Curr Diab Rep (2013) 13:6–11
DOI 10.1007/s11892-012-0332-1

Carbohydrates, Glycemic Index, and Pregnancy Outcomes in Gestational Diabetes

Jimmy Chun Yu Louie · Jennie C. Brand-Miller · Robert G. Moses

Published online: 11 October 2012

Abstract This review critically evaluates the current evidence regarding the effect of the dietary glycemic index (GI) on pregnancy outcomes in gestational diabetes mellitus (GDM). Current evidence, although limited, consistently supports the advantages of, and has demonstrated no disadvantages of, a low-GI diet. We conclude that pregnant women with GDM are likely to benefit from following a low-GI meal pattern, with no significant side effects, and consideration of the GI should be given when formulating a diet for GDM. However, until larger scale intervention trials are completed, an exclusive low-GI diet should not replace the current recommended diets for GDM from relevant government and health agencies. Further studies that intervene at an earlier stage of pregnancy are required.

Keywords Glycemic index · Carbohydrates · Pregnancy · Diet · Gestational diabetes mellitus

J. C. Y. Louie
School of Health Sciences, Faculty of Health and Behavioral Sciences, The University of Wollongong,
Wollongong, NSW 2522, Australia
e-mail: jlouie@uow.edu.au

J. C. Brand-Miller
Discipline of Nutrition and Metabolism, School of Molecular Bioscience, The University of Sydney,
Sydney, NSW 2006, Australia
e-mail: jennie.brandmiller@sydney.edu.au

R. G. Moses (✉)
Diabetes Centre, Illawarra Shoalhaven Local Health District,
Wollongong, NSW 2500, Australia
e-mail: Robert.moses@sesiahs.health.nsw.gov.au

Introduction

Gestational diabetes mellitus (GDM) is commonly defined as "any degree of glucose intolerance with onset or first recognition during pregnancy" [1]. It has been suggested that women who develop GDM may have preexisting β-cell dysfunction as a result of chronic insulin resistance [2–4]. This reduces their ability to further increase insulin secretion to compensate for the decreasing insulin sensitivity as their pregnancy progresses [2, 5, 6], and glucose tolerance is impaired as a result. However, once the pregnancy-related impairment of insulin sensitivity has disappeared, glucose tolerance is likely to return to normal levels, although pregnant women who have had GDM are at a high risk of developing type 2 diabetes in future years. Worldwide, the prevalence of GDM has continued to increase. This is likely to increase further after the adoption of the new International Association of Diabetes and Pregnancy Study Groups (IADPSG) guidelines [7–9], which have lower glucose diagnostic levels [10]. The causes and adverse outcomes of GDM are summarized in Fig. 1.

Pedersen hypothesized that maternal blood glucose was the main fuel for fetal growth [11], and subsequently, it has been shown that women with even a mildly elevated blood glucose level have a higher risk of giving birth to a large for gestational age (LGA; defined as birth weight ≥90th percentile of the relevant population) infant [10]. LGA infants have higher risks of childhood obesity [12, 13], cardiovascular diseases [14], and diabetes [12] later in life; therefore, one of the main objectives of GDM treatment is to maintain maternal blood glucose levels as close to normal as possible. Since carbohydrate (CHO) is the only macronutrient that significantly increases postprandial blood glucose response, moderation of CHO intake is usually recommended as the main and first-line strategy to achieve postprandial euglycemia [15].

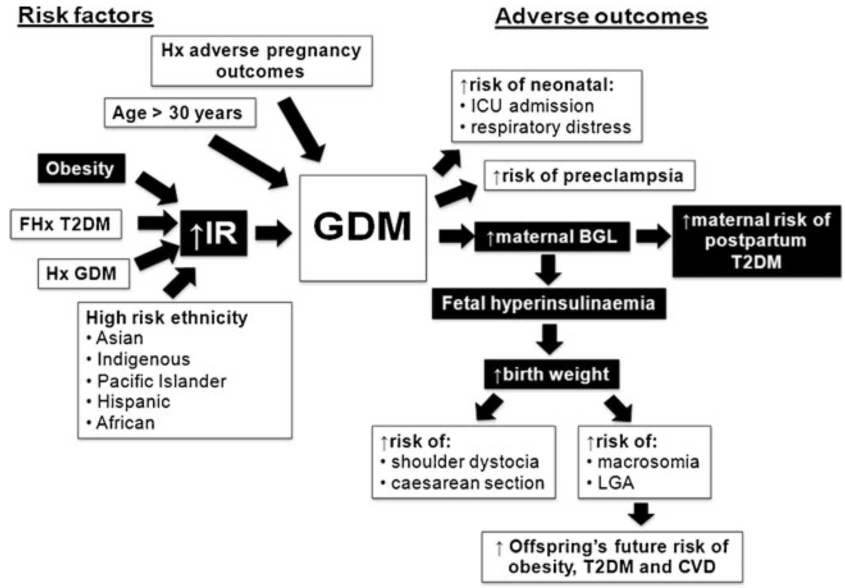

Fig. 1 The causes and adverse outcomes of gestational diabetes mellitus. Shaded boxes indicate factors/outcomes where a low glycemic index diet may be beneficial. GDM, gestational diabetes mellitus; IR, insulin resistance; T2DM, type 2 diabetes mellitus; CVD, cardiovascular diseases; FHx, family history; BGL, blood glucose level

Carbohydrates and Gestational Diabetes Mellitus

The main purpose of dietary management of GDM is to achieve postprandial euglycemia. A euglycemic GDM diet is usually one with a restricted CHO content [16, 17] that is evenly distributed throughout the day. This is because a diet with a higher proportion (>55 %) of energy from CHOs had been shown to cause higher levels of postprandial glycemia [18].

Despite the significant effect of CHO on postprandial glycemia, complete avoidance of CHO-containing foods is heavily discouraged, since many of them are nutritious, such as milk, yoghurt, and fruits. In addition, overrestriction of CHO in GDM may actually increase the risk of fetal macrosomia [19]. Therefore, instead of complete avoidance or heavy restriction of CHOs, moderation of CHO intake is usually recommended as the first-line strategy to achieve postprandial euglycemia [15, 20]. The American Diabetes Association recommends a diet with ≤40 % of energy derived from CHOs for pregnant women with GDM [17].

The Glycemic Index and Glycemic Load

Glycemic index (GI) and the related dietary term *glycemic load* (GL) are systematic and physiologically based measures of the glycemic burden of dietary CHOs. Specifically, the GI is an in vivo measure of the blood glucose response to a standard amount of CHOs from a food, relative to a reference food. It is defined methodologically as the average incremental area under the curve in response to the test food (numerator) versus the reference food (denominator), usually pure glucose or white bread, in 10 or more individuals. Examples of common low- and high-GI foods are given in Table 1. So far, the limited evidence available does not suggest that the GI of a food is different in pregnancy [21].

The Potential Benefits of Low Glycemic Index in Pregnancy

The first evidence supporting the beneficial effect of a low-GI diet in pregnancy came from Clapp [22], who investigated the effect of a low-GI diet on the pregnancy outcome of healthy gravidas. After following a low-GI weight maintenance diet from before pregnancy until 8 weeks gestation, the 12 subjects were randomized either to continue the low-GI diet ("aboriginal" CHO diet) or to a high-GI ("cafeteria" CHO) diet that is isoenergetic. Mothers on the high-GI diet were found to have gained more weight than were those who followed a low-GI diet (mean±*SEM*: low GI, 11.8±2.3 kg, vs. high GI, 19.7±1.2 kg; $p<.01$). Infants born to mothers in the high-GI group also had higher birth weight (mean±*SEM*: low GI, 3.27±0.12 kg, vs. high GI, 4.25±0.11 kg; $p<.01$) and higher fat mass (mean±*SEM*: low GI, 301±50 g, vs. high GI, 402±80 g; $p<.01$).

Epidemiological evidence from the Camden study [23] also found that a low-GI diet during pregnancy was associated with lower birth weight. They found that infants of mothers who had a dietary GI<50—that is, a low-GI diet—had a 116-g lower birth weight than did infants of mothers who had a dietary GI>60 ($p<.05$). The authors, however, did not find significant association between dietary GI and risk of LGA.

To test the hypothesis that low-GI diets may improve pregnancy outcomes—in particular, birth weight—Moses et al. [24] in 2006 conducted a randomized controlled trial

Table 1 Examples of common low and high glycemic index (GI) foods

Low-GI foods	High-GI foods
Heavy or dense wholegrain breads	White and wholemeal breads
Porridge made with traditional rolled oats	Cornflakes and puffed/extruded breakfast cereals
Pastas (except gnocchi)	Most rice (except basmati and Doongara™)
Most fruits (excluding melons)	Watermelon
Milk and yoghurts	Rice milk
Legumes	Potatoes and potato products

in 70 healthy women with a singleton pregnancy recruited in weeks 12–16 of gestation, investigating the effect of a low-GI diet on outcomes of pregnancy. There was a lower prevalence of LGA infants in the low-GI group (3 % vs. 33 % in the conventional diet group; p=.01), and the prevalence of SGA was similar (9 % vs. 7 %; nonsignificant). This has led to the hypothesis that a low-GI diet may be beneficial in GDM, where blood glucose control is strongly associated with pregnancy outcomes [25–27].

Evidence from well-designed, large-scale randomized trials suggests that treating even mild GDM results in marked improvement in pregnancy outcomes [25, 28]. By reducing maternal postprandial hyperglycemia in diabetic pregnancies through pharmacological and dietary strategies, risks of macrosomia (birth weight>4 kg) and later obesity in childhood could be reduced [13, 24, 29].

Apart from the quantity of CHO consumed, postprandial glycemia is also affected by the rate of CHO digestion and absorption. Low-GI foods, which are digested and absorbed more slowly than high-GI foods, have been found to reduce postprandial glycemia in healthy individuals [30]. Therefore, by following a low-GI meal pattern, postprandial hyperglycemia in pregnancy may be better controlled with minimal need of CHO restriction [29].

In addition to its potential beneficial effect on postprandial blood glucose responses, a low-GI diet may also lower the risk of developing GDM, via better weight control [31–35] and improved insulin sensitivity [33, 36] (Fig. 1). Evidence from the Nurses' Health Study II [37] found that women who had a dietary GI higher than 57 units had a 30 % increased risk of developing GDM, as compared with subjects whose dietary GI was below 51. When total CHO intake is taken into account as well—that is, dietary GL—the increase in risk for the group with the highest GL (greater than 138 units), as compared with the lowest quintile (GL<104 units), jumped from 30 % to 61 %.

Recent Evidence from Animal Studies

Smith et al. [38•] in 2009 conducted a novel study that provided significant evidence in a sheep model, which is considered comparable to human models in the studies of pregnancy and fetal development [38•], that a high-GI diet may be detrimental in pregnancy. They randomly assigned 104 ewes to receive oral administration of either 100 mL propylene glycol (n=51), which produces similar postprandial effect as a high-GI meal, or 100 mL water (n=53) twice per day. The authors found that lambs born to the ewes in the propylene glycol group had significantly higher birth weight (p=.032), ponderal index (calculated as birth weight in kilograms, divided by the cube of height in meters; p=.043), and plasma glucose levels (p<.001). These lambs also grew quicker, as evidenced by the achievement of the same carcass weight at an earlier age than the control lambs (p=.039). The authors therefore concluded that transient high glycemic intake was associated with higher birth weight and faster postnatal growth.

Recent Evidence from Intervention Studies

The number of interventional studies supporting the benefits of a low-GI diet during pregnancy complicated by GDM is limited. There are three published human studies in the last 3 years directly investigating the effect of a low-GI diet in pregnancy complicated with GDM.

Following the success in reducing the prevalence of LGA infants in healthy pregnancy using a low-GI diet [24], Moses et al. [39••] conducted a study to investigate the effect of a low-GI diet in GDM on postprandial glycemic control. In their study, 63 women with GDM were randomized to follow either a low-GI (n=31) or a conventional high-fiber diet with a higher GI (n=32) from 28 to 32 weeks of pregnancy until delivery. Dietary assessment at 36–37 weeks by a 3-day food record revealed an 8-unit difference in dietary GI between the groups (low-GI group, 48±1, vs. higher GI, 56±1; p=.018). As compared with the low-GI group, a significantly higher proportion of women in the higher GI group met the treatment criteria to commence insulin (low-GI group, 29 %, vs. high-GI group, 59 %; p=.023). For 19 women in the higher GI group who met the criteria to start insulin and were switched to a low-GI diet, 9 of them avoided the need of insulin to maintain optimal glycemic control. This suggests that a low-GI diet is effective in controlling postprandial blood glucose spikes.

Infants of mothers in the low-GI group had similar birth weight and birth weight percentiles as their counterparts in the higher GI group.

A more recent randomized controlled trial by Louie et al. [40••] provided evidence supporting the implication. In that study, 99 women with GDM 26–42 years of age were randomized to follow either a low-GI or conventional high-fiber diet from a mean of 28 weeks of gestation until the end of their pregnancy. The target difference between the diets was 10 unit (50 vs. 60), although at the end, only a 6-unit difference was achieved (47 vs. 53; $p<.001$), possibly due to the high awareness of GI, as well as high education level, among the study population. Women in both groups had similar pregnancy outcomes, including birth weight, birth weight percentile, and prevalence of macrosomia, as well as the need for insulin treatment. The authors concluded that a low-GI diet could be offered as a safe alternative dietary management strategy to women with GDM. Following a low-GI diet from early pregnancy, especially for women at high risk, may be beneficial, because the harm to the fetus caused by maternal hyperglycemia may be done before GDM is diagnosed at the end of second trimester, where universal GDM testing occurs in most countries [41].

A secondary analysis [42] of the baseline food intake data of the study subjects revealed that the majority of them were not meeting the nutritional requirement for fiber, folate, iodine, and iron, as well as having excessive amounts of sodium and folate. The analysis also found that lower dietary GI, but not GL or grain intake, was significantly associated with a higher intake of various micronutrients. That study, albeit having a small sample size, suggested that women with GDM may be at risk of nutrient inadequacy and that following a low-GI diet may reduce the risk.

The latest randomized trial of the effect of low-GI diet in GDM was conducted by Grant et al. [43••]. In their study, 47 pregnant women diagnosed with GDM or impaired glucose tolerance in pregnancy were randomized to follow a low-GI ($n=23$) or a control ($n=24$) diet from 28 weeks of pregnancy until delivery. They were also instructed to self-monitor their fasting and 2-h postprandial blood glucose level, using a home glucose meter, and these were reviewed by a clinician against clinical targets. Dietary assessment by 3-day food records revealed that the low-GI group achieved a 9-unit lower dietary GI than the control group at the end of the intervention (49±0.8 vs. 58 ± 0.5; $p=.001$), and 7 g more fiber (30±1.6 vs. 23±1.0; $p=.001$). They found that subjects in the low-GI group had fewer postprandial blood glucose values above the target (25.9 % vs. 30.3 %; $p=.003$). However, unlike the findings of Moses et al. [39••], there was no significant difference between the proportions of women requiring insulin, which could be due to the different clinical targets used to decide on insulin commencement (1 h postprandial≥8.0 mmol/L in Moses et al. vs. 2 h postprandial> 6.6 mmol/L in Grant et al.). A high-GI meal has been shown to cause a spike in postprandial blood glucose level close to 60 min after meals, with the 2-h postprandial level returning close to the fasting level in diabetic subjects [44]. Therefore, when GI of the foods/meals is concerned, 1-h postprandial blood glucose level may be more relevant. Lastly, unlike in the other studies [39••, 40••], infants of mothers in the low-GI group were ~200 g lower on average (3,124±124 g vs. 3,320±220 g), although this did not reach statistical significance with the number of subjects available.

Implications for Clinical Care

Current evidence, although still limited, suggests that pregnant women with GDM may benefit from following a low-GI meal pattern. A low-GI diet was found to be safe in GDM and produces similar pregnancy outcomes as a conventional high-fiber diet. The studies by Moses et al. [39••] and Grant et al. [43••] provided reasonable evidence that postprandial glycemia in GDM may be better controlled with a low-GI diet. Clinicians may therefore look at replacing high-GI CHO from their patients' diet with low-GI CHO before initiating insulin treatment. Because many nutritious foods, such as dairy foods and fruits, are naturally low GI, it may also be easier to plan a nutritionally adequate GDM diet by choosing more low-GI CHO foods over high-GI, highly processed foods.

Future Directions and Conclusions

On the basis of the current available evidence, we conclude that pregnant women may benefit, with no apparent disadvantage, from following a low-GI meal pattern. By reducing maternal glucose levels within the normal range, a healthy low-GI diet could reduce the risk of adverse pregnancy outcomes. Further studies that intervene at an earlier stage of pregnancy are required.

Disclosure Conflicts of interest: J.C.Y. Louie: none. J.C. Brand-Miller is a co-author of The New Glucose Revolution book series (Hodder and Stoughton, London; Marlowe and Co., NY; Hodder Headline, Sydney; and elsewhere), is the Director of the Glycemic Index Foundation, a not-for-profit that administers a food symbol program in Australia, manages the University of Sydney Glycemic Index testing service, has received honoraria from Pfizer Nutrition Pty Ltd, has received grant support from NHMRC, has received payment for manuscript preparation from Fonterra NZ Pty Ltd, 2012, Inovo Biologic Canada, 2010–2011, and has received travel/accommodations expenses covered or reimbursed from FINEST Conference 2012, A*Star Singapore Government, and Food Structure, Digestion and Health Conference, 2012, Riddett Institute, Massey University, NZ. R.G. Moses: none.

References

Papers of particular interest, published recently, have been highlighted as:
• Of importance
•• Of major importance

1. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2009;32:S62–67.
2. Kaaja R, Rönnemaa T. Gestational diabetes: pathogenesis and consequences to mother and offspring. Rev Diabet Stud. 2008;5:194–202.
3. Damm P, Vestergaard H, Kuhl C, Pedersen O. Impaired insulin-stimulated nonoxidative glucose metabolism in glucosetolerant women with previous gestational diabetes. Am J Obstet Gynecol. 1996;174:722–9.
4. Kautzky-Willer A, Prager R, Waldhausl W, et al. Pronounced insulin resistance and inadequate betacell secretioncharacterize lean gestational diabetes during and after pregnancy. Diabetes Care. 1997;20:1717–23.
5. Setji TL, Brown AJ, Feinglos MN. Gestational diabetes mellitus. Clin Diabetes. 2005;23:17–24.
6. Buchanan TA, Xiang AH, Buchanan TA, Xiang AH. Gestational diabetes mellitus. J Clin Invest. 2005;115:485–91.
7. Morikawa M, Yamada T, Yamada T, et al. Change in the number of patients after the adoption of IADPSG criteria for hyperglycemia during pregnancy in Japanese women. Diabetes Res Clin Pract. 2010;90:339–42.
8. Moses RG, Morris GJ, Petocz P, et al. The impact of potential new diagnostic criteria on the prevalence of gestational diabetes mellitus in Australia. Med J Aust. 2011;194:338–40.
9. O'Sullivan E, Avalos G, O'Reilly M, et al.: Atlantic Diabetes in Pregnancy (DIP): the prevalence and outcomes of gestational diabetes mellitus using new diagnostic criteria. Diabetologia 2011:1–6.
10. International Association of Diabetes and Pregnancy Study Groups Consensus Panel. International Association of Diabetes and Pregnancy Study Groups recommendations on the diagnosis and classification of hyperglycemia in pregnancy. Diabetes Care. 2010;33:676–82.
11. Pedersen J. Weight and length at birth of infants of diabetic mothers. Acta Endocrinol. 1954;16:330–42.
12. Wei JN, Li HY, Sung FC, et al. Birth weight correlates differently with cardiovascular risk factors in youth. Obesity. 2007;15:1609–16.
13. Hillier TA, Pedula KL, Schmidt MM, et al. Childhood obesity and metabolic imprinting: the ongoing effects of maternal hyperglycemia. Diabetes Care. 2007;30:2287–92.
14. Wang X, Liang L, Junfen FU, Lizhong DU. Metabolic syndrome in obese children born large for gestational age. Indian J Pediatr. 2007;74:561–5.
15. Hollander MH, Paarlberg KM, Huisjes AJM. Gestational diabetes: a review of the current literature and guidelines. Obstet Gynecol Surv. 2007;62:125–36.
16. Dornhorst A, Frost G. The principles of dietary management of gestational diabetes: reflection on current evidence. J Hum Nutr Diet. 2002;15:145–56. quiz 157–149.
17. American Diabetes Association. Nutritional management. Medical management of pregnancy complicated by diabetes. 2nd ed. American Diabetes Association Inc: Virginia, America; 1995.
18. Jovanovic-Peterson L, Peterson CM. Dietary manipulation as a primary treatment strategy for pregnancies complicated by diabetes. J Am Coll Nutr. 1990;9:320–5.
19. Romon M, Nuttens MC, Vambergue A, et al. Higher carbohydrate intake is associated with decreased incidence of newborn macrosomia in women with gestational diabetes. J Am Diet Assoc. 2001;101:897–902.
20. Jovanovic L. Achieving euglycaemia in women with gestational diabetes mellitus: current options for screening, diagnosis and treatment. Drugs. 2004;64:1401–17.
21. Lock DR, Bar-Eyal A, Voet H, Madar Z. Glycemic indices of various foods given to pregnant diabetic subjects. Obstet Gynecol. 1988;71:180–3.
22. Clapp 3rd JF. Diet, exercise, and feto-placental growth. Arch Gynecol Obstet. 1997;261:101–7.
23. Scholl TO, Chen X, Khoo CS, Lenders C. The dietary glycemic index during pregnancy: influence on infant birth weight, fetal growth, and biomarkers of carbohydrate metabolism. Am J Epidemiol. 2004;159:467–74.
24. Moses RG, Luebcke M, Davis WS, et al. Effect of a low-glycemic-index diet during pregnancy on obstetric outcomes. Am J Clin Nutr. 2006;84:807–12.
25. Crowther CA, Hiller JE, Moss JR, et al. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. N Engl J Med. 2005;352:2477–86.
26. Metzger BE, Lowe LP, Dyer AR, et al. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med. 2008;358:1991–2002.
27. Metzger BE, Lowe LP, Dyer AR, et al. Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study: Associations with neonatal anthropometrics. Diabetes. 2008;58:453–9.
28. Landon MB, Spong CY, Thom E, et al. A multicenter, randomized trial of treatment for mild gestational diabetes. N Engl J Med. 2009;361:1339–48.
29. Louie JC, Brand-Miller JC, Markovic TP, et al. Glycemic index and pregnancy: a systematic literature review. J Nutr Metab. 2010;2010:282464.
30. Brand-Miller JC, Stockmann K, Atkinson F, et al. Glycemic index, postprandial glycemia, and the shape of the curve in healthy subjects: analysis of a database of more than 1,000 foods. Am J Clin Nutr. 2009;89:97–105.
31. Turner-McGrievy GM, Jenkins DJ, Barnard ND, et al. Decreases in dietary glycemic index are related to weight loss among individuals following therapeutic diets for type 2 diabetes. J Nutr. 2011;141:1469–74.
32. Miller CK, Headings A, Peyrot M, Nagaraja H. A behavioural intervention incorporating specific glycaemic index goals improves dietary quality, weight control and glycaemic control in adults with type 2 diabetes. Public Health Nutr. 2011;14:1303–11.
33. Marsh K, Barclay A, Colagiuri S, Brand-Miller J. Glycemic index and glycemic load of carbohydrates in the diabetes diet. Curr Diab Rep. 2011;11:120–7.
34. Bailey WA, Westman EC, Marquart ML, Guyton JR. Low glycemic diet for weight loss in hypertriglyceridemic patients attending a lipid clinic. J Clin Lipidol. 2010;4:508–14.
35. Larsen TM, Dalskov SM, van Baak M, et al. Diets with high or low protein content and glycemic index for weight-loss maintenance. N Engl J Med. 2010;363:2102–13.
36. Armeno ML, Krochik AG, Mazza CS. Evaluation of two dietary treatments in obese hyperinsulinemic adolescents. J Pediatr Endocrinol Metab. 2011;24:715–22.
37. Zhang C, Liu S, Solomon CG, Hu FB. Dietary fiber intake, dietary glycemic load, and the risk for gestational diabetes mellitus. Diabetes Care. 2006;29:2223–30.
38. • Smith NA, McAuliffe FM, Quinn K, et al. Transient high glycaemic intake in the last trimester of pregnancy increases offspring birthweight and postnatal growth rate in sheep: a randomised

control trial. BJOG. 2009;116:975–83. *This is a novel study demonstrating the potential detrimental effect(s) of a high-GI diet in pregnancy, using a sheep model. Results of this study suggest that a high-GI diet during pregnancy may increase birth weight, as well as postnatal growth rate. Both of these have been positively associated with the risk of obesity later in life*.

39. •• Moses RG, Barker M, Winter M, et al. Can a low-glycemic index diet reduce the need for insulin in gestational diabetes mellitus? A randomized trial. Diabetes Care. 2009;32:996–1000. *This is the first randomized controlled trial to examine the benefits of a low-GI diet in GDM. The authors found that fewer women who followed a low-GI diet during their GDM pregnancy required insulin for optimal postprandial glycemic control and that 9 out of 19 women in the high-GI group who met the criteria to commence insulin treatment avoided insulin simply by switching to a low-GI diet. This novel finding suggests that the use of a low-GI diet may be of benefit to women with GDM, especially in terms of optimal postprandial glycemic control*.
40. •• Louie JC, Markovic TP, Perera N, et al. A randomized controlled trial investigating the effects of a low-glycemic index diet on pregnancy outcomes in gestational diabetes mellitus. Diabetes Care. 2011;34:2341–6. *This study is the first randomized controlled trial adequately powered to examine the effect(s) of a low-GI diet on birth weight of infants born to mothers with GDM. Although the authors failed to find significant differences in birth weight between the low-GI and higher GI groups, it provided data to suggest that an earlier low-GI dietary intervention may be necessary to improve outcomes of GDM pregnancy. It also provided evidence supporting the safety of a low-GI diet in GDM*.
41. Hillier TA, Vesco KK, Pedula KL, et al. Screening for gestational diabetes mellitus: a systematic review for the U.S. Preventive Services Task Force. Ann Intern Med. 2008;148:766–75.
42. Louie JCY, Markovic TP, Foote D, et al. The association between glycemic index, glycemic load, grain intake and nutrient adequacy in gestational diabetes mellitus [Abstract 375]. Presented at *The 16th International Congress of Dietetics*. Sydney, NSW, Australia; September 5–8, 2012.
43. •• Grant SM, Wolever TMS, O'Connor DL, et al. Effect of a low glycaemic index diet on blood glucose in women with gestational hyperglycaemia. Diabetes Res Clin Pract. 2011;91:15–22. *This is the latest randomized controlled trial investigating the effect of a low-GI diet on postprandial glycemic control. The authors found that a low-GI diet had resulted in significantly fewer 2-h postprandial blood glucose values above the treatment target of 6.6 mmol/L, which supports the finding of Moses et al. (39••) that a low-GI diet results in better glycemic control*.
44. Liatis S, Grammatikou S, Poulia KA, et al. Vinegar reduces postprandial hyperglycaemia in patients with type II diabetes when added to a high, but not to a low, glycaemic index meal. Eur J Clin Nutr. 2010;64:727–32.