Chapter 19
Treatments with Low Glycaemic Index Diets in Gestational Diabetes

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Key Points

- Gestational Diabetes Mellitus (GDM) is the carbohydrate intolerance that results from maternal inability to cope with increased insulin resistance associated with pregnancy.
- GDM management aims to achieve glycaemic control and promote adequate weight gain in the mother and also improve foetal outcomes.
- Diet is the cornerstone of GDM management.
- Low Glycaemic Index (GI) or glycaemic load (GL) diets by preventing postprandial glycaemic and insulinaemic peaks, attenuate cardiovascular risks; especially in subjects with obesity, insulin resistance or hyperinsulinaemia.
- Low-GI diets are beneficial only when they comply with current dietary guidelines and therefore require appropriate dietetic supervision.
- GDM subjects on low-GI diets have lower spikes in post-meal glycaemia and are less likely to require the initiation of insulin therapy when compared to those receiving standard diets with higher GI.
- Low-GI diets in GDM may also reduce central adiposity in the foetus.
- Low-GI diets are also likely to benefit GDM women in managing their glycaemia and body weight post-delivery.
- Current evidence raises no safety issues in using low-GI/GL diets in GDM management.
- However, further evidence is required to lend unequivocal support for the benefit of low-GI/GL diets in GDM treatment.

Keywords Gestational diabetes mellitus · Diet · Glycaemic index · Glycaemic load · Pregnancy

Abbreviations

GDM	Gestational diabetes mellitus
GI	Glycaemic index
GL	Glycaemic load
T2DM	Type 2 diabetes mellitus

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RCT Randomised-controlled trial
CVD Cardiovascular disease
SCFA Short-chain fatty acids

Introduction

Gestational Diabetes mellitus (GDM) is defined as the 'glucose intolerance first recognised during pregnancy' [1]. All pregnancies are accompanied by metabolic changes that promote adipose tissue accumulation in early gestation, followed by an increase in insulin resistance to provide adequate nourishment to the foetus [2]. The insulin resistance is accompanied by increased pancreatic insulin secretion to maintain maternal euglycaemia as the pregnancy progresses [2]. Hyperglycaemia results when the maternal insulin secretion is unable to meet the increased insulin demand [1, 3]. Therefore, the pathophysiology of GDM is similar to that of type 2 diabetes mellitus (T2DM); namely, marked insulin resistance and impairment of insulin secretion [4] and associated dyslipidaemia [5]. Thus management of postprandial glycaemia and insulin demand are essential targets for GDM management.

Diet is the cornerstone of GDM management [1, 6]. Dietary management for GDM has the following maternal goals: achieving glycaemic control, ensuring adequate weight gain and appropriate nutritional status. Achieving these goals ensures maternal and foetal health. More intensive medical management and increased surveillance are instituted in women who fail to respond adequately to diet therapy and increases treatment costs [7]. Most importantly, GDM increases long-term health risks for the mother and her offspring [1, 7] posing greater demand on healthcare resources.

Carbohydrates predominantly influence postprandial glycaemic response [8]. Therefore, carbohydrate restriction has historically been the prime focus of dietary management for GDM [1]. Restricting carbohydrates to provide around 45% of the energy is safe in GDM pregnancies [9], though evidence from randomised-controlled trials (RCTs) support the use of diets with reasonably high amount of complex carbohydrates [1]. Low-carbohydrate diets that are high in protein and fat intake, may increase risk for diabetes specifically among pregnant women [6] and can compromise foetal outcomes [7]. In the absence of concrete evidence to favour any particular diet, consensus panels for GDM have no specific recommendation but encourage the adoption of conventional healthy diets [1, 5].

As the role for low-carbohydrate diets is limited by their health concerns, the effect of carbohydrate quality (type) on glucose metabolism and insulin resistance has gathered interest [10]. Emerging evidence suggests that deterioration of glucose homeostasis can be prevented by monitoring both carbohydrate quantity and quality [10]. Concepts of glycaemic index (GI) and glycaemic load (GL) were born out of this need to describe the quality or type of carbohydrate foods.

This review aims to assess the current evidence for the treatment of GDM with low-GI/GL diets. The objective of this review is in-line with the professional societies' repeated calls for the consolidation of current evidence and efforts to bridge the knowledge deficits in this area to identify optimal diets for GDM women [5, 7, 11]. This is especially important because GDM affects a significant proportion of pregnant women globally, and alarmingly its prevalence is increasing [11].

Glycaemic Index (GI)

Carbohydrate foods even when consumed in equal amounts differ in their glycaemic effect. Hence physiological effects of carbohydrates are better described by their in vivo ability to raise blood glucose [12]. GI is such a physiological classification of carbohydrates [12], that ranks them on a scale of 0–100, in accordance to their postprandial glycaemic effect [13]. GI, therefore, reflects the rate of conversion of a carbohydrate into glucose [13]. Higher the GI value of a food, greater the postprandial glycaemic response it elicits [14] (Fig. 19.1).

The GI of a food is measured as 'the incremental area under the blood glucose response curve of a 50 g available carbohydrate portion of the food expressed as a percentage of the response after 50 g of glucose taken by the same subject' [14]. To simplify interpretation, foods are often classified into three categories based on their GI: high (GI >70), intermediate (GI between 55 and 70) and low (GI <55) [15].

Factors Affecting Glycaemic Index

The differences in GI of food depend on the type of sugar and or starch it contains [16], the extent of processing it has undergone [15] and the presence of factors that determine the rate of carbohydrate absorption [16]. Low-GI recommendations utilise these determinants to lower postprandial glucose responses.

Foods with a high content of fructose (fruits), and galactose (milk products) provide lower amounts of absorbable glucose, and thus have lower GI [16]. Beans and seeds have fibrous coats that slow down the access of enzymes to the starch inside [17, 18]. Beans and rolled oats are also rich in viscous fibre that delays gastric emptying [19], enzymatic starch hydrolysis [17] and consequently delay glucose absorption [19]. Basmati rice and legumes also contain a greater amylose: amylopectin ratio that slows down the rate of starch hydrolysis and glucose absorption [16]. The presence of organic acids in oranges [20] and legumes [18] reduce the rate of starch digestion and thereby elicit lower glycaemic responses. These foods are therefore recommended in low-GI diets. Small amounts

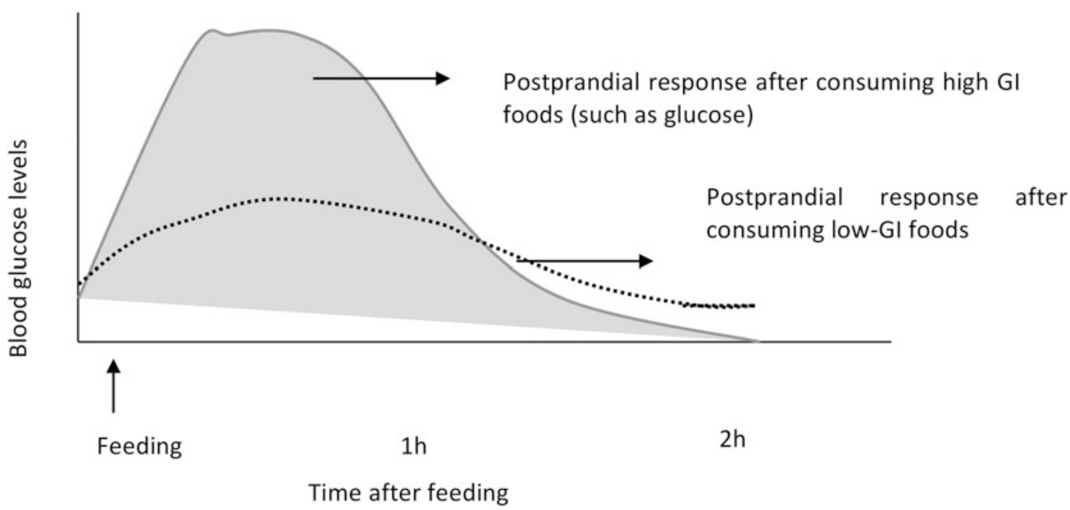


Fig. 19.1 Comparison of blood glucose *curves* after consumption of low- and high-GI foods. *Legend* GI: glycaemic index

of acetic acid (vinegar) when consumed along with the meal, reduces postprandial hyperglycaemia by 20% due to delayed gastric emptying and inhibition of digestive enzymes [21] and is a probable strategy to reduce meal GI. Furthermore, gelatinization of starch during heat treatment increases its availability to amylases and its GI [22]. Therefore, low-GI recommendations emphasise on the need to prevent overcooking of cereal foods like spaghetti and oatmeal.

Glycaemic Index of Mixed Meals

GI of individual foods in a meal has shown to predict the glycaemic response when eaten together [16] in different environments and for different cuisines [23]. The GI of a mixed meal is calculated as the sum of the proportional GI contributions of each carbohydrate component of the meal [16]. Daily diet GI is similarly calculated as the mean GI of meals consumed during the day [18].

A 15% reduction in dietary GI (~ 10 GI units for most populations) is thought to confer clinically significant health benefits [24, 25]. Given that staple cereals predominantly determine dietary GI, a 10 unit GI reduction is achieved by substituting usual high-GI staples with lower GI alternatives, while maintaining their prescribed serving size [26]. Another practical strategy to efficiently lower GI is to include one low-GI food in each meal, since GI works through the principle of averages [24]. A sample of dietary recommendations used to lower the GI of healthy diets is provided in Table 19.1.

Glycaemic Load (GL)

Due to its methodology of determination, GI may not reflect the glycaemic effect of a typical carbohydrate serving [27]. The glycaemic load (GL) concept was therefore invented to quantify the overall glycaemic effect of a portion of food [28]. The GL of a typical serving of food is the product of the amount of available carbohydrate it contains and its GI value [8]. GL of a serving is thus a measure of both carbohydrate quality and quantity [8] and accurately predicts postprandial glycaemia [29]. Accordingly, GL of a meal can be reduced either by reducing the amount of carbohydrate in diet, selecting foods that have lower GI or a combination of both [29] (Fig. 19.2).

While dietary GL can be reduced by different methods (Table 19.2), efforts that lower risks for T2DM [28] and cardiovascular disease (CVD) [3], reduce GL predominantly by lowering dietary GI, with minimal reduction to carbohydrate (compensated by slightly higher protein) content [29]. Thus, healthy low-GI/GL diets are essentially matched for calories, macronutrient distribution and other aspects of nutritional adequacy afforded by conventional healthy diets. The difference remains in the source of carbohydrates, primarily with respect to staples.

Possible Benefits of Low Glycaemic Index Diets in GDM Management

The advocacy for low-GI foods in promoting health draws from its ability to lower postprandial glycaemic and insulinaemic responses [17]. Chronic consumption of high-GI foods results in marked rise in glycaemia [17], and demands more insulin. This demand is initially compensated by increased insulin secretion [12]. This increased insulin demand exacerbates insulin resistance [12]. Hyperinsulinaemia and insulin resistance that are central to GDM pregnancies [2], eventually lead to β-cell fatigue and increased cardiovascular risks [12] as shown in Fig. 19.3.

Table 19.1 Dietary recommendations use to lower dietary GI without causing major macronutrient changes

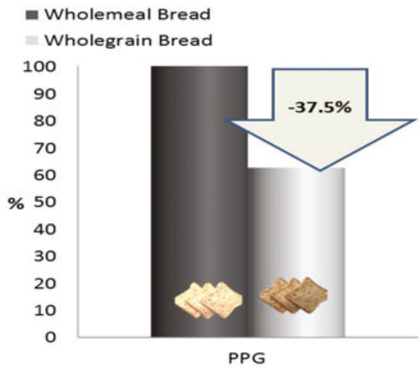
Food	Low-GI	Moderate-GI	High-GI
Recommendation	Encouraged	Moderation advised	Discouraged
Cereals and grains			
Rice	Parboiled	Basmati rice, brown rice, white rice with yoghurt (curd rice)	White rice, fragrant rice, Jasmine rice, glutinous rice
Bread	Multi-grain bread	Pita bread, chapatti made from wheat atta with dhal	White bread, wholemeal bread
Breakfast cereal	Muesli, coarse oat bran	Quick cooking/instant oats	Cornflakes, chocolate coated cornflakes, sugar coated cornflakes
Noodle and pasta	Macaroni, fettuccini spaghetti, noodles (al-dente)	Udon noodles plain	Rice noodles (fried)
Biscuits	Cream crackers—high calcium	Digestive biscuits, wholemeal biscuits, oatmeal biscuits	Wafers, sugar coated biscuits
Vegetables	Green peas, carrot, green vegetables	Sweet corn, sweet potato, yam	Pumpkin, tapioca potato
Fruits	Apple, orange, pear, plum, strawberry, dates	Grapes, banana, papaya, mango, raisins, pineapple	Watermelon, lychee
Legumes and nuts	Baked beans, kidney beans, soya beans, chick peas, lentils (dhal), mung beans, dried peas Nuts—though low in GI, moderation is encouraged	–	–
Dairy products	Skim milk, low-fat milk, low-fat yoghurt	Condensed sweetened milk	–

Source Adapted from Shyam et al. [86]
Note Serving size recommendations need to be adhered to even when using low-GI options
Legend GI: glycaemic index

In contrast, when compared with high-GI diets, low-GI diets show a slower and more sustained glycaemic response [30]. They prevent exaggerated postprandial glycaemic excursions during pregnancy [31]. Additionally, low-GI meals diminish glycaemic response to the subsequent meal [32]. Besides improving glycaemic control [33], low-GI diets improve insulin sensitivity [34] and increase β-cell function in individuals with impaired glucose tolerance [35, 36] and T2DM [37]. These actions prevent the degeneration of the glucose tolerance [16] and suggest the potential benefit of low-GI diet in GDM management.

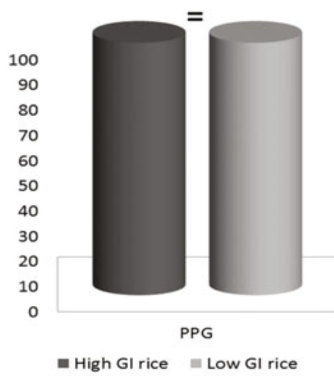
Low-GI foods also lead to the increased secretion of anorexic gut hormones which induce satiety and suppress appetite [39, 40]. Therefore voluntary energy intake is reduced for the rest of the day after a low-GI meal is consumed [41]. Moreover, low-GI diets prevent decreases in fat oxidation induced by hyperglycaemia and hyperinsulinaemia; and increases lipolysis [38, 42]. The postulated mechanisms of action of dietary GI in modulating fat oxidation and body weight gain [42–47] are compiled in Fig. 19.4. Furthermore, low-GI diets increase protein retention in both normal and hyperinsulinaemic men [48] and favour lean body mass retention [38]. Whether these mechanisms can further optimise body weight management in GDM women, who are more likely to be obese and gain more weight during and after pregnancy [1], remains to be established.

	Whole meal bread	Whole Grain Bread
Serving size (3 Carb ex)	=	
CHO (g)	45	45
GI	70	45
GL	32	20



Comparison of the postprandial glycaemic responses of breads varying in GI, when the portion size is maintained constant

	High GI Rice	Low GI Rice
GL	36 =	36
GI	60	80
Permissible CHO (g)	60	45
Serving size	4 ex	3 ex
	2 cups	1 ½ cups



Comparison of the potential serving size of rice varying in GI to maintain a constant GL

Fig. 19.2 Postulated practical application of GL concept. *Legend* GI: glycaemic index, GL: glycaemic load, CHO: carbohydrate amount (g), PPG: postprandial glycaemic response. *Top panel* shows that a similar portion of a lower GI option (wholegrain bread) versus a higher GI option (wholemeal bread) will reduce GL and hence result in lower postprandial glycaemic response. *Bottom panel* shows that theoretically a smaller serving size of a higher GI option (high-GI rice: e.g. glutinous rice) can have a similar GL as a slightly larger serving size of a lower GI option (low-GI rice: e.g. Basmati rice). However the increase in total calories as the number of carbohydrate exchange increases should be considered

Additionally, low-GI diets by virtue of increased production of short chain fatty acids (SCFA) from colonic fermentation [49], decrease the colonic luminal pH and stimulate the absorption of minerals such as calcium, potassium, magnesium, copper, zinc and selenium [50]. Colonic fermentation also increases folate availability and promotes normal homocysteine concentrations [16, 51]. Colonic fermentation moreover reduces inflammation by altering the bacterial species in the colon [52]. These effects of low-GI diets need to be verified in GDM women.

Table 19.2 Comparing options to lower dietary GL of a sample 1800 kcal diet

	Sample		Option A		Option B	
Diet	Standard healthy diet		Low-GI diet option		Low-carbohydrate diet option	
	g	% en	g	%en	g	% en
Carbohydrate	248	55	248	55	**180**	**40**
Protein	90	20	90	20	**68**	**15**
Fat	50	25	50	25	**70**	**35**
Diet GI	65		**50**		65	
Estimated diet GL	160		124		120	
Satisfies dietary guidelines	Yes		Yes		No	
Diet GI classification	Medium		Low		Medium	
Expected magnitude of dietary change	None		Medium		High	

Legend GI: glycaemic index, GL: glycaemic load
Bolded portions in Columns "Option A and B" highlight the changes made to the sample healthy diet to lower dietary GL. To achieve a similar reduction in dietary GL, the low-carbohydrate option increases fat intake and requires the implementation of drastic dietary changes

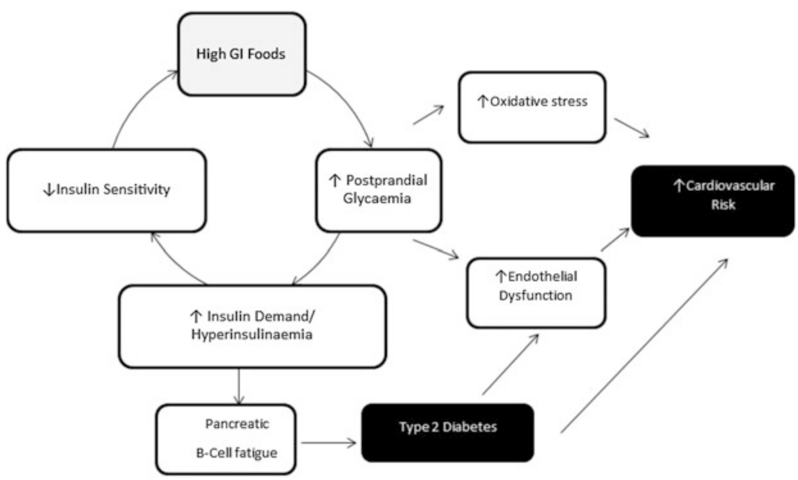


Fig. 19.3 Potential mechanisms of low-GI diets in the management of glucose homeostasis and cardiovascular risks. Legend: ↑: increase; ↓: decrease, *GI* glycaemic index

Interestingly, low-GI diets are especially beneficial to those with central obesity, insulin resistance, hyperinsulinaemia, diabetes, hypertension and metabolic syndrome [24, 53–56]. In such populations, low-GI/GL diet favours weight reduction, glycaemic control and CVD risk reduction, suggesting its potential success in the management of GDM, a condition that shares many of these risks.

In light of the pathophysiological similarities between GDM and T2DM [4], it is pertinent to note that data adds moderate to strong support for the use of low-GI diets in diabetes management [57–60]. American Diabetes Association grades the evidence to support the substitution of high-GL foods with those with lower GL, to modestly improve glycaemic control in diabetes at "level C" [61].

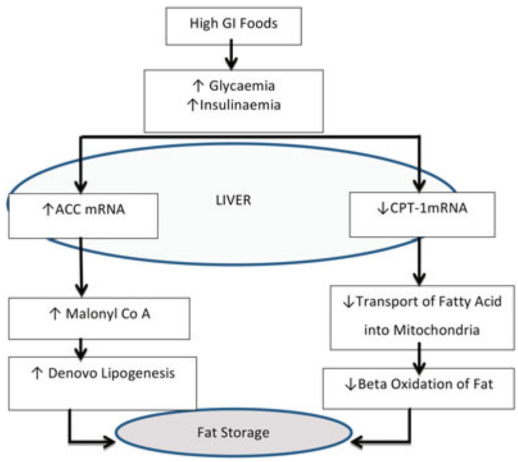


Fig. 19.4 GI and fat oxidation. Legend: ↑: increase; ↓: decrease, *GI* glycaemic index. High-GI foods reduce hepatic carnitine palmitoyltransferase-1(CPT-1) messenger RNA (mRNA) expression [42]. CPT-1 transport fatty acids into mitochondria for oxidation. High-GI foods concomitantly increase hepatic acetyl-CoA carboxylase (ACC) mRNA expression. ACC catalyses the formation of malonyl-CoA. Malonyl-CoA is a potent inhibitor of CPT-1, resulting in decreased fatty acid oxidation [42, 43]. Thus, high-GI foods lower lipolysis and facilitate fat storage

Current Evidence for the Role of Low Glycaemic Index and Glycaemic Load Diets in GDM Management

For Maintaining or Achieving Glycaemic Control During Pregnancy

Limited evidence supports the effectiveness of low-GI diet in maintaining a good glycaemic control in GDM pregnancies. Only three recent RCTs have investigated the impact of low-GI diet on blood glucose-related parameters [62–64].

Moses et al.'s intervention on women with GDM ($n = 63$) at 28 weeks of gestation, found only 29% ($n = 9$) of women receiving low-GI diets required insulin, as compared to 59% ($n = 19$) of women on a conventional-high-fibre-higher-GI-diet [62]. Eventually, 50% ($n = 9$) of these 19 women avoided insulin use after changing to a low-GI diet. However, the final GI of women in both groups were statistically similar and it was noted that increased fibre intake, reduction in carbohydrate intake and self-restriction of energy which occurred in both groups may have interfered with the study outcomes.

Grant and colleagues reported a pilot study ($n = 47$) on the feasibility and effectiveness of a low-GI diet on glycaemic control of GDM women [63]. In contrast to Moses et al. [62], Grant et al. reported lower dietary GI in the low-GI vs. the control group (49 vs. 58, $p = 0.001$). Improvements in glycaemic control in both groups were reported, but 58% of low-GI group had postprandial glucose within target as compared to 49% of control group ($p < 0.001$). This study was not powered to detect the small difference in self-monitored blood glucose (0.1–0.2 mmol/L) and postprandial blood glucose (1.2 mmol/L) observed between the study groups.

The most recent study by Hu et al. [64] was a relatively short 5-day intervention that compared the effectiveness of a low-GI staple versus a normal diabetic control diet among GDM women ($n = 140$) in Guangdong, China. Similar to the earlier studies, postprandial glucose levels were significantly reduced in both groups. However, post-intervention glucose levels taken after each meal were significantly reduced only in the low-GI group. There were also significantly greater reductions in glucose values from baseline in low-GI compared to the control group. The researchers observed a reduction in glucose parameters after breakfast in this group, though low-GI staple foods were only consumed at lunch and dinner. While the generalisability of the study findings may be limited to Asian women and the feasibility of adhering to a low-GI staple diet can be questioned due to a very short intervention period, the study nevertheless has set a good precedence for future exploration in this area.

Cumulatively these findings suggest that lowering the GI of standard diets by substituting high-GI staples with low-GI options may improve management of glycaemia in GDM women and reduce the likelihood of requiring insulin therapy. This interpretation is further supported by a meta-analysis [65] involving 257 participants that confirmed lesser use of insulin in the low-GI diet group (RR = 0.767, 95% CI = 0.597–0.986, $p = 0.039$) compared to those in control group. This translates into 13 out of 100 GDM women avoiding the use of insulin by adopting a low-GI diet during pregnancy [65].

Prevention of Complications in Pregnancy and Neonatal Outcomes

Foeto-placental overgrowth and higher infant body fat has been associated with high-GI intake during pregnancy, while low-GI diet reduces these tendencies [62, 66]. A small but intensive study by Moses and colleagues [62] showed that the consumption of low-GI diet in the second and third trimesters in normoglycaemic mothers reduced foetal birth weight, foetal percentile and Ponderal index. However PREGGIO, a similar but larger trial [67] found that an early intervention at 20 weeks of gestation did not result in significant differences in similar neonatal outcomes.

Accordingly, Louie et al. reported no significant difference in pregnancy outcomes such as birth weight, birth weight centile, prevalence of macrosomia, Ponderal index and adverse pregnancy outcomes after a low-GI dietary intervention that included a minimum of three face-to-face counselling sessions with a dietician [68]. The researchers postulated that a relative small five-point difference in GI between the study groups, early nutrition counselling for both groups, relatively lower GI than norm at the baseline, timing (third trimester) and short duration of the intervention (6–7 weeks) may have contributed to the findings. Another justification for the lack of difference may be the high proportion of participants with normal BMI (68%) and the researchers are now hypothesising low-GI diet to be more efficient among overweight and obese pregnant mothers who have higher level of insulin resistance and deficiency of β-cells [69]. However, it can be concluded that both low-GI and high fibre diet produce optimal pregnancy outcomes and this further strengthens the argument for safety of low-GI diet in the management of GDM.

The significant relationship between maternal glycaemic control and neonatal outcomes has been well-established [70–73]. Higher fasting glucose during initiation of diet therapy was associated with increased neonatal fat mass and elevated C-peptide among women treated for mild GDM in a multicentre RCT [71]. A higher prevalence of elevated C-peptide levels and neonatal outcomes such as macrosomia and large-for-gestational age babies were found among women with higher fasting blood glucose at the final two weeks of gestation. The findings were consistent with an earlier study which described fasting glucose levels to be associated with neonatal adiposity and increased skinfold thickness in neonates, regardless of whether maternal GDM was treated with diet or insulin [72]. Expectedly, secondary analysis of the ROLO study [70] found low-GI dietary intervention in pregnancy to have a beneficial effect on neonatal central adiposity, which was also positively associated with mother's postprandial glucose. Although the study was conducted among normoglycaemic pregnant women, modest reductions in GI and GL were sufficient to lower neonatal waist: length ratio in the intervention group. This indicates that improved dietary carbohydrate quality may be associated with reduced neonatal central adiposity rather than birth weight. More importantly, epidemiologic studies among healthy pregnant women have found associations between high diet GI and congenital malformations such as neural tube defects, musculoskeletal and gastrointestinal defects [74].

These findings illustrate the importance of carbohydrate quality during pregnancy to promote neonatal well-being. However, while available data indicates the potential role of low-GI diets in reducing fat mass and central adiposity in neonates born to GDM mothers, there is insufficient evidence to establish the benefit of low-GI diets in preventing excessive maternal weight gain, foetal abnormalities, pregnancy complications or adverse pregnancy outcomes.

Prevention of GDM Recurrence and Overt Development of Diabetes

There is limited evidence relating low-GI diets and recurrence of GDM or development of T2DM among women with prior GDM. A recent Asian study [75] among women with prior GDM, compared the effectiveness of low-GI diet and conventional healthy dietary recommendations and reported improvements in glucose tolerance with low-GI educational intervention. The greatest improvement in glucose tolerance was observed among women with higher baseline insulin levels and in the lowest quartile of dietary GI at six months. The researchers also noted a significant reduction in 2-h post-75 g-oral glucose tolerance test (2HPP). In contrast, 2HPP levels increased in the comparison group, resulting in a significant difference in 2HPP changes between groups (Mean difference = 2.4 mmol/L, $p = 0.004$). It was suggested that a reduction of 2HPP by more than 0.84 mmol/L may halve the risk for T2DM and low-GI diet may be able to deliver that especially among women with a history of GDM and higher insulin levels.

In another distinctive study by Ostman and colleagues [76], seven women with impaired glucose tolerance and history of GDM were provided with either low-GI/high-fibre or high-GI/low-fibre bread products, during two consecutive 3-week periods, separated by a three-week washout period. The women receiving low-GI/high fibre bread had 35% lower insulin response to intravenous glucose challenge, though no effect was found on fasting glucose, insulin or lipid markers within the short 3 weeks of intervention. However, the sustainability of the effect remains to be established.

Concerns with the Use of Low-GI Diets

Since its inception, the utility of GI concept has been voraciously debated citing methodological issues and nutritional concerns [18].

Methodological Issues

Among the technical objections, the applicability of GI in mixed meals is predominantly questioned. However, studies reporting a lack of association between GI and glycaemic response when foods are taken as part of a mixed meal [77, 78] are thought to be methodologically flawed [14]. When analysed using standardised methods, the relative glycaemic impact of mixed meals is reportedly predicted by the amount of available carbohydrate they contain and the GI of their components [14].

The practical applicability of GI concept is also limited by the lack of a comprehensive GI database [24, 27]. While the international listing of GI and GL values is indeed comprehensive [13], determination of GI values of local foods is a work in progress in many countries. GI determination is cost and labour intensive and simplified methods have been devised to appropriately match foods and assign GI values to those with unknown values, till more local GI values become available [79]. As many factors affect the GI of a food, including its species, maturity (ripeness), storage time, processing and cooking method, [27], technical uncertainties exist in this estimation process. Estimation of diet GI and GL require in-depth knowledge of carbohydrate intake [16] and food composition tables lack the intricate detail necessary to accurately match foods and their glycaemic response [80]. However, this limitation affects not only the reliability of estimating dietary GI but of all other nutrients as well. Specific biomarkers to assess diet intake, including diet GI, need to be established and will improve the objectivity of dietary assessments.

The relationship between the dietary fibre content and the GI of a food despite being modest [81], confounds existing evidence for the health impact of low-GI diets which are also consistently higher in dietary fibre [27]. While the proponents of GI point to validity and reproducibility of GI values determined in standardised laboratories [29], phenotypic differences among populations in response to starch exist [82] and may limit the application of GI values as it is currently determined.

Nutritional Concerns

Nutritional concerns in using GI stem from the fear that it may incite public to consume foods low in GI but high in fat and sugars like ice-cream, cookies, etc. [18, 27, 83]. However, GI proponents argue that GI should be applied only to low-fat starchy foods [84]. GI was never meant to be used in "isolation", but as an adjunct to other healthy eating principles [27]. Therefore the GI concept cannot replace, but should rather supplement existing nutritional strategies [18]. Perhaps the best approach to include GI education in diabetes counselling is to focus on individualisation [83] and this requires appropriate dietetic supervision.

Practical Issues with Implementation

The complexity of the GI/GL concepts make it difficult for patients to comprehend and implement the recommendations [18, 83, 85]. However, many low-GI diet books for weight control and wellness have been well-received in the West. Whether this acceptance can be extended to the other parts of the globe remains to be answered. Interestingly, various efforts at developing simplified GI-education modules have been successful. Categorising carbohydrates with simple terms like "gushers" and "tricklers" may ease patient comprehension of the concept [17]. Asian RCTs have shown that adults can be counselled to follow low-GI diets without having to memorise GI values [24, 86]. However, these are findings from clinical trials run by trained researchers and the practicality of providing GI-education in conventional healthcare settings remains to be proven.

Another concern with low-GI diets is that it can limit food choices and compromise nutritional adequacy [18, 83]. This may be especially important when dealing with pregnant women. Although traditional Indian and Greek cuisines include more low-GI foods than typical Western diets [83], adopting these food patterns may not be practical for all. Furthermore, food industries face challenges in producing palatable low-GI foods [84]. The issue is further compounded by the absence of a universally accepted logo that would facilitate consumer recognition of low-GI products.

While trials lasting a year or more show similar rates of adherence to low-GI and standard diets across continents [37, 87, 88], feasibility of long-term adherence to low-GI diets is unknown. While it may be possible to plan low-GI diets economically [86], its cost-effectiveness also remains to be established.

Recommendations

While emerging evidence suggests the possible benefit of low-GI diets in GDM management, there is urgent need for validation of the results. Optimising caloric intake to individual needs, restricting saturated fat, and distributing carbohydrates throughout the day will aid management of body weight and prevent of the degeneration of glucose tolerance in GDM women. Dietary recommendations should continue to encourage a moderate carbohydrate diet (45–50%), with adequate dietary fibre (25–30 g). Accordingly, dietary recommendations should encourage the inclusion of whole grains, beans, rolled oats, low-fat dairy and lean meat products, while being mindful of the daily energy needs. These strategies while in-line with conventional dietary goals, will also lower diet GI. Switching to low-GI-staples (such as whole grain breads, low-GI rice varieties and pasta) can be encouraged, taking cues from individual preferences. While adopting low-GI diets, there is a need to continue monitoring the portion sizes since postprandial glycaemia is affected by both the quantity and quality of carbohydrates. Low-GI diets that satisfy other nutritional considerations are acceptable in the treatment of GDM.

Conclusions

Existing evidence suggests that lowering the GI of conventional healthy diets may be beneficial in GDM treatment for managing maternal glycaemia and neonatal adiposity. However, a few practical issues in implementing low-GI dietary recommendations remain unresolved at present. There is an urgent need for adequately powered, well-controlled trials to further investigate the feasibility, acceptability, adherence, safety, clinical and cost effectiveness of low-GI dietary recommendations in GDM management.

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References

1. Metzger BE, Buchanan TA, Coustan DR, de Leiva A, Dunger DB, Hadden DR, et al. Summary and recommendations of the fifth international workshop-conference on gestational diabetes mellitus. Diab Care. 2007;30(Suppl. 2):S251–S60.
2. Barbour LA, McCurdy CE, Hernandez TL, Kirwan JP, Catalano PM, Friedman JE. Cellular mechanisms for insulin resistance in normal pregnancy and gestational diabetes. Diab Care. 2007;30(Suppl. 2):S112–9.
3. Barclay AW, Petocz P, McMillan-Price J, Flood VM, Prvan T, Mitchell P, et al. Glycemic index, glycemic load, and chronic disease risks-a meta-analysis of observational studies. Am J Clin Nutr. 2008;87(3):627–37.
4. Solomon CG, Willett WC, Carey VJ, Rich-Edwards J, Hunter DJ, Colditz GA, et al. A prospective study of pregravid determinants of gestational diabetes mellitus. JAMA. 1997;278(13):1078–83.
5. Butte FN. Carbohydrate and lipid metabolism in pregnancy: normal compared with gestational diabetes mellitus. Am J Clin Nutr. 2000;71(suppl):1256S–61S.
6. Moses RG, Cefalu WT. Considerations in the management of gestational diabetes Mellitus: “You Are What Your Mother Ate!”. Diabetes Care. 2016;39:13–5.
7. Hernandez TL, Anderson MA, Chartier-Logan C, Friedman JE, Barbour LA. Strategies in the nutritional management of gestational diabetes. Clin Obstet Gynecol. 2013;56(4):803–15.
8. Foster-Powell K, Holt SH, Brand-Miller JC. International table of glycemic index and glycemic load values: 2002. Am J Clin Nutr. 2002;76:5–56.

9. Cypryk K, Kaminska P, Kosinski M, Pertynska-Marczewska M, Lewinski A. A comparison of the effectiveness, tolerability and safety of high and low carbohydrate diets in women with gestational diabetes. Endokrynol Pol. 2007;58(4):314–9.
10. Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, et al. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. New Engl J Med. 2001;345(11):790–7.
11. Different types of dietary advice for women with gestational diabetes mellitus (Review) [Internet]. Wiley; 2013.
12. Augustin LSFS, Jenkins DJ, Kendall CW, La Vecchia C. Glycemic index in chronic disease: a review. Eur J Clin Nutr. 2001;56:1049–71.
13. GI database [Internet]. 2012. Available from http://www.glycemicindex.com.
14. Wolever TM, Yang M, Zeng XY, Atkinson F, Brand-Miller JC. Food glycemic index, as given in Glycemic Index tables, is a significant determinant of glycemic responses elicited by composite breakfast meals. Am J Clin Nutr. 2006;83:1306–12.
15. Jenkins D, Wolever T, Taylor R, Barker H, Fielden H, Baldwin J, et al. Glycemic index of foods: a physiological basis for carbohydrate exchange. Am J Clin Nutr. 1981;34(3):362–6.
16. Wolever TMS. The glycaemic index: a physiological classification of dietary carbohydrate. Oxfordshire, UK: CABI Publishing; 2006 227 p.
17. Burani J. Gushers and Tricklers: Practical use of the glycemic index [Powerpoint Presentation]. Marco Island, Florida: University of Sydney; 2006 [cited 2010 11 March]. Available from http://www.glycemicindex.com/glycemic.index.ppt.
18. Kalergis M. The role of the glycemic index in the prevention and management of diabetes: a review and discussion. Can J Diab. 2005;29:27–38.
19. Jenkins DJJA, Wolever TM, Collier GR, Rao AV, Thompson LU. Starchy foods and fiber: reduced rate of digestion and improved carbohydrate metabolism. Scand J Gastroenterol Suppl. 1987;129:132–41.
20. Yoon J, Thompson L, Jenkins D. The effect of phytic acid on in vitro rate of starch digestibility and blood glucose response. Am J Clin Nutr. 1983;38(6):835–42.
21. Johnston CS, Stelewska I, Long CA, Harris LN, Ryals RH. Examination of antiglycemic properties of vinegar in healthy dults. Ann Nutr Metab. 2010;56(1):74–9.
22. Holm J, Lundquist I, Bjorck I, Eliasson A, Asp N. Degree of starch gelatinization, digestion rate of starch in vitro, and metabolic response in rats. Am J Clin Nutr. 1988;47(6):1010–6.
23. Chew I, Brand J, Thorburn A, Truswell A. Application of glycemic index to mixed meals. Am J Clin Nutr. 1988;47:53–6.
24. Yusof BNM. A randomized control trial of low glycemic index against conventional carbohydrate exchange diet on glycemic control and metabolic parameters in type 2 diabetes [Thesis]. Kuala Lumpur: Universiti Kebangsaan Malaysia; 2008.
25. Goff LM, Frost GS, Hamilton G, Thomas EL, Dhillo WS, Dornhorst A, et al. Carbohydrate-induced manipulation of insulin sensitivity independently of intramyocellular lipids. Br J Nutr. 2003;89(03):365–74.
26. Amano Y, Sugiyama M, Lee JS, Kawakubo K, Mori K, Tang AC, et al. Glycemic index-based nutritional education improves blood glucose control in Japanese adults: a randomized controlled trial. Diab Care. 2007;30(7):1874–6.
27. Miller J. The glycaemic index of foods. Asia Pacific J Clin Nutr. 1993;2:107–10.
28. Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. JAMA. 1997;277(6):472–7.
29. Augustin LSA, Kendall CWC, Jenkins DJA, Willett WC, Astrup A, Barclay AW, et al. Glycemic index, glycemic load and glycemic response: an International Scientific Consensus Summit from the International Carbohydrate Quality Consortium (ICQC). Nutr Metab Cardiovasc Dis. 25(9):795–815.
30. Wolever TM. The glycemic index. World Rev Nutr Diet. 1990;62:120–85.
31. Louie JCY, Brand-Miller JC, Markovic TP, Ross GP, Moses RG. Glycemic index and pregnancy: a systematic literature review. J Nutr Metab. 2011;2010.
32. Brighenti F, Benini L, Del Rio D, Casiraghi C, Pellegrini N, Scazzina F, et al. Colonic fermentation of indigestible carbohydrates contributes to the second-meal effect. Am J Clin Nutr. 2006;83(4):817–22.
33. Jenkins DA, Kendall CC, McKeown-Eyssen G, et al. Effect of a low–glycemic index or a high–cereal fiber diet on type 2 diabetes: a randomized trial. JAMA. 2008;300(23):2742–53.
34. Barr S, Reeves S, Sharp K, Jeanes YM. An isocaloric low glycemic index diet improves insulin sensitivity in women with polycystic ovary syndrome. J Acad Nutr Diet. 2013;113(11):1523–31.
35. Wolever TMS, Mehling C. High-carbohydrate low-glycaemic index dietary advice improves glucose disposition index in subjects with impaired glucose tolerance. Br J Nutr. 2002;87(5):477–87.
36. Laaksonen DE, Toppinen LK, Juntunen KS, Autio K, Liukkonen K-H, Poutanen KS, et al. Dietary carbohydrate modification enhances insulin secretion in persons with the metabolic syndrome. Am J Clin Nutr. 2005;82(6):1218–27.

37. Wolever TM, Gibbs AL, Mehling C, Chiasson JL, Connelly PW, Josse RG, et al. The Canadian Trial of Carbohydrates in Diabetes (CCD), a 1-y controlled trial of low-glycemic-index dietary carbohydrate in type 2 diabetes: no effect on glycated hemoglobin but reduction in C-reactive protein. Am J Clin Nutr. 2008;87(1): 114–25.
38. Brand-Miller JC, Holt SH, Pawlak DB, McMillan J. Glycemic index and obesity. Am J Clin Nutr. 2002;76 (1):281S–5S.
39. Lavin J, Wittert G, Andrews J, Yeap B, Wishart J, Morris H, et al. Interaction of insulin, glucagon-like peptide 1, gastric inhibitory polypeptide, and appetite in response to intraduodenal carbohydrate. Am J Clin Nutr. 1998; 68(3):591–8.
40. Holt S, Brand J, Soveny C, Hansky J. Relationship of satiety to postprandial glycaemic, insulin and cholecystokinin responses. Appetite. 1992;18(2):129–41.
41. Ludwig DS, Majzoub JA, Al-Zahrani A, Dallal GE, Blanco I, Roberts SB. High glycemic index foods, overeating, and obesity. Pediatrics. 1999;103:e26.
42. Wolfe R. Metabolic interactions between glucose and fatty acids in humans. Am J Clin Nutr. 1998;67(3): 519S–26S.
43. Pawlak D, Denyer GS, Brand-Miller JC. Long term feeding with high glycemic index starch leads to obesity in mature rats. Proc Nutr Soc Aust. 2000;24:215 (abstract).
44. Zurlo F, Lillioja S, Esposito-Del Puente A, Nyomba BL, Raz I, Saad MF, et al. Low ratio of fat to carbohydrate oxidation as predictor of weight gain: study of 24-h RQ. Am J Physiol Endocrinol Metab. 1990;259(5):E650–7.
45. Simoneau J-A, Veerkamp JH, Turcotte LP, Kelley DE. Markers of capacity to utilize fatty acids in human skeletal muscle: relation to insulin resistance and obesity and effects of weight loss. FASEB J. 1999;13(14):2051–60.
46. Febbraio MA, Keenan J, Angus DJ, Campbell SE, Garnham AP. Preexercise carbohydrate ingestion, glucose kinetics, and muscle glycogen use: effect of the glycemic index. J Appl Physiol. 2000;89(5):1845–51.
47. Kiens B, Richter E. Types of carbohydrate in an ordinary diet affect insulin action and muscle substrates in humans. Am J Clin Nutr. 1996;63(1):47–53.
48. Howe JC, Rumpler WV, KM. B. Dietary starch composition and level of energy intake alter nutrient oxidation in 'carbohydrate-sensitive' men. J Nutr. 1996;126:2120–9.
49. FAO/WHO. Carbohydrates in Human nutrition: report of a joint FAO/WHO expert consultation. In: FAO/WHO, editor. Rome 14–18 April 1997.
50. Metzler-Zebeli BU, Hooda S, Mosenthin R, Ganzle MG, Zijlstra RT. Bacterial fermentation affects net mineral flux in the large intestine of pigs fed diets with viscous and fermentable nonstarch polysaccharides. J Anim Sci. 88(10):3351–62.
51. Houghton L, Green T, Donovan U, Gibson R, Stephen A, O'Connor D. Association between dietary fiber intake and the folate status of a group of female adolescents. Am J Clin Nutr. 1997;66(6):1414–21.
52. Neish AS, Gewirtz AT, Zeng H, Young AN, Hobert ME, Karmali V, et al. Prokaryotic regulation of epithelial responses by inhibition of IκB-α ubiquitination. Science. 2000;289(5484):1560–3.
53. Frost G, Wilding J, Beecham J. Dietary advice based on the glycaemic index improves dietary profile and metabolic control in type 2 diabetic patients. Diab Med. 1994;11(4):397–401.
54. Ebbeling CB, Leidig MM, Sinclair KB, Seger-Shippee LG, Feldman HA, Ludwig DS. Effects of an ad libitum low-glycemic load diet on cardiovascular disease risk factors in obese young adults. Am J Clin Nutr. 2005;81 (5):976–82.
55. Frost GS, Brynes AE, Bovill-Taylor C, Dornhorst A. A prospective randomised trial to determine the efficacy of a low glycaemic index diet given in addition to healthy eating and weight loss advice in patients with coronary heart disease. Eur J Clin Nutr. 2004;58(1):121–7.
56. Ghani RA, Shyam S, Arshad F, Wahab NA, Chinna K, Safii NS, et al. The influence of fasting insulin level in post-gestational diabetes mellitus women receiving low-glycaemic-index diets. Nutr Diabetes. 2014;4:e107.
57. Wheeler ML, Dunbar SA, Jaacks LM, Karmally W, Mayer-Davis EJ, Wylie-Rosett J, et al. Macronutrients, food groups, and eating patterns in the management of diabetes: a systematic review of the literature, 2010. Diab Care. 2012;35(2):434–45.
58. Rivellese AA, Giacco R, Costabile G. Dietary carbohydrates for diabetics. Curr Atherosclerosis Rep. 2012; 14(6):563–9.
59. Ajala O, English P, Pinkney J. Systematic review and meta-analysis of different dietary approaches to the management of type 2 diabetes. Am J Clin Nutr. 2013;97(3):505–16.
60. Mann JI, Te Morenga L. Diet and diabetes revisited, yet again. Am J Clin Nutr. 2013;97(3):453–4.
61. Evert AB, Boucher JL, Cypress M, Dunbar SA, Franz MJ, Mayer-Davis EJ, et al. Nutrition therapy recommendations for the management of adults with diabetes. Diab Care. 2013;36(11):3821–42.
62. Moses RG, Barker M, Winter M, Petocz P, Brand-Miller JC. Can a low-glycemic index diet reduce the need for insulin in gestational diabetes mellitus? A randomized trial. Diab Care. 2009;32(6):996–1000.
63. Grant SM, Wolever TMS, O'Connor DL, Nisenbaum R, Josse RG. Effect of a low glycaemic index diet on blood glucose in women with gestational hyperglycaemia. Diab Res Clin Pract. 2011;91(1):15–22.

64. Hu Z-G, Tan R-S, Jin D, Li W, Zhou X-Y. A low glycemic index staple diet reduces postprandial glucose values in Asian women with gestational diabetes mellitus. J Investig Med. 2014;62(8):975–9.
65. Viana LV, Gross JL, Azevedo MJ. Dietary intervention in patients with gestational diabetes mellitus: a systematic review and meta-analysis of randomized clinical trials on maternal and newborn outcomes. Diab Care. 2014; 37(12):3345–55.
66. Moses RG, Luebcke M, Davis WS, Coleman KJ, Tapsell LC, Petocz P, et al. Effect of a low-glycemic-index diet during pregnancy on obstetric outcomes. Am J Clin Nutr. 2006;84(4):807–12.
67. Moses RG, Casey SA, Quinn EG, Cleary JM, Tapsell LC, Milosavljevic M, et al. Pregnancy and glycemic index outcomes study: effects of low glycemic index compared with conventional dietary advice on selected pregnancy outcomes. Am J Clin Nutr. 2014;99(3):517–23.
68. Louie JCY, Markovic TP, Perera N, Foote D, Petocz P, Ross GP, et al. A randomized controlled trial investigating the effects of a low–glycemic index diet on pregnancy outcomes in gestational diabetes mellitus. Diab Care. 2011;34(11):2341–6.
69. Catalano P, Vargo K, Bernstein I, Amini S. Incidence and risk factors associated with abnormal postpartum glucose tolerance in women with gestational diabetes. Am J Obstet Gynecol. 1991;165:914–9.
70. Horan MK, McGowan CA, Gibney ER, Donnelly JM, McAuliffe FM. Maternal low glycaemic index diet, fat intake and postprandial glucose influences neonatal adiposity–secondary analysis from the ROLO study. Nutr J. 2014;13(1):1.
71. Durnwald CP, Mele L, Spong CY, Ramin SM, Varner MW, Rouse DJ, et al. Glycemic characteristics and neonatal outcomes of women treated for mild gestational diabetes. Obstet Gynecol. 2011;117(4):819.
72. Uvena-Celebrezze J, Fung C, Thomas A, Hoty A, Huston-Presley L, Amini S, et al. Relationship of neonatal body composition to maternal glucose control in women with gestational diabetes mellitus. J Matern Fetal Neonatal Med. 2002;12(6):396–401.
73. Pedersen J. Diabetes and pregnancy; blood sugar of newborn infants during fasting and glucose administration. Ugeskrift laeger. 1952;114(21):685.
74. Parker SE, Werler MM, Shaw GM, Anderka M, Yazdy MM. Dietary glycemic index and the risk of birth defects. Am J Epidemiol. 2012;176(12):1110–20.
75. Shyam S, Arshad F, Ghani RA, Wahab NA, Safii N, Nisak MY, et al. Low glycaemic index diets improve glucose tolerance and body weight in women with previous history of gestational diabetes: a six months randomized trial. Nutr J. 2013;12(1):1.
76. Östman E, Frid A, Groop L, Björck I. A dietary exchange of common bread for tailored bread of low glycaemic index and rich in dietary fibre improved insulin economy in young women with impaired glucose tolerance. Eur J Clin Nutr. 2006;60(3):334–41.
77. Hollenbeck CBCA, Reaven GM. Comparison of plasma glucose and insulin responses to mixed meals of high, intermediate- and low-glycemic potential. Diab Care. 1988;11:323–9.
78. Laine DCTW, Levitt MD, Bantle JP. Comparison of predictive capabilities of diabetic exchange lists and glycemic index of foods. Diab Care. 1987;10(4):387–94.
79. Louie JC-Y, Flood V, Turner N, Everingham C, Gwynn J. Methodology for adding glycemic index values to 24-hour recalls. Nutrition. 2010;30:1–6.
80. Wolever T, Jenkins D, Jenkins A, Josse R. The glycemic index: methodology and clinical implications. Am J Clin Nutr. 1991;54(5):846–54.
81. Wolever T. Relationship between dietary fiber content and composition in foods and the glycemic index. Am J Clin Nutr. 1990;51(1):72–5.
82. Mandel AL, Breslin PAS. High endogenous salivary amylase activity is associated with improved glycemic homeostasis following starch ingestion in adults. J Nutr. 2012;142(5):853–8.
83. Palmer S. Finding a place for the glycemic index. Today's Dietitian [Internet]. 2009;11(6):8 p. Available from http://www.todaysdietitian.com/newarchives/060109p8.shtml.
84. Brand-Miller J, Foster-Powell K, Nutr M. Diets with a low glycemic index: from theory to practice. Nutr Today. 1999;34(2):64–72.
85. Beebe C. Diets with a low glycemic index: not ready for practice yet!! Nutr Today. 1999;34(2):82–6.
86. Sangeetha-Shyam, Fatimah A, Rohana AG, Norasyikin AW, Karuthan C, Nik Shanita S, et al. Lowering dietary glycaemic index through nutrition education among Malaysian women with a history of gestational diabetes mellitus. Mal J Nut. 2013;19(1):9–23.
87. Sichieri R, Moura AS, Genelhu V, Hu F, Willett WC. An 18-mo randomized trial of a low-glycemic-index diet and weight change in Brazilian women. Am J Clin Nutr. 2007;86(3):707–13.
88. Shyam S, Arshad F, Ghani RA, Wahab NA, S. NS, Chinna K, et al. Effect of including glycaemic index (GI) nutrition education, within the conventional healthy dietary recommendation framework, on body weight and composition of women with prior gestational diabetes mellitus: results from a one-year randomised controlled trial. Mal J Nutr. 2015;21(3):269–83.