

Whole grain consumption and weight gain: a review of the epidemiological evidence, potential mechanisms and opportunities for future research

Pauline Koh-Banerjee^{1*} and Eric B. Rimm^{1,2,3}

Departments of ¹Nutrition and ²Epidemiology, Harvard School of Public Health, Boston, MA 02115, USA

³Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA 02115, USA

The epidemiological data that directly examine whole grain *v.* refined grain intake in relation to weight gain are sparse. However, recently reported studies offer insight into the potential role that whole grains may play in body-weight regulation due to the effects that the components of whole grains have on hormonal factors, satiety and satiation. In both clinical trials and observational studies the intake of whole-grain foods was inversely associated with plasma biomarkers of obesity, including insulin, C-peptide and leptin concentrations. Whole-grain foods tend to have low glycaemic index values, resulting in lower postprandial glucose responses and insulin demand. High insulin levels may promote obesity by altering adipose tissue physiology and by enhancing appetite. The fibre content of whole grains may also affect the secretion of gut hormones, independent of glycaemic response, that may act as satiety factors. Future studies may examine whether whole grain intake is directly related to body weight, and whether the associations are primarily driven by components of the grain, including dietary fibre, bran or germ.

Whole grains: Refined grains: Dietary fibre: Body weight

The prevalence of obesity in the USA is at its highest levels in history; an estimated 97 million adults are overweight (Expert Panel on the Identification, Evaluation and Treatment of Overweight in Adults, 1998). Obesity dramatically increases the risk of morbidity from chronic diseases, including hypertension, dyslipidaemia, type 2 diabetes, cardiovascular disease and certain types of cancer (Lapidus *et al.* 1984; Larsson *et al.* 1984; Lundgren *et al.* 1989; Freedman *et al.* 1995; Expert Panel on the Identification, Evaluation and Treatment of Overweight in Adults, 1998). Despite its public health importance, there is no scientific consensus on the optimal diet for the prevention and treatment of obesity. Much controversy exists over the roles that specific nutrients have on the aetiology of obesity, including the effects of dietary fats and carbohydrates. Due to the complex physical and chemical nature of foods, the investigation of whole foods in the development of obesity deserves further scientific analysis. In particular, diets that are characterized by high whole grain content and low glycaemic load may be of particular importance in the age-related progression to hyperinsulinaemia and obesity, due to

their potential influence on metabolic and hormonal effects. However, few studies to date have examined the potential roles of whole grain intake on body weight.

Whole grain consumption and chronic disease risk

Walker (1947), Burkitt (1952), Cleave (1956) and Trowell (1972) pioneered the concept that highly-refined foods contribute to Western diseases including coronary artery disease. Since that time, whole-grain foods have been associated with a reduced risk for several chronic diseases including CHD (Rimm *et al.* 1996; Jacobs *et al.* 1998; Willett, 1998; Liu *et al.* 1999), diabetes (Salmeron *et al.* 1997*a,b*) and certain types of cancer (Adlercreutz, 1990). Whole-grain foods may protect against chronic diseases by altering serum cholesterol profiles, exerting antioxidant properties and anti-thrombotic action, and through their favourable effects on vascular reactivity and insulin sensitivity (Anderson & Hanna, 1999). Whole grains are rich sources of dietary fibre, resistant starch, vitamins, minerals, phyto-oestrogens, antioxidants and other important nutrients

Abbreviation: GI, glycaemic index.

***Corresponding author:** Pauline Koh-Banerjee, fax +1 617 432 2435, email pkoh@hsph.harvard.edu

(Anderson *et al.* 2000). Structurally, whole grains are composed of the endosperm (approximately 80 %, w/w), the germ and the bran of the grain (Anderson *et al.* 2000). Bran contains the seed coat that comprises fatty acids, phytochemicals and fibre (Southgate, 1995). During the milling process, the bran (outer layer) and germ (inner layer) are separated from the starchy endosperm (middle layer), which leads to the loss of many nutrients and fibre (Slavin *et al.* 1997). For this reason, refined grains are nutritionally inferior to whole grains because they contain lower amounts of fibre, minerals, vitamins, phenols, phyto-oestrogens and unsaturated fatty acids (Jacobs *et al.* 1998; Willett, 1998). The whole-grain foods that are commonly consumed in Western cultures include dark bread, whole-grain breakfast cereals, popcorn, oatmeal and brown rice (Liu *et al.* 1999). However, most of the grain products consumed in the USA are highly refined (Slavin, 1994).

While whole-grain foods have been hypothesized to protect against obesity, epidemiological data that directly examine whole grain *v.* refined grain intake in relation to obesity are sparse. In the Iowa Women's Health Study whole grain intake was inversely correlated with body weight and fat distribution in comparison with a weaker direct relationship for refined grain consumption and body size (Jacobs *et al.* 1998). In the Coronary Artery Risk Development in Young Adults (CARDIA) study whole grain intake was inversely related to BMI at 7-year follow-up of the participants of the study. No association was observed between whole grain intake and the waist:hip ratio (Pereira *et al.* 1998).

While there are few direct studies on the association between whole grain intake and obesity, there are several studies that indicate an association between whole grain intake and insulin sensitivity, thereby providing indirect support for a causal role of whole grain consumption on body-weight regulation. Pereira *et al.* (1998) reported that whole grain intake was negatively related to fasting insulin levels even after adjustment for BMI, Mg and fibre. Similar associations were observed for cereal-bran intake and insulin levels. In the Framingham Offspring Study diets rich in whole grains were inversely associated with BMI, the waist:hip ratio, total cholesterol and LDL-cholesterol, and fasting insulin levels (McKeown *et al.* 2002). In another study among US male health professionals a dietary pattern characterized by higher intakes of fruit, vegetables, whole grains and poultry was inversely associated with plasma biomarkers of obesity, including insulin, C-peptide and leptin concentrations (Fung *et al.* 2001). In a clinical trial the isoenergetic replacement of refined rice with whole grain and legume powder as a source of carbohydrate in a meal showed marked beneficial effects on glucose and insulin concentrations in patients with coronary artery disease (Jang *et al.* 2001).

Carbohydrates with different physical forms, chemical structures, particle sizes and fibre content can induce distinct plasma glucose and insulin responses (Jenkins *et al.* 1981). The glycaemic index (GI) classifies foods based on the physiological response that they entail in comparison with the same amount of carbohydrate from a standard source, either white bread or pure glucose (Wolever & Jenkins, 1986). The glycaemic load represents the combi-

nation of quality and quantity of carbohydrates consumed (Liu *et al.* 1999). Whole-grain foods tend to have low GI values due to their starch content, particle size, extent of refinement and high content of viscous fibre (Liu *et al.* 2000). In contrast to refined-grain products, whole-grain foods are digested and absorbed more slowly, resulting in smaller postprandial glucose responses and insulin demand on the pancreatic β cells (Slavin, 1994). Refined-grain foods more than double the glycaemic and insulinaemic responses compared with whole-grain foods (Jacobs *et al.* 1998; Liu *et al.* 1999). Metabolic studies have shown that the insulin demand induced by various types of carbohydrate largely depends on the type or extent of digestibility of the starch content (Jenkins *et al.* 1981; Wolever & Jenkins, 1986). The starch in grains is composed of both long chains of glucose (amylose) and highly-branched chains of glucose (amylopectin). Since fewer glucose molecules are released when amylose chains are hydrolysed as compared with amylopectin chains, a high amylose:amylopectin content is associated with decreased glucose responses and insulin demand (Hallfrisch & Behall, 2000). Behall *et al.* (1988) observed that insulin responses were lower among men and women who consumed maize crackers containing 70 % amylose compared with 70 % amylopectin. Jenkins *et al.* (1988a) reported that lower glycaemic responses were associated with an increasing proportion of whole cereal grains in test breads.

Based on the epidemiological data, increased consumption of whole grains has been recommended to improve insulin sensitivity and to lower serum insulin concentrations (Jacobs *et al.* 1998; Willett, 1998; Anderson & Hanna, 1999). High insulin levels may promote obesity by altering adipose tissue physiology, shunting metabolic fuels from oxidation to storage and by increasing appetite (Ludwig *et al.* 1999b). Hyperinsulinaemia has been associated with excessive weight gain among adults and children in some (Odeleye *et al.* 1997; Folsom *et al.* 1998) but not all epidemiological studies (Folsom *et al.* 1998). In one crossover study researchers assessed the effects of high-GI foods on eating patterns and obesity among obese teenage boys. On three separate occasions subjects consumed identical test meals at breakfast and lunch that had low, medium or high GI values. The high- and medium-GI meals were designed to have similar macronutrient composition, fibre content and palatability, and all meals for each subject had equal energy content. Voluntary energy intake after the high-GI meal was 53% greater than after the medium-GI meal. In addition, the high-GI meal resulted in higher serum insulin levels, lower plasma glucagon levels, lower post-absorptive plasma glucose and serum fatty acid levels, and elevated plasma adrenaline (Ludwig *et al.* 1999a).

Dietary fibre component of whole grains

The carbohydrates in starchy foods with a low GI are mainly from less-processed grain products and dried legumes that maintain their original fibre content (Salmeron *et al.* 1997a,b). Indeed, dietary fibre has been found to explain approximately 40 % of the variance in the glycaemic response to foods (Trout *et al.* 1993). Fibre is a complex class of substances that can be broadly classified into soluble and

insoluble fibre types. The solubility of fibre depends on the extent to which the fibre dissolves in water or forms a gel (Howarth *et al.* 2001). In practical terms fibre consists of the edible NSP portion of plant foods that includes cellulose, hemicellulose, pectins, β -glucans, fructans, gums, mucilages and algal polysaccharides.

In cross-sectional observational studies fibre generally has been inversely associated with body weight (Alfieri *et al.* 1995) and body fat (Miller *et al.* 1994; Nelson & Tucker, 1996). The beneficial effects of fibre on energy regulation have been observed with soluble and insoluble fibres, although there are relatively few data that directly compare the effects of different fibre types (Howarth *et al.* 2001). Furthermore, the studies that have investigated the effects of dietary fibre on body weight have been limited by factors including cross-sectional design or short duration of follow-up, inadequate adjustment for confounding factors, and over-adjustment for total energy intake (Pereira & Ludwig, 2001). In one longitudinal investigation fibre was inversely associated with BMI at all levels of fat intake after adjustment for lifestyle factors and other confounding factors among young adults in the CARDIA study (Ludwig *et al.* 1999b). Fibre consumption further predicted insulin levels, 10-year weight gain and other cardiovascular disease risk factors including blood pressure and cholesterol levels after adjustment for possible confounding influences (Ludwig *et al.* 1999b).

Fibre may regulate body weight through its intrinsic effects and hormonal responses. High-fibre food may promote satiation (lower meal energy content) and satiety (longer duration between meals) due to its bulk (Raben *et al.* 1994) and relatively low energy density (Pereira & Ludwig, 2001), leading to decreased energy intake. Importantly, whole-grain products provide 20–50% of their fibre in the soluble or viscous forms (Chen & Anderson, 1986). Grains with high levels of soluble fibre, such as oats, rye and barley, may improve insulin sensitivity by slowing absorption of macronutrients (Hallfrisch & Behall, 2000). In contrast to insoluble fibre, soluble fibre results in highly viscous intestinal contents with gel-like properties that may delay gastric emptying and/or intestinal absorption (Hallfrisch & Behall, 2000). In the small intestine soluble fibre may blunt postprandial glycaemic and insulinaemic responses (Jenkins *et al.* 1988b) that are linked to reductions in the rate of return of hunger and subsequent energy intake in several previous studies (Roberts, 2000). It was observed that oat gum added to a glucose solution reduced glucose and insulin responses of healthy adults (Jenkins *et al.* 1978). Similar results were observed when highly viscous oat extracts were consumed by middle-aged men and women (Hallfrisch & Behall, 2000).

Fibre may also affect secretion of gut hormones, including cholecystokinin, independent of glycaemic response, that may act as satiety factors or alter glucose homeostasis (Pereira & Ludwig, 2001). Some studies have shown prolonged increases in circulating cholecystokinin after ingestion of fibre-rich meals relative to energy-matched low-fibre meals (Holt *et al.* 1992; Bourdon *et al.* 1999). Cholecystokinin is secreted from cells in the small intestine on ingestion of food, and functions in the stimulation of pancreatic secretion, regulation of gastric emptying and

central inducement of satiety (Liddle, 1997). While fibre has been thought to promote satiety and alter metabolic fuel partitioning in favour of fat oxidation due to its hormonal effects (Ludwig, 2000), it is unknown whether the type of fibre is differentially related to body weight. Salmeron *et al.* (1997a,b) found that diets with a high glycaemic load and low cereal fibre content were positively associated with risk of non-insulin-dependent diabetes mellitus among both adult males and females in the USA. Hyperinsulinaemia, a manifestation of insulin resistance, plays an important role in the development and progression of diabetes (Salmeron *et al.* 1997a,b) and is further associated with obesity. It is plausible that the chronically-high insulin demand that is induced by the high glycaemic load and low fibre may contribute to obesity.

Antioxidant role in insulin sensitivity

The antioxidants that are contained within whole grains may also contribute to insulin sensitivity by protecting against oxidative stress (Slavin *et al.* 1997). Most of the antioxidants in grains are contained in the bran and germ, which have a thick-walled cellular structure that inhibits extraction (Miller *et al.* 2000). Oxidative stress has been associated with reduced insulin-dependent glucose disposal and diabetic complications (Oberley, 1988). Mg and vitamin E in particular are thought to be involved in insulin metabolism (Frost *et al.* 1996; Slavin *et al.* 1997) that may prevent or mitigate hyperinsulinaemia (Fukagawa *et al.* 1990; Feskens *et al.* 1994; Marshall *et al.* 1997). In clinical trials Mg supplementation provided beneficial effects on insulin sensitivity among patients with non-insulin-dependent diabetes mellitus (Paolisso *et al.* 1989) and in normal subjects (Paolisso *et al.* 1992). Furthermore, in the Health Professionals Study (Salmeron *et al.* 1997a) and Nurses' Health Study (Colditz *et al.* 1992) an independent inverse association between Mg intake and risk of non-insulin-dependent diabetes mellitus was observed.

It is currently unknown whether bran or germ intakes are independently associated with body weight, as there are no current studies in this area. However, it is plausible that the bran component of whole grains may be involved in body-weight regulation due to its insulin-sensitizing effects. Researchers have reported that wheat bran consumption improves glucose tolerance (Anderson & Chen, 1979), and it was reported that long-term wheat bran administration improved glucose tolerance better than pectin administration (Brodribb & Humphreys, 1976). Further, in the Nurses' Health Study CHD analysis (Liu *et al.* 1999) bran itself was more strongly inversely associated with CHD than most whole grains. It is unknown whether these effects were mediated in part by influence of wheat bran on postprandial insulin levels.

Future research directions

Future prospective studies may address the question of whether whole grain intake is directly related to body weight and obesity, and whether the associations are primarily driven by fibre, some other dietary component of whole-grain foods, or some other related aspect of diet. Thus, it will be of interest

to ascertain the independent effects of bran, germ and the different types of fibre on body weight. Furthermore, the classification of whole-grain foods that was developed by Jacobs *et al.* (1998) was a semi-quantitative estimate based on the number of servings of whole grains consumed per d. In this classification cereals that contained at least 25% by weight whole grain content were considered as whole-grain foods. Future studies may employ quantitative estimates of whole grain intake that adhere to the recent US Food and Drug Administration (1994) guidelines that restrict the whole grain label to foods with $\geq 51\%$ by weight whole grain ingredient per reference amount customarily consumed. The potential application of these studies is high, given the rising prevalence of obesity in the USA, and the opportunity for increased intake of whole grains and dietary fibre. It has been estimated that the average whole grain intake in the USA is less than one serving per d (Cleveland *et al.* 2000). The low whole grain intake has been attributed to lack of consumer awareness of the benefits and sources of whole-grain foods (Adams & Engstrom, 2000). It has been further estimated that Americans only consume an average of 14–15 g fibre/d (Alaimo *et al.* 1994), far short of the recommended daily intake of 20–35 g.

References

- Adams JF & Engstrom A (2000) Helping consumers achieve recommended intakes of whole grain foods. *Journal of the American College of Nutrition* **19**, Suppl. 3, 339S–344S.
- Adlercreutz H (1990) Western diet and western diseases; some hormonal and biochemical mechanisms and associations. *Scandinavian Journal of Clinical and Laboratory Investigation* **201**, Suppl., 3–23.
- Alaimo K, McDowell M, Briefel R, Bischof A, Caughman C & Johnson L (1994) *Dietary Intake of Vitamins, Minerals, and Fiber of Persons Aged 2 Months and Over in the United States: Third National Health and Nutrition Examination Survey, Phase 1, 1988–91. Advance Data from Vital and Health Statistics*: no. 258. Hyattsville, MD: National Center for Health Statistics.
- Alfieri M, Pomerleau J, Grace DM, *et al.* (1995) Fiber intake of normal weight, moderately obese and severely obese subjects. *Obesity Research* **3**, 541–547.
- Anderson J & Hanna T (1999) Whole grains and protection against coronary heart disease: what are the active components and mechanisms? *American Journal of Clinical Nutrition* **70**, 307–308.
- Anderson JW & Chen WJ (1979) Plant fiber: carbohydrate in lipid metabolism. *American Journal of Clinical Nutrition* **32**, 346–363.
- Anderson JW, Hanna TJ, Peng X & Kryscio RJ (2000) Whole grain foods and heart disease risk. *Journal of the American College of Nutrition* **19**, Suppl. 3, 291S–299S.
- Behall KM, Scholfield DJ, Yuhaniak I & Canary J (1988) Effect of starch structure on glucose and insulin responses in adults. *American Journal of Clinical Nutrition* **47**, 428–432.
- Bourdon I, Yokoyama W, Davis P, Hudson C, Backus R, Richter D, Knuckles B & Schneeman BO (1999) Postprandial lipid, glucose, insulin and cholecystokinin responses in men fed barley pasta enriched with beta-glucan. *American Journal of Clinical Nutrition* **69**, 55–63.
- Brodribb AJ & Humphreys DM (1976) Diverticular disease: three studies. *British Medical Journal* **1**, 424–426.
- Burkitt DP (1952) Acute abdomens. British and Bagand compared. *East African Medical Journal* **19**, 189–192.
- Chen W-J & Anderson JW (1986) Hypocholesterolemic effects of soluble fibers. In *Dietary Fiber: Basic and Clinical Aspects*, pp 275–286 [GV Vahouny and D Kritchevsky, editors]. New York: Plenum.
- Cleave TL (1956) The neglect of the natural principles in current medical practice. *Journal of the Royal Naval Medical Service* **42**, 55–60.
- Cleveland LE, Moshfegh AJ, Albertson AM & Goldman JD (2000) Dietary intake of whole grains. *Journal of the American College of Nutrition* **19**, Suppl. 3, 331S–338S.
- Colditz GA, Manson JE, Stampfer MJ, Rosner B, Willett WC & Speizer FE (1992) Diet and risk of clinical diabetes in women. *American Journal of Clinical Nutrition* **55**, 1018–1023.
- Expert Panel on the Identification, Evaluation and Treatment of Overweight in Adults (1998) Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: Executive summary. *American Journal of Clinical Nutrition* **68**, 899–917.
- Feskens EJ, Loeber JG & Kromhout D (1994) Diet and physical activity as determinants of hyperinsulinemia: the Zutphen Elderly Study. *American Journal of Epidemiology* **140**, 350–360.
- Folsom AR, Vitelli LL, Lewis CE, Schreiner PJ, Watson RL & Wagenknecht LE (1998) Is fasting insulin concentration inversely associated with rate of weight gain? Contrasting findings from the CARDIA and ARIC study cohorts. *International Journal of Obesity and Related Metabolic Disorders* **22**, 48–54.
- Freedman D, Williamson D, Croft J, Ballew C & Byers T (1995) Relation of body fat distribution to ischemic heart disease. The National Health and Nutrition Examination Survey (NHANES I). *American Journal of Epidemiology* **142**, 53–63.
- Frost G, Keogh B, Smith D, Akinsanya K & Leeds A (1996) The effect of low-glycemic carbohydrate on insulin and glucose response in vivo and in vitro in patients with coronary heart disease. *Metabolism* **45**, 669–672.
- 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. *American Journal of Clinical Nutrition* **52**, 524–528.
- Fung TT, Rimm EB, Spiegelman D, Rifai N, Tofler GH, Willett WC & Hu FB (2001) Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk. *American Journal of Clinical Nutrition* **73**, 1–2.
- Hallfrisch J & Behall KM (2000) Mechanisms of the effects of grains on insulin and glucose responses. *Journal of the American College of Nutrition* **19**, Suppl. 3, 320S–325S.
- Holt S, Brand J, Soveny C & Hansky J (1992) Relationship of satiety to postprandial glycemic, insulin and cholecystokinin responses. *Appetite* **18**, 129–141.
- Howarth N, Saltzman E & Roberts S (2001) Dietary fiber and weight regulation. *Nutrition Reviews* **59**, 129–139.
- Jacobs DR Jr, 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. *American Journal of Clinical Nutrition* **68**, 248–257.
- 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. *Arteriosclerosis, Thrombosis and Vascular Biology* **21**, 2065–2071.
- Jenkins D, Wesson V, Wolever TM, Jenkins AL, Kalmusky J, Guidici S, Csima A, Josse RG & Wong GS (1988a) Whole-meal versus wholegrain breads: proportion of whole or cracked grain and the glycaemic response. *British Medical Journal* **297**, 958–960.
- Jenkins DJ, Jenkins AL, Wolever TMS, Vuksan V, Brighenti F, Cunnane S, Rao AV, Thompson LV & Josse RG (1988b) Lente

- carbohydrate or slowly absorbed starch: physiological and therapeutic implications. In *Dietary Fiber: Chemistry, Physiology and Health Effects*, pp. 247–259 [D Kritchevsky, C Bonfield and J Anderson, editors]. New York: Plenum Press.
- 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. *British Medical Journal* **1**, 1392–1394.
- Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM, Bowling AC, Newman HC, Jenkins AL & Goff DV (1981) Glycemic index of foods: a physiological basis for carbohydrate exchange. *American Journal of Clinical Nutrition* **34**, 362–366.
- Lapidus L, Bengtsson C, Larson B, Pennert K, Rybo E & Sjoström L (1984) Distribution of adipose tissue and risk of cardiovascular disease: a 12 year follow up of participants in the population study of women in Gothenburg, Sweden. *British Medical Journal* **289**, 1261–1263.
- Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Björntorp P & Tibblin G (1984) Abdominal adipose tissue distribution, obesity and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *British Medical Journal* **288**, 1401–1404.
- Liddle RA (1997) Cholecystokinin cells. *Annual Reviews of Physiology* **59**, 221–242.
- Liu S, Manson J, Stampfer M, Rexrode K, Hu F, Rimm E & Willett W (2000) Whole grain consumption and risk of ischemic stroke in women. A prospective study. *Journal of the American Medical Association* **284**, 1534–1540.
- Liu S, Stampfer M, Hu FB, Giovannucci E, Rimm E, Manson J, Hennekens C & Willett W (1999) Whole-grain consumption and risk of coronary heart disease: results from the Nurses' Health Study. *American Journal of Clinical Nutrition* **70**, 412–419.
- Ludwig D, Majzoub J, Al-Zahrani A, Dallal G, Blanco I & Roberts S (1999a) High glycemic index foods, overeating and obesity. *Pediatrics* **103**, 1–6.
- Ludwig DS (2000) Dietary glycemic index and obesity. *Journal of Nutrition* **130**, Suppl., 280–283.
- Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, Van Horn L, Slattery ML & Jacobs DR Jr (1999b) Dietary fiber, weight gain, and cardiovascular disease risk factors in young adults. *Journal of the American Medical Association* **282**, 1539–1546.
- Lundgren H, Bengtsson C, Blohme G, Lapidus L & Sjoström L (1989) Adiposity and adipose tissue distribution in relation to incidence of diabetes in women: results from a prospective population study in Gothenburg, Sweden. *International Journal of Obesity* **13**, 413–423.
- 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. *American Journal of Clinical Nutrition* **76**, 390–398.
- Marshall JA, Bessesen DH & Hamman RF (1997) High saturated fat and low starch and fibre are associated with hyperinsulinaemia in a non-diabetic population: the San Luis Valley Diabetes Study. *Diabetologia* **40**, 430–438.
- Miller HE, Rigelhof F, Marquart L, Prakash A & Kanter M (2000) Antioxidant content of whole grain breakfast cereals, fruits and vegetables. *Journal of the American College of Nutrition* **19**, Suppl. 3, 312S–319S.
- Miller WC, Niederpruem MG, Wallace JP & Lindeman AK (1994) Dietary fat, sugar, and fiber predict body fat content. *Journal of the American Dietetic Association* **94**, 612–615.
- Nelson LH & Tucker LA (1996) Diet composition related to body fat in a multivariate study of 293 men. *Journal of the American Dietetic Association* **96**, 771–777.
- Oberley LW (1988) Free radicals and diabetes. *Free Radical Biology and Medicine* **5**, 113–124.
- Odeleye O, de Courten M, Pettitt D & Ravussin E (1997) Fasting hyperinsulinemia is a predictor of increased body weight gain and obesity in Pima Indian children. *Diabetes* **46**, 1341–1345.
- Paolisso G, Sgambato S, Gambardella A, Pizza G, Tesaro P, Varricchio M & D'Onofrio F (1992) Daily magnesium supplements improve glucose handling in elderly subjects. *American Journal of Clinical Nutrition* **55**, 1161–1167.
- Paolisso G, Sgambato S, Pizza G, Passariello N, Varricchio M & D'Onofrio F (1989) Improved insulin response and action by chronic magnesium administration in aged NIDDM subjects. *Diabetes Care* **12**, 265–269.
- Pereira A, Jacobs D, Slattery M, Ruth K, Van Horn L, Hilner J & Kushi L (1998) The association of whole grain intake and fasting insulin in a biracial cohort of young adults: the CARDIA study. *CVD Prevention* **1**, 231–242.
- Pereira MA & Ludwig DS (2001) Dietary fiber and body-weight regulation. *Pediatric Clinics of North America* **48**, Suppl. 4, 1–9.
- Raben L, Christensen NJ, Madsen J, Holst JJ & Astrup A (1994) Decreased postprandial thermogenesis and fat oxidation but increased fullness after a high-fiber meal compared with a low-fiber meal. *American Journal of Clinical Nutrition* **59**, 1386–1394.
- Rimm EB, Ascherio A, Giovannucci E, Spiegelman D, Stampfer M & Willett W (1996) Vegetable, fruit and cereal fiber intake and risk of coronary heart disease risk among men. *Journal of the American Medical Association* **275**, 447–451.
- Roberts SB (2000) High-glycemic index foods, hunger and obesity: is there a connection? *Nutrition Reviews* **58**, 163–70.
- Salmeron J, Ascherio A, Rimm E, Colditz G, Spiegelman D, Jenkins D, Stampfer M, Wing A & Willett W (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 among women. *Journal of the American Medical Association* **277**, 472–477.
- Slavin J (1994) Whole grains and health: separating the wheat from the chaff. *Nutrition Today* **29**, 6–10.
- Slavin J, Jacobs D & Marquart L (1997) Whole-grain consumption and chronic disease: protective mechanisms. *Nutrition in Cancer* **27**, 14–21.
- Southgate DAT (1995) The structure of dietary fibre. In *Dietary Fiber in Health and Disease* pp. 25–36 [D Kritchevsky and C Bonfield, editors]. St Paul, MN: Eagan Press.
- Trout DL, Behall KM & Osilesi O (1993) Prediction of glycemic index for starchy foods. *American Journal of Clinical Nutrition* **58**, 873–878.
- Trowell H (1972) Ischemic heart disease and dietary fiber. *American Journal of Clinical Nutrition* **25**, 926–932.
- US Food and Drug Administration (1994) *Center for Food Safety and Applied Nutrition: A Food Labeling Guide*. Docket 99P–2209. Health Claim Notification for Wholegrain Foods. <http://www.cfsan.fda.gov/label.html/>
- Walker ARP (1947) The effects of recent changes of food habits and bowel motility. *South African Medical Journal* **21**, 590–592.
- Willett WC (1998) The dietary pyramid: does the foundation need repair? *American Journal of Clinical Nutrition* **68**, 218–219.
- Wolever TM & Jenkins DJ (1986) The use of the glycemic index in predicting the blood glucose response to mixed meals. *American Journal of Clinical Nutrition* **43**, 167–172.