

Special Article

DIET IN THE TREATMENT OF DIABETES*

MARK L. WAHLQVIST

Med. J. Aust., 1980, 2: 64-68.

THERE ARE basically three ways in which diet could lead to the manifestation of diabetes mellitus. One would be by damage

Professor M. L. Wahlqvist, B.Med.Sc., M.D. (Adelaide), M.D. (Uppsala), F.R.A.C.P., is Professor of Human Nutrition at Deakin University, and Consultant Physician, Prince Henry's Hospital, Melbourne.

* Based on the paper delivered at the Centenary Scientific Meeting of the Australian Medical Association (Victorian Branch), Melbourne, October 6, 1979.

Address for reprints: Professor M. L. Wahlqvist, Section of Human Nutrition, Deakin University, Geelong, Vic. 3217.

to the pancreatic beta-cells which manufacture insulin. Malnourished individuals who eat foods containing excessive amounts of cyanogenetic glycoside, such as cassava, may be so affected.¹ Theoretically, toxins, such as streptozotocin, produced by soil microorganisms, could also play a role. However, in most cases of insulin-deficient diabetes seen in affluent societies, a genetic predisposition signified by HLA phenotype and an infective viral insult²⁻³ seem the most likely pathogenetic factors. Nevertheless, alcohol abuse with pancreatic

damage is of growing importance as an aetiological consideration in affluent societies. Second, the rate or extent of absorption of glucose, and the related insulin response may be modified by diet. Third, the advent of obesity allows the expression of maturity-onset diabetes in those who are genetically predisposed.⁴⁻⁵

The management of the diabetic individual and especially, from a preventive point of view, of the family in which diabetes has been identified, requires attention to these dietary pathogenetic

factors. Since maturity-onset diabetes is the most prevalent form of diabetes in Australia, avoidance of energy intake which exceeds energy needs is the most important preventive measure.

TOWARDS A HIGH CARBOHYDRATE DIET

More than is generally recognized, the contribution of carbohydrate to energy in the diabetic diet resembles that in the diet of the population at large. Thus, in Japan, it is an average of about 70% and, in Australia, about 40%.⁶ There has been no evidence that those on the high carbohydrate diets fare less well. Indeed, when dietary carbohydrate is restricted, even in healthy subjects, glucose tolerance may be impaired.⁷ Epidemiological studies, such as the eleven populations study of West and Kalbfleisch⁸ indicate that diabetes is less prevalent in populations whose total carbohydrate intake is higher.

Where insulin response to dietary carbohydrate is deficient, or glucose disposal is impaired, the even distribution of carbohydrate intake throughout the day ought to be recommended. However, as newer insulin delivery systems (artificial pancreas) become available, even this advice may be unnecessary.

The "slowness" or "fastness" of carbohydrate absorption is not conferred by the chain length of the polymer.⁹ That is to say, digestion is not rate-limiting, given normal exocrine pancreatic and mucosal brush-border enzyme activity.

One important consequence of a high carbohydrate diet is that, isoenergetically, the proportion of dietary fat intake will be lower.

TOWARDS A HIGH FIBRE DIET

Population observations by Trowell indicate that diabetes is less prevalent in those populations who consume a high-fibre diet.¹⁰

Studies with different fibre sources and types, such as bran,^{11 12 13} pectin, and guar,^{12 13} show that in healthy subjects and in diabetics, the postprandial rise in blood glucose concentration and the insulin response (where present) are less pronounced when fibre is included in the diet. One implication of such studies is that, for a given genetic predisposition and level of obesity, maturity-onset diabetes will not be as evident with a high fibre diet.

In the present management of diabetes, one of the difficult aspects of carbohydrate control is postprandial hyperglycaemia. An increase in the intake of dietary fibre is likely to be a significant contribution to this aspect of diabetic control. It would also appear that the ingestion of dietary fibres of certain types may contribute to the lipid-lowering potential of a diet.^{14 15}

This new dimension means greater flexibility in diabetic dietary management, and greater acceptability to diabetics. Items such as wholemeal bread, fruit, potatoes

and peas will find a revised place in diabetic diets. There is now a need to examine the impact of intact foods on diabetic control, rather than be limited to predictions on the basis of food composition tables in respect of absorbable carbohydrates. Indeed, there may well be factors in addition to fibre, such as amylase inhibitors,¹⁶ and glucose tolerance factor,¹⁷ which may modify blood glucose concentration. An emphasis on whole-grain cereals, fruit, and vegetables in diabetic diets can be expected to raise the daily fibre intake from present low levels of less than 15 g to more than 30 g.¹⁸ Such an approach also allows a variety of dietary fibres to be ingested, each with potentially different modes of action.

TOWARDS A LOW-FAT DIET

Diabetics are prone to atherosclerotic cardiovascular disease in which a principal risk factor is hyperlipoproteinaemia.^{19 20} In a Melbourne diabetic clinic, insulin-dependent diabetics who had already been given dietary advice, had a 21% prevalence of hypercholesterolaemia (>6.2 mmol/L) and a 38% prevalence of hypertriglyceridaemia (>2.0 mmol/L).²¹ In general, when carbohydrate control is improved, lipid control is improved.²² However, in a high-risk group, it becomes relatively more important to correct any residual lipid abnormality. In this situation, a diet which is low in the contribution of saturated fat to energy intake (for example, 10%), with a polyunsaturated to saturated fat ratio (P/S ratio) of 1.0 to 1.5, and a daily cholesterol intake down to as near to 300 mg as practical, is recommended. These dietary recommendations are more easily accommodated with a high carbohydrate diet. The effect of carbohydrate to induce hypertriglyceridaemia is, if it occurs, temporary.²³

DIABETES AND THE AUSTRALIAN DIETARY GUIDELINES

A working party on nutrition policy for Australia, set up under the auspices of the Australian Association of Dietitians, presented its "Dietary Guidelines for Australians" at the Fourth Scientific Meeting of the Nutrition Society of Australia, in Perth in August 1979. The committee included representatives of the medical profession, the dietetic profession, the Australian Department of Health, the food industry, and the two Australian Professors of Human Nutrition. It had inputs from consumers. Its guidelines were as follows:

1. Eat a variety of foods each day.
2. Encourage breast feeding.
3. Prevent and control obesity.
4. Decrease total fat intake.
5. Decrease consumption of sucrose.
6. Limit alcohol consumption.
7. Increase consumption of complex carbohydrate and dietary fibre.
8. Reduce sodium intake.
9. Encourage water intake.

Ideally, such guidelines ought to be relevant to diabetics as well as to non-

diabetics. It will be seen that Points 1, 3, 4, 5 and 7 correspond with the general nutritional approach for diabetes presented above. In respect of alcohol consumption, a modest amount (10 g to 20 g a day) is compatible with good diabetic control, but an excessive amount may be associated with excessive energy intake, interference with therapy with orally administered agents or with insulin, declining pancreatic beta-cell function, and hepatic cirrhosis with associated impairment of glucose tolerance.²⁴ Moderation in sodium intake for diabetics who are prone to hypertension would seem to be wise dietary counsel. To encourage water intake is to displace other energy-dense beverages, to improve oral hygiene, and to provide a sometimes forgotten alternative to alcohol. As fibre intake increases, water intake probably should also increase since, in some respects (as in gel formation) its function is water dependent.

NUTRITIONAL PRIORITIES IN DIABETIC MANAGEMENT

Each diabetic needs to have his nutritional priorities arranged and these include (i) weight control; (ii) carbohydrate control; (iii) lipid control; and (iv) social function of food.

A first priority for overweight diabetics with the maturity-onset disease is weight control.

REFERENCES

1. McMILLAN, D. E., and GEEVARGHESE, P. J., Dietary cyanide and tropical malnutrition diabetes, *Diabet. Care*, 1979, 2: 202.
2. DRASH, A. L., The etiology of diabetes mellitus, *New Engl. J. Med.*, 1979, 300: 1211.
3. YOON, J.-W., AUSTIN, M., ONODERA, T., and NOTKINS, A. L., Virus-induced diabetes mellitus: Isolation of a virus from the pancreas of a child with diabetic ketoacidosis, *New Engl. J. Med.*, 1979, 300: 1173.
4. WESTLUNG, K., and NICOLAYSEN, R., Ten year mortality and morbidity, related to serum cholesterol: A follow up of 3751 men aged 40-49, *Scand. J. clin. Lab. Invest.*, 1972, 30 (Suppl. 127): 1.
5. ZIMMET, P., Epidemiology of diabetes and its macrovascular manifestations in Pacific population: Medical effects of social progress, *Diabetes Care*, 1979, 2: 144.
6. WAHLQVIST, M. L., McDONALD, J., and FLINT, D. M., Diet and diabetes mellitus, *Food Nutr. Notes Rev.*, 1979, 36: 180.
7. WILKERSON, H. L. C., HYMAN, H., KAUFMAN, M., et alii, Diagnostic evaluation of oral glucose tolerance tests in non-diabetic subjects after various levels of carbohydrate intake, *New Engl. J. Med.*, 1960, 262: 1047.
8. WEST, K. M., and KALBFLEISCH, J. M., Influence of nutritional factors on prevalence of diabetes, *Diabetes*, 1971, 20: 99.
9. WAHLQVIST, M. L., WILMSHURST, E. G., MURTON, C. R., and RICHARDSON, E. N., The effect of chain length on glucose absorption and the related metabolic response, *Amer. J. clin. Nutr.*, 1978, 31: 1978.
10. TROWELL, H., Diabetes mellitus and dietary fiber of starch foods, *Amer. J. clin. Nutr.*, 1978, 31: S53.
11. BRODRIBB, A. J. M., and HUMPHREYS, D. M., Metabolic effect of bran in patients with diverticular disease, *Brit. med. J.*, 1976, 1: 428.
12. JENKINS, D. J. A., WOLEVER, T. M. S., LEEDS, A. R., et alii, Dietary fibres, fibre analogues, and glucose tolerance: Importance of viscosity, *Brit. med. J.*, 1978, 1: 1392.
13. WAHLQVIST, M. L., MORRIS, M. J., LITTLEJOHN, G. O., et alii, The effects of dietary fibre on glucose tolerance in healthy males, *Aust. N.Z. J. Med.*, 1979, 9: 154.

- ¹⁴ ANDERSON, J. W., and CHEN, W. L., Plant fibre: Carbohydrate and lipid metabolism, *Amer. J. clin. Nutr.*, 1979, 32: 346.
- ¹⁵ ANDERSON, J. W., MIDGELEY, W. R., and WEDMAN, B., Fiber and diabetes, *Diabet. Care*, 1979, 2: 369.
- ¹⁶ WALTON, R. J., SHERIF, I. T., NOY, G. A., and ALBERTI, K. G. M. M., Improved metabolic profiles in insulin-treated diabetic patients given alpha-glucosidase-hydrolase inhibitor, *Brit. med. J.*, 1979, 1: 220.
- ¹⁷ MERTZ, W., Effects and metabolism of glucose tolerance factor, in *Nutritional Reviews: Present Knowledge in Nutrition*, 4th ed., Hegsted, D. M., Chichester, C. O., Darby, W. J., *et alii* (eds), The Nutrition Foundation, Washington, 1976: p. 365.
- ¹⁸ *McCance and Widdowson's The Composition of Foods*, 4th ed., revised by A. A. Paul and D. A. T. Southgate, Medical Research Council's Special Report No. 297, Her Majesty's Stationery Office, London, 1978.
- ¹⁹ JARRETT, J., Diabetes and the heart: Coronary heart disease, *Clin. Endocr. Metab.*, 1977, 6: 389.
- ²⁰ KANNEL, W. B., and MCGEE, D. L., Diabetes and glucose tolerance as risk factors for cardiovascular disease: The Framingham study, *Diabet. Care*, 1979, 2: 120.
- ²¹ WAHLQVIST, M. L., ENNIS, G. C., and LORDING, D. W., Unpublished data.
- ²² COURT, J. M., DUNLOP, M., and HILL, M., A study of plasma-lipid concentrations in diabetic children, *J. hum. Nutr.*, 1978, 32: 285.
- ²³ ANTONIS, A., and BERSHON, I., The influence of diet on serum triglycerides in South Africa white and Bantu prisoners, *Lancet*, 1961, 1: 3.
- ²⁴ SAMAN, N. A., STONE, D. B., and ECKHARDT, R. D., Serum glucose, insulin and growth hormone in chronic hepatic cirrhosis, *Arch. Intern. Med.*, 1969, 124: 149.