NON-DIGESTIBLE CARBOHYDRATES
AND GLYCAEMIC CONTROL

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ABSTRACT

Mechanisms are indicated whereby fibre and polyols could reduce blood glucose and insulin concentrations and so potentially help to reduce the prevalence of diabetes, CHD, and certain cancers. Seven are identified independent of associated substances or antioxidant or lignans. Further, high cereal fibre intake additionally lowers the starch:fibre ratio thus lowering the glycaemic index of total carbohydrate; this may be helped by fiber as cell walls in whole grain causing some starch to be unavailable. It seems that glycaemic index expressed as g equivalents per g available carbohydrate does not account for the full effects of non-digestible carbohydrates on lowering of blood glucose in some circumstances, whereas glycaemic load in units of g glucose equivalents per day (or per unit food) would. Overall, the glycaemic load appears stronger at present than glycaemic index in several circumstances: the epidemiology on development of type-2 diabetes and heart disease when combined from different studies; the epidemiology of HDL, TG, and C-reactive protein; the epidemiology of haemorrhagic stroke; the interventions on body weight in ad-libitum studies; and the interventions on glycated proteins and fasting blood glucose in diabetics. Information appears more consistent than is generally discussed in the literature and appears independent of the concepts of whole grain and reduced energy density. Scope exists for a reduction in glycaemic load by replacing high glycaemic available carbohydrate with either low-glycaemic available carbohydrate or fibre or polyols and this potentially could reduce the prevalence of diabetes and CHD by up to thirty percent.

INTRODUCTION

Interest in non-digestible carbohydrates grew with the dietary fibre hypothesis that linked an absence of dietary fibre with the prevalence of a variety of western diseases, with notable contributions supporting the hypothesis from medical researchers in South Africa [1,2]. Further impetuous arose when it was recognised that diabetics could, given medication, tolerate carbohydrate provided the foods included plenty of dietary fibre [3]. We now recognise this is not the entire picture, since further glycaemic control in diabetic patients develops when a low rather than high glycaemic carbohydrate diet is consumed [4]. The general picture for glycaemic control is that a high-fiber, low-glycaemic and low-saturated fat diet is optimal [5].

The need for such diets to be low in saturated fats and higher in polyunsaturated fats originates from concerns about fats and coronary heart disease rather than the low fibre intake that a high-fat diet might imply [6,7]. This is especially important for diabetics who are at a much increased risk of CHD. The carbohydrate was simply seen as a means to displace dietary fat as an energy source. With obesity being a major problem and a risk factor for type-2 diabetes, then energy balance has become of major importance [8], and weight loss has for some time been recognised as important to the survival of newly diagnosed type-2 diabetics [9]. It is clearly preferable to limit the intake of both saturated fats and high-glycaemic carbohydrate as energy sources. Nutrition debates of fat versus carbohydrate now seem somewhat futile, rather the issue is the adverse influence of saturated fats plus high glycaemic carbohydrates versus the relative benefit of other energy yielding and reduced energy substrate, which includes non-digestible carbohydrate.
Non-digestible carbohydrates play a role in reducing food intake via influences on food composition and structure and so facilitate low-energy density food production and formulation. Low saturated fat diets (perhaps more particularly diets low in rapidly available C12,14, and 16 fatty acids) that are also moderately low in glycaemic load should be seen as preferable to high-carbohydrate low-glycaemic index diets in obesity, diabetes, stroke, coronary heart disease, and cancers in total. The former better informs the manufacturer and the consumer. Emphasis on high carbohydrate diets of low glycaemic index is perhaps only necessary in those who are unable to control saturated fat intake by any other means.

While reduced saturated fat diets improve plasma lipid profiles, which signifies reduced CHD risk, there is epidemiological evidence of low saturated fat diets associate with better glycaemic control [10], presumably by improving insulin sensitivity and reducing the glycaemic response to diet in the longer term. Good glycaemic control among non-diabetics is also an indicator of lower risk for coronary heart disease [11]. Reduced food intake and exercise are likely to operate similarly, improving long-term glycaemic control via sensitising tissues to insulin, and so reducing the risk of diseases that emerge from the metabolic syndrome. Although studies on CHD look for improved plasma lipid markers, they could as well examine protein glycation (HbA1c). Glycaemic control may be recommended as the first approach in managing dislipidaemia [12].

Failure to recognise correctly the variety of dietary determinants of adverse glycaemic control has, I believe, lead to an unduly narrow glycaemic index concept. While this concept was initially a major break though in the area of carbohydrates and health, there is now a risk that too narrow an understanding of the glycaemic concept, as glycaemic index, may impede the further development, prescription, and understanding of optimal diets. Too narrow a focus will have both economic and health implications. In diabetics, medication alone or in combination with some prior ‘good’ (lower fat) dietary practice is still insufficient as deterioration of glycaemic control still occurs, measurable as a ‘coefficient of treatment failure’ (annual rate of rise in HbA1c)[13,14]. Today the situation is that a good proportion of diseases such as type-2 diabetes mellitus, CHD, stroke, and certain cancers are potentially avoidable in a good proportion of people, yet these diseases are set to overburden governmental and private health budgets. However, it is not my remit here to dwell further on the variety of ways in which high-glycaemic carbohydrates can be replaced in the diet to health advantage, rather it is to focus on the use of non-digestible carbohydrates for this purpose.

NON-DIGESTIBLE CARBOHYDRATES, POST-PRANDIAL GLYCAEMIA AND DIABETES

The impact of added fibre in various forms on the acute glycaemic response to glucose is well known [15]. There is also a considerable literature interrelating high-fibre, high-carbohydrate diets, diabetes and coronary risk factors including improvement in total cholesterol but deterioration in triglycerides and glycaemic control (HbA1c) when the carbohydrate is rapidly available [16-19]. It is not intended to review these here, rather more recent aspects will be addressed. Nevertheless, it is worth noting that fibre is often mentioned by diabetes associations as a means to help lower postprandial glycaemia from available carbohydrates and to aid body weight maintenance. Replacing carbohydrate with non-digestible fiber or polyols can be equally or more effective [20,21] and in both cases complete control over insulinemia is possible for meals of limited size, which may reduce a reported coronary artery disease risk from compensatory hyperinsulinaemia [22]. In some foodstuffs it is technically possible to use polyols when fibre cannot be used, due to the need for crystallinity, likewise it is possible to use polyols in some circumstances when intense sweeteners cannot be used, due to the need for bulk. It is emerging that some low glycaemic polyols, for example erythritol and isomalt, even appear better tolerated in the gastrointestinal tract than some frequently used carbohydrates classified as fibre.

THE GLYCAEMIC RESPONSE CONCEPT

Various terms exist to describe the relationship between glycaemic response and health (Fig.1). Although current information generally places glycaemic load ahead of the glycaemic index in the strength of associate with health (see above), for the moment no consensus has developed on
which, index or load or other term is most useful in food production and communication with the consumer. Hence, I believe it is currently preferable to refer to the glycaemic response concept, rather than the glycaemic index concept. Moreover, it appears that glycaemic load is central to all expressions of the glycaemic response. Direct measures avoid certain inaccuracies and pitfalls (legend to Fig. 1). Due to their recent introduction the direct measures are not in widespread use. Neither national nor international agreement exists on a preferred approach.

GLYCATION AND HEALTH

Elevated concentrations of glycated haemoglobin (HbA1c) in diabetes is a risk factor for retinopathy, peripheral vascular disease (limb amputation) and death [11,23]. In a population of men without recognised diabetes it is a substantial factor pointing to a history of heart disease and stroke, and is a risk factor for ischemic heart disease and high blood pressure [11].

GLYCATION AND DIET INCLUDING NON-DIGESTIBLE CARBOHYDRATES

Intervention studies have shown in diabetes mellitus a reduction in glycated proteins (HbA1c and/or the more rapidly responsive fructosamine) using protein [24], polyol [20], fibre [5], low-glycaemic carbohydrate in place of high-glycaemic carbohydrate [4], fructose alone as a low glycaemic carbohydrate [25-28] but not with monounsaturated fats or total fats. A lack of benefit of ‘non-glycaemic’ fats is presumable due to long-term influence on insulin sensitivity and subsequent potentiation (or more with saturates) of post-prandial glycaemic excursions [5]. Lowering the glycaemic response to diet using an inhibitor of carbohydrate digestion (acarbose) also lowers glycated protein [29]. Drugs for the control of post-prandial glycaemia also lower the glycated protein concentration [30]. There can be little doubt that protein glycation is related to post-prandial glycaemia, and that both are related to diet composition. This is a marked advance as prior to all these studies only fasting glycaemia was thought significant. There seems little doubt too that the glycaemic response concept underlying the variety of food based approaches to glycaemic control is distinctly different to other nutrition and health concepts such as the whole grain food concept and the energy density concept.

The effectiveness of replacing high with low-glycaemic carbohydrate for reducing glycated protein concentrations in diabetics appears to depend on the severity of the condition Greater reduction occurs when the fasting blood glucose concentration is high (author, unpublished). This somewhat invalidate simple fixed-effects meta-analyses [4,5,31]; thus the significance and magnitude of effect for management of severe disease has been underestimated.

BLOOD GLUCOSE CONTROL

The diagnosis, treatment and severity of diabetes mellitus has long been assessed from the fasting blood glucose value. However, this is a less stable measure than is glycated protein mentioned. As with glycated protein, intervention studies indicate low-glycaemic carbohydrate in place of high-glycaemic carbohydrate reduces fasting glucose, again with greatest benefit for severe diabetes (author unpublished). There is evidence that non-digestible carbohydrate used either to replace available carbohydrate or to reduce food intake (so limiting both high-glycaemic carbohydrate and saturated fat intake) can achieve a similar response [20], as might be expected.

REDUCTION OF POSTPRANDIAL GLYCAEMIA AND INSULINAEMIA WITH UNAVAILABLE CARBOHYDRATES – FIBRE AND POLYOLS

Replacement of higher glycaemic starches, maltodextrins and sugars with polyols or fibre in food products can contribute to a lower post-prandial glycaemia. This arises from non-absorption of carbohydrate, and with some polyols the slow conversion of monosaccharide polyol to glucose in the liver. Table 1 shows the glycaemic and insulinaemic loads from certain fibres and polyols. It has been established that values in healthy people, type-2 diabetics and type-1 diabetics with an artificial pancreas have similar glycaemic and insulinaemic responses to polyols, and that these are essentially independent of dose [20]. Several approaches are possible for the classification or
bANDING of the glycaemic response to foods according to different modes of expression (see Table 1). All the fibre and polyols fall within the low and very low or non-glycaemic bands.

**POLYOLS AS NON-DIGESTIBLE CARBOHYDRATE**

Addition of 6g/meal of isomalt (90% unavailable) four times per day at mealtimes lowers glycated haemoglobin in type-2 diabetics [20]. The effect seen is as great as that occurring on average with low-glycaemic carbohydrate diets, and was sufficient during the 3 month of treatment to reverse deterioration in glycated protein with time.

This treatment also lowered fasting glucose, as well as lowering postprandial glycaemia. These effects were modest at first though progressive with time up to 3 months.

**FIBRE AS NON-DIGESTIBLE CARBOHYDRATE**

Consistent with there being no effect of low-glycaemic carbohydrate on glycated protein in healthy people, randomly bonded glucose fibre showed no influence on glycated haemoglobin concentrations in healthy people [32] while doses of 20 g/meal reduced fasting glucose in diabetics over 12 weeks [33]. Similarly, improved glycaemic control (glycated haemoglobin) was observed in type-2 diabetics following guar gum ingestion [34-37] though evidently not in insulin sensitive type-1 diabetics [38] or in non-diabetic subjects [39], and not always in type-2 diabetics [40] possibly because fasting glucose was already well controlled [41] or because saturated fat intake was low so that glucose control would already have been optimised [42]. The finding of benefit in one circumstance and not another is reminiscent of similar findings for glycaemic index of available carbohydrates – i.e. the response depends on the severity of the condition, and possibly on the fat or saturated fat content of the treatment diet.

With low GI foods the main effect may be argued to arise from resistance carbohydrate that remains ‘hidden’ by food tables or analytical methods, while with added fibre similar effects may be due to the reduction in the glycaemic response. Soluble fiber from corn bran also lowered glycated haemoglobin in people with impaired glucose tolerance but not in healthy people [43]. There is evidence of possible improvement due to the consumption of Konjac mannan in diabetics too [44] and a study with better experimental control shows there is evidence in high risk type-2 diabetics [45], though no effect was reported with of lupine fibre [47]. It remains to be seen whether studies showing lack of effect in diabetics are the result of underpowered studies, studies under circumstances not expected to show effectiveness of fibre (e.g. low saturated fat diets) or whether some fibre preparations can have no effect and why this should be so. In diets of comparable high carbohydrate content, those also with high fibre content show improved glycaemic control in diabetics [5]. In type-1 diabetes there is population cross sectional evidence relating higher fibre intake to lower glycated protein [48], presumably the population included some insulin resistant patients. The general phenomenon is nevertheless relevant under circumstances commonly encountered in free living populations in western culture, and is consistent with fibre offering protection against the development of type-2 diabetes [49,50] and heart disease [51-54].

It follows that with general observations of saturated fats raising and dietary fibre lowering protein glycation, the replacement of saturated fats with fibre would lower protein glycation from raised levels, and this has been observed [55].

**MODES OF ADMINISTRATION OF NON-DIGESTIBLE CARBOHYDRATE: MECHANISMS**

A detailed discussion of mechanisms is beyond the scope of this narrative review; however, non-digestible carbohydrate is likely to act in different ways dependent on the mode of its administration in foods and diets. Firstly, replacement of available with unavailable carbohydrate will lower postprandial glycaemia and insulinaemia with attendant benefits. Second, dietary fibre may act via the colon as a source of fermentable carbohydrate, the molecular mechanisms are yet to be established though may include a reduction in free fatty acids due to the appearance of short-chain fatty acids in plasma [56] or to a colonic incretin response [58,59]. Third, when
unavailable carbohydrate replaces fat in the diet the fat is less able to increase free fatty acid concentrations and would likely favour an improved sensitivity to insulin. A fourth mechanism in free-living people (when caloric intake is not fixed) may be an increase in satiety and so a reduction of food intake (thereby lowering both glycaemia and post-prandial free fatty acids), this applies also to polyols and in animals may contribute to survival longer in old age [21]. A fifth mechanism may be inhibition of digestive enzymes to cause a lowering of post-prandial glycaemia from disaccharides, oligosaccharides and polysaccharides (though apparently not from monosaccharides when the fibre is randomly polymerised glucose, so implicating slower digestion rather than impaired absorption) [60]. Similarly, there is potential for inhibition of digestion of maltose (cf. from starch) by the polyol isomalt, which comprises both competitive and non-competitive inhibitory action on the maltase activity of human jejuna biopsies [61]. A potential sixth mechanism with unavailable carbohydrate from polyols is a more rapid passage of digesta through the uppermost absorptive region of the small intestine (the duodenum and jejunum), which could lower glycaemia from absorbable or digestible mono, di and polysaccharides because absorption from the distal intestine could be less rapid, as observed with free glucose [62]. None of these mechanisms are reflected in the concept of glycaemic index of available carbohydrate. Hence, the glycaemic index of available carbohydrate can be problematic for communicating the potential benefits of polyols, dietary fibre, and so foods as a whole.

A seventh mechanism is the slowing down of the digestive process due to either viscosity or cell-wall polysaccharides causing inaccessibility of enzymes to starch or limitation of diffusion of sugars. The glycaemic index concept based on assessment of 25-50 g available carbohydrate captures only a part of this mechanism. When 100 g wholemeal bread replaces 100 g white bread, the glycaemic load is lower (more fibre less starch) but this is not captured in the glycaemic index methodology since the methodology dictates that increased amount of food are eaten to compensate for the dilution of starch with fibre. Thus whereas in practice people tend to eat smaller amounts of higher fibre foods, the glycaemic index methodology requires a reversal of this practice to assure the same ‘available’ carbohydrate load.

In whole grain cereals the cell wall fibre may also cause some of the starch measured as available in vitro to be unavailable in vivo. This would confound accuracy of the glycaemic index expression but not the accuracy of the glycaemic load expression and may contribute to observed benefits of cereal fibre in the prevention of diabetes and CHD [49,50,63].

**FOOD MIXTURES**

In general, mixtures of carbohydrate foods result in predictable glycaemic responses in mixed meals. Similarly, the very low glycaemic responses to polyols of varying unavailable carbohydrate content are retained or reduced further when these products are consumed in mixtures with monosaccharides, disaccharides, starches, proteins and fats [20].

In general, inclusion of fat with carbohydrate lowers the glycaemic response. Often this is attributed to slower emptying of the stomach. However, this explanation would lower the insulin response too. In fact, the insulin response is elevated when fat is mixed in with either available carbohydrates or polyols [20]. Thus at least two mechanisms operate. One is a lower rate of stomach emptying, another is an elevation of incretins, hormones released intestinally by the occurrence of fat absorption. Incretins amplify the insulin response to elevated glucose concentrations.

As noted earlier, while saturated fats may lower the glycaemic response to foods acutely, in the long term they elevate the response through influence on the metabolic state, including the development of diabetes and heart disease. Thus, total fats and especially or only saturated fats, elevate the long-term glycaemic load and could be assigned a glycaemic load equivalent to that of glucose. It remains to be established, in epidemiological studies for example, whether fats especially saturated fats have additive effects with glycaemic load in the development of metabolic diseases.
AVOIDANCE OF HYPERINSULIN SECRETION WITH CARBOHYDRATE-FAT MIXTURES

Hyper-insulin secretion is a contributor to heart disease, as indicated by epidemiological studies from Helsinki [64], Paris [65], and Australia [66], and when the pancreas has a low capacity for insulin excretion it is thought to cause beta-cell exhaustion and so type-2 diabetes. Such associations may not always be linear [66]. Excessive carbohydrate or excessive fat or both in excess in the diet is a cause of hyper-insulin secretion. The insulin hypersecretion can be avoided by completely replacing available carbohydrate in fatty foods with very low or non-glycaemic polyols [20]. Further study would be advantageous.

EMPHASIS RELATED TO HIGH FAT FOODS

That high glycaemic foods should not carry reduced or low fat claims appears not to have entered discussion. Conversely, there is some agreement (in the absence of all perspectives) that high fat foods should not carry claims about other health benefits, for example about inclusion of reduced or low glycaemic carbohydrate. The latter may well be a mistake, as too may be the bias 'for carbohydrate- against fat', which as discussed is too simplistic. In principle, it seems to be precisely the higher fat foods that ought to be modified to contain non-glycaemic counterparts of higher glycaemic available carbohydrates, thus reducing in the diet or eliminating in certain foods the insulin hypersecretion. For example, chocolate is a high fat food containing available carbohydrate and causes an insulin response much higher than expected from its low glycaemic response or even for its fat content [20,67]. There may well be an improvement in the health aspect of such confessions by choosing chocolate with very low or non-glycaemic carbohydrate. This would be additional to any benefits from anti-oxidants etc in the cocoa [68].

ELIMINATION OF THE GLYCAEMIC RESPONSE OR JUST LOWERING IT

The benefits of low and non-glycaemic carbohydrates such as polyols and fibre is not the elimination of postprandial glycaemia and insulin secretion altogether, but the elimination of too high a post-prandial glycaemia and hypersecretion of insulin. This is achievable with low-glycaemic available carbohydrate and with non-glycaemic carbohydrates polyols and fibre. Potentially, the replacement of carbohydrate with non-digestible carbohydrate may be more effective for haemorrhagic stroke than either the lowering of glycaemic load or glycaemic index. This is because a stronger relationship is reported with carbohydrate intake. Whereas once we in the UK considered high carbohydrate diets as safe on the based on experiences of such diets in Asian countries, we now lean that the high carbohydrate may be responsible for the high contribution of haemorrhagic stroke to cardiovascular disease mortality whereas cereal fibre may be protective [69].

CONSISTENCY OF INFORMATION ABOUT REDUCED GLYCAEMIA

Body Weight

Controversy has arisen about the role of carbohydrate versus fat, and glycaemic index in body weight control. The state of the evidence at present is that low glycaemic carbohydrate diets eaten ad libitum promote weight loss or reduce weight gain (Livesey, 2004). Certain caveats remain. Firstly, this is not proved beyond doubt - although the data looks consistent there is insufficient evidence to be sure that publication bias is not a significant factor. Second, body weight reduction may not result in all circumstances. Third, pharmacological means to reducing the glycaemic response through using acarbose is not consistently effective in body weight reduction. Fourth, as with the high protein diets, results longer than one year are not available. Studies on non-digestible carbohydrate generally indicate a lower energy intake, whether assessed directly in the short-term or whether assessed from differences in body weight in the mid-term or body weight over 6 years in prospective studies (Livesey G, unpublished). The extent to which this lowering of body weight, usually not more than 2 kg, underemphasises differences in body fat and associated glycaemic
control is unclear; prospective studies usually adjust for differences in energy intake and so the benefits may go unrecognised.

Generally, studies with non-digestible carbohydrates have not replaced available carbohydrate, rather the non-digestible carbohydrate has been supplemental; such study designs would not be expected to lower the glycaemic load except by reducing food intake generally. Detailed studies in animals or epidemiological studies in humans control for energy intake, thus this aspect of non-digestible carbohydrates has not been extensively examined. The full potential of non-digestible carbohydrate in body weight control remains to be examined and exploited.

There are also several pieces of evidence for body weight reduction with the polyol isomalt [21]. Such reduction occurs in a 12-week study in healthy people eating **ad libitum**. In addition, when caloric intake is controlled body weight reduction with isomalt is at least as effective as halving the amount of sucrose or maltodextrin or other carbohydrate it replaces; this being entirely due to the lower energy value of the polyol (2 kcal/g, half that of sucrose). Together these two pieces of information point to the lack of caloric compensation to the reduced energy value from isomalt in the **ad libitum** setting. This is evident too when 5% of starch is replaced with isomalt in rats and mice; this results in reduced food intake, reduced body weight, and improved longevity [21]. Here the mechanisms include ‘spontaneous’ caloric restriction or reduced glycaemia or both.

**Markers of Health**

Consistency between studies with which both fasting blood glucose and glycated protein respond to low-glycaemic carbohydrate diets in diabetics has been noted already above. That is little or no effect occurs when there is already very good glycaemic control while low-glycaemic treatment effects arise to a greater extent with increasing severity of the condition as marked by fasting blood glucose. Similar observations apply to triglycerides; that is greater absolute effects occur in those who have elevated triglyceride concentrations, with no effect when these concentrations are already low. In epidemiological studies, the HDL-cholesterol concentration is generally higher (better) in both men and women who consume low glycaemic carbohydrate diets. Poor association between HDL-cholesterol and GI in population studies arises only when the range of GI values is narrow, or for reasons unexplained the results for one in four quintiles falls away from the general pattern. The strength of the relationship when studies are combined is similar in men and women. HDL-cholesterol outcomes from intervention studies on reduced glycaemic index are mixed; the critical question is whether HDL-cholesterol and other lipid parameters are dependent on the severity of the metabolic perturbations remains to be addressed.

There is little reason to believe observations between studies differ one from another by anything other than severity of the condition or for statistical reason (underpowered study, outlying data, too narrow range of input data). Of course, dietary and other factors may affect the severity of the disease condition. A further cause of variation between studies currently available is that obese individual show greater responses to low glycaemic diets than do normal weight individuals, at least for the development of diabetes, heart disease, elevated triglycerides and elevated high-sensitivity C-reactive protein (chemical pathological markers of increased risk for heart disease) [21].

**Populations**

Combined information about glycaemic load and the development of disease (diabetes and heart disease) from key epidemiological literature shows a statistically significant relationship exists. All the available data appear consistent when plotted together from studies [49,50,63,70,71]. No significant effect may be observed in individual studies such as that of Meyer [70] and is due to both a narrow range of GL at the low end of the spectrum where little happens. The success of the Nurses’ Heath Studies (I and II) and the Men’s Health Professional Study might be attributed a broader range of GL covering importantly higher GL values. Thus, there is nothing odd about the studies from Willett’s laboratories as some have suggested. The totality of evidence at present appears to suggest that GL is of greater importance than GI. Thus after combining studies the significance of GL versus diabetes and heart disease combined is $P<0.001$, whereas the
significance of GI vs these conditions is $P<0.4$. Individual studies may show inconsistent results when comparing GL versus GI, when fat intake may be confounding.

**Glycaemic Load vs Glycaemic Index and Implications for Non-Digestible Carbohydrate**

There is growing evidence that glycaemic load is more significantly related to diabetes and CHD development than is glycaemic index to these conditions (above). This and any underlying reason has yet to be established, though a likely reason is that glycaemic response to foods is determined by several factors not just GI, such as the amounts of carbohydrate consumed, and fat or carbohydrate replaced by protein, fibre and polyols. In addition, plasma triglycerides, hsC-reactive protein, HDL-cholesterol, body weight, and the development of certain cancers appear influenced by glycaemic load more than by glycaemic index [21]. The implication is that reducing glycaemic load by means other that elevating fat intake would be beneficial; this includes the replacement of available carbohydrate with polyols and fibre when this is technologically feasible and desirable, and in addition to other low glycaemic starchy foods.

**OVERALL POTENTIAL**

Potential health benefits of elevated fibre intake have been communicated on several occasions. More recently, these have been emphasised by the Institute of Medicine [15]. Those for polyols have been reviewed recently [20]. Even small reductions in glycaemic load, of the order of 20-30g daily, may have useful impact on CHD development, and theoretically may lower the incidence of this disease by as much as 30%. This is sometimes difficult to appreciate due to our being accustomed to larger differences in 'carbohydrate load' having a smaller impact. The sensitivity to GL appears due to a balance between the benefits of reduced glycaemia from carbohydrate and the benefits of reduced (saturated) fat intake. When protein, polyol, and fibre replace available carbohydrate, the significance of glycaemic load appears to be uncovered. This explains the usefulness also of the inhibitor of carbohydrate digestion acarbose [72-74], which used intensively may also reduce the prevalence of heart disease by as much as thirty percent. While greater reduction following longer periods of treatment is possible, this remains to be established.

**Figure 1.** Glycaemic load and related terms under the glycaemic response concept, with permission [21].

Glycaemic load is the central or common concept. One approach to its derivation is indirectly as the product of *available carbohydrate [75] and †glycaemic index [76], though this is sometimes inaccurate when total carbohydrate is low-glycaemic [21]. Two other approaches originated independently and are the ‡glycaemic glucose equivalent [77,78] and net carbohydrate now measurable directly as a §glycaemic response index or glycaemic load equivalent [79]. The latter two are ostensibly identical (here symbolized by the curved parallel line of equality) and derive from similar direct method(s) of determination. The direct approaches avoid many inherent pitfalls in the indirect approach or calculation method [21,78].
Table 1. Glycaemic and insulinaemic loads from polyols and other unavailable carbohydrates.

<table>
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<th>Load value</th>
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<th>Glycaemic</th>
<th>Insulinaemic</th>
<th>GI b</th>
<th>GL c</th>
<th>GL d</th>
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<td>(g glucose eq. per g product)</td>
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<td>&gt;20</td>
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<td>Intermediate (I)</td>
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<td>&gt;80 to 120</td>
<td>&gt;10-20</td>
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<td>&gt;4 to 10</td>
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<td>Sorbitol</td>
<td>9</td>
<td>11</td>
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<tr>
<td>Mannitol</td>
<td>0</td>
<td>0</td>
<td>V</td>
<td>V</td>
<td>V</td>
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<tr>
<td>Isomalt</td>
<td>9</td>
<td>6</td>
<td>V</td>
<td>V</td>
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</tr>
<tr>
<td>Lactitol</td>
<td>4</td>
<td>4</td>
<td>V</td>
<td>V</td>
<td>V</td>
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</tr>
<tr>
<td>&quot;Fibre&quot; f</td>
<td></td>
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<tr>
<td>Dietary fibre (generally)</td>
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<td>0</td>
<td>V</td>
<td>V</td>
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<tr>
<td>Polydextrose</td>
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<td>-</td>
<td>V</td>
<td>V</td>
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<tr>
<td>Inulin</td>
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<td>V</td>
<td>V</td>
<td>V</td>
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</tr>
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<td>Fibersol-2</td>
<td>10</td>
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<td>V</td>
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</tr>
</tbody>
</table>

a. GI, glycaemic index; GL, glycaemic load
b. Based on total carbohydrate
c. Based on 50 g ingested daily, as in a number of studies
d. Based on a 10g serving size (or exchange rates) noted by Foster-Powell et al [80].
e. Classifications based on www.glycaemicindex.com and Livesey [20].
f. Data combined from Livesey [20], Foster-Powel [80] and personal communications
g. 1, High polymer; 2, low maltitol; 3, intermediate maltitol; 4, high maltitol; from [20].

REFERENCES