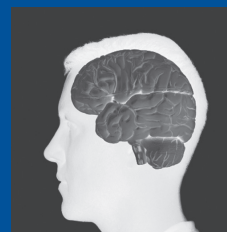
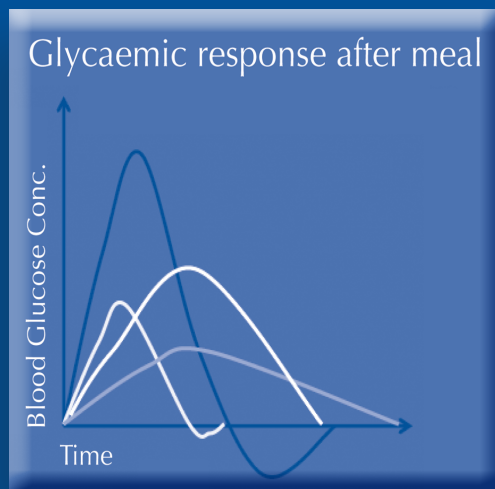


FOOD, GLYCAEMIC RESPONSE AND HEALTH



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FOOD, GLYCAEMIC RESPONSE AND HEALTH

by
Michele Sadler



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FOREWORD

Carbohydrates typically provide the major energy contribution to our diet. They also lead to rises in blood sugar or blood glucose (glycaemia). The glycaemic response to a food or meal is the effect that food or meal has on blood glucose levels after consumption. It is normal for blood glucose and insulin levels to rise after eating and then return again to fasting levels over a short period of time. This is particularly so after consumption of meals rich in certain carbohydrates. Reducing the size and duration of rises in blood glucose after meals is particularly important for people with diabetes, and may also be of benefit to the general population. Several tools have been developed to help quantify and communicate the effect of food on glycaemic response. These include glycaemic index (GI), glycaemic load (GL) and glycaemic glucose equivalents (GGE).

In 1981, the idea of classifying carbohydrates according to their GI was first published. Since then, many studies have been undertaken to determine the impact of altering the blood glucose-raising potential (glycaemic challenge) of the diet on a wide range of short- and long-term health outcomes. However, evaluating the impact of a single dietary change on health is notoriously complex, and opinions on the relevance of GI, GL and GGE have been divided.

The Dietary Carbohydrates Task Force of ILSI Europe has, over a period of several years, studied selected aspects of the science behind glycaemic response and health, seeking to contribute a balanced perspective on the subject. Firstly, the group commissioned an expert consultation on GI methodology. A comprehensive meta-analysis of intervention studies linking glycaemic response with health outcomes was then undertaken. This culminated in a Workshop entitled *Glycaemic response and health*, held in Nice, France, on 6–8 December 2006.

The Workshop analysed in detail the findings of the meta-analysis and, whilst the conclusions were in many cases still far from clear, several key points have emerged and been summarized in a supplement of the January 2008 issue of the *American Journal of Clinical Nutrition*.

Dietary interventions that lower the GI and/or GL of the diet improve fasting blood glucose in individuals with impaired blood glucose control, e.g. diabetics. Similarly, glycated proteins (associated with tissue damage) are reduced, with the effect being greater in subjects with poorer glycaemic control. Insulin sensitivity can also be improved, with the magnitude of the effect being greater, the greater the initial level of dysfunctionality. It should be noted that reductions in the GI and/or GL of the diet are often associated with changes beyond only the available carbohydrate source. These include fat and protein levels and unavailable carbohydrate content, such as dietary fibre.

It is becoming evident that modifying the glycaemic response of the diet should not be seen as a stand-alone strategy but rather as an element of an overall balanced diet and lifestyle.

This Concise Monograph seeks to provide a balanced overview of the ILSI Europe findings to date linking glycaemic response with health outcomes. It includes a summary of the most recently available information and sections on the glycaemic properties of foods and on the glycaemic concept in action. In the tradition of ILSI Europe Concise Monographs, it is hoped that this volume will inform healthcare professionals, students and motivated consumers alike, becoming a useful educational and reference tool.

Julian Stowell
Danisco Ltd (UK)

INTRODUCTION

Carbohydrates in foods are traditionally classified according to their chemical structure, i.e. as mono-, di-, oligo- and polysaccharides. Such a classification gives little information about how carbohydrates behave in the gastrointestinal tract, or their rate or ability to be digested and absorbed in the small intestine and thus contribute to blood glucose concentration. Because this variable physiological property of carbohydrates has the potential to influence health, it is important to fully understand this relationship. During the past 30 years much attention has been focused on measuring and classifying carbohydrates on the basis of their glycaemic effects. One aim of such research is to enable improved glycaemic control through the dietary management of postprandial blood glucose concentrations.

In most countries around the globe, dietary guidelines recommend that most of the daily energy consumption should derive from (available) carbohydrates, which are typically glycaemic. Efforts have been made to classify carbohydrates as energy source on the basis of the glycaemic response they mediate. The scope to influence glycaemic response has also been considered. Dietary fibres are another type of dietary carbohydrates. A daily intake of 25 g dietary fibre is recommended due to its association with various health benefits, including effects on the blood glucose response (EFSA, 2010a).

This monograph explains the concept of the glycaemic response and discusses the potential benefits of dietary management of postprandial glycaemia for various health indicators, including a number of key public health priorities.

Definitions

Glycaemic response

After eating a meal, the digestible or glycaemic carbohydrates are absorbed from the intestine into the bloodstream, producing an increase in blood glucose concentration. In time and in response to its tissue disposal, facilitated by the hormone insulin, the blood glucose concentration falls back to or below fasting levels. The magnitude of the rise and fall in blood glucose and the duration over which it occurs has been termed the blood glucose (or glycaemic) response. The glycaemic response varies for different carbohydrates. Fully and readily digestible carbohydrates such as glucose, maltodextrin and cooked potato starch produce a rapid increase in blood glucose, followed by an equally rapid fall. Insulin is released in response to the initial rise in blood glucose and causes blood glucose to fall. Insulin release can also be triggered by other food components such as certain proteins. Over-compensation can result in blood glucose falling below baseline values.

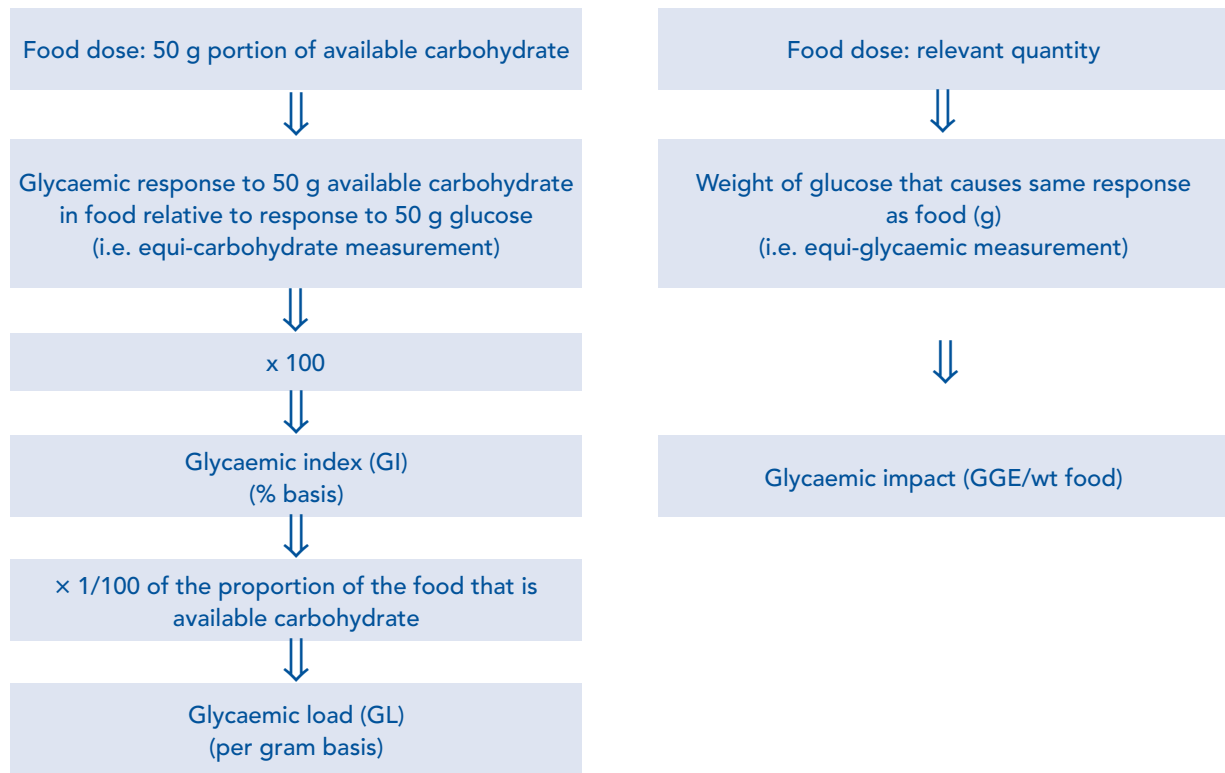
More slowly digestible carbohydrates or minimally processed starchy foods produce a different response. Compared with rapidly digestible carbohydrates they show a slower and more prolonged increase in blood glucose, rising to a lower peak. Non-digestible carbohydrates (e.g. dietary fibres such as polydextrose and inulin) elicit a negligible direct blood glucose response (see Role of unavailable carbohydrate, page 11). However, there are also some absorbed carbohydrates such as fructose and xylitol (which is partially absorbed) that do not result in increased blood glucose levels. In addition to the available carbohydrate level of food, the rest of the composition of food can also influence the glycaemic response. Different fat, protein and unavailable carbohydrate levels can influence the speed with which glucose can enter the blood, e.g. via reduction in the speed of gastric emptying and intestinal transit or the level of access of digestive enzymes.

Because blood glucose concentration is influenced by the foods we eat, it is important to have appropriate descriptors to express the differences in the glycaemic response that are characteristic of different carbohydrate-rich foods. Historically the GI has been the main focus of research but more recently GL and GGE have been proposed as alternative descriptors (Figure 1).

These indicators of the glycaemic properties of carbohydrate-rich foods each give different information and, depending on the information required and how it will be used, there is currently much discussion about which is the most appropriate communication tool.

FIGURE 1

Comparison of glycaemic index (GI), glycaemic load (GL) and glycaemic glucose equivalents (GGE)



Source: Monro and Shaw (2008)

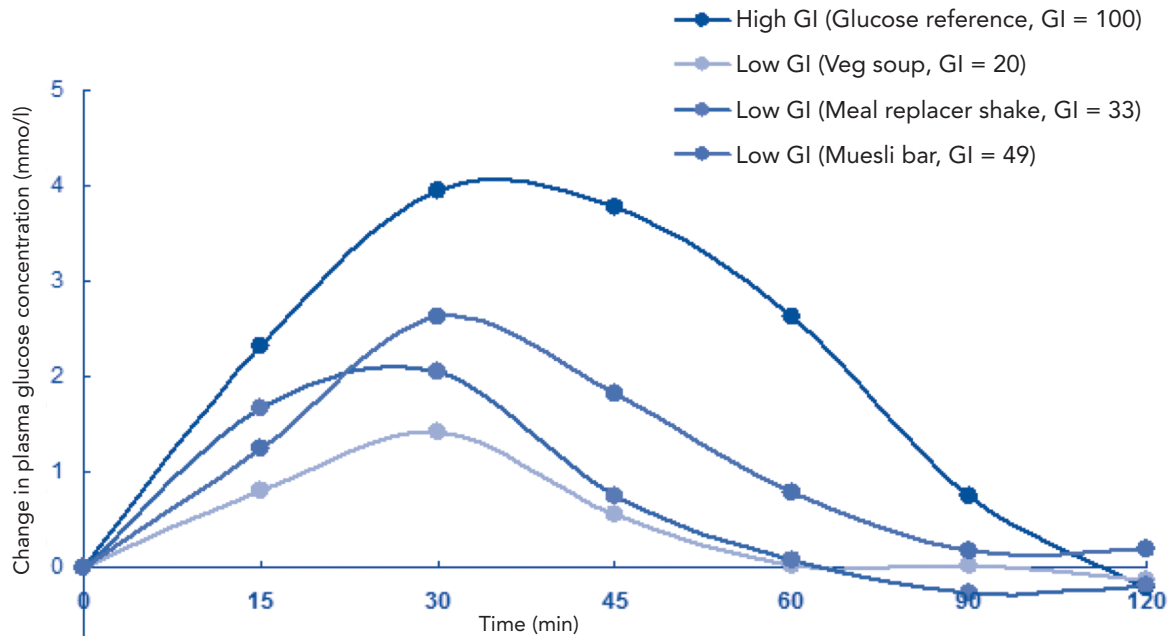
Glycaemic index (GI)

The term “glycaemic index” was defined in the early 1980s as one way to numerically express the effect of available carbohydrates in a food on blood glucose concentration (Jenkins, 1981). This research established a method to link foods containing different amounts of available carbohydrates with different glycaemic responses in the blood. Carbohydrate exchange lists previously used in the dietary management of diabetes mellitus were based on food portions containing similar amounts of carbohydrate. GI research clearly showed that similar amounts of carbohydrate from different foods elicit different glycaemic responses (Figure 2).

The GI represents a measure of the average concentration of glucose in the blood following a test food, ingredient or meal (usually containing 50 g available carbohydrate), over a set period of time (usually 2 hours), and is expressed relative to a standard or reference food (usually glucose or white bread). By definition, the reference food has a GI of 100 (Brouns, 2005).

The GI provides a means of expressing the glycaemic potency of a meal or snack. Because it gives comparative information about the glycaemic properties of the available carbohydrate in a food on a weight for weight basis, substitution of a food with a higher GI value for a food with a lower GI value reduces the glycaemic

FIGURE 2
Blood glucose response curves for high and low GI foods



The GI of a food is calculated from the area under the curve (for further details see How GI is calculated, page 5)

response, assuming that a similar amount is eaten. Knowledge of the GI is thus useful for meal planning or choosing between different carbohydrate staples such as potatoes, pasta or rice within a meal. In such situations, the GI value does not give information about the total glycaemic potency of the meal because this is dependent on the amount and other components of the food that is eaten.

Glycaemic load (GL)

Unlike GI values, which are based on a set amount of available carbohydrate present in a test food or meal relative to a standard, GL provides additional information about the glycaemic quantity in relation to portion size. The concept of GL incorporates the total available carbohydrate in the food portion in addition to the glycaemic quality of the carbohydrate, both being factors that influence postprandial blood sugar concentration. GL thus refers to the cumulative exposure to postprandial glycaemia over a specified time period, which may be used as an indicator of insulin demand.

Glycaemic glucose equivalents (GGE)

GGE is a measure of the glycaemic impact of a food and is an alternative measure to GI or GL. Advocates of GGE contest that it is a more advanced expression of the glycaemic potency of foods than GI or GL.

How to measure the glycaemic response

Expression of the glycaemic index

How GI is calculated

GI is measured in human subjects, *in vivo*. For each individual subject, the GI value of the test food is calculated as follows:

GI value of test food = [IAUC of test food/individual subject's average IAUC of the reference food] × 100*

The overall GI is calculated as:

The mean (± SEM) GI value for 10 or more subjects

**The incremental area under the curve (IAUC) includes the area above the baseline and under the curve, ignoring the area beneath the baseline (see Figure 2). This method of calculating the area under the curve is recommended by FAO (FAO/WHO, 1998).*

As GI refers to the glycaemic effect of available carbohydrate in food relative to the effect of an equal amount of glucose (i.e. weight glucose/weight available carbohydrate = 50 g/50 g = 1), the GI is an equi-carbohydrate measure.

Hence, the GI is a measure of the glycaemic impact of foods and does not relate to the quantity or the portion size of the food that is eaten. GI values of different foods vary, e.g. with glucose as the reference food, the GI of boiled soya beans is 15±5 and the GI of boiled potato is 82±7 (Table 1). GI values can theoretically exceed 100, the value assigned to the reference food (glucose). This could occur in cases where a food elevates blood glucose concentration to a greater degree than glucose itself, or in association with more rapid gastric emptying. GI tables can therefore provide guidance on the glycaemic influence of foods, but do not provide absolute accuracy.

TABLE 1

Examples of GI values

Food no. in tables	Food item	GI
945	Apple (Golden Delicious)	39±3
102	Baguette, traditional French	57±9
960	Banana	62±9
1624	Carrot (boiled), mean of 4 studies	39±4
452	Cornflakes with skimmed milk	65±5
1206	Beef lasagne	47±7
1566	Fructose (50 g portion)	23±1
776	Ice cream, raspberry ripple	55±3
N/A	Isomaltulose	32±3
1611	Peas (boiled)	51±6
1651	Potato (boiled), mean of 4 studies	82±7
1659	French fries	70±6
1049	Orange juice	46±6
1017	Prunes	29±4
1018	Raisins	64±11
562	Rice, Basmati (boiled), mean of 6 studies	57±4
1124	Soya beans (boiled)	15±5
1367	Spaghetti (boiled)	44±3
1608	Sugar (sucrose)	65±9
172	White bread	59±11
234	Wholemeal bread	68±9
895	Yogurt (low-fat, natural), mean of 4 foods	19±6

Reference food: Glucose

Data are means ± SEM

Adapted from tables given in Atkinson *et al.* (2008)

It is important that GI values are not used in isolation when selecting foods, but in the context of dietary recommendations on macronutrients. For example, advice for foods that are low GI but high in saturated fat remains “eat them in moderation”. Also, some fruits for example may be high in GI but are low in fat, provide dietary fibre and are a useful source of vitamins and minerals.

Over the past two or three decades, the GI values of many diverse foods have been tested by *in vivo* human measurements. Atkinson *et al.* (2008) have systematically tabulated both published and unpublished sources of GI values. Atkinson *et al.* considered the quality of the data and separated values in healthy subjects from those in individuals with impaired glucose metabolism. The correlation coefficient for 20 staple foods tested in both healthy and diabetic subjects was $r=0.94$ ($P<0.001$).

Summary tables of GI values are available in the scientific literature and other media, including the internet (<http://www.glycemicindex.com>) and the appendix to Atkinson *et al.* (2008), that are convenient for use by healthy and diabetic consumers.

Reproducibility of GI measurements

A number of methodological variables that effect the determination of GI values in human testing have been observed. These include the method of blood sampling, choice of reference food (usually glucose or white bread), measures to reduce intra-individual variation (the reference food is tested in each subject at least twice) and the method of calculating the AUC. Also, the restrictions placed on the subject’s diet and activity the day before testing can impact the outcomes. A recommended method is available that aims to standardise such variables (FAO/WHO, 1998). Availability of an approved method is important for regulatory and enforcement purposes.

How GI is measured

The FAO/WHO Joint Expert Consultation *Carbohydrates in Human Nutrition* (FAO/WHO, 1998) established a standard methodology for measuring GI. More recently, Brouns *et al.* (2005) published a review of GI testing. For detailed information about the methodology (e.g. details of test procedure, blood sampling, quality control) the reader is referred to the above texts and to Wolever *et al.* (2008). Salient points of GI testing include:

- GI is measured *in vivo* in a minimum of 10 healthy human volunteers in the fasting state. To assist standardisation of conditions subjects are not allowed to:
 - (i) Consume food and drink other than water for 10 or more hours prior to the test
 - (ii) Consume alcohol on the previous evening
 - (iii) Undertake vigorous exercise on the morning of the test
- GI is measured over a 2-hour period starting at ingestion of the test food.
- The weighed portion of the test food is calculated to contain 50 g of glycaemic carbohydrate, or 25 g if the portion size is unreasonably large. Foods providing less than 10 g glycaemic carbohydrate per usual serving are not suitable for GI testing.
- The test food is measured in each subject two to three times on separate days.
- A numerical GI value should only be assigned to a food following *in vivo* testing, and not by mathematical calculation from the GI value of the individual ingredients of food items.

Standards are also required to assess the performance of laboratories making GI measurements. An inter-laboratory study assessed the magnitude of variation of the means and standard deviations of GI values measured in different laboratories globally. This study determined the extent to which sources of methodological variation may explain the inter-laboratory variation in GI measurements (Wolever, 2008). GI testing of two food products, cheese puffs and fruit leather, was compared in 28 participating laboratories in a total of 314 subjects. Consistent with previous studies, the analysis found that ethnicity, gender, age and body mass index (BMI) were not related to intra-individual variation in glycaemic response or to GI values in individual subjects. It was concluded that it is not necessary to control these factors and that it may not be necessary for subjects to avoid coffee or tea with the test meal.

Hence, when tested by appropriate methods, GI is the same in different subjects and can therefore be viewed as a property of the food and not of the subject in whom it is tested. GI can thus be measured with validity in most subjects and the results apply to most of the healthy population. However, very high individual values can occur (outliers), and it is recommended that these values are excluded (Brouns, 2005). Important sources of variation affecting the accuracy of GI values included how these outlier values are handled and how the AUC is calculated. The results of the study showed that greater than two standard deviations (SD) above the mean is an appropriate definition of GI outliers for a group of 10 or more subjects. It was also determined that the intra-individual variability of the AUC (expressed as the mean value of coefficient of variation for the reference food) should be less than 30% (Wolever, 2008).

Factors affecting the precision of GI measurement were identified as (i) the precise analytical method for glucose analysis – duplicating blood samples or glucose analyses was found to be beneficial, and (ii) subject preparation – asking subjects to consume a normal meal and to avoid unusual exercise the night before the test, also had benefits.

Expression of the glycaemic load (GL) and glycaemic glucose equivalents (GGE)

How GL is calculated

GL is calculated from the GI value of a food as follows:

$$GL = [GI \times \text{total available carbohydrate in the food portion}] / 100$$

The GL descriptor is not expressed in units and relates to the serving size. For example, the GL of instant mashed potato with a GI of 82 and with 25 g carbohydrate per serving $[(82 \times 25)/100]$ is 21. Use of GL requires tables of the GI values of foods and information about the carbohydrate content per serving.

GL is practically useful for managing postprandial glycaemia by manipulating both the choice of carbohydrate-containing food and the portion size that is eaten. However, determination of the portion size is a practical limitation, as this will vary with different pack sizes, and for foods where the consumer selects the portion size, such as for rice and pasta.

How GGE is calculated

GGE is calculated as:

$$GGE/g = (IAUC_{\text{food}} / IAUC_{\text{glucose}}) \times (\text{weight glucose} / \text{weight food}) \times 1 g$$

$$(IAUC_{\text{food}} / IAUC_{\text{glucose}}) = 1 \text{ (i.e. equi-glycaemic)}$$

$$\text{Relative glycaemic impact (RGI)} = GGE/g \times \text{food weight subsequently consumed}$$

Relative glycaemic impact (RGI) is defined by the American Association of Cereal Chemists as “the weight of glucose that would induce a glycaemic response equivalent to that induced by a given amount of food” (Monro, 2008). RGI expresses the relative glycaemic potential of a food in grams of GGE, per specified amount of food. As a numerical expression, GGE is thus similar to that of other food components (e.g. micrograms of retinol equivalents) in a way that GI and GL are not. GGE differs

from GI and GL because it refers to food rather than to available carbohydrate, is expressed in grams whereas GI is an index without units, and depends on the quantity of food consumed, whereas GI does not.

If a serving of food has a glycaemic impact equivalent to that of 15 g of glucose, the food is said to have a GGE content of 15 g. The GGE descriptor thus expresses a set amount of food that equals a certain amount of glucose in its effect, or expresses the weight of glucose that would have the same effect as a given weight of food. GGE is thus a “virtual food component”, meaning that it can be expressed in terms of a weight of a reference food, such as glucose per unit weight. GGE thus has the potential to be used on food labels and within food tables.

When studying glycaemic response and health, GGE could also be very useful. Potential uses include:

- To express the RGI of foods as their GGE content on food labels or in food tables
- To demonstrate the effects of different portion sizes, since GGE is directly related to the quantity consumed
- To demonstrate the effects of combining foods by adding together GGE values
- To assess the quantities of foods that may be exchanged for the same glycaemic effect and publish these in food exchange tables for glycaemic management
- For use in meal planning in combination with systems for monitoring blood glucose and insulin delivery.

Relevance of GI, GL and GGE in the context of a mixed meal

The GI value of an individual food might differ when the food is eaten in combination with other foods. The protein, fat and fibre content, the bulk of food eaten, and other factors can all influence the measured glycaemic response. The rate of gastric emptying, rate of absorption and insulin demand can all be altered depending on the food matrix.

A key question frequently raised is how meaningful are GI, GL and GGE measures relating to individual foods when we generally eat foods in combination or as part of a meal.

When eaten in the context of a test meal, often referred to as a “mixed meal”, the substitution of low glycaemic foods for higher glycaemic foods reduces the glycaemic response to the meal.

Conclusions

Historically, GI has been the key descriptor for the glycaemic properties of carbohydrates and the main focus of scientific research. Over time, additional expressions of glycaemic response have been developed, depending on the information required and how it will be used. GI provides an indication of the glycaemic impact of carbohydrates present in food, and is useful for meal planning and choosing between different carbohydrate staples within a meal. It does not give information that relates to the quantity consumed. GI should be used in the context of dietary recommendations on macronutrients and not in isolation.

GL has the advantage of providing information about glycaemic quantity, based on serving size, and glycaemic quality. It is useful for managing postprandial glycaemia by manipulating both the choice of carbohydrate-containing food and the portion size.

GGE is an alternative measure to GI and GL. It provides an estimate of the glycaemic impact arising from an ingested quantity of food and is a more complex concept. A potential advantage of GGE is that because of the way it is measured and expressed it is more amenable for potential use in the nutritional labelling panel along with nutrient declarations, provided consumers can understand the concept.

Glycaemic labelling of foods must be based on a generally accepted relationship between the glycaemic properties of foods and health outcomes. The evidence linking glycaemic response and health is discussed in the section starting on page 12.

GLYCAEMIC PROPERTIES OF FOODS

Impact of the food matrix on glycaemic response

GI values of the same food are subject to variation depending on the physical state of the food. However, variation is true of all nutritional values, e.g. the vitamin and mineral content of foods.

Food-related factors that influence the glycaemic impact of a food include:

- The nature and amount of available carbohydrate contained in a food:
 - The physical form of the carbohydrate (e.g. particle size, degree of hydration)
 - The nature of starch (amylose, amylopectin) or starch hydrolysis products
 - The content of monosaccharides (glucose, fructose, galactose, mannose, tagatose)
 - The content of disaccharides (sucrose, isomaltulose, trehalose, lactose)
 - The content of oligosaccharides (maltodextrins)
- The nature and amount of other food components:
 - Fat
 - Protein
 - Dietary fibres
 - Organic acids
 - Phytochemicals
- Food form and effects of processing and preparation
 - Degree of cooking
 - Physical form (solid versus liquid)
 - Particle size of food

The rate of starch breakdown in the digestive tract has a major influence on GI values. The susceptibility of starch to hydrolysis is thus an important influence, e.g. amylopectin is hydrolysed by pancreatic alpha-amylase more rapidly than amylose. Other important factors include access of the digestive enzymes to starch, which is influenced by the physical form of the food and its structure. For example, the ripeness of fruit, and physical changes to food in food processing and preparation – whether food is served pureed, in large or small pieces, cooked or raw – all influence GI values. The viscosity in the gastrointestinal tract and resulting rate of absorption of the monosaccharides are further determinants of the GI.

Diet and glycaemic response

Factors linked to eating behaviour and physiological factors relating to individuals also influence the glycaemic response. These factors include:

- How often and when food is eaten
- How quickly food is ingested
- The composition of meals and snacks
- The rate of gastric emptying
- Digestive responses
- Non-blood glucose stimulation of insulin release
- Individual variation

Nutrients that affect gastric emptying and viscosity of the gut contents include fat, protein and fibre. For example, fat delays gastric emptying and is therefore predicted to lower the GI of a food or meal.

Influence of previous meal on glycaemic response

The carbohydrate content of a meal or snack and its postprandial period has been shown to influence the glycaemic response of the next meal eaten (Nilsson, 2006). Hence, glycaemic response and GI values can vary

depending on previous consumption. For example, it has been demonstrated that the incorporation of one key low GI food with each meal reduces the GI of the diet and has a statistically significant effect on lowering fasting and 24-hour glucose profiles in healthy subjects and people with diabetes (Brynes, 2005).

It has been shown that consuming low GI foods rather than high GI foods has a positive effect on lowering postprandial glucose levels after the following meal. Similarly, if a high GI snack is eaten, this raises the glycaemic response at the next meal (Frost, 2005). This has implications for the glycaemic impact of the daily diet.

Can the GI of a meal be calculated from the individual components?

There is consensus among experts that the GI of a meal can reasonably be estimated by calculation from measured GI values of the individual meal components (Howlett, 2008). However, the extent to which the observed response to the meal can be related to its GI value is determined by a number of factors including the magnitude of the difference in GI between the intervention and the control diets, the accuracy of the GI values of the individual meal components, and whether the difference in GI is the only variable between the test and control meal.

Glycaemic impact of the diet

Postprandial glycaemia and health

Eating more frequent snacks and meals, as opposed to a traditional pattern of three meals a day, means that a higher proportion of the day is spent in the postprandial state. The absorption and metabolism of different carbohydrates influences postprandial glucose, insulin and non-esterified fatty acid concentrations. Postprandial metabolism thus mediates an important influence on disease risk including cardiovascular disease (CVD) and other diseases related to insulin resistance such as type 2 diabetes.

Insulin response

As carbohydrate intake increases, serum insulin concentration increases, though this may not be in proportion. Various factors influence the insulin response including BMI, age and gender, gastric emptying, gut hormone release, and viscosity of the gut contents. The insulin response also varies in different groups of subjects, such as those with impaired glucose tolerance, obese subjects and type 2 diabetics.

Although still an area of debate, lower versus higher GI foods tend to show an attenuated insulin response. In non-diabetics, the insulin response to foods with a wide range of GI values are highly correlated. The possible exception is dairy products, which show a higher insulin response than expected because whey protein stimulates insulin secretion.

Role of unavailable carbohydrate

A key finding from the Livesey meta-analysis (see Influence of glycaemic response on health indicators, page 12) is that, in most of the studies, a decrease in GI and/or GL was accompanied by increased levels of unavailable carbohydrate, i.e. dietary fibre (Livesey, 2008a). Hence, the specific effects of the individual changes in GI and/or GL and unavailable carbohydrate cannot be totally disentangled.

Although GI is measured and calculated on the basis of available carbohydrates, the presence of unavailable carbohydrates such as fibre can also have an effect on the glycaemic response. This may occur due to the slowing down of digestion whilst maintaining the available carbohydrate content or by substitution with a higher fibre diet, reducing the available carbohydrate intake. Livesey *et al.* found that when non-digestible carbohydrates are used to replace highly digestible carbohydrates in diets and in food products they are capable of reducing the glycaemic response, and hence the glycaemic impact of the diet.

Conclusions

Tables of GI values are easily accessible on the Internet and in the scientific literature. There is good correlation for staple foods tested in both healthy and diabetic subjects, such that the tables are applicable to both of these groups.

Methodological variables that affect the determination of GI values in human testing have been observed and are accounted for in recommended methodology. GI can be measured with validity in most subjects and the results apply to most of the healthy population. In an inter-laboratory study, sources of variation in GI values included how outlier values are handled, how the AUC is calculated, the choice of analytical method for glucose analysis, and subject preparation.

Factors that influence GI values include the food matrix, factors linked to eating behaviour and physiological factors. The carbohydrate content and GI of a meal or snack has been shown to influence the glycaemic response of the next meal eaten following the postprandial period. Substituting available carbohydrate with unavailable carbohydrates is another means whereby the glycaemic response to the diet can be reduced. There is consensus among experts that the GI of a meal can be reasonably estimated by calculation from the measured GI values of the individual meal components. The intake of a single low glycaemic food may have impact on 24-hour blood glucose control parameters.

INFLUENCE OF GLYCAEMIC RESPONSE ON HEALTH INDICATORS

There is clear scientific evidence that glycaemic response can influence health outcomes related to key public health priorities like type 2 diabetes and blood glucose control. In Western countries, impaired glucose metabolism is already prevalent in a substantial part of the general adult population. There is suggestive, but not yet proven, evidence that glycaemic response might play a role in weight management and CVD.

A systematic review and meta-analysis has investigated the glycaemic impact of foods on measures relevant to health maintenance and disease management, i.e. whether foods with a low impact on blood glucose have a high impact on reducing risk factors for disease (Livesey, 2008a, 2008b). The markers or outcome measures chosen in the analysis are relevant to a number of health parameters, but require further validation with respect to their ability to predict disease risk (Howlett, 2008).

Livesey *et al.* comprehensively addressed the contribution of GL compared with GI, the role of unavailable carbohydrate in studies of reduced GI, and whether there are dose-dependent effects of GI on markers of health risk. Altering one dietary component may alter other components and hence the review also analysed which dietary factors are important in affecting markers of health risk.

The meta-analysis investigated effects related to glycaemic response in healthy subjects, glucose intolerant subjects, patients with type 1 and type 2 diabetes, subjects at risk of coronary heart disease (CHD), and hyperlipidaemic

groups. A total of 45 relevant human intervention studies (published before January 2005) were included in the meta-analysis and the key findings are summarised in the following sections.

Blood glucose concentration and glycaemic control

In diabetic patients, blood glucose control is crucial to avoid hyperglycaemia, hypoglycaemia and ketoacidosis. It is also vital in preventing secondary complications such as renal disease, retinopathy, neuropathy and atherosclerosis. Blood glucose is controlled through management with diet *alone* or with diet and drugs.

Although diabetes mellitus is a recognised disease state, there is a continuum of fasting and post-load glucose tolerance with potential health impacts, ranging from normal values in healthy subjects to impaired fasting glucose (IFG) and impaired glucose tolerance (IGT) (Wolever, 1999a). The oral glucose tolerance test (OGTT) is used to assess the degree of elevation of blood glucose and to test for diabetes mellitus and impaired glucose tolerance. OGTT measures how quickly a 75 g dose of glucose is cleared from the blood in a fasting subject. OGTT is thus suitable to detect mild abnormalities of glucose tolerance and has a potential role in the early detection of poor glycaemic control and diabetes prevention.

The definition of diabetes mellitus is based on general consensus cut-offs for blood glucose concentration (fasting plasma glucose at or above 7.0 mmol/l, 2-hour plasma glucose at or above 11.1 mmol/l after 75 g oral glucose tolerance test). Subjects with IFG and/or IGT may have higher glycation (the bonding of protein or lipid with sugar molecules as indicated by glycated haemoglobin, HbA1c, concentrations) and are at increased risk of developing diabetic complications. They might therefore benefit from dietary management.

HbA1c is a marker of longer-term blood glucose control that depends on the blood glucose concentrations over the previous 3 months. HbA1c concentrations in diabetics are typically $\geq 6.5\%$, while values of 5–5.5 % are indicative of impaired glucose tolerance, depending on the assay. Fructosamine is an intermediate measure that is sensitive to changes in blood glucose control over 2–3 weeks, though is seldom used. Both markers result from non-physiological chemical reactions between free glucose and proteins. Such reactions also occur with proteins in vessels and peripheral tissues that are exposed to glucose, which can cause functional damage including ageing.

The issue discussed in the sections below is the value of low GI and low GL foods (including the role of unavailable carbohydrate) in contributing to the improved management of these states, in helping to prevent adverse health effects, and in helping to prevent secondary complications of diabetes. This is determined from studies that have investigated the impact of low GI foods and diets on endpoints such as:

- Fasting blood glucose levels
- Postprandial blood glucose levels
- 24-Hour blood glucose levels
- Concentrations of glycated proteins such as HbA1c and fructosamine

These studies have been conducted against a background of epidemiological evidence that suggest that lower GI diets might be beneficial for health, in particular for people with type 2 diabetes.

Fasting blood glucose concentration

For normal fasting blood glucose levels, the Livesey meta-analysis (2008a) found no difference in the effect of lower GI versus higher GI treatment (study population mean approximately 5 mmol/l). It should be noted that 5 mmol/l is the optimum blood glucose concentration and any

consistent fall of fasting blood glucose below 4.5 mmol/l is counterbalanced by hormonal regulation. A beneficial association was found for low GI compared with a higher GI treatment when the mean fasting blood glucose concentration in the study population was above 5 mmol/l.

The evidence base included 36 studies reporting fasting blood glucose concentration (in venous or capillary blood), with change in GI ranging from -4 to -32 points and with study durations of 2–26 weeks. Subjects included healthy adults, adults with glucose intolerance, type 1 and type 2 diabetics, and subjects at risk of CHD.

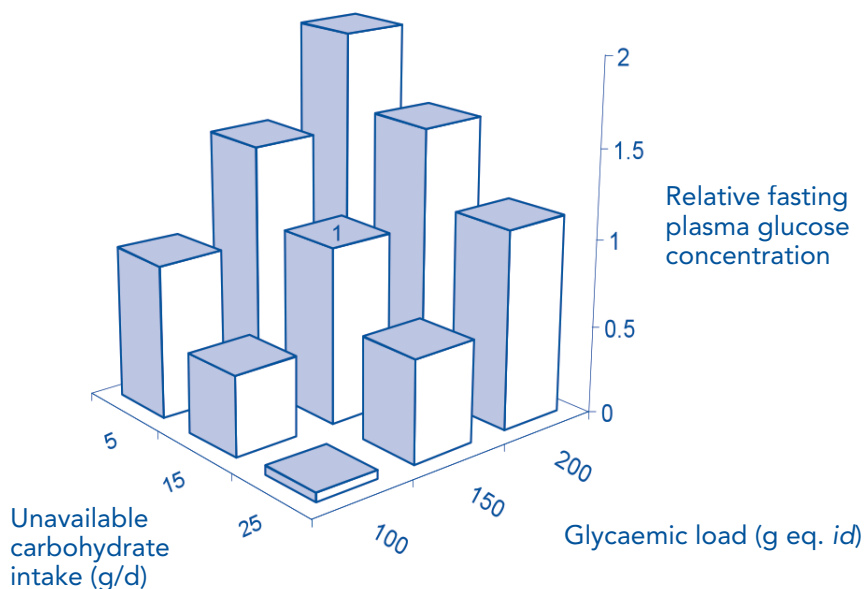
The meta-analysis found that many interventions that set out to lower the GI of the diet also resulted in an increased

intake of unavailable carbohydrate and a varied intake of available carbohydrate. This resulted in a concomitant decrease in GL. Further analysis of the data revealed that lowering GL and raising unavailable carbohydrate (fibre) both independently act to control fasting blood glucose (Figure 3), and that the unavailable carbohydrate is as effective as GL in lowering fasting blood glucose.

A distinction between replacement of available carbohydrates by dietary fibre and slower digestion due to unavailable carbohydrates cannot be made through this analysis. Only studies with elevated fasting blood glucose (>5 mmol/l) were used for the analysis shown in Figure 3.

FIGURE 3

Implications of the effect of unavailable carbohydrate intake and glycaemic load for fasting blood glucose concentrations



Source: Livesey (2008a)

The meta-analysis found that GL is better reduced by a reduction in GI than by a reduction in available carbohydrate. A further conclusion was that the size of the effect is dependent on the status of the subjects consuming the diet, i.e. the effect was greater in subjects with poor fasting blood glucose control.

Maximum control of fasting blood glucose was achieved by elevation of unavailable carbohydrate together with a lowering of GI. Unavailable carbohydrate had a stronger impact than GL on a per gram weight basis, but remained equally important when taken together with the range of intakes.

In conclusion, sustained reductions in glycaemic response by using lower GI diets or foods (including the use of low GI carbohydrates) have a beneficial effect on elevated fasting blood glucose. Since elevated fasting blood glucose is a risk factor for type 2 diabetes, this effect relates to possibilities for risk reduction. The effect is greater in subjects with poorer control of blood glucose, including those with type 1 and type 2 diabetes. For individuals with fasting blood glucose in excess of 5 mmol/l, the fasting blood glucose is reduced by consuming lower GI foods and diets, or by a lower GL. Higher intakes of unavailable carbohydrate have an additive effect to that of lower GI or GL (directly via stomach emptying or indirectly via fermentation).

Such dietary manipulations thus have potential in helping diabetics to manage their blood glucose concentration. The meta-analysis found that optimum reduction in fasting blood glucose occurs with GI of <45, GL of ≤ 100 g (glucose equivalents)/day, or intakes of unavailable carbohydrate ≥ 25 g per day. Whether the beneficial effects found in people with impaired glucose metabolism are also beneficial for healthy subjects (i.e. people with normal glucose metabolism) cannot be concluded from these data. However, it is important to notice that a substantial number of people in the current general population have impaired blood glucose tolerance.

Postprandial and 24-hour blood glucose

GI is a measure of the postprandial glucose response, which supports by definition a short-term effect of low GI foods on postprandial glycaemic control. Second meal and whole-day randomised controlled trials also support the short-term impact of consuming low GI foods on postprandial glycaemic control and demonstrate a positive impact of low GI diets over a period of 24 hours, both in the laboratory and in free living studies.

Henry *et al.* (2006) investigated the effect on 24-hour blood glucose of high or low GI bread eaten within identical diets. The low GI bread resulted in a lower mean glucose response compared with the high GI bread, and the IAUC following the low GI bread was consistently lower than for the higher GI bread. A simple dietary change can thus favourably alter blood glucose control.

Glycated proteins

The concentration of glycated proteins such as fructosamine and HbA1c can be used as a measure for the retrospective average blood glucose levels over a prolonged period: 2–3 months for HbA1c and 2–3 weeks for glycated fructosamine. The Livesey meta-analysis (2008a) found that interventions to lower GI reduced blood glycated protein concentration, with a trend for a greater treatment effect in those with poor glycaemic control. This was significant for HbA1c and fructosamine combined, and for fructosamine alone.

The evidence base included 28 studies reporting endpoints of glycated albumin, glycated plasma protein, fructosamine and HbA1c. Reductions in GI ranged from –5 to –31 points and study durations were up to 12 weeks. Subjects included healthy adults, adults with impaired glucose intolerance, type 1 and type 2 diabetic patients and adults at risk of CHD.

As with fasting blood glucose, variability in the effects on glycated proteins was dependent on the variation in

GI and/or GL, and unavailable carbohydrate. Bivariate analysis found that variation in fructosamine concentration is explained by variation in the intake of unavailable carbohydrate and GI, whereas the effect of available carbohydrate intake was not significant.

Similar to the findings for fasting blood glucose, the meta-analysis determined that optimum reduction in fructosamine occurs with GI of <45, GL of ≤ 100 g (glucose equivalents)/day, or intakes of unavailable carbohydrate ≥ 25 g per day.

A beneficial long-term effect of low GI foods on markers of blood glucose control has also been supported in two previous meta-analyses and systematic reviews (Brand Miller, 2003a; Opperman, 2004) each citing over 50 individual papers.

The review by Opperman, of 16 pertinent studies concluded that low GI diets significantly reduced fructosamine and HbA1c. In the review by Brand-Miller of 14 pertinent studies with diabetic patients, it was estimated that low GI diets reduced HbA1c by 0.43% points compared with high GI diets. Taking the data for HbA1c and fructosamine together, after adjusting for baseline differences, glycated proteins were reduced 7.4% more on a low GI diet compared with a high GI diet.

Plasma insulin concentration and insulin resistance

Insulin is a hormone secreted by the pancreas. It has a central role in the control of carbohydrate metabolism by signalling insulin-sensitive tissues such as muscle and adipose tissue to absorb glucose, thus lowering blood glucose concentration. When blood glucose falls to normal physiological values insulin release is slowed. In healthy subjects, fasting insulin levels range between 8 and 17 $\mu\text{U/ml}$ and in those with insulin resistance from 17 to 25 $\mu\text{U/ml}$.

Individual responses to insulin vary considerably and a muted response (i.e. "less than normal") is termed "insulin resistance" (BNF, 2005). In this condition, normal levels of insulin have insufficient effect on muscle and adipose tissue, and blood glucose levels stay raised. This stimulates the release of more insulin, raising plasma insulin levels, which has various physiological effects in the body.

Reduced gluco-regulatory responses to insulin include both a reduction in the maximal response (i.e. unresponsiveness) and a shift to the right of the dose-response curve (insulin insensitivity).

There are various techniques for measuring insulin sensitivity (BNF, 2005). These include the insulin tolerance test in which insulin is given intravenously and the decline in glucose concentration is measured over the short term (e.g. 20 minutes); the clamp method in which insulin is infused followed by determination of the amount of glucose required to "clamp" or maintain the blood glucose concentration; the homeostatic model assessment (HOMA), which is based on an index of fasting glucose and insulin concentrations and modelling based on results of frequently sampled intravenous glucose tolerance tests.

Fasting insulin

The Livesey meta-analysis found no treatment effects or evidence of trends on fasting insulin concentrations where insulin levels were less than 100 pmol/l (14.4 $\mu\text{U/ml}$). Similarly to fasting blood glucose concentration (see Fasting blood glucose concentration, page 13) this value (100 pmol/l) represents an optimum, and lower levels would not necessarily reflect an improvement. However, an effect of treatment was observed in subjects with hyperinsulinaemia (>100 pmol/l). The evidence base included 18 studies.

Insulin sensitivity

In the Livesey meta-analysis there were 18 relevant studies, which used a number of techniques to measure insulin sensitivity. Subjects included healthy adults, adults

with impaired glucose intolerance, type 1 and type 2 diabetic patients and adults at risk of CHD.

For all studies, lower GI and higher unavailable carbohydrate interventions resulted in a statistically significant 20% improvement in insulin sensitivity. When analysed by subjects' health status, lower GI resulted in significant increases in insulin sensitivity in non-diabetics and type 2 diabetic patients. Analysed by body weight, lower GI resulted in significant increases in insulin sensitivity in overweight subjects, obese subjects and in both of these groups together. Combined means for all of the methods of measuring insulin sensitivity were positive towards improved sensitivity, and statistically significant for some (insulin tolerance test and frequently sampled intravenous glucose tolerance tests) but not for other techniques.

Plasma lipids

Fasting plasma triacylglycerols

Elevated plasma triacylglycerols (also called triglycerides) are a risk factor for CHD (BNF, 2005). The Livesey meta-analysis found a significant effect of GL on fasting triacylglycerols in 30 studies that measured both fat and GL, following treatment with lower GI and/or GL interventions and after adjustment for fat intake. The effect was significant in the 30 studies combined and in studies with subjects in the normal range of body weight. Overall, a 10% fall in fasting triacylglycerols requires a lowering of GL by 30–100 g (glucose equivalents)/day. The effect was only evident when fat intake was considered.

In total, 32 studies reported on fasting plasma triacylglycerols and subjects included healthy adults, hyperlipidaemic subjects, type 1 and type 2 diabetic patients, subjects with impaired glucose intolerance and adults at risk of CHD. There was no clear evidence for a difference following treatment with lower GI, and no interaction with available and unavailable carbohydrate.

Plasma cholesterol concentrations

The Livesey meta-analysis did not analyse the effect of GI and/or GL, and hence the potential influence of unavailable carbohydrate, on plasma cholesterol concentrations. The European Food Safety Authority (EFSA) opinion on dietary reference values for carbohydrates and dietary fibre comments that conflicting results with respect to the effect of GI and/or GL on serum lipids might be due to methodological problems in designing experimental diets that are similar in dietary composition except for differences in GI (EFSA, 2010a).

Three other systematic reviews of intervention trials suggest a positive impact of low GI on plasma total and low density lipoprotein (LDL) cholesterol concentrations (Opperman, 2004; Kelly, 2004; Thomas, 2007). Thomas *et al.* investigated the effect of low GI or low GL diets on changes in body mass, and additionally on total, LDL and HDL cholesterol. Based on analysis of 3 relevant trials, the total cholesterol decreased significantly more with low GI diets than with higher GI diets (weighted mean difference -0.22 mmol/l, 95% confidence interval (CI) -0.43 to -0.02 , $P < 0.05$). The decrease in LDL cholesterol was significantly greater with lower than with higher GI diets (weighted mean difference -0.24 mmol/l, 95% CI -0.44 to -0.05 , $P < 0.05$).

Opperman *et al.* (2004) analysed 16 randomised controlled trials of low vs high GI diets on blood cholesterol concentration. For 13 studies that measured the effect on total cholesterol (in healthy and diabetic subjects, and subjects with CHD), lowering the GI of the intervention diet by 22 (SD 8) units, significantly reduced serum total cholesterol concentration by -0.33 mmol/l (95% CI -0.47 to -0.18 , $P < 0.0001$). Larger reductions in total cholesterol concentrations were observed in subjects with elevated total cholesterol at baseline (>5.2 mmol/l). For 10 studies that measured the effect on LDL-cholesterol (including healthy and diabetic subjects and subjects with CHD),

lowering the GI by 21 (SD 10) units tended to reduce LDL-cholesterol, with an overall reduction of -0.15 mmol/l (95% CI -0.31 to -0.00 , $P=0.06$). Larger decreases in LDL-cholesterol were reported for longer studies in well-controlled type 2 diabetic subjects.

Results from epidemiological studies suggest an association between low GI foods and maintenance of high density lipoprotein (HDL) cholesterol concentrations. A cross-sectional study of middle-aged adults found that the GI of the diet was significantly inversely related to serum HDL-cholesterol concentration, suggesting that low GI diets may help to preserve HDL-cholesterol levels, a desirable effect. A negative relation between GI and HDL-cholesterol (i.e. high GI was associated with low HDL-cholesterol concentration), was observed by Ford and Liu (2001). However, intervention trials have generally shown that HDL cholesterol concentration is unaffected by low GI diets.

Satiety

The Livesey meta-analysis (2008a) did not analyse the effect of GI and/or GL on satiety. However, several studies suggest a beneficial effect of low GI meals on short-term markers of satiety. Studies undertaken in both children and adults have used validated visual analogue scales to measure subjective feelings of satiety and have measured the impact on energy intake at a subsequent meal, where food intake has been measured 3–4 hours after consuming a low or a high GI meal.

In a systematic review by Bornet *et al.* (2007) it was found that in short-term studies (1 day or less), low-glycaemic foods or meals have a higher satiating effect than high-glycaemic foods or meals. Whether this was directly affected by changes in glycaemic response could not be determined. When lowering the GI of a food, other nutritional factors are often changed as well. Confounding factors that could influence both GI and satiety were

taken into consideration in the analysis. However, the authors concluded that it is difficult to tease out the separate effect of the lowering of postprandial glycaemia per se and the effect of higher fibre intake. In studies where these compositional factors were controlled, no consistent relationship between GI and satiety was found (Alfenas, 2005). More studies are needed to investigate the relation between blood glucose responses, satiety and food intake in the long term.

Body weight

In the Livesey meta-analysis (2008a), 23 relevant studies assessed changes in body weight in relation to changing from high to low GI diets. Subjects included healthy adults, adults with impaired glucose intolerance, type 1 and type 2 diabetic patients, and adults at risk of CHD. It is important to know that body weight was not a search item for this meta-analysis and it is likely that several studies investigating the relationship between low GI and body weight have not been included.

The meta-analysis found that reduction in GL was associated with a fall in body weight, and vice versa. The trend was statistically significant for all studies and occurred for *ad libitum* and limited controlled intakes studies, but not for studies with controlled food intake, i.e. with fixed energy intake. Combining the first two of these categories, reductions in body weight occur when GL was reduced by more than 17 g (glucose equivalents)/day and most consistently when GL is reduced by at least 42 g (glucose equivalents)/day (95% confidence interval). Lower GI, lower available carbohydrate and lower metabolisable energy intake were also associated with reductions in body weight.

In a systematic review published after the Livesey meta-analysis, Thomas *et al.* (2007) investigated the effect of low GI or low GL diets on changes in body weight. Published

in the *Cochrane Database of Systematic Reviews*, the analysis included 6 studies. The meta-analysis found that overweight or obese people following low GI and/or low GL diets lost more weight than those consuming control diets. Low GI and/or low GL diets resulted in significantly greater reductions in body mass, body mass index and total fat mass. Research with longer follow-up is needed to determine whether low GI and/or low GL diets help to improve body weight in the long term. In addition, it will be important to determine whether different approaches to lowering GI and/or GL have the same benefits, or whether specific components particularly contribute towards these effects.

The Diogenes 8 European Country Dietary Intervention Study has investigated the efficacy of 5 reduced-fat diet groups, differing in GI and protein content, on the prevention of weight regain. A difference in GI of >10 units between diets was associated with benefits for weight maintenance, though the effect was less marked than that for a higher protein diet (Larsen, 2010).

Other health indicators

Several studies have reported on the relation between GI and GL and the incidence of various types of cancer. The findings of such research, assessing associations and investigating heterogeneous endpoints, is inconsistent and shows only small, if any, relationships.

Recovery from exercise and sports performance

It has been suggested that consuming low GI carbohydrate foods prior to exercise may attenuate insulin-mediated metabolic effects and help to maintain carbohydrate availability.

A number of studies have investigated whether eating low GI carbohydrates is more effective than higher GI

foods in influencing sports performance and recovery from exercise.

Some data have shown that eating a low GI meal before exercise results in a higher rate of fat oxidation during exercise compared with a high GI meal, and the same effect has been observed in trained men and women consuming low GI carbohydrates before endurance exercise. Some data have also suggested that increased endurance capacity following a low GI post-exercise (recovery) diet is a consequence of increased fat oxidation following the low GI diet. The GI of carbohydrates has also been shown to influence glycogen storage in skeletal muscle and circulating non-esterified fatty acid concentrations. Several studies suggest that high GI foods are beneficial for faster energy uptake while undertaking physical exercise. This is especially important in endurance sports.

However, studies have used different designs with different feeding protocols, different amounts of carbohydrate, and different ranges of GI and hence not all studies demonstrate that lower GI food enhances the metabolic response to exercise or exercise performance.

Cognitive function

The brain is dependent on glucose for its energy source, with consumption estimated at 6 g/hour, which might increase with more demanding tasks. Research has thus focussed on glucose administration and cognitive function. Positive effects of glucose versus non-caloric controls on memory functions have been reported in several studies, including studies in the elderly and in children (Manning, 1990; Benton and Stevens, 2008). Investigations into the effects of rapidly and slowly available glucose suggest a favourable effect of lower glycaemic responses later in the morning (Benton, 2003; Wesnes, 2003). However, a recent meta-analysis of 8 studies on GI and/or GL considering heterogeneous endpoints found that the overall evidence was insufficient to reach firm conclusions (Gilsenan, 2009).

Conclusions

There is a growing body of scientific evidence that compositional changes affecting the glycaemic response to foods is associated with beneficial health outcomes related to key public health priorities, including type 2 diabetes, weight management and CHD.

Often, dietary interventions to lower GI have also resulted in an increased intake of unavailable carbohydrate and a varied intake of available carbohydrate and other macronutrients. Many studies investigating lower GI interventions have actually used lower GL interventions. Hence, it is difficult to disentangle the specific effects of these various dietary manipulations. Furthermore, for certain outcomes, it is uncertain whether effects can be attributed to changes in the blood glucose itself, or to other characteristics of low GI and low GL diets. It is suggested that changes in unavailable carbohydrate (e.g. dietary fibre) independent of GI, may have at least as big an effect on health outcome as GI itself.

Dietary interventions to lower GI and/or GL have been shown to:

- Improve fasting blood glucose in subjects with poorer blood glucose control
- Reduce blood glycated protein concentration, with greater effects in subjects with poorer glycaemic control
- Improve insulin sensitivity in non-diabetics, overweight subjects, obese subjects and patients with type 2 diabetes, particularly in association with higher unavailable carbohydrate intake
- Reduce fasting insulin in subjects with hyperinsulinaemia
- Reduce fasting triacylglycerols among those with the highest baseline concentrations

Optimum reduction in fasting blood glucose and fructosamine occurs with a GI of <45, GL of ≤ 100 g (glucose

equivalents)/day or intakes of unavailable carbohydrate ≥ 25 g/day. A 10% fall in fasting triacylglycerols requires a reduction in GL of 30–100 g (glucose equivalents)/day.

Higher intakes of unavailable carbohydrate have an additive effect on reducing fasting blood glucose to that of lower GI or GL. A combination of these two dietary manipulations achieves optimum effects.

Evidence from prospective randomised controlled trials supports the view that lowering the GI of the diet by 10 points has beneficial effects on total and LDL-cholesterol, with larger decreases in LDL-cholesterol reported for longer studies in well-controlled type 2 diabetics.

Overall, there is evidence of positive health effects from a reduction in dietary glycaemic impact in diabetic patients, but only weak evidence for an effect in healthy subjects. It should be realised, however, that within the general population there is a continuum from normal to impaired glucose tolerance. The greater the divergence of glycaemic control from the norm, the greater the potential benefits of reducing GI or GL or increasing intake of unavailable carbohydrate. This is consistent with the fact that a decrease in blood glucose below the optimum that is maintained in healthy subjects would lead to hypoglycaemia and thus be considered detrimental.

Short-term studies (1 day or less) suggest that low-glycaemic foods or meals might have a higher satiating effect than high-glycaemic foods or meals. Studies are needed to investigate the longer-term effects, and relating any such effects to body weight loss or body weight maintenance.

Lower GI, lower GL, lower available carbohydrate and lower metabolisable energy intake are associated with reductions in body weight. In these studies, a reduction in GL of more than 17 g (glucose equivalents)/day was necessary for reducing body weight.

In 2010, the European Food Safety Authority (EFSA) published a Scientific Opinion on the substantiation of health claims related to carbohydrates that induce low/reduced glycaemic responses and carbohydrates with a low glycaemic index (EFSA, 2010b). In the summary of this opinion it was stated:

The Panel considers that carbohydrates that induce a low/reduced glycaemic response and carbohydrates with a low glycaemic index (e.g. <55) are not sufficiently characterised. On the basis of the data presented, the Panel concludes that a cause and effect relationship has not been established between the consumption of carbohydrates that induce a low/reduced glycaemic response or carbohydrates with a low glycaemic index and the claimed effects.

Subsequently, in 2011 the EFSA provided an additional opinion relating to glycaemic response (EFSA, 2011). In the summary of this EFSA opinion it was stated that: “The EFSA acknowledges that a reduction of postprandial blood glucose may be a beneficial physiological effect (as long it does not disproportionately increase the insulinemic response).” In this context, the EFSA has therefore approved reductions of postprandial glucose as well as lower glucose rises as mediated by specific ingredients such as pectin, hydroxymethylcellulose, resistant starch, sucralose, polyols, polydextrose and isomaltulose.

GLYCAEMIC CONCEPT IN ACTION

Guidelines for glycaemic labelling

Global dietary guidelines recommend that 45–60% of energy intake is derived from carbohydrates (EFSA, 2010a; USDA, 2005; Mann, 2007; FAO/WHO, 1998). These guidelines refer to available carbohydrates, but do not take into account the different glycaemic responses.

Of the different glycaemic concepts available, glycaemic index has mainly been used, especially for people with impaired glucose tolerance to control blood glucose. In addition, communication on GI as a food property has also been used in various countries.

There are global differences in the use of GI by health professionals, such as Diabetes Associations, and their recommendations for diabetic patients. For example, Diabetes Australia is supportive of the concept and recommends diabetics to eat more low and intermediate GI foods, in the context that GI is only one part of a sound approach to healthy eating for diabetics. Diabetes UK recommends diabetics to include a low GI food with each meal or snack. In North America, health professionals have communicated less on the use of GI.

Labelling

There has been much discussion globally on how to regulate labelling the glycaemic properties of foods and there is considerable variation in the ways that regulatory agencies around the world approach this issue. Where statements or claims have been used on food products, GI is the most commonly used, with relatively little use of GL or GGE.

For the practical use of GI values, and to assist communication of the concept, it is useful to refer to low, medium and high GI foods. However, the dose–response relation between GI and health outcomes has not been quantified and such categorisation is therefore subjective. The following cut-offs for categories of foods are based on the work of Brand Miller (Brand Miller, 2003b) and are related to improvement in glycaemic control in subjects with diabetes:

- Low GI: 55 and below
- Medium GI: 56–69
- High GI: 70 and above

Although categorisation is useful, a drawback is the risk of suggested overprecision because the numerical GI values themselves represent ranges and provide guidance for ranking.

Experiences from around the world

Australia and New Zealand

Australia and New Zealand are the countries that have adopted GI labelling to the greatest extent. These countries have regulated the use of GI in relation to food communication in the draft standard 1.2.7 (March 2009) for *Nutrition, Health and Related Claims*, Food Standards Australia and New Zealand (FSANZ). Australia has an approved Australian Standard *Glycemic Index of Foods* (AS 4694 – 2007) for the determination of the GI of carbohydrate-containing foods (Standards Australia, 2007).

The GI Symbol programme, an independent GI certification programme, was introduced in 2002 as a quality marker to assist consumers with healthy eating. The programme consists of a symbol or signpost label indicating that the GI of the product has been measured and that the product conforms to set nutritional criteria for total fat, saturated fat, sodium and energy, and where appropriate fibre and calcium. A key aim of the

programme is that GI is not used in isolation but in the context of other dietary recommendations, making the symbol a credible signpost for healthier choices. The symbol is a registered trademark in Australia, the European Union, Japan and the United States.

The associated website (www.glycemicindex.com) provides a comprehensive database of GI values for foods that have been tested around the world. The database also includes values for GL and grams of carbohydrate per serving.

In a global consumer research survey about the use of nutrition labelling, 21,261 consumers from 38 markets were interviewed online in spring 2005. Australia had the highest percentage of respondents (82%) that were aware of the low GI diet, closely followed by 76% in New Zealand. Globally awareness was 41% (Nielsen, 2005).

South Africa

Legislation for glycaemic index labelling has previously been discussed in South Africa, but glycaemic index claims were not included in the South African labelling regulations published in March 2010 (South Africa Department of Health, 2010). In the absence of legislation, the Glycaemic Index Foundation South Africa (GIFSA) runs an endorsement scheme with four logos for use by industry. The GIFSA logo aims to direct consumers to low GI, lower fat and reduced sodium alternatives. Four variations of the logo distinguish between “frequent foods” (low GI, very low fat), “often foods” (low GI, low fat), “special treats” (intermediate GI, lower fat) and “best after exercise” (high GI). Use of the Diabetes South Africa logo on “frequent” and “often” foods provides third party verification that the endorsed product meets appropriate criteria and is suitable for diabetics.

Europe

Europe is moving towards a harmonised regulatory system for nutrition and health claims based on the prior

approval of health claims, and with nutrition content claims restricted to those on a permitted list. The regulation covers all commercial communications including product labelling, advertising and the presentation of food products sold to consumers.

EFSA has stated that the reduction of postprandial glycaemic responses (as long as postprandial insulinaemic responses are not disproportionately increased) may be a beneficial physiological effect. EFSA has issued several favourable opinions for different ingredients like resistant starch, pectins and several others for which they consider that a cause and effect relation have been established between the consumption of the ingredient in drinks or foods and the reduction of postprandial glucose response. Concomitantly, the EFSA acknowledges that impaired glucose tolerance is common in the general population of adults. The respective food ingredients comprise available carbohydrates as well as fibres (EFSA, 2010b).

Prior to this, GI claims such as “low GI” have been in use in various European countries. In the global consumer research survey on the use of nutrition labelling, 9% of European consumers were found to check labels regularly for low GI. Consumer awareness of low GI was highest in Portugal (74%) followed by Italy (73%), France (61%), Sweden (60%) and Spain (53%) (Nielsen, 2005).

USA

In the USA, structure–function claims do not require prior approval, and claims relating to the glycaemic properties of foods can be freely made. Regarding disease risk reduction claims, the FDA operates a system of prior approval and as yet no such claims have been established for GI or other measures of glycaemic response. The Glycaemic Research Institute (a commercial organisation) in the USA has developed Seals of Approval for use on food labels, such as “low glycaemic” and “diabetic friendly”. In the global consumer research survey on the use of nutrition labelling, although America had one

of the highest scores for percentage of respondents interested in carbohydrate labelling, this did not translate to information about GI labelling.

Conclusions

There seems to be agreement across the world that low GI foods and diets are generally beneficial for people with diabetes and with impaired glucose metabolism. Whether low GI is considered beneficial for healthy people is perceived, legislated and communicated differently in different countries. Clearer insight into the cause and effect of low GI foods on health is important before authorities, such as those in Europe and the USA, will endorse the communication of GI-associated claims to consumers. This situation arises also because there are many different ways of lowering GI or GL, not all of which have the same effects on health. In Europe, consequently, the beneficial effects of ingredients that trigger lower rises in postprandial glucose as well as the reduction of postprandial blood glucose by fibres has recently been positively assessed for subjects with impaired glucose intolerance.

Overall, reducing the glycaemic impact of the diet in association with an enhanced fibre intake, other aspects being equal, can be seen as a valid nutritional objective for those consuming a typical “Western” style diet. However, more research is needed to better understand the underlying mechanisms in different populations. This becomes more important the more the prevalence of hidden impaired glucose intolerance increases in the general population.

ABBREVIATIONS AND GLOSSARY

Abbreviations

AUC	Area under the curve
BMI	Body mass index
CHD	Coronary heart disease
CI	Confidence interval
CVD	Cardiovascular disease
EFSA	European Food Safety Authority
FDA	US Food and Drug Administration
FSANZ	Food Standards Australia and New Zealand
GI	Glycaemic index
GIFSA	Glycaemic Index Foundation South Africa
GL	Glycaemic load
GGE	Glycaemic glucose equivalents
GRI	Glycaemic Research Institute
HbA1c	Glycated haemoglobin
HDL	High density lipoprotein
HOMA	Homeostatic model assessment of insulin resistance
IAUC	Incremental area under the (blood glucose response) curve
IFG	Impaired fasting glucose
IGT	Impaired glucose tolerance
IU	International Unit
LDL	Low density lipoprotein
OGTT	Oral glucose tolerance test
RCT	Randomised controlled trial
RGI	Relative glycaemic impact
SD	Standard deviation
SEM	Standard error of mean
USDA	US Department of Agriculture

Glossary

Absorbed/absorption: The passage of materials, originating from the diet, from the gastrointestinal tract through the wall of the intestine into the blood.

Blood glucose, fasting: Plasma glucose concentration measured after an overnight fast. The normal level in early morning is ≤ 5.6 mmol/l; impaired fasting glucose is defined as a level of 6.1–7.0 mmol/l and may be a risk factor for type 2 diabetes.

Blood glucose, postprandial: Plasma glucose concentration following a meal; it is raised and prolonged in diabetics.

Body mass index: Body weight (kg) divided by height (m) squared; defines optimal (18.5–24.9 kg/m²), overweight (25–29.9 kg/m²) and obese (≥ 30 kg/m²).

Carbohydrates: Components of foods containing carbon, hydrogen and oxygen. The term carbohydrate encompasses simple sugars, monosaccharides (e.g. glucose) and disaccharides (e.g. sucrose), oligosaccharides (several monosaccharide units) and polysaccharides. Starch is the only important food polysaccharide that can be digested in the small intestine. The indigestible polysaccharides are the main components of “dietary fibre”.

Case control study: An observational study that compares the exposure to a suspected cause of a disease in people having that disease (the cases) to the exposure in those without that disease (controls); exposure is thus assessed retrospectively. See also “cross-sectional study”.

Cholesterol: Lipid (sterol) made in the body from acetyl-CoA and present in the diet; a constituent of cell membranes (especially in nervous system tissues), blood and atherosclerotic plaques.

Cholesterol, total: Sum of concentrations of cholesterol in very low, low and high density lipoproteins (VLDL, LDL and HDL).

Claim: Any message or representation, including pictorial, graphic or symbolic representation, that states, suggests or implies that a food has particular characteristics.

Clinical study: Study of any therapeutic, prophylactic or diagnostic agent conducted in human subjects.

Cohort study: Prospective observational study in which data on exposure to suspected causes of e.g. a disease, are collected in a selected/recruited group of people who do not yet have the disease(s) under investigation. The subjects are then followed for a period of time, after which it can be assessed whether development of the disease is related to the (presence of) suspected causes.

Confounding factors, confounders: A certain exposure may be associated with a disease or other outcome, without this association being causal. This can result from a third factor being a cause of both; such a factor is referred to as “confounder”.

Coronary heart disease: Restriction of blood flow to the coronary arteries, often characterised by chest pains (angina). May result in a heart attack. The main cause of reduced blood flow is the accumulation of atherosclerotic plaques, a disease known as atherosclerosis. The blockage of an already narrowed artery is thrombosis.

Cardiovascular disease: Any one of numerous abnormal conditions characterised by dysfunction of the heart and blood vessels, e.g. coronary heart disease, congestive heart failure, stroke, cerebrovascular events and peripheral artery disease.

Cross-sectional study: Study design that relates the rates of a certain exposure to the levels of an outcome of interest in a number of individuals or populations. A key feature is that exposure and outcome are measured at the same point in time.

Diabetes mellitus: Metabolic disorder characterised by a chronically elevated glucose level in the blood resulting from either an absolute or relative shortage of insulin. In the case of the first, the pancreas fails to secrete insulin (type 1 or juvenile diabetes). In the case of the latter, the main problem is that target tissues are insensitive to insulin (type 2 or maturity onset diabetes). Patients with type 1 diabetes require regular administration of insulin. In contrast, patients with type 2 diabetes may actually have a high blood concentration of insulin (hyperinsulinaemia).

Dietary fibre: The edible parts of plants or analogues of carbohydrates that are resistant to digestion and absorption in the small intestine and undergo complete or partial fermentation in the large intestine.

Endpoint: Variable or outcome that is relevant in itself, e.g. survival time after medical surgery, time to run a marathon, gastrointestinal discomfort, or risk of a disease. The level of a surrogate or intermediate endpoint – also referred to as “marker” or “biomarker” – is in itself not relevant, but is indirectly relevant because it reflects a relevant endpoint. See also “(bio) marker”.

Energy intake: The chemical energy in foods and drinks that can be metabolized to produce energy available to the body.

Epidemiology: Study of health and the occurrence of diseases and their predictors and causes.

Fructosamine: Glycated serum protein complex reflecting the average blood glucose concentration of the past 2–3 weeks. It is used in the management of insulin therapy for diabetes mellitus.

Glycated haemoglobin (HbA1c): Form of haemoglobin used primarily to identify the average plasma glucose concentration over the past 8–10 weeks.

Glycaemic (available) carbohydrates: Total carbohydrate minus non-glycaemic carbohydrate, i.e. carbohydrates that are absorbed into the bloodstream as carbohydrates and capable of increasing blood glucose levels and capable of being metabolized when consumed.

Glycaemic control: Degree to which favourable levels of glycaemia are achieved in diabetic patients. Strict glycaemic control, e.g. near normal, reduces the risk of complications.

Glycaemic index: The incremental area under the blood glucose response curve of a 50 g digestible carbohydrate portion of a test food expressed as a percentage of the response to the same amount of digestible carbohydrate from a standard food (glucose or bread) taken by the same subject. This is a measure of the effect on blood glucose levels produced by consuming a given quantity of a test carbohydrate food, expressed relative to the effect on blood glucose levels produced by consuming the same amount of a reference carbohydrate food, usually glucose.

Glycaemic response: The rise in blood glucose concentration following the ingestion of a food. Glycaemic response is a measure of the increase in postprandial blood glucose concentration after consuming a carbohydrate-containing food.

Health claim: Any representation that states, suggests or implies that a constituent of a food influences health.

High density lipoprotein (HDL): Plasma lipoprotein with high density; contains relatively low amounts of cholesterol and other lipids and a high amount of protein. It is regarded as beneficial because it transports cholesterol from atherosclerotic plaques to the liver, from where it is eliminated in the intestine (see also lipoproteins and low-density lipoproteins, LDL).

Homeostatic model assessment of insulin resistance (HOMA): Method quantifying insulin resistance and beta-cell functions from fasting glucose and insulin concentrations.

Hyperinsulinaemia: Higher than normal level of insulin in the blood.

Hyperlipidaemia: Abnormal elevations of fat (lipids) in the blood, including LDL-cholesterol, and triglycerols.

Hyperglycemia: Higher than normal level of glucose in the blood.

Incremental area under the (blood glucose response) curve (IAUC): The area under the curve is calculated as the incremental area under the blood glucose response curve, including all the area below the curve above the fasting or baseline concentration and ignoring the area beneath the fasting concentration.

Insulin: Hormone secreted by the pancreas that has a central role in the control of carbohydrate metabolism by lowering blood glucose levels.

Insulin resistance: Impaired sensitivity of target tissues to insulin, impairing insulin-stimulated uptake of glucose by tissues.

Intervention study: Study in which investigators intervene by allocating and establishing one or more treatments ("interventions") to or in certain subjects, after which they observe outcomes of interest. See also "observational study" and "randomised controlled trial".

Lipoproteins: Particles of protein and lipids that enable lipids to be transported by the blood in the plasma.

Low density lipoproteins (LDL): Plasma lipoproteins containing high concentrations of lipids (which are low in density compared to that of water), including cholesterol. Increased concentrations are a risk factor for coronary heart disease.

(Bio)Marker: Variable that is of interest because it marks or reflects a certain phenomenon of interest. See also “endpoint”.

Meta-analysis: Quantitative summary of several individual studies of a similar type. Both intervention and observational studies can be meta-analysed. See also “pooled analysis”.

Observational study: Study in which researchers do not intervene but only observe outcomes of interest and the levels of suspected causes, e.g. cohort or case-control study. See also “cross-sectional study” and “intervention study”. Observational studies are often loosely referred to as epidemiological studies.

Pooled analysis: Analysis of the combined, original data of several individual studies. See also “meta-analysis”.

Postprandial: Occurring after a meal.

Quintile: One fifth of a distribution of a certain parameter.

Randomised controlled trial (RCT): Study design in which subjects are randomly allocated to study groups. As a result, the groups will not be expected to differ systematically, except with regard to an intervention that one group will undergo and the other will not. As a result, the effects observed can principally be ascribed to the intervention. See also “intervention study”.

Relative glycaemic impact: Weight of glucose that would induce a glycaemic response equivalent to that induced by a given amount of a specific food.

Relative risk: Ratio of the outcome rate among persons exposed to a certain factor divided by the outcome rate among persons not exposed.

Satiety: Feeling of fullness and inhibition of further food intake once a meal has finished. It is brought about by the food engaging with body systems to maintain this inhibition over eating and to control appetite.

Sugars: Simple carbohydrates, generally with a sweet taste and soluble in water. The term is usually reserved for mono- and disaccharides. The disaccharide sucrose is commonly referred to as “sugar”.

Unavailable carbohydrates: Carbohydrates that are not absorbed in the small intestine and that become available in the colon for fermentation or excretion (e.g. dietary fibre).

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