Glycaemic index effects on fuel partitioning in humans

E. O. Díaz, J. E. Galgani and C. A. Aguirre

Laboratory of Energy Metabolism and Stable Isotopes, Institute of Nutrition and Food Technology (INTA), University of Chile, Macul, Santiago, Chile

Address for correspondence: Dr EO Díaz-Bustos, Laboratory of Energy Metabolism and Stable Isotopes, Institute of Nutrition and Food Technology (INTA), University of Chile, Ave El Líbano 5524, Macul, Santiago, Chile. E-mail: ediaz@inta.cl

Summary

The purpose of this review was to examine the role of glycaemic index in fuel partitioning and body composition with emphasis on fat oxidation/storage in humans. This relationship is based on the hypothesis postulating that a higher serum glucose and insulin response induced by high-glycaemic carbohydrates promotes lower fat oxidation and higher fat storage in comparison with low-glycaemic carbohydrates. Thus, high-glycaemic index meals could contribute to the maintenance of excess weight in obese individuals and/or predispose obesity-prone subjects to weight gain. Several studies comparing the effects of meals with contrasting glycaemic carbohydrates for hours, days or weeks have failed to demonstrate any differential effect on fuel partitioning when either substrate oxidation or body composition measurements were performed. Apparently, the glycaemic index-induced serum insulin differences are not sufficient in magnitude and/or duration to modify fuel oxidation.

Keywords: Body composition, fuel oxidation, glycaemic index, obesity.

Introduction

Dietary carbohydrates provide the most important source of energy in most parts of the world, representing between 45 and 60% of total energy consumed in the population (1). Carbohydrates have traditionally been classified as simple sugars and complex carbohydrates. Glucose, fructose, lactose and sucrose are the main simple sugars, whereas the main complex carbohydrate in the human diet is starch. An intense discussion on the adequate proportion of simple vs. complex carbohydrates in the diet was held recently. The 1998 FAO (Food and Agriculture Organization of the United Nations) Expert Committee (1) recommended classifying carbohydrates according to their glycaemic effect.

Based on serum insulin response modulation by glycaemic index (GI), several studies have focused on the effect of GI on fuel partitioning and obesity (2,3). Differences in serum insulin response could produce different insulin action on body tissues, which would accelerate glucose uptake and oxidation and at the same time stimulate fat storage. The aim of this review was to analyse published research evaluating the role of GI on fuel partitioning under resting conditions with particular emphasis on the likely effect of high-glycaemic carbohydrates on fat storage and obesity.

GI is defined as the increase in glycemia caused by the ingestion of 50 g of available carbohydrates relative to an equal amount of carbohydrates from glucose or white bread (4). The capacity of different carbohydrates to modify serum glucose concentrations (5,6), depends on their molecular structure, presence of other macronutrients or fibre in the same food, food processing, cooking, food storage and ripeness (7). The use of this concept has permitted the classification of carbohydrates in a more physiologic manner based on their impact on serum glucose concentration. Using this approach, a large number of studies, summarized in several reviews (2,3,8,9,10) have assessed the effect of GI on several outcomes such as β-cell function, serum triacylglycerol concentrations, serum non-esterified fatty acids (NEFA) levels, glycaemic control, food intake regulation and body weight.

GI largely depends on the rate at which the carbohydrate is digested in the small intestine. The digestion process determines the rate of glucose entry from the gut into the

bloodstream (8) and the rate of glucose disposal. In general, quickly digested and absorbed carbohydrates will produce a higher increase in postprandial glycemia. The higher glycemia will induce an increase in pancreatic insulin secretion promoting glucose uptake to counteract the rise in serum glucose, sometimes leading to a reactive hypoglycaemia. The higher serum insulin response induced by high-glycaemic carbohydrates has led to the hypothesis that its ingestion will cause a higher glucose uptake and oxidation, with a subsequent reduction in fat oxidation (3). This review will address if GI is a determining factor in fuel partitioning.

Glycaemic index effects on fuel partitioning

Short-term studies

Ritz *et al.* (11) evaluated fuel oxidation after the ingestion of 50 g of carbohydrates from glucose and manioc starch in non-obese healthy subjects for 6 h using a crossover design. Expected changes in serum glucose and insulin concentration were observed, with a GI for starch in relation to glucose of 57% and an insulinemic index of 68%. Suppression of serum NEFA concentration was similar for both carbohydrates between fasting and 180 min. Thereafter, serum NEFA concentration increased, returning to near fasting levels at 300 min for starch, whereas with glucose, serum NEFA concentration increased to almost twice the fasting value. In the 6-h period there were no differences in total energy expenditure or diet-induced thermogenesis. Cumulative carbohydrate oxidation was higher for the starch load ($P = 0.0002$); this difference was exclusively explained by an increased carbohydrate oxidation after 200 min. Cumulative fat oxidation was not different between carbohydrate loads. However, a decreased fat oxidation for starch from 220 min to the end of the test was reported.

Korach-André *et al.* (12) evaluated non-protein respiratory quotient after ingestion of parboiled and polished rice in healthy individuals. Consistent with previous data on starch digestion rate, the authors stated that parboiled rice is less susceptible to digestion than polished rice. Using a crossover design, fasting subjects received a large amount of starch from rice [~270 g; 5 g (dry mass)/kg (body mass)]. Both rices were intrinsically and artificially enriched in ^{13}C. A priming dose of ^{2}H-glucose was infused to assess plasma glucose appearance and liver glucose production. Non-protein respiratory quotient was higher after ingestion of parboiled rice than after ingestion of polished rice during the whole period ($P < 0.05$). A decreased cumulative fat oxidation for parboiled rice was observed ($P < 0.05$), which was maintained throughout the entire period. No differences for carbohydrate oxidation were reported. Total energy expenditure data at fasting and 8 h-postprandial period were not reported, but increased energy expenditure after ingestion of polished rice might be inferred, because carbohydrate and protein oxidation were similar and fat oxidation was higher. Calculations of diet-induced thermogenesis were not available. No differences in the rates of plasma glucose appearance, liver glucose production and serum glucose, insulin, NEFA and lactate concentrations were found during the whole period. The authors attributed this unexpected result to the large dose of ingested carbohydrate (~270 g).

Mid-term studies

Díaz *et al.* (13) evaluated the effect of high- and low-glycaemic breakfast and lunch on fuel oxidation in obese women by a crossover design. Serum glucose, NEFA, insulin and plasma glucagon responses were only evaluated after breakfast for 5 h. Non-protein respiratory quotient was measured in a respiratory chamber after breakfast and lunch. Expected differences for serum glucose and insulin response were observed (Fig. 1). There were no differences

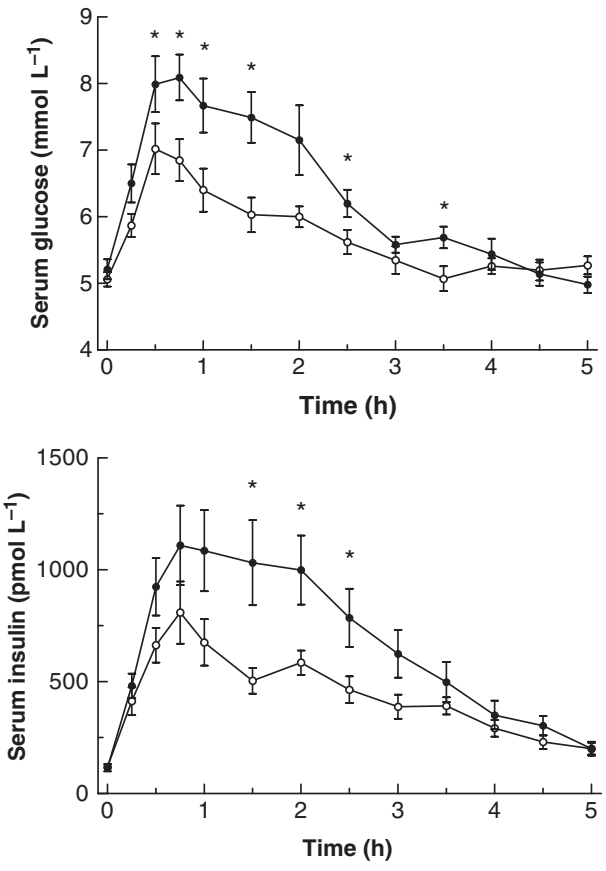

Figure 1 Serum glucose and insulin concentration after the ingestion of a low-GI (○) or high-GI (●) breakfast in obese women. Data are mean ± SE. Statistical analysis by paired *t*-test. *$P < 0.05$. GI, glycaemic index.

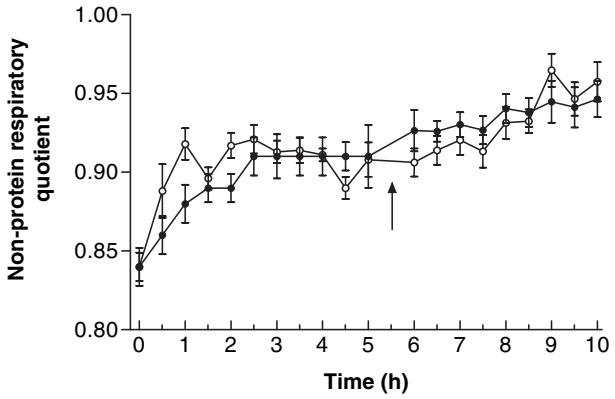

Figure 2 Non-protein respiratory quotient after the ingestion of a low-GI (○) or high-GI (●) breakfast and lunch in obese women. Data are mean ± SE. Statistical analysis by paired *t*-test. Arrow indicates lunch time. GI, glycaemic index.

in the serum NEFA and plasma glucagon profiles between the two types of glycaemic breakfasts. Similarly, no differences between the postprandial non-protein respiratory quotient after breakfast and lunch were observed (Fig. 2).

Sparti *et al.* (14) studied the effect of two different diets containing carbohydrates with high or low *in vitro* digestion rate on 24-h fuel oxidation in lean subjects using a crossover design. Serum glucose response was not evaluated in this study. Energy expenditure and energy and macronutrient balance over the 24 h were not affected by the diets. However, after lunch and dinner carbohydrate oxidation was increased for the highly digestible carbohydrate diet ($P < 0.05$). This profile was reversed during the night ($P < 0.05$).

Würsch *et al.* (15) tested the effects of two isoenergetic breakfasts containing 57 g of carbohydrates (49% of calories) from potato (high GI) or bean (low GI) flakes in healthy men. Both breakfasts were prepared by adding whole-fat milk and butter and supplied for 7 days in a crossover design with 1-week washout period. Postprandial effects were measured on days 1 and 7 for 6 h. Serum glucose area under the curve was higher for potato breakfast at 1 and 2 h ($P < 0.05$), but not at 6 h. Serum insulin area under the curve was higher for potato during the whole 6-h period. Cumulative 6-h energy expenditure was 3% (~20 kcal) higher for the potato breakfast ($P < 0.002$). Cumulative 6-h substrate oxidation was similar in both test meals; however, during the early postprandial period for the potato meal (between 60 and 90 min) an increased carbohydrate oxidation for potato was observed ($P < 0.05$). After the same meal, decreased fat oxidation at 90 min ($P < 0.01$), 150, 180 and 210 min ($P < 0.05$) was observed. No significant differences were found between days 1 and 7 for each experimental breakfast.

Long-term studies

Studies with a longer duration are valuable because dietary factors affecting fuel partitioning may go undetected in the short term, but may be expressed in the long term through changes in body composition. The main disadvantage with this type of study is that dietary control under free-living conditions is difficult and extremely demanding, and as such, compromises results reliability.

Using a crossover design, Howe *et al.* (16) studied the effect on 24-h fuel oxidation of diets providing 55% of energy as starch with 70% as amylose (low-glycaemic response) or 70% as amylopectin (high-glycaemic response). The study was conducted with subjects with normal or exacerbated serum insulin response to an oral glucose tolerance test. The study design included 10 weeks of replacement of customary starchy foods with one of the experimental starches followed by 4 weeks during which the diets were cooked and weighed at the research unit, and were consumed in this same place and in the home. A washout of 2 weeks in between dietary periods was included. At the end of each experimental period, two 24-h indirect calorimetry measurements were performed. For one measurement subjects were tested during energy balance and in the other, under energy excess conditions (1.25-fold total energy expenditure). On each test day, the same starch foods were consumed consistent with the experimental period.

Total energy expenditure and sleep energy expenditure were not affected by diet or insulinemic subject condition. Similarly, 24-h respiratory quotient under energy excess and energy balance conditions was not modified by diet or subject type. Data about temporal fat and carbohydrate oxidation profile were not reported. Protein oxidation increased only for the amylopectin-enriched diet in both subject types after energy excess compared with energy balance. However, it was not indicated if protein oxidation under energy balance or energy excess conditions differed as a function of the diet type consumed.

The concern regarding this study is that serum glucose and insulin response were measured 1 week before fuel oxidation measurements, and only for the isolated experimental starch instead of for the whole meal.

Kiens *et al.* (17) studied the effect of two isoenergetic diets containing high- or low-GI carbohydrates. These diets were provided for 30 days in lean subjects using a crossover design. The outcomes were muscle glycogen and muscle triacylglycerol concentrations, which were measured before and after both dietary treatments. In addition, insulin sensitivity using an euglycaemic clamp at two serum insulin levels (370 and 2400 pmol L^{-1}) was assessed. The authors found that body weight was unaltered after either dietary period, which implies that observed changes were not linked to differences in energy balance. At the end of the

low-GI period a decrease in muscle glycogen concentration by 12–13% was observed in comparison with the measurement before low-GI diet and the end of the high-GI period ($P < 0.05$). No effect on muscle triacylglycerol concentration with either GI diet for the initial vs. final comparison was observed. On the other hand, the authors reported a 22% ($P < 0.05$) reduction in muscle triacylglycerol concentration when comparing the values at the end of both dietary periods. However, this change was comparable with the modification seen after the 3-week washout period (on average 23%), where no dietary modification was introduced. No statistical analysis was available for this comparison.

A factor to be considered is related to the determination of human muscle triacylglycerol concentration, which has a variability of ~24% (18) as determined by repeated human muscle measurements at rest. Thus, Wendling *et al.* proposed that a change higher than this value should be observed in order to be considered meaningful (18).

With regards to insulin sensitivity, there were no differences between the high- and low-GI diets at the low insulin infusion rate. On the other hand, at the high insulin infusion rate, lower insulin sensitivity after the low-GI diet was reported. The relevance of this finding for supraphysiological serum insulin concentration remains to be elucidated.

Bouché *et al.* (19) evaluated the effect of a high- and low-GI diet on body composition in overweight men for 5 weeks using a crossover design. Body composition was assessed using dual-energy X-ray (DEXA) before and after each dietary period. A body fat mass reduction of 2.7% (0.52 kg) after the low-GI dietary period was observed. This change was not associated with dietary intake or body weight modifications. This finding requires additional confirmation, because detected difference was close to the DEXA measurement error (20).

In a longer study, Sloth *et al.* (21) evaluated the effect of diets containing starches with contrasting *in vitro* digestion rate for 10 weeks in overweight women using a parallel design. Before, during and after each diet, body weight, body composition (using DEXA) and insulin sensitivity (using HOMA: Homostatic model assessment) were assessed. No significant differences between diets were found for the above outcomes.

Glycaemic index effects on body weight

A role for GI in body weight regulation has been attributed to direct anabolic effects of insulin or indirectly through modifications in appetite resulting in a higher energy intake in response to a higher-GI diet.

High fasting serum insulin concentration (22) or high first-phase serum insulin response to intravenous glucose (23) have been proposed as risk factors for weight gain. This may have lead Ludwig to state that the 'functional hyperinsulinemia associated with high-GI diets may promote weight gain by preferentially directing nutrients away from oxidation in muscle and towards storage in fat (24)'. Evidence for this hypothesis is still lacking since no effects of GI on fuel partitioning have been demonstrated to date. Even if different serum insulin concentrations were able to differentially modify fuel partitioning, this does not imply a change in energy balance (discussed below) capable of explaining body weight changes. Indeed, in some studies, subjects classified by their fasting serum insulin levels, did not gain (25) or lose (26) body weight to a different extent. Many studies advocating the effects of low-GI diets on body weight have not considered that these diets are rich in other dietary components such as fibre, non-digestible carbohydrates, low-fat or with lower energy density (27–29). Such studies are not included in this review.

With regards to energy expenditure, differences in the thermogenic effect induced by different carbohydrate types could be observed. Some studies comparing glucose vs. starch have not detected differences in postprandial energy expenditure or thermogenesis (11,30). On the other hand, some studies did not find differences in energy expenditure when mixed glycaemic meals were compared (13,14,16), while in other studies, an increased energy expenditure after a high-glycaemic breakfast was detected (15).

In relation to energy intake, GI has been associated with food intake regulation, but considering that a similar number of studies verify and refute GI effect (28,31), available evidence is not conclusive.

Some of the studies that support the effect of a low-GI diet on body weight were undertaken by Slabber *et al.* (32) and Spieth *et al.* (33). Their conclusions, however, need to be reconsidered in several respects.

Slabber *et al.* (32) provided two energy-restricted diets to two hyperinsulinemic groups of obese women for 12 weeks to test the effect of GI on body weight loss. The two diets were a conventionally balanced diet and a diet based on selected low-GI foods designed to elicit a low serum insulin concentration response. Once the 12-week period was finished, a subsample from each group was chosen to follow a crossover study. Thus, after a 12-week washout period, the contrasting diet was given for another 12 weeks. The results of the non-crossover study showed similar weight loss for both groups (low-insulinemic response diet -9.3 ± 2.5 kg; conventional diet: -7.4 ± 4.2 kg, mean ± SD, $P = 0.14$). In the crossover study, however, a significantly higher weight loss was found after the low-insulinemic response diet (–7.4 vs. –4.5 kg, SD not given; $P = 0.04$). From this part of the study, the authors concluded that an energy-restricted, low-insulinemic response diet induced higher weight loss compared with a conventional energy-restricted diet.

This conclusion is not necessarily correct due to the lack of some critical information. For instance (i) glycaemic and/

or insulinemic responses of tests diets can not be ascertained because they were not assessed; (ii) body weight at the beginning of the crossover study was not provided (only the average change in body weight was given) therefore selection of subjects might have been biased because of greater or lower weight losses in the first part of the study; (iii) energy intake during the crossover study was not mentioned and finally; (iv) subsample characteristics were not given. The lack of data on food intake, energy expenditure and body weight during each dietary period, limits the confidence of any energy balance estimate, the latter being essential to ascribe the changes in body weight to a certain diet.

In a less controlled and parallel design, Spieth *et al.* (33) prescribed a low-fat, reduced-energy diet or a low-GI, unrestricted diet to two groups of obese children. Subjects in the two groups were followed on average for 4.3 months. Additionally, some subjects were given behavioural therapy. At the end of the dietary period, weight loss was recorded and adjusted by age, sex, ethnicity, duration of follow-up, behavioural therapy and baseline body mass index. The authors needed to perform this adjustment either because all these variables were not considered in the group separation criteria or, because they were not provided to all subjects (e.g. behavioural treatment). The main conclusion was a higher adjusted weight loss after the low-GI diet vs. the low-fat diet. The following problems were mentioned by the authors: lack of random order in the diet prescription, no dietary intake monitoring, differences in macronutrient intake provided by the two diets, heterogeneity of study subjects and selection bias. All these flaws lead the authors to conclude that the study results must be considered as suggestive but not strong enough to establish the health benefits of low-GI diets.

Finally, as indicated by different studies quoted above (16,17,19,21) no effect of GI on body weight was evident. This question has been reviewed by Saris (29).

Insulin and fat oxidation relationship

Considering the literature reviewed above, in general, no differential effect on fuel partitioning is observed when meals with contrasting GI are supplied. The lack of a relationship between serum insulin concentration and fat oxidation suppression may be explained by: (i) the magnitude of the difference in serum insulin concentration induced by high- vs. low-glycaemic carbohydrates; and (ii) duration at which this difference in GI-induced postprandial serum insulin response is maintained.

Concerning the magnitude of the difference in serum insulin response, a critical component for testing the GI effects will be to quantify the difference on serum insulin responses induced by the different types of dietary carbohydrates. In several published studies, the ratio between serum insulin area under the curve for the high- vs. low-glycaemic meals on average ranged from 0.8 to 1.9. Some of these studies have measured fuel oxidation (11–13,15,16,30), whereas others have not (17,19,34, 35,36,37). From our data (13), the ratio for serum insulin area under the curve for the high- vs. low-GI breakfasts was at the highest level (on average 1.81). As indicated by the studies discussed above, the GI-induced difference in serum insulin response has been accompanied by negligible changes in fuel oxidation when mixed meals have been supplied.

With regard to duration over which the difference in serum insulin concentration is maintained, it usually peaks in a relatively short time although its effects may prevail much longer via effects on gene expression of metabolically relevant proteins. Usually, serum insulin peak concentration is reached 45 min post dose or earlier, as can be observed in Fig. 1. The difference in serum insulin concentration after the ingestion of glycaemic carbohydrates is maintained for less than 45 min. Therefore, some effect on fuel partitioning will be accounted for by serum insulin differences in this time period.

From our data (13), the maximal GI-mediated serum insulin concentration difference occurred between 1 and 2 h after glycaemic breakfast. For the high- and low-GI breakfasts, serum insulin concentration during this period reached about 1000 and 600 pmol L^{-1}, respectively. This difference in serum insulin concentration was about 200–500 pmol L^{-1} ($P < 0.05$). When substrate oxidation at the end of this period of maximal serum insulin concentration difference was determined, no difference in the non-protein respiratory quotient was detected (Fig 2. P = NS).

Another factor to be considered when examining the effect of GI on fuel partitioning is related to the difference in serum insulin response for a subsequent glycaemic meal. Some studies have supplied contrasting glycaemic meals at breakfast, then, at lunch a standard meal was provided. From these studies, an improvement in glucose tolerance accompanied by lower serum insulin response after a low-GI breakfast was suggested (38,39). On the other hand, using a similar design, Wolever *et al.* (34) supplied isocaloric breakfasts with different carbohydrate content (high and low) and GI (high and low), which were followed by a standard lunch. In general, no differential effects of GI after lunch were found except for an increased serum glucose and insulin response when the low-carbohydrate, low-GI breakfast was given.

Studies testing the postmeal effects of contrasting glycaemic breakfasts and lunches have found different results. Brynes *et al.* (40) evaluated the effect of a high- vs. low-GI breakfast and lunch after day 1 and after 24 days of dietary intervention. All meals had similar energy and macronutrient content. Furthermore, GI values were similar for break-

fast and lunch in each glycaemic category. Both meals induced similar differences in serum insulin response after breakfast and lunch. However, the opposite has also been reported; in one study, serum glucose response between contrasting glycaemic meals differed only at breakfast with no differences after lunch (41). Perhaps a similar situation could be expected for serum insulin response. The latter might be another explanation for the null effect of GI on fuel oxidation after lunch, as shown in Fig. 2.

Taking into account all the above arguments, we speculate that under postprandial conditions, GI-induced serum insulin differences are not sufficient in magnitude and/or duration to modify fat oxidation.

In contrast with human studies, animal studies comparing effects of different glycaemic meals tended to show the expected response. Accordingly, rats fed high-GI starch (high amylopectin) when compared with rats fed isocaloric diets containing low-GI starch (high-amylose) for 3–7 weeks have shown a higher fat storage (42) accompanied by increased lipogenic enzymes expression (43). This implies that under similar macronutrient composition and energy content, GI modified fuel partitioning sufficiently to change body composition. Most recently, another study on weight-stable animals for 18 weeks confirmed these findings. Rats and obesity-prone mice treated with a high-GI diet had almost twice the amount of fat stored than those on the low-GI diet (44).

Studies using oral fructose

Studies using fructose as a 'low-GI carbohydrate' (45) are considered separately since fructose metabolism differs from starch-based low-GI carbohydrates.

Fructose bypasses the first rate-limiting enzymes of glycolysis (46), and thus, fructose is expected to be more readily oxidized, and as such directs fatty acids away from oxidation. This leads to lower fat oxidation after the fructose dose as compared with the glucose dose (30), which is in contrast with the postulated hypothesis on the effect of low-glycaemic carbohydrates on fat oxidation. On the other hand, the differential effect of fructose on fat oxidation tends to disappear when fructose is not provided as the only source of energy but instead combined with other foods in the same meal (47).

To further elucidate, Blaak *et al*. (30) provided four different carbohydrates as: 75 g of glucose, sucrose, fructose and corn starch to be ingested. Different profiles of serum glucose and insulin were observed postprandially. Serum NEFA concentration, however, had similar suppression levels for all carbohydrates types between fasting and about 180 min post dose, rising more with glucose after this time period as compared with the other carbohydrates. Carbohydrate oxidation was higher after fructose and sucrose, whereas fat oxidation was higher after glucose and starch.

Delarue *et al*. (48) studied the effect of fish oil supplementation on substrate oxidation after ingestion of glucose and fructose in normal subjects. This study did not aim to evaluate differences between carbohydrate types; therefore, statistical analysis was not available. However, the results observed are of interest in the context of this review. The authors provided an oral dose of glucose and fructose on two separate days. The expected profiles of serum glucose and insulin were observed, consistent with the type of carbohydrate. Carbohydrate oxidation was, on average, 25% higher for fructose than for the glucose test. In contrast, fat oxidation was, on average, 35% lower with fructose as compared with glucose. Although statistical analyses were not available, these results had the same trend to the above study (30).

In a tightly controlled study by McDevitt *et al*. (47), normal-weight and obese subjects were overfed for 96 h while remaining confined in a respiratory chamber. Energy intake corresponded to 1.5-fold of their measured energy requirements. The energy excess was given as glucose, sucrose or fructose, which was approximately 127 g of carbohydrate. The same protocol of activities was performed at fixed times on each occasion. Their results showed no differences in fat and carbohydrate oxidation; similarly, there were no differences between treatments in fat or carbohydrate balance during the entire study period. Blood sample measurements were not performed in this study.

The above information shows that fructose should not be considered a low-glycaemic carbohydrate, at least when discussing fuel metabolism assessment. On the other hand, its differential effect on serum insulin is explained by other mechanisms, which are different to those mechanisms postulated for starch.

Conclusions

This review examined the effect of GI on fuel partitioning (Table 1). Short-, medium- and long-term studies failed to demonstrate that meals or diets of contrasting GI have significant effects on carbohydrate and fat oxidation and body composition. It is possible that no effects are observed because the GI-induced serum insulin differences are not sufficient in magnitude and/or duration to modify fuel oxidation.

An exception was observed with isolated oral fructose vs. glucose, in which case a lower fat oxidation was observed with the former. These results are likely explained by the differential fructose hepatic oxidation rate.

In conclusion, the hypothetical beneficial effects of low-GI diets on fuel partitioning need further support.

Conflict of Interest Statement

No conflict of interest was declared.

Table 1 Effects of glycaemic index (GI) on fuel oxidation and body composition (controlled studies)

Study	Subjects	Study design	Variables	Outcome
Ritz *et al.* (11)	6 healthy, lean	50 g glucose (high GI) or manioc starch (low GI). Random, crossover	6 h-cumulative fuel oxidation	No change
Korach-André *et al.* (12)	8 healthy, lean	270 g parboiled (low GI) or polished (high GI) rice. Random, crossover	8 h-cumulative fuel oxidation	No change on carbohydrate oxidation Lower fat oxidation with parboiled rice*
Díaz *et al.* (13)	12 healthy, obese	Breakfast (112 g carbohydrate) and lunch (163 g carbohydrate) of high and low GI. Random, crossover	10 h-cumulative fuel oxidation	No change
Sparti *et al.* (14)	14 healthy, lean	Meals with carbohydrates of high and low *in vitro* digestion rate. Random, crossover	24 h-cumulative fuel oxidation	No change
Würsch *et al.* (15)	6 healthy, lean	57 g carbohydrate from potatoes (high GI) and beans (low GI). Random, crossover. Intervention for 7 days	6 h-cumulative fuel oxidation	No change
Howe *et al.* (16)	9 healthy and 13 hyperinsulinemic	Enriched-carbohydrate diets in amylose (low GI) or amylopectin (high GI). Random, crossover. Intervention for 14 weeks	24 h-cumulative fuel oxidation	No change
Kiens *et al.* (17)	7 healthy, lean	High- and low-GI diets. Random, crossover. Intervention for 30 days	Muscle TG and glycogen content. IS† at 2 insulin doses	No TG changes. Lower glycogen and IS with low-GI diet (high insulin dose)*
Bouché *et al.* (19)	15 overweight	High- and low-GI diets. Random, crossover. Intervention for 5 weeks	Body fat	Decreased with low-GI diet*
Sloth *et al.* (21)	23 and 22 overweight	Replacements of usual foods with carbohydrates of high and low *in vitro* digestion rate. Random, parallel. Intervention for 10 weeks	Body fat	No change

*$P < 0.05$.
†Insulin sensitivity by euglycaemic-hyperinsulinemic clamp.
TG, triacylglycerols.

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