



Physiological effects of cereal dietary fibre

Nils-Georg Asp, Inger Björck & Margareta Nyman

Department of Applied Nutrition and Food Chemistry, Chemical Center, University of Lund, PO Box 124, S-221 00 Lund, Sweden

Cereal dietary fibre constitutes about half of the total fibre intake in Sweden. The amount of fibre available is 21 g/person/day or 1.75 g/MJ. Increased faecal bulk and shortening of intestinal transit time are well-documented effects, that are inversely proportional to the fermentability of the dietary fibre. Wheat bran, oat husks, and corn bran are fibre sources with high bulking effect, whereas fibre from the endosperm of cereals is more extensively fermented. Resistant starch in processed cereals has properties similar to dietary fibre, and must be considered in analysis as well as regarding physiological effects of dietary fibre. Oat groats and oat bran have serum cholesterol lowering effects related to the viscous soluble β -glucans. Rye and barley contain similar levels of viscous soluble fibre but are not as thoroughly investigated. Effects on postprandial glucose and insulin levels are also partly related to viscous soluble dietary fibre, but structural properties in, e.g., whole kernels and pasta products are more important in this respect. Phytate in whole grain flour and bran preparations inhibits iron and zinc absorption in single meal tests. Its role for mineral status in persons consuming a mixed, Western diet remains to be established.

INTRODUCTION

Cereals form the quantitatively most important source of dietary fibre in many countries, constituting about half of the 21 g of fibre available per person per day in Sweden. This corresponds to 1.75 g/MJ. The Nordic recommendation is 3 g/MJ. For a long time cereal fibre was thought of mainly as wheat bran, which is the most thoroughly investigated source of fibre. The dietary fibre of wheat bran is characterized by a low solubility in water and a high lignin content. Oat husks and corn bran are other sources of insoluble, lignified cereal fibre, in which xylan, arabinoxylan and cellulose are the main polysaccharides (Schimberni *et al.*, 1982; Nyman, 1985; Nyman & Asp, 1988).

The great interest in oats as a source of dietary fibre stems mainly from its high content of soluble, viscous polysaccharides, mainly β -glucans. A unique property of oats is that these β -glucans are distributed roughly proportional to the insoluble fibre in various parts of the oat grain. Therefore, the soluble fibre constitutes about 40% of the total fibre in both oat flour and oat bran (Frölich & Nyman, 1988). Barley and rye have a similar content of viscous, soluble polysaccharides, but a more even distribution in different parts of the grain

(Nyman *et al.*, 1984). Therefore, brans from these cereals have comparatively lower soluble fibre content.

DELIMITATION OF DIETARY FIBRE

The definition of dietary fibre as polysaccharides and lignin that are not digested by endogeneous enzymes in the human intestinal tract (Trowell *et al.*, 1976) is widely accepted. This includes resistant starch, i.e. starch not digested in the small intestine. Another point of view, however, is to include only non-starch polysaccharides. NSP (Cummings & Englyst, 1991). For labelling and legislation purposes, it must be decided whether to include or exclude resistant starch and lignin in the dietary fibre. Obviously, these undigestible components are nutritionally more similar to NSP than to digestible carbohydrates.

From the scientific point of view, however, no strict definition is needed (Asp *et al.*, 1992b). Resistant starch fractions and lignin, as well as other undigestible material, should be measured and characterized as far as possible, in addition to NSP, in any preparation used to investigate physiological effects.

All methods for dietary fibre or NSP analysis

employ precipitation in 78–80% aqueous ethanol (or dialysis) to separate the dietary fibre polysaccharides from low-molecular weight material (Asp *et al.*, 1983; Englyst & Cummings, 1984; Theander & Westerlund, 1986; Asp *et al.*, 1992b). Cereals contain 1–4% undigestible fructans (Aspinall, 1970; Nilsson *et al.*, 1986) that add to the fermentable material. These are not determined as dietary fibre or NSP. Chemically modified food starches (Östergård *et al.*, 1988) or starches subjected to dry heating (Siljeström *et al.*, 1989) are degraded by amylases to partly undigestible fragments that are soluble in 80% ethanol. The delimitation between dietary fibre/NSP and oligosaccharides by alcohol precipitation is arbitrary and has no immediate nutritional justification.

FERMENTATION AND FAECAL BULKING

The faecal bulking capacity of dietary fibre is inversely proportional to its fermentation in the large bowel. A rat model has been developed and evaluated at our department, to predict fermentability and bulking capacity of different types of dietary fibre in man and to investigate the effect of processing on these properties. A 4–6-day adaptation period is used, followed by a 5-day balance period with collection of feed residues and faeces (Nyman & Asp, 1985). Comparison with human experiments has shown a good agreement regarding both faecal dry weight increment and fermentation of neutral sugar constituents when feeding wheat bran, apple, cabbage and guar gum (Nyman *et al.*, 1986), and recently also with variously processed carrots (Wisker *et al.*, 1990; Nyman *et al.* 1991). It should be noted,

however, that human faeces has a much higher water content (about 75%) than rat faeces (about 30%).

Chemical structure, solubility and lignification are some properties of dietary fibre of great importance for the fermentability. For example, oat husks with a high content of cellulose, xylan and lignin, were almost completely resistant to fermentation (Nyman & Asp, 1988). It gave a prominent faecal dry weight increment, almost exclusively due to remaining dietary fibre. Oat bran fibre with a high β -glucan content, on the other hand, was rather extensively fermented. Between 19 and 38% remained in the faeces, and accounted for only half of the dry weight increment (Nyman & Asp, 1988).

Table 1 shows percent dietary fibre remaining in the faeces of rats and faecal dry weight increment when feeding a number of different sources of cereal dietary fibre. Dietary fibre from the wheat endosperm was as easily fermented as purified pectin and much more available to the microbial flora than the fibre in whole grain flour. This is noteworthy in relation to the fact that the monomeric composition of the dietary fibre polysaccharides is similar in the endosperm and outer layers. One important difference, however, is the lignification of the outer layers. The higher proportion of soluble fibre in the endosperm is also likely to be of importance for the fermentability (Nyman *et al.*, 1985).

Table 1 also shows that two processes — extrusion cooking and puffing — increased the fermentability of wheat fibre (Björck *et al.*, 1984; Nyman *et al.*, 1987). An increased water solubility of the fibre was seen. However, these processes also caused depolymerization of starch (Siljeström *et al.*, 1986). A similar depolymerization of dietary fibre polysaccharides probably also occurred and might have altered other physiological properties of the fibre (see below).

Table 1. Faecal recovery of dietary fibre and faecal dry weight increment in rats after intake of different sources of cereal fibre^a

	Faecal recovery of dietary fibre (percent)	Faecal dry weight increment (g/g fibre in feed)
Refined wheat flour	17	0.6
Whole grain wheat flour	62	1.1
Wheat bran	58	0.8
Refined rye flour	36	0.8
Whole grain rye flour	56	1.0
Refined barley flour	16	0.5
Whole grain barley flour	69	1.0
Unprocessed wheat flour	24	— ^b
Extruded wheat flour	12	— ^b
Unprocessed whole grain wheat	74	1.1
Puffed whole grain wheat	58	1.0
Pectin	19	0.5

^aData compiled from Björck *et al.* (1984), Nyman (1985) and Nyman *et al.* (1985, 1986, 1987).

^bNot analysed.

Other processes such as sour dough baking, conventional baking, autoclaving, flaking and drum-drying seemed to have only minor effects on the fermentability of dietary fibre as judged from similar dry matter digestibilities (Håkansson *et al.*, 1987; Siljeström *et al.*, 1988).

The products formed during colonic fermentation, i.e. acetate, propionate and butyrate, contribute to the bulking capacity, but also have potential effects on lipid and carbohydrate metabolism (Jenkins *et al.*, 1987a,b). A recent study in rats showed lower total short-chain fatty acid content in the caecum in animals fed oat bran than with corresponding amounts of pectin and guar gum, despite an almost complete fermentation of all materials (Berggren *et al.*, 1992).

BLOOD CHOLESTEROL LOWERING EFFECT

It is well documented that viscous soluble types of dietary fibre have a blood cholesterol lowering effect. The low density lipoprotein cholesterol (LDL) is lowered, whereas the high density lipoprotein cholesterol (HDL) as well as fasting triglyceride levels are not altered (Truswell & Beynen, 1992). The originally postulated mechanism behind this effect, i.e. sequestration of bile acids in the small intestine, has recently been substantiated by direct experiments in humans with ileostomy (Andersson, 1992). Dietary fibre with cholesterol lowering effects increases the amount of bile acids and/or cholesterol in the ileostomy effluent.

Wheat bran increases HDL-cholesterol in the rat (Asp *et al.*, 1981). Although a few small studies indicate a similar effect in man, the overall conclusion that dietary fibre practically does not alter HDL-cholesterol levels seems valid (Truswell & Beynen, 1992).

VARIATION IN DIETARY FIBRE AND β -GLUCAN CONTENT OF OATS

The dietary fibre and β -glucan content of 150 oat samples grown in Sweden during 1986–1989 were recently reported (Asp *et al.*, 1992a). The average dietary fibre content was 9.7 g/100 g dry matter with a range of 5.0–13.4. On an average, 36% was soluble as measured with the enzymatic, gravimetric method of Asp *et al.* (1983). The β -glucan content was 4.6 (3.5–6.7) g/100 g dry matter. The β -glucan content correlated with the total as well as the insoluble fibre content, but surprisingly not with amount of soluble fibre.

The dietary fibre and β -glucan content of oat bran also varies considerably depending on both the raw material and the milling technique. The American Association of Cereal Chemists (AACC) recently defined oat bran as containing at least 17 g dietary fibre/100 g (AOAC method) and being processed with

a yield not exceeding 50% of the oat groats used as raw material. Obviously, this is just slightly higher than the fibre content in the most fibre-rich variety in our study. Thus oat bran should be regarded as a fibre/ β -glucan enriched oat flour rather than a fibre concentrate or isolate.

Although one negative study (Swain *et al.*, 1990) attracted much attention, there is a substantial overall documentation that oat flour and oat bran has a significant, although in most studies modest, blood cholesterol lowering effect (Truswell & Beynen, 1992). Optimal effect amounting to about 5% reduction in serum cholesterol seems to be obtained with 56 g oat bran or 84 g oat flour/day, corresponding to about 10 g total oat fibre or 4 g oat β -glucan.

Animal experiments have shown that the blood cholesterol lowering effect of oat bran is related to the β -glucan content and abolished by depolymerization of the β -glucans by β -glucanase treatment (Tietzen *et al.*, 1990). Thus, minor components such as saponins and tocotrienol seem to be of less importance, but more studies are needed to document their possible effects in man.

Oat bran has recently been shown to lower blood cholesterol also in subjects with ileostomy, who have practically no fermentation of fibre (Zhang *et al.*, 1992). Thus, inhibition of cholesterol synthesis by propionate does not seem to be a major mechanism in man, although some additional effect cannot be excluded.

POSTPRANDIAL GLUCOSE AND HORMONAL RESPONSES

The glycaemic response has for a long time been regarded as important in the dietary management of diabetes, but there is now an increasing interest in foods eliciting low glycaemic responses also in relation to satiety, blood pressure, blood cholesterol and physical performance (Haber *et al.*, 1977; Ducimetiere *et al.*, 1980; Jenkins *et al.*, 1987a,b; Brand *et al.*, 1990, 1991; Sherman *et al.*, 1991; Thomas *et al.*, 1991). Possibly, it might also be related to ageing of tissues since the glycosylation of tissue proteins is influenced by extracellular glucose concentration (Cerami *et al.*, 1987).

One well-documented effect of dietary fibre is the lowering of postprandial blood glucose and insulin responses. This hypoglycaemic effect has been connected to the viscous character of certain fibre, is abolished by depolymerization and concomitant loss of viscosity (Jenkins *et al.*, 1978), and is similar for purified oat gum and guar gum (Braaten *et al.*, 1991). However, most studies on this topic have been conducted with added purified fibre, and little is known about the impact of naturally occurring levels of viscous fibre in common foods.

In cereal foods, several factors are capable of affecting the glycaemic response. Some are related to properties of the starch moiety as such (degree of gelatinization, amylose/amylopectin ratio, extent of retrogradation, starch-lipid interactions), whereas others are influenced by the presence of certain substances intrinsic to the food (antinutrients, organic acids, viscous dietary fibre) or to the type and extent of processing (Yoon *et al.*, 1983; Holm *et al.*, 1985; Ross *et al.*, 1987; Würsch, 1988; Todesco *et al.*, 1991). Although the presence of dietary fibre *per se* is probably somewhat less important than previously assumed, the preservation in food of an intact fibre matrix is increasingly being recognized as an important determinant of postprandial glycaemia.

The glycaemic response is often expressed as the glycaemic index (GI), i.e. the ratio of the area under the blood glucose response curve as a percentage of the area after a standard, either glucose solution or white wheat bread (Jenkins *et al.*, 1981). Cereal products are found over the whole range from low to high GI, with no obvious correlation to dietary fibre content (Jenkins *et al.*, 1983). Consequently, pasta products as a group have low GI, despite a generally low content of dietary fibre (Wolever *et al.*, 1986; Granfeldt & Björck, 1991; Granfeldt *et al.*, 1991), whereas most conventional bread products, including wholemeal breads, have among the highest GI's (Würsch, 1988). In contrast, pumpnickel bread with intact rye grains including boiled barley grains has been reported to produce comparatively low glycaemic responses (Jenkins *et al.*, 1986).

In a recent study, the potential of exchanging flour for pre-heated grains from different cereals was evaluated as a means of reducing postprandial glycaemia to bread (Liljeberg *et al.*, 1992). Approximately 20% of the starch was provided in the form of white wheat flour and 80% in the form of wheat, rye, oat and barley grains. Apart from the reference white bread, a barley bread made from milled grains was also included. The pumpnickel-type bread products from wheat, rye and barley produced significantly lower GI's in healthy subjects than the reference bread. The GI's with intact grains from rye and barley (GI 49–58) were in the lower range of that reported for pasta. The beneficial properties of these pumpnickel-type products are interpreted in terms of a lowered digestion rate of starch when present in a more or less intact fibre matrix. In contrast, the glucose responses to the barley meal bread and the bread with pre-heated oat grains were not significantly different from those with white bread. The lack of effect in the case of oat grain bread was probably due to the integrity of the oat grains having been damaged during pre-heating and subsequent baking.

These results highlight the importance of maintaining the botanical structure as intact as possible, and

suggest that the dietary fibre in oats and barley was not effective in reducing glycaemia at the level tested (2 and 3 g soluble fibre/50 g starch, respectively). However, enrichment of bread with oat bran (corresponding to 4 g soluble fibre/50 g starch) significantly reduced both glucose (GI 72) and insulin responses in healthy subjects (Holm & Björck, 1992).

INHIBITION OF MINERAL ABSORPTION

The inhibiting effect on mineral absorption of fibre-rich foods is related to their phytate content as well as to tannins and possibly other minor components. In spite of a mineral binding capacity *in vitro* of fibre polysaccharides rich in uronic acid residues, there is no evidence that dietary fibre as such inhibits mineral absorption *in vivo* (Rossander *et al.*, 1992).

When evaluating the nutritional importance of phytic acid, it should be noted that phytate-rich outer layers of cereal grains also contain high levels of minerals. A decreased percent absorption, therefore, does not necessarily mean a lower total amount of minerals absorbed. Thus, whole grain cereals have been considered a good source of zinc in spite of the phytate content (Sandström *et al.*, 1980).

The present knowledge regarding the inhibitory effect of phytate on mineral absorption is based on single meal studies with radioactive isotopes. Recently, however, one semi-long-term study was published (Cook *et al.*, 1991) supporting that inhibitors of iron absorption decreased the retention of labelled iron administered during a four-week period. Further long-term studies are needed to evaluate the importance of phytate in mixed diets.

Based on the present knowledge, the development of processes decreasing the phytate content without removing minerals seems desirable. It cannot be excluded that high intake of phytate-rich foods, e.g. oat bran, might impair the mineral status, especially if the products are processed with inactivation of phytase.

CONCLUSIONS

Cereals are quantitatively the most important source of dietary fibre in most diets. Physiological effects of cereal dietary fibre polysaccharides, such as faecal bulking, lowering of blood cholesterol, and decrease in postprandial glucose and hormone responses are well documented. However, other properties of foods and diets may modify or simulate fibre effects. This is especially evident regarding structural effects on postprandial glucose and hormonal response.

REFERENCES

- Andersson, H. (1992). In *Resistant Starch*, ed. N.-G. Asp, *Eur. J. Clin. Nutr.*, **46**, (Suppl. 2), S69–S76.

- Asp, N.-G., Bauer, H., Nilsson-Ehle, P., Nyman, M. & Öste, R. (1981). *Br. J. Nutr.*, **46**, 385-93.
- Asp, N.-G., Johansson, C.G., Hallmer, H. & Siljeström, M. (1983). *J. Agric. Food Chem.*, **31**, 476-82.
- Asp, N.-G., Mattsson, B. & Önning, G. (1992a). *Europ. J. Clin. Nutr.*, **46**, 31-7.
- Asp, N.-G., Schweizer, T.F., Southgate, D.A.T. & Theander, O. (1992b). In *Dietary Fibre — a Component of Food*, ed. T.F. Schweizer & C.A. Edwards. Springer-Verlag, London, pp. 57-101.
- Aspinall, G.O. (1970). *Polysaccharides*, ed. G.O. Aspinall, Pergamon Press, Oxford, pp. 69-84.
- Berggren, A., Björck, I., Nyman, M. & Eggum, B.O. (1992). In *Topics in dietary fibre research*, Abstract, p. 62. Symposium within the COST 92 programme, Rome/Viterbo, 5-7 May 1992.
- Björck, I., Nyman, M. & Asp, N.-G. (1984). *Cereal Chem.*, **61**, 174-9.
- Braaten, J.T., Wood, P.J., Scott, F.W., Riedel, K.D., Poste, L.M. & Collins, M.W. (1991). *Am. J. Clin. Nutr.*, **53**, 1425-30.
- Brand, J.C., Holt, S., Soveny, C. & Hansky, J. (1990). *Proc. Nutr. Soc. Aust.*, **15**, 209 (abstract).
- Brand, J.C., Colagiuri, S., Crossman, S., Allen, A. & Truswell, A.S. (1991). *Diabetes Care*, **14**, 95-101.
- Cerami, A., Vlassana, H. & Brownlee, M. (1987). *Scientific American*, May, 90-6.
- Cook, J.D., Dassenko, S.A. & Lynch, S.R. (1991). *Am. J. Clin. Nutr.*, **54**, 712-22.
- Cummings, J.H. & Englyst, H.N. (1991). *Trends in Food Science & Technology*, April, 99-103.
- Ducimetiere, P., Eschwege, E., Papoz, L., Rickard, J.L., Claude, J.R. & Rosselin, G. (1980). *Diabetologia*, **19**, 205-10.
- Englyst, H.N. & Cummings, J.H. (1984). *Analyst*, **109**, 937-42.
- Frölich, W. & Nyman, M. (1988). *J. Cereal Sci.*, **7**, 73-82.
- Granfeldt, Y. & Björck, I. (1991). *J. Cereal Sci.*, **14**, 47-61.
- Granfeldt, Y., Hagander, B. & Björck, I. (1991). *Eur. J. Clin. Nutr.*, **45**, 489-99.
- Haber, G.B., Heaton, K.W., Murphy, D. & Burroughs, L.F. (1977). *Lancet*, October, 679-82.
- Håkansson, B., Jägerstad, M., Öste, R., Åkesson, B. & Jonsson, L. (1987). *J. Cereal Sci.*, **6**, 269-82.
- Holm, J. & Björck, I. (1992). *Am. J. Clin. Nutr.*, **55**, 420-9.
- Holm, J., Björck, I., Asp, N.-G. & Lundquist, I. (1985). *J. Cereal Sci.*, **3**, 193-206.
- Jenkins, D.J.A., Wolever, T.M.S., Leeds, A.R. *et al.* (1978). *Br. Med. J.*, **1**, 1392-4.
- Jenkins, D.J.A., Wolever, T.M.S., Taylor, R.H. *et al.* (1981). *Am. J. Clin. Nutr.*, **34**, 362-6.
- Jenkins, D.J.A., Wolever, T.M.S., Jenkins, A.L. *et al.* (1983). *Diabetes Care*, **6**, 155-9.
- Jenkins, D.J.A., Wolever, T.M.S., Jenkins, A.L. *et al.* (1986). *Am. J. Clin. Nutr.*, **43**, 516-20.
- Jenkins, D.J.A., Jenkins, A.L., Wolever, T.M.S., Collier, G.R., Venket Rao, A. & Thompson, L.U. (1987a). *Scand. J. Gastr.*, **22**, Suppl. 128, 132-41.
- Jenkins, D.J.A., Wolever, T.M.S. & Kalmusky, J. (1987b). *Am. J. Clin. Nutr.*, **46**, 66-71.
- Liljeberg, H., Granfeldt, Y. & Björck, I. (1992). *Eur. J. Clin. Nutr.*, **46**, 561-75.
- Nilsson, U., Nilsson, B. & Dahlqvist, A. (1986). *Food Chem.*, **22**, 95-106.
- Nyman, M. (1985). Fermentation of dietary fibre in the intestinal tract. Thesis, Dept of Food Chemistry, University of Lund, Sweden, p. 27.
- Nyman, M. & Asp, N.-G. (1985). *Br. J. Nutr.*, **54**, 635-43.
- Nyman, M. & Asp, N.-G. (1988). *Am. J. Clin. Nutr.*, **48**, 274-9.
- Nyman, M., Asp, N.-G., Pedersen, B., Bach-Knudsen, K.E. & Eggum, B.O. (1984). *Cereal Chem.*, **61**, 14-19.
- Nyman, M., Asp, N.-G., Pedersen, B. & Eggum, B.O. (1985). *J. Cereal Sci.*, **3**, 207-19.
- Nyman, M., Asp, N.-G., Cummings, J.H. & Wiggins, H. (1986). *Br. J. Nutr.*, **55**, 487-96.
- Nyman, M., Björck, I., Håkansson, B. & Asp, N.-G. (1987). *J. Cereal Sci.*, **5**, 67-72.
- Östergård, K., Björck, I. & Gunnarson, A. (1988). *Stärke*, **40**, 58-66.
- Ross, S.W., Brand, J.C., Thorburn, A.W. & Truswell, A.S. (1987). *Am. J. Clin. Nutr.*, **46**, 631-5.
- Rossander, L., Sandberg, A.-S. & Sandström, B. (1992). In *Dietary Fibre — a Component of Food*, ed. T.F. Schweizer & C.A. Edwards. Springer-Verlag, London, pp. 176-216.
- Sandström, B., Arvidsson, B., Cederblad, A. & Björn-Rasmussen, E. (1980). *Am. J. Clin. Nutr.*, **33**, 739-45.
- Schimberni, M., Cardinale, F., Sodimi, G. & Canella, M. (1982). *Lebensm.-Wiss. u. Techn.*, **15**, 337-9.
- Sherman, W.M., Peden, M.C. & Wright, D.A. (1991). *Am. J. Clin. Nutr.*, **54**, 866-70.
- Siljeström, M., Westerlund, E., Björck, I., Holm, J., Asp, N.-G. & Theander, O. (1986). *J. Cereal Sci.*, **4**, 315-23.
- Siljeström, M., Björck, I., Eliasson, A.C., Lönner, C., Nyman, M. & Asp, N.-G. (1988). *Cereal Chem.*, **65**, 1-8.
- Siljeström, M., Björck, I. & Westerlund, E. (1989). *Stärke*, **41**, 95-100.
- Swain, J.F., Rouse, I.L., Curley, C.B. & Sacks, F.M. (1990). *New England J. Med.*, **322**, 147-52.
- Theander, O. & Westerlund, E.A. (1986). *J. Agric. Food Chem.*, **34**, 330-6.
- Thomas, D.E., Brotherhood, J.R. & Brand, J.C. (1991). *Int. J. Sports Med.*, **12**, 180-6.
- Tietjen, J.L., Nevins, D.J. & Schneeman, B.O. (1990). *FASEB Journal*, **4**, A527.
- Todesco, T., Rao, A.V., Bosello, O. & Jenkins, D.J.A. (1991). *Am. J. Clin. Nutr.*, **54**, 860-5.
- Trowell, H., Southgate, D.A.T., Wolever, T.M.S., Leeds, A.R.L., Gasull, M.A. & Jenkins, D.J.A. (1976). *Lancet*, **i**, 967.
- Truswell, A.S. & Beynen, A.C. (1992). In *Dietary Fibre — a Component of Food*, ed. C.A. Edwards & T.F. Schweizer. Springer-Verlag, London, 295-332.
- Wisker, E., Feldheim, W. & Schweizer, T.F. (1990). In *Dietary Fibre: Chemical and Biological Aspects*, ed. D.A.T. Southgate, K. Waldron, I.T. Johnson & G.R. Fenwick. The Royal Society of Chemistry, Cambridge, pp. 318-20.
- Wolever, T.M.S., Jenkins, D.J.A. & Kalmursky, J. *et al.* (1986). *Diabetes Care*, **9**, 401-4.
- Würsch, P. (1988). *Wld. Rev. Nutr. Diet.*, **67**, 1-46.
- Yoon, J.H., Thompson, L.U. & Jenkins, D.J.A. (1983). *Am. J. Clin. Nutr.*, **38**, 835-42.
- Zhang, J.-X., Hallmans, G., Andersson, H., Bosaeus, I., Åman, P., Tidehag, P., Stenling, R., Lundén, E. & Dahlgren, S. (1992). *Am. J. Clin. Nutr.*, **56**, 99-105.