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REVIEW

Cereal grains, legumes and diabetes

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This review examines the evidence for the role of whole grain foods and legumes in the aetiology and management of diabetes. MedLine and SilverPlatter ('Nutrition' and 'Food Science FSTA') databases were searched to identify epidemiological and experimental studies relating to the effects of whole grain foods and legumes on indicators of carbohydrate metabolism. Epidemiological studies strongly support the suggestion that high intakes of whole grain foods protect against the development of type II diabetes mellitus (T2DM). People who consume $\sim$3 servings per day of whole grain foods are less likely to develop T2DM than low consumers ($<$3 servings per week) with a risk reduction in the order of 20–30%. The role of legumes in the prevention of diabetes is less clear, possibly because of the relatively low intake of leguminous foods in the populations studied. However, legumes share several qualities with whole grains of potential benefit to glycaemic control including slow release carbohydrate and a high fibre content. A substantial increase in dietary intake of legumes as replacement food for more rapidly digested carbohydrate might therefore be expected to improve glycaemic control and thus reduce incident diabetes. This is consistent with the results of dietary intervention studies that have found improvements in glycaemic control after increasing the dietary intake of whole grain foods, legumes, vegetables and fruit. The benefit has been attributed to an increase in soluble fibre intake. However, prospective studies have found that soluble fibre intake is not associated with a lower incidence of T2DM. On the contrary, it is cereal fibre that is largely insoluble that is associated with a reduced risk of developing T2DM. Despite this, the addition of wheat bran to the diets of diabetic people has not improved indicators of glycaemic control. These apparently contradictory findings might be explained by metabolic studies that have indicated improvement in glucose handling is associated with the intact structure of food. For both grains and legumes, fine grinding disrupts cell structures and renders starch more readily accessible for digestion. The extent to which the intact structure of grains and legumes or the composition of foods in terms of dietary fibre and other constituents contribute to the beneficial effect remains to be quantified. Other mechanisms to help explain improvements in glycaemic control when consuming whole grains and legumes relate to cooking, type of starch, satiety and nutrient retention. Thus, there is strong evidence to suggest that eating a variety of whole grain foods and legumes is beneficial in the prevention and management of diabetes. This is compatible with advice from around the world that recommends consumption of a wide range of carbohydrate foods from cereals, vegetables, legumes and fruits both for the general population and for people with diabetes.
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Introduction

Prevalence rates of type II diabetes mellitus (T2DM) have been escalating throughout the world, to an extent that the condition is considered to have reached epidemic proportions in many countries. While the highest prevalence rates appear to occur among indigenous peoples who have undergone rapid changes in their lifestyle, all populations have been affected to some extent. The greatest number of cases of T2DM within the next 20 years is predicted to occur in China and India, countries that earlier probably had relatively low rates of diseases that were considered to be associated with Western affluence. Diet is implicated in the aetiology of T2DM. Energy intake in excess of requirements, high intakes of saturated and *trans* fatty acids, and high intakes of fibre-depleted refined grain foods have been proposed as contributory factors. Obesity, especially when the excess adiposity is centrally distributed, and reduced physical activity are well-established risk factors for the development of T2DM. However, the roles of individual dietary components as promotive or protective factors are less clearly established. Sucrose, other sugars and rapidly

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digested starches may have an indirect role by promoting obesity (WHO/FAO, 2003). High intakes of fat, especially saturated fatty acids may increase resistance to the action of insulin, the underlying abnormality in many cases of T2DM, as well as promoting a high-energy intake (Maron *et al*, 1991). The suggestion that whole grain foods might protect against the development of diabetes as well as being useful in the management of people who have already developed T2DM is relatively recent (Jenkins *et al*, 1986; Mann, 2001, 2002).

The first documented dietary prescription in the treatment of presumed diabetes appeared in the *Papyrus Ebers*, written around 1500 BC. The use of wheat grains, grapes, honey and berries was advocated (Leeds, 1979). Before the discovery of insulin in 1921, several other dietary treatments had been tried in the management of diabetes. These ranged from diets high in carbohydrates to replace lost sugar, to diets that virtually excluded carbohydrates by the strict use of animal foods to facilitate the substitution of fat and protein for carbohydrate. Some diets involved near starvation (Vinik *et al*, 1997). Presumably most of the patients being treated were those with type I diabetes mellitus (T1DM) and exogenous insulin provided the means for logical therapy and a more liberal intake of dietary carbohydrate. There are also some descriptions of overweight patients who presumably suffered from what would now be diagnosed as T2DM and they too were given widely differing dietary advice. A recent Food and Agriculture Organization (FAO)/World Health Organization (WHO) report recommends that the bulk of carbohydrate-containing foods consumed be those rich in nonstarch polysaccharides and with a low glycaemic index (GI), and that appropriately processed cereals, vegetables, legumes and fruits are particularly good food choices (FAO/WHO, 1998). Diabetes organisations in many countries have generally endorsed this approach in their advice for treatment of both types of diabetes as well as for reducing the risks of T2DM (Diabetes Australia, Diabetes UK, American Diabetes Association, European Association for the Study of Diabetes), although the United States has not wholeheartedly endorsed the concept of the GI. Furthermore, there is no consistent advice regarding the optimal proportion of total energy that should be provided by carbohydrates. The European Association for the Study of Diabetes (EASD) suggest that this may be based upon cultural or personal preference and nature of the metabolic abnormality (Anon, 2000). There is also no general agreement with regard to the proportions of total carbohydrate that should be provided by intact whole grain and legume foods, and processed foods from these sources. This issue is of considerable importance in societies consuming much of their carbohydrate from processed food sources.

Defining the term ''whole grain'' has presented difficulties both in terms of analysing and interpreting research studies and in making recommendations. Several epidemiological studies have defined whole grain foods as those products that comprise $\geq$25% whole grain content or bran by weight (Liu *et al*, 2000; Meyer *et al*, 2000; Fung *et al*, 2002; McKeown *et al*, 2002). The United States Food and Drug Administration (FDA) require foods to contain $>$51% by weight of whole grain ingredients in order for health claims to be made in respect of whole grains and disease (FDA, 1999). It has been suggested that analysing the product for its fibre content may provide a check of compliance with the requirement for overall whole grain content. However, neither of these definitions takes account of the structure of the grain. This property is important to the glycaemic response to food. For example, popcorn is made from a whole kernel of corn but has a disrupted structure. The GI of whole corn is 53 ± 4 whereas plain popcorn has a GI of 72 ± 7 (Foster-Powell *et al*, 2002). Milling of wheat efficiently separates the bran, germ and endosperm to produce a finely ground flour (Campbell & Penfield, 1979; North American Millers Association, 2000). Reconstitution of the separated parts produces ''wholemeal flour''. However, bread made from finely ground wholemeal flour has a GI of 71 ± 2 (mean of 13 studies), no different to that of white bread (Foster-Powell *et al*, 2002). In contrast, bread containing a substantial proportion of whole or cracked wheat kernels has a GI some 20–30% lower than white bread (Foster-Powell *et al*, 2002). Grain structure therefore affects the metabolism of the carbohydrate content possibly by influencing the rate of digestion and absorption. The fact that grain structure is seldom taken into account in epidemiologic and dietary intervention studies makes interpretation of the scientific literature imprecise. Nevertheless, this paper reviews existing research regarding cereal grain foods and legumes in the prevention and management of diabetes and considers the extent to which existing recommendations might be strengthened to take new information into account.

Epidemiology

The earliest suggestion that refining whole foods may be implicated in the aetiology of T2DM stems from observations that the frequency of diabetes increased in populations as unprocessed or lightly processed foods were replaced by refined products (Trowell, 1978). In support of his observations made in East Africa, Trowell described the British experience with the less processed ''National flour'' which replaced refined grains between 1940 and the mid-1950s (Trowell, 1974). Coincident with its introduction, the diabetes death rate fell and rose again after the use of National flour had ceased. Many other lifestyle-related factors could have contributed to these temporal changes, but Trowell's work greatly influenced subsequent research.

Cross-sectional studies

The diets of 2941 subjects from the Framingham Offspring Cohort were analysed for whole grain intake and risk factors for T2DM (McKeown *et al*, 2002). Food that contained $\geq$25% whole grain or bran by weight was classified as whole grain in accordance with the definition suggested by Jacobs *et al*

(1998). Both men and women reported a greater intake of refined grains (22.0 and 18.5 servings/week) than whole grains (8.3 and 8.8 servings/week), respectively. Inverse associations across quintiles of whole grain intake were found for body mass index (P for trend = 0.06), waist-to-hip ratio ($P = 0.005$) and fasting insulin ($P = 0.03$). These associations could not be explained by potential confounding factors (eg: age, sex, smoking, physical activity).

Prospective studies

Three large prospective studies carried out in the United States have examined the effect of whole grain foods on T2DM (Table 1). The definition of ''whole grain foods'' was the one suggested by Jacobs *et al* (1998) and a serving size was based on commonly used units, for example, one slice of bread or 1 cup of breakfast cereal. After adjustment for potential confounding factors, people consuming around 3 servings per day of whole grain products had a risk reduction in the order of 20–30% compared with low consumers of whole grain products with a dose–response across quintiles of whole grain food intake (Liu *et al*, 2000; Meyer *et al*, 2000; Fung *et al*, 2002). A smaller study carried out in Finland did not find a statistically significant difference in incident T2DM when comparing consumers of whole grain foods above and below the extreme quartiles of intake, although there was a trend suggesting a reduced risk of developing T2DM among larger consumers of whole grain foods (Montonen *et al*, 2003). Adding dietary glycaemic load into the models did not change the association between whole grain food intake and incident T2DM (Liu *et al*, 2000; Meyer *et al*, 2000). Intake of insoluble fibre was inversely associated with T2DM risk, whereas intake of soluble fibre was not strongly associated with risk (Meyer *et al*, 2000). Fung *et al* (2002) found that after adjustment for cereal fibre, magnesium intake, and glycaemic load, that the inverse association between whole grain food intake and risk of T2DM was largely explained by cereal fibre.

Liu *et al* (2000) calculated a ratio of refined:whole grain food intake and found a trend across quintiles for incident T2DM. Risk increased as the ratio of refined:whole grain food intake increased. After adjustment for age and energy intake, women in the highest quintile of refined whole grain food intake had a 57% (95% CI: 36, 82) greater risk of incident T2DM compared with women in the lowest intake ratio (ie: greater risk for low consumers of whole grain foods or high consumers of refined grain foods). Other prospective studies that have found an inverse association of incident T2DM with increasing intake of whole grain foods have not found a positive association with refined grain intake (Meyer *et al*, 2000; Fung *et al*, 2002). This appears contradictory because a high consumer of whole grain foods might, almost by definition, be expected to be a low consumer of refined grain foods. However, the definition of whole grain used in the studies was a product that contained $\geq 25\%$ whole grain or bran by weight (Jacobs *et al*, 1998). Thus, a person

Table 1 Prospective studies that have assessed the association between whole grain intake and the relative risk of developing type II diabetes mellitus

Reference	*Subjects* (n)	*Country*	*Follow-up (y)*	*Cases* (n)	*Multivariate adjustments*[a]	*Relative risk (refined grain)*	P *for trend*[b]	*Relative risk (whole grain)*	P *for trend*
Liu *et al* (2000)	Female nurses (75 521)	USA	10	1879	Physical activity, alcohol intake, family history of diabetes, supplement use	1.11 (0.94, 1.30)	0.26	0.73 (0.63, 0.85)[c]	<0.001
Meyer *et al* (2000)	Women (35 988)	USA	6	1141	Physical activity, waist-to-hip ratio, education, alcohol intake	0.87 (0.70, 1.08)	0.36	0.79 (0.65, 0.96)[c]	0.0089
Fung *et al* (2002)	Male health workers (42 898)	USA	≤ 12	1197	Physical activity, family history of diabetes, alcohol, fruit, vegetable intakes	1.08 (0.87, 1.33)	0.69	0.70 (0.57,0.85)[c]	0.0006
Montonen *et al* (2003)	Men and women (4316)	Finland	10	156	Vegetable, fruit, berry intakes	0.62 (0.36, 1.06)	0.05	0.65 (0.36,1.18)	0.02

[a]In addition to the variables listed in the table, all studies adjusted for age, body mass index, smoking and energy intake.
[b]p for trend across percentiles of grain intake.
[c]Significantly different relative risk in high consumers (comparison of groups above or below the extreme percentiles of grain intake (quintiles in the USA studies, quartiles in the Finnish study).

consuming 3 servings/day of a whole grain product in which whole grain was not the major constituent part (ie: 25–49%) would have been classified as a high consumer of whole grain foods despite the majority of the product being made up of non-whole grain material. Montonen *et al* (2003) used a definition of whole grain foods that was restricted to products that contained grains (as opposed to bran products) and applied mainly to breads, flours and minimally disrupted grain products such as porridge, gruel and a traditional rice pudding. It was suggested that this difference in classification better differentiated whole grain intake from refined grain intake than in previous studies. Despite this, there was an inverse association between incident T2DM for both whole grain food intake (P for trend $= 0.02$) and refined grain food intake (P for trend $= 0.05$). Thus, although there is some evidence that a high intake of refined grain food is associated with an increased risk for developing T2DM (Liu *et al*, 2000), this is not a consistent result. Unravelling the effects of whole- and refined-grain intakes on incident T2DM is likely to require a definition of whole grain that incorporates grain structure. In addition, the analysis should take account of the absolute intake of whole grain to separate out the effect of the refined grain content of the food.

Only one of these studies reported legume intake (Meyer *et al*, 2000). Neither fibre from legumes nor intake of mature beans was related to incident diabetes. However, dietary intake, as defined by extreme quintiles, of total grains (<13–>33 servings/week) far exceeded that of legumes (<1.5–>4.5 servings/week). Thus, in this population of North American women living in Iowa the intake of legumes may have been too small to show an effect on diabetes risk.

Dietary data on cereal fibre intake, as distinct from whole grain food intake, were collected from 42 759 men and 65 173 women (Salmeron *et al*, 1997a, b). During 6 y of follow-up, 523 men and 915 women were diagnosed with T2DM. When comparing people above or below the extreme quintiles of cereal fibre intake, there was a 30% (95% CI: 4, 49) and 28% (95% CI:10, 42) reduction in risk of incident diabetes for the men and women, respectively. The greatest risk occurred in people with diets that had a high dietary glycaemic load (GI $\times$ amount of carbohydrate) and a low intake of cereal. Stevens *et al* (2002) recorded the dietary intakes of 12 251 middle-aged men and women. During 9 y of follow-up, 1447 cases of diabetes were reported (Stevens *et al*, 2002). Cereal fibre was inversely associated with risk of diabetes in white and African-American subjects but statistically significant only in white people with a hazard ratio of 0.75 for diabetes (95% CI: 0.60, 0.92) comparing people above or below the extreme quintiles of cereal fibre intake. Of the prospective studies that analysed dietary intakes for different forms of fibre, none found an association between soluble fibre and/or fibre derived largely from fruit and vegetables, and incident T2DM (Salmeron *et al*, 1997a, b; Meyer *et al*, 2000; Stevens *et al*, 2002; Montonen *et al*, 2003).

The issue as to whether protection against T2DM is afforded by cereal fibre intake or from whole grain intake, or whether both factors make an independent contribution, cannot be definitively determined from these studies. However, an indication that whole grain food is beneficial was found in The Iowa Women's Health Study in which 11 040 women were matched on total grain fibre intake (Jacobs *et al*, 2000). A lower mortality rate (RR 0.83, 95% CI 0.73, 0.94) was found among women consuming predominantly fibre from whole grain foods compared with women consuming a similar amount of fibre predominantly from refined grain sources (Jacobs *et al*, 2000). A number of dietary and physical characteristics were different between the groups so that it would not be possible to attribute the lower rate of mortality to fibre source alone. None of the prospective studies attempted to determine whether grain structure (intact or disrupted) was associated with incident T2DM. Nevertheless, the results are consistent with the suggestion that consumption of whole grain foods imparts health benefits over and above the effect of fibre.

Short- and medium-term dietary intervention studies

Dietary intervention studies were selected if it was possible to determine that whole grain foods and/or legumes had been compared with more refined sources of carbohydrate (Table 2). Adding such foods into a diet can be achieved by carbohydrate displacement of other foods (usually fatty foods) or by replacing refined products with whole grain foods. Early intervention studies were often designed to increase the carbohydrate content and reduce the fat content of a diet. In later studies, the carbohydrate content of test diets was kept constant but the type of carbohydrate and/or structure of the food differed between diets. In studies where the aim was to increase dietary fibre content, fruit and vegetables were often increased in addition to unrefined grains. Whole grain foods have also been used in investigations to determine whether low GI foods lower blood glucose levels in healthy people and in those with diabetes. Such studies often involved increased use of pasta as well as legumes and unrefined grain foods. Different studies have involved the use of the major grains (wheat, rice, rye, oats, barley, corn), legumes (beans, lentils, peas), and whole fruit. Typically, these foods have been used in combination with each other making it difficult to disentangle the effects of individual foods. Thus, most of these studies do not provide a clear indication as to which food or combination of foods has a beneficial influence over carbohydrate metabolism, or whether food structure is important in this respect.

The majority of studies listed in Table 2 show some improvement in measures of glucose metabolism in association with the use of unrefined foods. Improvements include the discontinuation or reduction in insulin or oral hypoglycaemic agents, fewer hypoglycaemic events, lowering of fasting blood glucose, lowering of glycosylated protein concentrations, and reduced urinary glucose and C-peptide output. Apart from these positive outcomes, several studies

Table 2 Intervention studies that have investigated the effect of dietary modification on glycaemic control using whole grain or legume foods

Author	*Subject no. and description*	*Study design*	*Diet*[a]	*C/Ft/Pr*[b]	*Fibre (g)*	*Diet modifiers*	*Positive outcomes*[c]
Kiehm *et al* (1976)	13 T2DM men	Serial 1 wk/2 wk	ADA	43/34/23	5 (crude)	(no sugar)	Lower fasting plasma glucose (26%)
		Metabolic ward	HCHF	75/9/16	14 (crude)	Natural foods	Discontinued or decreased diabetic drug use in 9 of 13 men
Simpson *et al* (1979b)	18 T2DM men and women	Randomised crossover 6 wk	LC	34/50/16	35.5	(no sugar)	Lower basal and fasting glucose (12%)
		Outpatient	HC	60/25/15	78.0	Wholemeal bread + vegetables	Lower preprandial blood glucose Lower glycosylated haemoglobin (11%)
Simpson *et al* (1979a)	12 T1DM men and women	Randomised crossover 6 wk	LC	34/50/16	35.5	(no sugar)	Lower basal blood glucose
		Outpatient	HC	60/25/15	78.0	Wholemeal bread + vegetables	Lower preprandial blood glucose
Anderson and Ward (1979)	20 lean T2DM men	Serial 1 wk/2 wk Metabolic ward	ADA	43/37/20	26	(no sugar)	Discontinued or decreased insulin use in 11 of 20 men
			HCHF	70/9/21	65	Whole grain, cereals, legumes, vegetable, fruit	
Anderson *et al* (1980)	11 T2DM men	Control run in crossover 2 wk	Control			Whole wheat bread, rice, beans, oat bran, fruit vegetables (no sugar)	(with reference to control diet)
		Metabolic ward	HCLF	70/18/12	12 g/Mcal		Lower insulin doses (both diets)
			HCHF	70/18/12	34 g/Mcal		Lower fasting glucose (HCHF) (9%)
Rivellese *et al* (1980)	8 T2DM adults	Randomised crossover 10 d	Usual diet	53/30/17	16	(restricted simple sugars)	(HF *vs* both usual diet and LF)
		Metabolic ward	LF	42/37/21	20	Legumes, wholemeal bread, vegetables, fruit	Lower mean daily blood glucose
			HF	53/30/17	54		Lower insulin dose in all four using insulin
Simpson *et al* (1981)	18 T2DM	Randomised crossover 6 wk	LC	40/39/21	17.6	Legumes and wholemeal bread	Lower preprandial blood glucose
	9 T1DM adults	Outpatient	HCHF	61/18/21	96.6		Lower 24 h glycosuria Lower HbA_{1C} (type II subjects) (10%)
Kay *et al* (1981)	5 elderly	Serial 2 wk	LF	Same	10	Oats, wholemeal bread, legumes	Lower basal plasma insulin
	T2DM	Metabolic ward	HF		30		Lower basal plasma GIP
Kinmonth *et al* (1982)	10 T1DM children	Randomised crossover 6 wk	Refined	55/30/15	20	Wholemeal bread, cereals, legumes, fruit, vegetable	Lower blood glucose during last 4 wk of diet
		Outpatient	Unrefined	55/30/15	60		More preprandial negative urinary glucose test Lower insulin dose in 6/10 children
Ward *et al* (1982)	7 T2DM adults	Randomised crossover 6 wk	LC	40/40/20	15	(no added refined sugar)	Increased monocyte insulin binding activity
		Outpatient	HC	60/18/22	100	Legumes and cereals	Lower fasting plasma glucose

Table 2 *Continued*

Author	*Subject no. and description*	*Study design*	*Diet*[a]	*C/Ft/Pr*[b]	*Fibre (g)*	*Diet modifiers*	*Positive outcomes*[c]
Hjollund *et al* (1983)	18 T2DM adults	Parallel control 3 wk	Usual	34/50/16	24	Whole grain cereals and vegetables	Lower fasting plasma & 24 h urinary glucose
		Outpatient	HFHC/Lfat	51/32/17	53		Increased insulin action & cellular binding Increased *in vitro* insulin sensitivity
Riccardi *et al* (1984)	6 T1DM	Randomised crossover 10 d	LCLF	42/37/21	20	Legumes, vegetables, wholemeal bread, fruit	Lower daily blood glucose
	8 T2DM	Metabolic ward	HCLF	53/30/17	16		
			HCHF	53/30/17	54		
Hollenbeck *et al* (1985)	6 T1DM women	Randomised crossover 4/6 wk	Usual diet	45/40/15	28	Brown rice, legumes, whole grain bread and cereals	
		Outpatient	HC/Lfat	65/20/15	50		
Hollenbeck *et al* (1986)	6 T2DM adults	Randomised crossover 4 wk	Normal fibre	60/25/15	11/Mcal	Whole wheat bread, cereals, fruit, peanuts	
		Metabolic ward	High fibre	60/25/15	27/Mcal		
Jenkins *et al* (1987)	6 non-diabetic adults	Randomised crossover 2 wk	High GI	62/20/19	21	Oat bran, pumpernickel bread, legumes, pasta	Lower serum fructosamine
		Metabolic ward	Low GI	59/21/20	26		Lower 24 h urinary C-peptide
Karlstrom *et al* (1987)	15 T2DM adults	Randomised crossover 3 wk	Control diet	47/33/20	24	Peas and beans	Lower mean postprandial glucose
		Metabolic ward	Leguminous		37		Lower mean urine glucose excretion
Jenkins *et al* (1988)	8 T2DM adults	Randomised crossover 2 wk	High GI	53/26/21	28	Oat bran, bulgur, pumpernickel bread, legumes, pasta	Lower fasting blood glucose, AUC
		Metabolic ward	Low GI	54/24/22	34		Lower HbA_{1C} and fructosamine (both diets) Lower urinary C-peptide:creatinine ratio
Parillo *et al* (1988)	6 T1DM with renal failure	Randomised crossover 10 d	LCLFLP	40/51/9	22	Legumes, vegetable, fruit	Lower daily blood glucose (postprandial)
		Metabolic ward	HCHF	50/38/12	65		
Simpson *et al* (1988)	13 T2DM adults	Serial 3 wk/2 wk	LCLF	28/49/22	21	Bran cereal, wholewheat bread	Lower fasting plasma glucose (25%)
		Metabolic ward	HCHF	60/21/19	57		Lower urinary glucose
			LCLF	28/49/22	21		Lower pancreatic immunorcactive glucagons (with reference to baseline)
O'Dea *et al* (1989)	10 T2DM men	Randomised crossover 2 wk	LCLFHP	23/15/62	13	Unrefined cereals, pasta, brown rice, wholemeal bread, legumes	
		Outpatient	LCHF	27/55/18	14		Lower fasting blood glucose (17%) HCHF, LCLFAP
			HCLF	63/12/25	20		Lower mean change in plasma glucose to OGTT (LCLFHP)
			HCHF	65/10/24	45		

Table 2 *Continued*

Author	*Subject no. and description*	*Study design*	*Diet*[a]	*C/Ft/Pr*[b]	*Fibre (g)*	*Diet modifiers*	*Positive outcomes*[c]
Fukagawa *et al* (1990)	12 non-diabetics	Serial 3/4 wk	Usual diet	43/42/18	16–18	(no sucrose)	Lower fasting blood glucose (5%)
		Meals under supervision	HCHF	68/14/18	68–88	Whole grain, cereals, legumes, vegetable, fruit	Lower fasting plasma insulin Increased glucose disposal rate (clamp)
Anderson *et al* (1991)	10 T2DM adults	Randomised crossover 4 wk	LCLF	39/41/20	5	Whole grain or bran cereals, beans, fruit, vegetables	Lower basal insulin requirements
			HCHF	70/10/20	35		Increased glucose disposal per unit insulin
Fontvieille *et al* (1992)	12 T1DM	Randomised crossover 5 wk	High GI	45/36/19	27	Rice, rye bread, peas, beans, pasta, biscuits	Lower fasting blood glucose (11%)
	6 T2DM	Outpatient	Low GI	46/36/18	27		Lower plasma fructosamine
Frost *et al* (1994)	51 T2DM adults	Randomised parallel 12 wk	BDA	44/32/22	14	Pumpernickel bread, oats, barley, pasta, legumes, fruit	Lower serum fructosamine
			Low GI	49/25/23	21		Lower within group fasting glucose (21%)
Pick *et al* (1998)	11 T2DM men	Randomised crossover 12 wk	White bread	60/24/16	28	Barley bread, pastries, pasta, cereal	Reduction in oral diabetic medicine (4 of 7)
		Outpatient	Barley	61/23/16	39		
Järvi *et al* (1999)	20 T2DM adults	Randomised crossover 24 d	High GI	55/28/16	34	Both diets included:-Whole grain barley, legumes, pasta, maize flour, parboiled rice	Lower fasting plasma glucose and improved insulin sensitivity (both diets), lower HbA_{1C} (low GI) and fructosamine, plasminogen inhibitor-1 activity (low vs high GI).
		Outpatient	Low GI	55/28/16	38		
Giacco *et al* (2000)	63 T1DM adults	Randomised parallel 24 wk	LF	55/28/17	15	Legumes, fruit, vegetables	Lower daily blood glucose
		Outpatient	HF	52/28/20	37		Fewer hypoglycaemic events Lower HbA_{1C}
Chandalia *et al* (2000)	13 T2DM overweight adults	Randomised crossover 6 wk	ADA	55/30/15	24	Vegetables, oat bran, oatmeal, whole wheat bread, fruits, legumes	Lower daily preprandial plasma glucose
		Outpatient	HF	55/30/15	50		Lower daily urinary glucose
Jang *et al* (2001)	76 nondiabetic men	Randomised parallel 16 wk	White rice	69/16/15	23	Brown rice, barley, black beans, sesame seeds, Job's tears	Lower fasting serum glucose and insulin, lower AUC (OGTT), lower β-cell function and improved insulin sensitivity (HOMA)
		Outpatient	Roasted grain powder	61/20/19	26		
Pereira *et al* (2002)	11 nondiabetic adults	Randomised crossover 6 wk	Refined grains	55/31/16	18	80% wheat, 20% oats, rice, corn, barley, rye	Lower fasting insulin
		Outpatient	Whole grains	54/32/17	28		Lower insulin resistance (HOMA) Higher rate of glucose infusion (clamp)

Table 2 *Continued*

Author	*Subject no. and description*	*Study design*	*Diet*[a]	*C/Ft/Pr*[b]	*Fibre (g)*	*Diet modifiers*	*Positive outcomes*[c]
McAuley *et al* (2002)	79 insulin resistant adults	Randomised parallel 4 mo	Control	46/22/16	22	Fish, nuts, seeds, whole grain, pasta, rice, fruit, legumes, vegetables	(Intensive with reference to control)
		Outpatient	Modest	54/27/20	27		Improved insulin sensitivity (clamp)
			Intensive	51/30/19	30		
Jimenez-Cruz *et al* (2003)	14 T2DM adults	Randomised crossover 6 wk	High GI	63/20/17	25	Oranges, beans, yogurt, pasta, corn tortillas	Lower fasting serum glucose
		Outpatient	Low GI	58/22/20	34		Lower HbA_{1C}

[a]ADA: American Diabetic Association, HC: high carbohydrate, LC: low carbohydrate, HF: high fibre, LF: low fibre, HP: high protein, Lfat: low fat, GI: glycaemic index, BDA: British Diabetic Association.
[b]CHO/Ft/Pr: proportion of energy intake derived from carbohydrate, fat, and protein, respectively.
[c]HbA_{1C}: glycated haemoglobin concentration, GIP: gastric inhibitory polypeptide, AUC: area under the curve, OGTT: oral glucose tolerance test, HOMA: homeostasis model assessment.
wk, weeks; d, days; mo, months.

monitored metabolic profiles over a number of hours and showed improved glucose control and lower insulin responses over a prolonged period (Simpson *et al*, 1979a, b, 1981; Brand *et al*., 1991; Järvi *et al*, 1999).

Improvements in insulin sensitivity have been shown in a number of ways. In a crossover study using low (control) and high carbohydrate diets, Ward *et al* (1982) found greater monocyte insulin receptor binding after the high carbohydrate diet compared with the control period. Hjollund *et al* carried out intravenous insulin tolerance tests in people with T2DM and found that *in vivo* insulin action (expressed as a rate constant for plasma glucose disappearance) had increased in the group receiving a low-fat, high-starch, high-fibre diet compared with baseline, while no change had occurred in the control group. *In vitro*, adipocyte and monocyte insulin receptor binding had increased in the experimental dietary group (Hjollund *et al*, 1983). Fukagawa *et al* used the euglycaemic clamp in a crossover study comparing a usual diet (control) with a high-carbohydrate high-fibre diet, in nondiabetic men. Mean glucose disposal was 43% higher after the high-carbohydrate high-fibre dietary period compared with the control period (Fukagawa *et al*, 1990). Jang *et al* compared glucose handling in a group receiving a white rice breakfast (control group) with a group receiving a powdered breakfast made from coarsely ground whole grains and legumes (powder group). Comparison of net differences between the two groups showed that the glucose and insulin response areas following an OGTT were lower in nondiabetic subjects after receiving the powder breakfast. Using the homeostasis model assessment (HOMA), insulin resistance and β-cell function were improved in the powder group relative to the control group in nondiabetic subjects (Jang *et al*, 2001). Pereira *et al* compared insulin sensitivity in overweight people with T2DM on diets that incorporated refined or whole grains. A greater rate of glucose infusion was achieved during a euglycaemic hyperinsulinaemic clamp test on the whole grain diet (Pereira *et al*, 2002). McAuley *et al* placed normoglycaemic insulin-resistant adults on a diet and exercise intervention programme that included increased consumption of whole grain foods. Weight loss and enhanced insulin sensitivity using the insulin clamp technique was demonstrated on an intensive lifestyle intervention programme (McAuley *et al*, 2002).

Chandalia *et al* (2000) and Giacco *et al* (2000) showed improved measures of glycaemic control in people with diabetes by increasing the dietary fibre content. Although the authors attributed the improvement in glycaemic control to an increased intake of dietary fibre, much of the additional fibre was contained in whole foods having an intact structure. These studies therefore do not rule out the possibility of an independent effect of food structure on glycaemic control. The effect of food structure was examined by Järvi *et al* (1999) in a study where the dietary fibre content was kept constant but the overall dietary glycaemic index (dietary GI) differed between two diets, achieved largely by

altering the food structure. The low and the high GI diets were sufficiently different from the subjects habitual diets to improve glycaemic control during both the dietary periods. Additional improvements were also found between diets. After the low GI dietary period, serum fructosamine concentrations and plasminogen activator inhibitor-1 activity were lower compared with the high GI period. A difference between baseline and postintervention HbA_{1C} concentrations was found only after the low GI diet.

Two studies reported no changes in glycaemic control after dietary intervention. Hollenbeck *et al* provided six women with T1DM with diets that theoretically differed substantially in their carbohydrate and fat content. The high carbohydrate diet contained 65% carbohydrate and 20% fat and in theory included brown rice, legumes, lentils and whole grain breads and cereals. In contrast to most other studies, participants selected prepared foods during the control and high carbohydrate periods. Food choices were similar during both the dietary periods; beans and lentils that had been provided during the high carbohydrate period were not generally chosen. Thus, failure to consume sufficient quantities of the whole grain and legume foods may have accounted for the lack of effect (Hollenbeck *et al*, 1985). In a second study, Hollenbeck *et al* provided six people with T2DM with prepared meals differing in fibre content in a crossover study. During the high-fibre period, exchanging white bread with whole wheat bread, fruit juice with whole fruit, and refined cereals with whole grain cereals provided the additional fibre. Peanuts were included as a snack. No difference in measures of glucose and lipid metabolism was observed between the two diets. A sample 1-day menu showed that the exchange foods accounted for a relatively small proportion of the daily food intake. Indeed, there appeared to be reluctance by the investigators to make large dietary changes, claiming that dietary fibre intake had been increased to ''acceptable and practical limits''. Thus, once again it seems likely that the amounts of unrefined foods used were insufficient to produce changes in glucose handling (Hollenbeck *et al*, 1986). Parillo *et al* reported a ''remarkable'' improvement in blood glucose control on a diet containing grains and legumes in diabetic patients with chronic renal failure. However, a note of caution was expressed concerning the high dietary phosphate content of the diet that may be inappropriate in uremic patients (Parillo *et al*, 1988).

Considering the scope of these dietary intervention studies, there is little doubt that diets containing substantial intakes of whole grain foods, fruit, vegetables and legumes are associated with an improvement in insulin sensitivity and other indicators of carbohydrate metabolism including improved glycaemic control in people with diabetes. The studies reviewed here do not provide any clear indication as to which component(s) of whole grains or legumes produce these beneficial effects, although the study by Järvi *et al* (1999) suggests that the structure of the grain is important. Owing to dietary modifications that included changes in intake of a variety of foods, it is not possible to draw conclusions regarding individual foods.

Long-term dietary intervention studies

Turner *et al* compared the effectiveness of a low-fat, high-carbohydrate, high-fibre diet, and hypoglycaemic drug treatment on glycaemic control in newly diagnosed T2DM. After 9 y of follow-up, only 8% of diet-treated subjects had fasting plasma glucose <7.8 mmol/l. In contrast, 24% of subjects assigned to sulphonylurea treatment had fasting plasma glucose <7.8 mmol/l. No details of dietary compliance were provided nor was it stated whether the use of whole grain foods had been encouraged (Turner *et al*, 1999). Story *et al* instructed 14 men with diabetes (three T1DM, 11 T2DM), all of whom were using insulin, to consume a high-carbohydrate, high-fibre diet and monitored their glycaemic control during a 4 y period. Consumption of whole grain breads and cereals, dried beans, vegetables and fruit was encouraged. Insulin therapy was discontinued in all of the type II subjects although sulphonylurea therapy was subsequently introduced to five of the 11 subjects. Changes in insulin doses in type I subjects appeared to be related to adherence to diet (Story *et al*, 1985). However, these findings need to be interpreted with caution since the study was uncontrolled. Lifestyle intervention programmes involving exercise and dietary advice have shown reductions in incident diabetes (Pan *et al*, 1997; Tuomilehto *et al*, 2001; Anon, 2002; Knowler *et al*, 2002). In these studies, people with impaired glucose tolerance (IGT) were randomised to intervention groups or to a control group. Healthier eating was encouraged in all studies with the frequent consumption of whole grain foods specifically mentioned in the study by Tuomilehto *et al* (2001). Incident diabetes was reduced by 31–58% in the intervention groups relative to the control groups.

Thus, long-term dietary intervention studies provide confirmation that dietary modification can reduce the risk of progression from IGT to T2DM, and produce sustained benefit in people with diabetes. However, due to changes in several components of the diet it is not possible to determine to what extent the increased intake of whole grain foods and legumes might have contributed. Nevertheless, it should be noted that the benefit of lifestyle change was greater than expected from the relatively small weight reduction.

Mechanisms

Understanding the mechanisms by which whole grains and legumes might improve glycaemic control may facilitate the formulation of more specific dietary recommendations aimed at reducing risk of developing diabetes as well as treating those who have diabetes, than is possible at present.

Table 3 Comparison of glycaemic index between grains with intact or disrupted structures

Reference	*Study subjects*	*Disrupted structure*	*GI*[a]	*Intact structure*	*GI*
Jenkins *et al* (1986)	Diabetics ($n=15$–17)	Wholemeal wheat bread	96 ± 5	Wheat kernels	63 ± 6*
Jenkins *et al* (1986)	Diabetics ($n=15$–17)	Wholemeal wheat bread	96 ± 5	Cracked wheat kernels	65 ± 4*
Jenkins *et al* (1986)	Diabetics ($n=14$)	Wholemeal rye bread	89 ± 6	Rye kernels	47 ± 5*
Jenkins *et al* (1988)	Diabetics ($n=6$–8)	Wholemeal wheat bread	92 (11)	75% cracked wheat bread	69 (4)*
Jenkins *et al* (1988)	Diabetics ($n=6$–8)	Barley flour bread	96 (6)	75% barley kernel bread	39 (7)*
Liljeberg *et al* (1992)	Non-diabetics	80% Wholemeal barley flour bread	94.9 ± 15.1	80% barley kernel bread	57.1 ± 10.3
Granfeldt *et al* (1994)	Non-diabetics ($n=9$–10)	Barley flour porridge	65 ± 9**	Barley kernels	35 ± 8*
Granfeldt *et al* (1995)	Non-diabetics ($n=9$)	Rolled oat porridge	93 ± 7	Oat kernel porridge	60 ± 7*

[a]GI with reference to white wheat bread (GI = 100); mean ± s.d., mean (s.e.).
*Significantly different from GI of food with disrupted structure.
**Significantly different from white wheat bread.

Grain size and structure

The postprandial glycaemic response to grains having an intact structure would appear to be lower than when the grain structure has been disrupted (Table 3). Liljeberg *et al* (1992) also found lower insulin responses to test meals comprising 80% intact barley grains compared with food made from wholemeal barley flour. Although grain structure is the most likely explanation for the measured differences in glycaemic response, different methods of preparation and cooking (eg: boiling compared with baking) might also have had an effect. The size of grains or their products would appear to have a complex association with digestion and absorption. For example, the glycaemic response to whole wheat kernels was no different to that of cracked wheat kernels (Jenkins *et al*, 1986). A possible explanation is that a whole grain of wheat is crushed during chewing whereas cracked wheat particles, having a smaller size, may be difficult to disrupt further. In theory, similar sized particles might be delivered to the small intestine thereby eliciting a similar glycaemic response. The extent of grinding provides another example of the effect of grain particle size on absorption and digestion. Postprandial glucose and insulin responses were lower in healthy people and in people with T2DM after consuming scones made from coarsely ground flour (40% of the wheat particles retained on a 1 mm sieve) compared with finely ground flour (Heaton *et al*, 1988; O'Donnell *et al*, 1989). This effect is likely to be due to less starch being digested and absorbed in the small intestine since in nondiabetic patients with ileostamies, the starch content of the effluent was 42% higher after the coarsely ground compared with the finely ground flour meal (O'Donnell *et al*, 1989). A slow rate of starch hydrolysis using ''stone ground'' flour has been attributed to an amylase inhibitor naturally present in the wheat germ (Snow & O'Dea, 1981). The process of passing the wheat through a roller mill appears to eliminate the amylase inhibitor activity because the starch in wholemeal flour, which included milled wheat germ, was hydrolysed at a rate identical to that of white flour (Snow & O'Dea, 1981). O'Dea *et al* (1980) found that ground rice meals elicited much higher peak responses of glucose and insulin than meals consisting of whole grains of rice. An effect on glycaemic response is also found when comparing mixed meals having different grain structures. A meal consisting of parboiled rice, whole beans, and bread containing whole wheat grains elicited areas under the plasma insulin and blood glucose response curves that were 39 and 42% lower, respectively, compared with a meal of sticky rice, ground beans and bread made from wholemeal flour (Järvi *et al*, 1995). Disruption of intact legumes also has an effect on the rate of carbohydrate digestion. Cooked white kidney beans, lentils and yellow peas had substantially lower *in vitro* starch digestion rates (50–75% lower) compared with ''dry milled and cooked'' pulses (Würsch *et al*, 1986). The slower rate of digestion was attributed to the starch being entrapped in fibrous thick-celled walls. Similarly, the rate of starch hydrolysis was five-fold greater when using ground cooked lentils compared with cooked mashed whole lentils, while blending the lentils after cooking gave an intermediate rate (Wong & O'Dea, 1983). *In vivo*, higher glycaemic and/or insulinaemic responses were found following meals of ground pulses compared with intact pulses (O'Dea & Wong, 1983; Golay *et al*, 1986).

These results suggest that incomplete chewing might be partly responsible for the lower glycaemic responses to intact or partially intact grains. In support of this suggestion, microscopical examination of the digestas of *in vitro* digestive systems found intact kernels after bread containing whole grains had been chewed, expectorated and incubated using the digestive systems (Holm & Bjorck, 1992; Liljeberg *et al*, 1992). Thus, food with an intact botanical structure may delay or render some of the starch unavailable for absorption, indicating that structural disruption of cereal grains and pulses has an effect on carbohydrate metabolism independent of the fibre content.

Fibre

There are many definitions of dietary fibre. When considered as those constituents of plant foods that escape digestion in

the upper intestine, it has been suggested that the components include nonstarch polysaccharides, lignin, resistant oligosaccharides and resistant starch (FAO/WHO, 1998). Nonstarch polysaccharides are derived predominantly from plant cell walls. On a dry weight basis they account for 14% of rye flour, 8% of pearl barley, 7% of porridge oats, 3% of white wheat flour and 2% of brown rice (MacDougall & Selvendran, 2001). Nonstarch polysaccharides have been classified as soluble and insoluble but the distinction is not straightforward. β-Glucan is predominantly water soluble but not entirely, and 35% of the arabinoxylans from wheat endosperm cell walls are water soluble (MacDougall & Selvendran, 2001).

Much of the interest in dietary fibre and diabetes has centred on the effects of soluble fibre. Some studies in people with or without diabetes have shown that soluble fibre added to test drinks or to test meals induces a low glycaemic response. Fibres used to demonstrate this effect were extracted and purified from various sources and have included guar gum, glucomannan, psyllium, pectin, β-glucan, and arabinoxylan (Jenkins *et al*, 1976; Doi *et al*, 1979; Wolever *et al*, 1991; Tappy *et al*, 1996; Lu *et al*, 2000). The term soluble fibre includes a variety of compounds, some of which have gel-forming properties and others of which do not. Guar gum (galactomannan) and Konjac-Mannan (glucomannan) form gels having high viscosities, and it is this property that has been suggested as the mechanism by which these fibres reduce postprandial glycaemia (Jenkins *et al*, 1978). When the viscous property of guar gum was removed by partial hydrolysis, the nonviscous guar added to a glucose meal had no effect on postprandial measures of glucose and insulin responses compared with a control beverage (Jenkins *et al*, 1978). Of these fibre extracts, guar gum has been the most frequently used to investigate the effect of adding supplemental fibre to the diets of diabetic people. Results of these studies have been mixed, with some showing improvement in measures of glycaemic control (Aro *et al*, 1981; Ray *et al*, 1983), while others found no effect (Cohen *et al*, 1980; Carroll *et al*, 1981). Compliance may have been an issue, because guar gum in the quantities given was not well tolerated by all participants. Also, the method of adding fibre affects the glycaemic response, with best results obtained when fibre is mixed with food (Wolever *et al*, 1991). Glucomannan added to biscuits that were given to people with insulin resistance and to people with T2DM lowered serum fructosamine concentrations and improved indicators of blood lipids, but did not lower fasting blood glucose or insulin concentrations compared with control biscuits containing added wheat bran (Vuksan *et al*, 1999, 2000). Adding the fibre and protein fractions isolated from beans to potato flakes tested the role of fibre in starch utilisation (Tappy *et al*, 1986). Various measures of *in vitro* and *in vivo* responses were lower using 50 g of starch from beans compared with responses to 50 g of starch from potato flakes that included the bean fibre fraction. It was suggested that the slow carbohydrate property of the beans might be related to structure rather than to the fibre content.

Despite some evidence therefore to suggest that an increased intake of soluble fibre improves glycaemic control, soluble fibre intake is not associated with incident T2DM in prospective studies (Meyer *et al*, 2000; Montonen *et al*, 2003). A possible explanation is that the quantity of fibre consumed in an average diet does not contain sufficient soluble fibre, particularly of the gel-forming type, to have a distinguishable effect on glycaemic control. The range of median intakes of soluble fibre across quintiles of soluble fibre intake in 35 988 middle-aged to elderly North American women was 4–8 g/day (Meyer *et al*, 2000). In contrast, long-term intervention studies in which improvement in glycaemic control has been found have provided soluble fibre (guar gum) in quantities of around 10–20 g/day (Aro *et al*, 1981; Ray *et al*, 1983; Jones *et al*, 1985). It is possible to increase soluble fibre intake to this extent from dietary changes. Chandalia *et al* (2000) increased the average soluble fibre intake of people with T2DM from 8 to 25 g/day. Improvements in glycaemic control were attributed largely to an increase in soluble fibre intake. A glycaemic-lowering mechanism is plausible because oat bran and oatmeal were used, products that have been found to increase the supernatant viscosity of the intestinal contents of rats (Gallaher *et al*, 1999). In humans, the addition of oat bran and oat gum to meals induced lower postprandial glucose and insulin responses in healthy controls and in people with T2DM compared with a wheat meal (Braaten *et al*, 1994). Although increasing viscosity is a probable mechanism by which oat bran and gum slow the rate of digestion and absorption of glycaemic carbohydrate, there is also an independent effect of grain structure. Boiled oat kernels elicited lower postprandial glucose and insulin responses than cold rolled oats (muesli) or boiled rolled oat porridge (Granfeldt *et al*, 1995). Thus, soluble fibre has been shown to have some effect on indicators of glycaemic control in supplemental form and when added as oat products, and is associated with better glycaemic control when dietary changes result in a two-fold increase in soluble fibre intake, but it is not associated with risk reduction in incident T2DM at the levels consumed in the population groups studied.

Several studies have shown that an increased consumption of total dietary fibre is associated with improvements in measures of glycaemic control (Table 2). However, the use of whole foods as a means of increasing the dietary fibre content cannot separate the effect of fibre from that of food structure. *In vitro*, bread made from 80% whole wheat grains was either mechanically ground, or chewed and expectorated, before being tested using digestive systems (Holm & Bjorck, 1992). There was no difference in the hydrolysis index between the ground grain bread (105%) and white reference bread (100%) indicating that the presence of fibre made no difference to the rate of *in vitro* starch hydrolysis. In contrast, the chewed sample had a hydrolysis index that was 21% lower than the reference white bread. The presence of

whole or partially disintegrated grains in the digestas indicated that incomplete chewing of the grains had prevented or delayed enzymic action. *In vivo*, no difference in the GI was found comparing bread meals made from milled wholemeal or white wheat flour (Jenkins *et al*, 1986). These results indicate that the fibre contained in the bread made from milled wholemeal flour made no difference to postprandial glycaemic response. Further, the addition of wheat bran, a rich source of insoluble fibre, to the diets of diabetic people has been shown to have little effect on measures of glycaemic control (Cohen *et al*, 1980; Beattie *et al*, 1988). A recent, well-controlled 6-month randomised crossover trial involving 23 people with T2DM found that wheat bran added to bakery products made no difference to fasting blood glucose or HbA_{1C} concentrations (Jenkins *et al*, 2002).

Results from these metabolic and intervention studies appear to be in conflict with prospective studies that show a protective effect of cereal fibre on the development of T2DM (Salmeron *et al*, 1997a, b; Stevens *et al*, 2002). It is possible that cereal fibre is a marker for whole grain foods. Certainly, the risk reduction of 20–30% for T2DM found in studies that have recorded whole grain intake (Table 1) is similar to the risk reduction found when recording cereal fibre intake. Resolution of this issue requires a definition of whole grain that differentiates fibre obtained from grains having an intact structure as distinct from cereal fibre that has been derived from processed grains in which the bran, including outer layers and the aleurone layer, and the germ have been separated from the endosperm. While substantial increases in soluble fibre improves glycaemic control in people with diagnosed diabetes, the role of soluble fibre in the prevention of diabetes is less clear. This may be because the range of usual dietary intake is insufficient to see an effect and that doubling or trebling soluble fibre intake is required. To help achieve this, oats and barley, and in particular oat bran, are good sources of β-glucans, soluble fibres that are highly viscous.

Cooking

Ungelatinised starch granules resist digestion by α-amylase. Gelatinisation normally occurs during cooking in a process that is dependent on available water, time and temperature. Undercooking rice produced lower *in vitro* digestibility and blood glucose responses compared with fully cooked rice (Panlasigui *et al*, 1991). Retrogradation is a process in which gelatinised starch reassociates to form aggregate compounds that are partly resistant to attack by α-amylase. Retrogradation may occur during cooling of foods, freezing, freeze-drying and storage (Englyst & Cummings, 1985). Retrogradation and structure are plausible explanations for the lower glycaemic response reported in some studies to parboiled rice compared with nonparboiled rice (Wolever *et al*, 1986). The glycaemic-lowering effect may be dependent on the extent of parboiling. Rice that had been processed using modern methods of high-temperature water steeping and pressure cooking had a GI lower than that obtained with the nonparboiled rice (Larsen *et al*, 2000). The extent to which the glycaemic effect of grains other than rice is influenced by cooking has been less fully investigated. The consequences of gelatinisation and retrogradation are clearly not consistent. Incomplete gelatinisation of muesli (62%) made no difference to glycaemic or insulinaemic responses compared with cooked porridge that was essentially completely gelatinised (97–99%) (Granfeldt *et al*, 1995). Clearly more work is required to determine the extent to which glycaemic response to food can be altered by methods of food preparation.

Amylose content

Starch is a complex glucose polymer that can be present as amylopectin, a highly branched molecule or amylose that predominantly has a linear structure. Starches with high amylose content have high gelatinisation temperature that may not be reached in conventional cooking practices and amylose retrogrades at a faster rate and to more amylase-resistant crystalline structures than amylopectin. These properties render amylose less susceptible to digestion than amylopectin and provide a means to manipulate starch digestion based on the relative proportions of the starch fractions. The proportion of starch present as amylose in different varieties of grain varies considerably, particularly for varieties of corn, barley and rice. Grains with different amylose contents have been tested to determine whether starch structure affects the rate and extent of digestibility. Postprandial glucose and insulin response curves were approximately two-fold smaller, and the rate of starch hydrolysis was slower, using corn bread made from flour extracted from a high amylose corn variety (70–75% amylose) compared with corn bread made from ''ordinary'' corn flour (25% amylose) (Granfeldt *et al*, 1995). However, despite experimental evidence suggesting that amylose content is an important determinant of glycaemic control, several data sets suggest that the effect may not be as striking as first considered or that the structure of the grain may be relevant.

Three varieties of rice differing in amylose contents were fed to 33 healthy adults. The rices contained nil, $\sim$15% or $\sim$25% of carbohydrate in the form of amylose. The areas under the 3 h plasma glucose curves were not different among rice varieties, although the 25% amylose rice induced a lower insulin response compared with the rice varieties containing less amylose (Goddard *et al*, 1984). Four varieties of barley containing 7, 33, 42 and 44% amylose as a percentage of starch were fed to healthy adults either as boiled whole kernels (all four varieties) or as ground barley porridge (the 33 and 42% varieties) (Granfeldt *et al*, 1994). Despite a large variation in the amylose contents, the effect on plasma glucose and insulin responses was marginal when making comparisons among kernel meals or between the

ground meals. Although a grain variety having a high amylose/amylopectin ratio might be predicted to induce a lower glycaemic response to one containing a higher proportion of amylopectin, the evidence suggests that this is not true in all cases. In the study by Granfeldt *et al* (1994), an intact grain structure elicited lower glucose and insulin responses than the ground meals. Thus, the effect on measures of carbohydrate metabolism determined by structure and possibly particle size appears to be appreciably greater than the effect of starch type.

Satiety

It has been suggested that unrefined carbohydrate foods with low energy density will prolong eating time and induce satiation at a low energy intake (Weinsier *et al*, 1982). Duncan *et al* provided 10 obese and 10 nonobese subjects with diets low or high in energy density. The low energy density diet included large amounts of whole grain foods, legumes, fruits and vegetables. All food was provided in a randomised crossover design for a period of 5 days on each diet and the subjects were encouraged to eat to satiation. Eating time was longer and satiation was reached at a lower mean daily energy intake during the low energy density diet (Duncan *et al*, 1983). In studies that have replaced low carbohydrate diets with equicaloric high carbohydrate, high fibre diets, subjects have commented on the increased volume of food (O'Dea *et al*, 1980; Kinmonth *et al*, 1982; Chandalia *et al*, 2000). This effect may be partly due to the larger volume of unrefined foods causing gastric distension sufficient to induce satiation at a low energy intake. Liquid meals fed to rats has shown that gastric volume rather than nutrient load in the stomach inhibits food intake (Phillips & Powley, 1996). Apart from the bulk of food, it has been hypothesised that high glycaemic foods promote a more rapid return to hunger because of counterregulatory hormonal responses to rapid increases in postprandial glycaemia (Roberts & Heyman, 2000). This explanation is consistent with the finding of a positive association between the particle size of wheat and satiety (Holt & Miller, 1994). Satiety may also be influenced by substrate oxidation. A diet high in cereal and legume intake elicited a lower and delayed rise of postprandial carbohydrate oxidation and was associated with lower hunger feelings than a diet of refined carbohydrates (Sparti *et al*, 2000). These studies support a role for high fibre, low glycaemic foods in promoting satiety, but although reaching satiety is a plausible mechanism whereby energy intake might be restricted, it is not clear to what extent a diet high in unrefined foods achieves this. In some large population studies, whole grain intake has been positively associated with total energy intake (Liu *et al*, 2000; Meyer *et al*, 2000). This apparent discrepancy between whole grain foods promoting satiety in acute studies yet being positively associated with energy intake on a population basis may be explained in part by a positive association between whole grain food intake and level of physical activity (Liu *et al*, 2000; Meyer *et al*, 2000).

Second meal effect

The ''second meal effect'' describes the glycaemic and/or other physiological responses to a ''standard'' meal fed several hours after the feeding of a previous meal. It has been shown that consumption of a breakfast having a low GI lowers the glycaemic response to a standard lunch taken 4 h later compared with the same lunchtime meal following a higher GI breakfast (Jenkins *et al*, 1982). A similar finding was obtained when an evening meal having a low GI lowered the following day's glycaemic response to a standard test breakfast (Wolever *et al*, 1988). The reason for this effect is not known. Rapidly digested carbohydrate can result in large rises in insulin followed by rapid falls in blood glucose, leading to the suggestion that serum fatty acid release is stimulated resulting in impaired carbohydrate tolerance (Jenkins *et al*, 1982). Another suggested mechanism is that the fermentation of undigested carbohydrate produces short-chain fatty acids in the gut that when absorbed suppress hepatic glucose output and serum-free fatty acids (Thorburn *et al*, 1993). To what extent the second meal effect might be influenced by the rate of carbohydrate digestion or by fermentable carbohydrate is uncertain. To address this question, Liljeberg *et al* (1999) conducted a series of experiments in which comparisons were made between high fibre/low fibre breads, spaghetti and bread (similar fibre contents), and bread with and without a spread of raw potato starch. The results showed that the content of fermentable carbohydrates did not influence glycaemia after the second meal. In another study, a second meal effect to a standardised lunch (lower blood glucose, insulin and triglycerides) was found following a spaghetti breakfast made from 100% Durum wheat compared with a white wheat bread breakfast (Liljeberg & Bjorck, 2000). The fibre contents of the breakfast meals were not reported but typically these products have a relatively low content of fermentable fibre suggesting that the low glycaemic property of spaghetti was predominantly responsible for the second meal effect in this study. These results indicate that meals having low glycaemic indices beneficially affect the glycaemic response to the following meal.

Nutrient retention

Some minerals are partially depleted during the refining of grains. Of particular interest in diabetes is magnesium. Refining wheat to white flour results in a 33% retention of magnesium in Australian flour compared with a 15% retention in the United States (Schroeder, 1971; Mugford, 1983; United States Department of Agriculture, 2003). Low blood levels of magnesium are associated with impaired insulin secretion and function, poor glycaemic control, and diabetic complications (Paolisso *et al*, 1992; White &

Campbell, 1993; Rosolova *et al*, 1997). Urinary excretion of magnesium is raised in people with diabetes and it has been suggested that hypomagnesaemia is a possible consequence of diabetes (Khan *et al*, 1999). However, prospective studies have found both low serum magnesium and low dietary magnesium intakes to be inversely associated with the incidence of T2DM, suggesting that low magnesium intake and status predates the diabetic condition (Colditz *et al*, 1992; Salmeron *et al*, 1997a, b; Kao *et al*, 1999; Meyer *et al*, 2000). Further, Liu *et al* (2000) found that the median dietary magnesium intakes of female nurses increased from 248 to 342 mg/day across quintiles of whole grain intake. The average requirement for magnesium in adult women estimated from balance studies is 255 mg/day (Institute of Medicine, 1999). This suggests that a diet largely devoid of whole grain foods, in which grain products are consumed in amounts typified by North American nurses, may result in a marginal magnesium intake, providing a plausible mechanism whereby whole grain foods protect against T2DM. Detracting from this argument is the finding of lower bioavailability of minerals from less refined grains due to higher faecal excretion rates (Reinhold *et al*, 1976). It is possible that mineral absorption is impaired by the presence of fibre and antinutrients such as phytic acid. However, a study in rats found that wholewheat flour was associated with higher magnesium absorption and bioavailability than white wheat flour (Levrat-Verny *et al*, 1999). Therefore, in the case of magnesium the four-fold higher content in wholewheat flour compared with white flour more than compensated for any impairment in absorption. Magnesium intake does not fully explain the protective effect of whole grain foods, because the association between whole grain food intake and incident T2DM was still significant (relative risk 0.81, 95% CI: 0.69, 0.96) after adjustment for fibre, magnesium and vitamin E intake (Liu *et al*, 2000). Thus, although results from prospective studies carried out in North America support the suggestion of a role for low dietary magnesium intakes in the aetiology of T2DM, whole grain intake has a protective effect beyond that of magnesium intake. An independent contribution to risk is difficult to assess because magnesium intake is correlated with other components of whole grain foods including fibre.

Discussion

The evidence from epidemiological studies as well as dietary intervention and metabolic studies strongly support the suggestion that whole grain foods and legumes protect against the development of T2DM and improve glycaemic control in those with T1DM and T2DM. The risk reduction is evident even when foods containing as little as 25% whole grain are consumed. Large prospective studies have consistently found that people consuming about three servings per day of whole grain foods are less likely to develop T2DM than people consuming <3 servings per week with a risk reduction in the order of 20–30% (Liu *et al*, 2000; Meyer *et al*, 2000; Fung *et al*, 2002). The association is robust after controlling for other risk factors such as age, body mass index, physical activity, total energy intake, smoking and alcohol intake with a dose–response across quintiles of whole grain food intake. Randomised controlled trials using lifestyle interventions that have included the use of whole grain foods have shown the potential to delay progression of IGT to T2DM and to reduce insulin resistance. It is not possible to determine the extent to which the different aspects of lifestyle interventions contribute to improved glycaemic control. Dietary factors might include an increase in dietary fibre and magnesium intake, and a lowering of dietary GI.

Some prospective studies have found cereal fibre intake to be inversely associated with incident T2DM (Salmeron *et al*, 1997a, b; Stevens *et al*, 2002). Fibre *per se* may have a beneficial effect, although these studies do not exclude the possibility that cereal fibre is a marker for another component of whole grain foods that is wholly or partially responsible for providing the benefit. The finding of a lower mortality rate among women consuming predominantly fibre from whole grain foods compared with women consuming an equal amount of fibre predominantly from refined grain sources would suggest an independent benefit of eating the whole grain over the fibre (Jacobs *et al*, 2000). Increasing fibre intake by increasing consumption of foods rich in fibre has been found to improve glycaemic control (Chandalia *et al*, 2000). However, an independent effect of fibre cannot be assumed because foods used to provide the additional fibre also tended to have an intact structure. Adding cereal fibre alone into the diet of people with T2DM for 3 months did not improve measures of glycaemic control (Jenkins *et al*, 2002).

While there are several plausible mechanisms by which whole grain foods and legumes might reduce diabetes risk and improve glycaemic control, some uncertainties remain. The evidence that whole grain foods and the largely insoluble forms of dietary fibre derived from cereals protect against T2DM is strong and consistent among prospective studies. It is possible that this protection is afforded by the intact structure of the cereal grains and pulses slowing digestion and partially restricting absorption of the glycaemic carbohydrate. Long-term benefit in diabetes prevention might also relate to differences in nutrient content between refined and unrefined grains. The strongest evidence of this is linked to the higher magnesium content of unrefined grains. While cereal-derived foods appear to be particularly beneficial in terms of reducing diabetes risk (Table 1), the use of whole grain cereals, vegetables, pulses and fruits rich in dietary fibre and with a low GI are effective in improving glycaemic control in those who have established diabetes (Table 2). It seems appropriate therefore to recommend in the context of both prevention and management of diabetes that a variety of whole grain foods and legumes be consumed. Of greater practical importance is the issue as to

whether much additional benefit is likely to be gained from consuming the intact grain or whether most of the benefit can be derived from whole grain products that have been partially processed to facilitate consumption. Multivariate analyses of some of the epidemiological studies suggest that the constituents of whole grain account for some of the beneficial effect, an indication that is confirmed by some experimental studies. However, neither epidemiological nor experimental studies exclude a potential additional benefit from consuming the intact structure. Indeed, there is evidence from the studies of Järvi *et al* (1995, 1999) that leaving the structure of the plant food intact does confer considerable benefit. Confirmation of the benefit of plant structure is required and it would certainly be possible to devise an appropriate experimental approach.

Most of the epidemiological studies showing a protective effect of whole grain foods on incident diabetes used the definition of whole grain defined by Jacobs, which does not distinguish whether the grain is intact or simply includes the appropriate constituents. Currently, there is no internationally agreed definition of whole grain food that may include a variety of products including the intact grain, cracked grain, coarsely ground grain, finely milled reconstituted grain or a blend of these constituents mixed with refined flour and other ingredients. Perhaps the best indication of the need for such a definition is wholemeal bread. Milling of the grain results in wholemeal flour that, when made into bread, has a glycaemic effect no different to that of bread made from refined white flour. Consumption of intact grains on the other hand has been shown to produce a lower glycaemic response compared with an equivalent intake of bread made from wholemeal flour (Jenkins *et al*, 1986). Thus, grains having an intact structure may offer a benefit in terms of glycaemic control over finely ground ''wholemeal'' products.

In the absence of agreed Food Standards, a lack of definition of whole grain products may lead to consumers in many countries being misled with regard to the potential benefits of whole grain foods. The definition developed by the American Association of Cereal Chemists may be the most appropriate interim definition until the issue has been finally resolved: ''Whole grains may be intact, ground, cracked or flaked grains where the components—endosperm, germ and bran—are present in substantially the same proportions as they exist in the intact grain.'' This definition relates to the constituents of the grain rather than to the structure and does not address the issue as to what proportion of a food must consist of whole grain in order to be classified as a whole grain food. In terms of lessening the acute effect of consuming grains on postprandial glucose metabolism, ''ground'' might best be interpreted as ''stone ground'', rather than finely milled reconstituted wholemeal flour.

When considering the benefits of whole grain foods in relation to diabetes, it is important to emphasise that cereal grains and legumes may confer benefits that extend beyond their effects on glycaemia; cardiovascular disease is a common cause of serious morbidity and mortality in diabetes. Whole grain foods have been clearly shown to be associated with a reduced risk of ischaemic heart disease (Truswell, 2002). Part of the favourable effect may be due to lowering of lipoprotein-mediated risk, notably a reduction in plasma total cholesterol and/or LDL cholesterol and triglycerides (Järvi *et al*, 1999; Chandalia *et al*, 2000). In addition, the reduction in plasminogen activator inhibitor-1 activity found when comparing low and high GI diets (partially accounted for by a difference in consumption of cereal grains and legumes) might explain some of the benefit via a reduction in thrombogenic risk (Järvi *et al*, 1999). The most convincing experimental data supporting the benefit of cereal grains and legumes are derived from studies in which comparisons are made between them and refined carbohydrate foods in diets with comparable macronutrient composition (Chandalia *et al*, 2000). However, it seems likely that even greater benefit will ensue in terms of both glucose and lipoprotein metabolism if low energy-dense unrefined foods replace an appreciable proportion of high-fat and high-sugar energy-dense foods, especially if saturated fatty acids are thereby reduced.

Given the epidemic nature of T2DM, it is important to emphasise that a diet high in unrefined cereal grain foods and legumes is appropriate for the general population as well as for high-risk families and individuals with diabetes. Thus, there should be no need for those with T1DM and T2DM to have meals that differ from those which are appropriate for the rest of the family and the community. Unfortunately, substantial barriers to achieving an increased consumption of whole grain and legume foods remain (Adams & Engstrom, 2000). These include consumer preference for refined products, lack of availability of whole grains and whole grain products in supermarkets and restaurants, limited choice, price, lack of knowledge of health benefit, product labelling, unfamiliarity with cooking techniques, lack of recipes, familial and cultural eating habits, and reluctance to change. Substantial initiatives from the government and private sectors are needed to make whole grain foods and legumes a high priority dietary component. It is also important for health professionals and the general public to appreciate the distinction between whole grain cereal foods and legumes, and other foods relatively high in carbohydrate that are refined and rapidly digested and absorbed. In particular, there is a need for a consistent definition of the term ''whole grain'' and appropriate food standards regarding the proportion of whole grains required in a food in order for it to be classed as a whole grain food, which will together help guide consumer choices.

Summary points

Whole grain foods are protective against the development type II diabetes.

Whole grain foods and legumes improve indicators of glucose, lipid and lipoprotein metabolism in people with diabetes and in healthy people.

The acute metabolic advantage in glucose handling appears at least in part to be due to the intact structure of the grain or legume.

The extent to which the intact structure of grains and legumes contribute to the beneficial effect in terms of prevention and management of diabetes remain to be quantified.

References

Adams JF & Engstrom A (2000): Helping consumers achieve recommended intakes of whole grain foods. *J. Am. Coll. Nutr.* **19**, 339S–344S.

Anderson JW & Ward K (1979): High-carbohydrate, high-fiber diets for insulin-treated men with diabetes mellitus. *Am. J. Clin. Nutr.* **32**, 2312–2321.

Anderson JW, Chen WJ & Sieling B (1980): Hypolipidemic effects of high-carbohydrate, high-fiber diets. *Metabolism* **29**, 551–558.

Anderson JW, Zeigler JA, Deakins DA, Floore TL, Dillon DW, Wood CL, Oeltgen PR & Whitley RJ (1991): Metabolic effects of high-carbohydrate, high-fiber diets for insulin-dependent diabetic individuals. *Am. J. Clin. Nutr.* **54**, 936–943.

Anon (2000): Recommendations for the nutritional management of patients with diabetes mellitus. *Eur. J. Clin. Nutr.* **54**, 353–355.

Anon (2002): The Diabetes Prevention Program (DPP): description of lifestyle intervention. *Diabetes Care* **25**, 2165–2171.

Aro A, Uusitupa M, Voutilainen E, Hersio K, Korhonen T & Siitonen O (1981): Improved diabetic control and hypocholesterolaemic effect induced by long-term dietary supplementation with guar gum in type 2 (insulin-independent) diabetes. *Diabetologia* **21**, 29–33.

Beattie VA, Edwards CA, Hosker JP, Cullen DR, Ward JD & Read NW (1988): Does adding fibre to a low energy, high carbohydrate, low fat diet confer any benefit to the management of newly diagnosed overweight type II diabetics? *BMJ. (Clin. Res. Ed)* **296**, 1147–1149.

Braaten JT, Scott FW, Wood PJ, Riedel KD, Wolynetz MS, Brule D & Collins MW (1994): High beta-glucan oat bran and oat gum reduce postprandial blood glucose and insulin in subjects with and without type 2 diabetes. *Diabet. Med.* **11**, 312–318.

Brand JC, Colagiuri S, Crossman S, Allen A, Roberts DC & Truswell AS (1991): Low-glycemic index foods improve long-term glycemic control in NIDDM. *Diabetes Care* **14**, 95–101.

Campbell AM & Penfield MP (1979): *Starch and Flour. The Experimental Study of Food*. Houghton: Miffling Company.

Carroll DG, Dykes V & Hodgson W (1981): Guar gum is not a panacea in diabetes management. *N. Z. Med. J.* **93**, 292–294.

Chandalia M, Garg A, Lutjohann D, von Bergmann K, Grundy SM & Brinkley LJ (2000): Beneficial effects of high dietary fiber intake in patients with type 2 diabetes mellitus. *N. Engl. J. Med.* **342**, 1392–1398.

Cohen M, Leong VW, Salmon E & Martin FI (1980): Role of guar and dietary fibre in the management of diabetes mellitus. *Med. J. Aust.* **1**, 59–61.

Colditz GA, Manson JE, Stampfer MJ, Rosner B, Willett WC & Speizer FE (1992): Diet and risk of clinical diabetes in women. *Am. J. Clin. Nutr.* **55**, 1018–1023.

Doi K, Matsuura M, Kawara A & Baba S (1979): Treatment of diabetes with glucomannan (konjac mannan). *Lancet* **1**, 987–988.

Duncan KH, Bacon JA & Weinsier RL (1983): The effects of high and low energy density diets on satiety, energy intake, and eating time of obese and nonobese subjects. *Am. J. Clin. Nutr.* **37**, 763–767.

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

FAO/WHO (1998): Carbohydrates in human nutrition. FAO Food and Nutrition Paper-66.

FDA (1999): Health claim notification for whole grain foods. Internet: http://www.cfsan.fda.gov/~dms/flgrains.html accessed 10th March 2003.

Fontvieille AM, Rizkalla SW, Penfornis A, Acosta M, Bornet FR & Slama G (1992): The use of low glycaemic index foods improves metabolic control of diabetic patients over five weeks. *Diabet. Med.* **9**, 444–450.

Foster-Powell K, Holt SH & Brand-Miller JC (2002): International table of glycemic index and glycemic load values: 2002. *Am. J. Clin. Nutr.* **76**, 5–56.

Frost G, Wilding J & Beecham J (1994): Dietary advice based on the glycaemic index improves dietary profile and metabolic control in type 2 diabetic patients. *Diabet. Med.* **11**, 397–401.

Fukagawa NK, Anderson JW, Hageman G, Young VR & Minaker KL (1990): High-carbohydrate, high-fiber diets increase peripheral insulin sensitivity in healthy young and old adults. *Am. J. Clin. Nutr.* **52**, 524–528.

Fung TT, Hu FB, Pereira MA, Liu S, Stampfer MJ, Colditz GA & Willett WC (2002): Whole-grain intake and the risk of type 2 diabetes: a prospective study in men. *Am. J. Clin. Nutr.* **76**, 535–540.

Gallaher DD, Wood KJ, Gallaher CM, Marquart LF & Engstrom AM (1999): Intestinal contents supernatant viscosity of rats fed oat-based muffins and cereal products. *Cereal Chem.* **76**, 21–24.

Giacco R, Parillo M, Rivellese AA, Lasorella G, Giacco A, D'Episcopo L & Riccardi G (2000): Long-term dietary treatment with increased amounts of fiber-rich low-glycemic index natural foods improves blood glucose control and reduces the number of hypoglycemic events in type 1 diabetic patients. *Diabetes Care* **23**, 1461–1466.

Goddard MS, Young G & Marcus R (1984): The effect of amylose content on insulin and glucose responses to ingested rice. *Am. J. Clin. Nutr.* **39**, 388–392.

Golay A, Coulston AM, Hollenbeck CB, Kaiser LL, Wursch P & Reaven GM (1986): Comparison of metabolic effects of white beans processed into two different physical forms. *Diabetes Care* **9**, 260–266.

Granfeldt Y, Hagander B & Bjorck I (1995): Metabolic responses to starch in oat and wheat products. On the importance of food structure, incomplete gelatinization or presence of viscous dietary fibre. *Eur. J. Clin. Nutr.* **49**, 189–199.

Granfeldt Y, Liljeberg H, Drews A, Newman R & Bjorck I (1994): Glucose and insulin responses to barley products: influence of food structure and amylose-amylopectin ratio. *Am. J. Clin. Nutr.* **59**, 1075–1082.

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

Hjollund E, Pedersen O, Richelsen B, Beck-Nielsen H & Sorensen NS (1983): Increased insulin binding to adipocytes and monocytes and increased insulin sensitivity of glucose transport and metabolism in adipocytes from non-insulin-dependent diabetics after a low-fat/high-starch/high-fiber diet. *Metabolism* **32**, 1067–1075.

Hollenbeck CB, Coulston AM & Reaven GM (1986): To what extent does increased dietary fiber improve glucose and lipid metabolism in patients with noninsulin-dependent diabetes mellitus (NDDM)? *Am. J. Clin. Nutr.* **43**, 16–24.

Hollenbeck CB, Riddle MC, Connor WE & Leklem JE (1985): The effects of subject-selected high carbohydrate, low fat diets on glycemic control in insulin dependent diabetes mellitus. *Am. J. Clin. Nutr.* **41**, 293–298.

Holm J & Bjorck I (1992): Bioavailability of starch in various wheat-based bread products: evaluation of metabolic responses in

healthy subjects and rate and extent of *in vitro* starch digestion. *Am. J. Clin. Nutr.* **55**, 420–429.

Holt SH & Miller JB (1994): Particle size, satiety and the glycaemic response. *Eur. J. Clin. Nutr.* **48**, 496–502.

Institute of Medicine (1999): *Magnesium, Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride*. Washington DC: National Academy Press.

Jacobs Jr DR, Meyer KA, Kushi LH & Folsom AR (1998): Whole-grain intake may reduce the risk of ischemic heart disease death in postmenopausal women: the Iowa Women's Health Study. *Am. J. Clin. Nutr.* **68**, 248–257.

Jacobs DR, Pereira MA, Meyer KA & Kushi LH (2000): Fiber from whole grains, but not refined grains, is inversely associated with all-cause mortality in older women: the Iowa women's health study. *J. Am. Coll. Nutr.* **19**, 326S–330S.

Jang Y, Lee JH, Kim OY, Park HY & Lee SY (2001): Consumption of whole grain and legume powder reduces insulin demand, lipid peroxidation, and plasma homocysteine concentrations in patients with coronary artery disease: randomized controlled clinical trial. *Arterioscler. Thromb. Vasc. Biol.* **21**, 2065–2071.

Järvi AE, Karlstrom BE, Granfeldt YE, Bjorck IE, Asp NG & Vessby BO (1999): Improved glycemic control and lipid profile and normalized fibrinolytic activity on a low-glycemic index diet in type 2 diabetic patients. *Diabetes Care* **22**, 10–18.

Järvi AE, Karlstrom BE, Granfeldt YE, Bjorck IM, Vessby BO & Asp NG (1995): The influence of food structure on postprandial metabolism in patients with non-insulin-dependent diabetes mellitus. *Am. J. Clin. Nutr.* **61**, 837–842.

Jenkins DJ, Goff DV, Leeds AR, Alberti KG, Wolever TM, Gassull MA & Hockaday TD (1976): Unabsorbable carbohydrates and diabetes: Decreased post-prandial hyperglycaemia. *Lancet* **2**, 172–174.

Jenkins DJ, Kendall CW, Augustin LS, Martini MC, Axelsen M, Faulkner D, Vidgen E, Parker T, Lau H, Connelly PW, Teitel J, Singer W, Vandenbroucke AC, Leiter LA & Josse RG (2002): Effect of wheat bran on glycemic control and risk factors for cardiovascular disease in type 2 diabetes. *Diabetes Care* **25**, 1522–1528.

Jenkins DJ, Wolever TM, Buckley G, Lam KY, Giudici S, Kalmusky J, Jenkins AL, Patten RL, Bird J & Wong GS *et al.* (1988): Low-glycemic-index starchy foods in the diabetic diet. *Am. J. Clin. Nutr.* **48**, 248–254.

Jenkins DJ, Wolever TM, Collier GR, Ocana A, Rao AV, Buckley G, Lam Y, Mayer A & Thompson LU (1987): Metabolic effects of a low-glycemic-index diet. *Am. J. Clin. Nutr.* **46**, 968–975.

Jenkins DJ, Wolever TM, Jenkins AL, Giordano C, Giudici S, Thompson LU, Kalmusky J, Josse RG & Wong GS (1986): Low glycemic response to traditionally processed wheat and rye products: bulgur and pumpernickel bread. *Am. J. Clin. Nutr.* **43**, 516–520.

Jenkins DJ, Wolever TM, Leeds AR, Gassull MA, Haisman P, Dilawari J, Goff DV, Metz GL & Alberti KG (1978): Dietary fibres, fibre analogues, and glucose tolerance: importance of viscosity. *BMJ* **1**, 1392–1394.

Jenkins DJ, Wolever TM, Taylor RH, Griffiths C, Krzeminska K, Lawrie JA, Bennett CM, Goff DV, Sarson DL & Bloom SR (1982): Slow release dietary carbohydrate improves second meal tolerance. *Am. J. Clin. Nutr.* **35**, 1339–1346.

Jimenez-Cruz A, Bacardi-Gascon M, Turnbull WH, Rosales-Garay P & Severino-Lugo I (2003): A flexible, low-glycemic index mexican-style diet in overweight and obese subjects with type 2 diabetes improves metabolic parameters during a 6-week treatment period. *Diabetes Care* **26**, 1967–1970.

Jones DB, Slaughter P, Lousley S, Carter RD, Jelfs R & Mann JI (1985): Low-dose guar improves diabetic control. *J. R. Soc. Med.* **78**, 546–548.

Kao WH, Folsom AR, Nieto FJ, Mo JP, Watson RL & Brancati FL (1999): Serum and dietary magnesium and the risk for type 2 diabetes mellitus: the Atherosclerosis Risk in Communities Study. *Arch. Intern. Med.* **159**, 2151–2159.

Karlstrom B, Vessby B, Asp NG, Boberg M, Lithell H & Berne C (1987): Effects of leguminous seeds in a mixed diet in non-insulin-dependent diabetic patients. *Diabetes Res* **5**, 199–205.

Kay RM, Grobin W & Track NS (1981): Diets rich in natural fibre improve carbohydrate tolerance in maturity-onset, non-insulin dependent diabetics. *Diabetologia* **20**, 18–21.

Khan LA, Alam AM, Ali L, Goswami A, Hassan Z, Sattar S, Banik NG & Khan AK (1999): Serum and urinary magnesium in young diabetic subjects in Bangladesh. *Am. J. Clin. Nutr.* **69**, 70–73.

Kiehm TG, Anderson JW & Ward K (1976): Beneficial effects of a high carbohydrate, high fiber diet on hyperglycemic diabetic men. *Am. J. Clin. Nutr.* **29**, 895–899.

Kinmonth AL, Angus RM, Jenkins PA, Smith MA & Baum JD (1982): Whole foods and increased dietary fibre improve blood glucose control in diabetic children. *Arch. Dis. Child.* **57**, 187–194.

Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA & Nathan DM (2002): Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N. Engl. J. Med.* **346**, 393–403.

Larsen HN, Rasmussen OW, Rasmussen PH, Alstrup KK, Biswas SK, Tetens I, Thilsted SH & Hermansen K (2000): Glycaemic index of parboiled rice depends on the severity of processing: study in type 2 diabetic subjects. *Eur. J. Clin. Nutr.* **54**, 380–385.

Leeds AR (1979): The dietary management of diabetes in adults. *Proc. Nutr. Soc.* **38**, 365–371.

Levrat-Verny MA, Coudray C, Bellanger J, Lopez HW, Demigne C, Rayssiguier Y & Remesy C (1999): Wholewheat flour ensures higher mineral absorption and bioavailability than white wheat flour in rats. *Br. J. Nutr.* **82**, 17–21.

Liljeberg H & Bjorck I (2000): Effects of a low-glycaemic index spaghetti meal on glucose tolerance and lipaemia at a subsequent meal in healthy subjects. *Eur. J. Clin. Nutr.* **54**, 24–28.

Liljeberg HG, Akerberg AK & Bjorck IM (1999): Effect of the glycemic index and content of indigestible carbohydrates of cereal-based breakfast meals on glucose tolerance at lunch in healthy subjects. *Am. J. Clin. Nutr.* **69**, 647–655.

Liljeberg H, Granfeldt Y & Bjorck I (1992): Metabolic responses to starch in bread containing intact kernels versus milled flour. *Eur. J. Clin. Nutr.* **46**, 561–575.

Liu S, Manson JE, Stampfer MJ, Hu FB, Giovannucci E, Colditz GA, Hennekens CH & Willett WC (2000): A prospective study of whole-grain intake and risk of type 2 diabetes mellitus in US women. *Am. J. Public Health* **90**, 1409–1415.

Lu ZX, Walker KZ, Muir JG, Mascara T & O'Dea K (2000): Arabinoxylan fiber, a byproduct of wheat flour processing, reduces the postprandial glucose response in normoglycemic subjects. *Am. J. Clin. Nutr.* **71**, 1123–1128.

MacDougall AJ & Selvendran RR (2001): Chemistry, architecture, and composition of dietary fiber from plant cell walls. In *The Handbook of Dietary Fiber* eds Cho SS & Dreher ML. New York: Marcel Dekker Inc.

Mann JI (2001): Dietary fibre and diabetes revisited. *Eur. J. Clin. Nutr.* **55**, 919–921.

Mann JI (2002): Diet and risk of coronary heart disease and type 2 diabetes. *Lancet.* **360**, 783–789.

Maron DJ, Fair JM & Haskell WL (1991): Saturated fat intake and insulin resistance in men with coronary artery disease. The Stanford Coronary Risk Intervention Project Investigators and Staff. *Circulation* **84**, 2020–2027.

McAuley KA, Williams SM, Mann JI, Goulding A, Chisholm A, Wilson N, Story G, McLay RT, Harper MJ & Jones IE (2002): Intensive lifestyle changes are necessary to improve insulin sensitivity: a randomized controlled trial. *Diabetes Care* **25**, 445–452.

McKeown NM, Meigs JB, Liu S, Wilson PW & Jacques PF (2002): Whole-grain intake is favorably associated with metabolic risk factors for type 2 diabetes and cardiovascular disease in the Framingham Offspring Study. *Am. J. Clin. Nutr.* **76**, 390–398.

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

Montonen J, Knekt P, Jarvinen R, Aromaa A & Reunanen A (2003): Whole-grain and fiber intake and the incidence of type 2 diabetes. *Am. J. Clin. Nutr.* **77**, 622–629.

Mugford D (1983): Nutritional composition of Aust wheat and bakers' flour. *Austral. Bakers Millers J.* **86**, 27–29.

North American Millers Association (2000): How Wheat Flour is Milled. Internet: http://www.namamillers.org/prd_w_mill.html accessed 10th March 2003.

O'Dea K & Wong S (1983): The rate of starch hydrolysis *in vitro* does not predict the metabolic responses to legumes *in vivo*. *Am. J. Clin. Nutr.* **38**, 382–387.

O'Dea K, Nestel PJ & Antonoff L (1980): Physical factors influencing postprandial glucose and insulin responses to starch. *Am. J. Clin. Nutr.* **33**, 760–765.

O'Dea K, Traianedes K, Ireland P, Niall M, Sadler J, Hopper J & De Luise M (1989): The effects of diet differing in fat, carbohydrate, and fiber on carbohydrate and lipid metabolism in type II diabetes. *J. Am. Diet. Assoc.* **89**, 1076–1086.

O'Donnell LJ, Emmett PM & Heaton K.W (1989): Size of flour particles and its relation to glycaemia, insulinaemia, and colonic disease. *BMJ.* **298**, 1616–1617.

Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB, Liu PA, Jiang XG, Jiang YY, Wang JP, Zheng H, Zhang H, Bennett PH & Howard BV (1997): Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. *The Da Qing IGT and Diabetes Study. Diabetes Care* **20**, 537–544.

Panlasigui LN, Thompson LU, Juliano BO, Perez CM, Yiu SH & Greenberg GR (1991): Rice varieties with similar amylose content differ in starch digestibility and glycemic response in humans. *Am. J. Clin. Nutr.* **54**, 871–877.

Paolisso G, Sgambato S, Gambardella A, Pizza G, Tesauro P, Varricchio M & D'Onofrio F (1992): Daily magnesium supplements improve glucose handling in elderly subjects. *Am. J. Clin. Nutr.* **55**, 1161–1167.

Parillo M, Riccardi G, Pacioni D, Iovine C, Contaldo F, Isernia C, De Marco F, Perrotti N & Rivellese A (1988): Metabolic consequences of feeding a high-carbohydrate, high-fiber diet to diabetic patients with chronic kidney failure. *Am. J. Clin. Nutr.* **48**, 255–259.

Pereira MA, Jacobs Jr DR, Pins JJ, Raatz SK, Gross MD, Slavin JL & Seaquist ER (2002): Effect of whole grains on insulin sensitivity in overweight hyperinsulinemic adults. *Am. J. Clin. Nutr.* **75**, 848–855.

Phillips RJ & Powley TL (1996): Gastric volume rather than nutrient content inhibits food intake. *Am. J. Physiol.* **271**, R766–R769.

Pick ME, Hawrysh MI & Toth E (1998): Barley bread products improve glycemic control of type 2 subjects, *Int. J. Food. Sci. Nutr.* **49**, 71–78.

Ray TK, Mansell KM, Knight LC, Malmud LS, Owen OE & Boden G (1983): Long-term effects of dietary fiber on glucose tolerance and gastric emptying in noninsulin-dependent diabetic patients. *Am. J. Clin. Nutr.* **37**, 376–381.

Reinhold JG, Faradji B, Abadi P & Ismail-Beigi F (1976): Decreased absorption of calcium, magnesium, zinc and phosphorus by humans due to increased fiber and phosphorus consumption as wheat bread. *J. Nutr.* **106**, 493–503.

Riccardi G, Rivellese A, Pacioni D, Genovese S, Mastranzo P & Mancini M (1984): Separate influence of dietary carbohydrate and fibre on the metabolic control in diabetes. *Diabetologia* **26**, 116–121.

Rivellese A, Riccardi G, Giacco A, Pacioni D, Genovese S, Mattioli PL & Mancini M (1980): Effect of dietary fibre on glucose control and serum lipoproteins in diabetic patients. *Lancet* **2**, 447–450.

Roberts SB & Heyman MB (2000): Dietary composition and obesity: do we need to look beyond dietary fat? *J. Nutr.* **130**, 267S.

Rosolova H, Mayer Jr O & Reaven G (1997): Effect of variations in plasma magnesium concentration on resistance to insulin-mediated glucose disposal in nondiabetic subjects. *J. Clin. Endocrinol. Metab.* **82**, 3783–3785.

Salmeron J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, Stampfer MJ, Wing AL & Willett WC (1997a): Dietary fiber, glycemic load, and risk of NIDDM in men. *Diabetes Care* **20**, 545–550.

Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing AL & Willett WC (1997b): Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* **277**, 472–477.

Schroeder HA (1971): Losses of vitamins and trace minerals resulting from processing and preservation of foods. *Am. J. Clin. Nutr.* **24**, 562–573.

Simpson HC, Simpson RW, Lousley S, Carter RD, Geekie M, Hockaday TD & Mann JI (1981): A high carbohydrate leguminous fibre diet improves all aspects of diabetic control. *Lancet* **1**, 1–5.

Simpson RW, Mann JI, Eaton J, Carter RD & Hockaday TD (1979a): High-carbohydrate diets and insulin-dependent diabetics. *BMJ* **2**, 523–525.

Simpson RW, Mann JI, Eaton J, Moore RA, Carter R & Hockaday TD (1979b): Improved glucose control in maturity-onset diabetes treated with high-carbohydrate-modified fat diet. *BMJ* **1**, 1753–1756.

Simpson RW, McDonald J, Wahlqvist ML, Balasz N, Sissons M & Atley L (1988): Temporal study of metabolic change when poorly controlled noninsulin-dependent diabetics change from low to high carbohydrate and fiber diet. *Am. J. Clin. Nutr.* **48**, 104–109.

Snow P & O'Dea K (1981): Factors affecting the rate of hydrolysis of starch in food. *Am. J. Clin. Nutr.* **34**, 2721–2727.

Sparti A, Milon H, Di Vetta V, Schneiter P, Tappy L, Jequier E & Schutz Y (2000): Effect of diets high or low in unavailable and slowly digestible carbohydrates on the pattern of 24-h substrate oxidation and feelings of hunger in humans. *Am. J. Clin. Nutr.* **72**, 1461–1468.

Stevens J, Ahn K, Juhaeri, Houston D, Steffan L & Couper D (2002): Dietary fiber intake and glycemic index and incidence of diabetes in African-American and white adults: the ARIC study. *Diabetes Care* **25**, 1715–1721.

Story L, Anderson JW, Chen WJ, Karounos D & Jefferson B (1985): Adherence to high-carbohydrate, high-fiber diets: long-term studies of non-obese diabetic men. *J. Am. Diet. Assoc.* **85**, 1105–1110.

Tappy L, Gugolz E & Wursch P (1996): Effects of breakfast cereals containing various amounts of beta-glucan fibers on plasma glucose and insulin responses in NIDDM subjects. *Diabetes Care* **19**, 831–834.

Tappy L, Wursch P, Randin JP, Felber JP & Jequier E (1986): Metabolic effect of pre-cooked instant preparations of bean and potato in normal and in diabetic subjects. *Am. J. Clin. Nutr.* **43**, 30–36.

Thorburn A, Muir J & Proietto J (1993): Carbohydrate fermentation decreases hepatic glucose output in healthy subjects. *Metabolism* **42**, 780–785.

Trowell H (1974): Diabetes mellitus death-rates in England and Wales 1920-70 and food supplies. *Lancet* **2**, 998–1002.

Trowell H (1978): Diabetes mellitus and dietary fiber of starchy foods. *Am. J. Clin. Nutr.* **31**, S53–S57.

Truswell AS (2002): Cereal grains and coronary heart disease. *Eur. J. Clin. Nutr.* **56**, 1–14.

Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, Keinanen-Kiukaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V & Uusitupa M (2001): Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N. Engl. J. Med.* **344**, 1343–1350.

Turner RC, Cull CA, Frighi V & Holman RR (1999): Glycemic control with diet, sulfonylurea, metformin, or insulin in patients with type 2 diabetes mellitus: progressive requirement for multiple therapies (UKPDS 49). UK Prospective Diabetes Study (UKPDS) Group. *JAMA* **281**, 2005–2012.

United States Department of Agriculture (2003): National Nutrient Database for Standard Reference Release 15. Internet: www.nal.usda.gov/fnic/foodcomp accessed 7th July 2003.

Vinik AI, Wing RR & Lauterio TJ (1997): Nutritional management of the person with diabetes. *Ellenberg and Rifkin's Diabetes Mellitus* eds Porte D & Sherwin RS Connecticut: Appleton and Lange.

Vuksan V, Jenkins DJ, Spadafora P, Sievenpiper JL, Owen R, Vidgen E, Brighenti F, Josse R, Leiter LA & Bruce-Thompson C (1999): Konjac-mannan (glucomannan) improves glycemia and other associated risk factors for coronary heart disease in type 2 diabetes. A randomized controlled metabolic trial. *Diabetes Care* **22**, 913–919.

Vuksan V, Sievenpiper JL, Owen R, Swilley JA, Spadafora P, Jenkins DJ, Vidgen E, Brighenti F, Josse RG, Leiter LA, Xu Z & Novokmet R (2000): Beneficial effects of viscous dietary fiber from Konjac-mannan in subjects with the insulin resistance syndrome: results of a controlled metabolic trial. *Diabetes Care* **23**, 9–14.

Ward GM, Simpson RW, Simpson HC, Naylor BA, Mann JI & Turner RC (1982): Insulin receptor binding increased by high carbohydrate low fat diet in non-insulin-dependent diabetics. *Eur. J. Clin. Invest.* **12**, 93–96.

Weinsier RL, Johnston MH, Doleys DM & Bacon JA (1982): Dietary management of obesity: evaluation of the time-energy displacement diet in terms of its efficacy and nutritional adequacy for long-term weight control. *Br. J. Nutr.* **47**, 367–379.

White Jr JR & Campbell RK (1993): Magnesium and diabetes: a review. *Ann. Pharmacother.* **27**, 775–780.

WHO/FAO (2003): Diet, nutrition, and the prevention of chronic diseases. WHO Technical Report Series 916.

Wolever TM, Jenkins DJ, Kalnusky J, Jenkins A, Giordano C, Guidici S, Josse R & Wong GS (1986): Comparison of regular and parboiled rices: explanation of discrepancies between reported glycemic responses to rice. *Nutr. Res.* **6**, 349–357.

Wolever TM, Jenkins DJ, Ocana AM, Rao VA & Collier GR (1988): Second-meal effect: low-glycemic-index foods eaten at dinner improve subsequent breakfast glycemic response. *Am. J. Clin. Nutr.* **48**, 1041–1047.

Wolever TM, Vuksan V, Eshuis H, Spadafora P, Peterson RD, Chao ES, Storey ML & Jenkins DJ (1991): Effect of method of administration of psyllium on glycemic response and carbohydrate digestibility. *J. Am. Coll. Nutr.* **10**, 364–371.

Wong S & O'Dea K. (1983): Importance of physical form rather than viscosity in determining the rate of starch hydrolysis in legumes. *Am. J. Clin. Nutr.* **37**, 66–70.

Würsch P, Del Vedovo S & Koellreutter B (1986): Cell structure and starch nature as key determinants of the digestion rate of starch in legume. *Am. J. Clin. Nutr.* **43**, 25–29.