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Review Articles

The Diabetic Diet, Dietary Carbohydrate and Differences in Digestibility

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Over the past three years there has been a move in the West to increase the carbohydrate content of the diabetic diet. In 1979 the Food and Nutrition Committee of the American Diabetes Association published dietary recommendations [1] advocating that the carbohydrate intakes of Type 1 (insulin-dependent) diabetic patients be increased to 50%–60% of total calories. This was followed in 1980 by advice from the Special Report Committee of the Canadian Diabetes Association that the diets of all diabetic patients should be 45% carbohydrate or more, with no upper limit placed [2]. Most recently, in 1981, the Nutrition Sub-committee of the British Diabetic Association took a similar stand, suggesting that half or more of the energy content of the diet should be derived from carbohydrate [3]. The overall aim was to reduce fat consumption in the hope that in the longterm this would reduce the risk of cardiovascular disease. In addition, it was recommended that simple sugar intake be restricted and that where possible carbohydrate should be taken in the form of fibre-rich unprocessed foods.

However, after the American Diabetes Association report, concern was expressed that official liberalisation of a previous carbohydrate restriction might be used as a licence to consume those carbohydrate foods which would compromise good diabetic control [4, 5]. This matter has been well debated [6–8]. One outcome has been to re-emphasise the possible value of fibre in improving diabetic control at a given carbohydrate intake, and another has been to highlight the inadequacy of our knowledge of possible differences in physiological responses to different foods. If the carbohydrate and fibre intakes of diabetic patients are to be increased, which foods are to be used? Should they be new foods, or more of what the diabetic is already eating?

The advice and the dilemma are succintly summed up in the British Diabetic Association recommendations [3]: "A general increase in the fibre content of the diabetic diet is a useful therapeutic measure [6971]. More effective normalisation of post-prandial glycaemia may be achieved by a more selective approach to dietary carbohydrate and by consideration of the glycaemic effects of foods and meals in their entirety rather than on the basis of sucrose or total carbohydrate alone. More research is urgently needed to unravel this complicated but fundamental aspect of dietary advice." It is therefore proposed to review both some of the influences which have helped to fashion current advice, and more recent ideas which may provide answers in the future.

Why the Increase in Carbohydrate?

Based on experimental and epidemiological data [9–12] and contemporary dietary measures used in the management of the hyperlipidaemic patient [13, 14], it was felt appropriate to reduce the fat intake of the diabetic. In view of the significant excess cardiovascular mortality, suffered especially by Type 2 (non-insulin dependent) diabetic patients [15, 16], this measure was to be applied irrespective of the presence of hyperlipidaemia.

The other theme which has undoubtedly influenced contemporary thinking is the so-called 'dietary fibre hypothesis' of Trowell and Burkitt [17]. This emphasised that freedom from a wide spectrum of diseases, including colonic cancer, diabetes and cardiovascular disease, might be related to eating diets composed largely of unrefined starchy staples and containing substantial amounts of dietary fibre. As the 'dietary fibre hypothesis' evolved in the early 1970's, it carried with it the implicit assumption that increased carbohydrate intakes, necessary to obtain adequate fibre intake from unprocessed foods, were also beneficial. To a large extent, in the absence of solid laboratory data, and (many would claim) even less solid epidemiology [18], this idea has influenced a number of official pronouncements on diet including those of the Senate Select Committee on the U.S. Dietary Goals in 1977 [19]. The population as a whole was thus advised to eat less fat and more unprocessed carbohydrate foods. Again the aim was to reduce the risk of cardiovascular disease. As further evidence has accumulated, the Heart Foundations [20, 21] and Diabetes Associations [1–3] have endorsed this general move. Sheer weight of support does not indicate the rightness of a manoeuvre. Nevertheless many facets of what may be termed dietary fibre 'prophecy' have subsequently been shown to be true or at least to have lead to fruitful exploration of underdeveloped areas in nutrition, gastroenterology and metabolism.

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The Fibre Hypothesis and High Carbohydrate High Fibre Diets

Before the current interest in fibre, raising carbohydrate intakes from 40% to 60% had been reported to have no deleterous effect in diabetic subjects [22–24]. On the contrary, there were data which demonstrated that high carbohydrate diets might even produce a small improvement in glucose tolerance [25, 26]. Nevertheless if taken as liquid formula diets, they could result in higher postprandial blood glucose levels [27, 28]. In addition they tended to produce hypertriglyceridaemia. This was more marked if liquid formula rather than solid foods were taken [29, 30]. Such findings gave an early indication of the possible importance of the form in which the carbohydrate was fed in determining the physiological response.

The pioneer work of Anderson was the first to test the use of high carbohydrate diets in the context of the fibre hypothesis. He provided 70% carbohydrate diets containing 64 g dietary fibre to non-obese middle aged men previously controlled on low doses of insulin and taking 35% carbohydrate low-fibre diets (American Diabetes Association diets of the time) [31–35]. The fibre in these diets was derived from cereals (40%), starchy vegetables and legumes (20%), other vegetables (31%), and fruit (9%) taken as whole foods, as opposed to fibre supplements [34]. This was achieved by substantially reducing the intake of animal produce. In a series of reports Anderson and his team demonstrated their ability to withdraw insulin therapy in those on low doses of insulin (< 30 U/day) and to reduce the insulin requirements in those on higher doses while maintaining or improving diabetic control [31–35]. These trials were carried out under carefully controlled conditions in a metabolic ward. Since then patients have been maintained as outpatients for periods of over 3 years with a reduction in carbohydrate intake to 60% [32].

It might be said that such work merely represented an educational experience in dietary control and that the insulin reduction seen was dependent not so much on the nature of the diet but rather on the enforced dietary discipline. Nevertheless it demonstrated that an increased carbohydrate intake, provided it was in the form of unprocessed high fibre foods, need in no way worsen the diabetes or result in the elevated triglyceride levels seen with diets high in refined carbohydrate [27–30]. On the contrary, the studies showed lower triglyceride levels in those whose values were already raised [33] and a significantly lower serum cholesterol in the group as a whole [31]. This work has encouraged others to explore a similar approach.

Mann and his colleagues raised the carbohydrate intake from 40% to 60% in the form of diets high in cereal fibre (largely as wholemeal bread). This lowered glycosylated haemoglobin in Type 2 diabetes, and basal (03.00–07.00 h) and fasting blood glucose levels in both Type 1 [36] and Type 2 [37] diabetic patients, over the course of 6-week outpatient studies. Taking advantage of differences in regional taste to increase carbohydrate and fibre intake (54 g/day), workers in Naples used fibre largely from legumes (45%) and less commonly consumed vegetables (37%) (eg. egg-plant, artichokes, beansprouts and fennel), to reduce post-prandial glycaemia as well as total and low density lipoprotein cholesterol in a mixed group of diabetic patients [38]. Comparison of the high-fibre, high-carbohydrate treatment with a low-fibre, high-carbohydrate diet demonstrated a clear effect of fibre, in that fasting blood glucose and 24-h urinary glucose values were significantly lower and serum triglyceride levels were also reduced. This study did not indicate any benefit from increased carbohydrate intake, but did show an advantage when high-fibre foods were taken. Further work emphasizing the value of legumes demonstrated that most aspects of diabetic control were improved on a 60% carbohydrate diet which provided 150 g fibre/day, 64% from legumes and the remainder from cereals [39]. Seventy-three percent of this diet's 1920 calories were obtained from wholemeal bread, haricot beans, butter beans, kidney beans and dried peas and emphasized the extent of the dietary change required. Such diets also produced a small decrease in body weight. However the results were more impressive in Type 1 than in Type 2 diabetes [37].

These studies, following in the wake of the fibre hypothesis, lent support to a possible role of fibre in the diabetic diet. They did not indicate the extent to which high fibre or the combination of high fibre and high carbohydrate in the diet was responsible. Subsequent studies have shown that simple substitution of high- for low-fibre foods is effective [40], and there is also evidence that fibre and carbohydrate may act synergistically [41]. The least that can be said is that when the two were combined the diabetes was if anything improved, the triglyceride levels not raised and the cholesterol levels in all studies lowered. However, it is important to stress that consistent benefits from high carbohydrate diets have, to date, been reported only in studies involving high-fibre foods often with meal plans very different from those eaten in conventional Western societies. Much of the success of Anderson's diets therefore depends on the patient's ability to alter his established eating patterns and the capacity of his medical advisors to draw up a manageable diet plan. Nevertheless such work suggests a possible change in approach to the management of diabetes, although these prototype treatments would need considerable modification for general application.

Purified Fibre Supplements

A separate approach to the treatment of diabetes, stimulated by the dietary fibre hypothesis, was the use of dietary fibre supplements. At the time these appeared capable of providing a clear indication of the potential value of fibre in diabetes, and of allowing investigation of the mechanism of action. Taken as sachets or premixed into certain foods (eg. bread) they would prove acceptable as

a means of increasing fibre intake without further dietary manipulation.

Early on it had been suggested that fibre might act as a barrier to diffusion of nutrients from the lumen to the gut mucosa [42]. In this way absorption from fibre-rich foods might be slower, utilising a greater length of small intestine (Fig. 1 A), than that from more refined foods (Fig. 1 B) [43]. In healthy individuals, the slower absorption was expected to result in a flatter and more prolonged blood response in terms of both glucose and hormones (Fig. 2 A) while rapid absorption would result in high rises, perhaps with a subsequent undershoot in blood glucose (Fig. 2 B) [43].

Coincident with the first publication showing that high-fibre high-carbohydrate diets improved diabetic control was a report which demonstrated that supplementation of meals by the viscous fibres guar and pectin flattened both blood glucose profiles and, in Type 2 diabetic patients, insulin responses [44]. This was followed by metabolic ward studies in which addition of 25 g guar daily to the diet reduced urinary glucose loss by 50% in a mixed group of Types 1 and 2 diabetic patients [45].

In normal volunteers, it was shown that the glucose tolerance at lunch time was improved if fibre was taken with a glucose load at breakfast [46]. This second meal effect may have been due to the lower non-esterified fatty acid and ketone body levels seen after taking fibre. Such metabolic events may in turn have resulted from the slower rate of carbohydrate absorption from the gut. Similar changes were seen in the response to a standard lunch if the preceding breakfast contained slowly digested rather than rapidly digested carbohydrate foods [47]. These findings emphasize that fibre may affect both the meal with which it is taken and the subsequent meal. Further studies in normal subjects using xylose as a marker of carbohydrate absorption have confirmed that viscous fibre prolongs absorption time without evidence of carbohydrate malabsorption [48].

The effect on post-prandial blood glucose response was much more marked with viscous than with particulate fibres such as wheat bran [48]. Although a direct relationship with viscosity has not always been confirmed, if viscous materials are rendered non-viscous then their effect on post-prandial responses is greatly reduced or abolished [48, 49]. Nevertheless in longer term studies, particulate fibre preparations, taken in high fibre breads, have been reported to benefit blood glucose control in diabetes [50–53]. Other mechanisms may therefore be operating.

The importance of these studies lay in the emphasis they placed on events within the gastrointestinal tract in determining changes seen in the peripheral blood. In this way a rationale was provided for the influence of unabsorbable carbohydrate on carbohydrate metabolism. Subsequent studies have extended these observations to illustrate marked differences in gut hormone response to high- compared with low-fibre meals, including reduced GIP [53, 54] and, in certain situations, lower enteroglucagon responses [54]. The long-term effects on villous

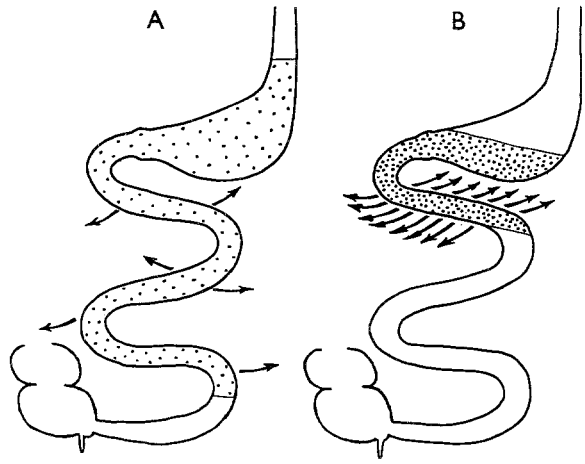

Fig. 1. Schematic representation of stomach and small intestine showing *(A)* slow digestion and absorption of energy-dilute food in a 'fibre-rich' diet and *(B)* rapid digestion and absorption of energy-dense food from low fibre diets

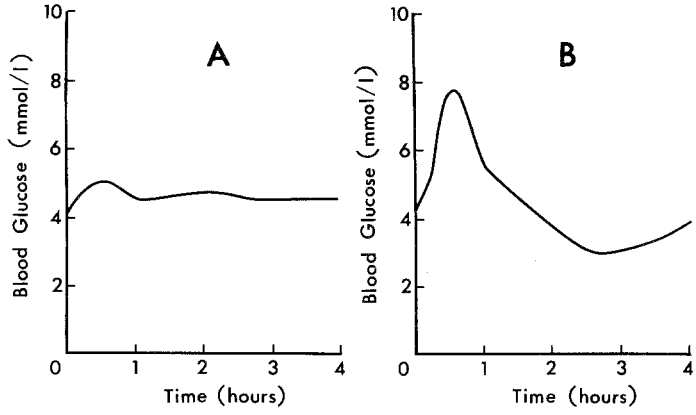

Fig. 2. Schematic representation of the post-prandial glycaemia following *(A)* slow absorption of starchy fibre-rich meals and *(B)* rapid absorption with undershoot due to excessive insulin release following refined, fibre-depleted carbohydrate foods

morphology, absorptive capacity, endocrine responses and whole body metabolism remain to be assessed in man [55]. In young rats addition of pectin to the diet reduced villous height and number [55]. Nevertheless, in normal volunteers dietary supplementation with 30 g pectin for 6 weeks did not result in flatter blood glucose profiles following a standard breakfest test meal [56]. Overall, however, longer term studies have indicated a beneficial effect on urinary glucose loss and insulin requirement in diabetic patients. By way of further explanation studies using the artificial pancreas have confirmed a small but significant decrease in insulin requirement (11%) during guar supplementation (20 g/day) for 24 h [57].

One of the debates which remains is the relative importance of a reduced rate of gastric emptying, observed to occur with viscous fibre [58–60], as opposed to a reduced rate of small intestinal absorption [61–63]. This matter is important in that glucose tolerance tests in patients with autonomic neuropathy were shown to be unaffected by guar [64]. It was argued that with already delayed gastric emptying in this condition guar was with-

out further influence [64]. However, it has also been shown that even after complete gastrectomy, guar still reduced the blood glucose rise after oral glucose by 20% [61].

A major impediment to the therapeutic use of fibre supplements is the requirement that they should be intimately mixed with the food, to simulate a situation analogous to the relationship found in unprocessed foods [65]. Inadequately mixed they have been shown to be largely ineffective [65–67]. At present the clinical use of purified fibre supplements is therefore severely limited both by this requirement and by the unpalatability of viscous materials. Only two products, an experimental guar crispbread [68, 69] and granulate [70] have been found to be palatable and effective and neither is produced commercially. Although the potential for practical impact of the fibre hypothesis might at present seem bleak, a number of useful lessons have been learnt.

Conclusions from Experimental Work on the Fibre Hypothesis

Firstly, increased carbohydrate intake need not result in impaired blood glucose control in the diabetic patient, provided it is given in the correct form [22–26, 31–39, 41, 71]. Secondly, unabsorbable food constituents (ie. dietary fibre) may reduce post-prandial blood glucose profiles and urinary glucose loss in diabetic patients [41, 44–46, 50–53, 57, 68, 69]. Additionally a combination of high carbohydrate and high fibre in the diet may both improve diabetic control and reduce blood lipids [31–39, 41]. Finally, modification of the rate of nutrient absorption together with the alteration in endocrine response may be a valuable objective of dietary therapy for the future.

For practical application, such lessons must be translated into actual foods for use in the diabetic diet. A guiding principle for their selection might be the rate at which individual foods release their nutrients on digestion in vitro, and their effect on blood glucose levels in vivo. Some work has already been undertaken in these areas.

Individual Foods – The Glycaemic Response

The concept of simple carbohydrate exchange has been challenged by the demonstration that not all starchy foods produce the same glycaemic response [72–77]. Comparing five starchy foods, including bread, rice, potato and maize, Crapo et al. demonstrated significant differences in both the character of the glycaemic response and the total amount of insulin secreted [72]. They concluded that this was related to differences not in the fibre but in the digestibility of the different starches. They reasoned that the more rapidly digested bread and potato caused higher rises in blood glucose and insulin levels. These findings were demonstrated in normal volunteers [72] and in Type 2 diabetic patients [73]. Schauberger et al. extended this approach in a study of 12 foods in normal volunteers, again noting the large differences in glycaemic response to the same amount of carbohydrate [74].

Not only the fibre and the nature of the starch but also the form in which the food is eaten has been suggested as an important determinant of the glycaemic response. Thus higher rises were seen after puréed as opposed to whole apples [75], ground compared with whole rice [76, 77] and cooked versus uncooked starch [78]. By comprehensive testing along these lines both the foods and the food factors responsible for determining the glycaemic response can be identified.

The Bean, an Example of Slow Release Carbohydrate

The bean is an example of how, on the basis of such experimental results, new foods may be identified for introduction into the diabetic diet. Although relatively neglected in the Western diet, beans first attracted interest as a source of fibre because they are eaten as staples in those parts of the world where cardiovascular disease is less common [79–81]. In addition, the bean fibre, guar, was among the first dietary fibres to be used successfully in diabetic [45, 46] and hyperlipidaemic treatment studies [82, 83]. This prompted tests in both normal volunteers and diabetic patients where it was shown that the leguminous seeds, lentils and soya beans, produced only 20%–40% of the blood glucose rise seen after consumption of the same amount of carbohydrate taken as wholemeal bread [84].

These results with legumes led to a comparison between a range of legumes and cereal products [85]. The blood glucose responses after testing eight legumes were only 50% as high as those after a wide range of other starchy foods, including breads, pasta, breakfast cereals, cereal products and root vegetables [85]. Such studies highlighted the possible beneficial role legumes might play in the diabetic diet. Their possible usefulness in the diabetic diet has been borne out by the Naples [38] and Oxford [39] studies, where much of the fibre came from the consumption of large amounts of cooked beans.

The Glycaemic Response, Digestibility and Anti-nutrients

One of the assumptions latent in the fibre hypothesis is that the rate of digestion and hence of absorption is a major determinant of the glycaemic response. If this is so it will be influenced by the many factors, other than fibre, which alter digestibility. The possible closeness of this relationship has only recently been demonstrated. Fourteen foods were digested with human saliva and pancreatic juice in vitro in a dialysis system and the results compared with the blood glucose response in healthy volunteers after feeding the same 14 foods individually as test meals. The carbohydrate released over 5 h of digestion in vitro was highly significantly related to the area under the 2 h glucose response curve ($r = 0.8618$, $n = 14$, $p < 0.001$) [86].

In this group of foods the negative relationship of fibre to the glycaemic response barely reached significance despite the fact that the legumes, as very rich

Table 1. Glycaemic index: the area under the blood glucose response curve for each food expressed as a percentage of the area after taking the same amount of carbohydrate as glucose. Data from normal individuals [95]

	Glycaemic index (%)		Glycaemic index (%)		Glycaemic index (%)
Grain, cereal products		*Fresh Legumes*		*Fruit*	
Bread (white)	69	Broad beans[a]	79	Apples (Golden Delicious)	39
Bread (wholemeal)	72	Frozen peas	51	Bananas	62
Buckwheat	51			Oranges	40
Millet	71	*Root Vegetables*		Orange juice	46
Pastry	59	Beetroot[a]	64	Raisins	64
Rice (brown)	66	Carrots[a]	92		
Rice (white)	72	Parsnips[a]	97	*Sugars*	
Spaghetti (wholemeal)	42	Potato (instant)	80	Fructose	20
Spaghetti (white)	50	Potato (new)	70	Glucose	100
Sponge cake	46	Potato (sweet)	48	Maltose	105
Sweetcorn	59	Swede[a]	72	Sucrose	59
		Yam	51		
Breakfast cereals		*Dried and Tinned Legumes*		*Dairy Products*	
'All-bran'	51	Beans (tinned, baked)	40	Ice cream	36
Cornflakes	80	Beans (butter)	36	Milk (skimmed)	32
Muesli	66	Beans (haricot)	31	Milk (whole)	34
Porridge Oats	49	Beans (kidney)	29	Yoghurt	36
'Shredded Wheat'	67	Beans (soya)	15		
'Weetabix'	75	Beans (tinned soya)	14	*Miscellaneous*	
		Peas (blackeye)	33	Fish fingers	38
Biscuits		Peas (chick)	36	Honey	87
Digestive	59	Peas (marrowfat)	47	'Lucozade'	95
Oatmeal	54	Lentils	29	'Mars bar'	68
'Rich Tea'	55			Peanuts[a]	13
'Ryvita'	69			Potato crisps	51
Water	63			Sausages	28
				Tomato soup	38

[a] Only 25 g carbohydrate portion given.

sources of fibre, showed the lowest glycaemic responses and slowest rates of digestion [86]. A wide range of food factors might be responsible for such differences in digestibility. These include: enzyme inhibitors, lectins, phytates, tannins, and starch-protein, starch-lipid interactions. It is worthy of note that beans are rich sources of these so-called anti-nutrients.

Many of these factors along with fibre are consumed in relatively large amounts in the diets of more primitive cultures but have been reduced in concentration both by processing and by food preferences in the Western diet. The α-amylase inhibitor of wheat may survive baking [87] and in purified form has been shown to reduce the glycaemic response to starch-containing foods [88]. Recently an α-glycoside hydrolase inhibitor (Acarbose, Bayer) has been developed commercially expressly to reduce the rates of digestion of starch and absorption of sucrose. This is now marketed as a means of reducing the glycaemic response in the treatment of diabetes [89–91]. One can only speculate that fibre has paved the way for other anti-nutrients to gain respectability as nutritional or pharmacological agents in the treatment of disease; and more may appear with time. This in itself marks quite a reversal in thinking over the past two decades.

The Colon and Carbohydrate Absorption

If slower absorption of carbohydrate is a goal, what then of the carbohydrate which is not absorbed in the small intestine? One result is the flatulence noted with beans. Due to colonic bacterial metabolism of carbohydrate, hydrogen, methane and carbon dioxide are formed. However, in addition to the gases, short-chain acids (including lactic, propionic and butyric acids) are also produced [92]. There is evidence from human and rat studies that substantial amounts of these are absorbed from the colon to be used in metabolic processes within the body [92, 93]. In short, a degree of ruminant function is now being ascribed to the human colon. The nature of the carbohydrate and other food factors influencing small intestinal digestibility will determine the extent of colonic absorption. For example, it has been estimated that, due to the presence of gluten and the starch-protein interaction, 10%–20% of bread carbohydrate eaten is not absorbed in the small intestine [94] and will therefore be available for absorption as lactate and short-chain fatty acids by the colon.

Although the calorific value and metabolic effects of prolonged absorption of these metabolites is not known, they will not contribute to the post-prandial rise in blood glucose levels of the meal from which they are generated. At present there can only be speculation as to whether they contribute to the improved carbohydrate tolerance seen in the meal which follows one containing high fibre or slowly released carbohydrate [46, 47]. Thus dietary factors may not only influence the rate of small intestinal absorption, but may also alter the balance between small intestinal and colonic absorption.

Selection of Carbohydrate Foods

As more information becomes available on those factors which influence digestibility or directly influence the glycaemic response to foods, it will be possible to give more specific advice on how the carbohydrate content of the diabetic diet may best be increased. Until such time, use may be made of tables based on physiological testing, both to allow advice to be given and to advance the basic understanding of factors influencing the effects of foods. The table shown as an example (Table 1) [95] gives the glycaemic index of foods tested in normal individuals. The glycaemic index is derived from:

$$\frac{\text{area under the 2 h glucose response curve for a food}}{\text{area under the 2 h glucose response curve for the equivalent amount of glucose}} \times 100$$

Table 1 illustrates many of the points already made. These include: the low glycaemic response seen with beans, the lack of effect of cereal fibre, the effect of food form, and the relatively low blood glucose response to sugars [96] in fruit, biscuits, 'All-bran' and dairy products (probably explained by the small response to sucrose, fructose and lactose as such).

Such tables may form a useful basis for dietary planning, but the lack of explanation which can be given for the observed differences illustrates that we have much to learn of the food factors which determine the glycaemic response.

Conclusion

The last decade has seen much interest in the physiological response to carbohydrate foods. Interest in dietary fibre emphasised the possible influence of events within the gastrointestinal tract on carbohydrate metabolism. Links have been established between digestibility and the glycaemic response to foods. However, the influence of food factors on these processes is ill understood. Nevertheless food form, certain types of fibre, other anti-nutrients and the nature of the starch-protein interaction may be major determinants. Genetic differences in the responses of different individuals to the same food remain to be explored. For example, the protein, gliadin, may act as a lectin [97] in susceptible individuals to the extent of causing villous atrophy (coeliac disease) and so severely limiting absorption. Coeliac disease and Type 1 diabetes are linked both in occurrence [98] and in the frequency with which sufferers share the same tissue antigens (HLA-B8 and DW3) [99–102]. Less dramatic alterations in absorptive capacity may be seen in subclinical coeliac disease or with other anti-nutrients. These, therefore, represent other ways in which food can modify the glycaemic response. Perhaps in those with impaired carbohydrate metabolism a mildly reduced absorptive capacity could be beneficial.

In view of the present state of knowledge, the decisions of the American and Canadian Diabetes Associations and the British Diabetic Association to increase carbohydrate intake [1–3] may be seen as a worthwhile move to encourage not so much gastronomic licence, but, some would say, more frugal living. The frugality might be lightened by inclusion of new foods and preservation of certain ethnic dishes. Indeed choice for the diabetic may ultimately be greatly enhanced as indicated by two recently published diet guides [103, 104]. The advice was not intended to be thrust on all with a sweep of the pen but it will give support to those willing to prescribe, and those willing to accept, such diets. In addition, it has provided a tremendous impetus to further activity for those working in this field. For those unable to take the diets, continued exploration along these lines may result in new pharmaceutical approaches to the management of diabetes.

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References

1. Committee of the American Diabetes Association on Food and Nutrition (1979) Special Report: Principles of nutrition and dietary recommendations for individuals with diabetes mellitus. Diabetes Care 2: 520–523
2. Special Report Committee (1981) Guidelines for the nutritional management of diabetes mellitus: A special report from the Canadian Diabetes Association. J Can Dietet Assoc 42: 110–118
3. The Nutrition Sub-committee of the British Diabetic Association's Medical Advisory Committee: Dietary recommendations for diabetes for the 1980's, Final Draft July 1981
4. Reaven GM (1980) How much carbohydrate? Diabetologia 19: 409–413
5. Reaven GM (1981) How much carbohydrate: Diabetologia 20: 508–509 (Letter)
6. Jarrett RJ (1981) How much carbohydrate? Diabetologia 20: 507 (Letter)
7. Mann JI (1981) How much carbohydrate? Diabetologia 20: 507–508 (Letter)
8. Jarrett RJ (1981) More about carbohydrates. Diabetologia 21: 427–428 (Letter)
9. Dayton S, Pearce ML, Goldman H, Harrish A, Harnish A, Plotkin D, Shickman M, Winfield M, Zager A, Dixon W (1968) Controlled trial of a diet high in unsaturated fat for prevention of atherosclerotic complications. Lancet 2: 1060–1062
10. Rinzler SH (1968) Primary prevention of coronary heart disease by diet. Bull NY Acad Med 44: 936–949
11. Miettinen M, Turpeinen O, Karvonen MN, Elosuo K, Poavilainen E (1972) Effect of cholesterol-lowering diet on mortality from coronary heart disease and other causes: a twelve year clinical trial in men and women. Lancet 2: 835–838
12. Kawate R, Yamakido M, Nishimato Y, Bennett PH, Hamman RF, Knowler WC (1979) Diabetes mellitus and its vascular complications in Japanese migrants on the island of Hawaii. Diabetes Care 2: 161–170
13. Fredrickson DS, Levy RI, Lees RS (1967) Fat transport in lipoproteins – an integrated approach to mechanisms and disorders. N Engl J Med 276: 215–225
14. Lewis B (1980) The hyperlipidaemias: clinical and laboratory practice. Blackwell Scientific Publications. Oxford
15. Ostrander LD, Francis T, Hayner NS, Kjelsberg MD, Epstein FH (1965) The relationship of cardiovascular disease to hyperglycemia. Ann Intern Med 62: 1188–1198

16. Garcia M, McNamara P, Gordon T, Kannel WB (1974) Morbidity and mortality in diabetics in the Framingham population: sixteen year follow-up study. Diabetes 23: 105–111
17. Trowell HC, Burkitt DP (1975) Concluding considerations. In: Burkitt DP, Trowell HC (eds). Refined carbohydrate foods and disease. Academic Press, London, pp 333–345
18. Mendeloff AI (1978) Dietary fiber and gastrointestinal diseases: some facts and fancies. Med Clin N Amer 62: 165–171
19. Senate Select Committee on Nutrition and Human Needs (1977) Dietary Goals for the United States. US Government Printing Office, Washington, DC
20. Turner RWD (1978) Perspectives in coronary prevention. Postgrad Med J 54: 141–148
21. Report of Nutrition Committee of the American Heart Association (1982) Rationale of the diet-heart statement of the American Heart Association. Circulation 65: 839A–854A
22. Stone DB, Connor WE (1965) Prolonged effects of a low cholesterol, high carbohydrate diet upon the serum lipids in diabetic patients. Diabetes 12: 127–132
23. Weinsier RL, Seeman A, Henera MG, Assal JP, Soeldner JS, Gleason RE (1974) High and low carbohydrate diets in diabetes mellitus. Study of effects on diabetic control, insulin secretion and blood lipids. Ann Intern Med 80: 332–341
24. Singh I (1955) Low-fat diet and therapeutic dose of insulin in diabetes mellitus. Lancet 1: 422–425
25. Anderson JW (1977) Effect of carbohydrate restriction and high carbohydrate diets in men with chemical diabetes. Am J Clin Nutr 30: 402–408
26. Brunzell JD, Lerner RL, Hazard WR, Porte D, Bierman EL (1971) Improved glucose tolerance with high carbohydrate feeding in mild diabetes. N Engl J Med 284: 521–524
27. Farquhar JW, Frank A, Gross RC, Reaven GM (1966) Glucose, insulin and triglyceride responses to high and low carbohydrate diet in man. J Clin Invest 45: 1648–1656
28. Reaven GM (1979) Effect of variations in carbohydrate intake on plasma glucose, insulin, and triglyceride responses in normal subjects and patients with chemical diabetes. In: Camerini-Davalos RA, Hanover BA (eds). Treatment of Early Diabetes, Plenium Press, New York, pp 253–262
29. Reiser S, Hallfrisch J, Michaelis OE, Lagar FL, Martin RF, Prather ES (1979) Isocaloric exchange of dietary starch and sucrose in humans. Am J Clin Nutr 32: 1659–1669
30. Antar MA, Little JA, Lucas C, Buckley GC, Csima A (1970) Interrelationships between dietary carbohydrate and fat in hyperlipidemic patients. Part 3. Synergistic effect of sucrose and animal fat on serum lipids. Atherosclerosis 11: 191–201
31. Kiehm TG, Anderson JW, Ward K (1976) Beneficial effects of a high carbohydrate high fiber diet in hyperglycemic men. Amer J Clin Nutr 29: 895–899
32. Anderson JW, Ward K (1978) Long-term effects of high carbohydrate, high fiber diets on glucose and lipid metabolism: A preliminary report on patients with diabetes. Diabetes Care 1: 77–82
33. Anderson JW, Chen WL (1979) Plant fiber carbohydrate and lipid metabolism. Am J Clin Nutr 32: 346–363
34. Anderson JW, Ward K (1979) High carbohydrate high fiber diets for insulin-treated men with diabetes mellitus. Am J Clin Nutr 32: 2312–2321
35. Anderson JW (1979) High carbohydrate, high fiber diets for patients with diabetes. In: Camerini-Davalos RA, Hanover BA (eds). Treatment of Early Diabetes. Plenum Press, New York, pp 263–273
36. Simpson RW, Mann JI, Eaton J, Carter RD, Hockaday TDR (1979) High carbohydrate diets in insulin-dependent diabetes. Brit Med J 2: 523–525
37. Simpson RW, Mann JI, Eaton J, Moore RA, Carter R, Hockaday TDR (1979) Improved glucose control in maturity onset diabetes treated with carbohydrate-modified fat diet. Br Med J 1: 1752–1756
38. 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
39. Simpson HCR, Simpson RW, Lousley S, Carter RD, Geekie M, Hockaday TDR, Mann JI (1981) A high carbohydrate leguminous fibre diet improves all aspects of diabetic control. Lancet 1: 1–5
40. 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
41. Jenkins DJA, Wolever TMS, Bacon S, Nineham R, Lees R, Rowden R, Love M, Hockaday TDR (1980) Diabetic diets: high carbohydrate combined with high fibre. Am J Clin Nutr 33: 1729–1733
42. Southgate DAT AT (1973) Fibre and the other unavailable carbohydrates and their effects on the energy value of the diet. Proc Nutr Soc 32: 131–136
43. Jenkins DJA, Wolever TMS (1981) Slow release carbohydrate and the treatment of diabetes. Proc Nutr Soc 40: 227–235
44. Jenkins DJA, Leeds AR, Gassull MA, Wolever TMS, Goff DV, Alberti KGMM, Hockaday TDR (1976) Unabsorbable carbohydrates and diabetes: decreased postprandial hyperglycaemia. Lancet 2: 172–174
45. Jenkins DJA, Wolever TMS, Hockaday TDR, Leeds AR, Haworth R, Bacon S, Apling EC, Dilawari J (1977) Treatment of diabetes with guar gum. Lancet 2: 779–780
46. Jenkins DJA, Wolever TMS, Nineham R, Sarson DL, Bloom SR, Ahern J, Alberti KGMM, Hockaday TDR (1980) Improved glucose tolerance four hours after taking guar with glucose. Diabetologia 19: 21–24
47. Jenkins DJA, Wolever TMS, Taylor RH, Griffiths C, Krzeminska K, Lawrie JA, Bennett CM, Goff DV, Sarson DL, Bloom SR (1982) Slow release carbohydrate improves second meal tolerance. Am J Clin Nutr 35: 1339–1346
48. Jenkins DJA, Wolever TMS, Leeds AR, Gassull MA, Dilawari JB, Goff DV, Metz GL, Alberti KGMM (1978) Dietary fibres, fibre analogues and glucose tolerance: importance of viscosity. Br Med J 1: 1392–1394
49. O'Connor N, Tredger J, Morgan L (1981) Viscosity differences between various guar gums. Diabetologia 20: 612–615
50. Bosello O, Ostuzzi R, Armellini F, Micciolo RM, Ludovico AS (1980) Glucose tolerance and blood lipids in bran fed patients with impaired glucose tolerance. Diabetes Care 3: 46–49
51. Miranda PM, Horiwitz DL (1978) High fiber diets in the treatment of diabetes mellitus. Ann Intern Med 88: 482–486
52. Monnier LH, Blotman MJ, Colette C, Monnier MP, Mirouze J (1981) Effects of dietary fibre supplementation in stable and labile insulin-independent diabetics. Diabetologia 20: 12–17
53. Morgan LM, Gondler TJ, Tsiolakis D, Marks V, Alberti KGMM (1979) The effect of unabsorbable carbohydrate on gut hormones: Modification of post-prandial GIP secretion by guar. Diabetologia 17: 85–89
54. Jenkins DJA, Leeds AR, Bloom SR, Sarson DL, Albuquerque RH, Metz GL, Alberti KGMM (1980) Pectin and post-gastric surgery complications: Normalisation of postprandial glucose and endocrine responses. Gut 21: 574–579
55. Tasman-Jones C (1980) Effects of dietary fiber on the structure and function of the small intestine. In: Spiller GA, Kay RM (eds). Medical Aspects of Dietary Fiber. Plenum Medical Books, New York, London, pp 67–74
56. Jenkins DJA, Leeds AR, Houston H, Hinks L, Alberti KGMM, Cummings JH (1977) Carbohydrate tolerance in man after six weeks of pectin administration. Proc Nutr Soc 36: 60A (Abstract)
57. Christiansen JS, Bonnevie-Nielsen V, Svendsen PA, Rubin P, Ronn B, Nerup J (1980) Effect of guar gum on 24-hour insulin requirements of insulin-dependent diabetic subjects as assessed by an artificial pancreas. Diabetes Care 3: 659–662
58. Leeds AR, Ralphs DN, Boulos P, Ebied F, Metz GL, Dalawari J, Elliott A, Jenkins DJA (1978) Pectin and gastric emptying in the dumping syndrome. Proc Nutr Soc 37: 23 (Abstract)
59. Leeds AR, Ralphs DNL, Ebied F, Metz G, Dilawari JB (1981) Pectin in the dumping syndrome: Reduction of symptoms and plasma volume changes. Lancet 1: 1075–1978
60. Holt S, Heading RC, Carter DC, Prescott LF, Tothill P (1979) Effect of gel fibre on gastric emptying and absorption of glucose and paracetamol. Lancet 1: 636–639

61. Taylor RH (1979) Gastric emptying, fibre and absorption. Lancet 1: 872 (Letter)
62. Elsenhaus B, Sufke U, Blume R, Caspary WF (1980) The influence of carbohydrate gelling agents on rat intestinal transport of monosaccharides and neutral amino acids in vitro. Clin Sci 59: 373–380
63. Johnson IT, Gee JM (1980) Inhibitory effect of guar gum on the intestinal absorption of glucose in vitro. Proc Nutr Soc 39: 52 (Abstract)
64. Levitt NS, Vinik AI, Sive AA, Child PT, Jackson WPU (1980) The effect of dietary fiber on glucose and hormone responses to a mixed meal in normal subjects and in diabetic subjects with and without autonomic neuropathy. Diabetes Care 3: 515–519
65. Jenkins DJA, Nineham R, Craddock C, Craig-McFeely P, Donaldson K, Leigh T, Snook J (1979) Fibre and diabetes. Lancet 1: 434–435 (Letter)
66. Williams DRR, James WPT, Evans IE (1980) Dietary fibre supplementation of a 'normal' breakfast administered to diabetics. Diabetologia 18: 379–383
67. Cohen M, Leong VW, Salmon E, Martin FIR (1980) The role of guar and dietary fibre in the management of diabetes mellitus. Med J Aust 1: 59–61
68. Jenkins DJA, Wolever TMS, Nineham R, Taylor R, Metz GL, Bacon S, Hockaday TDR (1978) Guar crispbread in the diabetic diet. Br Med J 2: 1744–1746
69. Jenkins DJA, Wolever TMS, Taylor RH, Reynolds D, Nineham R, Hockaday TDR (1980) Diabetic glucose control, lipids, and trace elements on long term guar. Br Med J 1353–1354
70. Aro A, Uusitripa M, Vontilainen E, Hersio K, Korhonen T, Sirtonen (1981) Improved diabetic control and hypocholesterolemic effect induced by long-term dietary supplementation with guar gum in Type 2 (insulin-dependent) diabetes. Diabetologia 21: 29–33
71. Hockaday TDR, Hockaday JM, Mann JI, Turner RC (1978) Prospective comparison of modified-fat-high-carbohydrate with standard low-carbohydrate dietary advice in the treatment of diabetes: one year follow-up study. Br J Nutr 39: 357–362
72. Crapo PA, Reaven G, Olefsky J (1977) Post-prandial plasma-glucose and insulin responses to different complex carbohydrates. Diabetes 26: 1178–1183
73. Crapo PA, Kolterman OG, Waldeck N, Reaven GM, Olefsky JM (1980) Postprandial hormonal responses to different types of complex carbohydrate in individuals with impaired glucose tolerance. Am J Clin Nutr 33: 1723–1728
74. Schauberger G, Brinck UC, Suldner G, Spaethe R, Niklas L, Otto H (1978) Exchange of carbohydrates according to their effect on blood glucose. Diabetes 26: 415 (Abstract)
75. Haber GB, Heaton KW, Murphy D, Burroughs LF (1977) Depletion and disruption of dietary fibre: Effects on satiety, plasma-glucose, and insulin. Lancet 2: 679–682
76. 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
77. O'Dea K, Snow P, Nestel P (1981) Rate of starch hydrolysis in vitro as a predictor of metabolic responses to complex carbohydrate in vivo. Am J Clin Nutr 34: 1991–1993
78. Collings P, Williams C, MacDonald I (1981) Effect of cooking on serum glucose and insulin responses to starch. Br Med J 282–1032
79. Grande F, Anderson JT, Keys A (1965) Effect of carbohydrates of leguminous seeds, wheat and potatoes on serum cholesterol in man. J Nutr 86: 313–317
80. Marthur KS, Khan MA, Sharma RD (1968) Hypocholesterolemic effect of Bengal Gram: a longterm study in Man. Br Med J 1: 30–31
81. Sirtori CR, Agrandi E, Conti F, Mantero O, Gatti E (1977) Soybean-protein diet in the treatment of type II hyperlipoproteinaemia. Lancet 1: 275–277
82. Jenkins DJA, Leeds AR, Slavin B, Mann J, Jepson EM (1979) Dietary fibre and blood lipids: reduction of serum cholesterol in type II hyperlipidemia by guar gum. Am J Clin Nutr 32: 16–18
83. Jenkins DJA, Reynolds D, Slavin B, Leeds AR, Jenkins AL, Jepson EM (1980) Dietary fibre and blood lipids: treatment or hypercholesterolemia with guar crispbread. Am J Clin Nutr 33: 575–581
84. Jenkins DJA, Wolever TMS, Taylor RH, Ghafari H, Jenkins AL, Barker H, Jenkins MJA (1980) Rate of digestion of foods and postprandial glycaemia in normal and diabetic subjects. Br Med J 2: 14–17
85. Jenkins DJA, Wolever TMS, Taylor RH, Barker H, Fielden H (1980) Exceptionally low blood glucose response to dried beans: comparison with other carbohydrate foods. Br Med J 2: 578–580
86. Jenkins DJA, Ghafari H, Wolever TMS, Taylor RH, Barker HM, Fielden H, Jenkins AL, Bowling AC (1982) Relationship between the rate of digestion of foods and post-prandial glycaemia. Diabetologia 22: 6
87. Militzer W, Ikeda C, Kreen E (1946) The preparation and properties of an amylase inhibitor of wheat. Arch Biochem 9: 309–320
88. Puls W, Keup V (1973) Influence of an α-glucoside inhibitor (Bay d7791) on blood glucose, serum insulin and NEFA in starch loading tests in rats, dogs and man. Diabetologia 9: 97–101
89. Walton RJ, Sharif IT, Nog GA, Alberti KGMM (1979) Improved metabolic profiles in insulin treated diabetic patients given an α-glucosidehydrolase inhibitor. Br Med J 1: 220–221
90. Gerard J, Luyckx AS, Lefebvre PJ (1982) Long-term improvement of metabolic control in insulin-treated diabetes by the α-glucoside inhibitor acarbose. Diabetologia 21: 446–451
91. Hillebrand I, Bochine K, Kink H, Berchtold P (1979) The effects of the α-glucosidase inhibitor Bay g5421 (Acarbose) on meal stimulated elevations of circulating glucose, insulin and triglyceride levels in man. Res Exp Med (Berk) 175: 81–86
92. Bond JH, Levitt MD (1976) Fate of soluble carbohydrate in the colon of rats and man. J Clin Invest 57: 1158–1164
93. Bond JA, Currier BE, Buchwald H, Levitt MD (1980) Colonic conservation of malabsorbed carbohydrate. Gastroenterology 78: 444–447
94. Anderson IH, Levine AS, Levitt MD (1981) Incomplete absorption of the carbohydrate in all-purpose wheat flour. N Eng J Med 304: 891–892
95. Jenkins DJA, Wolever TMS, Taylor RH, Barker HM, Fielden H, Baldwin JM, Bowling AC, Newman HC, Jenkins AL, Goff DV (1981) Glycemic index of foods: a physiological basis for carbohydrate exchange. Amer J Clin Nutr 34: 362–366
96. Lenner RA (1976) Studies of glycemia and glucosuria in diabetics after breakfast meals of different composition. Am J Clin Nutr 29: 716–725
97. Weiser MM, Douglas AP (1976) An alternative mechanism for gluten toxicity in coeliac disease. Lancet 1: 567–569
98. Hooft C, Devos E, Van Danme J (1969) Coeliac disease in a diabetic child. Lancet 2: 161 (Letter)
99. Falchuk ZM, Rogetine GN, Strober W (1972) Predominance of histocompatibility antigen HLA-8 in patients with gluten sensitive enteropathy. J Clin Invest 51: 1602–1605
100. Keuning JJ, Pena AS, Van Leeuwen A, Van Hooff JP, Van Rood JJ (1976) HLA-DW3 association with coeliac disease. Lancet 1: 506–507
101. Cudworth AG (1978) Type 1 diabetes mellitus. Diabetologia 14: 281–291
102. Sachs JA, Cudworth AG, Jaraquemade D, Gorsuch AN, Festenstein H (1980) Type 1 diabetes and the HLA-D locus. Diabetologia 18: 41–43
103. Anderson JW (1981) Diabetes: a practical new guide to healthy living. Martin Dunitz, London
104. Mann JI (1981) The high-fibre eating programme. Martin Dunitz, London

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