Labelling of foods for glycaemic index — advantages and problems

C S Venter, M Slabber, H H Vorster

Food labelling has two aims — to inform the consumer of the composition of the food, and to assist him/her in the selection of a healthy diet. Labelling of foods and food products for the glycaemic index (GI) informs consumers how to choose carbohydrate-containing foods or beverages based on physiological effects. Requirements of a possible GI label should be rigorously examined and recommendations made to relevant bodies for consideration. Only foods/beverages that make a meaningful contribution to dietary carbohydrate intake should be labelled. Clear directions are needed regarding standardised methodology in accredited laboratories, including clarity on issues such as the reference (standard),

The glycaemic index (GI) is a classification of the blood glucose-raising potential of carbohydrate foods. It is defined as the incremental blood glucose area following the test food, expressed as the percentage of the corresponding area following a carbohydrate-equivalent load of a reference product.¹ With white bread as reference, GIs range from less than 20% to approximately 120%. The main causes of these large differences in GI are differences in the rate of digestion or absorption of the carbohydrates, as well as the digestive/fermentation fate of carbohydrates in the small and large gut (to glucose versus short-chain fatty acids).

Low-GI foods release glucose to the blood at a slower rate.² There are a number of long-term implications of altering the rate of breakdown and absorption, or GI, of dietary carbohydrate. There is good evidence that low-GI foods improve overall blood glucose control in people with type 2 diabetes,³⁶ reduce serum lipids in people with hypertriglyceridaemia,⁷ and improve insulin sensitivity.⁸⁹ In addition, low-GI foods are associated with high high-density lipoprotein (HDL) cholesterol¹⁰ and reduced risk for the development of type 2 diabetes and cardiovascular disease.¹⁰⁻¹² Furthermore, it has been shown that when low-glycaemic carbohydrates are incorporated into an energy-deficient diet,

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Department of Human Nutrition, University of the Free State, Bloemfontein M Slabber, PhD total ('available') carbohydrate of the test food, number and characteristics of experimental subjects, capillary versus venous blood samples, analytical method for determination of blood glucose value and method of calculation of the area under the glucose curve. Furthermore, it will have to be decided whether the label should indicate low, moderate or high GI with the reference ranges or the specific number, using the standard deviation or 95% confidence interval to illustrate individual variation. The short- and long-term effects of low- and high-GI foods, and the place of each in the context of both the other nutrient contributions of the food and the total diet, should be understood by the consumer. This is a major challenge.

there is a greater fall in insulin resistance than can be accounted for by weight loss alone.¹³ For athletes, low-GI carbohydrate foods have been recommended before prolonged exercise to promote carbohydrate availability.¹⁴ Moderate- to high-GI foods and drinks are considered appropriate during prolonged exercise, and high-GI carbohydrates are the best choice to enhance glycogen storage after exercise by promoting greater glucose and insulin response.¹⁴

These effects prompted the joint Food and Agricultural Organisation/World Health Organisation (FAO/WHO) expert consultation on 'Carbohydrates in human nutrition'¹⁵ and more recently Riccardi and Rivellese⁹ to endorse the usefulness of the GI in diet planning. However, according to Franz *et al.*¹⁶ the usefulness of low-GI diets in persons with type 1 diabetes is controversial. Therefore, the American Diabetes Association is of the opinion that the evidence of long-term benefit from the use of low-GI foods is not sufficient to recommend low-GI diets as a primary strategy in food/meal planning.¹⁷

Although the benefits of using GI to counsel people are still a matter of debate¹⁸ and legislation regarding food labelling has not yet been approved, the GI already appears on the label of some South African beverages. Therefore, guidelines are needed in terms of public use of the GI concept through health and education professionals. Many issues have to be resolved in this regard. This article attempts to highlight some of the issues to be debated in order to avoid confusion and to educate consumers to see the GI in context of the total diet and other nutrients of the foodstuff.

Advantages of labelling

Food labelling has two main aims - to inform consumers of the composition of the food, and to assist them in the selection of a healthy diet. These two aims are not always easy to reconcile because the health benefit of different carbohydratecontaining foods cannot readily be communicated simply from a description of their composition. The GI can be used, in conjunction with information on food composition, to guide food choices. Labelling for GI informs consumers how to choose carbohydrate-containing foods based on expected physiological effects (e.g. blood glucose-raising potential). Traditionally, foods containing significant amounts of carbohydrates have been categorised according to the structural classification of the principally occurring carbohydrate. This has led to the categorisation of carbohydrate-containing foods as 'simple' (containing mono-, di- and oligosaccharides) and 'complex' (containing polysaccharides or starches). Although this classification system may have been intended as a convenient education tool for the lay person, it led to the inaccurate belief that simple carbohydrates cause large and rapid changes in blood glucose levels and are generally not nutrient-rich whereas complex carbohydrate foods are digested and absorbed more slowly, producing a flatter and more sustained blood glucose response, and contain significant amounts of other nutrients, including dietary fibre. This is a major oversimplification and is inaccurate regarding the effect of carbohydrate-rich foods on blood glucose levels. For example, several carbohydrate-rich foods containing predominantly sugars (e.g. fruit and low-fat fruit yogurt) produce a flattened blood glucose curve, and provide protein, fibre, micronutrients, a large array of nonnutrient physiologically active compounds and very little fat. On the other hand, a number of foods high in complex carbohydrates (e.g. bread and potatoes) produce a high blood glucose response, and might be considered less nutritious. Clearly, another system is needed to describe blood glucose responses to carbohydrate-rich foods. Labelling foods for GI may eliminate problems with understanding of the terms 'complex' and 'simple' carbohydrate, terminology which should not be used.15

Problems of GI labelling

Foodstuffs to be labelled

Foods/beverages that make a meaningful contribution to dietary carbohydrate intake should be labelled. GIs are now available for a considerable number of carbohydrate foods.¹⁹ The GIs of some groups of carbohydrate foods — starchy foods, fruit and milk products — are given in Table I. Although there are traditional indigenous starchy products with a low GI, such as legumes and pasta, it is evident that the major

sources of carbohydrates in a Western diet are found in the upper GI range. That is, most potato products, common bread and breakfast cereals have high GIs, often higher than sucrose. A high dietary fibre content is not a prerequisite for low GI properties, and the naturally occurring levels of viscous fibre in common cereals have only a marginal impact on glycaemia.²⁰ Wholemeal cereal products may therefore produce GIs as high as those of white bread. However, dietary fibre as part of an intact botanical structure, as in barley and pumpernickel bread, may be effective in reducing glycaemia.²⁰

In order to implement a well-balanced low-GI diet, a much wider range of low-GI products will be required. In particular, whereas there are many options for including low-GI foods at lunch and dinner, few such alternatives are available among most common breads, muffins, scones and breakfast cereals on the market,²¹ a fact that seriously limits efforts to reduce dietary GI. Moreover, the GI features of breakfast may be particularly important.²¹ Metabolic control improved significantly in subjects with type 2 diabetes simply by exchanging the conventional high-GI breakfast for a low-GI one.²² There is also evidence from studies in healthy subjects that a low-GI breakfast may have beneficial metabolic effects extending beyond the postprandial phase.^{21,23} The GI features of breakfast may be more crucial for the glycaemic response at lunch than is the GI of the lunch per se. The technological means exist to lower the GI of starchy foods significantly, for example by choice of raw material and/or optimising the processing conditions.²¹ The development of low-GI products is a challenge to the food industry.

Labelling low-carbohydrate foods for GI is not meaningful. Some foods are so low in carbohydrate that they have no measurable effect on blood sugar levels. It is suggested that only foods containing 10 g carbohydrate per 100 g portion or supplying 40 - 50% of energy from carbohydrate should be labelled. Furthermore, consumers should be educated that the GI of food is not the only factor that will determine whether the food should be included in the diet or not. Some low-GI foods may not be a good choice because they are high in fat and/or low in other nutrients and phytochemicals.

Composite foods and food products

Composite foods, for example tinned butter bean soup with added modified starch and sugar, present a special problem. Food scientists/nutritionists will have to decide whether the GI of composite food products should be determined in physiological experiments, or whether they may be calculated on the basis of their composition and published values, using the same principles as in calculating the GI of mixed meals.¹⁵

Wording and symbols on the label

The short- and long-term effects of low- and high-GI foods, and the place of each in the context of both the other nutrient



Table I. The glycaemic index of son	ne popular foods ¹⁹
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Table I. The glycaemic index of some popular foods ¹⁹ GI GI GI				
	Food (gluce		(bread = 100)	
High GI	Glucose	97	138	
(70 and above)	Lucozade	95	137	
	Cornflakes	84	119	
	Cocopops	77	110	
	Rice Krispies	82	117	
	Shredded wheat	83	118	
	Instant mashed potato	83	118	
	Baked potato	85	121	
	French fries	75		
	Maize meal porridge	74	106	
	White bread	70	101	
	Wholewheat bread	69	99	
	Weetbix	70	101	
	Watermelon	72	103	
	Honey	73	104	
	Rice (low amylose)	87	124	
	Pumpkin	75	107	
	Ensure (vanilla)	75	107	
Moderate GI	Grapenuts	67	96	
(55 - 69)	One-minute oats	66	94	
	Oat porridge	61	87	
	Oat bran (raw)	55	78	
	Muesli (not toasted)	56	80	
	Muffins	62	88	
	Soft drinks	68	97	
	Orange juice	57	74	
	Sucrose	65	92	
	Popcorn	55	79	
	Ice cream	61	87	
	Banana, over-ripe	52	74	
	Mango	55	79	
	Brown rice	55	80	
	Basmati rice	58	83	
	Couscous	65	93	
Low GI	All-Bran	42	60	
(below 55)	Build-Up (Nestle)*	51	74	
(201011-00)	Muesli, toasted	43	61	
	Special K	54	77	
	Wheat, whole kernel	41	59	
	Barley	25	36	
	Bulgar	48	68	
	Banana, under-ripe	30	43	
	Cherries	22	32	
	Grapefruit	25	36	
	Grapes	43	62	
	Orange	43	62	
	Peach, fresh	28	40	
	Pear	33	47	
	Baked beans	40	57	
	Butter beans	31	44	
	Chickpeas	33	47	
	Kidney beans	27	42	
	Lentils	29	41	
	Soya beans	18	25	
	Spaghetti	41	59	
	Macaroni	45	64	
	Instant noodles	47	67	
	Green peas	48	68	
* Determined by the Nutrition Research Group, Potchefstroom University.				
	e ryunnon Research Group, ro	Acheistrooli	i Oniversity.	

contributions of the food and the total diet, should be understood by the consumer. This will influence the wording of the claim, which should not be misleading.

In Australia, the most advanced country in terms of knowledge of the GI of foods and publicising the information to consumers, a GI symbol programme is being jointly developed by the University of Sydney, Diabetes Australia, and the Juvenile Diabetes Foundation.²⁴ Foods that meet specific nutrition criteria and have been tested for their GI by an accredited laboratory will be authorised to display the symbol. The actual GI value and a short explanation will appear next to the nutrition information box. A similar GI symbol could be designed for South Africa. Furthermore, it will have to be decided whether the label should indicate low, moderate or high GI with the reference ranges or the specific number, using the standard deviation (SD) or 95% confidence interval (CI) as determined in an accredited laboratory. For example, the GI of tinned butter beans could be 'low', or the GI indicated as 41 (SD 10.9). Because of variance in food composition, external and internal environments and genotype, variation in glycaemic response occurs between and within persons. Variety in response presents a major challenge. A possible solution might be to use terms such as 'usually' or 'generally'. For example, the GI of a product can be labelled as being 40 (95% CI 35.6 - 50.5), with an explanation that the user can expect, with 95% confidence, that the GI of the product will fall between 35.6 and 50.5, with a usual value of about 40.

Methodology for determining GI

One of the major problems regarding labelling foods with GI values is the lack of standardised methodology among different researchers in determining the GI. Several factors contribute to the variability of the glycaemic response and are indicated in Table II. To circumvent this problem most published GI tables have provided conversion factors or have presented tables using different methods alongside each other.²⁵

Before the GI of a specific food is determined for labelling purposes standardisation of methodology is of utmost importance to render the GI universally applicable and acceptable.²⁷ Furthermore, trained researchers in the wellcontrolled experimental environment of an accredited laboratory should perform the tests with accuracy and precision to warrant reliability and comparability of measurements. Protocols should also comply with research ethical standards. In the following paragraphs a detailed description of current method, based on the available scientific literature, is discussed.

Description of current method

Portions of test foods and a standard food (glucose or white bread) containing 50 g available carbohydrate are fed to

Table II. Factors contributing to the variability of glycaemic responses

Inter- and intra-individual variation in blood glucose ${\rm response}^{\rm ^{25,26}}$

Different methods to calculate the area under the blood glucose response $\mbox{curve}^{\mbox{\tiny 27}}$

Use of different standard foods¹⁹

Definition of available carbohydrate portion of food^{1,28}

Practised standards of sample food preparation²⁹

Volume and type of drinks consumed with test meals²⁶

Method of blood sampling³⁰

Biochemical assay³¹

Number of subjects used¹⁵

Preparation of subjects before test days²⁷ Length of time over which studies are conducted³²

healthy or diabetic subjects in random order on separate occasions after an overnight 10 - 12-hour fast.³³ The same subject should repeat the standard food test at least three times, with the mean result used as the reference to calculate the GIs of the test foods.¹⁵ Blood samples are taken during fasting and at regular intervals of up to 120 minutes for healthy subjects, and 180 minutes for diabetic subjects. The normal dose of insulin or oral hypoglycaemic agent, if applicable, is taken after the fasting blood sample and 5 - 10 minutes before ingestion of the test meal.³³

Subjects should be prepared for the test 3 days in advance by ingesting a diet rich in carbohydrate (60% of total energy) and with 20% of the energy derived from both protein and fat.²⁷ This diet ensures optimal substrate induction of enzyme synthesis and activation²⁷ and prevents ketogenesis and gluconeogenesis, which may occur after a period of carbohydrate restriction.³⁴ Subjects should consume a standard pre-evening meal with an average of 50% of the total energy from carbohydrates, 30% from fat and 20% from protein to standardise potential second-meal effects.³⁵ During the course of the study, subjects should consume a weight-maintenance diet,³⁶ and normal or usual physical activity should be maintained.

Subjects

Many subject characteristics affect the glycaemic response to a given food including health status, type and treatment of diabetes mellitus, body mass index (BMI), age, gender, ethnicity and background knowledge of GI studies.³⁷ Godsland³⁸ states that volunteers from the lay public show an increased within-individual variance compared with laboratory staff who are more aware of the importance of following a specific protocol.

Within- and between-subject variation

The variability of the glycaemic response for a given food for

any one individual is similar to that seen for the oral glucose tolerance test.²⁶ In three recent studies within-subject variation of healthy subjects to glucose varied from $19\%^{39}$ to 63%,⁴⁰ while a fairly consistent picture of fasting plasma glucose variability of 14 - 20% in type 2 diabetic patients was shown.⁴¹ These results are in agreement with those of similar studies.^{42,43}

There is also variation between individual subjects in the glycaemic response to a food and in the GI of the same food. According to Wolever²⁶ the variability between individuals is larger than within individual subjects. While some studies confirmed this^{44,45} phenomenon, others found greater within-than between-subject variation in both healthy³⁹ and type 2 diabetic patients.⁴¹ The latter findings have an important practical application in GI determinations for research or labelling purposes as they suggest that it would not be necessary to use the same subjects repeatedly and that larger groups of subjects could be used less often, provided that the group is homogeneous.

Subjects' health status, age, ethnicity, BMI and kind of treatment (for diabetic patients) are all factors that might contribute to variations and will subsequently be discussed.

Health status

When determining the GI of a specific food, subjects from the healthy population or type 1 or type 2 diabetic subjects might be included for determining the GI of a specific food. Although studies on different population samples have resulted in different GI values for the same food, the rank order of GIs for different foods has been found to be essentially the same between healthy and diabetic subjects,^{36,46} although higher GIs have been reported in type 2 diabetics.⁴⁷ Ideally, repetitive tests on all three groups would give more meaningful and useful results. However, if a specific food formula or feed is developed for a specific target population and labelling purposes, the health status of specific characteristics of subjects included should preferably be in agreement with those of the target population, i.e. patients with type 1 diabetes or athletes.

Before subjects can be typified as either healthy or diabetic, their individual glucose tolerance should be determined using the standard 2-hour glucose tolerance test.⁴⁸

In healthy subjects no drugs should be taken that may affect glucose tolerance. Type 2 diabetic patients included in GI studies should be well controlled as variability in measurement has been found to be larger in poorly controlled diabetics.⁴⁶ Glycated haemoglobin concentrations that are indicative of the time-averaged blood glucose concentration over the past 1 - 3 months should be measured⁴⁸ and be within the acceptable range of 7 - 8% to ensure that diabetic subjects are well controlled. Furthermore, serum and urine creatinine concentrations should be in the normal ranges to ensure that subjects have normal renal function. To decrease variability between type 2 diabetics, they should be treated with diet



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alone or diet and metformin rather than sulphonylureas as intensive glucose control with metformin appears to decrease the risk of diabetes-related endpoints in overweight diabetic patients. Metformin is also associated with less weight gain and fewer hypoglycaemic attacks than are sulphonylureas.⁵⁰

Age

With increasing age, dietary changes⁵¹ and lower physical activity⁵² may affect glucose tolerance. However, Wolever and co-workers⁵³ found no significant differences in glycaemic responses between adults and children.

Ethnicity

There is a lack of data on the effect of ethnicity independent of the background diet. Walker and Walker²⁸ could find no significant differences in blood glucose response between different race groups, but Summerson and co-workers⁵⁴ have shown race-related differences in the control of diabetes in adults. Therefore, in studies using diabetic subjects it might be advisable to use subjects from the same ethnic group only.

Gender

Rasmussen and co-workers⁵⁵ failed to show a significant influence of gender on glycaemic responses in middle-aged male and female type 2 diabetic subjects.

Body mass index

Although the presence of obesity as a variable has not been studied adequately, obese subjects may show altered glucose tolerance due to insulin resistance that is associated with abdominal obesity.⁵⁶ Therefore, subjects in the normal BMI range of 18.5 - 24.9 kg/m²¹⁵ are included in a non-diabetic study sample. However, approximately 80% of type 2 diabetic subjects are obese or have a history of obesity at the time of diagnosis.⁵⁷ Therefore, a reference BMI range of 20 - 35 kg/m² will be more representative of the general type 2 diabetic population when determining the GI.

It can be concluded that comparison of the absolute glycaemic responses both within- and between subjects is unreliable.⁵⁸ However, the problem of within- and betweensubject variation is diminished when the glycaemic response to any given food is indexed to the GL.²⁵ Moreover, by expressing the glycaemic response as the GI, the variation that may occur with age, gender, BMI, and race as well as glucose tolerance and its treatment are also controlled for.⁵⁹ However, to optimise results the study population should be homogeneous with regard to age, weight, height and BMI.^{38,58,60}

Number of subjects

Homogeneous, intensive studies on the GI have generally used 6 - 20^{44,61} subjects. Truswell⁶² suggests that it is essential to test at least 10 subjects to obtain reasonable values in GI studies.

Recently power calculations based on the smallest SD have shown that at least 24 subjects are necessary to determine the GI of bread with 80% accuracy in both healthy individuals³⁹ and type 2 diabetic patients.⁴¹ However, Nell³⁹ has shown that larger groups of subjects (24 - 90) should be used if foods on a scale of 0 - 100 are consistently classified as having a low (0 -55%), moderate (> 55 - 70%), or high (70+%) GI (using glucose as standard/reference).

Standard food

Glucose dissolved in water was initially used as standard food, and was assigned a value of 100.1 White bread was later regarded as being more physiologically standard.²⁶ Subjects may experience the sweetness of glucose as nauseating and the high osmotic load may cause delayed gastric emptying which may affect the results.63 Furthermore, glucose contains no other macronutrients whereas almost all natural foods contain some fat and protein. Protein stimulates insulin secretion and the serum insulin response may therefore be larger after bread than after oral glucose, despite lower blood-glucose responses.⁶⁴ Fat delays gastric emptying and small intestinal motility.65 Wolever and co-workers43 proved that results from studies with different standard foods might be compared if adjusted proportionally. Glucose-based values are multiplied by 1.38 to convert them to bread-based values since the glycaemic response of glucose is, on average, 38% greater than that of bread.²⁶

Glucose as standard food later proved to give higher variability (2 - 3 times) in glycaemic response and it was suggested that starchy meals may allow more precise assessment of carbohydrate tolerance.⁴³ The magnitude of these findings was investigated in three recent studies of healthy^{39,40} and type 2 diabetic subjects⁴¹ respectively. It was shown that using white bread as standard in healthy or 'normal' subjects would ensure the lowest variation in the glycaemic responses.³⁹ However, in type 2 diabetic subjects glucose proved to be a more consistent test meal in GI calculations.⁴¹

If glucose is used as standard it should be purchased in bulk and selected from the same batch. Fifty grams of glucose powder should be weighed in separate portions and dissolved in 200 - 250 ml water. Glucose solutions should be served at the same temperature. If white bread is used as standard food, each sample should provide 50 g available carbohydrate as determined by food composition tables. To avoid differences in the quality and quantity of carbohydrate load, all bread used should come from the same batch and supplier. Bread crusts must be removed because of the influence of the Maillard reaction on the availability of carbohydrate from the crust.66 Bread is not a consistent food and it goes stale, losing water when standing at usual indoor temperatures.62 White bread ingested on different days as standard food should be frozen and thawed according to methods prescribed for test foods to ensure uniformity.

Availability of carbohydrate

Food composition tables are used to determine the nutritional composition of different test foods to ensure that 50 g of 'available' carbohydrate is ingested by subjects. If the carbohydrate content of the test food is low, 25 g of the test food should be used with 25 g of available carbohydrate as glucose or white bread as standard.^{1,67} Bulky foods like carrots, for example, contain about 5 g carbohydrate/100 g food when raw and 4 g carbohydrate/100 g when boiled.⁴² To take in 50 g of available carbohydrate would require the unphysiological amount of 1 kg carrots or more.⁴² The term 'available' carbohydrate is unfortunate; it means total carbohydrate minus dietary fibre content. Resistant starch is 'available carbohydrate' (available in the colon) and should be included in the 50 g carbohydrate portion.²⁴

Test foods

Test foods are given in random order on separate days and should provide 50 g of available carbohydrate. Test foods should be purchased in bulk and selected from the same batch to ensure uniformity of shelf life and similarity of management during production, maturity and processing procedures. Cooked test foods should be prepared beforehand, frozen in portioned amounts in plastic bags or sealed containers at –18 to –30°C. Required foods should be removed from the freezer on the night before the test session, thawed at room temperature and reheated if necessary in a microwave oven at precise times.³⁹ A digital scale is used to weigh individual dry food portions into precise portions containing 50 g carbohydrate each. Standardised equipment, cooking methods and utensils should be used to prepare cooked food products.

Volume and type of drinks consumed with test meals

An accompaniment could be given with dry test foods, otherwise they might be unpleasant to consume. This accompaniment should be low in energy, very low in carbohydrate and kept the same for different foods compared.⁶² For labelling purposes clear statements should be made regarding the accompaniments used in the experimental protocol.

Blood sampling

The GI was based on measurement of glucose responses in whole capillary finger-prick blood due to the simplicity and non-invasiveness of the method of blood sampling, allowing for extensive screening of foods.¹²⁶ Venous blood was later used in GI tests. Glycaemic responses in capillary blood are greater than those in venous blood or plasma and therefore may allow for detection of smaller differences in glycaemic responses to different foods.^{15,68} Studies in which GIs were calculated both

from analyses of capillary and venous blood have shown either no differences in GI values³⁰ or differences after ingestion of some foods.⁵³ Venous blood from an antecubital vein is recommended because of higher glucose values of 1.1 - 3.8 mmol/l in capillary than venous blood after ingestion of glucose.⁶⁹ Current recommendations are that capillary blood sampling is preferred to determining the GI, but venous blood sampling is also acceptable.¹⁵ Blood glucose concentrations are then determined with a glucose oxidase peroxidase reagent. Wolever and co-workers³¹ collect capillary blood samples (110 -200 µl) into tubes containing 410 µg of sodium fluoride and 250 µg of potassium oxalate (as anticoagulants) before the samples are frozen. An automatic glucose analyser (such as the 27AM glucose analyser, Yellow Springs, OH) is used for accurate glucose determination.

Calculation of the area under the curve (AUC) (Fig. 1)

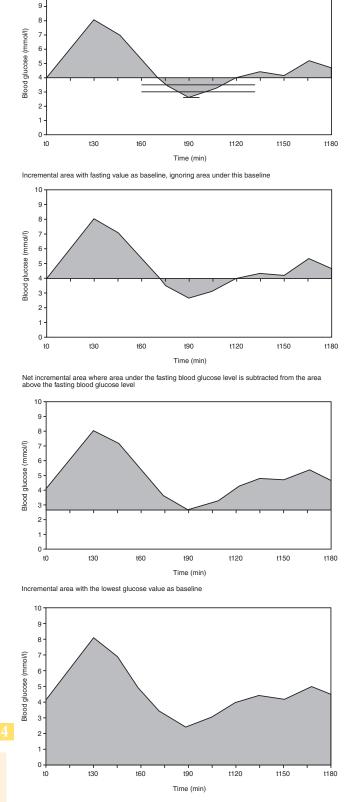
Four different methods to calculate the incremental AUC have been documented by different research groups.^{15,26,27}

1. Incremental AUC. Wolever²⁶ defined the incremental area as the area under the glucose response curve with the fasting glucose value as baseline, and considers it the only method to calculate the GI.⁷⁰ The formula is as follows: At/2 + At + (B - C)A)t/2 + Bt + (C - B)t/2 + Ct + (D - C)t/2 + Dt + (E - D)t/2, etc. where A, B, C, D, and E represent positive blood-glucose increments, and t is the time interval between blood samples. If the blood glucose increment D is positive (i.e. greater than the fasting value) and E is negative (i.e. less than fasting value), only the area above the fasting value (between D and E) is used. If the value E occurs t minutes after value D, a straight line drawn between points D and E equals the fasting bloodglucose value at a time T after D, where T < t. Thus the area above the curve is given by DT/2. Because $T/t = D/(D + \{E\})$ (where $\{E\}$ = absolute value of E), thus, T + Dt(D + $\{E\}$). Therefore $DT/2 = D^2t/2(D + \{E\})$. The overall equation simplifies to Area = $(A + B + C + D/2)t + D^2t/2(D + \{E\})$.

This equation ignores any area under the fasting bloodglucose value. However, in an abnormal physiological condition as frequently seen in hypoglycaemic non-diabetic subjects, an undershoot of blood-glucose values occurs after initial high glucose concentration. Therefore, this method will not always give a true representation of the glucose response to specific foods.

2. Net incremental AUC. The net incremental AUC is a variant of Wolever's method and was used by several researchers.⁷¹⁻⁷⁴. In this method the area under the fasting blood-glucose curve is subtracted from the area above the fasting blood-glucose curve. Again, a difference between the incremental and the net incremental areas will only be detected in cases where the postprandial blood-glucose concentration drops below the fasting value.²⁶





Total area under the curve, i.e. the area under the blood glucose curve and above a blood glucose value of zero

Fig. 1. Different methods for calculating the area under the glucose response curve (adapted from Vorster et al.²⁷).

3. Incremental area with the lowest glucose value as baseline (AUCmin). Vorster and co-workers²⁷ proposed an incremental area with the lowest glucose value as baseline to calculate the GI. They have shown that the sharp rise in the curve when glucose is used as the standard results in a hypoglycaemic response at approximately 90 minutes in a large number of healthy subjects, which is absent when slowly absorbed ('lente') carbohydrate is consumed. Therefore, in healthy subjects experiencing hypoglycaemia or blood glucose levels below the fasting level, the method ignoring these will not reflect the true picture. According to this method, hypoglycaemia is regarded as a physiologically undesirable state, as is hyperglycaemia. If blood glucose values remain above the fasting value (as can be expected in diabetics) this method will give the same results as the incremental AUC of Wolever et al.⁷⁰

4. Total AUC. Reaven and co-workers⁷⁵ used the total AUC that is defined as the area under the glucose curve and above a blood glucose value of zero. Wolever²⁶ has shown that this method will give values 3 - 10 times greater for normal subjects and 2 - 5 times greater for diabetics than the incremental area for the same data. This method has been criticised^{26,32} as being insensitive for detecting differences between the postprandial glycaemic response of different meals.

The main source of error in determining the GI could be the method of calculating the AUC. Confusion evolved when per cent differences between the 'total' AUCs for different foods were compared with the differences in their GIs.^{76,77} Thornburn *et al.*⁶⁷ also queried whether the base of the AUC should be a line extended to the right on the graph from the fasting blood glucose concentration, or a line drawn horizontally, which may occur after the peak rise, between 2 and 3 hours after the test meal. The former is usually applied. Recently, Nell³⁹ found that the AUC^{min} method showed less variation than the incremental AUC method and suggested that the AUC^{min} method is a more relevant physiological method to use in GI calculations.

It is clear that there is still no agreement between researchers on this issue. Moreover, authors have not always made it sufficiently clear exactly how the AUC was calculated. Current knowledge indicates that GI calculations should be done with the AUC^{min} method. However, methods used to analyse data should be documented clearly.

Conclusions and recommendations

There is an important body of evidence in support of the therapeutic potential of a low-GI diet in persons with type 2 diabetes and dyslipidaemia. There are also indications of a preventive role against type 2 diabetes and cardiovascular disease. To exploit the metabolic potential of a low GI fully, the requirements of a possible GI label should be rigorously examined and recommendations made to international bodies

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for consideration. Foods that meet specific nutrition criteria and have been tested for their GI by an accredited laboratory, may be eligible to label the GI value and give a short explanation near the nutrition information panel. This may educate the consumer about the use of the GI concept in a balanced diet and challenge the food industry to reformulate and develop low-GI versions of their products (for the benefit of persons with diabetes, cardiovascular disease and metabolic syndrome) and high-GI versions (for the benefit of physically active persons). In this way, consumers and health professionals will be able to make informed choices about the quality of carbohydrate in foods. However, issues such as the potential to abuse the concept and to mislead consumers will have to be debated before labelling for the GI can be implemented.

Therefore, permission or legislation to label the GI of foods, food products and beverages should be accompanied by clear instructions/directions regarding the following: (*i*) which foods/beverages/products may be labelled (minimum of 10 g carbohydrate per serving/portion, or 40 - 50% carbohydrate energy as total energy); (*ii*) standardised methodology in an accredited laboratory, including clarity on issues such as the reference, total ('available') carbohydrate of test food, number and type of subjects, capillary versus venous blood, method of calculation of AUC; (*iii*) way to express the GI on products, using SDs and/or 95% CIs to reflect variability; and (*iv*) claims of benefits of low-GI values.

References

- Jenkins DJA, Wolever TMS, Taylor RH, et al. Glycemic index of foods: a physiological basis for carbohydrate exchange. Am J Clin Nutr 1981; 34: 362-366.
- 2. Björck I, Liljeberg H, Östman E. Low glycaemic index foods. Br J Nutr 2001; 83: S149-S155.
- Brand JC, Colagiuri S, Crossman S, et al. Low-glycaemic index foods improve long-term glycaemic control in NIDDM. *Diabetes Care* 1991; 14: 95-101.
- Wolever TMS, Jenkins DJA, Vuksan V, *et al*. Beneficial effect of low-glycaemic index diet in overweight NIDDM subjects. *Diabetes Care* 1992; **15**: 562-566.
 Frost G, Wilding J, Beecham J. Dietary advice based on the glycaemic index improves dietary
- Fronce, When g, Decembry Decembry 2 diabetic patients. Diabet Med 1994; 11: 397-401.
 Iarvi AE, Karlström BE, Granfeld YE, et al. Improved elycaemic control and lipid profile and
- Jarvi AE, Karlström BE, Granfeldt YE, et al. Improved glycaemic control and lipid profile and normalised fibrinolytic activity on a low-glycemic index diet in type 2 diabetic patients. *Diabetes Care* 1999; 22: 10-18.
- Jenkins DJA, Wolever TMS, Kalmusky J, et al. Low-glycemic index diet in hyperlipidemia: use of traditional starchy foods. Am J Clin Nutr 1987; 46: 66-71.
- Frost G, Leeds A, Trew G, Margara R, Dornhorst A. Insulin sensitivity in women at risk of coronary heart disease and the effect of a low glycemic diet. *Metabolism* 1998; 47: 1245-1251.
- Riccardi G, Rivellese AA. Dietary treatment of the metabolic syndrome the optimal diet. Br J Nutr 2000; 83: suppl. 1, 5143 - 5148.
- 10 Frost G, Leeds AA, Doré CJ, et al. Glycaemic index as determinant of serum HDL-cholesterol concentration. Lancet 1999; 353: 1045-1048.
- Salmerón J, Manson JE, Sampfer MJ, et al. Dietary fibre, glycemic load, and risk of insulindependent diabetes mellitus in women. JAMA 1997; 277: 472-477.
- Salmerón J, Ascherio A, Rimm EB, et al. Dietary fibre, glycaemic load, and risk of NIDDM in men. Diabetes Care 1997; 20: 545-550.
- Slabber M, Barnard HC, Kuyl JM, Dannhauser A, Schall R. Effects of a low-insulin-response, energy-restricted diet in weight loss and plasma insulin concentrations in hyperinsulinemic obese females. Am J Clin Nutr 1994; 60: 48-53.
- Burke LM, Collier GR, Hargreaves M. Glycemic index a new tool in sport nutrition? Int J Sport Nutr 1998; 8: 401-415.
- FAO/WHO. Carbohydrates in human nutrition: report of a joint FAO/WHO expert consultation. FAO Food Nutr Pap 1998: 66: 1-140.
- Franz MJ, Bantle JP, Beebe CA, et al. Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. *Diabetes Care* 2002; 25: 148-198.
- American Diabetes Association. Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications: Position statement. *Diabetes Care* 2002; 25: S50-S60.
- 18. Wolever TMS. The glycemic index: flogging a dead horse? Diabetes Care 1997; 20: 452 456.

- Foster-Powel K, Miller JB. International tables of glycemic index. Am J Clin Nutr 1995; 62: 871S-893S.
- Liljeberg H, Björck I. Bioavailability of starch in bread products. Postprandial glucose and insulin responses in healthy subjects and *in vitro* resistant starch content. *Eur J Clin Nutr* 1994; 48: 151-163.
- Liljeberg HGM, Åkerberg AKE, Björck IME. Effect of the glycemic index and content of indigestible carbohydrates of cereal-based breakfast meals on glucose tolerance at lunch in healthy subjects. Am J Clin Nutr 1999; 69: 647-655.
- Golay A, Koellreuter B, Bloise D, Assal J-P, Würsch P. The effect of muesli or cornflakes at breakfast on carbohydrate metabolism in type 2 diabetic patients. *Diabetes Res Clin Pract* 1992; 15: 135-142.
- Jenkins DKA, Wolever TMS, Taylor RH, et al. Slow release dietary carbohydrate improves second meal tolerance. Am J Clin Nutr 1982; 35: 1339-1346.
- Brand-Miller J, Gilberson H. Practical aspects of meal planning using the glycaemic index. FAO/Danone Vitapole Workshop. Glycaemic index and health: the quality of the evidence. Bandol, France: Danone Vitapole, 2001.
- Frost G, Dornhorst A. The relevance of the glycaemic index to our understanding of dietary carbohydrates. *Diabet Med* 2000; 17: 336-345.
- Wolever TMS. The glycaemic index. In: Bourne GH, ed. Aspects of some vitamins, minerals and enzymes in health and disease. World Rev Nutr Diet 1990; 62: 120-185.
- Vorster HH, Venter CS, Silvis N. The glycaemic index of foods: a critical evaluation. South African Journal of Food Science and Nutrition 1990; 2: 13-17.
- Walker ARP, Walker BF. Glycemic index of South African foods determined in rural blacks a population at low risk to diabetes. *Hum Nutr Clin Nutr* 1984; 38C: 215-222.
- Soh NL, Brand-Miller J. The glycaemic index of potatoes: the effect of variety, cooking method and maturity. *Eur J Clin Nutr* 1999; **53:** 249-254.
 Granfeldt Y, Hagander B, Björck I. Metabolic responses to starch in oat and wheat products.
- Granteidt Y, rlagander B, bjorck I. Metabolic responses to starch in oat and wheat products. On the importance of food structure, incomplete gelatinisation or presence of viscous dietary fibre. Eur J Clin Nutr 1995; 49: 189-199.
- Wolever TMS, Vuksan V, Eshuis H, *et al*. Effect of method of administration of psyllium on glycemic response and carbohydrate digestibility. *J Am Coll Nutr* 1991; **10**: 364-371.
 Gannon MC, Nuttal FQ. Factors affecting interpretation of postprandial glucose and insulin
- areas. *Diabetes Care* 1987; 10: 759-763.33. Wolever TMS, Jenkins DJA, Jenkins AL, *et al*. The glycemic index: methodology and clinical
- implications. Am J Clin Nutr 1991; 54: 846-854.
 Zilva JF, Pannal PR, Mayne PD. Clinical Chemistry in Diagnosis and Treatment. 5^a ed. London:
- Lloyd Luke, 1988: 226.
 Gresse A, Vorster HH. The glycaemic index and second meal effect of a typical African meal in black non-insulin dependent diabetic subjects. *South African Journal of Food Science and*
- Nutrition 1992; 4: 64-69.
 Crapo PA, Insel J, Sperling M, Kolterman OG. Comparison of serum glucose, insulin and glucagon responses to different types of complex carbohydrate in non-insulin dependent diabetic patients. *Am J Clin Nutr* 1981; 34: 184-190.
- Jenkins DJA, Wolever TMS, Wong CS, et al. Glycaemic responses to foods: possible differences between insulin-dependent and non-insulin dependent diabetics. Am J Clin Nutr 1984; 40: 965-970.
- Godsland IF. Intra individual variation: significant changes in parameters of lipid and carbohydrate metabolism in the individual and intra-individual variation in different test populations. Ann Clin Biochem 1985; 22: 618-624.
- Nell T. The variation and application of the glycaemic index of foods. PhD thesis, Potchefstroom University for Christian Higher Education, 2000.
- Aginsky J, Visser ME, Levitt NS. The inter- and intraindividual variation in glycemic response to glucose and white bread in healthy male students. *JEMDSA* 2000; 5: 53.
- 41. Kruger L, Slabber M, Joubert G, et al. The intra- and inter individual variation of blood glucose response to white bread and glucose as determined in patients with type 2 diabetes mellitus. South African Journal of Clinical Nutrition (in press).
- Oleerton RL, Playle R, Ahmed K, et al. Day to day variability of fasting plasma glucose in newly diagnosed type 2 diabetic subjects. *Diabetes Care* 1999; 22: 394-397.
- Wolever TMS, Vuksan V, Palmason C. Less variation of postprandial blood glucose after starchy test meals than oral glucose. *Nutr Res* 1996; 16: 899-905.
- Wolever TMS, Nuttal FQ, Lee R, et al. Prediction of the relative blood glucose response of mixed meals using the white bread glycemic index. Diabetes Care 1985; 8: 418-428.
- Wolever TMS, Csima A, Jenkins DJA, et al. The glycemic index: variation between subjects and predictive differences. J Am Coll Nutr 1989, 8: 235-247.
- Wolever TMS, Jenkins DJA. The use of the glycemic index in predicting the blood glucose response to mixed meals. *Am J Clin Nutr* 1986; 43: 167-172.
 Jenkins DJA. Wolever TMS. Buckley G. *et al.* Low glycemic index starchy foods in the diabe
- Jenkins DJA, Wolever TMS, Buckley G, et al. Low glycemic index starchy foods in the diabetic diet. Am J Clin Nutr 1988; 48: 248-254.
- Franz M. Medical nutrition therapy for diabetes mellitus and hypoglycaemia of nondiabetic origin. In: Mahan LK, Escott-Stumo S, eds. *Krause's Food, Nutrition, and Diet Therapy*. 10th ed Philadelphia: WB Saunders, 2000: 742-780.
- Society for Endocrinology, Metabolism and Diabetes of South Africa. Proposed guidelines for diagnosis and management of diabetes mellitus 2002. Minutes of the SEMDSA Annual General Meeting held on 8 April 2002, Lord Charles Hotel, Somerset West, Cape.
- UK Prospective Diabetes Study Group. Effect of intensive blood glucose control with metformin on complications in overweight patients with type 2 diabetes. *Lancet* 1998; 352: 854-865.
- Tessari P. Changes in protein, carbohydrate, and fat metabolism with aging: Possible role of insulin. Nutr Rev 2000; 58: 11-19.
- Fukagawa NK, Anderson JW, Hageman G, et al. High carbohydrate, high fibre diets increase peripheral insulin sensitivity in healthy young and old adults. Am J Clin Nutr 1990; 52: 524-528.
- Wolever TMS, Jenkins DJA, Kalmusky J, et al. Glycemic response to pasta: effect of food form, cooking and protein enrichment. Diabetes Care 1988; 9: 401-404.



ARTICLES

- Summerson JS, Konen JC, Dignan MB. Race related differences in metabolic control among adults with diabetes. S Afr Med J 1992; 85: 953-956.
- Rasmussen OW, Gregersen S, Dorup J, et al. Day to day variation of blood glucose and insulin responses in type 2 diabetic subjects after starch-rich meal. Diabetes Care 1992; 15: 522-524.
- Castillo MJ, Scheen AJ, Jandrian B, et al. Relationship between metabolic clearance rate of insulin and body mass index in a female population ranging from anorexia nervosa to severe obesity. Int J Obes Relat Metab Disord 1994; 18: 47-53.
- Marion JF. Nutritional care in diabetes mellitus and reactive hypoglycemia. In: Krause MV, Mahan LK, eds. *Krause's Food, Nutrition and Diet Therapy.* 8th ed. Philadelphia: WB Saunders, 1984: 479-509.
- Weyman-Daum M, Fort P, Recker B, et al. Glycemic response in children with insulindependent diabetes mellitus after high- or low-glycemic-index breakfast. Am J Clin Nutr 1987; 46: 798-803.
- Jenkins DJA, Wolever TMS, Jenkins AL, et al. The glycaemic index of foods tested in diabetic patients; a new basis for carbohydrate exchange favouring the use of legumes. Diabetologia 1983; 24: 257-264.
- Inoescu-Tirgoviste C, Popa E, Sintu E, et al. Blood glucose and plasma insulin responses to various carbohydrates in type 2 (non-insulin dependant) diabetes. *Diabetologia* 1983; 14: 80-84.
- 61. Venter CS, Vorster HH, Van Rooyen A, et al. Comparison of the effects of maize porridge consumed at different temperatures on blood glucose, insulin and acetate levels in healthy volunteers. South African Journal of Food Science and Nutrition 1990; 2: 2-5.
- 62. Truswell AS. Glycaemic index of foods. Eur J Clin Nutr 1992; 46S: S91 S101
- Thompson DG, Wingate DL, Thomas M, et al. Gastric emptying as a determinant of the oral glucose tolerance test. Gastroenterology 1982; 82: 51-55.
- Krezowski PA, Nuttal FQ, Gannon MC, et al. Insulin and glucose responses to various starchcontaining foods in type 2 diabetic subjects. *Diabetes Care* 1987; 10: 205-212.
- 65. Hunt SM, Groff JL. Advanced Nutrition and Human Metabolism. New York: West, 1990.

- Robinson CH, Lawler MR, Chenoweth WL, et al. Normal and Therapeutic Nutrition. 7th ed New York: Macmillan, 1986: 759.
- Thornburn AW, Brand JC, Truswell AS. The glycaemic index of foods. *Med J Aust* 1986; 144: 580-582.
- Jackson RA, Blick PM, Matthews JA, et al. Comparison of peripheral glucose uptake after oral glucose loading and a mixed meal. *Metabolism* 1983; 32: 706-710.
- Porte D, Sherwin RS. Ekkenberg and Rifkin's Diabetes Mellitus: Theory and Practice. 5th ed. USA: Appleton and Lange, 1997: 366.
- Wolever TMS, Jenkins DJA, Jenkins AL. The glycemic index: methodology and clinical implications. Am J Clin Nutr 1991; 54: 846-854.
- Bantle JP, Laine DC, Castle GW, et al. Postprandial glucose and insulin responses to meals containing different carbohydrates in normal and diabetic subjects. N Engl J Med 1983; 309: 7-12.
- Nuttal FQ, Moorandian AD, DeMarais R, et al. The glycemic effect of different meals approximately isocaloric and similar in protein, carbohydrate and fat content as calculated using the ADA exchange lists. Diabetes Care 1983; 6: 432-435.
- Gannon MC, Nuttal FQ, Krezowski PA, et al. The serum insulin and plasma glucose response to milk and fruit products in type 2 (non-insulin dependant) diabetic patients. *Diabetologia* 1986; 29: 784-791.
- Laine DC, Thomas W, Levitt MD, et al. Comparison of predictive capabilities of diabetic exchange lists and glycemic index of foods. *Diabetes Care* 1987; 10: 387-394.
- Reaven GM, Chen Y-DI, Golay A, et al. Documentation and hyperglucagonemia throughout the day in nonobese and obese patients with non-insulin dependant diabetes mellitus. J Clin Endocrinol Metab 1987; 64: 106-110.
- Coulston AM, Hollenbeck CB, Swiskocki ALM, et al. Effect of source of dietary carbohydrate on plasma glucose and insulin responses to mixed meals in subjects with NIDDM. Diabetes Care 1987; 10: 395-400.
- Hollenbeck CB, Coulston AM, Reaven GM. Glycemic effects of carbohydrates: a different perspective. Diabetes Care 1986; 9: 641-647.

A community-based growth monitoring model to complement facility-based nutrition and health practices in a semi-urban community in South Africa

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Objective. To assess the feasibility of a community-based growth monitoring model in alleviating the shortcomings in health and nutrition surveillance of preschool-aged children as practised by the health services.

Method. Baseline community and health facility practice surveys and interactive workshops with the community were conducted before the study. Eleven women were trained to drive the community-based growth monitoring project. Health facility practice information was collected before and after establishment of the community-based growth monitoring system.

Results. The health facility practice reached 12 - 26% of the preschool population per month compared with 70 - 100% per 3-week session in the community-based growth

monitoring system. The community-based growth monitoring system increased growth monitoring coverage of preschool children by more than 60%. Attendance of preschool children aged 12 months and older varied between 10% and 14% at the health facility practice compared with 80 - 100% in the community-based growth monitoring system. This made the system more conducive for monitoring and targeting of malnourished children for health and nutrition interventions.

Conclusion. The community-based growth monitoring model demonstrated that community participation and mobilisation can increase preschool child growth monitoring coverage extensively and contribute to improved health and nutrition surveillance.

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