

## Food, nutrition and diabetes

**D**iabetes mellitus was so-called because of the characteristic loss of large quantities of sweet urine. This was the outward evidence of a serious problem of metabolism. During the later nineteenth and early twentieth centuries, it became clear that diabetes was due to an inadequate supply of a hormone produced in the pancreas, called insulin. The blood glucose, often referred to as 'blood sugar', rises and above a certain level spills over from the blood into the urine. Because of this, extra water is passed, so leading to the loss of large quantities of urine and also to thirst.

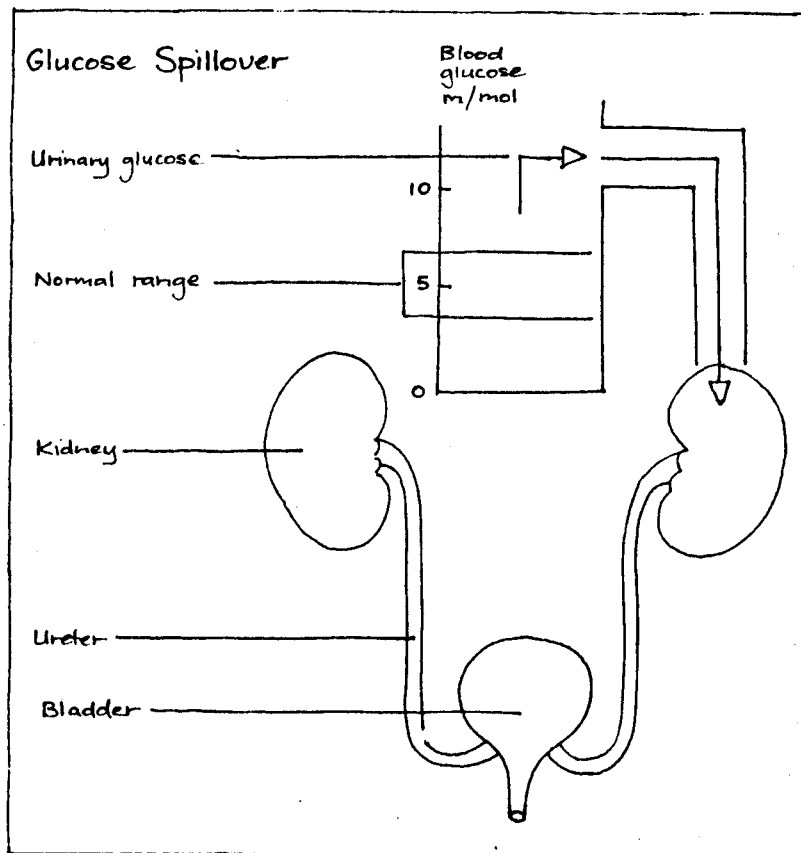
Most health organizations now recommend that glucose tolerance, or the body's handling of glucose, be assessed by measurement of blood glucose for two hours after ingestion of 75 g glucose in the fasting state. The acceptable limits are shown in Fig. 8.1.

Symptoms of diabetes are excessive thirst and excess urination because glucose is being excreted in the urine. More water is required to handle the glucosuria (glycosuria). Glucose generally 'spills over' from blood into urine when blood concentrations are above 10 mmol/l, the normal renal threshold, that is, the level above which glucose will be excreted in urine.

The classical situation in which blood glucose concentrations outside the normal range are found is in juvenile onset diabetes (JOD) where there is little or no insulin secretion by the beta cells of the islets of the pancreas which are responsible for the body's insulin production.

However, a relative lack of insulin can also occur. In

**Figure 8.1** Glucose is found in the urine when blood concentrations exceed the 'renal threshold', usually about 10 mmol/l. However, normal blood glucose concentrations range between 3–8 mmol/l.



Source: Wahlqvist, M.L. *Food and Nutrition in Australia*

maturity onset diabetes (MOD), despite higher than normal plasma insulin concentrations, blood glucose concentrations are still abnormally high. This suggests that there is resistance to insulin action. In most maturity onset diabetes, obesity is present, and this is thought to lead to the insulin resistance.

There is actually a strong hereditary tendency to MOD in certain families, but the disease does not appear unless obesity develops. Most MOD is preventable and families known to have the condition should be strongly counselled against becoming overweight.

### **Food metabolism and hormones**

We must provide a continuous supply of fuels in amounts required by body tissues, such as muscle, heart and brain. Fuels obtained from food (carbohydrate, fat and protein) may be processed in the liver, stored in the liver, fat or muscle, and routed through blood to the organ that needs them.

**Table 8.1 Hormonal response to food**

Fuels in blood		Hormones in blood	
Mixed meal	Glucose	Insulin	↑
	Amino acids		
	Free fatty acids		
	Triglycerides		

Fuels in blood include carbohydrates — glucose, lactate and pyruvate; and lipids (fats) — free fatty acids, triglycerides, ketone bodies and acetate. The concentration of each of these fuels in blood is affected by insulin, the hormone that is inadequate in diabetes. However, glucose has been the one most talked about in connection with diabetes, since, when it rises in blood, it spills over in the urine.

Glucose in blood may come from glucose-containing food; in addition, our bodies make it during fasting and during exercise, first in the liver and later in the kidney. It can be made from amino acids, the building blocks of protein, and also from lactic acid and from glycerol, but not from fat. A lack of insulin leads not only to reduced removal of glucose from the bloodstream — for example, after a meal containing glucose — but also to an increased manufacture of glucose by the body.

A hormone is a substance produced in one site and trans-

ported through the tissues to act at a distant site. One such, the insulin produced by the beta cells of the pancreatic islets, acts on liver, muscle, fatty tissue and other sites. It is the only hormone that can lower blood glucose concentration. Others raise blood glucose and are known as counter-regulatory hormones; these include adrenalin (catecholamine), cortisol (steroid hormone produced by the adrenal gland), glucagon (produced by the alpha cells of the pancreatic islets) and growth hormone (produced by the anterior pituitary gland at the base of the brain).

**Table 8.2 Control of blood glucose levels**

	Lower blood glucose	Raised blood glucose
Level of physical activity	Increased	Decreased
Nutrients		Carbohydrate and ethanol
Metabolites		Free fatty acids, (actic acid, some amino acids, glycerol)
Hormones	Insulin, Insulin-like activity	Glucorticoids (cortisol), Glucagon, Catecholoamines (adrenalin), Growth hormone

The fuels themselves also help to regulate each other. For example, as tissues in the fasting state take up more free fatty acid, they take up less glucose. The feed-back control between fuels contributes to a process of homeostasis, a term describing the maintenance of a steady internal environment within the body.

Not all food carbohydrates raise blood glucose equally. In the first place, not all carbohydrate is composed entirely of glucose units. For example, starch is a long chain consisting entirely of glucose units; sucrose is a disaccharide consisting of one molecule of glucose and another of fructose.

Pure starch raises blood glucose to the same extent as another disaccharide, maltose does if we take in the same amount. However, sucrose raises blood glucose to only half the extent

that maltose does. And fructose, when eaten alone, causes a very small increase in blood glucose, because a small amount of fructose can be converted to glucose in the liver, and released to the blood.

The sensitivity of the body to insulin also depends on the amount of carbohydrate in the diet. Thus, if normal healthy people are deprived of carbohydrate, their metabolism will become more like that of a diabetic. Conversely, as the proportion of carbohydrate in the diet increases in the diabetic, so blood glucose control improves. This process can, however, take days or weeks, and studies of diet in diabetes have not always taken this timing into account.

Another reason for the confusion about the place of carbohydrate in the diet of diabetics is that the reduction of excess weight improves diabetic control. Of course, if too much food is eaten for energy need, added weight can come from carbohydrate as well as from fat, protein and alcohol. As part of a general reduction in energy intake, a reduction in carbohydrate intake is advised. Studies where this has been done and where weight has decreased (with an associated improvement of blood glucose) have attributed the improvement to the reduction in carbohydrate intake rather than to the reduction in weight. In actual fact, the percentage contribution of carbohydrate to energy intake has sometimes increased.

Many factors in food other than carbohydrate affect the blood glucose. For example, the presence of fat slows down the emptying of the stomach and therefore decreases the rate of absorption of glucose. This immediate effect of fat (referred to as an 'acute' effect) may not be the same as its long-term ('chronic') effects on blood glucose. Then, too, the amino acids derived from protein influence the secretion of insulin from the pancreas and also the secretion of some of the counter-regulatory hormones.

Ethanol (or alcohol) acutely lowers blood glucose, and may do so to the extent that it produces the state of 'hypoglycaemia' (blood glucose concentration low enough to produce symptoms). However, the alcoholic drink may also contain glucose, which will tend to offset the hypoglycaemic effect of the ethanol.

**Table 8.3 Properties of food affecting blood glucose**

---

1. Physical
    - Particle size
    - Viscosity
    - Acidity
  2. Absorbable carbohydrate
    - Monomeric components (glucose, fructose, etc.)
    - Relative contribution to energy intake
  3. Dietary fibre
  4. Non-carbohydrate macro-nutrients
    - Protein
    - Fat
  5. Micro-nutrients
    - Chromium
    - Zinc
  6. Hypoglycaemic factors
    - Ethanol
    - Hypoglycin
    - Bongkreik acid
  7. Enzymes and enzyme inhibitors
- 

There is considerable interest in the role that roughage or dietary fibre plays in the regulation of blood glucose. We are now learning that these components of food have influence well beyond the regulation of bowel function. All dietary fibre, apart from lignin (a component of woody tissue), is carbohydrate, but — unlike other forms of carbohydrate — it is not digested in the small bowel. Hence, it is sometimes called 'non-absorbable carbohydrate ligning', or 'non-starch poly-saccharide'.

However, some breakdown of fibre does take place in the large bowel or colon, through the action of bacteria. Dietary fibres make up a varied group of chemicals with different ways of affecting blood glucose. The viscous (thick, sticky) properties of some, such as gums, slow down the rate of absorption of glucose. Others, ingested over a long period, may change the structure of the small bowel and so alter its absorptive capacity. Dietary fibre may also carry other substances that influence the digestion of carbohydrate, such as amylase (a starch-digesting enzyme).

On the whole, the presence of dietary fibre helps to identify

foods that may keep the blood glucose concentration within more narrow limits. On present evidence it seems preferable to obtain dietary fibre from as wide a variety of sources as possible, including whole-grain cereals and different kinds of vegetables and fruit.

Micronutrients, the vitamins and minerals in food, may also be important in regulating blood glucose. For example, some women who develop diabetes during pregnancy respond favourably to supplementation with vitamin B6. Poor glucose handling in children with protein energy malnutrition can respond to zinc administration. Elderly people who develop diabetes can be helped sometimes by the administration of the trace element chromium, found in high concentration in brewer's yeast and in pepper.

Thus, when considering the relation between food and blood glucose, we must consider as well as eating patterns, food as a whole, rather than just its glucose content. This is because of the complex effects that various food components have on body metabolism. And it is also because a lack of insulin leads to many more metabolic problems than a raised blood glucose.

### **Consequences of insulin inadequacy**

The consequences of insulin inadequacy can be thought of as acute (happening within hours or days) and chronic (happening over weeks, months or years). The acute manifestations, when the pancreas produces almost no insulin, not only include high blood glucose (hyperglycaemia), but also high blood concentrations of free fatty acids and the acidic ketone bodies. Where insulin is deficient, fatty tissue breaks down, releasing free fatty acids that are converted to ketone bodies. The rise of ketones makes the blood more acid, and this itself is life-threatening. Ketones appear in the urine and gives the breath a sweetish acetone smell. Breathing can be particularly heavy because of the need to get rid of excess carbon dioxide through the lungs.

Some diabetics have enough insulin to stop ketone formation, but not enough to prevent a progressive rise in blood glucose. Such diabetics progressively move into drowsiness and coma.

If too much insulin is given to a diabetic, blood glucose concentration can fall to levels associated with hypoglycaemia. In the first instance, the counter-regulatory hormones will rise in an effort to bring the blood glucose back towards normal. In particular, the patient will notice the various effects of adrenalin, including a feeling of apprehension and anxiety, a change in mood, awareness of a rapid heart beat (palpitations), tremor and sweating. As the blood glucose falls further, the brain becomes deprived of its important glucose fuel supply, and drowsiness and then coma take over. It is important, therefore, to recognise hypoglycaemia before the development of drowsiness. Repeated episodes of hypoglycaemia can lead to brain damage.

Blood fats — cholesterol and triglycerides — can also rise as a result of an inadequate supply of insulin. When elevated, these blood fats constitute risk factors for the common disease of the arteries, known as atherosclerosis, which leads to heart attacks and strokes (see chapter 7). The rises can occur because of decreased removal of blood fats from the blood in diabetes or because of their increased formation in the liver as part of the fat-protein complexes known as lipoproteins.

Among the chronic complications of diabetes mellitus, one group is associated with microvascular disease (disease of the small blood vessels). The vessels affected in particular are those of the retina, (which receives light at the back and the inside of the eye), the kidneys and the limbs (peripheral blood vessels).

**Table 8.4 Long-term complications of diabetes**

- 
1. Microvascular disease
    - Retina
    - Kidneys
    - Peripheral (limbs)
  2. Macrovascular disease
    - Coronary
    - Cerebral
    - Peripheral
  3. Lens opacification (cataracts)
  4. Nerve problems
  5. Skin problems
-



A growing body of evidence — from studies in experimental animals and from prospective studies in people — suggests that the degree of blood glucose control is important in the development of microvascular disease. Indeed, the World Health Organization criteria for the diagnosis of diabetes reflect the fact that individuals showing the typical diabetic blood glucose response run the risk of microvascular disease.

Of course, the development of such disease may also depend on factors other than glucose, such as genetic (inherited) predisposition and the tendency of small blood cells (platelets) which are the clotting factor in blood, to clump together too much. Diet itself may adversely affect small blood vessels in ways other than through glucose: it does appear to influence platelet aggregation, and fish oils have the ability to decrease platelet stickiness.

Macrovascular disease, or atherosclerotic vascular disease, affects large or medium-size arteries. It develops slowly and principally involves depositing cholesterol in the inner part of the arteries. When it affects the coronary arteries, a reduction in blood flow to the heart leads to heart disease. This can have various forms, including heart attacks (myocardial infarction), angina pectoris (pain in the chest because of lack of blood supply to the heart muscle), abnormal heart rhythm and heart failure.

When the disease affects the arteries supplying the brain, the carotid and vertebral arteries in the neck, and the cerebral

**Table 8.5 Glucose Tolerance Test**

Orally administered 75 g glucose  
World Health Organization (WHO) criteria  
Blood glucose (mmol/l.)

Time	Normal	Impaired	Diabetic
0 min	0.0	6.0 – 7.9	8.0
60 min	10.0	10.00–13.9	14.0
120 min	8.0	8.0 –10.9	11.0

This standardisation of the Glucose Tolerance Test is most useful for comparing individuals or populations and for deciding whether or not a person is diabetic. However, it should be remembered that we do not ordinarily take in glucose by itself, but rather in combination with other foods that can be expected to modify its absorption. Thus, in a mixed diet, in healthy individuals, it is unusual for the blood glucose to exceed 8.0 millimoles per litre.

arteries within the skull, then strokes can ensue. When the arteries affected supply the lower limbs, pain on walking can develop and gangrene or death of the tissues of the lower limbs can occur.

Not only diabetes, but also impaired glucose tolerance can increase the likelihood of macrovascular disease. It is not clear whether this is due to the blood glucose itself, to associated high insulin levels (although still inadequate for needs), to associated high lipid levels or to some other factor. Also, it must be remembered that risk factors for atherosclerotic disease act to reinforce each other — that is, they act synergistically. This makes it much more important when a diabetic smokes cigarettes, has high blood fats or has high blood pressure, since these are also major risk factors for atherosclerotic vascular disease.

Cataract formation, which makes the lens of the eye opaque, is also more common in the diabetic. This may occur because, when blood glucose concentration is high, some tissues form more sorbitol (a kind of sugar) which cannot get out of the cell. Water then moves into the cell to restore balance. The formation of sorbitol within cells in diabetes should not be confused with the possible effects of dietary sorbitol, commercially used in some foods for diabetics. Sorbitol in food is largely converted to fructose in the liver and does not end up in tissues like the lens of the eye.

Neuropathy (or abnormal function of nerves, either sensory or motor) can also occur in poorly controlled diabetes, as can a variety of skin lesions.

Thus, there are many reasons for avoiding development of diabetes if at all possible and for finding ways of modifying its expressions.

### **Development of diabetes**

The kind of diabetes that often occurs in young people, which requires insulin very soon after diagnosis, appears to develop because some injurious agent damages the beta cells of the pancreas that make insulin. One sequence of events, for which

there is growing evidence, begins with susceptible individuals, identifiable by their genetically determined tissue type (like a blood group). However, many genetically susceptible individuals may never encounter the injurious agent.

The agent may be a virus, which may directly attack the beta cells of the pancreas; alternatively, the antibodies that develop to fight the viral infection may damage the beta cells. It is common to find antibodies directed at the beta cells of the pancreas in the early stages of insulin-dependent diabetes mellitus (IDDM).

There has been little evidence that food intake patterns play a role in the development of IDDM. However, it has been reported from Iceland recently that some IDDM sufferers might have been exposed during foetal life to N-nitroso-compounds in the smoked or cured mutton traditionally eaten in December.

In Australia, possibly 2 to 3 per cent of diabetics have abused alcohol in sufficient quantities to damage the beta cells of the pancreas or, alternatively, to damage the liver sufficiently to disrupt the carbohydrate metabolism. However, the notion that sucrose (cane sugar) in some ways damages the pancreas, often held by diabetics themselves, is not well supported.

In an important international study of the prevalence of diabetes, Dr Kelly West and his colleagues showed that the higher the carbohydrate in energy intake and the lower the fat, the lower the likelihood of diabetes mellitus.

Obesity is associated with an increased risk of developing diabetes mellitus. The kind of diabetes that develops with overweight, however, usually does not require treatment with insulin at the time of diagnosis, although it may be required

**Table 8.6 Alcohol and diabetes**

Unfavourable effects	Favourable effects
Pancreatic damage	Social function
Cirrhosis impairs glucose tolerance	Increase in HDL (high-density lipoprotein)
High energy value	
Hypoglycaemia	
Oral agent interaction	
Other organic effects	

later on. Patients usually have higher levels than the blood insulin levels of non-diabetics, suggesting that they have some kind of resistance to the action of insulin, perhaps related to the extra fatty tissue.

Weight reduction alone can often return the blood glucose profile to normal. These diabetics can usually be managed either by diet alone or by a combination of diet and tablets. About 90 per cent of diabetics in Australia are non-insulin-dependent diabetics (NIDDM) and 80 to 90 per cent of these are overweight.

In developing countries, many diabetics are slim and do not require insulin. Several reasons have been suggested why they may have diabetes: they may have liver disease arising from previous hepatitis, or they might have ingested certain food toxins that have partially damaged the beta cells of the pancreas. Some evidence suggests that people whose staple diet is cassava, which contains a cyanogenetic glycoside food toxin, have more diabetes. (Cyanide administration can also produce diabetes in rats.) Zinc and chromium deficiencies may also play a role.

Diabetes can be secondary to iron overload of the pancreas and liver. In susceptible people, alcohol abuse can increase the risk of iron overload because alcohol increases the absorption of iron. This condition is known as bronze diabetes or haemochromatosis, because of the pigmentation of the skin that occurs.

### Extent of the problem

In Australia, diabetes mellitus affects 2 to 3 per cent of the population. (This figure comes from surveys of the populations of Toowoomba, Goulburn, Canberra and Busselton.) However, as already mentioned, most of these diabetics are of the NIDDM type. So the prevalence of IDDM is only a fraction of 1 per cent.

Not all populations have a diabetes prevalence rate as low as this. As Dr Paul Zimmet and his colleagues showed, Nauruans, who have dramatically changed their diet patterns, decreased their level of physical activity and become much more over-

weight over the last 50 years, have a prevalence of diabetes mellitus as high as 40 per cent.

Within Australia, different rates of diabetes prevail according to ethnic origin. Aboriginal Australians have been estimated to have a diabetes prevalence among adults from 9 to 20 per cent, about ten times the rate of adult Caucasian Australians. The high prevalence seems to have developed as the traditional hunter-gatherer life style has been abandoned. Clearly, this raises interesting questions about the roles of diet, exercise and other factors in the development of diabetes. Dietary factors that have been suggested include: an energy intake in excess of need; a reduction in dietary fibre intake; alcohol abuse; and an increased contribution of fat, especially of the saturated kind, to energy intake.

Dr Kerin O'Dea and her colleagues believe that fat intake may have something to do with the development of diabetes in Aboriginal Australians. They have pointed out that some traditional Aboriginal groups have a high-protein, low-fat, low-carbohydrate intake, and that this changes to a lower-protein, higher-fat diet as they move to cities.

It may also be that Aboriginal Australians, Nauruans and others have a more natural tendency to develop diabetes than other people in the same environmental conditions. It has been argued that a 'thrifty genotype' (hereditary constitution) allowed them to conserve energy better and survive at an earlier stage of evolution and migration.

### **Sweetness and diabetes**

We have good evidence that people are born with a preference for sweet things. The human foetus, for instance responds with swallowing movements to an alteration of sweetness of the amniotic fluid. And hunter-gatherers, who probably retain the food style which has been experienced for the longest period of our evolution, seek out sweet things. It may be that this preference has survival value, that it aids the recognition of foods that are sometimes energy-dense, fulfilling energy needs and sometimes safer to eat.

**Table 8.7 Sweetness**

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

Nowadays we think of sucrose as the yardstick against which to measure the sweetness of other compounds. Fructose and xylitol taste sweeter than sucrose, while glucose, lactose, maltose and sorbitol taste less so. Mostly, compounds with sweetness are carbohydrates and also have an energy value, but this is not necessarily the case. A new sweetener, aspartama, is actually a dipeptide, consisting of two amino acids.

Sweeteners with an energy value are known as 'nutritive', those without any are termed 'non-nutritive'. The best known non-nutritive sweeteners are saccharin and cyclamates. In recent years their possible adverse effects on health have aroused concern, especially that of the Food and Drug Administration of the United States. This is because some work on experimental animals has suggested a possible increase in the risk of bladder cancer. However, diabetics, who have used these non-nutritive sweeteners the longest, show no evidence of an excess of bladder cancer over non-diabetics. Caution should still be exercised in their use, however, since they are increasingly being consumed by younger and younger individuals. Thus, children who take food and beverages sweetened this way may be exposed for a whole lifetime. This duration of exposure and its risk has yet to be evaluated.

Diabetics who must lose weight to improve their carbohydrate control must not be confused about so-called 'diabetic foods'. These are often sweetened with, for example, sorbitol instead of sucrose, but sorbitol, being a carbohydrate, has an energy value similar to that of sucrose, although it may not be quite as completely absorbed. Thus, these 'diabetic foods' can add to a weight problem just as other foods do.

Whether or not low energy foods sweetened with saccharine or cyclamates aid in weight control has also yet to be sub-

stantiated. Clearly, fewer kilojoules are ingested, but the preference for sweet things may still contribute to a greater total energy intake than required.

### Can diet help?

Nutritional management of diabetes can be helpful in several ways. First, it can help to avoid further pancreatic damage by control of obesity, avoidance of food toxins where appropriate, and by reduction in alcohol consumption.

Second, it can normalise blood glucose. The non-insulin-dependent diabetic who has a limited insulin reserve for need, can use that reserve more effectively with a more even spread of carbohydrate through the day. On the other hand, a relatively greater contribution of carbohydrate to energy requirements will increase the sensitivity of the body to whatever insulin is available.

The insulin-dependent diabetic can arrange food intake first on general nutritional grounds, and then tailor the insulin dosage to that food intake pattern. However, the administration of insulin under the skin (subcutaneous) usually makes it impossible to generate more than four slowly rising and falling peaks of blood insulin during a 24-hour period. Thus, a knowledge of carbohydrate in food, and its distribution at fairly regular intervals through the day is helpful.

Third, diet control can normalise blood fats. A reduction in saturated fat and cholesterol, with an increase in the amount of

**Table 8.8 Energy intake as carbohydrate in insulin-dependent diabetics (Caucasian Australians)**

	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

Table 8.9 Nutritional characteristics of some foods in the daily diet of a diabetic

Food item	Average size serve (g)	Energy to/serve	Carbohydrate (g/serve)	Number of 10-g portions per serve	Energy from Carbohydrate (%)	Energy from fat (%)	Dietary fibre (g/serve)
<b>BREAKFAST</b>							
Cornflakes	30	470	25	2.5	88	5	3
All Bran	10	115	5	0.5	73	19	3
Bran	10	87	3	0.5	58	24	5
Bread-wholemeal	30	275	12.5	1.5	77	12	3
Bread-white	30	...	15	1.5	83	7	1
Apricots-stewed (no added sugar)	100	...	5.0	0.5	85	0	2
Cod-fried in batter	90	750	5	1	11	50	0
Cod-grilled	90	...	0	0	0	13	0
Lamb chops — loin grill (lean)	90	835	0	0	0	50	0
Lamb chops — loin grill (with fat)	90	1325	0	0	0	74	0
Chicken-breast	75	.65	0	0	0	33	0
Beef-grill (lean)	50	820	0	0	0	51	0
Potatoes — boiled	90	310	15	1.5	81	0	1



Potatoes — chips	30	320	10	1	53	37	0
Rice — boiled	30	155	10	1	93	0	0
Spagnetti — boiled	60	300	10	1	57	0	0
Peas, frozen — boiled	60	105	5	0.5	80	0	7
Peas, fresh — boiled	60	134	5	0.5	63	0	3
Beans, fresh — boiled	80	10	0	0	0	0	1
Beans, haricot — boiled	50	200	10	1	83	0	4
Tomatoes — raw	50	54	2.5	0	71	0	2
Sweetcorn kernels	60	312	15	1.5	83	12	2
Lettuce	50	25	0	0	0	0	1
Cauliflower — boiled	50	20	0	0	0	0	1
SNACK ITEMS							
Apple	100	200	10	1	83	0	2
Banana	50	300	20	2	71	0	3
Dried dates	30	630	20	2	53	0	7
Dried apricots	20	155	10	1	93	0	5
Rye crispbread (Ryvita)	20	273	15	1.5	92	0	2
Hamburger with onion	75	623	1.5	0	3	73	0
Milk	200ml	...	10	1.5	42	51	0

Suggested energy distribution for a 25-year-old adult diabetic male requiring 11 500 kilojoules per day:

Carbohydrate: 45%–60% (310 g–410 g carbohydrate)

Fat : 25%–35% (75 g–105 g fat)

polyunsaturated fat, can help reduce the blood cholesterol level. The increased polyunsaturated-to-saturated fat ratio will also help reduce blood triglyceride concentration. In order to make this change, for a given level of energy intake, the diabetic will need to increase the carbohydrate intake, which, as has been indicated, has a favourable effect on carbohydrate control in its own right. When the kind of carbohydrate used is associated with dietary fibre, this too helps blood glucose and blood fat control. Such a dietary approach may also have a favourable effect on the prevention of heart disease in other ways, including reduced platelet aggregation.

In short, the diet for the diabetic should keep body weight as near to desirable as possible, characteristically emphasise whole-grain cereals, vegetables (especially leguminous vegetables, peas, beans, lentils etc.) and fruits, and contain little animal fat and little alcohol. None of these characteristics need detract from the enjoyment of food and beverages.

### Further reading

- Brunzell, J.D. 'Use of fructose, sorbitol, or xylitol as a sweetener in diabetes mellitus' *Journal of the American Dietetic Association*, 73, 499, 1978
- Burkitt, D., and Trowell, H. (eds) 'Western Diseases.' London: Edward Arnold, 1981
- Gastineau, C.F. Effects of organ failure on nutrient absorption, transportation and utilisation: endocrine system. In 'Human Nutrition. A Comprehensive Treatise' R.B. Alfin-Slater and D. Kritchevsky (eds) volume 4. New York: Plenum Press, 1979
- Helgason, T. and Jonasson, M.R. Evidence for a food additive as a cause of ketosis-prone diabetes. *Lancet*, 2, 1981, 716-20
- O'Dea, M. Spargo, R.M. and Akkerman, K. The effect of transition from traditional to urban life-style on the insulin secretory response in Australian Aborigines. *Diabetes Care*, 3, 1980, 31-7
- Proust, A.J. and Smithurst, B.A. Epidemiology of diabetes mellitus in Australia. *Medical Journal of Australia*, 2, 1968, 769-72
- Sharon, N. Carbohydrates, *Scientific American*, November 1980, 80-97
- Simpson, H.C.R. Simpson, R.W. Lousley, S. et al A high-carbohydrate leguminous fibre diet improves all aspects of diabetic control. *Lancet*, 1, 1981, 1-5
- Smithurst, B.A. Wallace, B.C. and Proust, A.J. The Toowoomba and

- Goulburn diabetes surveys. *Medical Journal of Australia* 2, 1968, 775-7
- Smithurst, B.A. Epidemiological survey of diabetes in Toowoomba, Queensland. *Journal of Chronic Diseases*, 22, 1969, 153-64
- Taylor, R. and Reid, M. Admission to hospital for diabetes in Aborigines and other Australians, rural New South Wales, 1977-78. *Community Health Studies*, 5, 1981, 142-6
- Wahlqvist, M.L. McDonald, J. and Flint, D.M. 'Diet and diabetes mellitus' *Food and Nutrition, Notes and Reviews*, 36, 1979, 180-6
- Wahlqvist, M.L. Diet in the treatment of diabetes. *Medical Journal of Australia*, 2, 1980, 64-6
- Wahlqvist, M.L. (ed.) 'Food and Nutrition in Australia.' Sydney: Cassell, 1981
- Walborn, T.A. Curnow, D.H. Wearne, J.T. *et al.* Diabetes detected by blood sugar measurements after a glucose load; report from the Busselton survey, 1966 *Medical Journal of Australia*, 2, 1968, 778-83
- Wise, P.H. Edwards, F.M. Thomas, D.W. *et al.* Hyperglycaemia in the urbanized Aboriginal: the Davenport survey. *Medical Journal of Australia* 2, 1970, 1001-06
- Wise, P.H. Edwards, F.M. Craig, R.J. *et al.* Diabetes and associated variables in the South Australian Aboriginal. *Australian and New Zealand Journal of Medicine*, 6, 1976, 191-6