International Journal of Food Sciences and Nutrition,
December 2012; 63(8): 1001–1013

informa
healthcare

Wheat bran: its composition and benefits to health, a European perspective

LEO STEVENSON[1], FRANKIE PHILLIPS[1], KATHRYN O'SULLIVAN[2], & JENNY WALTON[2]

[1]*Food & Nutrition Research Group, Faculty of Education, Community & Leisure, Liverpool John Moores University, Liverpool, UK, and* [2]*Department of Nutrition and Scientific Affairs, Kellogg Company GB Ltd, Manchester, UK*

Abstract
Wheat bran is a concentrated source of insoluble fibre. Fibre intakes are generally lower than recommendations. This paper reviews the physiological effects of wheat bran and the health benefits it may provide in terms of the prevention of diseases such as colon and breast cancers, cardiovascular disease, obesity and gastrointestinal diseases. In recognition of the weight of evidence, the European Food Safety Authority has recently approved two health claims for wheat bran and gastrointestinal health.

Keywords: *dietary fibre, wheat bran, wheat germ, phytic acid, cancer, CVD, dietary antioxidants, gastrointestinal health, health claims*

Introduction

Wheat, a type of grass plant, is second only to rice as the main human food crop. Commercially, *Triticum aestivum vulgare* and *Triticum turgidum durum* (hard wheat, mainly used in pasta products) are of most importance (Macrae et al. 1993). The wheat grain or 'caryopsis', which is harvested for human nutrition, is composed of a number of different tissues: the germ (or embryo); the endosperm, which is packed with starch grains to provide energy for germination; the thick cell-walled aleurone layer, encasing the endosperm; and the pericarp. The bran fractions consist of the pericarp, testa, and hyaline and aleurone layers. By weight, the wheat caryopsis is composed of an outer branny husk (14–16% of the grain), the germ or embryo (2–3%), and the central endosperm (mainly starch: 81–84%) (Pomeranz 1988).

Conventional milling of wheat grains is based on separating the endosperm (which produces white flour when milled) from the bran layers and embryo. The aleurone cells, along with the other bran layers and the embryo, are removed to form the bran fraction. Although some processing is necessary for palatability, safety and even nutrient bioavailability (Topping 2007), there has been interest in the potential health benefits of high fibre food products for several years.

In terms of health, epidemiological (as well as experimental) evidence is accumulating to show that fibre may reduce the risk of certain chronic diseases, in particular cardiovascular disease (CVD), metabolic syndrome, type 2 diabetes and certain cancers (Fung 2002; Koh-Banerjee et al. 2004; Sayhoun et al. 2006; Seal 2006; de Munter et al. 2007; Schatzkin et al. 2007; Mellen et al. 2009). Although it has been suggested that it is the complex combination of components in the wholegrain matrix that may work together to impart health benefits (Slavin 2003; Fardet 2010), others have suggested the particular role of the bran component (Jensen et al. 2004). In several epidemiological studies, analysis of data obtained for whole grains and adjusted for intake of its constituents (namely cereal bran, germ and endosperm) showed an independent association only for the wheat bran component (Vitaglione et al. 2008).

Correspondence: Kathryn O'Sullivan, Department of Nutrition and Scientific Affairs, Kellogg Company GB Ltd, Kellogg Building, Talbot Rd, Manchester M16 OPU, UK. Tel: + 44 161 869200. E-mail: kathryn.O'sullivan@kellogg.com

ISSN 0963-7486 print/ISSN 1465-3478 online
DOI: 10.3109/09637486.2012.687366

Physiological effects of wheat bran and wheat germ

Nutritionally, bran fractions produced by milling are rich in fibre, minerals, vitamin B6, thiamine, folate and vitamin E and some phytochemicals, in particular antioxidants such as phenolic compounds (Shewry 2009). However, bioavailability is affected by the food matrix as well as processing conditions. Bran is used in the production of brown and wholemeal flours, hence retaining some of the valuable nutritional components that are depleted when these fractions are further removed in the refinement of white flour.

The physiological effects of wheat bran can be split into nutritional effects (from the nutrients present), mechanical effects (mainly on the gastrointestinal tract, due to the fibre content) and antioxidant effects (arising from the phytonutrients present such as phenolic acid and alkylresorcinols). Table I lists the major bioactive compounds found in wholegrain wheat, wheat bran and germ fractions.

Studies have reported that the majority of beneficial antioxidant phytochemicals (including phenolic acid and alkylresorcinols) in wholewheat grain are present in the germ/bran fractions. In wholegrain wheat flour, the bran/germ fractions contained 83% of total phenolic content (Adom et al. 2005). Consequently, the bran fraction has higher antioxidant activity than other milled fractions (Liyana-Pathirana and Shahidi 2007).

Although the antioxidant capacity of cereal fractions, such as wheat bran, is well reported, Perez-Jimenez and Saura-Calixto (2005) suggest that the antioxidant capacity may be underestimated in the literature, since the laboratory extraction methods used do not always allow for a complete release of antioxidant compounds, and non-extractable polyphenols (which may be released in the gut after colonic fermentation) with a high antioxidant capacity are also often ignored.

Both wheat variety and growing conditions can significantly alter the antioxidant profiles, concentrations and properties of compounds such as phenolic acids, carotenoids and tocopherols found in wheat brans (Zhou et al. 2004; Menga et al. 2010; Shewry et al. 2010), although wheat bran fractions seem to consistently retain their radical scavenging and chelating capacities. Additional work has shown that it is the aleurone layer (wheat bran fraction) that consistently has the highest antioxidant capacity among wheat fractions and that ferulic acid in particular (a derivative of the phenolic acid cinnamic acid) accounts for up to 60% of this antioxidant capacity (Mateo Anson et al. 2008; Vaher et al. 2010).

It has been suggested that the antioxidant phytochemicals found in wheat bran fractions may modulate cellular oxidative status and prevent biologically important molecules such as DNA, proteins and membrane lipids from oxidative damage, and that this consequently plays a role in reducing the risk of chronic diseases such as CVD and cancer (Zhou et al. 2004).

The phenolic antioxidants present in wheat bran have been shown to inhibit LDL oxidation, possibly by binding with apolipoprotein-B (Yu et al. 2005; Liyana-Pathirana and Shahidi 2007). Alkylresorcinols, another antioxidant found in wheat bran, has been shown to inhibit platelet binding to fibrinogen, stimulate thromboxane production and inhibit triglyceride formation, suggesting a potential role for phenolic compounds found in the bran fractions in CVD (Ross et al. 2004).

More recent work has also shown that wheat bran phenolic compounds, such as feruloyl oligosaccharides, protect against free radical-induced oxidative damage in human erythrocytes (Wang 2009).

Table I. Average content (g/100 g food) of bioactive compounds in wholegrain wheat and wheat bran and germ fractions.

Bioactive compound	Wholegrain wheat	Wheat bran	Wheat germ
α-Linoleic acid (18:3*n*−3)	−*	0.16	0.53
Sulphur compounds	0.5	0.7	1.2
Total free glutathione	0.007	0.038	0.27
Fibre (as AOAC)	13.2	44.6	17.7
Lignins	1.9	5.6	1.5
Oligosaccharides	1.9	3.7	10.1
Phytic acid	0.9	4.2	1.8
Minerals and trace elements	1.12	3.39	2.51
B vitamins	0.0091	0.0303	0.0123
Vitamin E (tocopherols and tocotrienols)	0.0047	0.0095	0.0271
Carotenoids	0.00034	0.00072	−*
Polyphenols	0.15	1.10	>0.37
Phenolic acids	0.11	1.07	>0.07
Flavonoids	0.037	0.028	0.300
Lignans	0.0004	0.0050	0.0005
Alkylresorcinol	0.07	0.27	−*
Phytosterols	0.08	0.16	0.43

Note: From Fardet (2010); * No data found.

Arabinoxylan is an important source of antioxidant phenolic compounds, including alkylresorcinols and phenolic acids (including ferulic acid). When delivered to the colon complexed with arabinoxylan, these phenolic compounds may be released by fermentation to have potentially beneficial effects (Vitaglione et al. 2008). Alkylresorcinols can be incorporated into cell membranes and play a role *in vivo*, in membrane function.

Ferulic acid like most of the phenolic compounds in wholegrain wheat fractions exists in bound form (approximately 76%), usually bound to arabinoxylans and other indigestible polysaccharides (Liu 2007; Mateo Anson et al. 2009). Although processing (such as thermal treatments and fermentation) may improve the release of ferulic acid and other bound phenolics, some authors question the availability of such phenolics for absorption in the gastrointestinal tract (Mateo Anson et al. 2009). Others suggest the importance of bound phytochemicals, and that unlike phytochemicals from fruit and vegetables (which are mainly in free or soluble conjugate forms and readily available in the upper gastrointestinal tract) they may have a more site-specific effect in the colon, suggesting that wheat bran may impart greater health benefits when consumed as part of a wider diet (Liu 2007). Although most of the phenolics in wheat bran are bound to cell wall materials that are difficult to digest, they may reach the colon in tact, where they undergo colonic digestion by microflora and consequently may exert their potential health benefits locally. For example, gastrointestinal esterase can release ferulic acid (a potent antioxidant) from bran (Andreasen et al. 2001). Saura-Calixto (2011) points out that the presence of bound antioxidants, such as polyphenols and carotenoids, in dietary fibre may significantly affect the physiological properties and health effect of dietary fibre, and that perhaps dietary fibre and antioxidants should be approached jointly in nutrition and health studies because of their common fate in the gut.

Other biologically important components in wholegrain fractions include sulphur containing amino acids (methionine and cystine). These two amino acids are found in higher levels in the wheat bran (0.6%) (Fardet 2010). Both methionine and cysteine are precursors of glutathione (an intracellular antioxidant) and contribute to the control of cell oxidative status (Metayer et al. 2008). Lignans, also found in wheat bran, have been shown to have anti-tumour properties in mice and human cells that may be mediated by cytostatic and apoptotic mechanisms (Qu et al. 2005).

Wheat bran and wheat germ fractions also contain almost all of the B-group vitamins: thiamine, riboflavin, niacin, pantothenic acid, pyridoxine, biotin and folates, with wheat bran and wheat germ fractions containing about 30.3 mg and 12.3 mg B vitamins/100 g respectively. They are also a source of vitamin E and the carotenoids. Other bioactives found in wholegrain fractions (such as ferulic acid, magnesium, zinc, copper, inositols, policosanol and melatonin) have also been suggested to have a role in promoting mental health (Fardet 2010).

Phytic acid and wheat bran

The bioavailability of minerals in wheat bran is under debate because of the presence of the 'anti–nutrient' phytic acid.

Phytic acid is a naturally occurring organic compound present in cereals, usually as myoinositol hexaphosphate. It is concentrated in the external covers in the pericarp and aleurone layer of the grain as well as, at lower levels, in the germ (Cheryan 1980); 90% of the phytic acid in grain is in the aleurone layer with 10% in the embryo (Dost and Tokul 2005). Consequently, the amount of phytic acid is greatly determined by the fractions removed during milling: white flour has almost no phytate. Wheat contains around 1.13% phytate (dry weight) (Cheryan 1980) or 3% expressed as gross product (Pointillart and Gueguen 1992).

Phytic acid content ranges from 200 to 400 mg/100 g in refined flour and 600–1000 mg/100 g in whole flour (Febles et al. 2002). In wheat bran, it ranges from 3116 to 5839 mg/100 g dry weight (Bilgicli and Ibanoglu 2007). An average 2–3 tablespoon serving of commercial wheat bran is estimated to contain 200–300 mg of phytic acid. There is a lack of data on average daily intakes of phytic acid. In Spain, where daily bread consumption is calculated at 151 g/day, phytic acid intake from bread is estimated at 159 mg/day (if white bread was consumed) to 350 mg/day (wholewheat bread) (Garcia-Estepa et al. 1999). On the basis of a similar calculation, phytic acid intake is estimated at 96 g/day (if white bread is consumed) to 211 mg/day (wholemeal bread).

Most of the minerals in wheat kernels are present as complexes with phytic acid. Mature wheat grain has high phytase activity, hydrolysing phytates and making the minerals nutritionally available (Brinch-Pedersen et al. 2002). However, the presence of phytate has been considered as an anti-nutrient in humans because of its effect on the bioavailability of iron, magnesium, zinc and calcium. While the mechanism is not entirely understood, it is suggested that phytic acid binds strongly with these mineral cations to form phytate–mineral complexes, changing their solubility, functionality absorption and digestibility (Rickard and Thompson 1997). Consequently, the complex cannot be absorbed or easily hydrolysed by the human body and so there is an adverse effect on bioavailability of minerals (Harland and Harland 1980).

Human studies have shown that the absorption of calcium, iron, magnesium and zinc is significantly lower in diets high in phytic acid (Kies 1985; Sandstrom and Lonnerdal 1989; Heaney et al. 1991; Larsson et al. 1996; Sandberg et al. 1999).

Historical studies have shown a decreased absorption of calcium after phytic acid was added to white bread (Harris 1955). More recently, studies have shown that wheat bran, when digested with milk (e.g. as breakfast cereal), reduces the absorbability of the calcium in milk (Weaver et al. 1991), possibly through physical entrapment, adsorption or ionic binding. Weaver et al. (1996) showed, in a randomized cross-over study of healthy women, that with comparable calcium load, fractional calcium absorption was significantly lower when the diet was supplemented with 16 g/day wheat bran; the impact of wheat bran on calcium availability continued even at calcium loads as high as 75 mmol, suggesting that the ability of wheat bran to bind calcium is not saturated at doses higher than those provided by most meals. Consequently, it has been suggested that high intakes of insoluble fibre may contribute to osteoporosis. Whether this is of physiological significance in terms of bone health has been explored further. Short-term (4 weeks) and longer-term (2 years) studies have demonstrated that wheat bran in the diet had no significant effect on bone turnover markers in young and older women (Zitterman et al. 1999; Chen et al. 2004). Therefore, despite the negative effects of wheat bran on calcium bioavailability, there is insufficient evidence to suggest that it has any detrimental effect on bone metabolism, causing accelerated bone loss.

Zinc absorption follows a dose-dependent response with phytate (Navert et al. 1985). Hunt et al. (2008) showed that diets higher in phytic acid may necessitate greater intakes of zinc, and suggested that humans only minimally adapt to increase zinc absorption from diets high in phytic acid with a phytate:zinc ratio $>15-20$. However, if the diet is low in phytic acid, absorption of zinc is up-regulated when zinc intakes are habitually low. Currently, US and Canadian diets have a typical phytate: zinc ratio of 2, which would make the current zinc intakes of 7.1 mg (women) and 9.0 mg (men) adequate. Meeting current US recommendations on increasing wholegrain consumption would lead to a phytate: zinc ratio of 8, which Hunt et al. (2008) calculate to equate to a zinc intake of 9.2 mg and 13.4 mg for women and men respectively. There are currently no data available on the UK dietary phytate: zinc ratio, but assuming it is similar to the US ratio, current UK intakes of zinc (7.8 mg and 10.1 mg for women and men respectively; meeting the RNI of 7.0 and 9.5); it could be anticipated that increasing consumption of whole grains (and, consequently, a higher wheat bran intake) could have a negative impact on zinc status in UK adults: RNIs may need to be adjusted accordingly.

There is no consensus on the effect of wheat bran on iron bioavailability. Animal studies have shown that iron from wheat bran forms a monoferric phytate, which is highly available, possibly because it remains soluble at intestinal pH and hydrolyses to aid absorption, whereas other iron–phytate complexes (e.g. from oat bran) have been shown to be unavailable (Fly and Czarnecki-Maulden 1996). At moderate levels (providing 2–4% of total dietary fibre), the authors concluded that iron from wheat bran is readily available. However, some human studies have shown a decrease in iron retention when fed wheat bran (Cook et al. 1983; Hallberg et al. 1987), while others like Dintzis et al. (1985) showed no change. Indeed, according to Gibson et al. (2006), phytic acid begins to lose its inhibitory effect on iron at ratios of $< 1.0{:}1.0$ (phytate:iron), but the effect is still present as low as 0.2:1.0.

There is a potential public health nutrition problem of iron and zinc deficiency for populations whose diets are mainly cereal and legume-based, or those whose diets are marginal in essential minerals (Raboy 2001). Vegetarians, whose intakes of phytate are typically higher than those of omnivores, appear to be most affected by phytate-containing foods as despite having similar intakes of, e.g., iron and zinc as omnivores the bioavailability may be compromised (Agte et al. 1999). Nevertheless, most studies of vegetarians indicate that iron and zinc status is adequate, and it appears that there may be some degree of physiological adaption of the gastrointestinal tract to increase absorption of trace elements and so overcome the presence of phytic acid (Gibson 1994).

The degree to which phytic acid can influence nutritional status, however, is dependant on a number of food and host-related factors: the amount digested in the gut, the content of phytate and minerals in the food and the nutritional status (replete or deficient in a nutrient), and the overall diet of the individual can all have an impact on mineral bioavailability (McKevith 2004).

Processing significantly affects the bioavailability of minerals in the presence of phytates; germination, fermentation and baking, leading to phytate hydrolysis, have been shown to have a beneficial impact on mineral bioavailability (Watzke 1998).

Animal studies have shown that other components of fibre, inulin and oligofructose, act as potent enhancers of mineral bioavailability in plant-derived foods and human studies, have partially confirmed these findings (Coudray et al. 1997; Van den Heuvel et al. 1999; Vitali et al. 2008). Furthermore, preliminary human studies have also shown that carotenoids (lycopene, lutein and zeaxanthin) increased iron absorption from a wheat-based breakfast by 8.4–14.4% (Garcia-Casal 2006), but this used white wheat flour, likely to be low in phytate.

However, the presence of phytic acid does not only imply nutritional problems. In contrast to the reported anti-nutrient properties of phytic acid, studies have shown a potentially beneficial role for the compound. In particular, phytic acid is reported to lead to delayed post-prandial absorption (Yoon et al. 1983), decrease in plasma cholesterol and triglycerides (Katayama 1995), inhibition of hypercalcuria and renal stone

development (Grases et al. 1998, 2000) and anti-carcinogenic effects (Thomson and Zhang 1991; Huang et al. 1997), the latter being purported to be owing to antioxidant action. The antioxidant functions of phytic acid are thought to result mainly from its iron and copper chelating properties, although mechanisms are not yet fully understood (Minihane and Rimbach 2002).

Dietary fibre and wheat bran

The fibre content of the wholewheat grain ranges from 11.6% to 12.7% dry weight (Carson and Edwards 2009). Most of the fibre that is in the outer layers of the grain (pericarp and seed coat) is typically called wheat bran. It is one of the richest sources of fibre, 46% is non-starch polysaccharide (NSP). The main NSPs present are arabinoxylan, cellulose and beta-glucan that are respectively 70%, 24% and 6% of the NSP of the bran (Maes and Delcour 2002). The concentration of soluble fibre in wheat is significantly less than in other cereals, e.g. barley and oats, 3–11% and 3–7% respectively, compared with less than 1% in wheat (dry weight) (Wood 1997). The amount and type of fibre in wheat, and specifically in wheat bran, is shown in Table II.

Wheat consumption in the diet

Cereals are staple foods in western countries, and typically contribute about 50% of dietary fibre intake (Lambo et al. 2005). The recommendation that cereals form the basis of the diet is well recognized in the majority of countries with dietary guidelines including most EU member states, the USA and Canada.

Table III shows the cereal and cereal product consumption by adults in France and also the % contribution to average intakes of energy and fibre (AFSSA 2009). Similar data for UK adults (Hoare et al. 2004) show that cereal products contribute 37% of fibre intake and 28% of total kcal intake, while in Belgium it is estimated that average consumption is 186.5 g/day, with cereal and cereal products contributing 22.3% to total energy and 34% to fibre intakes (De Vriese et al. 2005).

Wheat is the most heavily consumed grain in the world. For example, in the UK wheat consumption is more than 10 times as much as rice (which is mostly consumed as white rice), and oats (419,000 tonnes in 2008/2009), or maize (305,000 tonnes). Usage of wheat for flour and starch milling in 2008/2009 was 6.1 million tonnes. Total human and industrial consumption of wheat for the same year was estimated at 6.836 million tonnes (FAO 2010), implying wheat consumption of nearly 110 kg per capita. Data for consumption of wheat bran *per se* are not currently available; however, bran for human consumption is produced by flour millers rather than other wheat users. It is estimated that it accounts for approximately 10% of their total output of co-products (i.e. products other than flour); this would amount to 112,000 tonnes in 2008/2009 (Alex Waugh, NABIM, personal communication). Wheat is consumed in Europe typically as bread, pasta, breakfast cereals and biscuits, cakes and pastries. With information taken from manufacturer's webites, Table IV shows sources of wheat bran in the diet.

Table II. Fibre concentration in wheat and wheat bran.

	Total fibre (g/100 g)	Insoluble fibre (g/100 g)	Soluble fibre (g/100 g)
Wheat grain	11.6–17.0	10.2–14.7	1.4–2.3
Wheat bran	36.5–52.4	35.0–48.4	1.5–4.0

Note: From Vitaglione et al. (2008).

Dietary fibre and wheat bran intakes

The European-recommended dietary fibre intake is 25 g/day based on the AOAC method. In the UK, fibre recommendations based on NSP are set at 18 g/day. For children, this varies and the general rule for health care professionals is age of child plus 5 g. Average dietary fibre intakes in Europe range from 10 to 20 g/day in young children and from 16 to 29 g/day in adults (European Food Safety Authority [EFSA] 2010). These recommendations cover total fibre intake and there is currently no established guidelines that differentiate between fibre type and source.

Fibre intakes are ascertained through dietary survey either a full dietary recall including portion sizes, diet diaries or food frequency questionnaires. These methods are often time-consuming and when dealing with a large dataset take a great deal of time, effort, attention to detail and consistency of methodology. The DINE questionnaire (Roe et al. 1994) was developed to be administered and scored in under 10 minutes by primary care staff without specialized nutritional knowledge. The questionnaire gives an output of low, medium or high for fat and fibre intakes. This validated questionnaire is in wide use and is currently being modified to provide a more numeric value for fibre intake based on more modern AOAC fibre values.

The majority of data from cohort and epidemiological studies used to demonstrate the potential health benefits of wholegrain consumption have been derived from dietary assessment methods that were not originally designed to quantify wholegrain intakes – the majority relied on semi-quantitative food frequency questionnaires using a limited range of wholegrain foods with varying descriptions of wholegrain foods (Seal 2006).

Wheat bran and health benefits

Studies have shown that wheat bran may have a beneficial effect on the prevention of diseases,

Table III. Cereal products consumption and % contribution to energy and fibre intakes by French adults.

		Consumption (g/person/day)				
	Age (years)	Males	Females	Total	% contribution to energy	% contribution to fibre
Bread and rusk	18–34	104.1	70.3	83.8	14.7	20.8
	35–54	149.7	89.8	117.0		
	55–79	163.5	101.7	136.1		
Breakfast cereals	18–34	6.7	7.9	7.4	0.9	1.7
	35–54	3.5	4.7	4.1		
	55–79	4.1	3.2	3.7		
Pastas	18–34	64.7	39.2	49.3	2.0	3.5
	35–54	47.5	28.5	37.1		
	55–79	35.8	22.8	30.1		
Rice and hard wheat/semolina	18–34	33.9	21.7	26.5	1.4	0.9
	35–54	32.9	24.1	28.1		
	55–79	22.1	16.7	19.7		
Other cereals	18–34	0.3	0.7	0.6	0.1	0.2
	35–54	0.7	0.8	0.7		
	55–79	0.3	0.4	0.3		
Pastry/bakery products	18–34	20.6	14.3	16.8	2.1	1.5
	35–54	13.6	11.5	12.4		
	55–79	8.6	6.0	7.4		
Sweet and savoury biscuits/bars	18–34	15.3	14.5	14.8	2.0	1.6
	35–54	8.8	8.1	8.4		
	55–79	5.7	4.7	5.3		
Cakes	18–34	42.2	35.6	38.2	6.0	4.0
	35–54	41.8	38.6	40.0		
	55–79	32.0	37.1	34.2		

Note: From AFSSA (2009).

including some cancers (in particular colorectal cancer), CVD, obesity and some gastrointestinal diseases, including diverticular disease, constipation and irritable bowel syndrome (IBS) (Fardet 2010).

Wheat bran and cancer

Bowel cancer is a major cause of mortality in the UK. The European Prospective Investigation into Cancer and Nutrition (EPIC) (Bingham et al. 2003) recommended that people eating low fibre diets could significantly reduce risk of colorectal cancer, by 40%, by eating more fibre-rich foods. Similarly, the World Cancer Research Fund's report on cancer and diet, physical activity and weight suggested that foods containing fibre decrease risk of colorectal cancer (WCRF/AICR 2007).

The Familial Adenomatous Polyposis Trial (De Cosse et al. 1989) is a randomized controlled trial that investigated the effects of wheat bran fibre, with or without supplements of ascorbic acid and alpha-tocopherol, on development of rectal adenomas in patients with familial adenomatous polyposis (precursor to colorectal cancer). After 4 years of follow-up,

Table IV. Sources of wheat bran in the diet.

Product	Fibre per 100 g AOAC	Standard portion size (g)	Fibre per portion (g)
Bran shreds breakfast cereal	27	40	10.8
Bran flakes breakfast cereal;	15	30	4.5
Shredded wheat breakfast cereal	11.8	45	5.3
Extruded pillos breakfast cereal	9.9	45	4.5
Whole wheat biscuits breakfast cereal	10	37.5	3.8
Wholemeal bread	7	36	2.5
Granary bread	5.3	36	1.9
Brown bread	5	36	1.8
Sliced white bread	2.5	36	0.9
Dried wholemeal pasta (uncooked weight)	9	44	4.0
Dried wholemeal pasta (uncooked weight)	9	87	7.8
Dried wholemeal pasta (uncooked weight)	9	130	11.7
Dried white pasta (uncooked weight)	2.8	75	2.1
White pasta (cooked weight)	1.2	180	2.2
Wheat couscous (dry weight)	5	60	3.0

Note: Compiled from Manufacturer's websites 2011.

there was some evidence of decreased number of polyps and therefore reduced risk of cancer where patients consumed at least 50% of their prescribed fibre.

In another randomized control trial of patients following colonic polyp removal surgery, Alberts et al. (1996) found that a diet supplemented with wheat bran cereal (13.5 g/day) reduced faecal bile acid concentration. Bile acids are considered to play a role in colorectal cancer risk. However, later follow-up studies, after 3 years (Alberts et al. 1997), failed to show any significant effect of the high wheat bran diet on the development of colorectal adenomas.

The Australian Polyp Prevention Project (Maclennan et al. 1995) reported that a low fat wheat bran supplemented diet (25 g wheat bran/day) reduced the incidence of large colorectal adenomas, suggesting that wheat bran, alongside a low-fat diet, inhibits the development of malignant adenomas. These studies support, to some extent, that a wheat bran supplemented diet may be protective against colorectal cancer and polyps.

Furthermore, animal studies have demonstrated a significant protective effect of wheat bran on colon carcinogenesis in rats fed a high-fat western-style diet (Alabaster et al. 1996). Wheat bran, in addition to psyllium (50:50), led to enhanced protection and synergistic effects may inhibit different phases of the carcinogenic process, with wheat bran phytic acid inhibiting earlier stages and psyllium inhibiting later stages (Alabaster et al. 1993).

The protective mechanisms of wheat bran, especially in terms of colon cancer, fall into three categories (Lupton and Turner 1999). The first is the established effect on dilution of potential carcinogens and promoters of carcinogens – a more bulky stool reduces access to the cells lining the colon. Second, it is well established that wheat bran accelerates transit of faecal material through the colon, such that rapid transit reduces access of the colonic epithelial cells to faecal constituents. However, not all fibre has the same ability to dilute contents of the lumen, or the same potential to accelerate colonic transit. Animal models have shown that wheat bran is the best diluter and has the shortest transit time compared with pectin, guar gum, oat bran and cellulose (Gazzaniga and Lupton 1987; Lupton and Meacher 1988).

The third is the effect of fermentation of wheat bran to Short Chain Fatty Acids (SCFA) (including butyric acid) throughout the colon. Studies have found that SCFAs may modulate carcinogenesis through their effects on proliferation, differentiation and apoptosis of colonocytes, as well as stimulation of the immune system (Topping and Clifton 2001; Schley and Field 2002).

Clausen et al. (1991) have shown that wheat bran doubles the production of SCFAs and *in vitro* fermentation, resulted in reduced production of butyrate in subjects with colonic adenomas or suffering from colon cancers. Rat model studies have shown that butyric acid may stimulate, rather than inhibit colonic epithelial cell proliferation (Lupton and Kurtz 1993). The authors concluded that the effect of SCFAs on colonocytes is different in normal cells from transformed cells. Butyrate seems to inhibit growth of transferred cells while enhancing proliferation in normal human and rat mucosa.

An animal model study (Zoran et al. 1997) showed that although oat bran produces more butyric acid in the colon, wheat bran is more protective against colon carcinogenesis, reducing incidence of tumours. In a study involving patients with a history of colon cancer fed 13.5 g of wheat bran fibre for 2 months, Alberts et al. (1990) concluded that wheat bran fibre supplementation can inhibit DNA synthesis and epithelial cell proliferation within rectal mucosa crypts of patients at high risk for colon cancer.

Finally, studies have shown that phytic acid may block PI-3 kinase activation, which accelerates apoptosis and may be protective against colorectal cancer development (Huang et al. 1997). Other data suggest that some of the phytochemical compounds in wheat bran, including beta-sitosterol, may also have a beneficial effect on colon cancer (Waliszewski et al. 1997)

Colon cancer may not be the only cancer linked to fibre. A potentially protective effect of fibre has also been observed in breast cancer research. Cade et al. (2007) suggest that in pre-menopausal women total fibre is protective against breast cancer, in particular fibre from cereals and possibly fruit. Their work that involved analysis of a large cohort of adult women found that in pre-menopausal, but not post-menopausal, women a statistically significant inverse relationship was found between total fibre intake and risk of breast cancer. The top quintile of fibre intake was associated with a hazard ratio of 0.48 [95% confidence interval (CI) 0.24–0.96] compared with the lowest quintile. Pre-menopausally, fibre from cereals was inversely associated with risk of breast cancer and fibre from fruit had a borderline inverse relationship. Mechanisms of effect are postulative, but plausible mechanisms could be the role in fibre and weight management and the potential for fibre to bind with estrogens (Goldin et al. 1986; Rose et al. 1991; Stoll 1996).

Wheat bran and CVD

The cardiovascular benefits from wholegrains are only supported where the wholegrain contains significant amounts of fibre or bran. In a double-blind placebo-controlled crossover study of wholegrain cereal and wheat bran, Costabile et al. (2008) reported a significant reduction in total serum cholesterol. After consuming a wheat bran-based breakfast cereal for 3 weeks, containing approximately 13.5 g of fibre, serum cholesterol was reduced from 5.576 to

4.385 mmol/l in those participants with the highest quintile of serum cholesterol. Furthermore, no reduction was found in beneficial HDL-C, suggesting beneficial effects on CVD risk.

In a cross-sectional analysis of US adolescents, Carlson et al. (2011) found that increasing dietary fibre was associated with lower risk of metabolic syndrome. In a prospective cohort study of 42,850 males, Jensen et al. (2004) examined the effect of added bran in the diet, in addition to wholegrain intakes, on risk of coronary heart disease (CHD). However, while the type of bran was not identified, the authors reported that it was predominantly wheat and oat bran. The relative risk of CHD in men with the highest intake of added bran was 0.70 (95% CI: 0.60–0.82) compared with men who had no added bran. He et al. (2010) recently reported on the mortality and CVD-specific mortality of 7822 women with type 2 diabetes from the Nurses Health Study. Compared with those women with the lowest intake of bran, women with the highest intake have all-cause mortality relative risk of 0.72 (95% CI: 0.56–0.92) and relative risk for CVD-specific mortality was 0.65 (95% CI: 0.43–0.99).

Wheat bran and obesity

Evidence from epidemiological studies suggests an inverse relationship between intake of dietary fibre and weight gain and obesity, while fibre consumption is associated with increased satiety and decreased energy intake (Freeland et al. 2009). It has also been proposed that dietary fibre increases faecal energy loss (Astrup et al. 2010). Viscous fibre is thought to exert the greatest effect on appetite regulation, but studies using wheat bran have also reported a reduction in food intake following a test meal with wheat bran, but it is not clear whether this effect is long-lasting in terms of management of obesity (Freeland et al. 2009).

The effect of wheat bran on postprandial appetite-regulating hormones is less well studied, although a recent animal study (Neyrinck et al. 2008) investigated the effect of wheat bran on GLP-1 secretion, and found no effect on body weight, adipose tissue mass, glucose or insulin resistance. However, this study did demonstrate an impact of wheat bran on inflammation, including decreased inflammatory cytokines.

Wheat bran and digestive health

In terms of digestive health, wheat bran can offer several beneficial effects. Wheat bran has an effect on faecal bulking, delays gastric emptying and accelerates small bowel transit (McIntyre et al. 1997). Generally, faecal bulking has been linked with a number of potentially beneficial effects as summarized in Table V.

Faecal bulk is a result of multiple interactions between the food, the host and the gut ecosystem (Eastwood 1993). The bulking effects of fibre are greatest with cereal fibre, especially products high in insoluble NSP such as wheat bran (Topping 2007) Wheat bran is so effective at faecal bulking that it is the reference against which other foods are measured for their faecal bulking efficiency (Monro 2002).

The mechanism by which wheat bran increases stool weight has been studied by Chen et al. (1998), who showed that a 30 g/day supplement was associated with a mean wet stool weight increase of 52.4 g/day. Wheat bran was shown to increase faecal concentrations of sugars (glucose, arabinose and xylose) and mass of plant material more than oat bran, although oat bran had a greater effect on increasing bacterial mass. With wheat bran, the increase in stool weight was largely a result of undigested plant fibre (50–60%) but increased bacteria contributed 12–17% of the increase. These studies reported no increase in the proportion of water in the stool, which supports other reports (Cummings 1993).

Both the European Food Safety Authority and the UK Scientific Advisory Committee conclude that the mean increase in daily faecal weight is approximately 5 g per 1 g wheat bran consumed compared to other fibres such as fruit and vegetables (4.1 g/g), gums

Table V. Effects of faecal bulking.

Property/effect	Consequent effect
Bulkiness	Bulk transfer
Bulk transfer	Toxin removed, colonic exposure reduced, decreased transit time
	Replenishment of substrates for fermentation – decreased colon cancer risk
Decreased transit time	Less protein putrefaction to harmful products – decreased colon cancer risk
	Less time for dehydration and stool hardening
Increased water load	Diluted colon contents, stool softening
	Pressure distribution – decreased risk of diverticulitis and haemorrhoids
Replenishment	Provides substrate for bacterial growth.
	Butyrate produced by fermentation protects against colorectal cancer.
	Short chain fatty acid production decreases solubility of carcinogenic bile acids
Binding	Decreased toxin / carcinogen activity
Pressure distribution	Reduces risk of diverticulitis and haemorrhoids by reducing pressure points
Distension	Stimulates defaecation, preventing stagnation
Defaecation	Digestive Comfort and continued flow, sense of well-being

Note: From Monro (2002).

including psyllium (4 g/g), soya products (2.5 g/g) and pectin least of all (1.2 g/g) (SACN 2008).

Transit time is also affected by wheat bran. Payler et al. (1975) showed that adding 20 g/day of wheat bran reduced transit time from 2.75 to 2.0 days, and the authors also confirmed that while bran accelerates slow transit time, it may also slow down fast transit time (that is less than 1 day). The likely mechanism for the increase in transit time is the high content of cellulose and hemicelluloses in wheat bran, which soften and expand the stool.

Insoluble fibre (the main component of wheat bran) is not readily broken down by gastrointestinal microflora and it increases faecal bulk, shortening colonic transit time. Soluble fibre dissolves in water to form gel and may be digested by the colonic microflora, increasing bacterial numbers and thus increasing bulk.

Wheat bran and IBS

Increasing fibre intake has been suggested as an initial treatment for IBS, although there are conflicting data on its effectiveness. Conversely, wheat is often associated with increased symptoms and reducing, but not necessarily excluding, wheat intake may be beneficial in IBS management (NICE 2008). Nevertheless, Bijkerk et al. (2003, 2009) reported that most GPs recommend an increase in fibre for patients with IBS, advising the addition of insoluble fibre in the form of bran. A more recent randomized controlled trial showed that while soluble fibre (psyllium) was effective at reducing symptoms of IBS, wheat bran (20 g/day) was not, especially at the onset of treatment (Bijkerk et al. 2009).

A meta-analysis of the effect of wheat bran on stool weight and transit time (Muller-Lissner 1988) showed that wheat bran increased stool weight and decreased transit time in healthy controls and patients with IBS and chronic constipation. The NICE (2008) guideline consensus on IBS states that wheat bran should not be recommended for people with IBS as it is ineffective in the management of symptoms, and may even increase symptoms for some people – an increase in fibre, if needed should be in the form of soluble fibre.

Other gut conditions, however, may be helped by wheat bran. Painter et al. (1972) showed that there was marked relief from symptoms of diverticular disease with a high residue, low sugar diet, including 12–14 g of raw unprocessed wheat bran per day (range: 3–45 g). More recently, a Cochrane review of evidence has found that adding more wheat and bran fibre to the diet was effective at alleviating constipation during pregnancy, increasing frequency of defaecation, and is preferable to taking stimulant laxatives, which may have undesired side effects (Jewell and Young 2001).

Wheat bran as a prebiotic

Colonic microflora has a profound effect on health. The gut flora components can be modified by dietary means, such as increasing prebiotic intake. Prebiotics are defined as non-digestible food ingredients that beneficially affect host health by selectively stimulating the growth and/or activity of one or a limited number of bacteria in the colon (Gibson and Roberfroid 1995). It is not the prebiotic itself that brings about change, but its effect on the gut microflora (Wang et al. 2009). There is a growing body of evidence to support the beneficial health effects of prebiotics on bowel health and risk of colon cancer and CVD (Costabile et al. 2008). Prebiotic components of dietary fibre in wheat bran (including beta-glucans) may be fermented by colonic microflora to form SCFA, resulting in physiological changes to the colonic contents, affecting bulking, water retention capacity and viscosity. Butyric acid is one such SCFA that has been recognized as a fuel for colonocytes and also contributes to faecal pH, influencing colonic function

Costabile et al. (2008) found a more modest change in gut microflora after consuming a wheat bran cereal, compared with a wholegrain cereal. Markers of colonic metabolic output (including ferulic acid and SCFAs) also increased.

Wheat bran and health claims within the European Union

The EFSA has been tasked to preapprove any nutrition and health claim for market in the EU. In this relatively early stage of the legislation, the EFSA is working through a list of over 40,000 so-called 'generally accepted claims'. Eighty percent of these claims have been rejected on several grounds from insufficient characterization of the food to cause and effect relationship not being established. In October 2010, the EFSA panel passed opinion on two claims pertaining to the benefits of wheat bran. The two approved claims are as follows:

> Increase in faecal bulk
> The claimed effect is 'intestinal health: faecal bulking'. The target population is assumed to be the general population. The panel considers that an increase in faecal bulk might be a beneficial physiological effect. In weighing the evidence, the panel took into account that the majority of the human intervention studies showed a consistent effect of wheat bran fibre on faecal bulk and that no threshold dose for the effect can be established. A linear dose dependent relationship was demonstrated in several studies.
> The claimed effects are 'gut health' and 'intestinal transit time, intestinal health'. The target population is assumed to be the general population. In the context of the clarifications provided by Member States, the panel assumes that the claimed effect refers to a reduction in intestinal transit time. The panel considers that a reduction in intestinal transit time within the normal range might be a

beneficial physiological effect. In weighing the evidence, the panel took into account that the studies provided consistently indicated that wheat bran fibre consumed at an amount of at least 10 g/day decreased intestinal transit time.

Wheat bran is easy to characterize and the evidence for the effect on stool weight and transit time is unequivocal. The conditions of use for the claim, i.e. the amount manufacturers need to have in the food in order to make the claim, still need to be approved by the European Commission.

Summary

In conclusion like many European countries, UK dietary intakes of fibre are in region of 13 g versus the dietary recommendation of 18 g (25 g AOAC). Promotion of foods high in wheat bran will help achieve this recommendation. Given the prevalence of constipation, less than desirable stool weight and slow digestive transit health recommendations should promote and include advice on wheat bran fibre as it is established to be the benchmark in promoting laxation and more expedient transit time. This is endorsed by European Food Safety Authority who has recently approved two health claims for wheat bran for faecal bulking and transit time. In addition, there is now strong evidence that fibre, and in particular wheat bran fibre, may have health benefits in terms of prevention of diet-related diseases.

Declaration of interest: Kellogg Company and Liverpool John Moores University collaborated on the preparation of this article. Liverpool John Moores University received a financial grant for the contribution of their time.

References

Adom K, Sorrels M, Liu RH. 2005. Phytochemicals and antioxidant activity of milled fractions of different wheat varieties. J Agric Food Chem 53:2297–2306.

AFSSA. 2009. Report of the 2006/2007 Individual and National Survey on Food Consumption 2 (INCA 2). Available at: http://www.anses.fr/Documents/PASER-Ra-INCA2.pdf. Last accessed March 2012.

Agte VV, Tarwadi KV, Chiplonkar SA. 1999. Phytate degradation during traditional cooking: significance of the phytic acid profile in cereal-based vegetarian meals. J Food Compos Anal 12: 161–167.

Alabaster O, Tang Z, Frost A, Shivapurkar N. 1993. Potential synergism between wheat bran and psyllium: enhanced inhibition of colon cancer. Cancer Lett 75:53–58.

Alabaster O, Tang Z, Shivapurkar N. 1996. Dietary fibre and the chemopreventive modulation of colon carcinogenesis. Mutat Res 350:185–197.

Alberts DS, Einspahr J, Rees-McGee S, Ramanujam P, Buller MK, Clark L, et al. 1990. Effects of dietary wheat bran fiber on rectal epithelial cell proliferation in patients with resection for colorectal cancers. J Natl Cancer Inst 82(15):1280–1285.

Alberts DS, Einspahr J, Ritenbaugh C. 1997. The effect of wheat bran fibre and calcium supplementation on rectal mucosal proliferation rates in patients with resected adenomatous polyps. Cancer Epidemiol Biomarkers Prev 6:161–169.

Alberts DS, Ritenbaugh C, Story JA. 1996. Randomised double blinded placebo controlled study of the effect of wheat bran fibre and calcium on faecal bile acids in patients with respected adenomatous colon polyps. J Natl Cancer Inst 88:81–92.

Andreasen MF, Kroon PA, Williamson G, Garcia-Conesa MT. 2001. Intestinal release and uptake of phenolic antioxidant diferulic acids. Free Radical Biol Med 31:304–314.

Astrup A, Kristensen NT, Gregersen A, Belza A, Lorenzen JK, Due A, Larsen TM. 2010. Can bioactive foods affect obesity. Ann N Y Acad Sci 1190:25–41.

Bijkerk CJ, de Wit NJ, Muris JWM, Whorwell PJ, Knottnerus JA, Hoes AW. 2009. Soluble or insoluble fibre in irritable bowel syndrome in primary care? Randomised placebo controlled trial. Br Med J 339:b3154.

Bijkerk CJ, de Wit NJ, Stalman WAB, Knottnerus JA, Hoes AW, Muris JWM. 2003. Irritable bowel syndrome in primary care: the patient's and doctors' views on symptoms, etiology and management. Can J Gastroenterol 17:363–368.

Bilgicli N, Ibanoglu S. 2007. Effect of wheat germ and wheat bran on the fermentation activity, phytic acid content and colour of tarhana. J Food Eng 78:681–686.

Bingham SA, Day NE, Luben R. 2003. Dietary fibre in food and protection against colorectal cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC): an observational study. Lancet 361:1496–1501.

Brinch-Pedersen H, Lisbeth DS, Preben BH. 2002. Engineering crop plants: getting a handle on phosphate. Trends Plant Sci 7: 118–124.

Cade JE, Burley VJ, Greenwood DC. 2007. Dietary fibre and risk of breast cancer in the UK Women's Cohort Study. Int J Epidemiol 36(2):431–438.

Carson GR, Edwards NM. 2009. Criteria of wheat and flour quality. In: Khan K, Shewry P, editors. Wheat chemistry and technology. 4th ed., St Paul, MN: American Association of Cereal Chemists. pp 97–118.

Carlson JJ, Eisenmann JC, Norman GJ, Oritz KA, Young PC. 2011. Dietary fibre and nutrient density are inversely associated with metabolic syndrome in US adolescents. J Am Diet Assoc 111: 1688–1695.

Chen HL, Haack VS, Janecky CW, Vollendorf NW, Marlett JA. 1998. Mechanisms by which wheat bran and oat bran increase stool weight in humans. Am J Clin Nutr 68:711–719.

Chen Z, Stini WA, Marshall JR, Martínez ME, Guillén-Rodríguez JM, Roe D, Alberts DS. 2004. Wheat bran fiber supplementation and bone loss among older people. Nutrition 20:747–751.

Cheryan M. 1980. Phytic acid interactions in food systems. Crit Rev Food Sci Nutr 13:297–335.

Clausen MR, Bonnen H, Mortensen PB. 1991. Colonic fermentation of dietary fibre to short chain fatty acids in patients with adenomatous polyps and colonic cancer. Gut 32:923–928.

Cook JD, Noble NL, Morck TA, Lynch SR, Petersburg SJ. 1983. Effect of fiber on nonheme iron absorption. Gastroenterology 85: 1354–1358.

Costabile A, Klinder A, Fava F, Napolitano A, Fogliano V, Leonard C. 2008. Wholegrain wheat breakfast cereal has a prebiotic effect on the human gut microbiota. Br J Nutr 99:110–120.

Coudray C, Bellanger J, Castiglia-Delavaud C, Remesy C, Vermorel V, Rayssignuier Y. 1997. Effect of soluble or partly soluble dietary fibres supplementation on absorption and balance of magnesium, calcium, iron and zinc in healthy young men. Eur J Clin Nutr 51:349–365.

Cummings JH. 1993. The effect of dietary fiber on fecal weight and composition. In: Spiller GA, editor. Dietary fibre in human nutrition. Boca Raton, FL: CRC Press. p 263–350.

De Cosse JJ, Miller HH, Lesser ML. 1989. Effect of wheat fibre and vitamins C and E on rectal polyps in patients with familial adenomatous polyposis. J Natl Cancer Inst 81:1290–1297.

De Vriese S, De Backer G, De Henauw S, Huybrechts I, Kornitzer K, Leveque A, et al. 2005. The Belgian Food Consumption survey: aims, design and methods. Arch Public Health 63:1–16.

de Munter JS, Hu F, Spiegelman D, Franz M, van Dam RM. 2007. Wholegrain, bran and germ intake and risk of type 2 diabetes: a prospective cohort study and systematic review. PLoS Med 4: 1389–1395.

Dintzis FR, Watson PR, Stanstead HH. 1985. Mineral content of brans passed through the human GI tract. Am J Clin Nutr 41: 901–908.

Dost K, Tokul O. 2005. Determination of phytic acid in wheat and wheat products by reverse phase high performance liquid chromatography. Anal Chim Acta 558:22–27.

Eastwood M. 1993. Diet fibre and colorectal disease: a critical appraisal. In: Phillips SF, Pemberton JH, Shorter RG, editors. The large intestine: physiology, pathophysiology and disease. New York: Raven Press. pp 209–222.

EFSA. 2010. Scientific opinion on the substantiation of health claims related to wheat bran fibre and increase in faecal bulk (ID3066) reduction in intestinal transit time (ID 828, 839, 3067, 4699) and contribution to the maintenance or achievement of a normal body weight (ID 829) pursuant to Article 13(1) of Regulation (EC) No 1924/20061. EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA) 2.3. EFSA J 8(10): 1817.

FAO. 2010. Food Outlook. Global Market Analysis. http://www.fao.org/docrep/011/ai482e/ai482e03.htm#32. Accessed 14 July 2010.

Fardet A. 2010. New hypotheses for the health protective mechanisms of whole-grain cereals: what is beyond fibre? Nutr Res Rev 23:65–134.

Febles CI, Arias A, Hardisson A, Rodriguez-Alvarez C, Sierra A. 2002. Phytic acid level in wheat flours. J Cereal Sci 36:19–23.

Fly AD, Czarnecki-Maulden GL. 1996. Iron bioavailability from diets containing high-fibre breakfast cereals and crackers. Nutr Res 16:267–278.

(Food Standards Agency). Hoare J, Henderson L, Bates C, Prentice A, Birch M, Swan G, Farron M. 2004. The National Diet and Nutrition Survey (NDNS): adults age 19–64. Volume 5. London: HMSO.

Freeland KR, Anderson GH, Wolever TMS. 2009. Acute effects of dietary fibre and glycaemic carbohydrate on appetite and food intake in healthy males. Appetite 52:58–64.

Fung T. 2002. Whole-grain intake and the risk of type 2 diabetes: a prospective study in men. Am J Clin Nutr 76:535–540.

Garcia-Casal MN. 2006. Carotenoids increase iron absorption from cereal-based food in the human. Nutr Res 26:340–344.

Garcia-Estepa RM, Guerra-Hernandez E, Garcia-Villanova B. 1999. Phytic acid content in milled cereal products and breads. Food Res Int 32:217–221.

Gazzaniga JM, Lupton JR. 1987. Dilution effect of dietary fibre sources: an in vivo study in the rat. Nutr Res 7:1261–1268.

Gibson RS. 1994. Content and bioavailability of trace elements in vegetarian diets. Am J Clin Nutr 59(Suppl.):1223S–1232S.

Gibson GR, Roberfroid MB. 1995. Dietary modulation of the human colonic microbiota: introducing the concept of prebiotics. J Nutr 125:1401–1412.

Gibson RS, Perlas L, Hotz C. 2006. Improving the bioavailability of nutrients in plant foods at the household level. Proc Nutr Soc 65:160–168.

Goldin BR, Aldercreutz H, Gorbach SL, Woods MN, Dwyer JT, Conlon T, et al. 1986. The relationship between estrogen levels and diets of Caucasian: American and oriental immigrant women. Am J Clin Nutr 44:945–953.

Grases F, Garcia-Gonzalez R, Torres A, Llobera A. 1998. Effects of phytic acid on renal stone formation in rats. Scand J Urol Nephrol 32:262–267.

Grases F, Simonet BM, March JG, Pieto RM. 2000. Inositol hexakisphosphate in urine: the relationship between oral intake and urinary excretion. Br J Urol Int 85:138–142.

Hallberg L, Rossander L, Skanberg A. 1987. Phytates and the inhibitory effect of bran on iron absorption in man. Am J Clin Nutr 45:988–996.

Harland BF, Harland J. 1980. Fermentative reduction of phytate in rye, white and whole wheat breads. Cereal Chem 57:226–229.

Harris RS. 1955. Phytic acid and its importance in human nutrition. Nutr Rev 13:257–259.

He M, van Dam RM, Rimm E, Hu FB, Qi L. 2010. Whole-grain, cereal fiber, bran, and germ intake and the risks of all-cause and cardiovascular disease-specific mortality among women with type 2 diabetes mellitus. Circulation 121:2162–2168.

Heaney RP, Weaver CM, Fitzsimmons ML. 1991. Soybean phytate content: effect on calcium absorption. Am J Clin Nutr 53: 745–747.

Huang C, Ma WY, Hecht SS, Dong Z. 1997. Inositol hexaphosphate inhibits cell transformation and activator protein 1 activation by targeting phosphatidylinositol-3 kinase. Cancer Res 57: 2873–2878.

Hunt JR, Bieseigel M, Johnson LK. 2008. Adaptation in human zinc absorption as influenced by dietary zinc and bioavailability. Am J Clin Nutr 87:1336–1345.

Jensen MK, Koh-Banerjee P, Hu F. 2004. Intakes of wholegrain, bran, and germs and the risk of coronary heart disease in men. Am J Clin Nutr 80:1492–1499.

Jewell D, Young G. 2001. Interventions for treating constipation in pregnancy, Cochrane Database Syst Rev 2001(2). Art. No.: CD001142. doi: 10.1002/14651858.CD001142.

Katayama T. 1995. Effects of dietary sodium phytate on the hepatic and serum levels of lipids. Biosci Biotechnol Chem 59: 1159–1160.

Kies C. 1985. Effect of dietary fat and fibre and calcium bioavailability. In: Kies C, editor. Nutritional bioavailability of calcium. ACS Symposium Series 275. Washington, DC: American Chemical Society. pp 175–187.

Koh-Banerjee P, Franz M, Sampson L, Liu S, Jacobs DR, Spiegelman D. 2004. Changes in wholegrain, bran and cereal fibre consumption in relation to 8 year weight gain among men. Am J Clin Nutr 80:1237–1245.

Lambo AM, Oste R, Nyman ME. 2005. Dietary fibre in fermented oat and barley β-glucan rich concentrates. Food Chem 89: 283–293.

Larsson M, Hulthen L, Sandstrom B, Sandberg AS. 1996. Improved zinc and iron absorption from breakfast meals containing malted oats with reduced phytate content. Br J Nutr 76:677–688.

Liu RH. 2007. Wholegrain phytochemicals and health. J. Cereal Sci. 46(3):207–219.

Liyana-Pathirana CM, Shahidi F. 2007. The antioxidant potential of milling fractions from bread, wheat and durum. J Cereal Sci 45: 238–247.

Lupton JR, Kurtz PP. 1993. Relationship of colonic luminal short chain fatty acids and pH to in vivo cell proliferation. J Nutr 123: 1522–1530.

Lupton JR, Meacher MM. 1988. Radiographic analysis of the effect of dietary fibres on rat colonic transit time. Am J Physiol 255: 633–639.

Lupton JR, Turner ND. 1999. Potential protective mechanisms of wheat bran fibre. Am J Med 106:24–27.

Maclennan R, Macrae F, Bain C. 1995. Randomized trial of intake of fat, fibre and betacarotene to prevent colorectal adenomas: the Australian Polyp Prevention Project. J Natl Cancer Inst 87: 1760–1766.

Macrae R, Robinson RK, Sadler MJ. 1993. Encyclopaedia of food science, food technology and nutrition. London: Academic Press.

Maes C, Delcour JA. 2002. Structural characterisation of water extractable and water unextractable arabinoxylans in wheat bran. J Cereal Sci 35:315–326.

Mateo Anson N, van den Berg R, Havenaar R, Bast A, Haenen GRMM. 2008. Ferulic acid from aleurone determines the antioxidant potency of wheat grain (*Triticum aestivum* L.). J Agric Food Chem 56:5589–5594.

Mateo Anson N, van den Berg R, Havenaar R, Bast A, Haenen GRMM. 2009. Bioavailability of ferulic acid is determined by its bioaccessibility. J Cereal Sci 49:296–300.

Menga V, Fares C, Troccoli A, Cattivelli A, Baiano A. 2010. Effects of genotype, location and baking on the phenolic content and some antioxidant properties of cereal species. Int J Food Sci Technol 45:7–16.

McIntyre ARM, Vincent AC, Perkins RC, Spiller RC. 1997. Effect of bran, ispaghula, and inert plastic particles on gastric emptying and small bowel transit in humans: the role of physical McIntyre factors. Gut 40:223–227.

McKevith B. 2004. Nutritional aspects of cereals. Nutr Bull 29: 111–142.

Mellen BP, Walsh TF, Herrington DM. 2009. Wholegrains intake and cardiovascular disease: a meta-analysis. Nutr Metab Cardiovasc Dis 18:283–290.

Metayer S, Seiliez I, Collin A, Duchene S, Mercier Y, Geraert P-A, Tesseraud S. 2008. Mechanisms through which sulphur amino acids control metabolism and oxidative status. J Nutr Biochem 19(4):207–221.

Minihane AM, Rimbach G. 2002. Iron absorption and the iron binding and antioxidant properties of phytic acid. Int J Food Sci Technol 37:741–748.

Monro JA. 2002. New approaches to providing nutrition information. In: Henry CKJ, Chapman C, editors. The nutrition handbook for food processors. Boca Raton, NC: CRC Press. pp 165–192.

Muller-Lissner SA. 1988. Effect of wheat bran on weight of stool and gastrointestinal transit time: a meta-analysis. Br Med J 296: 615–617.

Navert B, Sandstron B, Cederblad A. 1985. Reduction of the phytate content of bran by leavening in bread and its effect on zinc absorption in man. Br J Nutr 53:47–53.

Neyrinck AM, de Backer F, Cani P, Bindels LB, Stroobants A, Portelle D, Delzenne NM. 2008. Immunomodulatory properties of two wheat bran fractions – aleurone enriched and crude fractions – in obese mice. Int Immunopharmacol 8:1423–1432.

NICE (National Institute for Health and Clinical Excellence). 2008. Irritable bowel syndrome in adults: diagnosis and management of irritable bowel syndrome in primary care. Clinical Practice Guideline 61. London: NICE.

Painter NS, Almeida AZ, Colebourne KW. 1972. Unprocessed bran in treatment of diverticular disease of the colon. Br Med J 2: 137–140.

Payler DK, Pomare EW, Heaton KW, Harvey RF. 1975. The effect of wheat bran on intestinal transit. Gut 16:209–213.

Perez-Jimenez J, Saura-Calixto F. 2005. Literature data may underestimate the actual antioxidant capacity of cereals. J Agric Food Chem 52:6108–6114.

Pomeranz Y. 1988. Chemical composition of kernel structures. In: Pomeranz Y, editor. Wheat: chemistry and technology. 3rd ed, Berlin: Springer. pp 99.

Pointillart A, Gueguen L. 1992. Influence des fibres alimentaires sur la biodisponsabilite des mineraux. Bana 8:157–182.

Qu H, Madl RL, Takemoto DL, Baybutt RC, Wang W. 2005. Lignans are involved in the antitumor activity of wheat bran in colon cancer SW480 cells. J Nutr 135:598–602.

Raboy V. 2001. Seeds for a better future: low phytate grains help to overcome malnutrition and reduce pollution. Trends Plant Sci 6: 458–462.

Rickard ES, Thomson LU. 1997. Interactions and effects of phytic acid. In: Shahidi F, editor. Antinutrients and phytochemicals in foods. Washington, DC: American Chemical Society.

Roe L, Strong C, Whiteside C, Neil A, Mant D. 1994. Dietary intervention in primary care: validity of the DINE method for diet assessment. Fam Pract 11(4):375–381.

Rose DP, Goldman M, Connolly JM, Strong LE. 1991. High-fiber diet reduces serum estrogen concentrations in premenopausal women. Am J Clin Nutr 54:520–525.

Ross AB, Kamal-Eldin A, Aman P. 2004. Dietary alkylresorcinols: absorption, bioactivities and possible use as biomarkers of wholegrain wheat and rye rich foods. Nutr Rev 62:81–95.

Sandberg AS, Brune M, Carlsson NG, Hallberg L, Skoglund E, Rossander-Hulthen L. 1999. Inositol phosphates with different numbers of phosphate groups influence iron absorption in humans. Am J Clin Nutr 70:240–246.

Sandstrom B, Lonnerdal B. 1989. Promoters and antagonists of zinc absorption. In: Mills CF, editor. Zinc in human biology. Berlin: Springer–Verlag. pp 57–78.

Sayhoun NR, Jacques PF, Zhang XL, Juan W, McKeown NM. 2006. Whole grain is inversely associated with the metabolic syndrome and mortality in older adults. Am J Clin Nutr 83: 124–131.

Saura-Calixto F. 2011. Dietary fibre as a carrier of dietary antioxidants: an essential physiological function. J Agric Food Chem 59(1):43–49.

Scientific Advisory Committee Nutrition (SACN). 2008. Statement on dietary fibre. http://www.sacn.gov.uk/pdfs/final_sacn_position_statement_for_website_dietary_fibre.pdf. Accessed April 2012.

Schatzkin A, Mouw T, Park Y, Subar AF, Kipnis V, Hollenbeck A, Leitzmann MF, Thompson FE. 2007. Dietary fibre and wholegrain consumption in relation to colorectal cancer in the NIH AARP diet and health study. Am J Clin Nutr 85: 1353–1360.

Schley PD, Field CJ. 2002. The immune enhancing effects of dietary fibres and prebiotics. Br J Nutr 97:S221–S230.

Seal CJ. 2006. Wholegrains and CVD risk. Proc Nutr Soc 65:24–34.

Shewry P. 2009. The Healthgrain programme opens new opportunities for improving wheat for nutrition and health. Nutr Bull 34:225–231.

Shewry PR, Piironen V, Lampi AM, Edelmann M, Kariluoto S, Nurmi T, et al. 2010. Effects of genotype and environment on the content and composition of phytochemicals and dietary fiber components in rye in the HEALTHGRAIN diversity screen. J Agric Food Chem 58(17):9372–9383.

Slavin J. 2003. Why wholegrains are protective: biological mechanisms. Proc Nutr Soc 62:129–134.

Stoll DA. 1996. Can supplementary dietary fiber suppress breast cancer growth? Br J Cancer 73:557–559.

Thomson LU, Zhang L. 1991. Phytic acid and minerals: effect on early markers of risk for mammary and colon carcinogenesis. Carcinogenesis 12:2041–2045.

Topping D. 2007. Cereal complex carbohydrates and their contribution to human health. J Cereal Sci 46:220–229.

Topping DL, Clifton PM. 2001. Short chain fatty acids and human colonic function: roles of resistant starch and nonstarch polysaccharides. Physiol Rev 81:1031–1064.

Vaher M, Matso K, Levandi T, Helmja K, Kaljurand M. 2010. Phenolic compounds and the antioxidant activity of the bran, flour and whole grain of different wheat varieties. Procedia Chemistry 2(1):76–82.

Van den Heuvel E, Muys T, van Dokkum W, Schaafsma G. 1999. Oligofructose stimulates calcium absorption in adolescents. Am J Clin Nutr 69:544–548.

Vitaglione P, Napolitano A, Fogliano V. 2008. Cereal dietary fibre: a natural functional ingredient to deliver phenolic compounds into the gut. Trends Food Sci Technol 19:451–463.

Vitali D, Dragojevic, IV, Sebecic B. 2008. Bioaccessibility of Ca, Mg, Mn and Cu from wholegrain tea biscuits: impact of proteins, phytic acid and polyphenols. Food Chem 110:62–68.

Waliszewski P, Blaszczyk M, Wolinska-Witort E. 1997. Molecular study of sex steroid receptor gene expression in human colon and in colorectal carcinomas. J Oncol 64:3–11.

Wang J, Sun B, Cao Y, Tian Y. 2009. Protection of wheat bran feruloyl oligosaccharides against free radical induced oxidative

damage in normal human erythrocytes. Food Chem Technol 47: 1591–1599.

Wang Y. 2009. Prebiotics: present and future in food science and Technology. Food Res Int 42:8–12.

Watzke H. 1998. Impact of processing on bioavailability examples of minerals in foods. Trends Food Sci Technol 9:320–327.

World Cancer Research Fund/American Institute for Cancer Research. 2007. Food, nutrition, physical activity and the prevention of cancer: a global perspective. Washington, DC: American Institute for Cancer Research.

Weaver CM, Heaney RP, Teegarden D, Hinders SM. 1996. Wheat bran abolishes the inverse relationship between calcium load size and absorption fraction in women. J Nutr 126:303–307.

Weaver CM, Heaney RP, Martin BR, Fitzsimmons ML. 1991. Human calcium absorption from whole wheat products. J Nutr 121:1769–1775.

Wood P. 1997. Functional foods for health: opportunities for novel cereal processes and products. In: Campbell GM, Webb C, McKee SL, editors. Cereals novel uses and processes. New York: Plenum Press. pp 233–239.

Yoon JH, Thompson LU, Jenkins DJ. 1983. The effect of phytic acid on in vitro rate of starch digestibility and blood glucose response. Am J Clin Nutr 38:835–842.

Yu L, Zhou K, Parry JW. 2005. Inhibitory effect of wheat bran extracts on human LDL oxidation and free radicals. LWT 38: 463–470.

Zitterman A, Scheld K, Danz A, Stehle P. 1999. Wheat bran supplementation does not affect biochemical markers of bone turnover in young adult women with recommended calcium intake. Br J Nutr 82:431–439.

Zoran DL, Turner ND, Taddeo SS. 1997. Wheat bran diet reduced tumor incidence in a rat model of colon cancer independent of effects on distal luminal butyric acid concentrations. J Nutr 127: 2217–2225.

Zhou K, Su L, Yu L. 2004. Phytochemicals and antioxidant properties in wheat bran. J Agric Food Chem 52:6108–6114.