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2 Benefits of fibre itself are uncertain

Robert Tattersall, Peter Mansell

A former dean of the Harvard Medical School is said to have told students at graduation, "Half of what we have taught you is wrong. Unfortunately, we don't know which half!"¹ The diet for non-insulin dependent diabetes is a case in point. Many doctors were taught simplistically that too much glucose in the blood could be treated only by eating as little carbohydrate as possible. They therefore prescribed low carbohydrate diets that were high in fat as this was the only affordable way of making up the energy. Currently, most diabetes associations, including the British Diabetic Association,² recommend high carbohydrate, high fibre, low fat diets for all diabetics, although this is tempered by the advice that "any dietary strategy—for example, calorie counting, slimming clubs, etc—which reduces energy intake in the obese non-insulin dependent diabetic is acceptable if it is nutritionally sound."³

We agree that many patients with non-insulin dependent diabetes are hyperlipidaemic⁴ and that this may contribute to their exorbitant rates of arterial disease. It is also a reasonable premise that eating less saturated fat will be beneficial and this is best achieved by a reciprocal increase in dietary carbohydrate. Nevertheless, diets high in carbohydrate and fibre raise the same questions as any other innovation in treatment. Do they work and, even if they do, will people follow them?

History of high fibre diets and diabetes

High carbohydrate diets for diabetes have been in vogue intermittently for over 100 years. For example, in the early 1900s van Noorden's "oat cure" was greeted as an astounding and mysterious phenomenon and followed by much wasted effort to show the superiority of oatmeal over other carbohydrates (a foretaste of the minutiae of the glycaemic index). The fibre story began in 1880 when Dr T R Allinson (whose 100% stoneground wholemeal flour is still available) wrote, "One great curse of this country is constipation, which is caused in great measure by white bread. From this constipation come piles, varicose veins, headaches, miserable feelings, dullness and other ailments."⁵ Dr Allinson was struck off the medical register, and only in the late 1960s was his idea that Western diseases are due to a deficiency of dietary fibre resurrected by Cleave, Burkitt, and Trowell. Their hypothesis that the excess of constipation, appendicitis, cancer of the colon, and diabetes in Europeans compared with Africans was due to a deficiency of dietary fibre is now well accepted, although the epidemic of diabetes in developing countries is probably a multifactorial process in which genes, lack of exercise, and obesity are as or more important than a deficiency of fibre.

Even if a lack of fibre does cause diabetes, however, it does not necessarily follow that eating it in normal or excessive quantities will be beneficial in treating the established disease. In the mid-1970s Jenkins *et al* in Oxford showed that adding the unabsorbable polysaccharides guar and pectin reduced postprandial blood glucose concentrations in both normal and diabetic

people, with a flattening of the insulin response in the controls and a need for less insulin in the diabetic subjects.⁶ Not all fibres were equal. Highly viscous ones (such as guar) worked best, gum tragacanth and methylcellulose were less effective, and pectin and bran were unimpressive. To reduce blood glucose concentra-

... The glycaemic response to a particular food correlates only weakly with the total fibre content.

tions the fibre had not only to be viscous but also to be incorporated into the food—giving it as a capsule, sprinkling it on the food, or taking it before a meal did not work. This differential effect of various fibres and the difficulty in defining exactly what is being fed to patients have led to many problems. It is easy to define fibre as "components of plant material which resist human digestive enzymes,"⁷ but life becomes difficult when we are warned that:

Fibre-rich foods contain a variety of fibres and the effects of each cannot be determined using natural foods. On the other hand, when fibres are extracted from natural foods, their actions may not resemble those of the same fibres when fed as an integral part of the intact food. The fibre-nutrient relationship in foods is disrupted by cutting, cooking and chewing. . . .⁸

For simplicity we can say that insoluble fibre—for example, wheat bran—speeds up intestinal transit, increases faecal bulk, and is good for the bowels, whereas soluble fibre (such as guar) gums you up, delays gastric emptying, and may lower blood glucose concentrations. The glycaemic response to a particular food correlates only weakly with the total fibre content and may have more to do with the chemical and mechanical forms in which the carbohydrate is held.⁸

Clinical trials of high fibre diets

The earliest clinical trials of high fibre diets were performed in the late 1970s, and one English and one American study were particularly influential. In Oxford a six week crossover study in 18 patients with non-insulin dependent diabetes compared a standard low carbohydrate diet with a "high carbohydrate diet containing leguminous fibre."⁹ Most of the patients were taking sulphonylureas and their glycaemic control was already good with the low carbohydrate diet; their mean fasting blood glucose concentration of 6.7 mmol/l was reduced to 5.7 mmol/l by the test diet. What was not fully appreciated by many was that the daily provision of fibre in the test diet was a massive 96.6 g, of which "64% was derived from leguminous sources, most of the remainder being cereal fibre in the form of wholemeal bread . . . beans were consumed twice daily, usually at breakfast and supper." In retrospect, it was unclear whether the improved diabetic control (such as

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it was) was due to the high carbohydrate intake, the slow rate of digestion of the legume starch, or the fibre. In the United States Anderson and Ward using a diet containing 70% carbohydrate, 19% protein, 11% fat, and 36 g fibre/1000 calories (4184 MJ) showed significant decreases in fasting and postprandial glucose concentrations.¹⁰ Much was made of the fact that many patients (all overweight with non-insulin dependent diabetes) discontinued taking insulin or tablets, but what was not emphasised was that most patients lost weight and the greatest reduction in glucose concentrations and insulin doses was in those who lost most weight.

These and other studies which have shown a beneficial effect of high fibre diets are criticised by Reaven and Coulston *et al* as "short-term and using diets with an enormous and impractical enrichment of fibre in patients who were already well controlled."^{11 12}

In the more typical patients with late onset diabetes and poor glycaemic control the results are conflicting but generally unimpressive. In Oxford 11 out of 15 patients with non-insulin dependent diabetes who completed a fibre study had a fasting plasma glucose concentration of 9.6 mmol/l while eating their usual diet (26 g fibre/day). The fasting plasma glucose concentration was 8.4 mmol/l after six weeks of eating a low carbohydrate diet (12.7 g fibre/day) and 6.8 mmol/l with a high carbohydrate diet containing 67 g fibre/day.¹³ Coulston *et al*, however, found a significant deterioration in blood glucose concentration after only 15 days of a 60% carbohydrate diet containing 38 g fibre.¹² The same group found that raising the fibre from 11.0 to 22.7 g/1000 cal (4184 MJ)/day had no significant effect on fasting or postprandial plasma glucose, insulin, triglyceride, or cholesterol concentrations.¹⁴ In Nottingham we treated 33 outpatients with poorly controlled non-insulin dependent diabetes for six months with a high fibre diet and found a rise in fasting plasma glucose concentrations from 10.8 mmol/l to 12.6 mmol/l with no change in lipid concentrations.¹⁵ Deterioration in diabetic control was greatest in those who ate most fibre. In Sheffield 24 overweight patients in whom non-insulin dependent diabetes was newly diagnosed were given a low energy, high carbohydrate, low fat diet supplemented with either cereal fibre or guar gum.¹⁶ There were no differences between the groups in weight or control of blood glucose concentrations after 20 weeks, and the only measurable effect of the guar was to increase diarrhoea and flatulence.

Conclusions

Published work on high fibre diets is enormous and this review is obviously selective. Nevertheless, even if you accept that extremely high intakes of fibre (over 60 g/day) work under the conditions of metabolic wards in the short term, we doubt whether most ordinary middle aged people will eat such a diet as outpatients. In Nottingham, where we asked our volunteers to increase their intake of fibre with natural foods, the mean increase was 16 g/day and only two of the 33 patients managed to eat more than 50 g/day.¹⁵ In a follow up study in Oxford¹⁷ only 15 of the 22 study patients tried to continue a 60% carbohydrate high fibre diet, and after two years those who had made the effort averaged 41% of total energy as carbohydrate and 27 g dietary fibre a day, similar to what motivated outpatients in Nottingham managed after six months. It is hard to prevent an increased intake of energy with a high fibre, high carbohydrate diet without becoming vegetarian. Furthermore, even if diabetic patients want to make major increases in their fibre intake it is hard to explain which natural foods will be beneficial.

For example, only bread that contains intact wholegrains reduces the glycaemic response,¹⁸ and bread advertised as wholegrain may not contain the intact grains but merely flour milled from wholegrains. A similar problem arises with breakfast cereals advertised as being "high in bran or natural fibre." Most do not contain enough fibre, even of the right sort, to make any difference to diabetic control, and some contain the wrong sort of fibre.

To summarise, diets that contain a lot of beans may improve glycaemic control and reduce lipid concentrations in some diabetics, but how far these improvements are due to the fibre content is dubious.¹⁶ Furthermore, very high fibre diets (the only ones which may be effective) represent a revolutionary change in the eating habits of the average Briton and we suspect that many diabetics will see the inconvenience and socially unacceptable side effects of such diets as outweighing any rather nebulous longer term benefits. Since medicine began doctors have been liberal dispensers of conflicting nutritional advice and this phenomenon is currently at a peak. Many of the proffered recommendations are scientifically dubious or premature, confusing patients and reducing the credibility of the adviser. High fibre diets are good for the bowels but inappropriate for many middle aged diabetics. We agree with Nuttall that:

The almost religious fervour for imposing high carbohydrate, high-fibre diets not only on diabetic persons but on the population at large is premature... quotations from "experts" or "expert committees" are not a substitute for analysis of data. For many patients the quality of life associated with having eggs and bacon for breakfast rather than beans or a high-fibre cereal is worth a possible modest reduction in life expectancy... the impact of diet modification is uncertain but it is not likely to affect the vascular aging process significantly.¹⁶

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