

A Randomized Controlled Trial Investigating the Effects of a Low-Glycemic Index Diet on Pregnancy Outcomes in Gestational Diabetes Mellitus

JIMMY CHUN YU LOUIE, MNUTRDIET, APD¹
TANIA P. MARKOVIC, MBBS, PHD²
NIMALIE PERERA, MBBS²
DEBORAH FOOTE, BSC DIPNUTRDIET, APD³

PETER PETOCZ, PHD⁴
GLYNIS P. ROSS, MBBS²
JENNIE C. BRAND-MILLER, PHD¹

OBJECTIVE—The prevalence of gestational diabetes mellitus (GDM) is rising. There is little evidence to demonstrate the effectiveness of one dietary therapy over another. We aimed to investigate the effect of a low-glycemic index (LGI) versus a conventional high-fiber diet on pregnancy outcomes, neonatal anthropometry, and maternal metabolic profile in GDM.

RESEARCH DESIGN AND METHODS—Ninety-nine women (age 26–42 years; mean \pm SD prepregnancy BMI 24 ± 5 kg/m²) diagnosed with GDM at 20–32 weeks' gestation were randomized to follow either an LGI ($n = 50$; target glycemic index [GI] ~ 50) or a high-fiber moderate-GI diet (HF) ($n = 49$; target GI ~ 60). Dietary intake was assessed by 3-day food records. Pregnancy outcomes were collected from medical records.

RESULTS—The LGI group achieved a modestly lower GI than the HF group (mean \pm SEM 47 ± 1 vs. 53 ± 1 ; $P < 0.001$). At birth, there was no significant difference in birth weight (LGI 3.3 ± 0.1 kg vs. HF 3.3 ± 0.1 kg; $P = 0.619$), birth weight centile (LGI 52.5 ± 4.3 vs. HF 52.2 ± 4.0 ; $P = 0.969$), prevalence of macrosomia (LGI 2.1% vs. HF 6.7%; $P = 0.157$), insulin treatment (LGI 53% vs. HF 65%; $P = 0.251$), or adverse pregnancy outcomes.

CONCLUSIONS—In intensively monitored women with GDM, an LGI diet and a conventional HF diet produce similar pregnancy outcomes.

Diabetes Care 34:2341–2346, 2011

Gestational diabetes mellitus (GDM) is commonly defined as any degree of glucose intolerance with onset or first recognition during pregnancy (1). In developed nations, between 4 and 8% of pregnant women are presently affected (2–4), and the prevalence will rise dramatically if the guidelines of the new International Association of Diabetes in Pregnancy Study Groups (IADPSG) are adopted (3). The main adverse outcome

of GDM is excessive fetal growth resulting in higher risk of large-for-gestational-age (LGA) infants (birth weight >90 th centile). Higher birth weight has been linked with childhood obesity (5), cardiovascular disease (6), and diabetes (5) later in life.

In the medical management of GDM, the primary goal is to maintain maternal blood glucose concentrations, especially postprandial levels, within an acceptable range (7). Interventions that

reduce postprandial glucose levels, including dietary strategies such as carbohydrate restriction, have been shown to be effective in reducing LGA and later obesity in type 1 diabetic offspring (8).

Postprandial glycemia can be reduced without carbohydrate restriction by slowing down the rate of carbohydrate digestion and absorption. Compared with moderate- or high-glycemic index (GI) foods containing similar amount of carbohydrates, low-GI (LGI) foods have been demonstrated to reduce postprandial glucose in healthy individuals (9). The GI of various foods has been shown to be the same in pregnancy as in the nonpregnant state (10). An LGI meal pattern may therefore represent an alternative strategy for reducing postprandial glycemia in GDM without restricting carbohydrate (11).

The effect of an LGI eating pattern on obstetric outcomes in GDM has been little studied. Moses et al. (12) found that a significantly higher proportion of women in the higher-GI group met the criteria to commence insulin compared with women in the LGI group. In addition, 47% of the women in the high-GI group who met the criteria for insulin commencement avoided insulin by switching to an LGI diet. However, they found no significant differences in key fetal and obstetric outcomes.

To our knowledge, this study is the first randomized controlled trial to determine the efficacy of an LGI diet versus a conventional healthy diet on pregnancy outcomes in GDM. Our hypothesis was that an LGI diet would reduce birth weight (primary end point), birth weight centile, ponderal index, and the prevalence of LGA infants.

RESEARCH DESIGN AND METHODS

This study was a two-arm parallel randomized controlled trial based at the Diabetes Antenatal Clinic of the Royal Prince Alfred Hospital, Camperdown, Australia. With the exception of the study dietitian (J.C.Y.L.), who provided the

From the ¹School of Molecular Bioscience and Boden Institute of Obesity, Nutrition, Exercise and Eating Disorders, University of Sydney, Sydney, Australia; the ²Department of Endocrinology, Royal Prince Alfred Hospital, Camperdown, Australia; the ³Department of Nutrition and Dietetics, Royal Prince Alfred Hospital, Camperdown, Australia; and the ⁴Department of Statistics, Macquarie University, Sydney, Australia.

Corresponding author: Jennie C. Brand-Miller, jennie.brandmiller@sydney.edu.au.

Received 26 May 2011 and accepted 29 July 2011.

DOI: 10.2337/dc11-0985. Clinical trial reg. no. ACTRN12608000218392, www.anzctr.org.au.

This article contains Supplementary Data online at <http://care.diabetesjournals.org/lookup/suppl/doi:10.2337/dc11-0985/-/DC1>.

© 2011 by the American Diabetes Association. Readers may use this article as long as the work is properly cited, the use is educational and not for profit, and the work is not altered. See <http://creativecommons.org/licenses/by-nc-nd/3.0/> for details.

dietary education, all study personnel and participants were blinded to dietary assignment.

Subject recruitment, randomization, and stratification

Women aged 18–45 years diagnosed with GDM by a 75-g oral glucose tolerance test at 20–32 weeks' gestation, with an otherwise healthy singleton pregnancy, were eligible for the study. GDM diagnosis was based on the modified Australasian Diabetes in Pregnancy Society (ADIPS) criteria: fasting blood glucose level (BGL) ≥ 5.5 mmol/L, 1-h BGL ≥ 10.0 mmol/L, or 2-h BGL ≥ 8.0 mmol/L. Most women were tested at 26–32 weeks, but testing occurred earlier in those at high risk. Women who had special dietary requirements (including vegetarianism/veganism), preexisting diabetes, or pregnancy achieved by assisted reproduction and those who smoked or consumed alcohol during pregnancy were excluded. A total of 482 women were approached between September 2008 and November 2010, of whom 99 met the inclusion criteria and agreed to participate. The enrolled subjects were centrally randomized to study diet by computer-generated random numbers, stratified by BMI (BMI < 30 vs. ≥ 30 kg/m²) and weeks of gestation (< 28 or ≥ 28 weeks). The allocation sequence was unpredictable and concealed from the recruiter. Participants received routine GDM care regardless of dietary assignment, including instructions to monitor BGL before breakfast and 1 h after meals. The treating endocrinologist (T.P.M. or N.P.) reviewed the subjects every 2–4 weeks prior to 36 weeks and then every week until delivery. Insulin treatment was commenced if the mean fasting BGL or 1-h postprandial BGL in the preceding week exceeded 5.2 and 7.5 mmol/L, respectively.

Demographics and dietary assessment

At enrollment, demographic information, family history of diabetes, and ethnicity were recorded. Subjects were asked to recall their prepregnancy weight, were weighed, and were asked to complete a 3-day food record (including 2 weekdays and 1 weekend day) at baseline and again at 36–37 weeks' gestation. A two-dimensional food model booklet was provided to the subjects to assist in portion size estimation. Last recorded weight before delivery was obtained from the medical record.

Dietary interventions

Subjects were randomized to one of two healthy diets of similar protein (15–25%),

fat (25–30%), and carbohydrate (40–45%) content—one with an LGI (target GI ≤ 50) and the other with a high-fiber content and moderate GI, similar to the Australian population average (HF) (target GI ~ 60) (13–15). Both study diets provided all essential nutrients for pregnancy other than iron and iodine, which were supplemented as appropriate by the treating endocrinologist. The baseline 3-day food diary provided information on baseline dietary composition and served as the basis of individualized dietary counseling. Sample menus and their nutritional analyses are given in Supplementary Table A1.

Subjects attended at least three face-to-face visits with the study dietitian, scheduled to coincide with regular antenatal visits. A 24-h recall of all food and drink intake was conducted during each session to assess compliance. In the case of noncompliance, suitable alternative foods were encouraged. Food sample baskets containing key foods for the assigned diet were provided to promote product recognition and dietary adherence. The content of the sample baskets is listed in Supplementary Table A2.

Data collection

Subjects provided blood samples at baseline and ~ 36 weeks' gestation. Pregnancy outcomes, including birth weight, infant length, infant head circumference, and the need for emergency caesarean section, were obtained from the electronic medical records system. Gestational age was based on the last menstrual period and early pregnancy ultrasound. Birth weight centile was calculated using a macro program for Microsoft Excel (available from <http://www.gestation.net>) that adjusted for ethnic differences (16). The calculated birth weight centile was used to categorize the infant as small for gestational age (birth weight < 10 th centile), normal, or LGA (birth weight > 90 th centile). Ponderal index, an estimate of neonatal adiposity, was calculated as birth weight in kg \times infant length (m)⁻³. Macrosomia was defined as birth weight > 4 kg.

Nutritional analysis and assessment of compliance

The study dietitian entered the food records into Australian nutrition analysis software based on AUSNUT2001 (FoodWorks Professional 2009; Xyris Software, Brisbane, Australia). The GI of individual food items was assigned according to a published method (17). Dietary glycemic

load (GL) was calculated as follows: \sum GI \times available carbohydrate of each food in a day/100. Dietary GI was calculated as follows: (dietary GL/total daily available carbohydrate) $\times 100$. Subjects were deemed compliant if their final dietary GI was ≤ 50 in the LGI group and > 50 in the HF group.

Power calculation

Based on previous data, the study was designed to provide 80% statistical power to detect an ~ 260 g difference in birth weight, with 60 subjects in each group. Recruitment was halted at 99 subjects because the SD in birth weight among the study population was smaller than expected. In the primary analysis, the observed SD of 416 g in birth weight provided 80% power to detect a group difference of 246 g in birth weight or an $\sim 17\%$ point difference in birth weight centile.

Statistical analyses

A biostatistician blinded to the diet allocation performed the statistical analyses. The primary analysis included all women randomized who attended at least one dietary education session but excluded those with preterm delivery (< 37 weeks; $n = 4$; two from each group) regardless of compliance. All statistical analyses were performed with SPSS (version 19; IBM Australia, St. Leonards, Australia). Results for continuous data are reported as mean \pm SD or mean \pm SEM, and categorical data (e.g., need for insulin) are reported as percentage. Pearson χ^2 test was used to test for differences between groups for categorical data, and continuous data were tested using one-way ANOVA. A paired t test was used to assess within-group changes from baseline to final outcomes.

The study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects/patients were approved by the Human Research Ethics Committee of the Sydney South West Area Health Service (Royal Prince Alfred Hospital Zone). Informed consent was obtained from all subjects in this study.

RESULTS—The flow of subjects through the study is shown in Supplementary Fig. 1. Of the 99 subjects recruited, four delivered prematurely (< 37 weeks) and three withdrew before the first dietary instruction session, leaving 92 subjects in the primary analysis. Subject characteristics are shown in Table 1.

Table 1—Subject characteristics

	LGI	HF	<i>P</i> *
<i>n</i>	47	45	—
Age (years)	34.0 ± 4.1	32.4 ± 4.5	0.062
Prepregnancy BMI (kg/m ²)	23.9 ± 4.4	24.1 ± 5.7	0.837
Ethnicity (%)			
Asian	59.6	55.6	0.697
Caucasian	31.9	40.0	0.419
Others	8.5	4.4	0.430
Week of gestation at diagnosis	26.1 ± 4.0	26.0 ± 4.3	0.951
Family history of type 2 diabetes (%)			
Maternal	23.4	20.0	0.692
Paternal	21.3	33.3	0.194
Week of gestation at start of intervention	29.0 ± 4.0	29.7 ± 3.5	0.410
75-g OGTT results (mmol/L)			
Fasting	4.6 ± 0.5	4.7 ± 0.7	0.279
1 h	9.4 ± 1.4	9.7 ± 1.6	0.501
2 h	8.6 ± 1.2	8.0 ± 1.3	0.024
Nulliparous (%)	61.7	64.4	0.785

Data are mean ± SD except for ethnicity, family history of type 2 diabetes, and nulliparous, which are expressed as percentages. OGTT, oral glucose tolerance test. **P* values calculated by one-way ANOVA for continuous variables and Pearson χ^2 for categorical variables. *P* < 0.05 indicates statistical significance.

At baseline, subjects in the LGI group had significantly higher 2-h postload blood glucose levels (LGI 8.6 ± 1.2 mmol/L vs. HF 8.0 ± 1.3 mmol/L; *P* = 0.024) but were otherwise similar to those in the HF group. At baseline, both groups had a relatively LGI diet (LGI 49 ± 1 vs. HF 52 ± 2) (Table 2). At the end of the intervention (36–37 weeks' gestation), the diets were matched for macro- and micronutrients, but the LGI group had a significantly lower GI and GL than the HF group as per protocol (both *P* < 0.001). Compared with data at baseline, intake of fat, fiber,

calcium, iron, zinc, and folate significantly increased in subjects in the LGI group. Subjects in the HF group had increased energy intake and GL but not GI. The results were similar in the secondary analysis of “compliers” only except that compliers in the LGI group (*n* = 30) had significantly decreased their GI, whereas those in the HF group (*n* = 34) remained unchanged from baseline (data not shown).

At the end of the intervention, biochemical parameters were similar between groups (Table 3). The results were

similar in the compliers-only analysis (data not shown). In the primary analysis, there were no significant differences between groups in any of the pregnancy outcomes (Table 4). Fewer women in the LGI group gained an excessive amount of weight according to the American Institute of Medicine guidelines (LGI 25% vs. HF 42%; *P* = 0.095). Compliers in the LGI group appeared to gain less weight than those in the HF group (LGI 11.2 ± 0.9 kg vs. HF 13.7 ± 1.0 kg; *P* = 0.073). There was no significant difference in fetal abdominal circumference at 36–37 weeks' gestation (mean ± SEM LGI 327.6 ± 19.2 mm vs. HF 322.6 ± 14.6 mm; *P* = 0.186). Additional analyses with adjustments for ethnicity (Asian vs. Caucasian), BMI, oral glucose tolerance test results; baseline characteristics including daily intakes of energy, monounsaturated fatty acid, polyunsaturated fatty acid, and sodium; fasting BGL; fasting insulin; homeostasis model assessment of insulin resistance; and total cholesterol did not change the lack of significance of the between-group comparisons.

CONCLUSIONS—Contrary to our hypothesis, this randomized controlled trial of an LGI diet versus a conventional high-fiber diet found no differences in key pregnancy outcomes in GDM. Average infant birth weight, birth weight centile, and ponderal index were within healthy norms in both groups. One explanation for the findings is that both groups of women achieved a relatively LGI diet, with only a modest 5-point difference between

Table 2—Baseline and end-of-intervention diet analysis

	Baseline		<i>P</i> *	End of intervention		<i>P</i> *	<i>P</i> †	
	LGI	HF		LGI	HF		LGI	HF
<i>n</i>	44	40		42	42			
Energy (kJ)	7,240 ± 240	6,630 ± 260	0.089	7,680 ± 260	8,090 ± 300	0.307	0.141	<0.001
Protein (g)	99.2 ± 4.4	93.1 ± 5.4	0.389	107.5 ± 4.2	107.2 ± 5.6	0.971	0.100	0.049
Total fat (g)	70.2 ± 3.7	61.4 ± 3.1	0.073	71.2 ± 3.5	75.3 ± 5.6	0.532	<0.001	0.029
Saturated fat (g)	23.5 ± 1.3	22.3 ± 1.4	0.553	24.2 ± 1.3	28.8 ± 3.1	0.181	0.515	0.090
Monounsaturated fat (g)	27.0 ± 1.7	22.4 ± 1.3	0.035	27.4 ± 1.6	26.8 ± 1.7	0.797	0.887	0.034
Polyunsaturated fat (g)	12.6 ± 0.9	10.1 ± 0.7	0.032	13.5 ± 0.8	12.5 ± 1.3	0.488	0.465	0.065
Total available carbohydrate (g)	165.1 ± 5.4	155.0 ± 7.9	0.289	177.8 ± 5.9	194.8 ± 6.2	0.051	0.066	<0.001
Sugars (g)	59.1 ± 3.3	56.0 ± 15.5	0.470	66.9 ± 4.1	70.5 ± 2.7	0.464	0.087	<0.001
Starch (g)	105.0 ± 3.5	99.6 ± 8.1	0.523	111.1 ± 4.1	124.6 ± 5.6	0.056	0.190	0.001
Dietary fiber (g)	23 ± 1	21 ± 1	0.245	27 ± 1	25 ± 1	0.222	0.001	0.012
Calcium (mg)	887 ± 54	915 ± 41	0.680	1,080 ± 62	1,030 ± 43	0.507	0.005	0.013
GI	49 ± 1	52 ± 2	0.171	47 ± 1	53 ± 1	<0.001	0.187	0.600
GL	81 ± 3	84 ± 6	0.598	84 ± 3	105 ± 4	<0.001	0.453	0.001

Data are mean ± SEM. **P* values calculated by one-way ANOVA to test for difference between groups. †*P* values calculated by paired sample *t* test to test for difference compared with baseline.

Table 3—Biochemical parameters at baseline and end of intervention

	Baseline					End of intervention				
	LGI		HF		P*	LGI		HF		P*
	n	Mean ± SEM	n	Mean ± SEM		n	Mean ± SEM	n	Mean ± SEM	
BGL (mmol/L)	44	4.7 ± 0.1	42	4.6 ± 0.1	0.665	42	4.3 ± 0.1	32	4.4 ± 0.1	0.464
Insulin (pmol/L)	44	73.1 ± 9.4	42	70.5 ± 5.3	0.813	40	83.8 ± 16.1	30	73.0 ± 5.2	0.525
HOMA2-IR (%)	44	1.3 ± 0.2	42	1.3 ± 0.1	0.780	38	1.2 ± 0.1	39	1.3 ± 0.1	0.670
Fructosamine (μmol/L)	43	202.3 ± 2.5	41	199.9 ± 2.3	0.479	41	196.3 ± 2.3	40	193.7 ± 2.2	0.412
HbA _{1c} (%)	44	5.4 ± 0.1	42	5.4 ± 0.1	0.995	43	5.5 ± 0.1	41	5.5 ± 0.0	0.665

HOMA2-IR, homeostasis model assessment of insulin resistance. *P values calculated by one-way ANOVA to test for difference between groups.

groups. Irrespective of dietary assignment, all had received early nutrition counseling in a group setting. Thus, on enrollment, both groups were found to be consuming a diet with a lower GI than population norms. Compared with routine care in another Australian study (18), both dietary interventions resulted in a lower prevalence of LGA (9 vs. 22%), macrosomia (4 vs. 21%), and emergency caesarean section (16 vs. 20%). Hence, in the setting of intensive medical management of GDM, our findings suggest that both an LGI and HF diet produce optimal pregnancy outcomes.

Our findings increase the evidence supporting the safety and efficacy of an LGI diet in GDM. Moses et al. (12) also found no significant differences in key

fetal and obstetric outcomes between subjects who followed an LGI diet (GI = 48) versus a higher-GI diet (GI = 56). However, unlike in the current study, they found that a significantly higher proportion of women in the higher-GI group met the criteria to commence insulin (59 vs. 29% in the LGI group). In addition, almost one-half of the women in the higher-GI group who met the criteria for insulin commencement avoided insulin by switching to an LGI diet. Their insulin treatment protocol, however, was different from that of the current study, in which more stringent criteria were used as the basis for insulin treatment.

A recent Canadian study (19), in which women with GDM or impaired glucose tolerance monitored their own

blood glucose levels, found that those who were randomized to an LGI diet versus those assigned to the conventional diet had a greater proportion of their 2-h postprandial levels on or below the treatment target. Although there was a tendency for higher birth weight in the control group, the study was a pilot and underpowered to detect a statistically significant difference.

Another explanation for our findings is the relatively normal weight of most of our subjects (68% had a BMI <25 kg/m²). It is possible that an LGI diet may be more effective among overweight and obese gravidas with higher degrees of insulin resistance and β-cell deficiency (20). Rhodes et al. (21) reported higher head circumference and a lower proportion of early delivery (<38 weeks' gestation) in overweight and obese nondiabetic pregnant women assigned to a low GL diet. However, there was no significant difference in birth weight, ponderal index, or pregnancy weight gain, which are more sensitive to maternal glycemic control (22).

The lack of difference in our study may also relate to the timing and duration of the intervention. Dietary instruction began at the start of the third trimester (29 weeks' gestation) and lasted, on average, 6–7 weeks. It is likely that maternal hyperglycemia during the first and second trimester will also drive excessive fetal growth. In a post hoc analysis of women who started dietary intervention before 25 weeks of gestation (10 from the LGI group and 5 from the HF group), those in the LGI group showed a tendency to lower birth weight (LGI 3.2 ± 0.2 kg vs. HF 3.5 ± 0.1 kg; P = 0.224) and lower birth centile (LGI 45.3 ± 11.0 vs. HF 57.5 ± 12.2; P = 0.476), suggesting that an earlier intervention may be beneficial. However, apart from a small number of high-risk women who are screened early, in most countries GDM screening occurs

Table 4—Pregnancy outcomes by diet group

	LGI		HF		P*
	n	Value	n	Value	
Gestational age (weeks)	47	39.1 ± 0.1	45	39.2 ± 0.1	0.552
Birth weight (kg)	47	3.3 ± 0.1	45	3.3 ± 0.1	0.619
Birth weight centile	47	52.5 ± 4.3	45	52.2 ± 4.0	0.969
LGA (%)	47	12.8	45	4.4	0.157
Small for gestational age (%)	47	10.6	45	8.9	0.778
Macrosomia (%)	47	2.1	45	6.7	0.286
Infant head circumference (cm)	43	34.4 ± 0.2	39	34.6 ± 0.3	0.478
Infant length (cm)	47	49.7 ± 0.3	45	49.7 ± 0.3	0.995
Ponderal index (kg/m ³)	47	27.2 ± 0.3	45	27.0 ± 0.4	0.614
Maternal weight gain (kg)	44	11.9 ± 0.7	43	13.1 ± 0.9	0.305
Below target (%)†		31.8		25.6	0.520
Within target (%)†		43.2		32.6	0.307
Above target (%)†		25.0		41.9	0.095
Insulin treatment (%)	47	53.2	45	65.1	0.251
Final daily insulin dose (units)	47	17.7 ± 4.1	43	20.0 ± 3.8	0.676
Emergency caesarean (%)	44	20.5	44	11.6	0.263

Data are mean ± SEM or percent. *P values calculated by one-way ANOVA for continuous variables and Pearson χ² for categorical variables. P < 0.05 indicates statistical significance. †Based on Institute of Medicine. Weight gain during pregnancy: reexamining the guidelines [article online], 2009. Available from <http://www.iom.edu/-/media/files/report%20files/2009/weight-gain-during-pregnancy-reexamining-the-guidelines/report%20brief%20-%20weight%20gain%20during%20pregnancy.pdf>.

at 26–28 weeks' gestation (23,24), which means that any intervention in GDM will be necessarily short. A more viable test of our hypothesis would therefore be an appropriately powered study in women at high risk of developing GDM (e.g., women with a BMI >30 kg/m² or previous GDM), starting on or before the start of the second trimester, to determine the effect of an LGI diet on both pregnancy outcome and risk of developing GDM.

The failure to achieve the target GI of ~60 in the HF group could reflect high recognition of the GI concept among Australians diagnosed with diabetes, particularly among those with higher education (in the current study, two of three subjects had a university degree). In the group education session conducted soon after diagnosis, all the women, irrespective of future dietary assignment, were encouraged to limit total carbohydrate to ~180 g per day and to consume a greater proportion as fruit and dairy products—changes which are likely to lower the GI of the overall diet. Self-monitoring of blood glucose levels was also encouraged and may have provided feedback that discouraged consumption of high glycemic foods. Finally, the use of data collected from medical record may be subject to inaccuracy, e.g., birth weights were measured and entered by different staff, therefore biasing the result toward the null hypothesis.

In conclusion, we found that both an LGI diet and a conventional high-fiber diet produced comparable pregnancy outcomes in women with GDM. Both groups achieved a relatively low GI diet and had mean birth weight, birth weight centile, and pregnancy weight gain within population norms. An LGI diet appears to be a safe alternative to the traditional pregnancy diet for women with GDM and expands the range of dietary strategies that can be offered. Further studies in overweight and obese individuals and earlier interventions in women with risk factors for GDM are warranted.

Acknowledgments—This study was funded by Australian National Health and Medical Research Council Project grant ID632889.

J.C.B.-M. is a coauthor of *The New Glucose Revolution* book series, the director of a not-for-profit GI-based food endorsement program in Australia, and manages the University of Sydney GI testing service. The authors thank Roche Diagnostics Australia for donation of

glucose meters. No other potential conflicts of interest relevant to this article were reported.

J.C.Y.L. was involved in the conception and design of the study, designed the study diets, provided dietary education to the study participants, collected data, drafted the manuscript, was involved in the interpretation of data, was involved in the subsequent edits to the manuscript, and read and approved the final manuscript. T.P.M. was involved in the conception and design of the study, provided medical management to the study participants, was involved in the interpretation of data, was involved in the subsequent edits to the manuscript, and read and approved the final manuscript. N.P. was involved in the conception and design of the study, provided medical management to the study participants, assisted in the blood pathology analyses, was involved in the interpretation of data, was involved in the subsequent edits to the manuscript, and read and approved the final manuscript. D.F. was involved in the conception and design of the study, designed the study diets, was involved in the interpretation of data, was involved in the subsequent edits to the manuscript, and read and approved the final manuscript. P.P. was involved in the conception and design of the study, analyzed data, was involved in the subsequent edits to the manuscript, and read and approved the final manuscript. J.C.B.-M. was involved in the conception and design of the study, designed the study diets, was involved in the interpretation of data, was involved in the subsequent edits to the manuscript, and read and approved the final manuscript.

The authors thank Ms. Dawn Tan of the Discipline of Nutrition and Metabolism, School of Molecular Bioscience, The University of Sydney, Sydney, Australia, for her help in the GI assignment process and thank Uncle Toby's Australia and George Weston Foods Australia for their donations of sample foods.

References

1. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care* 2009;32(Suppl. 1):S62–S67
2. Lamberg S, Raitanen J, Rissanen P, Luoto R. Prevalence and regional differences of gestational diabetes mellitus and oral glucose tolerance tests in Finland. *Eur J Public Health*. 23 December 2010 [Epub ahead of print]
3. Moses RG, Morris GJ, Petocz P, San Gil F, Garg D. The impact of potential new diagnostic criteria on the prevalence of gestational diabetes mellitus in Australia. *Med J Aust* 2011;194:338–340
4. Ferrara A. Increasing prevalence of gestational diabetes mellitus: a public

health perspective. *Diabetes Care* 2007;30 (Suppl. 2):S141–S146

5. Wei JN, Li HY, Sung FC, et al. Birth weight correlates differently with cardiovascular risk factors in youth. *Obesity (Silver Spring)* 2007;15:1609–1616
6. Wang X, Liang L, Junfen FU, Lizhong DU. Metabolic syndrome in obese children born large for gestational age. *Indian J Pediatr* 2007;74:561–565
7. Hoffman L, Nolan C, Wilson JD, Oats JJ, Simmons D; The Australasian Diabetes in Pregnancy Society. Gestational diabetes mellitus—management guidelines. *Med J Aust* 1998;169:93–97
8. Hillier TA, Pedula KL, Schmidt MM, Mullen JA, Charles M-A, Pettitt DJ. Childhood obesity and metabolic imprinting: the ongoing effects of maternal hyperglycemia. *Diabetes Care* 2007;30:2287–2292
9. Brand-Miller JC, Stockmann K, Atkinson F, Petocz P, Denyer G. 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
10. Lock DR, Bar-Eyal A, Voet H, Madar Z. Glycemic indices of various foods given to pregnant diabetic subjects. *Obstet Gynecol* 1988;71:180–183
11. Louie JC, Brand-Miller JC, Markovic TP, Ross GP, Moses RG. Glycemic index and pregnancy: a systematic literature review. *J Nutr Metab* 2010;2010:282464
12. 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. *Diabetes Care* 2009;32:996–1000
13. Barclay AW, Brand-Miller JC, Mitchell P. Macronutrient intake, glycaemic index and glycaemic load of older Australian subjects with and without diabetes: baseline data from the Blue Mountains Eye study. *Br J Nutr* 2006;96:117–123
14. O'Sullivan TA, Bremner AP, O'Neill S, Lyons-Wall P. Glycaemic load is associated with insulin resistance in older Australian women. *Eur J Clin Nutr* 2010;64:80–87
15. O'Sullivan TA, Lyons-Wall P, Bremner AP, et al. Dietary glycaemic carbohydrate in relation to the metabolic syndrome in adolescents: comparison of different metabolic syndrome definitions. *Diabet Med* 2010; 27:770–778
16. Mongelli M, Figueras F, Francis A, Gardosi J. A customized birthweight centile calculator developed for an Australian population. *Aust N Z J Obstet Gynaecol* 2007;47: 128–131
17. Louie JC-Y, Flood V, Turner N, Everingham C, Gwynn J. Methodology for adding glycemic index values to 24-hour recalls. *Nutrition* 2011;27:59–64
18. Crowther CA, Hiller JE, Moss JR, McPhee AJ, Jeffries WS, Robinson JS; Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS) Trial Group. Effect

- of treatment of gestational diabetes mellitus on pregnancy outcomes. *N Engl J Med* 2005;352:2477–2486
19. 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. *Diabetes Res Clin Pract* 2011; 91:15–22
 20. Catalano PM. Management of obesity in pregnancy. *Obstet Gynecol* 2007;109: 419–433
 21. Rhodes ET, Pawlak DB, Takoudes TC, et al. Effects of a low-glycemic load diet in overweight and obese pregnant women: a pilot randomized controlled trial. *Am J Clin Nutr* 2010;92:1306–1315
 22. Metzger BE, Lowe LP, Dyer AR, et al.; HAPO Study Cooperative Research Group. Hyperglycemia and adverse pregnancy outcomes. *N Engl J Med* 2008;358:1991–2002
 23. Hillier TA, Vesco KK, Whitlock EP, Pettitt DJ, Pedula KL, Beil TL. *Screening for Gestational Diabetes Mellitus: U.S. Preventive Services Task Force Evidence Syntheses*. Rockville, MD, Agency for Healthcare Research and Quality, 2008, p. 60
 24. Australasian Diabetes In Pregnancy Society. Gestational diabetes mellitus—management guidelines [article online], 2002. Available from <http://www.ranzcog.edu.au/publications/statements/coll-end-statements/ADIPS-gdm-management-guidelines.pdf>. Accessed 15 April 2011