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Low glycaemic-index foods

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Accumulating data indicate that a diet characterized by low glycaemic-index (GI) foods not only improves certain metabolic ramifications of insulin resistance, but also reduces insulin resistance *per se*. Epidemiological data also suggest a protective role against development of non-insulin-dependent diabetes mellitus and cardiovascular disease. A major disadvantage in this connection is the shortage of low-GI foods, and many common starchy staple foods, such as bread products, breakfast cereals and potato products, have a high GI. Studies in our laboratory show that it is possible to significantly lower the GI of starchy foods, for example by choice of raw material and/or by optimizing the processing conditions. Such low-GI foods may or may not influence glucose tolerance at a subsequent meal. Consequently, certain low-GI breakfasts capable of maintaining a net increment in blood glucose and insulin at the time of the next meal significantly reduced post-prandial glycaemia and insulinaemia following a standardized lunch meal, whereas others had no 'second-meal' impact. These results imply that certain low-GI foods may be more efficient in modulating metabolism in the long term. Although the literature supports a linear correlation between the GI and insulinaemic index (II) of foods, this is not always the case. Consequently, milk products elicited elevated IIs, indistinguishable from a white bread reference meal, despite GIs in the lower range. This inconsistent behaviour of milk products has not been acknowledged, and potential metabolic consequences remain to be elucidated.

Glycaemic index: Insulinaemic index: Metabolic syndrome: Second-meal effect: Carbohydrates: Starch: Resistant starch: Dietary fibre

The glycaemic index (GI) was introduced by Jenkins and co-workers in the early 1980s, and is a concept for ranking of carbohydrate foods based on their effects on post-prandial glycaemia (Jenkins *et al.* 1981). The GI is defined as the incremental blood glucose area following the test food, expressed as the percentage of the corresponding area following a carbohydrate equivalent load of a reference product. With white bread as reference, GIs range from less than 20 to approximately 120 %. The main cause for these large differences in GI is differences in the rate of digestion or absorption of the carbohydrates, and low-GI foods thus release glucose to the blood at a slower rate. The concept appears to rank foods similarly in diabetic and non-diabetic individuals (Crapo *et al.* 1981), although originally the identification of foods of low-GI character was considered mainly in diabetes. Today there is an important body of evidence to support a therapeutic potential of low-GI diets, not only in diabetes but also in subjects with dyslipidaemia (Jenkins *et al.* 1987*a*). More recent data also support the preventive potential of such a diet against the development of non-insulin-dependent diabetes mellitus (NIDDM) and cardiovascular disease (Salmerón *et al.* 1997*a*, *b*; Frost *et al.* 1999). Consequently the low-GI diet has emerged as an interesting tool in combating diseases linked to the metabolic syndrome.

GI of food products

GIs are now available for a considerable number of carbohydrate foods (Foster-Powell & Miller, 1995). The GIs of some groups of carbohydrate foods – starchy foods, fruit and milk products – are displayed in Table 1. For comparison, GIs of certain low molecular-weight carbohydrates are also included. Although there are traditional indigenous starchy products with a low GI, such as legumes, pasta, some rice, sour-dough bread and bulgur-type products, it is evident that the major sources of carbohydrates in a western diet are found in the upper GI range. That is, most potato products, common bread and breakfast cereals have high GIs, often higher than for sucrose. Although the range in GI

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Table 1. Approximate range in glycaemic index of some starchy foods, fruits and dairy products in comparison with low molecular-weight carbohydrates (glucose, sucrose, lactose, fructose)*

	Starchy foods	Fruits	Dairy products
Glucose, GI = 138			
Potato products	80, 98–120		
Breakfast cereals	74, 96–131		
Common bread	89–131		
Sucrose, GI = 92			
Fruit cocktail, ripe banana, canned apricots, raisins		79–93	
Rice	60–80, 120		
Sour-dough bread	83		
Pasta products	40–70		
Kernel-based bread	35–75		
Lactose, GI = 65			
Apples, oranges, pears, unripe bananas, apple-, orange-, pineapple-juice		45–74	
Milk, yoghurt			15–60
Fructose, GI = 27			
Barley kernels	30–40		
Legumes	12–70		

* Sources: (Wolever, 1990; Granfeldt & Björck, 1991; Liljeberg *et al.* 1992; Tovar *et al.* 1992; Granfeldt *et al.* 1994; Liljeberg & Björck, 1994; Foster-Powell & Miller, 1995).

is most pronounced in the case of starchy products, fruits may also differ. Consequently GI properties are not related to the molecular weight of the carbohydrate component *per se*. In fact, milk products and certain fruits have low GIs despite their content of low molecular-weight carbohydrates. Nor is a high dietary fibre content a prerequisite for low-GI properties, and the naturally occurring levels of viscous fibre in common cereals have only a marginal impact on glycaemia. Wholemeal cereal products thus produce GIs as high as those of white bread. Instead, dietary fibre as part of an intact botanical structure, as in barley and pumpernickel bread, may be effective in reducing glycaemia (Liljeberg & Björck, 1994).

Correlation between GI and II

The physiological relevance of the GI for ranking has been questioned. One point of criticism has been that ranking based on glycaemia does not provide information regarding the insulinogenic effect. In Fig. 1, the GI values have been

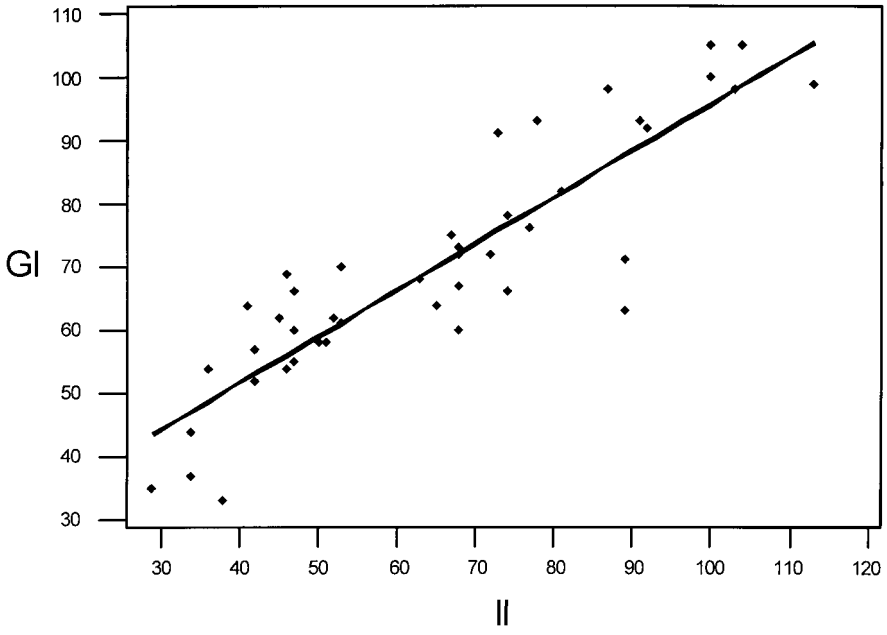

Fig. 1. Correlation between glycaemic index (GI) and insulinemic index (II) for 43 starchy foods. $y = 22{\cdot}1164 + 0{\cdot}736858x$, $r^2 = 78{\cdot}9\,\%$, $P < 0{\cdot}001$. Combined data from Björck *et al.* 1996; Liljeberg & Björck, 1998; Åkerberg *et al.* 1998*a*; Liljeberg *et al.* 1999; V. Skrabanja, H. Liljeberg, I. Kreft and I. Björck, unpublished results; H. Liljeberg and I. Björck, unpublished results.

plotted versus insulinaemic response, expressed as II, for a range of starchy foods in healthy subjects. From our data there appears to be a correlation, high-GI, starchy foods being more insulinogenic. A similar correlation has been obtained for starchy foods and common sugars (Miller *et al.* 1995; Holt *et al.* 1996). Since hyperinsulinaemia itself has been implicated in the development of insulin resistance (DelPrato *et al.* 1994), this correlation supports the relevance of a ranking system based on glycaemia – at least in the case of starchy foods.

Metabolic benefits of a low-GI diet

During the past 10 years an important number of studies have identified a low-GI diet as beneficial in relation to the metabolic syndrome. Several semi-long-term dietary interventions are available for healthy subjects and subjects with metabolic disease. With few exceptions, these studies have shown that a low-GI diet not only improves certain metabolic ramifications of insulin resistance, but also reduces insulin resistance *per se*.

In diabetics, several semi-long-term studies have indicated improved blood glucose control as manifested by lowered day-long glycaemia, lowered HbA1c and improved glucose tolerance (Brand *et al.* 1991). Other metabolic benefits include lowered day-long plasma insulin excursions, and improvements in insulin sensitivity (Järvi *et al.* 1999). A low-GI diet also appears to be a useful adjunct to the management of hyperlipidaemia, as judged from lowered cholesterol and triglyceride levels (Jenkins *et al.* 1985, 1987*a*). A lowering of cholesterol levels has also been observed in healthy subjects (Jenkins *et al.* 1987*b*). In addition to improvements in glucose and lipid metabolism there are indications of improvements in fibrinolytic activity. A low-GI diet was thus found to dramatically lower, and even normalize PAI1 levels in subjects with NIDDM (Järvi *et al.* 1999). This is an important finding, as hyperfibrinogenaemia is common in NIDDM. Concerning the key feature, there are reports of improved insulin sensitivity from direct measurements, not only in diabetics (Wolever *et al.* 1992; Järvi *et al.* 1999), but also in patients with CHD and in healthy subjects (Frost *et al.* 1998). The finding that a low-GI diet reduced fasting insulin levels in parallel to a weight loss in obese women is also noteworthy (Slabber *et al.* 1994) and should be evaluated further.

Preventive potential of a low-GI diet

In two prospective studies, dietary GI was positively associated with risk of NIDDM in men (Salmerón *et al.* 1997*a*) and in women (Salmerón *et al.* 1997*b*), suggesting a preventive role of low-GI diets. A recent study also suggests a significant negative correlation between serum HDL-cholesterol and dietary GI in both men and women (Frost *et al.* 1999). In this study, dietary GI was a stronger predictor of serum HDL-cholesterol than dietary fat intake. There is also evidence that a low-GI diet may reduce the risk of myocardial infarction in women (Liu *et al.* 1998). Taken together, a low-GI diet appears to have not only a therapeutic role, but also a preventive potential.

Shortage of low-GI foods

From the above data it seems relevant to promote increased consumption of low-GI foods. This was done recently by the Joint FAO/WHO expert consultation 'Carbohydrates in Human Nutrition' (FAO/WHO, 1998). However, in order to implement a well-balanced low-GI diet, a much wider range of low-GI products will be required. In particular, whereas there are many options to compose lunch and dinner of low-GI foods, few such alternatives are available among the most common bread and breakfast cereals on the market. The shortage of commercial low-GI products within these food groups seriously limits the utility of the concept. The lack of low-GI products to be ingested at breakfast and evening meal jeopardizes efforts to reduce dietary GI. Moreover, it may be that the GI features of these meals may be particularly important. Consequently, in subjects with type II diabetes it was possible to improve metabolic control simply by exchanging the conventional high-GI breakfast for a low-GI meal (Golay *et al.* 1992). There is also evidence from studies in healthy subjects that a low-GI breakfast meal may have beneficial metabolic effects extending beyond the post-prandial phase. The GI features of the breakfast are thus more crucial for the glycaemic response at lunch than is the GI of the lunch *per se* (Liljeberg *et al.* 1999; see Second-meal effects).

Means of optimizing the GI features of starchy foods

Choice of raw material and processing conditions

Today most of the differences in GI between foods can be explained, and the food factors identified provide a tool that can be used to optimize the GI of food products. Some are related to the choice of raw material, and others to the choice of food process and processing conditions.

With respect to starchy foods, a high degree of crystallinity within the starch substrate will favour a lowered rate of amylolysis, and hence a lower GI. A highly ordered starch structure can be obtained by preserving the starch crystallinity present in native granules, i.e. avoiding gelatinization. In most ready-to-eat food items, the starch crystallinity is generally lost as the commonly applied food processing conditions result in more-or-less complete gelatinization. Flaked cereal muesli-type products represent an exception in this respect. However, despite a degree of gelatinization around 40 %, flaked cereals frequently induce high metabolic responses of glucose and insulin (Granfeldt *et al.* 1995). In order to make use of the slow release properties of ungelatinized starch, an extremely low degree of gelatinization will be required. This cannot be achieved by use of traditional flaking procedures. However, by applying very gentle roasting conditions instead of the more extensive steaming used commercially prior to flaking, it is possible to maintain a high starch crystallinity in the finished flaked product (Y. Granfeldt, A.-C. Eliasson and I. Björck, unpublished results). Such minimally processed wheat flakes were shown to produce a favourably low glycaemia in healthy subjects, similar to that of a raw wheat flake.

Yet another tool to increase starch crystallinity in the product is to promote retrogradation of gelatinized starch, i.e. by use of selected time/temperature cycles

(H. Fredriksson, I. Björck, R. Andersson, H. Liljeberg, J. Silverio, A.-C. Eliasson and P. Åman, unpublished results). High amylose-containing crops are particularly interesting in this respect, in that the retrogradation of the amylose component can easily be obtained under commonly used conditions for food processing, and the selection of cereal genotypes with a high amylose content opens possibilities to significantly lower the glycaemic response. Consequently, maize bread (Arepas) produced from high-amylose maize (70 % amylose, starch basis) displayed an importantly lower post-prandial glycaemia than a corresponding bread product from ordinary maize (25 % amylose) (Granfeldt *et al.* 1995). It is possible that not only retrogradation, but also incomplete gelatinization adds to the favourable properties of high-amylose maize bread. Less is known about the potential of cereal crops containing intermediate amylose levels. Also, high-amylose genotypes of barley are available, containing approximately 45 % amylose. However, barley bread containing 70 % of high-amylose barley maintained a high GI (GI = 99) when processed under conventional baking conditions (Åkerberg *et al.* 1998*a*). Instead, baking of a high-amylose barley bread under so-called pumpernickel baking conditions (120°C, 20 h) resulted in a GI of 71, possibly by providing more favourable conditions for amylose retrogradation.

A highly organized food form, at molecular level as in pasta (Granfeldt & Björck, 1991), or at a tissue level as in leguminous (Tovar *et al.* 1992) and kernel-based products (Liljeberg *et al.* 1992), may also induce an enzymatic barrier and reduce GI.

The presence of certain food components may also have an impact on glycaemic response. Consequently, it is well established that the addition of viscous dietary fibre may reduce glycaemic response to a carbohydrate meal (Braaten *et al.* 1991). In this case, the mechanism at a gastrointestinal level is more related to a reduced motility than to a reduced rate of starch digestion. Within a few years it will be possible to design and select much more consciously the nutritional properties of food ingredients. By including a barley genotype, Prowashonupana, which contains higher levels of viscous dietary fibre (20 %, dry weight basis) than conventional cereals, it is possible to significantly lower the glycaemic impact of bread and porridge products in healthy subjects (Liljeberg *et al.* 1996). Consequently, by exchanging 50 % of conventional wholemeal barley flour for Prowashonupana, the GIs of porridge and flat bread products were lowered by approximately 30 units.

More recently, it has also been established that the presence of certain organic acids, such as those produced upon sourdough fermentation, may reduce glycaemia either by reducing the gastric emptying rate (Liljeberg & Björck, 1996, 1998) or by reducing the rate of starch digestion (Liljeberg *et al.* 1995). This effect of organic acids has opened up new interest in the nutritional potential of food fermentation.

Resistant starch – an accompanying feature of low-GI foods

To conclude, it is not only desirable but also possible to lower the GI of common foods. For most starchy food products a reduction in GI appears to be accompanied by a higher content of resistant starch (RS). Consequently, when plotting RS content versus GI for eleven starchy foods, a high correlation was obtained using a quadratic regression model (Fig. 2). The RS content was analysed according to Åkerberg *et al.* (1998*b*), and includes all major forms: resistant B-type starch, retrograded starch and physically inaccessible starch. The products included were spaghetti, flakes, bread (flour- and kernel-based, regular and high-amylose genotypes), and

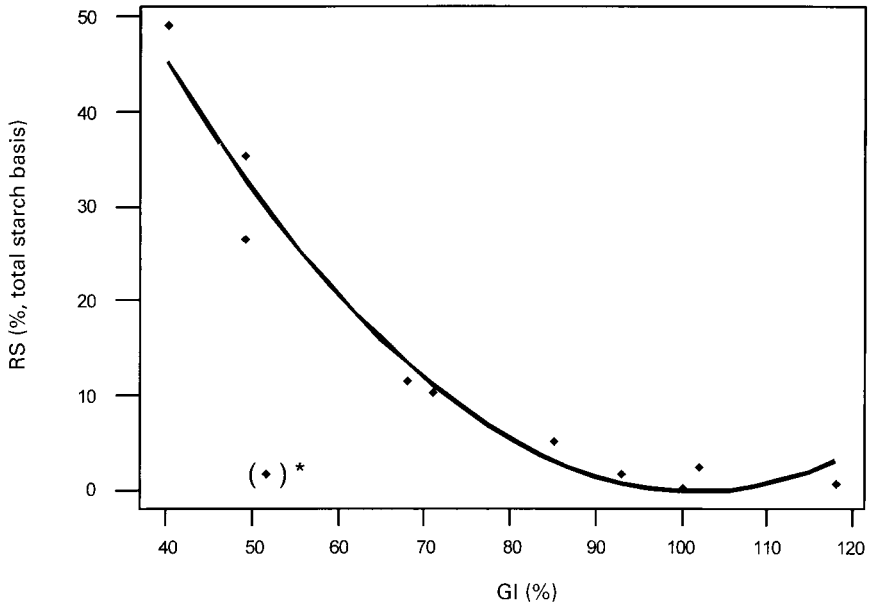

Fig. 2. Correlation between resistant starch (RS) and glycaemic index (GI) for 10 starchy foods. $y = 123{\cdot}536 - 2{\cdot}43393x + 1{\cdot}20^{-2}x^2$, $r^2 = 96{\cdot}8\,\%$, $P < 0.001$.
*, Results for spaghetti not included. Combined data from Granfeldt & Björck, 1991; Liljeberg *et al.* 1992; Liljeberg & Björck, 1994; Granfeldt *et al.* 1995; Åkerberg *et al.* 1998*a, b*; Liljeberg *et al.* 1999; Y. Granfeldt, H. Liljeberg and I. Björck, unpublished results; H. Liljeberg and I. Björck, unpublished results.

mashed potatoes. Food factors that reduce the rate of starch digestion, such as retrogradation of the amylose component or encapsulation within botanical structures, thus seem to render a starch fraction resistant to amylases. As indicated in Fig. 2, the spaghetti product was an exception in that it displayed a low GI and a low RS content. This may imply that the starch interactions responsible for pasta texture reduce the overall rate of starch digestion, but have only a minor impact on RS formation.

A high RS content will add to the total amount of indigestible carbohydrates reaching the colon for fermentation. Resistant starch can thus be expected to contribute to the colonic generation of short-chain fatty acids, with potential beneficial effects on glucose and lipid metabolism (Thorburn *et al.* 1993). It has been suggested that RS is particularly prone to generating butyric acid upon colonic fermentation (Scheppach *et al.* 1988), which may suggest a specific role of RS in the maintenance of a healthy colonic epithelium.

Potential metabolic differences between low-GI foods

When optimizing the GI properties of foods, it is essential to know whether there may also be metabolic differences among low-GI foods. The present paper addresses two issues: firstly, potential differences in so-called second-meal effects, and secondly, the lack of agreement between GI and II that we have identified for one important non-starchy food group.

Second-meal effects

It has been shown that the GI of breakfast may influence glycaemia and insulinaemia at a subsequent standardized lunch in healthy subjects (Jenkins *et al.* 1982). The cause of this second-meal effect is probably that a prolonged absorptive phase following breakfast will favour a more efficient suppression of free fatty acids, thus improving insulin sensitivity at the time of the next meal (Wolever *et al.* 1995). This mechanism has been implicated as partly responsible for the long-term benefits of low-GI foods.

The second-meal effect was studied at lunch in healthy subjects given breakfasts varying in GI features (Liljeberg *et al.* 1999). The subjects were given a white bread breakfast, or three low-GI test breakfasts, ranging in GI from 52 to 64. In the case of a spaghetti and a barley-based breakfast, the lunch produced only 60 or 70 % of the corresponding glycaemic area following the reference breakfast. In contrast, no significant effect was noted at lunch in the case of the breakfast with white bread dressed with vinegar, despite a comparatively low GI. As an indicator of a prolonged absorptive phase, there was a net increment in glycaemia when commencing lunch in the case of these two low-GI breakfasts – but not with the meal with added vinegar. This might suggest that, in addition to low-GI features (measured as commonly for GI determination), the presence of a very late glycaemic response may promote a second-meal effect. Consequently it cannot be excluded that low-GI foods may differ in capacity to induce a second-meal effect.

Inconsistency between GI and II for milk products

Another issue concerns whether all low-GI foods can be expected to induce low insulinaemic responses. As discussed above, it is well established that a ranking of starchy foods based on GI can be expected to run in parallel with a ranking based on II. Until recently, this consistency was believed to be a general entity of carbohydrate foods. In a recent study, the GI and II for milk products were measured in healthy subjects (E. Östman, H. Liljeberg and I. Björck, unpublished results). The test products were regular milk, two types of fermented milk, or a carbohydrate equivalent amount of pure lactose. With white bread as reference, the GIs were very low for the milk products and ranged from 12 to 30. This low range in GI is in accordance with data from the literature. However, the IIs of the milk products were high and similar to that of white bread. This inconsistency has not been acknowledged, and milk products appear to be an exception in that the IIs cannot be predicted from the GIs. The fact that lactose induced a substantially lower II than the milk products indicates that some other milk component adds to the insulin response. An insulinotrophic effect of milk and fruit juices has been reported previously from studies in type II diabetic subjects (Gannon *et al.* 1986; Bucalossi *et al.* 1990), but with food products having a very different gross composition, making interpretation regarding mechanism difficult.

The insulinogenic features of milk observed in healthy subjects in the present study is likely to increase insulinaemia from mixed meals. Accordingly, we have observed that inclusion of milk (150 ml) with a barley-based breakfast increased the II of the meal by approximately 20 units (Liljeberg *et al.* 1996). The potential metabolic consequences of this insulinotrophic capacity of milk need to be elucidated.

Conclusions

There is an important body of evidence in support of a therapeutic potential of a low-GI diet in subjects with NIDDM and dyslipidaemia. There are also indications of a preventive role against NIDDM and cardiovascular disease. However, to fully exploit the metabolic potential of a low GI, a wider range of low-GI foods is necessary. The shortage of low-GI alternatives is particularly pronounced among bread and breakfast cereals. The technological means exist to provide such foods, and the development of low-GI products is a challenge for the food industry.

Low-GI foods appear to differ in second-meal effects and, in the case of milk, in insulinogenic properties. The insulinotrophic features of milk need to be acknowledged, and the potential metabolic effects in a mixed diet evaluated.

Sometimes even modest modifications of current food processes may significantly reduce GI. However, as yet few commercial low-GI/high-RS food products have appeared, and the introduction of such products on the market will probably await demand from health professionals involved in the treatment and prevention of diabetes and related disorders.

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