Dietary fiber and carbohydrate metabolism[1,2]

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Relevant aspects of carbohydrate metabolism

We usually think of blood glucose as representing the body's carbohydrate metabolism and it is a useful index (1). The concentration at any point reflects the balance between entry of glucose into blood from the gut or from gluconeogenesis, principally in the liver, and removal by peripheral tissues for energy metabolism, storage as glycogen, or conversion to fat or, with hyperglycemia, removal via glycosuria. Conceivably, any of these events might be affected directly or indirectly by dietary fiber (**Fig 1**). Of them, only some of those in the gut are definite but current work on dietary fiber physiology is making others more likely.

Some regulators of blood glucose, hormonal, and other energy substrates, may be affected by dietary fiber. For example, insulin secretion may be modulated by gut hormones where release is in turn influenced by dietary fiber (2, 3). Volatile fatty acids (VFA) are products of dietary fiber metabolism by colonic microflora and reach significant concentrations in the splanchnic circulation (4–7). If energy balance is altered by dietary fiber, free fatty acid (FFA) flux may in turn be altered. These are questions deserving further investigation. More so since variables like serum insulin and plasma free fatty acids have predictive power for health outcomes in their own right (8).

Isolating dietary fiber effects

Several acute studies show an effect of some isolated dietary fiber types, such as pectin and guar, on blood glucose response to a carbohydrate load (9). Differences in acute blood glucose responses to foods high or low in dietary fiber may not be seen (10) or may be attributed to any of several factors (11–16): physical properties, such as particle size (3, 17, 18), viscosity (9), processing (19); or antinutrients, such as (1, 20) phytic acid, tannic acid, lectins, saponins, and enzyme inhibitors. In acute studies the effects of and interactions with the macronutrients carbohydrate, fat, and protein need to be taken into account (21, 22).

Perhaps one of the only long-term studies of the effect of increased dietary fiber intake on blood glucose in nondiabetics is that of Brodribb and Humphreys (23) who studied patients with diverticular disease for 6 mo. A lower blood glucose response to a load was seen. In diabetics, long-term, bran and guar improve (24), but pectin does not (25), blood glucose control.

With time, use of Anglo-Celtic style diets, high in carbohydrate and dietary fiber and low in fat (HCHFLFat), can improve insulin sensitivity where it is impaired by a decrease in fasting (basal) blood glucose (26–28). The effect is not always seen (29). Instead, improvement in overall blood glucose profiles during the day can be attributable to reduced incremental response to a meal (29). Part of this effect is attributable to an increased intake of absorbable carbohydrate over several days (30). Some of the effect may be attributable to reduced fat (31, 32). At least, where Italian-style diets are used, the improvement in incremental blood glucose response after 10 d can be attributed to dietary fiber (29). Assuming some of the effect on basal blood glucose is attributable to dietary fiber, after changing from a low-carbohydrate, low-fiber (LCLF) to high-carbohydrate, high fiber (HCHF) diet, up to an average of 14 d is required for the full effect to be seen (33) at least in type II diabetes. Simpson and Wahlqvist (unpublished observations) examined the question as to whether in type II diabetes after 3 wk on an Anglo-Celtic HCHF diet there is a differential blood

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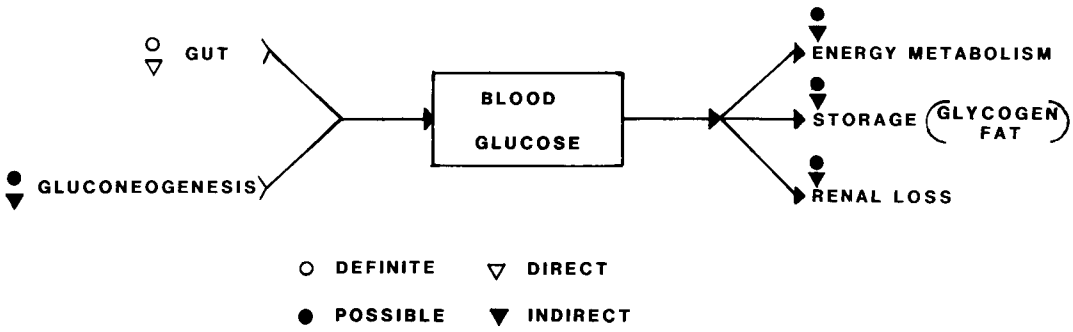

FIG 1. Sites of dietary fiber action on glucose flux.

glucose or plasma insulin response to a LCLF or HCHF test meal; there is not. Thus, an effect of dietary fiber from food may not be evident acutely or with all background diets.

Chronic administration of some fiber types (wheat bran or guar) to the experimental animal alters intestinal morphology (34), an effect which might contribute to reduced glucose absorption (35). Viscous fiber alters the activity of enzymes involved in luminal digestion (36). More work is required on the effects of dietary fiber on mucosal enzyme function.

Some methods of assay for dietary fiber (37–40) include resistant *starch* (41). The Englyst method (42) and its modification (43) allow an estimate of resistant starch to be made (44). The Association of Official Analytical Chemists (AOAC) method presumably includes most resistant starch in the figure for dietary fiber (45). Incomplete absorption of starch is now recognized to occur in a variety of circumstances in health or in ileostomy patients (46), with protein-starch and lipid-starch interactions (11), and with cooking (44) although higher temperature and pressure may make starch more bioavailable through disruption of starch granules (19). This phenomenon has to be taken into account when identifying effects of dietary fiber if nonstarch polysaccharide and lignin is what is meant.

Mechanisms of action

The first task when considering mechanisms of action of dietary fiber and carbohydrate metabolism is to distinguish between acute and longer-term effects (**Table 1**). Putative mechanisms can be broadly categorized into those operating in the gut (on transit, digestion, or absorption), those acting on gluconeogenesis, those acting on the regulation of blood glucose (be it hormonally or through other energy substrates), and those acting on peripheral glucose utilization (47, 48). Clearly, mechanisms outside the gut must be indirect. This systematic analysis allows possible mechanisms presently inadequately explored to form the basis of hypothesis development and testing. Much work remains to be done.

TABLE 1
Dietary fiber effects on carbohydrate metabolism*

Mechanisms	Acute	Longer-term
Gut		
Transit		
Upper gut	G	W
Colon	W	W
Digestion		
Luminal	G	W
Mucosal	?	?
Absorption	?	W
Gluconeogenesis		
Fasting	NA	W
Exercise	?	?
Regulation of blood glucose		
Hormonal		
Gut hormones	G	W
Insulin	G	W
Glucagon	W	W
Steroids	?	?
Other energy substrates		
Free fatty acids	?	W
Volatile fatty acids	F	W
Peripheral glucose utilization		
Energy metabolism	?	?
Storage	?	W
Renal loss	W	W

* Grades of evidence: G (good), F (Fair), W (Weak), ? (Don't know), and NA (not applicable).

The physical structure endowed on food by dietary fiber may alter access of luminal enzymes to food carbohydrate (1, 17). Activation or inhibition of gut enzymes involved in digestion of starch or simple sugars may be a consequence of food factors associated with dietary fiber (1). Effect on absorption may be a consequence of effects on gastric or small intestinal motility (1, 49) of dietary fiber-nutrient interactions (50) or changes in intestinal mucosal morphology (34). Yet another mechanism by which dietary fiber may have an effect on carbohydrate metabolism is through the generation of VFA by colonic microflora and the absorption of VFA into the splanchnic circulation where it may influence hepatic metabolism (4).

Dietary fiber types

Dietary fiber is chemically heterogeneous and not all dietary fiber types have similar effects on blood glucose response to a meal whether it be acutely (51) or long-term (29, 33, 52). In many ways a statement about food source of dietary fiber may be the most helpful in indicating the physiological or pathophysiological relevance of dietary fiber types. With current methods of dietary fiber analysis, it is possible to make this a consideration of dietary fiber chemistry (42, 43).

Leguminous vegetables are of particular interest insofar as carbohydrate metabolism is concerned. Their use is generally associated with lower blood glucose profiles in diabetics, both acutely (13, 53) and long-term (28). However, the exaggerated use of a particular food over another raises questions of the risk-benefit ratio, especially since legumes contain antinutrients (1, 20).

Caution is required in extrapolation of findings of the effects of parent foods (eg, wholemeal flour as bread) to more derivative products (eg, extruded foods) (54) where, at the least, the physicochemical properties have changed.

Initial work on carbohydrate metabolism with isolates of dietary fiber was with pectin and guar. Not nearly enough work has been done on the growing array of other commercial preparations of dietary fiber in the marketplace, used principally for management of bowel disorders, or of dietary fiber products used as thickeners in the food industry. Modified starches should also be investigated in relation to potential dietary fiber-like properties (55).

Subgroups of the population

The effects of dietary fiber are not the same in all subgroups of the population and this applies to carbohydrate metabolism as well as to other areas of physiology and pathophysiology. We know very little about children or about special effects in pregnancy or lactation. The prevalence of impaired glucose tolerance and of type II diabetes increases with advancing years and it can be expected that the role of dietary fiber will change with age. Ethnic food style may need to be taken into account since each food culture provides a different background total dietary-fiber intake and a variety of dietary-fiber types (28, 29).

The metabolic response to dietary-fiber-rich foods differs between healthy subjects, those with type I diabetes and those with type II diabetes (3, 21, 56). The alteration of dietary fiber intakes in diabetes should not focus only on the blood glucose outcome but should be mindful of the causes of premature death and excess morbidity in diabetes, principally atherosclerotic vascular disease, renal failure, and retinopathy. Much more work is required to evaluate the pathways connecting dietary fiber and these endpoints (57–60). Since impaired glucose tolerance is a risk factor for macrovascular disease, its potential for modification by altered dietary fiber intakes needs consideration in its own right.

Yet another consideration is the extent to which medication used in the management of carbohydrate disorders might interact with dietary fiber and the extent to which medication might alter the handling of dietary fiber.

Conclusions

Dietary-fiber isolates, used in amounts unlikely to be obtained from Western foods, can lower blood glucose and alter hormonal profiles (especially for insulin and gastrointestinal polypeptides) likely to influence carbohydrate metabolism. Effects are seen both acutely and in the longer term (days to weeks). Effects in healthy subjects are less impressive than in diabetic subjects.

Many studies examine the effects of high-carbohydrate, high-fiber diets on carbohydrate metabolism but few seek to identify a dietary-fiber effect independent of carbohydrates or other food components. When this is done, acutely, in healthy subjects and in type II diabetic subjects, differences in blood glucose responses to meals with and without fiber are negligible. Differences in insulin and gastrointestinal polypeptides responses may be of biological importance. Here we are talking about 12–22 g fiber from a variety of foods at a given meal. Most of the glycemic effect differences between foods is unlikely to be attributable to dietary fiber.

Longer term, in diabetic subjects a food dietary-fiber effect distinct from carbohydrate may operate on fasting (basal) blood glucose or the glucose response to a meal, possibly depending on the food culture examined (eg, Anglo-Australian vs Italian). Dietary-fiber intakes per day range from ~15–20 g/d to ~50 g/d. As far as dietary fiber isolates are concerned, effects are seen longer term with bran in nondiabetic and diabetic subjects, with guar in diabetic subjects, but not with pectin in diabetic subjects.

The mechanisms of dietary fiber action on carbohydrate metabolism may operate at various sites. With acute studies the upper gut is the most important site but with longer-term studies, metabolic changes in the upper gut may be important as well as the consequences of fermentation in the lower gut.

In the final analysis, we will always need to evaluate dietary fiber effects in food and meals. The effects on particular metabolic events should not be dissociated from overall health outcomes. Therapeutic use of dietary fiber will markedly distort the dietary fiber chemistry profile usually obtained from food. Such use presents the challenge of evaluating risk-benefit ratios as with other therapeutic modalities.

References

1. Jenkins DA, Jenkins AL, Wolever TMS, Thompson LH, Rao RV. Simple and complex carbohydrates. Nutr Rev 1986;44:44–9.
2. Collier G, O'Dea K. Effect of physical form of carbohydrate on the postprandial glucose, insulin and gastric inhibitory polypeptide responses in type 2 diabetes. Am J Clin Nutr 1982;36:10–4.
3. Simpson RW, McDonald J, Wahlqvist ML, Atley L, Outch K. Food physical factors have different metabolic effects in non-diabetics and diabetics. Am J Clin Nutr 1985;42:462–9.
4. Topping DL, Illman RJ. Bacterial fermentation in the human large bowel: time to change from the roughage model of dietary fibre? Med J Aust 1986;144:307–9.
5. Topping DL, Illman RJ, Taylor MN, McIntosh GH. The effects of wheat bran and porridge oats on hepatic portal venous volatile fatty acids in the pig. Ann Nutr Metab 1985;29:325–31.
6. Cummings JH, Englyst HN, Wiggins HS. The role of carbohydrates in lower gut function. Nutr Rev 1986;44:50–4.
7. Pomare EW, Branch WJ, Cummings JH. Carbohydrate fermentation in the human colon and its relation to acetate concentrations in venous blood. J Clin Invest 1985;75:1448–54.
8. Wahlqvist ML, Lo CS, Myers K, Simpson RW. Plasma insulin and free fatty acids as risk factors for arterial compliance in type-2 diabetes. Recent Adv Clin Nutr 1986;2:330–2.
9. Jenkins DJ, Wolever TM, Leeds AR, et al. Dietary fibres, fibre analogues, and glucose tolerance: importance of viscosity. Br Med J 1978;1:1392–4.
10. Simpson RW, McDonald J, Wahlqvist M, Balasz N, Dunlop M. Effect of naturally occurring dietary fibre in Western foods on blood glucose. Aust NZ J Med 1981;11:484–7.
11. Thornburn AW, Brand JC, Truswell AS. The glycaemic index of foods. Med J Aust 1986;144:580–2.
12. Fitz-Henry AW, Brand JC. Rate of digestion and absorption of Australian Aboriginal starchy bushfoods. Proc Nutr Soc Aust 1982;7:214(abstr).
13. Jenkins DJA, Wolever TMS, Jenkins AL, et al. The glycaemic index of foods tested in diabetic patients: a new basis for carbohydrate exchange favouring use of legumes. Diabetologia 1983;24:257–64.
14. Jenkins DJA, Ghafari H, Wolever TMS, et al. Relationship between rate of digestion of foods and post-prandial glycaemia. Diabetologia 1982;22:450–5.
15. Jenkins DJA. Lente carbohydrate: a newer approach to the dietary management of diabetes. Diabetes Care 1982;5:634–41.
16. Wahlqvist ML, Wilmshurst EG, Murton CR, Richardson EN. The effect of chain length on glucose absorption and the related metabolic response. Am J Clin Nutr 1978;31:1998–2001.
17. O'Dea K, Nestel PJ, Antonoff L. Physical factors influencing post-prandial glucose and insulin responses to starch. Am J Clin Nutr 1980;33:760–5.
18. Haber GB, Heaton KW, Murphy D, Burroughs LF. Depletion and disruption of dietary fibre: effects on satiety, plasma glucose and serum insulin. Lancet 1977;ii:679–82.
19. Brand JC, Nicholson PL, Thorburn AW, Truswell AS. Food processing and the glycemic index. Am J Clin Nutr 1985;42:1192–6.
20. Nachbar MS, Oppenheim JD. Lectins in the United States diet: a survey of lectins in commonly consumed foods and a review of the literature. Am J Clin Nutr 1980;33:2338–45.
21. Simpson RW, McDonald J, Wahlqvist ML, Atley L, Outch K. Macronutrients factors have different metabolic effects in non-diabetics and diabetics. Am J Clin Nutr 1985;449–53.
22. Collier G, McLean A, O'Dea K. The effect of coinges-

tion of fat on the metabolic responses to slowly and rapidly absorbed carbohydrates. Diabetologia 1984;26: 50–4.
23. Brodribb AJM, Humphreys DM. Metabolic effect of bran in patients with diverticular disease. Brit Med J 1976;1:428–30.
24. Frølich W, Aaseth J, Ødegaard B, Aagenaes Ø. Improvement in long-term diabetic control after high fibre (bran and guar) diets. Diabetologia 1983;25:200.
25. Gardner DF, Schwartz RN, Krista MS, Merimee TJ. Dietary pectin and glycemic control in diabetes. Diabetes Care 1984;7:143–5.
26. Simpson RW, Mann JI, Eaton J, Moore RA, Carter R, Hockaday TDR. Improved glucose control in maturity onset diabetes treated with high carbohydrate-modified fat diet. Br Med J 1979;i:1753–6.
27. Simpson RW, Mann JI, Eaton J. High-carbohydrate diets and insulin dependent diabetics. Br Med J 1979;ii:523–5.
28. Simpson HCR, Simpson RW, Lousley S, et al. A high carbohydrate leguminous fibre diet improves all aspects of diabetic control. Lancet 1981;i:1–4.
29. Riccardi G, Rivellese A, Pacione D, Genovese S, Mastranzo P, Mancini M. Separate influence of dietary carbohydrate and fibre on the metabolic control in diabetes. Diabetologia 1984;26:116–21.
30. Wilkerson HLC, Hyman H, Kaufman M, McCuiston AC, Francis J. Diagnostic evaluation of oral glucose tolerance tests in non-diabetic subjects after various levels of carbohydrate intake. N Engl J Med 1960;262: 1047–53.
31. Chisholm KW, O'Dea K. A low fat diet improves metabolic control in insulin-deficient rats. Proc Nutr Soc Aust 1984;9:119(abstr).
32. O'Dea K. Marked improvement in carbohydrate and lipid metabolism in diabetic Australian Aborigines after temporary revision to traditional lifestyle. Diabetes 1984;33:596–603.
33. Simpson RW, McDonald J, Wahlqvist ML, Balasz N, Sissons M, Atley L. Temporal study of metabolic change when noninsulin-dependent diabetics changed from a low- to high-carbohydrate fiber diet. Am J Clin Nutr (in press).
34. Dryden PA, Burcher E, Read RSD, Gollan RA. Effect of bran on histology of rat intestine. Proc Nutr Soc Aust 1982;7:215(abstr).
35. Dryden PA, Read RSD, Kestin M, Jones GP, Burcher E, McLennan E. The effect of chronic ingestion of dietary fibre on the rate of absorption of glucose in rats. Proc Nutr Soc Aust 1981;6:160(abstr).
36. Isaksson G, Lundquist I, Ihse I. Effect of dietary fiber on pancreatic enzyme activity in vitro: importance of viscosity, pH, ionic strength, absorption and time of incubation. Gastroenterology 1982;82:918–24.
37. Southgate DAT. The determination of food carbohydrates. London, UK: Applied Science Publishers, 1976.
38. Southgate DAT. The analysis of dietary fibre. In: Spiller GA, ed. Fibre in human nutrition. New York, NY: Plenum Press, 1976:73–102.
39. Southgate DAT, White MA. Commentary on the results obtained by the different laboratories using the Southgate method. In: James WPT, Theander O, eds. The analysis of dietary fibre in food. New York, NY: Marcel Dekker, 1981;37–70.
40. Southgate DAT. Determination of carbohydrates in foods. II Unavailable carbohydrates. J Sci Food Agric 1969;20:331.
41. Eastwood MA. What does the measurement of dietary fibre mean? Lancet 1986;i:1487–8.
42. Englyst HN, Wiggins HS, Cummings JH. Determination of the nonstarch polysaccharides in plant foods by gas-liquid chromatography of constituent sugars as alditol acetates. Analyst 1982;107:307–18.
43. Jones GP, Briggs DR, Wahlqvist ML, Flentje LM. Dietary fibre content of Australian foods. 1 Potatoes. Food Technol Aust 1985;37:81–3.
44. Jones GP, Briggs DR, Wahlqvist ML, Flentje LM. The effect of retrograded starch on the determination of dietary fibre in food. Proc Nutr Soc Aust 1982;7: 162(abstr).
45. Association of Official Analytical Chemists. Total dietary fiber in foods. J Assoc Off Anal Chem 1985;68: 399.
46. Englyst HN, Cummings JH. Digestion of the polysaccharides of some cereal foods in the human small intestine. Am J Clin Nutr 1985;42:778–87.
47. Mann JI, Diabetes mellitus: nutritional aspects of etiology and management. Recent Adv Clin Nutr 1986;2:255–70.
48. Andersson AH, Asp N-G, Hallmans G. Kost och diabetes. Naringsforksning 1986;30:78–89.
49. Cooke AR. Control or gastric emptying and motility. Gastroenterology 1975;68:804–16.
50. Stafford H, Jones GP, Wahlqvist ML, Read RSD. Measurement of mouth to cecum transit time. Recent Adv Clin Nutr 1986;2:336–8.
51. Wahlqvist ML, Moris MJ, Littlejohn GO, Bond A, Jackson RVJ. The effects of dietary fibre on glucose tolerance in healthy males. Aust NZ J Med 1979;9: 154–8.
52. Jenkins DJA, Hockaday TDR, Howarth R, et al. Treatment of diabetes with guar gum: reduction of urinary loss in diabetes. Lancet 1971;ii:779–80.
53. Thorne MJ, Thompson LU, Jenkins DJA. Factors affecting starch digestibility and the glycemic response with special reference to legumes. Am J Clin Nutr 1983;38:481–8.
54. Bjorck I, Asp N-G, Birkhed D, Lundquist I. Effects of processing of availability of starch for digestion in vitro and in vivo. 1 Extrusion cooking of wheat flours and starch. J Cereal Sci 1984;2:91–103.
55. Wurzburg OB. Nutritional aspects and safety of modified food starches. Nutr Rev 1986;44:74–9.
56. Jenkins DJA, Wolever TMS, Wong GS, et al. Glycemic responses to foods: possible differences between insulin-dependent and non-insulin-dependent diabetes. Am J Clin Nutr 1984;40:971–81.
57. Hughes RE. A new look at dietary fibre. Hum Nutr Clin Nutr 1986;40C:81–6.
58. Lewis B, Mann JI, Mancini M. Reducing the risks of coronary heart disease in individuals and in the population. Lancet 1986;i:956–9.
59. Lean MEJ, James WPT. Prescription of diabetic diets in the 1980s. Lancet 1986;i:723–5.
60. Fisher N, Berry CS, Fearn T, Gregory JA, Hardy J. Cereal dietary fiber consumption and diverticular disease: a lifespan study in rats. Am J Clin Nutr 1985;42: 788–804.