Macronutrient Composition or Social Determinants? Impact on Infant Outcomes With Gestational Diabetes Mellitus

Kimberly K. Trout,1 Carol J. Homko,2 Lisa Wetzel-Effinger,3 Wadia Mulla,2 Ricardo Mora,4 Joanna McGrath,3 Lisa Basel-Brown,5 Angelina Arcamone,3 Parichehr Sami,2 and Kepher H. Makambi6

ABSTRACT
The purpose of this study was to examine, through a randomized, controlled trial, the effects of a maternal carbohydrate-restricted diet on maternal and infant outcomes in gestational diabetes mellitus (GDM). Women diagnosed with GDM were randomly allocated into one of two groups: an intervention group that was placed on a lower-carbohydrate diet (35–40% of total calories) or a control group that was placed on the usual pregnancy diet (50–55% carbohydrate). A convenience sample of participants diagnosed with GDM (ages 18–45 years) was recruited from two different sites: one urban and low-income and the other suburban and more affluent. Individual face-to-face diet instruction occurred with certified diabetes educators at both sites. Participants tested their blood glucose four times daily. Specific socioeconomic status indicators included enrollment in the Supplemental Nutrition Program for Women, Infants and Children or Medicaid-funded health insurance, as well as cross-sectional census data. All analyses were based on an intention to treat. Although there were no differences found between the lower-carbohydrate and usual-care diets in terms of blood glucose or maternal-infant outcomes, there were significant differences noted between the two sites. There was a lower mean postprandial blood glucose (100.59 ± 7.3 mg/dL) at the suburban site compared to the urban site (116.3 ± 15 mg/dL) ($P < 0.01$), even though there was no difference in carbohydrate intake. There were increased amounts of protein and fat consumed at the suburban site ($P < 0.01$), as well as lower infant complications ($P < 0.01$). Further research is needed to determine whether these disparities in outcomes were the result of macronutrient proportions or environmental conditions.

Gestational diabetes mellitus (GDM) is the most common type of diabetes found in pregnancy, with higher prevalence in racial/ethnic groups at greater risk for perinatal health disparities. It is more common in African-American women, who have poorer pregnancy outcomes compared to non-Hispanic whites (1,2). Dietary manipulation has been referred to as the cornerstone of GDM treatment, yet there is no consensus in the scientific community as to what constitutes optimal dietary management of GDM (3). Previous studies have generally examined either the effects of calorie restriction (4–7) or carbohydrate manipulation (8–12) on maternal and infant outcomes. Some women with GDM are unable to maintain ideal blood glucose control with dietary management alone and also may require exogenous insulin or an oral hypoglycemic medication to maintain euglycemia (13).

Potential complications for women with GDM range from immediate increased pregnancy risks to long-term health implications such as increased risk for metabolic syndrome.
and cardiovascular disease after pregnancy. Increased maternal glucose levels cross the placenta and result in increased fetal glucose levels and augmented secretion of fetal insulin. Although the pathophysiological mechanisms of the fetal sequelae from GDM are not completely understood, there is evidence that fetal hyperinsulinemia contributes to the risk of infant hypoglycemia after birth and to infant macrosomia (14–18). The multicenter Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study revealed that increasing levels of maternal hyperglycemia result in increasing rates of infant adverse outcomes in a continuous and linear manner (19). With plasma glucose levels just one standard deviation (SD) above the mean for fasting, 1-hour, and 2-hour postprandial periods, the odds ratios for infant birth weight above the 90th percentile were 1.38 (95% CI 1.32–1.44), 1.46 (1.39–1.53), and 1.38 (1.32–1.44), respectively.

Infant macrosomia, defined as an infant birth weight ≥4,000 g, can result in birth injuries to infants and increased cesarean section rates for mothers, as well as other complications. These complications result in increased costs for care. A study conducted in Finland found that total mean health care costs (with adjustments for age, BMI, and education) were 25.1% higher in women diagnosed with GDM than in those without GDM (20).

Research also has suggested that there are long-term health implications for infants and children related to GDM. A multiethnic sample of 9,439 maternal-child pairs screened for GDM found that increasing hyperglycemia in pregnancy was associated with an increased risk of childhood obesity 5–7 years after birth (P <0.0001) (21). The investigators concluded that their results support the increasingly identified concept of “metabolic imprinting,” also referred to as “fetal programming.” (22). These terms describe a phenomenon through which the maternal intrauterine environment can affect the long-term health of the fetus/child through the alteration of various metabolic, immune, vascular, and possibly other health parameters, thus underscores an urgent need for discovery of efficacious and cost-effective means to optimally care for women with GDM.

Major et al. (10) were among the first investigators to test the hypothesis that carbohydrate restriction may help women with GDM achieve euglycemia. In a nonrandomized study, women with diet-controlled GDM were placed on either a low-carbohydrate (<42% of total calories, n = 21) or a high-carbohydrate (>45% of total calories, n = 21) diet within 1 week of GDM diagnosis (between 24 and 28 weeks of gestation). In this study, 1-hour postprandial blood glucose levels were significantly reduced in the low-carbohydrate group (P <0.04). The investigators also found that fetal macrosomia was significantly less in the low-carbohydrate group (P <0.035), and there were lower rates of cesarean deliveries for macrosomia and cephalopelvic disproportion (P <0.037). More recently, Hernandez et al. (8) used a crossover design to compare differing diet types for women with GDM in the highly controlled environment of a clinical research center unit. Women with GDM were randomized to isocaloric diets that were either higher in complex carbohydrate (60%) and lower in fat (15%) (HC/LF [CHOICE] diet) or lower in carbohydrate (40%) and higher in fat (45%) (LC/HF), with protein levels held constant at 15% in both groups. One-hour (P <0.01) and 2-hour (P <0.001) postprandial blood glucose levels were lower on the LC/HF diet than on the HC/LF (CHOICE) diet. Glucose area under the curve (AUC) was measured via a 24-hour continuous glucose monitoring system, and 24-hour profiles were 6% lower with the LC/HF diet (P = 0.02). However, the study authors noted that, despite lower postprandial and AUC blood glucose results with the LC/HF diet, acceptable targets were still attained with the HC/LF (CHOICE) diet with the additional benefit of a more favorable cardiovascular profile than was achieved with the LC/HF diet. This conclusion was reached on the basis of a lower free fatty acids AUC for the HC/LF (CHOICE) diet (19%, P <0.01). Other studies have shown similar results, suggesting that higher levels of carbohydrate with concomitant lower levels of fat produce favorable outcomes (3,5,8,9,12,15,23).

In this study, we aimed to evaluate through a randomized, controlled trial conducted at two socioeconomically diverse sites: 1) the effects of a maternal carbohydrate-restricted diet (35–40% of total calories) versus the usual pregnancy diet (carbohydrate intake 50–55% of total calories) on maternal outcomes of blood glucose control, weight gain, common medical comorbidities, and incidence of medical procedures, and 2) the effects of a maternal carbohydrate-restricted diet versus the usual pregnancy diet on infant outcomes of birth weight, incidence of macrosomia, and adverse perinatal events.

Methods
Women diagnosed with GDM were allocated randomly into one of two groups: an intervention group that was placed on a lower-carbohydrate diet (35–40% of total calories) versus a control group that followed the usual pregnancy diet (50–55% carbohydrates).

A convenience sample of subjects diagnosed with GDM (aged 18–45 years) whose GDM was controlled via diet alone or diet plus oral medication (e.g., glyburide) were recruited to enter the study. Participants were recruited from two sites; Site A was an urban teaching hospital, and Site B was a suburban community hospital. Potential participants were screened with a 50-g oral glucose tolerance test (OGTT) at 24–28 weeks’ gestation without regard to time of

72 SPECTRUM.DIABETESJOURNALS.ORG

FEATURE ARTICLE / MACRONUTRIENTS OR SPECIAL DETERMINANTS?
day or interval since their last meal (13). Women whose 1-hour screening test plasma glucose result was ≥135 mg/dL underwent a 100-g OGTT and were diagnosed with GDM based on the Carpenter-Coustan criteria (24–26). Women who were diagnosed with GDM by these criteria and were at ≤35 weeks of gestation were eligible for inclusion. Women were excluded if they had a multifetal pregnancy or gestational diabetes or required insulin for their diabetes at the time of enrollment. Women with any other significant medical or psychiatric comorbidities (e.g., cardiovascular disease or preexisting hypertension) also were excluded, as were smokers and users of alcohol or illicit drugs, due to the possible effects of these habits on fetal growth.

Institutional Review Board
The study received approval from the institutional review board (IRB) of Villanova University and the respective IRBs of each of the two clinical facilities participating.

Procedures
Participants were notified by their obstetrical provider of their diagnosis of GDM. Those interested in participating were screened for eligibility via a questionnaire administered by a study team member. For women who were eligible and willing to participate, a study team member answered questions to potential participants’ satisfaction and reviewed and secured signatures on a written informed consent form before enrollment. Enrollment and participation were completely voluntary.

Once accepted and enrolled into the study, participants were randomly assigned to either the control (usual pregnancy diet) or intervention group. Results were analyzed based on an intention to treat; therefore, results were included from all participants who enrolled and were randomly assigned to either group. Both groups received the current maternal-fetal surveillance standard of care as determined by their obstetrical provider. Changes in diabetes therapy (e.g., the decision to initiate pharmacotherapy) were at the discretion of participants’ providers and not the research team.

Women in both groups received diabetes education and self-management training by a certified diabetes educator (CDE). Participants in both groups were instructed in self-monitoring of blood glucose (SMBG) with portable reflectance memory meters (OneTouch Ultra, LifeScan, Milpitas, Calif., or Ascencia Countour, Bayer HealthCare, Whippany, N.J.). Women in both groups were asked to perform SMBG four times daily (fasting and 2 hours after meals). Participants were asked to bring their blood glucose and food logs or meter records to their prenatal care appointments so their providers could review their results. Women in both groups were seen for clinical evaluation every 2 weeks until 36 weeks’ gestation, after which they were seen weekly. Women in both groups also self-monitored urine ketones each morning and had urine ketones checked by staff at each prenatal visit with Ketostix reagent strips (Bayer HealthCare).

Carbohydrate-Restricted Diet
Participants in the intervention group received a total maximum daily carbohydrate gram count that was set after consultation with the CDE in relation to each participant’s total daily caloric intake. Participants were instructed that they should meet a minimum carbohydrate level (calculated in grams at 35% of total calories) recommended by the CDE and that they should not exceed a maximum carbohydrate level (calculated in grams at 40% of total calories) recommended by the CDE. For example, if the participant was instructed to consume 2,200 kcal/day:

- Minimum carbohydrate intake: 2,200 kcal/day × 0.35 = 770 carbohydrate kcal/4 = 193 g of carbohydrate daily
- Maximum carbohydrate intake: 2,200 kcal/day × 0.40 = 880 carbohydrate kcal/4 = 220 g of carbohydrate daily

Therefore, this participant would be instructed to eat a minimum of 193 g but not to exceed 220 g of carbohydrate daily.

Participants were encouraged to divide their total carbohydrate allotment into three meals and three snacks daily. They received detailed instructions on carbohydrate gram counting from the CDE or another trained study team member. Participants practiced weighing and measuring actual foods and calculating the carbohydrate content of those foods. Instructions included the use of measuring cups, a calibrated gram scale (Polder, Port Chester, N.Y.), and a pocket-sized book listing carbohydrate grams for portions of commonly eaten foods (27).

Participants also were encouraged to eat healthful foods, with written examples provided in the instructional sheet reviewed with each participant, such as:

“Here are some examples of healthy carbohydrate foods that you should include in your diet:
- Grains, including bread, rice, pasta, and low-sugar cereals
- Starchy vegetables such as potatoes, corn, and beans
- Fruit
- Low-fat milk and yogurt”

Figure 1 shows a sample menu plan provided to participants.

Adherence to the prescribed diet was ensured through review of food logs by the CDE, perinatal nurses, and prenatal care providers (in most cases, perinatologists). Participants who did not complete food logs were questioned orally about diet adherence during review of their blood glucose records with their care provider.

Usual Pregnancy Diet
Participants in the control group underwent identical procedures in all
Macronutrients or Special Determinants?

Aspects of the study, including carbohydrate counting and recording food intake, and following the SMBG protocol delineated above. The only difference between the intervention and control groups was that participants in the control group had a carbohydrate intake level set at 50–55% of total calories.

Statistical Analyses

Before testing hypotheses, descriptive statistics were computed for all subjects. The results were examined to determine the presence of marked skewness, outliers, and systematic missing data. Appropriate statistics were computed between the demographic variables (e.g., ethnicity and age at delivery) and the dependent outcome measures to determine the presence of any significant confounding variables. All statistical analyses were based on an intention to treat, meaning that results from all randomized subjects were analyzed. In addition, the intention-to-treat analysis accounted for noncompliance of subjects and minor deviations from protocols. Intention-to-treat analysis reduces the likelihood of a biased treatment effect. For continuous outcome variables, the Student’s t test for independent samples was used to assess the significance of the difference between the two groups. A χ² test or Fisher’s exact test was used to examine the association between categorical variables and the intervention group.

Results

A total of 68 women were enrolled (Site A, n = 54; Site B, n = 14). Statistical significance was not reached for the primary outcome variable of a lower mean 2-hour postprandial blood glucose (2hPPBG) in the intervention group compared to the control group, as had been hypothesized (Table 1). Fasting blood glucose was not significantly different between the two groups. (A difference in this variable had not been expected.) Surprisingly, there was a significant difference in mean 2hPPBG between the two study sites. Participants from Site A (an urban teaching center) had a mean 2hPPBG of 116.30 ± 15.13 mg/dL compared to 100.59 ± 7.30 mg/dL for participants from Site B (a suburban community hospital) (P <0.01).

Tables 1 and 2 illustrate that there were no differences in individual or composite maternal comorbidities or neonatal complications between the intervention and control groups. There was a trend toward a lower primary cesarean section rate between the intervention and control groups (29.4 and 40.6%, respectively), although this was not statistically significant (P = 0.34). When looking at composite maternal complications by site, there were no differences between Site A and Site B (48.1 and 50.0%, respectively), as shown in Table 3. Complications included in the composite score included incidence of gestational hypertension, preeclampsia, polyhydramnios, postpartum hemorrhage, and urinary tract infection.

There was a difference in composite neonatal complications by site (Site A 40.4%; Site B 0%; P <0.01) (Table 3). Neonatal complications included in this calculation included incidence of macrosomia (birth weight ≥ 4000 g) and respiratory distress syndrome (RDS) requiring respiratory support. However, the statistical significance of the difference between the two groups was not reached (P = 0.13).
weight ≥4,000 g), shoulder dystocia, respiratory distress syndrome, hypoglycemia, and admission to the neonatal intensive care unit. There were no neonatal deaths, bone fractures, or nerve palsies at either site for either group. Nutritional comparisons by site are shown in Table 4.

**Demographic and Environmental Data**

The median household income in the zip code surrounding Site A was $22,654, significantly lower than that of Site B, which was $87,347 (Table 5). There was also a significant difference in the number of individuals living below the poverty line, with Site A having 42.9% compared to 4.3% in Site B (28). In our study sample, more participants from Site A than from Site B were recipients of Medicaid-funded health insurance (96 and 28.6%, respectively) ($P<0.0001$). The area surrounding

---

**TABLE 1. Means and Percentages of Maternal Characteristics by Study Group**

| Maternal Characteristics                  | Lower-Carbohydrate Diet Group (n = 37) | Usual Pregnancy Diet Group (n = 31) | P     |
|-------------------------------------------|----------------------------------------|------------------------------------|-------|
| 2hPPBG (mg/dL)                            | 111.56 ± 14.45                         | 113.91 ± 16.18                     | 0.61  |
| Fasting blood glucose (mg/dL)             | 90.52 ± 8.53                           | 91.97 ± 12.08                      | 0.65  |
| BMI (kg/m²)                               | 33.84 ± 8.84                           | 31.80 ± 8.68                       | 0.34  |
| Total pregnancy weight gain (lb)          | 27.24 ± 16.02                          | 25.68 ± 17.08                      | 0.71  |
| Age at delivery (years)                   | 30.09 ± 6.15                           | 29.63 ± 5.19                       | 0.74  |
| Weight change from study entry to birth (lb) | 4.75 ± 6.20                           | 4.41 ± 6.24                       | 0.85  |
| Weeks of gestation at study entry         | 29.17 ± 2.78                           | 30.50 ± 2.85                       | 0.032 |
| Weeks of gestation at delivery            | 37.78 ± 1.66                           | 37.76 ± 1.74                       | 0.96  |

**Percentage (%)**

| Need for insulin therapy                  | 8.8                                    | 6.3                                | 0.69  |
| Oral medication use:                      |                                        |                                    |       |
| Before enrollment                         | 32.4                                   | 34.4                               | 0.98  |
| After enrollment                          | 2.9                                    | 3.1                                |       |
| Incidence of induction of labor           | 35.3                                   | 34.4                               | 0.94  |
| Incidence of primary cesarean section     | 29.4                                   | 40.6                               | 0.34  |

**Composite maternal complications**

| None                                      | 52.0                                   | 50.0                               | 0.81  |
| ≥1                                       | 47.1                                   | 50.0                               |       |

**TABLE 2. Means and Percentages of Neonatal Characteristics by Study Group**

| Neonatal Characteristics                  | Lower-Carbohydrate Diet Group          | Usual Pregnancy Diet Group         | P     |
|-------------------------------------------|----------------------------------------|------------------------------------|-------|
| Birth weight (g)                          | 3,409.53 ± 527.91                      | 3,377.28 ± 589.91                  | 0.81  |
| Infant head circumference (cm)            | 35.09 ± 3.80                           | 33.95 ± 1.77                       | 0.13  |
| Abdominal girth (cm)                      | 31.78 ± 2.83                           | 31.56 ± 3.17                       | 0.77  |

**Percentage (%)**

| Birth weight ≥4,000 g                     | 11.8                                   | 12.5                               | 0.93  |
| Incidence of shoulder dystocia           | 2.9                                    | 0                                  | 0.25  |
| Incidence of hypoglycemia                | 9.7                                    | 26.9                               | 0.09  |
| Admission to neonatal intensive care unit| 20.6                                   | 12.5                               | 0.38  |

**Composite infant complications**

| None                                      | 67.6                                   | 68.8                               | 0.92  |
| ≥1                                       | 32.4                                   | 31.1                               |       |
Site A is classified as a “food desert”—an area having limited access to transportation and more than one-half mile away from a supermarket (29) (Figure 2). Limited availability of transportation indicates less access to supermarkets and often forces individuals to obtain foods from convenience stores that not only contain an abundance of “empty calories,” but also have higher prices compared to foods in the supermarkets located outside of the neighborhoods (30,31).

Discussion and Conclusions
The primary aim of this randomized, controlled trial was to test the hypothesis that a lower-carbohydrate diet (compared to the usual pregnancy diet) would result in lower postprandial blood glucose levels in women with GDM. Although postprandial blood glucose trended in the lower direction for the intervention group, the decrease did not reach statistical significance and, therefore, the null hypothesis could not be rejected. Several other outcome variables also trended in the direction of the hypothesis (e.g., lower primary cesarean section rates in the intervention group); however, the results may not have been significant due to a type II error, which occurs when one falsely rejects the null hypothesis, most commonly resulting from too small a sample size.

There were significantly different results between the two study sites; Site B had a lower mean postprandial blood glucose level and a lower composite rate of infant complications. Analysis of nutritional data revealed that there were no differences in mean daily intake of carbohydrate between sites or in mean daily glycemic index of foods (Table 4), but there were higher daily average intakes of fat ($P<0.01$) and protein ($P=0.01$) at Site B.

Although there has been much discussion in the literature regarding the impact of glycemic index and glycemic load on blood glucose control, an even greater impact might be attributed to the role of other macronutrients (i.e., protein and fat) in blunting the effects of carbohydrate within the context of mixed meals. A similar observation was made by Zeevi et al. (32), who noted that the utility of the glycemic index is somewhat limited because it corresponds to a single type of food as opposed to the variety of foods found in typical meals. Some studies have suggested that increasing dietary protein (in particular, whey protein) may play a role in blunting glucose excursions (33).

The HAPO study reported strong, continuous associations between ele-

---

### TABLE 3. Mean Maternal 2hPPBG and Percentage of Maternal and Infant Complications by Site

| Results                        | Site A ($n=54$) | Site B ($n=14$) | $P$  |
|--------------------------------|-----------------|-----------------|------|
| Mean (± SD)                    |                 |                 |      |
| Maternal 2hPPBG (mg/dL)        | 116.30 ± 15.13  | 100.59 ± 7.30   | <0.01|
| Maternal complications         |                 |                 | 0.89 |
| None                           | 51.9            | 50.0            |      |
| ≥1                             | 48.1            | 50.0            |      |
| Infant complications           |                 |                 | <0.01|
| None                           | 59.6            | 100.0           |      |
| ≥1                             | 40.4            | 0               |      |

### TABLE 4. Nutritional Comparisons by Site

| Mean Daily Intake               | Site A ($n=16$)* | Site B ($n=10$)* | $P$  |
|--------------------------------|-----------------|-----------------|------|
| Total intake (kcal)             | 1,614           | 1,887           | NS   |
| Carbohydrates (g)               | 178.0           | 179.51          | NS   |
| Glycemic index                  | 61.13           | 59.28           | NS   |
| Glycemic load                   | 90.24           | 94.28           | NS   |
| Fat (g)                         | 65              | 89              | <0.01|
| Protein (g)                     | 82.3            | 100.17          | <0.01|
| Fiber (g)                       | 14.68           | 18.66           | NS   |

*Reflects number of subjects who were able to provide adequate nutrition data for analysis.

---

*FIGURE 2.* Food desert classifications of sites and surrounding areas.
vation of maternal glucose levels (even those below the diagnostic threshold for diabetes) and increased infant mean birth weight and cord C-peptide levels (19), resulting in the types of complications found in Site A of this study. However, the question of whether the differences in infant outcomes between the two sites can be explained fully by differences in macronutrient intake and mean blood glucose values remains unanswered. An alternative explanation could attribute such differences in infant outcomes to socioeconomic disparities between the two sites. A large body of epidemiological evidence underscores the effects of social determinants on infant morbidity and mortality. The two sites in this study differed markedly in socioeconomic status of participants, which may have translated into differences in the availability of fresh, healthy foods. Thus, differences in rates of neonatal complications potentially could be attributed to the stress of economic and racial disparities, the physiological effects of which (including effects on glucose homeostasis) are not completely elucidated.

One of the major limitations of this study is that only a limited number of women (n = 26) completed nutrition logs, raising the possibility that women who did not complete the logs were eating differently. Because of the intention-to-treat analysis, all other data (e.g., glucose levels and birth outcomes) were included from all randomized participants without regard to evidence of treatment adherence.

Healthy behaviors such as getting adequate physical activity, eating nutritious food, and avoiding harmful substances are within a person’s control and are known factors to promote healthy pregnancy outcomes. However, socioeconomic factors appear to play an important role as well. Numerous studies have identified socioeconomic and racial disparities in rates of infant deaths and low birth weight, with black infants at nearly triple the risk compared to non-Hispanic whites. This study could not determine whether the lower rate of infant complications found at Site B could be primarily attributed to the lower mean postprandial blood glucose values or to differences in race/ethnicity and socioeconomic conditions. However, this study does support the notion that social determinants may affect outcomes for infants of mothers with GDM. Additional well-designed trials with larger sample sizes are needed to better determine optimal diets for women with GDM, as well as social determinants that can affect the weight and health of the infants of mothers with GDM.

Duality of Interest
No potential conflicts of interest relevant to this article were reported.

References
1. Nguyen BT, Cheng YW, Snowden JM, Esakoff TF, Frias AE, Caughey AB. The effect of race/ethnicity on adverse perinatal outcomes among patients with gestational diabetes mellitus. Am J Obstet Gynecol 2012;207:322e1–322e6
2. American College of Obstetricians and Gynecologists. Gestational diabetes mellitus. (Practice Bulletin No. 137) Obstet Gynecol 2013;122:406–416
3. Han S, Crowther CA, Middleton P, Healey E. Different types of dietary advice for women with gestational diabetes mellitus. Cochrane Database Syst Rev 2013;3:CD009275
4. Algert S, Shragg P, Hollingsworth DR. Moderate caloric restriction in obese women with gestational diabetes. Obstet Gynecol 1985;65:487–491
5. Ho LF, Benzie IF, Lao TT. Relationship between caloric intake and pregnancy outcome in diet-treated gestational diabetes mellitus. Nurs Health Sci 2005;7:15–20
6. Knopp RH, Magee MS, Raisys V, Benedetti T, Bonet B. Hypocaloric diets and ketogenesis in the management of obese gestational diabetic women. J Am Coll Nutr 1991;10:649–667
7. Rae A, Bond D, Evans S, North F, Roberman B, Walters B. A randomised controlled trial of dietary energy restriction in the management of obese women with gestational diabetes. Aust N Z J Obstet Gynaecol 2000;40:416–422
8. Hernandez TL, VanPelt RE, Anderson MA, et al. A higher-complex carbohydrate diet in gestational diabetes mellitus achieves glucose targets and lowers postprandial lipids: a randomized crossover study. Diabetes Care 2014;37:1254–1262
9. Cypryk K, Kamiuńska P, Kosinski M, Pertyńska-Marczewska M, Lewiński A. A comparison of the effectiveness, tolerability and safety of high and low carbohydrate diets in women with gestational diabetes. Endokrynol Pol 2007;58:314–319
10. Major CA, Henry MJ, DeviCena M, Morgan MA. The effects of carbohydrate restriction in patients with diet-controlled gestational diabetes. Obstet Gynecol 1998;91:600–604
11. Nolan CJ. Improved glucose tolerance in gestational diabetic women on a low fat, high carbohydrate diet. Diabetes Care 1991;14:68–72

TABLE 5. Demographic Comparisons of Site A and Site B

| Census Data                          | Site A   | Site B   |
|--------------------------------------|----------|----------|
| Total population (n)                 | 54,133   | 35,704   |
| White (non-Hispanic) (n [%])         | 8,160 (15.1) | 31,796 (89.1) |
| Black/African American (n [%])      | 30,998 (57.3) | 2,007 (5.6) |
| Hispanic/Latino (n [%])             | 21,656 (40.1) | 553 (1.5) |
| Asian (n [%])                        | 684 (1.3) | 1,237 (3.5) |
| Median household income ($)          | 22,654   | 87,347   |
| Individuals below poverty line (%)   | 42.9     | 4.3      |

| Study Sample                        |          |          |
|-------------------------------------|----------|----------|
| Total sample size (n)               | 54       | 14       |
| White (non-Hispanic) (n [%])        | 4 (7.4)  | 9 (64.3) |
| Black/African American (n [%])      | 25 (46.3) | 5 (35.7) |
| Hispanic/Latino (n [%])             | 23 (42.6) | 0 (0)    |
| Asian (n [%])                       | 2 (3.7)  | 0 (0.0)  |
high unrefined carbohydrate diet. Aust N Z J Obstet Gynaecol 1984;24:174–177
12. 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
13. Coustan DR. Medical Management of Pregnancy Complicated by Diabetes. 5th ed. Alexandria, Va., American Diabetes Association, 2000
14. Giapros VI, Challa AS, Cholevas VI, Evangelidou EN, Bairaktari ET, Andronikou SK. Vitamin D, parathormone, and insulin resistance in children born large for gestational age. J Pediatr Endocrinol Metab 2014;27:1145–1150
15. Olmos P, Martelo G, Reimer V, et al. [Nutrients other than glucose might explain fetal overgrowth in gestational diabetic pregnancies.] Rev Med Chil 2013;141:1441–1448
16. Eslamian L, Akbari S, Marsoosi V, Jamal A. Effect of different maternal metabolic characteristics on fetal growth in women with gestational diabetes mellitus. Iran J Reprod Med 2013;11:325–334
17. Stein RG, Meinusch M, Diessner J, Dietl J, Höning A, Zollner U. Amniotic fluid insulin and C-peptide as predictive markers for fetal macrosomia, birth injuries, and delivery complications? Med Sci Monit 2014;20:54–58
18. Durnwald CP, Mele L, Spong CY, et al. Glycemic characteristics and neonatal outcomes of women treated for mild gestational diabetes. Obstet Gynecol 2011;117:819–827
19. HAPO Study Cooperative Research Group, Metzger BE, Lowe LP, Dyer AR, et al. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med 2008;358:1991–2002
20. Kolu P, Raitanen J, Rissanen P, Luoto R. Health care costs associated with gestational diabetes mellitus among high-risk women: results from a randomised trial. BMC Pregnancy Childbirth 2012;12:71
21. Hiller TA, Pedula KL, Schmidt MM, Mullen JA, Charles MA, Pettitt DJ. Childhood obesity and metabolic imprinting: the ongoing effects of maternal hyperglycemia. Diabetes Care 2007;30:2287–2292
22. Durnwald C, Landon M. Fetal links to chronic disease: the role of gestational diabetes mellitus. Am J Perinatol 2013;30:343–346
23. Griejer JA, Grzeskowiak LE, Clifton VL. Preconception dietary patterns in human pregnancies are associated with preterm delivery. J Nutr 2014;144:1075–1080
24. Cheng YW, Block-Kurbisch I, Caughey AB. Carpenter-Coustan criteria compared with the National Diabetes Data Group thresholds for gestational diabetes mellitus. Obstet Gynecol 2009;114:326–332
25. Berggren EK, Bogges KA, Stuebe AM, Jonsson Funk M. National Diabetes Data Group versus Carpenter-Coustan criteria to diagnose gestational diabetes. Am J Obstet Gynecol 2011;205:253.e1–253.e7
26. Carpenter MW, Coustan DR. Criteria for screening tests for gestational diabetes. Am J Obstet Gynecol 1982;144:768–773
27. Borushek A. The Calorie King Calorie, Fat & Carbohydrate Counter. Costa Mesa, Calif., Family Health Publications, 2009
28. U.S. Census Bureau. American FactFinder. 2010. Available from http://factfinder.census.gov. Accessed 8 July 2015
29. U.S. Department of Agriculture Economic Research Service. Overview: the Food Access Research Atlas. Available from http://www.ers.usda.gov/data-products/food-access-research-atlas.aspx. Accessed 8 July 2015
30. Beaulac J, Kristjansson E, Cummings S. A systematic review of food deserts, 1966–2007. Prev Chronic Dis 2009;6:A105
31. Walker RE, Keane CR, Burke JG. Disparities and access to healthy food in the United States: a review of food deserts literature. Health Place 2010;16:876–884
32. Zeevi D, Korem T, Zmora N, et al. Personalized nutrition by prediction of glycemic responses. Cell 2015;163:1079–1094
33. Graf S, Egeri S, Heer M. Effects of whey protein supplements on metabolism: evidence from human intervention studies. Curr Opin Clin Nutr Metab Care 2011;14:569–580