreasing fasting total cholesterol concentration (0.16mmol/L, 95% CI -0.28, 0.04; p=0.01), but in some of the trials that also result in weight loss in both experimental groups a difference in the reduction of fasting total cholesterol concentration is reported. The diets vary both carbohydrate (between 5% and 65% energy) and fat (between 18% and 40% energy), including saturated fatty acid intakes, between groups. Saturated fatty acid intakes, in particular, are reduced in most of the higher carbohydrate diets. In those trials which try to maintain saturated fatty acid intakes at similar levels between experimental groups there is still a lower intake of between 1-3% energy in the higher carbohydrate diets (Golay *et al.*, 2000; Couture *et al.*, 2003; Colette *et al.*, 2003; Pelkman *et al.*, 2004; Appel *et al.*, 2005; Due *et al.*, 2008b; Jebb *et al.*, 2010; Tierney *et al.*, 2011; de Souza *et al.*, 2012). It is not possible, therefore, to exclude confounding by concomitant decreases in saturated fatty acid intake or possibly weight loss on the effect on fasting total cholesterol concentration. Of the trials identified in the update search, three report no significant effect of diets differing in the proportion of carbohydrate and fat on fasting total cholesterol concentration (Howard *et al.*, 2010; Tierney *et al.*, 2011; Brooking *et al.*, 2012). One reports a higher carbohydrate and lower fat diet to reduce fasting total cholesterol concentration as compared with a lower carbohydrate and higher fat diet, with a higher saturated fatty acid content, but not a higher monounsaturated fatty acid content (Jebb *et al.*, 2010). The other trial reports weight loss in both experimental groups and a higher carbohydrate and lower fat diet to result in a greater reduction in fasting total cholesterol concentration as compared with a lower carbohydrate and higher fat diet (Haufe *et al.*, 2011).

5.35. An meta-analysis has been conducted for fasting LDL cholesterol concentration, but the heterogeneity is too high to report the meta-analysis pooled estimate (I^2=76%). There is, however, no evidence of an effect of higher carbohydrate, lower fat diets on fasting LDL-cholesterol concentration, which is inconsistent with effects on fasting total cholesterol concentration (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 123-4)

Higher carbohydrate, lower fat diets and fasting total cholesterol concentration
• Effect • Adequate evidence • A diet higher in carbohydrate and lower in fat may decrease fasting total cholesterol concentration, but it is not possible to exclude confounding by other variables, e.g. a concomitant reduction in saturated fat intake and/or weight loss • The effect is biologically relevant

Fasting HDL-cholesterol

5.36. Twenty two randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat on fasting HDL-cholesterol were included in the meta-analysis (Campos *et al.*, 1995; Nelson *et al.*, 1995; Ginsberg *et al.*, 1998; Turley *et al.*, 1998; Zambon *et al.*, 1999; Golay *et al.*, 2000; Wolever & Mehling, 2002; Foster *et al.*, 2003; Colette *et al.*, 2003; Couture *et al.*, 2003; Lovejoy *et al.*, 2003; Clifton *et al.*, 2004; Ley *et al.*, 2004; Pelkman *et al.*, 2004; Segal-Isaacson *et al.*, 2004; Cornier *et al.*, 2005; Bhargava, 2006; Petersen *et al.*, 2006; Howard *et al.*, 2006b; Ebbeling *et al.*, 2007; Due *et al.*, 2008b; Frisch *et al.*, 2009). Seven trials were subsequently identified in the update search (Jebb *et al.*, 2010; Howard *et al.*, 2010; Foster *et al.*, 2010; Haufe *et al.*, 2011; Tierney *et al.*, 2011; de Souza *et al.*, 2012; Brooking *et al.*, 2012) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 93-99; Update search table p 64).

5.37. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat on fasting HDL-cholesterol concentration (0.03mmol/L, 95% CI -0.06, 0.01; p=0.11). Several of the trials are weight loss trials and diets vary both carbohydrate (between 5% and 65% energy) and fat (between 18% and 40% energy) between groups. Of the trials identified in the update search, four report no significant effect of diets differing in the proportion of carbohydrate and fat on fasting HDL-cholesterol concentration (Howard *et al.*, 2010; Haufe *et al.*, 2011; de Souza *et al.*, 2012; Brooking *et al.*, 2012). One trial reports a decrease in fasting HDL-cholesterol concentration on the higher total carbohydrate, lower fat diet compared with the lower carbohydrate, higher fat diets (both higher saturated and higher monounsaturated fatty acid diets (Jebb *et al.*, 2010). Another trial reports an increase in change from baseline fasting HDL-cholesterol concentration in lower carbohydrate, higher fat (both higher saturated and higher monounsaturated fatty acid) diets as compared with higher carbohydrate lower fat diets (Tierney *et al.*, 2011). A follow-up of a trial included in the meta-analysis reports fasting HDL-cholesterol concentration to be lower after the higher carbohydrate, lower fat diet as compared with the lower carbohydrate lower fat diet (Foster *et al.*, 2010).

Higher carbohydrate, lower fat diets and fasting HDL-cholesterol concentration
• No effect • Adequate evidence

Fasting total cholesterol:HDL-cholesterol ratio

5.38. Five randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat on fasting total cholesterol:HDL-cholesterol ratio were included in the meta-analysis (Ginsberg *et al.*, 1998; Colette *et al.*, 2003; Ley *et al.*, 2004; Pelkman *et al.*, 2004; Howard *et al.*, 2006b). One trial was subsequently identified in the update search (Foster *et al.*, 2010) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 195-7; Update search table 64).

5.39. A revised meta-analysis was conducted subsequent to the cardiometabolic health review (Annex 1, additional meta-analyses), which found no statistically significant effect was demonstrated with higher carbohydrate, lower fat diets compared with lower carbohydrate, higher fat diets on fasting total cholesterol:HDL-cholesterol ratio (-0.03, 95% CI -0.12, 0.05; p=0.734). The diets vary both carbohydrate (between 40% and 64% energy) and fat (between 18% and 39% energy), including saturated fatty acid intakes, between groups. The trial identified in the update search reports no significant effect on the total cholesterol:HDL ratio between dietary groups (Foster *et al.*, 2010).

Higher carbohydrate, lower fat diets and fasting total cholesterol:HDL-cholesterol ratio
• No effect • Moderate evidence

Fasting LDL-cholesterol:HDL-cholesterol ratio

5.40. Five randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat on fasting LDL-cholesterol:HDL-cholesterol ratio were included in the meta-analysis (Turley *et al.*, 1998; Zambon *et al.*, 1999; Colette *et al.*, 2003; Pelkman *et al.*, 2004; Due *et al.*, 2008b) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 207-208).

5.41. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat on fasting LDL-cholesterol:HDL cholesterol ratio (0.04, 95% CI -0.36, 0.44; p=0.84). The diets vary both carbohydrate (between 40% and 64% energy) and fat (between 18% and 39% energy) between groups.

Higher carbohydrate, lower fat diets and fasting LDL-cholesterol:HDL-cholesterol ratio
• No effect • Moderate evidence

Higher carbohydrate, average protein diets compared with lower carbohydrate higher protein diets

Fasting total cholesterol

5.42. Six randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein on fasting total cholesterol were included in the meta-analysis (Due *et al.*, 2004; Appel *et al.*, 2005; Noakes *et al.*, 2005; Leidy *et al.*, 2007; Claessens *et al.*, 2009; Delbridge *et al.*, 2009). Two trials were subsequently identified in the update search (Toscani *et al.*, 2011; Gogebakan *et al.*, 2011) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 69-70; Update search table 67). The proportion of carbohydrate in the diets varies from 40-63% energy and protein from 14-31% energy.

5.43. An effect is demonstrated, with higher carbohydrate, average protein diets resulting in less of a reduction in fasting total cholesterol concentration (0.15mmol/L, 95% CI 0.01, 0.28; p=0.03) as compared with the lower carbohydrate, higher protein diet. Body weight is kept constant in one of the trials (Appel *et al.*, 2005), but the other four are weight loss trials. When the difference in weight loss between experimental groups in these trials is plotted on a forest plot it is proportional for each trial to the increase in fasting blood cholesterol. The heterogeneity, however, is too high to report the meta-analysis pooled estimate (I^2=97). It is not possible, therefore, to exclude confounding by concomitant weight loss on the effect on fasting total cholesterol. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein on fasting total cholesterol concentration.

Higher carbohydrate, average protein diets and fasting total cholesterol concentration
• Effect • Limited evidence • A higher carbohydrate, average protein diet may result in less of a reduction in fasting cholesterol concentration as compared with the lower carbohydrate, higher protein diet, but it is not possible to exclude confounding by other variables, e.g. less weight loss in one of the experimental groups • The effect is biologically relevant

Fasting HDL-cholesterol

5.44. Five randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein on fasting HDL-cholesterol concentration were included in the meta-analysis (Due *et al.*, 2004; Appel *et al.*, 2005; Noakes *et al.*, 2005; Claessens *et al.*, 2009; Delbridge *et al.*, 2009). Two trials were subsequently identified in the update search (Toscani *et al.*, 2011; Gogebakan *et al.*, 2011) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 96; Update search table 69). The proportion of carbohydrate in the diets varies from 40-63% energy and protein from 14-31% energy.

5.45. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein on fasting HDL-cholesterol concentration (0.0mmol/L, 95% CI -0.04, 0.04; p=0.98). The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein on fasting HDL-cholesterol concentration.

Higher carbohydrate, average protein diets and fasting HDL-cholesterol concentration
• No effect • Adequate evidence

Fasting LDL-cholesterol

5.46. Five randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein on fasting LDL-cholesterol concentration were included in the meta-analysis (Due *et al.*, 2004; Appel *et al.*, 2005; Noakes *et al.*, 2005; Claessens *et al.*, 2009; Delbridge *et al.*, 2009). Two trials were subsequently identified in the update search (Toscani *et al.*, 2011; Gogebakan *et al.*, 2011) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 125-126; Update search table 68). The proportion of carbohydrate in the diets varies from 40-63% energy and protein from 14-31% energy.

5.47. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein on fasting LDL-cholesterol concentration (0.06mmol/L, 95% CI -0.03, 0.16; p=0.20). The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein on fasting HDL-cholesterol concentration.

Higher carbohydrate, average protein diets and fasting LDL-cholesterol concentration
• No effect • Adequate evidence

Fasting triacylglycerol

5.48. Six randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein on fasting triacylglycerol concentration were included in the meta-analysis (Due *et al.*, 2004; Appel *et al.*, 2005; Noakes *et al.*, 2005; Leidy *et al.*, 2007; Claessens *et al.*, 2009; Delbridge *et al.*, 2009). Two trials were subsequently identified in the update search (Toscani *et al.*, 2011; Gogebakan *et al.*, 2011) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 160-161; Update search table 71). The proportion of carbohydrate in the diets varies from 40-63% energy and protein from 14-31% energy.

5.49. An effect is demonstrated, with higher carbohydrate, average protein diets resulting in less of a reduction in fasting triacylglycerol concentration (0.18mmol/L, 95% CI 0.07, 0.29; p=0.001) as compared with the lower carbohydrate, higher protein diet. The effect could be due to difference in weight loss between the experimental groups, as discussed in paragraphs 5.27 and 5.43 above, in relation to systolic blood pressure and fasting cholesterol concentration. It is not possible, therefore, to exclude confounding by concomitant weight loss on the effect on fasting triacylglycerol. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein on fasting triacylglycerol concentration.

Higher carbohydrate, average protein diets and fasting triacylglycerol concentration
• Effect • Limited evidence • A higher carbohydrate, average protein diet may result in less of a reduction in fasting triacylglycerol concentration as compared with the lower carbohydrate, higher protein diet, but it is not possible to exclude confounding by other variables, e.g. less weight loss in one of the experimental groups • The effect is biologically relevant

Higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate, average or higher fat and higher protein diets

Fasting total cholesterol

5.50. Twenty three randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein and fat on fasting total cholesterol were included in the meta-analysis (Clevidence *et al.*, 1992; Golay *et al.*, 1996; Brehm *et al.*, 2003; Johnston *et al.*, 2004; Meckling *et al.*, 2004; Sharman *et al.*, 2004; Brehm *et al.*, 2005; Dansinger *et al.*, 2005; Ebbeling *et al.*, 2005; Layman *et al.*, 2005; Krauss *et al.*, 2006; McMillan-Price *et al.*, 2006; Noakes *et al.*, 2006; Keogh *et al.*, 2007; Mahon *et al.*, 2007; Meckling & Sherfey, 2007; Maki *et al.*, 2007b; Keogh *et al.*, 2008; Lasker *et al.*, 2008; Phillips *et al.*, 2008; Stoernell *et al.*, 2008; Sacks *et al.*, 2009; de Luis *et al.*, 2009b). Five trials were subsequently identified in the update search (Al-Sarraj *et al.*, 2010; Krebs *et al.*, 2010; Klemsdal *et al.*, 2010; Lim *et al.*, 2010; Wycherley *et al.*, 2010) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 70-71; Update search table 72).

5.51. An effect is demonstrated, with higher carbohydrate, lower fat, average protein diets resulting in a decrease in fasting total cholesterol concentration (-0.26mmol/L, 95% CI -0.40, 0.12; p<0.001), but in some of the trials that also result in weight loss in both experimental groups a difference in the reduction of fasting total cholesterol concentration is reported. The trials vary carbohydrate (from 4% to 67% energy), fat (from 10% to 54% energy), including saturated fatty acid intakes, and protein (from 18% to 37% energy) between groups. Saturated fatty acid intakes, in particular, are reduced in most of the higher carbohydrate diets, although weight loss differences between experimental groups appears to be less of an issue. In those trials which try to maintain saturated fatty acid intakes at similar levels between experimental groups there is still a

lower intake of between 1-3% energy in the higher carbohydrate diets (Golay *et al.*, 1996; Krauss *et al.*, 2006; Mahon *et al.*, 2007; Stoernell *et al.*, 2008; Sacks *et al.*, 2009; de Souza *et al.*, 2012). It is not possible, therefore, to exclude confounding by concomitant decreases in saturated fatty acid intake on the effect on fasting total cholesterol concentration. Three trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein and fat on fasting cholesterol concentration. One trial reported a significant decrease in total cholesterol with very low carbohydrate, very low fat and high unsaturated fat diets compared to the control diet (Lim *et al.*, 2010). One trial reported a significantly greater increase in total cholesterol with a low carbohydrate diet compared to a high carbohydrate low fat diet (Wycherley *et al.*, 2010).

Higher carbohydrate, lower fat, average protein diets and fasting total cholesterol concentration
• Effect • Adequate evidence • A higher carbohydrate, lower fat, average protein diet may decrease fasting total cholesterol concentration, but it is not possible to exclude confounding by other variables, e.g. a concomitant reduction in saturated fat intake and/or weight loss • The effect is biologically relevant

Fasting HDL-cholesterol

5.52. Twenty seven randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein and fat on fasting HDL-cholesterol were included in the meta-analysis (Clevidence *et al.*, 1992; Golay *et al.*, 1996; Brehm *et al.*, 2003; Johnston *et al.*, 2004; Pereira *et al.*, 2004; Sharman *et al.*, 2004; Meckling *et al.*, 2004; Brehm *et al.*, 2005; Ebbeling *et al.*, 2005; Layman *et al.*, 2005; Appel *et al.*, 2005; Krauss *et al.*, 2006; McMillan-Price *et al.*, 2006; Noakes *et al.*, 2006; Keogh *et al.*, 2007; Gardner *et al.*, 2007; Leidy *et al.*, 2007; Mahon *et al.*, 2007; Meckling & Sherfey, 2007; Maki *et al.*, 2007b; Keogh *et al.*, 2008; Phillips *et al.*, 2008; Stoernell *et al.*, 2008; Layman *et al.*, 2009; Morgan *et al.*, 2009; Sacks *et al.*, 2009; de Luis *et al.*, 2009b). Seven trials were subsequently identified in the update search (Al-Sarraj *et al.*, 2010; Hernandez *et al.*, 2010; Klemsdal *et al.*, 2010; Lim *et al.*, 2010; Wycherley *et al.*, 2010; Krebs *et al.*, 2010; Wood *et al.*, 2012) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 97-99; Update search table 74).

5.53. An effect is demonstrated, with higher carbohydrate, lower fat, average protein diets decreasing fasting HDL-cholesterol concentration (-0.06mmol/L, 95% CI -0.10, -0.02; p=0.006), but in some of the trials that also result in weight loss in both experimental groups a lower relative increase of fasting HDL-cholesterol concentration is reported. Nearly all of the trials are weight loss trials. The trials vary carbohydrate (from 4% to 67% energy), fat (from 54% to 10% energy), including saturated fatty acid intakes, and protein (from 18% to 37% energy) between groups. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein and fat on fasting HDL-cholesterol concentration.

5.54. The fat content of the diet may affect fasting HDL-cholesterol concentration, with higher intake raising concentrations, which may in turn be affected by fatty acid composition (Mensink *et al.*, 2003).

Higher carbohydrate, lower fat, average protein diets and fasting HDL-cholesterol concentration
• Effect • Moderate evidence • A higher carbohydrate, lower fat, average protein diet may decrease fasting HDL-cholesterol concentration, but it is not possible to exclude confounding by other variables, e.g. a concomitant reduction in fat intake • The effect is biologically relevant

Fasting LDL-cholesterol

5.55. Twenty four randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein and fat on fasting LDL-cholesterol were included in the meta-analysis (Clevidence *et al.*, 1992; Brehm *et al.*, 2003; Johnston *et al.*, 2004; Pereira *et al.*, 2004; Sharman *et al.*, 2004; Meckling *et al.*, 2004; Brehm *et al.*, 2005; Dansinger *et al.*, 2005; Layman *et al.*, 2005; Krauss *et al.*, 2006; McMillan-Price *et al.*, 2006; Noakes *et al.*, 2006; Gardner *et al.*, 2007; Keogh *et al.*, 2007; Leidy *et al.*, 2007; Mahon *et al.*, 2007; Meckling & Sherfey, 2007; Maki *et al.*, 2007b; Keogh *et al.*, 2008; Phillips *et al.*, 2008; Stoernell *et al.*, 2008; Morgan *et al.*, 2009; Sacks *et al.*, 2009; de Luis *et al.*, 2009b). Six trials were subsequently identified in the update search (Al-Sarraj *et al.*, 2010; Hernandez *et al.*, 2010; Klemsdal *et al.*, 2010; Krebs *et al.*, 2010; Lim *et al.*, 2010; Wycherley *et al.*, 2010) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 126-8; Update search table 73).

5.56. An effect is demonstrated, with higher carbohydrate, lower fat, average protein diets decreasing fasting LDL-cholesterol concentration (-0.27mmol/L, 95% CI -0.36, 0.18; p<0.001), but in some of the trials a difference in the reduction of fasting HDL-cholesterol concentration is reported. Nearly all trials employ energy restricted weight loss diets. The trials vary carbohydrate (from 4% to 67% energy), fat (from 54% to 10% energy), including saturated fatty acid intakes, and protein (from 18% to 37% energy) between groups. Saturated fatty acid intakes, in particular, are reduced in most of the higher carbohydrate diets, although weight loss differences between experimental groups appears to be less of an issue (see paragraph 5.51). It is not possible, therefore, to exclude confounding by concomitant decreases in saturated fatty acid intake on the effect on fasting LDL cholesterol concentration. Four of the trials identified in the update search report a significant effect of diets differing in the proportion of carbohydrate to protein and fat on fasting LDL cholesterol concentration. Two other trials report higher carbohydrate, lower fat, average protein including lower saturated fatty acid, diets reduce fasting LDL-cholesterol concentration (Hernandez *et al.*, 2010; Lim *et al.*, 2010).

Higher carbohydrate, lower fat, average protein diets and fasting LDL-cholesterol concentration
• Effect • Adequate evidence • A higher carbohydrate, lower fat, average protein diets may decrease fasting LDL-cholesterol concentration, but it is not possible to exclude confounding by other variables, e.g. a concomitant reduction in saturated fat intake • The effect is biologically relevant

Fasting triacylglycerol

5.57. Twenty seven randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein and fat on fasting triacylglycerol were included in the meta-analysis (Clevidence *et al.*, 1992; Golay *et al.*, 1996; Brehm *et al.*, 2003; Johnston *et al.*, 2004; Pereira *et al.*, 2004; Meckling *et al.*, 2004; Sharman *et al.*, 2004; Brehm *et al.*, 2005; Dansinger *et al.*, 2005; Ebbeling *et al.*, 2005; Layman *et al.*, 2005; O'Brien *et al.*, 2005; Seshadri *et al.*, 2005; Krauss *et al.*, 2006; McMillan-Price *et al.*, 2006; Noakes *et al.*, 2006; Keogh *et al.*, 2007; Gardner *et al.*, 2007; Mahon *et al.*, 2007; Meckling & Sherfey, 2007; Maki *et al.*, 2007b; Keogh *et al.*, 2008; Phillips *et al.*, 2008; Stoernell *et al.*, 2008; Morgan *et al.*, 2009; Sacks *et al.*, 2009; de Luis *et al.*, 2009b). Seven trials were subsequently identified in the update search (Al-Sarraj *et al.*, 2010; Klemsdal *et al.*, 2010; Lim *et al.*, 2010; Hernandez *et al.*, 2010; Wycherley *et al.*, 2010; Krebs *et al.*, 2010; Wood *et al.*, 2012) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 162-164; Update search table 75).

5.58. An effect is demonstrated, with higher carbohydrate, average protein and lower fat diets resulting in less of a reduction in fasting triacylglycerol concentration, as indicated from the forest plot, but the heterogeneity is too high to report the meta-analysis pooled estimate (I^2=82%). Nearly all trials employ energy restricted weight loss diets. The trials vary carbohydrate (from 4% to 67% energy), fat (from 54% to 10% energy), including saturated fatty acid intakes, and protein (from 18% to 37% energy) between groups. Of the trials identified in the update search five report no significant effect of diets differing in the proportion of carbohydrate to protein and fat on fasting triacylglycerol concentration. One reports an increase in fasting triacylglycerol concentration in response to a higher carbohydrate, average protein and lower fat diet (Wood *et al.*, 2012). One trial reported a significantly greater increase in fasting triacylglycerol with a carbohydrate restricted diet compared to a higher carbohydrate, average protein and lower fat diet (Al-Sarraj *et al.*, 2010)

5.59. The fat content of the diet affects fasting triacylglycerol concentration, with higher intakes reducing concentrations (Mensink *et al.*, 2003), thus the effect may be due to a concomitant change in fat intake in relation to carbohydrate intake.

Higher carbohydrate, lower fat, average protein diets and fasting triacylglycerol concentration
• Effect • Moderate evidence • A higher carbohydrate, lower fat, average protein diet may result in less of a reduction in fasting triacylglycerol concentration as compared with a lower carbohydrate, average or higher fat and higher protein diet, but it is not possible to exclude confounding by other variables, e.g. a concomitant reduction in fat intake • The effect is biologically relevant

Fasting total cholesterol:HDL-cholesterol ratio

5.60. Nine randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein and fat on fasting total cholesterol:HDL-cholesterol ratio were included in the meta-analysis (Clevidence *et al.*, 1992; Johnston *et al.*, 2004; Sharman *et al.*, 2004; Dansinger *et al.*, 2005; Krauss *et al.*, 2006; McMillan-Price *et al.*, 2006; Mahon *et al.*, 2007; Maki *et al.*, 2007b; Layman *et al.*, 2009) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 197).

5.61. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein and fat on fasting cholesterol:HDL-cholesterol ratio (0.06, 95% CI -0.14, 0.27; p=0.42). Nearly all trials employ energy restricted weight loss diets. The trials vary carbohydrate (from 5% to 67% energy), fat (from 46% to 10% energy) and protein (from 18% to 37% energy) between groups.

Higher carbohydrate, lower fat, average protein diets and fasting total cholesterol:HDL-cholesterol ratio
• No effect • Moderate evidence

Fasting LDL-cholesterol:HDL-cholesterol ratio

5.62. Three randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein and fat on fasting LDL-cholesterol:HDL-cholesterol ratio were included in the meta-analysis (Clevidence *et al.*, 1992; Dansinger *et al.*, 2005; Layman *et al.*, 2009) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 209-210).

5.63. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein and fat on fasting LDL-cholesterol:HDL-cholesterol ratio (0.10, 95% CI -0.34, 0.14; p=0.43). All trials employ energy restricted weight loss diets. The trials vary carbohydrate (from 10% to 67% energy), fat (from 41% to 19% energy) and protein (from 15% to 30% energy) between groups.

Higher carbohydrate, lower fat, average protein diets and fasting LDL-cholesterol:HDL-cholesterol ratio
• No effect • Limited evidence

Higher carbohydrate diets and fasting non-HDL cholesterol

5.64. Three randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein and/or fat on fasting non-HDL-cholesterol were included in the meta-analysis (Pelkman *et al.*, 2004; Howard *et al.*, 2006b; Gardner *et al.*, 2007). One trial was subsequently identified in the update search (Toscani *et al.*, 2011) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 130-131; Update search table 70).

5.65. An effect is demonstrated, with higher carbohydrate diets resulting in more of a reduction in fasting non-HDL-cholesterol concentration (-0.03mmol/L, 95% CI 0, -0.06, 0.00; p=0.04) as compared with a lower carbohydrate diet. All trials employ energy restricted weight loss diets. The trials vary carbohydrate (from 18% to 64% energy), fat (from 44% to 20% energy) and protein (from 16% to 30% energy) between groups. One trial aims to maintain saturated fatty acid intakes at similar levels between experimental groups (Pelkman *et al.*, 2004). The other two trials reduce saturated fatty acid intakes in the higher carbohydrate group relative to other experimental groups. One trial contributes 87% to the pooled estimate and results in more weight loss and reduced saturated fatty acid intake in the higher carbohydrate diet group (Howard *et al.*, 2006b) (see paragraph 5.39). It is not possible, therefore, to exclude confounding by concomitant decreases in saturated fatty acid intake on the effect on fasting non-HDL cholesterol concentration. The trial identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein and/or fat on fasting non-HDL-cholesterol concentration.

Higher carbohydrate diets and fasting non-HDL cholesterol concentration
• Effect • Limited evidence • A higher carbohydrate diet may result in more of a reduction in fasting non-HDL-cholesterol concentration as compared with a lower carbohydrate diet, but it is not possible to exclude confounding by other variables, e.g. a concomitant reduction in saturated fat intake and/or weight loss • The effect is biologically relevant

Higher carbohydrate diets and fasting non-esterified fatty acids

5.66. Nine randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein and/or fat on fasting non-esterified fatty acids were included in the meta-analysis (Helge, 2002; Wolever & Mehling, 2003; Due *et al.*, 2004; Cornier *et al.*, 2005; Lofgren *et al.*, 2005; Noakes *et al.*, 2005; McMillan-Price *et al.*, 2006; Claessens *et al.*, 2009; Kirk *et al.*, 2009) (Cardio-metabolic review, the hyperlipidaemias and blood lipids chapter p 191).

5.67. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein and/or fat on fasting non-esterified fatty acid concentration (0mmol/L, 95% CI -0.04, 0.05; p=0.82). All trials employ energy restricted weight loss diets. The trials vary carbohydrate (from 30% to 63% energy), fat (from 41% to 10% energy) and protein (from 15% to 35% energy) between groups.

Higher carbohydrate diets and fasting non-esterified fatty acid concentration
• No effect • Adequate evidence

Higher carbohydrate diets and tumour necrosis factor- α

5.68. Three randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein and/or fat on blood tumour necrosis factor-α were included in the meta-analysis (Sharman & Volek, 2004; Seshadri *et al.*, 2005; de Luis *et al.*, 2007). No further trials were identified in the update search (Cardio-metabolic review, markers of inflammation chapter p 26 and 33).

5.69. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein or fat on blood tumour necrosis factor-α concentration (0.16pg/mL, 95% CI -0.20, 0.51; p=0.39). All trials employ energy restricted weight loss diets. The trials vary carbohydrate (from 8% to 51% energy), fat (from 25% to 63% energy) and protein (from 16% to 25% energy) between groups.

Higher carbohydrate diets and inflammatory markers
• No effect • Limited evidence

Carbohydrate diets and C-reactive protein

5.70. Sixteen randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein and/or fat on C-reactive protein (CRP) (Cardio-metabolic review, markers of inflammation chapter p20-21). Two trials could not be included in the meta-analyses as they did not report the necessary variance data (O'Brien *et al.*, 2005; Stoernell *et al.*, 2008). The trials have been stratified according to whether fat or protein, or both, were adjusted as a result of changes in carbohydrate intake.

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

5.71. Five randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat on blood CRP were included in the meta-analysis (Lovejoy *et al.*, 2003; Dansinger *et al.*, 2005; Johnston *et al.*, 2006; Phillips *et al.*, 2008; Due *et al.*, 2008b) Three trials were subsequently identified in the update search (Petersson *et al.*, 2010; Jebb *et al.*, 2010; Haufe *et al.*, 2011) (Cardio-metabolic review, markers of inflammation chapter p20-1; Update search table 108).

5.72. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat on blood CRP concentration (0.38mg/L, 95% CI -1.10, 0.33; p=0.29). Nearly all trials employ energy restricted weight loss diets. The trials vary both carbohydrate (from 20% to 65% energy) and fat (from 20% to 40% energy) between groups. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to fat on blood CRP concentration.

Higher carbohydrate, lower fat diets and CRP concentration
• No effect • Adequate evidence

Higher carbohydrate, average protein diets compared with lower carbohydrate higher protein diets

5.73. Three randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein on blood CRP were included in the meta-analysis (Due *et al.*, 2005; Noakes *et al.*, 2005; Mahon *et al.*, 2007) One trial was subsequently identified in the update search (Gogebakan *et al.*, 2011) (Cardio-metabolic review, markers of inflammation chapter p23, 29-33; Update search table 109).

5.74. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein on blood CRP concentration (0.49mg/L, 95% CI -0.19, 1.16; p=0.16). All trials employ energy restricted weight loss diets. The trials vary both carbohydrate (from 10% to 60% energy) and protein (from 18% to 37% energy) between groups. The trial identified in the update search reports no significant effect of a diet differing in the proportion of carbohydrate to protein on blood CRP concentration.

Higher carbohydrate, average protein diets and CRP concentration
• No effect • Limited evidence

Higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate, average or higher fat and higher protein diets

5.75. Six randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat and protein on blood CRP were included in the meta-analysis (Pereira *et al.*, 2004; Sharman & Volek, 2004; McMillan-Price *et al.*, 2006; Noakes *et al.*, 2006; de Luis *et al.*, 2007; Keogh *et al.*, 2008). Two trials were subsequently identified in the update search (Klemsdal *et al.*, 2010; Lim *et al.*, 2010) (Cardio-metabolic review, markers of inflammation chapter p24, 29-33; Update search table 110).

5.76. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat and protein on blood CRP concentration (0.09mg/L, 95% CI -0.42, 0.61; p=0.72). Nearly all trials employ energy restricted weight loss diets. The trials vary both carbohydrate (from 10% to 67% energy), fat (from 10% to 54% energy) and protein (from 18% to 37% energy) between groups. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to fat and protein on blood CRP concentration.

Higher carbohydrate, lower fat, average protein and diets CRP concentration
• No effect • Adequate evidence

Type 2 diabetes mellitus

5.77. Seven cohort studies were identified that presented evidence on total carbohydrate intake as % energy and incidence of type 2 diabetes mellitus (Feskens *et al.*, 1995; Monterrosa *et al.*, 1995; Schulze *et al.*, 2004a; Lindstrom *et al.*, 2006; Gunderson *et al.*, 2007; Villegas *et al.*, 2007; Schulze *et al.*, 2008). Five cohort studies could not be included in meta-analysis, which left an insufficient number of studies to enable a meta-analysis to be performed. One cohort study was subsequently identified in the update search (Simila *et al.*, 2012) (Cardio-metabolic review, diabetes chapter p 51, 56-60; Update search table 117).

5.78. The seven cohort studies present conflicting results, but the larger studies that took important covariates into consideration indicate a lack of association between total carbohydrate intake as % energy and incidence of type 2 diabetes mellitus. The cohort study identified in the update search indicates that higher intakes of total dietary carbohydrate as % energy are associated with a lower incidence of type 2 diabetes.

5.79. Nine cohort studies were identified that presented evidence on total carbohydrate intake as g/day and incidence of type 2 diabetes mellitus; two studies could not be included in analysis because the results were unadjusted for any potential confounders (Leonetti *et al.*, 1996; Montonen *et al.*, 2007) The remaining seven cohort studies were included in the meta-analysis (Salmeron *et al.*, 1997a; Salmeron *et al.*, 1997b; Meyer *et al.*, 2000; Hodge *et al.*, 2004; Barclay *et al.*, 2007; Villegas *et al.*, 2007; Schulze *et al.*, 2008). Two further cohort studies were identified in the update search (Sluijs *et al.*, 2010; Mekary *et al.*, 2011) (Cardio-metabolic review, diabetes chapter p 51-60; Update search table p 117). A further meta-analysis was performed which included the studies found in the update search (Greenwood *et al.*, 2013). One study conducted in China presents a very different background diet, and the risk estimates in this cohort are markedly elevated in particular sub groups of women, therefore the findings were not included in the analysis (Villegas *et al.*, 2007). The results from the later meta-analysis were used.

5.80. No significant association is indicated between total carbohydrate intake as g/day and incidence of type 2 diabetes mellitus (RR 0.96, 95% CI 0.86, 1.08 for each 70g/day increase; p=0.5).

Total carbohydrate (% energy and g/day) and type 2 diabetes mellitus
• No association • Moderate evidence

Higher carbohydrate diets and impaired glucose tolerance

5.81. One randomised controlled trial was identified that presented evidence on diets differing in the proportion of carbohydrate in relation to impaired glucose tolerance (Swinburn *et al.*, 2001). Two trials were subsequently identified in the update search (Toscani *et al.*, 2011; Gogebakan *et al.*, 2011) (Cardio-metabolic review, diabetes chapter p 180, 182; Update search table 136).

5.82. No significant effect is reported of diets differing in the proportion of carbohydrate intake on impaired glucose tolerance in any of the trials.

Higher carbohydrate diets and impaired glucose tolerance
• No effect • Limited evidence

Glycaemia and insulinaemia

Cohort studies

5.83. Four cohort studies were identified that presented evidence on total carbohydrate intake as either g/day or % energy and glycaemia (Feskens *et al.*, 1995; Leonetti *et al.*, 1996; Mayer-Davis *et al.*, 2006; Schroeder *et al.*, 2007). No further cohort studies were identified in the update search (Cardio-metabolic review, diabetes chapter p 183).

5.84. The outcome measure is defined as either blood glucose level or area under the curve following a two-hour glucose tolerance test. One study also presents results according to fasting glucose concentration (Mayer-Davis *et al.*, 2006). Due to variation in methodologies a meta-analysis is not possible. None of the studies indicate an association between total carbohydrate intake as either g/day or % energy and glycaemia.

Total carbohydrate (% energy) and Glycaemia
• No association • Moderate evidence

Randomised controlled trials

5.85. Forty six randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to fat and/or protein in relation to measures of glycaemia. Four trials could not be included in the meta-analyses as they did not report the necessary data (Kirkwood *et al.*, 2007; Mahon *et al.*, 2007; Clifton *et al.*, 2008; Dale *et al.*, 2009). The trials have been stratified according to whether fat or protein, or both, were adjusted as a result of changes in carbohydrate intake (Cardio-metabolic review, diabetes chapter p184-186). Where there are insufficient studies to categorise the dietary manipulation under one of the three subheadings, trials have been considered under a heading of higher carbohydrate diets.

Higher carbohydrate diets and area under the curve for blood glucose

5.86. Four randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to fat or protein in relation to blood glucose response two hours after an oral glucose tolerance test (Swinburn *et al.*, 2001; Foster *et al.*, 2003; Lasker *et al.*, 2008; Due *et al.*, 2008a). No further trials were identified in the update search (Cardio-metabolic review, diabetes chapter p185)

5.87. No significant effect is reported for diets differing in the proportion of carbohydrate intake on glucose tolerance test in any of the trials.

Higher total carbohydrate diets and oral glucose tolerance test
• No effect • Adequate evidence

Fasting blood glucose

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

5.88. Twenty six randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat on fasting glucose were included in the meta-analysis (Racette *et al.*, 1995; Golay *et al.*, 1996; Golay *et al.*, 2000; Swinburn *et al.*, 2001; Helge, 2002; Colette *et al.*, 2003; Landry *et al.*, 2003; Lovejoy *et al.*, 2003; Wolever & Mehling, 2003; Clifton *et al.*, 2004; Segal-Isaacson *et al.*, 2004; Dansinger *et al.*, 2005; Lofgren *et al.*, 2005; Raatz *et al.*, 2005; Petersen *et al.*, 2006; Howard *et al.*, 2006b; Ebbeling *et al.*, 2007; Gardner *et al.*, 2007; Maki *et al.*, 2007b; Phillips *et al.*, 2008; Due *et al.*, 2008a; Frisch *et al.*, 2009; Grau *et al.*, 2009; Kirk *et al.*, 2009; Morgan *et al.*, 2009; Sacks *et al.*, 2009). Five trials were subsequently identified in the update search (Goree *et al.*, 2011; Haufe *et al.*, 2011; Shikany *et al.*, 2011; Tierney *et al.*, 2011; Brooking *et al.*, 2012) (Cardio-metabolic review, diabetes chapter p185-188; Update search table 138). Shikany et al. (2011) presents data from the same trial as Howard et al (2006b) but over a longer time period.

5.89. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat on fasting glucose concentration (-0.01mmol/L, 95% CI, -0.06, 0.04; p=0.75). Nearly all trials employ energy restricted weight loss diets that vary both carbohydrate (between 5% and 65% energy) and fat (between 18% and 40% energy) between groups. Of the five trials identified in the update search three report no significant effect, while one reports lower carbohydrate, higher fat diet to increase fasting glucose concentration (Goree *et al.*, 2011).

Higher carbohydrate, lower fat diets and fasting blood glucose concentration
• No effect • Adequate evidence

Higher carbohydrate, average protein diets compared with lower carbohydrate higher protein diets

5.90. Five randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein on fasting glucose were included in the meta-analysis (Due *et al.*, 2004; Noakes *et al.*, 2005; Bowden *et al.*, 2007; Leidy *et al.*, 2007; Claessens *et al.*, 2009). Three trials were subsequently identified in the update search (Weickert *et al.*, 2011; Gogebakan *et al.*, 2011; Toscani *et al.*, 2011) (Cardio-metabolic review, diabetes chapter p189-190; Update search table 139).

5.91. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein on fasting glucose concentration (-0.08mmol/L, 95% CI, -0.26, 0.09; p=0.36). Nearly all trials are energy restricted weight loss trials. The proportion of carbohydrate in the diets varies from 40-63% energy and protein from14-31% energy. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein on fasting glucose concentration.

Higher carbohydrate, average protein diets and fasting blood glucose concentration
• No effect • Adequate evidence

Higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate, average or higher fat and higher protein diets

5.92. Twelve randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat and protein on fasting glucose were included in the meta-analysis analysis (Brehm *et al.*, 2003; Johnston *et al.*, 2004; Meckling *et al.*, 2004; Sharman *et al.*, 2004; Seshadri *et al.*, 2005; McMillan-Price *et al.*, 2006; Noakes *et al.*, 2006; Keogh *et al.*, 2007; Meckling & Sherfey, 2007; Lasker *et al.*, 2008; Keogh *et al.*, 2008; de Luis *et al.*, 2009b). Four trials were subsequently identified in the update search (Klemsdal *et al.*, 2010; Hernandez *et al.*, 2010; Krebs *et al.*, 2010; Lim *et al.*, 2010; Wood *et al.*, 2012) (Cardio-metabolic review, diabetes chapter p190-191; Update search table 140).

5.93. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat and protein on fasting glucose concentration (0.02mmol/L, 95% CI -0.14, 0.17; p=0.84). Nearly all trials employ energy restricted weight loss diets. The trials vary carbohydrate (from 4% to 67% energy), fat (from 10% to 54% energy) and protein (from 18% to 37% energy) between groups. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to fat and protein on fasting glucose concentration.

Higher carbohydrate, lower fat, average protein diets and fasting blood glucose concentration
• No effect • Adequate evidence

Fasting blood insulin

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

5.94. Thirty randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to fat on fasting insulin (Peterson & Jovanovic-Peterson, 1995; Racette *et al.*, 1995; Golay *et al.*, 1996; Golay *et al.*, 2000; Swinburn *et al.*, 2001; Helge, 2002; Colette *et al.*, 2003; Landry *et al.*, 2003; Lovejoy *et al.*, 2003; Wolever & Mehling, 2003; Clifton *et al.*, 2004; Cornier *et al.*, 2005; Dansinger *et al.*, 2005; Lofgren *et al.*, 2005; Petersen *et al.*, 2006; Howard *et al.*, 2006b; Ebbeling *et al.*, 2007; Gardner *et al.*, 2007; Kirkwood *et al.*, 2007; Maki *et al.*, 2007b; Phillips *et al.*, 2008; Tinker *et al.*, 2008; Due *et al.*, 2008b; Dale *et al.*, 2009; Grau *et al.*, 2009; Kirk *et al.*, 2009; Morgan *et al.*, 2009; Sacks *et al.*, 2009; Sloth *et al.*, 2009; de Luis *et al.*, 2009a). Five trials were subsequently identified in the update search (Jebb *et al.*, 2010; Goree *et al.*, 2011; Haufe *et al.*, 2011; Shikany *et al.*, 2011; Tierney *et al.*, 2011; Brooking *et al.*, 2012) (Cardio-metabolic review, diabetes chapter p 209-10; Update search table 141). Shikany et al (2011) presents the data from the same trial as Howard et al (2006b) but over a longer follow-up period.

5.95. Due to variation between the different methodologies used to measure fasting insulin concentration, it was not possible to conduct a meta-analysis. Twenty four trials report no significant effect of diets differing in the proportion of carbohydrate to fat on fasting insulin; six trials do report an effect (Swinburn *et al.*, 2001; Clifton *et al.*, 2004; Dansinger *et al.*, 2005; Due *et al.*, 2008b; Kirk *et al.*, 2009; de Luis *et al.*, 2009a). One of these trials reports that the extent of weight loss predicts the decrease in insulin concentration regardless of dietary group (Dansinger *et al.*, 2005). Nearly all trials employ energy restricted weight loss diets that vary both carbohydrate (between 5% and 65% energy) and fat (between 18% and 40% energy) between groups. It is not possible, therefore, to exclude confounding by concomitant weight loss on fasting insulin concentration. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to fat on fasting insulin concentration.

Higher carbohydrate, lower fat diets and fasting blood insulin concentration
• No effect • Adequate evidence

Higher carbohydrate, average protein diets compared with lower carbohydrate higher protein diets

5.96. Five randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to protein on fasting insulin (Due *et al.*, 2004; Noakes *et al.*, 2005; Clifton *et al.*, 2008; Claessens *et al.*, 2009; Sacks *et al.*, 2009). Two trials were subsequently identified in the update search (Gogebakan *et al.*, 2011; Toscani *et al.*, 2011) (Cardio-metabolic review, diabetes chapter p 211; Update search table 142).

5.97. Due to variation between the different methodologies used to measure fasting insulin concentration, it was not possible to conduct a meta-analysis. All trials report no significant effect from diets differing in the proportion of carbohydrate to protein on fasting insulin concentration. Nearly all trials are weight loss trials. The proportion of carbohydrate in the diets varies between 40-63% energy and protein between 14-31% energy. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein on fasting insulin concentration.

Higher carbohydrate, average protein diets and fasting blood insulin concentration
• No effect • Moderate evidence

Higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate, average or higher fat and higher protein diets

5.98. Seventeen randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to fat and protein on fasting insulin (Golay *et al.*, 1996; Brehm *et al.*, 2003; Johnston *et al.*, 2004; Meckling *et al.*, 2004; Sharman *et al.*, 2004; Dansinger *et al.*, 2005; Layman *et al.*, 2005; Seshadri *et al.*, 2005; McMillan-Price *et al.*, 2006; Noakes *et al.*, 2006; Keogh *et al.*, 2007; Mahon *et al.*, 2007; Meckling & Sherfey, 2007; Keogh *et al.*, 2008; Lasker *et al.*, 2008; Sacks *et al.*, 2009; de Luis *et al.*, 2009a). Three trials were subsequently identified in the update search (Klemsdal *et al.*, 2010; Hernandez *et al.*, 2010; Lim *et al.*, 2010) (Cardio-metabolic review, diabetes chapter p 212; Update search table 143).

5.99. Due to variation between the different methodologies used to measure fasting insulin concentration, it was not possible to conduct a meta-analysis. One trial reports that a higher proportion of dietary carbohydrate to fat and protein increased fasting insulin concentration (Seshadri *et al.*, 2005); whereas all other trials report no significant effect on fasting insulin concentration. Nearly all trials employ energy restricted weight loss diets. The trials vary carbohydrate (from 4% to 67% energy), fat (from 10% to 54% energy) and protein (from 18% to 37% energy) between groups. The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to fat and protein on fasting insulin concentration.

Higher carbohydrate, lower fat, average protein diets and fasting blood insulin concentration
• No effect • Adequate evidence

Insulin response to oral glucose tolerance test

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

5.100. Five randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to fat on the insulin response to an oral glucose tolerance (Swinburn *et al.*, 2001; Foster *et al.*, 2003; Raatz *et al.*, 2005; Due *et al.*, 2008a; Frisch *et al.*, 2009). No further trials were identified in the update search (Cardio-metabolic review, diabetes chapter p 210).

5.101. Due to variation between the different methodologies used to measure fasting insulin concentration, it was not possible to conduct a meta-analysis. Four trials report no significant effect of diets differing in the proportion of carbohydrate to fat on the insulin response to an oral glucose tolerance. One trial reports that a higher carbohydrate, lower fat diet lowered the insulin response to an oral glucose tolerance test (Swinburn *et al.*, 2001).

Higher carbohydrate, lower fat diets and insulin response to oral glucose tolerance test
• No effect • Limited evidence

Higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate, average or higher fat and higher protein diets

5.102. One randomised controlled trial was identified that presented evidence on diets differing in the proportion of carbohydrate to fat and protein on the insulin response to an oral glucose tolerance (Lasker *et al.*, 2008). Two trials were subsequently identified in the update search (Krebs *et al.*, 2010; Hernandez *et al.*, 2010)(Cardio-metabolic review, diabetes chapter p 212; Update search table 143).

5.103. One trial reports that a higher carbohydrate, lower fat and average protein diet lowered the insulin response to an oral glucose tolerance test (Lasker *et al.*, 2008). One trial identified in the update search found that subjects in the lower carbohydrate group had a lower insulin response to an oral glucose tolerance test (Hernandez *et al.*, 2010); whereas the other trial found no effect of the intervention on insulin response (Krebs *et al.*, 2010).

Higher carbohydrate, lower fat, average protein diets and insulin response to oral glucose tolerance test
• No effect • Limited evidence

Insulin resistance/sensitivity

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

5.104. Thirteen randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to fat on measures of insulin resistance/sensitivity (Helge, 2002; Wolever & Mehling, 2002; Foster *et al.*, 2003; Lofgren *et al.*, 2005; Raatz *et al.*, 2005; Johnston *et al.*, 2006; Maki *et al.*, 2007b; Tinker *et al.*, 2008; Due *et al.*, 2008a; Grau *et al.*, 2009; Kirk *et al.*, 2009; Sacks *et al.*, 2009; de Luis *et al.*, 2009a). Five trials were subsequently identified in the update search (Jebb *et al.*, 2010; Goree *et al.*, 2011; Haufe *et al.*, 2011; Shikany *et al.*, 2011; Tierney *et al.*, 2011) (Cardio-metabolic review, diabetes chapter p 229-230; Update search table 144). Shikany et al (2011) reports data from the same trial as Tinker et al (2008) but over a longer time period.

5.105. Due to variation between the different methodologies used to measure insulin resistance/sensitivity, it was not possible to conduct a meta-analysis. Three report an effect of varying the proportion of total carbohydrate to fat on measures of insulin resistance/sensitivity. Two of these trials report improvements in both diet groups, but this is greater in the lower total carbohydrate intervention (Due *et al.*, 2008a; Kirk *et al.*, 2009). One trial reports that in subjects with the *FTO* gene *TT* genotype only, there is a beneficial effect on insulin release in those who consume a higher carbohydrate, lower fat diet (Petersen *et al.*, 2006; Grau *et al.*, 2009). Nearly all trials employ energy restricted weight loss diets that varied both carbohydrate (from 5% to 65% energy) and fat (from 20% to 40% energy) between groups. The four trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to fat on the insulin resistance/sensitivity.

Higher carbohydrate, lower fat diets and insulin resistance/sensitivity
• No effect • Limited evidence

Higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate, average or higher fat and higher protein diets

5.106. Fifteen randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate to fat and protein on measures of insulin resistance/sensitivity (Helge, 2002; Wolever & Mehling, 2002; Johnston *et al.*, 2004; Pereira *et al.*, 2004; Sharman *et al.*, 2004; Ebbeling *et al.*, 2005; O'Brien *et al.*, 2005; Seshadri *et al.*, 2005; McMillan-Price *et al.*, 2006; Mahon *et al.*, 2007; Maki *et al.*, 2007b; Gray *et al.*, 2008; Lasker *et al.*, 2008; Sacks *et al.*, 2009; de Luis *et al.*, 2009a). One trial was subsequently identified in the update search (Krebs *et al.*, 2010) (Cardio-metabolic review, diabetes chapter p 231-232; Update search table 146).

5.107. Due to variation between the different methodologies used to measure insulin resistance/sensitivity, it was not possible to conduct a meta-analysis. Four trials report an effect of diets differing in the proportion of carbohydrate to fat and protein on measures of insulin resistance/sensitivity. Overall, findings from the fifteen trials tend to show improvements in insulin resistance in both dietary groups studied, which is likely to be a reflection of decreasing weights in the majority of the studies. Nearly all trials employ energy restricted weight loss diets that varied carbohydrate (from 12% to 57% energy), fat (from 54% to 20% energy) and protein (from 18% to 37% energy) between groups. The trial identified in the update search reports no significant effect of diets differing in the proportion of carbohydrate to fat and protein on the insulin resistance/sensitivity.

Higher carbohydrate, lower fat, average protein diets and insulin resistance/sensitivity
• No effect • Moderate evidence

Higher carbohydrate diets and haemoglobin A1c

5.108. Six randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate in relation to glycosylated haemoglobin proteins, five of which were included in the meta-analysis (Wolever & Mehling, 2003; Seshadri *et al.*, 2005; Claessens *et al.*, 2009; Frisch *et al.*, 2009; Sloth *et al.*, 2009). One trial could not be included in an analysis because it did not provide any measures of variation (Dyson *et al.*, 2007). One trial was subsequently identified in the update search (Weickert *et al.*, 2011) (Cardio-metabolic review, diabetes chapter p 241-242; Update search table 147).

5.109. No significant effect is demonstrated for diets differing in the proportion of carbohydrate on haemoglobin A1c concentration (0.01%, 95% CI -0.06, 0.05; p=0.82). The largest trial contributed 87% to the pooled estimate (Frisch *et al.*, 2009). The trial identified in the update search reports no significant effect of diets differing in the proportion of carbohydrate in relation to haemoglobin A1c concentration.

Higher carbohydrate diets and haemoglobin A1c concentration
• No effect • Adequate evidence

Obesity

Higher carbohydrate diets and fat free mass

5.110. Four randomised controlled trials presented evidence on diets differing in the proportion of carbohydrate in relation to change in fat free mass. One trial could not be included in the meta-analysis because the percentage energy difference in carbohydrate between the intervention groups was less than 5% (Dale *et al.* 2009). Three trials were included in a meta-analysis (Due *et al.*, 2004; Delbridge *et al.*, 2009; Frisch *et al.*, 2009). Two of the trials varied the proportion of carbohydrate to protein (Due *et al.*, 2004; Delbridge *et al.*, 2009) and the other varied the proportion of carbohydrate to fat (Frisch *et al.*, 2009). One trial was subsequently identified in the update search (Foster *et al.*, 2010), which varied the proportion of carbohydrate to fat and protein (Cardio-metabolic review, obesity chapter p 88-89; Update search table 199).

5.111. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein or fat on loss of fat mass (0.03kg, 95% CI -0.77, 0.83; p=0.95). The trial identified in the update search reports no significant effect of diets differing in the proportion of carbohydrate to fat and protein on fat free mass.

Higher carbohydrate diets and fat free mass
• No effect • Moderate evidence

Higher carbohydrate diets and waist to hip ratio

5.112. Four randomised controlled trials presented evidence on diets differing in the proportion of carbohydrate in relation to waist to hip ratio. One could not be included in a meta-analysis because the percentage of energy from carbohydrates was less than 5% between the intervention groups (McManus *et al.* 2001). Three trials were included in the meta-analysis (Due *et al.*, 2004; Howard *et al.*, 2006a; Gardner *et al.*, 2007). One trial varied the proportion of carbohydrate to protein (Due *et al.*, 2004); one varied the proportion of carbohydrate to fat (Howard *et al.*, 2006a); and the other varied the proportion of carbohydrate to fat and protein (Gardner *et al.*, 2007). One trial was subsequently identified in the update search (Klemsdal *et al.*, 2010) which varied the proportion carbohydrate to fat and protein (Cardio-metabolic review, obesity chapter p 96-97; Update search table 198).

5.113. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein and/or fat on change in waist to hip ratio (0.00, 95% CI -0.01, 0.01; p=0.87). The trial identified in the update search reports waist to hip ratio to be decreased by the higher carbohydrate diet compared with the lower carbohydrate higher fat and protein diet.

Higher carbohydrate diets and waist to hip ratio
• No effect • Limited evidence

Total carbohydrate and body weight change

5.114. Twenty randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate in relation to body weight were considered for inclusion into meta-analysis. Six trials could not be included in the meta-analysis for various reasons (McManus *et al.*, 2001; Ebbeling *et al.*, 2007; Gardner *et al.*, 2007; Clifton *et al.*, 2008; Dale *et al.*, 2009; Sacks *et al.*, 2009) (Cardio-metabolic review, obesity chapter p23-25). The trials have been stratified according to whether fat or fat and/or protein were adjusted as a result of changes in carbohydrate intake.

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

5.115. Eight randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat in relation to on body weight were included in a meta-analysis (Sheppard *et al.*, 1991; Shah *et al.*, 1996; Swinburn *et al.*, 2001; Ebbeling *et al.*, 2005; Bhargava, 2006; Howard *et al.*, 2006a; Frisch *et al.*, 2009; Greenberg *et al.*, 2009). Two follow-up analyses from a trial included in the meta-analysis (Howard *et al.*, 2006a) were identified in the update search (Howard *et al.*, 2010; Shikany *et al.*, 2011) however, these reported on a smaller number of participants and over a shorter time period than the original paper and were not considered further. One additional trial identified in the update search reported no effect of diets differing in the proportion of carbohydrate on body weight (Foster *et al*. 2010) (Cardio-metabolic review, obesity chapter p 23-26; Update search table 192).

5.116. No significant effect is demonstrated for higher carbohydrate, lower fat diets on body weight change (-0.93kg, 95% CI -1.87, 0.01kg; p=0.05). The result is of borderline statistical significance, but is in the opposite direction to the borderline effect for fat mass (see paragraph 5.126)

Higher carbohydrate, lower fat diets and body weight
• No effect • Limited evidence

Higher carbohydrate, average protein diets and higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate higher protein diets and lower carbohydrate, average or higher fat and higher protein diets

5.117. Six randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein or to fat and protein in relation to body weight were included in a meta-analysis (Foster *et al.*, 2003; Due *et al.*, 2004; Dansinger *et al.*, 2005; Keogh *et al.*, 2007; Delbridge *et al.*, 2009; Layman *et al.*, 2009). Three trials were subsequently identified in the update search (Klemsdal *et al.*, 2010; Wycherley *et al.*, 2010; Lim *et al.*, 2010) (Cardio-metabolic review, obesity chapter p 26-27; Update search table 193).

5.118. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein or fat and protein on body weight change (0.48kg, 95% CI -0.79, 1.74kg; p=0.46). The trials identified in the update search report no significant effect of diets differing in the proportion of carbohydrate to protein or fat and protein on body weight. The later follow-up analysis after two years also reports no significant effect.

Higher carbohydrate, average protein diets and higher carbohydrate, lower fat, average protein diets and body weight
• No effect • Adequate evidence

Total dietary carbohydrate and body mass index

5.119. Eight randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate in relation to body mass index were considered for inclusion into meta-analysis. Two trials could not be included in a meta-analysis as they did not report the necessary data (McManus *et al.*, 2001; Dale *et al.*, 2009) (Cardio-metabolic review, obesity chapter p28-29). The trials have been stratified according to whether fat or fat and/or protein were adjusted as a result of changes in carbohydrate intake.

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

5.120. Four randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat in relation to body mass index were included in a meta-analysis (Swinburn *et al.*, 2001; Bhargava, 2006; Howard *et al.*, 2006a; Frisch *et al.*, 2009). No further trials were identified in the update search (Cardio-metabolic review, obesity chapter p 30).

5.121. Due to high heterogeneity between trials (I^2=80%), it is not possible to report the meta-analysis pooled estimate. The forest plot does show that all four trials demonstrate a change in the same direction, with a lower BMI at the end of the intervention following a higher carbohydrate, lower fat diet. The magnitude of this change between trials is inconsistent.

Higher carbohydrate, lower fat diets and body mass index
• Effect • Limited evidence • The direction of the effect demonstrates energy restricted, higher carbohydrate, lower fat diets may be beneficial to reducing body mass index • The effect is biologically relevant

Higher carbohydrate, average protein diets and higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate higher protein diets and lower carbohydrate, average or higher fat and higher protein diets

5.122. Four randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein or to fat and protein in relation to body mass index were included in a meta-analysis (Due *et al.*, 2004; Dansinger *et al.*, 2005; Gardner *et al.*, 2007; Delbridge *et al.*, 2009). One trial was subsequently identified in the update search (Wycherley *et al.*, 2010) (Cardio-metabolic review, obesity chapter p 31; Update search table 193).

5.123. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein or protein and fat on body mass index change (0.26 kg/m^2, 95% CI -0.46, 0.98; p=0.48) The trial identified in the update search reports no significant effect of diets differing in the proportion of carbohydrate to protein or protein and fat on change in body mass index.

Higher carbohydrate, average protein diets and higher carbohydrate, lower fat, average protein diets and body mass index
• No effect • Limited evidence

Total dietary carbohydrate and total body fat

5.124. Six randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate in relation to fat mass were considered for inclusion into meta-analysis. Three trials could not be included in a meta-analysis because the difference in the percentage of energy from carbohydrate was less than 5% (McManus *et al.*, 2001; Clifton *et al.*, 2008; Dale *et al.*, 2009). The trials have been stratified according to whether fat or fat and/or protein were adjusted as a result of changes in carbohydrate intake.

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

5.125. Three randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat and protein in relation to fat mass were included in a meta-analysis (Ebbeling *et al.*, 2007; Gardner *et al.*, 2007; Frisch *et al.*, 2009). No further trials were identified in the update search (Cardio-metabolic review, obesity chapter p 90-91).

5.126. No significant effect is demonstrated for higher carbohydrate, lower fat diets on change in

fat mass (0.30kg, 95% CI -0.01, 0.62; p=0.06). The result is of borderline statistical significance, but is in the opposite direction to the borderline effect for body weight (see paragraph 5.116).

Higher carbohydrate lower fat diets and fat mass
• No effect • Limited evidence

Higher carbohydrate, average protein diets and higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate higher protein diets and lower carbohydrate, average or higher fat and higher protein diets

5.127. Three randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein or to fat and protein in relation to fat mass were included in a meta-analysis (Due *et al.*, 2004; Delbridge *et al.*, 2009; Layman *et al.*, 2009). One trial was subsequently identified in the update search (Foster *et al.*, 2010) (Cardio-metabolic review, obesity chapter p 91, 92; Update search table 199)

5.128. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to protein or fat and protein on change in fat mass (-0.57kg, 95% CI -2.58, 1.44; p=0.58). The trial identified in the update search reports no significant effect on fat mass of diets differing in the proportion of carbohydrate to protein or fat and protein.

Higher carbohydrate, average protein diets and higher carbohydrate, lower fat, average protein diets and fat mass
• No effect • Limited evidence

Total dietary carbohydrate and waist circumference

5.129. Nine randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate in relation to waist circumference were considered for inclusion into meta-analysis. Two trials could not be included in the analysis (McManus *et al.*, 2001; Dale *et al.*, 2009). The trials have been stratified according to whether fat or fat and/or protein were adjusted as a result of changes in carbohydrate intake.

Higher carbohydrate and lower fat diets compared with lower carbohydrate higher fat diets

5.130. Three randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat and protein in relation to waist circumference were included in a meta-analysis (Bhargava, 2006; Howard *et al.*, 2006a; Frisch *et al.*, 2009). Two follow-up analyses from a trial included in the meta-analysis (Howard *et al.*, 2006a) were identified in the update search (Howard *et al.*, 2010; Shikany *et al.*, 2011) however, these reported on a smaller number of participants and over a shorter time period than the original paper and were not considered further (Cardio-metabolic review, obesity chapter p 94; Update search table 197).

5.131. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat on change in waist circumference (0.04cm, 95% CI -1.26, 1.34; p=0.96).

Higher carbohydrate, lower fat diets and waist circumference
• No effect • Limited evidence

Higher carbohydrate, average protein diets and higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate higher protein diets and lower carbohydrate, average or higher fat and higher protein diets

5.132. Four randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to protein or fat and protein in relation to waist circumference were included in a meta-analysis (Due *et al.*, 2004; Dansinger *et al.*, 2005; Keogh *et al.*, 2007; Delbridge *et al.*, 2009). No further trials were identified in the update search (Cardio-metabolic review, obesity chapter p 95).

5.133. Due to high heterogeneity between trials (I^2=86%), it is not possible to report the meta-analysis pooled estimate. The forest plot shows there is no consistent effect overall on change in waist circumference, with only one trial reporting a difference between experimental groups (Due *et al.*, 2004).

Higher carbohydrate, average protein diets and higher carbohydrate, lower fat, average protein diets and waist circumference
• No effect • Limited evidence

Total dietary carbohydrate and energy intake

5.134. Sixty three randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate in relation to energy intake were identified. Seventeen trials could not be included in a meta-analysis as they did not report the necessary data (Schlundt *et al.*, 1993; Stubbs *et al.*, 1996; Saltzman *et al.*, 1997; Harvey-Berino, 1998; Johnstone *et al.*, 2000; McManus *et al.*, 2001; Drummond *et al.*, 2003; Brehm *et al.*, 2003; Ebbeling *et al.*, 2005; Mazlan *et al.*, 2006; Rumpler *et al.*, 2006; Keogh *et al.*, 2007; Meckling & Sherfey, 2007; Park *et al.*, 2007; Kirkwood *et al.*, 2007; de Luis *et al.*, 2007; Clifton *et al.*, 2008; Dale *et al.*, 2009). The majority of these studies found no statistical difference in energy intakes with varying carbohydrate consumption. The remaining studies were analysed according to whether the carbohydrate was replaced with fat and/or protein. For details on trial design, see table 6.3 of the systematic review (Cardio-metabolic review, energy intake and eating motivation chapter p 13-54). Due to high heterogeneity between trials is was not possible to report the meta-analysis pooled estimate for diets differing in the proportion of carbohydrate to fat and for diets differing in the proportion of carbohydrate to protein in relation to energy intake. This evidence for both these dietary manipulations was considered too inconsistent for a conclusion to be drawn and, therefore, they have been listed in table 5.4. However, there is no suggestion of an effect between these two dietary modifications and energy intake. Only the analysis for diets differing in the proportion of carbohydrate to fat and protein is given below.

Higher carbohydrate, lower fat and average protein diets compared with lower carbohydrate, average or higher fat and higher protein diets

5.135. Seventeen randomised controlled trials that presented evidence on diets differing in the proportion of carbohydrate to fat and protein in relation to energy intake were included in a meta-analysis (Borkman *et al.*, 1991; Volek *et al.*, 2004; Meckling *et al.*, 2004; Sharman & Volek, 2004; Brehm *et al.*, 2005; Dansinger *et al.*, 2005; Layman *et al.*, 2005; Nickols-Richardson *et al.*, 2005; Seshadri *et al.*, 2005; McMillan-Price *et al.*, 2006; Dyson *et al.*, 2007; Rankin & Turpyn, 2007; Keogh *et al.*, 2008; Lasker *et al.*, 2008; Stoernell *et al.*, 2008; Layman *et al.*, 2009; Morgan *et al.*, 2009). Only two trials provide an energy restriction goal (Borkman *et al.*, 1991; Volek *et al.*, 2004) (Cardio-metabolic review, energy intake and eating motivation chapter p 68).

5.136. No significant effect is demonstrated for diets differing in the proportion of carbohydrate to fat and protein on energy intake (20kJ, 95% CI -282, 323kJ; p=0.90) (5kcal, 95 % CI -67, 77kcal).

Higher carbohydrate, lower fat, average protein diets and energy intake
• No effect • Adequate evidence

Colo-rectal health

5.137. Six cohort studies were identified that presented evidence on total carbohydrate intake as g/day and colo-rectal cancer incidence, all of which were included in the meta-analysis (Terry *et al.*, 2003; Higginbotham *et al.*, 2004; Michaud *et al.*, 2005; Larsson *et al.*, 2007; Strayer *et al.*, 2007; Howarth *et al.*, 2008). One cohort study was subsequently identified in the update search (Li *et al.*, 2011) (Colo-rectal health review p174-180; Update search table 221)

5.138. No significant association is indicated between carbohydrate intake as g/day and colo-rectal cancer incidence (RR 1.00, 95% CI 0.87, 1.14 for each 70g/day increase; p=0.99). The cohort study identified in the update search also indicates no significant association between total carbohydrate intake as g/day and colo-rectal cancer incidence.

Total carbohydrate (g/day) and colo-rectal cancer
• No association • Adequate evidence

5.139. Four cohort studies were identified that presented evidence on total carbohydrate intake as g/day and colon and rectal cancer incidence separately, all of which were included in the meta-analysis (Terry *et al.*, 2003; Michaud *et al.*, 2005; Larsson *et al.*, 2007; Howarth *et al.*, 2008). One cohort study was subsequently identified in the update search (Li *et al.*, 2011) (Colo-rectal health review p174-180; Update search table 221).

5.140. No significant association is indicated between carbohydrate intake as g/day and colon cancer incidence (RR 0.99, 95% CI 0.89, 1.08 for each 70g/day increase; p=0.75) or rectal cancer incidence (RR 0.99, 95% CI 0.86, 1.14 for each 70g/day increase; p=0.86). The cohort study identified in the update search also indicates no significant association between total carbohydrate intake as g/day and colon or rectal cancer incidence.

Total carbohydrate (g/day) and colon cancer
• No association • Adequate evidence

Total carbohydrate (g/day) and rectal cancer
• No association • Adequate evidence

Children and adolescents

Body mass index and body fatness

5.141. Four cohort studies and four trials were conducted in children and adolescents aged five years or more. All trials were conducted in obese children and adolescents and were weight-loss trials, but only two had a duration of 12 months or more.

Cohort studies

5.142. Three cohort studies were identified that presented evidence on total carbohydrate intake as % energy in relation to change in BMI (Twisk *et al.*, 1998; Boreham *et al.*, 1999; Magarey *et al.*, 2001). One study could not be included in a meta-analysis as it did not report the necessary data (Boreham *et al.*, 1999), which left an insufficient number of trials to enable a meta-analysis to be performed. No further studies were identified in the update search (Cardio-metabolic review, obesity chapter p 22).

5.143. Overall, there was no evidence of an association between total carbohydrate intake and follow-up BMI in any of these studies of children and adolescents.

5.144. Three cohort studies were identified that presented evidence on total carbohydrate intake as % energy in relation to body fatness and fat distribution (Twisk *et al.*, 1998; Boreham *et al.*, 1999; Johnson *et al.*, 2008). The assessments of body fatness were insufficiently comparable to enable a meta-analysis to be performed. No further studies were identified in the update search (Cardio-metabolic review, obesity chapter p 87).

5.145. Two of the studies indicate no association between total carbohydrate intake and body fatness in children and adolescents, but one study indicates a higher total carbohydrate intake is associated with increased triceps skinfold measurement (Magarey *et al.*, 2001).

Total carbohydrate intake (% energy) and body mass index and body fatness
• No association • Limited evidence

Randomised controlled trials

5.146. Two randomised controlled trials were identified that presented evidence on diets differing in the proportion of carbohydrate in relation to change in BMI and body fatness (Ebbeling *et al.*, 2003; Demol *et al.*, 2009).

5.147. The two trials, in obese adolescents, provide contradictory results of a low carbohydrate diet on BMI and total body fat changes as compared with high carbohydrate diets (Ebbeling *et al.*, 2003; Demol *et al.*, 2009). In one trial in 55 obese adolescents aged 12-18 years all dietary groups lost body fat, and changes in BMI, BMI-percentile or fat mass did not vary by diet group (Demol *et al.*, 2009). All diets were equally effective in reducing body fatness as well as reducing insulin resistance and some fasting blood lipid measurements. In the other trial in 14 obese adolescents aged 13 to 21 years the lower carbohydrate diet decreased BMI and fat mass relative to the high carbohydrate group (Ebbeling *et al.*, 2003). Energy intake and insulin resistance were also reduced in the low carbohydrate group. The age range of the subjects, however, also includes adults.

5.148. The two other trials that were identified had a duration of 12 weeks (Sondike *et al.*, 2003) and 13 weeks (Krebs *et al.*, 2010) and explored the effects of a low carbohydrate diet and a high carbohydrate diet in obese adolescents on weight loss and fasting blood lipid concentrations. Overall, no significant differences were reported between the groups on changes in fasting blood lipid concentrations. One of the trials also reports no significant difference between dietary groups on changes to measures of blood glucose, insulin and insulin sensitivity (Krebs *et al.*, 2010).

5.149. Overall, the evidence provided from the trials is insufficient to enable conclusion to be drawn.

Outcomes where there is insufficient evidence

5.150. The tables below detail the exposures and outcomes where there are too few studies or trials that meet the inclusion criteria for the review (e.g. by study duration, publication date, health of subjects, design of intervention) to draw a conclusion. A full description of the studies can be found in the relevant systematic reviews.

Table 5.1 Insufficient evidence-cohort studies

Risk factor/health outcome/measure	**Exposure**
Coronary calcification Aortic calcification Pulse wave velocity Blood pressure Fasting blood lipids Fibrinogen Body weight change Weight gain Fat distribution Energy intake Impaired glucose tolerance Insulinaemia Hb1Ac	Total carbohydrate (g/day and % energy)
Cardiovascular disease events Coronary events Stroke Type 2 diabetes mellitus	Dietary patterns

Table 5.2. Insufficient evidence-randomised controlled trials

Risk factor/health outcome	**Exposure**
Total cardiovascular disease Pulse wave velocity Augmentation index IL-6 Serum-amyloid A, ICAM-1 VCAM-1 E-selectin P-selectin, tPA, Factor VII Total body fat Hip circumference Type 2 diabetes mellitus	Higher carbohydrate diets
Insulin resistance	Higher carbohydrate, lower protein diets

Table 5.3. Inconsistent evidence-cohort studies

Measure	**Exposure**
Body weight	Total carbohydrate

Table 5.4 Inconsistent evidence-randomised controlled trials

Risk factor/health outcome/measure	**Exposure**
Apolipoproteins A-1, B Lipoprotein a Clotting factors Central and peripheral fat Eating motivation	Higher carbohydrate diets
LDL cholesterol TAG Energy intake	Higher carbohydrate and lower fat diets
Waist circumference	Higher carbohydrate, lower fat and/or lower protein diets
Energy intake	Higher carbohydrate, lower protein diets

Summary and conclusions

5.151. This assessment is based on 48 prospective cohort studies and 164 randomised controlled trials investigating the relationship between total carbohydrate intake and the various health/disease outcomes included in this review. There is a lack of evidence on total carbohydrate intake in relation to oral health. Three prospective cohort studies and four randomised controlled trials were conducted in children or adolescents. With observational studies there is substantial potential for biases and the possibility of confounding by an extraneous variable that correlates with both the dependent variable and the independent variable (residual confounding) and any associations must be interpreted with caution.

5.152. For many outcomes, there is insufficient evidence from cohort studies to permit a conclusion to be drawn in terms of total carbohydrate intake. No association is indicated between total carbohydrate intake and the incidence of cardiovascular disease endpoints, type 2 diabetes mellitus, glycaemia or colo-rectal cancer. In children and adolescents limited evidence indicates that there is no association between total carbohydrate intake and body mass index or body fatness.

5.153. Cohort studies estimate total carbohydrate intake from subjects with unrestricted food choice. Total carbohydrate is the sum of the sugars, starches and dietary fibre in the diet and, therefore, a general term that encompasses several nutritional components which are diverse. Any or all of these components maybe increased to raise total carbohydrate intake. Therefore, in some cohort studies it is not reported how carbohydrate intakes were achieved. As the components are linked with differing health outcomes, it may be more difficult to detect an association for total carbohydrate intake. A further limitation is that variation or changes in carbohydrate intakes may affect micronutrient intakes, which is unlikely to be accounted for in cohort studies.

5.154. Randomised controlled trials assess the effect of varying total carbohydrate intake, by reciprocally varying fat, type and quantity, and/or protein intake. These trials indicate no significant effect of varying total carbohydrate intake on vascular function, inflammatory markers and risk factors for type 2 diabetes mellitus. Higher total carbohydrate intake is shown to have mixed effects on fasting blood lipid concentrations, but it is not possible to exclude confounding by a concomitant reduction in saturated fatty acid intake and total fat and/or differences in weight loss between experimental groups; equally, higher carbohydrate intake is shown to affect systolic blood pressure, but it is not possible to exclude confounding by differences in weight loss between experimental groups (see paragraphs 5.6 and 5.7). A higher carbohydrate, average protein diet results in less of a reduction in systolic blood pressure as compared with a lower carbohydrate, higher protein diet, but this appears to be due to greater weight-loss in the higher carbohydrate experimental group rather than to dietary differences. This caveat applies to all cardio-metabolic risk factors investigated in these trials.

5.155. The trials do provide evidence that an energy-restricted higher carbohydrate, lower fat diet, as compared with a lower carbohydrate, higher fat diet, may be beneficial as a dietary strategy to reducing body mass index, but there is high heterogeneity between trials and the evidence is limited due to a relatively small number of trials.

5.156. Overall, prospective cohort studies indicate that total carbohydrate is neither detrimental nor beneficial to cardio-metabolic health. The randomised controlled trials do indicate some effects on cardiovascular risk factors, but it is not possible to exclude confounding by a concomitant reduction in saturated fatty acid intake and total fat and/or differences in weight loss between experimental groups. There is some evidence that an energy restricted, higher carbohydrate, lower fat diet may be an effective strategy in reducing body mass index and body weight. Prospective cohort studies indicate that total carbohydrate intake is not associated with risk of colo-rectal cancer.

Chapter 6. Sugars, sugar alcohols, sugars-sweetened foods and beverages

6.1. This assessment is based on 67 prospective cohort studies and 41 randomised controlled trials investigating the relationship between sugars, individual sugars, sugars-sweetened foods and beverages and sugar alcohol intake and cardio-metabolic, colo-rectal and oral health outcomes. Thirty six prospective cohort studies and nine randomised controlled trials were conducted in children and adolescents and are considered separately at the end of the chapter. Links to the individual systematic reviews and update search are given in Annex 1 and the relevant page or table is given in the text below.

6.2. Evidence on health/disease outcomes have been discussed in detail only where there are sufficient data for a conclusion to be drawn, from studies meeting the pre-agreed inclusion criteria. Outcomes where there are too few such studies to reach a conclusion, are listed at the end of the chapter (see tables 6.1 and 6.2). Outcomes in which the evidence was considered too inconsistent to make a valid judgement are listed in table 6.3. Therefore, not all of the 67 cohort studies and 41 randomised controlled trials are listed in this chapter.

6.3. For the prospective cohort study meta-analyses the relative risks for total sugars intake are presented for 50g/day, for individual sugars 20g/day and for sugars-sweetened beverages 100ml/day.

6.4. The term ‘sugars’ is used in this chapter to refer to all dietary sugars in cohort studies. For randomised controlled trials, ‘sugars’ is used when considering trials where the exposure cannot be clearly attributed to specific sugar(s) types investigated (e.g. sucrose, lactose, glucose or fructose).

6.5. There is a lack of evidence to enable conclusions to be drawn on sugars intake and colo-rectal cancer and no studies were identified for any other colo-rectal health outcome.

Sugars

Coronary events

6.6. Three cohort studies were identified that presented evidence on sugars intake and incidence of coronary events (Fehily *et al.*, 1993; Liu *et al.*, 2000c; Beulens *et al.*, 2007). The exposure measures used in the studies were not sufficiently comparable to enable a meta-analysis to be performed. Two cohort studies were subsequently identified in the update search (Sieri *et al.*, 2010; Burger *et al.*, 2011) (Cardio-metabolic review, cardiovascular disease chapter p 31-32; Update search table 6).

6.7. No significant association is indicated between sugars consumption and incidence of coronary events in any of the studies.

Sugars (g/day) and coronary events
• No association • Moderate evidence

Blood pressure

6.8. Five randomised controlled trials were identified that presented evidence on diets differing in the proportion of sugars in relation to blood pressure (Surwit *et al.*, 1997; Vasilaras *et al.*, 2001; Poppitt *et al.*, 2002; Raben *et al.*, 2002; Black *et al.*, 2006), three of which were included in a meta-analysis (Surwit *et al.*, 1997; Raben *et al.*, 2002; Black *et al.*, 2006). Two very small trials could not be included in a meta-analysis as they did not report the necessary data (Vasilaras *et al.*, 2001; Poppitt *et al.*, 2002) . No further trials were identified in the update search (Cardio-metabolic review, incident hypertension and blood pressure chapter p103-107).

6.9. No significant effect is demonstrated for diets differing in the proportion of sugars on systolic blood pressure (1.4mmHg, 95% CI -5.4, 8.3mmHg; p=0.69) or diastolic blood pressure (3.1mmHg, 95% CI -0.2, 6.3mmHg; p=0.06). The result is of borderline statistical significance. One trial included in the meta-analysis compared the consumption of sucrose sweetened foods and drinks to non-calorically sweetened foods and beverages (Raben *et al.*, 2002). The other two trials compared higher and lower sucrose diets, one of which was a weight loss trial (Surwit *et al.*, 1997).

Sugars and systolic and diastolic blood pressure
• No effect • Limited evidence

Fasting blood lipids

Fasting total cholesterol concentration

6.10. Six randomised controlled trials were identified that presented evidence on diets differing in the proportion of sugars in relation to fasting total cholesterol, of which four were included in a meta-analysis (Ryle *et al.*, 1990; Surwit *et al.*, 1997; Saris *et al.*, 2000; Black *et al.*, 2006). Two trials could not be included in a meta-analysis as they did not report the necessary data (Poppitt *et al.*, 2002; Drummond *et al.*, 2003). No further trials were identified in the update search (Cardio-metabolic review, hyperlipidaemias and blood lipids chapter 244-249).

6.11. No significant effect is demonstrated for diets differing in the proportion of sugars on fasting total cholesterol concentration (0.14mmol/L, 95% CI -0.11, 0.39; p=0.28). Two of the included trials are weight loss trials (Surwit *et al.*, 1997; Saris *et al.*, 2000).

Sugars and fasting total cholesterol concentration
• No effect • Limited evidence

Fasting LDL-cholesterol concentration

6.12. Four randomised controlled trials were identified that presented evidence on diets differing in the proportion of sugars in relation to fasting LDL-cholesterol, of which three were included in a meta-analysis (Surwit *et al.*, 1997; Saris *et al.*, 2000; Black *et al.*, 2006). One trial could not be included in a meta-analysis as it did not report the necessary data (Poppitt *et al.*, 2002). No further trials were identified in the update search (Cardio-metabolic review, hyperlipidaemias and blood lipids chapter p255-257)

6.13. No significant effect is demonstrated for diets differing in the proportion of sugars on fasting LDL-cholesterol concentration (0.10mmol/L, 95% CI -0.18, 0.38; p=0.49). Two of the included trials are weight loss trials (Surwit *et al.*, 1997; Saris *et al.*, 2000).

Sugars and fasting LDL-cholesterol concentration
• No effect • Limited evidence

Fasting HDL-cholesterol concentration

6.14. Four randomised controlled trials were identified that presented evidence on diets differing in the proportion of sugars in relation to fasting HDL-cholesterol, of which three were included in a meta-analysis (Surwit *et al.*, 1997; Saris *et al.*, 2000; Black *et al.*, 2006). One trial could not be included in a meta-analysis as it did not report the necessary data (Poppitt *et al.*, 2002). No further trials were identified in the update search (Cardio-metabolic review, hyperlipidaemias and blood lipids chapter 250-254).

6.15. No significant effect is demonstrated for diets differing in the proportion of sugars on fasting LDL-cholesterol concentration (-0.03mmol/L, 95% CI -0.09, 0.02; p=0.21). Two of the included trials are weight loss trials (Surwit *et al.*, 1997; Saris *et al.*, 2000).

Sugars and fasting HDL-cholesterol concentration
• No effect • Limited evidence

Fasting triacylglycerol concentration

6.16. Five randomised controlled trials were identified that presented evidence on diets differing in the proportion of sugars in relation to fasting triacylglycerol (Surwit *et al.*, 1997; Saris *et al.*, 2000; Poppitt *et al.*, 2002; Sorensen *et al.*, 2005; Black *et al.*, 2006). Three trials could not be included in a meta-analysis as they did not report the necessary data (Poppitt *et al.*, 2002; Sorensen *et al.*, 2005; Black *et al.*, 2006), which left an insufficient number of trials to enable a meta-analysis to be performed. No further trials were identified in the update search (Cardio-metabolic review, hyperlipidaemias and blood lipids chapter p258-261).

6.17. Two trials investigate substitution of dietary fat with either 'simple' or 'complex' carbohydrates; however these terms are not defined (Saris *et al.*, 2000; Poppitt *et al.*, 2002). One reports fasting triacylglycerol concentration to be raised by lower fat, higher simple carbohydrate diet compared with the lower fat higher complex carbohydrate diet (Poppitt *et al.*, 2002), while the other reports no significant effect (Saris *et al.*, 2000). The three remaining trials investigate the effect of higher and lower sucrose diets and report no significant effect on fasting triacylglycerol concentration.

Sugars and fasting triacylglycerol concentration
• No effect • Limited evidence

Energy intake

6.18. Nine randomised controlled trials were identified that presented evidence on diets differing in the proportion of sugars in relation to energy intake, of which seven were included in a meta-analysis (Drummond & Kirk, 1998; Saris *et al.*, 2000; Poppitt *et al.*, 2002; Raben *et al.*, 2002; Brynes *et al.*, 2003; Drummond *et al.*, 2003; Reid *et al.*, 2007). Two trials could not be included in a meta-analysis as they did not report the necessary data (Mazlan *et al.*, 2006; Volp *et al.*, 2008) (Cardio-metabolic review, energy intake and eating motivation chapter p99-106).

6.19. An effect is demonstrated, with diets higher in sugars causing higher energy intakes (1274 kJ/day, 95% CI 889, 1660 kJ/day; p=<0.001) (304 kcal/day, 95% CI 212, 397 kcal/day). As energy intake in excess of requirements can lead to weight gain over time, higher energy consumption is deemed to be detrimental to health. There is variation in study design with respect to how the sugars intervention is delivered. In five of the trials the intervention involves substitution of the macronutrient content of the diet (e.g. in the fat to carbohydrate ratio and the carbohydrate constituents) (Drummond & Kirk, 1998; Saris *et al.*, 2000; Poppitt *et al.*, 2002; Brynes *et al.*, 2003; Drummond *et al.*, 2003). In the two other trials, the intervention involves the replacement of sucrose with non-caloric sweeteners, particularly in drinks (Raben *et al.*, 2002; Reid *et al.*, 2007), thus these trials demonstrate incomplete compensation for the sugars energy by insufficient voluntary reduction in intake of other foods or drinks. Nevertheless, the outcomes of the included

trials are fairly consistent, with very low heterogeneity (I^2=0%). Only one trial provides diets that are designed to be iso-energetic and differ in the type of carbohydrate provided (Brynes *et al.*, 2003). None of the included trials has an energy restriction goal. The first follow up at the end of the intervention ranges from 4 weeks to 6 months.

Sugars and energy intake
• Effect • Adequate evidence • The direction of the effect demonstrates that greater consumption of sugars is detrimental to health • The effect is biologically relevant at a population level in free living individuals not subject to energy restriction

Type 2 diabetes mellitus

6.20. Five cohort studies were identified that presented evidence on diets differing in the proportion of sugars in relation to incidence of type 2 diabetes mellitus , of which three were included in a meta-analysis (Hodge *et al.*, 2004; Barclay *et al.*, 2007; Montonen *et al.*, 2007). Two studies could not be included in a meta-analysis as they did not report the necessary data (Feskens *et al.*, 1995; Janket *et al.*, 2003). One cohort study was subsequently identified in the update search (Sluijs *et al.*, 2010) (Cardio-metabolic review, diabetes and glycaemia chapter p 62-65; Update search table 121).

6.21. The heterogeneity is too high (I^2=82%) to report the meta-analysis pooled estimate, but the studies provide no consistent evidence of an association between diets differing in the proportion of sugars in relation to incidence of type 2 diabetes mellitus. The cohort study identified in the update search reports no significant association between sugars intake and the incidence of type 2 diabetes mellitus.

Sugars (g/day) and type 2 diabetes mellitus
• No association • Limited evidence

Blood glucose

6.22. Five randomised controlled trials were identified that presented evidence on diets differing in the proportion of sugars in relation to blood glucose (Ryle *et al.*, 1990; Surwit *et al.*, 1997; Bantle *et al.*, 2000; Saris *et al.*, 2000; Black *et al.*, 2006). No further trials were identified in the update search (Cardio-metabolic review, diabetes and glycaemia chapter p248-251).

6.23. Due to the variation in the sugars interventions used in these trials, it is not appropriate to combine them in a meta-analysis. There are a diverse range of interventions in the trials. One compares a higher glucose diet to a higher fructose diet (Bantle *et al.*, 2000); one compares a higher glucose, lower soluble fibre diet, which includes a guar preparation, to a lower glucose, higher soluble fibre diet (Ryle *et al.*, 1990); one compares a higher sucrose group and a lower sucrose group (Black *et al.*, 2006) and one randomised subjects to three diets, lower fat, higher 'simple carbohydrates', lower fat 'complex carbohydrates' and a control group (Saris *et al.*, 2000). Two of the trials are weight loss trials (Bantle *et al.*, 2000; Saris *et al.*, 2000). All the trials conducted in adults report no significant effect of diets differing in the proportion of sugars in relation to blood glucose concentration.

Sugars and blood glucose concentration
• No effect • Limited evidence

Blood insulin

6.24. Four randomised controlled trials were identified that presented evidence on diets differing in the proportion of sugars in relation to blood insulin (Ryle *et al.*, 1990; Surwit *et al.*, 1997; Saris *et al.*, 2000; Black *et al.*, 2006). No further trials were identified in the update search (Cardio-metabolic review, diabetes and glycaemia chapter p252-255).

6.25. Due to variation between the different methodologies used to measure insulin concentration, it was not possible to conduct a meta-analysis. Three trials that compare diets higher and lower in sugars report no significant effect of diets differing in the proportion of sugars in relation to blood insulin concentration (Ryle *et al.*, 1990; Saris *et al.*, 2000; Black *et al.*, 2006). One trial reports that subjects fed a higher fructose diet have lower daylong area under the curve insulin concentration as compared with a higher glucose diet (Bantle et al. 2000). Two of the trials are weight loss trials (Bantle *et al.*, 2000; Saris *et al.*, 2000).

Sugars and blood insulin concentration
• No effect • Limited evidence

Individual sugars

Type 2 diabetes mellitus

6.26. Six cohort studies were identified that presented evidence on sucrose intake in relation to incidence of Type 2 diabetes mellitus, of which four were included in a meta-analysis (Meyer *et al.*, 2000; Janket *et al.*, 2003; Montonen *et al.*, 2007; Schulze *et al.*, 2008). Two studies could not be included in a meta-analysis as they did not report the necessary data (Colditz *et al.*, 1992; Monterrosa *et al.*, 1995). No further cohort studies were identified in the update search (Cardio-metabolic review, diabetes and glycaemia chapter p 68-74).

6.27. A borderline association is indicated in the meta-analysis between higher sucrose intake and the reduced incidence of type 2 diabetes mellitus (RR 0.94, 95% CI 0.89, 1.00 for each 20g/day increase; p=0.05). Of the two studies that could not be included in the meta-analysis, however, one reports mean sucrose intake as % energy to be slightly greater in cases than controls (Monterrosa *et al.*, 1995). The other study, which is much larger than the other studies identified, reports a non-significant RR of 1.16 for those in the highest quintile of sucrose intake as compared with the lowest (Colditz *et al.*, 1992). Overall the evidence meeting the inclusion criteria supports an interpretation of no significant association.

Sucrose (g/day) and type 2 diabetes mellitus
• No association • Limited evidence

6.28. Four cohort studies were identified that presented evidence on glucose or fructose intake in relation to incidence of type 2 diabetes mellitus, of which three were included in a meta-analysis (Meyer *et al.*, 2000; Montonen *et al.*, 2007; Schulze *et al.*, 2008). One study could not be included in a meta-analysis as it did not report the necessary data (Janket *et al.*, 2003). No further cohort studies were identified in the update search (Cardio-metabolic review, diabetes and glycaemia chapter p 66-67).

6.29. The heterogeneity is too high for studies investigating glucose (I^2=80%) or fructose (I^2=83%) intakes in relation to incidence of type 2 diabetes mellitus to report the meta-analysis pooled estimate. Two studies specify that glucose and fructose are analysed individually rather than as part of sucrose (Meyer *et al.*, 2000; Montonen *et al.*, 2007), but this is not stated in the other two studies (Janket *et al.*, 2003; Schulze *et al.*, 2008). Two of the studies report an association between higher glucose or higher fructose intakes and higher incidence of type 2 diabetes mellitus (Meyer *et al.*, 2000; Montonen *et al.*, 2007), while the other two studies observe no significant association (Janket *et al.*, 2003; Schulze *et al.*, 2008).

6.30. Three of the cohort studies above also report data on lactose intake in relation to incidence of type 2 diabetes mellitus (Meyer *et al.*, 2000; Janket *et al.*, 2003; Montonen *et al.*, 2007). As one study did not report the necessary data to be included in a meta-analysis (Janket *et al.*, 2003), an insufficient number of studies was available to enable a meta-analysis to be performed. All of these studies report no significant association between lactose intake and the incidence of type 2 diabetes mellitus.

Glucose, fructose or lactose (g/day) and type 2 diabetes mellitus
• No association • Limited evidence

6.31. There is a lack of evidence from trials exploring the effect of individual sugars on cardio-metabolic health outcomes to draw a conclusion (see table 6.1). A commentary on the evidence base for fructose consumption has been provided in Annex 3.

Sugars-sweetened beverages

Type 2 diabetes mellitus

6.32. Six cohort studies were identified that presented evidence on the relationship between sugars-sweetened beverage consumption and the incidence of type 2 diabetes mellitus (Schulze *et al.*, 2004b; Paynter *et al.*, 2006; Dhingra *et al.*, 2007; Montonen *et al.*, 2007; Palmer *et al.*, 2008; Nettleton *et al.*, 2009). These were not combined into a meta-analysis due to variation in both serving size and the definition for sugars-sweetened beverage. One cohort study was subsequently identified in the update search (de Koning *et al.*, 2011) (Cardio-metabolic review, diabetes and glycaemia chapter p137-143; Update search table 122).

6.33. A meta-analysis has been performed which included results from several large cohort studies published subsequently to the original review and update search (Greenwood *et al.*, 2014). Nine cohort studies in eleven publications were identified that presented evidence on the relationship between sugars-sweetened beverages and the incidence of type 2 diabetes mellitus (Schulze *et al.*, 2004b; Paynter *et al.*, 2006; Dhingra *et al.*, 2007; Montonen *et al.*, 2007; Palmer *et al.*, 2008; Nettleton *et al.*, 2009; Odegaard *et al.*, 2010; Bhupathiraju *et al.*, 2013; The InterAct Consortium, 2013; Fagherazzi *et al.*, 2013; Eshak *et al.*, 2013). Two publications reported on the EPIC cohort (The InterAct Consortium, 2013; Fagherazzi *et al.*, 2013), results from the smaller cohort were not included in meta-analyses (Fagherazzi *et al.*, 2013). Two publications reported on the Health Professional's Follow-up Study (de Koning *et al.*, 2011; Bhupathiraju *et al.*, 2013) results from the shorter follow-up are not included in meta-analyses (de Koning *et al.*, 2011). One study could not be included in a meta-analysis as it did not report the necessary data to estimate a dose-response trend (Nettleton *et al.*, 2009). Three cohorts only presented results for all soft drinks combined, and fruit juice or fruit and so were excluded (Montonen *et al.*, 2007; Dhingra *et al.*, 2007; Odegaard *et al.*, 2010; Eshak *et al.*, 2013). One other study included orange and grapefruit juice in its definition of sugars-sweetened soft drinks and was therefore excluded (Paynter *et al.*, 2006). Five cohort studies, in four publications,

that presented evidence on the relationship between sugars-sweetened beverages and the incidence of type 2 diabetes mellitus were included in a meta-analysis (Schulze *et al.*, 2004b; Palmer *et al.*, 2008; Bhupathiraju *et al.*, 2013; The InterAct Consortium, 2013). The studies only included sugars-sweetened carbonated beverages as their exposure measure, except one study that also included diluted syrups (The InterAct Consortium, 2013).

6.34. An association is indicated between greater sugars-sweetened beverage consumption and higher incidence of type 2 diabetes mellitus (RR=1.07, 95% CI 1.05, 1.08 for each 100ml/day increase; p<0.001), with a heterogeneity of I^2=65%.

Sugars-sweetened beverages (ml/day) and type 2 diabetes mellitus
• Association • Moderate evidence • The direction of the association indicates that greater consumption of sugars-sweetened beverages is detrimental to health • The association is biologically relevant

Colon cancer

6.35. A pooled analysis of ten cohort studies was identified that presented evidence on the relationship between sugars-sweetened carbonated soft drinks and incidence of colon cancer (Zhang *et al.*, 2010). At the time of the review, none of the studies included in the pooled analysis had published individually on the relationship between sugars-sweetened carbonated soft drinks and incidence of colon cancer. No further studies were identified in the update search (Colo-rectal health review, p181-183).

6.36. No significant association is indicated between sugars-sweetened carbonated soft drink intake and the incidence of colon cancer (RR 0.94, 95% CI 0.66-1.32, P_{trend}=0.91; comparing consumption greater than 550g/day to non-consumers).

Sugars-sweetened beverages (g/day) and colon cancer
• No association • Adequate evidence

Sugar alcohols

Colo-rectal health

6.37. Three randomised controlled trials were identified that presented evidence on sugar alcohol (polyol) supplementation in relation to faecal wet weight (Van Es *et al.*, 1986; Sinaud *et al.*, 2002; Gostner *et al.*, 2005). The data were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Colo-rectal health review p68).

6.38. Two trials report an effect of lactitol or maltitol supplementation on increasing faecal weight (Van Es *et al.*, 1986; Sinaud *et al.*, 2002). One trial reports no significant effect of the sugar alcohol isomalt on faecal weight (Gostner *et al.*, 2005). From these trials, sugar alcohol intake is found to have a low faecal bulking effect with the resulting increase in faecal wet weight being 0.5-1g per gram of sugar alcohol consumed. Sugar alcohols are likely to be consumed in small amounts in the diet.

Sugar alcohols and faecal weight
• Effect • Limited evidence • The direction of the effect demonstrates that greater consumption of sugar alcohols is of minor benefit to health because of the limited presence of sugar alcohols in the diet • The effect is potentially biologically relevant

6.39. Three randomised controlled trials were identified that presented evidence on sugar alcohol supplementation in relation to faecal bacteria content (Ballongue *et al.*, 1997; Gostner *et al.*, 2005; Finney *et al.*, 2007). The data were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Colo-rectal health review p86).

6.40. Two trials, report lactitol 20g/d and the sugar alcohol isomalt at 30g/day to increase faecal *Bifidobacterium* content (Ballongue *et al.*, 1997; Gostner *et al.*, 2005) In the other trial supplementation of lactitol at 5 or 10g/day has no significant effect on faecal bacterial content in one trial (Finney *et al.*, 2007).

Sugar alcohols and faecal bacteria content
• Effect • Limited evidence • Whether the effect is beneficial or of biological relevance is currently unclear

6.41. Three randomised controlled trials were identified that presented evidence on sugar alcohol supplementation in relation to faecal pH (Ballongue *et al.*, 1997; Gostner *et al.*, 2006; Finney *et al.*, 2007). The data were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Colo-rectal health review p99).

6.42. Two trials report an effect of lactitol supplementation on decreasing faecal pH at doses of 10g/day or more. The other trial reports no significant effect of the sugar alcohol isomalt on faecal pH (Gostner *et al.*, 2006).

Sugar alcohols and faecal pH
• No effect • Limited evidence

6.43. Three randomised controlled trials were identified that presented evidence on sugar alcohol supplementation in relation to faecal short chain fatty acid content (Ballongue *et al.*, 1997; Gostner *et al.*, 2006; Finney *et al.*, 2007). The data were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Colo-rectal health review p99).

6.44. One trial reports an effect of lactitol supplementation on increasing faecal acetate and lowered faecal propionate content (Ballongue *et al.*, 1997). One trial reports no significant effect of supplementation with the sugar alcohol isomalt on faecal short chain fatty acid content (Gostner *et al.*, 2006). It is not possible to determine the effect of the other trial as results are only reported from baseline and graphically (Finney *et al.*, 2007).

Sugar alcohols and faecal short chain fatty acid content
• No effect • Limited evidence

Polydextrose

Colo-rectal health

6.45. Two randomised controlled trials were identified that presented evidence on polydextrose supplementation in relation to faecal bacteria content (Jie *et al.*, 2000; Hengst *et al.*, 2008). The data were insufficiently comparable to enable a meta-analysis to be performed. Two trials were subsequently identified in the update search (Boler *et al.*, 2011; Costabile *et al.*, 2012) (Colo-rectal health review p86; Update search table 216).

6.46. One trial reports an effect of polydextrose supplementation on increasing faecal *Bifidobacterium* content and reducing the number of *Bacteriodes* (Jie *et al.*, 2000). Three trials report no significant effect of polydextrose supplementation on faecal bacterial content (Hengst *et al.*, 2008; Costabile *et al.*, 2012).

Polydextrose and faecal bacteria content
• No effect • Moderate evidence

Children and adolescents

6.47. Eight prospective cohort studies and three randomised controlled trials were conducted in children and adolescents aged five years or more in relation to cardio-metabolic outcomes. Twenty eight prospective cohort studies and six randomised controlled trials were conducted in infants, children and adolescents in relation to oral health outcomes.

Body mass index and body fatness

Sugars-sweetened beverages

Cohort studies

6.48. Six cohort studies were identified that presented evidence on sugars-sweetened beverage intake and BMI in children or adolescents aged 5 years or more (Phillips *et al.*, 2004; Kvaavik *et al.*, 2005; Striegel-Moore *et al.*, 2006; Libuda *et al.*, 2008; Fiorito *et al.*, 2009; Nissinen *et al.*, 2009). The outcome measures were insufficiently comparable to enable a meta-analysis to be performed. No further studies were identified in the update search (Cardio-metabolic review, obesity chapter p 61).

6.49. Three European cohort studies indicate no significant association between sweetened beverage consumption and BMI (Kvaavik *et al.*, 2005; Libuda *et al.*, 2008; Nissinen *et al.*, 2009). Three US cohorts indicate consumption of sugars-sweetened beverages is associated with increasing BMI.

6.50. Three studies also assessed BMI in relation to unsweetened fruit juice consumption and indicate no significant association between baseline consumption and BMI at follow-up (Striegel-Moore *et al.*, 2006; Libuda *et al.*, 2008; Fiorito *et al.*, 2009).

6.51. Collectively, these studies provide conflicting evidence concerning the relationship between sugars-sweetened beverages and BMI, with the US studies tending to find small but positive associations and the European studies tending to report no evidence of a statistical association.

Sugars-sweetened beverages (g/day, energy/day and servings/day) and BMI
• No association • Limited evidence

6.52. Five cohort studies were identified that presented evidence on sugars-sweetened beverage intake and body fatness and fat distribution (Phillips *et al.*, 2004; Mundt *et al.*, 2006; Johnson *et al.*, 2007; Libuda *et al.*, 2008; Fiorito *et al.*, 2009). Two cohorts did not recruit males (Phillips *et al.*, 2004; Fiorito *et al.*, 2009). The outcome measures were insufficiently comparable to enable a meta-analysis to be performed. No further studies were identified in the update search (Cardio-metabolic review, obesity chapter p 132). Percentage body fat was measured in three of the studies (Phillips *et al.*, 2004; Libuda *et al.*, 2008; Fiorito *et al.*, 2009); total body fat in one study (Johnson *et al.*, 2007), and development of fat mass in another (Mundt *et al.*, 2006). The methods used for measuring body fat included skinfold thickness (Libuda *et al.*, 2008), dual-energy X-ray absorptiometry (Mundt *et al.*, 2006; Johnson *et al.*, 2007; Fiorito *et al.*, 2009) and bioimpedance (Phillips *et al.*, 2004).

6.53. No significant association between sugars-sweetened beverage intake and body fatness and fat distribution is indicated by four of the five cohort studies. One study indicates sugars-sweetened beverage consumption at age 5 is a significant predictor of adiposity at ages 5 through 15 years (Fiorito *et al.*, 2009). No significant association between unsweetened fruit juice consumption and body fatness and fat distribution is indicated by the three studies that determine this exposure (Johnson *et al.*, 2007; Libuda *et al.*, 2008; Fiorito *et al.*, 2009). Overall there is no consistent evidence of a change in body fat amount or distribution with sugars-sweetened beverage consumption assessed in childhood or adolescence.

Sugars-sweetened beverages (g/day, energy/day and servings/day) and body fatness
• No association • Limited evidence

Randomised controlled trials

6.54. One randomised controlled trial was identified that presented evidence on the relationship between reducing sugars-sweetened beverage consumption and change in BMI (James *et al.*, 2004). Two randomised controlled trials investigating an effect of reducing sugars-sweetened beverage consumption in children and adolescents in relation to BMI were subsequently identified after the cut-off for both the cardio-metabolic review and the update search (Ebbeling *et al.*, 2012; de Ruyter *et al.*, 2012). The outcome and exposure measures were insufficiently comparable to enable a meta-analysis to be performed.

6.55. A cluster randomised controlled trial of primary school children in the UK provided data on carbonated beverage consumption and BMI and BMI z-score[5] at baseline and one year after an intervention study (James *et al.*, 2004). A total of 644 children aged 7-11 years were randomised to intervention or control groups. Drinks diaries completed over 3 days revealed that carbonated drinks consumption decreased by 0.6 glasses in the intervention group (half of these were carbonated drinks with sugar) but increased by 0.2 glasses in the control group (mean difference 0.7, 95% CI 0.1 to 1.3). A limitation in the interpretation of this trial is that the intervention concerned all types of carbonated beverage, whether non-calorically- or sugars-sweetened which were, therefore, not exclusively sugars-sweetened. The intervention and control group children increased BMI and z-score to a similar degree, with no significant between-group difference in the changes. The percentage of overweight and obese children, however, increased in the control group by 7.5%, compared with a decrease in the intervention group of 0.2% (mean difference 7.7%, 95% CI 2.2 to 13.1%). A follow up two years after the educational programme's discontinuation with data from 434 of the original 644 children reported no differences between the groups in terms of the prevalence of overweight (James *et al.*, 2007).

6.56. A trial in the USA randomised 224 overweight and obese adolescents to either a group who were instructed to consume non-caloric beverages in place of sugars-sweetened drinks for a year or a control group who received no intervention (Ebbeling *et al.*, 2012). The intervention group received home delivery of water or non-calorically sweetened beverages to replace sugars-sweetened beverages for 1 year. After one year the experimental group consumed fewer sugars-sweetened beverages (mean ±sem 0.2±0.4 servings/day compared with 0.9±1.1 servings/day; a serving is 355ml). At two years, the consumption of sugars-sweetened beverages remained lower (mean ±sem 0.4±0.5 servings/day compared with 0.8±0.8 servings/day) and the consumption of unsweetened beverages remained higher in the experimental group. At one year, the intervention group gained significantly less weight (mean difference ± sem -1.9±0.9 kg; p= 0.04) and had a smaller increase in BMI (mean difference ± sem -0.57±0.28 kg/m^2; p = 0.045) compared with the control group; percentage fat mass change, as determined by electrical impedance measures, also tended to be less (mean difference ±SD -0.7±0.4%; p=0.12) in comparison with the control group. After one year, the experimental group no longer received non-caloric drinks and both groups were followed up for a further year without receiving any intervention. At the end of the two year period there was no significant difference in weight (mean difference ± sem -0.8±1.4 kg, P = 0.55) or BMI (mean difference ± sem -0.30±0.40 kg/m^2, P = 0.46) between the groups.

[5] A BMI z score or standard deviation score indicates how many units (of the standard deviation) a child's BMI is above or below the average BMI value for their age group and sex. For instance, a z score of 1.5 indicates that a child is 1.5 standard deviations above the average value, and a z score of –1.5 indicates a child is 1.5 standard deviations below the average value. (National Obesity Observatory, 2011)

6.57. A double-blinded placebo-controlled trial randomised 641 normal-weight Dutch children to receive 250 ml/day of a sugars-free, non-caloric sucralose-sweetened beverage or a similar sucrose-containing beverage that provided 435kJ (104 kcal) per serving (de Ruyter *et al.*, 2012). Compliance was measured by assessing urinary sucralose in a random sample of children. At the end of the 18 month intervention children receiving the non-calorically sweetened beverage had a lower BMI z-score, skinfold thickness, waist to hip ratio and less fat mass as determined by electrical impedance measures in comparison with children receiving the sucrose sweetened beverage. Measurements were also obtained from those children that did not complete the trial showing the results were similar for drop-outs. This trial has good retention rates, is sufficiently powered and provides a direct test of whether non-caloric beverage intake can have an impact on weight gain.

6.58. One of the trials investigates all carbonated beverages, not just sugars-sweetened ones, in relation to BMI, so this does not represent a direct test of the effect of sugars-sweetened beverages (James *et al.*, 2004). The other two trials do provide direct tests of the effect of sugars-sweetened beverages and both report effects of the consumption of sugars-sweetened beverages on increasing weight gain and BMI (Ebbeling *et al.*, 2012; de Ruyter *et al.*, 2012). These trials demonstrate inadequate compensation for the sugars energy by insufficient voluntary reduction in intake of other foods or drinks, which leads to weight gain. One trial, in particular, is of a high standard (de Ruyter *et al.*, 2012), and warrants upgrading the judgement that can be reached from two trials as inconclusive to effect based on limited evidence (see Annex 2, paragraph A2.21).

Sugars-sweetened beverages and BMI
• Effect • Limited evidence • The direction of the effect demonstrates that greater consumption of sugars-sweetened beverages is detrimental to health • The effect is biologically relevant

Oral Health

Sugars

6.59. Five cohort studies were identified that presented evidence on the relationship between sugars intake and the incidence of dental caries in the mixed and permanent dentition in children, three of which adjusted their results for tooth brushing (Rugg-Gunn *et al.*, 1984; Rugg-Gunn *et al.*, 1987; Szpunar *et al.*, 1995; Ruottinen *et al.*, 2004) one of which did not (Campain *et al.*, 2003). Two papers report on the same cohort study (Rugg-Gunn *et al.*, 1984; Rugg-Gunn *et al.*, 1987). The data on measures of dietary exposure, caries incidence/prevalence and risk assessment methods were insufficiently comparable to enable a meta-analysis to be performed. No further studies were identified in the update search (Oral health review p34 and p77).

6.60. All three cohort studies that adjust their results for tooth brushing report an association between higher sugars consumption and greater risk of developing dental caries of some type. One study observes an association with fissure caries only, but not for overall two year caries increment or approximal surface caries (Rugg-Gunn *et al.*, 1984; Rugg-Gunn *et al.*, 1987). The study that did not adjust its results for tooth brushing observes no significant association between intake of higher sugars consumption and the incidence of dental caries (Campain *et al.*, 2003).

Amount of sugars consumed (g/day and % energy) and dental caries in mixed and permanent dentition
• Association • Moderate evidence • The direction of the association indicates that greater consumption of sugars is detrimental to oral health • The association is biologically relevant

6.61. Three cohort studies were identified that presented evidence on the relationship between frequency of sugars intake and the incidence of dental caries in the mixed and permanent dentition in children, all of which adjusted their results for tooth brushing (Rugg-Gunn *et al.*, 1984; Rugg-Gunn *et al.*, 1987; Szpunar *et al.*, 1995; Levine *et al.*, 2007). Two papers report on the same cohort study (Rugg-Gunn *et al.*, 1984; Rugg-Gunn *et al.*, 1987). The data on measures of dietary exposure, caries incidence/prevalence and risk assessment methods were insufficiently comparable to enable a meta-analysis to be performed. No further studies were identified in the update search (Oral health review p36 and p77).

6.62. All three cohort studies that adjust their results for tooth brushing report no significant association between the frequency of sugars consumption and risk of developing dental caries. One study does report the frequency of bedtime sugars consumption from drinks to be associated with greater dental caries prevalence (Levine *et al.*, 2007).

Frequency of sugars (servings/day) consumed and dental caries in mixed and permanent dentition
• No association • Limited evidence

Sugars-containing beverages[6]

6.63. Seven cohort studies in eleven publications were identified that presented evidence on the relationship between sugars-containing beverage consumption and the incidence of dental caries in deciduous dentition in children, all of which adjusted their results for tooth brushing (Grytten *et al.*, 1988; Grindefjord *et al.*, 1995; Grindefjord *et al.*, 1996; Tada *et al.*, 1999; Levy *et al.*, 2003; Mariri *et al.*, 2003; Sakuma *et al.*, 2007; Ismail *et al.*, 2008; Warren *et al.*, 2008; Lim *et al.*, 2008; Ismail *et al.*, 2009). Three cohorts were reported in more than one publication: (Grindefjord *et al.*, 1995; Grindefjord *et al.*, 1996) reported on one cohort; (Ismail *et al.*, 2008; Lim *et al.*, 2008; Ismail *et al.*, 2009) reported on one cohort; and (Mariri *et al.*, 2003; Levy *et al.*, 2003) reported on one cohort. The data on measures of dietary exposure, caries incidence/prevalence and risk assessment methods were insufficiently comparable to enable a meta-analysis to be performed. No further studies were identified in the update search (Oral health review p37-39).

6.64. An association is indicated between greater consumption of sugars-containing beverages and increased dental caries incidence in deciduous dentition in five cohort studies (Grindefjord *et al.*, 1995; Grindefjord *et al.*, 1996; Levy *et al.*, 2003; Mariri *et al.*, 2003; Sakuma *et al.*, 2007; Ismail *et al.*, 2008; Warren *et al.*, 2008; Lim *et al.*, 2008; Ismail *et al.*, 2009). The two other cohort studies observe no significant association between sugars-containing beverage consumption and the development of dental caries at 36 months (Grytten *et al.*, 1988) or between-meal sweet drink intake and 18 months dental caries increment in deciduous dentition (Tada *et al.*, 1999).

Amount and frequency of sugars-containing beverages consumption (servings/day or ounces/day) and dental caries in deciduous dentition
• Association • Adequate evidence • The direction of the association indicates that greater consumption of sugars-sweetened beverages is detrimental to oral health • The association is biologically relevant

[6] For studies on the deciduous dentition, sugars-sweetened beverages included sugar sweetened carbonated beverages, non-carbonated fruit drinks and fruit juice. Some studies did not specify what they defined as sugars-sweetened/containing beverages.

Sugars-containing foods[7]

6.65. Eleven cohort studies were identified that presented evidence on the relationship between the amount of sugars-containing foods and/or sugars confectionery consumed and the incidence of dental caries in deciduous dentition in children, five of which adjusted their results for tooth brushing (Grytten *et al.*, 1988; Grindefjord *et al.*, 1995; Grindefjord *et al.*, 1996; Tada *et al.*, 1999; Levy *et al.*, 2003; Mariri *et al.*, 2003; Sakuma *et al.*, 2007) and six of which did not (Persson *et al.*, 1985; Wilson & Ashley, 1989; Holbrook, 1993; Holbrook *et al.*, 1995; Pienihakkinen *et al.*, 2004; Law & Seow, 2006) Two cohorts were reported in more than one publication: (Grindefjord *et al.*, 1995; Grindefjord *et al.*, 1996) reported on one cohort; and (Mariri *et al.*, 2003; Levy *et al.*, 2003) reported on one cohort. The data on measures of dietary exposure, caries incidence/prevalence and risk assessment methods were insufficiently comparable to enable a meta-analysis to be performed. One study was subsequently identified in the update search (Fontana *et al.*, 2011), which did not adjust for tooth brushing (Oral health review p37-39 and p70; Update search table 233).

6.66. Two studies that adjust for tooth brushing indicate an association between greater consumption of sugars confectionery and greater risk of dental caries at 3.5 years and 18 months (Grindefjord *et al.*, 1995; Grindefjord *et al.*, 1996; Sakuma *et al.*, 2007). The three other studies that adjust for tooth brushing indicate no significant association between consumption of sugars-sweetened food or sweet confectionery and dental caries (Grytten *et al.*, 1988; Tada *et al.*, 1999; Mariri *et al.*, 2003). All six of the cohort studies that do not adjust their results for tooth brushing indicate an association between higher consumption of sugars-containing foods or confectionery and greater risk of dental caries. The study identified in the update search reports that snacking on candy, cake, cookies, ice cream or dried fruit are not associated with dental caries incidence, but two dietary snacking behaviours are associated with caries ('child does not snack on fresh fruit' and 'child does snack on popcorn').

Amount and frequency of sugars-containing foods and/or sugars confectionery consumption (servings/day or servings/week) and dental caries in deciduous dentition
• Association • Limited evidence • The direction of the association indicates that greater consumption of sugars-containing foods and/or sugars confectionery is detrimental to oral health • The association is biologically relevant

[7] Sugars containing foods include sweets/candy, between meal sweet food intake, sugars-containing foods, sugars-containing snacks, sucrose rich foods, sugar-starch foods.

6.67. Eight cohort studies in nine publications were identified that presented evidence on the relationship between the frequency of consumption of sugars-containing foods and/ sugars confectionery and the incidence of caries in the mixed/permanent dentition, five of which adjusted their results for tooth brushing (Mattila *et al.*, 2001; Leroy *et al.*, 2005; Mattila *et al.*, 2005; Kallestal & Fjelddahl, 2007; Ollila & Larmas, 2007; Tamaki *et al.*, 2009) and three of which that did not (Wilson & Ashley, 1989; Petti & Hausen, 2000; Bruno-Ambrosius *et al.*, 2005). Two articles reported on same cohort (Mattila *et al.*, 2001; Mattila *et al.*, 2005), which followed-up a birth cohort until the children were 10 years of age. The data on measures of dietary exposure, caries incidence/prevalence and risk assessment methods were insufficiently comparable to enable a meta-analysis to be performed. Two additional studies were identified in the update search, both of which adjusted their results for tooth brushing (Ferreira *et al.*, 2011; Alm *et al.*, 2012) (Oral health review p40 and p70; Update search table 234)

6.68. Three studies report an association between higher frequency of consumption of sugars in confectionery and greater dental caries incidence (Mattila *et al.*, 2001; Mattila *et al.*, 2005; Kallestal & Fjelddahl, 2007; Ollila & Larmas, 2007). Two studies report no significant association between either intake of sugars confectionery or sweet snacks and dental caries incidence (Leroy *et al.*, 2005; Tamaki *et al.*, 2009). Of the studies identified in the update search one reports an association between frequency of consumption of sugars confectionary at age one year and dental caries development at age 15 years (Alm *et al.*, 2012) and the other in children and adolescents with cerebral palsy also found a significant association between higher frequency of sugars consumption and dental caries (Ferreira *et al.*, 2011). Of the studies that did not adjust for tooth brushing one indicates that the number of between meal snacks containing more than 10% sugars is associated with two-year dental caries increment, but not three-year caries increment (Wilson & Ashley, 1989) and the other two indicate no association between sugars confectionery and snack consumption and dental caries risk.

Frequency of sugars-containing foods and/or sugars confectionery consumption (servings/day or servings/week) and dental caries in mixed and permanent dentition
• Association • Moderate evidence • The direction of the association indicates that higher frequency of consumption of sugars-containing foods and/or sugars in confectionery is detrimental to oral health • The association is biologically relevant

Sugar alcohols

6.69. Six randomised controlled trials were identified that presented evidence on sugar alcohol containing gum and dental caries incidence in both the mixed and permanent dentition in children and adolescents (Finn *et al.*, 1978; Glass, 1983; Beiswanger *et al.*, 1998; Alanen *et al.*, 2000; Machiulskiene *et al.*, 2001; Szoke *et al.*, 2001). The data on measures of dietary exposure, caries incidence/prevalence and risk assessment methods were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Oral heath review p41-43).

6.70. Four trials report an effect of sugar-free chewing gum containing sorbitol, mannitol or xylitol in reducing caries incidence in comparison with not using a chewing gum (Beiswanger *et al.*, 1998; Alanen *et al.*, 2000; Machiulskiene *et al.*, 2001; Szoke *et al.*, 2001). Two trials report no significant effect of sugar-free chewing gum containing sorbitol and/or mannitol in reducing caries incidence in comparison to a no gum control group (Finn *et al.*, 1978; Glass, 1983). In the trials that employ a 'no gum' control group, it is unclear whether it is specifically the sugar alcohol or the act of chewing and the concomitant increase in salivary flow that contributed to the effect.

Sugar alcohols and dental caries incidence in both the mixed and permanent dentition
• Effect • Moderate evidence • The direction of the effect demonstrates that use of chewing gum containing sugar alcohols in comparison with not using a chewing gum is beneficial to oral health • The effect is biologically relevant

Outcomes where there is insufficient evidence

6.71. The tables below detail the exposures and outcomes where there are too few studies or trials that meet the inclusion criteria for the review (e.g. by study duration, publication date, health of subjects, design of intervention) to draw a conclusion. A full description of the studies can be found in the relevant systematic reviews.

Table 6.1 Insufficient evidence-cohort studies

Risk factor/health outcome/measure	**Exposure**
cardiovascular disease events	monosaccharides disaccharides
Coronary events	monosaccharides disaccharides sugars rich foods sugars-sweetened beverages
Stroke	total sugars monosaccharides disaccharides sugars-sweetened beverages
Incident hypertension	fructose sugars-sweetened beverages
Blood pressure	sugars rich foods
Total cholesterol	sugars rich foods
HDL-cholesterol	sucrose sugars-sweetened beverages
Body weight change	sucrose sugars rich foods sugars-sweetened beverages
Weight gain	sucrose sugars rich foods sugars-sweetened beverages
Body fatness and fat distribution	sugars rich foods sugars-sweetened beverages
Energy intake	sugars-sweetened beverages
Type 2 diabetes mellitus	sugars rich foods
Impaired glucose tolerance	total sugars
Glycaemia	sugars-sweetened beverages
Insulinaemia	total sugars sugars-sweetened beverages
Insulin resistance/sensitivity	sugars sugars-sweetened beverages
Colo-rectal, colon and rectal cancer	sugars, fructose, sucrose lactose (colon cancer only)
Caries in the deciduous dentition	frequency of sugars intake sweetened comforter (dummy) fruit juice
Caries in the mixed and permanent dentition	frequency of sugars-sweetened beverages
Oral cancer	sugars-sweetened beverages

Table 6.2 Insufficient evidence-randomised controlled trials

Risk factor/health outcome/measure	**Exposure**
Vascular compliance	sugars
Fasting blood lipids	glucose, fructose sucrose sugars-sweetened beverages
CRP, haptoglobin and transferrin	sugars
Body weight	sugars-sweetened beverages
Weight gain	sugars-sweetened beverages
Energy intake	sugars rich foods sugars-sweetened beverages
Eating motivation	sucrose
Impaired glucose tolerance	sugars
Glycosylated blood proteins	sugars
Glycaemia	sugars-sweetened beverages
Insulinaemia	sugars-sweetened beverages
Insulin resistance	sugars-sweetened beverages
Transit time	sugar alcohols polydextrose
Constipation	polydextrose
Caries in the deciduous dentition	monosaccharides sugar alcohols
Caries in the mixed and permanent dentition	sugars rich foods
Periodontal disease	sugars

Table 6.3 Inconclusive evidence

Measure	**Exposure**
Faecal pH	polydextrose
Faecal SCFA	polydextrose

Summary and conclusions

6.72. This assessment is based on 67 prospective cohort studies and 41 randomised controlled trials investigating the relationship between sugars, individual sugars, sugars-sweetened foods and beverages and sugar alcohol intake and cardio-metabolic, colo-rectal and oral health outcomes. Thirty six prospective cohort studies and nine randomised controlled trials were conducted in children and adolescents. The majority of the evidence on sugars, sugars-sweetened foods and beverages and sugar alcohols is derived from cohort studies. There are very few data on individual sugars such as glucose, fructose or sucrose. Due to the paucity of studies, there is a lack of evidence to draw conclusions on the impact of sugars intake on the majority of cardio-metabolic outcomes in adults, including body weight. There is also a lack of evidence to assess the impact of sugars intake on oral health in adults, as all included studies and trials were conducted in children and adolescents. With observational studies there is substantial potential for biases and the possibility of confounding by an extraneous variable that correlates with both the dependent variable and the independent variable (residual confounding) and any associations must be interpreted with caution.

6.73. The WHO draft guideline for sugars intake for adults and children (WHO, 2014) was published in the final stages of the drafting of this report. The reviews on sugars intake in relation to body weight and oral health (Te Morenga *et al.*, 2013; Moynihan & Kelly, 2014), conducted as part of the WHO draft guideline for sugars intake for adults and children, employed different inclusion criteria to the reviews conducted to inform this report. Consequently, different studies and trials were considered to inform the WHO draft guideline as compared with this report, e.g. non-randomised trials, population and cross-sectional studies were also included in the systematic reviews (see Chapter 1 and Annex 2 for the inclusion criteria used in the reviews informing this report).

6.74. Very few trials on fructose met the inclusion criteria for this report because many were either not randomised or they were of insufficient duration. No studies specifically investigating the effects of high fructose corn syrup were identified that met the inclusion criteria. As there is concern around fructose consumption and its implications to cardio-metabolic health, evidence from trials that did not meet the inclusion criteria for this report and its implications to public health were considered. Details of this can be found in Annex 3.

6.75. Randomised controlled trials conducted in adults indicate that increasing sugars intake when consuming an *ad libitum* diet, either through the substitution of other macronutrient components or by replacement of non-caloric sweeteners by sugars, leads to an increase in energy intake. Evidence from trials conducted in children and adolescents indicates that consumption of sugars-sweetened beverages, as compared with non-calorically sweetened beverages, results in weight gain and an increase in body mass index. Trials in which sugars are consumed in place of non-caloric sweeteners demonstrate incomplete compensation for the sugars energy by insufficient voluntary reduction in intake of other foods or drinks, which leads to weight gain.

6.76. In cohort studies, intake of sugars or individual sugars is not associated with the incidence of type 2 diabetes mellitus. Randomised controlled trials indicate there appears to be no effect of diets differing in sugars content on glycaemia, insulinaemia or insulin resistance, but the evidence is limited. A greater risk of developing type 2 diabetes mellitus, however, is associated with the consumption of sugars-sweetened beverages in cohort studies , but there are too few trials on glycaemia, insulinaemia and insulin resistance to draw firm conclusions with regard to sugars-sweetened beverage intake. There is no association between sugars intake and the risk of coronary events. No evidence was available on sugars consumption in relation to cardiovascular diseases, but one cohort study indicates a detrimental association with higher added sugars intakes (Yang *et al.*, 2014). In trials, varying sugars intake has no effect on blood pressure or fasting cholesterol concentrations, but the evidence for this is limited.

6.77. The evidence for a relationship between sugars intake and dental caries comes from cohort studies and trials conducted in children and adolescents. There is a paucity of studies and trials in adults. Cohort studies that adjusted results for tooth brushing frequency were given more weight during the analysis than those that did not. Greater consumption (i.e. the amount) of total sugars and sugars-containing foods and beverages are associated with a greater risk of dental caries in the deciduous and permanent dentitions. Greater frequency of consumption of sugars-containing foods and beverages, but not total sugars, is also associated with greater risk of dental caries in the deciduous and permanent dentitions. Identifying the relationship between dental caries and sugars intake is confounded by oral hygiene and global preventative measures that have reduced the incidence of caries worldwide. Nevertheless, caries is present in 31% of the adult population of England and Wales, with variations in social class being very apparent (White *et al.*, 2011), and in many people oral hygiene does not meet the best possible standards (Steele & O' Sullivan, 2011).

6.78. Randomised controlled trials indicate that the use of chewing gum containing sugar alcohols reduces risk of dental caries, as compared with not using a chewing gum, but it is unclear whether it is specifically the sugar alcohols or the act of chewing and the concomitant increase in salivary flow that contributed to the effect.

6.79. Randomised controlled trials on dental erosion and sweetened beverage consumption were considered, but no direct comparison between non-calorically sweetened beverages and sugars-sweetened beverages was identified. Details of the evidence on acidic foods and drinks and dental erosion can be found in Annex 4.

6.80. Trials provide limited evidence that intake of some sugar alcohols increases faecal weight and affects faecal bacterial content, e.g. *Bifidobacteria* spp. Sugar alcohols are not commonly consumed in the diet, however, and despite the large doses supplied in the trials, the interventions did not produce a large faecal bulking effect. The increase in faecal weight, therefore, is considered to be of minor biological relevance. The health impact of an effect on faecal bacteria is currently uncertain, thus, whether this observation is beneficial or biologically relevant cannot be determined.

Chapter 7. Starch and starch-rich foods

7.1. This assessment is based on 38 prospective cohort studies and four randomised controlled trials investigating the relationship between intake of starch and starch-rich foods in relation to cardio-metabolic health outcomes. There were no prospective cohort studies or randomised controlled trials conducted in children and adolescents. Links to the individual systematic reviews and update search are given in Annex 1 and the relevant page or table is given in the text below.

7.2. Evidence on health/disease outcomes has been discussed in detail only where there are sufficient data for a conclusion to be drawn, from studies meeting the pre-agreed inclusion criteria. Outcomes where there are too few studies to reach a conclusion are listed at the end of the chapter (see tables 7.1 and 7.2). Outcomes in which the evidence was considered too inconsistent to make a valid judgement are listed in table 7.3. Therefore, not all 38 cohort studies and four randomised controlled trials will be listed in this chapter.

7.3. For the prospective cohort study meta-analyses the relative risks for starch intake are presented for 50g/day. For refined grain intake relative risks are presented for each half servings/day increase, equivalent to approximately one standard deviation, in refined grains intake (Lang et al., 2003). A limitation in cohort studies is that it is unclear how starch is determined.

Starch

Coronary events

7.4. Four cohort studies were identified that presented evidence on total starch intake and incidence of fatal or non-fatal coronary events (Fehily *et al.*, 1993; Pietinen *et al.*, 1996; Liu *et al.*, 2000c; Beulens *et al.*, 2007). Due to variation in the exposures reported and the lack of age-adjustment in one cohort study (Fehily et al., 1993), there were too few studies to undertake a meta-analysis. Three cohort studies were subsequently identified in the update search (Sieri *et al.*, 2010; Burger *et al.*, 2011; Wallstrom *et al.*, 2012) (Cardio-metabolic review, cardiovascular disease chapter p 36; Update search table 11).

7.5. No association between starch intake and incidence of coronary events is indicated in any of the cohort studies, except one (Burger et al., 2011), which indicates a higher starch intake as g/day is associated with greater incidence of coronary events in men, but not in women.

Starch (g/day) and coronary events
• No association • Moderate evidence

Type 2 diabetes mellitus

7.6. Eight cohort studies were identified that presented evidence on total starch intake and incidence of type 2 diabetes mellitus, four of which were included in a meta-analysis (Meyer *et al.*, 2000; Hodge *et al.*, 2004; Barclay *et al.*, 2007; Schulze *et al.*, 2008). Four studies could not be included in a meta-analysis as they did not report the necessary data (Feskens *et al.*, 1995; Monterrosa *et al.*, 1995; Leonetti *et al.*, 1996; Janket *et al.*, 2003). One cohort study was subsequently identified in the update search (Sluijs et al., 2010) (Cardio-metabolic review, diabetes chapter p75; Update search table 123).

7.7. No association is indicated between total starch intake and type 2 diabetes mellitus (RR 1.00, 95% CI 0.84, 1.19 for each 50g/day increase; p=0.96). The studies that are not included in the analysis do not provide evidence of an association between starch intakes and risk of type 2 diabetes mellitus. The study identified in the update search indicates a higher starch intake is associated with a greater incidence of type 2 diabetes mellitus (Sluijs *et al.*, 2010).

Starch (g/day) and type 2 diabetes mellitus
• No association • Moderate evidence

Starch rich foods

Refined grains

Total cardiovascular disease events

7.8. Three cohort studies were identified that presented evidence on refined grains intake and combined incidence of stroke and coronary events, all of which were included in the meta-analysis (Liu *et al.*, 2000b; Sahyoun *et al.*, 2006; Jacobs, Jr. *et al.*, 2007). No further cohort studies were subsequently identified in the update search (Cardio-metabolic review, cardiovascular disease chapter p 153-154 and 159)

7.9. No association is indicated between refined grains intake and the combined incidence of stroke and coronary events (RR 1.00, 95% CI 0.98, 1.01 for each half serving/day increase; p=0.5). Two of the cohort studies report no association between refined grains intake and fatal cardiovascular disease events (Sahyoun *et al.*, 2006; Jacobs, Jr. *et al.*, 2007).

Refined grains (serving/day) and total cardiovascular disease events
• No association • Moderate evidence

Type 2 diabetes mellitus

7.10. Four cohort studies were identified that presented evidence on refined grains intake and incidence of type 2 diabetes mellitus, all of which were included in a meta-analysis (Meyer *et al.*, 2000; Liu *et al.*, 2000a; Fung *et al.*, 2002; Montonen *et al.*, 2003). One cohort study was subsequently identified in the update search (Yu et al., 2011) (Cardio-metabolic review, diabetes chapter p174; Update search table 130).

7.11. No association is indicated between refined grains intake and incidence of type 2 diabetes mellitus (RR 1.00, 95% CI 0.98, 1.01, for each half serving/day increase; p=0.3). The cohort study identified in the update search indicates no association between refined grain intake and the incidence of type 2 diabetes.

Refined grains (serving/day) and type 2 diabetes mellitus
• No association • Moderate evidence

Rice

Type 2 diabetes mellitus

7.12. Three cohort studies were identified that presented evidence on rice intake and incidence of type 2 diabetes mellitus (Liu *et al.*, 2000a; Hodge *et al.*, 2004; Villegas *et al.*, 2007). Due to variation in the exposures reported a meta-analysis could not be conducted. Six cohort studies were subsequently identified in the update search (Sun *et al.*, 2010; Nanri *et al.*, 2010; Yu *et al.*, 2011), one publication reported on data from three cohorts in the US (Sun et al., 2010), including a cohort reported on previously (Liu et al., 2000a), and another publication reported on two cohorts in Japan (Nanri et al., 2010). (Cardio-metabolic review, diabetes chapter p128; Update search table 132). A meta-analysis has subsequently reported white rice intake in relation to the incidence of type 2 diabetes mellitus (Hu et al., 2012), which includes seven cohorts identified above (Hodge *et al.*, 2004; Villegas *et al.*, 2007; Nanri *et al.*, 2010; Sun *et al.*, 2010).

7.13. An association is indicated between higher white rice intake and greater incidence of type 2 diabetes mellitus (RR 1.11, 95% CI 1.08, 1.14 for each 158g serving/day increase; $P_{trend}<0.001$). When the three Asian (Japanese and Chinese) cohorts and the four Western (US and Australian) cohorts were considered separately no association is indicated in the Western cohorts (RR 1.12, 95% CI 0.94, 1.33), but an association is indicated in the Asian cohorts (RR 1.55, 95% CI 1.20, 2.01) between increased white rice intake incidence of type 2 diabetes mellitus. One smaller cohort study in a Chinese population identified in the update search, but not included in the meta-analysis (Yu et al., 2011), reports no association between rice intake and the incidence of type 2 diabetes mellitus.

7.14. In the UK, the median portion of rice is 180g with a 75th centile of 235g (Henderson et al., 2003). Overall, UK population intakes are low, the mean intake for cooked white rice amongst consumers is 28g/day (Bates et al., 2012). The levels of intake at which a detrimental association is indicated may only be relevant for a small proportion of the UK population where white rice is consumed on a daily basis. It is therefore unlikely that there would be many people in the UK consuming the very high intakes of rice seen in studies conducted in Asian countries.

White rice (serving/day) and type 2 diabetes mellitus
• Association with white rice intake • Moderate evidence • The direction of the association indicates that high consumption of white rice is detrimental to health • The association is biologically significant; however, the association is largely derived from reported intakes that are substantially greater than typical current diets within the UK

7.15. Several of the US cohort studies also report on brown rice intake in relation to incidence of type 2 diabetes mellitus (Sun et al., 2010) and observe a protective association between brown rice intake and incidence of type 2 diabetes mellitus (RR 0.89, 95% CI 0.81, 0.97 for two or more servings a week as compared with one a month).

Brown rice (serving/day) and type 2 diabetes mellitus
• Association with brown rice intake • Limited evidence • The direction of association indicates higher intake of brown rice is beneficial to health • The association is biologically relevant

Potatoes

7.16. Cooking methods (e.g. fried or boiled) for potatoes were not always reported in the cohort studies, which maybe a confounding factor in the conclusions reported on associations with cardio-metabolic health outcomes.

Total cardiovascular disease events

7.17. Three cohort studies were identified that presented evidence on intake of potatoes and incidence of total cardiovascular disease (Joshipura et al., 2009; Panagiotakos et al., 2009). One publication reported on data from two cohorts (Joshipura et al., 2009). Due to variation in the exposures reported no meta-analysis was conducted. No further cohort studies were subsequently identified in the update search (Cardio-metabolic review, cardiovascular disease chapter p 128-130).

7.18. Two cohort studies report an association between greater consumption of potatoes and risk of fatal and non-fatal cardiovascular disease events (Joshipura et al., 2009); the strength and statistical significance of these associations varied by total carbohydrate consumption subgroup (tending to increase in higher carbohydrate consumers). The other cohort study reports a higher potato consumption in cases of cardiovascular disease than the other members of the cohort, but this study only provides age-adjusted estimates (Panagiotakos et al., 2009).

7.19. Two cohort studies were identified that presented evidence on intake of potatoes and incidence of fatal coronary events (Knekt et al., 1994; Pietinen et al., 1996). Two cohort studies were identified in one publication that presented evidence on intake of potatoes and incidence of fatal and non-fatal ischaemic stroke (Joshipura et al., 1999). No further cohort studies were subsequently identified in the update search (Cardio-metabolic review, cardiovascular disease chapter p 131-134). The studies were combined to conduct a meta-analysis of the combined incidence of stroke and coronary events. Two studies could not be included in a meta-analysis as they did not report the necessary data to estimate a dose-response trend (Knekt *et al.*, 1994; Joshipura *et al.*, 2009), one of which reported no association between potato consumption and fatal coronary events for men or women (Knekt et al., 1994).

7.20. The heterogeneity is too high (I^2=90%) to report the meta-analysis pooled estimate, but the studies provide no consistent evidence of an association between potato consumption and incidence of total cardiovascular disease.

Potatoes (serving/day) and total cardiovascular disease events
• No association • Limited evidence

Type 2 diabetes mellitus

7.21. Four cohort studies were identified that presented evidence on intake of potatoes and incidence of type 2 diabetes mellitus, three of which were included in a meta-analysis (Hodge *et al.*, 2004; Montonen *et al.*, 2005; Halton *et al.*, 2006). One study could not be included in a meta-analysis as they did not report the necessary data to estimate a dose-response trend (Villegas et al., 2007). No further cohort studies were subsequently identified in the update search (Cardio-metabolic review, diabetes chapter p 132-134).

7.22. A borderline association is indicated between higher potato consumption and higher incidence of type 2 diabetes mellitus (RR 1.07, 95% CI 1.00, 1.15 for each half serving/day increase; p=0.04). The cohort study that is not included in the meta-analysis reports a protective association between potato consumption and incidence of type 2 diabetes mellitus, but this is in a Chinese cohort where potatoes are not a major source of starch and in this study, sweet potatoes (Ipomoea batatas) are also classified with potatoes (Solanum tuberosum).

Potatoes (serving/day) and type 2 diabetes mellitus
• Association • Limited evidence • The direction of the association indicates that greater consumption of potatoes is detrimental to health, but it is not possible to exclude confounding by other variables, e.g. cooking methods such as frying • The association is biologically relevant

Outcomes where there is insufficient evidence

7.23. The tables below detail the exposures and outcomes where there are too few studies or trials that meet the inclusion criteria for the review (e.g. by study duration, publication date, health of subjects, design of intervention) to draw a conclusion. A full description of the studies can be found in the relevant systematic reviews.

Table 7.1 Insufficient evidence- -cohort studies

Risk factor/health outcome/measure	**Exposure**
Total cardiovascular disease	starch rice
Stroke	starch refined grains rice potatoes
Incident hypertension	refined grains
Type 2 diabetes mellitus	starch rich foods potatoes
Impaired glucose tolerance	potatoes
Glycaemia	starch potatoes
Insulinaemia	starch
Glycosylated blood proteins	starch
Colo-rectal cancer	starch
Coronary events	refined grains rice potatoes
Weight gain	starch
Fat distribution	starch
Percentage body fat	starch
Waist circumference	refined grains

Table 7.2 Insufficient evidence-randomised controlled trials

Health outcome	**Exposure**
Dental caries	starch

Table 7.3 Inconsistent evidence

Health outcome	**Exposure**
Total cardiovascular disease	potatoes

Summary and conclusions

7.24. This assessment is based on 38 prospective cohort studies and four randomised controlled trials investigating the relationship between intake of starch or starch-rich foods and cardio-metabolic health outcomes. There is a lack of available evidence on the relationship between starch or starch-rich foods and colo-rectal and oral health outcomes or cardiovascular risk factors. There were no prospective cohort studies or randomised controlled trials conducted in children and adolescents. With observational studies there is substantial potential for biases and the possibility of confounding by an extraneous variable that correlates with both the dependent variable and the independent variable (residual confounding) and any associations must be interpreted with caution.

7.25. Prospective cohort studies indicate there is no association between starch intake and incidence of coronary events or type 2 diabetes mellitus. Cohort studies also indicate there is no association between refined grains intake and risk of type 2 diabetes mellitus.

7.26. Cohort studies do indicate an association between greater consumption of white rice and greater risk of type 2 diabetes mellitus, but this finding is largely based on data from Asian populations (Japanese and Chinese) consuming levels of rice which are not generally observed in the UK. Higher consumption of brown rice is associated with a reduction in the risk for incident type 2 diabetes mellitus, but the evidence is limited due to the small number of studies. A higher consumption of potatoes is associated with a greater risk of type 2 diabetes mellitus, but it is not possible to exclude confounding by other dietary variables and the evidence is limited due to the small number of studies.

7.27. Overall, the available evidence in relation to cardio-metabolic outcomes indicates no association with dietary starch when consumed in the amounts eaten in the typical UK diet.

Chapter 8. Dietary fibre

8.1. This assessment is based on 74 prospective cohort studies and 131 randomised controlled trials investigating the relationship between dietary fibre ('total dietary fibre', non-starch polysaccharide and its constituents) and whole grains intake and cardio-metabolic, colo-rectal and oral health outcomes. There were three prospective cohort studies conducted in children and adolescents. Randomised controlled trials investigating the effects of dietary fibre isolates are considered separately in Annex 5. Links to the individual systematic reviews and update search are given in Annex 1 and the relevant page or table is given in the text below.

8.2. Evidence on health/disease outcomes have been discussed in detail only where there are sufficient data for a conclusion to be drawn, from studies meeting the pre-agreed inclusion criteria. Outcomes where there are too few such studies to reach a conclusion, are listed at the end of the chapter (see tables 8.1 and 8.2). Outcomes in which the evidence was considered too inconsistent to make a valid judgement are listed in table 8.3. Therefore, not all 74 cohort studies and 131 randomised controlled trials are listed in this chapter.

8.3. The evidence relating to dietary fibre is considered under diets containing differing amounts of dietary fibre and its constituent fibres and foods containing dietary fibre. Prospective cohort studies do not always report the method used to determine dietary fibre, but among those that do the American Association of Official Analytical Chemists methods 985.29 and 991.43 (Prosky *et al.*, 1988; Lee *et al.*, 1992) is the most frequently used, and only a few studies report using the non-starch polysaccharide method (Englyst *et al.*, 1994).

8.4. For the prospective cohort study meta-analyses the relative risks for dietary fibre, cereal fibre and insoluble fibre intake are presented for each 7g/day increase in intake. For soluble, insoluble, vegetable and fruit fibre the relative risks are presented for each 4g/day increase and for legume fibre the relative risk is presented for each 1g/day increase. The relative risks for total cereal intake and whole grains intake are presented for each 0.5 servings/day increase and for non-soy legumes for each 0.25 servings/day increase in intake. Each of these values is equivalent to approximately one standard deviation in adult intakes, as based on UK data from the National Diet and Nutrition Survey (Bates *et al.*, 2009).

Dietary fibre

Cardiovascular disease

8.5. Four cohort studies were identified that presented evidence on dietary fibre intake and cardiovascular disease incidence. (Liu *et al.*, 2002; Bazzano *et al.*, 2003; Laaksonen *et al.*, 2005; Drogan *et al.*, 2007). Two studies could not be included in a meta-analysis as they did not report the necessary data, leaving an insufficient number of studies to enable a meta-analysis to be performed. Five cohort studies were subsequently identified in the update search (Eshak *et al.*, 2010; Kokubo *et al.*, 2011; Park *et al.*, 2011; Wallstrom *et al.*, 2012; Chuang *et al.*, 2012) (Cardio-metabolic review, cardiovascular disease chapter p 39-41; Update search table 13). A further meta-analysis was performed which included the studies found in the update search (Threapleton *et al.*, 2013d) and also included three later studies (Buyken *et al.*, 2010; Akbaraly *et al.*, 2011; Threapleton *et al.*, 2013a). The results from the later meta-analysis were used.

8.6. An association is indicated between higher consumption of dietary fibre and a reduced incidence of cardiovascular disease (RR 0.91, 95% CI 0.88, 0.94 for each 7g/day increase; $p<0.001$).

Dietary fibre (g/day) and cardiovascular disease
• Association • Moderate evidence • The direction of the association indicates higher consumption of dietary fibre is beneficial to health • The association is biologically relevant

Coronary events

8.7. Eleven cohort studies were identified that presented evidence on dietary fibre intake and coronary events (Fehily *et al.*, 1993; Knekt *et al.*, 1994; Pietinen *et al.*, 1996; Rimm *et al.*, 1996; Todd *et al.*, 1999; Appleby *et al.*, 1999; Wolk *et al.*, 1999; Liu *et al.*, 2002; Bazzano *et al.*, 2003; Mozaffarian *et al.*, 2003; Streppel *et al.*, 2008), eight of which were included in a meta-analysis. Three cohort studies, that could not be included in the meta-analysis as they did not report the necessary data, indicated no significant association between dietary fibre and coronary events (Fehily *et al.*, 1993; Knekt *et al.*, 1994; Todd *et al.*, 1999). Three cohort studies were subsequently identified in the update search (Eshak *et al.*, 2010; Kokubo *et al.*, 2011; Wallstrom *et al.*, 2012) (Cardio-metabolic review, cardiovascular disease chapter p 42-47, 50-51; Update search table 14). A further meta-analysis was performed which included the studies found in the update search (Threapleton *et al.*, 2013d) and also included two later studies (Crowe *et al.*, 2012; Threapleton *et al.*, 2013a). The results from the later meta-analysis were used.

8.8. An association is indicated between higher consumption of dietary fibre and a reduced incidence of coronary events (RR 0.91, 95% CI 0.87, 0.94 for each 7g/day increase; p<0.001).

Dietary fibre (g/day) and coronary events
• Association • Adequate evidence • The direction of the association indicates higher consumption of dietary fibre is beneficial to health • The association is biologically relevant

Stroke

8.9. Four cohort studies were identified that presented evidence on dietary fibre intake and stroke, all of which were included in a meta-analysis (Ascherio *et al.*, 1998; Bazzano *et al.*, 2003; Oh *et al.*, 2005; Larsson *et al.*, 2009). Three cohort studies were subsequently identified in the update search (Eshak *et al.*, 2010; Kokubo *et al.*, 2011; Wallstrom *et al.*, 2012) (Cardio-metabolic review, Cardiovascular disease chapter p 48-52; Update search table 15). A further meta-analysis was performed which included the studies found in the update search (Threapleton *et al.*, 2013c). The results from the later meta-analysis were used.

8.10. An association is indicated between higher consumption of dietary fibre and a reduced incidence of haemorrhagic plus ischemic stroke (RR 0.93, 95% CI 0.88, 0.98 for each 7g/day increase; p=0.002).

Dietary fibre (g/day) and stroke
• Association • Adequate evidence • The direction of the association indicates higher consumption of dietary fibre is beneficial to health • The association is biologically relevant

Fasting blood lipids

8.11. Five randomised controlled trials were identified that presented evidence on consumption of diets higher in dietary fibre in relation to total cholesterol, LDL-cholesterol, HDL-cholesterol and triacylglycerol concentration, all of which were included in a meta-analysis (Kesaniemi *et al.*, 1990; Aller *et al.*, 2004; Thompson *et al.*, 2005; Andersson *et al.*, 2007; Olendzki *et al.*, 2009),. No further trials were identified in the update search (Cardio-metabolic review, hyperlipidaemia and blood lipid review chapter p 281-296).

8.12. No significant effect is demonstrated for the consumption of diets higher in dietary fibre relative to low dietary fibre diets on fasting total cholesterol concentration (-0.08mmol/L, 95% CI -0.27, 0.11; p=0.4), fasting LDL-cholesterol concentration (-0.02mmol/L, 95% CI -0.20, 0.15; p=0.8), fasting HDL-cholesterol concentration (-0.07mmol/L, 95% CI -0.17, 0.04; p=0.2) or fasting triacylglycerol concentration (-0.06mmol/L, 95% CI -0.27, 0.15; p=0.57).

Dietary fibre and fasting total cholesterol, LDL-cholesterol, HDL-cholesterol and triacylglycerol concentration
• No association • Adequate evidence

Body weight change

8.13. Four cohort studies were identified that presented evidence on dietary fibre intake and body weight change (Colditz *et al.*, 1990; Ludwig *et al.*, 1999; Iqbal *et al.*, 2006; Hays *et al.*, 2006). The data were insufficiently comparable for a meta-analysis to be performed. One cohort study was subsequently identified in the update search (Du *et al.*, 2010) (Cardio-metabolic review, obesity chapter p 47-49; Update search table 186).

8.14. No significant association between dietary fibre intake, as grams/day, and body weight change is indicated in two studies (Colditz *et al.*, 1990; Iqbal *et al.*, 2006). Two studies assess the fibre density of the diet in relation to body weight change: one indicates higher fibre-density consumption is associated with less body weight gain (Ludwig *et al.*, 1999) and the other indicates higher fibre-density consumption is associated with greater body weight gain (Hays *et al.*, 2006). The study identified in the update search indicates higher intake of dietary fibre is associated with less body weight gain (Du *et al.*, 2010).

8.15. Overall, these cohort studies provide no evidence of a consistent association between dietary fibre intake and body weight change.

Dietary fibre (g/day) and body weight change
• No association • Moderate evidence

Energy intake

8.16. Six randomised controlled trials were identified that presented evidence on the effects of dietary fibre rich diets in relation to energy intakes, all of which were included in a meta-analysis (Kesaniemi *et al.*, 1990; Sciarrone *et al.*, 1993; Turpeinen *et al.*, 2000; Thompson *et al.*, 2005; Andersson *et al.*, 2007; Olendzki *et al.*, 2009) (Cardio-metabolic review, energy intake chapter p 111-114).

8.17. No significant effect of the consumption of a dietary fibre rich diet on energy intake (-445.9kJ/day, 95% CI -957.6, 65.82; p=0.09). The majority of the studies did not make allowance for the metabolisable energy that is available in fibre due to fermentation therefore the majority of studies will have over-estimated the decrease in energy intake.

Dietary fibre and energy intake
• No effect • Moderate evidence

Type 2 diabetes mellitus

8.18. Eleven cohort studies were identified that presented evidence on dietary fibre intake and type 2 diabetes mellitus (Salmeron *et al.*, 1997a; Salmeron *et al.*, 1997b; Meyer *et al.*, 2000; Stevens *et al.*, 2002; Montonen *et al.*, 2003; Hodge *et al.*, 2004; Schulze *et al.*, 2004a; Lindstrom *et al.*, 2006; Barclay *et al.*, 2007; Schulze *et al.*, 2007b; Wannamethee *et al.*, 2009), ten of which were included in a meta-analysis. One cohort study, that could not be included in a meta-analysis as it did not report the necessary data, indicated no statistically significant association between dietary fibre intake and type 2 diabetes mellitus (Lindstrom *et al.*, 2006). Three cohort studies were identified in the update search (Sluijs *et al.*, 2010; Hopping *et al.*, 2010; Sakurai *et al.*, 2012) (Cardio-metabolic review, diabetes and glycaemia chapter p 81-84; Update search table 124).

8.19. An association is indicated between higher consumption of dietary fibre and a reduced incidence of type 2 diabetes mellitus (RR 0.94, 95% CI 0.90, 0.97 for each 7g/day increase; p=0.001). Two of the studies identified in the update search indicate higher dietary fibre intake is associated with a reduction in the incidence of type 2 diabetes mellitus (Sluijs *et al.*, 2010; Hopping *et al.*, 2010) and the other study indicates no significant association between dietary fibre intake and the incidence of type 2 diabetes (Sakurai *et al.*, 2012).

Dietary fibre (g/day) and type 2 diabetes mellitus
• Association • Adequate evidence • The direction of the association indicates a higher consumption of dietary fibre is beneficial to health • The association is biologically relevant

Fasting blood glucose

8.20. Four randomised controlled trials were identified that presented evidence on dietary fibre in relation to blood glucose, all of which were included in a meta-analysis (Aller *et al.*, 2004; Thompson *et al.*, 2005; Andersson *et al.*, 2007; Olendzki *et al.*, 2009). Two trials were identified in the update search (Venn *et al.*, 2010; Weickert *et al.*, 2011) (Cardio-metabolic review, diabetes and glycaemia chapter p 268-272; Update search table 158).

8.21. No consistent effect is demonstrated for dietary fibre intake on fasting blood glucose concentration on the forest plot, but the heterogeneity is too high to report the meta-analysis pooled estimate (I^2=82%). The two trials identified in the update search reports no significant effect of dietary fibre intake on fasting blood glucose concentration.

Dietary fibre and fasting blood glucose concentration
• No effect • Limited evidence

Fasting insulin

8.22. Three randomised controlled trials were identified that presented evidence on dietary fibre in relation to fasting insulin concentration (Aller *et al.*, 2004; Thompson *et al.*, 2005; Andersson *et al.*, 2007). No further trials were identified in the update search (Cardio-metabolic review, diabetes and glycaemia chapter p 273-276).

8.23. Due to variation between the different methodologies used to measure fasting insulin concentration, it was not possible to conduct a meta-analysis. All trials report no significant effect dietary fibre diets on fasting insulin concentration.

Dietary fibre and fasting insulin concentration
• No effect • Moderate evidence

Faecal weight and intestinal transit time

8.24. Five randomised controlled trials were identified that presented evidence on dietary fibre in relation to transit times and faecal weight (Beyer & Flynn, 1978; Kelsay *et al.*, 1978; Stasse-Wolthuis *et al.*, 1979; Stasse-Wolthuis *et al.*, 1980; Kesaniemi *et al.*, 1990) The data on dietary intakes were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health review, p 34-35 39, 44-46, 49).

8.25. An effect of higher fibre diets, containing cereals, fruits and vegetables, is demonstrated on increasing faecal wet weights and decreasing intestinal transit times. The effect sizes are similar to the effect sizes with wheat fibre. The increase in faecal weight is approximately a 4g increase in wet weight per 1g dietary fibre.

Dietary fibre and faecal weight and intestinal transit time
• Effect • Adequate evidence • The direction of the effect demonstrates higher consumption of dietary fibre is potentially beneficial to health • The effect is potentially biologically relevant

Colo-rectal cancer

8.26. Eleven cohort studies were identified that presented evidence on dietary fibre intake and colo-rectal cancer (Pietinen *et al.*, 1999; Mai *et al.*, 2003; Lin *et al.*, 2005; Michels *et al.*, 2005; Otani *et al.*, 2006; Shin *et al.*, 2006; Nomura *et al.*, 2007; Schatzkin *et al.*, 2007; Wakai *et al.*, 2007; Butler *et al.*, 2008; Murphy *et al.*, 2012). One cohort study, that could not be included in a meta-analysis as it did not report the necessary data, indicated that a higher total fibre intake was associated with decreased incidence of colo-rectal cancer (Butler *et al.*, 2008) (Colo-rectal health review, p 152-60, 164; Update search table 222). In the colorectal health review, Bingham et al. (2005) was included in the meta-analysis (Bingham *et al.*, 2005). A further analysis was performed to include more recent data, as identified in the update search, from the EPIC cohort (Murphy et al 2012). The pooled estimate provided in paragraph 8.28 is taken from the updated meta-analysis (Annex 1 additional meta-analyses).

8.27. A pooling project reported a meta-analysis of dietary fibre intake, and its constituent fibres, with colo-rectal, colon or rectal cancer incidence (Park *et al.*, 2005) containing the following 13 cohorts (van den Brandt *et al.*, 1990; Kato *et al.*, 1997; Bandera *et al.*, 1997; Pietinen *et al.*, 1999; Terry *et al.*, 2001; Sieri *et al.*, 2002; Terry *et al.*, 2002; Mai *et al.*, 2003; McCullough *et al.*, 2003; Larsson *et al.*, 2005; Lin *et al.*, 2005; Michels *et al.*, 2005; McCarl *et al.*, 2006) (Colo-rectal health review p152-156). The pooling project indicated no significant association between dietary fibre intake, or intake of constituent fibres, with colo-rectal, colon or rectal cancer incidence. In the colo-rectal health review dietary fibre meta-analyses for colo-rectal, colon or rectal cancer incidence were performed which included the pooled analysis and excluded the cohorts contained within it, but there was little difference in the results as compared with when the pooled analysis was excluded and the individual cohorts were included (Colo-rectal health review p152-172).

8.28. An association is indicated between higher consumption of dietary fibre and a reduced incidence of colo-rectal cancer (RR 0.92, 95% CI 0.87, 0.97 for each 7g/day increase; p=0.002).

Dietary fibre (g/day) and colo-rectal cancer
• Association • Adequate evidence • The direction of the association indicates higher consumption of dietary fibre is beneficial to health • The association is biologically relevant

Colon cancer

8.29. Eight cohort studies were identified that presented evidence on dietary fibre intake and colon cancer, all of which were included in a meta-analysis (McCullough *et al.*, 2003; Michels *et al.*, 2005; Bingham *et al.*, 2005; Otani *et al.*, 2006; Shin *et al.*, 2006; Nomura *et al.*, 2007; Schatzkin *et al.*, 2007; Wakai *et al.*, 2007). Murphy et al. (2012) provided data from the same cohort as Bingham et al. 2005, but with a longer follow up period (Murphy *et al.*, 2012). Therefore an updated analysis was performed which included Murphy et al. (2012) in place of Bingham et al. (2005) (Annex 1 additional meta-analyses). The pooled estimate is given in paragraph 8.30. A cohort study identified in the update search was not included as it reported data from Scandinavian cohorts within the EPIC study, which was already included (Hansen *et al.*, 2012) (Colo-rectal health review, p 152 -158, 165, 168; Update search table 222).

8.30. An association is indicated between higher consumption of dietary fibre and a reduced incidence of colon cancer (RR 0.93, 95% CI 0.89, 0.98 for each 7g/day increase; p=0.007).

Dietary fibre (g/day) and colon cancer
• Association • Adequate evidence • The direction of the association indicates higher consumption of dietary fibre is beneficial to health • The association is biologically relevant

Rectal cancer

8.31. Eight cohort studies were identified, in seven publications, that presented evidence on dietary fibre intake and rectal cancer, all of which were included in a meta-analysis (Michels *et al.*, 2005; Otani *et al.*, 2006; Shin *et al.*, 2006; Nomura *et al.*, 2007; Schatzkin *et al.*, 2007; Wakai *et al.*, 2007; Murphy *et al.*, 2012) (Colo-rectal health review, p 152-158, 169, 172; Update search table 222). In the colorectal health review, Bingham et al. (2005) was included in the meta-analysis (Bingham *et al.*, 2005). A further analysis was performed to include more recent data, as identified in the update search, from the EPIC cohort (Murphy et al 2012). The pooled estimate provided in paragraph 8.32 is taken from the updated meta-analysis (Annex 1 additional meta-analyses).

8.32. A significant association is indicated between higher consumption of dietary fibre and a reduced incidence of rectal cancer (RR 0.91, 95% CI 0.86, 0.97 for each 7g/day increase; p=0.007).

Dietary fibre (g/day) and rectal cancer
• Association • Limited evidence • The direction of the association indicates higher consumption of dietary fibre is beneficial to health • The association is biologically relevant

Insoluble fibre

Cardiovascular disease

8.33. One cohort study was identified that presented evidence on insoluble fibre intake and coronary events (Liu *et al.*, 2002). One cohort study was subsequently identified in the update search (Eshak *et al.*, 2010) (Cardio-metabolic review, cardiovascular disease chapter p 57-58, 62; Update search table 16). A further meta-analysis was performed which included the study found in the update search (Threapleton *et al.*, 2013d) and also included one later study (Threapleton *et al.*, 2013a). The results from the later meta-analysis were used.

8.34. An association between higher consumption of insoluble fibre and a reduced incidence of cardiovascular disease is indicated (RR 0.82, 95% CI 0.70, 0.96 for each 7g/day increase; p=0.02).

Insoluble fibre (g/day) and cardiovascular disease
• Association • Limited evidence • The direction of the association indicates higher consumption of insoluble fibre is beneficial to health • The association is biologically relevant

Coronary events

8.35. Three cohort studies were identified that presented evidence on insoluble fibre intake and coronary events, all of which were included in a meta-analysis (Pietinen *et al.*, 1996; Rimm *et al.*, 1996; Liu *et al.*, 2002) . Two cohort studies were subsequently identified in the update search (Eshak *et al.*, 2010; Kokubo *et al.*, 2011) (Cardio-metabolic review, cardiovascular disease chapter p 57-58, 62; Update search table 17). A further meta-analysis was performed which included the studies found in the update search (Threapleton *et al.*, 2013d) and also included one later study (Threapleton *et al.*, 2013a). One study could not be included in a meta-analysis as it did not report the necessary data (Kokubo *et al.*, 2011). The results from the later meta-analysis were used.

8.36. An association between higher consumption of insoluble fibre and a reduced incidence of coronary events is indicated (RR 0.82, 95% CI 0.68, 0.99 for each 7g/day increase; p=0.03). The study not included in the analysis indicates non-significant protective association between intake of insoluble fibre and fatal or non-fatal events.

Insoluble fibre (g/day) and coronary events
• Association • Moderate evidence • The direction of the association indicates higher consumption of insoluble fibre is beneficial to health • The association is biologically relevant

Type 2 diabetes mellitus

8.37. Three cohort studies were identified that presented evidence on insoluble fibre intake and type 2 diabetes mellitus, all of which were included in a meta-analysis (Meyer *et al.*, 2000; Montonen *et al.*, 2003; Schulze *et al.*, 2007b). No further studies were identified in the update search (Cardio-metabolic review, diabetes and glycaemia chapter p 111-114).

8.38. An association between higher consumption of insoluble fibre and a reduced incidence of type 2 diabetes mellitus is indicated (RR 0.84, 95% CI 0.78, 0.91 for each 7g/day increase; p<0.001).

Insoluble fibre (g/day) and type 2 diabetes mellitus
• Association • Limited evidence • The direction of the association indicates higher consumption of insoluble fibre is beneficial to health • The association is biologically relevant

Soluble fibre

Cardiovascular disease

8.39. Two cohort studies was identified that presented evidence on soluble fibre intake and coronary events (Liu *et al.*, 2002; Bazzano *et al.*, 2003). One cohort study was subsequently identified in the update search (Eshak *et al.*, 2010) (Cardio-metabolic review, cardiovascular disease chapter p 56-58, 62; Update search table 16). A further meta-analysis was performed which included the study found in the update search (Threapleton *et al.*, 2013d) and also included one later study (Threapleton *et al.*, 2013a). The results from the later meta-analysis were used.

8.40. No significant association is indicated between consumption of soluble fibre and the incidence of cardiovascular disease (RR 0.88, 95% CI 0.75, 1.03 for each 4g/day increase; p=0.10).

Soluble fibre (g/day) and cardiovascular disease
• No association • Limited evidence

Coronary events

8.41. Four cohort studies were identified that presented evidence on soluble fibre intake and coronary events, all of which were included in a meta-analysis (Pietinen *et al.*, 1996; Rimm *et al.*, 1996; Liu *et al.*, 2002; Bazzano *et al.*, 2003) . Two cohort studies were subsequently identified in the update search (Eshak *et al.*, 2010; Kokubo *et al.*, 2011) (Cardio-metabolic review, cardiovascular disease chapter p 57-58, 62; Update search table 17). A further meta-analysis was performed which included the studies found in the update search (Threapleton *et al.*, 2013d) and also included one later study (Threapleton *et al.*, 2013a). One study could not be included in a meta-analysis as it did not report the necessary data (Kokubo *et al.*, 2011). The results from the later meta-analysis were used.

8.42. No significant association is indicated between consumption of soluble fibre and the incidence of coronary events (RR 0.89, 95% CI 0.78, 1.02 for each 4g/day increase; p=0.09). The study not included in the analysis indicates no significant association between intake of soluble fibre and fatal or non-fatal events.

Soluble fibre (g/day) and coronary events
• No association • Limited evidence

Type 2 diabetes mellitus

8.43. Three cohort studies were identified that presented evidence on soluble fibre and type 2 diabetes mellitus, all of which were included in a meta-analysis (Meyer *et al.*, 2000; Montonen *et al.*, 2003; Schulze *et al.*, 2007b). No further studies were identified in the update search (Cardio-metabolic review, diabetes and glycaemia chapter p 112-114).

8.44. An association between higher consumption of soluble fibre and a reduced incidence of type 2 diabetes mellitus is indicated (RR 0.86, 95% CI 0.76, 0.97 for each 4g/day increase; p=0.01).

Soluble fibre (g/day) and type 2 diabetes mellitus
• Association • Limited evidence • The direction of the association indicates higher consumption of soluble fibre is beneficial to health • The association is biologically relevant

Fruit fibre

Cardiovascular disease

8.45. One cohort study was identified that presented evidence on fruit fibre intake and cardiovascular disease (Liu *et al.*, 2002). Two cohort studies were subsequently identified in the update search (Eshak *et al.*, 2010; Chuang *et al.*, 2012) (Cardio-metabolic review, cardiovascular disease chapter p 78-81, 84; Update search table 21). A further meta-analysis was performed which included the studies found in the update search (Threapleton *et al.*, 2013d) and also included one later study (Buyken *et al.*, 2010). The results from the later meta-analysis were used.

8.46. A non-significant borderline association is indicated between consumption of fruit fibre and the incidence of coronary events (RR 0.96, 95% CI 0.93, 1.00 for each 4g/day increase; p=0.06).

Fruit fibre (g/day) and cardiovascular disease
• No association • Limited evidence

Coronary events

8.47. Six cohort studies were identified that presented evidence on fruit fibre intake and coronary events, all of which were included in a meta-analysis (Pietinen *et al.*, 1996; Rimm *et al.*, 1996; Wolk *et al.*, 1999; Liu *et al.*, 2002; Mozaffarian *et al.*, 2003; Streppel *et al.*, 2008). One cohort study was subsequently identified in the update search (Eshak *et al.*, 2010) (Cardio-metabolic review, cardiovascular disease chapter p 78-81, 84; Update search table 22). A further meta-analysis was performed which included the studies found in the update search (Threapleton *et al.*, 2013d) and also included two later studies (Crowe *et al.*, 2012; Threapleton *et al.*, 2013a). The results from the later meta-analysis were used.

8.48. No significant association is indicated between consumption of fruit fibre and the incidence of coronary events (RR 0.92, 95% CI 0.83, 1.01 for each 4g/day increase; p=0.09).

Fruit fibre (g/day) and coronary events
• No association • Limited evidence

Type 2 diabetes mellitus

8.49. Nine cohort studies were identified that presented evidence on fruit fibre intake and type 2 diabetes mellitus incidence, all of which were included in a meta-analysis (Salmeron *et al.*, 1997a; Salmeron *et al.*, 1997b; Meyer *et al.*, 2000; Stevens *et al.*, 2002; Montonen *et al.*, 2003; Hodge *et al.*, 2004; Schulze *et al.*, 2004a; Barclay *et al.*, 2007; Schulze *et al.*, 2007b). One cohort study was subsequently identified in the update search (Hopping *et al.*, 2010) (Cardio-metabolic review, diabetes and glycaemia review chapter p 98-102; Update search table 125).

8.50. No significant association is indicated between consumption of fruit fibre and the incidence of type 2 diabetes mellitus (RR 0.99, 95% CI 0.95, 1.04 for each 4g/day increase; p=0.7).The study identified in the update search indicates no significant association between consumption of fruit fibre and the incidence of type 2 diabetes mellitus.

Fruit fibre (g/day) and type 2 diabetes mellitus
• No association • Adequate evidence

Colo-rectal cancer

8.51. Six cohort studies were identified that presented evidence on fruit fibre and colo-rectal cancer, all of which were included in a meta-analysis (Michels *et al.*, 2005; Lin *et al.*, 2005; Bingham *et al.*, 2005; Wakai *et al.*, 2007; Nomura *et al.*, 2007; Schatzkin *et al.*, 2007). One cohort study that was a later follow-up of the EPIC cohort (Bingham *et al.*, 2005) was subsequently identified in the update search, but it reported fruit and vegetable fibre combined and could not be included in the meta-analysis (Murphy *et al.*, 2012). (Colo-rectal health review, p 152-155, 281, 284; Update search table 224).

8.52. No significant association is indicated between fruit fibre consumption and incidence of colo-rectal cancer (RR 0.96, 95% CI 0.91, 1.02 for each 4g/day increase; p=0.206).

Fruit fibre (g/day) and colo-rectal cancer
• No association • Adequate evidence

Vegetable fibre

Cardiovascular disease

8.53. One cohort study was identified that presented evidence on vegetable fibre intake and cardiovascular disease (Liu *et al.*, 2002). Two cohort studies were subsequently identified in the update search (Eshak *et al.*, 2010; Chuang *et al.*, 2012) (Cardio-metabolic review, cardiovascular disease chapter p 78-81, 84; Update search table 23). A further meta-analysis was performed which included the studies found in the update search (Threapleton *et al.*, 2013d) and also included one later study (Buyken *et al.*, 2010). The results from the later meta-analysis were used.

8.54. An association is indicated between higher consumption of vegetable fibre and a reduced incidence of coronary events (RR 0.92, 95% CI 0.87, 0.96 for each 4g/day increase; p<0.001).

Vegetable fibre (g/day) and cardiovascular disease
• Association • Limited evidence • The direction of the association indicates greater consumption of vegetable fibre is beneficial to health • The association is biologically relevant

Coronary events

8.55. Six cohort studies were identified that presented evidence on vegetable fibre intake and coronary events, all of which were included in a meta-analysis (Pietinen *et al.*, 1996; Rimm *et al.*, 1996; Wolk *et al.*, 1999; Liu *et al.*, 2002; Mozaffarian *et al.*, 2003; Streppel *et al.*, 2008). One cohort study was subsequently identified in the update search (Eshak *et al.*, 2010) (Cardio-metabolic review, cardiovascular disease chapter p 78-81, 84; Update search table 24). A further meta-analysis was performed which included the studies found in the update search (Threapleton *et al.*, 2013d) and also included two later studies (Crowe *et al.*, 2012; Threapleton *et al.*, 2013a). The results from the later meta-analysis were used.

8.56. A non-significant borderline association is indicated between consumption of vegetable fibre and the incidence of coronary events (RR 0.94, 95% CI 0.89, 1.00 for each 4g/day increase; p=0.03).

Vegetable fibre (g/day) and coronary events
• Association • Limited evidence • The direction of the association indicates greater consumption of vegetable fibre is beneficial to health • The association is biologically relevant

Type 2 diabetes mellitus

8.57. Nine cohort studies were identified that presented evidence on vegetable fibre and type 2 diabetes mellitus, all of which were included in a meta-analysis (Salmeron *et al.*, 1997a; Salmeron *et al.*, 1997b; Meyer *et al.*, 2000; Montonen *et al.*, 2003; Hodge *et al.*, 2004; Schulze *et al.*, 2004a; Barclay *et al.*, 2007; Schulze *et al.*, 2007b; Wannamethee *et al.*, 2009). One cohort study was subsequently identified in the update search (Hopping *et al.*, 2010) (Cardio-metabolic review, diabetes and glycaemia chapter p 103-107; Update search table 126).

8.58. No significant association is indicated between consumption of vegetable fibre and the incidence of type 2 diabetes mellitus (RR 0.99, 95% CI 0.94, 1.04 for each 4g/day increase; p=0.7). The study identified in the update search indicates higher consumption of vegetable fibre is associated with reduced incidence of type 2 diabetes mellitus in men, but not women.

Vegetable fibre (g/day) and type 2 diabetes mellitus
• No association • Adequate evidence

Colo-rectal cancer

8.59. Seven cohort studies were identified, in six publications, that presented evidence on vegetable fibre and colo-rectal cancer, all of which were included in a meta-analysis (Michels *et al.*, 2005; Lin *et al.*, 2005; Bingham *et al.*, 2005; Wakai *et al.*, 2007; Nomura *et al.*, 2007; Schatzkin *et al.*, 2007). One cohort study that was a later follow-up of the EPIC cohort (Bingham *et al.*, 2005) was subsequently identified in the update search, but it reported fruit and vegetable fibre combined and could not be included in the meta-analysis (Murphy *et al.*, 2012) (Colo-rectal health review, p 152-155, 276, 279; Update search table 224).

8.60. No significant association is indicated between vegetable fibre consumption and incidence of colo-rectal cancer (RR 0.99, 95% CI 0.96, 1.02 for each 4g/day increase; p=0.59). The study identified in the update search reports follow-up data from the EPIC study included in the meta-analysis (Bingham *et al.*, 2005) and indicates consumption of vegetable fibre is not associated with incidence of colo-rectal cancer.

Vegetable fibre (g/day) and colo-rectal cancer
• No association • Adequate evidence

Fruit and vegetable fibre

Faecal weight and intestinal transit time

8.61. Six randomised controlled trials were identified that presented evidence on fruit and vegetable fibre in relation to transit time and faecal weight (Stasse-Wolthuis *et al.*, 1980; Tinker *et al.*, 1991; Wisker *et al.*, 1994a; Wisker *et al.*, 1994b; Cherbut *et al.*, 1997; Johnson *et al.*, 2006). The data on dietary intakes were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health review, p 32, 36, 39, 45, 47, 51).

8.62. One trial reports carrot consumption increases faecal weight and moisture content (Wisker *et al.*, 1994b). Potato fibre consumption is reported to increase faecal weight in one trial (Cherbut *et al.*, 1997) and consumption of prunes is reported to increase faecal weight in one trial (Tinker *et al.*, 1991). A higher mixed fruit and vegetable intake is reported to increase faecal weight, with a less consistent increase in faecal moisture content (Stasse-Wolthuis *et al.*, 1980; Lampe *et al.*, 1992). Three trials report on intestinal transit times and generally observe a decrease in response to higher fruit and vegetable fibres intake (Stasse-Wolthuis *et al.*, 1980; Cherbut *et al.*, 1997; Johnson *et al.*, 2006).

8.63. Conclusions are limited to those foods studied and cannot be broadened to other fruit and vegetable fibres without further studies being conducted.

Fruit and vegetable fibre and faecal weight
• Effect • Moderate evidence • The direction of the effect demonstrates higher consumption of fibre from carrots, potato[8], prunes or citrus fruits is potentially beneficial to health • The effect is potentially biologically relevant

Fruit and vegetable fibre and intestinal transit times
• Effect • Limited evidence • The direction of the effect demonstrates greater consumption of fibre from carrots, potato, prunes or citrus fruits is potentially beneficial to health • The effect is potentially biologically relevant

Legume fibre

Type 2 diabetes mellitus

8.64. Three cohort studies were identified that presented evidence on legume fibre intake and incidence of type 2 diabetes mellitus, all of which were included in a meta-analysis (Meyer *et al.*, 2000; Stevens *et al.*, 2002; Hodge *et al.*, 2004). No further studies were identified in the update search (Cardio-metabolic review, diabetes and glycaemia chapter p 108-110).

8.65. No significant association is indicated between consumption of legume fibre and the incidence of type 2 diabetes mellitus (RR 1.01, 95% CI 0.98, 1.04 for each 1g/day increase; p=0.6).

Legume fibre (g/day) and type 2 diabetes mellitus
• No association • Limited evidence

[8] Potatoes are botanically classified as a vegetable, but they are classified nutritionally as a starchy food

Faecal weight and intestinal transit time

8.66. Five randomised controlled trials were identified that presented evidence on legume fibre in relation to transit time and faecal weight (Tsai *et al.*, 1983; Effertz *et al.*, 1991; Lampe *et al.*, 1992; Stephen *et al.*, 1995; Johnson *et al.*, 2006). The data on dietary intakes were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health review, p 45).

8.67. An effect of higher legume fibre (pea fibre, lentils or soy polysaccharide) intake on increased faecal weight is demonstrated in all trials. Little or no significant effect of higher legume fibre on intestinal transit time is demonstrated.

Legume fibre and faecal weight
• Effect • Moderate evidence • The direction of the effect demonstrates higher consumption of legume fibres is potentially beneficial to health • The biological relevance is unclear due to size of supplements, and it is unclear whether this finding is applicable to all legume fibres

Legume fibre and intestinal transit time
• No effect • Limited evidence

Colo-rectal cancer

8.68. Five cohort studies were identified that presented evidence on legume fibre and colo-rectal cancer, all of which were included in a meta-analysis (Lin *et al.*, 2005; Bingham *et al.*, 2005; Wakai *et al.*, 2007; Nomura *et al.*, 2007; Schatzkin *et al.*, 2007) (Colo-rectal health review, p 286, 288).

8.69. No significant association is indicated between legume fibre consumption and incidence of colo-rectal cancer (RR 0.98, 95% CI 0.94, 1.02 for each 1g/day increase; p=0.275)

Legume fibre (g/day) and colo-rectal cancer
• No association • Moderate evidence

Legume intake

Cardiovascular disease events

8.70. Five cohort studies were identified in four publications that presented evidence on legume (non-soy) intake and total cardiovascular disease events (Bazzano *et al.*, 2001; Joshipura *et al.*, 2009; Nagura *et al.*, 2009; Panagiotakos *et al.*, 2009). The data were insufficiently comparable for a meta-analysis to be performed. One cohort study was subsequently identified in the update search (Park *et al.*, 2011) (Cardio-metabolic review, cardiovascular Disease chapter p 120-121).

8.71. Two cohort studies indicate higher legume (non-soy) consumption is associated with a reduced incidence of cardiovascular disease (Bazzano *et al.*, 2001; Nagura *et al.*, 2009). The other three cohort studies indicate no significant association between legume (non-soy) consumption and the incidence of cardiovascular disease. The study identified in the update search indicates an association between higher legume (non-soy) consumption and a reduced incidence of cardiovascular disease in women, but not men.

8.72. Six cohort studies were identified that presented evidence on legume-intake (non-soy) and combined incidence of coronary events and stroke, all of which were included in a meta-analysis (Fraser *et al.*, 1992; Joshipura *et al.*, 1999; Bazzano *et al.*, 2001; Joshipura *et al.*, 2009; Nagura *et al.*, 2009; Panagiotakos *et al.*, 2009) (Cardio-metabolic review, cardiovascular disease chapter p 126-127).

8.73. No significant association is indicated between legume (non-soy) consumption and the incidence of cardiovascular disease (RR 0.96, 95% CI 0.90, 1.03 for each 0.25 serving/day; p=0.2). There is considerable heterogeneity in the data (I^2= 73%). One study, which only adjusts results for age, has a strong influence on heterogeneity When this study is excluded from the analysis heterogeneity is reduced (I^2=53%) and there is still no significant association between legume (non-soy) consumption and the incidence of cardiovascular disease (RR 0.98, 95% CI 0.93, 1.04 for each 0.25 serving/day).

Legume intake (serving/day) and cardiovascular disease events
• No association • Moderate evidence

Cereal fibre

Cardiovascular disease

8.74. One cohort study was identified that presented evidence on cereal fibre intake and cardiovascular disease incidence. (Liu *et al.*, 2002). One cohort study was subsequently identified in the update search (Chuang *et al.*, 2012) (Cardio-metabolic review, cardiovascular disease chapter p 63-65; Update search table 19). A meta-analysis was performed (Threapleton *et al.*, 2013d) which included the studies already identified and three other studies (Buyken *et al.*, 2010; Baer *et al.*, 2011; Threapleton *et al.*, 2013a). The results from this meta-analysis were used.

8.75. A non-significant borderline association is indicated between consumption of cereal fibre and the incidence of cardiovascular disease (RR 0.92, 95% CI 0.84, 1.00 for each 7g/day increase; p=0.06).

Cereal fibre (g/day) and cardiovascular disease
• No association • Limited evidence

Coronary events

8.76. Eight cohort studies were identified that presented evidence on cereal fibre intake and coronary events (Fehily *et al.*, 1993; Pietinen *et al.*, 1996; Rimm *et al.*, 1996; Wolk *et al.*, 1999; Liu *et al.*, 2002; Mozaffarian *et al.*, 2003; Streppel *et al.*, 2008; Kaushik *et al.*, 2009), six of which were included in a meta-analysis. Two cohort studies could not be included in a meta-analysis as they did not report the necessary data. One indicated no significant association between cereal fibre and coronary events (Kaushik et al., 2009) while the other study did not report a statistical comparison (Fehily et al., 1993). One cohort study was subsequently identified in the update search (Eshak et al., 2010) (Cardio-metabolic review, Cardiovascular Disease chapter p 65-70, 73-75; Update search table 20). A further meta-analysis was performed which included the study found in the update search (Threapleton *et al.*, 2013d) and also included three other studies (Baer *et al.*, 2011; Crowe *et al.*, 2012; Threapleton *et al.*, 2013a). The results from the later meta-analysis were used.

8.77. An association between higher consumption of cereal fibre and a reduced incidence of cardiovascular disease is indicated (RR 0.84, 95% CI 0.76, 0.94 for each 7g/day increase; p=0.003).

Cereal fibre (g/day) and coronary events
• Association • Adequate evidence • The direction of the association indicates higher consumption of fibre in cereals is beneficial to health • The association is biologically relevant

Blood pressure

8.78. Five randomised controlled trials were identified that presented evidence on oat bran and β-glucans in relation to blood pressure (Swain *et al.*, 1990; Saltzman *et al.*, 2001; Davy *et al.*, 2002b; He *et al.*, 2004; Maki *et al.*, 2007a), four of which were included in a meta-analysis (Saltzman *et al.*, 2001; Davy *et al.*, 2002b; He *et al.*, 2004; Maki *et al.*, 2007a). One trial could not be included in a meta-analysis as it did not report the necessary data (Swain *et al.*, 1990). One trial was subsequently identified in the update search (Charlton *et al.*, 2012) (Cardio-metabolic review, blood pressure and incident hypertension chapter p 94-102; Update search table 52).). All trials compared oat bran and/or oat meal containing diets to a refined wheat control. Two trials also supplemented subjects with refined oat β-glucan (Maki *et al.*, 2007a; Charlton *et al.*, 2012). One of the trials was a weight-loss trial (Saltzman *et al.*, 2001).

8.79. An effect is demonstrated for higher oat bran and β-glucan consumption on reducing systolic blood pressure (-2.86 mmHg, 95% CI -4.87, -0.85 mmHg; p<0.01).

8.80. An effect is demonstrated for higher oat bran and β-glucan consumption on reducing diastolic blood pressure (-1.45 mmHg, 95% CI -2.68, -0.22 mmHg; p=0.02).

8.81. The trial not included in the meta-analysis reports no significant effect of oat bran on blood pressure (Swain *et al.*, 1990). The trial identified in the update search reports no significant effect of β-glucan-rich oat products on blood pressure

Oat bran and β-glucans and systolic and diastolic blood pressure
• Effect • Moderate evidence • The direction of the effect demonstrates higher consumption of oat bran and oat β-glucans is beneficial to health • The effect is biologically relevant

Fasting blood lipids

8.82. Seven randomised controlled trials were identified that presented evidence on oat bran and β-glucan supplementation in relation to fasting total cholesterol, HDL- and LDL-cholesterol concentration (Swain *et al.*, 1990; Johnston, 1998; Romero *et al.*, 1998; Saltzman *et al.*, 2001; Davy *et al.*, 2002b; Chen *et al.*, 2006; Keenan *et al.*, 2007). All of the trials, except one (Swain *et al.*, 1990), also presented evidence on oat bran and β-glucan supplementation in relation to fasting triacylglycerol concentration. One trial was subsequently identified in the update search (Charlton *et al.*, 2012) (Cardio-metabolic review, hyperlipidaemia and blood lipids chapter p 398-409; Update search tables 80-83). All trials, except one that supplemented subjects with refined barley β-glucans (Keenan *et al.*, 2007), compared oat bran and/or oat meal containing diets to a refined wheat control or in one case wheat bran (Romero *et al.*, 1998). One trial also supplemented subjects with refined oat β-glucan (Charlton *et al.*, 2012). One of the trials was a weight-loss trial (Saltzman *et al.*, 2001).

8.83. An effect is demonstrated for the consumption of oat bran or β-glucan supplements on lowering fasting total cholesterol concentration (-0.27mmol/L, 95% CI -0.43, -0.10; p=0.002). The trial identified in the update search reports no significant effect of β-glucan-rich oat products on fasting total cholesterol concentration.

8.84. An effect is demonstrated for the consumption of oat bran or β-glucan supplements on lowering fasting LDL-cholesterol concentration (-0.22mmol/L, 95% CI -0.34, -0.10; p<0.001). The trial identified in the update search reports no significant effect of β-glucan-rich oat products on fasting total cholesterol concentration.

8.85. No significant effect is demonstrated for the consumption of oat bran or β-glucan supplements on fasting HDL-cholesterol concentration (0.02mmol/L, 95% CI -0.04, 0.08; p=0.55). The trial identified in the update search reports no significant effect of β-glucan-rich oat products on fasting HDL-cholesterol concentration.

8.86. An effect is demonstrated for the consumption of oat bran or β-glucan supplements on lowering fasting triacylglycerol concentration (-0.17mmol/L, 95% CI -0.31, -0.02; p=0.03). The trial identified in the update search reports no significant effect of β-glucan-rich oat products on fasting triacylglycerol concentration.

Oat bran and β-glucans and fasting total cholesterol concentration
• Effect • Moderate evidence • The direction of the effect demonstrates higher consumption of oat bran or β-glucan supplementation is beneficial to health • The effect is biologically relevant

Oat bran and β-glucans and fasting HDL-cholesterol concentration
• No effect • Adequate evidence

Oat bran and β-glucans and fasting LDL- cholesterol concentration
• Effect • Adequate evidence • The direction of the effect demonstrates higher consumption of oat bran or β-glucan supplementation is beneficial to health • The effect is biologically relevant

Oat bran and β-glucans and fasting triacylglycerol concentration
• Effect • Moderate evidence • The direction of the effect demonstrates higher consumption higher consumption of oat bran or β-glucan supplementation is beneficial to health • The effect is biologically relevant

Fasting blood glucose

8.87. Four randomised controlled trials were identified that presented evidence on oat bran and β-glucan supplements in relation to fasting blood glucose concentration, all of which were included in a meta-analysis (Saltzman *et al.*, 2001; Chen *et al.*, 2006; Maki *et al.*, 2007a; Smith *et al.*, 2008). Two trials were subsequently identified in the update search (Bays *et al.*, 2011; Charlton *et al.*, 2012) (Cardio-metabolic review, diabetes and glycaemia chapter p 308-310; Update search table 155). Two trials compared oat bran containing diets to a refined wheat control (Saltzman *et al.*, 2001; Chen *et al.*, 2006). Four trials supplemented subjects with refined β-glucans, either from barley or oats (Maki *et al.*, 2007a; Smith *et al.*, 2008; Bays *et al.*, 2011; Charlton *et al.*, 2012). One of the trials was a weight-loss trial (Saltzman *et al.*, 2001).

8.88. No significant effect is demonstrated for the consumption of oat bran, barley or oat β-glucan supplements on fasting blood glucose concentration (-0.08 mmol/L, 95% CI -0.21, 0.06; p=0.25). The two trials identified in the update search report no significant effect of barley or oat β-glucan supplements on fasting blood glucose concentration.

Oat bran and β-glucans and fasting blood glucose concentration
• No effect • Adequate evidence

Type 2 diabetes mellitus

8.89. Eleven cohort studies were identified that presented evidence on cereal fibre and incidence of type 2 diabetes mellitus, all of which were included in a meta-analysis (Salmeron *et al.*, 1997a; Salmeron *et al.*, 1997b; Meyer *et al.*, 2000; Stevens *et al.*, 2002; Montonen *et al.*, 2003; Hodge *et al.*, 2004; Schulze *et al.*, 2004a; Barclay *et al.*, 2007; Krishnan *et al.*, 2007; Schulze *et al.*, 2007b; Wannamethee *et al.*, 2009). One cohort study was subsequently identified in the update search (Hopping *et al.*, 2010) (Cardio-metabolic review, diabetes and glycaemia chapter p 92-97; Update search table 127).

8.90. An association between higher consumption of cereal fibre and a reduced incidence of type 2 diabetes mellitus is indicated (RR 0.79, 95% CI 0.72, 0.87 for each 7g/day increase; $p<0.001$). The study identified in the update search indicates higher cereal fibre consumption is associated with a reduced incidence of type 2 diabetes mellitus.

Cereal fibre (g/day) and type 2 diabetes mellitus
• Association • Adequate evidence • The direction of the association indicates higher consumption of fibre in cereals is beneficial to health • The association is biologically relevant

Fasting insulin

8.91. Four randomised controlled trials were identified that presented evidence on β-glucan in relation to fasting insulin concentration, all of which were included in a meta-analysis (Saltzman *et al.*, 2001; Chen *et al.*, 2006; Maki *et al.*, 2007a; Smith *et al.*, 2008). Two trials were subsequently identified in the update search (Bays *et al.*, 2011; Charlton *et al.*, 2012) (Cardio-metabolic review, diabetes and glycaemia chapter p 311-312; Update search table 161). Two trials compared oat bran containing diets to a refined wheat control (Saltzman *et al.*, 2001; Chen *et al.*, 2006). Four trials supplemented subjects with refined β-glucans, either from barley or oats (Maki *et al.*, 2007a; Smith *et al.*, 2008; Bays *et al.*, 2011; Charlton *et al.*, 2012). One of the trials was a weight-loss trial (Saltzman *et al.*, 2001).

8.92. Due to variation between the different methodologies used to measure fasting insulin concentration, it was not possible to conduct a meta-analysis. All of the trials identified in the systematic review report no significant effect of oat bran or β-glucan supplements on fasting insulin concentration. Of the trials identified in the update search, one reports no significant effect of β-glucan supplements on fasting insulin concentration and the other reports a dose of 6g/day refined β-glucans, but not 3g/day, to reduce fasting insulin concentration (Bays *et al.*, 2011).

Oat bran and β-glucans and fasting insulin concentration
• No effect • Moderate evidence

Energy intake

Oat fibre and β-glucans

8.93. Fifteen randomised controlled trials in 17 publications were identified that presented evidence on oat bran/oatmeal, barley fibre or β-glucan supplements in relation to energy intake (Whyte *et al.*, 1992; Abrahamsson *et al.*, 1994; Johnston, 1998; Reynolds *et al.*, 2000; Davy *et al.*, 2002a; Davy *et al.*, 2002b; Kerckhoffs *et al.*, 2003; He *et al.*, 2004; Robitaille *et al.*, 2005; Chen *et al.*, 2006; Keenan *et al.*, 2007; Theuwissen & Mensink, 2007; Maki *et al.*, 2007a; Maki *et al.*, 2007b; Smith *et al.*, 2008; Sundberg, 2008; Kohl *et al.*, 2009), twelve of which were included in a meta-analysis (Whyte *et al.*, 1992; Abrahamsson *et al.*, 1994; Reynolds *et al.*, 2000; Davy *et al.*, 2002a; Kerckhoffs *et al.*, 2003; Robitaille *et al.*, 2005; Chen *et al.*, 2006; Theuwissen & Mensink, 2007; Maki *et al.*, 2007a; Maki *et al.*, 2007b; Smith *et al.*, 2008; Sundberg, 2008). Two trials reported energy intake data in two publications (Davy *et al.*, 2002a; Davy *et al.*, 2002b) and (He *et al.*, 2004; Chen *et al.*, 2006). Three trials could not be included in the meta-analyses as they did not report the necessary data (Johnston, 1998; Keenan *et al.*, 2007; Kohl *et al.*, 2009). (Cardio-metabolic review, energy intake and eating motivation chapter p 156-159). Wheat products or wheat fibre was the comparison group for most of the studies.

8.94. No significant effect is demonstrated for the consumption of oat bran, barley fibre or β-glucan supplements on energy intake (48kJ, 95% CI -265kJ, 362kJ; p=0.76). The trials not included in the meta-analysis report no significant effect of oat bran or purified β-glucan supplements on energy intake (Johnston, 1998; Keenan *et al.*, 2007; Kohl *et al.*, 2009).

Oat bran and β-glucans and energy intake
• No effect • Adequate evidence

Cereal fibre excluding oat fibre

8.95. Five randomised controlled trials were identified that presented evidence on cereal bran (excluding oat bran) in relation to energy intake, all of which were included in a meta-analysis (Tredger *et al.*, 1991; Sanders & Reddy, 1992; Jenkins *et al.*, 1998; Vuksan *et al.*, 1999; Jenkins *et al.*, 2000) (Cardio-metabolic review, energy intake and eating motivation chapter p. 153-155). Bran has been defined as the edible outer covering of any seed. Three trials investigated wheat bran in comparison to a lower fibre control product and two trials reported data on rice bran and cocoa bran. Two trials compared wheat bran to either sugar beet bran (Tredger *et al.*, 1991) or rice bran (Sanders & Reddy, 1992). None of the trials were energy restriction trials.

8.96. No significant effect is demonstrated for cereal bran consumption on energy intake (-56 kJ, 95% CI -612 kJ, 499 kJ; p=0.84).

Cereal fibre excluding oat fibre and energy intake
• No effect • Moderate evidence

Faecal weight and intestinal transit time

Wheat fibre

8.97. Fourteen randomised controlled trials were identified that presented evidence on wheat fibre in relation to faecal weight, all of which were included in a meta-analysis (Southgate *et al.*, 1976; Stasse-Wolthuis *et al.*, 1980; Andersson *et al.*, 1983; Spiller *et al.*, 1986; Stevens *et al.*, 1988; Tomlin & Read, 1988; Cummings *et al.*, 1996; Jenkins *et al.*, 1998; Vuksan *et al.*, 1999; Jenkins *et al.*, 1999a; McRorie *et al.*, 2000; McIntosh *et al.*, 2003; Bird *et al.*, 2008; Vuksan *et al.*, 2008) One trial provided two estimates on faecal output (Jenkins *et al.*, 1999a). No further trials were identified in the update search (Colo-rectal health Review p 34-42).

8.98. Twelve randomised controlled trials were identified that presented evidence on wheat fibre in relation to intestinal transit time (Connell & Smith, 1974; Jenkins *et al.*, 1975; Cummings *et al.*, 1976; Wyman *et al.*, 1976; Stasse-Wolthuis *et al.*, 1980; Andersson *et al.*, 1983; Spiller *et al.*, 1986; Stevens *et al.*, 1988; Tomlin & Read, 1988; Vuksan *et al.*, 1999; Muir *et al.*, 2004; Vuksan *et al.*, 2008) (Colo-rectal health review, p 34-41, 43).

8.99. An effect is demonstrated for wheat fibre (between 10-29g/day) on decreasing intestinal transit time on the forest plot, but the heterogeneity is too high to report the meta-analysis pooled estimate. An investigation into the heterogeneity demonstrated that the amount of reduction in transit times in response to wheat fibre is greater when initial intestinal transit times are longer and *vice versa*.

Wheat fibre and faecal weight and intestinal transit time
• Effect • Adequate evidence • The direction of the effect demonstrates higher consumption of fibre from wheat is potentially beneficial to health • The effect is potentially biologically relevant

Non-wheat cereal fibre

8.100. Five randomised controlled trials were identified that presented evidence on non-wheat cereal fibres in relation to transit time or faecal weight (Tomlin & Read, 1988; Cherbut *et al.*, 1997; Grasten *et al.*, 2000; McIntosh *et al.*, 2003; Bird *et al.*, 2008). The data on dietary intakes were insufficient to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health review, p 44-46).

8.101. An effect on increasing faecal weight is reported for a novel barley (containing more resistant starch and non-starch polysaccharide than normal barley) (Bird *et al.*, 2008), a higher rye-fibre diet (McIntosh *et al.*, 2003), whole grain rye bread (Grasten *et al.*, 2000) maize fibre (Cherbut *et al.*, 1997) and rice bran (Tomlin & Read, 1988). The effects on faecal weight are the same or similar to the effect sizes for wheat fibre. Total intestinal transit time reductions are demonstrated in the three trials that measured transit time (Tomlin & Read, 1988; Cherbut *et al.*, 1997; Grasten *et al.*, 2000).

Non-wheat cereal fibre and faecal weight
• Effect • Adequate evidence • The direction of the effect demonstrates higher consumption of mixed non-wheat cereal fibre is potentially beneficial to health • The effect is potentially biologically relevant

Non-wheat cereal fibre and intestinal transit time
• Effect • Limited evidence • The direction of the effect demonstrates higher consumption of mixed non-wheat cereal fibre is potentially beneficial to health • The effect is potentially biologically relevant

Faecal pH and short chain fatty acid content

Wheat fibre

8.102. Eight randomised controlled trials were identified that presented evidence on wheat bran in relation to faecal pH and short chain fatty acid content (Jenkins *et al.*, 1975; Cummings *et al.*, 1976; Lampe *et al.*, 1992; Jenkins *et al.*, 1998; Jenkins *et al.*, 1999a; McIntosh *et al.*, 2003; Muir *et al.*, 2004; Bird *et al.*, 2008). The data on measures of faecal short chain fatty acid content were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health review, p 98-99, 102-103).

8.103. Wheat bran increases faecal weight, with a concomitant increase in total daily faecal short chain fatty acid excretion, but most trials observe no significant effect on the concentration or relative proportions of faecal short chain fatty acid concentrations. One trial observes wheat bran to increase faecal butyrate concentrations compared with a lower fibre bread control (Lampe *et al.*, 1992), and another trial observes finely ground, but not coarse ground, wheat bran to increase faecal butyrate concentration (Jenkins *et al.*, 1999a).

Wheat fibre and faecal pH and short chain fatty acid content
• No effect • Moderate evidence

Non-wheat cereal fibre

8.104. Six randomised controlled trials were identified that presented evidence on cereal intake in relation to faecal pH and short chain fatty acid content (Noakes *et al.*, 1996; Grasten *et al.*, 2000; McIntosh *et al.*, 2003; Grasten *et al.*, 2007; Bird *et al.*, 2008; Carvalho-Wells *et al.*, 2010). The data on measures of faecal short chain fatty acid content were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health review, p 98, 102-103).

8.105. Three trials investigate the effect of rye bran on faecal pH and short chain fatty acid content: one reports a higher rye-fibre diet to reduce faecal pH and increase faecal butyrate concentration (McIntosh *et al.*, 2003), one reports the same, but in male subjects only (Grasten *et al.*, 2000) and the other reports no significant effect (Grasten *et al.*, 2007). An effect on reducing faecal pH and increasing faecal butyrate concentration is reported for a novel barley (containing more resistant starch and non-starch polysaccharide than normal barley) (Bird *et al.*, 2008). No significant effect on faecal pH or short chain fatty acid content is reported for maize bran (Carvalho-Wells *et al.*, 2010) or oat bran (Noakes *et al.*, 1996).

Non-wheat cereal fibre and faecal pH and short chain fatty acid content
• No effect • Moderate evidence

Constipation

8.106. Ten randomised controlled trials were identified that presented evidence on cereal fibre and constipation (Sculati & Giampiccoli, 1984; Anderson & Whichelow, 1985; Finlay, 1988; Brown & Everett, 1990; Mantle, 1992; Badiali *et al.*, 1995; Howard *et al.*, 2000; Rees *et al.*, 2005; Hongisto *et al.*, 2006; Holma *et al.*, 2010) (Colo-rectal health review, p 121-142). The data were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search. The trials were conducted in three subject groups: hospitalised or institutionalised patients (Finlay, 1988; Brown & Everett, 1990; Mantle, 1992; Howard *et al.*, 2000), patients with self-reported constipation (Sculati & Giampiccoli, 1984; Badiali *et al.*, 1995; Rees *et al.*, 2005; Hongisto *et al.*, 2006; Holma *et al.*, 2010) and one in pregnant women (Anderson & Whichelow, 1985). Improvement in constipation was measured as either a decrease in laxative use or a self-reported improvement in symptoms of constipation.

8.107. In the four trials in hospitalised or institutionalised patients, cereal fibre (wheat bran) is reported as effective in reducing laxative use in two trials (Brown & Everett, 1990; Howard *et al.*, 2000) and the other two trials report a tendency for cereal fibre to reduce laxative use (Finlay, 1988; Mantle, 1992). Only one of the four trials, observes an increase in bowel frequency in response to cereal fibre (Brown & Everett, 1990). One of the other trials reports a higher proportion of patients with 2-7 bowel motions a week of soft or firm stools in response to wheat bran supplementation (Mantle, 1992).

8.108. In the trials of adults with self-reported constipation, two trials report higher rye bread consumption to increase bowel frequency, improve stool consistency and result in less strain at defecation relative to lower-fibre bread consumption (Hongisto *et al.*, 2006; Holma *et al.*, 2010). One trial reports a wheat fibre preparation increases bowel frequency, improves stool consistency and lessens abdominal pain (Sculati & Giampiccoli, 1984). Two trials in subjects from gastroenterology outpatient clinics report no significant effect of higher wheat bran consumption on bowel frequency, strain at defecation or other symptoms (Badiali *et al.*, 1995; Rees *et al.*, 2005).

8.109. One trial, investigating the effect of wheat-bran on constipation in pregnancy, reports wheat bran supplementation to increase bowel movement frequency and corn fibre supplementation to have a non-significant tendency to increase bowel movement frequency (Anderson & Whichelow, 1985). No significant effect of either wheat or corn fibre supplementation on stool consistency or abdominal pain is demonstrated.

Cereal fibre and constipation
• Effect • Moderate evidence • The direction of the effect demonstrates higher consumption of fibre from cereals is beneficial to health • The effect is biologically relevant

Intestinal transit time

8.110. Five randomised controlled trials were identified that presented evidence on wheat fibre in relation to total intestinal transit time in outpatients with constipation or patients with self-reported constipation (Corinaldesi *et al.*, 1982; Badiali *et al.*, 1995; Rees *et al.*, 2005; Hongisto *et al.*, 2006; Holma *et al.*, 2010). No further trials were identified in the update search (Colo-rectal health review, p 127, 132, 139-140). The transit time methodologies were insufficiently comparable to allow a meta-analysis. Three trials supplemented subjects with wheat bran fibre relative to placebo (Corinaldesi et al. 1982, Badiali et al. 1995, Rees et al. 2005). Two trials assessed the effect of rye bread relative to a lower-fibre bread (Hongisto *et al.*, 2006; Holma *et al.*, 2010).

8.111. Two trials observe an effect of wheat bran (20g/day) on reducing intestinal transit time. The other trial supplementing with wheat bran (10-20g/day) reports a non-significant tendency for intestinal transit time to be reduced (Rees *et al.*, 2005). In the other two trials consumption of rye bread decreases intestinal transit time in one trial and one trial reports a non-significant tendency for a decrease (Hongisto *et al.*, 2006), relative to a lower fibre bread control.

Wheat fibre and intestinal transit time in patients with constipation
• Effect • Limited evidence • The direction of the effect demonstrates higher consumption of wheat bran fibre is potentially beneficial to health • The effect is potentially biologically relevant

Colo-rectal adenoma recurrence

8.112. Three randomised controlled trials were identified that presented evidence on wheat fibre in relation to colo-rectal adenoma recurrence (MacLennan *et al.*, 1995; Alberts *et al.*, 2000; Ishikawa *et al.*, 2005). A pooled analysis of the Wheat Bran Fiber Trial (Alberts *et al.*, 2000) and the Polyp Prevention Trial (a lower fat, high fibre dietary intervention) (Schatzkin *et al.*, 2000) was also identified (Jacobs *et al.*, 2006). No further trials were identified in the update search (Colo-rectal health review, p 199-203).

8.113. All three trials report no significant effect of cereal supplements on recurrence of colo-rectal adenomas. The pooled analysis reports an adjusted odds ratio for colo-rectal adenoma recurrence in all subjects on both trials in relation to the intervention and observes no significant association (OR 0.91, 95% CI 0.78, 1.06). A separate analysis for men and women observes the intervention is associated with a reduced odds of colo-rectal recurrence in men (OR 0.81, 95% CI 0.67, 0.98), but not in women.

Wheat fibre and colo-rectal adenomas
• No effect • Moderate evidence

Colo-rectal cancer

8.114. Six cohort studies were identified, in five publications, that presented evidence on cereal fibre intake and colo-rectal cancer, all of which were included in a meta-analysis (Michels *et al.*, 2005; Lin *et al.*, 2005; Nomura *et al.*, 2007; Schatzkin *et al.*, 2007; Murphy *et al.*, 2012) (Colo-rectal health review, p 269-271, 274; Update search table 223). In the colorectal health review, Bingham et al. (2005) was included in the meta-analysis (Bingham *et al.*, 2005). A further analysis was performed to include more recent data, as identified in the update search, from the EPIC cohort (Murphy et al 2012). The pooled estimate provided in paragraph 8.115 is taken from the updated meta-analysis (Annex 1 additional meta-analyses)

8.115. An association is indicated between higher consumption of cereal fibre and a reduced incidence of colo-rectal cancer (RR 0.92, 95% CI 0.89, 0.96, for each 7g/day increase; p=0.005).

Cereal fibre (g/day) and colo-rectal cancer
• Association • Moderate evidence • The direction of the association indicates higher cereal fibre consumption is beneficial to health • The association is biologically relevant

Higher dietary fibre breakfast cereals

Coronary events

8.116. Four cohort studies were identified that presented evidence on breakfast cereal and coronary events (Key *et al.*, 1996; Jacobs, Jr. *et al.*, 1998; Liu *et al.*, 1999; Liu *et al.*, 2003), three of which were included in a meta-analysis. One cohort study, that could not be included in a meta-analysis as it did not report the necessary data, indicated no significant association between bran cereals and coronary events (Key *et al.*, 1996). No further studies were identified in the update search (Cardio-metabolic review, cardiovascular disease chapter p 113-114, 117-118).

8.117. An association is indicated between higher dietary fibre breakfast cereal consumption and reduced incidence of coronary events (RR 0.87, CI 0.80-0.94 for each 0.5 serving/day increase; p=0.001). The studies define high dietary breakfast cereals as either having greater than 25% whole grains or bran content by weight or bran containing breakfast cereals.

Higher dietary fibre breakfast cereals (serving/day) and coronary events
• Association • Limited evidence • The direction of the association indicates higher consumption of higher fibre breakfast cereals is beneficial to health • The association is biologically relevant

Energy intake

8.118. Six randomised controlled trials were identified that presented evidence on breakfast cereal in relation to energy intake, all of which were included in a meta-analysis (Kirk *et al.*, 1997; Kleemola *et al.*, 1999; Mattes, 2002; Waller *et al.*, 2004; Rodriguez-Rodriguez *et al.*, 2008; Zaveri & Drummond, 2009) (Cardio-metabolic review, Energy intake and eating motivation chapter p 162-165). The trials assessed the effect of incorporating ready-to-eat breakfast cereals (or cereal bars) into the diet as compared with a 'no change' protocol.

8.119. No consistent effect is demonstrated, between breakfast cereal consumption and energy intake, as observed from the forest plot, but the heterogeneity is too high to report the meta-analysis pooled estimate (I^2=80%). Only one trial reports a significant effect with the replacement of two meals/day with higher carbohydrate ready to eat breakfast cereal reducing energy intake (Mattes, 2002), but the results from other trials show no consistent effect.

Higher dietary fibre breakfast cereals and energy intake
• No effect • Moderate evidence

Type 2 diabetes mellitus

8.120. Three cohort studies were identified that presented evidence on higher dietary fibre breakfast cereals and incidence of type 2 diabetes mellitus, all of which were included in a meta-analysis (Liu *et al.*, 2000a; Hodge *et al.*, 2004; Kochar *et al.*, 2007) (Cardio-metabolic review, diabetes and glycaemia chapter p 126-127).

8.121. An association is indicated between higher dietary fibre breakfast cereal consumption and reduced incidence of type 2 diabetes mellitus (RR 0.89, CI 0.86-0.92 for each 0.5 serving/day increase; p<0.001). The studies define high dietary breakfast cereals as either having greater than 25% whole grains or bran content by weight, wheat germ, muesli and other breakfast cereal or cold whole grain types.

Higher dietary fibre breakfast cereals (serving/day) and type 2 diabetes mellitus
• Association • Moderate evidence • The direction of the association indicates higher consumption of higher fibre breakfast cereals is beneficial to health • The association is biologically relevant

Whole grain Bread

Type 2 diabetes mellitus

8.122. Four cohort studies were identified that presented evidence on whole grain bread and incidence of type 2 diabetes mellitus (Liu *et al.*, 2000a; Hodge *et al.*, 2004; Simmons *et al.*, 2007; Schulze *et al.*, 2007a), three of which were included in a meta-analysis. One study could not be included in a meta-analysis as it did not report the necessary data (Simmons *et al.*, 2007). No further studies were identified in the update search (Cardio-metabolic review, diabetes and glycaemia chapter p 123-125).

8.123. An association is indicated between higher whole grain bread consumption and reduced incidence of type 2 diabetes mellitus (RR 0.93, CI 0.90-0.96 for each 0.5 serving/day increase; $p<0.001$). The study not included in the meta-analysis indicates higher whole grain bread consumption is associated with reduced incidence of type 2 diabetes mellitus.

Whole grain bread (serving/day) and type 2 diabetes mellitus
• Association • Moderate evidence • The direction of the association indicates higher consumption of whole grain bread is beneficial to health • The association is biologically relevant

Total cereals

Cardiovascular disease

8.124. Four cohort studies were identified that presented evidence on total cereals and total cardiovascular disease(Jacobs, Jr. *et al.*, 2001; Liu *et al.*, 2003; Drogan *et al.*, 2007; Panagiotakos *et al.*, 2009). Two studies could not be included in a meta-analysis as they did not report the necessary data, leaving an insufficient number of studies to enable a meta-analysis to be performed. (Cardio-metabolic review, cardiovascular disease chapter p 106-107).

8.125. One study indicates no significant association between total cereal consumption and cardiovascular disease (Panagiotakos *et al.*, 2009). The other three studies indicate higher consumption of higher fibre and total cereal types (Liu *et al.*, 2003) and whole grain bread (Jacobs, Jr. *et al.*, 2001; Drogan *et al.*, 2007) is associated with a reduced incidence of cardiovascular disease. The results are consistent with those relating to whole grains consumption.

Total cereals (serving/day) and cardiovascular disease
• Association • Moderate evidence • The direction of the association indicates higher whole grains consumption is beneficial to health • The association is biologically relevant

Whole grains

8.126. The definitions of whole grains vary between studies. The term 'whole grains' can have several meanings from 'whole of the grain' through to physically intact structures. Whole grains include whole wheat, whole-wheat flour, wheat flakes, bulgar wheat, whole and rolled oats, oatmeal, oat flakes, brown rice, whole rye and rye flour and whole barley. A precise definition is needed and the role of intact versus milled grains established (Cummings & Stephen, 2007). A commonly used definition of whole grains is that of the American Association of Cereal Chemists[9]. The definition of whole grains is discussed further in Chapter 2, paragraphs 2.37 to 2.39.

Cardiovascular disease

8.127. Six cohort studies from nine publications were identified that presented evidence on whole grains intake and incident cardiovascular disease events (Jacobs, Jr. *et al.*, 1998; Liu *et al.*, 1999; Liu *et al.*, 2000b; Steffen *et al.*, 2003; Jensen *et al.*, 2004; Sahyoun *et al.*, 2006; Drogan *et al.*, 2007; Jacobs, Jr. *et al.*, 2007; Nettleton *et al.*, 2008), five of which were included in a meta-analysis. Four publications were not included in the meta-analysis. Two publications were an early analysis (Jacobs, Jr. *et al.*, 1998; Liu *et al.*, 1999) of data that is more fully presented data in later publications (Liu *et al.*, 2000b; Jacobs, Jr. *et al.*, 2007) . Two publications presented data on the same study (Steffen *et al.*, 2003; Nettleton *et al.*, 2008) and the data from the former was used in meta-analysis. One study only presented results for individual sources of whole grains, which were in broad agreement with the results from the meta-analysis (Drogan *et al.*, 2007). One cohort study was subsequently identified in the update search (Nilsson *et al.*, 2012) (Cardio-metabolic review, cardiovascular disease chapter p. 141-142, 149-150; Update search table 35).

8.128. An association is indicated between higher consumption of whole grains and a reduced incidence of cardiovascular disease (RR 0.95, 95% CI 0.92, 0.97 for each 0.5 serving/day; $p<0.001$). The study identified in the update search indicates no significant association between whole grains intake and incidence of cardiovascular disease.

Whole grains (serving/day) and cardiovascular disease
• Association • Moderate evidence • The direction of the association indicates higher whole grains consumption is beneficial to health • The association is biologically relevant

[9] Whole grains consist of the 'intact, ground, cracked or flaked caryopsis (grain), whose principal components-the starchy endosperm, germ and bran-are present in the same relative proportions as they exist in the intact caryopsis' (American Association of Cereal Chemists International, 1999). To be considered whole grain, a food may contain intact grains, minimally processed grains, or milled grains from which no component has been removed during the milling process.

Stroke

8.129. Three cohort studies were identified that presented evidence on whole grains intake and stroke events, all of which were included in a meta-analysis (Liu *et al.*, 2000b; Steffen *et al.*, 2003; Jacobs, Jr. *et al.*, 2007). No further studies were identified in the update search (Cardio-metabolic review, Cardiovascular disease chapter p 146-148).

8.130. An association is indicated between higher consumption of whole grains and a reduced incidence of stroke (RR 0.96, 95% CI 0.93, 0.99 for each 0.5 serving/day; p=0.02).

Whole grains (serving/day) and stroke
• Association • Limited evidence • The direction of the association indicates higher whole grains consumption is beneficial to health • The association is biologically relevant

Hypertension

8.131. Three cohort studies were identified that presented evidence on whole grains intake and incident hypertension, all of which were included in a meta-analysis (Steffen *et al.*, 2005; Wang *et al.*, 2007; Flint *et al.*, 2009). No further studies were identified in the update search (Cardio-metabolic review, cardiovascular disease chapter p 39-42).

8.132. An association is indicated between higher consumption of whole grains and a reduced incidence of hypertension (RR 0.95, 95% CI 0.93, 0.96 for each 0.5 serving/day; p<0.001).

Whole grains (serving/day) and hypertension
• Association • Limited evidence • The direction of the association indicates higher whole grains consumption is beneficial to health • The association is biologically relevant

Blood pressure

8.133. Three randomised controlled trials were identified that presented evidence on whole grains intake in relation to blood pressure, all of which were included in the meta-analyses for systolic and diastolic blood pressure (Davy *et al.*, 2002b; Howard *et al.*, 2006b; Andersson *et al.*, 2007; Tinker *et al.*, 2008). Two studies presented data from the same trial (Howard *et al.*, 2006b; Tinker *et al.*, 2008), and the data from Howard et al. (2006b) was used in the meta-analysis. Three trials were subsequently identified in the update search (Brownlee *et al.*, 2010; Tighe *et al.*, 2010; Kristensen *et al.*, 2012) (Cardio-metabolic review, incident hypertension and blood pressure chapter p 114-120; Update search table 54). All trials compared whole grain diets to refined grain control diets. One of the trials is a weight-loss trial (Kristensen *et al.*, 2012).

8.134. No significant effect is demonstrated for whole grains consumption on systolic blood pressure (0.2 mmHg, 95% CI, -1.6, 2.0 mmHg; p=0.85). Of the trials identified in the update search two report no significant effect (Brownlee *et al.*, 2010; Kristensen *et al.*, 2012). One reports higher whole grains consumption to reduce systolic blood pressure (Tighe *et al.*, 2010).

8.135. No significant effect is demonstrated for whole grains consumption on diastolic blood pressure (-0.3 mmHg, 95% CI, -0.9, 0.4 mmHg; p=0.43). The three trials identified in the update search report no significant effect of whole grains consumption on diastolic blood pressure.

8.136. One trial, contributes 92% to the pooled estimate for systolic blood pressure and 97% for diastolic blood pressure (Howard *et al.*, 2006b). This trial results in a very small increase in whole grains consumption (less than one serving per day) and also results in weight loss differences between experimental groups.

Whole grains and systolic and diastolic blood pressure
• No effect • Moderate evidence

Fasting blood lipids

8.137. Five randomised controlled trials were identified that presented evidence on whole grains intake in relation to total cholesterol, LDL-cholesterol, HDL-cholesterol and triacylglycerol concentration, all of which were included in the meta-analysis (Saltzman *et al.*, 2001; Davy *et al.*, 2002a; Howard *et al.*, 2006b; Andersson *et al.*, 2007; Kim *et al.*, 2008). Three trials were subsequently identified in the update search (Tighe *et al.*, 2010; Brownlee *et al.*, 2010; Kristensen *et al.*, 2012) (Cardio-metabolic review, hyperlipidaemias and blood lipids chapter p 430-443; Update search tables 95-98). All trials compared whole grain diets to refined grain control diets. Three of the trials are weight-loss trials (Saltzman *et al.*, 2001; Kim *et al.*, 2008; Kristensen *et al.*, 2012).

8.138. No significant effect is demonstrated for whole grains consumption on fasting total cholesterol concentration (0.04mmol/L, 95% CI -0.12, 0.20; p=0.49). The trials identified in the update search report inconsistent effects of whole grains consumption on fasting total cholesterol concentration: one no significant effect (Brownlee *et al.*, 2010); one a decrease with refined grain consumption (Tighe *et al.*, 2010); and the other reports a decrease with whole grains consumption (Kristensen *et al.*, 2012).

8.139. No significant effect is demonstrated for whole grains consumption on fasting triacylglycerol concentration (-0.02mmol/L, 95% CI -0.06, 0.03; p=0.46). The trials identified in the update search report no significant effect of whole grains consumption on fasting triacylglycerol concentration.

8.140. No consistent effect is demonstrated for whole grains consumption on either fasting HDL- or LDL- cholesterol concentration on the forest plot, but the heterogeneity is too high to report the meta-analysis pooled estimate (I^2=77% and 79%, respectively). The heterogeneity may be due to differences in subjects' weight change between the trials. One trial reports a higher BMI with higher whole grains consumption, as compared with refined grain consumption (Andersson *et al.*, 2007), while the weight loss trials report greater weight loss after higher whole grains consumption, as compared with refined grain consumption (Saltzman *et al.*, 2001; Howard *et al.*, 2006b; Kim *et al.*, 2008; Kristensen *et al.*, 2012). The trials identified in the update search report no significant effect on HDL-cholesterol concentration and inconsistent effects of whole grains consumption on LDL-cholesterol concentration: one no significant effect (Brownlee *et al.*, 2010); one a decrease with refined grain consumption (Tighe *et al.*, 2010); and the other reports a decrease with whole grains consumption (Kristensen *et al.*, 2012).

Whole grains and fasting total cholesterol and triacylglycerol concentration
• No effect • Adequate evidence

Energy intake

8.141. Three randomised controlled trials were identified that presented evidence on whole grains intake in relation to energy intake, all of which were included in a meta-analysis (Turpeinen *et al.*, 2000; Howard *et al.*, 2006a; Andersson *et al.*, 2007; Tinker *et al.*, 2008). Two publications reported on the same trial (Howard *et al.*, 2006a; Tinker *et al.*, 2008) and the data from Tinker et al (2008) was used in the meta-analysis (Cardio-metabolic review, energy intake and eating motivation chapter p 170-172). All trials compared whole grain diets to refined grain control diets.

8.142. An effect is demonstrated for higher whole grains consumption on reduced energy intake (-360 kJ, 95% CI -642 kJ, -79 kJ; p=0.01). One trial contributes 75% to the pooled estimate (Tinker *et al.*, 2008) and is responsible for the significant effect. The dietary intervention in this study is to promote a decrease in fat intake and increases in vegetable, fruit, and grain consumption, with the increase in whole grains intake being small (daily whole grains servings increased from 1.1 to 1.4).

Whole grains and energy intake
• Effect • Limited evidence • The direction of the effect demonstrates higher whole grains consumption may be beneficial to health, but it is not possible to exclude confounding by other dietary variables • The effect is biologically relevant

Type 2 diabetes mellitus

8.143. Eight cohort studies in seven publications (de Munter et al. 2007 reported on the Nurse's Health Study and Nurse's Health Study 2) were identified that presented evidence on whole grains intake and incidence of type 2 diabetes mellitus, all of which were included in a meta-analysis (Meyer *et al.*, 2000; Liu *et al.*, 2000c; Fung *et al.*, 2002; Montonen *et al.*, 2003; Van Dam *et al.*, 2006; de Munter *et al.*, 2007; Fisher *et al.*, 2009). Two publications reported on the same study (Liu *et al.*, 2000c; de Munter *et al.*, 2007) and the data from the most recent was used in meta-analysis (de Munter *et al.*, 2007). One publication reporting a longer follow up period of three cohorts already included was identified in the update search (Sun *et al.*, 2010) (Cardio-metabolic review, diabetes and glycaemia chapter p 165-172; Update search table 129). A further meta-analysis was performed (Aune *et al.*, 2013) which included the studies found in the update search and also included three later studies (Ericson *et al.*, 2013; Parker *et al.*, 2013; Wirstrom *et al.*, 2013). The results from the later meta-analysis were used.

8.144. An association is indicated, between higher whole grains consumption and reduced incidence of type 2 diabetes, as indicated from the forest plot, but the heterogeneity is too high to report the meta-analysis pooled estimate (I^2=82%).

Whole grains (serving/day) and type 2 diabetes mellitus
• Association • Moderate evidence • The direction of the association indicates higher whole grains consumption is beneficial to health • It is not possible to quantify the magnitude of the association due to differences in definition and serving size used. It is therefore not possible to determine the biological relevance of this finding

Fasting blood glucose

8.145. Four randomised controlled trials were identified that presented evidence on whole grains intake in relation to fasting blood glucose, all of which were included in a meta-analysis (Saltzman *et al.*, 2001; Howard *et al.*, 2006b; Andersson *et al.*, 2007; Kim *et al.*, 2008; Tinker *et al.*, 2008). Two publications reported on the same trial (Howard *et al.*, 2006b; Tinker *et al.*, 2008) and the data from (Howard *et al.*, 2006b) were used in meta-analysis. Four trials were subsequently identified in the update search (Tighe *et al.*, 2010; Brownlee *et al.*, 2010; Lankinen *et al.*, 2011; Kristensen *et al.*, 2012) (Cardio-metabolic review, diabetes and glycaemia chapter p 325-327; Update search table 159). All trials compared whole grain diets to refined grain control diets. Three of the trials are weight-loss trials (Saltzman *et al.*, 2001; Kim *et al.*, 2008; Kristensen *et al.*, 2012).

8.146. No significant effect is demonstrated for whole grains consumption on fasting blood glucose concentration (-0.05mmol/L, 95% CI -0.12, 0.02; p=0.14). The trials identified in the update search report no significant effect of whole grains consumption on fasting blood glucose concentration.

Whole grains and fasting blood glucose concentration
• No effect • Adequate evidence

Fasting blood insulin

8.147. Four randomised controlled trials were identified that presented evidence on whole grains intake in relation to fasting insulin (Saltzman *et al.*, 2001; Howard *et al.*, 2006b; Andersson *et al.*, 2007; Kim *et al.*, 2008; Tinker *et al.*, 2008). Two publications reported on the same trial (Howard *et al.*, 2006b; Tinker *et al.*, 2008). Four trials were subsequently identified in the update search (Tighe *et al.*, 2010; Brownlee *et al.*, 2010; Lankinen *et al.*, 2011; Kristensen *et al.*, 2012) (Cardio-metabolic review, diabetes and glycaemia chapter p 328-329; Update search table 162). All trials compared whole grain diets to refined grain control diets. Three of the trials are weight-loss trials (Saltzman *et al.*, 2001; Kim *et al.*, 2008; Kristensen *et al.*, 2012).

8.148. Due to variation between the different methodologies used to measure fasting insulin concentration, it was not possible to conduct a meta-analysis. All of the trials identified in the systematic review and the update search report no significant effect of whole grains consumption on fasting insulin concentration.

Whole grains and fasting blood insulin concentration
• No effect • Adequate evidence

Insulin sensitivity/resistance

8.149. Three randomised controlled trials were identified that presented evidence on whole grains intake in relation to insulin sensitivity (Saltzman *et al.*, 2001; Howard *et al.*, 2006b; Andersson *et al.*, 2007; Tinker *et al.*, 2008). Two publications reported on the same trial (Howard *et al.*, 2006b; Tinker *et al.*, 2008). Four trials were subsequently identified in the update search (Tighe *et al.*, 2010; Brownlee *et al.*, 2010; Lankinen *et al.*, 2011; Kristensen *et al.*, 2012) (Cardio-metabolic review, diabetes chapter p 330-332; Update search table 166). All trials compared whole grain diets to refined grain control diets. Two of the trials are weight-loss trials (Saltzman *et al.*, 2001; Kristensen *et al.*, 2012).

8.150. Due to variation between the different methodologies used to measure fasting insulin concentration, it was not possible to conduct a meta-analysis. All of the trials identified in the systematic review and the update search report no significant effect of whole grains consumption on insulin sensitivity/resistance.

Whole grains and insulin sensitivity/resistance
• No effect • Adequate evidence

Colo-rectal cancer

8.151. Three cohort studies were identified that presented evidence on whole grains intake and colo-rectal cancer incidence, all of which were included in a meta-analysis (Pietinen *et al.*, 1999; Larsson *et al.*, 2005; Schatzkin *et al.*, 2007). No further studies were identified in the update search (Colo-rectal health review, p 154-155, 291-292, 297).

8.152. A non-significant borderline association is indicated between higher whole grains consumption and reduced incidence of colo-rectal cancer (RR 0.97, CI 0.94-1.00 for each 0.5 serving/day increase; p=0.077).

Whole grains (serving/day) and colo-rectal cancer
• No association • Limited evidence

Colon cancer

8.153. Three cohort studies were identified that presented evidence on whole grains intake and colon cancer incidence, all of which were included in a meta-analysis (McCullough *et al.*, 2003; Larsson *et al.*, 2005; Schatzkin *et al.*, 2007). No further studies were identified in the update search (Colo-rectal health review, p 154-155, 291-292, 297).

8.154. An association is indicated between higher whole grains consumption and reduced incidence of colon cancer (RR 0.97, CI 0.95-0.99 for each 0.5 serving/day increase; p=0.015).

Whole grains (serving/day) and colon cancer
• Association • Limited evidence • The direction of the association indicates higher whole grains consumption is beneficial to health • The association is biologically relevant

Children and adolescents

2.40. Three cohort studies were conducted in children and adolescents aged five years or more.

Body fatness and fat distribution

8.155. Three cohort studies were identified that presented evidence on diets differing in dietary fibre content and body fatness and fat distribution (Johnson *et al.*, 2008; Cheng *et al.*, 2009; Albertson *et al.*, 2009). One study determined grams of fibre per day and anthropometric data at puberty onset and over the subsequent 4 years (Cheng *et al.*, 2009). Another study determined dietary fibre density in children aged 5 and 7 years and fatness at 9 years among UK children (Johnson *et al.*, 2008). The other study calculated the cumulative percentage of days that girls consumed cereal during childhood (between ages 11.5 and 18.6 years) in relation to percentage body fat at age 18.6 years of age (Albertson *et al.*, 2009). It is unclear in this study if this refers to breakfast cereals only or to total cereals. The outcomes of the studies were assessed differently: by skinfold measurements (Cheng *et al.*, 2009), dual-energy X-ray absorptiometry (Johnson *et al.*, 2008) or bioimpedance (Albertson *et al.*, 2009).

8.156. No significant association is indicated between dietary fibre intake and body fatness in two of the studies, but one does indicate that girls who ate cereal on a greater percentage of days during childhood had a lower percentage body fat and were more likely to exhibit higher levels of physical activity (Albertson *et al.*, 2009). Overall, none of the results support a strong relationship between dietary fibre intake and adiposity in children.

Dietary fibre (g/day) and body fatness
• No association • Limited evidence

Outcomes where there is insufficient evidence

8.157. The tables below detail the exposures and outcomes where there are too few studies or trials that meet the inclusion criteria for the review (e.g. by study duration, publication date, health of subjects, design of intervention) to draw a conclusion. A full description of the studies can be found in the relevant systematic reviews.

Table 8.1 Insufficient evidence-cohort studies

Risk factor/health outcome/measure	**Exposure**
Total cardiovascular disease	legume fibre
Coronary events	whole grain foods bread legumes
Stroke	soluble and insoluble fibre fibre in fruit fibre in vegetables (including potatoes) fibre in cereals total cereal intake legume intake
Fibrinogen	dietary fibre
Coronary and aortic calcification	dietary fibre
Vascular function	whole grains consumption
Incident hypertension	dietary fibre intake total cereal intake legume intake
Blood pressure	dietary fibre
Fasting blood lipids	dietary fibre intake breakfast cereals
Fibrinogen	dietary fibre intake
Change in body weight	dietary fibre intake, total cereal intake whole grains intake
Body fatness and fat distribution	dietary fibre, cereal intake whole grains intake
Type 2 diabetes mellitus	whole legume intake
Impaired glucose tolerance and altered fasting glycaemia	dietary fibre bread legumes
Insulinemia and insulin resistance/sensitivity	dietary fibre
Insulin resistance	dietary fibre
Colo-rectal cancer	soluble and insoluble fibre whole grains intake
Colon cancer	soluble and insoluble fibre vegetable fibre fruit fibre legume fibre

Rectal Cancer	soluble and insoluble fibre cereal fibre whole grains intake vegetable fibre fruit fibre legume fibre
Colo-rectal adenoma recurrence	dietary fibre

Table 8.2 Insufficient evidence-randomised controlled trials

Risk factor/health outcome/measure	**Exposure**
Blood pressure	dietary fibre breakfast cereals legumes
Fasting blood lipids	breakfast cereals legumes dietary fibre oat fibre and β-glucans whole grains intake
IL-6, PAI-1,CRP, fibrinogen	dietary fibre intake whole grains intake
Change in body weight and BMI	whole grains intake
Body fatness and fat distribution	dietary fibre whole grains intake
Energy intake	legumes
Eating motivation	oat fibre and β-glucans breakfast cereal consumption
Type 2 diabetes mellitus	whole grains intake
Impaired glucose tolerance	oat fibre and β-glucans
Fasting glycaemia	breakfast cereals legumes
Insulin resistance/sensitivity	dietary fibre oat fibre and β-glucans legumes
Insulinemia	breakfast cereals legumes
Glycosylated blood proteins	dietary fibre
Dental caries	dietary fibre
Intestinal transit time	whole grains intake
Stool frequency	whole grains intake oat fibre and β-glucans
Faecal weight	oat fibre and β-glucans
Faecal bacteria populations	whole grains intake wheat bran legume fibre oat fibre and β-glucans
Faecal weight in people with constipation	cereal fibre

Table 8.3 Inconsistent evidence

Risk factor/health outcome	**Exposure**
Coronary events	total cereal intake
HDL and LDL-cholesterol	whole grains intake

Summary and conclusions

8.158. This assessment is based on 74 prospective cohort studies and 131 randomised controlled trials investigating the relationship between dietary fibre intake and cardio-metabolic health and colo-rectal health outcomes. There is a lack of available evidence on dietary fibre intake in relation to oral health outcomes. Three prospective cohort studies were conducted in children and adolescents. With observational studies there is substantial potential for biases and the possibility of confounding by an extraneous variable that correlates with both the dependent variable and the independent variable (residual confounding) and any associations must be interpreted with caution.

8.159. Overall the evidence from prospective cohort studies indicates diets rich in dietary fibre are associated with a lower incidence of cardiovascular diseases, coronary events, stroke and type 2 diabetes mellitus, colo-rectal cancer, colon and rectal cancer; no association with change in body weight in adults or body fatness in children and adolescents is indicated. Although the definitions used to define whole grains vary between studies, higher whole grains consumption is associated with a lower incidence of cardiovascular disease, stroke, hypertension, type 2 diabetes mellitus and colon cancer, although the evidence is based on a smaller number of studies than for dietary fibre. Higher cereal fibre consumption is associated with a lower incidence of coronary events, type 2 diabetes mellitus, colo-rectal cancer, although the evidence is more limited for constituents of dietary fibres due to the smaller number of studies.

8.160. Randomised controlled trials indicate there is no effect of dietary fibre intake on cardiovascular or type 2 diabetes mellitus risk factors. Trials indicate dietary fibre, wheat fibre and other cereal fibres decrease intestinal transit times and increase faecal mass. Trials found that legume fibre increased faecal weight. Higher cereal fibre consumption is shown to be beneficial in the treatment of constipation. Trials indicate no effect of cereal fibre supplements on recurrence of colo-rectal adenomas (colo-rectal adenomas can be a precursor of colo-rectal cancer). Trials in subjects receiving oat fibre and β-glucan indicate this cereal fibre component has beneficial effects on fasting blood lipid concentrations and blood pressure. The doses of oat bran and isolated β-glucan used in these trials generally exceed levels currently consumed in a typical UK diet, but recent and future developments of novel food ingredients may substantially increase the intake of isolated β-glucan.

8.161. Randomised trials indicate that dietary fibre intake has no effect on body weight change or energy intake. There is limited evidence from trials, however, that a higher whole grains intake may decrease total energy intake, but more evidence is required to draw firm conclusions.

8.162. Overall, prospective cohort studies provide mainly adequate evidence that a diet higher in dietary fibre is associated with a lower incidence of cardiovascular diseases, coronary events, type 2 diabetes mellitus, colo-rectal cancer, colon and rectal cancer. The randomised controlled trials indicate no effect of dietary fibre on cardio-metabolic risk factors, except for relatively high doses of β-glucan fibre, but do indicate a beneficial effect of dietary fibre in the treatment of constipation and on decreasing intestinal transit times and increasing faecal mass.

Chapter 9. Non-digestible oligosaccharides and resistant starch

9.1. This assessment is based on 87 randomised controlled trials investigating the relationship between supplements of resistant starch, oligosaccharide or inulin and cardio-metabolic and colo-rectal. Adverse effects on gastrointestinal symptoms such as bloating, borborygmi or flatulence, have not been considered. There were no prospective cohort studies investigating the relationship between oligosaccharides and the relevant health/disease outcomes. Twenty three randomised trials were conducted in infants, children and adolescents. Links to the individual systematic reviews and update search are given in Annex 1 and the relevant page or table is given in the text below.

9.2. Evidence on health/disease outcomes have been discussed in detail only where there are sufficient data for a conclusion to be drawn, from studies meeting the pre-agreed inclusion criteria. Outcomes where there are too few such studies to reach a conclusion, are listed at the end of the chapter (see table 9.1). Outcomes in which the evidence was considered too inconsistent to make a valid judgement are listed in table 9.2. Therefore, not all 87 randomised controlled trials will be listed in this chapter.

9.3. Information on resistant starch, non-digestible oligosaccharide or inulin intake in the UK is not available from the National Diet and Nutrition Survey. Based on the analytically determined inulin and fructo-oligosaccharide (fructans) content of specific foods (cereals, fruits and vegetables) and consumption data from the USA and Europe, it has been estimated that the intake of inulin-type fructans ranges between 1 and 4 g/day (van Loo *et al.*, 1995). A study in 66 subjects in the UK (Dunn *et al.*, 2011) estimates mean intakes of inulin and oligofructose to be approximately 4.0g/day and 3.8g/day respectively. The main source of inulin-type fructans in a typical Western diet are wheat and onions. Fructans are the storage carbohydrates in artichokes and chicory with small amounts of low molecular weight forms found in wheat, rye, asparagus and members of the onion, leek and garlic family (Cummings & Stephen, 2007).

9.4. National statistics for consumption of starchy foods have been used to estimate the intake of resistant starch in Europe as 4.1 g/day (no variance data given) with variation in mean intakes from 3.2 to 5.7 between different European countries (Asp *et al.*, 1996). The calculations are based on literature data using the Englyst method (Englyst *et al.*, 1992) or separate analyses of foods with the Englyst method or the modified Berry method (Champ *et al.*, 2003). Resistant starch intake in Sweden is estimated to be 3.2g/day (no variance data given) (Liljeberg Elmstahl, 2002), based on consumption data from the 1997-1998 national survey and calculated from the individual foods analysed using a modified Berry method (Akerberg *et al.*, 1998). In the UK, mean resistant starch intake has been estimated at about 2.8g/day (no variance data given), derived from published food and food ingredient values for resistant starch in conjunction with the average weekly consumption of these foods (and foods prepared from the food ingredients) by the general UK population (Annual Report of the Food Survey Committee, 1985) (Tomlin & Read, 1990).

9.5. Using national food consumption data from surveys conducted during the 1980s and determination of food resistant starch content by a modified Berry method (Berry, 1986; Champ *et al.*, 2003), Italian intakes of resistant starch are estimated at 8.5 g/day (no variance data given), with regional differences (from 7.2 g/day in the north-west to 9.2 g/day in the south) (Brighenti *et al.*, 1998). In Australia, intakes of resistant starch are estimated to be between 3.4 and 9.4g/day using the 1995 National Nutrition Survey and published values of food resistant starch content. The calculations are based on literature data obtained using the Englyst method (Roberts *et al.*, 2004). In the USA, resistant starch mean intake is estimated to be approximately 4.9 g/day (range 2.8 to 7.9 g/day) based on the 1999-2002 National Health and Nutrition Examination Surveys and literature data of food resistant starch concentrations using the modified Berry method (Murphy *et al.*, 2008).

Non-digestible oligosaccharides

Fasting blood lipid concentrations

9.6. Five randomised controlled trials were identified that presented evidence on non-digestible oligosaccharide or inulin supplementation and fasting blood lipids, all of which were included in a meta-analysis (Davidson *et al.*, 1998; Jackson *et al.*, 1999; Letexier *et al.*, 2003; Forcheron & Beylot, 2007; Genta *et al.*, 2009). No further trials were identified in the update search (Cardio-metabolic review, hyperlipidaemias and blood lipids chapter p303-306).

9.7. An effect of non-digestible oligosaccharide supplementation, as compared with unsupplemented controls, on reducing fasting LDL-cholesterol is demonstrated (-0.39mmol/L, 95% CI -0.76, -0.03 mmol/L; p=0.04). The trial that reports the greatest effect of non-digestible oligosaccharide supplementation on fasting LDL-cholesterol concentration, as compared with the unsupplemented controls, also reports greater weight loss in the supplemented group as compared with the unsupplemented controls (Genta *et al.*, 2009).

9.8. No significant effect of non-digestible oligosaccharide or inulin supplementation on fasting total cholesterol (-0.13mmol/L, 95% CI -0.55, 0.30; p=0.57), HDL-cholesterol (0.04mmol/L, 95% CI -0.12, 0.20; p=0.60) or triacylglycerol concentration (-0.13mmol/L, 95% CI -0.27, 0.00; p=0.06) is demonstrated. Three trials administer 10g/day inulin compared with a maltodextrin control (Jackson *et al.*, 1999; Letexier *et al.*, 2003; Forcheron & Beylot, 2007). One trial administers approximately 12g/day fructo-oligosaccharides (as yacon syrup) compared with a similar dose of placebo syrup (Genta *et al.*, 2009). The other trial administers 18g/day inulin incorporated into chocolate, spreads and sweeteners and compared with un-supplemented products (Davidson *et al.*, 1998).

Non-digestible oligosaccharides and fasting LDL-cholesterol concentration
• Effect • Limited evidence • The direction of the effect demonstrates the change is beneficial. The magnitude of the effect is biologically relevant, but is inconsistent with the effects on other fasting lipid concentrations. The supplement doses used are above levels currently consumed in typical diets in the UK, but recent and future developments of novel food ingredients may substantially increase the intake of these oligosaccharides. • The effect is biologically relevant

Non-digestible oligosaccharides and fasting total cholesterol, HDL-cholesterol and triacylglycerol concentration
• No effect • Limited evidence

Energy intake

9.9. Eleven randomised controlled trials in adults were identified that presented evidence on non-digestible oligosaccharide or inulin supplementation and energy intake, eight of which were included in a meta-analysis (Luo *et al.*, 1996; Pedersen *et al.*, 1997; Davidson *et al.*, 1998; Cani *et al.*, 2006; Pasman *et al.*, 2006; Forcheron & Beylot, 2007; Cani *et al.*, 2009; Parnell & Reimer, 2009). Three trials could not be included in a meta-analysis as they did not report the necessary data (Letexier *et al.*, 2003; Van den Heuvel *et al.*, 2004; Genta *et al.*, 2009)(Cardio-metabolic review, Energy Intake and Eating Motivation review, p115-118).

9.10. Most trials administer either fructo-oligosaccharides or inulin, compared with a maltodextrin control; seven trials administer doses between 10-18g/day (Pedersen *et al.*, 1997; Davidson *et al.*, 1998; Letexier *et al.*, 2003; Cani *et al.*, 2006; Forcheron & Beylot, 2007; Genta *et al.*, 2009; Cani *et al.*, 2009), two trials administer doses of 20-21g/day (Luo *et al.*, 1996; Parnell & Reimer, 2009). Two trials administer a modified dextrin in a dose-response manner at doses of 30, 40, 60 or 80 g/day (Van den Heuvel *et al.*, 2004; Pasman *et al.*, 2006)

9.11. No significant effect of non-digestible oligosaccharide or inulin supplementation and energy intake is demonstrated (-378kJ, 95% CI -791, 36kJ; p=0.07). The result is of borderline statistical significance. When the dose-response effect is explored by plotting the trials by order of difference in oligosaccharide intake between study groups, it appears that only at doses greater than 20g/day is there any suggestion of an effect. The three trials not included in the meta-analysis report no significant effect of non-digestible oligosaccharide supplementation and energy intake.

Non-digestible oligosaccharides and energy intake
• No effect • Moderate evidence

Fasting blood glucose

9.12. Five randomised controlled trials in adults were identified that presented evidence on non-digestible oligosaccharide or inulin supplementation and fasting blood glucose concentration, four of which were included in a meta-analysis (Jackson *et al.*, 1999; Letexier *et al.*, 2003; Forcheron & Beylot, 2007; Genta *et al.*, 2009). One trial could not be included in a meta-analysis as it did not report the necessary data (Parnell & Reimer, 2009). No further trials were identified in the update search (Cardio-metabolic review, diabetes chapter p281-282).

9.13. Three trials administer 10g/day inulin compared with a maltodextrin control (Jackson *et al.*, 1999; Letexier *et al.*, 2003; Forcheron & Beylot, 2007). One trial administers approximately 12g/day fructo-oligosaccharides (as yacon syrup) compared with a similar dose of placebo syrup (Genta *et al.*, 2009). The other trial administers 21g/day inulin compared with a maltodextrin control (Parnell & Reimer, 2009).

9.14. No significant effect of non-digestible oligosaccharide or inulin supplementation on fasting blood glucose concentration is demonstrated (-0.13mmolo/L, 95% CI -0.46, 0.20; p=0.79). The trial not included in the meta-analysis reports fasting glucose concentration to be reduced by non-digestible oligosaccharide supplementation and increased in the control group, which reflects the difference in body weight change between the experimental groups (Parnell & Reimer, 2009). Whether there is an overall effect of non-digestible oligosaccharide supplementation compared with control is unclear.

Non-digestible oligosaccharides and fasting blood glucose concentration
• No effect • Adequate evidence

Fasting blood insulin

9.15. Five randomised controlled trials in adults were identified that presented evidence on non-digestible oligosaccharide or inulin supplementation and fasting blood insulin concentration (Jackson *et al.*, 1999; Letexier *et al.*, 2003; Forcheron & Beylot, 2007; Genta *et al.*, 2009; Parnell & Reimer, 2009). No further trials were identified in the update search (Cardio-metabolic review, diabetes chapter p284-285).

9.16. Three trials administer 10g/day inulin compared with a maltodextrin control (Jackson *et al.*, 1999; Letexier *et al.*, 2003; Forcheron & Beylot, 2007). One trial administers approximately 12g/day fructo-oligosaccharides (as yacon syrup) compared with a similar dose of placebo syrup (Genta *et al.*, 2009). The other trial administers 21g/day inulin compared with a maltodextrin control (Parnell & Reimer, 2009).

9.17. Due to variation between the different methodologies used to measure fasting insulin concentration, it was not possible to conduct a meta-analysis. No significant effect of non-digestible oligosaccharide supplementation on fasting blood insulin concentration is demonstrated in three of the trials (Jackson *et al.*, 1999; Letexier *et al.*, 2003; Forcheron & Beylot, 2007). One trial reports fasting insulin concentration to be reduced by oligosaccharide supplementation and increased in the control group, which reflects the difference in body weight change between the experimental groups (Parnell & Reimer, 2009). Whether there is an overall effect of non-digestible oligosaccharide supplementation compared with control is unclear. The other trial reports an effect of non-digestible oligosaccharide supplementation on reducing fasting blood insulin concentration as compared with the unsupplemented control, which reflects difference in body weight change between the experimental groups (Genta *et al.*, 2009).

Non-digestible oligosaccharides and fasting blood insulin concentration
• No effect • Moderate evidence

Faecal weight

9.18. Nine randomised controlled trials in adults were identified that presented evidence on non-digestible oligosaccharide or inulin supplementation in relation to faecal weight, of which eight were included in the meta-analyses (Ito *et al.*, 1990; Bouhnik *et al.*, 1996; Alles *et al.*, 1999; Van Dokkum *et al.*, 1999; Causey *et al.*, 2000; Den Hond *et al.*, 2000; Tahiri *et al.*, 2001; Scholtens *et al.*, 2006b). There was no indication that doses of less than 10g/day had any effect on faecal wet weight, so only data from doses of 10g/day or more were used. One trial was not included in the meta-analyses as this supplemented subjects with a 3g/day dose of non-digestible oligosaccharide (Swanson *et al.*, 2002). No further trials were identified in the update search (Colo-rectal health review p69-78).

9.19. Three trials supplemented subjects with galacto-oligosaccharide (doses 10-15g/day) (Ito *et al.*, 1990; Alles *et al.*, 1999; Van Dokkum *et al.*, 1999); three trials supplemented subjects with inulin (doses 15-20g/day) (Van Dokkum *et al.*, 1999; Causey *et al.*, 2000; Den Hond *et al.*, 2000); and four trials supplemented subjects with fructo-oligosaccharide (doses 10-30g/day) (Bouhnik *et al.*, 1996; Van Dokkum *et al.*, 1999; Tahiri *et al.*, 2001; Scholtens *et al.*, 2006b). All trials compared supplemented groups to either a sugar or maltodextrin control.

9.20. An effect of non-digestible oligosaccharide and inulin supplementation (10-30g/day; mean 15.4g/day) on increasing wet faecal weight is demonstrated (20g/day, 95% CI 6, 35; p=0.006 – data from eight trials). An effect of non-digestible oligosaccharide and inulin supplementation (10-20g/day; mean 14.1g/day) on increasing wet faecal weight is demonstrated (18g/day, 95% CI 3, 33; p=0.02 – data from seven trials).

9.21. An effect of fructo-oligosaccharide and inulin supplementation (10-30g/day; mean 16.4g/day) on increasing wet faecal weight is demonstrated (23g/day, 95% CI 7, 40; p=0.006; data from six trials). An effect of fructo-oligosaccharide and inulin supplementation (10-20g/day; mean 14.6/day) on increasing wet faecal weight is demonstrated (20g/day, 95% CI 3, 38; p=0.023; data from five trials)

9.22. No significant effect of galacto-oligosaccharide supplementation (10-15g/day; mean 13g/day) on wet faecal weight is demonstrated (13g/day, 95% CI-11, 37; p=0.292; data from three trials). This is limited by the small number of trials supplementing with galacto oligosaccharide.

9.23. Overall, there appears to be no difference in the faecal bulking capacity of fructo-oligosaccharide and inulin, which broadly equated to a 1-1.5g increase in faecal wet weight per 1g non-digestible oligosaccharide intake. The data are limited in relation to the faecal bulking capacity of galacto-oligosaccharide due to there being only three trials.

Non-digestible oligosaccharides and faecal weight
• Effect • Adequate evidence • The direction of the effect demonstrates the change is potentially beneficial to health. The supplement doses used are above levels currently consumed in typical diets in the UK, but recent and future developments of novel food ingredients may substantially increase the intake of these oligosaccharides. • The effect is potentially biologically relevant.

Faecal pH and short chain fatty acid content

9.24. Fifteen randomised controlled trials in adults were identified that presented evidence on non-digestible oligosaccharide or inulin supplementation in relation to faecal pH or SCFA content (Bouhnik *et al.*, 1996; Bouhnik *et al.*, 1999; Alles *et al.*, 1999; Van Dokkum *et al.*, 1999; Causey *et al.*, 2000; Den Hond *et al.*, 2000; Tuohy *et al.*, 2001; Tahiri *et al.*, 2001; Bouhnik *et al.*, 2004; Bouhnik *et al.*, 2006; Scholtens *et al.*, 2006b; Bouhnik *et al.*, 2007; Kleessen *et al.*, 2007; Ramnani *et al.*, 2010; Walton *et al.*, 2010). No further trials were identified in the update search (Colo-rectal health review p100). The data on measures of faecal pH and short chain fatty acid content were insufficiently comparable to allow a meta-analysis. Nine trials supplemented subjects with fructo-oligosaccharides (doses 2.5-30g/day) (Bouhnik *et al.*, 1996; Van Dokkum *et al.*, 1999; Bouhnik *et al.*, 1999; Tahiri *et al.*, 2001; Tuohy *et al.*, 2001; Swanson *et al.*, 2002; Bouhnik *et al.*, 2004; Bouhnik *et al.*, 2006; Scholtens *et al.*, 2006b). Three trials supplemented subjects with galacto-oligosaccharides (doses 8.5-15g/day) (Alles *et al.*, 1999; Van Dokkum *et al.*, 1999; Bouhnik *et al.*, 2004), six trials supplemented subjects with inulin (Van Dokkum *et al.*, 1999; Causey *et al.*, 2000; Bouhnik *et al.*, 2004; Bouhnik *et al.*, 2007; Kleessen *et al.*, 2007; Ramnani *et al.*, 2010) and one trial supplemented subjects with manno-oligosaccharides derived from coffee (Walton *et al.*, 2010). All trials compared supplemented groups to either a sugar or maltodextrin control.

9.25. Eight trials determine faecal SCFA content in response to non-digestible oligosaccharide or inulin supplementation and none report an effect on overall SCFA content. Five trials report no significant effect of non-digestible oligosaccharide or inulin supplementation (3-15g/day) on individual faecal SCFA content (Alles *et al.*, 1999; Swanson *et al.*, 2002; Kleessen *et al.*, 2007; Ramnani *et al.*, 2010; Walton *et al.*, 2010) three report an increase in the concentration or proportion of faecal acetate to higher doses of non-digestible oligosaccharide or inulin (15-30g/day) (Van Dokkum *et al.*, 1999; Causey *et al.*, 2000; Scholtens *et al.*, 2006b). None of the trials report an effect of non-digestible oligosaccharide or inulin supplementation on faecal pH. All trials compared supplemented groups to either a sugar or maltodextrin control.

Non-digestible oligosaccharides and faecal short chain fatty acid content and pH
• No effect • Limited evidence

Faecal bacteria

Fructo-oligosaccharides

9.26. Six randomised controlled trials in adults were identified that presented evidence on fructo-oligosaccharide supplementation (doses 2.5-20g/day) in relation to faecal bacteria content (Bouhnik *et al.*, 1996; Bouhnik *et al.*, 1999; Tuohy *et al.*, 2001; Tannock *et al.*, 2004; Bouhnik *et al.*, 2004; Bouhnik *et al.*, 2006). The data on measures of faecal bacteria content were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health review p85). All trials compared supplemented groups to either a sugar or maltodextrin control.

9.27. An effect of fructo-oligosaccharide supplementation on increasing faecal *Bifidobacterium* spp. content relative to the control group is generally reported at doses of 10g/day or more, but not at lower doses.

Fructo-oligosaccharides and faecal bacteria
• Effect at doses of 10g/day or more • Adequate evidence • Whether the effect is beneficial and of biological relevance is currently unclear. The supplement doses used are above levels currently consumed in typical diets in the UK, but recent and future developments of novel food ingredients may substantially increase the intake of these oligosaccharides.

Galacto-oligosaccharides

9.28. Seven randomised controlled trials in adults were identified that presented evidence on galacto-oligosaccharide supplementation (doses 2.5-14.4g/day) in relation to faecal bacteria content (Ito *et al.*, 1990; Alles *et al.*, 1999; Gopal *et al.*, 2003; Bouhnik *et al.*, 2004; Tannock *et al.*, 2004; Depeint *et al.*, 2008; Vulevic *et al.*, 2008) The data on measures of faecal bacteria content were insufficiently comparable to allow a meta-analysis. One trial was subsequently identified in the update search (Walton *et al.*, 2012) (Colo-rectal health review p85; Update search table 215). All trials compared supplemented groups to either a sugar or maltodextrin control.

9.29. An effect of galacto-oligosaccharide supplementation on increasing faecal *Bifidobacterium* spp. content relative to the control group is generally reported at doses of 10g/day or more, but not at lower doses. The trial identified in the update search reports galacto-oligosaccharide supplementation (8g/day) to increase faecal *Bifidobacterium* spp. content relative to the control group.

Galacto-oligosaccharides and faecal bacteria
• Effect at doses of 8g/day or more • Adequate evidence • Whether the effect is beneficial and of biological relevance is currently unclear. The supplement doses used are above levels currently consumed in typical diets in the UK, but recent and future developments of novel food ingredients may substantially increase the intake of these oligosaccharides.

Inulin

9.30. Seven randomised controlled trials in adults were identified that presented evidence on inulin supplementation (doses 5-15g/day) in relation to faecal bacteria content (Bouhnik *et al.*, 2004; Fuller *et al.*, 2007; Kleessen *et al.*, 2007; Bouhnik *et al.*, 2007; Calame *et al.*, 2008; Costabile *et al.*, 2010; Ramnani *et al.*, 2010). The data on measures of faecal bacteria content were insufficiently comparable to allow a meta-analysis. One trial was subsequently identified in the update search (Slavin & Feirtag, 2011). (Colo-rectal health review p85; Update search table 215). All trials compared supplemented groups to either a sugar or maltodextrin control.

9.31. Four trials report inulin supplementation (5-10g/day) to increase faecal *Bifidobacterium* spp. content relative to control, (Fuller *et al.*, 2007; Kleessen *et al.*, 2007; Ramnani *et al.*, 2010; Costabile *et al.*, 2010). No significant effect of inulin supplementation (5-10g/day) on faecal bacteria content is reported in three trials (Bouhnik *et al.*, 2004; Bouhnik *et al.*, 2007; Calame *et al.*, 2008). The trial identified in the update search reports no significant effect inulin supplementation (20g/day) on faecal *Bifidobacterium* spp. content. Overall, the evidence is more inconsistent than for fructo- and galacto-oligosaccharides.

Inulin and faecal bacteria
• No effect • Limited evidence

Calcium absorption

9.32. Most calcium absorption occurs in the small intestine, but about 5% has been shown to occur in the colon (Barger-Lux *et al.*, 1989). Experimental work shows that short-chain fatty acids may stimulate calcium (Trinidad *et al.*, 1996; Trinidad *et al.*, 1997; Trinidad *et al.*, 1999) and magnesium (Coudray *et al.*, 2003) absorption in the colon, suggesting that increased colonic fermentation of carbohydrate may stimulate mineral absorption. Trials conducted in children and adolescents are considered in the children and adolescents section at the end of the chapter (see paragraph 9.49).

9.33. Four randomised controlled trials were identified that presented evidence on non-digestible oligosaccharide or inulin supplementation in relation to calcium fractional absorption, all of which were included in meta-analyses (see Annex 1, additional meta-analyses). The trials supplemented subjects with doses of 10-20g/day (Van den Heuvel *et al.*, 1998; van den Heuvel *et al.*, 2000; Tahiri *et al.*, 2003; Holloway *et al.*, 2007). No further trials were identified in the update search (Colo-rectal health review p115). All trials compared supplemented groups to either a sugar or maltodextrin control.

9.34. No significant effect of non-digestible oligosaccharide or inulin supplementation on fractional calcium absorption is demonstrated (0.47%, 95% CI -3.36, 4.29; p=0.81). This is in contrast to the trials in children and adolescents where an effect is demonstrated (see paragraph 9.49).

Non-digestible oligosaccharide or inulin and fractional calcium absorption
• No effect • Moderate evidence

Resistant starch

Energy intake

9.35. Four randomised controlled trials were identified that presented evidence on resistant starch supplementation in relation to measures of energy intake, three of which were included in a meta-analysis (De Roos *et al.*, 1995; Heijnen *et al.*, 1996; Jenkins *et al.*, 1998). One trial was not included in the meta-analysis as it was unclear which type of resistant starch subjects were supplemented with (Ells *et al.*, 2005) (Cardio-metabolic review, energy intake and eating motivation chapter, p126-129). The trials compared supplemented groups to either a sugar or non-supplemented test foods control. The data were analysed separately for raw resistant starch (RS_2) and retrograde resistant starch (RS_3).

9.36. No significant effect of raw resistant starch (RS_2) supplementation on measures of energy intake is demonstrated (18.5 kJ/day, 95% CI -710.0, 747.1 kJ/day; p=0.96).

9.37. No significant effect of retrograde resistant starch (RS_3) supplementation on measures of energy intake is demonstrated (-102.8 kJ/day, 95% CI -824.1, 618.6 kJ/day; p=0.78).

Resistant starch (RS_2 and RS_3) and energy intake
• No effect • Limited evidence

Faecal weight

9.38. Ten randomised controlled trials were identified that presented evidence on resistant starch supplementation in relation to faecal weight, of which seven were included in a meta-analysis (Phillips *et al.*, 1995; Cummings *et al.*, 1996; Silvester *et al.*, 1997; Heijnen *et al.*, 1998; Jenkins *et al.*, 1998; Behall *et al.*, 2002; Muir *et al.*, 2004). There was no indication that doses of 12g/day or less had any effect on faecal wet weight, so only data from doses greater than 12g/day were used. Two trials supplemented subjects with resistant starch at doses of 12/day or less and were not included in the meta-analyses (Tomlin & Read, 1990; Stewart *et al.*, 2010). Two trials reported on chemically modified resistant starch (RS_4) in relation to faecal wet weight, which was an insufficient number to enable a meta-analysis to be performed. Resistant starch supplementation appeared to have little effect on intestinal transit time (Tomlin & Read, 1990; Cummings *et al.*, 1996; Silvester *et al.*, 1997; Behall *et al.*, 2002; Muir *et al.*, 2004) or faecal moisture content (Cummings *et al.*, 1996; Silvester *et al.*, 1997; Heijnen *et al.*, 1998; Vermorel *et al.*, 2004). No further trials were identified in the update search (Colo-rectal health review p54-63). The data were analysed separately for raw resistant starch (RS_2) and retrograde resistant starch (RS_3). Six trials reported on the effects of RS_2 (Cummings *et al.*, 1996; Silvester *et al.*, 1997; Jenkins *et al.*, 1998; Heijnen *et al.*, 1998; Behall *et al.*, 2002; Muir *et al.*, 2004); three trials reported on the effects of RS_3 (Cummings *et al.*, 1996; Heijnen *et al.*, 1998; Jenkins *et al.*, 1998) and one trial reported on the effects of RS1, 2 and 3 combined (Phillips *et al.*, 1995).

9.39. An effect of raw resistant starch (RS_2) supplementation on increasing faecal weight is demonstrated (38g/d, 95% CI 23, 53g/day; p<0.001; data from six trials). The doses range from 21.5-37g/day (mean 28.3g/day).

9.40. An effect of retrograde resistant starch (RS_3) supplementation on increasing faecal weight is demonstrated (46g/d, 95% CI 23, 68g/day; p<0.001; data from three trials). The doses range from 17.4-32g/day (mean 24.1g/day).

9.41. An effect of resistant starch types 1, 2 and 3 supplementation combined on increasing faecal weight is demonstrated (40g/d, 95% CI 26, 54g/day; p<0.001; data from seven trials). The doses range from 17-37g/day (mean 27g/d). Of the trials that investigate chemically modified resistant starch (RS_4) in relation to faecal wet weight, one reports 100g/day increases faecal weight (Vermorel *et al.*, 2004), while the other observes no significant effect on faecal weight of 12g/day of three different corn and tapioca modified starches (Stewart *et al.*, 2010).

9.42. There appears to be no difference in the faecal bulking capacity of the different types of resistant starch (1, 2 and 3), which broadly equated to a 1-2g increase in faecal wet weight per 1g resistant starch. The data are limited with regard to chemically modified resistant starch (RS_4) due to there being only two trials.

Resistant starch (RS_1, RS_2 and RS_3) and faecal weight
• Effect • Adequate evidence • The direction of the effect demonstrates the change is potentially beneficial. The supplement doses used are above levels currently consumed in typical diets in the UK, but recent and future developments of novel food ingredients may substantially increase the intake of resistant starches. • The effect is potentially biologically relevant

Faecal pH and short chain fatty acid content

9.43. Ten randomised controlled trials were identified that presented evidence on resistant starch supplementation in relation to faecal pH or short chain fatty acid content (Phillips *et al.*, 1995; Noakes *et al.*, 1996; Cummings *et al.*, 1996; Silvester *et al.*, 1997; Heijnen *et al.*, 1998; Jenkins *et al.*, 1998; Muir *et al.*, 2004; Pasman *et al.*, 2006; Fastinger *et al.*, 2008; Stewart *et al.*, 2010). No further trials were identified in the update search (Colo-rectal health review p99 and 105). The data on measures of faecal pH and short chain fatty acid content were insufficiently comparable to allow a meta-analysis.

9.44. An effect of supplementation with retrograded, granular and high amylose resistant starches (RS_1, RS_2 and RS_3) on lowering faecal pH is demonstrated in four trials (Phillips *et al.*, 1995; Noakes *et al.*, 1996; Silvester *et al.*, 1997; Muir *et al.*, 2004), but two trials report no significant effect of 30g/day (Heijnen *et al.*, 1998) and 12g/day (Stewart *et al.*, 2010). Three trials investigate the effect of chemically modified starches (RS_4). One trial reports that tapioca dextrin RS_4 supplementation lowers faecal pH, but other sources of RS_4 do not (Stewart *et al.*, 2010); the two other trials report no significant effect of RS_4 on faecal pH (Pasman *et al.*, 2006; Fastinger *et al.*, 2008).

9.45. An effect of supplementation with retrograded, granular and high amylose resistant starches (RS_1, RS_2 and RS_3) on increasing the faecal concentration or proportion of butyrate is demonstrated in five trials (Phillips *et al.*, 1995; Noakes *et al.*, 1996; Cummings *et al.*, 1996; Jenkins *et al.*, 1998; Muir *et al.*, 2004) but two trials report no significant effect of 30g/day (Heijnen *et al.*, 1998) and 12g/day (Stewart *et al.*, 2010). Of the three trials investigating chemically modified starches (RS_4), one reports all faecal SCFA concentrations, except butyrate, to be lowered by the RS4 intervention (Fastinger *et al.*, 2008), but the other two report no significant effect (Pasman *et al.*, 2006; Stewart *et al.*, 2010)

9.46. Overall, resistant starch (RS_1, RS_2 and RS_3) at doses of 20-40g/day generally lower faecal pH and increase either the concentration or proportion of faecal butyrate. There is little evidence for an effect of chemically modified resistant starch (RS_4) on these faecal parameters.

Resistant starch (RS_1, RS_2 and RS_3) and faecal short chain fatty acid content
• Effect • Moderate evidence • Whether the effect is beneficial or of biological relevance is currently unclear. The supplement doses used are above levels currently consumed in typical diets in the UK, but recent and future developments of novel food ingredients may substantially increase the intake of resistant starches.

Resistant starch (RS_1, RS_2 and RS_3) and faecal pH
• Effect • Limited evidence • Whether the effect is beneficial or of biological relevance is currently unclear. The supplement doses used are above levels currently consumed in typical diets in the UK, but recent and future developments of novel food ingredients may substantially increase the intake of resistant starches.

Faecal bacteria

9.47. Four randomised controlled trials were identified that presented evidence on resistant starch supplementation in relation to faecal *Bifidobacterium* spp. content (Jenkins *et al.*, 1999c; Pasman *et al.*, 2006; Fastinger *et al.*, 2008; Beards *et al.*, 2010). No further trials were identified in the update search (Colo-rectal health review p85). The data on measures of faecal bacteria content were insufficiently comparable to allow a meta-analysis. Three trials supplemented subjects with chemically modified resistant starch (RS_4) (Pasman *et al.*, 2006; Fastinger *et al.*, 2008; Beards *et al.*, 2010), the other trials supplemented subjects with either raw resistant starch (RS_2) or retrograde resistant starch (RS_3) (Jenkins *et al.*, 1999c).

9.48. No significant effect of supplementation with any of the resistant starches on faecal *Bifidobacterium* spp. content is demonstrated.

Resistant starch (RS_2, RS_3 and RS_4) and faecal bacteria
• No effect • Adequate evidence

Infants, children and adolescents

Calcium absorption

9.49. Five randomised controlled trials were identified that presented evidence on non-digestible oligosaccharide or inulin supplementation in relation to calcium fractional absorption, all of which were included in meta-analyses (van den Heuvel *et al.*, 1999; Griffin *et al.*, 2003; Griffin & Abrams, 2005; Abrams *et al.*, 2005; van den Heuvel *et al.*, 2009) (Annex 1, additional meta-analyses). The trials supplemented subjects with doses of 5-15g/day (Colo-rectal health review p115). All trials compared supplemented groups to either a sugar or maltodextrin control.

9.50. A significant effect is demonstrated for non-digestible oligosaccharide or inulin supplementation on increased fractional calcium absorption (4.95%, 95% CI 1.62, 8.27; p=0.003). This is in contrast to the trials in adults where no effect is demonstrated (see paragraph 9.34).

Non-digestible oligosaccharide or inulin and fractional calcium absorption
• Effect • Moderate evidence • Whether the effect is beneficial and of biological relevance is currently unclear. The supplement doses used are above levels currently consumed in typical diets in the UK, but recent and future developments of novel food ingredients may substantially increase the intake of these oligosaccharides.

Faecal bacteria

Infants up to three months of age

9.51. Fourteen trials in fifteen publications randomised controlled trials were identified that presented evidence on supplementation of infant formula (2-8g/L) with non-digestible oligosaccharide (mainly galacto-oligosaccharide) or inulin on faecal bacterial concentration or proportion in infants up to three months of age (Moro *et al.*, 2002; Ben *et al.*, 2004; Bakker-Zierikzee *et al.*, 2005; Knol *et al.*, 2005; Fanaro *et al.*, 2005; Brunser *et al.*, 2006; Scholtens *et al.*, 2006a; Waligora-Dupriet *et al.*, 2007; Kim *et al.*, 2007; Scholtens *et al.*, 2008; Ben *et al.*, 2008; Costalos *et al.*, 2008; Magne *et al.*, 2008; Fanaro *et al.*, 2009; Nakamura *et al.*, 2009). Ben et al. 2004 and Ben et al. 2008 are the same study. Only one trial supplemented infant formula with inulin (average intake 1.5g/day) (Kim *et al.*, 2007). The data on measures of faecal bacterial concentration or proportion were insufficiently comparable to allow a meta-analysis. Two trials were subsequently identified in the update search (Veereman-Wauters *et al.*, 2011; Salvini *et al.*, 2011) (Colo-rectal health report p95; Update search table 218).

9.52. Six trials report no significant effect on the faecal concentration or proportion of *Bifidobacterium* spp. (Bakker-Zierikzee *et al.*, 2005; Brunser *et al.*, 2006; Scholtens *et al.*, 2006a; Waligora-Dupriet *et al.*, 2007; Costalos *et al.*, 2008; Nakamura *et al.*, 2009), while eight report that non-digestible oligosaccharide supplementation of infant formula, as compared with unsupplemented controls, increases the faecal concentration or proportion of *Bifidobacterium* spp. (Moro *et al.*, 2002; Ben *et al.*, 2004; Fanaro *et al.*, 2005; Knol *et al.*, 2005; Kim *et al.*, 2007; Scholtens *et al.*, 2008; Ben *et al.*, 2008; Magne *et al.*, 2008; Fanaro *et al.*, 2009).

9.53. Both the trials identified in the update search report supplementation of infant formula with non-digestible oligosaccharide (8g/L), as compared with unsupplemented controls, increases the faecal concentration or proportion of *Bifidobacterium* spp (Veereman-Wauters *et al.*, 2011; Salvini *et al.*, 2011).

Non-digestible oligosaccharides and infant faecal bacteria
• Effect • Moderate evidence • Supplementation of infant formula with non-digestible oligosaccharides increases faecal *Bifidobacterium* spp. concentration or proportion. Whether this effect is beneficial or of biological relevance is currently unclear

Infants more than three months of age

9.54. Four randomised controlled trials were identified that examined the effect of non-digestible oligosaccharide (mainly galacto-oligosaccharide) supplementation of either infant formula 2-8g/L, follow-on formula (5g/L) or weaning foods (2.5-4g/day) on faecal bacterial concentration or proportion in infants more than three months of age (Brunser *et al.*, 2006; Scholtens *et al.*, 2006a; Waligora-Dupriet *et al.*, 2007; Fanaro *et al.*, 2009). The data on measures of faecal bacterial concentration or proportion were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health report p 95).

9.55. Three trials report no significant effect of supplementation of infant formula or weaning foods with either non-digestible oligosaccharides on the faecal concentration or proportion of *Bifidobacterium* spp. (Brunser *et al.*, 2006; Scholtens *et al.*, 2006a; Waligora-Dupriet *et al.*, 2007), while one trial reports an increase in the faecal concentration of *Bifidobacterium* spp. (Fanaro *et al.*, 2009).

Non-digestible oligosaccharides and infant faecal bacteria
• No effect • Limited evidence

Faecal pH and short chain fatty acid content

9.56. Nine randomised controlled trials examining the effect of non-digestible oligosaccharides or inulin supplementation of infant formula (2-8g/L) or weaning foods (2.5-4g/day) on faecal pH or short chain fatty acid content in infants (Moro *et al.*, 2002; Ben *et al.*, 2004; Bakker-Zierikzee *et al.*, 2005; Fanaro *et al.*, 2005; Knol *et al.*, 2005; Scholtens *et al.*, 2006a; Kim *et al.*, 2007; Ben *et al.*, 2008; Scholtens *et al.*, 2008). Only one trial supplemented infant formula with inulin (average intake 1.5g/day)(Kim *et al.*, 2007). All trials were conducted in infants aged less than three months, except one which was conducted in infants aged four to six months and supplemented weaning foods (Scholtens *et al.*, 2006a).The data on measures of faecal pH and short chain fatty acid content were insufficiently comparable to allow a meta-analysis. No further studies were identified in the update search (Colo-rectal health report p95).

9.57. Of the five trials that investigated faecal short chain fatty acid content, four trials report supplementation of infant formula with non-digestible oligosaccharides or inulin, as compared with unsupplemented controls, increases the faecal concentration or proportion of faecal acetate (Ben *et al.*, 2004; Bakker-Zierikzee *et al.*, 2005; Knol *et al.*, 2005; Ben *et al.*, 2008),; one trial, in older infants (aged four to six months), reports no significant effect (Scholtens *et al.*, 2006a). All trials report supplementation of infant formula with non-digestible oligosaccharides or inulin, as compared with unsupplemented controls, lowers faecal pH, but one trial in older infants reports supplementation of weaning foods with non-digestible oligosaccharides has no effect on faecal pH (Scholtens *et al.*, 2006a).

Non-digestible oligosaccharides or inulin and infant faecal pH and short chain fatty acid content
• Effect in infants aged less than three months • Adequate evidence • Supplementation of infant formula with either non-digestible oligosaccharides or inulin increases faecal acetate content and lowers pH. Whether this effect is beneficial or of biological relevance is currently unclear

Outcomes where there is insufficient evidence

9.58. The tables below detail the exposures and outcomes where there are too few studies or trials that meet the inclusion criteria for the review (e.g. by study duration, publication date, health of subjects, design of intervention) to draw a conclusion. A full description of the studies can be found in the relevant systematic reviews.

Table 9.1 Insufficient evidence-randomised controlled trials

Risk factor/health outcome/measure	**Exposure**
Apolipoproteins	non-digestible oligosaccharides
LDL-cholesterol:HDL-cholesterol ratio Apolipoproteins	non-digestible oligosaccharides
Body weight change	non-digestible oligosaccharides
Eating motivation	resistant starch
Insulin resistance	non-digestible oligosaccharides
Glycaemia	non-digestible oligosaccharides
Insulin levels	non-digestible oligosaccharides and inulin
Faecal bacteria	non-digestible oligosaccharides
Faecal short chain fatty acid	RS_4
Faecal pH	RS_4
Mineral absorption	resistant starch
Constipation	non-digestible oligosaccharides and/or inulin
Diarrhoea	non-digestible oligosaccharides
Adenoma recurrence	resistant starch
Magnesium absorption	non-digestible oligosaccharides

Table 9.2 Inconsistent evidence-randomised controlled trials

Measure	**Exposure**
Eating motivation	non-digestible oligosaccharides

Summary and Conclusions

9.59. This assessment is based on 87 randomised controlled trials investigating the relationship between supplements of resistant starch, oligosaccharide or inulin and cardio-metabolic and colo-rectal outcomes. There were no prospective cohort studies investigating the relationship between oligosaccharides and the relevant health/disease outcomes. There was no evidence, which met the inclusion criteria, for this report identified on non-digestible oligosaccharides, inulin or resistant starch intake in relation to oral health outcomes. Twenty three randomised trials were conducted in infants, children and adolescents.

9.60. Nearly all the trials investigating non-digestible oligosaccharides supplement subjects with fructo-oligosaccharide, galacto-oligosaccharide or inulin. The data are too limited to draw conclusions on other non-digestible oligosaccharides that may be found naturally occurring in foods or produced using industrial processes. The non-digestible oligosaccharides and inulin used in trials often differ in their degree of polymerisation, but it is unclear from the data whether these differences have an impact on the outcomes investigated.

9.61. Randomised controlled trials that supplemented subjects with non-digestible oligosaccharides or inulin indicate a beneficial effect on fasting LDL-cholesterol concentration, but no effect is indicated on fasting total cholesterol, HDL-cholesterol or triacylglycerol concentration. Trials indicate no effect of non-digestible oligosaccharide supplementation on fasting glucose and fasting insulin concentration or energy intake. The trials do indicate non-digestible oligosaccharides cause an increase in faecal weight and an effect on faecal bacterial content, with an increase in the content of *Bifidobacterium* spp., but no effect on faecal short chain fatty acid content. Trials indicate that non-digestible oligosaccharides or inulin supplementation increases the fractional absorption of calcium in children and adolescents, but has no effect in adults.

9.62. Randomised controlled trials that supplemented subjects with resistant starch indicate no effect of resistant starch on energy intake. Resistant starch (types 1, 2 and 3 combined) is shown to cause an increase in faecal weight, have no effect on faecal bacterial content, but modify faecal short chain fatty acid content, with an increase in the concentration or proportion of butyrate and a lowering of pH. A limited number of trials indicate little effect of resistant starch type 4 on these faecal parameters.

9.63. The supplement doses of resistant starch, non-digestible oligosaccharide or inulin at which the effects described above become apparent is above levels currently consumed in typical diets in the UK, but recent and future developments of novel food ingredients may substantially increase the intake of these carbohydrates. The actual intake and extent of use of these novel foods is, as yet, unknown. The doses required for these effects may cause adverse gastrointestinal symptoms such as bloating, borborygmi and flatulence in the majority of the population (Coussement, 1999; Bishop *et al.*, 2009; Bonnema *et al.*, 2010). The effects on faecal parameters demonstrate the colonic fermentation of these carbohydrates, but whether they have an effect on health outcomes is unclear; equally the biological relevance of the effect of non-digestible oligosaccharide and inulin supplementation on calcium fractional absorption in children is unclear.

9.64. Randomised controlled trials conducted in infants aged less than three months tend to report that supplementation of infant formula with non-digestible oligosaccharides (mainly galacto-oligosaccharide, but also fructo-oligosaccharide) increase the faecal content of *Bifidobacterium* spp., lower faecal pH and increase faecal acetate concentration, as compared with unsupplemented infant formula. These effects are less consistent in trials conducted in infants aged more than three months. It is, however, unclear what the relationship is between these faecal parameters and relevant outcomes on health. The human intestinal microbiota is a complex ecosystem, consisting of several hundred different bacterial species and *Bifidobacterium* spp. is just one factor in the infant intestinal microbiota. In the first year of life, the infant intestinal tract progresses from sterility to extremely dense colonization, ending with a mixture of microbes that is broadly very similar to that found in the adult intestine. Being breast fed, mode of birth, perinatal antibiotics and country of birth are major factors influencing the composition of the infant intestinal microbiota (Fallani *et al.*, 2010). The composition of oligosaccharides in human milk contain is very complex, with great diversity of structure, and does not contain the non-milk oligosaccharides fructo-oligosaccharide and galacto-oligosaccharide (Boehm & Stahl, 2007).

Chapter 10. Glycaemic index and load

10.1. This assessment is based on 36 prospective cohort studies and 31 randomised controlled trials investigating the relationship between the glycaemic characteristics of carbohydrate intake and cardio-metabolic and colo-rectal health outcomes. There were no prospective cohort studies or randomised controlled trials conducted in children and adolescents. Links to the individual systematic reviews and update search are given in Annex 1 and the relevant page or table is given in the text below.

10.2. Evidence on health/disease outcomes have been discussed in detail only where there are sufficient data for a conclusion to be drawn, from studies meeting the pre-agreed inclusion criteria. Outcomes where there are too few such studies to reach a conclusion, are listed at the end of the chapter (see tables 10.1 and 10.2). Outcomes in which the evidence was considered too inconsistent to make a valid judgement are listed in table 10.3. Therefore, not all 36 cohort studies and 31 randomised controlled trials will be listed in this chapter.

10.3. Glycaemic index (GI) and glycaemic load (GL) are used as measures of the glycaemic characteristics of the diet (see paragraph 2.19 for a discussion of the limitations of these terms). The GI is a relative measure of the plasma glucose response induced by a specific ingredient, food or meal, as compared with the response induced by the same amount (usually 50g) of available carbohydrate from a reference source, such as pure glucose or an alternative source (such as white bread) which has been calibrated against it (Brouns *et al.*, 2005). The GL is the product of a specific food's GI and its carbohydrate content (Brouns *et al.*, 2005), therefore taking into account both the quality and quantity of carbohydrate consumed.

10.4. For the prospective cohort study meta-analyses the relative risks are presented for every two GI unit increment in GI and for every 20 GL unit increase, which are equivalent to approximately one standard deviation GI and GL. The approximate population mean GL is 120 units. These figures for GI and GL are based on published UK and European data from the EPIC study, in which the UK average for GI and GL (±sd) in women are 54.5 ± 4.8 and 122.0 ± 21.4, and in men are 56.0± 4.0 and 153.4 ± 25.9, respectively (van Bakel *et al.*, 2009).

10.5. In the cardio-metabolic health review, trials investigating GI and GL were combined into a single meta-analysis. Subsequently these trials have been separated into GI and GL studies and further meta-analyses have been performed. These additional analyses form part of annex 1 which has been included as online supplementary material. The pooled estimates for the trial data in this chapter are, therefore, taken from the additional meta-analyses document rather than the cardio-metabolic health review. The difference between these two types of trials is that the glycaemic index trials do not vary carbohydrate quantity, but change the quality to modify the GI. The GL trials reduce carbohydrate intake, resulting in a higher proportion of fat, often including saturated fatty acids, and/or protein intake, as well as changing the carbohydrate quality to modify the GI. Nearly all of the trials resulted in weight loss and consideration is given to whether an effect demonstrated on the studied parameters could be due to greater weight loss in one of the experimental groups.

Glycaemic Index

Total cardiovascular disease events

10.6. One cohort study was identified that presented evidence on GI and the incidence of total cardiovascular disease (Levitan *et al.*, 2007), which indicated GI was not associated with the incidence of all cardiovascular disease mortality. Five cohort studies were identified that presented evidence on GI and coronary and stroke events combined, all of which were included in a meta-analysis (Van Dam *et al.*, 2000; Oh *et al.*, 2005; Beulens *et al.*, 2007; Levitan *et al.*, 2007; Kaushik *et al.*, 2009). One cohort study was subsequently identified in the update search, which pooled four Danish cohort studies together (Grau *et al.*, 2011) (Cardio-metabolic review, cardiovascular disease chapter p 160-168; Update search table 36).

10.7. No significant association is indicated between GI and incidence of cardiovascular disease events (RR 1.02, 95% CI 0.99, 1.06, for each two GI unit increase), although the result is of borderline statistical significance (p=0.07). The pooled analysis paper (Grau *et al.*, 2011) indicates a protective association for a higher GI intake in men for both cardiovascular mortality and morbidity, but no significant association is indicated in women.

Glycaemic index (unit/day) and total cardiovascular disease events
• No association • Limited evidence

Coronary events

10.8. Four cohort studies were identified that presented evidence on GI and coronary event incidence, all of which were included in a meta-analysis (Van Dam *et al.*, 2000; Beulens *et al.*, 2007; Levitan *et al.*, 2007; Kaushik *et al.*, 2009). Five cohort studies were subsequently identified in the update search (Levitan *et al.*, 2010; Sieri *et al.*, 2010; Hardy *et al.*, 2010; Grau *et al.*, 2011; Burger *et al.*, 2011), one of which was a pooled analysis of four Danish cohort studies (Grau *et al.*, 2011) *(*Cardio-metabolic review, cardiovascular disease chapter p 162-169; Update search table 37).

10.9. No significant association is indicated between GI and incidence of coronary events (RR 1.02, 95% CI 0.96, 1.08, for each two GI unit increase; p=0.2); however, one study (Levitan *et al.*, 2007), had a strong influence on the outcome, but only presented results for myocardial infarction. Of the five studies identified in the update search, three indicate no significant association (Levitan *et al.*, 2010; Sieri *et al.*, 2010; Burger *et al.*, 2011). The pooled analysis of four cohort studies indicates a protective association for a higher GI intake in men, but not women (Grau *et al.*, 2011). The other study (Hardy *et al.*, 2010) indicates a greater risk of coronary events in African American women with a higher GI diet.

Glycaemic index (unit/day) and coronary events
• No association • Moderate evidence

Stroke

10.10. Three cohort studies were identified that presented evidence on GI and fatal and non-fatal stroke incidence, all of which were included in a meta-analysis (Oh *et al.*, 2005; Levitan *et al.*, 2007; Kaushik *et al.*, 2009). Two cohort studies were subsequently identified in the update search (Oba *et al.*, 2010b; Burger *et al.*, 2011) (Cardio-metabolic review, cardiovascular disease chapter p 164-170; Update search table 38).

10.11. No significant association is indicated between GI and incidence of stroke events (RR=1.01, 95% CI 0.98, 1.05, for each two GI unit increase). Of the two cohort studies identified in the update search, one indicates no significant association (Burger *et al.*, 2011), whereas the other (Oba *et al.*, 2010b) indicates a higher GI intake is associated with an higher incidence of mortality from total stroke in women, but not men.

Glycaemic index (unit/day) and stroke
• No association • Limited evidence

Blood pressure

10.12. Four randomised controlled trials were identified that presented evidence on GI in relation to systolic blood pressure and diastolic blood pressure, all of which were included in a meta-analysis (Bellisle *et al.*, 2007; Abete *et al.*, 2008; Jensen *et al.*, 2008; Philippou *et al.*, 2009b). Two trials were subsequently identified in the update search (Jebb *et al.*, 2010; Gogebakan *et al.*, 2011) *(*Cardio-metabolic review, blood pressure chapter p 121-126; Update search tables 47-48; Additional meta-analyses)

10.13. No significant effect is demonstrated for GI on systolic blood pressure (-0.54 mmHg 95% CI -4.08, 2.99; p=0.76) or diastolic blood pressure (0.60 mmHg 95% CI -2.06, 3.25; p=0.66). The two trials identified in the update search (Jebb *et al.*, 2010; Gogebakan *et al.*, 2011) report no statistically significant effect of GI on SBP or DBP.

Glycaemic index and systolic and diastolic blood pressure
• No effect • Moderate evidence

Fasting blood lipids

Fasting total-, LDL-, HDL-cholesterol and triacylglycerol

10.14. Ten randomised controlled trials were identified that presented evidence on GI in relation to fasting blood lipid concentration, all of which were included in a meta-analysis (Wolever & Mehling, 2003; Raatz *et al.*, 2005; McMillan-Price *et al.*, 2006; Bellisle *et al.*, 2007; Sichieri *et al.*, 2007; Abete *et al.*, 2008; Jensen *et al.*, 2008; Philippou *et al.*, 2008; Philippou *et al.*, 2009a; Philippou *et al.*, 2009b). Two trials were subsequently identified in the update search (Jebb *et al.*, 2010; Gogebakan *et al.*, 2011) (Cardio-metabolic review, hyperlipidaemias and blood lipids chapter, p448-476; Update search table p 99-102; Additional meta-analyses).

10.15. An effect of a higher GI diet is demonstrated resulting in less of a reduction in fasting total cholesterol concentration (0.20 mmol/L, 95% CI 0.08, 0.33; p=0.002) and fasting LDL-cholesterol concentration (0.21 mmol/L, 95% CI 0.10, 0.32; p<0.001) as compared with a lower GI diet. No significant effect of GI is demonstrated on fasting HDL-cholesterol concentration (0.01 mmol/L, 95% CI -0.05, 0.06; p=0.81) or fasting triacylglycerol concentration (-0.04 mmol/L, 95% CI -0.17, 0.10; p=0.59). The trials identified in the update search report no statistically significant effect of GI on fasting blood lipid concentrations.

10.16. A confounding factor in interpreting the effects demonstrated above is that body weight loss is generally less in the higher GI diet experimental groups (A meta-analysis of the trials above is almost significant, p=0.069). Fasting blood lipid concentrations are modified by body weight change (Poobalan *et al.*, 2004; Te Morenga *et al.*, 2010; Dow *et al.*, 2013), any differences, therefore, may not be attributable to the carbohydrate component of the dietary intervention. Weight loss, however, generally results in a reduction in fasting triacylglycerol concentrations as well as reducing fasting LDL-cholesterol (Dow *et al.*, 2013).

Glycaemic index and fasting total cholesterol concentration
• Effect • Moderate evidence • A higher GI diet may result in less of a reduction in fasting total cholesterol concentration as compared with a lower GI diet, but it is not possible to exclude confounding by other variables • The effect is biologically relevant

Glycaemic index and fasting LDL- cholesterol concentration
• Effect • Moderate evidence • A higher GI diet may result in less of a reduction in fasting LDL concentration as compared with a lower GI diet, but it is not possible to exclude confounding by other variables • The effect is biologically relevant

Glycaemic index and fasting HDL-cholesterol and triacylglycerol concentration
• No effect • Moderate evidence

Fasting total cholesterol:HDL-cholesterol ratio

10.17. Four randomised controlled trials were identified that presented evidence on GI in relation to the ratio of fasting total cholesterol:HDL-cholesterol ratio, all of which were included in a meta-analysis (McMillan-Price *et al.*, 2006; Bellisle *et al.*, 2007; Philippou *et al.*, 2008; Philippou *et al.*, 2009b) (Cardio-metabolic review, hyperlipidaemias and blood lipids review, p 480-482; Additional meta-analyses).

10.18. No significant effect of GI is demonstrated on fasting total cholesterol:HDL-cholesterol ratio (0.01, 95% CI -0.20, 0.21; p=0.95).

Glycaemic index and fasting total cholesterol:HDL-cholesterol ratio
• No effect • Moderate evidence

Fasting non-esterified fatty acids

10.19. Three randomised controlled trials were identified that presented evidence on GI in relation to fasting non-esterified fatty acid concentration, all of which were included in a meta-analysis (Wolever & Mehling, 2002; McMillan-Price *et al.*, 2006; Jensen *et al.*, 2008) (Cardio-metabolic review, hyperlipidaemias and blood lipids chapter, p 477-479; Additional meta-analyses).

10.20. No significant effect of GI is demonstrated on fasting non-esterified fatty acid concentration (0.01 mmol/L, 95% CI -0.05, 0.08; p=0.74).

Glycaemic index and fasting non-esterified fatty acid concentration
• No effect • Limited evidence

Markers of inflammation (C-reactive protein)

10.21. One randomised controlled trial was identified that presented evidence on GI in relation to C-reactive protein concentration (McMillan-Price *et al.*, 2006). Two trials were subsequently identified in the update search (Jebb *et al.*, 2010; Gogebakan *et al.*, 2011) (Cardio-metabolic review, markers of Inflammation chapter p 46-50; Update search table 113).

10.22. None of the trials report a significant effect of GI on C-reactive protein concentration.

Glycaemic index and C-reactive protein concentration
• No effect • Limited evidence

Eating motivation

10.23. Seven randomised controlled trials were identified that presented evidence on GI in relation to subjective reports of appetite in adults (Herrmann *et al.*, 2001; Sloth *et al.*, 2004; Alfenas & Mattes, 2005; Bellisle *et al.*, 2007; de Rougemont *et al.*, 2007; Sichieri *et al.*, 2007; Philippou *et al.*, 2009b) (Cardio-metabolic review, energy intake and eating motivation chapter, p182-186).

10.24. Due to variation between trials in design, the method of assessing eating motivation and the nature of each intervention, it is not possible to combine these trials using meta-analysis. One trial reports higher intensity of hunger and desire to eat in the higher GI diet group (Bellisle *et al.*, 2007), but all other trials report no significant effect of dietary GI on subjective ratings of appetite.

Glycaemic index and eating motivation
• No effect • Moderate evidence

Type 2 diabetes mellitus

10.25. Twelve cohort studies were identified that presented evidence on GI and type 2 diabetes mellitus incidence (Salmeron *et al.*, 1997a; Salmeron *et al.*, 1997b; Meyer *et al.*, 2000; Stevens *et al.*, 2002; Hodge *et al.*, 2004; Schulze *et al.*, 2004a; Schulz *et al.*, 2006; Villegas *et al.*, 2007; Krishnan *et al.*, 2007; Barclay *et al.*, 2007; Mosdol *et al.*, 2007; Sahyoun *et al.*, 2008). Four cohort studies were subsequently identified in the update search (Sluijs *et al.*, 2010; Mekary *et al.*, 2011; Simila *et al.*, 2011; Sakurai *et al.*, 2012) (Cardio-metabolic review, Diabetes chapter p145-154; Update search table 133). An updated meta-analysis was performed (Greenwood *et al.*, 2013) that included all identified studies and one additional study (van Woudenbergh *et al.*, 2011). The results of this further analysis are given below.

10.26. An association is indicated between a higher GI and a higher incidence of type 2 diabetes mellitus incidence (RR 1.03, 95% CI 1.01, 1.06, for each two GI unit increase; p=0.01).

Glycaemic index (unit/day) and type 2 diabetes mellitus
• Association • Adequate evidence • The direction of the association indicates consumption of a higher GI diet is detrimental to health, but it is not possible to exclude confounding by other variables • The association is biologically relevant

Fasting blood glucose

10.27. Ten randomised controlled trials were identified that presented evidence on GI in relation to fasting blood glucose concentration, all of which were included in a meta-analysis (Wolever & Mehling, 2003; Raatz *et al.*, 2005; McMillan-Price *et al.*, 2006; Bellisle *et al.*, 2007; Sichieri *et al.*, 2007; Jensen *et al.*, 2008; Philippou *et al.*, 2008; Abete *et al.*, 2008; Philippou *et al.*, 2009a; Philippou *et al.*, 2009b). Three trials were subsequently identified in the update search (Vrolix & Mensink, 2010; Gogebakan *et al.*, 2011; Krog-Mikkelsen *et al.*, 2011)(Cardio-metabolic review, diabetes and glycaemia chapter p 333; Update search table 139,178; additional meta-analyses).

10.28. No significant effect of GI is demonstrated on fasting blood glucose concentration (-0.01 mmol/L, 95% CI -0.09, 0.07 mmol/L; p=0.85). Of the trials identified in the update search, one reports no significant effect of GI on fasting blood glucose concentration (Gogebakan *et al.*, 2011), while the other (Krog-Mikkelsen *et al.*, 2011) reports fasting blood glucose concentration to be lower in the higher GI diet group.

Glycaemic index and fasting blood glucose concentration
• No effect • Moderate evidence

Fasting insulin

10.29. Nine randomised controlled trials were identified that presented evidence on GI in relation to fasting insulin concentration (Wolever & Mehling, 2003; Raatz *et al.*, 2005; McMillan-Price *et al.*, 2006; Bellisle *et al.*, 2007; Sichieri *et al.*, 2007; Abete *et al.*, 2008; Jensen *et al.*, 2008; Philippou *et al.*, 2009a; Philippou *et al.*, 2009b). Two trials were subsequently identified in the update search (Gogebakan *et al.*, 2011; Krog-Mikkelsen *et al.*, 2011)(Cardio-metabolic review, diabetes chapter p 340-344; Update search table 142,180).

10.30. Due to variation between trials in the method used to measure fasting insulin concentration, it is not possible to combine these trials using meta-analysis. The trials provide no consistent evidence of an effect of GI on fasting insulin concentration. Of the trials identified in the update search both report no significant effect of GI on fasting insulin concentration.

Glycaemic index and fasting insulin concentration
• No effect • Moderate evidence

Insulin sensitivity/resistance

10.31. Seven randomised controlled trials were identified that presented evidence on GI in relation to insulin sensitivity/resistance (Sloth *et al.*, 2004; Raatz *et al.*, 2005; McMillan-Price *et al.*, 2006; Bellisle *et al.*, 2007; Sichieri *et al.*, 2007; Abete *et al.*, 2008; Philippou *et al.*, 2009b). Two trials were subsequently identified in the update search (Jebb *et al.*, 2010; Krog-Mikkelsen *et al.*, 2011) Cardio-metabolic review, diabetes chapter p 345-350; Update search tables 141,181)

10.32. Due to variation between trials in the method used to measure insulin sensitivity/resistance, it is not possible to combine these trials using meta-analysis. All the trials provide no evidence of an effect of GI on insulin sensitivity/resistance. Of the trials identified in the update search both report no significant effect of GI on insulin sensitivity/resistance.

Glycaemic index and insulin sensitivity/resistance
• No effect • Moderate evidence

Colo-rectal cancer

10.33. Seven cohort studies were identified that presented evidence on GI and colo-rectal cancer incidence, all of which were included in the meta-analysis (Higginbotham *et al.*, 2004; Michaud *et al.*, 2005; McCarl *et al.*, 2006; Larsson *et al.*, 2007; Strayer *et al.*, 2007; Weijenberg *et al.*, 2008; George *et al.*, 2009). One cohort study was identified in the update search (Li *et al.*, 2011) (Colo-rectal health review p 186-197; Update search table 221).

10.34. No significant association is indicated between GI and incidence of colo-rectal cancer (RR 1.01, 95% CI 0.99, 1.03, for each two GI unit increase). The study identified in the update search (Li *et al.*, 2011) indicates no significant association between GI and risk of colo-rectal cancer in women.

Glycaemic index (unit/day) and colo-rectal cancer
• No association • Adequate evidence

Glycaemic load

Total cardiovascular disease events

10.35. One cohort study was identified that presented evidence on GL and the incidence of total cardiovascular disease (Levitan *et al.*, 2007), which indicated GL was not associated with the incidence of all cardiovascular disease mortality. Three cohort studies were identified that presented evidence on GL and the incidence of coronary and strokes events combined, all of which were included in a meta-analysis (Oh *et al.*, 2005; Levitan *et al.*, 2007; Beulens *et al.*, 2007). One cohort study was subsequently identified in the update search, which pooled four Danish cohort studies together (Grau *et al.*, 2011) (Cardio-metabolic review, cardiovascular disease chapter p 171 and 177; Update search table 39).

10.36. An association is indicated between a higher GL and a higher incidence of coronary events and strokes combined (RR 1.06, 95% CI 1.01, 1.12, for each 20 GL unit increase; p=0.03). The pooled analysis study identified in the update search indicated no significant association between GL and cardiovascular disease in men, but indicated an association with higher incidence of cardiovascular disease morbidity, but not mortality, in women (Grau *et al.*, 2011).

Glycaemic load (unit/day) and total cardiovascular disease events
• Association • Limited evidence • The direction of the association indicates consumption of a higher GL diet is detrimental to health, but it is not possible to exclude confounding by other variables • The association is biologically relevant

Blood pressure

10.37. Three randomised controlled trials were identified that presented evidence on GL in relation to systolic blood pressure and diastolic blood pressure, all of which were included in a meta-analysis (Pereira *et al.*, 2004; Ebbeling *et al.*, 2005; Maki *et al.*, 2007b). All trials were weight loss trials. No trials were identified in the update search *(*Cardio-metabolic review, blood pressure chapter p 121-126; Additional meta-analyses).

10.38. No significant effect is demonstrated for GL on systolic blood pressure (1.92 mmHg 95% CI-0.76, 4.60; p=0.16), but an effect is demonstrated for a higher GL diet resulting in less of a reduction in diastolic blood pressure as compared with a lower GL diet (3.10 mmHg 95% CI 0.25, 5.95; p=0.03).

Glycaemic load and systolic blood pressure
• No effect • Limited evidence

Glycaemic load and diastolic blood pressure
• Effect • Limited evidence • A higher GL diet may result in less of a reduction in diastolic blood pressure as compared with a lower GL diet, but it is not possible to exclude confounding by other dietary variables • The effect is biologically relevant

Fasting blood lipids

Fasting total-, LDL-, HDL-cholesterol and triacylglycerol

10.39. Five randomised controlled trials were identified that presented evidence on GL in relation to fasting blood lipid concentration and all of these trials were included in a meta-analysis (Pereira *et al.*, 2004; Ebbeling *et al.*, 2005; Das *et al.*, 2007; Ebbeling *et al.*, 2007; Maki *et al.*, 2007b). All trials were weight loss trials. One trial was subsequently identified in the update search (Vrolix & Mensink, 2010) (Cardio-metabolic review, hyperlipidaemias and blood lipids chapter, p448-476; Update search table 103-106; Additional meta-analyses). All trials were weight loss trials except the trial identified in the update search.

10.40. No significant effect of GL is demonstrated on fasting total cholesterol concentration (-0.01 mmol/L, 95% CI -0.14, 0.13; p=0.94) and fasting LDL-cholesterol concentration (0.07 mmol/L, 95% CI -0.05, 0.19; p=0.29). The heterogeneity test for fasting HDL-cholesterol concentration is over 75%, so the results are not reported, but the trials present no evidence for an effect. An effect of a higher GL diet is demonstrated resulting in less of a reduction in fasting triacylglycerol concentration (0.13 mmol/L, 95% CI 0.03, 0.24; p=0.012) as compared with the lower GL diet. The trial identified in the update search reports no significant effect of GL on fasting blood lipid concentrations.

Glycaemic load and fasting total cholesterol, LDL-cholesterol and HDL-cholesterol concentration
• No effect • Limited evidence

Glycaemic load and fasting triacylglycerol concentration
• Effect • Limited evidence • A higher GL diet may result in less of a reduction in fasting triacylglycerol concentration than a lower GL diet, but it is not possible to exclude confounding by other dietary variables • The effect is borderline biologically relevant

Markers of inflammation (C-reactive protein)

10.41. Two randomised controlled trials were identified that presented evidence on GL in relation to C-reactive protein concentration (Pereira *et al.*, 2004; Pittas *et al.*, 2006). One trial was subsequently identified in the update search (Vrolix & Mensink, 2010) (Cardio-metabolic review, markers of Inflammation chapter p 46-50; Update search table p 114).

10.42. One trial reports higher C-reactive protein concentration in the higher GL diet group (Pittas *et al.*, 2006), but the other two trials report no significant effect (Pereira *et al.*, 2004; Vrolix & Mensink, 2010).

Glycaemic load and C-reactive protein concentration
• No effect • Limited evidence

Body weight change

10.43. Three randomised controlled trials were identified that presented evidence on GL in relation to body weight change (Ebbeling *et al.*, 2005; Das *et al.*, 2007; Ebbeling *et al.*, 2007). One trial could not be included in a meta-analysis as it did not report the necessary data, leaving an insufficient number or trials to enable a meta-analysis to be performed (Cardio-metabolic review, obesity chapter p55-57).

10.44. None of the trials report an effect of GL on body weight change.

Glycaemic load and body weight change
• No effect • Limited evidence

Type 2 diabetes mellitus

10.45. Eleven cohort studies were identified that presented evidence on GL and type 2 diabetes mellitus incidence, all of which were included in the meta-analysis (Salmeron *et al.*, 1997a; Salmeron *et al.*, 1997b; Meyer *et al.*, 2000; Stevens *et al.*, 2002; Hodge *et al.*, 2004; Schulze *et al.*, 2004a; Schulz *et al.*, 2006; Villegas *et al.*, 2007; Krishnan *et al.*, 2007; Mosdol *et al.*, 2007; Sahyoun *et al.*, 2008). Six cohort studies were subsequently identified in the update search (Hopping *et al.*, 2010; Sluijs *et al.*, 2010; Mekary *et al.*, 2011; Yu *et al.*, 2011; Simila *et al.*, 2011; Sakurai *et al.*, 2012) (Cardio-metabolic review, Diabetes chapter p 155-164; Update search table 134). A further meta-analysis was performed which included the studies found in the update search (Greenwood *et al.*, 2013) as well as later study (van Woudenbergh *et al.*, 2011). The results from the later meta-analysis were used.

10.46. An association is indicated between a higher GL and a higher incidence of type 2 diabetes mellitus (RR 1.03, 95% CI 1.00, 1.05, for each 20 GL unit increase; p=0.02).

Glycaemic load (unit/day) and type 2 diabetes mellitus
• Association • Adequate evidence • The direction of the association indicates higher GL is detrimental to health, but it is not possible to exclude confounding by other variables • The association is biologically relevant

Fasting blood glucose

10.47. Three randomised controlled trials were identified that presented evidence on GL in relation to fasting blood glucose concentration, all of which were included in a meta-analysis (Ebbeling *et al.*, 2007; Das *et al.*, 2007; Maki *et al.*, 2007b). One trial was subsequently identified in the update search (Vrolix & Mensink, 2010) (Cardio-metabolic review, diabetes chapter p 333; Update search table 179; Additional meta-analyses).

10.48. No significant effect of GL is demonstrated on fasting blood glucose concentration (-0.13 mmol/L, 95% CI -0.40, 0.13 mmol/L; p=0.33). The trial identified in the update search report reports no significant effect of GL on fasting glucose concentration.

Glycaemic load and fasting blood glucose concentration
• No effect • Limited evidence

Fasting insulin

10.49. Three randomised controlled trials were identified that presented evidence on GL in relation to fasting insulin concentration (Ebbeling *et al.*, 2007; Das *et al.*, 2007; Maki *et al.*, 2007b). One trial was subsequently identified in the update search (Vrolix & Mensink, 2010) (Cardio-metabolic review, diabetes chapter p 340-344; Update search table 182).

10.50. Due to variation between trials in the method used to measure fasting insulin concentration, it is not possible to combine these trials using meta-analysis. The trials provide no evidence of an effect of GL on fasting insulin concentration. The trial identified in the update search also reports no significant effect of GL on fasting insulin concentration.

Glycaemic load and fasting insulin concentration
• No effect • Limited evidence

Insulin sensitivity/ resistance

10.51. Four randomised controlled trials were identified that presented evidence on GL in relation to insulin sensitivity/resistance (Pereira *et al.*, 2004; Ebbeling *et al.*, 2005; Pittas *et al.*, 2006; Maki *et al.*, 2007b). One trial was subsequently identified in the update search (Vrolix & Mensink, 2010) (Cardio-metabolic review, diabetes chapter p 345-350; Update search table 182).

10.52. Due to variation between trials in the method used to measure insulin sensitivity/resistance, it is not possible to combine these trials using meta-analysis. One trial reports an effect of GL on insulin sensitivity/resistance (Pereira *et al.*, 2004), but the other trials, including the trial identified in the update search, provide no evidence of an effect of GL on insulin sensitivity/resistance.

Glycaemic load and insulin sensitivity/ resistance
• No effect • Limited evidence

Colo-rectal cancer

10.53. Nine cohort studies investigating GL were identified which provided data on diets with a higher or lower GL and their relationship to the incidence of colo-rectal cancer, all of which were included in the meta-analysis (Terry *et al.*, 2003; Higginbotham *et al.*, 2004; Michaud *et al.*, 2005; McCarl *et al.*, 2006; Larsson *et al.*, 2007; Strayer *et al.*, 2007; Howarth *et al.*, 2008; Weijenberg *et al.*, 2008; George *et al.*, 2009). One cohort study was identified in the update search (Li *et al.*, 2011) (see Colo-rectal health review p 186-197; Update search table 221).

10.54. No significant association is indicated between GL and incidence of colo-rectal cancer (RR 1.00, 95% CI 0.97, 1.03, for each 20 GL unit increase). The study identified in the update search (Li *et al.*, 2011) indicates no significant association between GI and risk of colo-rectal cancer in women.

Glycaemic load (unit/day) and colo-rectal cancer
• No association • Adequate evidence

Outcomes where there is insufficient evidence

10.55. The tables below detail the exposures and outcomes where there are too few studies or trials that meet the inclusion criteria for the review (e.g. by study duration, publication date, health of subjects, design of intervention) to draw a conclusion. A full description of the studies can be found in the relevant systematic reviews.

Table 10.1 Insufficient evidence-cohort studies

Risk factor/health outcome/measure	**Exposure**
Fasting blood lipids Body weight change BMI Body fatness Glycaemia and glucose tolerance Glycosylated blood proteins	Glycaemic Index
Stroke events Fasting blood lipids Body weight change and BMI Body fatness and fat distribution Glycaemia and glucose tolerance Glycosylated blood proteins	Glycaemic Load

Table 10.2 Insufficient evidence- randomised controlled trials

Risk factor/health outcome/measure	**Exposure**
Pulse wave velocity PAI-1 Body weight change and BMI Body fat mass and distribution Weight regain Glycosylated blood proteins	Glycaemic index
Blood pressure Fasting blood lipids PAI-1 Body weight change and BMI Body fat mass and distribution Glycosylated blood proteins Eating motivation	Glycaemic load

Table 10.3 Inconsistent evidence

Risk factor/health outcome/measure	Exposure
Coronary events	Glycaemic load
Energy intake	Glycaemic index/load

Summary and conclusions

10.56. This assessment is based on 36 prospective cohort studies and 31 randomised controlled trials investigating the glycaemic characteristics in the diet and the health/disease outcomes included in this review. No evidence was identified which met the inclusion criteria for this review on dietary glycaemic index and load in relation to oral health. There were no prospective cohort studies or randomised controlled trials conducted in children and adolescents. With observational studies there is substantial potential for biases and the possibility of confounding by an extraneous variable that correlates with both the dependent variable and the independent variable (residual confounding) and any associations must be interpreted with caution.

10.57. The evidence provided by prospective cohort studies indicates that a diet higher in glycaemic index or load is associated with a greater risk of type 2 diabetes mellitus. There is no evidence from prospective cohort studies to suggest an association between glycaemic index and cardiovascular disease or coronary heart disease; glycaemic load is associated with a greater risk of cardiovascular diseases, but the evidence is limited due to the small number of studies. There is no evidence for an association between glycaemic index or load and colo-rectal cancer incidence.

10.58. The evidence provided by randomised controlled trials is limited due to the design of the trials mainly being weight loss trials. These trials indicate a higher glycaemic index diet results in less reduction in fasting blood total cholesterol and LDL-cholesterol concentrations as compared with a lower glycaemic index diet, but this could also be due to less weight loss in the higher glycaemic index diet group as compared with the lower glycaemic index diet group. A higher glycaemic load diet results in less reduction in diastolic blood pressure and fasting triacylglycerol concentration as compared with a lower glycaemic load diet, but this is based only on three trials and it is not possible to exclude confounding by other dietary variables, although difference in weight loss between experimental groups appears not to be a factor in these trials. The interventions in trials investigating glycaemic index and load in relation to cardio-metabolic risk factors include other factors that mean a cause effect relationship cannot be established.

10.59. Glycaemic index is a measure of the blood glucose response to a portion of a food containing a fixed quantity of available carbohydrates. There are, however, many influences on the glycaemic index and load over and above content and quality of carbohydrates in a food, both physiological and dietary, not all of which are understood. The effects and associations on health described in the meta-analyses cannot be specifically attributed to the GI or GL and may be a result of other associated factors such as dietary fibre, protein or fat content, as well as food processing and storage (Brouns *et al.*, 2005; Venn & Green, 2007).

10.60. Higher and lower GI/GL diets will, in most cases, differ in many ways other than the carbohydrate fraction. As such, the majority of the literature on GI and GL does not allow for certainty that the carbohydrate content of an exposure is the sole influence on the GI or GL of a diet. It also limits the confidence in assigning cause-effect relationships for outcomes based on variation in diet GI or GL.

Chapter 11. Dietary reference values

11.1. The proposed carbohydrate dietary reference values are for adults and children aged 2.0 years or more and have been made in the context of an energy intake that is appropriate to maintain a healthy weight (SACN, 2011).

Total Carbohydrate

11.2. The carbohydrate dietary reference value was previously set to facilitate the recommended reduction in the average contribution of total fat to dietary energy in the population to about 35% with protein intake comprising 15% of dietary energy (COMA, 1994). At that time it was recommended that 'complex carbohydrates' and sugars in fruits and vegetables should replace some dietary fat, to achieve approximately 50% the proportion of dietary energy derived from total carbohydrates (COMA, 1994).

11.3. Concern has been raised that high intakes of total carbohydrate may be deleterious to health, but this report concludes that total carbohydrate intakes, at levels generally recommended in the UK diet, are not associated with the health outcomes examined. Specifically, total carbohydrate intake shows no association with the incidence of cardiovascular disease endpoints, type 2 diabetes mellitus, glycaemia or colo-rectal cancer. In children and adolescents, limited evidence indicates that there is no association between total carbohydrate intake and body mass index or body fatness. Overall, there is no evidence to warrant a change to the total carbohydrate dietary reference value previously set by COMA of 50% dietary energy.

11.4. It is recommended that:

- The dietary reference value for total carbohydrate should be maintained at a population average of approximately 50% of dietary energy.

Sugars

11.5. The previous dietary reference value referred to non-milk extrinsic sugars, and recommended that the population average should be no more than 10% of total dietary energy (COMA, 1991). This dietary reference value was based on the observation that dental caries was rare in populations whose intakes were estimated to be approximately 10% of dietary energy (based on national sugar supply data from food balance sheets and household consumption data). It was also advised that intakes in excess of 30% dietary energy should be avoided as it may lead to an increase in plasma concentrations of glucose, insulin and lipids.

11.6. There are several definitions of sugars in current use internationally. The present UK term of 'non-milk extrinsic sugars', which is used in national surveys, encompasses sugars in unsweetened fruit juice and honey, as well as sugars that are added to food and drink. It also includes 50% of the weight of sugars found in dried, stewed or canned fruit. The 50% figure is arbitrary and is used to account for the partial breakdown of the cellular structure during processing (Buss *et al.*, 1994; Bates *et al.*, 2012), but proves problematic

when trying to estimate the sugars composition of certain foods. The term 'added sugars' as used by the European Food Safety Authority and US Institute of Medicine includes sugars that are added to foods either during manufacture or by the consumer, but does not capture the sugars present in unsweetened fruit juice or honey. The definition of 'free sugars', as used by the World Health Organization, captures all sugars added to foods and those present in fruit juice and honey but does not apply the figure of 50% of sugars in dried and cooked fruit. Therefore, the definition of free sugars is similar to non-milk extrinsic sugars but overcomes the problem of trying to account for the additional sugars from processed fruit. In addition, the term non-milk extrinsic sugars is used exclusively by the UK and does not lend itself to being easily understood compared to the term free sugars or added sugars.

11.7. In light of the issues above, it is proposed that the UK adopts the definition of 'free sugars' in place of 'non-milk extrinsic sugars'. Free sugars are defined as all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and unsweetened fruit juices. Under this definition, lactose naturally present in milk and milk products and sugars contained within the cellular structure of foods would be excluded. In this report the term "sugars" is used because this enables other sugars (e.g. glucose, fructose, lactose) to be included in addition sucrose. This is important because it will ensure that any replacement of sucrose by high fructose corn syrups (isoglucose) in the production of food and drinks is captured.

11.8. Since the dietary reference values were last considered, the quality of the evidence indicating that a high intake of free sugars is detrimental to several health outcomes has strengthened. Higher consumption of sugars, and sugars- containing foods and beverages is associated with greater risk of dental caries. Randomised controlled trials conducted in adults indicate that increasing sugars intake as part of an *ad libitum* diet, either through the substitution of other macronutrient components or by replacement of non-caloric sweeteners by sugars, leads to an increase in energy intake. Consumption of sugars-sweetened beverages is associated with a greater risk of type 2 diabetes mellitus. Randomised controlled trials conducted in children and adolescents indicate that consumption of sugars-sweetened beverages, as compared with non-calorically sweetened beverages, resulted in weight gain and an increase in body mass index. This finding suggests that there was insufficient voluntary reduction in intake of other foods or drinks, thus implying incomplete appetite compensation for the additional energy supplied by the sugars, and thus leading to weight gain.

11.9. The data on sugars intake, taken from randomised controlled trials in adults, of both the higher and lower sugars intervention groups were plotted against daily energy intake (see figure 1). The graph included all the studies that had been combined in the meta-analysis from the systematic review except for Reid et al. (2007) which only provided data on carbohydrate intake and not sugars consumption (Drummond & Kirk, 1998; Saris *et al.*, 2000; Poppitt *et al.*, 2002; Raben *et al.*, 2002; Brynes *et al.*, 2003; Drummond *et al.*, 2003; Reid *et al.*, 2007) (see chapter 6, paragraph 6.18). The consumption of sugars ranged from 4% to 32% of energy intake. The data show a clear dose response relationship such that total energy intake increases as the percentage of energy from sugars increases. Although there is limited evidence relating to sugars intakes below 10% of energy intake, there is little reason to doubt that the relationship continues to be approximately linear at lower percentages of energy from sugars. Similarly for the link

between sugars intake and oral health, specifically caries, there appears to be continuous benefit as percentage energy from sugars decreases. Therefore it appears that in relation to both improving oral health and reducing the risk of weight gain, sugars should provide no more than 10% of dietary energy.

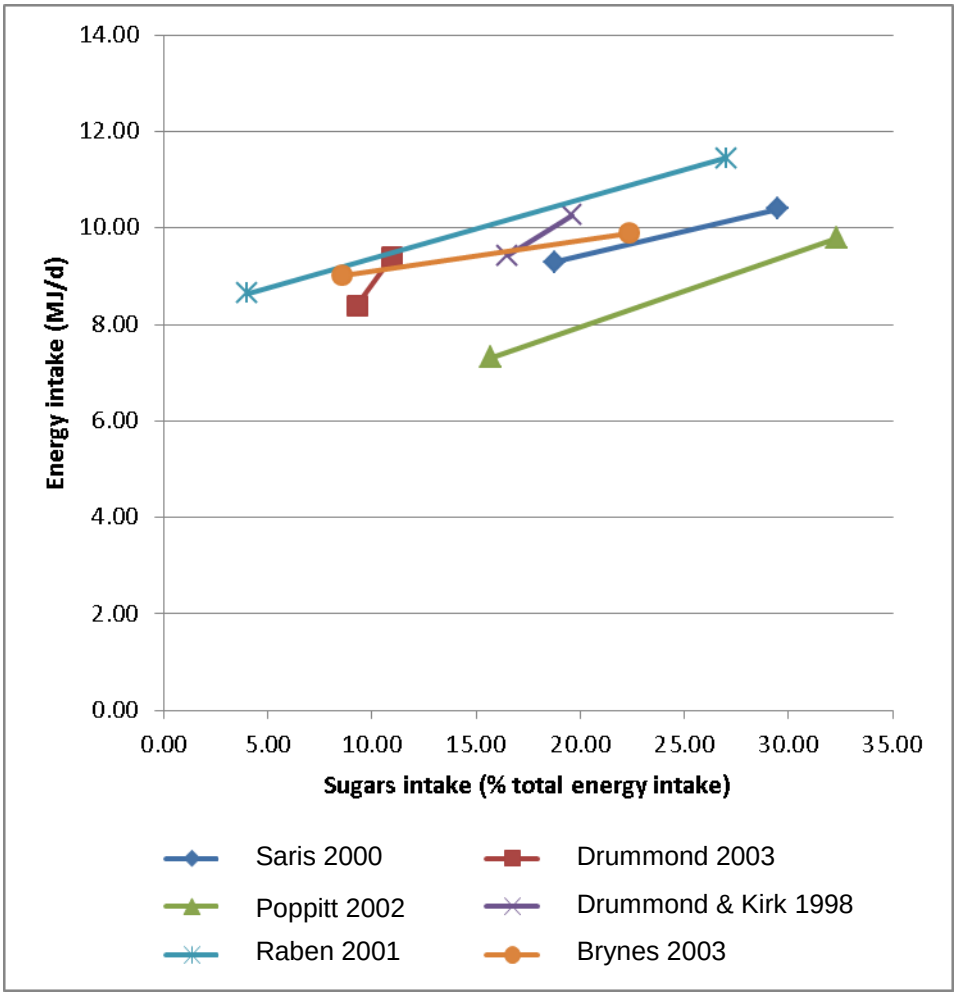
11.10. In order for an individual to achieve a recommendation to consume less than 10% of dietary energy from free sugars, the population average needs to be less than this figure. To inform what this value should be, advice from Calorie Reduction Expert Group was considered. It was estimated that a 418kJ/person/day (100kcal/person/day) reduction in energy intake of the population would address energy imbalance and lead to a moderate degree of weight loss in some individuals (Calorie Reduction Expert Group, 2011). The amount of energy delivered for each percentage of sugars was calculated from the randomised controlled trials listed in paragraph 11.8; it was estimated that there was a 117kJ (28kcal) lower energy intake for every 1% reduction in sugars in the diet. This figure assumes no dietary compensation for the additional energy supplied in the higher sugars diets, which may not reflect true dietary behaviour and, therefore, the estimate should be treated with some caution. Nonetheless these calculations provide a useful guide on which to make an informed judgement on the evidence. To achieve an average reduction in energy intakes by 418kJ (100kcal/person/day), intake of free sugars would have to be reduced by approximately 4% of energy. From figure 1, there appears to be even greater reduction in energy intake when sugars are consumed at 5% of energy, however there are few data at this level of intake to draw firm conclusions. To reflect the uncertainty around the figure whilst also acknowledging that there is evidence showing that average intakes of free sugars should be below 10% of dietary energy, it was considered that the population average free sugars intake should be around 5% of dietary energy.

11.11. Given the high rates of obesity in the UK, reducing the population's energy intakes is likely to be beneficial to health. Obesity occurs when energy intake from food and drink consumption is greater than energy expended over a prolonged period. In the UK, the prevalence of obesity increased sharply during the 1990s and early 2000s. For example in England, the proportion of adults who were categorised as obese (BMI 30kg/m^2 or over) increased from 13.2% of men in 1993 to 24.4% in 2012 and from 16.4% of women in 1993 to 25.1% in 2012 (Health and Social Care Information Centre, 2013a). In addition in England, 9.7% of boys and 8.8% of girls (all children 9.3%) aged 4.0-5.0 years and 20.4% of boys and 17.4% of girls (all children 18.9%) aged 10.0-11.0 years were also classified as obese, according to the British 1990 population monitoring definition of obesity (≥95th centile) (Health and Social Care Information Centre, 2013b). Higher rates of obesity were observed in children living in more deprived areas compared to those living in the least deprived. Obesity is associated with greater risks of type 2 diabetes mellitus, hypertension, coronary artery disease and hyperlipidaemia as well as some types of cancer and other diseases (Foresight, 2007) . Since free sugars intake is a dietary factor shown to increase energy intake, decreasing the population intake of free sugars is a step that could be taken to help reduce the current UK over-consumption of energy.

11.12. Reducing consumption of free sugars would also help to lower the risk of dental caries which continues to be a widespread problem in the UK. From the Adult Dental Health Survey, it was found that 31% of adults in England, Wales and Northern Ireland experienced dental caries in either the crown or root of the tooth (Steele & O' Sullivan, 2011). In 2012 almost a third (27.9%) of 5 year olds in England had tooth decay (Public Health England, 2013). There are also stark inequalities across the regions for example, 21.2% of five-year olds had tooth decay in South East England compared to 34.8% in the North West of England with even greater inequalities within local authority areas. In the Children's Dental Health Survey, 57% of eight year olds had some kind of dental caries in their primary dentition. In terms of permanent dentition, 14%, 34% and 49% of 8, 12 and 15 year olds had obvious dental caries (Lader *et al.*, 2003). In 2013, the prevalence of diabetes mellitus was 6% of the adult population. It is estimated that 90% of these cases are type 2 diabetes mellitus (Diabetes UK, 2014). Given that the evidence in this report found that sugars sweetened beverages are associated with a higher risk of type 2 diabetes mellitus and that obesity is also linked with this outcome, lowering sugars intake may confer additional health benefits.

Figure 1: The relationship in randomised controlled trials between daily energy intake and the percentage of total energy intake consumed as sugars.

To convert from MJ to Kcal, multiply value by 239



11.13. It is recommended that:

- The definition for 'free sugars' be adopted in the UK and that this comprises all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and unsweetened fruit juices, excluding lactose when naturally present in milk and milk products.
- The dietary reference value for free sugars should be set at a population average of around 5% of dietary energy for age-groups from 2.0 years upwards. This is based on the need to limit free sugars to no more than 10% of total energy intake at an individual level, which is likely to lead to a population average free sugars intake of around 5% of total energy.
- The consumption of sugars-sweetened beverages should be minimised, in both children and adults.

Dietary fibre

11.14. In 1991 COMA set a dietary reference value for fibre, defined as non-starch polysaccharides, that recommended the population average intake should be 18g/ day, with a minimum of 12 g/day and a maximum of 24 g/day for individuals (COMA, 1991). Non-starch polysaccharides are determined using the Englyst method of analysis (Englyst et al., 1994). Guidance on high intakes stated there was a lack of evidence of benefit associated with intakes of non-starch polysaccharides in excess of 32g/day. It was noted that such intakes were not seen in self-selected diets and potentially undesirable effects could not be excluded. The dietary reference value was based on the effect of non-starch polysaccharide on increasing faecal weight and the observation that a lower incidence of bowel disease was observed in populations with higher faecal weights.

11.15. For this report, it was agreed that components would be considered in the context of SACN's position statement on dietary fibre. This states that for extracted natural carbohydrate components or synthetic carbohydrate products to be defined as dietary fibre, beneficial physiological effects, similar to those demonstrated for the naturally integrated dietary fibre component of foods, must be demonstrated by accepted scientific evidence. Such effects include increasing stool bulk, decreasing intestinal transit time, decreasing post-prandial glycaemia or the lowering of total cholesterol and LDL-cholesterol concentration (SACN, 2008). However, evidence limited only to effects on gut fermentation or the nature of the microbiota is not sufficient to satisfy this definition. In this report, there is evidence to show that non-digestible oligosaccharides and resistant starch increase faecal mass. On this basis, SACN consider that these two components can be considered as dietary fibre. With the inclusion of resistant starch and non-digestible oligosaccharides, this broadens the definition of fibre beyond non-starch polysaccharides. Therefore it is recommended that dietary fibre should be defined as all carbohydrates that are naturally integrated components of foods and that are neither digested nor absorbed in the small intestine and has a degree of polymerisation of three or more monomeric units, plus lignin.

11.16. The majority of the evidence on fibre and health considered in this report uses the Association of Official Analytical Chemists (AOAC) definition of fibre therefore broadening the definition allows the UK to be aligned with the research base and permits intakes to be directly compared with different countries.

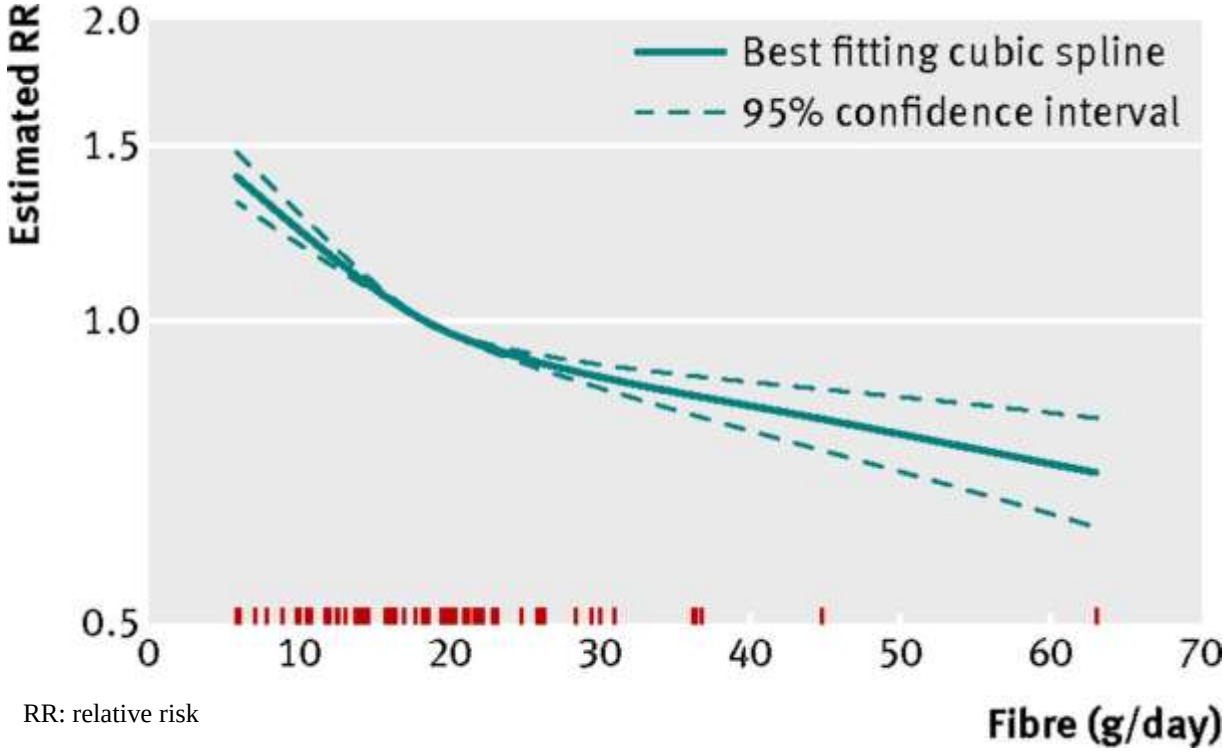
11.17. The broader definition of fibre is measured by the AOAC method and is colloquially known as AOAC fibre. There are different AOAC methods available; the older methods (AOAC 985.29 and 991.43) capture non-starch polysaccharides, some resistant starches, lignin and some inulin, but they do not measure most non-digestible oligosaccharides (Prosky et al., 1988; Lee et al., 1992). It is these methods which have been used by the studies included in this report. A newer method of analysis is now available (AOAC 2009.01) which is able to determine all the components included in the proposed definition of fibre above (McCleary et al., 2010; McCleary et al., 2012).

11.18. Since the dietary reference values were last considered, the quality of the evidence indicating that a diet rich in dietary fibre (mostly defined as AOAC fibre) reduces the risk of type 2 diabetes mellitus, cardiovascular disease and colo-rectal cancer has strengthened considerably. Despite inconsistency between studies in the definitions of whole grains, greater consumption of whole grains is associated with a lower incidence of cardiovascular disease, hypertension, type 2 diabetes mellitus and colon cancer.

11.19. Cardiovascular disease, type 2 diabetes mellitus and colorectal cancer are of great public health significance in the UK. It is estimated that in 2010, 10.1% of women and 11.7% of men were suffering from cardiovascular disease (coronary heart disease and stroke combined)(British Heart Foundation, 2012) . The prevalence of type 2 diabetes is given in paragraph 11.11 There were approximately 41,600 new cases of colorectal cancer diagnosed in the UK in 2011 and it is the third most common cancer in both men and women (Cancer Research UK, 2014a). For deriving the dietary reference value for dietary fibre, non-linear dose response plots included in meta-analyses of prospective cohort studies investigating cardiovascular disease, coronary heart disease, stroke, type 2 diabetes mellitus and colorectal cancer were considered (Figures 2-6) (Aune *et al.*, 2011; Threapleton *et al.*, 2013b; Threapleton *et al.*, 2013c; Threapleton *et al.*, 2013d). The plots indicate that a non-linear dose-response relationship exists such that as dietary fibre intake increases the risk of developing these diseases decreases. From these data, it is apparent that intakes of 30g/day and above (as defined using the AOAC methods 985.29 and 991.43) are associated with the greatest health benefits in reducing the incidence of cardiovascular diseases, type 2 diabetes mellitus and colo-rectal cancer. Therefore, a dietary reference value for fibre of 30g per day is proposed as it is an amount which was shown in the evidence reviewed to be associated with reduced health risk.

Figure 2: Risk of CVD in cohort studies with increasing intake of total fibre

Threapleton et al. 2013c BMJ 347, f6879 (the lines at the bottom of the plot represents where a risk estimate has been provided for a given intake within each study)



RR: relative risk

Figure 3: Risk of CHD in cohort studies with increasing intake of total fibre

Threapleton et al. 2013c. BMJ 347, f6879 (the lines at the bottom of the plot represents where a risk estimate has been provided for a given intake within each study)

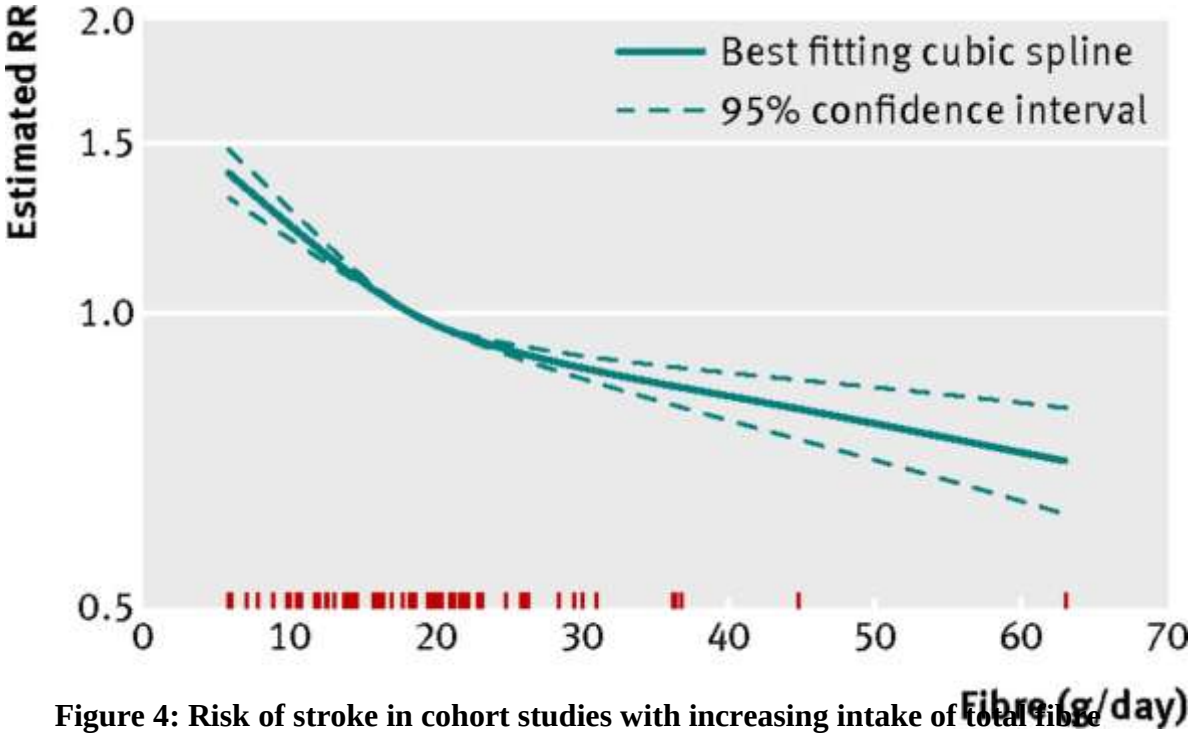
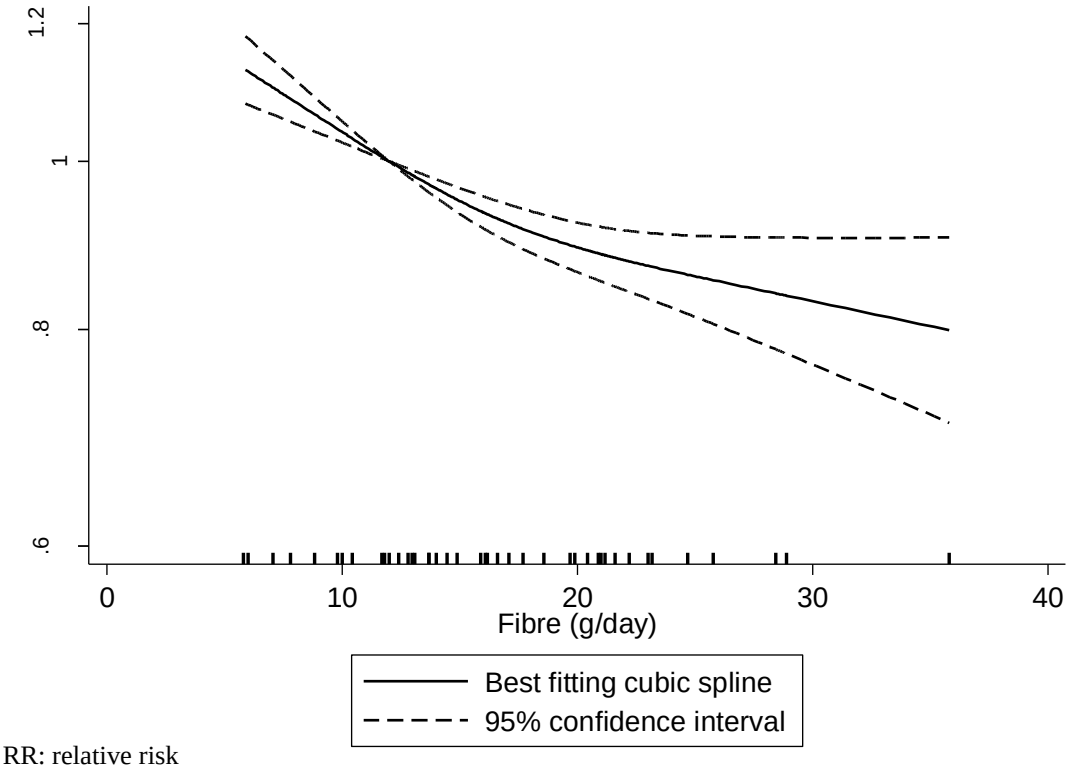


Figure 4: Risk of stroke in cohort studies with increasing intake of total fibre

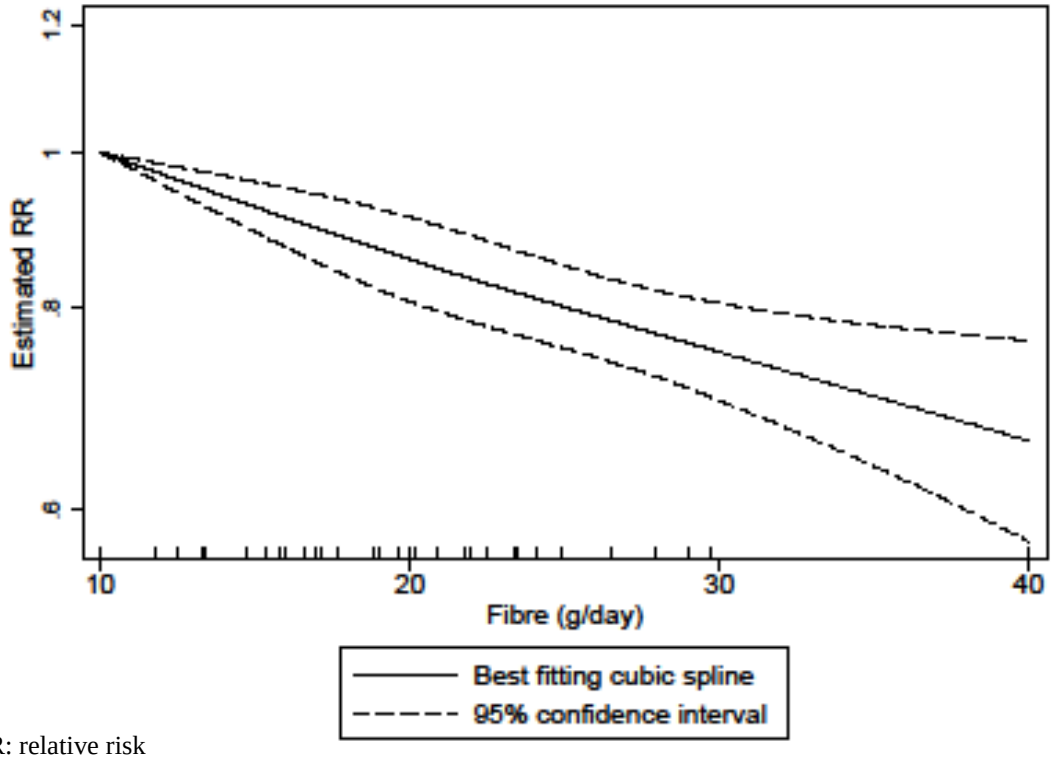
Threapleton et al. 2013b *Stroke* **44**, 1360-1368 (the lines at the bottom of the plot represents where a risk estimate has been provided for a given intake within each study)



RR: relative risk

Figure 5: Risk of type 2 diabetes mellitus in cohort studies with increasing intake of total fibre

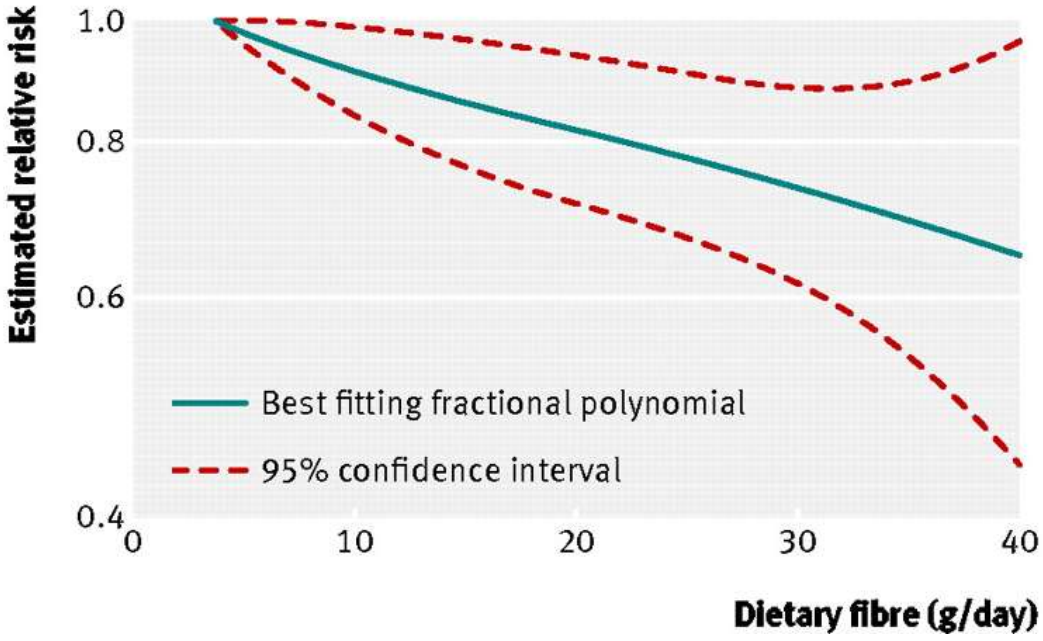
Figure supplied by Victoria Burley based on data in Threapleton et al. 2013a (the lines at the bottom of the plot represents where a risk estimate has been provided for a given intake within each study)



RR: relative risk

Figure 6: Risk of colorectal cancer in cohort studies with increasing intake of total fibre

Aune et al 2011 *BMJ* 343, d6617



11.20. The dietary reference value is based on evidence in which the consumption of a variety of foods rich in dietary fibre as a naturally integrated component is associated with beneficial health outcomes. A diet rich in these foods will also usually be rich in micronutrients and phytochemicals that may have additional health benefits.

11.21. There is a paucity of evidence in relation to the effects of dietary fibre intake in infants and children upon which to base a dietary reference value, e.g. there are no laxation trials and little information is available with regard to possible adverse effects of high intakes of dietary fibre on growth. The National Diet and Nutrition Survey indicates that the upper 2.5 percentile of non-starch polysaccharide intakes in infants (10-13g/day; 13-17g/day AOAC method 985.29 and 991.43) and children aged 4.0-10.0 (19g/day; 25g/day AOAC method 985.29 and 991.43) and 11.0-18.0 years (23g/day; 30g/day AOAC method 985.29 and 991.43) are not associated with any adverse effects on growth (Lennox *et al.*, 2011; Bates *et al.*, 2012). This is supportive of setting a dietary reference value for children as a proportion of the adult dietary reference value, providing they are able to achieve an adequate energy intake and are thriving. The fibre dietary reference value for children would then be based on the energy dietary reference value for a given age range (SACN, 2011) as a proportion of the adult value. The proposed fibre dietary reference values for children and adolescents that are detailed in paragraph 11.21 have been rounded to the nearest multiple of five and are informed by comparative intakes of dietary fibre in different age groups in the NDNS.

11.22. It is recommended that:

- Dietary fibre should be defined as all carbohydrates that are naturally integrated components of foods and that are neither digested nor absorbed in the small intestine and have a degree of polymerisation of three or more monomeric units, plus lignin. Dietary fibre is to be chemically determined using the Association of Official Analytical Chemists (AOAC) method 2009.01 (McCleary et al., 2010; McCleary et al., 2012).
- The dietary reference value for dietary fibre should be 30g/day, as a mean value for the adult population, as defined using the AOAC methods 985.29 and 991.43[10] (Prosky *et al.*, 1988; Lee *et al.*, 1992). The previous dietary reference value of a population average of 18g/day non-starch polysaccharide, defined by the Englyst method, equates to about 23-24 g/day dietary fibre if analysed using these AOAC methods. It is recommended that the dietary reference value is increased to 30g from the evidence described in paragraph 11.18 and it is also one standard deviation above the current DRV.

[10] As the Association of Official Analytical Chemists methods 985.29 and 991.43 used to define the dietary reference value do not measure all of the components of dietary fibre, as described in the dietary fibre definition, the value may need to be revised as the more recent Association of Official Analytical Chemists methods begin to be widely used.

- To correspond to the new adult DRV for fibre, the average intake for children aged 2.0-5.0 years should approximate 15g/day, children aged 5.0-11.0 years should consume on average about 20g/day, children aged 11.0-16.0 years should consume on average about 25 g/day and adolescents aged 16.0-18.0 years about 30g/day. These values have been rounded to the nearest multiple of five and are informed by comparative intakes of dietary fibre in different aged groups in the NDNS. No specific recommendation is made for children aged 2.0 years and less, due to the absence of information, but a diet containing increasing amounts of whole grains, pulses, fruits and vegetables is encouraged in the context of a normal growth pattern.
- Dietary fibre intake should be largely achieved from a variety of foods, such as whole grains, pulses (e.g. kidney beans, haricot beans, lentils), potatoes, vegetables and fruits, where it is a naturally integrated component. At this time, it is not known whether extracted or isolated dietary fibres would convey the range of health benefits associated with the consumption of dietary fibre rich foods.

Chapter 12. Overall summary and conclusions

12.1. This report considered the evidence for a role of dietary carbohydrate in cardio-metabolic, colo-rectal and oral health. Systematic reviews on cardio-metabolic, colo-rectal and oral health were commissioned to inform the evaluation on carbohydrates and health. Evidence from both prospective cohort studies and randomised controlled trials was included in the reviews.

Approach taken in the consideration of the evidence

12.2. The evidence was assessed using the SACN *Framework for the Evaluation of Evidence* (SACN, 2012) and graded according to the system described in Annex 2. This system was devised specifically to consider the evidence included in the Carbohydrates and Health report. In accord with the SACN *Framework for Evaluation of Evidence,* strict inclusion and exclusion criteria were applied in the systematic reviews to ensure the evidence considered was of sufficient quality to be able to draw sound conclusions (SACN, 2012). This restricted the evidence considered to either prospective cohort studies or randomised controlled trials. A total of 225 prospective cohort studies and 403 randomised controlled trials met the inclusion criteria for this report. For the individual systematic reviews and update search see Annex 1.

12.3. There is potential for biases with observational studies and the possibility of confounding by extraneous variables that correlate with both the dependent variable and the independent variable (residual confounding); hence, any associations must be interpreted with caution. The definitions used in cohort studies to characterise and quantify a specific dietary exposure, e.g. 'whole grains', have been used as described even though they may vary between studies.

12.4. Randomised controlled trials can provide strong evidence for a causal relationship between diet and disease risk, yet a limitation is that they generally investigate markers and risk factors, but not actual disease outcomes. Trials are only typically carried out for short periods whilst chronic diet over many years is more relevant to health. Consideration of disease outcomes in relation to carbohydrate intake is, therefore, often dependent on prospective cohort studies. Many of the randomised controlled trials involve mixed interventions that modify other dietary components, e.g. the proportion and type of fat or micronutrient content. They also often involve energy restriction with the aim of producing weight loss. Both of these could potentially affect the outcomes considered. This limits the conclusions that can be drawn; in particular this applies to the effect of variations in total carbohydrate intake and glycaemic index and glycaemic load on cardio-metabolic risk factors. A further limitation is that the dietary interventions vary greatly between trials examining a specific outcome and it is often not possible to consider dose-response effects; this is especially so with regard to trials varying total carbohydrate intake.

12.5. For many of the outcomes considered in this report, there was insufficient evidence of the required quality. Where possible, the dose-response relationships between carbohydrate intakes and health outcomes have been considered and used to inform the dietary reference values.

Total dietary carbohydrate

12.6. Prospective cohort studies indicate no association between total carbohydrate intake and the incidence of cardiovascular disease endpoints, type 2 diabetes mellitus, glycaemia or colo-rectal cancer. Cohort studies and trials conducted in children and adolescents indicate no association between total carbohydrate intake and body mass index or body fatness. There is a lack of evidence available for oral health outcomes in relation to total carbohydrate intake.

12.7. Randomised controlled trials assess the effect of varying total carbohydrate intake and reciprocal variation of fat type and quantity and/or protein intake. No effect of varying total carbohydrate intake on vascular function, inflammatory markers and risk factors for type 2 diabetes mellitus has been demonstrated. Fasting blood lipid concentrations and blood pressure are affected, but it is not possible to exclude confounding by a concomitant reduction in fat intake and/or differences in weight loss between experimental groups. This caveat applies to all the cardio-metabolic risk factors investigated. The trials provide evidence that an energy-restricted higher carbohydrate, lower fat diet, as compared with a lower carbohydrate average fat diet, may be effective in reducing body mass index, but the evidence is based on only four trials and there is high heterogeneity between them.

12.8. Overall, the evidence from both prospective cohort studies and randomised controlled trials indicates that total carbohydrate intake appears to be neither detrimental nor beneficial to cardio-metabolic health and colo-rectal health.

Sugars and sugars-sweetened foods and beverages

12.9. Prospective cohort studies indicate that sugars or sugars-sweetened beverage intake is not associated with the incidence of colo-rectal cancer. There is no association between the incidence of type 2 diabetes mellitus and total or individual sugars intake, but a greater risk is associated with a higher intake of sugars-sweetened beverages. There is insufficient evidence to enable conclusions to be drawn in relation to cardiovascular disease endpoints. Prospective cohort studies, conducted in children and adolescents, indicate that higher consumption (i.e. the amount) of sugars, sugars-containing foods and sugars containing beverages is associated with a greater risk of dental caries in the deciduous and permanent dentitions. A higher frequency of consumption of sugars-containing foods and beverages, but not total sugars, is also associated with greater risk of dental caries in the deciduous and permanent dentitions. Identifying the relationship between dental caries and sugars intake is confounded by oral hygiene and global preventative measures that have reduced the incidence of caries worldwide.

12.10. Randomised controlled trials conducted in adults indicate that increasing sugars intake when consuming an *ad libitum* diet, either through the substitution of other macronutrient components or by replacement of non-caloric sweeteners by sugars, leads to an increase in energy intake. It appears that this relationship may be linear so that as the intake of sugars increases, the more energy is consumed (Figure 1, chapter 11). Randomised controlled trials conducted in children and adolescents demonstrate that consumption of sugars-sweetened beverages, as compared with non-calorically sweetened beverages, results in weight gain and an increase in body mass index. Trials in which sugars are consumed in place of non-caloric sweeteners demonstrated insufficient voluntary reduction in intake of other foods or drinks implying incomplete appetite compensation for the additional energy supplied by the sugars, and leading to weight gain. Trials examining cardiovascular risk factors, inflammatory markers and risk factors for type 2 diabetes mellitus demonstrate no effects of increasing sugars intake. There is also insufficient evidence to assess the link between individual sugars and sugars-sweetened foods and beverages and cardio-metabolic outcomes.

12.11. The WHO draft guideline for sugars intake for adults and children (WHO, 2014) was published in the final stages of the drafting of this report. The reviews on sugars intake in relation to body weight and oral health (Te Morenga *et al.*, 2013; Moynihan & Kelly, 2014), conducted as part of the WHO draft guideline for sugars intake for adults and children, employed different inclusion criteria to the reviews conducted to inform this report e.g. WHO included studies of shorter duration. Different studies and trials were considered to inform the WHO draft guideline as compared with this report, e.g. non-randomised trials, population and cross-sectional studies were also included in WHO's systematic reviews (see Chapter 1 and Annex 2 for the inclusion criteria used in the reviews informing this report). It should be noted that WHO's draft recommendations to limit sugar consumption are based on evidence for reducing the risk of dental caries.

12.12. Overall, there were very few studies on individual sugars, such as glucose, fructose or sucrose, which met the inclusion criteria for this report. Prospective cohort studies indicate higher consumption of sugars and sugars-containing foods and beverages is associated with a greater risk of dental caries. Prospective cohort studies indicate that greater consumption of sugars-sweetened beverages is associated with risk of type 2 diabetes mellitus. The randomised controlled trials indicate that a higher sugars intake increases energy intake and that the consumption of sugars-sweetened beverages results in weight gain and an increase in body mass index.

Starch and starch-rich foods

12.13. Prospective cohort studies indicate no association between starch and refined grain intake and cardiovascular disease endpoints and type 2 diabetes mellitus. There is a lack of evidence relating colo-rectal cancer and oral health to starch and starch-rich foods intake.

12.14. Cohort studies indicate an association between greater consumption of white rice and risk of type 2 diabetes mellitus in Asian populations (in Japan and China) consuming amounts of white rice which are not generally achieved in the UK. It is therefore uncertain whether the detrimental association is relevant to the whole UK population. Consumption of brown rice is associated with a reduction in the risk of type 2 diabetes mellitus, but the evidence is limited to a small number of studies. A higher consumption of potatoes is associated with risk of type 2 diabetes mellitus, but it is not possible to exclude confounding by other dietary variables, e.g. cooking methods such as frying. Moreover, the evidence is limited to a small number of studies.

12.15. Overall, the available evidence in relation to cardio-metabolic outcomes indicates no association with dietary starch intake when consumed in the amounts typical of the UK diet.

Dietary fibre

12.16. Prospective cohort studies indicate that a diet rich in dietary fibre is associated with a lower incidence of cardiovascular diseases, coronary events, type 2 diabetes mellitus and colo-rectal cancer. Over the ranges of intakes studied, it was found that as more dietary fibre was consumed a greater reduction in risk was observed (see figures 2-6 chapter 11). No association is indicated between dietary fibre intake and body weight. There is a lack of evidence for oral health in relation to dietary fibre intake. Although the definitions used to define whole grains vary between studies, higher whole grains consumption is associated with a lower incidence of cardiovascular disease, hypertension, type 2 diabetes mellitus and colon cancer. Higher cereal fibre consumption is associated with a lower incidence of coronary events, type 2 diabetes mellitus, colo-rectal and colon cancer, although the evidence is more limited for individual types of dietary fibre due to the smaller number of studies.

12.17. Randomised controlled trials indicate no effect of dietary fibre intake on cardiovascular or type 2 diabetes mellitus risk factors. Trials indicate that dietary fibre, wheat fibre and other cereal fibres decrease intestinal transit times and increase faecal mass. Higher cereal fibre consumption is beneficial in the treatment of constipation. Trials demonstrate no effect of cereal fibre supplements on recurrence of colo-rectal adenomas (which can be a precursor of colo-rectal cancer). Trials in subjects receiving oat bran and isolated β-glucan demonstrate beneficial effects on fasting blood lipid concentrations and blood pressure. The doses of oat fibre and β-glucan used in these trials generally exceed levels currently consumed in a typical UK diet, but recent and future developments of food ingredients may substantially increase the intake of isolated β-glucan.

12.18. Overall there is strong evidence from cohort studies that increased intake of dietary fibre, cereal fibre and wholegrain is associated with a lower risk of cardiometabolic disease and colorectal cancer. Randomised controlled trials indicate that dietary fibre, wheat fibre and other cereals fibres promote good colo-rectal function by increasing faecal mass and decreasing intestinal transit times. Randomised controlled trials also indicated that higher intake of oat bran and isolated β-glucan leads to lower total cholesterol, LDL and TAG concentrations and lower blood pressure.

12.19. No prospective cohort studies have examined the relationship between non-digestible oligosaccharides or resistant starch and health/disease outcomes. Randomised controlled trials in which diets were supplemented with non-digestible oligosaccharides demonstrated a beneficial effect on fasting blood lipid concentrations, but no effect on fasting glucose and insulin concentration or energy intake. Trials demonstrated that non-digestible oligosaccharides cause an increase in faecal mass and an effect on faecal bacterial content, but no effect on faecal short chain fatty acid levels. Non-digestible oligosaccharides or inulin supplementation increases fractional absorption of calcium in children, but not in adults. Resistant starch supplementation has no effect on energy intake or faecal bacterial content, but increases faecal mass and modifies faecal short chain fatty acid levels.

12.20. Overall randomised controlled trials in adults indicate that supplementation with non-digestible oligosaccharides improved blood lipid concentrations, increased faecal mass and bacterial content. Resistant starch supplementation increased faecal mass and short chain fatty acid content. The health significance of the effects on faecal parameters and net calcium absorption in children is unclear. While some effects are demonstrated for both non-digestible oligosaccharides and resistant starch, the supplement doses in trials currently exceed estimated dietary intakes in the UK, but a large number of foods containing these compounds have already been authorised in Europe and the potential or actual impact of these on overall intakes is unclear.

Glycaemic index and load

12.21. Prospective cohort studies indicate that a diet higher in glycaemic index or load is associated with a greater risk of type 2 diabetes mellitus. There is no evidence from prospective cohort studies to suggest an association between glycaemic index and cardiovascular disease or coronary heart disease. Glycaemic load is associated with a greater risk of cardiovascular disease, but the evidence is limited due to the small number of studies. The available evidence does not suggest an association between glycaemic index or load and colo-rectal cancer incidence. There is a lack of evidence for oral health in relation to glycaemic index or load.

12.22. Randomised controlled trials have assessed the effect of varying glycaemic index primarily by changing the quality of the dietary carbohydrate sources. Trials assessing the effect of varying glycaemic load have reduced carbohydrate intake, reciprocally varied fat, type and quantity, and/or protein intake, as well as changing the quality of dietary carbohydrate. These trials indicate no effect of varying glycaemic index or load on vascular function, inflammatory markers, risk factors for type 2 diabetes mellitus and obesity. The trials indicate a higher glycaemic index diet may affect fasting blood lipid concentrations, but it is not possible to exclude confounding due to differential weight changes between the groups. A higher glycaemic load diet has been shown to reduce diastolic blood pressure and fasting blood triacylglycerol concentration to a lesser degree than the lower glycaemic load diet, but it is not possible to exclude confounding by other dietary variables and the evidence is limited due to the small number of trials.

12.23. Higher and lower glycaemic index or load diets will, in most cases, differ in many ways other than just the carbohydrate fraction. It is not possible, therefore, to assign cause-effect relationships for outcomes based on variation in diet glycaemic index or load, as the nature of the intervention includes a number of factors.

Dietary carbohydrate recommendations

Carbohydrate dietary reference values for aged 2.0 years and above

12.24. The dietary reference values for total carbohydrate, free sugars, starch and sugars contained within the cellular structure of food, and milk sugars have been proposed in the context of an energy intake which is appropriate to maintain a healthy weight (SACN, 2011).

12.25. It is recommended that:

- The dietary reference value for total carbohydrate should be maintained at a population average of approximately 50% of dietary energy.

12.26. It is recommended that:

- The definition for 'free sugars' be adopted in the UK. This comprises all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and unsweetened fruit juices. Under this definition lactose when naturally present in milk and milk products is excluded.
- The dietary reference value for free sugars should be set at a population average of around 5% of dietary energy for age-groups from 2.0 years upwards. This is based on the need to limit free sugars to no more than 10% of total energy intake at an individual level, which is likely to lead to a population average free sugars intake of around 5% of total energy.
- With the proposed reduction in the population reference intake of free sugars, the energy should be replaced with starches, sugars contained within the cellular structure of foods and in milk and milk products.
- The consumption of sugars-sweetened beverages should be minimised in both children and adults.

12.27. It is recommended that:

- Dietary fibre should be defined as all carbohydrates that are naturally integrated components of foods and that are neither digested nor absorbed in the small intestine and have a degree of polymerisation of three or more monomeric units, plus lignin. Dietary fibre is to be chemically determined using the Association of Official Analytical Chemists (AOAC) method 2009.01 (McCleary et al., 2010; McCleary et al., 2012).
- The dietary reference value for dietary fibre for an adult population average should be 30g/day, as defined using the AOAC methods 985.29 and 991.43[11] (Prosky *et al.*, 1988; Lee *et al.*, 1992). The previous dietary reference value of 18g/day of non-starch polysaccharides, defined by the Englyst method, equates to about 23-24 g/day of dietary fibre if analysed using these AOAC methods, thus the new recommendation represents an increase from this current value.
- The average intake for children aged 2.0 to 5.0 years should approximate 15g/day,

[11] As the Association of Official Analytical Chemists methods 985.29 and 991.43 used to define the dietary reference value do not measure all of the components of dietary fibre, as described in the dietary fibre definition, the value may need to be revised as the more recent Association of Official Analytical Chemists methods begin to be widely used.

children aged 5.0 to 11.0 years should consume on average about 20g/day, children aged 11.0 to 16.0 years should consume on average about 25 g/day and adolescents aged 16.0 to 18.0 years about 30g/day. No specific recommendation is made for children aged under 2.0 years, due to the absence of information, but a diet containing increasing amounts of whole grains, pulses, fruits and vegetables is encouraged.

- Dietary fibre intake should be largely achieved from a variety of foods, such as whole grains, pulses (e.g. kidney beans, haricot beans, lentils), potatoes, vegetables and fruits, where it is a naturally integrated component. At this time, it is not known whether extracted or isolated dietary fibres would convey the range of health benefits associated with the consumption of dietary fibre rich foods.

UK carbohydrate intakes

12.28. The UK National Diet and Nutrition Survey (NDNS) provide robust data at national population level on intakes of carbohydrates, sugar and fibre. Latest available data covering 2008/09 – 2011/12 were compared with the Dietary Reference Values set by COMA in 1991. Mean intakes of total carbohydrate met or were close to meeting the dietary reference value. Mean intakes of non-milk extrinsic sugars exceeded the current dietary reference value (10% of total energy intake) in all age groups and were highest in children aged 4.0-10.0 years and children aged 11.0-18.0 years (14.7% and 15.4% of total energy intake respectively). Sugars sweetened beverages (including fizzy drinks, energy drinks and squashes and cordials) provided 30% of non-milk extrinsic sugars intake in the 11.0-18.0 year age group and 16% of intake in younger children and adults. Fruit juice provided 10% of non-milk extrinsic sugars intake in the 11.0-18.0 year age group and 13% in younger children.

12.29. Other sources of non-milk extrinsic sugars were table sugar, preserves and confectionery and biscuits, cakes, buns, pastries and breakfast cereals. Mean intakes of dietary fibre (defined as non-starch polysaccharides) in all age groups were below the current dietary reference value of 18g/day set for adults. Mean intakes were less than 14g/day in adults and 11-12g/day in children. Cereals and cereal products were the main sources of fibre intake in all age groups, followed by vegetables and potatoes. There is evidence from NDNS and other surveys of a socio-economic gradient in intakes of both sugars and fibre, with higher intakes of non-milk extrinsic sugars as a percentage of energy for adults and lower intakes of non-starch polysaccharides for both adults and children in the lower income groups.

12.30. Changing the definition of sugars from non-milk extrinsic sugars to free sugars would slightly reduce mean intakes as a percentage of total energy intake but setting the dietary reference value for free sugars to no more than 5% of total energy intake means that current mean intakes in all age groups would be at least twice the dietary reference value and three times the dietary reference value in the 11-18 year age group.

12.31. Changing the definition of fibre from non-starch polysaccharides to AOAC would increase mean intakes by 3-5g in each age group. Setting the dietary reference value for AOAC fibre at 30g/day for adults means that current mean intakes would be 10-11g below the dietary reference value for men and 13g below for women. For children, setting the dietary reference value for AOAC fibre to 15g for the 2.0-5.0 year age group and 20g for the 5.0-11.0 year age group means that current mean intakes would be 4-6g below the dietary reference value. For older children aged 11.0-16.0 years, setting the dietary reference value at 25g means that current mean intake would be 9g below the dietary reference value.

Recommended dietary pattern in relation to carbohydrate

12.32. The evidence considered in this report endorses a dietary pattern concerning carbohydrates that is based on whole grains, pulses (e.g. kidney beans, haricot beans, lentils), potatoes, vegetables and fruits, but limiting the amounts consumed of table sugar and rich sources of free sugars, such as preserves and sweet spreads, fruit juice, confectionery, biscuits, buns and cakes. The report also provides evidence that sugars sweetened beverages should be consumed in minimal (i.e. infrequently and in small) amounts.

12.33. The National Diet and Nutrition Survey shows that, as a whole, the population consumes more than the recommended amount of sugars and the intakes of fibre are below current advice. With the proposed increase of the dietary reference value for fibre and the reduction of the dietary reference value for free sugars, the difference between recommendations and the population's intake would become even greater for both. In order to address this imbalance, there needs to be a change in the population's diet so that people derive a greater proportion of dietary energy from carbohydrate-rich foods that are low in free sugars and high in dietary fibre.

Research recommendations

To be added to the finalised report.

Annex 1. Cardio-metabolic, colo-rectal and oral heath systematic reviews, additional meta-analyses and update search

Links to these are to be included

Annex 2. Summary of review methodology

A2.1. For the cardio-metabolic health review, literature searches were performed to identify evidence on carbohydrates and cardiovascular disease, type 2 diabetes mellitus and obesity, and their associated risk markers. Prospective cohort studies and randomised controlled trials were included and the literature searches were performed from 1990 up to November and December 2009. A start date of 1990 was chosen, as the evidence prior to that was considered by the Committee on Aspects of Food Policy (COMA). To be included in the review, cohort studies investigating cardiovascular disease had to be adjusted for age and smoking as important confounders and for type 2 diabetes mellitus studies had to be adjusted for age and body mass index as important confounders. For more details on how the review was conducted, refer to the introductory section of the cardio-metabolic health review (see Annex 1).

A2.2. For the colo-rectal health review, literature searches were performed for trials on normal colo-rectal function (bowel habit/function, faecal microflora, fermentation products and calcium and magnesium absorption) and impaired colo-rectal function (constipation, prevention of diarrhoea, diverticular disease and irritable bowel syndrome). In terms of colo-rectal cancer risk, prospective studies on colo-rectal cancer and trials investigating the recurrence of colo-rectal adenomas were included. The literature searches were performed from the inception of the relevant database, regardless of the start date, up to November 2010. To be included in the review, cohort studies investigating colo-rectal cancer had to be adjusted for alcohol intake, smoking, physical activity, age and overweight/obesity as important confounders. For more detail on the review methodology, refer to the colo-rectal health review see Annex 1.

A2.3. For the oral health review, literature searches were performed for cohort studies and randomised controlled trials on dental caries, periodontal disease, tooth wear (including tooth loss) and oral mucosal lesions (including oral cancer). To be included in the review, cohort studies investigating dental caries, erosion or periodontal disease must have been adjusted for smoking status (in adults), brushing frequency and age. For oral cancer, cohort studies were included only if they adjusted for smoking status, alcohol intake and age as they are important confounders. Searches were conducted from the inception of the relevant databases up to January 2011 regardless of start date. For more details on the review methodology refer to the oral health review. Fruit juice search terms were part of the search strategy for oral health, but not for colo-rectal or cardio-metabolic health.

A2.4. Studies conducted in pregnant women were excluded from the cardio-metabolic health review because pregnancy represents a unique physiological and metabolic state and any changes observed could not be extrapolated to the general population. Studies conducted in children aged less than 5 years of age were also excluded from the cardio-metabolic health review.

A2.5. Outcomes included in the update search were prioritised as follows: cardiovascular disease, coronary events, stroke, total cholesterol, LDL cholesterol, HDL cholesterol, triacylglycerol, type 2 diabetes mellitus, glycaemia, impaired glucose tolerance, Hb1Ac, insulinaemia, insulin resistance, C-reactive protein, vascular function, dental caries, periodontal disease, faecal output (transit time, faecal weight), faecal bacteria, faecal pH and short chain fatty acid content, constipation and colo-rectal cancer. The data from the included studies were extracted into tables using the same format as in the respective systematic reviews. These can be found in Annex 1 of this report. In the update search, studies investigating the effect of different polymorphisms were not included.

Data analysis

A2.6. Where there were three or more studies which were sufficiently similar in design included in the systematic reviews, a meta-analysis was conducted using a random effects model. For meta-analyses of cohort studies, a dose response approach was used to quantify the relationship between dietary intakes on particular health outcomes. In the colo-rectal health review this was referred to as the 'per unit analysis'. Meta-analyses using the highest versus lowest quantile approach were also presented in the colo-rectal health review, however in this report only the results from the dose response meta-analyses have been used since they are more informative.

A2.7. Where studies only reported results by population subgroup within the same study (e.g. men and women, colon and rectal cancer), a fixed effect model was used to combine the results in order to obtain a single estimate. A fixed effect model was used in this situation, since there should be no heterogeneity between results from the same study. The single estimate was subsequently included in the random effects model.

A2.8. The I^2 statistic was used to denote heterogeneity and provides the percentage of variation across the studies, which is due to systematic differences between studies. In general, an I^2 statistic of 0-25% reflects low heterogeneity, 25-75% is medium heterogeneity and >75% is high heterogeneity. It was agreed that if the result produced an I^2 of more than 75%, the pooled estimate would not be presented because it indicates that there is excessive heterogeneity and the result would have little meaning. In this case, the studies were presented in a forest plot only.

A2.9. In the systematic reviews, where meta-analyses were available they have been used to inform conclusions but were not used as the sole basis on which to judge the evidence. Those studies which provided insufficient information to be incorporated into a meta-analysis were considered in support of this evidence.

A2.10. For studies investigating fibre intake in relation to health outcomes, the cardio-metabolic health review applied a conversion factor to studies using non-starch polysaccharide so they could be compared with studies using AOAC before being included in a meta-analysis. For the colo-rectal health review, studies measuring both non-starch polysaccharide and AOAC were included in a meta-analysis without applying a conversion factor.

A2.11. For more details on the statistical approaches used, refer to the original cardio-metabolic health and colo-rectal health systematic reviews (see annex 1). Meta-analyses were not possible to conduct in the oral health review and for some outcomes in the colo-rectal health review because the studies were not sufficiently comparable, therefore data were extracted into tables and are discussed in the narrative.

Grading system for judging the evidence

A2.12. It was considered important to grade the evidence included in this report in order to convey the strength and quality of evidence identified. Established grading systems from the World Cancer Research Fund, The Working Group on Grading of Recommendations, Assessment, Development and Evaluation (GRADE) and the German Nutrition Society were evaluated, however they were considered not to offer the flexibility required for a risk assessment of this nature. Using the SACN framework for the evaluation of evidence as a basis, a grading system specifically for use in this report was devised. Details of this system have been provided in the following paragraphs.

A2.13. The scientific evidence included in the commissioned systematic reviews and the update search was used to draw conclusions on the relationship between carbohydrates and different health outcomes/intermediate markers. An approach to judge, assess and categorise the evidence based on the nature of the relationship observed and the amount of available evidence was agreed.

A2.14. In keeping with the Framework for the Evaluation of Evidence, the word 'association' was used to describe the evidence from cohort studies and the word 'effect' was used when referring to evidence from randomised controlled trials.

A2.15. Expert judgement was used to determine the exact grading. This included taking account of study quality, study size and methodological considerations, which may have resulted in the upgrading or downgrading of evidence, where appropriate.

A2.16. Generally, evidence from three or more cohort studies was used to judge if there was an association particularly where a meta-analysis had been performed. However, this is not a strict cut-off and expert judgement was applied to determine whether the quality of the evidence permitted a conclusion to be made. Where an association appeared to be present, the evidence was considered as adequate, moderate or limited. The number of studies acted as an approximate guide only, since issues surrounding study design, study size, methodology and definition of exposures affect how evidence is graded.

- Adequate – five or more cohort studies and a meta-analysis have been performed. Alternatively, where there are a total of five or more studies identified in both the systematic review and/or the update search combined, which consistently showed the same outcome without a meta-analysis.
- Moderate – meta-analysis on 3-4 cohort studies may be present. Alternatively, a total of five studies identified from the systematic review and/or update search combined, but without a meta-analysis.
- Limited – three to four cohort studies have been included, but with no meta-analysis, and there is some indication that the results are in the same direction. However, the

evidence may also be considered limited if there are a number of studies but the biological plausibility is unclear or the methodology is not precise.

A2.17. Generally, there needed to be three or more randomised controlled trials to determine if there was evidence of an effect or not. The expert judgement was used to determine if the quality of these data permitted a conclusion to be drawn. Where an effect appeared to be present, the evidence was considered as adequate, moderate or limited. The number of studies was used as an approximate guide only, since issues surrounding study design, study size methodology and definition of exposures affected the grading of the evidence.

- Adequate – where there were three or more randomised controlled trials that had been included in a meta-analysis. Alternatively, there were a total of four or more studies identified in both the systematic review and/or the update search combined, which consistently showed the same outcome without a meta-analysis.
- Moderate – There may be a meta-analysis present or a total of three or more randomised controlled trials identified from both the systematic review and/or the update search combined, which are consistently showing the same outcome without a meta-analysis.
- Limited –If there were a number of studies but the biological plausibility is unclear, the methodology is not precise or the exposures are heterogeneous.

A2.18. The evidence was normally considered as insufficient where there were fewer than three studies of the same type. Alternatively, if there were more studies but they were of poor quality, the evidence may have also been considered as insufficient.

A2.19. Evidence was deemed inconsistent according to statistical considerations i.e. in a meta-analysis, when I^2 >75%, the confidence intervals do not overlap or if the results of individual studies are not in the same direction. When the I^2 was greater than 75%, but the forest plot suggested there was evidence of a direction for an outcome expert judgement was used to upgrade the conclusion, where appropriate.

A2.20. In addition to the evidence grading, if there was an effect or association shown statements were provided on the following:

- Whether the effect or association indicated beneficial or adverse effects with higher intakes of carbohydrate or carbohydrate components
- Whether the effect or association was biologically relevant, noting that some changes may be important at a population level, but not at an individual level. This statement also contains, where appropriate, an indication as to whether the intakes reported in the studies were achievable through diets typically consumed in the UK.

Upgrading criteria

A2.21. Specific factors, when present, were used to upgrade the judgement reached. Expert judgement was used to apply the factors (listed below)

- Presence of a plausible biological gradient ('dose response') in the association or effect. Such a gradient need not be linear and may even be 'U'-shaped, so long as this can be explained plausibly.
- Data from a large, well designed randomised controlled trial, which is adequately powered.
- A relatively large summary effect size after appropriate control for confounders. The pooled estimates from observational studies included in the meta-analysis have a relative risk above 1.2 for greater risk or below 0.8 for decreased risk for an agreed increment of intake.
- Evidence from appropriately controlled experiments demonstrating one or more plausible and specific mechanisms in humans.
- Consistent direction of effect, despite a small effect size.
- Evidence was also downgraded if there were concerns around study design, if the exposure was heterogeneous or the methodology used to measure the exposure or outcome was not precise.
-

Concluding statements for judging the strength of the evidence

Randomised controlled trials	Prospective cohort studies
Effect – Adequate evidence	Association – Adequate evidence
Effect – Moderate evidence	Association – Moderate evidence
Effect – Limited evidence	Association – Limited evidence
No effect - Adequate evidence	No Association - Adequate evidence
No effect – Moderate evidence	No Association – Moderate evidence
No effect – Limited evidence	No Association – Limited evidence

A2.22. Note – the following concluding statements were applied to both randomised controlled trials and prospective cohort studies:

- No conclusion- insufficient evidence.
- No conclusion- inconsistent evidence.

A2.23. When an effect or association was identified, a comment as to whether the relationship was beneficial or adverse with higher intakes of carbohydrate or carbohydrate components was included. A final comment was added concerning whether the effect or association is biologically relevant; noting that some changes may be small but important at a population level and whether the intakes reported in the studies are achievable through diet. Biological relevance was be judged by drawing on the expertise of the committee. For findings on blood lipids, blood pressure and BMI published literature was also used to inform biological relevance (Stamler *et al.*, 1993; He & Whelton, 1999; Cullen, 2000; Lewington *et al.*, 2007; Flegal *et al.*, 2013; Liu *et al.*, 2013; Asayama *et al.*, 2014). A finding was considered to be biologically relevant when it was considered that people could change their diet to impact on disease risk in a way that the level of alteration in the dietary component could be achievable within a normal diet.

Annex 3. Commentary on the evidence on fructose and health

A3.1. Fructose intakes, through the use of the sweetener high fructose corn syrup (HFCS, also known as isoglucose), have increased in parallel with obesity rates. Current intakes of fructose (as separate from sucrose) in the UK are estimated to provide 3.4% of total energy (3.5% of food energy) in adults, but the National Diet and Nutrition Survey (NDNS) does not capture intakes of HFCS; therefore there are no data on current intakes of this sweetener in the UK. However HFCS is more commonly used in the US than in Europe. HFCS is virtually excluded from Europe at the moment by EU quotas, so that it currently accounts for less than 5% of sweeteners. Products sweetened with HFCS are not necessarily significantly higher in fructose than other foods as it has a similar composition to sucrose, which is 50% glucose and 50% fructose.

A3.2. Fructose is metabolised differently compared with other monosaccharides and, for this reason, some have argued that it promotes lipid production and insulin resistance. A few trials have demonstrated that supplementing subjects with high levels of pure fructose (without similar amounts of glucose) leads to increased fasting blood lipid concentrations, particularly triacylglycerol (TAG), fat deposition, and lower insulin sensitivity compared with those consuming glucose (Teff *et al.*, 2004; Swarbrick *et al.*, 2008; Stanhope *et al.*, 2009; Teff *et al.*, 2009; Stanhope *et al.*, 2011a; Stanhope *et al.*, 2011b). None of these trials met the inclusion criteria for the reviews conducted to inform this report because they were either not randomised trials (Swarbrick *et al.*, 2008; Stanhope *et al.*, 2009; Stanhope *et al.*, 2011a; Stanhope *et al.*, 2011b) or they were of insufficient duration, i.e. less than six weeks (Teff *et al.*, 2004; Teff *et al.*, 2009).

A3.3. A randomised controlled trial performed in overweight men found that there was no difference in body weight or levels of hepatic TAG if subjects consumed pure fructose or glucose as part of an isocaloric diet (Johnston *et al.*, 2013). However when subjects consumed glucose or fructose in addition to an *ad libitum* diet, body weight increased together with hepatic, calf muscle and serum levels of TAG, but these did not differ significantly between the fructose or glucose intervention. These results imply that the changes demonstrated were as a result of the additional energy consumed. A systematic review of trials investigating the effect of feeding subjects pure fructose found that overall there was no significant effect on body weight when the intervention involved substituting fructose for another carbohydrate using an isocaloric diet (Sievenpiper *et al.*, 2012). However when fructose was provided in addition to the normal diet, weight significantly increased. The authors noted that a number of the trials they reviewed were not randomised, provided large doses of fructose and were of short duration, with only one of the trials being longer than four weeks.

A3.4. A systematic review and meta-analysis of randomised and non-randomised trials found that there was no statistically significant effect of feeding subjects pure fructose compared with isoenergetic amounts of other carbohydrates on the concentrations of total, LDL or HDL cholesterol. However when the analysis was stratified by dose, it was found that very high doses of 100g or more of fructose significantly increased the concentrations of total and LDL cholesterol. Of the twenty four trials included, only eleven of them were randomised, nine studies were conducted in diabetics and only nine studies had a duration of 6 weeks or longer (Zhang *et al.*, 2013).

A3.5. It is important to note that in these trials participants consumed sweetened beverages containing high doses of either pure fructose or pure glucose (25-30% of their daily energy intake). However glucose and fructose are not commonly consumed in pure form within the normal diet, since they are either consumed as components of sucrose or high fructose corn syrup. Sucrose consists of 50% glucose and 50% fructose and high fructose corn syrup contains glucose and fructose in ratios similar to sucrose. The body absorbs free fructose and glucose, or the same sugars derived from sucrose and HFCS, in exactly the same way. Therefore it appears unlikely that fructose, as consumed as a component of HFCS, causes metabolic abnormalities or promotes weight gain more than other sugars consumed in an isocaloric diet (Klurfeld *et al.*, 2013).

A3.6. There was only one trial that was eligible to be included in the cardio-metabolic health review in which subjects were supplemented with either glucose or fructose for six weeks (Bantle *et al.*, 2000). The sugars provided 17% of daily energy, 14% of which were added to foods and drinks in their pure form with the remaining 3% being consumed from naturally occurring fructose or glucose. At the end of the intervention TAG concentrations were significantly higher in men following fructose supplementation. However there were no significant differences between the fructose and glucose interventions for any of the other fasting blood lipid concentrations measured.

A3.7. There are few data from trials investigating fructose intake akin to how it would be consumed in the diet, or as HFCS. One trial investigated the effect of consuming different amounts of sucrose and HFCS sweetened, low-fat milk as part of an energy restricted diet, on weight loss and fasting blood lipids in overweight and obese subjects (Lowndes *et al.*, 2012). There were no differences in the extent of weight loss and measures of adiposity or fasting blood lipid concentrations between the sucrose and HFCS containing diets. Another trial by the same group demonstrated that consuming sucrose or HFCS sweetened low-fat milk as part of an *ad libitum* diet had no effect on the amount of fat in the liver or skeletal muscle or most fasting blood lipid concentrations. TAG concentrations increased, as did body mass, but this did not differ between the dietary groups (Bravo *et al.*, 2013).

A3.8. Therefore on balance, it is considered that there is insufficient evidence to demonstrate that fructose intake, as consumed in the diet, leads to adverse health outcomes independent of any effects related to its presence as part of total and free sugars.

Annex 4. Dietary acids and tooth wear (including dental erosion)

Introduction

A4.1. Teeth wear in function, as a consequence there would be an expected increase in wear with increasing age of the individual. There are also some intrinsic and extrinsic variables, however, that increase the rate of tooth wear. These can act as individual factors but most likely act in combination during any wear process.

A4.2. There are three types of wear described in the dental literature.

A4.3. Abrasion, in which tooth substance is removed by the abrasive action of an external substance or device, for example a 'smoker's toothpaste' or incorrect use of interdental cleaning aids.

A4.4. Attrition in which tooth substance is removed as a consequence of teeth rubbing together in contact, effectively polishing each other, but when combined with a clenching or tooth grinding habit this can result in rapid destruction of teeth.

A4.5. Erosion in which tooth substance is removed as a consequence of its dissolution by acids from either foods or regurgitated gastric content. This type of wear is different to caries because the acids are not generated in the mouth and it occurs at lower pH than the caries process (typically pH of less than 3 compared with 5.5 or 6 for enamel and dentine respectively for caries). As a consequence of the more aggressive acid attack, the sub-surface demineralisation that characterises dental caries does not occur, rather the tooth surface simply dissolves. The dissolution of mineral from either enamel or dentine results in a residual softened surface that can then be worn away much more rapidly by attrition from tooth-to-tooth contact or by contact with the lips, cheeks and tongue or by tooth brushing, or by hard fibrous foods during chewing. In terms of the study of wear this process would more accurately be described as corrosion, but is universally described as erosion in the dental literature.

A4.6. Dental erosion is a multi-factorial condition in which an interplay between chemical (e.g. food acidity and its calcium, phosphorus and fluoride content), biological (e.g. salivary flow, buffering capacity and acquired pellicle) and behavioural factors (e.g. oral hygiene and eating and drinking habits) underlies large inter-individual variation (Lussi & Jaeggi, 2008).

A4.7. The patterns of wear produced by these three different mechanisms have some characteristic features, so for example when wear occurs on a surface of a tooth that cannot be in contact with another, it can't be produced by attrition. So some attribution of ear mechanisms can be made based on appearance of the teeth. These will tend to be more accurate in younger people where a 'pure' mechanism may have been acting for a short period of time. In adults this linkage is much more difficult to make as a single observation will show the outcomes of a lifetime's function and wear.

A4.8. The Children's Dental Health series of national surveys of oral health reported the prevalence of tooth wear (including dental erosion) in 1993 and 2003. A comparison between the two Children's Dental Health surveys of 1993 and 2003 observed that there has been little change in the proportion of five-year-olds with tooth wear: 52% in 1993 and 53% in 2003. There was, however, an increase in the prevalence of tooth wear on permanent teeth, which was statistically significant at age 15 where 27% upper incisors had tooth wear palatally in 1993 compared with 33% in 2003 (Chadwick et al., 2006).

A4.9. The national surveys of Adult Dental Health reported the prevalence of tooth wear (including dental erosion) in 1998 and 2009 (Steele & O' Sullivan, 2011); the 2009 survey included data from England, Wales and Northern Ireland, but not Scotland. The prevalence of tooth wear in England has increased since the 1998 survey, when two thirds (66%) of the dentate population showed signs of wear compared with over three quarters (76%) in the 2009 survey. There have also been small increases in the proportion of adults with moderate wear, 11 per cent in 1998 compared with 15 per cent in 2009. The greatest increase was in the youngest three age groups; 15 percentage points, 10 percentage points and 13 percentage points for those aged 16 to 24, 25 to 34 and 35 to 44 years, respectively. For adults under the age of 65 moderate and severe tooth wear has increased since 1998, but for those aged 65 and over, there has been a small decrease. While the increase in moderate tooth wear was small, moderate tooth wear in 16 to 34 year olds was suggestive of rapid tooth wear.

A4.10. The study designs for all prospective cohort studies have been considered in the next section, while the study designs for all randomised controlled trials have been considered in the following section. The results from both prospective cohort studies and randomised controlled trials have been considered together for each dietary exposure.

Prospective cohort studies

A4.11. The initial search identified three articles, which were assessed as full-text articles. All three were eligible for inclusion (Lussi & Schaffner, 2000; Dugmore & Rock, 2004; El Aidi *et al.*, 2011).

A4.12. The study design details have been summarised in Table A4.1. All studies investigated tooth wear in mixed and permanent dentition. In one study (Lussi & Schaffner, 2000) the tooth wear indices (facial, oral and occlusal surfaces) were based upon diagnostic criteria used in a previous study (Lussi et al., 1991). Another study assessed tooth wear using the criteria used in 1993 survey of Children's Dental Health (Dugmore & Rock, 2004): a Tooth Wear Index that recorded all three main types of tooth wear irrespective of aetiology (Smith & Knight, 1984). For the third study (El Aidi et al., 2011) the criteria for the clinical assessment of tooth wear were a modification (van Rijkom et al., 2002) of diagnostic criteria developed previously (Lussi, 1996).

A4.13. Cohort sizes ranged from 55 to 1149 and the follow-up period ranged from two to six years. Dietary assessment was by a simple questionnaire in two studies and by food frequency questionnaire in the other (El Aidi et al., 2011). All studies investigated consumption of acidic foods and drinks in relation to tooth wear. Multivariate logistic regression analysis was used in all studies to explore associations. The confounders considered by the studies investigating carbohydrate and dental caries risk have been summarised in Table A4.2. Only one study reported its funding sources which were mainly commercial (El Aidi et al., 2011); 67% of studies did not report funding sources.

Table A4.1. Prospective cohort studies of acidic food and drink intake and risk of dental erosion

Study	Country	Sex	Baseline age (y)	Cohort size	Mean follow-Up duration (y)	Statistical method	Fluoride intake / water content	Dental erosion assessment and method	Dietary assessment method	Dietary components investigated/question asked	Funding source
Lussi & Schaffner, 2000	Switzerland	Mixed	26-50	55	6	MLR	NR	Tooth wear; clinical assessment	Questionnaire	Consumption of fruits, citrus fruits, fruit juice, apple juice, vegetables and yoghurt	NR
Dugmore & Rock, 2004	England	Mixed	12	1149	2	MLR	NR	Tooth Wear Index; clinical assessment	Questionnaire	Consumption of apples, oranges or grapefruit, other fruit, chips with vinegar or tomato sauce, chocolate or sweets (yes or no) Daily frequency of glasses/cans of the water, milk, tea/coffee, chocolate, squash, fruit juice or carbonated soft drink.	NR
El Aidi et al., 2011	The Netherlands	Mixed	mean 11.9	572	3	MLR	NR	Tooth wear; clinical assessment	Food frequency questionnaire every 6 months	Consumption on the previous day of acidic drinks, water, tea, dairy products, yoghurt products, milk products, acidic fruit and non-acidic fruit, sour vegetables, pickled vegetables, cheese, chewing gum, red sauces, curry, chilli sauce and white/yellow sauces	Radboud University Nijmegen, the Dutch Dairy Association, Dutch Sugar Bureau and Dutch Soft Drinks Association

MLR, multivariate logistic regression; mth, month; y, year.

Table A4.2. Confounders considered in prospective studies investigating acidic food and drink intake and dental erosion risk

Study	Age	Sex	Tooth-brushing habits	SES	Gingival index	Plaque index	Calculus	Fluoride intake	Baseline caries prevalence	Ethnicity
Lussi & Schaffner, 2000	Y		Y							
Dugmore & Rock, 2004			Y	Y	Y	Y	Y		Y	Y
El Aidi et al., 2011			Y			Y				

Randomised controlled trials

A4.14. Thirteen articles were identified as eligible (Rugg-Gunn *et al.*, 1998; West *et al.*, 1998; West *et al.*, 1999; Hughes *et al.*, 1999a; Hughes *et al.*, 1999b; Hughes *et al.*, 2002; West *et al.*, 2003; West *et al.*, 2004; Hooper *et al.*, 2004; Hooper *et al.*, 2005; Venables *et al.*, 2005; Caglar *et al.*, 2008).

A4.15. A summary of the trial designs has been given for those trials investigating tooth wear in relation to soft drinks and fruit juices (see Table A4.3). All trials had an in situ design whereby assessment of enamel or dentine blocks contained within intra-oral appliance was investigated. The wear was determined by surface profilometry.

A4.16. The enamel or dentine blocks were exposed to dietary factors by either applying solutions ex vivo, once the intra-oral appliances were removed and then replacing them afterwards, or by exposing dental blocks to dietary factors in vivo, e.g. eating food or consuming drinks while the intra-oral appliance was being worn. In the trial where solutions were dipped onto the dental blocks, subjects were instructed to remove the intra-oral palatal appliances during meals.

A4.17. All trials were cross-over in design with either no washout period or a washout period ranging from two days to one week. The experimental period ranged from seven to twenty one days. The funding sources were mainly commercial, reflecting product modifications to reduce the erosive potential of soft drinks.

A4.18. One of the trials used a split-mouth intra-oral palatal appliance where the blocks on each side of the mouth were exposed to different test solutions (Rugg-Gunn et al., 1998).

A4.19. In a series of trials by the same authors investigating the effect of fruit juices and soft drinks on dental erosion (see Table A4.3), intra-oral appliances were worn between 9am and 5pm and removed for one hour over lunch (West et al., 1998; Hughes et al., 1999b; Hughes et al., 1999a; West et al., 1999; Hughes et al., 2002; West et al., 2003; Hooper et al., 2004; West et al., 2004; Hooper et al., 2005; Hooper et al., 2007). In another trial, investigating the effect of a malt-based drink on dental erosion, intra-oral appliances were worn between 9am and 4pm and removed for one hour over lunch (Caglar et al., 2008). In one trial investigating the effect of sport drink consumption on enamel loss when subjects participated in planned exercise, the intra-oral appliances were worn during the exercise period between 7:00am and 9:10am (Venables et al., 2005).

A4.20. Most of the trials investigated the effect of modifying soft drinks, by the addition of calcium compounds, to reduce their erosive potential, but this aspect has not been included in the review (Rugg-Gunn et al., 1998; Hughes et al., 1999b; Hughes et al., 1999a; West et al., 1999; Hughes et al., 2002; Hunter et al., 2003; West et al., 2003; Hooper et al., 2004; West et al., 2004; Hooper et al., 2005; Venables et al., 2005; Hooper et al., 2007). Several trials investigated the effect of juice drinks non-calorically sweetened with aspartame, acesulfame K and saccharin.

Table A4.3. Soft drink and fruit juice trial design

Study	Trial design	Country	Fluoride	Age (y)	Subject characteristics	Dental erosion method and exposure time	Duration (d)	Dietary assessment method	Basal diet	Intervention	Control intervention	Funding source
Rugg-Gunn et al., 1998	XO – no washout	England	NR	NR	11 adults	Bovine permanent incisor enamel mouth palatal appliances analysed by profilometer and scanning electron microscopy	7	NR	No restrictions – volunteers asked to maintain similar eating and drinking patterns during experimental periods	Dental blocks dipped with either non-calorically sweetened phosphoric acid-based cola drink or non-calorically sweetened citric acid-based orange drink for 15 minutes 4 times/d	Dental blocks dipped with distilled water 4 times/d	Proctor and Gamble Ltd.
West et al., 1998	XO – 1 wk washout	England	No tooth brushing allowed when appliance *in situ*	mean 24, range 20-30	10 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer and surface microhardness	15	Compliance questionnaire completed each study day	Only tea, coffee or water could be consumed while the appliances were *in situ*	250ml orange juice 4 times/d	250ml mineral water 4 times/d	Smith-Kline Beecham Consumer Healthcare
Hughes et al., 1999a	XO – 2 ½ d washout	England	No tooth brushing allowed when appliance *in situ*	mean 28, range 20-34	12 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer	15	Compliance questionnaire completed each study day	Only tea, coffee or water could be consumed while the appliances were *in situ*	250ml orange juice 4 times/d	250ml mineral water 4 times/d	Smith-Kline Beecham Consumer Healthcare
Hughes et al., 1999b	XO – 2 d washout	England	No tooth brushing allowed when appliance *in situ*	NR	15 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer	15	Compliance questionnaire completed each study day	Only tea, coffee or water could be consumed while the appliances were *in situ*	250ml non-calorically sweetened orange drink or non-calorically sweetened apple and blackcurrant juice drink 4 times/d	250ml mineral water 4 times/d	Smith-Kline Beecham Consumer Healthcare
West et al., 1999	XO – no washout	England	No tooth brushing allowed when appliance *in situ*	mean 27, range 20-39	12 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer	15	Compliance questionnaire completed each study day	Only tea, coffee or water could be consumed while the appliances were *in situ*	250ml orange juice or blackcurrant juice drink 4 times/d	250ml mineral water 4 times/d	Smith-Kline Beecham Consumer Healthcare
Hughes et al., 2002	XO – 2 d washout	England	No tooth brushing allowed when appliance *in situ*	mean 29, range 22-42	12 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer and ultrasonication	10	Compliance questionnaire completed each study day, drinks were sipped under supervision	Only tea, coffee or water could be consumed while the appliances were *in situ*	250ml orange juice 4 times/d	250ml mineral water 4 times/d	GlaxoSmithKline Consumer Healthcare
West et al., 2003	XO – 2 d weekend washout	England	No tooth brushing allowed when appliance *in situ*	median 32, range 17-56	15 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer	20	Compliance questionnaire completed each study day	Only tea, coffee or water could be consumed while the appliances were *in situ*	250ml non-calorically sweetened carbonated orange juice drink 4 times/d	250ml mineral water 4 times/d	GlaxoSmithKline Consumer Healthcare
Hooper et al., 2004	XO – no washout	England	No tooth brushing allowed when appliance *in situ*	Mean 33	21 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer	15	Compliance questionnaire completed each study day	Only tea, coffee or water could be consumed while the appliances were *in situ*	250ml sugars-sweetened citric acid-based sports drink 4 times/d	250ml mineral water 4 times/d	GlaxoSmithKline Consumer Healthcare

NR, not reported; y, year; d, day

Soft drink and fruit juice trial design continued

Study	Trial design	Country	Fluoride	Age (y)	Subject characteristics	Dental erosion method and exposure time	Duration (d)	Dietary assessment method	Basal diet	Intervention	Control intervention	Funding source
West et al., 2004	XO – no washout	England	No tooth brushing allowed when appliance *in situ*	mean 34	16 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer	15	Compliance questionnaire completed each study day	Only tea, coffee or water could be consumed while the appliances were *in situ*	250ml non-calorically sweetened apple and blackcurrant juice drink 4 times/d	250ml mineral water 4 times/d	GlaxoSmithKline Consumer Healthcare
Hooper et al., 2005	XO –16hr overnight washout	England	No tooth brushing allowed when appliance *in situ*	range 19-45	10 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer	10	Compliance questionnaire completed each study day, sipping of drinks was supervised	No other food and drink could be consumed while the appliance was *in situ*	1.5litres sugars-sweetened citric acid-based sports drink consumed within 1 hour/d	1.5litres mineral water consumed within 1 hour/d	NR
Venables et al., 2005	XO – no washout	England	NR	mean 22	19 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer	21	NR, sipping of drinks was supervised	No restrictions	1.4 litres sugars-sweetened citric acid-based sports drink consumed within two hours, 5 days a week during planned exercise	1.4 litres mineral water consumed within 2 hours, 5 days a week during planned exercise	GlaxoSmithKline Consumer Healthcare
Hooper et al., 2007	XO – 2 d washout	England	No tooth brushing allowed when appliance *in situ*	18+	15 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer	10	Compliance questionnaire completed each study day	No other food and drink could be consumed while the appliances were *in situ*	250ml non-calorically sweetened citric acid-based drink 4 times/d	250ml mineral water 4 times/d	GlaxoSmithKline Consumer Healthcare
Caglar et al., 2008	XO – no washout	Turkey	NR	range 21-23	10 adults	Human third molar teeth enamel in palatal appliances analysed by profilometer	10	NR	No other food and drink could be consumed while the appliances were *in situ*	250ml/d malt drink	250ml/d mineral water	NR

NR, not reported; y, year; d, day

Risk of bias assessment

A4.21. A summary of the risk of bias assessment has been given in Table A4.4.

A4.22. All trials reported being randomised. Allocation concealment was not reported in any of the trials. One trial reported the method random sequence generation. Only two trials reported having any drop-outs, which ranged from 11-14%. The missing outcome data in these trials were balanced in numbers across intervention groups, with similar reasons for missing data across groups.

A4.23. Due to the nature of the interventions all trials were open to participants since the volunteers were either able to identify the treatments by the flavour and consistency of the solutions or the intervention involved consumption of specific foods. All of the trials reported that the trials were blind with respect to the assessors.

TableA4.4. Risk of bias assessment

Study	Randomisation	Sequence generation	Allocation concealment	Blinding	Incomplete outcome data	Dropouts (%)
Rugg-Gunn et al., 1998	Y	Random number tables	NR	Open to participants and personnel, but assessors blind	No missing outcome data	0
West et al., 1998	Y	NR	NR	Open to participants and personnel, but assessors blind	No missing outcome data	0
Hughes et al., 1999a	Y	NR	NR	Open to participants and personnel, but assessors blind	No missing outcome data	0
Hughes et al., 1999b	Y	NR	NR	Open to participants and personnel, but assessors blind	No missing outcome data	0
West et al., 1999	Y	NR	NR	Open to participants and personnel, but assessors blind	No missing outcome data	0
Hughes et al., 2002	Y	NR	NR	Open to participants and personnel	No missing outcome data	0
West et al., 2003	Y	NR	NR	Open to participants and personnel	No missing outcome data	0
Hooper et al., 2004	Y	NR	NR	Open to participants and personnel, but assessors blind	Missing outcome data unlikely to be related to outcome	14
West et al., 2004	Y	Computer generated randomisation sequence	NR	Open to participants and personnel, but assessors blind	No missing outcome data	13
Hooper et al., 2005	Y	NR	NR	Open to participants and personnel, but assessors blind	No missing outcome data	0
Venables et al., 2005	Y	NR	NR	Open to participants and personnel, but assessors blind	Missing outcome data unlikely to be related to outcome	11
Hooper et al., 2007	Y	NR	NR	Open to participants and personnel, but assessors blind	No missing outcome data	0
Caglar et al., 2008	Y	NR	NR	Open to participants and personnel, but assessors blind	No missing outcome data	0

yes indicates low risk of bias; no indicates high risk of bias; NR, not reported

Results

A4.24. Data on measures of dietary exposure or interventions used and risk assessment methods were insufficiently comparable to allow quantitative synthesis.

A4.25. For the following dietary exposures no relevant articles were identified: glycaemic index and load, dietary fibre, non-digestible oligosaccharides, polyols, sugar, starch, infant feeding, carbohydrate rich foods and total carbohydrate.

Prospective cohort studies

A4.26. The findings from all prospective cohort studies of acidic food and drink consumption and risk of tooth wear have been summarised in Table A4.5.

TableA4.5. Results from prospective cohort studies

Study	% with enamel loss at baseline	% with dentine exposed at baseline	% with enamel loss at follow-up	% with dentine exposed at follow-up	Dietary exposure	Reported association
Lussi & Schaffner, 2000	10-15*	3-8	30-51	8-26	Consumption of fruits, citrus fruits, fruit juice, apple juice, vegetables and yoghurt	Consumption of dietary acids was associated with significantly increased dental erosion
Dugmore & Rock, 2004	56	2	65	9	Consumption of apples, oranges or grapefruit, other fruit, chips with vinegar or tomato sauce, chocolate or sweets Daily frequency of glasses/cans of the water, milk, tea/coffee, chocolate, squash, fruit juice or carbonated soft drink	Carbonated soft drink consumption was significantly associated with increased tooth wear
El Aidi et al., 2011	32	NR	42	NR	Consumption on the previous day of acidic drinks, water, tea, dairy products, yoghurt products, milk products, acidic fruit and non-acidic fruit, sour vegetables, pickled vegetables, cheese, chewing gum, red sauces, curry, chilli sauce and white/yellow sauces	Consumption of alcoholic mixed drinks and sour vegetables and tooth grinding during sleep were significantly associated with increased erosive wear incidence. Consumption of yoghurt and milk products was negatively associated with the incidence of erosive wear. No significant association was indicated between carbonated soft drink consumption and erosive wear incidence or progression

* Data given as ranges in the report; NR, not reported.

A4.27. A small study in adults investigated the association between the consumption of erosive foodstuffs and tooth wear after six years follow-up (Lussi & Schaffner, 2000). An assessment of fruits, citrus fruits, fruit juice, apple juice, vegetables and yoghurt consumption was used to assess dietary acid ingestion. Multiple linear regression analysis revealed age and dietary acid consumption to explain 28% of the variability in the progression of tooth wear between baseline and follow-up. A subgroup analysis of subjects who accounted for most of the total tooth wear progression observed (about a third of cohort subjects) indicated that, compared with the rest of the cohort, their intake of dietary acids and the hardness of their tooth brush bristles were significantly higher and the buffering capacity of their saliva significantly lower.

A4.28. A study of twelve year old children followed-up at 14 years of age reported no significant associations with tooth wear regarding dental cleanliness, gingival health, eating apples, chips with tomato sauce or vinegar, citrus fruit, sweets or chocolate; or drinking coffee, chocolate or squash (Dugmore & Rock, 2004). Experience of caries and carbonated soft drink consumption were observed to increase the chances of tooth wear by around 50%, whilst an orthodontic anomaly, the presence of calculus or consumption of fruits other than apples or citrus types (OR 0.61; 95% CI 0.45-0.84) appeared to confer a protective effect. High consumption of carbonated soft drink was the principle factor associated with tooth wear, increasing by around 50% for each daily additional intake: carbonated soft drink consumption (yes compared with no) OR 1.46 (95% CI, 1.08 1.97); carbonated soft drink consumption (less than compared with three or more per day) OR 2.16 (95% CI, 1.46 3.18); and carbonated soft drink consumption (less than compared with four or more per day) OR 2.23 (95% CI, 1.41 3.54). All odds ratios were adjusted for all variables, both significant and non-significant.

A4.29. The relationship between a broad collection of food items and the incidence and progression of erosive tooth wear among adolescents was investigated in one study (El Aidi et al., 2011). Incidence was defined as the percentage of subjects without tooth wear at baseline developing tooth wear over the course of the study. Progression was defined as the percentage of subjects with tooth wear at baseline showing an increase in severity score at the final examination. In multivariate analyses significant associations were indicated between the incidence of erosive tooth wear and alcoholic mixed drinks (odds ratio, OR 1.82; 95% CI 1.03-3.23), sour vegetables (OR 1.16; 95% CI 1.02-1.32) and tooth grinding during sleep (OR 4.03; 95% CI 1.09-13.67). The intake of yoghurt products was negatively associated with the incidence of erosive wear (OR 0.79; 95% CI 0.66-0.94). Erosive wear was less likely to progress in subjects who consumed milk and yoghurt products (OR 0.89; 95% CI 0.82-0.97 and 0.76; 95% CI 0.60-0.98, respectively). Yoghurt, despite a low pH, has hardly an erosive effect in vitro due to its high calcium and phosphate content (Lussi et al., 2004). Carbonated soft drink consumption was not associated with either the incidence (OR 1.03; 95% CI .0.99-1.08) or progression (OR 0.97; 95% CI .0.91-1.02) of erosive tooth wear and nor were any of the other acidic drinks investigated.

A4.30. To test whether interaction between factors gave an added or reduced risk, both the incidence and progression model were extended with interactions between the combined acidic products (carbonated soft drink, fruit lemonade, lemonade squash, energy/sports drink, alcoholic mixed drink and sour vegetables) and biological factors. The interaction of acidic products and tooth grinding resulted in a significant extra risk (OR 1.20; 95% CI 1.01-1.42). Overall this study indicates that factors such as tooth grinding during sleep play a large role in tooth wear in the cohort studied. The consumption of carbonated soft drinks in this cohort (mean intake of 8.8 glasses/week) was lower than that indicated in the Dugmore and Rock study where a positive association between carbonated soft drink and tooth wear was found: 40.9% of the 12-year-olds drank three or more glasses per day; by the age of 14 this increased to 45% (Dugmore & Rock, 2004). The prevalence of dental erosion in the cohort studied by Dugmore and Rock was also higher than that indicated in the cohort studied by El Aidi et al.

Randomised controlled trials

A4.31. The findings from all trials investigating the effect of acidic food and drink on tooth wear have been summarised in Table A4.6. As many of the trials provide no variance data only the means for enamel erosion have been tabulated.

A4.32. One trial investigated the effect on bovine enamel dental block demineralisation in response to either water, an non-calorically sweetened phosphoric acid-based cola drink (pH 3.1) or an non-calorically sweetened citric acid-based orange drink (pH 3.6) for 15 minutes four times a day for one week (Rugg-Gunn et al., 1998). The exposure of enamel slabs to the phosphoric acid-based cola drink resulted in a deeper depth of enamel loss, as determined by profiling casts of the of the enamel slabs and scanning electron microscopy, compared with the distilled water control. There was no difference in depth of enamel loss between distilled water and the citric acid-based orange drink treatments. The finding that non-calorically sweetened phosphoric acid-based cola drink was more erosive to enamel than the non-calorically sweetened citric acid-based orange drink suggested that the pH of the drink may have been a factor.

Table A4.6. Results of trials investigating the effect of acidic food and drink on tooth wear

Study	Intervention	Control enamel lesion depth	Intervention enamel lesion depth	Results
Rugg-Gunn et al., 1998	non-calorically sweetened phosphoric acid-based cola drink	depth of loss of enamel 3.8µm	depth of loss of enamel 12.8 µm	The intervention significantly increased enamel surface loss
	non-calorically sweetened citric acid-based orange drink		depth of loss of enamel 5.9µm	No effect
West et al., 1998	orange juice	depth of gain of enamel 0.05 µm	depth of loss of enamel 2.77µm	The intervention significantly increased enamel surface loss
Hughes et al., 1999a	orange juice	depth of loss of enamel 0.19µm	depth of loss of enamel 2.54µm	The intervention significantly increased enamel surface loss
Hughes et al., 1999b	non-calorically sweetened orange drink	depth of loss of enamel 0.08µm	depth of loss of enamel 8.29µm	The intervention significantly increased enamel surface loss
	non-calorically sweetened apple and blackcurrant juice drink		depth of loss of enamel 2.04µm	The intervention significantly increased enamel surface loss
West et al., 1999	orange juice	depth of loss of enamel 0.05µm	depth of loss of enamel 1.70µm	The intervention significantly increased enamel surface loss
	sugars-sweetened blackcurrant juice drink		depth of loss of enamel 2.75µm	The intervention significantly increased enamel surface loss
Hughes et al., 2002	orange juice	depth of loss of enamel 0.18µm	depth of loss of enamel 2.03µm	The intervention significantly increased enamel surface loss
West et al., 2003	non-calorically sweetened carbonated orange drink	depth of loss of enamel 0.11µm	depth of loss of enamel 4.92µm	The intervention significantly increased enamel surface loss
Hooper et al., 2004	sugars-sweetened citric acid-based sports drink	depth of loss of enamel 0.01µm	depth of loss of enamel 3.91µm	The intervention significantly increased enamel surface loss
West et al., 2004	non-calorically sweetened apple and blackcurrant juice drink	NR	mean difference to control of depth of loss of enamel 4.67µm	The intervention significantly increased enamel surface loss
Hooper et al., 2005	sugars-sweetened citric acid-based sports drink	depth of loss of enamel 0.04µm	depth of loss of enamel 4.08µm	The intervention significantly increased enamel surface loss
Venables et al., 2005	sugars-sweetened citric acid-based sports drink	depth of loss of enamel 0.14µm	depth of loss of enamel 4.24µm	The intervention significantly increased enamel surface loss
Hooper et al., 2007	non-calorically sweetened citric acid-based drink	depth of loss of enamel 0.00µm	depth of loss of enamel 6.04µm	The intervention significantly increased enamel surface loss
Caglar et al., 2008	malt-based drink	depth of loss of enamel 0.26µm	depth of loss of enamel 0.59µm	The intervention significantly increased enamel surface loss

NR, not reported.

A4.33. Three trials investigated the effect of one litre a day of orange juice (pH 3.7-3.9) consumed over fifteen days on dental erosion in relation to a water control (West et al., 1998; Hughes et al., 1999a; West et al., 1999). All demonstrated a progressive loss of enamel with time during the orange juice consumption period, but not the water consumption period, as determined by profiling the enamel slabs during the intervention period. After fifteen days the mean enamel loss ranged from 1.70 – 2.77µm following

orange juice consumption, while water consumption had little effect on enamel loss, 0.05 – 0.19µm. One trial also performed a surface microhardness analysis of enamel that had either been exposed or not exposed to the oral environment (PVC tape was used to cover part of the enamel to prevent exposure during the experimental periods) (West et al., 1998). There was no difference in demineralisation between the exposed and unexposed areas after the water consumption period, but following the orange juice consumption period the exposed enamel was significantly different from the unexposed enamel. The exposed-unexposed difference was greater for orange juice than for water (p=0.049).

A4.34. One trial investigated the effect of one litre a day of orange juice (pH 3.3) consumed over ten days on dental erosion in relation to a water control (Hughes et al., 2002). There was a progressive loss of enamel with time during the orange juice consumption period, but not the water consumption period, as determined by profiling the enamel slabs during the intervention period. After fifteen days the mean enamel loss was 2.03µm following orange juice consumption, while water consumption had little effect on enamel loss, 0.18µm. The mean softening depth of the enamel was determined using ultrasonication. Orange juice consumption resulted in a deeper softening depth of the enamel than water consumption.

A4.35. The effect of one litre a day of non-calorically sweetened orange drink (pH 3.0) and an non-calorically sweetened apple and blackcurrant juice drink (pH 3.4) over fifteen days on dental erosion was investigated in relation to a water control (Hughes et al., 1999b). A progressive loss of enamel with time was demonstrated during the orange drink and apple and blackcurrant drink consumption periods, but not the water consumption period, as determined by profiling the enamel slabs during the intervention period. After fifteen days the mean enamel loss was 8.29µm following non-calorically sweetened orange drink and was 2.04µm following non-calorically sweetened apple and blackcurrant juice drink, while water consumption had little effect on enamel loss, 0.08µm.

A4.36. Another trial also investigated the effect of an non-calorically sweetened blackcurrant juice drink (pH 3.6) over fifteen days on dental erosion in relation to a water control (West et al., 2004). A progressive loss of enamel with time was demonstrated during the apple and blackcurrant drink consumption periods, but not the water consumption period, as determined by profiling the enamel slabs during the intervention period. After fifteen days the mean difference in enamel loss between the non-calorically sweetened apple and blackcurrant juice drink and water consumption was 4.67µm ($p<0.001$).

A4.37. The effect of one litre a day of sugars-sweetened blackcurrant juice drink (pH 2.9) over fifteen days on dental erosion was investigated in relation to orange juice and a water control (West et al., 1999). A progressive loss of enamel with time was demonstrated during the blackcurrant juice drink consumption period, but not the water consumption period, as determined by profiling the enamel slabs during the intervention period. After fifteen days the mean enamel loss was 2.75µm following sugars-sweetened blackcurrant juice drink consumption, while water consumption had little effect on enamel loss, 0.05µm.

A4.38. Carbonated drinks are potentially more erosive than non-carbonated drinks due to the additional carbonic acid present. The effect of one litre a day of non-calorically sweetened carbonated orange juice drink (pH 3.1) over twenty days on dental erosion was investigated in relation to a water control (West et al., 2003). A progressive loss of enamel with time was demonstrated during the non-calorically sweetened carbonated orange juice drink consumption period, but not the water consumption period, as determined by profiling the enamel slabs during the intervention period (West et al., 2003). After fifteen days the mean enamel loss was 3.19µm and after twenty days this was 4.92µm following non-calorically sweetened carbonated orange juice drink consumption, while water consumption had little effect on enamel loss, 0.11µm after twenty days.

A4.39. The effect of one litre a day of sugars-sweetened citric acid-based sports drink (pH3.2) over fifteen days on dental erosion was investigated in relation to a water control (Hooper et al., 2004). A progressive loss of enamel with time was demonstrated during the sugars-sweetened citric acid-based sports drink consumption period, but not the water consumption period, as determined by profiling the enamel slabs during the intervention period. After fifteen days the mean enamel loss was 3.91µm following sugars-sweetened citric acid-based sports drink consumption, while water consumption had little effect on enamel loss, mean erosion 0.01µm.

A4.40. The effect of one and a half litres a day of sugars-sweetened citric acid-based sports drink (pH3.2), consumed within one hour, over ten days on dental erosion was investigated in relation to a water control (Hooper et al., 2005). A progressive loss of enamel at five and ten days duration was demonstrated during the sugars-sweetened citric acid-based sports drink consumption period, but not the water consumption period, as determined by profiling the enamel slabs during the intervention period. After ten days the mean enamel loss was 4.08µm following sugars-sweetened citric acid-based sports drink consumption, while water consumption had little effect on enamel loss, mean erosion 0.04µm.

A4.41. The effect of 1.4 litres sugars-sweetened citric acid-based sports drink (pH3.2) consumed within two hours, five days a week during planned exercise on dental erosion was investigated in relation to a water control over twenty one days (Venables et al., 2005).. After twenty one days, fifteen days of exposure, the mean enamel loss, as determined by profiling the enamel slabs, was 4.24µm following sugars-sweetened citric acid-based sports drink consumption, while water consumption had little effect on enamel loss, mean erosion 0.14µm.

A4.42. The effect of one litre a day of non-calorically sweetened citric acid-based drink (pH3.4) over ten days on dental erosion was investigated in relation to a water control (Hooper et al., 2007). A progressive loss of enamel with time was demonstrated during the non-calorically sweetened citric acid-based drink consumption period, but not the water consumption period, as determined by profiling the enamel slabs during the intervention period. After fifteen days the mean enamel loss was 6.04µm following non-calorically sweetened citric acid-based drink consumption, while water consumption had no effect on enamel loss, mean erosion 0.00µm.

A4.43. The effect of 250ml a day of a malt-based drink (pH4.3) over ten days on dental erosion was investigated in relation to a water control. After ten days the mean enamel loss was higher (0.59µm) following non-calorically sweetened citric acid-based drink consumption, while water consumption had less effect on enamel loss, mean erosion 0.26µm.

A4.44. In all trials a large degree of variation in enamel loss was demonstrated, with some subjects showing negligible erosion and others showing large degrees of erosion, e.g. the range of responses in one trial in response to acidic soft drink consumption was 0.07µm to 22.06µm enamel erosion (Hooper et al., 2007).

Summary and conclusions

A4.45. Prospective cohort studies of acidic food and drink consumption and risk of tooth wear provided some evidence that a higher consumption of dietary acids may result in more tooth wear. *In situ* trials demonstrated that consumption of fruit juices (e.g. orange juice and apple and blackcurrant juice) and acidic soft drinks and sports drinks (whether non-calorically sweetened or sugars-sweetened) resulted in a progressive loss of enamel and dentine from dental blocks. No direct comparison between non-calorically sweetened soft drinks and sugars-sweetened soft drinks on dental erosion was conducted in the trials included. Individuals showed a large degree of variation in response to the erosive challenge ranging from almost negligible erosion to large degrees of erosion, reflecting the multi-factorial nature of dental erosion.

Annex 5. Fibre isolates

A5.1. This assessment is based on 43 randomised controlled trials investigating the relationship between dietary fibre isolates and cardio-metabolic health and colo-rectal health outcomes. No randomised trials were conducted in children and adolescents.

A5.2. Evidence on health/disease outcomes have been discussed in detail only where there are sufficient data for a conclusion to be drawn, from studies meeting the pre-agreed inclusion criteria. Outcomes where there are too few such studies to reach a conclusion, are listed at the end of the chapter (see table A5.1).

Psyllium

A5.3. Although psyllium husk is a source of soluble fibre it is unlike anything that occurs in the diet, and is essentially a pharmaceutical preparation (Marlett *et al.*, 2000). It is only present in the diet as a dietary supplement.

Fasting blood lipids

A5.4. Two randomised controlled trials were identified that presented evidence on psyllium supplements in relation to fasting blood lipids (total cholesterol; HDL- and LDL-cholesterol and triacylglycerol concentration) (Bell *et al.*, 1990; Romero *et al.*, 1998). One trial was subsequently identified in the update search (Pal *et al.*, 2011) (Cardio-metabolic review, hyperlipidaemias and blood lipid chapter p 351-359; Update search tables 76-79). Body weights were unchanged in all trials. The effect of psyllium supplements was compared with a placebo.

A5.5. An effect of psyllium supplements on lowering fasting total cholesterol and LDL-cholesterol concentration is reported in all three trials.

A5.6. No significant effect of psyllium supplementation on either fasting HDL-cholesterol or triacylglycerol concentration is reported in any of the trials.

Psyllium and fasting total cholesterol concentration
• Effect • Limited evidence • The direction of the effect demonstrates consumption of psyllium supplements is beneficial to health • The effect is biologically relevant, but only achieved through psyllium supplementation

Psyllium and fasting LDL-cholesterol concentration
• Effect • Limited evidence • The direction of the effect demonstrates consumption of psyllium supplements is beneficial to health • The effect is biologically relevant, but only achieved through psyllium supplementation

Fasting HDL-cholesterol or triacylglycerol concentration
• No effect • Limited evidence

Faecal weight and intestinal transit time

A5.7. Four randomised controlled trials were identified that presented evidence on psyllium supplements in relation to faecal weight and transit time (Spiller *et al.*, 1979; Stevens *et al.*, 1988; Marteau *et al.*, 1994; Vuksan *et al.*, 2008). The transit time methodologies were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health review, p 45, 48-53). The effect of psyllium supplements was compared with a placebo.

A5.8. An effect of psyllium supplements (10-19g/d dietary fibre) on increasing faecal weights is demonstrated all trials. Two trials compare the effects of wheat bran and psyllium on increasing faecal weight: one reports a greater effect using a mixture of psyllium and wheat bran compared with wheat bran alone (Vuksan *et al.*, 2008); the other reports wheat bran and psyllium have similar effects on increasing faecal weight (Stevens *et al.*, 1988). One trial compares psyllium to a cellulose/pectin mixture and reports both equally increase faecal wet weight, but only cellulose/pectin mixture significantly decreases transit time (Spiller *et al.*, 1979). None of the trials report a significant effect of psyllium supplements on reducing intestinal transit time, but all trials show a non-significant tendency to reduce transit time.

Psyllium and faecal weight
• Effect • Limited evidence • The direction of the effect demonstrates consumption of psyllium supplements is potentially beneficial to health • The effect is potentially biologically relevant, but only achieved through psyllium supplementation

Psyllium and intestinal transit time
• No effect • Limited evidence

Constipation

A5.9. Four randomised controlled trials were identified that presented evidence on psyllium supplements in relation to constipation symptoms in outpatients with constipation or patients with self-reported constipation (Fenn *et al.*, 1986; Ashraf *et al.*, 1995; Cheskin *et al.*, 1995; Ashraf *et al.*, 1997). The data were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Colo-rectal health review, p 134,137, 139). The effect of psyllium supplements was compared with a placebo.

A5.10. An effect of psyllium supplements on increasing bowel motion frequency is demonstrated in three trials. The other trial reports a non-significant tendency for psyllium supplements to increase bowel frequency (Cheskin *et al.*, 1995). An effect of psyllium supplements on improving faecal consistency and abdominal pain is reported in the two larger trials (Fenn *et al.*, 1986; Ashraf *et al.*, 1995), but the other two trials report no effect.

Psyllium and frequency of bowel movements
• Effect • Moderate evidence • The direction of the effect demonstrates consumption of psyllium supplements is beneficial to health • The effect is biologically relevant, but only achieved through psyllium supplementation

Psyllium and faecal consistency and abdominal pain
• Effect • Limited evidence • The direction of the effect demonstrates consumption of psyllium supplements is beneficial to health • The effect is biologically relevant, but only achieved through psyllium supplementation

Pectin

Faecal weight and intestinal transit time

A5.11. Three randomised controlled trials were identified that presented evidence on purified pectin in relation to relation to faecal weight and transit times (Spiller *et al.*, 1980; Stasse-Wolthuis *et al.*, 1980; Hillman *et al.*, 1983). The transit time methodologies were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health review, p 34-35, 39, 45, 48-50).

A5.12. No significant effect is demonstrated for purified pectin (5-15g/day) on faecal weight or intestinal transit time.

Pectin and faecal weight and intestinal transit time
• No effect • Limited evidence

Cellulose

Faecal weight and intestinal transit time

A5.13. Three randomised controlled trials were identified that presented evidence on cellulose and in relation to faecal weight and transit times (Spiller *et al.*, 1980; Hillman *et al.*, 1983; Behall *et al.*, 1987). The transit time methodologies were insufficiently comparable to allow a meta-analysis. No further trials were identified in the update search (Colo-rectal health review, p 34-35, 39, 45, 48-50).

A5.14. An effect of cellulose (14-23g/day) is demonstrated on increasing faecal weight in all trials. Two trials report an effect of cellulose on reducing intestinal transit time, but one trial reports no significant effect in subjects with a relatively fast initial intestinal transit time (Behall *et al.*, 1987). The effect sizes are comparable to wheat fibre.

Cellulose and faecal weight and intestinal transit time
• Effect • Moderate evidence • The direction of the effect demonstrates consumption of cellulose is potentially beneficial to health • The effect is potentially biologically relevant, but demonstrated at concentration of intake achieved through supplementation

Mixed isolated fibre interventions

Gums and gelling agents

Blood Pressure

A5.15. Three randomised controlled trials were identified that presented evidence on isolated gums and gelling agents in relation to blood pressure (Bell *et al.*, 1990; Pasman *et al.*, 1997a; Schwab *et al.*, 2006) One trial could not be included in a meta-analysis as it did not report the necessary data, leaving an insufficient number of studies to enable a meta-analysis to be performed (Pasman *et al.*, 1997a) (Cardio-metabolic review, incident hypertension and blood pressure chapter p 88-93). The trials included in the analysis supplemented subjects with either pectin, guar gum, chitosan, or konjac-mannan in comparison with a placebo.

A5.16. No significant effect is demonstrated for various isolated gums and gelling agent supplements on systolic blood pressure (-0.82mmHg, 95% CI -5.2,-3.6; p=0.72).

A5.17. No consistent effect is demonstrated for various isolated gums and gelling agent supplements on diastolic blood pressure on the forest plot, but the heterogeneity is too high to report the meta-analysis pooled estimate (I^2=81%).

A5.18. The two trials that are not included in the meta-analysis report no significant effect of either guar gum or arabino-galactans on blood pressure (Pasman *et al.*, 1997a; Marett & Slavin, 2004)

Isolated gums and gelling agent supplements and systolic or diastolic blood pressure
• No effect • Moderate evidence

Fasting blood lipids

A5.19. Thirteen randomised controlled trials were identified that presented evidence on isolated gums and gelling agents in relation to fasting blood lipids (Bell *et al.*, 1990; Ryle *et al.*, 1990; Landin *et al.*, 1992; Vido *et al.*, 1993; Mee & Gee, 1997; Pasman *et al.*, 1997b; Panlasigui *et al.*, 2003; Marett & Slavin, 2004; Lehtimaki *et al.*, 2005; Schwab *et al.*, 2006; Garcia *et al.*, 2006; Wood *et al.*, 2007; Reppas *et al.*, 2009) Two trials could not be included in meta-analysis (Vido *et al.*, 1993; Pasman *et al.*, 1997b), but demonstrated no significant effect of fibre isolates and gum supplements on fasting blood lipids. One trial only provided data for fasting triacylglycerol concentration (Landin *et al.*, 1992). No further trials were identified in the update search (Cardio-metabolic review, hyperlipidaemia and blood lipids p 364-370. Three of the trials demonstrated differential weight loss between experimental groups (Vido *et al.*, 1993; Schwab *et al.*, 2006; Wood *et al.*, 2007). The trials compared guar gum, pectin, gum arabic, carrageenan, arabinogalactan, chitosan, arabinoxylan, konjac mannan or methylcellulose supplements with a control or placebo.

A5.20. No significant effect is demonstrated for various isolated gums and gelling agent supplements on fasting total cholesterol concentration (-0.15mmol/L, 95% CI -0.32, 0.01; p=0.07). A borderline non-significant effect is demonstrated.

A5.21. No significant effect is demonstrated for various isolated gums and gelling agent supplements on fasting LDL-cholesterol concentration (-0.09mmol/L, 95% CI -0.25, 0.07; p=0.29).

A5.22. No significant effect is demonstrated for various isolated gums and gelling agent supplements on fasting HDL-cholesterol concentration (0.0mmol/L, 95% CI -0.04, 0.05; p=0.89).

A5.23. No significant effect is demonstrated for various isolated gums and gelling agent supplements on fasting triacylglycerol concentration (-0.01mmol/L, 95% CI -0.09, 0.07; p=0.84).

Isolated gums and gelling agent supplements and fasting total cholesterol concentration
• No effect • Limited evidence

Isolated gums and gelling agent supplements and fasting HDL-cholesterol concentration
• No effect • Moderate evidence

Isolated gums and gelling agent supplements and fasting LDL-cholesterol concentration
• No effect • Limited evidence

Isolated gums and gelling agent supplements and fasting triacylglycerol concentration
• No effect • Adequate evidence

Energy intake

A5.24. Eleven randomised controlled trials were identified that presented evidence on isolated gums and gelling agents in relation to energy intake (Tredger *et al.*, 1991; Haskell *et al.*, 1992; Pasman *et al.*, 1997b; Panlasigui *et al.*, 2003; Marett & Slavin, 2004; Schwab *et al.*, 2006; Garcia *et al.*, 2007; Mattes, 2007; Pelkman *et al.*, 2007; Wood *et al.*, 2007; Paxman *et al.*, 2008), seven of which were included in a meta-analysis (Tredger *et al.*, 1991; Pasman *et al.*, 1997b; Schwab *et al.*, 2006; Garcia *et al.*, 2007; Pelkman *et al.*, 2007; Wood *et al.*, 2007; Paxman *et al.*, 2008). Four trials could not be included in meta-analysis but demonstrated no statistically significant effect of soluble fibre isolates on energy intake (Haskell *et al.*, 1992; Panlasigui *et al.*, 2003; Marett & Slavin, 2004; Mattes, 2007). No further trials were identified in the update search (Cardio-metabolic review, energy intake and eating motivation chapter p 144-149). The trials compared guar gum, pectin, gum arabic, carrageenan, algal polysaccharides, arabinogalactan, chitosan or arabinoxylan supplements with a placebo.

A5.25. An effect is demonstrated for various isolated gums and gelling agent supplements on reducing energy intake (-567 kJ, 95% CI -931, -202 kJ; $p<0.01$) (-135 kcal, 95% CI -223, -48 kcal).

Isolated gums and gelling agent supplements and energy intake
• Effect • Adequate evidence • The direction of the effect demonstrates higher consumption of fibre isolates and gum supplements is beneficial to health • The effect is biologically relevant, but demonstrated at intakes achieved through supplementation

Eating motivation

A5.26. Five randomised controlled trials were identified that presented evidence on isolated gums and gelling agents in relation to eating motivation (Pasman *et al.*, 1997b; Heini *et al.*, 1998; Schwab *et al.*, 2006; Mattes, 2007; Pelkman *et al.*, 2007). (Cardio-metabolic review, Energy intake and eating motivation chapter p 150-152). The trials compared either guar gum, pectin or glucomannan with a placebo.

A5.27. Due to variation in study designs, the method of assessing eating motivation and the nature of each intervention, it is not possible to combine these studies using meta-analysis. Overall, the trials provide inconsistent evidence of the effects of various isolated gums and gelling agent supplements on subjective ratings of eating motivation

Isolated gums and gelling agent supplements and eating motivation
• No effect • Limited evidence

Fasting blood glucose

A5.28. Nine randomised controlled trials were identified that presented evidence on isolated gums and gelling agents in relation to fasting blood glucose (Bell *et al.*, 1990; Ryle *et al.*, 1990; Landin *et al.*, 1992; Pasman *et al.*, 1997a; Marett & Slavin, 2004; Lehtimaki *et al.*, 2005; Schwab *et al.*, 2006; Wood *et al.*, 2007; Garcia *et al.*, 2007), eight of which were included in a meta-analysis (Bell *et al.*, 1990; Ryle *et al.*, 1990; Landin *et al.*, 1992; Marett & Slavin, 2004; Lehtimaki *et al.*, 2005; Schwab *et al.*, 2006; Wood *et al.*, 2007; Garcia *et al.*, 2007). One trial could not be included in meta-analysis as it did not report the necessary data, but demonstrated no significant effect of soluble fibre isolates in relation to fasting blood glucose (Pasman *et al.*, 1997a). No further trials were identified in the update search (Cardio-metabolic review, diabetes chapter p 293-298). The trials compared either guar gum, pectin, arabinogalactan, chitosan, arabinoxylan or konjac mannan supplements with a placebo.

A5.29. No significant effect is demonstrated for isolated gums and gelling agent supplements on fasting blood glucose concentration (-0.11mmol/L, 95% CI -0.26, 0.05; p=0.18).

Isolated gums and gelling agent supplements and fasting blood glucose concentration
• No effect • Moderate evidence

Fasting blood insulin

A5.30. Eight publications relating to seven randomised controlled trials were identified that presented evidence fibre isolates and gum supplements in relation to fasting insulin (Ryle *et al.*, 1990; Landin *et al.*, 1992; Pasman *et al.*, 1997a; Marett & Slavin, 2004; Schwab *et al.*, 2006; Garcia *et al.*, 2006; Wood *et al.*, 2007; Garcia *et al.*, 2007). Two papers presented evidence from one trial (Garcia *et al.*, 2006; Garcia *et al.*, 2007). No further trials were identified in the update search (Cardio-metabolic review, diabetes chapter p 299-302). The trials compared either guar gum, pectin, arabinogalactan, chitosan, arabinoxylan or konjac mannan supplements with a placebo.

A5.31. Due to variation in the methodologies used to measure insulin concentration, it is not possible to combine these studies using meta-analysis. Overall these trials do not provide evidence of an effect of various isolated gums and gelling agent supplements on fasting blood insulin concentration.

Isolated gums and gelling agent supplements and fasting blood insulin concentration
• No effect • Moderate evidence

Mixed gums and gelling agents

Fasting blood lipids

A5.32. Four randomised controlled trials were identified that presented evidence on mixed isolated gums and gelling agents and fasting blood lipid concentrations, all of which were included in a meta-analysis (Haskell *et al.*, 1992; Jensen *et al.*, 1997; Knopp *et al.*, 1999; Salas-Salvado *et al.*, 2008) No further trials were identified in the update search (Cardio-metabolic review, hyperlipidaemias and blood lipids chapter p 316-330). One trial was a weight loss trial (Salas-Salvado *et al.*, 2008). The trials compared either acacia gum, a mixture of psyllium, pectin, guar gum, and locust bean gum, guar gum and pectin, or psyllium and glucomannan with a placebo.

A5.33. An effect is demonstrated for mixed isolated gums and gelling agent supplements and on reducing fasting total cholesterol concentration (-0.36mmol/L, 95% CI -0.50, -0.23; $p<0.001$).

A5.34. No significant effect is demonstrated for mixed isolated gums and gelling agent supplements on fasting HDL-cholesterol concentration (-0.04mmol/L, 95% CI -0.11, 0.04; $p=0.36$).

A5.35. An effect is demonstrated for mixed isolated gums and gelling agent supplements on reducing fasting LDL-concentration (-0.29mmol/L, 95% CI -0.45, -0.12; $p=0.001$),

A5.36. No significant effect is demonstrated for mixed isolated gums and gelling agent supplements on fasting triacylglycerol concentration (0.00mmol/L, 95% CI -0.18, 0.18; $p=1.0$).

Mixed isolated gums and gelling agents and fasting total cholesterol concentration
• Effect • Moderate evidence • The direction of the effect demonstrates higher consumption of soluble fibre isolates and gum supplements are beneficial to health • The effect is biologically relevant, but only demonstrated at intakes achieved through supplementation

Mixed isolated gums and gelling agents and fasting HDL-cholesterol concentration
• No effect • Moderate evidence

Mixed isolated gums and gelling agents and fasting HDL-cholesterol concentration
• Effect • Moderate evidence • The direction of the effect demonstrates higher consumption of soluble fibre isolates and gum supplements are beneficial to health • The effect is biologically relevant, but only demonstrated at intakes achieved through supplementation

Mixed isolated gums and gelling agents and fasting triacylglycerol concentration
• No effect • Moderate evidence

Markers of inflammation C-reactive protein

A5.37. Three randomised controlled trials were identified that presented evidence on mixed isolated gums and gelling agents in relation to C-reactive protein concentration (Wood *et al.*, 2006; Salas-Salvado *et al.*, 2008; King *et al.*, 2008). No further trials were identified in the update search (Cardio-metabolic review, inflammation chapter p 39-43). One trial was a weight loss trial (Salas-Salvado *et al.*, 2008). The trials compared either psyllium, konjac mannan or psyllium and glucomannan with a placebo.

A5.38. No significant effect of mixed isolated gums and gelling agent supplements on C-reactive protein concentration is reported in any of the trials.

Mixed isolated gums and gelling agents and C-reactive protein concentration
• No effect • Limited evidence

Mixed dietary fibre isolates

Energy intake

A5.39. Three randomised controlled trials were identified that presented evidence on mixed soluble and insoluble fibre isolates in relation to energy intake, all of which were included in a meta-analysis (Rigaud *et al.*, 1990; Hunninghake *et al.*, 1994; Jenkins *et al.*, 1999b). No further trials were identified in the update search (Cardio-metabolic review, energy intake and eating motivation chapter p 133-135). The trials compared either cereal and citrus fibre, a mixture of guar gum, pectin, soy, corn bran, pea fibre or coarse bran wheat fibre breakfast cereal with either a control or placebo.

A5.40. No significant effect is demonstrated for mixed soluble and insoluble fibre isolates on energy intake (-405kJ, 95% CI -1017, 207 kJ; p=0.20).

Mixed dietary fibre isolates and energy intake
• No effect • Limited evidence

Outcomes where there is insufficient evidence

A5.41. The table below details the exposures and outcomes where there are too few studies or trials that meet the inclusion criteria for the review (e.g. by study duration, publication date, health of subjects, design of intervention) to draw a conclusion. A full description of the studies can be found in the relevant systematic reviews.

Table A5.1 Insufficient evidence-randomised controlled trials

Risk factor/health outcome/measure	**Exposure**
Blood pressure	psyllium an isolated fibres
Total cholesterol (Hyperlipidaemias and blood lipids chapter)	isolated fibres
HDL-cholesterol (Hyperlipidaemias and blood lipids chapter)	isolated fibres
LDL-cholesterol (Hyperlipidaemias and blood lipids chapter)	isolated fibres
Fasting triacylglycerol (Hyperlipidaemias and blood lipids chapter)	isolated fibres
LDL:HDL cholesterol Ratio (Hyperlipidaemias and blood lipids chapter)	psyllium and isolated fibres
Total cholesterol:HDL-cholesterol ratio (Hyperlipidaemias and blood lipids chapter)	psyllium and isolated fibres
Apolipoproteins (Hyperlipidaemias and blood lipids chapter)	isolated fibres
TNF- α, IL-6, Fibrinogen, plasminogen (Markers of Inflammation chapter)	isolated fibres

Change in body weight and BMI (Obesity chapter)	guar gum
Body fatness and fat distribution (Obesity chapter)	guar gum
Energy intake in adults (Energy Intake and Eating Motivation chapter)	psyllium
Energy intake (Energy Intake and Eating Motivation chapter)	psyllium
Eating motivation (Energy Intake and Eating Motivation chapter)	isolated fibres
Fasting glycaemia (Diabetes chapter)	psyllium and isolated fibres
Insulin resistance/sensitivity (Diabetes chapter)	isolated fibres
Insulinemia (Diabetes chapter)	isolated fibres
Glycosylated blood proteins (Diabetes chapter)	isolated fibres
Intestinal transit time (Colo-rectal health review)	isolated fibres
Faecal weight (Colo-rectal health review)	isolated fibres
Faecal bacteria populations (Colo-rectal health review)	isolated fibres
Faecal pH and short chain fatty acid content (Colo-rectal health review)	isolated fibres
Symptoms of constipation (Colo-rectal health review)	isolated fibres
Intestinal transit times in people with constipation (Colo-rectal health review)	psyllium
Irritable bowel syndrome (Colo-rectal health review)	fibre supplements

Table A5.2 Inconsistent evidence

Risk factor	**Exposure**
Diastolic blood pressure (Blood pressure chapter)	isolated fibres

Summary and conclusions

A5.42. This assessment is based on 43 randomised controlled trials investigating the relationship between dietary fibre isolates and cardio-metabolic health and colo-rectal health outcomes. No randomised trials were conducted in children and adolescents.

A5.43. Trials supplementing subjects with psyllium indicate beneficial effects on fasting blood lipid concentrations, faecal weight and the treatment of constipation. Although psyllium husk is a source of soluble fibre it is unlike anything that occurs in the diet, and is essentially a pharmaceutical preparation. The major types of soluble fibre derived from food are pectin or oat β-glucan, but trials with psyllium are not indicative of the possible efficacy of these types of soluble fibre. The peculiar property of psyllium, which may make it particularly effective as a laxative, is the presence of a poorly fermentable arabinoxylan fraction that stays intact all the way through the colon (Marlett *et al.*, 2000).

A5.44. Trials indicate extracted cellulose decreases intestinal transit times and increase faecal mass, although extracted pectin has no effect.

A5.45. Trials in subjects receiving supplements of mixed fibre isolates indicate these components can have beneficial effects on fasting blood lipid concentrations and decrease energy intake. The evidence for any specific extracted/isolated fibre, however, needs to be based on cause-effect evidence, and that requires clear specification of exposures. The evidence from mixed fibre interventions, therefore, contributes to a view on the potential for some specific combinations of these materials to meet the dietary fibre definition, but it is not possible to draw conclusions on the specific components.

Annex 6. Glossary of terms

To be completed.

Annex 7 – Dietary sources and intakes of carbohydrates

Table 3.1

Average daily intake of total carbohydrate, by age and sex

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Total carbohydrate	**Sex and age group (years)**												
	Boys		**Men**		**Girls**		**Women**		**Total**				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	219	265	251	231	205	211	197	187	151	212	239	224	206
Median	218	259	243	223	203	209	193	187	150	209	233	216	199
Sd	48	73	76	75	45	59	61	50	37	47	72	74	66
Upper 2.5 percentile	318	449	407	389	305	335	336	303	228	308	398	389	362
Lower 2.5 percentile	136	150	122	113	132	103	92	99	79	134	115	101	107
% food and drink energy (excluding energy from alcohol)													
Mean	52.2	50.9	47.8	46.9	52.0	51.1	48.2	47.5	50.6	52.1	51.0	48.0	47.2
Median	52.1	50.8	47.6	47.3	52.2	51.0	48.2	47.4	50.6	52.1	50.9	47.8	47.4
sd	4.8	5.5	6.8	6.3	5.1	5.6	7.2	6.5	5.7	4.9	5.5	7.0	6.4
Upper 2.5 percentile	62.0	62.0	61.3	58.3	62.5	61.1	61.7	59.6	61.6	62.3	61.4	61.4	59.5
Lower 2.5 percentile	43.1	40.3	35.4	34.0	43.0	39.7	33.8	33.9	40.4	43.0	39.9	34.5	33.9
% total energy (including energy from alcohol)													
Mean	52.2	50.5	45.1	44.9	52.0	50.7	46.3	46.5	50.6	52.1	50.6	45.7	45.8
Median	52.1	50.4	44.9	45.6	52.2	50.8	46.4	46.2	50.6	52.1	50.6	45.8	46.2
sd	4.8	5.7	7.7	7.2	5.1	5.8	7.7	6.9	5.7	4.9	5.7	7.7	7.1
Upper 2.5 percentile	62.0	61.7	60.7	57.5	62.5	61.0	61.4	59.6	61.6	62.3	61.3	61.1	59.4
Lower 2.5 percentile	43.1	39.3	30.7	29.3	43.0	39.2	30.9	32.3	40.4	43.0	39.3	30.8	29.8
Number of participants (unweighted)	*665*	*744*	*1126*	*317*	*612*	*753*	*1571*	*436*	*604*	*1277*	*1497*	*2697*	*753*

Table 3.2

Average daily intake of starch by age and sex

Aged 1.5 years and over — *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Starch	Sex and age group (years)												
	Boys		**Men**		**Girls**		**Women**		**Total**				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	118.6	149.3	145.5	128.9	110.8	120.7	112.6	98.3	75.9	114.8	135.4	129.0	111.8
Median	117.9	145.7	143.9	127.1	108.2	122.6	111.3	95.4	73.6	112.5	131.8	125.5	106.4
sd	28.8	41.3	44.3	45.5	26.6	35.6	36.2	28.7	22.0	28.0	41.2	43.7	40.0
Upper 2.5 percentile	178.2	236.6	245.6	218.5	170.7	185.1	185.2	150.4	122.3	174.4	228.0	228.5	195.1
Lower 2.5 percentile	65.8	76.4	66.7	56.0	61.0	48.1	48.4	41.0	35.7	64.6	58.7	52.1	48.3
% food and drink energy (excluding energy from alcohol)													
Mean	28.4	28.9	27.9	26.3	28.3	29.2	27.7	25.2	25.5	28.3	29.1	27.8	25.7
Median	28.3	28.7	28.0	26.6	28.0	29.3	27.6	25.2	25.5	28.1	29.0	27.8	25.6
sd	4.6	4.9	5.9	5.7	5.1	5.5	6.2	5.9	5.6	4.8	5.2	6.0	5.8
Upper 2.5 percentile	38.3	39.0	40.2	38.1	38.3	39.1	41.4	38.5	36.9	38.3	39.1	40.6	38.1
Lower 2.5 percentile	19.4	19.4	16.9	14.6	18.9	18.6	15.7	14.1	15.2	19.2	19.2	16.1	14.6
% total energy (including energy from alcohol)													
Mean	28.4	28.7	26.4	25.1	28.3	29.1	26.7	24.7	25.5	28.3	28.9	26.5	24.9
Median	28.3	28.5	26.4	25.2	28.0	29.1	26.3	24.4	25.5	28.1	28.8	26.3	24.8
sd	4.6	5.1	6.3	5.9	5.1	5.6	6.4	6.0	5.6	4.8	5.3	6.4	6.0
Upper 2.5 percentile	38.3	39.0	39.7	37.1	38.3	39.1	41.4	38.5	36.9	38.3	39.0	40.2	37.1
Lower 2.5 percentile	19.4	19.1	14.0	13.2	18.9	17.9	14.5	13.6	15.2	19.2	18.6	14.3	13.2
Number of participants (unweighted)	*665*	*744*	*1126*	*317*	*612*	*753*	*1571*	*436*	*604*	*1277*	*1497*	*2697*	*753*

Table 3.3

Average daily intake of total sugars by age and sex

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Total sugars	**Sex and age group (years)**												
	Boys		**Men**		**Girls**		**Women**		**Total**				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	100.2	115.7	105.6	102.4	94.6	90.4	84.6	88.4	75.6	97.4	103.4	95.1	94.5
Median	97.7	108.2	100.8	97.6	91.5	85.0	78.2	83.2	73.8	94.7	96.5	87.6	88.2
sd	32.7	47.7	48.3	46.6	30.6	37.6	39.4	34.8	25.2	31.8	44.8	45.3	41.0
Upper 2.5 percentile	168.4	245.8	221.6	203.9	153.7	173.4	176.1	175.8	129.2	164.5	216.9	204.2	196.2
Lower 2.5 percentile	45.6	43.8	32.0	32.4	41.0	29.5	26.6	32.3	30.5	43.8	35.0	30.6	32.4
% food and drink energy (excluding energy from alcohol)													
Mean	23.8	22.0	19.8	20.6	23.8	21.8	20.5	22.3	25.1	23.8	21.9	20.2	21.6
Median	23.6	21.6	19.2	20.3	23.7	21.1	19.9	21.9	24.6	23.6	21.3	19.5	21.2
sd	5.8	6.4	6.6	6.6	5.4	6.9	6.8	6.3	5.9	5.6	6.7	6.7	6.5
Upper 2.5 percentile	36.2	36.8	35.5	34.3	35.2	37.9	35.3	36.5	38.2	35.4	37.1	35.5	36.3
Lower 2.5 percentile	13.5	11.1	9.3	8.5	13.7	9.7	9.1	10.1	14.1	13.5	9.9	9.1	9.8
% total energy (including energy from alcohol)													
Mean	23.8	21.8	18.7	19.7	23.8	21.6	19.6	21.8	25.1	23.8	21.7	19.1	20.9
Median	23.6	21.5	18.1	18.8	23.7	20.9	19.0	21.2	24.6	23.6	21.1	18.5	20.5
sd	5.8	6.3	6.3	6.5	5.4	6.8	6.6	6.2	5.9	5.6	6.6	6.5	6.4
Upper 2.5 percentile	36.2	36.2	33.8	34.3	35.2	36.8	34.8	35.0	38.2	35.4	36.8	34.4	35.0
Lower 2.5 percentile	13.5	11.0	8.8	8.1	13.7	9.7	8.7	9.9	14.1	13.5	9.9	8.7	9.2
Number of participants (unweighted)	*665*	*744*	*1126*	*317*	*612*	*753*	*1571*	*436*	*604*	*1277*	*1497*	*2697*	*753*

Table 3.4

Average daily intake of intrinsic and milk sugars by age and sex

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Intrinsic and milk sugars	Sex and age group (years)												
	Boys		Men		Girls		Women		Total				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	37.2	31.7	37.2	43.8	36.1	26.6	35.3	42.2	39.5	36.7	29.2	36.3	42.9
Median	34.7	28.2	33.4	42.9	33.6	24.1	32.6	40.0	38.0	34.0	26.2	32.9	41.1
sd	14.7	16.6	20.0	20.3	15.3	13.3	17.6	18.5	15.6	15.0	15.3	18.8	19.3
Upper 2.5 percentile	72.2	71.4	84.3	91.9	71.0	58.7	72.4	81.3	73.9	71.0	68.0	78.7	87.5
Lower 2.5 percentile	13.5	9.7	10.7	13.3	12.3	9.2	11.1	15.0	11.6	13.2	9.6	10.8	13.6
% food and drink energy (excluding energy from alcohol)													
Mean	9.0	6.1	7.2	9.0	9.2	6.6	8.8	10.9	13.2	9.1	6.3	8.0	10.1
Median	8.5	5.6	6.4	8.4	8.6	5.8	8.0	10.2	13.2	8.6	5.7	7.3	9.4
sd	3.4	2.7	3.7	3.8	3.8	3.5	4.2	4.2	4.6	3.6	3.1	4.1	4.1
Upper 2.5 percentile	16.5	12.4	16.5	18.5	18.7	16.2	18.7	21.4	22.5	17.4	13.9	17.6	20.5
Lower 2.5 percentile	3.9	2.3	2.6	3.6	3.5	2.5	3.3	4.5	5.2	3.6	2.5	2.8	3.8
% total energy (including energy from alcohol)													
Mean	9.0	6.0	6.8	8.7	9.2	6.6	8.5	10.6	13.2	9.1	6.3	7.7	9.7
Median	8.5	5.5	6.1	8.3	8.6	5.8	7.8	10.0	13.2	8.6	5.7	6.9	9.2
sd	3.4	2.7	3.7	3.7	3.8	3.5	4.2	4.1	4.6	3.6	3.1	4.0	4.1
Upper 2.5 percentile	16.5	12.4	16.2	17.8	18.7	16.2	18.5	20.5	22.5	17.4	13.9	17.1	19.2
Lower 2.5 percentile	3.9	2.2	2.3	3.5	3.5	2.5	2.9	4.1	5.2	3.6	2.3	2.6	3.6
Number of participants (unweighted)	*665*	*744*	*1126*	*317*	*612*	*753*	*1571*	*436*	*604*	*1277*	*1497*	*2697*	*753*

Table 3.5

Average daily intake of non-milk extrinsic sugars by age and sex

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Non-milk extrinsic sugars (NMES)	**Sex and age group (years)**												
	Boys		**Men**		**Girls**		**Women**		**Total**				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	63.0	84.0	68.4	58.5	58.5	63.9	49.2	46.2	36.1	60.8	74.2	58.8	51.6
Median	58.5	77.9	62.2	53.1	54.8	59.3	41.6	39.8	32.9	57.0	66.8	49.2	42.9
sd	29.2	42.5	41.8	40.3	25.2	33.9	35.0	25.5	19.3	27.4	39.8	39.7	33.4
Upper 2.5 percentile	128.6	189.3	169.1	141.7	114.1	142.1	130.9	117.4	81.9	122.2	165.1	157.9	137.7
Lower 2.5 percentile	19.5	18.4	10.8	8.5	18.5	11.4	5.9	7.5	7.8	19.3	14.9	8.2	8.5
% food and drink energy (excluding energy from alcohol)													
Mean	14.8	16.0	12.7	11.6	14.6	15.2	11.6	11.5	11.9	14.7	15.6	12.1	11.5
Median	14.3	15.2	11.6	10.9	14.1	14.4	10.8	10.8	11.2	14.2	14.9	11.2	10.9
sd	5.6	6.6	6.4	6.3	5.1	6.4	6.6	5.1	5.4	5.3	6.5	6.5	5.7
Upper 2.5 percentile	27.9	32.4	27.6	24.3	25.4	29.8	25.8	23.6	25.0	26.3	31.1	26.3	24.3
Lower 2.5 percentile	5.8	4.5	2.8	1.9	5.6	3.6	2.0	2.4	3.6	5.8	4.1	2.4	2.1
% total energy (including energy from alcohol)													
Mean	14.8	15.8	11.9	11.1	14.6	15.0	11.1	11.2	11.9	14.7	15.4	11.5	11.2
Median	14.3	15.2	11.0	10.6	14.1	14.3	10.1	10.7	11.2	14.2	14.7	10.6	10.7
sd	5.6	6.4	6.0	6.2	5.1	6.3	6.3	5.1	5.4	5.3	6.4	6.1	5.6
Upper 2.5 percentile	27.9	31.2	25.0	23.8	25.4	29.0	25.1	23.4	25.0	26.3	29.6	25.0	23.7
Lower 2.5 percentile	5.8	4.5	2.7	1.8	5.6	3.6	1.8	2.4	3.6	5.8	4.1	2.2	1.8
Number of participants (unweighted)	*665*	*744*	*1126*	*317*	*612*	*753*	*1571*	*436*	*604*	*1277*	*1497*	*2697*	*753*

Table 3.6

Average daily intake of intrinsic and milk sugars and starch by age and sex

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Intrinsic and milk sugars and starch	**Sex and age group (years)**												
	Boys		**Men**		**Girls**		**Women**		**Total**				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
% food and drink energy (excluding energy from alcohol)													
Mean	37.4	35.0	35.1	35.3	37.4	35.9	36.6	36.1	38.7	37.4	35.4	35.8	35.7
Median	37.4	35.1	34.5	34.9	37.3	35.8	36.1	35.4	38.2	37.3	35.3	35.3	35.2
sd	5.0	5.4	6.8	6.6	5.6	5.9	7.0	6.5	5.4	5.3	5.7	7.0	6.5
Upper 2.5 percentile	47.6	45.3	50.0	46.2	48.2	48.2	51.9	50.3	50.4	48.2	47.2	50.4	49.6
Lower 2.5 percentile	28.1	24.1	23.2	22.9	27.7	25.0	23.9	22.2	28.0	27.7	24.4	23.6	22.2
% total energy (including energy from alcohol)													
Mean	37.4	34.7	33.3	33.8	37.4	35.7	35.2	35.3	38.7	37.4	35.2	34.2	34.6
Median	37.4	34.8	32.9	33.8	37.3	35.6	34.7	34.9	38.2	37.3	35.2	33.8	34.6
sd	5.0	5.7	7.5	7.0	5.6	6.1	7.5	6.7	5.4	5.3	5.9	7.6	6.9
Upper 2.5 percentile	47.6	45.3	49.8	46.2	48.2	48.2	51.9	49.8	50.4	48.2	47.2	50.3	48.9
Lower 2.5 percentile	28.1	23.0	19.8	19.4	27.7	24.5	21.1	21.9	28.0	27.7	23.5	20.4	20.4
Number of participants (unweighted)	*665*	*744*	*1126*	*317*	*612*	*753*	*1571*	*436*	*604*	*1277*	*1497*	*2697*	*753*

Table 3.7

Average daily intake of glucose by age and sex

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-3 combined (2008/09 - 2010/11)*

Glucose	**Sex and age group (years)**												
	Boys		**Men**		**Girls**		**Women**		**Total**				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	15.3	20.1	20.0	17.0	14.5	15.9	15.2	14.8	11.1	14.9	18.1	17.6	15.7
Median	14.5	18.8	17.5	15.2	14.1	13.6	13.4	13.4	10.2	14.3	16.3	15.4	14.2
sd	6.9	10.6	12.8	9.9	6.2	9.9	9.2	8.5	5.9	6.6	10.5	11.4	9.2
Upper 2.5 percentile	31.9	48.9	57.5	48.1	27.0	38.6	39.4	39.3	27.1	30.7	43.8	52.2	40.8
Lower 2.5 percentile	5.3	4.3	4.4	3.3	4.2	3.2	2.7	2.8	2.5	4.8	3.5	3.6	3.0
% food and drink energy (excluding energy from alcohol)													
Mean	3.6	3.9	3.7	3.5	3.6	3.7	3.7	3.8	3.6	3.6	3.8	3.7	3.6
Median	3.4	3.6	3.4	3.3	3.4	3.5	3.3	3.4	3.5	3.4	3.5	3.3	3.3
sd	1.5	1.8	2.2	1.7	1.4	2.0	1.9	1.9	1.7	1.4	1.9	2.0	1.8
Upper 2.5 percentile	7.2	8.7	9.6	8.4	6.6	8.9	8.5	8.5	8.0	6.8	8.7	8.8	8.3
Lower 2.5 percentile	1.4	1.1	0.9	0.9	1.3	1.1	0.9	0.8	1.1	1.3	1.1	0.9	0.9
% total energy (including energy from alcohol)													
Mean	3.6	3.8	3.4	3.3	3.6	3.7	3.5	3.7	3.6	3.6	3.7	3.5	3.5
Median	3.4	3.6	3.1	3.0	3.4	3.4	3.2	3.3	3.5	3.4	3.5	3.2	3.2
sd	1.5	1.7	1.9	1.7	1.4	1.9	1.8	1.8	1.7	1.4	1.8	1.8	1.8
Upper 2.5 percentile	7.2	8.1	8.1	8.2	6.6	8.6	7.4	8.4	8.0	6.8	8.2	7.8	8.2
Lower 2.5 percentile	1.4	1.1	0.9	0.8	1.3	1.1	0.9	0.8	1.1	1.3	1.1	0.9	0.8
Number of participants (unweighted)	*311*	*342*	*519*	*131*	*302*	*324*	*667*	*174*	*303*	*613*	*666*	*1186*	*305*

Table 3.8

Average daily intake of fructose by age and sex

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-3 combined (2008/09 - 2010/11)*

Fructose

	Sex and age group (years)												
	Boys		**Men**		**Girls**		**Women**		**Total**				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	17.2	19.0	17.5	16.8	15.7	15.9	15.5	15.0	12.8	16.5	17.5	16.5	15.8
Median	15.9	16.8	15.6	14.7	14.6	14.2	14.0	12.9	11.3	15.1	15.9	14.6	13.5
sd	9.0	10.4	11.5	10.5	7.8	9.6	9.7	9.5	7.9	8.5	10.1	10.7	9.9
Upper 2.5 percentile	41.1	46.1	47.9	50.8	35.8	39.1	39.9	44.3	32.9	36.8	42.3	43.0	44.3
Lower 2.5 percentile	4.9	3.7	3.2	2.4	3.1	2.9	1.9	2.4	2.4	4.0	2.9	2.7	2.4
% food and drink energy (excluding energy from alcohol)													
Mean	4.1	3.6	3.3	3.4	3.9	3.8	3.8	3.8	4.2	4.0	3.7	3.5	3.6
Median	3.7	3.4	2.8	3.2	3.5	3.4	3.4	3.3	3.8	3.6	3.4	3.1	3.2
sd	2.1	1.8	2.1	1.9	1.9	2.1	2.1	2.2	2.3	2.0	1.9	2.1	2.1
Upper 2.5 percentile	10.1	7.8	8.2	8.2	9.0	8.8	9.0	10.2	10.5	9.2	8.7	8.6	9.3
Lower 2.5 percentile	1.2	0.9	0.8	0.6	0.9	0.8	0.6	0.8	1.1	1.1	0.9	0.7	0.7
% total energy (including energy from alcohol)													
Mean	4.1	3.6	3.1	3.3	3.9	3.7	3.6	3.7	4.2	4.0	3.7	3.4	3.5
Median	3.7	3.4	2.6	3.0	3.5	3.4	3.2	3.1	3.8	3.6	3.4	2.9	3.1
sd	2.1	1.8	2.1	1.8	1.9	2.0	2.0	2.1	2.3	2.0	1.9	2.0	2.0
Upper 2.5 percentile	10.1	7.2	7.8	7.7	9.0	8.8	8.1	9.8	10.5	9.2	8.1	8.0	8.9
Lower 2.5 percentile	1.2	0.9	0.7	0.5	0.9	0.8	0.6	0.8	1.1	1.1	0.9	0.7	0.7
Number of participants (unweighted)	*311*	*342*	*519*	*131*	*302*	*324*	*667*	*174*	*303*	*613*	*666*	*1186*	*305*

Table 3.9

Average daily intake of sucrose by age and sex

Aged 1.5 years and over — *National Diet and Nutrition Survey years 1-3 combined (2008/09 - 2010/11)*

Sucrose	**Sex and age group (years)**												
	Boys		**Men**		**Girls**		**Women**		**Total**				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	46.1	54.2	46.4	42.8	47.5	45.4	39.2	37.3	30.1	46.8	49.9	42.8	39.7
Median	45.0	49.1	42.7	38.1	43.6	42.6	35.1	35.8	28.5	44.6	45.5	38.2	36.3
sd	17.8	25.7	27.3	24.2	18.3	22.3	24.3	19.4	13.8	18.0	24.5	26.1	21.8
Upper 2.5 percentile	85.8	124.0	114.3	110.7	89.9	101.7	105.6	96.8	68.1	87.5	110.6	109.1	100.5
Lower 2.5 percentile	17.1	14.4	8.7	7.9	17.8	11.6	5.6	6.0	9.4	17.6	13.2	7.2	7.9
% food and drink energy (excluding energy from alcohol)													
Mean	10.8	10.4	8.6	8.7	11.6	10.6	9.4	9.3	9.8	11.2	10.5	9.0	9.1
Median	10.5	9.9	8.0	8.1	11.3	10.1	9.0	9.5	9.5	10.9	10.1	8.4	8.9
sd	3.3	4.0	4.1	4.4	3.3	4.0	4.6	3.9	3.6	3.3	4.0	4.4	4.1
Upper 2.5 percentile	17.9	19.7	18.9	20.7	18.7	19.2	20.1	18.8	18.0	18.5	19.7	19.4	19.8
Lower 2.5 percentile	4.7	3.5	2.4	2.0	5.6	3.7	2.1	1.8	4.1	5.2	3.6	2.3	2.0
% total energy (including energy from alcohol)													
Mean	10.8	10.3	8.1	8.4	11.6	10.5	9.0	9.1	9.8	11.2	10.4	8.5	8.8
Median	10.5	9.8	7.6	7.9	11.3	10.1	8.6	9.2	9.5	10.9	9.9	8.0	8.7
sd	3.3	3.9	4.1	4.4	3.3	4.0	4.5	3.8	3.6	3.3	3.9	4.3	4.1
Upper 2.5 percentile	17.9	19.7	18.9	19.9	18.7	19.1	19.4	18.4	18.0	18.5	19.2	18.9	18.9
Lower 2.5 percentile	4.7	3.5	2.1	1.6	5.6	3.7	2.0	1.8	4.1	5.2	3.6	2.1	1.8
Number of participants (unweighted)	*311*	*342*	*519*	*131*	*302*	*324*	*667*	*174*	*303*	*613*	*666*	*1186*	*305*

Table 3.10

Average daily intake of maltose by age and sex

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-3 combined (2008/09 - 2010/11)*

Maltose	**Sex and age group (years)**												
	Boys		**Men**		**Girls**		**Women**		**Total**				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	4.1	5.9	10.5	8.6	3.6	3.9	4.2	3.6	2.5	3.9	4.9	7.3	5.8
Median	3.9	4.7	6.7	5.3	3.4	3.5	3.4	3.2	2.3	3.5	4.0	4.4	3.8
sd	1.9	5.2	11.2	9.4	1.6	2.3	3.7	2.4	1.3	1.8	4.2	8.9	6.9
Upper 2.5 percentile	8.3	18.8	42.8	35.2	7.8	10.1	13.7	9.7	5.6	8.0	14.6	32.3	26.8
Lower 2.5 percentile	1.4	1.3	1.0	1.6	1.1	1.0	0.6	0.7	0.5	1.3	1.2	0.7	0.8
% food and drink energy (excluding energy from alcohol)													
Mean	1.0	1.1	1.9	1.8	0.9	0.9	1.0	0.9	0.8	0.9	1.0	1.5	1.3
Median	0.9	0.9	1.2	1.2	0.8	0.8	0.8	0.8	0.8	0.9	0.9	1.0	0.9
sd	0.4	1.0	2.0	1.9	0.3	0.5	1.0	0.6	0.4	0.4	0.8	1.6	1.4
Upper 2.5 percentile	1.8	3.3	8.4	7.9	1.7	2.2	3.3	2.6	1.9	1.8	2.7	5.6	6.1
Lower 2.5 percentile	0.4	0.3	0.3	0.3	0.3	0.3	0.2	0.2	0.2	0.4	0.3	0.2	0.2
% total energy (including energy from alcohol)													
Mean	1.0	1.1	1.7	1.7	0.9	0.9	1.0	0.9	0.8	0.9	1.0	1.3	1.2
Median	0.9	0.9	1.2	1.1	0.8	0.8	0.8	0.8	0.8	0.9	0.9	0.9	0.9
sd	0.4	0.8	1.4	1.5	0.3	0.4	0.8	0.6	0.4	0.4	0.6	1.2	1.2
Upper 2.5 percentile	1.8	3.0	5.3	6.4	1.7	2.0	3.0	2.6	1.9	1.8	2.6	4.6	5.1
Lower 2.5 percentile	0.4	0.3	0.3	0.3	0.3	0.3	0.2	0.2	0.2	0.4	0.3	0.2	0.2
Number of participants (unweighted)	*311*	*342*	*519*	*131*	*302*	*324*	*667*	*174*	*303*	*613*	*666*	*1186*	*305*

Table 3.11

Average daily intake of lactose by age and sex

Aged 1.5 years and over — *National Diet and Nutrition Survey years 1-3 combined (2008/09 - 2010/11)*

Lactose	**Sex and age group (years)**												
	Boys		**Men**		**Girls**		**Women**		**Total**				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	14.4	11.5	10.9	13.5	13.0	9.4	9.5	12.7	17.9	13.7	10.4	10.2	13.0
Median	13.2	8.8	8.6	11.3	11.7	7.8	8.3	11.6	16.2	12.5	8.3	8.4	11.4
sd	8.1	8.7	8.2	9.2	7.5	7.1	6.5	7.0	10.9	7.8	8.0	7.4	8.0
Upper 2.5 percentile	35.8	35.0	33.6	44.0	30.6	33.5	25.6	28.9	41.6	30.9	34.6	29.5	35.4
Lower 2.5 percentile	2.0	0.6	0.9	1.9	2.6	1.1	1.1	2.3	1.6	2.1	1.1	1.1	2.1
% food and drink energy (excluding energy from alcohol)													
Mean	3.4	2.2	2.0	2.8	3.2	2.3	2.4	3.3	6.0	3.3	2.2	2.2	3.0
Median	3.3	1.8	1.8	2.4	3.0	1.8	2.1	3.0	5.7	3.1	1.8	1.9	2.7
sd	1.8	1.5	1.4	1.6	1.7	1.6	1.7	1.7	3.4	1.8	1.6	1.6	1.7
Upper 2.5 percentile	8.0	5.9	5.5	6.8	7.4	6.4	5.8	7.9	13.4	7.6	6.3	5.6	7.1
Lower 2.5 percentile	0.5	0.1	0.2	0.4	0.7	0.3	0.3	0.6	0.5	0.6	0.2	0.3	0.5
% total energy (including energy from alcohol)													
Mean	3.4	2.2	1.9	2.6	3.2	2.2	2.3	3.2	6.0	3.3	2.2	2.1	3.0
Median	3.3	1.8	1.6	2.3	3.0	1.8	2.0	2.9	5.7	3.1	1.8	1.8	2.7
sd	1.8	1.5	1.3	1.6	1.7	1.6	1.7	1.7	3.4	1.8	1.6	1.6	1.7
Upper 2.5 percentile	8.0	5.9	5.4	6.6	7.4	6.4	5.7	7.1	13.4	7.6	6.3	5.6	6.7
Lower 2.5 percentile	0.5	0.1	0.2	0.3	0.7	0.3	0.3	0.5	0.5	0.6	0.2	0.3	0.5
Number of participants (unweighted)	*311*	*342*	*519*	*131*	*302*	*324*	*667*	*174*	*303*	*613*	*666*	*1186*	*305*

Table 3.12

Average daily intake of non-starch polysaccharide (NSP) by age and sex

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Non starch polysaccharide (NSP)	Sex and age group (years)												
	Boys		Men		Girls		Women		Total				
	4-10	11-18	19-64	65+	4-10	11-18	19-64	65+	1.5-3	4-10	11-18	19-64	65+
g/day													
Mean	11.5	12.8	14.7	14.9	10.7	10.7	12.8	13.1	8.2	11.1	11.8	13.7	13.9
Median	11.3	12.5	14.1	14.5	10.5	10.3	12.2	12.8	8.1	10.9	11.3	13.2	13.3
sd	3.2	4.3	5.3	5.6	3.2	3.5	4.5	4.2	2.7	3.2	4.1	5.0	4.9
Upper 2.5 percentile	18.8	23.1	27.4	26.9	17.8	18.3	23.1	21.9	14.3	18.6	21.6	25.1	26.1
Lower 2.5 percentile	5.9	5.7	6.2	5.7	5.4	4.5	5.2	6.5	3.2	5.7	4.7	5.5	6.1
Number of participants (unweighted)	*665*	*744*	*1126*	*317*	*612*	*753*	*1571*	*436*	*604*	*1277*	*1497*	*2697*	*753*

Table 3.13a

Total quantities consumed of selected carbohydrate-containing foods (grams) per day: males (including non-consumers), by age

Males aged 4 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Food group	Age group (years)									
	Boys						Men			
	4-10		11-18		Total boys		19-64		65+	
	Mean	sd	Mean	sd	Mean	sd	Mean	sd	Mean	sd
White bread	45	39	66	51	57	47	63	57	57	55
Wholemeal bread	10	23	10	26	10	24	19	39	23	39
Brown, granary and wheatgerm bread	15	28	13	30	14	29	16	32	16	34
Other breads[a]	2	9	2	11	2	10	3	12	2	10
High fibre breakfast cereals[b]	19	30	13	25	16	27	19	52	44	75
Other breakfast cereals[c]	10	13	13	19	12	17	7	13	6	12
Chips, fried and roast potatoes and potato products	39	35	58	52	50	46	47	51	41	43
Boiled, mashed and baked potatoes, potato salads and dishes	28	30	33	40	31	36	47	57	65	84
Table sugar, preserves and sweet spreads	6	8	7	10	7	10	14	18	19	27
Sugar confectionery	8	14	8	17	8	16	2	7	1	5
Chocolate confectionery	9	13	13	20	11	17	10	21	4	10
Fruit juice	106	138	92	147	98	143	59	129	50	96
Soft drinks, not low calorie[d]	139	174	310	314	234	275	160	256	54	142
Soft drinks, low calorie[d]	197	241	183	286	189	267	116	280	44	204
Number of participants (unweighted)	*665*		*744*		*1409*		*1126*		*317*	

[a] Breads made with non-wheat flour; Includes rye bread, gluten free, oatmeal bread, besan flour chappatis, soya and linseed bread
[b] Breakfast cereals with NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge
[c] Breakfast cereals with NSP content of less than 4g/100g. Includes cornflakes, sugar puffs
[d] Non alcoholic beverages are reported as consumed with diluent water.

Table 3.13b

Total quantities consumed of selected carbohydrate-containing foods (grams) per day: male consumers[a], by age

Male consumers aged 4 years and over — *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Food Group[b]	Age group (years)														
	Boys 4-10			11-18			Total boys			Men 19-64			65+		
	Mean	Median	% consumers	Mean	Median	% consumers	Mean	Median	% consumers	Mean	Median	% consumers	Mean	Median	% consumers
White bread	53	47	85	75	64	88	65	58	87	78	68	81	74	68	77
Wholemeal bread	39	34	26	46	31	21	42	34	23	58	43	33	59	53	38
Brown, granary and wheatgerm bread	44	35	35	48	36	26	46	36	30	50	38	32	55	40	28
Other breads[b]	26	18	8	38	33	6	32	21	7	35	23	8	34	22	7
High fibre breakfast cereals[c]	31	20	63	34	24	39	32	23	49	48	30	40	76	41	58
Other breakfast cereals[d]	17	14	60	26	23	48	22	16	53	22	19	31	23	23	26
Chips, fried & roast potatoes & potato products	48	40	80	75	68	78	63	52	79	71	53	67	66	55	62
Boiled, mashed & baked potatoes, potato salads & dishes	44	36	64	59	49	55	52	45	59	76	56	62	87	68	75
Table sugar, preserves and sweet spreads	9	6	66	12	8	61	11	8	63	20	15	68	27	20	70
Sugar confectionery	17	12	47	24	15	33	20	14	39	13	8	13	12	6	6
Chocolate confectionery	15	12	62	24	16	54	20	15	58	24	15	42	15	11	26
Fruit juice	164	125	65	186	150	49	175	133	56	147	100	40	151	125	33
Soft drinks, not low calorie[e]	203	145	68	387	315	80	312	238	75	289	196	55	184	137	29
Soft drinks, low calorie[e]	288	225	68	340	261	54	314	237	60	351	183	33	279	125	16
Number of participants (unweighted)	*665*			*744*			*1409*			*1126*			*317*		

[a] Per cent consumers is over the four days although the gram intake is per day.
[b] Breads made with non-wheat flour; Includes rye bread, gluten free, oatmeal bread, besan flour chappatis, soya and linseed bread
[c] Breakfast cereals with NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge
[d] Breakfast cereals with NSP content of less than 4g/100g. Includes cornflakes, sugar puffs
[e] Non-alcoholic beverages are reported as consumed with diluent water.

Table 3.14a

Total quantities of carbohydrate-containing foods consumed (grams) per day: females (including non-consumers), by age

Females aged 4 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Food group	Age group (years)									
	Girls				**Total**		**Women**			
	4-10		11-18		**Total girls**		19-64		65+	
	Mean	sd	Mean	sd	Mean	sd	Mean	sd	Mean	sd
White bread	43	36	48	39	46	38	40	41	29	37
Wholemeal bread	8	18	6	16	7	17	15	27	18	29
Brown, granary and wheatgerm bread	13	24	11	22	12	23	13	23	16	26
Other breads[a]	2	9	2	9	2	9	3	11	3	12
High fibre breakfast cereals[b]	16	24	10	20	13	22	21	43	38	53
Other breakfast cereals[c]	10	12	8	13	9	13	5	10	6	22
Chips, fried and roast potatoes and potato products	35	33	50	45	43	41	35	41	22	28
Other potatoes, potato salads and dishes	29	32	33	41	31	37	41	44	62	52
Table sugar, preserves and sweet spreads	5	8	6	9	5	8	9	13	11	12
Sugar confectionery	9	22	6	15	7	19	2	7	1	5
Chocolate confectionery	9	14	11	15	10	15	7	13	5	11
Fruit juice	79	100	73	113	76	107	48	96	48	88
Soft drinks, not low calorie[d]	117	150	210	244	169	212	112	202	53	96
Soft drinks, low calorie[d]	171	246	173	258	172	253	100	212	28	96
Number of participants (unweighted)	*612*		*753*		*1365*		*1571*		*436*	

[a] Breads made with non-wheat flour; Includes rye bread, gluten free, oatmeal bread, besan flour chappatis, soya and linseed bread

[b] Breakfast cereals with NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge

[c] Breakfast cereals with NSP content of less than 4g/100g. Includes cornflakes, sugar puffs

[d] Non alcoholic beverages are reported as consumed with diluent water.

Table 3.14b

Total quantities consumed of selected carbohydrate-containing foods (grams) per day: female consumers[a], by age

Female consumers aged 4 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Food Group[b]	Age group (years)														
	Girls						Total			Women					
	4-10		%	11-18		%	girls		%	19-64		%	65+		%
	Mean	Median	consumers	Mean	Median	consumers	Mean	Median	consumers	Mean	Median	consumers	Mean	Median	consumers
White bread	50	46	86	56	50	87	53	48	86	52	41	78	48	39	61
Wholemeal bread	31	23	26	29	21	21	30	22	23	40	30	38	41	30	45
Brown, granary and wheatgerm bread	36	28	36	34	23	31	35	25	33	36	30	36	42	40	38
Other breads[b]	22	12	10	31	24	7	26	20	8	28	21	11	34	23	10
High fibre breakfast cereals[c]	28	20	58	27	20	36	27	20	46	45	26	46	60	40	64
Other breakfast cereals[d]	17	15	59	19	16	43	18	15	50	17	15	31	18	12	35
Chips, fried and roast potatoes and potato products	45	38	78	65	55	77	56	47	77	56	47	62	45	41	49
Boiled, mashed & baked potatoes, potato salads & dishes	43	35	66	57	45	58	51	43	62	61	50	66	73	67	85
Table sugar, preserves and sweet spreads	8	6	63	9	6	61	9	6	62	14	10	63	15	12	74
Sugar confectionery	19	12	50	16	9	37	17	10	43	11	7	17	10	6	11
Chocolate confectionery	16	11	55	19	13	59	18	12	57	17	13	44	14	10	33
Fruit juice	135	113	59	153	125	48	144	120	53	118	83	41	121	100	40
Soft drinks, not low calorie[e]	167	113	70	279	222	75	231	170	73	221	133	51	144	111	37
Soft drinks, low calorie[e]	265	195	65	303	205	57	285	200	60	267	173	38	157	100	18
Number of participants (unweighted)	*612*			*753*			*1365*			*1571*			*436*		

[a] Per cent consumers is over the four days although the gram intake is per day.
b Breads made with non-wheat flour; Includes rye bread, gluten free, oatmeal bread, besan flour chappatis, soya and linseed bread
c Breakfast cereals with NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge
d Breakfast cereals with NSP content of less than 4g/100g. Includes cornflakes, sugar puffs
e Non-alcoholic beverages are reported as consumed with diluent water.

Table 3.15a

Total quantities of selected carbohydrate-containing foods consumed (grams) per day: all (including non-consumers), by age

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Food group	Age group (years)									
	1.5-3		4-10		11-18		19-64		65+	
	Mean	sd	Mean	sd	Mean	sd	Mean	sd	Mean	sd
White bread	26	25	44	37	57	47	52	51	42	47
Wholemeal bread	8	16	9	20	8	21	17	34	20	34
Brown, granary and wheatgerm bread	10	20	14	26	12	26	15	28	16	30
Other breads[a]	1	4	2	9	2	10	3	12	3	11
High fibre breakfast cereals[b]	19	29	18	27	11	23	20	47	41	63
Other breakfast cereals[c]	5	7	10	12	11	16	6	12	6	18
Chips, fried and roast potatoes and potato products	19	21	37	34	54	49	41	46	30	37
Boiled, mashed and baked potatoes, potato salads and dishes	21	25	28	31	33	40	44	51	63	68
Table sugar, preserves and sweet spreads	4	7	6	8	6	10	11	16	14	20
Sugar confectionery	4	9	9	18	7	16	2	7	1	5
Chocolate confectionery	5	7	9	13	12	18	9	17	4	11
Fruit juice	57	81	93	121	83	132	54	114	49	92
Soft drinks, not low calorie[d]	65	119	128	163	261	286	136	232	53	118
Soft drinks, low calorie[d]	183	273	185	244	178	273	108	248	35	153
Number of participants (unweighted)	*604*		*1277*		*1497*		*2697*		*753*	

[a] Breads made with non-wheat flour; Includes rye bread, gluten free, oatmeal bread, besan flour chappatis, soya and linseed bread

[b] Breakfast cereals with NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge

[c] Breakfast cereals with NSP content of less than 4g/100g. Includes cornflakes, sugar puffs

[d] Non alcoholic beverages are reported as consumed with diluent water.

Table 3.15b

Total quantities consumed of selected carbohydrate-containing foods (grams) per day: all consumers[a], by age

Aged 1.5 years and over *National Diet and Nutrition Survey years 1-4 combined (2008/09 - 2011/12)*

Food Group	Age group (years)														
	1.5-3		%	4-10		%	11-18		%	19-64		%	65+		%
	Mean	Median	consumers	Mean	Median	consumers	Mean	Median	consumers	Mean	Median	consumers	Mean	Median	consumers
White bread	32	27	82	52	46	85	65	58	88	65	53	80	61	51	68
Wholemeal bread	26	21	30	35	28	26	38	26	21	48	36	36	48	36	42
Brown, granary and wheatgerm bread	28	21	35	40	30	35	41	28	29	43	33	34	47	40	34
Other breads[b]	14	11	7	24	18	9	34	30	6	31	22	10	34	23	8
High fibre breakfast cereals[c]	28	19	66	29	20	60	30	21	37	46	28	43	66	40	61
Other breakfast cereals[d]	10	8	52	17	15	59	23	19	46	19	15	31	19	15	31
Chips, fried and roast potatoes and potato products	29	25	65	47	39	79	70	63	77	64	50	64	55	50	55
Boiled, mashed, baked potatoes, potato salads and dishes	32	25	65	44	36	65	58	46	57	69	55	64	79	68	80
Table sugar, preserves and sweet spreads	7	5	53	9	6	65	10	8	61	17	13	65	20	15	73
Sugar confectionery	13	9	31	18	12	49	20	13	35	12	7	15	11	6	9
Chocolate confectionery	9	7	52	15	11	59	22	15	56	20	14	43	15	10	30
Fruit juice	114	91	50	151	118	62	170	131	49	132	100	41	133	100	37
Soft drinks, not low calorie[e]	143	100	46	185	130	69	336	263	78	256	158	53	160	113	33
Soft drinks, low calorie[e]	285	206	64	277	201	67	321	225	55	306	175	35	207	100	17
Number of participants (unweighted)	*604*			*1277*			*1497*			*2697*			*753*		

a Per cent consumers is over the four days although the gram intake is per day.
b Breads made with non-wheat flour; Includes rye bread, gluten free, oatmeal bread, besan flour chappatis, soya and linseed bread
c Breakfast cereals with NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge
d Breakfast cereals with NSP content of less than 4g/100g. Includes cornflakes, sugar puffs
e Non-alcoholic beverages are reported as consumed with diluent water.

Table 3.16

Percentage contribution of food groups to average daily carbohydrate intake, by sex and age

Aged 1.5 years and over — *National Diet and Nutrition Survey Years 1-4 combined (2008/09 - 2011/12)*

Food group[a]	Sex and age group (years)														
	Boys		Total boys	Men		Girls		Total girls	Women		Total				
	4-10	11-18		19-64	65+	4-10	11-18		19-64	65+	1.5-3	4-10	11-18	19-64	65+
	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%
Cereals and cereal products	46	46	46	45	46	47	44	45	45	44	42	46	45	45	45
of which:															
Pasta, rice, pizza and other miscellaneous cereals	9	12	11	11	4	9	12	11	10	4	8	9	12	11	4
White bread	11	13	12	13	13	11	12	11	10	8	9	11	12	12	10
Wholemeal bread	2	2	2	4	4	2	1	2	4	4	2	2	2	4	4
Brown, granary and wheatgerm bread	4	2	3	3	3	3	3	3	3	4	3	3	3	3	4
Other breads[b]	0	0	0	1	0	1	0	0	1	1	0	0	0	1	1
High fibre breakfast cereals[c]	5	3	4	4	5	4	3	3	4	6	6	4	3	4	6
Other breakfast cereals[d]	4	4	4	2	2	5	3	4	2	2	3	4	4	2	2
Biscuits	5	4	5	3	4	5	5	5	5	5	5	5	4	4	4
Buns, cakes, pastries and fruit pies	5	3	4	4	7	6	4	5	4	7	3	5	4	4	7
Puddings	1	1	1	1	2	2	1	1	1	3	2	2	1	1	2
Milk and milk products	9	6	7	5	6	9	6	7	6	9	16	9	6	5	8
Eggs and egg dishes	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Fat spreads	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Meat and meat products	4	6	5	6	6	4	6	5	6	5	4	4	6	6	5
Fish and fish dishes	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Vegetables and potatoes	11	12	11	14	14	11	13	12	14	13	9	11	12	14	14
of which:															
Salad and other raw vegetables	0	0	0	1	1	0	0	0	1	1	0	0	0	1	1
Vegetables (not raw) including vegetable dishes	3	3	3	4	3	3	3	3	4	3	3	3	3	4	3
Chips, fried and roast potatoes and potato products	5	6	6	6	5	5	7	6	5	3	4	5	7	5	4
Other potatoes, potato salads and dishes	2	3	2	4	5	3	3	3	4	6	3	3	3	4	6
Savoury snacks	3	3	3	2	1	3	3	3	2	1	2	3	3	2	1
Nuts and seeds	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Fruit	6	3	4	5	8	7	4	5	7	10	10	7	3	6	9
Sugar, preserves and confectionery	7	7	7	8	8	7	7	7	7	7	5	7	7	7	7
of which:															
Sugars, including table sugar, preserves and sweet spreads	2	2	2	5	7	2	2	2	4	5	2	2	2	5	6
Sugar confectionery	2	2	2	0	0	3	2	2	1	0	1	2	2	1	0
Chocolate confectionery	3	3	3	2	1	3	3	3	2	1	2	3	3	2	1

Table 3.16 (continued)

Percentage contribution of food groups to average daily carbohydrate intake, by sex and age

Aged 1.5 years and over *National Diet and Nutrition Survey: Years 1-4 combined (2008/09 - 2011/12)*

Food group[a]	Sex and age group (years)														
	Boys		Total boys	Men		Girls		Total girls	Women		Total				
	4-10	11-18		19-64	65+	4-10	11-18		19-64	65+	1.5-3	4-10	11-18	19-64	65+
	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%
Non-alcoholic beverages	10	14	12	8	4	9	13	11	7	5	7	9	13	8	5
of which:															
Fruit juice	5	3	4	2	2	4	3	4	2	2	4	4	3	2	2
Soft drinks, not low calorie	5	11	8	5	2	5	9	7	5	2	3	5	10	5	2
Alcoholic beverages	0	1	0	4	2	0	1	0	2	1	0	0	1	3	1
of which:															
Beer, lager, cider and perry	0	1	0	3	2	0	1	0	1	0	0	0	1	2	1
Miscellaneous[e]	2	2	2	2	3	2	2	2	3	5	3	2	2	3	4
Average daily carbohydrate intake g	219	265	244	251	231	205	211	209	197	187	151	212	239	224	206
Bases (unweighted)	*665*	*744*	*1409*	*1126*	*317*	*612*	*753*	*1365*	*1571*	*436*	*604*	*1277*	*1497*	*2697*	*753*

[a] Food groups that contribute <0.5% to intake across all age/sex groups are excluded from the table. All other food groups are included.

b Breads made with non-wheat flour; Includes rye bread, gluten free, oatmeal bread, besan flour chappatis, soya and linseed bread

c Breakfast cereals with NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge

d Breakfast cereals with NSP content of less than 4g/100g. Includes cornflakes, sugar puffs

[e] In addition to dry weight beverages; soup, manufactured/retail and homemade; savoury sauces, pickles, gravies and condiments; and commercial toddler foods, Miscellaneous also includes nutrition powders and drinks.

Table 3.17

Percentage contribution of food groups to average daily non-milk extrinsic sugars (NMES) intake, by sex and age

Aged 1.5 years and over *National Diet and Nutrition Survey: Year 1-4 combined (2008/09 - 2011/12*

Food group[a]	Sex and age group (years)														
	Boys		Total boys	Men		Girls		Total girls	Women		Total				
	4-10 %	11-18 %		19-64 %	65+ %	4-10 %	11-18 %		19-64 %	65+ %	1.5-3 %	4-10 %	11-18 %	19-64 %	65+ %
Cereals and cereal products	28	22	25	19	29	29	23	26	24	29	25	29	22	21	29
of which:															
Pasta, rice, pizza and other miscellaneous cereals	1	1	1	1	0	1	1	1	1	0	2	1	1	1	0
High fibre breakfast cereals[b]	3	2	3	2	3	2	2	2	3	3	3	3	2	3	3
Other breakfast cereals[c]	4	5	5	2	1	5	4	4	3	2	3	5	4	3	2
Biscuits	8	7	7	5	7	8	8	8	7	6	8	8	7	6	7
Buns, cakes, pastries and fruit pies	9	5	7	6	13	10	6	8	8	13	6	9	6	7	13
Puddings	3	2	2	2	4	3	1	2	2	5	3	3	2	2	4
Milk and milk products	11	6	9	5	6	12	8	10	7	10	18	12	7	6	8
of which:															
Other milk and cream	2	2	2	1	1	2	2	2	1	1	1	2	2	1	1
Yoghurt, fromage frais and other dairy desserts	6	2	4	3	3	6	3	4	4	6	13	6	3	3	5
Ice cream	4	2	3	2	2	5	3	4	2	3	3	4	3	2	3
Eggs and egg dishes	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Fat spreads	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Meat and meat products	1	1	1	2	2	1	1	1	2	2	1	1	1	2	2
Fish and fish dishes	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Vegetables and potatoes	2	2	2	2	2	1	1	1	3	1	3	2	2	2	2
Savoury snacks	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Nuts and seeds	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Fruit	1	0	1	1	4	1	1	1	2	4	3	1	1	1	4
Sugar, preserves and confectionery	22	21	21	27	28	22	22	22	25	24	19	22	21	26	26
of which:															
Sugars, including table sugar, preserves and sweet spreads	7	8	8	19	24	7	8	8	16	19	7	7	8	17	21
Sugar confectionery	7	5	6	1	1	7	5	6	2	1	5	7	5	2	1
Chocolate confectionery	7	7	7	7	3	8	9	8	7	5	7	7	8	7	4
Non-alcoholic beverages	32	42	38	26	14	28	38	34	24	18	27	30	40	25	16
of which:															
Fruit juice	15	10	12	8	8	12	10	11	8	8	14	13	10	8	8
Soft drinks, not low calorie	16	31	24	16	6	15	27	22	15	9	10	16	29	16	8
Soft drinks, low calorie	1	1	1	0	0	1	1	1	0	0	2	1	1	0	0
Tea, coffee and water	0	0	0	1	0	0	0	0	0	0	0	0	0	1	0

Table 3.17 (continued)

Percentage contribution of food groups to average daily non-milk extrinsic sugars (NMES) intake, by sex and age

Aged 1.5 years and over *National Diet and Nutrition Survey: Years 1-4 combined 2008/09 - 2011/12*

Food group[a]	Sex and age group (years)														
	Boys		Total boys	Men		Girls		Total girls	Women		Total				
	4-10	11-18		19-64	65+	4-10	11-18		19-64	65+	1.5-3	4-10	11-18	19-64	65+
	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%
Alcoholic beverages	0	2	1	14	10	0	2	1	6	3	0	0	2	10	6
of which:															
Wine	0	0	0	1	1	0	0	0	2	1	0	0	0	2	1
Beer, lager, cider and perry	0	2	1	13	8	0	2	1	3	1	0	0	2	8	4
Miscellaneous[d]	3	4	3	4	5	5	4	4	6	9	5	4	4	5	7
Average daily non-milk extrinsic sugars (NMES) intake g	63.0	84.0	74.6	68.4	58.5	58.5	63.9	61.5	49.2	46.2	36.1	60.8	74.2	58.8	51.6
Bases (unweighted)	*665*	*744*	*1409*	*1126*	*317*	*612*	*753*	*1365*	*1571*	*436*	*604*	*1277*	*1497*	*2697*	*753*

[a] Food groups that contribute <0.5% to intake across all age/sex groups are excluded from the table. All other food groups are included.

b Breakfast cereals with NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge

c Breakfast cereals with NSP content of less than 4g/100g. Includes cornflakes, sugar puffs

[d] In addition to dry weight beverages; soup, manufactured/retail and homemade; savoury sauces, pickles, gravies and condiments; and commercial toddler foods, Miscellaneous also includes nutrition powders and drinks.

Table 3.18

Percentage contribution of food groups to average daily non-starch polysaccharide (NSP) intake, by sex and age

Aged 1.5 years and over *National Diet and Nutrition Survey Years 1-4 combined 2008/09 - 2011/12*

Food group[a]	Sex and age group (years)														
	Boys		Total	Men		Girls		Total	Women		Total				
	4-10	11-18	boys	19-64	65+	4-10	11-18	girls	19-64	65+	1.5-3	4-10	11-18	19-64	65+
	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%
Cereals and cereal products	43	43	43	40	40	42	41	41	37	38	41	42	42	39	39
of which:															
Pasta, rice, pizza and other miscellaneous cereals	8	11	10	8	3	8	11	10	7	3	7	8	11	8	3
White bread	9	11	10	10	9	9	10	9	8	6	7	9	11	9	7
Wholemeal bread	4	3	4	6	7	3	3	3	5	6	4	4	3	5	6
Brown, granary and wheatgerm bread	5	4	4	4	4	5	4	4	4	5	5	5	4	4	4
Other breads[b]	1	0	0	1	1	1	1	1	1	1	0	1	0	1	1
High fibre breakfast cereals[c]	8	5	6	5	8	7	5	5	6	8	10	7	5	5	8
Other breakfast cereals[d]	2	2	2	1	1	2	2	2	1	1	1	2	2	1	1
Biscuits	3	3	3	2	3	3	4	4	3	3	4	3	3	3	3
Buns, cakes, pastries and fruit pies	3	2	3	2	4	3	2	3	2	3	2	3	2	2	4
Puddings	1	1	1	1	1	1	1	1	0	1	0	1	1	1	1
Milk and milk products	1	1	1	0	1	1	1	1	1	1	3	1	1	1	1
Eggs and egg dishes	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Fat spreads	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Meat and meat products	8	12	10	12	9	8	11	9	10	7	7	8	11	11	8
Fish and fish dishes	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Vegetables and potatoes	27	28	28	31	32	27	30	28	32	33	24	27	29	31	32
of which:															
Salad and other raw vegetables	2	1	2	3	3	2	2	2	4	4	1	2	2	4	4
Vegetables (not raw) including vegetable dishes	14	13	13	15	16	13	12	13	16	18	14	14	12	16	17
Chips, fried and roast potatoes and potato products	8	10	9	8	6	7	11	9	6	4	5	7	10	7	5
Other potatoes, potato salads and dishes	4	4	4	5	6	4	5	4	5	7	3	4	4	5	7
Savoury snacks	4	4	4	2	1	4	4	4	2	1	2	4	4	2	1
Nuts and seeds	0	1	0	1	1	0	1	0	1	1	0	0	1	1	1
Fruit	12	6	8	8	11	13	8	10	10	14	16	13	7	9	12
Sugar, preserves and confectionery	1	2	2	1	0	1	1	1	1	1	1	1	2	1	1
Non-alcoholic beverages	1	1	1	0	0	1	1	1	0	0	1	1	1	0	0
Alcoholic beverages	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Miscellaneous[e]	2	2	2	3	3	2	3	2	3	4	3	2	2	3	4
Average daily non-starch polysaccharide (NSP) intake g	11.5	12.8	12.2	14.7	14.9	10.7	10.7	10.7	12.8	13.1	8.2	11.1	11.8	13.7	13.9

Table 3.18 (continued)

Percentage contribution of food groups to average daily non-starch polysaccharide (NSP) intake, by sex and age

Aged 1.5 years and over *National Diet and Nutrition Survey: Years 1-4 combined (2008/09 - 2011/12)*

Food group[a]	**Sex and age group (years)**														
	Boys		**Total**	**Men**		**Girls**		**Total**	**Women**		**Total**				
	4-10	11-18	**boys**	19-64	65+	4-10	11-18	**girls**	19-64	65+	1.5-3	4-10	11-18	19-64	65+
	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%
Bases (unweighted)	*665*	*744*	*1409*	*1126*	*317*	*612*	*753*	*1365*	*1571*	*436*	*604*	*1277*	*1497*	*2697*	*753*

[a] Food groups that contribute <0.5% to intake across all age/sex groups are excluded from the table. All other food groups are included.

b Breads made with non-wheat flour; Includes rye bread, gluten free, oatmeal bread, besan flour chappatis, soya and linseed bread

c Breakfast cereals with NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge

d Breakfast cereals with NSP content of less than 4g/100g. Includes cornflakes, sugar puffs

[e] In addition to dry weight beverages; soup, manufactured/retail and homemade; savoury sauces, pickles, gravies and condiments; and commercial toddler foods, Miscellaneous also includes nutrition powders and drinks.

Table 3.19a

Top 20 contributors to total carbohydrate intake: children aged 1½-3 years

1.5 - 3 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	MILK AND CREAM	15.8	10.3
2	FRUIT	15.7	10.3
3	WHITE BREAD	12.8	8.4
4	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	8.7	5.7
5	BISCUITS	7.3	4.8
6	PASTA AND PASTA DISHES	7.2	4.7
7	CHIPS, FRIED AND ROAST POTATOES AND POTATO PRODUCTS	6.0	3.9
8	SOFT DRINKS	5.9	3.8
9	VEGETABLES AND VEGETABLE BASED DISHES	5.4	3.5
10	FRUIT JUICE	5.3	3.5
11	BROWN GRANARY AND WHEATGERM BREAD	5.1	3.4
12	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	4.9	3.2
13	BOILED, MASHED AND BAKED POTATOES AND DISHES	4.2	2.8
14	BUNS CAKES AND PASTRIES	4.2	2.7
15	WHOLEMEAL BREAD	3.6	2.4
16	FROMAGE FRAIS AND DAIRY DESSERTS	3.4	2.2
17	RICE (INCLUDES RISOTTO, FRIED RICE)	3.4	2.2
18	CRISPS AND SAVOURY SNACKS	3.3	2.2
19	CHOCOLATE CONFECTIONERY	3.3	2.2
20	YOGURT	2.9	1.9

Table 3.19b

Top 20 contributors to total carbohydrate intake: children aged 4-10 years

4 - 10 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	WHITE BREAD	23.3	10.8
2	FRUIT	14.2	6.6
3	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	11.3	5.3
4	SOFT DRINKS	11.2	5.2
5	BISCUITS	10.8	5.0
6	MILK AND CREAM	10.5	4.9
7	BUNS CAKES AND PASTRIES	10.4	4.8
8	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	9.5	4.4
9	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	9.3	4.3
10	PASTA AND PASTA-BASED DISHES	9.3	4.3
11	FRUIT JUICE	8.6	4.0
12	VEGETABLES AND VEGETABLE-BASED DISHES	7.4	3.5
13	BROWN GRANARY AND WHEATGERM BREAD	7.0	3.2
14	CRISPS AND SAVOURY SNACKS	6.2	2.9
15	SUGAR CONFECTIONERY	5.8	2.7
16	BOILED, MASHED AND BAKED POTATOES AND HOMEMADE DISHES	5.7	2.7
17	CHOCOLATE CONFECTIONERY	5.7	2.6
18	PIZZA	4.9	2.3
19	RICE (INCLUDING RISOTTO, FRIED RICE)	4.5	2.1
20	WHOLEMEAL BREAD	4.1	1.9

Table 3.19c

Top 20 contributors to total carbohydrate intake: children 11-18 years

11 - 18 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	WHITE BREAD	30.2	12.6
2	SOFT DRINKS	24.8	10.4
3	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	16.5	6.9
4	PASTA AND PASTA-BASED DISHES	11.4	4.7
5	BISCUITS	10.7	4.5
6	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	9.5	4.0
7	MILK AND CREAM	8.3	3.5
8	FRUIT	8.2	3.4
9	BUNS CAKES AND PASTRIES	8.1	3.4
10	CRISPS AND SAVOURY SNACKS	7.9	3.3
11	PIZZA	7.9	3.3
12	CHOCOLATE CONFECTIONERY	7.8	3.3
13	FRUIT JUICE	7.7	3.2
14	VEGETABLES AND VEGETABLE-BASED DISHES	7.3	3.1
15	RICE INCLUDING RISOTTOS AND FRIED RICE	7.1	3.0
16	BOILED, MASHED AND BAKED POTATOES INCLUDING DISHES	6.9	2.9
17	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	6.0	2.5
18	BROWN GRANARY AND WHEATGERM BREAD	5.8	2.4
19	SUGAR CONFECTIONERY	4.7	1.9
20	TABLE SUGAR	4.6	1.9

Table 3.19d

Top 20 contributors to total carbohydrate intake : adults 19-64 years

Adults 19-64 yrs

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	WHITE BREAD	26.1	11.6
2	FRUIT	13.3	5.9
3	SOFT DRINKS	13.0	5.8
4	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	11.8	5.2
5	VEGETABLES AND VEGETABLE-BASED DISHES	11.3	5.0
6	BOILED, MASHED, BAKED POTATOES INCLUDING DISHES	9.3	4.1
7	RICE (INCLUDES RISOTTO, FRIED RICE)	9.2	4.1
8	TABLE SUGAR	9.1	4.0
9	BISCUITS	8.5	3.8
10	WHOLEMEAL BREAD	8.4	3.8
11	PASTA AND PASTA-BASED DISHES	8.3	3.7
12	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	8.1	3.6
13	BUNS CAKES, PASTRIES	7.6	3.4
14	MILK AND CREAM	7.3	3.2
15	ALCOHOLIC DRINKS	7.1	3.1
16	BROWN GRANARY AND WHEATGERM BREAD	7.0	3.1
17	CHOCOLATE CONFECTIONERY	5.6	2.5
18	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	5.0	2.2
19	FRUIT JUICE	5.0	2.2
20	PIZZA	4.3	1.9

Table 3.19e

Top 20 contributors to total carbohydrate intake : people aged 65 years and over

65+ years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	WHITE BREAD	22.1	11.0
2	FRUIT	17.3	8.6
3	BUNS CAKES AND PASTRIES	11.9	5.9
4	BOILED, MASHED AND BAKED POTATOES INCLUDING DISHES	11.9	5.9
5	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	10.0	5.0
6	MILK AND CREAM	9.2	4.6
7	TABLE SUGAR	9.2	4.6
8	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	9.0	4.5
9	VEGETABLES AND VEGETABLE-BASED DISHES	8.8	4.4
10	BISCUITS	8.2	4.1
11	WHOLEMEAL BREAD	8.0	4.0
12	BROWN GRANARY AND WHEATGERM BREAD	7.1	3.5
13	CEREAL- BASED PUDDINGS (INCLUDES SPONGE AND RICE PUDDING)	5.5	2.7
14	FRUIT JUICE	4.5	2.2
15	SOFT DRINKS	4.4	2.2
16	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	4.4	2.2
17	YOGURT	4.0	2.0
18	OTHER CEREALS[c]	3.8	1.9
19	JAM AND OTHER PRESERVES	3.8	1.9
20	PASTA AND PASTA-BASED DISHES	3.4	1.7

a Wholegrain and high fibre breakfast cereals: NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge
b Other breakfast cereals, not high fibre: NSP content of less than 4g/100g. Includes cornflakes, sugar puffs
c Other cereals: Includes flour (not rice flour), cous cous, bran, oats, semolina, papadums, dumplings, Yorkshire pudding

Table 3.20a

Top 20 contributors to total sugar intake: children aged 1½-3 years

1.5 - 3 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	MILK AND CREAM	15.1	20.0
2	FRUIT	14.9	19.7
3	SOFT DRINKS	5.8	7.7
4	FRUIT JUICE	5.3	7.0
5	FROMAGE FRAIS AND DAIRY DESSERTS	3.3	4.4
6	CHOCOLATE CONFECTIONERY	3.2	4.2
7	YOGURT	2.7	3.6
8	BISCUITS	2.7	3.6
9	VEGETABLES AND VEGETABLE-BASED DISHES	2.6	3.4
10	SUGAR CONFECTIONERY	2.2	2.9
11	BUNS CAKES AND PASTRIES	2.1	2.8
12	CEREAL-BASED PUDDINGS (INCLUDES SPONGES, RICE PUDDING)	1.6	2.1
13	ICE CREAM	1.6	2.1
14	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	1.4	1.9
15	JAM AND OTHER PRESERVES	1.4	1.8
16	TABLE SUGAR	1.4	1.8
17	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	1.2	1.6
18	WHITE BREAD	0.8	1.1
19	POWDERED BEVERAGES[c]	0.8	1.0
20	PASTA AND PASTA-BASED DISHES	0.7	0.9

Table 3.20b

Top 20 contributors to total sugar intake: children aged 4-10 years

4 - 10 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	FRUIT	13.6	13.9
2	SOFT DRINKS	11.2	11.4
3	MILK AND CREAM	10.5	10.7
4	FRUIT JUICE	8.6	8.8
5	BUNS CAKES AND PASTRIES	5.7	5.8
6	SUGAR CONFECTIONERY	5.5	5.6
7	CHOCOLATE CONFECTIONERY	5.3	5.4
8	BISCUITS	4.7	4.8
9	VEGETABLES AND VEGETABLE-BASED DISHES	3.6	3.6
10	ICE CREAM	3.2	3.2
11	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	3.0	3.1
12	YOGURT	3.0	3.1
13	TABLE SUGAR	2.3	2.4
14	FROMAGE FRAIS AND DAIRY DESSERTS	2.2	2.3
15	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	1.9	2.0
16	CEREAL-BASED PUDDINGS (INCLUDES SPONGE AND RICE PUDDING)	1.8	1.8
17	JAM AND OTHER PRESERVES	1.7	1.7
18	POWDERED BEVERAGES[c]	1.5	1.6
19	WHITE BREAD	1.4	1.5
20	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	1.2	1.2

Table 3.20c

Top 20 contributors to total sugar intake: children aged 11-18 years

11 - 18 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	SOFT DRINKS	24.7	23.9
2	MILK AND CREAM	8.3	8.0
3	FRUIT	7.9	7.6
4	FRUIT JUICE	7.6	7.4
5	CHOCOLATE CONFECTIONERY	7.1	6.9
6	BISCUITS	4.9	4.8
7	TABLE SUGAR	4.6	4.5
8	SUGAR CONFECTIONERY	4.4	4.2
9	BUNS CAKES AND PASTRIES	4.1	4.0
10	VEGETABLES AND VEGETABLE-BASED DISHES	3.7	3.6
11	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	3.0	2.9
12	ALCOHOLIC DRINKS	2.1	2.0
13	ICE CREAM	2.1	2.0
14	YOGURT	2.0	2.0
15	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	1.8	1.8
16	WHITE BREAD	1.8	1.7
17	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	1.4	1.4
18	CEREAL BASED PUDDINGS (INCLUDES SPONGE AND RICE PUDDING)	1.3	1.2
19	POWDERED BEVERAGES[c]	1.1	1.1
20	PIZZA	0.9	0.8

Table 3.20d

Top 20 contributors to <u>total sugar</u> intake: adults aged 19-64 years

Adults 19-64 yrs

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	SOFT DRINKS	13.0	13.4
2	FRUIT	12.7	13.1
3	TABLE SUGAR	9.1	9.4
4	MILK AND CREAM	7.3	7.5
5	ALCOHOLIC DRINKS	7.1	7.3
6	VEGETABLES AND VEGETABLE-BASED DISHES	6.1	6.3
7	CHOCOLATE CONFECTIONERY	5.1	5.3
8	FRUIT JUICE	5.0	5.1
9	BUNS CAKES AND PASTRIES	3.6	3.7
10	BISCUITS	3.3	3.4
11	YOGURT	2.8	2.9
12	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	2.3	2.4
13	JAM AND OTHER PRESERVES	2.2	2.3
14	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	1.8	1.8
15	WHITE BREAD	1.6	1.6
16	CEREAL-BASED PUDDINGS (INCLUDING SPONGE AND RICE PUDDING)	1.4	1.4
17	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	1.3	1.4
18	SUGAR CONFECTIONERY	1.2	1.3
19	ICE CREAM	1.2	1.2
20	POWDERED BEVERAGES[c]	0.8	0.8

Table 3.20e

Top 20 contributors to total sugar intake: people aged 65 years and over

65+ years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	FRUIT	16.6	18.0
2	TABLE SUGAR	9.2	10.0
3	MILK AND CREAM	9.1	9.9
4	BUNS CAKES AND PASTRIES	5.7	6.1
5	VEGETABLES AND VEGETABLE-BASED DISHES	5.7	6.1
6	FRUIT JUICE	4.5	4.9
7	SOFT DRINKS	4.4	4.8
8	YOGURT	3.8	4.1
9	JAM AND OTHER PRESERVES	3.8	4.1
10	CEREAL-BASED PUDDINGS (SPONGE AND RICE PUDDING)	3.7	4.0
11	ALCOHOLIC DRINKS	3.3	3.6
12	CHOCOLATE CONFECTIONERY	2.7	2.9
13	BISCUITS	2.7	2.9
14	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	2.1	2.2
15	POWDERED BEVERAGES[c]	1.9	2.1
16	ICE CREAM	1.5	1.7
17	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	1.4	1.5
18	WHITE BREAD	1.4	1.5
19	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	0.9	0.9
20	SUGAR CONFECTIONERY	0.7	0.8

a : Wholegrain and high fibre breakfast cereals: NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge
b: Other breakfast cereals, not high fibre: NSP content of less than 4g/100g. Includes cornflakes, sugar puffs
c: Powdered beverages: Includes drinking chocolate, cocoa, Ovaltine, Horlicks, malted drinks, milk shake powder

Table 3.21a

Top 20 contributors to starch intake: children aged 1.5-3 years

1.5 - 3 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	WHITE BREAD	12.0	15.4
2	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	7.3	9.4
3	PASTA AND PASTA-BASED DISHES	6.5	8.4
4	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	5.8	7.5
5	BROWN GRANARY AND WHEATGERM BREAD	4.7	6.1
6	BISCUITS	4.6	5.9
7	BOILED, MASHED AND BAKED POTATOES AND DISHES	4.0	5.2
8	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	3.7	4.8
9	WHOLEMEAL BREAD	3.4	4.3
10	RICE (INCLUDES RISOTTO, FRIED RICE)	3.3	4.3
11	CRISPS AND SAVOURY SNACKS	3.2	4.1
12	VEGETABLES AND VEGETABLE-BASED DISHES	2.8	3.6
13	BUNS CAKES AND PASTRIES	2.0	2.6
14	OTHER CEREALS[c]	1.7	2.2
15	PIZZA	1.6	2.1
16	WHITE FISH COATED OR FRIED	1.3	1.7
17	SAUSAGES AND SAUSAGE BASED DISHES	1.0	1.2
18	MEAT PIES AND PASTRIES	1.0	1.2
19	CEREAL-BASED PUDDINGS (INCLUDES SPONGE PUDDING, RICE PUDDING)	0.9	1.1
20	COATED CHICKEN AND TURKEY PRODUCTS	0.9	1.1

Table 3.21b

Top 20 contributors to starch intake: children aged 4-10 years

4 - 10 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	WHITE BREAD	21.8	18.7
2	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	11.0	9.4
3	PASTA AND PASTA-BASED DISHES	8.8	7.5
4	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	7.6	6.5
5	BROWN GRANARY AND WHEATGERM BREAD	6.4	5.5
6	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	6.3	5.4
7	BISCUITS	6.1	5.2
8	CRISPS AND SAVOURY SNACKS	5.9	5.1
9	BOILED, MASHED AND BAKED POTATOES INCLUDING DISHES	5.5	4.7
10	BUNS CAKES AND PASTRIES	4.7	4.0
11	RICE (INCLUDING RISOTTO AND FRIED RICE)	4.5	3.8
12	PIZZA	4.4	3.7
13	VEGETABLES AND VEGETABLE-BASED DISHES	3.9	3.3
14	WHOLEMEAL BREAD	3.8	3.3
15	OTHER CEREALS[c]	2.5	2.1
16	COATED CHICKEN AND TURKEY PRODUCTS	1.6	1.3
17	WHITE FISH COATED OR FRIED	1.5	1.3
18	SAUSAGES AND SAUSAGE-BASED DISHES	1.4	1.2
19	MEAT PIES AND PASTRIES	1.2	1.0
20	CEREAL- BASED PUDDINGS (INCLUDES SPONGE AND RICE PUDDING)	1.0	0.9

Table 3.21c

Top 20 contributors to starch intake: children aged 11-18 years

11 - 18 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	WHITE BREAD	28.3	20.8
2	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	16.0	11.8
3	PASTA AND PASTA-BASED DISHES	10.7	7.9
4	CRISPS AND SAVOURY SNACKS	7.6	5.6
5	PIZZA	7.1	5.2
6	RICE (INCLUDES RISOTTO AND FRIED RICE)	7.0	5.2
7	BOILED, MASHED AND BAKED POTATOES INCLUDING DISHES	6.6	4.9
8	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	6.5	4.8
9	BISCUITS	5.7	4.2
10	BROWN GRANARY AND WHEATGERM BREAD	5.3	3.9
11	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	4.6	3.4
12	BUNS CAKES AND PASTRIES	4.0	2.9
13	VEGETABLES AND VEGETABLE-BASED DISHES	3.6	2.6
14	WHOLEMEAL BREAD	3.4	2.5
15	OTHER CEREALS[c]	3.0	2.2
16	COATED CHICKEN AND TURKEY PRODUCTS	2.1	1.6
17	MEAT PIES AND PASTRIES	2.1	1.5
18	BURGERS AND KEBABS	1.6	1.2
19	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	1.4	1.0
20	SAUSAGES AND SAUSAGE-BASED DISHES	1.3	0.9

Table 3.21d

Top 20 contributors to starch intake: adults aged 19-64 years

Adults 19-64 yrs

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	WHITE BREAD	24.5	19.1
2	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	11.4	8.9
3	RICE (INCLUDES RISOTTO AND FRIED RICE)	9.1	7.1
4	BOILED, MASHED, BAKED POTATOES INCLUDING DISHES	8.9	6.9
5	PASTA AND PASTA-BASED DISHES	8.0	6.2
6	WHOLEMEAL BREAD	7.9	6.2
7	BROWN GRANARY AND WHEATGERM BREAD	6.5	5.0
8	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	6.4	5.0
9	VEGETABLES AND VEGETABLE-BASED DISHES	5.2	4.1
10	BISCUITS	5.2	4.0
11	CRISPS AND SAVOURY SNACKS	4.1	3.2
12	OTHER CEREALS[c]	4.0	3.1
13	BUNS CAKES AND PASTRIES	4.0	3.1
14	PIZZA	3.9	3.0
15	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	3.7	2.9
16	MEAT PIES AND PASTRIES	1.7	1.3
17	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	1.5	1.2
18	WHITE FISH COATED OR FRIED	1.3	1.0
19	OTHER BREAD[d]	1.3	1.0
20	SAUSAGES AND SAUSAGE-BASED DISHES	1.1	0.8

Table 3.21e

Top 20 contributors to starch intake: people aged 65 years and over

65+ years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	WHITE BREAD	20.7	19.0
2	BOILED, MASHED, BAKED POTATOES INCLUDING DISHES	11.3	10.4
3	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	8.6	8.0
4	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	7.9	7.3
5	WHOLEMEAL BREAD	7.4	6.8
6	BROWN GRANARY AND WHEATGERM BREAD	6.6	6.0
7	BUNS CAKES AND PASTRIES	6.3	5.8
8	BISCUITS	5.5	5.1
9	OTHER CEREALS[c]	3.6	3.3
10	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	3.5	3.2
11	PASTA AND PASTA-BASED DISHES	3.2	2.9
12	VEGETABLES AND VEGETABLE-BASED DISHES	3.2	2.9
13	RICE (INCLUDES RISOTTO AND FRIED RICE)	2.9	2.6
14	CEREAL-BASED PUDDINGS (INCLUDES SPONGE AND RICE PUDDING)	1.8	1.7
15	MEAT PIES AND PASTRIES	1.6	1.5
16	WHITE FISH COATED OR FRIED	1.6	1.4
17	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	1.5	1.4
18	SOUP	1.2	1.1
19	CRISPS AND SAVOURY SNACKS	1.1	1.0
20	OTHER BREAD[d]	1.0	0.9

a: Wholegrain and high fibre breakfast cereals: NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge
b: Other breakfast cereals, not high fibre: NSP content of less than 4g/100g. Includes cornflakes, sugar puffs
c: Other cereals: Includes flour (not rice flour), cous cous, bran, oats, semolina, papadums, dumplings, Yorkshire pudding
d: Other bread: Bread made with non-wheat flour; Includes rye bread, gluten free, oatmeal bread, besan flour chappatis, soya and linseed bread

Table 3.22a

Top 20 contributors to NSP intake: children aged 1.5-3 years

1.5 - 3 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	VEGETABLES AND VEGETABLE-BASED DISHES	1.5	18.5
2	FRUIT	1.3	16.3
3	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	0.8	10.1
4	WHITE BREAD	0.5	6.1
5	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	0.4	5.2
6	BROWN GRANARY AND WHEATGERM BREAD	0.4	5.2
7	PASTA AND PASTA-BASED DISHES	0.4	5.1
8	WHOLEMEAL BREAD	0.4	4.8
9	BOILED, MASHED AND BAKED POTATOES INCLUDING DISHES	0.3	3.9
10	BISCUITS	0.3	3.2
11	SAUSAGES AND SAUSAGE-BASED DISHES	0.2	2.3
12	CRISPS AND SAVOURY SNACKS	0.1	1.8
13	MILK AND CREAM	0.1	1.8
14	BUNS CAKES AND PASTRIES	0.1	1.4
15	PIZZA	0.1	1.2
16	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	0.1	1.2
17	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	0.1	1.2
18	OTHER CEREALS[c]	0.1	1.1
19	COMMERCIAL TODDLERS FOODS	0.1	0.9
20	MEAT PIES AND PASTRIES	0.1	0.9

Table 3.22b

Top 20 contributors to NSP intake: children aged 4-10 years

4 - 10 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	VEGETABLES AND VEGETABLE-BASED DISHES	2.1	18.4
2	FRUIT	1.5	13.1
3	WHITE BREAD	0.9	8.1
4	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	0.9	7.7
5	CHIPS, FRIED POTATOES AND FRIED POTATO PRODUCTS	0.8	7.3
6	BROWN GRANARY AND WHEATGERM BREAD	0.6	5.3
7	PASTA AND PASTA-BASED DISHES	0.5	4.4
8	WHOLEMEAL BREAD	0.4	4.0
9	BOILED, MASHED AND BAKED POTATOES AND DISHES	0.4	3.9
10	CRISPS AND SAVOURY SNACKS	0.4	3.3
11	BISCUITS	0.3	3.1
12	BUNS CAKES AND PASTRIES	0.3	2.6
13	PIZZA	0.3	2.5
14	SAUSAGES AND SAUSAGE BASED DISHES	0.3	2.3
15	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	0.2	1.7
16	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	0.2	1.5
17	OTHER CEREALS[c]	0.1	1.2
18	FRUIT JUICE	0.1	0.7
19	MEAT PIES AND PASTRIES	0.1	0.7
20	WHITE FISH COATED OR FRIED	0.1	0.7

Table 3.22c

Top 20 contributors to NSP intake: children aged 11-18 years

11 - 18 years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	VEGETABLES AND VEGETABLE-BASED DISHES	2.1	18.0
2	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	1.2	10.3
3	WHITE BREAD	1.2	10.1
4	FRUIT	0.9	7.2
5	PASTA AND PASTA-BASED DISHES	0.6	4.9
6	BOILED, MASHED, BAKED POTATOES INCLUDING DISHES	0.5	4.6
7	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	0.5	4.6
8	CRISPS AND SAVOURY SNACKS	0.5	4.2
9	BROWN GRANARY AND WHEATGERM BREAD	0.5	4.1
10	PIZZA	0.5	3.9
11	WHOLEMEAL BREAD	0.4	3.4
12	BISCUITS	0.4	3.2
13	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	0.3	2.3
14	SAUSAGES AND SAUSAGE-BASED DISHES	0.2	2.1
15	BUNS CAKES AND PASTRIES	0.2	2.1
16	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	0.2	1.8
17	OTHER CEREALS[c]	0.2	1.3
18	CHOCOLATE CONFECTIONERY	0.1	1.2
19	BURGERS AND KEBABS	0.1	1.1
20	MEAT PIES AND PASTRIES	0.1	1.0

Table 3.22d

Top 20 contributors to NSP intake: adults aged 19-64 years

Adults 19-64 yrs

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	VEGETABLES AND VEGETABLE DISHES	3.3	24.2
2	FRUIT	1.4	10.2
3	WHITE BREAD	1.0	7.6
4	WHOLEMEAL BREAD	0.9	6.6
5	CHIPS, FRIED POTATOES AND POTATO PRODUCTS	0.9	6.3
6	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	0.8	6.1
7	BOILED, MASHED, BAKED POTATOES INCLUDING HOMEMADE DISHES	0.7	5.3
8	BROWN GRANARY AND WHEATGERM BREAD	0.6	4.0
9	PASTA AND PASTA-BASED DISHES	0.5	3.3
10	BISCUITS	0.4	2.7
11	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	0.3	2.3
12	CRISPS AND SAVOURY SNACKS	0.3	2.0
13	PIZZA	0.3	1.9
14	OTHER CEREALS[c]	0.2	1.8
15	BUNS CAKES AND PASTRIES	0.2	1.8
16	SAUSAGES AND SAUSAGE-BASED DISHES	0.2	1.5
17	NUTS AND SEEDS	0.2	1.2
18	CHOCOLATE CONFECTIONERY	0.1	1.0
19	RICE (INCLUDES RISOTTO, FRIED RICE)	0.1	0.9
20	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	0.1	0.9

Table 3.22e

Top 20 contributors to NSP intake: people aged 65 years and over

65+ years

National Diet and Nutrition Survey years 1-2 combined (2008/09 - 2009/10)

Rank	NDNS Subsidiary food group	Contribution to intake (g/day)	Contribution to intake (%)
1	VEGETABLES AND VEGETABLE-BASED DISHES	3.3	25.0
2	FRUIT	1.8	13.5
3	WHOLEGRAIN & HIGH FIBRE BREAKFAST CEREALS[a]	1.1	8.5
4	BOILED, MASHED AND BAKED POTATOES INCLUDING DISHES	0.9	6.8
5	WHITE BREAD	0.9	6.6
6	WHOLEMEAL BREAD	0.9	6.6
7	CHIPS. FRIED POTATOES AND POTATO PRODUCTS	0.6	4.9
8	BROWN GRANARY AND WHEATGERM BREAD	0.6	4.2
9	BUNS CAKES AND PASTRIES	0.4	2.9
10	BISCUITS	0.4	2.7
11	OTHER CEREALS[c]	0.2	1.7
12	PASTA AND PASTA-BASED DISHES	0.2	1.5
13	SAVOURY SAUCES PICKLES GRAVIES & CONDIMENTS	0.2	1.3
14	SOUP	0.2	1.4
15	CEREAL-BASED PUDDINGS (INCLUDES SPONGE AND RICE PUDDING)	0.1	1.1
16	SAUSAGES AND SAUSAGE-BASED DISHES	0.1	1.0
17	NUTS AND SEEDS	0.1	0.9
18	OTHER BREAD[d]	0.1	0.8
19	OTHER BREAKFAST CEREALS (NOT HIGH FIBRE)[b]	0.1	0.7
20	CRISPS AND SAVOURY SNACKS	0.1	0.7

a: Wholegrain and high fibre breakfast cereals: NSP content of 4g/100g or more. Includes Muesli, shredded wheat, porridge
b: Other breakfast cereals, not high fibre: NSP content of less than 4g/100g. Includes cornflakes, sugar puffs
c: Other cereals: Includes flour (not rice flour), cous cous, bran, oats, semolina, papadums, dumplings, Yorkshire pudding
d: Other bread: Bread made with non-wheat flour; Includes rye bread, gluten free, oatmeal bread, besan flour chappatis, soya and linseed bread

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