Diet and diabetes mellitus

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Diet as a cause

Although glucose tolerance and blood glucose may well be modified by diet, a primary aetiological role for diet in the production of diabetes seems unlikely. In the case of pancreatic β cell failure in juvenile onset diabetes (JOD), a combination of genetic predisposition, manifest by tissue type and processes injurious to the β cells, probably infective and autoimmune, seem important.[1,2]

In maturity onset diabetes (MOD), inheritance is a strong determinant [3], although not usually evident unless there is excessive energy intake and concomitant obesity (body weight greater than ideal by 20 per cent or more) (Table 1 and Fig. 1 p.183).[4,5,6] In a Norwegian prospective study, there was a close correlation between the emergence of diabetes and overweight from as little as 10 per cent excess.[7] Obesity seems to increase resistance to insulin.[8] Avoidance of obesity in families or ethnic groups with a genetic predisposition to MOD is, therefore, of considerable importance from a preventive point of view. In Australia, the prevalence of obesity is up to 25 per cent in older age groups.[9] The prevalence of all forms of diabetes is about 2–3 per cent[10,11,12] and MOD is about ten times more common than JOD. Thus there is much scope for prevention of diabetes through weight control.

Table 1 Dietary aetiology of diabetes

1. Excessive energy intake
2. Severe malnutrition
3. Alcohol abuse
4. Iron overload
5. Food toxicants

Other less common dietary factors are alcohol abuse and iron overload which can both lead to pancreatic β cell destruction and reduced insulin secretion and also to hepatic cirrhosis with associated impairment of glucose tolerance. Food toxins may also contribute to β cell dysfunction.[13] Streptozotocin, produced by strains of the branching micro-organisms Streptomyces usually found in soil, or other toxic products of microflora could conceivably damage pancreatic islet cells if present in food in sufficient quantity.

Dietary management—present practice

For many years, diets where 40 per cent of energy is derived from carbohydrate have been recommended to diabetics in Australia. This is not unlike what has applied in Britain[14] and the U.S.A..[15,16] Clinicians have multiplied the number of grams of carbohydrate by 10 to give the energy intake in Calories. The low level of patient adherence to such diets in the U.S.A. has been documented.[17] Likewise we found that amongst a group of diabetic children in Victoria and diabetics attending a Melbourne teaching hospital clinic, who had been recommended diets, presumptively 40 per cent in energy from carbohydrate, carbohydrate actually provided between 20 and 60 per cent of energy intake (Table 2 and Fig. 2 p. 183). Information was obtained for children by a two day (one week day and one weekend day) dietary record and for adults by dietary history. There are implications here both for energy balance and for adherance to carbohydrate modification.

Despite recommendations to the contrary[18], an 'anti-carbohydrate' attitude persists amongst diabetics, dietitians and doctors. In part this derives from the fact that, for insulin dependent diabetics (IDD), prior to insulin availability, carbohydrate restriction was one of the only ways to control symptoms related to osmotic diuresis. It is now appreciated that insulin homeostasis relates more to total fuel requirements than to carbohydrate in particular and, where insulin is available, it is energy balance that matters more.

The 'anti-carbohydrate' attitude also derives from a failure to distinguish between refined and unrefined carbohydrate. Much of the case against sucrose is presumably a case against excessive energy intake and refined carbohydrate in general. There was a misconception, for example, that simple sugars were absorbed more quickly than starches, because of shorter chain length. This is not the case.[18] It is the form in which the starch is found and/or the accompanying non-absorbable carbohydrate (NAC) which modifies its absorption.[19–22] In the younger diabetics whose diet we studied, the more carbohydrate contributed to energy intake, the more it was unrefined (Fig. 3, p.183). In this sense, these diabetics were disadvantaged by their higher

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Table 2 Dietary patterns of insulin dependent diabetics

	Age	Number	Male/ Female	Energy intake as carbohydrate Range	< 40%	⩾ 40%
					%	%
Younger	7–15	14	9/5	27–59	29	71
Older	16–73	63	31/32	21–56	49	51

carbohydrate intake. We consider it reasonable to aim at a two-fold increase in the average dietary fibre intake for younger diabetics (Fig. 4, p. 183).

One dietary carbohydrate recommendation for diabetics, of whatever type, on which most concur is that the carbohydrate intake should be distributed evenly through the day. Where oral agents or insulin are to be used, their administrations are tailored accordingly.

A diet high in unrefined carbohydrate

High carbohydrate diets are frequently found in societies with little diabetes and little ischaemic heart disease.[23,24] They were characteristic of man's earliest diet, that of most hunter-gatherers.[24,25] The carbohydrate was, however, unrefined, which is to say it was both absorbable and non-absorbable. Non-absorbable carbohydrate (NAC) and the non-carbohydrate lignin together constitute dietary fibre. Diets high in unrefined carbohydrate either do not lead to any deterioration[26] or may actually improve[27,28] carbohydrate control in MOD. Both the increased carbohydrate and its unrefined nature may have significance for the management of diabetes.

There is not universal agreement that an increase in dietary absorbable carbohydrate is a good thing[29-31], but studies that indicate that carbohydrate restriction may be an advantage do not usually exclude the effect of other variables such as weight reduction or the change to an even distribution of carbohydrate.[29-31]

Studies where unrefined carbohydrate in the diet was increased indicate that not only the post-prandial rise in blood glucose[18,19], but also the fasting blood glucose might be lower on such regimes.[28,32,33]

There are several ways in which unrefined carbohydrate might favourably influence blood glucose (Table 3). One is that larger particles (K.O'Dea, personal communication) may be less digestible. Another way, which appears relevant to the gum guar, is that increased viscosity may slow absorption.[34] Non-absorbable carbohydrate may retain otherwise absorbable carbohydrate. Also, amylase inhibitors may be present in the NAC.[35,36] The sucrase and glucoamylase inhibitor BAYg 5421 have been shown to reduce post-prandial blood glucose.[37]

Table 3 Dietary fibre
—Ways to affect blood glucose

1. Digestibility
2. Viscosity
3. Retention of absorbable carbohydrate
4. Amylase inhibitors

Table 4 Favourable effects of increased unrefined carbohydrate for diabetics

1. Lower fasting and post-prandial glucose
2. Less reactive hypoglycaemia
3. Earlier satiety
4. Less saturated fat
5. Oral hygiene

Another advantage for adequate NAC intakes might be that the likelihood of reactive hypoglycaemia is reduced[20] and this may reduce an added stimulus to appetite (Table 4).

One of the most welcome features of the 'rediscovery' of unrefined carbohydrates for diabetics must be the reduced dependency on special foods with their costs and limited availability.

Role of sweeteners

The pleasurable aspects of food should not be forgotten. Wherever possible, it seems man makes food sweet and this is a special characteristic of urban society. Sucrose is the usual sweetener. It has been ascribed a reference sweetness of 1.0.[38] It is exceeded in sweetness by fructose and xylitol. Glucose, lactose, maltose and sorbitol are less sweet (Table 5). Each of these sweeteners is nutritive and adds to energy intake. The disaccharides lactose and maltose generate glucose. Fructose, xylitol and sorbitol are converted to triose phosphates which can in turn be converted into glucose—6—phosphate and thence to glucose.

Table 5 Sweetness

Less sweet	*'1.0'*	*More sweet*
Glucose Lactose Maltose Sorbitol	Sucrose	Fructose Xylitol

Table 6 Alcohol and diabetes

Unfavourable effects	*Favourable effects*
Pancreatic damage Cirrhosis impairs GT High energy value Hypoglycaemia Oral agent interaction Other organic effects	Social function Increase in HDL

Table 7 Older insulin dependent diabetics *—Alcohol intake*

	%
Frequency of users	29
Frequency of abusers (> 80 g/day)	2
Energy contribution for users	2 (1-9)

Non-nutritive sweeteners, saccharine and cyclamate, make life more acceptable for diabetics. Theoretically, they contribute to reduced energy intake, but the subtleties of their effect on appetite need to be investigated. Doubt has arisen about their use on account of their potential as bladder carcinogens.[39] However, there is no good evidence that diabetics who have used them have an increased incidence of bladder cancer.[40]

Management of hyperlipidaemia

Cardiovascular disease is the leading cause of death amongst diabetics and it is relatively more important than in non-diabetics.[41] Elevated serum cholesterol[42] and triglyceride[43, 44] and decreased high density lipoprotein (HDL)[45] concentrations are risk factors for atherosclerotic vascular disease.

It has been suggested that the low carbohydrate, high fat diet which diabetics have been prescribed in the past may have contributed to hyperlipidaemia and excess cardio vascular mortality in this group.[42] There is no evidence that, long term,[46] high carbohydrate diets increase serum triglycerides in diabetics.[15, 26, 28, 47] Serum cholesterol in diabetics is lowered by diets high in unrefined carbohydrate.[26, 28, 47] This is probably mainly because of the reduction in saturated fat and cholesterol intake that accompanies such diets. In our own study subjects, there is much to be gained by reduction in fat intake through an increase in unrefined carbohydrate intake (Fig. 5, p.184).

Generally speaking, good lipid control parallels good carbohydrate control.[48] Studies of the relationship between plasma lipids, including HDL, and the glycosalated haemoglobin HbA1c concentrations in blood, now becoming available, tend to support this view. HbA1c is of value in such assessments because it reflects the prevailing blood glucose over several weeks.[4]

Alcohol and diabetes

Alcohol may be an aetiological dietary factor in diabetes mellitus through excessive energy intake, pancreatitis, hepatic cirrhosis or the development of haemochromatosis (Table 1).

Alcohol is also important to consider in the dietary management of those who already have diabetes (Table 6). In our older insulin dependent diabetics, 29% were users of alcohol and 2% were abusers (on intakes of more than 80 g ethanol/day) (Table 7). In the users, the mean energy contribution of alcohol was 2% (range 1-9%) (Table 7). It is of interest that the majority of diabetics claimed not to use alcohol at all. This may have implications for the quality of their social lives. Provided modest amounts are imbibed and the energy and carbohydrate intakes are accounted for, alcohol is not contraindicated in diabetics on diet alone or those on insulin. Problems do arise with the interaction between alcohol and oral agents, the sulphonylureas.

Glucose tolerance factor

Glucose tolerance factor (GTF) is a chromium-nicotinic acid complex.[49] It can apparently be synthesised to a variable extent by man, possibly in liver.[49] Its richest dietary source is brewer's yeast. GTF appears to potentiate insulin activity. It is of interest that chromium concentrations in blood rise when insulin rises.[49] The existence of this factor and a hypoglycaemic factor (hypoglycin), in the akee fruit *(Blighia Sapida)* raise the possibility that other blood glucose modifying agents might be found in food.

Diet in special situations

Every effort must be made for diabetics to eat the way that non-diabetics, following a prudent diet, might eat. To do this, special situations must be anticipated.

Eating out and entertaining at home can be achieved by the diabetic without recourse to

Figure 1 Development of maturity onset diabetes mellitus.

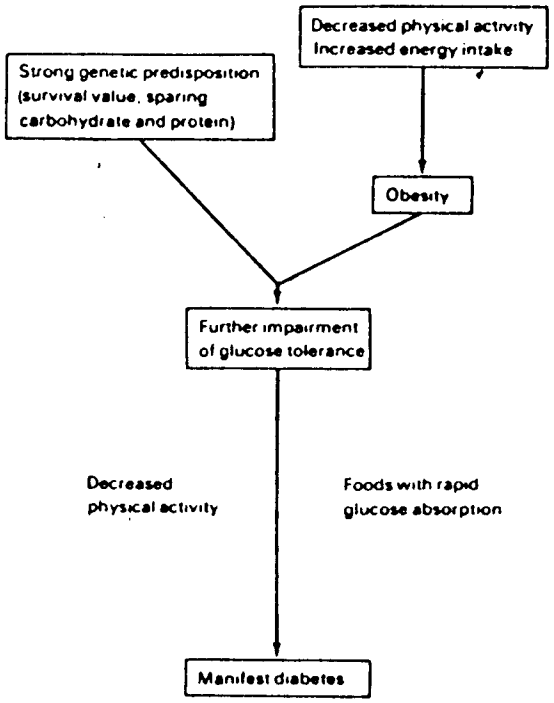


Figure 2 Average proportions of energy derived from carbohydrate for Australians in general (estimated from dietetic practice), older insulin-dependent diabetics (Adult IDD), younger insulin-dependent diabetics (children IDD) and the target intake.

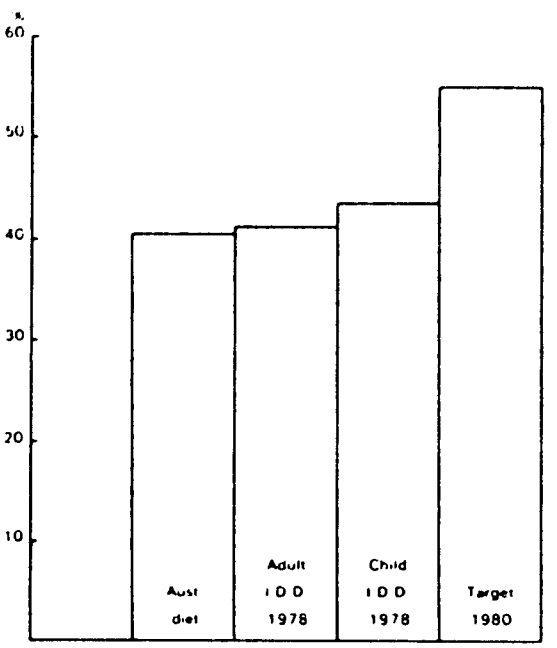


Figure 3 Relationship between the absorbable carbohydrate: non-absorbable carbohydrate (dietary fibre) ratio and proportion of energy derived from carbohydrate in a group of younger insulin-dependent diabetics resident in Victoria, Australia in 1978. 'r' is the correlation coefficient, 'n' the number of subjects and 'p' the level of significance.

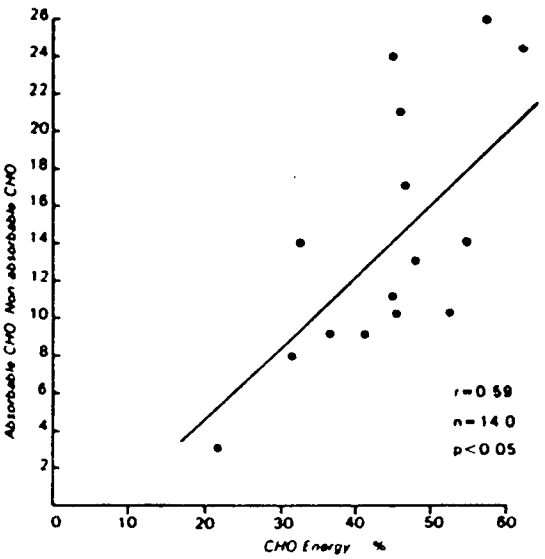


Figure 4 Estimated average dietary fibre intakes for Australians in general, older insulin-dependent diabetics (adult IDD) and younger insulin-dependent diabetics (child IDD) and a possible desirable target intake (dietary fibre values based on McCance & Widdowson's The Composition of Foods, *revised by A. A. Paul and D. A. T. Southgate, H.M.S.O., London, 1978).*

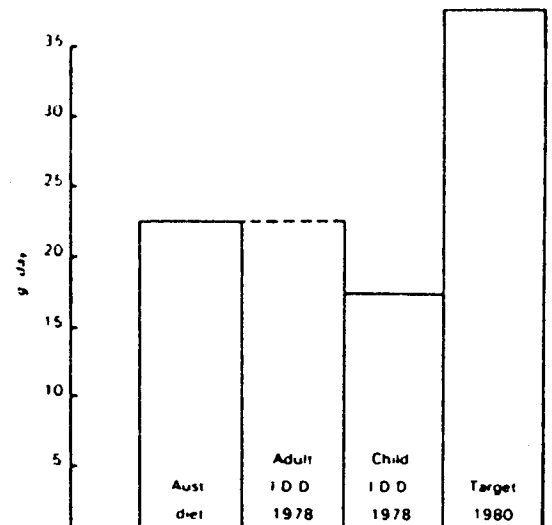
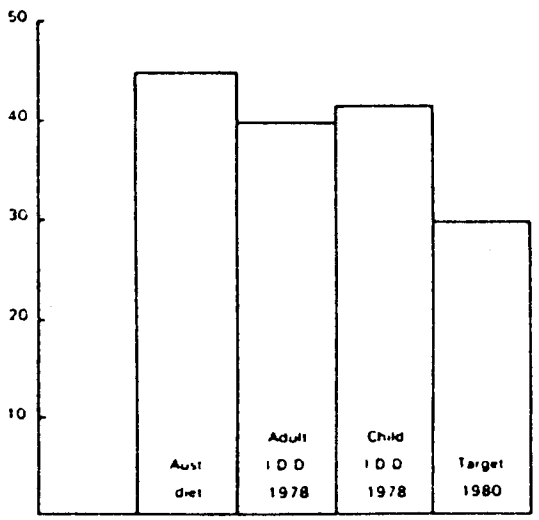


Figure 5 Average proportions of energy derived from fat for Australians in general (estimated from dietetic practice), older insulin-dependent diabetics (adult IDD), younger insulin-dependent diabetics (child IDD) and the target intake.



special products. The revised view of unrefined carbohydrate intake along with an understanding of the place of alcohol should facilitate these activities.

Regular exercise may reduce insulin requirements and improve carbohydrate and lipid control. Before an unusual increase in physical activity, extra carbohydrate should be ingested.

An additional dietary consideration in the pregnant diabetic is that the renal threshold for glucose falls. This can lead to a significant glucose and energy loss in the urine which must be met by dietary supplementation. For example, if a 24 hour urine were '1%' or '1 g' glucose/100 ml with a Clinitest or Diastix test and the urine volume were 3000 ml, this would represent a 30 g glucose or 120 calorie loss in 24 hours.

Nutrition education for the diabetic

For too long the dietary management of diabetes has concentrated almost entirely on carbohydrate and that without distinction between refined and unrefined. An adequate intake of all nutrients must be ensured. For MOD, energy balance is the first priority. The aims of nutritional advice for diabetes are summarised in Table 8.

It is time to learn more about the impact of whole foods on carbohydrate and lipid control and on maintenance of body weight. This will allow better nutritional advice than can be given on the basis of food compositional data with respect to carbohydrate and sucrose.

Table 8 Aims of nutritional advice for diabetics

1. Maintain ideal body weight
2. Maintain or improve glucose tolerance
3. Maintain as near as practicable euglycaemia
4. Maintain normolipidaemia including cholesterol, triglycerides and high density lipoprotein
5. Ensure an adequate intake of all nutrients
6. Allow ample physical activity
7. Consider the social function of food

REFERENCES

1. Nerup, J., Platz, P., Anderson, O. O., Christy, U., Lyngsoe, J., Poulsen, J. E., Ryder, L. P., Staub Nielsen, L., Thomsen, A., Svejgaard, A. (1974), HL-A Antigen & Diabetes Mellitus, *Lancet II*, 864.

2. Schernthaner, G., Mayr, W. R., Pacher, M., Ludgwig, H., Erd, W., and Eibl, M. (1975), HL-A8, W15, and T3 in Juvenile Onset Diabetes Mellitus. *Horm. Metab. Res. 7*, 521.

3. Tattersall, R. B. & Pyke, D. A. (1972). Diabetes in Indentical Twins. *Lancet III, 1120.*

4. Keen, H. & Jarrett, J. (1979). Diabetes Mellitus and its Complications. *Medicine Australia* (Series 1) 10 (2), 637.

5. Bray, G. A. (1979). Syndromes of Obesity: Pathogenesis and Treatment. *Medicine Australia* (Series 1) 10 (2), 690.

6. Cahill, G. F. Jr. (1978). Obesity and Diabetes. In *Recent Advances in Obesity* Vol.II. ed. G. Bray, Newman Publishing, 101.

7. Westlung, K. and Nicolaysen R. (1972), Ten Year Mortality and Morbidity, Related to Serum Cholesterol—a follow up of 3751 men ages 40–49. *Scan. J. Clin. Lab. Invest. 30* (Suppl.) *127*, 1.

8. Olefsky, J., Reaven, G. M., Farquar, J. W. (1974), Effects of Weight Reduction on Obesity: Studies of Lipid and Carbohydrate Metabolism in Normal and Hyperlipoproteinaemic Subjects. *J. Clin. Invest. 53*, 64.

9. Blacket, R. & Reader, R. (1979), Bodyweight and Coronary Heart Disease. Notes on Cardiovascular Diseases, National Heart Foundation of Australia.

10. Proust, A. J. & Smithurst, B. A. (1968), Epidemiology of Diabetes Mellitus in Australia. *Med. J. Aust. II*, 769.

11. Smithhurst, B. A., Wallace, B. C. & Proust, A. J. (1968), Toowoomba and Goulburn Diabetes Surveys. Med. J. Aust. *II*, 775.

12. Smithhurst, B. A. Epidemiological Survey of Diabetes in Toowoomba, Queensland, (1969), *J. Chron. Dist. 22*, 153.

13. Miller, J. A. and Miller, E. C. (1976), Carcinogens occurring naturally in foods. *Federation Proceedings 35*, 1316.

14. Truswell, A. S., Thomas, B. J. & Brown, A. M. (1975), Survey of Dietary Policy and Management in British Diabetic Clinics. *Brit. Med. J. 4*, 7.

15. West, K. M. (1976), Prevention and Therapy of Diabetes Mellitus. In Nutrition Review, *Present Knowledge in Nutrition*. Ed. D. M. Hegsted, C. O. Chichester, W. J. Darby, K. W. McNutt, R. M. Stalvey & E. H. Stotz. The Nutrition Foundation Inc., Washington, 356.

16. Bierman, E. L., Albring, M. J., Arky, R. A., Connor, W. E., Dayton, S., Spritz, N. and Steinberg, D. (1971), Principles of Nutrition and Dietary Recommendation for Patients with Diabetes Mellitus. *Diabetes 20*, 633.

17. West, K. M. (1973), Diet Therapy of Diabetes: An Analysis of Failure. *Ann. Int. Med. 79*, 425.

18. Wahlqvist, M. L., Wilmshurst, E. G., Murton, C. R. and Richardson, E. N. (1978) The Effect of Chain Length on Glucose Absorption and the Related Metabolic Response. *Am. J. Clin. Nutr. 31*, 1998.

19. Jenkins, D. J. A., Leeds, A. R., Wolever, T. M. S., Goff, D. V., Alberti, K. G. M. M., Gassull, M. A. and Hockaday, T. D. R. H. (1976) Unabsorbable Carbohydrate and Diabetes. Decreased post-prandial Hypoglycaemia. *Lancet II*, 172.

20. Wahlqvist, M. L., Morris, M. J., Littlejohn, G. O., Bond, A. & Jackson, R. V. J. (1979). The Effects of Dietary Fibre on Glucose Tolerance in Healthy Males. *Aust. N.Z. J. Med.* in press.

21. Brodribb, A. J. M. & Humphreys, D. M. (1976). Metabolic Effects of Bran in Patients with Diverticular Disease. *Brit. Med. J. 1*, 428.

22. Crap, P. A., Reaven, G. & Olefsky, J. (1977), Post-Prandial Plasma Glucose and Insulin Responses to differenct complex Carbohydrates. *Diabetes 26*, 1178.

23. West, K. M., (1974), Is the Risk of Becoming Diabetic Affected by Sugar Consumption *in* International Sugar Research Symposia Bethesda, Washington D.C., 33.

24. Trowell, H. (1977), Diabetes Mellitus and Dietary Fibre of Starch Foods. *Amer. J. Clin. Nut. 31*, S 53.

25. Truswell, A. S. (1977), Diet and Nutrition of Hunter-gatherers. *In Health and Disease in Tribal Societies* Ciba Symposium 49 (new series). Elsevier, Amsterdam/Oxford/New York, 213.

26. Hockaday, T. D. R. Hockaday, J. M., Mann, J. I. & Turner, R. C. (1978). Prospective Comparison of Modified-Fat-High-Carbohydrate with standard Low-Carbohydrate Dietary advice in the treatment of Diabetes. One Year Follow-up Study. *Brit. J. Nut. 39*, 357.

27. Anderson, J. W. (1977). Effect of Carbohydrate Restriction and High Carbohydrate Diet on Men with Clinical Diabetes. *Amer. J. Clin. Nut. 30*, 402.

28. Simpson, R. W., Mann, J. I., Eton, J. & Hockaday, T. D. R. Metabolic Effects of a High Carbohydrate-Modified Fat Diet in Maturity-Onset Diabetics (to be published.)

29. Rudnick, P. A. and Taylor, K. W. (1965). Effect of prolonged carbiohydrate restriction on Serum-Insulin levels in Mild Diabetes. *Brit. Med. J. 1*, 1225.

30. Hadden, D. R., Montgomery, D. A. D., Skelly, R. J., Trimble, E. R., Weaver, J. A., Wilson, E. A. & Buchanan, K. D. (1975), Maturity Onset Diabetes Mellitus. Response to Intensive Dietary Management. *Brit. Med. J. III*, 276.

31. Perkins, J. R., West, T. E. T., Sonksen, P. H., Lowy, C. and Iles, C. (1977), The Effects of Energy and Carbohydrate restriction in Patients with chronic Diabetes Mellitus. *Diabetologia 13*, 607.

32. Brunzell, J. D., Lerner, R. L., Porte, D. and Bierman, E. L. (1974), The Effect of a Fat Free, High Carbohydrate Diet on Diabetic Subjects with Fasting Hyperglycaemia. *Diabetes 23*, 138.

33. Keihm, T. G., Anderson, J. W., and Ward, K. (1976), Beneficial Effects of High Carbohydrate, High Fiber diet on Hyperglycemic Diabetic Men. *Amer. J. Clin. Nutr. 29*, 895.

34. Jenkins, D. J. A., Wolever, T. M. S., Leeds, A. R., Gassull, M. A., Harsman, P., Dilawari, J., Goff, D. V., Metz, G. L. & Alberti, K. G. M. M. (1978), Dietary fibres, fibre analogues, and glucose tolerance: Importance of Viscosity. *Brit. Med. J. 1*, 1329.

35. Frerichs, H., Daweke, H., Gries, F., Grunekle, D., Hessing, J., Jahnke, K., Keup, U., Miss, H., Otto, H., Puls, W., Schmidt, D. and Zumfelde, C. (1973). A novel Pancreatic Amylase Inhibitor (BAY d 7791) Experimental studies in Rats and Clinical Observations in Normal and Obese Diabetic and non-Diabetic Subjects. *Diabetologia 9*, 68.

36. Puls, W. & Keup, U. (1973), Influence of an α-Amylase Inibitor (BAY d 7791) on Blood Glucose, Serum Insulin and NEFA in starch loading tests in rats, dogs and man. *Diabetologia 9*, 97.

37. Walton, R. J., Sherif, I. T., Noy, G. A. and Alberti, K. G. M. M. (1979), Improved Metabolic Profiles in Insulin-Treated Diabetic Patients given an alpha-glucoside hydrolase inhibitor. *Brit. Med. J. 1*, 220.

38. Brunzell, J. D. (1978), Use of Fructose, Sorbitol or Xylitol as a sweetener in Diabetes Mellitus. *J. Amer. Diet. Assoc. 73*, 499.

39. Newbern, P. M. & Zeiger, E. (1978), Nutrition carcinogenesis and Mutogensis. *In Advances in Modern Toxicology* vol. 5. Ed. by W. G. Flamm & M. A. Mehlman. Hemisphere Publishing Corporation, Washington, London, 53.

40. Doll, R. and Armstrong, B. (1975), Bladder cancer mortality in diabetics in relation to saccharin consumption and smoking habits. *Brit. J. Prev. Soc. Med.* 29, 358.

41. Garcia, M. J., McNamara, P. M., Gordon, T. and Kannell, W. B. (1974), Morbidity and Mortality in Diabetics in the Framingham Population, sixteen-year follow-up study. *Diabetes* 23, *105*.

42. Stamler, J. (1975), Atherosclerotic Coronary Heart Disease *in Diabetes Mellitus*, 4th Edition. American Diabetes Assoc., 229.

43. Carlson, L. A., Bottiger, L. E. (1972), Ischaemic Heart Disease in relation to fasting values of Plasma Triglycerides and Cholesterol. Stockholm Prospective Study. *Lancet I 865*.

44. Pelkonen, R., Nikkila, E. A., Koskinen, S., Penttinen, K., and Sarna, S. (1977), Association of Serum Lipids and Obesity with Cardiovascular Mortality. *Brit. Med. J. II, 1185*.

45. Kannel, W. B. & Dawber, T. R. (1977), High Density Lipoprotein as a protective factor against Coronary Heart Disease. The Framingham Study. *Amer. J. Med. 62, 707*.

46. Antonis, A. & Bershon, I. (1961), The Influence of Diet on Serum Triglycerides in South African White and Bantu Prisoners. *Lancet 1, 3.*

47. Stone, D. B. & Connor, W. E. (1963), The Prolonged effects of a Low Cholesterol High Carbohydrate Diet upon the Serum Lipids in Diabetic Patients. *Diabetes 12, 127.*

48. Court, J. M. Dunlop, M. and Hill, M. (1978), A Study of Plasma-Lipid Concentrations in Diabetic Children. *J. Hum. Nut. 32,* 285.

49. Mertz, W. (1976), Effects and Metabolism of Glucose Tolerance Factor in Nutrition Reviews. *Present Knowledge in Nutrition.* 4th Edition. Ed. D. M. Hegsted, C. O Chichester, W. J. Darby, K. W. McNutt, R. M. Stalvey, E. H. Stotz. The Nutrition Foundation Inc. Washington, 365.