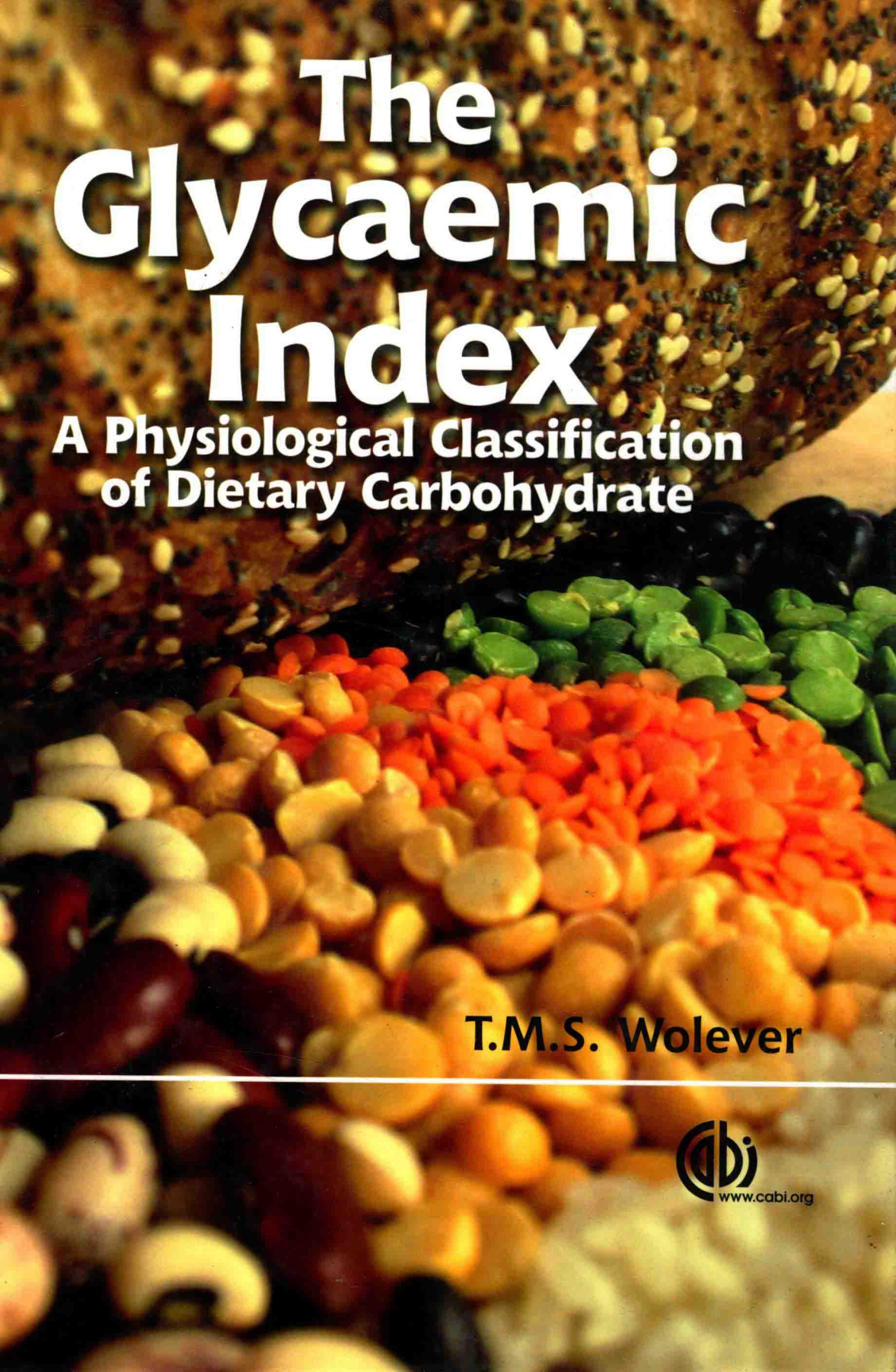
The Glycaemic Index

A Physiological Classification of Dietary Carbohydrate



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A Physiological Classification of Dietary Carbohydrate

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Preface

The term 'glycaemic index' (GI) first appeared in the literature in 1981. Initially, the concept was applied to the treatment of diabetes and there was vigorous debate and large differences of opinion about its role in this respect. The American Diabetes Association concluded that the GI had no clinical utility and it was not recommended in the management of diabetes. By contrast, Diabetes Australia was so strongly supportive it felt that GI was essential information for everyone with diabetes; the result was the world's first food labelling programme, the GI Symbol Program, operated by Glycemic Index Ltd, a non-profit corporation whose members are the University of Sydney, Diabetes Australia and the Juvenile Diabetes Research foundation. Nevertheless, for about 15 years after the initial publication, scientific interest in the GI was only moderate because it was considered relevant only to diabetes and did not have support from the American Diabetes Association.

However, public and scientific interest in the GI has exploded over the past 10 years as it became evident that GI-influenced human health and performance in many ways. Of particular interest has been the role of GI in the management of body weight, and more and more popular diet books make reference to it. Unfortunately, along with increased interest in the GI has come widespread promulgation of over-simplified, misleading and faulty information about it. Scientists, health professionals and lay people use the term 'glycaemic index' inappropriately. Foods are given incorrect GI values in popular books and peer-reviewed scientific publications. Incorrect methods are used to measure the GI of foods. The clinical utility of the GI is, more often than not, presented in an unbalanced fashion, with it either being praised with exaggerated benefits far beyond what is justified by any research, or being unjustifiably condemned as having no clinical utility. The results of clinical trials of low-GI diets are inappropriately extrapolated by some to apply to diets containing novel carbohydrates which reduce glycaemic responses by a different mechanism. Recommendations about how to use GI information are sometimes not consistent either with sound nutritional principles or with the way the GI was used in the clinical trials demonstrating benefits.

All this inappropriate information about GI is not helpful – at best it is confusing – at worst it could destroy the GI concept altogether. Either way, such misunderstanding reduces our ability to learn more about the role of the GI in health and disease and to determine how best to use the GI to help those who could derive benefit from it. Therefore, I offer this book in the hope that it will help students, scientists and health professionals to understand the current state of knowledge about GI, to evaluate properly studies and other information about the GI and to use the most appropriate and up-to-date methods in

their research, clinical work, product development or health education programs. In addition, I hope it will stimulate critical and creative thinking about the GI! It is not my intent to insist that my opinions about the GI are correct simply because that is the way it has always been done. I hope that the views presented in this book are based on the results of sound research and sound reasoning; I also hope that I am willing to be proven wrong.

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1

Historical Introduction

Over 150 years ago, Claude Bernard introduced the concept that the *milieu interieur*, the internal environment of the body, was controlled by homeostatic mechanisms. He was profoundly interested in carbohydrate metabolism, and considered that both the gut and the liver were central in the control of the blood glucose concentration. He suggested that carbohydrate was fed into the liver from the intestine and stored there (Bernard, 1848, 1855) and released (La Coudraie and Malloizel, 1881) as necessary to conserve the constancy of the *milieu interieur*. After his death, the liver continued to be considered central in the control of carbohydrate metabolism, a role later emphasized by the fact that individuals with liver disease may have episodes of hyperglycaemia (Johnston *et al.*, 1977) and hypoglycaemia (Sherlock, 1975). In addition, in individuals with diabetes who have normal liver function, there is a close relationship between hepatic glucose output and the fasting blood glucose (FBG) concentration (DeFronzo, 1988). With the discovery of the endocrine function of the pancreas, and later insulin itself, the role of the gastrointestinal tract in the regulation of carbohydrate metabolism was minimized. However, the small intestine is now known to be the largest endocrine organ in the body, and by virtue of the gut hormones, once again, has been implicated in the control of carbohydrate metabolism (Bloom and Polack, 1981; Drucker, 1998). This role was further enhanced by the discovery that modification of intraluminal events within the upper gastrointestinal tract by dietary fibre may profoundly affect the blood glucose and endocrine responses elicited by carbohydrate foods.

1.1 The Dietary Fibre Hypothesis

Interest in dietary fibre arose from the observations of Dennis Burkitt and Hugh Trowell, medical missionaries who noted, during their years of medical service in Africa, that many of the diseases common in developed countries were virtually unknown among rural Ugandans living on their traditional diets (Burkitt and Trowell, 1975). It is recognized that many factors have a role in the development of non-infectious diseases, including the effects of physical activity and obesity. The role of diet is less clear and much more controversial. Nevertheless, Burkitt and Trowell suggested that there was an association between a lack of fibre in the diet and many 'Western' diseases including coronary heart disease (CHD) (Trowell, 1972), diabetes (Trowell, 1973, 1974) and colon cancer (Burkitt, 1971). These observations have been confirmed by many subsequent epidemiological studies, and the role of dietary fibre in reducing the risk of heart disease (Lupton and Turner, 2003), diabetes (Salmerón *et al.*, 1997a,b; Wolever *et al.*, 1997a) and colon cancer (Bingham *et al.*, 2003; Peters *et al.*, 2003) is now well established.

Since it is not absorbed, dietary fibre influences disease risk by influencing events within the lumen of the gastrointestinal tract. In the 1970s, this was a novel concept, and the mechanisms discovered for dietary fibre led not only to the glycaemic index (GI), but also to the development of novel pharmacological (e.g. α-glucosidase inhibitors) and nutritional approaches (e.g. fructo-oligosaccharides) to prevent and treat various

disorders. The mechanisms of action suggested for dietary fibre included: dilution of the energy density of the diet, resulting in reduced food intake and obesity; altered rates of gastric emptying; reduced rate and/or extent of the absorption of carbohydrate, fatty acids and cholesterol; the passage of fibre into the colon, leading to stool bulking and reduced transit times; and colonic fermentation leading to the production of short-chain fatty acids (SCFA) which influence local and systemic metabolism. For example, SCFA reduce the pH of the colonic contents, which in turn, alters intraluminal metabolism and absorption of sterol and nitrogen compounds. SCFA provide fuel for colonic mucosa and are absorbed and influence systemic metabolism. More recently, it has been shown that unabsorbed carbohydrates alter the populations of specific colonic bacterial species. This alteration, in turn, influences local and systemic immune function. These concepts will be discussed in more detail later in this book. Of most relevant to the development of the GI, however, was work on the effect of dietary fibre on postprandial glucose and insulin responses.

Early on it was suggested (Heaton, 1973), and later demonstrated both *in vitro* and *in vivo* (Elsenhaus *et al.*, 1980; Jenkins and Wolever, 1981; Blackburn *et al.*, 1984), that certain forms of dietary fibre, notably the viscous gelling agents such as guar gum, a galactomannan extracted from the cluster bean, *Cyampopsis tetragonoloba*, were able to reduce the rate of uptake of carbohydrate from the small intestine (Blackburn *et al.*, 1984) and reduce the excursions of blood glucose and insulin following the ingestion of fibre-containing test meals (Jenkins *et al.*, 1976). We showed that incorporation of guar into the diets of subjects with diabetes resulted in improved glycaemic control, as judged by reduced urinary glucose output (Jenkins *et al.*, 1977). In order to see if guar could have any practical use in the management of diabetes, an effective method of administration was required. If guar worked by trapping the food within an intraluminal gel and reducing the rate of digestion, it would have to be hydrated and intimately mixed with the food in order to reduce postprandial glucose responses; if guar were sprinkled on food, or taken between meals it was not effective in this respect (Wolever *et al.*, 1978). Although mixing guar with foods such as bread, instant potato, soups and cereals prior to eating was effective in reducing glucose and insulin responses, it made them highly viscous and unpalatable (Wolever *et al.*, 1979). Guar crispbread turned out to be one palatable solution to this problem (Jenkins *et al.*, 1978a) and patients with diabetes were able to use guar crispbread for periods of up to 1 year with some success (Jenkins *et al.*, 1980c).

Unfortunately, we were unable to develop the use of guar as a practical tool in the management of diabetes for several reasons. The manufacturer of guar crispbread was unable to continue to support the research programme, and we were unable to find any other industrial backing. Manufacturers felt that there was no future in guar-enriched foods (products which would now be known as 'functional foods'). In addition, we had to rely on the crude measure of urinary glucose output as an assessment of postprandial glucose levels. At this time, there was no way of measuring blood glucose concentrations throughout the day in patients with diabetes outside of a metabolic ward situation. Glycated haemoglobin had not yet been discovered, and portable glucose monitors had not yet been developed. Indeed, lancet devices using a fine needle had only just been developed and our colleagues in the diabetes clinic were assessing procedures for home glucose monitoring such as having patients place a drop of blood on a filter paper and mailing to the laboratory a small disc of blood-soaked paper punched out with a hole punch. The difficulty in assessing glycaemic control made it difficult to know whether dietary fibre supplements had any benefits. Therefore, we decided to turn our attention to studying the glycaemic responses elicited by high-fibre foods; work which lead directly to the development of the GI. Before describing this, however, it is important to review studies on the glycaemic impact of foods originating from other laboratories prior to the appearance of the GI.

1.2 Early Studies on the Glycaemic Effects of Carbohydrates

Clinicians were interested in the glycaemic effects of foods at a very early stage, even before blood glucose could be measured easily. Because of methodological limitations, the results of some of the early papers are often difficult to interpret or do not agree (but are not necessarily inconsistent) with our current understanding about the glycae-

mic effects of carbohydrates. However, results can be found from over 80 years ago which are completely consistent with modern results. The following is not a complete review of the early literature, but a sampling of those cited by later workers in the area and which therefore appear to have been influential in shaping understanding about the glycaemic effects of carbohydrates prior to the development of the GI.

1.2.1 Studies before 1970

Before 1970, it was almost universally held that available carbohydrates should be categorized as being 'simple' (i.e. mono- and disaccharides) or 'complex' (i.e. starch). The idea that simple carbohydrates elicit higher glycaemic responses than complex carbohydrates originated from the work of Allen prior to the 1920s in dogs; in these studies blood glucose was not even measured. Allen's objective was to determine the appropriate management of diabetes at a time when insulin and oral hypoglycaemic drugs were not available and diet was the only known treatment. Partially depancreatized dogs were fed various different diets with urinary glucose excretion being the measure of glycaemic control and rate of progression of diabetes. Allen found that feeding dogs glucose caused diabetes to progress more rapidly than starch, but that there was no difference between the effects of different starches such as bread, oatmeal, rice, potato or pearled barley (Allen, 1920). Allen attributed the difference between 'sugar' and starch to the rate of absorption, and concluded that: '... a rapid flood of carbohydrate is more injurious to the pancreatic function than a slow absorption'.

In the early 1920s, carbohydrate tolerance was studied by measuring glucose excretion in the urine or glucose concentration in the blood. Indeed, an experimental issue which interested many investigators at this time was to determine the renal threshold for glucose, since it was not known if people without diabetes ever had 'sugar' in their urine (Folin and Berglund, 1922). We now know that the chemical methods used to measure urinary glucose at that time reacted with substances other than glucose; thus, the results of these early papers are probably unreliable because of false positives.

During this early period, blood glucose responses elicited by various nutrients and foods were sometimes measured, but the results are often difficult to interpret or unreliable for a number of reasons such as the use of a very small number of subjects (often only one); non-standardized fasting times, starting times, blood sampling intervals and doses of nutrients fed; and a lack of description of the composition of the test meals. Some of these problems are illustrated by the results Folin and Berglund (1922) (Fig. 1.1). The glycaemic response elicited by 200 g glucose was determined in six subjects – the carbohydrate tolerance of the six subjects differed markedly and the blood sampling interval and time over which blood samples were taken also varied from subject to subject (Fig. 1.1, top). The responses elicited by various carbohydrates including maltose, starch, fructose, galactose and lactose were studied in one subject. None of them elicited an appreciable rise in blood glucose (Fig. 1.1). Unfortunately, these results cannot reliably be compared with those elicited by glucose because the subject's blood glucose concentration after oral glucose was not measured until 1.5 h after the glucose load. By this time, blood glucose was only moderately above the fasting concentration and most of the glycaemic response elicited by oral glucose had, presumably, been missed. To be fair to the authors, however, the major purpose of the study was to determine the relationship between 'blood sugar' and 'urine sugar' in an attempt to gain some understanding about the metabolism of different sugars. Thus, the timing of the blood samples was probably related to when the subjects could pass urine rather than a desire to comparing the glycaemic responses elicited by the different test meals.

Gray (1923) was concerned about how to diagnose diabetes and felt that measuring blood sugar was better than measuring urine sugar. However, there was concern at the time that feeding subjects large doses of glucose might be harmful because of Allen's conclusion (Allen, 1920) that glucose was more damaging to the pancreas than starch. Therefore, Gray (1923) reviewed the blood glucose responses elicited by various doses of glucose and other carbohydrates with a view to establishing normal ranges. The results presented in this paper are difficult to interpret for several reasons. The different test meals were tested in different groups of subjects

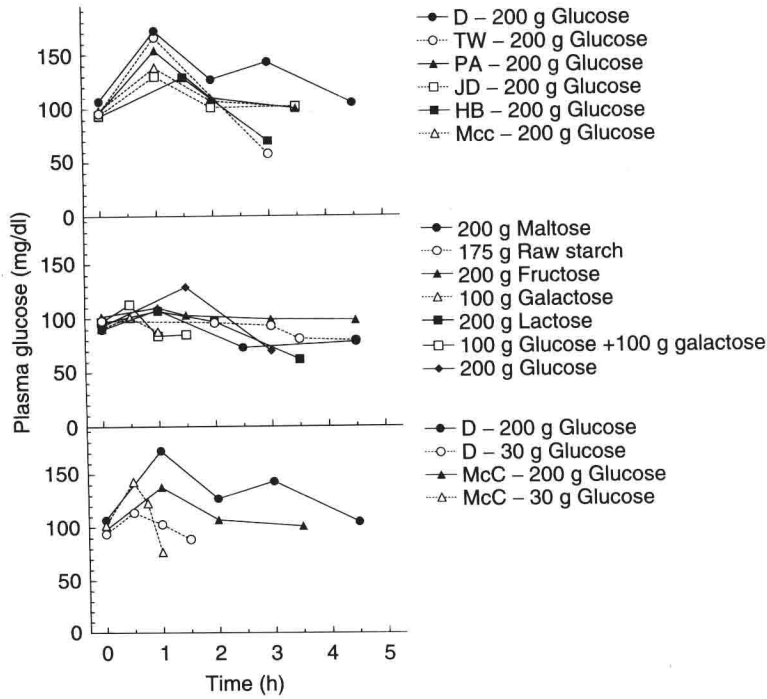

Fig. 1.1. Plasma glucose response curves published by Folin and Berglund (1922). Each curve is the response of a single individual. Top: six individuals' responses to 200 g glucose. Middle: one individual's (subject HB) response to various different carbohydrates. Bottom: two subjects' responses to 30 and 200 g glucose.

varying in size from $n = 4$ to 300. The doses of carbohydrates were not consistent, and the results of different doses were often combined in ranges. Also, curiously, the tables giving the mean blood glucose responses elicited by the different carbohydrates do not include the fasting value, and thus, one has to use the overall mean value for fasting glucose for all the subjects. This may not necessarily represent the fasting glucose for the group of subjects testing each test meal. The results from Gray (1923) suggest that glycaemic responses generally increase with the dose of glucose consumed, but 50 g glucose elicited a higher response in 21 subjects than 70–100 g glucose in 300 subjects (Fig. 1.2, left). These data do not fit the expected dose–response relationship derived from recent data (Fig. 1.2, right). By contrast, MacLean reported glycaemic responses elicited by 5, 10, 20 and 50 g glucose in the one subject (Fig. 1.2, centre) and these results fit the expected non-linear dose–response relationship very well (Fig. 1.2, right).

Another result from Gray (1923), which is not consistent with current concepts, is that sucrose and fructose elicited higher glycaemic responses than glucose (Fig. 1.3, top). On the other hand, MacLean (1922) showed that 50 g fructose elicited only about 15% of the glycaemic response elicited by 50 g glucose. Gray also shows that the responses elicited by starch and a mixed meal were similar to those elicited by glucose (Fig. 1.3, bottom), however, the nature of the starch and the composition of the mixed meal were not indicated.

Rowe and Rogers (1927) compared the glycaemic response elicited by 100 g glucose with that elicited by two shredded wheat biscuits and 3 oz milk, because the latter was being considered as a possible diagnostic test for diabetes. Twenty subjects consumed both test meals with blood glucose being measured at fasting and at 30, 90 and 150 min. These methods are suitable for comparing the glycaemic impact of the two test meals. I calculated the incremental areas under

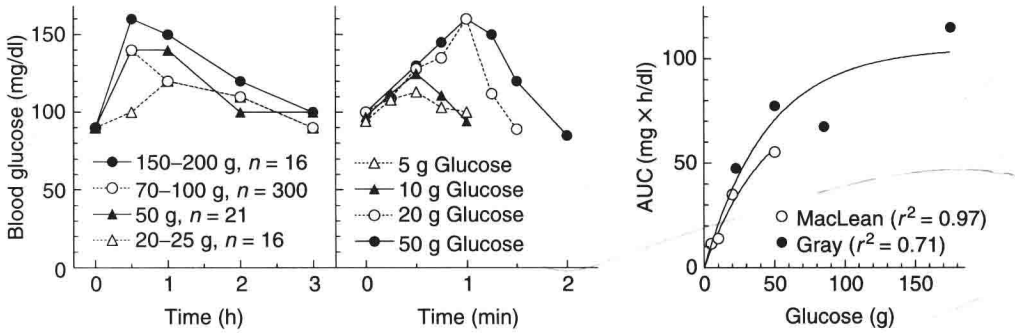

Fig. 1.2. Early dose–response curves: glycaemic responses elicited by different doses of glucose in normal subjects. Left, redrawn from Gray (1923). Centre, redrawn from MacLean (1922). Right, non-linear regression of incremental area under the curve (AUC) on dose of glucose; solid lines represent regression lines with the equation $AUC = K \times (1 - e^{-0.0222g})$ where K is a constant and g is the grams of glucose (the rate constant is the same as that derived in Fig. 4.1 using data from my laboratory).

the mean glucose response curves shown in the Chart 2 of Rowe and Roberts (1927), and found that the glycaemic response elicited by shredded wheat, 659 mg × min/dl, was 38% of that elicited by 100 g glucose, 1736 mg × min/dl. To see whether this result is similar to what would be expected based on our current knowledge, one needs to know the amount of available carbohydrate and GI of the test meals. Rowe and Rogers (1927) did not indicate the nutrient composition of the shredded wheat test meal, but according to current food tables it would contain 37 g of available carbohydrate and have a GI of 70 (48 g of shredded wheat = 33 g available carbohydrate with GI of 75 (Foster-Powell *et al.*, 2002) and 92 g milk = 4 g available carbohydrate with a GI of 32). Using the formula shown in Fig. 4.1, the expected relative response of the shredded wheat meal (37 g available carbohydrate, GI = 70) is 44% of that for 100 g glucose (100 g

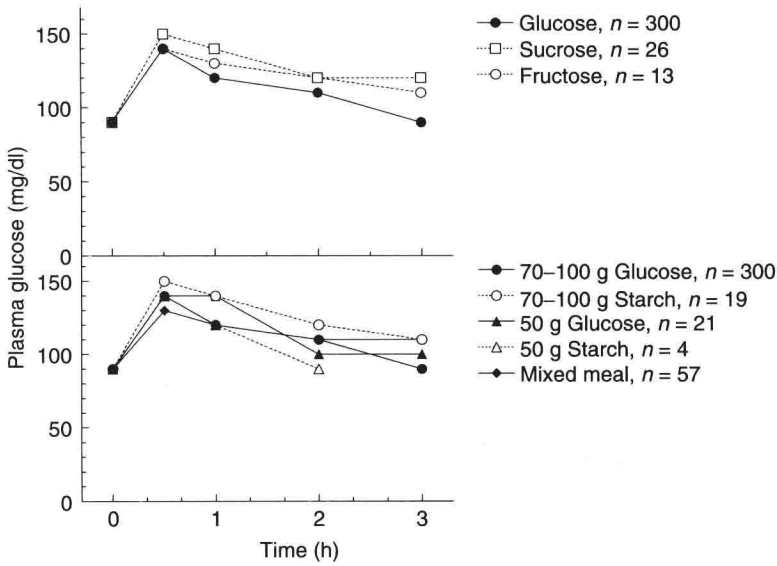

Fig. 1.3. Comparison of glycaemic responses elicited by different sugars (top) and glucose, starch and mixed meals (bottom) redrawn from Gray (1923).

available carbohydrate, GI = 100); a value very similar to the observed value of 38%.

During this period of time, it was generally felt that there were no significant differences in the assimilation of different starches (Allen, 1920), and there was evidence that there was little difference between cooked starches and glucose (Gray, 1923). Indeed, on this point, Allen (1920) concluded: 'Whenever permanent diabetes is present... starch brings on glycosuria more slowly than sugar, but just as surely.... The clinical lesson from such experiments is that even if a patient becomes free from glycosuria on withdrawal of sugar only, nevertheless other foods should also be limited'. The idea that different starchy foods have the same glycaemic impact was also put forward by MacLean (1922) based on a comparison of the glycaemic responses elicited by 250 g potatoes vs 100 g oatmeal (Fig. 1.4), probably based on studies in one subject. (Note also the different schedule of blood sampling for the different tests.) MacLean (1922) states that 'The ordinary starchy foods, such as potatoes, rice, corn-flour, and oatmeal, behave almost exactly like glucose when given in proportionate amounts'.

Although it was felt that all cooked starches elicited large glycaemic responses, it was known in the 1920s that raw starches had virtually no glycaemic effect in humans (Rosenthal and Ziegler, 1929) (Fig. 1.5). It was known at this time that raw starches were virtually completely digested during passage through the human alimentary tract (Langworthy and Deuel, 1922), but there was controversy as to the extent to which digestion and absorption occurred in the small intestine, as

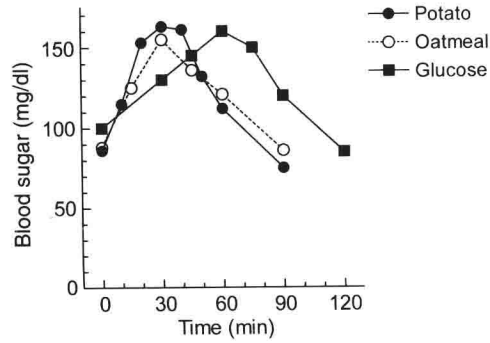

Fig. 1.4. Glycaemic responses elicited by 50 g glucose, 250 g potatoes and 100 g oatmeal. Redrawn from MacLean (1922).

opposed to breakdown by bacterial fermentation in the colon (Rosenthal and Ziegler, 1929). Those of us working in the carbohydrate field today tend to think that the significance of colonic fermentation in humans only began to be appreciated about 25 years ago; in fact, the discussion of Rosenthal and Ziegler (1929) reads like it was written recently and clearly shows that the importance of colonic fermentation was appreciated over 75 years ago.

Conn and Newburgh (1936) were mainly interested in comparing the effects of carbohydrate and protein on glycaemic responses, but as part of these studies they observed that portions of apple and bread containing 53 g available carbohydrate elicited lower glycaemic responses than 53 g glucose in patients with diabetes. Calculation of the area under the curve (AUC) from the

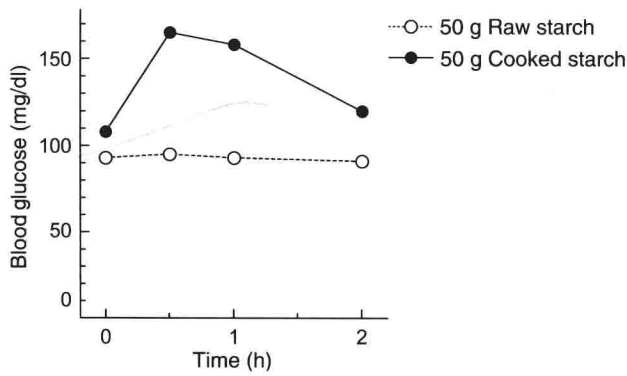

Fig. 1.5. Glycaemic response elicited by 50 g raw starch and 50 g cooked starch (250 g potato). Redrawn from Rosenthal and Ziegler (1929).

graphs of the data shown in this paper indicate that apple and bread elicited glycaemic responses 52% and 76% of that elicited by glucose, consistent with the GI values of these two foods, 38 and 71, respectively (Foster-Powell *et al.*, 2002).

1.2.2 Studies after 1970

After about 1970, authors began to be specifically interested in comparing the glycaemic responses elicited by different carbohydrates. Thus, study designs were more appropriate for this purpose, with groups of 8 to 20 subjects being studied with each subject testing each test meal. However, important details about the nature of the test foods and cooking methods used are sometimes lacking. In addition, the methods used to quantify glycaemic responses were evolving during this time and one can begin to see how differences in data analysis could influence markedly the interpretation of the results obtained.

Lütjens *et al.* (1975) compared the glycaemic responses elicited by 50 g carbohydrate portions of glucose, sucrose, bread and starch, in the form of potato. The way the potato starch was cooked and served is not indicated. The authors compared glucose and insulin concentrations at each point in time. They concluded that since neither the glucose nor insulin values showed any significant differences after the various carbohydrates, it was not important what type of carbohydrate is administered. However, the statistical analysis presented is not consistent with this conclusion because it shows that blood glucose concentrations at some time points differed significantly (Fig. 1.6). The authors did not calculate the areas under the blood glucose response curves (AUC), but doing so using the data presented indicates that sucrose, bread and potato, respectively, elicited glycaemic responses 76%, 71% and 53% that of glucose.

Crapo *et al.* (1977) published a series of papers in which the glucose and insulin responses elicited by 50 g carbohydrate portions of glucose, baked potato, rice, white bread and maize were determined in normal subjects, subjects with impaired glucose tolerance (IGT) (Crapo *et al.*, 1980) and subjects with type 2 diabetes (Crapo *et al.*, 1981). Although the same protocol was used in these papers, the method of data analysis differed. In the normal subjects, Crapo *et al.* (1977) found that the different carbohydrates elicited virtually identical total AUC values. Nevertheless, they concluded that different carbohydrate sources elicited different glycaemic responses on the basis of significantly different glucose concentrations between 30 and 60 min. For the studies in subjects with IGT and diabetes, Crapo *et al.* (1980, 1981) calculated incremental AUC (IAUC) values. In the paper with IGT subjects, there was no statistical comparison of the AUC values, but the authors noted that the responses elicited by bread, rice and maize were 25% to 36% lower than those elicited by glucose and potato. In the paper in subjects with diabetes, Crapo *et al.* (1981) found significant differences in AUC between the different carbohydrate sources, with bread, rice and maize eliciting glucose responses 11–41% lower than potato and glucose. When comparing the results of these studies, the authors did not compare the relative glycaemic responses (RGR), but pointed out that the difference in glycaemic response between foods in subjects with IGT or diabetes were two to three times greater than those in normal subjects (Crapo *et al.*, 1980, 1981).

Figure 1.7 shows my recalculation of the results of the studies by Crapo *et al.* (1977, 1980,

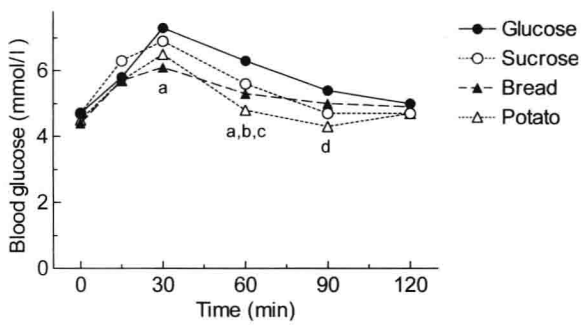

Fig. 1.6. Mean blood glucose responses elicited by 50 g carbohydrate from glucose, sucrose, bread or potato in nine normal subjects. Statistically significant differences ($P < 0.05$): a, glucose vs bread; b, glucose vs potato; c, sucrose vs potato; and d, sucrose vs bread. Redrawn from Lütjens *et al.* (1975).

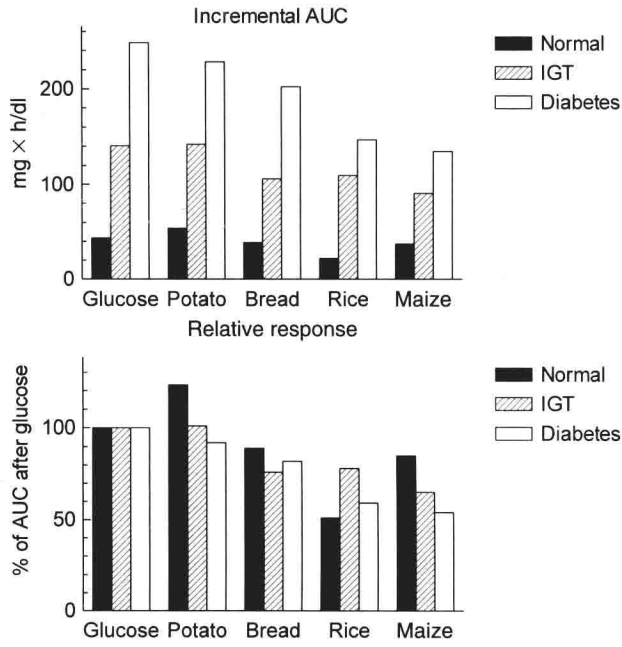

Fig. 1.7. Mean incremental areas under the curve (top) and relative responses (bottom) elicited by 50 g carbohydrate portions of glucose, baked potato, bread, rice and maize in normal subjects, and subjects with IGT and type 2 diabetes. Recalculated from Crapo *et al.* (1977, 1980, 1981).

1981). The top of Fig. 1.7 shows the IAUC values calculated from the mean glucose concentrations; it can be seen that subjects with IGT and diabetes have AUC values which are three- and fivefold greater than normal subjects, and that the differences in AUC between foods is larger in subjects with diabetes and IGT than without. However, when expressed relative to the AUC after glucose, the glycaemic responses elicited by potato, bread, rice and maize were similar in the different groups of subjects (Fig. 1.7, bottom). Indeed, using the SD of the relative responses of potato, rice, maize and bread as the measure of differences in glycaemic response between foods, the difference in relative response between foods is smaller in subjects with diabetes (SD = 18) and IGT (SD = 15) than in normal subjects (SD = 29).

Vaaler *et al.* (1980) measured the plasma glucose and insulin responses elicited by 300 kcal portions of glucose, potato, rice, white bread and brown bread. Since the test meals contained equal amounts of energy, their content of carbohydrate varied from 58 to 75. The authors did not calculate the AUC, but doing so from the mean glucose concentrations given, results in mean IAUC values of 111, 70, 77, 49 and 64 mg × h/dl, respectively, for glucose, potato, rice, white bread and brown bread. Thus, the responses of potato, rice, white bread and brown bread, expressed as a percentage of that for glucose, were 62%, 70%, 44% and 58%, respectively. However, these values cannot be considered approximations of the GI because the test meals contained different amounts of carbohydrate. After adjusting for this, using the regression equation shown in Fig. 4.1, the approximate 'GI' values of these four foods becomes 64, 72, 50 and 64, respectively.

The largest number of foods tested for glycaemic response prior to the GI was reported by Otto's group who expressed the glucose AUC elicited by 25 g carbohydrate portions of different foods in subjects with diabetes as a percentage of that elicited by 25 g glucose. This is nearly the same methodology as used for GI. However, at the time we published the first paper on GI (Jenkins *et al.*, 1981a) we were unaware of this work because, apart from a few abstracts (Schauberger *et al.*, 1977), only one paper was ever published by the group (Spaethe *et al.*, 1972). In addition, Otto used the information differently than we conceived; we envisaged the GI as being used as an aid to selecting carbohydrate foods to enable a high carbohydrate intake to be maintained with minimal glycaemic impact. By contrast, Otto's concept was to adjust the amounts of different carbohydrate foods consumed so as to maintain