Pregnancy and Glycemic Index Outcomes study: effects of low glycemic index compared with conventional dietary advice on selected pregnancy outcomes1–3

Robert G Moses, Shelly A Casey, Eleanor G Quinn, Jane M Cleary, Linda C Tapsell, Marianna Milosavljevic, Peter Petocz, and Jennie C Brand-Miller

ABSTRACT

Background: Eating carbohydrate foods with a high glycemic index (GI) has been postulated to result in fetoplacental overgrowth and higher infant body fat. A diet with a low glycemic index (LGI) has been shown to reduce birth percentiles and the ponderal index (PI).

Objectives: We investigated whether offering LGI dietary advice at the first antenatal visit would result in a lower fetal birth weight, birth percentile, and PI than providing healthy eating (HE) advice. This advice had to be presented within the resources of routine antenatal care.

Design: The Pregnancy and Glycemic Index Outcomes study was a 2-arm, parallel-design, randomized, controlled trial that compared the effects of LGI dietary advice with HE advice on pregnancy outcomes. Eligible volunteers who attended for routine antenatal care at <20 wk of gestation were randomly assigned to either group.

Results: A total of 691 women were enrolled, and 576 women had final data considered. In the LGI group, the GI was reduced from a mean (±SEM) of 56 ± 0.3 at enrollment to 52 ± 0.3 (P < 0.001) at the final assessment. There were no significant differences in primary outcomes of fetal birth weight, birth percentile, or PI. In a multivariate regression analysis, the glycemic load was the only significant dietary predictor (P = 0.046) of primary outcomes but explained <1% of all variation.

Conclusion: A low-intensity dietary intervention with an LGI diet compared with an HE diet in pregnancy did not result in any significant differences in birth weight, fetal percentile, or PI. This trial was registered at https://www.anzctr.org.au as ACTRN12610000174088.


INTRODUCTION

Prolonged effects of maternal nutrition on the intrauterine environment, perhaps exerted through epigenetic changes (1), have been established in animals and humans (2). Infants at the highest end of the distribution for birth weight or BMI are more likely to be obese in childhood, adolescence, and early adulthood than are other infants (3) and have greater risk of cardiovascular and metabolic complications in later life (4). Glucose is the main energy substrate for fetal growth (5), and different degrees of impaired glucose metabolism influence the fetal body composition, including body fat (6). There is no apparent threshold for these abnormalities to develop but, rather, a continuum of risk between maternal glucose concentrations (7) and the incidence of high birth weight and its inherent complications (8).

Maternal diet, particularly the quantity and quality of carbohydrate, influences maternal blood glucose concentrations (9). Different carbohydrate foods produce different glycemic responses. Jenkins et al (10) developed the glycemic index (GI)4 in 1981 as a method of ranking the postprandial glycemic response to equivalent portions of carbohydrate in different foods. A diet that is based on high-fiber, low-glycemic index (LGI) food choices has been shown to blunt the increase in insulin resistance in mid and late pregnancy typically seen in Westernized societies (11). The consumption of primarily carbohydrate foods with a high GI has been postulated to result in fetoplacental overgrowth and excessive maternal weight gain and lead to a higher infant body fat, whereas the intake of carbohydrate with an LGI predisposes individuals to a normal infant birth weight and normal maternal weight gain (12, 13). Because fetal birth weight and the ponderal index (PI) may predict chronic disease in later life, a low-GI diet may favorably influence child obesity and long-term outcomes.

In a small, relatively intensive study of 62 women with 5 dietary instruction visits, we have shown that an LGI diet consumed during the second and third trimester in healthy mothers reduced fetal birth weight, the fetal percentile, and the PI (13). However, to our knowledge, the effect of introducing an LGI diet,
with dietary resources that would be clinically reasonable in an antenatal environment, has not been subject to a large, randomized, controlled trial. We hypothesized that LGI dietary advice at the first antenatal visit would improve the fetal percentile and PI. For this purpose, the Pregnancy and Glycemic Index Outcomes (PREGGIO) study (https://www.anzctr.org.au; ACTRN12610000174088) was initiated.

SUBJECTS AND METHODS

The PREGGIO study was a 2-arm, parallel-designed, randomized, controlled trial that compared effects of 2 dietary advice strategies on selected pregnancy outcomes. The study was approved by the South Eastern Sydney Illawarra Area Health Service and University of Wollongong Human Research Ethics Committee (CT09/007).

Pregnant women who attended for their initial obstetric or midwife appointment at a publicly funded antenatal clinic (Wollongong Hospital) (public) with an intention to deliver in that hospital or with 2 private obstetricians with the intention of being delivered at the Illawarra Private Hospital (private) were invited to participate. Women were eligible if they met all of the following criteria: enrollment at <20 wk of gestation with a singleton pregnancy, age ≥18 y, ability to read and understand a consent form in English, and ability to comply with visit schedules. Exclusion criteria were known diabetes or previous gestational diabetes (GD), special dietary needs, the presence of medical conditions that could compromise the metabolic status (eg, thyroid disorders), or the use of medications that were likely to influence body weight. The medical and obstetric management (eg, serving sizes and brands, cooking methods, and ingredients) was clarified. Women were asked to estimate portions and their individual requirements for pregnancy (ie, that met energy needs and nutrient requirements). There was no intended transition period, was based on the International Association of Diabetes in Pregnancy Study Group criteria (15), which were later adopted by the Australasian Diabetes in Pregnancy Society (16). Women with diagnosed GD were withdrawn from the study and treated conventionally.

Testing for GD

All women were tested for GD at 24–28 wk of gestation. In 2010, the test was based on the Australasian Diabetes in Pregnancy Society criteria (14) and, from January 2011 (with a short transition period), was based on the International Association of Diabetes in Pregnancy Study Group criteria (15), which were later adopted by the Australasian Diabetes in Pregnancy Society (16). Women with diagnosed GD were withdrawn from the study and treated conventionally.

Diet

Women were randomly assigned to 1 of 2 dietary groups designated the LGI diet or healthy eating (HE) diet. Participants received a detailed dietary education tailored for the assigned diet and their individual requirements for pregnancy (ie, that met energy needs and nutrient requirements). There was no intended difference in the macronutrient distribution in diets. Women in both groups were counseled to adopt diets that were consistent with the nationally recommended nutritional intake for pregnant women (17) and recommendations of the Australian Guide to Healthy Eating (18).

Participants were provided with 1 of 2 sets of booklets (depending on their allocation) that included information on the choices for and serving sizes of carbohydrate-rich foods. The LGI group received specific information on LGI alternatives for relevant food groups. Specific dietary goals were provided to each participant that focused on differentiating between carbohydrate-rich foods (eg, replace white potato with an LGI potato or change breakfast cereals to rolled oats). Women in the HE group were counseled to follow a conventional healthy diet with recommended foods and serving sizes as noted in the Australian Guide to Healthy Eating and were not given any guidance on the GI. Study personnel were not blinded to the dietary assignment but were aware of the need for impartiality and equivalent treatment. Obstetric care providers were not specifically blinded to the study allocation but were also not informed.

Study protocol

A research dietitian explained aims and requirements of the study and assessed eligibility. Interested patients were provided with a participant information sheet, consent form, and 3-d food-record sheet. The first study visit was arranged to coincide with the next obstetric appointment. There were 4 contact points scheduled during the pregnancy.

First visit

At the first visit, women gave written informed consent. Women were randomly assigned by using computer-generated random numbers to 1 of 2 diet groups and stratified by pre-pregnancy BMI (in kg/m²); <30 compared with ≥30). The allocation sequence was unpredictable and concealed from the research dietitian. Demographic details, including ethnicity, country of birth, smoking status, and education were recorded, and basicanthropometric measures were undertaken, including of weight and height. Weight was measured to the nearest 0.1 kg on floor scales (HD-316, Tanita Scales; Wedderburn Pty Ltd) with subjects dressed in light clothes and without shoes. Height was measured to the nearest 0.1 cm against a wall by using a nonstretchable, fiberglass measuring tape (Gulick II; Country Technology Inc). The pre pregnancy weight by recall was recorded and used to calculate maternal BMI. The completed 3-d food record was reviewed, and extra details or information (eg, serving sizes and brands, cooking methods, and ingredients) were clarified. Women were asked to estimate portions and quantities on the basis of pictures, food models, and measuring tools (eg, cups and teaspoons). Diet education that was specific to the assigned diet group was given.

Second contact (phone call)

A phone call was made ~4 wk after the initial education and visit to ensure adherence to the prescribed diet and goals set, identify any barriers to adherence, and address any other dietary issues or concerns.

Third visit (midway assessment)

Dietitians reviewed participants face-to-face at ~28 wk of gestation before their obstetric appointment to monitor progress.
and address any new issues. At this visit, participants were also given another 3-d food record and asked to complete the record before their final visit scheduled at ~34 wk of gestation.

Fourth (final) visit

As late as possible, depending on the obstetric schedule, but at a minimum of 34 wk of gestation, the dietitian collected and reviewed the final 3-d food record and also measured the final weight of subjects.

The research dietitian was available for telephone queries outside of scheduled visits. An e-mail was sent each month to every participant who provided an e-mail address, and the content varied depending on the diet allocation. The content included nutrition tips for pregnancy and recipes. A total of 5 e-mails were scheduled to be sent to each participant.

Acceptability survey

To evaluate the acceptability of the recommended diet changes in pregnancy, at their final visit, subjects were asked to score 6 statements on a 5-point Likert scale (1 denoted “strongly agree,” and 5 denoted “strongly disagree”). Statements were as follows: “It was easy to follow the diet recommended during this study;” “I enjoyed the dietary changes that I made;” “The changes recommended were affordable;” “My family was accepting of the changes made to my eating habits;” “The study diet helped me meet the physical challenges of pregnancy;” and “I enjoyed a wide variety of foods in my eating plan.” In addition, women were also asked to indicate on a Likert scale how closely they followed the assigned diet (1 denoted “all of the time,” and 5 denoted “none of the time”).

Food-data entry

All data were entered into a Microsoft Access (Microsoft Office Excel 2007) database. Food-intake data were entered into a customized database that incorporates Australian food-composition tables and published GI values (glucose = 100 scale; FoodWorks 2009 Professional edition, version 6.0.2539; Xyris Software). When necessary, additional GI data were obtained from the University of Sydney’s online database (http://www.glycemicindex.com). Overall, the dietary GI was calculated as the weighted sum of the GI of all carbohydrate foods in the diet, with the weighting proportional to the contribution of each food to total carbohydrate intake. In general, unassigned carbohydrate was low (<5% of total carbohydrate). Because the target diets aimed for a similar carbohydrate content, it was intended that the glycemic load (GL) (the product of the GI and amount of carbohydrate) would be influenced only by differences in the GI. Therefore, any differences in fetal outcomes could be attributed to the nature of carbohydrate per se (ie, GI) rather than the simultaneous changes in both quantity and quality.

Power calculations

The enrollment of 622 subjects (311 subjects/group) gave 90% power to detect a 130-g difference in birth weight in a population in whom the average (±SD) birth weight was 3400 ± 500 g. We aimed to enroll 684 subjects to allow for a 10% dropout rate. In our earlier study (13), the intervention produced a 236-g difference in birth weight, but we did not expect to see such a large difference in this study because the intervention was to be less intensive because it has been adapted to test its effectiveness in a more-realistic antenatal setting. Because of a change in diagnostic criteria for GD, it was not practical to consider if the LGI diet reduced the prevalence of GD.

Statistical analysis

Comparisons between the HE and LGI groups were carried out by using independent-samples t tests for measured variables and chi-square tests for categorical variables. In additional analyses, relations between actual GI, GL, and birth outcomes (birth percentile and PI) were investigated by initially using linear regression and, then, general linear models that included the delivery method and fetal sex as factors. Analyses were carried out with SPSS version 20 software, with significance indicated at P < 0.05.

RESULTS

The flow of participants through the project is shown in Figure 1. In total, 691 women were enrolled in the PREGGIO study, with 354 women randomly assigned to the LGI group and 337 women randomly assigned to the HE group. In the LGI group, 58 women were later excluded (27 women had developed GD). In the HE group, 57 women were later excluded (28 women had developed GD). In the primary analysis, there were 296 women in the LGI group and 280 women in the HE group. The first woman was recruited on 19 February 2010, and the last woman was recruited on 19 September 2012 and delivered on 1 March 2013.

Maternal details and selected fetal outcomes are shown in Table 1. There were no significant differences in demographic characteristics between the 2 groups. With the use of a pre-pregnancy BMI ≥25 as indicating overweight and a BMI ≥30 as indicating obese, 21% of women in the LGI group were overweight and 12% of women in the LGI group were obese compared with 22% and 17% of women, respectively, in the HE group, with no significant difference between groups (P = 0.24).

Maternal energy intake and changes in the GI and GL are shown in Table 2. As anticipated, the total energy intake and macronutrient distribution were similar between groups at baseline and at the end of the study. At the final visit, as intended, the LGI group had reduced their GI and GL compared with the results at enrollment. Fiber intake and the macronutrient energy distribution were similar in both groups, with the exception of a small but significant increase in the percentage of energy derived from protein in the LGI group. From enrollment to final weight, the weight gain by women in the HE group of 10.3 ± 0.3 kg was similar to the weight gain of women in the LGI group of 10.2 ± 0.2 kg (P = 0.73).

There were no significant differences in the primary outcomes of fetal birth weight, birth percentile, or PI (Table 1). In the HE group, the prevalence of infants who were small for gestational age or large for gestational age (LGA) (7.9% and 10.4%, respectively) was similar to that in the LGI group (6.6% and 10.0%, respectively; P = 0.81). In the multivariate regression analysis,
maternal prepregnancy weight and pregnancy weight gain were significant predictors of fetal birth weight. Birth weight was significantly larger for mothers with higher prepregnancy weight ($P < 0.001$) and greater pregnancy weight gain ($P < 0.001$). However, there was no significant effect of the diet group when overweight and obese mothers were considered separately (BMI $\geq 25$; $n = 205$; $P = 0.72$). In terms of weight gain against recommendations, there was no evidence that the 2 diet groups had different proportions of mothers in the 3 weight-gain groups (low, recommended, and high; $P = 0.15$; data not shown).

In the cohort as a whole, the GL was the only significant dietary predictor ($P = 0.046$) of primary outcomes (birth percentile and PI) after adjustment for the type of delivery (spontaneous, cesarean, or instrumental) and sex. In each case, the $R^2$ for the GL was 0.26.

### Table 1

<table>
<thead>
<tr>
<th></th>
<th>HE group</th>
<th>LGI group</th>
<th>Significance ($P$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age (y)</td>
<td>29.9 ± 0.3</td>
<td>29.9 ± 0.3</td>
<td>0.96</td>
</tr>
<tr>
<td>Week enrolled</td>
<td>16.2 ± 0.1</td>
<td>16.5 ± 0.1</td>
<td>0.08</td>
</tr>
<tr>
<td>Gravida</td>
<td>2.2 ± 0.1</td>
<td>2.2 ± 0.1</td>
<td>0.74</td>
</tr>
<tr>
<td>Parity</td>
<td>0.8 ± 0.1</td>
<td>0.8 ± 0.1</td>
<td>0.82</td>
</tr>
<tr>
<td>Maternal height (cm)</td>
<td>165.0 ± 0.4</td>
<td>165.7 ± 0.4</td>
<td>0.26</td>
</tr>
<tr>
<td>Prepregnancy weight (kg)</td>
<td>67.5 ± 1.0</td>
<td>66.7 ± 0.8</td>
<td>0.54</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.7 ± 0.3</td>
<td>24.3 ± 0.3</td>
<td>0.35</td>
</tr>
<tr>
<td>Pregnancy weight gain (kg)</td>
<td>13.8 ± 0.3</td>
<td>14.1 ± 0.3</td>
<td>0.47</td>
</tr>
<tr>
<td>Gestational weeks at delivery</td>
<td>39.5 ± 0.1</td>
<td>39.5 ± 0.1</td>
<td>0.62</td>
</tr>
<tr>
<td>Fetal birth weight (g)</td>
<td>3443 ± 29</td>
<td>3465 ± 25</td>
<td>0.57</td>
</tr>
<tr>
<td>Fetal length (cm)</td>
<td>50.3 ± 0.2</td>
<td>50.3 ± 0.1</td>
<td>0.96</td>
</tr>
<tr>
<td>Birth percentile (%)</td>
<td>49.4 ± 0.2</td>
<td>51.4 ± 1.7</td>
<td>0.40</td>
</tr>
<tr>
<td>Ponderal index (g) × 100 ÷ length (cm)$^3$</td>
<td>2.70 ± 0.02</td>
<td>2.72 ± 0.01</td>
<td>0.53</td>
</tr>
</tbody>
</table>

$^1$ All values are means ± SEMs. The independent-samples $t$ test for differences between HE and LGI groups was used for measured variables, and the chi-square test was used for categorical variables. HE, healthy eating; LGI, low glycemic index.
value was small, which indicated that the dietary GI explained <1% of the variation. No other dietary variable was related to the primary outcome (data not shown).

The birth percentile and PI were also considered with respect to lowest and highest quintiles of GI achieved. For combined completers (HE plus LGI) there were no significant differences for the birth percentile \( (P = 0.52) \) or PI \( (P = 0.84) \).

In the HE group, 39 of women 280 (13.9%) had infants >4.0 kg, which was not significantly different to 31 of 296 women (10.5%) in the LGI group \( (P = 0.25) \). There was also no significant difference between the 6 women (2.1%) in the HE group compared with the 2 women (0.7%) in the LGI group \( (P = 0.17) \) who had infants >4.5 kg \( (P = 0.17) \).

For combined completers (HE plus LGI), and including women who were subsequently excluded because of a diagnosis of GD, 618 of 624 subjects (99%) had a glucose-tolerance test. The prevalence of GD in the LGI group (7.6%) and HE group (7.6%) was the same.

For combined completers, 566 of 576 women (98.3%) delivered at the intended hospital (public: \( n = 400 \); private: \( n = 166 \)). The women who were private were significantly older (31.4 ± 3 compared with 29.3 ± 1.6 years; \( P < 0.001 \)) and delivered at an earlier gestational week (39.0 ± 1 compared with 39.7 ± 1 week; \( P < 0.001 \)). There were no significant differences between public and private women with respect to fetal birth weight, fetal percentile, and PI.

The acceptability of the diet was similar in both groups (Table 3). With respect to how often women followed the assigned diet, the mean (±SEM) score for women in the LGI group was 2.26 ± 0.04 (maximum: 5), whereas women in the HE group scored 2.24 ± 0.04 \( (P = 0.73) \).

### DISCUSSION

The primary aim of this study was to assess effects on fetal variables of advice that was based on LGI foods compared with conventional HE advice. This advice was of low intensity and could be reasonably incorporated in the delivery of routine antenatal care. Our previous study (13), which has a smaller number of women but relatively intensive dietary advice, had showed a reduction in fetal birth weight, PI, and also the proportion of infants born LGA. In the current study, we did not expect to achieve the same outcomes because there was only 2 face-to-face diet sessions (compared with 5 sessions), and higher numbers were recruited to detect a smaller difference. Women allocated to the LGI group did lower their dietary GI by about 4 units (compared with 6 units in our earlier study), whereas the GI of women allocated to the HE group remained unchanged. Contrary to our hypothesis, despite this reduction of the GI, we showed that infants of women in the LGI group had similar birth weights, birth percentiles, and PI. Women in both groups also had a similar proportion of infants who were above the 90th percentile and below the 10th percentile (ie, LGA or small for gestational age, respectively).

A recent report from the ROLO study (19) showed that a small reduction in the GI (1–2 units) resulted in less maternal glucose intolerance (although there was no change in the prevalence of GD) and less gestational weight gain in the group allocated to an LGI diet. However, the population selected was targeted because

Table 3

Results of the acceptability questionnaire

<table>
<thead>
<tr>
<th></th>
<th>HE group</th>
<th>LGI group</th>
<th>Significance (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>It was easy to follow the diet...</td>
<td>2.06 ± 0.05</td>
<td>2.13 ± 0.05</td>
<td>0.31</td>
</tr>
<tr>
<td>I enjoyed the dietary changes...</td>
<td>2.14 ± 0.05</td>
<td>2.05 ± 0.05</td>
<td>0.22</td>
</tr>
<tr>
<td>The changes were affordable...</td>
<td>2.01 ± 0.05</td>
<td>2.06 ± 0.05</td>
<td>0.45</td>
</tr>
<tr>
<td>My family was accepting...</td>
<td>1.94 ± 0.05</td>
<td>1.91 ± 0.05</td>
<td>0.67</td>
</tr>
<tr>
<td>The study diet helped me...</td>
<td>2.22 ± 0.05</td>
<td>2.32 ± 0.04</td>
<td>0.14</td>
</tr>
<tr>
<td>I enjoyed a variety of foods...</td>
<td>1.98 ± 0.04</td>
<td>1.92 ± 0.04</td>
<td>0.34</td>
</tr>
</tbody>
</table>

\(^{1}\)All values are means ± SEMs. The independent-samples \( t \) test for differences between HE and LGI groups was used. HE, healthy eating; LGI, low glyemic index.
of the previous delivery of a macrosomic infant, and the comparison group received no dietary advice. In our study, in which both groups received equivalent dietary advice, the postenrollment gestational weight gain of ~10 kg was similar despite, in the LGI group, the achievement of about twice the reduction in the GI compared with that of women in the ROLO study. A glucose-tolerance test was done as part of normal obstetric practice and was ~12 wk after enrollment. We showed no difference in glucose concentrations between groups (data not shown). This comparative lack of effect may have been because we were dealing with a general obstetric population rather than a selected population.

We postulated that LGI dietary advice would reduce ambient glycemia in pregnant women and, thereby, improve fetal outcomes. In lean and overweight individuals, meals based on LGI foods led to a reduction in the postprandial glycemia (20, 21), and this reduction is likely to be applicable in pregnancy. Nonetheless, in the current study, birth percentiles and the PI were shown to be similar in both diet groups, which led us to conclude that there may have been an insufficient reduction of postprandial glucose concentrations to achieve the desired outcome.

Scholl et al (22) have proposed that eating foods with an LGI may lead to lower maternal glucose concentrations and less fuel for the fetus to develop a normal pattern of growth. In their study (an observational study), underprivileged women who reported a diet with a relatively LGI but higher intake of refined sugars gave birth to infants who were ~100 g lighter. In the current study, women randomly assigned to the LGI group had fetal variables that were similar to those in the HE group. When the dietary GL was considered as a continuous variable, there was a significant positive association between the dietary GL and both the birth percentile and PI. The GL per 1000 kJ is the strongest predictor of postprandial glycemia and insulinemia (23). Nonetheless, the GL explained only a very small proportion (1%) of the total variability in birth percentiles. As could be anticipated, maternal BMI and gestational age were more important predictors of birth outcomes.

Other studies have examined the feasibility of the use of LGI diets during pregnancy. Rhodes et al (24), in a pilot study, examined the effects of a low-GL diet for overweight and obese women. Whereas an LGI diet did improve certain aspects of pregnancy, there were no significant differences in birth weight z scores or variables of fetal adiposity. Grant et al (25) examined aspects of maternal glycemic control by comparing an LGI diet with a conventional diet. The study recruited women from a heterogenous ethnic background and was of relatively short duration. There was a nonsignificant reduction in the fetal size in the LGI group.

A secondary aim of the current study was to assess the tolerability and sustainability of an LGI diet. The 2 groups of women were well matched for initial macronutrient intakes. There were no significant differences in their responses to questions, and both diets were equally acceptable.

The strengths of our study include the randomized, controlled, parallel design, large sample size, high continuation rate, and detailed ascertainment of dietary measurements. Together, these factors increased the reliability and sensitivity of the data. A particular strength was the extensive knowledge of the GI of individual Australian foods (26). In previous studies, we confirmed that sample menus representative of the 2 diets produced differential day-long glucose and insulin postprandial responses as predicted by their calculated GI (27). An additional strength was that subjects were free-living women who represent an important target for early intervention and also included women from both public and private sectors. The overall quality of both diets was good, with food and nutritional intakes in accordance with recommendations for pregnancy.

Our study had limitations. Pregnancy outcomes depend on multiple factors and not just dietary intake. We did not control or assess physical activity, which is an important factor that affects the energy balance and, therefore, birth outcomes. Although the LGI dietary advice was given with the expectation that the GI would be reduced by ≥ 6 units, the women, on average, achieved much less. This result suggested that much more intensive interactions with dietitians are needed to produce clinically important outcomes. A greater frequency of contact has been recognized to increase the effectiveness of interventions (28). A lack of blinding of subjects and investigators to diet assignment can introduce a source of bias. Maternal weight gain and most measures of diet acceptance, however, were similar with both diets, which suggested no overt bias toward any one diet. We also lacked a control group that was not counseled on a healthy diet by dietitians. Thus, we were not able to consider the possibility that outcomes for both groups of women who received dietary advice were improved or otherwise compared with women who did not receive advice of any kind during pregnancy. Thus, we learned that additional considerations may be required on the intensity of the intervention, delivery of the intervention, and degree of difference in the GI that may be necessary.

In conclusion, the outcome of this study was neutral. Infants of women instructed to consume LGI carbohydrate foods during pregnancy were of normal size and had a similar PI to those of infants of women who received conventional HE advice. Although a high dietary GI during pregnancy is associated with adverse metabolic markers in young adults (29), the means and validity of reducing the GI during pregnancy still remain to be determined. This assessment is likely to require a more intensive dietary supervision than what is currently available both before and during pregnancy.

The authors’ responsibilities were as follows—RGM and JCB-M: conceived and designed the study; SAC, EGQ, JMC, and MM: conducted the research; SAC and JMC: analyzed dietary data; PP: analyzed data and performed the statistical analysis; RGM, LCT, and JCB-M: wrote the manuscript; and RGM: had primary study oversight and responsibility for the final content of the manuscript. RGM and JCB-M are coauthors of The Low GI Eating Plan for an Optimal Pregnancy (New York, NY: The Experiment, 2013) and The Bump to Baby Diet (Sydney, Australia: Hachette, 2012). SAC, EGQ, JMC, LCT, MM, and PP had no conflicts of interest.

REFERENCES


