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Health potential of polyols as sugar replacers, with emphasis on low glycaemic properties

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Polyols are hydrogenated carbohydrates used as sugar replacers. Interest now arises because of their multiple potential health benefits. They are non-cariogenic (sugar-free tooth-friendly), low-glycaemic (potentially helpful in diabetes and cardiovascular disease), low-energy and low-insulinaemic (potentially helpful in obesity), low-digestible (potentially helpful in the colon), osmotic (colon-hydrating, laxative and purifying) carbohydrates. Such potential health benefits are reviewed. A major focus here is the glycaemic index (GI) of polyols as regards the health implications of low-GI foods. The literature on glycaemia and insulinaemia after polyol ingestion was analysed and expressed in the GI and insulinaemic index (II) modes, which yielded the values: erythritol 0, 2; xylitol 13, 11; sorbitol 9, 11; mannitol 0, 0; maltitol 35, 27; isomalt 9, 6; lactitol 6, 4; polyglycitol 39, 23. These values are all much lower than sucrose 65, 43 or glucose 100, 100. GI values on replacing sucrose were independent of both intake (up to 50 g) and the state of carbohydrate metabolism (normal, type 1 with artificial pancreas and type 2 diabetes mellitus). The assignment of foods and polyols to GI bands is considered, these being: high (> 70), intermediate (> 55–70), low (> 40–55), and very low (< 40) including non-glycaemic; the last aims to target particularly low-GI-carbohydrate-based foods. Polyols ranged from low to very low GI. An examination was made of the dietary factors affecting the GI of polyols and foods. Polyol and other food GI values could be used to estimate the GI of food mixtures containing polyols without underestimation. Among foods and polyols a departure of II from GI was observed due to fat elevating II and reducing GI. Fat exerted an additional negative influence on GI, presumed due to reduced rates of gastric emptying. Among the foods examined, the interaction was prominent with snack foods; this potentially damaging insulinaemia could be reduced using polyols. Improved glycated haemoglobin as a marker of glycaemic control was found in a 12-week study of type 2 diabetes mellitus patients consuming polyol, adding to other studies showing improved glucose control on ingestion of low-GI carbohydrate. In general some improvement in long-term glycaemic control was discernible on reducing the glycaemic load via GI by as little as 15–20 g daily. Similar amounts of polyols are normally acceptable. Although polyols are not essential nutrients, they contribute to clinically recognised maintenance of a healthy colonic environment and function. A role for polyols and polyol foods to hydrate the colonic contents and aid laxation is now recognised by physicians. Polyols favour saccharolytic anaerobes and aciduric organisms in the colon, purifying the colon of endotoxic, putrefying and pathological organisms, which has clinical relevance. Polyols also contribute towards short-chain organic acid formation for a healthy colonic epithelium. Polyol tooth-friendliness and reduced energy values are affirmed and add to the potential benefits. In regard to gastrointestinal tolerance, food scientists and nutritionists, physicians, and dentists have in their independent professional capacities each now described sensible approaches to the use and consumption of polyols.

Diabetes; Coronary heart disease; Caries; Laxation; Digestive health; Glycaemic index; Insulinaemic index; Food energy; Sucrose; Sucrose replacer; Polyol; Erythritol; Xylitol; Sorbitol; Mannitol; Maltitol; Isomalt; Lactitol; Polyglycitol

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Abbreviations: DM, diabetes mellitus; FPG, fasting plasma glucose; GI, glycaemic index; GL, glycaemic load; GT, glucose tolerance; HbA_{1c}, glycated or glycosylated haemoglobin; II, insulinaemic index; RIR, relative insulin response.

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Introduction

Although polyols (hydrogenated carbohydrates) have been reviewed from various perspectives (Wang & van Eys, 1981; Ziesenitz & Siebert, 1987; Dills, 1989; Livesey, 1992, 2001; Zumbé *et al*. 2001) no reviews have considered their glycaemic indices (GI) and there has been little consideration of their prospects in respect of the health of the digestive tract other than their role in caries prevention. GI ranks foods and carbohydrates according to their ability to raise the concentration of glucose in the blood (Jenkins *et al*. 1981). Also the overall glycaemic load (GL) from the diet has implications for the development and management of metabolic syndrome, diabetes, and CHD and the control of metabolic markers such as glycated proteins (glycated haemoglobin (HbA_{1c}), fructosamine), plasma triacylglycerols, HDL and sensitivity to insulin (Brand *et al*. 1991; European Association for the Study of Diabetes, 1995, 2000; Salmerón *et al*. 1997*a*,*b*; Food and Agriculture Organization, 1998; Frost *et al*. 1998, 1999; Bär, 2000; Bastyr *et al*. 2000; Buyken *et al*. 2000, 2001; Canadian Diabetes Association, 2000; Diabetes UK, 2000, 2002; Liu *et al*. 2000*a*,*b*; Stratton *et al*. 2000; Bellisle, 2001; Ford & Liu, 2001; Gilbertson *et al*. 2001; Kapur & Kapur, 2001; Khaw *et al*. 2001; International Diabetes Institute Australia, 2002; Jenkins *et al*. 2002; Livesey, 2002*a*).

Widespread knowledge of the GI concept largely postdates many relevant studies on polyols and those studies having reported the GI of polyols have sometimes used calculation methods that are no longer acceptable. These studies are revisited to place information available in a modern context. The multiple potential health benefits from using polyols as replacers of sugars, maltodextrins and glucose syrups or in laxation are examined under the concepts of glycaemia and insulinaemia, reduced energy, caries reduction, and digestive health. The review begins with background on the definition, description and metabolism of polyols.

Polyols

Definition of 'polyol'

'Sugar replacer', 'sugar alcohol', 'hydrogenated carbohydrate', and 'polyol' are synonyms for a sub-class of carbohydrates present in foods. The defining characteristic is the occurrence of an alcohol group (>CH-OH) in place of the carbonyl group (>C=O) in the aldose and ketose moieties of mono-, di-, oligo- and polysaccharides; hence polyols are not sugars, and generally carry the suffix '-itol' in place of the suffix '-ose' according to modern carbohydrate nomenclature (McNaught, 1996). The name 'polyol' is an abridgement of 'polyalcohol' or 'polyhydric alcohol'. Preferred names are 'polyol' or 'hydrogenated carbohydrate'; the latter makes explicit that these substances are carbohydrate. Individual polyols are described in Table 1 and in more detail later (p. 164).

Classification amongst other carbohydrates

Because polyols are not sugars they are permitted in sugar-free and tooth-friendly products (European Communities, 1994). The distinction between sugars and polyols is important yet frequently overlooked, the consultation by the Food and Agriculture Organization (1998) being the most significant recent example. Sugars are legally defined for nutrition labelling purposes as mono- and disaccharide only. In contrast, polyols may be hydrogenated mono-, di-, but also oligo- and polysaccharide (Table 2). Polyols also contribute unavailable carbohydrate to fermentation analogous to dietary fibre to which it may contribute (American Association of Cereal Chemists, 2001). Examples of named carbohydrates in each subclass of food carbohydrates are given in Table 2 to help show their difference from polyols. The overall order in which the carbohydrates are listed here is governed by molecular weight or degree of polymerisation, as suggested by the Food and Agriculture Organization (1998). However, no real physiological meaning can be attached to this order, nor does this order help interpretation of carbohydrate terminology in regulatory food codes. To be usefully informative the nutrition information panel will in future require other information; in this context the GI, GL (GI × amount of carbohydrate) and other possible expressions of glycaemic potential are candidates for possible inclusion in future food labelling and food tables.

Individual polyols: description, absorption and metabolism

The physiological attributes of polyols, i.e. low cariogenicity, low glycaemia, low insulinaemia, low energy value, source of substrate for a healthy colon and intestinal tolerance are linked through the common property of polyols being difficult to digest or slow to metabolise yet relatively easy to ferment in the colon. This property results from the hindrance to digestion and absorption by the alcohol group that replaces the carbonyl group and the occurrence of saccharide linkages other than the α1–4 and α1–6 present in starches and sucrose. Thus, a low digestibility and/or slow hepatic glucose release is the determinant of their low glycaemic and insulinaemic response properties.

During the time polyols are resident in the mouth, they resist fermentation and acidogenesis by the micro-organisms of dental plaque (Willibald-Ettle & Schiweck, 1996; Kandelman, 1997) and are not absorbed via the stomach to any significant degree. Absorption that does occur is by passive diffusion of monosaccharide polyol along a concentration gradient (Herman, 1974). Disaccharide and higher polyols are too large to diffuse from the gut into the circulation in amounts more than 2 % of oral intake (Livesey, 1992). Some di-, oligo- and polysaccharide polyols may liberate glucose, but as their digestion is slow and incomplete this does not result in a substantial rise in blood glucose, as will be shown in later sections (p. 168). The small intestine is probably less permeable distally so that co-released monosaccharide polyol may be less readily absorbed than the same monosaccharide polyol taken orally. Once absorbed, monosaccharide polyols are excreted via the kidneys, oxidised directly or converted to glycogen or glucose in the liver; the route of metabolism and excretion depends on their structure. Unabsorbed carbohydrate from polyols is generally fermented completely by the colonic microflora (Livesey, 1992).

Table 1. Polyol specifications

Polyol	Formula	Saccharide type	Generic form	Molecular weight (Da)	Synonyms*	Further details†
Erythritol	$C_4H_{10}O_4$	Mono-	Tetritol	122·12	Hydrogenated erythrose *meso*-Erythritol Erthrite *tetra*-Hydroxybutane 1,2,3,4-Butanetetrol Erythrol Physitol	FNP 52/7
Xylitol	$C_5H_{12}O_5$	Mono-	Pentitol	152·15	Hydrogenated xylose Xylite	FNP 52/4
Mannitol	$C_6H_{14}O_6$	Mono-	Hexitol	182·17	Hydrogenated mannose D-Mannitol Mannite	FMP 52/4
Sorbitol	$C_6H_{14}O_6$	Mono-	Hexitol	182·17	Hydrogenated glucose D-Sorbitol Glucitol Sorbol Sorbit	FNP 52/4
Sorbitol syrup	Mixed mono- and smaller amounts of other hydrogenated saccharides‡				Hydrogenated glucose syrup D-Glucitol syrup	FNP 52/4
Lactitol	$C_{12}H_{24}O_{11}$	Di-	Hexopyranosyl-hexitol	344·3	Hydrogenated lactose β-D-Galactopyranosyl-1-4-D-sorbitol β-D-Galactopyranosyl-1-4-D-glucitol Lactositol Lactit Lactosbiosit	FNP 52/4
Isomalt	$C_{12}H_{24}O_{11}$	Mixed di-	Hexopyranosyl-hexitol	344·3	Hydrogenated isomaltulose Hydrogenated palatinose Mixture of α-D-glucopyranosyl-1-6-D-sorbitol and α-D-glucopyranosyl-1-1-D-mannitol	FNP 52/4
Maltitol	$C_{12}H_{24}O_{11}$	Di-	Hexopyranosyl-hexitol	344·3	Hydrogenated maltose α-D-Glucopyranosyl-1-4-D-sorbitol α-D-Glucopyranosyl-1-4-D-glucitol	FNP 52/4
Maltitol syrups	Mixed, ≥ 50 % di-, and lesser amounts of mono- and higher saccharides‡				Hydrogenated high-maltose glucose syrup Hydrogenated starch hydrolysate Dried maltitol syrup Maltitol syrup powder Several forms are available: Regular, about 53 % maltitol Intermediate, about 73 % maltitol High, about 98 % maltitol High polymer, about 50 % hydrogenated polymer	FNP 52/5
Polyglycitol	Mixed, < 50 % di- and of other especially oligo- and polysaccharides‡				Polyglucitol Hydrogenated starch hydrolysate	FNP 52/6

FNP 52, Food and Nutrition paper 52.
* Excluding proprietary names.
† Food and Agriculture Organization (1996–1999), addenda 4–7.
‡ For details, see p. 167.

Representative values for the absorption, fermentation and urinary excretion of polyols are shown in Table 3. The data are drawn from information collected by Livesey (1992), the Life Sciences Research Office (1994, 1999), and other material described later (pp. 166–168). For the present, no distinction is made between the results of digestibility studies assessing absorption from liquids and solids on the ground that such distinctions at moderate polyol intake are based on invasive methodology in which solids may increase the non-recovery of polyols at the ileum by increasing retention in the stomach and upper gastrointestinal tract rather than increasing absorption. Differences in the tolerance of polyols when consumed in liquid and solid meals are ascribable to different rates of stomach emptying rather than differences in the extent of digestion and absorption (Livesey, 1990*a*, 2001). Excessive intake might cause absorption to be lowered and potentially would affect the glycaemic response to polyols, though, as will be seen in subsequent dose–response data (p. 169), this appears not to happen.

Table 2. Classification of the major carbohydrates in foods (modified from Food and Agriculture Organization, 1998)

Class	DP	Sub-class	Examples
Monosaccharides	1	Sugars	Glucose, fructose, galactose
		Hydrogenated monosaccharides	Erythritol, xylitol, mannitol, sorbitol
Disaccharides	2	Sugars	Sucrose, maltose, lactose, trehalose
		Hydrogenated disaccharides	Maltitol, isomalt, lactitol
Oligosaccharides	3–9	Malto-oligosaccharides	Maltodextrins.
		Other oligosaccharides	Raffinose, stachyose, fructo-oligosaccharides, galacto-oligosaccharides.
		Hydrogenated oligosaccharides	Hydrogenated starch hydrolysate
Polysaccharides	>9	Starch	Amylose, amylopectin, modified starches
		NSP	Cellulose, hemicelluloses, pectins, etc
		Hydrogenated polysaccharides	Polyglycitol, hydrogenated polydextrose

DP, degree of polymerisation.

Table 3. Approximate absorption, fermentation and urinary excretion of polyols*

	Absorption (g/100 g)	Fermentation (g/100 g)	Urinary excretion (g/100 g)
Erythritol	90	10	90
Xylitol	50	50	<2
Sorbitol	25	75	<2
Mannitol	25	75	25
Isomalt	10	90	<2
Lactitol	2	98	<2
Maltitol	40	60	<2
Maltitol syrup			
Regular, intermediate, high	about 50†	about 50†	<2
High-polymer	about 40‡	about 60‡	–
Polyglycitol	about 40†	about 60†	<2

* Data are given to the nearest 5 %, except when close to zero, when data are to the nearest 2 % or for urinary excretion where an upper limit of 2 % appears. For references, see pp. 166–168.
† Data are based solely on glycaemic and insulinaemic responses, which may give a lower limit.
‡ Based on *in vitro* digestion.

Erythritol. This small (four-carbon, tetritol) molecule is absorbed readily by diffusion, with approximately 10 % escaping to the large intestine in man (Oku & Noda, 1990; Noda *et al*. 1994; Bornet *et al*. 1996*a*). Absorbed erythritol distributes widely through the tissues but its metabolism is minimal and being poorly reabsorbed via the kidneys it is essentially excreted unused in urine (Bernt *et al*. 1996).

Xylitol. Absorption of xylitol from the small intestine occurs less readily than the smaller molecule erythritol, causing more to be fermented in the large bowel. Estimates of the extent of fermentation range from 50 to 75 % (Livesey, 1992; Life Sciences Research Office, 1994) with the lower value being more consistent with the size of this molecule. Thus, based on D-arabitol as a non-metabolisable marker of pentitol absorption, a similar absorption of oral xylitol in man would suggest it to be 53 % (Bär, 1990). This is corroborated by the present author who has predicted its absorption based on molecular weight for a series of polyols (glycerol, erythritol, mannitol and lactitol) to be 48 % (see Livesey, 1992). On the basis of energy values for xylitol proposed by several experts and authorities, absorbability by consensus is 49 %; this being the average of values estimated by the Dutch Nutrition Council (1987), Bär (1990), Bernier & Pascal (1990), Livesey (1992); Life Sciences Research Office (1994), and Brooks (1995). The liver readily sequesters absorbed xylitol where it is dehydrogenated by a non-specific cytoplasmic NAD-dependent dehydrogenase (synonyms iditol dehydrogenase; polyol dehydrogenase). The xylulose so produced is phosphorylated via a specific xylulokinase to xylulose-5-phosphate, an intermediate of the pentose-phosphate pathway before conversion to glucose, which is only slowly released into the bloodstream or stored as glycogen (Keller & Froesch, 1972).

Mannitol. Various forms of evidence indicate that approximately 25 % of oral mannitol in solution is absorbed (reviewed in Livesey, 1992). Absorbed mannitol is excreted in urine because it is virtually non-metabolisable in the tissues (Nasrallah & Iber, 1969) and the remainder or unabsorbed mannitol is slowly fermented.

Sorbitol. Estimates of absorption from oral solutions range from 25 to 80 % of the ingested dose (Beaugerie *et al*. 1990; Livesey, 1992), with the lower value being

more consistent with the size of this molecule (Livesey, 1992) and the higher value possibly due to the use of invasive methodology and non-recovery. Slow and late $^{14}CO_2$ excretion from labelled sorbitol (compared with glucose) in non-invasive studies in human subjects suggests lower absorption (Tsuji *et al*. 1990) though this could also be due to the temporal storage of [^{14}C]carbon as glycogen. Absorbed sorbitol is practically metabolised fully as only a trace is excreted (Adcock & Grey, 1957). Dehydrogenation in the liver is via the non-specific cytoplasmic NAD-dependent dehydrogenase, as for xylitol, with the production of fructose then glycogen or glucose that may be slowly released into the bloodstream. Unabsorbed sorbitol is extensively fermented to short-chain organic acids and gases (Hyams, 1983), with a considerable yield of butyric acid *in vitro* (Mortensen *et al*. 1988; Clausen *et al*. 1998).

Sorbitol syrup. The biological response to sorbitol syrup is based on the combined individual responses to its constituents, which are mainly sorbitol and mannitol (Table 1, see earlier; p. 166).

Maltitol. This is a disaccharide polyol (moieties of glucose and sorbitol; Table 1) for which hydrolysis is required before absorption. Absorption in human subjects is reported to range from 5 to 80 % (Beaugerie et al. 1990; Life Sciences Research Office, 1999); the wide range is partly due to the use of invasive methods and partly due to the incorrect evaluation of results from non-invasive methods. Account needs to be taken of three non-invasive study approaches in human subjects. First, comparison of the time course of $^{14}CO_2$ production from [U^{14}C]maltitol, [U^{14}C]glucose (fully available) and [U^{14}C]fructo-oligosaccharides (fully unavailable) (see data in Livesey, 1993) indicates by the simplest of computational models a lower limit to absorption of 35 % for maltitol (10 g) in solution. Second, glycaemia and insulinaemia (see pp. 167–168) indicate a lower limit to absorption of 35 to 27 % respectively formaltitol (25–50 g) in solution. Third, based on indirect calorimetry following the ingestion of a high-polymer maltitol syrup containing 50 % maltitol and 50 % polymer and separate study of the polymer fraction (Sinaud et al. 2002) the energy value of maltitol can be estimated. This estimated energy value corresponds to maltitol absorption of approximately 32 % when consumed in three mixed solid meals interspersed by three maltitol drinks (totalling 50 g maltitol in 50 g polymer daily). On the basis of energy values for maltitol proposed by several authorities, absorbability by consensus is 45 % (Dutch Nutrition Council, 1987; Bär, 1990; Bernier & Pascal, 1990; Life Sciences Research Office, 1994, 1999; Brooks, 1995; Australia New Zealand Food Authority, 2001; American Diabetes Association, 2002; Food and Agriculture Organization, unpublished results). The products of hydrolysis by intestinal brush-border disaccharidases are glucose and sorbitol, the metabolism of which has been described earlier (p. 166).

Maltitol syrup(s). These are hydrogenated starch hydrolysates and consist of a mixture of sorbitol, maltitol, and hydrogenated oligo- and polysaccharides (Table 1). The terminology 'regular-, intermediate- and higher-maltitol syrups and high-polymer maltitol syrup' is applied here to conveniently identify four distinctly different products, all of which bear the same general name 'maltitol syrup'. Information on the availability of carbohydrate from hydrogenated oligo- and polysaccharide fractions of regular, intermediate- and high-maltitol syrups is not evidently available. However, based on glycaemic and insulinaemic response data (derived later, see p. 169), it is probably close to 50 %.

A maltitol syrup comprising 50 % maltitol and 50 % hydrogenated polymer has recently been introduced (Sinaud *et al*. 2002), which here is referred to as 'high-polymer maltitol syrup'. The high-polymer fraction is obtained by heating starch at high temperature and low moisture in the presence of an acid catalyst, which yields after separation a product with an average degree of polymerisation of about 17, the introduction of 1–2 and 1–3 glucosidic linkages and so a proportion of branched linkages. Digestibility of the high-polymer maltitol syrup *in vitro* is about 40 % based on hydrolysis with α-amylase and amyloglucosidase and the release of sorbitol and glucose (Sinaud *et al*. 2002). This value is consistent with the glycaemia and insulinaemia described in the present review.

Polyglycitol syrup. Similar to the maltitol syrups this is a hydrogenated starch hydrolysate, though it has more sorbitol (< 20 *v.* < 8 %) and less maltitol (< 50 *v.* ≥ 50 %). The absorption of carbohydrate from polyglycitol syrup is uncertain in extent. However, with a GI and insulinaemic index (II) similar to those for maltitol (see pp. 169–171) it probably has a similar small-intestinal digestibility, at about 40 %.

Isomalt. This is a mixed disaccharide polyol (Table 1). The products of hydrolysis are glucose, sorbitol and mannitol, the metabolism of which is described earlier (p. 166). However, a variety of studies including non-invasive methods in human subjects and methods in animals (Livesey, 1990*a*,*b*, 2000*a*) together with the present studies on glycaemia and insulinaemia suggest 0 to 14 % of isomalt is available as carbohydrate in man. On the basis of the energy values of isomalt suggested by various authorities and experts (Dutch Nutrition Council, 1987; Livesey, 1992; Life Sciences Research Office, 1994; Brooks, 1995) a consensus of approximately 90 % is fermented in the colon, with a stoichiometry *in vivo* and *in vitro* indicating relatively little H_2 gas production (Livesey *et al*. 1993).

Lactitol. Very little of this disaccharide polyol is absorbed, perhaps 2 % as lactitol and its hydrolysis products galactose and sorbitol. This is due to a very low activity of

β-galactosidase in the human intestine (Nilsson & Jägerstad, 1987; Grimble *et al*. 1988). The liver readily uses absorbed galactose and sorbitol in either hepatic glycogen storage or hepatic glucose production. Unabsorbed lactitol is completely fermented with a stoichiometry giving a generous yield of H_2 gas *in vivo* and *in vitro* (Livesey *et al*. 1993) and butyric acid *in vitro* (Clausen *et al*. 1998).

Glycaemia and insulinaemia

Definitions

Glycaemic index. GI is a measure of a specific property of carbohydrate in a food or meal or diet (Jenkins *et al*. 1981; Wolever *et al*. 1991; Food and Agriculture Organization, 1998). It is defined as 'the incremental area under the blood glucose response curve of a 50 g carbohydrate portion of a test food expressed as a percentage of the response to the same amount of carbohydrate from a standard food taken by the same subject' (Food and Agriculture Organization, 1998). In this definition carbohydrate usually means available carbohydrate, though has included for comparative purposes any carbohydrate that might replace available carbohydrate in a foodstuff (Pelletier *et al*. 1994; Bär, 2000; Zumbé *et al*. 2001; Foster-Powell *et al*. 2002; Sydney University's Glycaemic Index Research Service, 2002). The 50 g carbohydrate portion mentioned in the definition is not always practical and smaller portions (down to 25 g) can be used when this is more realistic of the conditions of consumption. The standard food mentioned in the definition is usually glucose in water or white bread. To avoid confusion, it is useful to express the GI relative to glucose (for example, G = 100 GI units), and to state the standard food (being well defined, glucose is preferred) and its GI. Measurements are usually made on eight to ten adults consuming each test food on one occasion and the standard food ideally on three occasions. The calculation of GI has been standardised (Food and Agriculture Organization, 1998) and applied here with the following additional instructions: calculations were performed on group mean plasma glucose responses to carbohydrate ingestion; this minimises a bias caused by discounting below-baseline areas that occur due to random effects. Baseline values were taken at zero time rather than averaged across time before zero time; this minimises a bias due to the fall in basal glucose concentrations with time in the basal state. In studies reporting GI values these calculations were still necessary to ensure a standard and consistent approach was used.

GI values obtained in normal individuals usually apply to those with abnormal carbohydrate metabolism (Wolever *et al*. 1987; Foster-Powell *et al*. 2002); namely patients with type 1 diabetes mellitus (DM) (previously called juvenile or insulin-dependent DM, which results from inadequate insulin secretion) and more commonly type 2 DM patients (previously called adult or late onset diabetes, which is associated with the resistance of tissues to insulin). A provisional WHO classification of diabetes is available (Alberti & Zimmet, 1998), together with useful desktop guides on type 1 and 2 DM (European Diabetes Policy Group, 1999*a*,*b*), and criteria for impaired glucose tolerance (GT) and impaired fasting glycaemia (Unwin *et al*. 2002).

Glycaemic load. GL is formally the product of the carbohydrate content and GI of a food and so is primarily a measure of the quantity and apparent quality of the carbohydrate in the food item and has units of weight (g). Foods with the same GL have practically the same impact on the integrated blood-glucose response, which in diabetes management is the main target.

Insulinaemic index. II is obtained under identical conditions to those for GI, simply replacing the measure of glucose with a measure of insulin. The index was introduced as a result of possible concern that blood-glucose responses might not adequately reflect the responses of the major anabolic hormone insulin, which is central to abnormal carbohydrate metabolism in DM (Holt *et al*. 1997; Wolever, 2000).

Insulin load. IL is calculated in the same way as GL, but replacing glucose measurements with insulin measurements.

Composite foods, meals and diets. The composite GL is the sum of GL from each food or ingredient item. Dividing this sum by the sum weight of the carbohydrate eaten gives the composite GI. Substitution of measures of glycaemia with measures of insulinaemia gives the composite insulin load and composite II.

Statistics. Studies from which GI and II values were calculated were generally of similar size and for simplicity were considered of equal weight when deriving overall means.

Time course of acute glycaemic responses to polyols

Glycaemic responses to sugars and polyols in fasted normal individuals of both sexes were summarised from the literature (Fig. 1). The curves are representative of 25 g doses taken in water or tea without milk or other nutrients (80 to 500 ml). The sugars (glucose and sucrose) result in higher responses 30–60 min after ingestion and lower glucose concentrations after 90 min than any of the polyols (erythritol, xylitol, sorbitol, mannitol, maltitol, isomalt, and lactitol). The responses to all polyols are lower or much lower than for sucrose. Glycaemic and insulinaemic responses (incremental areas) for sucrose and polyols, relative to equivalent intakes of glucose, were calculated for the studies represented in Fig. 1 and other studies. The responses were calculated using a wider range of intakes (10 to 70 g), type 1 and type 2 DM patients in addition to normal subjects, and maltitol syrups and polyglycitol in addition to the other polyols mentioned (Table 4). In the

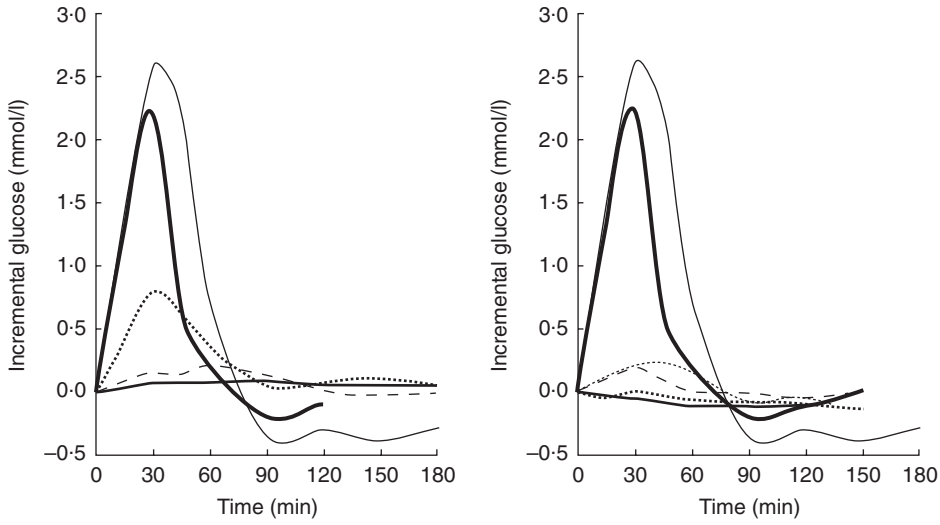

Fig. 1. (a), Glycaemic curves for glucose (—), sucrose (**—**) and polyols maltitol (.....), isomalt (– – –) and lactitol (**—**) in normal individuals; (b) glycaemic curves for glucose (—), sucrose (**—**) and polyols xylitol (·····), sorbitol (– – –), erythritol (·····) and mannitol (**—**) in normal individuals. Data from several publications were pooled to yield curves representative of 25 g doses (20–64 g for erythritol) in water or tea (80 to 500 ml) without other nutrients. In practice, individual studies used various doses, and dose was used as a covariate at each time point to obtain curves representing 25 g intake. Based on data in Table 4 for normal subjects.

majority of publications such calculations had either not been undertaken or had been undertaken incorrectly due to the literature pre-dating knowledge of the current GI calculation method. In type 1 DM patients supported by an artificial pancreas the rate of insulin delivery was in some cases used as a surrogate for insulinaemia.

Glucose and insulin measurements were invariably made on venous plasma or capillary blood. Glucose was the most common reference carbohydrate used. In a small number of cases sucrose was the reference carbohydrate, in which case responses were still expressed relative to glucose having 100 GI units. Statistical presentations (means, standard errors and differences) are omitted from Table 4 due to the possible heterogeneous nature of the data with respect to the level of polyol intake and the condition of subjects' carbohydrate metabolism, which are now examined.

Glycaemic responses in normal, type 1 and type 2 diabetes mellitus subjects

Information on glycaemic responses was available for sorbitol, isomalt and hydrogenated starch hydrolysates (intermediate- and high-maltitol syrups combined) in normal, type 1 and type 2 DM subjects and for maltitol in normal and type 1 DM subjects. Diabetics had HbA_{1c} values of less than 12 % indicating a degree of glucose control, thought less than aimed for nowadays. For each polyol, glycaemic responses expressed relative to glucose in both types of diabetes were similar to those in normal subjects (Fig. 2).

Relationship of glycaemic response to intake of polyols

Information was available for sucrose, maltitol, high-maltitol syrup, isomalt, lactitol and sorbitol to assess the relationship between intake and glycaemic response relative to the most commonly used reference, glucose (Fig. 3). Sorbitol, isomalt and lactitol had very low to little responses at all intakes and there was no association with dose. Responses tended to fall either significantly or numerically with increasing dose for sucrose ($P < 0{\cdot}02$), maltitol ($P = 0{\cdot}06$) and high-maltitol syrup ($P = 0{\cdot}16$). Such possible dose dependence is not limited to soluble carbohydrates as it is also observed for bread *v.* glucose (Jenkins *et al*. 1981; Wolever & Bolognesi, 1996; Lee & Wolever, 1998).

Sucrose is a carbohydrate often replaced by polyols in foodstuffs. When incremental glucose-response areas for polyols were re-expressed relative to a sucrose standard set at 65 GI units at all intakes (Fig. 3 (b)), the relative glycaemic response to all polyols was clearly independent of dose.

Glycaemic and insulinaemic indices of polyols

The achievement of low postprandial glycaemia is an important goal and has greater significance when accompanied by low insulinaemia. All polyols had lower GI and II values than either glucose or sucrose (Table 5).

Among these carbohydrates GI and II were related practically linearly (Fig. 4) with a slope of association of 0·75 (SE 0·05) (dimensionless); this slope is significantly less than might be expected ($P < 0{\cdot}0001$); for glucose the value would by definition fall on a line passing through the origin of slope 1·00.

Variations about mean GI and II values possibly increased with increasing value; homogeneity was achieved by transformation to the square root (Fig. 4 (b)). Observations falling below the line of identity (Fig. 4 (a) and (b)) are consistent with causing demand on the pancreas for insulin that is lower than that due to glucose, and

Table 4. Estimates of the relative glucose response (RGR) and relative insulin response (RIR) to sucrose and polyols (glucose = 100)

Reference (RGR, RIR)*	Intake (g)	RGR	RIR	Subjects	*n*	Composition (%) S	Ma	H	Source
Sucrose									
Glucose (100, 100)†	20	89	33	Normal	9 M				MacDonald *et al.* (1978)
Glucose (100, –)	20	87	na	Normal	12				Samata *et al.* (1985)
Glucose (100, –)	20	89	na	Type 2 DM	8				Samata *et al.* (1985)
Glucose (100, –)	20	79	na	Type 1 DM	6				Samata *et al.* (1985)
Glucose (100, 100)	25	58	58	Normal	4 M + 4 F				Lee & Wolever (1998)
Glucose (100, 100)	25	80	43	Normal	8 M				Pelletier *et al.* (1994)
Glucose (100, 100)†	35	58	23	Normal	9 M				MacDonald *et al.* (1978)
Glucose (100, 100)	50	58	45	Normal	4 M + 4 F				Lee & Wolever (1998)
Glucose (100, 100)†	50	64	43	Normal	9 M				MacDonald *et al.* (1978)
Glucose (100, –)	50	65	na	Normal	–				Brand-Miller *et al.* (1999)
Glucose (100, 100)†	70	75	45	Normal	9 M				MacDonald *et al.* (1978)
Glucose (100, 100)	100	58	67	Normal	4 M + 4 F				Lee & Wolever (1998)
Erythritol									
Glucose (100, 100)	17	21	3	Normal	5 M				Noda *et al.* (1994)
Glucose (100, 100)‡	64	0 (–5)	1	Normal	3 M + 3 F				Bornet *et al.* (1996*a*)
Glucose (100, 100)‡	20	0 (–20)	3	Type 2 DM	3 M + 8 F				Ishikawa *et al.* (1996)
Glucose (100, –)‡	40	3	–	Normal	6				PD Cock, Cerestar (unpublished results)
Xylitol									
Glucose (100, 100)	20	13	4	Normal	5 M + 5 F				Nguyen *et al.* (1993)
Glucose (100, 100)	25	9	31	Normal	8 M				Natah *et al.* (1997)
Glucose (100, 100)	30	14	12	Normal	3 M + 3 F				Salminen *et al.* (1982)
Glucose (100, –)	30	15	na	Normal	5 M + 5 F				Müller-Hess *et al.* (1975)
Glucose (100, 100)	50	7	14	Normal	30				Tong *et al.* (1987)
Glucose (100, 100)	50	18	14	Normal	5 M + 5 F				Müller-Hess *et al.* (1975)
Mannitol									
Glucose (100, 100)	25	0	0	Normal	5				Ellis & Krantz (1941)
Sorbitol									
Glucose (100, 100)	20	13	4	Normal	8 M				Nguyen *et al.* (1993)
Glucose (100, 100)†	20	7	36	Normal	9 M				MacDonald *et al.* (1978)
Sucrose (81, –)	20	14	7	Type 1 DM§	18 M + 6 F				Kaspar & Spengler (1984)
Glucose (100, –)	25	10	na	Normal	7				Ellis & Krantz (1941)
Glucose (100, 100)†	35	3	12	Normal	9 M				MacDonald *et al.* (1978)
Sucrose (68, 40)	40‖	11	19	Type 2 DM	10 M + 8 F				Petzoldt *et al.* (1982*b*)
Glucose (100, –)	50	14	na	Normal	2				Ellis & Krantz (1941)
Glucose (100, 100)†	50	6	15	Normal	9 M				MacDonald *et al.* (1978)
Glucose (100, 100)	50	8	6	Normal	9 (M + F)				Mimura *et al.* (1972)
Glucose (100, –)	50	4	na	Type 2 DM	13				Ellis & Krantz (1943)
Maltitol									
Glucose (100, 100)	20	44	23	Normal	5 M + 5 F	–	98	–	Nguyen *et al.* (1993)
Glucose (100, 100)	25	49	30	Normal	8 M	–	99	–	Pelletier *et al.* (1994)
Glucose (100, –)	50	25	na	Normal	9 (M + F)		98¶		Mimura *et al.* (1972)
Glucose (100, 100)	50	37	21	Type 2 DM	11 (M + F)		98¶		Mimura *et al.* (1972)
Glucose (100, 100)	50	39	29	Normal	12 M	–	99	–	Kamoi (1974)
Glucose (100, –)	50	31	na	Normal	14 M + 5 F	–	99	–	Kamoi (1974)
Glucose (100, –)	50	39	na	'Diabetic'	14 M + 7 F	–	99	–	Kamoi (1974)
Glucose (100, 100)	50	25	27	Type 2 DM	6	–	98	–	Slama (1989)
Glucose (100, 100)	50	29	33	Normal	6	–	98	–	Slama (1989)
Maltitol syrups									
High-maltitol syrup (about 89 % maltitol)									
Glucose (100, 100)‡	10	65	na	Normal	6	–	89	–	Secchi *et al.* (1986)
Glucose (100, 100)‡	25	48	na	Normal	6	–	89	–	Secchi *et al.* (1986)
Glucose (100, 100)	25	37	48	Normal	8 M	5	88	7	Pelletier *et al.* (1994)
Sucrose (71, 34)	30	55	28	Normal	8	2	88	10	Felber *et al.* (1987)
Glucose (100, 100)	35	47	28	Normal	8 M + 8 F	5	89	6	Kearsley *et al.* (1982)
Glucose (100, 100)	50	33	36	Normal	6	–	89	–	Secchi *et al.* (1986)
Glucose (100, 100)‡	50	50	na	Normal	6	–	89	–	Secchi *et al.* (1986)
Intermediate-maltitol syrup (about 70 % maltitol)									
Glucose (100, 100)	25	54	29	Normal	8 M	2	72	26	Pelletier *et al.* (1994)
Glucose (100, 100)	50	52	27	Normal	3 M + 3 F	7	69	33	Wheeler *et al.* (1990)
Glucose (100, 100)	50	52	na	Type 1 DM	3 M + 3 F	8	69	33	Wheeler *et al.* (1990)
Glucose (100, 100)	50	56	66	Type 2 DM	3 M + 3 F	9	69	33	Wheeler *et al.* (1990)

Table 4. *Continued*

Reference (RGR, RIR)*	Intake (g)	RGR	RIR	Subjects	*n*	Composition (%) S Ma H	Source
Regular-maltitol syrup (about 53 % maltitol)							
Glucose (100, 100)	20	43	19	Type 2 DM	5 M + 5 F	– >50 –	Nguyen *et al.* (1993)
Glucose (100, 100)	25	53	47	Normal	8 M	3 53 40	Pelletier *et al.* (1994)
Glucose (100, 100)	35	67	36	Normal	8 M + 8 F	7 53 40	Kearsley *et al.* (1982)
Glucose (100, 100)	66	54	52	Normal	6	5 55 40	Slama (1989)
Glucose (100, 100)	66	41	64	Type 2 DM	6	5 55 40	Slama (1989)
High-polymer maltitol syrup							
Glucose (100, 100)	50	47	23	Normal	6	– 50 50	Rizkalla *et al.* (2002)
Glucose (100, 100)	50	25	39	Type 2 DM	6	– 50 50	Rizkalla *et al.* (2002)
Polyglycitol syrup							
Glucose (100, 100)	25	32	16	Normal	3 M + 3 F	14 8 78	Wheeler *et al.* (1990)
Glucose (100, –)	30	45	na	Type 1 DM	3 M + 3 F	14 8 78	Wheeler *et al.* (1990)
Glucose (100, 100)	35	38	30	Type 2 DM	3 M + 3 F	14 8 78	Wheeler *et al.* (1990)
Isomalt							
Glucose (100, 100)	20	11	7	Type 1 DM§	18 M + 6 F		Kaspar & Spengler (1984)
Glucose (100, 100)	25	2	8	Normal	10		Sydney University Glycaemic Index Research Service (2002)
Sucrose (71, 34)	30	11	4	Normal	10 M		Thiébaud *et al.* (1984)
Glucose (100, 100)	50	7	5	Type 2 DM	8 F + 16 M		Petzoldt *et al.* (1982*a*)
Sucrose (65, 45)	50	12	3	Type 2 DM	24		Drost *et al.* (1980)
Sucrose (65, 45)	50	6	3	Type 2 DM	3 M + 9 F		Bachmann *et al.* (1984)
Glucose (100, 100)	50	12	15	Type 2 DM	6		Slama (1989)
Glucose (100, 100)	50	8	5	Normal	6		Slama (1989)
Sucrose (62, 47)	70	11	4	Normal	6		Keup & Püttner (1974)
Lactitol							
Glucose (100, 100)	25	3	6	Normal	8 M		Natah *et al.* (1997)
Glucose (100, 100)	25	7	1	Normal	7		Doorenbos (1977)
Sucrose (65, –)	50	7	na	Normal	8		Zaal & Ottenhof (1977)**

S, sorbitol; Ma, maltitol; H, hydrogenated saccharides with degree of polymerisation > 2; M, male; na, information not available; DM, diabetes mellitus; F, female.
* Values of RGR and RIR at the intakes of reference substrate used are shown in parentheses. These data are used to adjust to a glucose reference of 100 when the reference substrate in the study was other than glucose.
† Unpaired reference: data were adjusted for glucose responsiveness according to the treatment group's fasting glucose concentrations.
‡ Unpaired reference: data taken from a separate publication with adjustment for fasting glucose concentration. RGR data in parentheses are acutal, outside parentheses are conventional.
§ Subjects with artificial pancreas.
|| Intake was 4 x 10 g doses at hourly intervals over 240 min.
¶ 98 % assumed based on production by hydrogenation of maltose.
** Partially reported by van Velthuijsen (1990).

more than simply due to the GI of polyols and sucrose being lower than for glucose.

Composite glycaemic index, glycaemic load, insulinaemic index and insulin load: potential interactions

Interactions between polyols and sugar, and between polyols and foods. Seven studies involving polyols provided the possibility to assess whether the sum of the GL for meal components fed separately from one another would equal the GL of the entire meal (Table 6).

A meal composed of glucose and sorbitol (monosaccharide mixture) yielded a GL less than predicted from the sum of the loads for glucose and sorbitol separately (Table 6; cases 1 and 2). Incomplete hydrolysis cannot explain this result; possibly sorbitol slows stomach emptying or hurries the glucose to a site where absorption distally is less rapid (Livesey *et al*. 1998) or significantly dilutes luminal glucose concentration through its osmotic effect. A similar observation is made for a meal of a disaccharide mixture, sucrose and lactitol (Table 6; case 3). Likewise a similar result is observed for sorbitol taken in comparatively complex meals; a breakfast comprising mainly bread and butter (Table 6; cases 4 and 5) and a protein-and-carbohydrate-based breakfast, mainly scrambled eggs and farina cereal (1088 kJ (260 kcal); Akgün & Ertel, 1980) to which was added either sucrose or fructose or sorbitol (35 g) (Table 6; case 6). The last study was repeated in type 2 DM patients with similar results (Table 6; case 7).

Similar results were obtained when GI and load were replaced by II and load (Table 7), suggesting that the interaction affecting glycaemia was not the result of interaction to elevated insulin secretion.

The general case is evidently that a mixture involving a polyol yields a value less than the sum of its individual

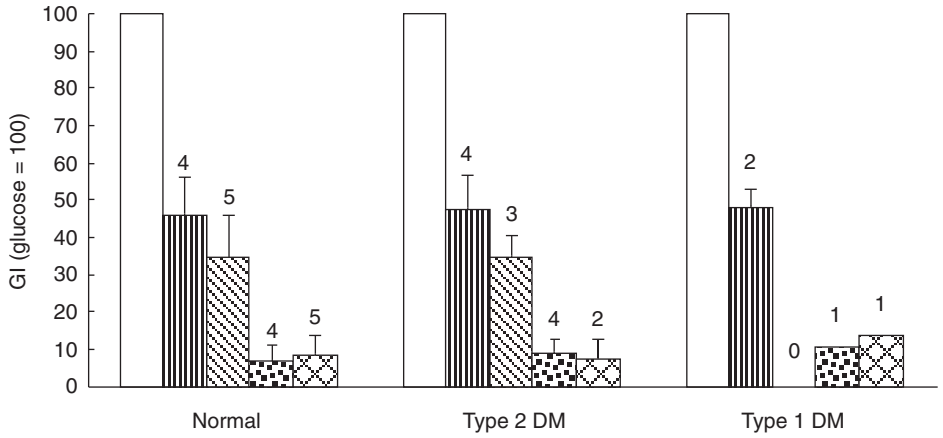

Fig. 2. The glycaemic response for four polyols relative to glucose (□) in normal, type 2 diabetes mellitus (DM) and type 1 DM subjects. Values are the means of the responses for individual studies shown in Table 4, which cites the sources of information for the calculations made. The numbers of studies represented are shown above each column and the vertical bars represent either standard deviation ($n \geq 3$ studies) or range (n 2) or are absent (n 1). Hydrogenated starch hydrolysate (▥) is equally weighted information combined from polyglycitol and regular maltitol syrup. (▨), Maltitol; (▩), isomalt; (▧), sorbitol.

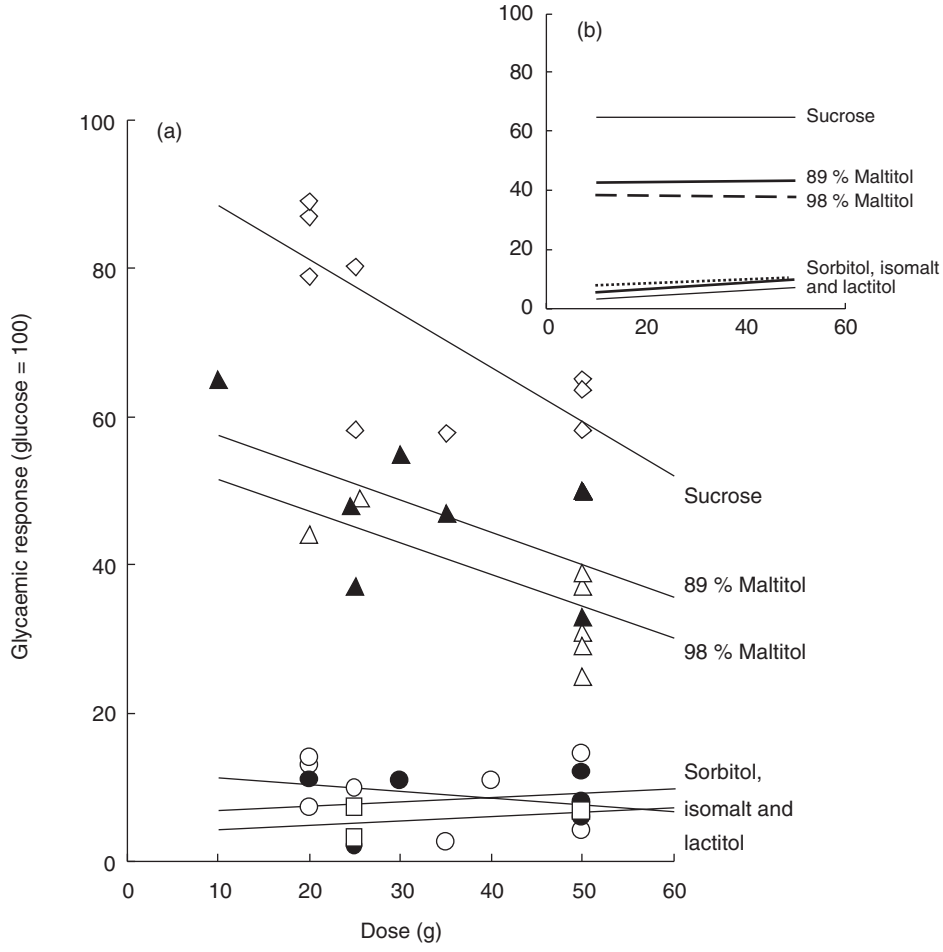

Fig. 3. Glycaemic response area for sucrose and polyols relative to glucose (glycaemic index (GI) = 100) (a) or sucrose (GI = 65) (b). Carbohydrates were taken in water or tea without other nutrients (80 to 500 ml). Data are inclusive of normal, type 2 diabetes mellitus (DM) and type 1 DM subjects and are from Table 4, which cites the sources of information used in the calculations. (◇), Sucrose; (▲), high-maltitol syrup; (△), maltitol; (○), sorbitol; (●), isomalt; (□), lactitol. Regression curves for glucose = 100 at each intake were:
Sucrose relative glucose response (RGR) = 95 (SE 8) + intake × (–0·70 (SE 0·24)), P = 0·02; High-maltitol syrup RGR = 62 (SE 9) + intake × (–0·43 (SE 0·27)), P = 0·16; Maltitol RGR = 56 (SE 10) + intake × (–0·42 (SE 0·23)), P = 0·10; Sorbitol RGR = 12 (SE 4) + intake × (–0·09 (SE 0·10)), P = 0·39; Isomalt RGR = 6 (SE 4) + intake × (0·06 (SE 0·06)), P = 0·53; Lactitol RGR = 3 (SE 5) + intake × (0·07 (SE 0·14)), P = 0·72.

Table 5. Glycaemic and insulinaemic indices of polyols*

Polyol	Glycaemic index (glucose = 100)			Insulinaemic index (glucose = 100)		
	Mean	SD	*n*†	Mean	SD	*n*†
Erythritol	0	17	4	2	1	3
Xylitol	13	4	6	11	5	4
Sorbitol	9	4	10	11	6	6
Mannitol	0	–	1	0	–	1
Isomalt	9	3	9	6	4	9
Lactitol	6	2	3	4	3‡	2
Maltitol	35	9	9	27	5	6
Maltitol syrups						
High-maltitol syrup	48	11	7	35	10	4
Intermediate-maltitol syrup	53	2	4	41	22	3
Regular-maltitol syrup	52	10	5	44	17	5
High-polymer maltitol syrup	36	11‡	2	31	8‡	2
Polyglycitol	39	7	3	23	7‡	2

* Data are the means of study values for relative glucose responses and relative insulin responses in Table 4, ignoring intake as a cause of variance when glucose is the reference carbohydrate. Observations obtained with > 50 g intake were excluded from the analysis. For the insulinaemic index, one observation on xylitol and one observation on sorbitol were excluded as outliers from the analysis due to their being > 6 standardised residuals from the results shown.

† No. of studies.

‡ Plus and minus half range of the two values.

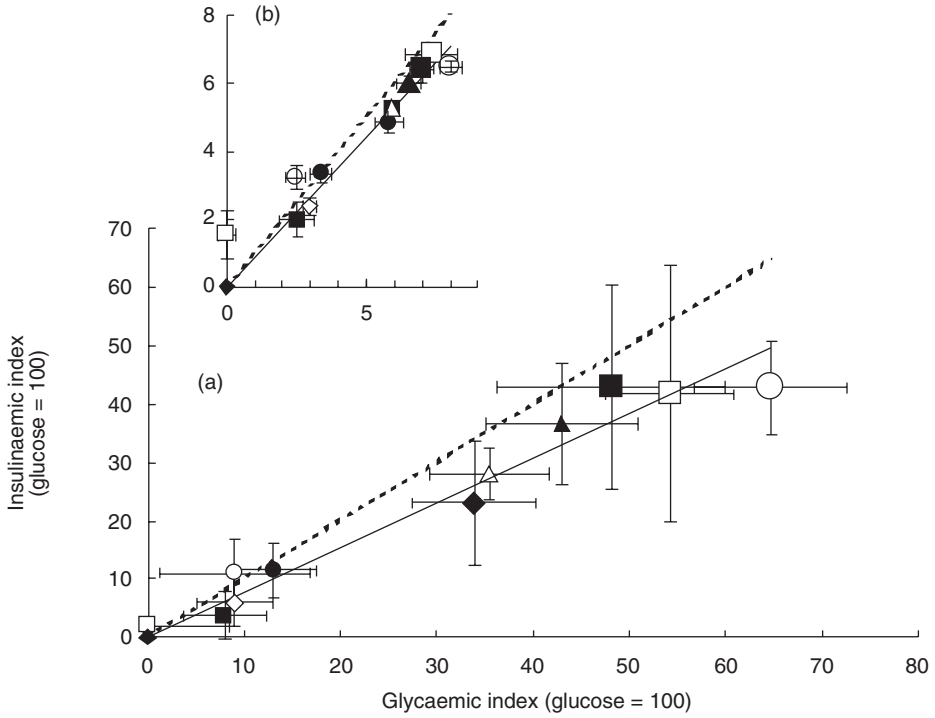

Fig. 4. Relationship (—; Slope = 0·75 (SE 0·05)) of the insulinaemic index to the glycaemic index for polyols and sucrose for untransformed data (a) and square root transformations (b). Data are from Table 5 and are means, with standard errors represented by vertical and horizontal bars (among studies) for sucrose (○), regular-maltitol syrup (□), intermediate-maltitol syrup (■), high-maltitol syrup (▲), polyglycitol (△), maltitol (◆), sorbitol (○), xylitol (●), isomalt (◇), lactitol (■), erythritol (□), and mannitol (◆). (- - -), Unity.

parts. A single instance departed from the general case and occurred in type 2 DM patients (Table 7; case 7). Here the interaction is as expected for the GL, but not for the insulinaemic load, and this could be due to a marked impairment of insulin secretion in the patients studied.

In conclusion, the GI and II and loads of the polyols apply approximately in the context of simple meals of sugars (glucose, sucrose), starches (bread) and protein (scrambled egg and farina cereal) and without overestimation (Tables 6 and 7). A similar conclusion was drawn for

Table 6. Interaction between polyols and other dietary components affecting glycaemic index*

		Glycaemic	
	Intake (g)	Index	Loadl† (g)
Case 1: normal subjects, *n* 16, 8 M + 8 F (Kearsley *et al.* 1982)			
Sorbitol	17·5	10	1·75
Glucose	17·5	100	17·5
Predicted sum for mixture			19·25
Observed for mixture			15·05
Observed/predicted value			0·78
Case 2: normal subjects, *n* 16, 8 M + 8 F (Kearsley *et al.* 1982)			
Sorbitol	15·1	10	1·51
Glucose	20·0	100	19·95
Predicted sum for mixture			21·46
Observed for mixture			17·50
Observed/predicted value			0·82
Case 3: normal subjects, *n* 8 (Zaal & Ottenhof,1977)			
Lactitol	50	6	3
Sucrose	50	65	32·5
Predicted sum for mixture			35·5
Observed for mixture			25·8
Observed/predicted value			0·73
Case 4: type 2 DM, *n* 12 (Drost *et al.* 1985)			
Bread (and butter)	36	70	25·2
Sorbitol	22	10	2·2
Predicted sum for mixture			27·4
Observed for mixture			16·7
Observed/predicted value			0·61
Case 5: type 1 DM‡, *n* 9, 3 M + 6 F (Vaaler *et al.* 1987)			
Bread (and butter)	75	70	52·5
Sorbitol	21	10	2·1
Predicted sum for mixture			54·6
Observed for mixture			49·8
Observed/predicted value			0·91
Cases 6 and 7: normal, *n* 10; type 2 DM, *n* 6 respectively (Akgün & Ertel, 1980)			
Protein and carb meal + sucrose		§	56·1 37·0
Protein and carb meal + fructose		§	41·4 22·3
'Protein and carb meal' predicted		§	33·4 14·3
Sorbitol	35	10	3·5 3·5
Predicted sum for mixture			36·9 17·8
Observed for mixture			11·6 15·4
Observed/predicted value			0·31 0·87

M, male; F, female; DM, diabetes mellitus; carb, carbohydrate; GI, glycaemic index.
* GI values are from Table 5 or calculated references cited.
† Glycaemic load = intake × GI/100.
‡ Supported with continuous subcutaneous insulin infusion.
§ Glycaemic loads calculated assuming the difference in glycaemic response between the sucrose and fructose meal was equal to the difference in glycaemic loads from sucrose (35 g × GI 65/100) and fructose (35 g × GI 23/100).

individual foods in the context of foods and more complex mixed meals (Collier *et al*. 1986; Wolever & Jenkins, 1986; Bornet *et al*. 1987). In both circumstances the composite GI (and GL) and II (and IL) were slightly less than predicted from the GI and II of individual components or foods. Importantly, the present results indicate that the potential benefits of low GI and II would not be diminished due to the co-ingestion of very low-GI polyols with protein and available carbohydrate in a meal context.

Interaction between polyols and fat. Chocolate is a source of both carbohydrate and fat. The glycaemic response to sucrose (GI 65 (SD 9)) is lower when in chocolate (GI 30 (SD 9)) (values recalculated from Pelletier *et al*. 1994). Similarly, the glycaemic response to maltitol (GI 35 (SD 7)) may be lower in chocolate (GI 29 (SD 7)) (Pelletier *et al*. 1994). These responses may be attributed to slower stomach emptying, but also to a higher insulin response in the presence of fat. Indeed, interactions between carbohydrate and fat are known to elevate insulinaemia and reduce glycaemia (Collier *et al*. 1988; Morgan *et al*. 1988). Thus, the II of sucrose (43 (SD 14)) is higher when in chocolate (76 (SD 24)); likewise the II of maltitol (27 (SD 10)) *v.* maltitol in chocolate (82 (SD 25)) (Pelletier *et al*. 1994). With other polyols (isomalt, erythritol) no such interactions were evident when comparing the present results for pure polyols (Table 5) with those from elsewhere for polyols eaten with fat, in chocolate (Gee *et al*. 1991; Bornet *et al*. 1996*b*).

Table 7. Interaction between polyols and other components affecting insulinaemic index*

	Intake (g)	Insulinaemic Index	Insulinaemic Load† (g equivalent)	
Case 1: normal subjects, *n* 16, 8 M + 8 F				
Sorbitol	17·5	11		1·9
Glucose	17·5	100		17·5
Predicted sum for mixture				19·4
Observed for mixture				13·7
Observed/predicted value				0·71
Case 2: normal subjects, *n* 16, 8 M + 8 F				
Sorbitol	15·1	11		1·7
Glucose	20·0	100		20·0
Predicted sum for mixture				21·6
Observed for mixture				13·1
Observed/predicted value				0·61
Case 3: no insulin data				
Case 4: type 2 DM, *n* 12				
Bread (and butter)	36	90		32·4
Sorbitol	22	11		2·4
Predicted sum for mixture				34·8
Observed for mixture				29·7
Observed/predicted value				0·85
Case 5: no insulin data				
Cases 6 and 7: normal, *n* 10; type 2 DM, *n* 6 respectively				
Protein and carb meal + sucrose		‡	33·4	64·8
Protein and carb meal + fructose		‡	23·6	55·0
'Protein and carb meal' predicted		‡	18·4	49·8
Sorbitol	35	11	3·9	3·9
Predicted sum for mixture			22·2	53·6
Observed for mixture			10·0	57·5
Observed/predicted value			0·45	1·07

M, male; F, female; DM, diabetes mellitus; carb, carbohydrate.
* Insulinaemic index values are from Table 5 or calculated from information in the references cited in Table 6. Cases 1–7 correspond to the glycaemic data in Table 6.
† Insulin load = intake × index/100.
‡ Insulinaemic loads calculated assuming the difference in glycaemic response between the sucrose and fructose meal was equal to the difference in insulinaemic loads from sucrose (intake of 35g × insulinaemic index of 43 divided by 100) and fructose (intake of 35g × insulinaemic index of 15 divided by 100).

Importantly, the potential benefit of a low glycaemic response *per se* to polyols is not lost when co-ingested with fat. The data would suggest, however, that to achieve low insulin responses in products that can be made only with appreciable amounts of fats then carbohydrate of particularly low glycaemic response would be needed. In view of a current understanding that high insulinogenic foods and diets may be adverse for health reasons it may be just as important (or possibly more important) to reduce the GI of carbohydrate in high-fat foods as it is to lower the amount of fat in the foods.

Polyol-based snack foods

Healthy individuals and individuals with disorders of carbohydrate metabolism alike can desire the sweet taste of foods requiring bulk sweeteners; sugars and polyols (Mehnert, 1971). A number of snack foods (which may also be eaten at mealtimes), sugars and polyols are listed in Table 8, ranked by II. The carbohydrate content of a reasoned portion, as used in these studies, is also shown and is about 25 g, much of which might be replaceable with polyols in manufactured goods.

Polyols rank very low on the II scale; however, this is not the case for all polyol products, thus (as discussed earlier; pp. 174–175) maltitol-based chocolate has an II comparable with sucrose-based chocolate, and much above the II for isomalt- and erythritol-based chocolate products. The latter two polyol products have II and GI values less than some fruits (oranges, apples, banana, grapes) and yoghurt. Both fruits and polyol products have II values that are less than for many other products. Some polyols may therefore be used to generate snack foods lower in II and GI than regular snack foods.

The lowering of insulinaemia between meals is well demonstrated for a polyol-based product by Bornet *et al*. (1996*b*). They fed sucrose- and erythritol-based chocolate between breakfast and lunch to type 2 DM patients, showing considerable savings on the demand for insulin (Fig. 5). Such responses are not limited to snacks since they are also observed after mixed meals as noted later (pp. 176–178). Thus also Hassinger *et al*. (1981) established that in diabetics requiring insulin, 30 g xylitol behaves as a low-

Table 8. Snack meals, foods, sugars and polyols ranked by insulinaemic index (II)

Meal item	Carbohydrate intake (g)	II	GI	II–GI	Reference
1 Cereal and milk	25	127	26	101	*
2 Chocolate confection	31	102	58	44	†
3 Glucose	–	100	100	0	
4 White bread	–	92	74	18	‡
5 Cheese, bread and milk	25	89	8	81	*
6 Peanut butter, bread and milk	25	88	14	73	*
7 Chocolate milk (drink)	25	81	24	57	*
8 Ice cream	26	79	52	27	†
9 Milk chocolate (bar)	25	79	25	54	*
10 Milk chocolate (bar)	26	78	23	56	*
11 Maltitol chocolate	25	82	30	52	§
12 Sucrose-based chocolate	25	76	30	46	§
13 Banana	32	68	58	10	†
14 Grapes	15	68	54	14	†
15 Yoghurt	25	63	35	28	*
16 Fried chipped potato	36·5	51	38	13	*
17 Peanut butter cup	25	51	10	41	*
18 Oranges	50·6	50	30	20	†
19 Potato chips (crisps)	25	49	23	26	*
20 Apples	18	49	38	11	†
21 Popcorn	27·4	45	45	0	†
22 Maltitol syrup (regular)	–	44	52	–8	‖
23 Sucrose	–	43	65	–22	‖
24 Maltitol syrup (high-polymer)	–	31	36	–5	‖
25 Maltitol	–	27	35	–8	‖
26 Polyglycitol	–	23	39	–16	‖
27 Peanuts	5·4	17	9	8	†
28 Isomalt chocolate	31	16	13	3	§
29 Fructose	–	15	23	–8	‖
30 Sorbitol	–	11	9	2	‖
31 Xylitol	–	11	13	–2	‖
32 Isomalt	–	6	9	–3	‖
33 Lactitol	–	4	6	–2	‖
34 Erythritol	–	2	0	2	‖
35 Erythritol chocolate	37	2	–	–	§
36 Mannitol	–	0	0	0	‖

GI, glycaemic index; II, insulinaemic index.
* Computed from Shively *et al.* (1986).
† Computed from Holt *et al.* (1997).
‡ Computed from Jenkins *et al.* (1981), Wolever & Bolognesi (1996*a*), and Lee & Wolever (1998).
§ Computed from Pelletier *et al.* (1994), Gee *et al.* (1991), and Bornet *et al.* (1996*a*).
‖ See Table 5.

glycaemic carbohydrate in the context of a high-protein mixed meal, reducing plasma glucose and insulin requirements by 50 % compared with sucrose.

Glycaemic control in groups of normal, type 1 and type 2 diabetes mellitus subjects

Markers of glycaemic control include fasting plasma glucose (FPG), glucose tolerance (GT) or 2 h post GT during a 75 g oral GT test, HbA_{1c} (glycosylated or glycated) concentrations and appearance of urinary glucose, all of which fall with improvement in glycaemic control (Alberti & Zimmet, 1998; Bastyr *et al*. 2000; Wang *et al*. 2002). The glucose response after a mixed meal (or meal GT) also provides an analogous measure to GT or 2 h post GT during a 75 g oral GT test. It is a relevant measure in longitudinal nutritional studies, though HbA_{1c} (and fructosamine as another marker of protein glycation) is probably the most relevant overall marker of glycaemic control and is now commonly used for this purpose. It is well established that both HbA_{1c} and fructosamine concentrations are reduced in diabetics by the consumption of low-glycaemic-carbohydrate diets (Jenkins *et al*. 2002), possibly more so when taken at each meal of the day.

When taken orally with meals at a readily tolerated dose, polyols may help to improve long-term glycaemic control in type 2 DM patients, as expected for low-glycaemic carbohydrates. Thus polyols have provided an example of how a low-glycaemic carbohydrate can benefit type 2 DM patients. A 12-week randomised controlled study of the impact of 6 g isomalt per meal (24 g daily) was undertaken on twenty-four subjects (twelve control and twelve parallel receiving isomalt). Measurements were made (Pometta *et al*. 1985) of HbA_{1c} (glycosylated) and FPG. In addition, the change in mealtime glycaemia was calculated by taking pre-treatment FPG as the baseline (change in this result then reflects the overall improvement due to the sum of chronic changes in FPG, meal GT and GI due to carbohydrate replacement).

The following data were subsequently ascertained by the present author's analysis. For the control group (no drugs, regular diet treatment alone) the underlying trend was for

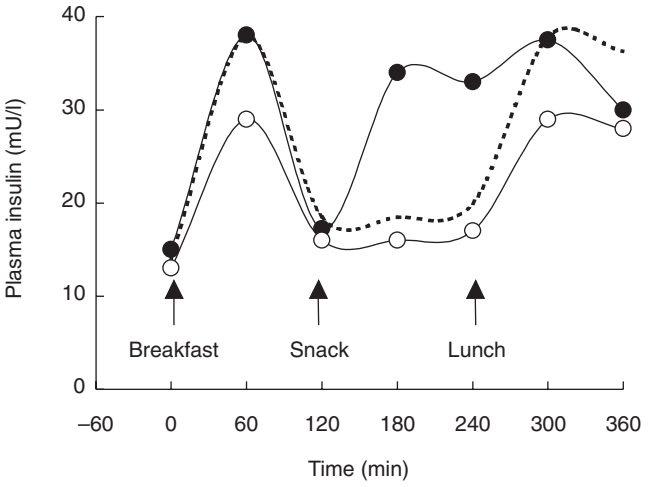

Fig. 5. Insulin demand between meals is reduced using a polyol-based snack food (Bornet *et al.* 1996b). (●), Sucrose-based chocolate snack between meals; (○), erythritol-based chocolate snack between meals. (---), Erythritol treatment group after adjustment upwards to account for differences in treatment-group mean responses to the breakfast (1·48 × area above the basal insulin concentration after breakfast).

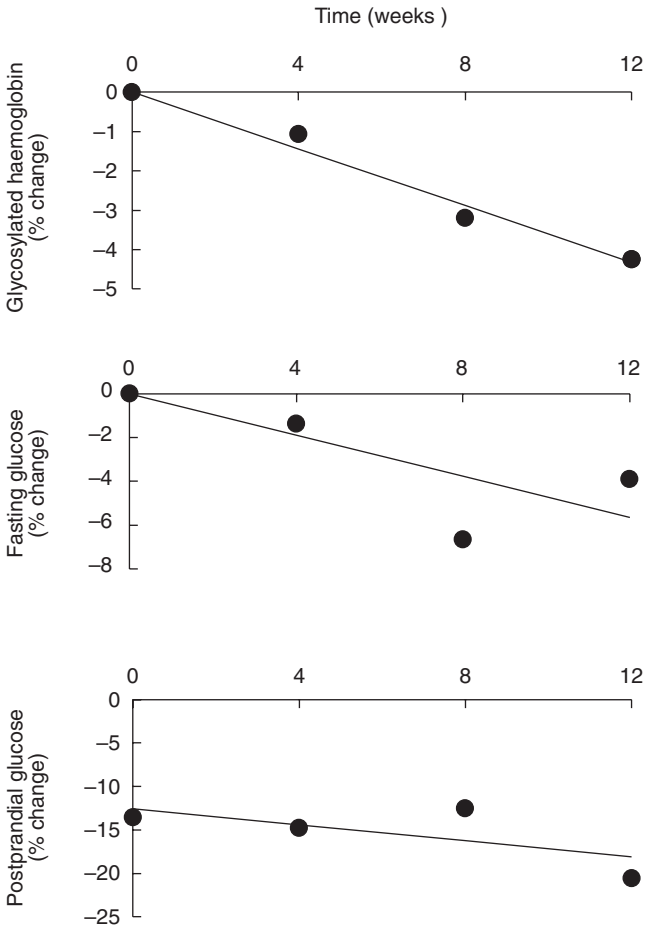

Fig. 6. Improvement in glycaemic control with 6 g isomalt per meal in type 2 diabetes mellitus (DM) patients. Isomalt was fed to twenty-four subjects, twelve controls and twelve type 2 DM patients, at a rate of 6 g per meal (24 g daily). Percentage change is 100 × treatment means difference/treatment means average. Means differences discounted the minor difference between treatment groups immediately after randomisation. The regression lines were:
% Glycosylated haemoglobin = −0·4 (SE 0·02) % per week, P = 0·0004;
Fasting glucose = −0·5 (SE 0·1) % per week, P = 0·04;
Postprandial glucose = (−12·5 (SE 2·7) %)* + (−0·5 (SE 0·4) % per week)†.
* P = 0·04, † P = 0·3. Data for these calculations were from the study of Pometta *et al.* (1985), which reported the data as figures; tabulated means data were kindly supplied by Palatinit GmbH (Mannheim, Germany).

glycaemic control to become progressively worse, though only slightly at an average rate of rise of HbA_{1c} of 0·022 (SE 0·006) % of the basal value per week (P = 0·035). This compares favourably with a worsening of twice this rate at approximately 0·05 % of the basal value per week calculated for conventionally controlled type 2 DM patients in other studies (UK Prospective Diabetes Study Group, 1998; Wallace & Matthews, 2000). The isomalt treatment group by contrast maintained or improved HbA_{1c} concentrations. The differences in the mean of treatment outcomes in the present study were expressed as a percentage of the average of means (Fig. 6), an appropriate statistical method for results comparisons (Altman, 1991). Mealtime glycaemia was immediately lower due to treatment with isomalt, by 12·5 (SE 2·7) %, a difference that tended to widen with time to 20 % lower after 3 months (Fig. 6) due to the combined improvement in FPG and meal GT. Relative to the control, the FPG fell at a significant rate of 0·5 (SE 0·1) % per week, while the corresponding fall for HbA_{1c} was at a significant rate of 0·4 (SE 0·02) % per week. The relative falls in FPG and HbA_{1c} were progressive with time and appeared not to have reached completion. These data contribute to the weight of data (Jenkins *et al*. 2002) showing that low-glycaemic carbohydrate ingestion by type 2 DM patients can improve blood glucose control.

Other long-term studies on the effects of polyols in normal individuals and diabetic patients have been undertaken. Many predate current concepts in glycaemic control and so require fresh interpretation. The objective of the studies of early design was usually to establish whether or not the polyols had adverse influences on metabolism, such as causing FPG, cholesterolaemia or triacylglycerolaemia to increase. For example: no such adverse effects were found in healthy individuals, a mixed group of mainly older schoolchildren (aged > 13 years) with some adults, when exchanging 50 g xylitol for sucrose for 2 years (Huttunen *et al*. 1975). The reduced GL due to this exchange is estimated to be 30 g daily, which is substantial. Reduced FPG was not observed, which suggests that the difference in GL is not of great importance in children or possibly young adults with a healthy metabolism. In another study, Abraham *et al*. (1981) investigated the exchange of 26 g sucrose for 30 g maltitol syrup for 4 weeks in type 2 DM patients. No adverse effects were observed and there was also no improvement in glycaemic control as indicated by

either FPG or fasting insulin, which can reflect the degree of insulin sensitivity and/or ß-cell function (Matthews *et al*. 1985). This is not surprising given the difference in GL between the treatments, estimated at present at just 2 g daily (due to sucrose, GI = 65 and 26 g intake daily *v.* maltitol syrup, GI = 50 at 30 g intake daily). Other studies have examined mainly type 1 DM children. The treatments were usually polyol *v.* 'no polyol' and the outcomes were usually no adverse effects on FPG and urinary glucose, for example with sorbitol (Steinke *et al*. 1961). Assuming the study followed the controlled plan, the supplementary sorbitol treatment group would have had an extra GL of 5 g daily; thus the study provided no information about the relationship of GI or GL to the degree of control of carbohydrate metabolism. Thannhauser & Meyer (1929) and Mehnert *et al*. (1960) undertook similar studies (of early design) in type 2 DM patients. Again no adverse effects of sorbitol (40 g) were observed, but again the experimental designs did not allow an assessment of the relationship between GL and the control of glucose metabolism. Another study was undertaken with parenteral xylitol (30 g daily for 1 week) because of expectations of reduced requirements for insulin secretion. Such treatment with xylitol lowered the FPG in some individuals of a mixed population of type 1 and 2 DM patients (Yamagata *et al*. 1965, 1969); amongst the type 2 DM patients the present author notes the xylitol to have consistently reduced urinary excretion of glucose, and this almost quantitatively in accordance with the degree of glucosuria observed before treatment.

Scope for replacement of sugars, maltodextrins and glucose syrups

Even quite small differences in GL due to carbohydrate exchange appear to be important. Thus in well-controlled type 2 DM patients a residual deterioration in plasma HbA_{1c} occurs at an average rate of 0·2 (SEM 0·04) units HbA1c % per year (0·1, 0·2 and 0·3 % per year in Pometta *et al*. 1985; Orchard *et al*. 1990; Wallace & Matthews, 2000 respectively). Intervention studies with low-GI diets show the reversal of deterioration during the period of study. Assuming linear responses, the minimum change in GL through change in carbohydrate quality needed to reverse the average deterioration is just 12 (SEM 2) g/d. Estimates for individual studies are 11, 8, 13, 19, 9, 14 and 12 g/d (for Jenkins *et al*. 1988; Brand *et al*. 1991; Wolever *et al*. 1992*a*,*b* (two treatments); Frost *et al*. 1994; Järvi *et al*. 1999; Giacco *et al*. 2000 respectively). Such reversal is seen with the polyol isomalt consumed at 24 g daily (Fig. 6). A similar conclusion arises from the examination of the upper quintiles of GI and advent of DM in men (Salmerón *et al*. 1997*a*) and in women (Salmerón *et al*. 1997*b*; Meyer *et al*. 2000), and CHD in women (Liu *et al*. 2000*b*). Thus a change in GL due to carbohydrate quality (not quantity) of 10 g glucose/d corresponds to a change in disease advent of 6, 27, 10 and 33 % respectively, with a mean of 19 (SEM 7) % ($P < 0·05$). Such a change in GL by exchanging carbohydrates could readily be achieved by replacing some sugars, maltodextrins and glucose syrups with tolerable amounts of polyols.

The consumption of sucrose in one population of US women ranged from the lowest quintile median of 26 g/d to the highest of 57 g/d, with a similar range for glucose and fructose combined (Meyer *et al*. 2000). This corroborates similar findings from elsewhere with men consuming more by weight than women in accordance with higher energy intakes (Glinsmann *et al*. 1986; Henderson *et al*. 2003). In terms of macronutrient exchange or replacement, it is more relevant to consider intakes per meal (rather than per d) because it is the meal that initiates an impulse to which metabolism responds (Livesey, 2000*b*). For an average three meals per d these sucrose consumption data correspond to an average meal sucrose intake of 8 to 19 g/meal; comparative values for glucose are from 4 to 10 g/meal (Meyer *et al*. 2000). Such quantities as polyol are tolerable and many individuals can tolerate more (Livesey, 2001; Marteau & Flourié, 2001). There is, therefore, a realistic potential for sugar replacers to exchange with sugars making a useful contribution towards a smaller glycaemic response to diet as a whole among those who would choose this approach.

The total replacement of dietary sugars nevertheless would be neither realistic nor expected, and in practice the potential benefit would probably be limited to reducing the upper range of sugar intakes. The range between the lower and upper quintiles in the study of Meyer *et al*. (2000) was just 10 g/meal for sucrose and 7 g/meal for glucose, a large part of which could potentially be replaced by polyols whenever desirable.

Food manufacturers will, however, consider foods not meals as products of their manufacture; likewise consumers buy food items, for which there is scope for sugar replacement to achieve reduced glycaemia. About 25 g per serving in foods is practical; however, usage of lower amounts across a broader range of food products may be more satisfactory. Unfortunately, this possibility is hampered at present by history; regulatory provisions in Europe currently limit the scope of use of sugar replacers (categorised as sweeteners and additives) but not other carbohydrates, which are considered as ingredients (Barlow, 2001; Howlett, 2001). This situation tends to limit the use of sugar replacers to confections and baked goods, and to elevate their content in such foods. A regulation permitting the broader use of polyols, as for low-digestible sugars of similar tolerance, would deserve consideration.

Assignment of polyols and foods to glycaemic index bands

Foods have GI values that span a continuous broad normal distribution, which can be divided into narrower bands (for example, very low, low, intermediate, high; Table 9). Banding can make it easier in practice for users to select appropriate diets, as noted by Black & Rayner, for the Coronary Prevention Group (1992), or appropriately low-glycaemic diets for diabetes control (Brand *et al*. 1991). Brand-Miller *et al*. (1999) suggest that GI > 70 would indicate a high-GI food while GI < 55 indicates a low-GI food, with intermediate GI being 55 to 70. These bands have been demonstrated in practice to be helpful in the selection of a low-GI diet (Brand *et al*. 1991), which without setting a precise value would just fall into the low-GI band. To

make a more stringent target for formulating low-GI foods, Bär (2000) recently suggested GI < 40, which here is called 'very-low GI' to avoid confusion with the low-GI band of Brand *et al*. (1991), Brand-Miller *et al*. (1999) and which coincidently occurs approximately at the mean less one standard deviation for the normal distribution of food GI values (G. Livesey, unpublished results). Some foods have such low GI values that the carbohydrate assessed inevitably includes unavailable or so-called 'non-glycaemic' carbohydrate (Jenkins *et al*. 1987; Food and Agriculture Organization, 1998), including resistant starch (Björck *et al*. 2000) and some polyols. Certain polyols (sorbitol, xylitol) additionally cause low increments in plasma glucose due to slow absorption and metabolism in the liver and, although glucogenic, they give only low glycaemic responses.

The use of nutrient banding to communicate nutritional value is still in its infancy (Black & Rayner, for the Coronary Prevention Group, 1992). Table 9 simply maps the polyols, fruits, sugars, and candies and snacks to the presently used bands and suggests an additional very-low band for GI based on currently available information.

Regular, intermediate- and higher-maltitol syrups fall into the low-GI band while other polyols fall into the very-low-GI band (erythritol, xylitol, sorbitol, mannitol, isomalt, lactitol, maltitol, high-polymer maltitol syrup, polyglycitol). There is an absence from Table 9 of information on polyols in goods other than confections, such as baked goods or jams. Reduced glycaemia and insulinaemia has been demonstrated in such products (Bakr, 1997) but there is inadequate information across the time course for the calculation of GI and II. It is possible to replace sucrose (and some maltodextrins and glucose syrups) with polyols in baked goods, preserves and candies, but it is not possible to do this with intense sweeteners which lack both volume or bulk mass.

Mixtures of polyols with sugars, fats, starch-based foods and protein-based foods were shown in the present review to yield lower GI than predicted for the component GI values. Until such time as a method is established to predict such lower GI values for the mixture, it is suggested that food products might, when polyol based, have GI values that are estimated from the GI values of the ingredients; this in the same way as GI values of meals are calculated from the GI values of the component foods.

Food energy values of polyols

Various articles concerned with blood glucose control and dental health report energy values for polyols incorrectly as 17 kJ (4 kcal)/g. This value was a supposition based on the approximate heats of combustion of polyols and an assumption that each polyol was fully absorbed and used in metabolism. Numerous investigations have now been undertaken and the polyols have been found to have different values lower than their heats of combustion (Table 10). The basis of derivation of polyol food energy is that carbohydrate absorbed via the small intestine and not excreted in the urine is fully available as energy, while carbohydrate entering the colon and fully fermented is only 50 % available as energy. This basis has widespread support, and so various reviewing bodies have derived similar (though not identical) energy values to those shown in Table 10 (see Livesey *et al*. 2000). Values obtained by indirect calorimetry

Table 9. Glycaemic index (GI) bands and assignment of polyols, fruits, sugars, and candies and snacks by GI shown*

Band	Polyols	GI	Fruits	GI	Sugars	GI	Candies and snacks	GI
High GI (GI >70–140)			Dates (dried)	103	Maltose	105	Jelly beans	87
			Watermelon	72	Glucose	100	Pretzels	83
							Corn chips	72
Intermediate GI (GI >55–70)			Pineapple	66	Sucrose	65	Regular candy	70
			Banana	55	Honey	58	Fruit chews	70
							Almond bar	68
							Power chocolate bar	58
							Chocolate confection	58
Low GI (GI >40–55)	Maltitol syrups				Lactose	46	Ice-cream	52
	Intermediate	53	Grapes	54			Chocolate	49
	Regular	52	Oranges	50			Yoghurt	46
	High	48					Popcorn	45
							Chocolate coated toffee and cookie bar	44
							Chocolate peanut confection	41
Very low GI (GI 0–40)	Polyglycitol	39	Plum	39	Fructose	23	Fried chipped potato	38
	Maltitol syrup (high-polymer)	36	Apple	38			Maltitol chocolate	30
			Cherries	22			Potato chips (crisps)	23
	Maltitol	35					Peanuts	14
	Xylitol	13					Isomalt chocolate	14
	Isomalt	9					Erythritol chocolate	2
	Sorbitol	9						
	Lactitol	6						
	Erythritol	0						
	Mannitol	0						

* For references, see Table 8 footnotes and Foster-Powell *et al.* (2002).

corroborate the formula approach (see also Livesey, 2002*b*). Values accepted in the USA under the process of 'self determination' are in reasonable agreement, while European regulations (European Communities, 1990) prescribe a single value for all permitted polyols.

The USA, Canada and Australia considered whether a single value for all polyols might be misleading to the public and allocate separate values to each polyol. In the context of the labelling of individual foods, and in the context of individual food products meeting energy-reduced claims in respect of low-energy food regulations (for example, Codex Alimentarius Commission, 1991), a single energy value is not easily sustainable.

For low-glycaemic foods or dental-remineralising candies and chewing gums made with polyols, it follows that such foods and dentifrices would also be lower in energy than the corresponding product made with sugars, maltodextrins or starches. Thus a candy of 25 g portion size and made with a polyol of 8 kJ/g, which may be consumed because it is tooth friendly or low glycaemic or both, would have a food energy content of 200 kJ compared with 425 kJ for similar candies based on sugars (> 50 % energy reduction). For a snack food of 1000 kJ with the same 25 g of carbohydrate this ingredient exchange would be a 20 % reduction in energy.

Dental aspects of polyols

The role of polyols in reducing dental caries may be regarded as a benefit to part of the digestive system and so an aspect of digestive health. Other such aspects are considered further later (p. 182).

Polyols are a poor source of energy for micro-organisms of the oral cavity. Sucrose, other sugars and high-GI starches, by contrast, are readily fermented by oral micro-organisms. Such carbohydrates are acidogenic and cause tooth decay (dental caries), whereas polyols effectively do not. For this reason polyols have been described as 'tooth friendly' and are permitted ingredients in sugar-free products (European Communities, 1994).

Five key factors are involved in dental caries: teeth, bacteria, sugar or starch, time and saliva. Bacteria in the mouth reside mainly in dental plaque. Many species reside there but few continue to ferment once a critical low pH of 5·7 is reached. In the main, mutans streptococci (*Streptococcus mutans* and *S. sobrinus*) and lactobacilli are involved in acidogenesis (British Nutrition Foundation, 2000). Saliva delivers amylase that may facilitate acidogenesis from starch, but also provides buffer capacity to wash away soluble carbohydrate, acids and immunoglobulins that aggregate bacteria. Other agents in saliva are effective in protecting the body from harmful pathogens: lysozyme digests certain bacteria, lactoferrin binds and deprives bacteria of Fe, sialoperoxidase reacts with H_2O_2 and salivary thiocyanate to form a potent antibacterial agent, and hypothiocyanite (British Nutrition Foundation, 2000). Saliva also provides Ca, which supports remineralisation of demineralised teeth. Increased salivary buffer capacity on mastication might contribute to reduced caries incidence and the sweetness of polyols and sugar-free chewing frequency have each been implicated in salivation rate (Rugg-Gunn, 1989; Birkhed & Bär, 1991; Dodds *et al*. 1991; Mäkinen *et al*. 1995, 1996) though direct evidence for this is scant.

Although dental caries has a multifactorial aetiology (Burt & Ismail, 1986) and has decreased in prevalence from values 40 years ago (König, 1990), it is still a highly prevalent disease. Current evidence indicates that it does not develop without either sugars and starches or bacteria in the mouth (National Research Council, 1989; British Nutrition Foundation, 2000), and cannot occur without an increase in acid production (Bibby, 1975; Burt & Ismail, 1986). Acidogenesis in human volunteers is measurable routinely by interdental-plaque-pH telemetry (Mühlemann, 1971).

Table 10. Food energy values of polyols (reference: sucrose, maltodextrins, starch at 17 kJ (4 kcal)/g)

	Potential energy (heat of combustion)*		Formula based on current availability data†		Indirect calorimetry‡		US 'self determined' and LSRO§		European regulations‖	
	kJ/g	kcal/g	kJ/g	kcal/g	kJ/g	kcal/g	kJ/g	kcal/g	kJ/g	kcal/g
Erythritol	17·2	4·1	1	0·2	na	na	1	0·2		
Isomalt	17	4·1	9	2·1	8	2	8	2		
Lactitol	17	4·1	8	2	8	1·9	8	1·9		
Maltitol	17	4·1	11	2·7	11¶	2·6¶	9	2·1		
Maltitol syrups										
Regular, intermediate, high	17·1	4·1	12	3	na	na	13	3	10	2·4
High-polymer	17·1	4·1	12	2·8	11¶	2·6¶				
Polyglycitol	17·1	4·1	12	2·8	na	na				
Mannitol	16·7	4·0	6	1·5	na	na	7	1.6		
Sorbitol	16·7	4·0	10	2·5	na	na	11	2·7		
Xylitol	17	4·1	12	3	na	na	10	2·4		

LSRO, Life Sciences Research Office; na, information not available.
* Potentially available had the polyol been fully available. Heats of combustion are calculated (Livesey, 1992).
† Formula value = heat of combustion × (available carbohydrate + 0.5 x fermentable carbohydrate) using data from Table 3.
‡ For studies on indirect calorimetry, see van Es *et al.* (1986), Sinaud *et al.* (2002) and Livesey (2002*a*).
§ US 'self determined' labelling values are given with support from LSRO (Life Sciences Research Office, 1994, 1999).
‖ European Communities (1990).
¶ Deduced from a high-polymer syrup and its polymer fraction based on Sinaud *et al.* (2002).

The technique is used for investigation of the compliance of tooth-friendly products with the requirements of the authority in Switzerland, where sugar-free products are the major form of confectionery (Imfeld, 1983, 1993).

Lack of acidogenic potential in polyols is the major mechanism minimising caries development in polyol-based candies and sweet goods (Table 11). It appears there are no real concerns about adaptation, that is, selection of polyol-fermenting acidogenic organisms (Table 11). Adaptation is not completely absent, but does not occur to any extent that would risk caries formation from acidogenesis (Toors, 1992). Acid production in plaque after sugar ingestion follows a characteristic curve, a rapid fall in pH followed by a slow rise, called a Stephan curve. A fall below the critical pH of 5·7 puts teeth under carious attack. According to this approach, Imfeld (1993) in his review was able to classify the polyols as either having 'no cariogenic potential' or having 'virtually no cariogenic potential' (see Table 11).

Caries prevention using polyols has been described as a 'passive process' as it is the absence of acidogenic substance rather than the presence of an active or bacteriostatic substance that is important (Imfeld, 1993). However, xylitol may also be bacteriostatic on one and possibly more strains of *S. mutans* (Waaler *et al*. 1992). The mechanism proposed was the reversible inhibition of essential metabolic pathways including the accumulation of xylitol-5-phosphate, an inhibitor of phosphoenolypyruvate production. The clinical significance has been reported as a reduction in virulence of *S. mutans* and modification of the plaque ecosystem including reductions in plaque quantity and adhesivity (reduced ability to adhere to the hard tissues). The quantitative contribution this makes to caries reduction is reported as unclear by some authors (Isokangas *et al*. 1991; Scheie *et al*. 1998; Alanen, 2001). Nevertheless, xylitol is commonly associated with reduced numbers of *S. mutans* (Hayes, 2001; Mäkinen *et al*. 2001), appears more effective than erythritol in reducing the mass of plaque in human subjects (Mäkinen *et al*. 2001), and is more effective than sorbitol in caries prophylaxis (Mäkinen *et al*. 1996). A difficulty with the interpretation of these comparisons is a lack of quantitative information on the separate roles of saliva stimulation and microbiological factors (Alanen, 2001). Interestingly, the reduced transmission of *S. mutans* from mother to offspring may explain a lower caries incidence in 2- to 5-year-old children after maternal xylitol consumption when the children were aged 3–24 months (Isokangas *et al*. 2000).

Less well known than the virtually non-acidogenic potential of polyols as sugar replacers is their limitation of plaque formation. Plaque is a conglomerate of bacteria and polysaccharides where acidogenesis takes place. The polysaccharides synthesised by oral bacteria bulk out the plaque, which in turn harbours these organisms and retains fermentation products, so depressing the pH further and reducing the ability of saliva to wash the organisms and acid away (Newbrun, 1982; Rolla *et al*. 1985). By contrast polyols are not substrates for polysaccharide and plaque synthesis. Isomalt, while not supplying substrate for polysaccharide synthesis (Bramstedt *et al*. 1976; Ciardi *et al*. 1983), might also inhibit this process from sucrose, as evident for some of the longer-chain hydrogenated isomalto-oligosaccharides (Tsunehiro *et al*. 1997). Polysaccharide synthesis is also lower with xylitol, lactitol, mannitol and sorbitol than with sucrose (Grenby *et al*. 1989).

Polyols also reverse the initial stages of dental caries by promoting remineralisation. This is preferable to tooth restoration except on advanced lesions (Featherstone, 2000). Stimulation of salivary flow facilitates remineralisation when induced between meals by confections containing a polyol; this is evident because the repair of early lesions is greater when such products are ingested than when no food is consumed (Leach, 1987). A recent and important observation is that polyols both slow demineralisation of tooth enamel and accelerate remineralisation of

Table 11. Cariogenic potential, bacteriostasis, inhibition of polysaccharides synthesis, remineralisation and adaptation

		Minor (active) mechanisms			
	Major (passive) mechanism: cariogenic potential* (based on acidogenesis)	Bacteriostasis	Inhibition of polysaccharide synthesis	Promotion of remineralisation	Concerns: significant adaptation
Erythritol	None to virtually none*	–	–	–	–
Xylitol	None	Yes†	–	Yes§	None‖
Sorbitol	Virtually none	–	–	–	None¶
Mannitol	Virtually none	–	–	–	–
Maltitol	Virtually none	–	–	–	–
Isomalt	None	–	Suggested‡	Yes§	None**
Lactitol	None	–	–	–	None††
Regular maltitol syrup	Virtually none	–	–	–	None‡‡

* After Imfeld (1993), except eythritol for which a preliminary classification is given here. This reflects the practical inability of oral bacteria to use these carbohydrates for acid production (or for plaque polysaccharide synthesis).
† Waaler *et al.* (1992). The quantitative contribution of this bacteriostatic mechanism to clinical outcome is unknown, though may explain advantages of xylitol over sorbitol and erythritol (see p. 181).
‡ Ciardi *et al.* (1983), Bramstedt *et al.* (1976). Quantitative contribution to clinical outcome is unknown.
§ Takatsuka (2000), Mäkinen *et al.* (1995).
‖ Toors (1992), Gehring *et al.* (1975).
¶ Toors (1992), Cornick & Bowen (1972).
** Van der Hoeven (1979, 1980).
†† Havenaar *et al.* (1978).
‡‡ Rugg-Gunn (1989).

demineralised lesions. Xylitol and particularly isomalt may be effective in this regard (Takatsuka, 2000).

On the basis of substantial studies in human subjects regarding caries the prophylactic properties of lactitol, isomalt and xylitol have been recommended (Imfeld, 1993; Featherstone, 1995; Mäkinen *et al*. 1996). Clinical trials on sorbitol (Birkhed & Bär, 1991), maltitol syrup (Rugg-Gunn, 1989) and xylitol (Mäkinen *et al*. 1996) indicate that they are non-cariogenic. Erythritol has been advanced as a potential new caries preventative (Kawanabe *et al*. 1992; Mäkinen *et al*. 2001).

Colonic health aspects

The colonic environment

Due to their ease of fermentation by gut flora, low-digestible carbohydrates are very important in human health. Such carbohydrates contribute fundamentally to the establishment of an anaerobic and acidic environment in the colon. Their fermentation enables saccharolytic anaerobes and aciduric organisms to grow in preference over putrefying, endotoxic, pathogenic, and procarcinogen-activating aerobic organisms (Hawksworth *et al*. 1971; Brown *et al*. 1974; Gracey, 1982; Hill, 1985; Hill *et al*. 1987; Rowland, 1991; Mitsouka, 1992; Screvola *et al*. 1993*a*,*b*; Mital & Garg, 1995).

Low-molecular-weight carbohydrate (lactulose) and polyol (lactitol) have long been acknowledged for their ability to reduce circulating levels of NH_3 and toxic microbial substances, the clinical utility of which is the treatment of hepatic encephalopathy (Blanc *et al*. 1992).

The acidic conditions associate with or normalise epithelial functions resulting in fewer pathologies and their markers, such as aberrant crypts (Samelson *et al*. 1985), large adenomas (Roncucci *et al*. 1993; Ponz de Leon & Roncucci, 1997; Biasco & Paganelli, 1999) and possibly tumours (Thornton, 1981). Lactic acid is of particular note; it is generated from all fermentable carbohydrates but especially those that readily undergo microbial glycolysis including polyols. A slow removal of lactic acid from the colon would help to maintain acidity and the growth of aciduric organisms such as the lactic acid bacteria, which are now widely promoted as probiotics. Butyric acid, which can be generated from polyols, sometimes in large amounts (Mortensen *et al*. 1988; Clausen *et al*. 1998), and possibly due to secondary fermentation of lactic acid, is widely recognised for its probable role in maintaining a healthy colonic epithelium. It is also recognised for its improvement of inflammatory conditions of the colonic mucosa (Roediger, 1990; Scheppach *et al*. 1995) and anti-neoplasic activity (Velazquez *et al*. 1996; Scheppach *et al*. 2001; for a review, see Brouns *et al*. 2002). Although faecal butyrate is not especially prominent amongst black South Africans who are renowned for their healthy colons, raised concentrations of short-chain organic acids (Segal *et al*. 1995) and acidity (Levy *et al*. 1994) are found in these individuals. This has been attributed to increased fermentation and a higher than usual entry into the colon (than in Westerners) of osmotic carbohydrate (Veitch *et al*. 1998; Segal, 2002).

These responses can generally be attributed to saccharolytic fermentation, ease of fermentation, and water entry into the colon with osmotic carbohydrates; thus responses have been reported for a wide range of low-digestible and fermentable carbohydrates including polyols in human subjects. For example, responses have been reported for lactulose (MacGillivary *et al*. 1959), lactitol (Felix *et al*. 1990; Screvola *et al*. 1993*b*; Ravelli *et al*. 1995; Tarao *et al*. 1995; Ballongue *et al*. 1997), isomalto-oligosaccharides (Kaneko *et al*. 1994), lactosucrose (Teramoto *et al*. 1996), and fructo-oligosaccharides (Gibson & Roberfroid, 1995; Tuohy *et al*. 2001). Gibson & Roberfroid (1995) have reported responses for inulin, Zhong *et al*. (2000) for polydextrose and Bird *et al*. (2000) for some resistant starches. For individuals with disaccharidase deficiencies, similar reports appear for lactose (Segal, 1998, 2002) and sucrose (Veitch *et al*. 1998; Segal, 2002), and incomplete absorption of fructose (Segal, 1998). Short-chain organic acids may also modify gastrointestinal motility and so could have a role in maintaining a regular bowel habit (Cherbut *et al*. 1998; Piche *et al*. 2000).

Constipation and laxation

Constipation may be defined most simply as ‘less than three bowel movements per week’ and is the most common gastrointestinal complaint in Western cultures, triggering considerable use of over-the-counter laxatives and consultations with medical practitioners (Royal College of General Practitioners, 1986; Sandler *et al*. 1990; Sweeney, 1997). It is particularly common in the elderly (Koch & Hudson, 2000), diabetics (Haines, 1995), children (Guimaraes *et al*. 2001) particularly those with developmental and neurological disability (Staiano *et al*. 2000; Tse *et al*. 2000), pregnancy (Signorelli *et al*. 1996), and in those with reduced food intake (anorexia, weight reduction, hospitalisation). It is also common in numerous other less prevalent circumstances (Baker *et al*. 1999; Nurko *et al*. 2001). Some drugs are causative, including the commonly used Al antacids and dietary Fe supplements (Baker *et al*. 1999).

Laxation is the ‘gentle stimulation of the bowel to render the motion slightly soft without causing any gripes’ (Macpherson, 1990). Laxative action has been established for acceptable intakes of xylitol, sorbitol, mannitol, isomalt, lactitol, maltitol and erythritol (Brin & Miller, 1974; Sheinin *et al*. 1974; Ornskov *et al*. 1988; Livesey, 2001; Marteau & Flourié, 2001). All act to promote hydration of the colonic contents. Usefully, polyols are obtainable by the public in tasty food items such as sugar-free, reduced-energy candies and other products. Studies have demonstrated the efficacy of polyols (crystalline or syrup formulations) in the elderly (Lederle *et al*. 1990) and in a multicentred study of the elderly both hospitalised and outpatients (Delas *et al*. 1991; Sacchetta *et al*. 2000), and in children (Ornskov *et al*. 1988; Pitzalis *et al*. 1996). Data from Spengler *et al*. (1987) indicate approximately 30 % less constipation even amongst young adults with ‘healthy colonic function’ consuming up to 48 g isomalt daily (thirty subjects, 84 d each), and without excess laxation. Also, lactitol and lactulose each show a reduced likelihood of slow transit occurring in physically inactive hospitalised individuals with healthy gastrointestinal tracts (Pontes *et al*. 1995).

Adequate drinking water has been recommended along with dietary fibre to enhance laxation (Gray, 1995; Anti *et al*. 1998). However, even this dual action may be inadequate (Benton *et al*. 1997). Rural South African and Asian diets are thought ideal for optimal stool formation. However, osmotic carbohydrate in these diets may be just as important as dietary fibre due to sucrase and lactase 'deficiency' in these populations (Veitch *et al*. 1998; Segal, 2002) promoting an adequate hydration of colonic contents. Westerners without lactase and sucrase deficiency could, logically, achieve the same goal with appropriate intakes of polyols.

A consensus of food and nutritional scientists and physicians has been established for polyol consumption: 'Each individual may experiment with intake amounts and make adjustments based on their own experience – as they may do routinely with everyday foods having the same effects when eaten to excess' (Salford Symposium Consensus, 2001). This was recommended because individuals vary in the magnitude of their response to polyol ingestion, as indeed they do in the degree to which constipation is experienced. Physicians have also recommended that individuals 'adjust the dose of polyol to a daily bowel movement for 1 to 2 months' (Baker *et al*. 1999; Nurko *et al*. 2001).

Tolerance

Low- and very-low glycaemic-carbohydrate foods can be a cause of unwanted gastrointestinal responses in sensitive individuals or when ingested to excess due to their reaching the colon. Increased gastrointestinal awareness is commonly experienced with high-fibre foods, some of which are low-glycaemic-carbohydrate foods such as beans, lentils and legumes. Other foods include cabbage, Brussels sprouts, brown bread, oatmeal porridge, rough-seeded fruits, honey, tamarinds, figs, prunes, raspberries, strawberries, stewed apples, aloes, rhubarb, cascara and senna (Macpherson, 1990; Friedman, 1991) and modest levels of fibre supplements (Stevens *et al*. 1987). Similar responses can occur without a change in food source by lowering of the GI using pharmacological means; the sucrase inhibitor acarbose results in elevated flatulence in up to 43 % of consumers and osmotic diarrhoea or laxation in up to 27 % (Sels *et al*. 1998). All such foods and carbohydrates can be a cause of increased colonic fermentation, flatulence, bloating and cramp. Feelings of bloating (as opposed to measurements of abdominal distension) are probably more common after overingestion of food in general, which is all too common. Furthermore, cramp appears to be secondary to faecal impaction in those with a poor bowel habit (McRorie *et al*. 2000), or in individuals with irritable bowel syndrome (Briet *et al*. 1995). In contrast to infectious diarrhoea, watery stools due to colonic fermentation of low-digestible carbohydrates are not a medical issue, and intakes of polyols comparable or greater than normal for dietary fibre are possible (Steinke *et al*. 1961; Sheinin *et al*. 1974; Spengler *et al*. 1987; Sinaud *et al*. 2002; A Lee, DN Storey, F Bornet and F Brouns, unpublished results).

Rapid transition from a diet that encourages constipation (diets low in polyols, dietary fibre and some slimming diets) to ones that promote laxation (high polyol, dietary fibre and high food intakes) may be a transient cause of discomfort (see McRorie *et al*. 2000). This may be avoided by varying the daily intake of polyol-based foods gradually over a period of 1 to 4 weeks (see Steinke *et al*. 1961; Baker *et al*. 1999; Salford Symposium Consensus, 2001; Nurko *et al*. 2001). Adaptation to polyols usually improves gastrointestinal tolerance (Tucker *et al*. 1981; Pometta *et al*. 1985; Briet *et al*. 1997) and may in part be psychological (Tucker *et al*. 1981) and occur with an increasing experience of fermentable carbohydrate consumption (Briet *et al*. 1997). Tolerance and intakes are greatest when polyols are consumed at regular intervals throughout the day (Livesey, 2001; Sinaud *et al*. 2002) as may be desirable in some diabetics (Warshaw & Powers, 1999). Consuming polyols in or with other foods will also improve tolerance by delaying stomach emptying (Livesey, 1990*a*, 2001; Marteau & Flourié, 2001). In this respect the co-ingestion of a high-cereal-fibre diet may be useful as it provides a matrix with which water combines to be retained in the large bowel. Some individuals are sensitive to polyols and should reduce or even avoid such foods altogether (Salford Symposium Consensus, 2001). Children more than younger or older adults are likely to consume larger amounts of freely available polyols; there is, however, no evidence that children are less able to tolerate polyols than are adults in terms of the weight of polyol per meal or d (Spengler *et al*. 1987; Paige *et al*. 1992; A Lee, Salford University, personal communication).

The scientific interpretation of consumer responses to polyols is difficult. Consumers generally indicate that they have diarrhoea whenever they notice a softening of their stool independently of whether it is inconveniencing and some 98 % of such occurrences do not meet commonly accepted criteria for clinical diarrhoea (McRorie *et al*. 2000). In agreement, a market survey of 1000 consumers of sugar-free products (polyols) has indicated that as little as 0·5 % of individuals make unprompted claims to the experience of adverse gastrointestinal responses (Stewart, 2001). This coincides with the rate observed in the absence of polyol consumption (Steinke *et al*. 1961; Spengler *et al*. 1987). Reported responses to polyols are often based on questionnaires that prompt volunteers to notice symptoms of intolerance, and so may be biased; thus when prompted such claims may increase five-fold (Stewart, 2001). Also the interpretation of scientific studies in a laboratory setting can be difficult due to substantial inter-individual variation in gastrointestinal responses to polyols (Livesey, 2001), adaptation (Marteau & Flourié, 2001) and other reasons (Barlow, 2001).

There are probably more non-diabetics who consume polyols than diabetics, though the latter have often been the subject of study. The American Diabetes Association (2001) has suggested, bearing in mind the varied responses among individuals, that the choice to consume particular types of carbohydrate including polyols must be an individual one, taking account of global dietary guidance and individual metabolic needs. Constipation can be common in diabetics and older individuals (Wegener *et al*. 1990) and older diabetics may indicate that polyol consumption improves bowel habit (Pometta *et al*. 1985). Idiopathic

diarrhoea also occurs in diabetics, but is not due to the increased use of polyols, and polyols are not contraindicated in diabetics when consumed in moderate amounts (Verina *et al*. 1995). Individuals with type 2 DM tolerate polyols equally as well as normal individuals (Zumbé & Brinkworth, 1992; Verina *et al*. 1995).

Conclusion

Polyols are found to provide acknowledged examples of clinical benefits in the treatment and regulation of bowel habit, and in the conditioning of the colonic environment. Intriguingly, appropriate consumption of low-digestible osmotic carbohydrates may be critically important in Westerners to achieve stool consistencies comparable with those of rural South Africans. These benefits add to the acknowledged properties of polyols as reduced-energy carbohydrates and to the benefits of tooth friendliness, where polyols may have a role in the repair as well as the prevention of caries. The low- to very-low-glycaemic and insulinaemic properties of polyols offer further potential health benefits on replacement of bulk in sugars, syrups and maltodextrins in foods for individuals with both normal and abnormal carbohydrate metabolism. Scope exists for such benefit within gastrointestinal tolerances, which can be improved by attention to the dose, timing, and diet during polyol consumption.

Information on the glycaemic and insulinaemic responses to polyol-based foods is scarce compared with information on polyols alone. Nevertheless, it is evident that interactions between polyols and macronutrients tend to reduce postprandial glycaemia, and interactions between sugars and fats that elevate postprandial insulinaemia can be attenuated or almost abolished using polyols. There is no reason to suppose that long-term use of polyols elevates protein glycation, a marker of glycaemic control, as do high-glycaemic carbohydrates, and there is evidence that the consumption of a polyol might reduce protein glycation, adding to similar observations for other low-glycaemic-carbohydrate diets.

On a technical note, as with carbohydrate foods tabulated in the international tables of GI (Foster-Powell *et al*. 2002), where data are available on polyols it is found acceptable to pool information on GI values from normal, type 1 and 2 DM patients to obtain a single value for each polyol applicable in all these conditions. Similarly, there is no more dose-dependency of GI values for polyols than for other carbohydrates.

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