The Influence of Maternal Weight and Glucose Tolerance on Infant Birthweight in Latino Mother–Infant Pairs

Edith C. Kieffer, PhD, MPH, Bahman P. Tabaei, MPH, Wendy J. Carman, PhD, MPH, George H. Nolan, MD, MPH, J. Ricardo Guzman, MPH, MSW, and William H. Herman, MD, MPH

Objectives. We assessed the influence of maternal anthropometric and metabolic variables, including glucose tolerance, on infant birthweight.

Methods. In our prospective, population-based cohort study of 1041 Latino mother–infant pairs, we used standardized interviews, anthropometry, metabolic assays, and medical record reviews. We assessed relationships among maternal sociodemographic, prenatal care, anthropometric, and metabolic characteristics and birthweight with analysis of variance and bivariate and multivariate linear regression analyses.

Results. Forty-two percent of women in this study entered pregnancy overweight or obese; at least 36% exceeded weight-gain recommendations. Twenty-seven percent of the women had at least some degree of glucose abnormality, including 6.8% who had gestational diabetes. Maternal multiparity, height, weight, weight gain, and 1-hour screening glucose levels were significant independent predictors of infant birthweight after adjustment for gestational age.

Conclusion. Studies of birthweight should account for maternal glucose level. Given the increased risk of adverse maternal and infant outcomes associated with excessive maternal weight, weight gain, and glucose intolerance, and the high prevalence of these conditions and type 2 diabetes among Latinas, public health professionals have unique opportunities for prevention through prenatal and postpartum interventions. (*Am J Public Health.* 2006;96:2201–2208. doi:10.2105/AJPH.2005.065953)

In the United States, low birthweight is a primary indicator of poor maternal and neonatal health.[1] Despite the substantial proportion of Latino mothers who live in poverty and receive inadequate prenatal care,[2] low birthweight is a rare outcome, especially among infants of Mexican ancestry. This observation, which is cited as an example of the "Hispanic paradox," has resulted in efforts to understand the apparently protective social and cultural factors that might explain this "good outcome."[3–14]

This focus has diverted research and program attention from other aspects of maternal and child health in this population. Obesity, impaired glucose tolerance, and type 2 diabetes are common among Latino women of childbearing age.[15,16] Intrauterine metabolic abnormalities associated with these conditions are linked to increased risk for fetal overgrowth, developmental anomalies, birth injuries, and subsequent obesity and type 2 diabetes in childhood, youth, and later life.[17–22] Increased birthweight secondary to intrauterine overnutrition may be a marker of poor maternal and infant health in populations with a high prevalence of maternal obesity and diabetes. Our study assessed the combined influence of maternal weight and other anthropometric and metabolic characteristics on the birthweights of Latino infants.

METHODS

Study Setting and Population

This prospective, population-based cohort study was conducted at the Community Health and Social Services (CHASS) Center, a designated federally qualified community health center in southwest Detroit. Most patients who attend CHASS are of Mexican ancestry, are uninsured, and have incomes below the poverty level. Prenatal care is provided by bilingual and bicultural staff. By contractual arrangement, high-risk and tertiary obstetric care and labor and delivery services are provided to CHASS patients by Henry Ford Hospital in Detroit.

All 1346 Latino women who entered prenatal care at CHASS between January 5, 1999, and February 28, 2001, were recruited to participate in this study. Nine women with twin pregnancies, 6 women who entered care in the final weeks of pregnancy, and 39 women who had participated during their previous pregnancy were excluded, which left 1292 eligible women. No consent was obtained for 104 women (8.0%) including 49 who refused, 39 who were younger than 18 years and could not obtain parent/guardian consent, 13 who changed provider, and 3 who miscarried before consent could be obtained. On average, these women were younger, thinner, and entered care later than did consenting women. Of the 1188 consenting women, 147 (12.4%) were subsequently excluded from analyses, including 10 who miscarried, 6 who had a stillbirth, 1 whose records were missing, and 130 who were lost to follow-up. They entered prenatal care a week earlier but were otherwise similar to the 1041 women who, with their infants, constituted the study population.

Data Collection

Standardized interviews, anthropometry, and medical record reviews were conducted by trained study staff. Blood was drawn by CHASS laboratory personnel and processed by the University of Michigan's Michigan Diabetes Research and Training Center laboratories according to standard protocols. Maternal and family health history, obstetric history, last menstrual period, and medical complications since the last menstrual period were obtained at the first prenatal visit and abstracted from the medical record. Acculturation variables (birthplace inside or outside the United States, duration of US residence, primary language of Spanish or English, urban vs rural childhood environment) were obtained by interview with study staff at the first prenatal visit. Hispanic Health and Nutrition Survey questions about smoking before and during pregnancy were used to assess smoking behavior.[23] An index of prenatal care adequacy, which was coded as adequate (yes/no), included the trimester prenatal care began and the number of prenatal care visits given the gestational age of the infant at birth.[24]

Maternal prepregnancy weight was assessed with a questionnaire. When prepregnancy weight was unknown or missing, and a weight was obtained within the first 10 weeks of pregnancy, predicted prepregnancy weight was estimated using procedures described by Siega-Riz.[8] Height was measured with a stadiometer and weight was measured with a beam balance scale at the first prenatal visit. Blood pressure, measured with a sphygmomanometer, and weight measurements were obtained at each prenatal visit. Prepregnancy body mass index (BMI) (weight [kg]/height [m]^2) was calculated and coded into 4 Institute of Medicine–recommended categories: underweight (<19.8), normal weight (19.8–26.0), overweight (>26.0–29.0), and obese (>29.0).[25] Weight gain was calculated by subtracting prepregnancy weight from the last weight recorded during prenatal care if the last visit occurred within 7 days of delivery. Women without a reported or predicted prepregnancy weight nor a last weight recorded within 7 days of delivery, were excluded from analyses of pregnancy weight gain.

Anthropometry was performed at the first prenatal visit and no later than 25 weeks of gestation to reduce the influence of fetal growth on the measurement of waist circumference. Anthropometry was adjusted for duration of gestation at the measurement visit during data analysis. Circumferences were measured to the nearest 0.5 cm with a flexible tape. Waist circumference, a measure of body fat distribution, was calculated by measuring at the narrowest horizontal circumference in the area between the ribs and iliac crest. Hip circumference was measured at the level of maximal extension of the buttocks. Arm circumference was measured at the midpoint of the upper arm. Triceps skinfold thickness was measured to the nearest millimeter using a Lange skinfold caliper at the triceps at the same level where the arm circumference was obtained.

Women with random glucose levels 126 mg/dL or higher at the first prenatal visit received immediate diabetes screening or diagnostic testing. All women without a diagnosis of diabetes received a 1-hour, 50-g oral glucose screening test at approximately 26 weeks gestation. Assays for insulin and lipids were performed on blood samples drawn 1 hour after a drink containing 50 g of glucose was consumed. There are no reference standards for insulin or lipid levels during pregnancy. Women with glucose screening test results 130 mg/dL or higher received a 3-hour, 100-g oral glucose tolerance test. Gestational diabetes mellitus (GDM) was diagnosed with American Diabetes Association criteria, i.e., 2 or more glucose values 95 mg/dL or higher at fasting, 180 mg/dL or higher at 1 hour, 155 mg/dL or higher at 2 hours, and 140 mg/dL or higher at 3 hours.[26] Women with 1 abnormal oral glucose tolerance test value were referred to the CHASS dietitian who provided nutrition counseling similar to that provided to women with GDM. Women with GDM were referred to the Henry Ford Hospital Endocrinology Department for diabetes management.

Gestational age was established by a single perinatologist who was blinded to the glucose and birth outcome status of the mother–infant pair. An initial ultrasound assessment was obtained immediately after the first prenatal visit. The last menstrual period date was used when the difference between that date and that estimated from ultrasound measurements was less than 8 days. In all other cases, the ultrasound data were used. Birthweight was measured in grams within an hour of birth on a Detecto recumbent scale (Cardinal Scale Manufacturing Co, Webb City, Mo). Birthweight was adjusted for gestational age with analysis of covariance and by including gestational age as a covariate in the linear regression models.

Statistical Analysis

All statistical analysis and test procedures were performed with SAS software version 6.12 (SAS Institute, Cary, NC). Descriptive statistics were obtained with mean and standard deviation for continuous variables and frequencies and proportions for categorical variables. Maternal sociodemographic, anthropometric, and metabolic characteristics by 1-hour glucose category (<100 mg/dL, 100–129 mg/dL, ≥130 mg/dL) and gestational diabetes status were assessed with 1-way analysis of variance using general linear models and the Cochran–Mantel–Haenszel χ^2 test. Differences within groups were assessed with the Tukey pairwise comparison procedure. Analysis of covariance was used to compare means between groups after adjustment for covariates. Bivariate and multivariate linear regression analyses were performed to assess the relationships among sociodemographic, biological, anthropometric, and metabolic characteristics as independent variables and birthweight as the dependent outcome variable after adjustment for gestational age.

The normality and linearity assumptions of linear regression models were assessed by univariate analyses and by categorizing each continuous variable into multiple dichotomous variables. Interaction terms between independent variables were also considered. None of the interaction effects was statistically significant. The coefficient of determination (R^2) and adjusted R^2 (for the multiple linear regression model) were used as a quantitative measure of how well the independent variables explained the outcome. Pearson correlation analyses and partial correlation analyses were performed to assess the strength of the relation between the outcome variable (birthweight) and the independent variables after adjustment for gestational age. Multicolinearity among anthropometric variables made it difficult to determine their independent effects if included in the multivariate linear regression model together. Therefore, to select the best possible multivariate model, a forward stepwise selection procedure was performed with maternal sociodemographic, health, prenatal care, and metabolic variables, and the anthropometric variables were forced into the stepwise model separately. A P value <.05 was defined as the level of statistical significance.

RESULTS

Maternal Characteristics and Infant Outcomes

The age of study participants averaged 25 years (Table 1). Ninety-two percent of

TABLE 1—Selected Maternal Characteristics and Infant Outcomes for 1041 Latino Mother–Infant Pairs

	Value	N
Age, y, mean ±SD	25.2 ±5.1	1041
Multiparous, no. (%)		
0	429 (41.2)	1041
1-3	574 (55.1)	1041
≥4	38 (3.7)	1041
Married, no. (%)	548.0 (53.8)	1019
Education, years, mean ±SD	8.7 ±3.0	1039
Gestational age at entry to care, weeks, mean ±SD	17.4 ±5.6	1041
Prenatal visits, number, mean ±SD	9.5 ±2.6	1041
Health history, no. (%)		
History of gestational diabetes	33.0 (5.4)	612
History of gestational hypertension	39.0 (6.4)	612
Family history of diabetes	194.0 (18.6)	1041
Family history of hypertension	227.0 (21.8)	1041
Pregravid weight, kg, mean ±SD	63.4 ±12.9	950
BMI, no. (%, by IOM category)[a]		
<19.8	63 (6.7)	947
19.8–26.0	489 (51.6)	947
26.1–29.0	171 (18.1)	947
29.1–34.0	174 (18.4)	947
>34.0	50 (5.3)	947
Pregravid height, cm, mean ±SD	156.3 ±6.0	1036
Pregravid BMI, kg/m^2, mean ±SD	25.9 ±5.0	946
Weight gain, kg, mean ±SD	12.9 ±6.2	938
Weight gain, %, by IOM category[a]		
Inadequate	46.9	938
Adequate	16.9	938
Excessive	36.2	938
Waist circumference, cm, mean ±SD	90.7 ±11.0	986
Hip circumference, cm, mean ±SD	102.8 ±10.1	986
Waist–hip ratio, mean ±SD	0.9 ±0.1	986
Upper-arm circumference, cm, mean ±SD	28.2 ±3.7	986
Triceps skinfold, mm, mean ±SD	27.2 ±7.5	985
Upper-arm fat area, cm, mean ±SD	44.0 ±34.1	985
1-h glucose, mg/dL, mean ±SD	116.2 ±27.9	1001
1-h glucose level, mg/dL, no. (%)		
<100	291.0 (29.1)	1001
100–129	439.0 (43.9)	1001
≥130	271.0 (27.1)	1001
Insulin, μU/mL, mean ±SD	95.5 ±65.0	917
HDL, mg/dL, mean ±SD	63.6 ±17.2	917
TG, mg/dL, mean ±SD	214.1 ±71.6	917
Gestational diabetes, no. (%)	68.0 ±6.8	1001
Infant birthweight, g, mean ±SD	3408 ±497	1041

Continued

participants were born in Mexico and, on average, arrived in the United States at age 20 years. Overall, participants had lived in the United States slightly less than 5 years and averaged slightly less than 9 years of education. Eighty-five percent of these women last attended school outside the United States. Seventy-seven percent spoke only Spanish; less than 1% spoke only English. Almost half had lived in rural areas before age 16 years. Fifty-four percent reported being married, and 59% percent reported previous pregnancies.

On average, study participants entered prenatal care at 17.4 weeks of gestation and had 9.5 visits. Because most women entered care in the second trimester, only 11% of the women were classified as receiving adequate prenatal care. Most women received intermediate levels of care; only 14% received an inadequate level of prenatal care. No participants reported type 1 or type 2 diabetes or chronic hypertension before pregnancy. Approximately 1 in 5 women reported a family history of diabetes or hypertension. Among parous women, 5.4% reported histories of gestational diabetes and 6.4% reported histories of gestational hypertension. Smoking before pregnancy was reported by 9% of women. Only 2% continued to smoke during pregnancy.

On average, the women weighed 139 pounds before pregnancy and were 5 feet, 2 inches tall. The average prepregnancy BMI was 25.9 kg/m^2. Only 7% of women were classified as underweight, whereas 42% were either overweight or obese before they became pregnant. The mean weight gain during pregnancy was 12.9 kg (28.4 pounds). Thirty-six percent of the women gained more than the recommended amount of weight during pregnancy, including more than half of overweight women and 38% of obese women. At the first prenatal visit, the mean waist, hip, and upper-arm circumferences were 90.7 cm, 102.8 cm, and 28.2 cm, respectively. The mean triceps skinfold measurement was 27.2 mm.

The mean glucose level derived from the 1-hour, 50-g glucose screen test was 116 mg/dL; and 27% of the women had abnormal glucose values of greater than 130 mg/dL. The mean 1-hour insulin level was 96 μU/mL, and the mean insulin–glucose ratio, a measure of insulin resistance, was 0.81. The mean 1-hour total cholesterol level was

TABLE 1—*Continued*

Infant birthweight, g, no. (%)		
<1500	5 (0.5)	1041
1500–2499	32 (3.1)	1041
2500–3999	913 (87.7)	1041
≥4000	91 (8.7)	1041
Infant gestational age, weeks, mean ±SD	39.3 ±1.9	1041
Gestational age at delivery, weeks, no. (%)		
<33	13 (1.3)	1041
33–36.9	67 (6.4)	1041
≥37	961 (92.3)	1041

Notes. SD = standard deviation; BMI = body mass index; HDL = high-density lipoprotein; TG = triglyceride.
[a]Institute of Medicine–recommended pregnancy weight-gain category by prepregnancy weight: BMI < 19.8, gain 28 to 40 lbs; BMI ≥ 19.8 to ≤ 26, gain 25 to 35 lbs; BMI > 26.0, gain 15 to 25 lbs.

223 mg/dL, the mean 1-hour high-density lipoprotein level was 64 mg/dL, and the mean 1-hour triglyceride level was 214 mg/dL. Women with 1-hour glucose levels of 130 mg/dL or higher were referred for 3-hour, 100-g oral glucose tolerance tests. Sixty-eight women (27% of women tested and 6.8% of the study population) had GDM.

The mean birthweight of infants born to study participants was 3408 g. Ninety-one infants (8.7%) weighed 4000 g or more. Among infants born to women with GDM, 13.2% weighed 4000 g or more compared with 8.8% among women without GDM. Only 3.6% of infants weighed less than 2500 g. The average gestational age of study infants was 39.3 weeks. Only 7.7% of infants were preterm (gestational age <37 completed weeks). Preterm birth was much more frequent (19.1%) among infants of mothers with GDM than among infants of mothers without GDM (5.9%) (data not shown).

Maternal Glucose Category, Maternal Characteristics, and Infant Outcomes

Mean maternal age and the percentage of women with a family history of diabetes were significantly higher with each successive maternal glucose category at 26 weeks gestation, and were greatest in women with GDM (Table 2). Maternal multiparity was not associated with significant differences in glucose category. There were no significant relations between maternal glucose category and parity, marital status, maternal education in years, any acculturation variable, prenatal care adequacy, smoking, or history of gestational hypertension (data not shown).

There were significant linear relations between maternal glucose categories and several anthropometric and metabolic variables. Mean BMI; waist, hip, and upper-arm circumference; and upper-arm fat area increased significantly with increasing glucose category, and were greatest in women with GDM. Women with GDM had significantly lower average weight gain than those without GDM, but weight gain was not significantly related to glucose category. Height was not significantly different by glucose category or GDM status. Insulin and triglyceride values increased significantly with each increase in glucose category and were highest among women with GDM. High-density lipoprotein values were not significantly different by glucose category or between women with GDM compared with those without GDM.

There was a significant linear trend among maternal glucose category, gestational age, and mean birthweight. Among women without GDM, mean gestational age was slightly but significantly higher with successively higher glucose category. Women with GDM had shorter gestations than did women without GDM in all glucose-level categories. Birthweights that were unadjusted for gestational length followed a similar trend, with successively greater birthweights at each increase in glucose-level category for women without GDM. Infants born to mothers with GDM had lower unadjusted birthweights than all mothers except those with glucose levels less than 100 mg/dL. After adjustment for gestational age, the significant linear increase in birthweight among infants of women without GDM persisted and continued among infants of GDM mothers, whose adjusted birthweights were the greatest. The significant linear increase in birthweight with increasing glucose level was maintained after further adjustment for maternal age, parity, BMI, weight gain, hypertensive disorders, and family history of diabetes.

Maternal Characteristics and Birthweight, Adjusted for Gestational Age

In bivariate regression analyses, birthweight was associated with multiparity and higher values of maternal age, as well as 7 anthropometric variables: BMI, weight gain, height, waist and hip circumference, triceps skinfold, upper-arm circumference, and upper-arm fat area after adjustment for gestational age (Table 3).

Each 10-mg/dL increase in the 1-hour glucose value was associated with a 19-g increase in birthweight adjusted for gestational age. Among women who had an oral glucose tolerance test because their screen glucose levels were greater than 130 mg/dL, each 10-unit increase in the fasting glucose value was significantly associated with a 41-g increase in birthweight adjusted for gestational age ($P = .04$). There was no significant relation between any other metabolic characteristic, nor adequate prenatal care, marital status, maternal education, acculturation, smoking, or family history of diabetes and birthweight adjusted for gestational age.

In the final multivariate model, significant independent predictors of birthweight were multiparity, BMI, height, weight gain, and 1-hour glucose value after adjustment for gestational age. Hip circumference was marginally significant. The maternal glucose value accounted for 16 g of increased birthweight for each 10-mg/dL increase in glucose value, after adjusting for the other variables in the model (Table 4).

DISCUSSION

In this 2-year, prospective, population-based cohort study of primarily Mexican

TABLE 2—Maternal Characteristics and Infant Outcomes by 1-Hour Glucose Categories for 1041 Latino Mother–Infant Pairs

	Glucose Level (mg/dL) of Non-GDM Mothers					GDM Mothers (n = 68)	
	<100 (n = 291), Mean ±SE	100–129 (n = 437), Mean ±SE	≥130 (n = 205), Mean ±SE	Overall (n = 933), Mean ±SE	P^a	Mean ±SE	P^b
Maternal age, years	23.3 ±0.3$_{xy}$	25.0 ±0.2$_x$	26.7 ±0.4	24.8 ±0.2	<.01	28.6 ±0.6	<.01
Body mass index, kg/m^2	24.8 ±0.2$_{xy}$	25.8 ±0.2	26.6 ±0.4	25.7 ±0.2	<.01	28.4 ±0.8	<.01
Weight gain, kg	12.9 ±0.3	13.4 ±0.3	12.7 ±0.5	13.0 ±0.2	NS	10.0 ±0.6	NS
Height, cm	156.0 ±0.4	156.2 ±0.3	156.7 ±0.4	156.3 ±0.4	NS	155.2 ±0.7	NS
Waist circumference, cm	87.1 ±0.6$_{xy}$	90.3 ±0.5$_x$	93.0 ±0.7	90.1 ±0.6	<.01	97.8 ±1.6	<.01
Hip circumference, cm	100.1 ±0.6$_{xy}$	102.7 ±0.5	104.5 ±0.7	102.8 ±0.3	<.01	107.2 ±1.3	<.01
Upper-arm circumference, cm	27.2 ±0.2$_{xy}$	28.1 ±0.2	28.8 ±0.3	28.0 ±0.2	<.01	30.7 ±0.5	<.01
Upper-arm fat area, cm	38.9 ±1.9$_x$	45.0 ±1.5	46.8 ±2.1	43.6 ±1.8	<.01	50.8 ±4.4	<.01
Insulin, μU/mL	64.8 ±2.6$_{xy}$	93.4 ±2.7$_x$	129.3 ±5.7	92.2 ±2.0	<.01	144.4 ±8.2	<.01
Glucose, mg/dL	86.4 ±0.6$_{xy}$	113.8 ±0.4$_x$	145.9 ±0.9	112.3 ±0.3	<.01	168.2 ±3.3	<.01
Triglyceride, mg/dL	200.8 ±3.7$_{xy}$	216.1 ±3.4	219.4 ±5.1	212.0 ±2.3	<.01	247.6 ±12.0	<.01
HDL, mg/dL	64.9 ±1.2	63.5 ±0.8	63.8 ±1.1	64.0 ±0.6	NS	57.9 ±1.6	NS
Gestational age, weeks	39.3 ±0.1$_x$	39.5 ±0.1	39.7 ±0.1	39.5 ±0.1	<.01	38.5 ±0.3	.01
Unadjusted birthweight, grams	3336.7 ±51.3$_x$	3419.0 ±44.4	3513.0 ±63.4	3422.9 ±53.0	<.01	3396.7 ±148.9	<.01
Birthweight, grams, adjusted for gestational age at delivery in weeks	3357.9 ±24.2$_x$	3404.0 ±19.7	3474.9 ±28.8	3412.3 ±24.2	<.01	3519.6 ±50.5	<.01
Birthweight, grams, adjusted for gestational age at delivery, body mass index, parity, maternal age, weight gain	3390.9 ±25.1	3396.2 ±20.0	3462.0 ±29.8	3408.9 ±25.0	<.01	3517.0 ±57.3	<.01
Multiparous, %	58.1	54.0	66.0	57.9	NS[c]	61.8	NS
Family history of diabetes, %	12.0	19.7	23.3	18.1	<.01[c]	26.5	<.01

Notes. GDM = gestational diabetes mellitus; NS = not significant ($P > .05$); subscript x = $P \leq .05$ versus glucose ≥130; subscript y = $P \leq .05$ versus glucose = 100–129.
[a]Analysis of variance using general linear model (glucose <100, 100–129, ≥130).
[b]Analysis of variance using general linear model (glucose <100, 100–129, ≥130, GDM).
[c]Cochran–Mantel–Haenszel χ^2 test for trend.

women residing in Detroit, we assessed the influence of maternal anthropometric and metabolic variables, including glucose tolerance, on infant birthweight. Maternal glucose level was significantly associated with birthweight, even when maternal weight and weight gain were considered. This confirms and extends the results of our previous study[22] and studies in other populations.[27–31] Maternal waist, hip, and upper-arm circumference and upper-arm fat area were associated with increasingly abnormal glucose categories among women with and without GDM. Greater values of these characteristics were also associated with higher infant birthweights in bivariate, but not multivariate, analyses that accounted for weight, weight gain, and glucose level.

More than a quarter of the study population had some degree of glucose intolerance, including 27% with an abnormal (≥130 mg/dL) 1-hour screening glucose value. Similar to our previous study, we found that among women without GDM, the highest birthweights occurred among infants of mothers whose screening glucose values were 130 mg/dL or higher.[22] Among women without GDM in our study, each 10-mg/dL increase in 1-hour glucose level was associated with a 16-g increase in birthweight, after adjusting for gestational age, parity, maternal BMI, height, pregnancy weight gain, and hip circumference. These variables attenuated but did not fully explain the effect of maternal glucose on birthweight. All women with an abnormal glucose screen were referred for further testing.[16,22]

Gestational diabetes was diagnosed in 6.8% of the women, which is higher than that found in a recent study of non-Hispanic White and African American women that used the same diagnostic criteria.[30] The percentage of women with 1 abnormal oral glucose tolerance test value was 3.3%. This condition, which has been defined as impaired glucose tolerance, has been associated with increased birthweight and macrosomia in African American women.[30] The number of women with impaired glucose tolerance in our study was too small to assess this relation. Women with GDM or impaired glucose tolerance received dietary and weight management counseling according to

TABLE 3—Bivariate Linear Regression Analyses of Maternal Characteristics and Birthweight Adjusted for Gestational Age: 933 Latino Mothers Without GDM and Their Infants

	Adjusted R^2 [a]	PE[b]	SE	*P*
Maternal age	0.31	7.7	2.5	<.01
Multiparous	0.31	43.3	11.1	<.01
BMI, kg/m^2	0.32	13.2	2.7	<.01
Weight gain, kg	0.30	8.2	2.2	<.01
Height, cm	0.02	10.8	2.5	<.01
Waist circumference, cm	0.31	6.2	1.2	<.01
Hip circumference, cm	0.32	9.1	1.3	<.01
Triceps skinfold, mm	0.32	10.3	1.7	<.01
Upper-arm circumference, cm	0.32	24.1	3.5	<.01
Upper-arm fat area, cm	0.30	1.1	0.4	<.01
Glucose, mg/dL	0.27	19.3	0.5	<.01
Triglyceride, mg/dL	0.26	3.2	0.2	.09

Notes. GDM = gestational diabetes mellitus; PE = parameter estimate; BMI = body mass index (kg/m^2).
[a]Adjusted for gestational age.
[b]Parameter estimate refers to the effect of a 1-unit increase in the maternal characteristic.

TABLE 4—Results of Multivariate Linear Regression Analyses[a] of Selected Maternal Characteristics and Birthweight: 933 Latino Mothers Without GDM and Their Infants

	PE[b]	SE	*P*
Multiparous	101.2	27.8	<.01
BMI, kg/m^2	30.7	6.2	<.01
Height, cm	10.2	2.8	<.01
Weight gain, kg	19.7	2.8	<.01
Glucose, mg/dL	15.9	6.3	.01
Hip circumference, cm	5.5	3.0	.06

Notes. GDM = gestational diabetes mellitus; PE = parameter estimate; BMI = body mass index (kg/m^2).
[a]Adjusted for gestational age.
[b]Parameter estimate refers to the effect of a 1-unit increase in the maternal characteristic.

clinic protocol. This practice may have reduced the impact of higher glucose levels on birthweight compared with those previously reported.[22,31]

We found no statistically significant independent associations between several other metabolic fuels and birthweight. A previous study found that the 1-hour triglyceride value was a significant predictor of birthweight among women with or at high risk for GDM but not among women with normal glucose status.[27] Another found a significant association between 1-hour triglyceride value and birthweight independent of BMI and weight gain, but glucose status was not included in their model.[32] In our study, when the glucose category was included, this relationship was attenuated and was not significant in our multivariate model.

Factors commonly used in perinatal research, policy, and practice, such as maternal age, education, marital status, and adequacy of prenatal care, showed no association with birthweight in our multivariate model after adjustment for gestational age.[1,2,21] We also found no association between any measure of acculturation and birthweight. Because more than 90% of our study participants were born in Mexico, and because their duration of US residence was short, there may have been insufficient variation in birthplace and acculturation levels to find significant associations. Others have found that low levels of acculturation and increased length of time in the United States were associated with higher infant birthweights.[13] Mexican-born women had significantly lower risks than did US-born Mexican women and non-Latino White women, whose risks of low birthweight were similar.[14]

Several limitations apply to this study. Our results may not apply to all US Latino women because a greater percentage of women in this study were Mexican and recent immigrants, and their levels of education and prenatal care were lower than US Latino women overall.[2] Nonetheless, our participants were representative of pregnant women in a growing number of communities throughout this country. Lack of first-trimester anthropometry, and more sensitive measures of metabolic status such as the fasting and 2-hour postglucose values of glucose, insulin, and lipids from the oral glucose tolerance test were not suitable for most women. This may have reduced our ability to detect the full influence of maternal anthropometric and metabolic characteristics on birthweight.

Overweight, obesity, abnormal glucose tolerance, and GDM were prevalent among Latino women in this study. Their infants' birthweights were influenced by these conditions and by maternal weight gain. These results suggest that research related to birth outcomes, including assessments of ethnic disparities in fetal growth, should include measures of glucose tolerance during pregnancy, maternal weight, and weight gain in addition to more commonly used sociodemographic, prenatal care, and behavioral variables. This work should not be limited to mothers with diabetes, but should include the whole range of glucose tolerance. Longitudinal studies that include anthropometry and metabolic measures from early until late in pregnancy are also needed to better assess the influence of the intrauterine metabolic environment on subsequent maternal, infant, and child health among Latino children, given their increased risk of early onset type 2 diabetes.[33]

Almost half of the women in this study entered pregnancy overweight or obese. More than a third of the women exceeded the Institute of Medicine weight gain recommendations, particularly if they were already overweight or obese. These percentages are similar to those for Latinas nationally.[34] However, these estimates are conservative. Calculating BMI using current standards for nonpregnant adults would increase the prevalence of both excessive prepregnancy weight and pregnancy-associated weight gain. Excessive maternal weight, weight gain, and glucose intolerance are each associated with adverse maternal and newborn outcomes and with increased risk for GDM and subsequent type 2 diabetes in both mothers and infants.[17–21,33,35–37]

Dietary modifications and increased physical activity during pregnancy may reduce excessive weight gain, improve maternal glucose status and decrease associated maternal and infant complications.[26,38–41] Similar lifestyle modifications and breastfeeding, along with postpartum glucose testing and diabetes education, may also reduce morbidity associated with undetected or untreated impaired glucose tolerance and type 2 diabetes.[20,26,39] Because most women have repeated contact with either public health or private health care systems during pregnancy and immediately after, public health professionals and prenatal care providers have unique opportunities to promote healthy outcomes among mothers and infants at risk for obesity and type 2 diabetes. ■

About the Authors

At the time of this study, Edith C. Kieffer and Wendy J. Carman were with the Department of Health Behavior and Health Education, University of Michigan, Ann Arbor. Bahman P. Tabaei and William H. Herman were with the Departments of Internal Medicine and Epidemiology, University of Michigan, Ann Arbor. George H. Nolan was with the Department of Obstetrics and Gynecology, Henry Ford Health System, Detroit, Mich. J. Ricardo Guzman was with Community Health and Social Services, Inc, Detroit.

Requests for reprints should be sent to Edith C. Kieffer, University of Michigan School of Social Work, South University, Room 3770, Ann Arbor, MI 48109-1106 (e-mail: ekieffer@umich.edu).

This article was accepted December 20, 2005.

Contributors

E.C. Kieffer led the study design and implementation and the writing of the article. B.P. Tabaei and W.J. Carman contributed to the analysis plan and conducted the statistical analysis. B. Tabaei also contributed to writing the article. G.H. Nolan and J.R. Guzman assisted with study design and interpretation. W.H. Herman contributed to study design and implementation and to writing the article. All authors helped to conceptualize ideas, interpret findings, and review drafts of the manuscript.

Acknowledgments

This study was supported by the National Institutes of Diabetes and Digestive and Kidney Diseases (grant R18 DK 062344); the Biostatistics and Measurement Cores of the Michigan Diabetes Research and Training Center (grant NIH5P60 DK20572); the General Clinic Center, National Institutes of Health (grant M01 RR00042); the Maternal and Child Health Bureau (grant R40 MC 00115-03); and the Detroit Community Academic Urban Research Center.

The authors thank Cathy Zavala Sanborn, Marilyn Lugo, and the staff and patients of Community Health and Social Services Center in Detroit.

Human Participant Protection

This study was approved by the University of Michigan's institutional review board.

References

1. *Healthy People 2000: National Health Promotion and Disease Prevention Objectives.* Washington DC: US Dept of Health and Human Services; 1991. DHHS publication PHS 91-50212.

2. Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Menacker F, Munson ML. Births: final data for 2002. *Natl Vital Stat Rep.* 2003;52:1-113.

3. Kieffer EC. Maternal obesity and glucose intolerance during pregnancy among Mexican-Americans. *Paediatr Perinat Epidemiol.* 2000;14:14–19.

4. Magana A, Clark NM. Examining a paradox: does religiosity contribute to positive birth outcomes in Mexican American populations? *Health Educ Q.* 1995;22: 96–109.

5. Fuentes-Afflick E, Hessol NA, Perez-Stable EJ. Testing the epidemiologic paradox of low birthweight in Latinos. *Arch Pedatr Adolesc Med.* 1999;153: 147–153.

6. Guendelman S, English P, Chavez G. Infants of Mexican immigrants. Health status of an emerging population. *Med Care.* 1995;33:41–52.

7. Balcazar H, Cole G, Hartner J. Mexican-Americans' use of prenatal care and its relationship to maternal risk factors and pregnancy outcome. *Am J Prev Med.* 1992;8:1–7.

8. Siega-Riz AM, Adair LS, Hobel CJ. Institute of Medicine maternal weight gain recommendations and pregnancy outcome in a predominantly Hispanic population. *Obstet Gynecol.* 1994;84:565–573.

9. Cobas JA, Balcazar H, Benin MB, Keith VM, Chong Y. Acculturation and low-birthweight infants among Latino women: a reanalysis of HHANES data with structural equation models. *Am J Public Health.* 1996;86:394–396.

10. Guendelman S, Abrams B. Dietary intake among Mexican-American women: generational differences and a comparison with White, non-Hispanic women. *Am J Public Health.* 1995;85:20–25.

11. Zambrana R, Scrimshaw SC, Collins N, Dunkel-Schetter C. Prenatal health behaviors and psychosocial risk factors in pregnant women of Mexican origin: the role of acculturation. *Am J Public Health.* 1997;87: 1022–1026.

12. Wolff CB, Portis M. Smoking, acculturation and pregnancy outcome among Mexican Americans. *Health Care Women Int.* 1996;17:563–573.

13. Balcazar H, Krull JL. Determinants of birth-weight outcomes among Mexican-American women: examining conflicting results about acculturation. *Ethn Dis.* 1999;9:410–422.

14. Cervantes A, Keith L, Wyshak G. Adverse birth outcomes among native-born and immigrant women: replicating national evidence regarding Mexicans at the local level. *Matern Child Health J.* 1999;3:99–109.

15. Harris MI, Flegal KM, Cowie CC, et al. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in US adults. The Third National Health and Nutrition Examination Survey, 1988-1994. *Diabetes Care.* 1998;21:518–524.

16. Kieffer EC, Carman WJ, Gillespie BW, Nolan GH, Worley SE, Guzman JR. Obesity and gestational diabetes among African-American and Latinas in Detroit: implications for disparities in women's health. *J Am Med Womens Assoc.* 2001;56:181–187.

17. Buchanan TA, Catalano PM. The pathogenesis of GDM: implications for diabetes after pregnancy. *Diabetes Reviews.* 1995;3:584–601.

18. Vohr BR, McGarvey ST. Growth patterns of large-for-gestational-age and appropriate-for-gestational-age infants of gestational diabetic mothers and control mothers at age 1 year. *Diabetes Care.* 1997;20: 1066–1072.

19. Silverman BL, Rizzo TA, Cho NH, Metzger BE. Long-term effects of the intrauterine environment. The Northwestern University Diabetes in Pregnancy Center. *Diabetes Care.* 1998;21(suppl 2):B142–B149.

20. Pettitt DJ, Knowler WC. Long-term effects of the intrauterine environment, birthweight and breast-feeding in Pima Indians. *Diabetes Care.* 1998;21(suppl 2): B138–B141.

21. Sermer M, Naylor CD, Gare DJ, et al. Impact of increasing carbohydrate intolerance on maternal-fetal outcomes in 3637 women without gestational diabetes. The Toronto Tri-Hospital Gestational Diabetes Project. *Am J Obstet Gynecol.* 1995;173:146–156.

22. Kieffer EC, Nolan GH, Carman WJ, Sanborn CZ, Guzman R, Ventura A. Glucose tolerance during pregnancy and birthweight in a Hispanic population. *Obstet Gynecol.* 1999;94:741–746.

23. National Center for Health Statistics. *Plan and operation of the Hispanic Health and Nutrition Examination Survey. 1982–1984.* Washington, DC: US Government Printing Office; 1985. Vital and Health Statistics. Series 1, No. 19. DHHS publication PHS 85–1321.

24. Alexander GR, Kotelchuck M. Quantifying the adequacy of prenatal care: a comparison of indices. *Public Health Rep.* 1996;111:408–418.

25. Institute of Medicine. *Nutrition During Pregnancy.* Washington, DC: National Academy of Sciences; 1990.

26. American Diabetes Association. Gestational diabetes mellitus. *Diabetes Care.* 2000;23(Suppl 1): S77–S79.

27. Nolan CJ, Riley SF, Sheedy MT, Walstab JE, Beischer NA. Maternal serum triglyceride, glucose tolerance and neonatal birthweight ratio in pregnancy. *Diabetes Care.* 1995;18:1550–1556.

28. Green JR, Schumacher LB, Pawson IG, Partridge JC, Kretchmer N. Influence of maternal body habitus and glucose tolerance on birthweight. *Obstet Gynecol.* 1991; 78:235–240.

29. Farmer G, Hamilton-Nicol DR, Sutherland HW, Ross IS, Russell G, Pearson DW. The ranges of insulin response and glucose tolerance in lean, normal and obese women during pregnancy. *Am J Obstet Gynecol.* 1992;167:772–777.

30. Saldana TM, Siega-Riz AM, Adair LS, Savitz DA, Thorp JM Jr. The association between impaired glucose tolerance and birthweight among Black and White women in central North Carolina. *Diabetes Care.* 2003; 26:656–661.

31. Scholl TO, Sowers MF, Chen X, Lenders C. Maternal glucose concentration influences fetal growth, gestation, and pregnancy complications. *Am J Epidemiol.* 2001;154:514–520.

32. Knopp RH, Magee MS, Walden CE, Bonet B, Benedetti TJ. Prediction of infant birthweight by GDM screening tests. Importance of plasma triglyceride. *Diabetes Care.* 1992;15:1605–1613.

33. Neufeld ND, Raffel LJ, Landon C, Chen YD, Vadheim CM. Early presentation of type 2 diabetes in Mexican-American youth. *Diabetes Care.* 1998;21:80–86.

34. Schieve LA, Cogswell ME, Scanlon KS. Trends in pregnancy weight gain within and outside ranges recommended by the Institute of Medicine in a WIC population. *Matern Child Health J.* 1998;2:111–116.

35. Cnattingius S, Bergstrom R, Lipworth L, Kramer MS. Prepregnancy weight and the risk of adverse pregnancy outcomes. *New Engl J Med.* 1998;338:147–152.

36. Rosenbloom AL, Joe JR, Young RS, Winter WE. Emerging epidemic of type 2 diabetes in youth. *Diabetes Care.* 1999;22:345–354.

37. Harris SB, Zinman B. Primary prevention of type 2 diabetes in high-risk populations. *Diabetes Care.* 2000;23:879–881.

38. Gunderson EP. Intensive nutrition therapy for gestational diabetes. Rationale and current issues. *Diabetes Care.* 1997;20:221–226.

39. Gregory KD, Kjos SL, Peters RK. Cost of non-insulin-dependent diabetes in women with a history of gestational diabetes: implications for prevention. *Obstet Gynecol.* 1993;81:782–786.

40. Horton ES. Exercise in the treatment of NIDDM. Applications for GDM? *Diabetes.* 1991;40(suppl 2):175–178.

41. Kjos SL, Henry O, Lee RM, Buchanan TA, Mishell DR Jr. The effect of lactation on glucose and lipid metabolism in women with recent gestational diabetes. *Obstet Gynecol.* 1993;82:451–455.