Maternal Obesity and Diabetes as Risk Factors for Adverse Pregnancy Outcomes: Differences Among 4 Racial/Ethnic Groups

Terry J. Rosenberg, PhD, Samantha Garbers, MPA, Heather Lipkind, MD, and Mary Ann Chiasson, DrPH

Objectives. We examined associations between obesity, diabetes, and 3 adverse pregnancy outcomes—primary cesarean delivery, preterm birth, and low birthweight (LBW)—by racial/ethnic group. Our goal was to better understand how these associations differentially impact birth outcomes by group in order to develop more focused interventions.

Methods. Data were collected from the 1999, 2000, and 2001 New York City birth files for 329 988 singleton births containing information on prepregnancy weight and prenatal weight gain. Separate logistic regressions for 4 racial/ethnic groups predicted the adverse pregnancy outcomes associated with diabetes. Other variables in the regressions included obesity, excess weight gain, hypertension, preeclampsia, and substance use during pregnancy (e.g., smoking).

Results. Chronic and gestational diabetes were significant risks for a primary cesarean and for preterm birth in all women. Diabetes as a risk for LBW varied by group. For example, whereas chronic diabetes increased the risk for LBW among Asians, Hispanics, and Whites (adjusted odds ratios = 2.28, 1.69, and 1.59), respectively, it was not a significant predictor of LBW among Blacks.

Conclusions. In this large, population-based study, obesity and diabetes were independently associated with adverse pregnancy outcomes, highlighting the need for women to undergo lifestyle changes to help them control their weight during the childbearing years and beyond. (*Am J Public Health.* 2005;95: 1544–1551. doi:10.2105/AJPH.2005.065680)

The prevalence of diabetes among American women continues to increase,[1,2] with Blacks and Hispanics more likely to be affected than non-Hispanic Whites.[3] Diabetes is the most common medical complication of pregnancy.[4] In the United States in 2002, 131 027 births included diabetes as a medical risk factor, translating to a rate of 32.8 per 1000 live births.[5] Because diabetes in pregnancy—both pregestational and gestational diabetes—can affect the mother and the infant, increases in the prevalence of diabetes among women of reproductive age is a public health concern.[6]

The associations between pregestational and gestational diabetes, adverse pregnancy and birth outcomes, and race have been well established in the literature. Pregnant women with pregestational type 1 and type 2 diabetes are more likely to have cesarean deliveries, macrosomic infants, fetal congenital malformations, and preterm deliveries.[7–9] Gestational diabetes has also been associated with adverse birth outcomes, including preterm birth, macrosomia and related shoulder dystocia, and cesarean deliveries.[10–15] Although both pregestational and gestational diabetes are strongly associated with higher birthweights, in the presence of vascular disease associated with diabetes, birthweight may be restricted.[4] Women with gestational diabetes have a 20% to 50% chance of developing type 2 diabetes in the next 5 to 10 years, and their offspring are at increased risk of developing diabetes and obesity later in life.[3,16,17]

Obesity is a major contributing factor to the 2 most common medical risks in pregnancy: diabetes and hypertension. The incidence of hypertension and preeclampsia is increased in pregnant women with pregestational diabetes and is related to both underlying vascular disease and pregestational hypertension.[18,19] Obesity has also been shown to be an independent risk factor for a longer, more difficult delivery and for a cesarean delivery.[20,21] Although obesity is associated with an increased risk of large-for-gestational age and macrosomic infants, obese women also have an increased risk of hypertensive disorders, including preeclampsia, which may be associated with low birthweight (LBW).[22] For these reasons, the role of prepregnancy weight and pregnancy weight gain, both in terms of their relation to these medical risks and as independent predictors of pregnancy and birth outcomes, are increasingly being examined.[8,19–23] Furthermore, 1 researcher has suggested that maternal obesity and diabetes may act "synergistically" to increase the risk of noncongenital defects in newborns.[9]

In pregnancy, gestational diabetes is often preexisting type 2 diabetes that has not been diagnosed.[4] The Fourth International Workshop-Conference on Gestational Diabetes and the American College of Obstetricians and Gynecologists have recommended that women of Hispanic, African, Native American, South or East Asian, Pacific Island, or Indigenous Australian ancestry, who are at higher risk for gestational diabetes, be screened for diabetes.[24] In a large prospective trial in Canada, Naylor et al.[25] identified Asian race as a risk factor for gestational diabetes. For the United States, Solomon et al.[26] found that the risk of gestational diabetes was increased among non-White women in the Nurses' Health Study Cohort II.

Given that rates of obesity and diabetes are higher among some racial and ethnic subgroups, particularly among Black women in the United States,[27] several analyses have examined the independent effects of obesity and diabetes and adverse birth outcomes by race. Saldana et al.[28] found a significant interaction between glucose status and race, so their analyses were stratified by race looking at Black and White mothers separately.

There is also evidence that the obesity-related risks during pregnancy vary by race/ethnicity, with obese Hispanic and Black women more likely to have adverse outcomes than obese White women.[29] Prior research has found racial differentials in the effects of impaired glucose tolerance and glucose levels on birth outcomes, with these conditions leading to higher levels of macrosomic babies among Black women but not among White women.[28,30,31]

The present analysis builds on previously published literature by using a large, population-based data set of births to the diverse population of women in New York, NY. The data set allows ample statistical power to conduct separate analyses examining the associations between diabetes, obesity, and 3 adverse pregnancy and birth outcomes—primary cesarean delivery, preterm birth, and LBW—in women of 4 different racial and ethnic subgroups. By conducting an analysis in this way, we were able to separate subgroup disparities in the prevalence of diabetes and associated risk factors (including obesity and hypertensive disorders) from the disproportionate impact these risk factors may have on the adverse pregnancy and birth outcomes of interest.

METHODS

Population

The data source was a combined New York City birth file for 1999, 2000, and 2001 (birth certificate data obtained from the New York City Department of Health and Mental Hygiene, Office of Vital Statistics). We restricted the analysis to the 329 988 live singleton births whose certificates included information on maternal prepregnancy weight and maternal weight gain during pregnancy (from a total of 373 325 live births for the 3-year period; some were multiple births, and 9.2% were missing the weight data).

Definition of Variables

The birth file contains 2 separate variables for diabetes—1 for chronic or overt diabetes (not specified as either type 1 or type 2) and 1 for gestational diabetes. It also contains 2 separate variables for hypertension (other than preeclampsia and eclampsia)—1 for chronic hypertension and 1 for pregnancy-induced hypertension. For this analysis, the 2 separate categories of diabetes and the 2 separate categories of hypertension were maintained. Maternal diabetes was divided into pregestational diabetes (including types 1 and 2) and gestational diabetes (including both diet- and insulin-treated subjects). Our rationale is that chronic diabetes has been shown to have a greater negative impact on pregnancy outcome than gestational diabetes.[4] When the file indicated that a woman had both the chronic and the pregnancy-related form of diabetes or hypertension, she was recoded to have only the chronic form. Preeclampsia (6981 cases) and eclampsia (228 cases) appear as separate variables on the birth file. Because eclampsia is a manifestation of very severe preeclampsia related to access to care rather than the risk factors discussed here, women diagnosed with eclampsia were excluded from the analysis. We accepted the diagnoses of all these conditions as determined by the physician completing the birth certificate, assuming that standard American College of Obstetrics and Gynecology definitions were used.

Because the birth file does not contain data on the mother's height, we could not compute body mass index; instead, we used the mother's prepregnancy weight as the measure of overweight and obesity. After constructing 5 categories of prepregnancy weight, we considered the women in the top 2 groups, 200 to 299 pounds (91 to 135 kg) and 300 pounds (136 kg) or more as overweight or obese. For a woman whose height is 5 feet 10 inches, a weight of 200 pounds corresponds to a body mass index of 29 kg/m^2, and a weight of 300 pounds corresponds to a body mass index of 43 kg/m^2. In all the logistic regressions, the modal group of women with prepregnancy weights of 100 to 149 pounds were chosen as the reference group ($n = 211\,288$). Excess weight gain was defined as 41 pounds (18 kg) or more, which is higher than the maximal weight gain of 40 pounds recommended for any group by the Institute of Medicine.[32]

The 3 outcome variables for the analysis were primary cesarean delivery, preterm birth (delivery before 37 weeks of gestation), and LBW (2500 grams or less). The analysis was limited to primary cesareans to avoid the complicating effect of a prior cesarean on the current delivery.

Sociodemographic variables were included in the analysis to control for potential confounding. These variables were mother's age (<20, 20 to 34, and >34), marital status, mother's education, mother's birthplace (US or foreign-born), prenatal care payer (e.g., Medicaid), social risk (smoking, alcohol, and illegal drug use), parity, and the trimester in which prenatal care began. Mother's age is an important confounder because both teenagers and women older than 35 years are more prone to develop preeclampsia. Additionally, because preeclampsia occurs most often in nulliparous women, parity is another important confounder.[22]

The 4 racial/ethnic groups created from the birth file were non-Hispanic Black (Black), non-Hispanic White (White), non-Hispanic Asian (Asian), and Hispanic.

Analysis

The bivariate associations between race/ethnicity and the key variables were first evaluated using analysis of variance (ANOVA) and χ^2 tests. In addition to their direct influences on pregnancy outcomes, obesity and diabetes indirectly affect pregnancy outcomes through their causal relation with preeclampsia; therefore, regression models predicting preeclampsia were tested next. These models evaluated the contributions of weight, weight gain, diabetes, and hypertension to preeclampsia in logistic regressions for each racial/ethnic group. The incidence of preeclampsia and hypertension is known to be increased in pregnant women with diabetes because of both pregestational hypertension and underlying vascular disease.[4] Furthermore, worsening hypertension and preeclampsia is often the mediating event that forces preterm delivery in diabetic women.[4,22] Multiple logistic regressions were then tested for the total population and within each racial/ethnic group for 3 adverse pregnancy outcomes—primary cesarean delivery, preterm birth, and LBW—with each regression including preeclampsia as a risk factor.

The data analysis was completed with SPSS 9.0 for Windows (SPSS Inc, Chicago, Ill).

TABLE 1—Risk Factors and Pregnancy Outcomes, by Race/Ethnicity: New York, NY, 1999–2001

	Non-Hispanic Blacks (n = 86 908)	Non-Hispanic Whites (n = 96 581)	Non-Hispanic Asians (n = 38 570)	Hispanics (n = 107 612)	Total (n = 329 988)
Mean age, y***	27.5	30.6	29.7	26.4	28.3
Prepregnancy weight, %***					
< 100 lb	1.7	1.8	8.1	2.9	2.9
100–149 lb	49.1	69.5	79.5	65.6	64.0
150–199 lb	37.5	24.0	11.6	26.2	26.8
200–299 lb	11.2	4.7	0.8	5.1	6.1
≥ 300 lb	0.5	0.1	0.0	0.1	0.2
Weight gain, %***					
< 41 lb	79.7	83.2	89.2	79.1	81.6
≥ 41 lb	20.3	16.8	10.8	20.9	18.4
Chronic diabetes, %***	0.4	0.3	0.4	0.3	0.3
Gestational diabetes, %***	3.7	2.6	6.6	3.5	3.7
Chronic hypertension, %***	1.7	0.6	0.5	0.7	0.9
Pregnancy hypertension, %***	1.9	1.2	0.7	1.4	1.4
Preeclampsia, %[a]***	2.9	1.3	1.2	2.6	2.1
Primary cesarean, %[b]***	16.2	14.7	14.4	13.8	14.7
Preterm birth, %***	10.5	5.1	5.9	7.8	7.5
Low birthweight, %***	9.7	4.1	5.7	6.1	6.4

[a]Women with eclampsia were excluded.
[b]Women with previous cesareans were excluded.
*P < .05; **P < .01; ***P < .001; for analysis of variance or χ^2 test comparing racial/ethnic groups.

RESULTS

Table 1 shows that the prevalence of all the key risk factors and pregnancy outcomes varied significantly by race/ethnicity (by ANOVA and χ^2 tests). White women and Asian women were the oldest, with mean ages of 30.6 and 29.7 years, respectively. Black women were significantly more likely to be in the 2 heaviest weight categories than in other subgroups: close to 12% of the Black women had a prepregnancy weight of 200 pounds or more compared with 5.2% of Hispanics, 4.8% of Whites, and less than 1% of Asians. Black women and Hispanic women were also more likely to have excess weight gain during pregnancy than women in the other 2 groups.

The prevalence of chronic diabetes ranged from 0.3% to 0.4% across all 4 groups. There was considerably more variation in the rates of gestational diabetes. Although 2.6% of White women developed gestational diabetes, 6.6% of the Asian women developed the same condition. Black women had the highest rates of both chronic hypertension (1.7%) and pregnancy-induced hypertension (1.9%). The remaining subgroups had less than 1% prevalence of chronic hypertension. Hispanics and Whites had the next highest percentages of pregnancy-induced hypertension at 1.4% and 1.2%, respectively.

The racial/ethnic differences in the prevalence of preeclampsia are important to understand as predictors of the 3 adverse outcomes: Blacks (2.9%) and Hispanics (2.6%) had much higher rates of preeclampsia than other women. Their rates were more than twice the rates of Whites and Asians. For the 3 outcome variables, Black women had the highest percentages of primary cesareans (16.2%), preterm births (10.5%), and LBW (9.7%).

Preeclampsia by Racial/Ethnic Group

Table 2 shows that in multivariate logistic regressions predicting preeclampsia for each of the racial/ethnic groups and for the total, as well as for prepregnancy weight, weight gain, diabetes, and hypertension, all had significant adjusted odds ratios (AORs). (Full results from this analysis and all others discussed in the article are available from the authors.) Obesity contributed the most to preeclampsia for White women (AOR = 2.67 and AOR = 3.36 for the 2 highest weight groups) compared with other women, whereas excess weight gain was a more important predictor of preeclampsia for Asians (AOR = 1.96) than for other women. The risk for preeclampsia increased as weight increased for women of all racial/ethnic subgroups and was highest for the heaviest group. Chronic diabetes among White women was a much stronger risk factor for preeclampsia (AOR = 5.31) than it was for other women. And, finally, for Asian women with either chronic hypertension or pregnancy-induced hypertension (AOR = 17.58 and AOR = 18.11, respectively), these factors conferred more serious risks than for other women. In brief, for all racial/ethnic groups, either chronic or pregnancy-induced hypertension was the strongest predictor of preeclampsia.

Models for 3 Adverse Outcomes in the Total Population

Table 3 provides data from models predicting 3 adverse outcomes in the total population before considering the race-stratified models. This table provides AORs and confidence intervals (CIs) for the most important risk factors in the analysis, the weight variables, the diabetes variables, and the hypertension variables.

With the exception of the lowest weight group, all the other risk factors significantly increased the likelihood of a primary cesarean delivery. The heaviest women were the most at risk for a primary cesarean (AOR = 2.59). Women with chronic diabetes and women with preeclampsia were also more than twice as likely to have a primary cesarean as were women without these conditions (AOR = 2.37 and AOR = 2.50, respectively).

For the total population, preeclampsia presented the greatest risk of a preterm birth (AOR = 5.07). Chronic diabetes and chronic hypertension were also significantly and positively related to the risk of a preterm birth (AOR = 2.54 and AOR = 2.34). In comparison with the other adverse outcomes, being underweight increased the likelihood of a preterm birth, whereas being in the heavier

TABLE 2—Association of Preeclampsia with Prepregnancy Weight, Prenatal Weight Gain, Diabetes, and Hypertension by Race/Ethnicity[a]

	Non-Hispanic Blacks (n = 86 908), AOR (95% CI)	Non-Hispanic Whites (n = 96 581), AOR (95% CI)	Non-Hispanic Asians (n = 38 570), AOR (95% CI)	Hispanics (n = 107 612), AOR (95% CI)	Total (n = 329 988), AOR (95% CI)
Prepregnancy weight, lb					
< 100	0.64 (0.41, 0.99)*	0.57 (0.29, 1.11)	0.63 (0.40, 1.00)*	0.90 (0.71, 1.14)	0.71 (0.59, 0.85)***
100–149	Reference	Reference	Reference	Reference	Reference
150–199	1.42 (1.29, 1.56)***	1.69 (1.49, 1.92)***	1.58 (1.22, 2.04)***	1.23 (1.12, 1.34)***	1.47 (1.39, 1.55)***
200–299	1.76 (1.56, 2.00)***	2.67 (2.20, 3.24)***	2.71 (1.40, 5.25)**	1.59 (1.37, 1.86)***	1.98 (1.82, 2.15)***
≥ 300[b]	2.66 (1.80, 3.92)***	3.36 (1.39, 8.14)**	. . .	2.02 (1.01, 4.03)*	2.78 (2.03, 3.81)***
Weight gain, lb					
< 41	Reference	Reference	Reference	Reference	Reference
≥ 41	1.44 (1.31, 1.58)***	1.65 (1.45, 1.88)***	1.96 (1.53, 2.50)***	1.47 (1.35, 1.60)***	1.54 (1.46, 1.63)***
Chronic diabetes	2.46 (1.71, 3.55)***	5.31 (3.36, 8.39)***	1.22 (0.45, 3.28)	2.58 (1.74, 3.82)***	2.77 (2.22, 3.47)***
Gestational diabetes	1.40 (1.18, 1.66)***	1.15 (0.86, 1.53)	1.00 (0.71, 1.41)	1.67 (1.42, 1.97)***	1.41 (1.27, 1.56)***
Chronic hypertension	8.11 (6.96, 9.46)***	8.82 (6.70, 11.60)***	17.58 (11.70, 26.42)***	8.57 (7.00, 10.49)***	8.97 (8.06, 9.99)***
Pregnancy hypertension	11.70 (10.29, 13.32)***	11.51 (9.58, 13.83)***	18.11 (12.60, 26.02)***	6.66 (5.74, 7.74)***	10.18 (9.37, 11.07)***

Note. AOR = adjusted odds ratio; CI = confidence interval.
[a]Women with eclampsia were excluded. Logistic regressions adjusted for maternal age (< 20, 20–34, > 34 years), marital status, mother's education, mother's birthplace, prenatal care payer, social risk (smoking, drinking, or substance abuse), parity, and trimester prenatal care began.
[b]For Asians this group was merged with the 200-to-299 group because of small numbers (3 individuals).
*P < .05; **P < .01; ***P < .001.

TABLE 3—Associations Between Prepregnancy Weight, Prenatal Weight Gain, Diabetes, Hypertension, Preeclampsia, and 3 Adverse Pregnancy Outcomes for All Women (n = 329 988)[a]

	Primary Cesarean, AOR (95% CI)	Preterm Birth, AOR (95% CI)	Low Birthweight, AOR (95% CI)
Prepregnancy weight, lb			
< 100	0.96 (0.91, 1.03)	1.23 (1.14, 1.32)***	1.85 (1.73, 1.98)***
100–149	Reference	Reference	Reference
150–199	1.32 (1.29, 1.36)***	1.02 (0.99, 1.05)	0.86 (0.83, 0.89)***
200–299	1.89 (1.81, 1.97)***	0.97 (0.91, 1.02)	0.77 (0.73, 0.82)***
≥ 300[b]	2.59 (2.13, 3.15)***	0.88 (0.67, 1.15)	0.66 (0.48, 0.90)**
Weight gain, lb			
< 41	Reference	Reference	Reference
≥ 41	1.38 (1.34, 1.41)***	0.54 (0.52, 0.57)***	0.41 (0.39, 0.43)***
Chronic diabetes	2.37 (2.05, 2.75)***	2.54 (2.18, 2.95)***	1.59 (1.32, 1.91)***
Gestational diabetes	1.47 (1.40, 1.55)***	1.28 (1.20, 1.36)***	0.88 (0.82, 0.96)**
Chronic hypertension	1.57 (1.43, 1.73)***	2.34 (2.12, 2.57)***	2.68 (2.42, 2.96)***
Pregnancy hypertension	1.35 (1.25, 1.46)***	1.82 (1.67, 1.98)***	2.06 (1.89, 2.25)***
Preeclampsia[c]	2.50 (2.36, 2.65)***	5.07 (4.79, 5.36)***	5.94 (5.61, 6.30)***

Note. AOR = adjusted odds ratio; CI = confidence interval.
[a]Logistic regression adjusted for maternal age (< 20, 20–34, > 34 years), marital status, mother's education, mother's birthplace, prenatal care payer, social risk, parity, and trimester prenatal care began.
[b]For Asians this group was merged with the 200-to-299 group because of small numbers (3 individuals).
[c]Women with eclampsia were excluded.
*P < .05; **P < .01; ***P < .001.

weight categories was not significantly related to this adverse outcome. Excess weight gain, in fact, protected against a preterm birth (AOR = 0.54).

Finally, all the major covariates were significantly related to LBW, although in differing directions. The hypertensive variables were the strongest contributors to an increased likelihood of an LBW infant. For chronic hypertension, pregnancy-induced hypertension, and preeclampsia, the increased risk was 2 to 5 times what it was in women without these conditions (AOR = 2.68, AOR = 2.06, and AOR = 5.94, respectively).

The negative associations of prepregnancy overweight, obesity, and excessive prenatal weight gain in the preterm and LBW regressions need to be considered in light of their positive association with macrosomia. Obese women were at an increased risk for a macrosomic infant, defined here as one weighing 4000 g or more. In our population, although 6.6% of women with a prepregnancy weight of 100 to 149 lb had a macrosomic infant, 16.4% of those in the 200- to 299-lb group, and 20.6% of those in the 300-lb-and-over group had a macrosomic infant.

TABLE 4—Summary of Associations Between Chronic and Gestational Diabetes and 3 Adverse Pregnancy Outcomes by Race/Ethnicity[a]

	Non-Hispanic Blacks (n = 86 908), AOR (95% CI)	Non-Hispanic Whites (n = 96 581), AOR (95% CI)	Non-Hispanic Asians (n = 38 570), AOR (95% CI)	Hispanics (n = 107 612), AOR (95% CI)	Total (n = 329 988), AOR (95% CI)
Primary cesarean					
Chronic diabetes	2.04 (1.66, 2.52)***	2.22 (1.61, 3.07)***	2.00 (1.32, 3.02)***	2.86 (2.17, 3.76)***	2.37 (2.05, 2.75)***
Gestational diabetes	1.55 (1.44, 1.68)***	1.23 (1.09, 1.38)***	1.52 (1.35, 1.70)***	1.54 (1.40, 1.69)***	1.47 (1.40, 1.55)***
Preterm birth					
Chronic diabetes	1.99 (1.54, 2.57)***	2.46 (1.72, 3.53)***	2.23 (1.40, 3.53)***	3.43 (2.68, 4.42)***	2.54 (2.18, 2.95)***
Gestational diabetes	1.31 (1.17, 1.46)***	1.20 (1.02, 1.41)*	1.34 (1.15, 1.56)***	1.25 (1.12, 1.40)***	1.28 (1.20, 1.36)***
Low birthweight					
Chronic diabetes	1.33 (0.99, 1.79)	1.59 (1.01, 2.50)*	2.28 (1.42, 3.68)***	1.69 (1.21, 2.35)**	1.59 (1.32, 1.91)***
Gestational diabetes	0.81 (0.70, 0.92)**	1.06 (0.87, 1.28)	1.17 (0.99, 1.39)	.71 (.61, .84)***	.88 (.82, .96)**

Note. AOR = adjusted odds ratio; CI = confidence interval.
[a]Logistic regressions adjusted for maternal age (< 20, 20–34, > 34 years), marital status, mother's education, mother's birthplace, prenatal care payer, social risk, parity, time trimester prenatal care began, as well as prepregnancy weight, prenatal weight gain, chronic hypertension, pregnancy-induced hypertension, and preeclampsia.
*P < .05; **P < .01; ***P < .001.

Significant differences in rates of macrosomia also were found by racial/ethnic subgroup: 10.5% for non-Hispanic Whites, 8.9% for Hispanics, 7.2% for non-Hispanic Blacks, and 5.5% for Asians (P<.001).

Primary Cesarean by Racial/Ethnic Group

Summary results from the logistic regressions predicting a primary cesarean by race/ethnicity appear in Table 4. For all the groups, chronic diabetes and gestational diabetes were significant risk factors for a primary cesarean. The level of risk varied, however, among racial/ethnic groups. Chronic diabetes appears to be a more serious risk for Hispanic women (AOR=2.86) than for other women, whereas gestational diabetes posed a similar risk across racial/ethnic subgroups (adjusted odds ratios ranged from 1.23 to 1.55).

In the full model (not shown here), chronic hypertension, pregnancy-induced hypertension, and preeclampsia together appeared to pose greater risks of a primary cesarean for Blacks than for other women.

Preterm Birth by Racial/Ethnic Group

Table 4 also presents summary results for the regressions predicting preterm birth. The diabetic conditions significantly increased the risk of a preterm birth for women in all 4 subgroups. The chronic form of diabetes appeared to pose greater risks for all women than the pregnancy-induced disorder, with the level of risk varying across the 4 groups. Although the increased risk of a preterm birth for Black women with chronic diabetes was 1.99, the increased risk for Hispanic women with the same condition was 3.43.

In the full model (not shown), preeclampsia presented the greatest increased risk of a preterm birth for Whites compared with other women.

LBW by Racial/Ethnic Group

A summary of the results of the logistic regressions predicting LBW (2500 g or less) is also presented in Table 4. Chronic diabetes showed more consistent increased risks for an LBW birth than the gestational condition. This condition posed the greatest increase in risk for Asian women (AOR=2.28). On the other hand, for 2 of the groups, Whites and Asians, gestational diabetes was not even a significant predictor of LBW. And for Blacks and Hispanics, gestational diabetes even appeared to lessen the risk of an LBW infant (AOR=0.81 and AOR=0.71).

As with the lower risk of LBW related to obesity and excess weight gain, the pattern found in Table 4 for gestational diabetes and LBW may be related to the positive association diabetes has with macrosomia.

DISCUSSION

Chronic diabetes and gestational diabetes were both significant, independent risk factors for a primary cesarean delivery and for preterm birth in all the racial/ethnic groups studied. Interestingly, the increased risk for an LBW birth conferred by either form of diabetes varied considerably among the 4 racial/ethnic groups studied, a finding that has not been clear in prior research. Likewise, although both weight measures—overweight or obesity and excess weight gain—were significant predictors of primary cesarean delivery for all racial/ethnic groups, each measure's effect on preterm birth and LBW varied by subgroup. Our results are similar to those of other researchers who found that maternal obesity and weight gain were independent risk factors for some adverse pregnancy outcomes (including cesarean delivery) beyond the risks conferred by pregestational and gestational diabetes.[8,33]

Our analyses suggest that diabetes and excess weight can adversely affect pregnancy and birth outcomes through 2 different causal paths and that the interplay of these risk factors may operate differently within each racial/ethnic group. One path through which diabetes and excess weight affect pregnancy and birth outcomes is through their contribution to the development of preeclampsia, which can trigger the preterm delivery of an LBW baby, often by cesarean delivery.[22] The second path is through the increased risk of a macrosomic infant. Pregnancy with a macrosomic infant often leads to labor dystocia, which can lead to an elevated cesarean delivery rate. To avoid a traumatic birth and possible injury, physicians are more likely to deliver a baby by cesarean delivery at or near term if a macrosomic infant is suspected. Evidence of these 2 alternative paths outside the United States was found in a recent Chinese study in which women with impaired glucose tolerance were at higher risk for both preterm births and macrosomia.[11] As further evidence of these divergent paths, the highest cesarean rates in our population were found for babies weighing 500 to 1999 g and for those weighing 4000 g and greater.

Overweight and obese mothers, and those with excessive weight gain, have higher risks for preeclampsia, which in turn increases the risk for both preterm birth and LBW. However, there were many obese women for whom there was little or no overlap with other conditions, and thus they were more likely to be at an increased risk for a macrosomic infant. This second profile underlies the seemingly "protective" effect of overweight, obesity, and excessive weight gain in the logistic regressions (see the results for all women in Table 3).

Unfortunately, the vital statistics files used for this study have limitations, including uneven and invalid reporting. Although prior research has found that maternal reports of prepregnancy weight are consistent with clinical record data,[34] analysis of birth certificate data in New York State (excluding New York City) found that the accuracy of birth certificate data for other variables is mixed.[35] Although the sensitivities for method of delivery and birthweight were 98% and 100%, respectively, the sensitivities for chronic hypertension and preexisting diabetes were 0% and 50%, respectively, suggesting that caution should be used. Sensitivity was higher for medical risk in pregnancy, including gestational diabetes (83%), pregnancy-related hypertension (72%), and preeclampsia (62%).[35] A similar study of low-risk pregnancies in Washington State found very low sensitivity for chronic hypertension (7.3%) but higher sensitivity for diabetes (52%).[36]

The birth certificate data are limited to live births, so we could not evaluate the association between our independent risk factors and miscarriage or stillbirth. Further, although the postoperative complications of cesarean delivery, including infection, operative injury, or extended hospital confinement, are related to the mother's obesity, we could not follow these adverse outcomes after a birth.[37–42] Finally, although preterm and LBW babies are at greater risk for morbidity and mortality early in their lives, again we could not measure these outcomes with the birth file.

Our analysis confirms findings from the National Health and Nutrition Examination Survey indicating that non-Hispanic Black women in their childbearing years are significantly more likely to be obese than are other women.[27] It also parallels the findings from another study in a large city that showed that obesity was most common among Black women and that among these women it increased the most over time.[43] Black women in our population were much more likely to be overweight or obese than were women in any other racial/ethnic group. Also, more than 1 in 5 of the Black women in our New York City population gained excess weight during pregnancy. Another study of Black women in New York City found that those who were overweight or obese before pregnancy had the highest weight gain and retained on average 20 pounds postpartum.[44] Numerous studies have shown that weight gain during pregnancy puts a woman at greater risk of excess weight before her next pregnancy and throughout her lifetime.[45–49]

Our finding that Asian women compared with others have a relatively high rate of chronic diabetes (0.4%) and a very high rate of gestational diabetes (6.6%) is consistent with a number of other studies performed in the United States and abroad.[7,50–52] Because Asians were less likely to be overweight or obese before pregnancy, and less likely to have excessive weight gain during pregnancy, early diabetes diagnosis and careful metabolic control for diabetic Asian mothers may be more important than weight control as preventive measures to improve their birth outcomes.

We recommend that although careful monitoring of diabetes during pregnancy can improve pregnancy outcomes for diabetic women, the longer-term public health approach should be to prevent type 2 diabetes and gestational diabetes by controlling women's weight over their lifetimes. Pregnancy and the postpartum period can be seen as a window of opportunity, or a "teachable moment,"[53] during which women are more open to counseling about the risks of being overweight or obese and more likely to make behavioral changes that will improve their health for many years to come. As recommended by other researchers, we suggest that lifestyle changes in nutrition and exercise be promoted by all primary care providers, including obstetricians, to avoid overweight and obesity during the childbearing years.[37,38,44,54] For women who have gestational diabetes, this strategy may be particularly crucial after delivery in order to prevent the development of type 2 diabetes later in life.[46] Counseling for women with diabetes mellitus or obesity is also critical to minimize the risk to the fetus in the next pregnancy. ■

About the Authors

Terry J. Rosenberg, Samantha Garbers, and Mary Ann Chiasson are affiliated with the Medical Health and Research Association (MHRA) of New York City, Inc, New York, NY. Heather Lipkind is with the Columbia University College of Physicians and Surgeons, Department of Obstetrics and Gynecology, New York, NY.

Requests for reprints should be sent to Terry Rosenberg, PhD, MHRA, 40 Worth St, Suite 720, New York, NY 10013 (e-mail: trosenberg@mhra.org).

This article was accepted April 21, 2005.

Contributors

T.J. Rosenberg originated the study, carried out the analysis, wrote much of the article, and completed the revisions. S. Garbers made suggestions for the analysis, wrote parts of the introduction, and contributed to the overall revisions. H. Lipkind provided detailed advice on the medical issues discussed in the article and made many suggestions for revisions. M.A. Chiasson was an adviser throughout the process and had considerable input into the revisions.

Acknowledgments

The authors are grateful to Claudette McKenzie of MHRA for designing, producing, and revising the tables.

Human Participant Protection

The study was exempted from review by the MHRA institutional review board because the analysis used data completely stripped of personal identifiers.

References

1. *Behavioral Risk Factor Surveillance System Online Prevalence Data.* Atlanta, Ga: Centers for Disease Control and Prevention; 2005.

2. Boyle JP, Honeycutt AA, Venkat Narayan KM, et al. Projection of diabetes burden through 2050 impact of changing demography and disease prevalence in the US. *Diabetes Care.* 2001;24:1936–1940.

3. *National Diabetes Fact Sheet: General Information and National Estimates on Diabetes in the United States, 2003.* Rev ed. Atlanta, Ga: Centers for Disease Control and Prevention; 2004.

4. Cunningham FG, Gant NF, Leveno KJ, et al. Diabetes. In: Cunningham FG, Gant NF, Leveno KJ, et al., eds. *Williams Obstetrics.* 21st ed. New York, NY: McGraw-Hill; 2001:1359–1381.

5. Martin JA, Hamilton BE, Sutton PD, et al. *Births: Final Data for 2002.* Hyattsville, Md: National Center for Health Statistics; 2003.

6. Beckles GLA, Thompson-Reid PE, eds. *Diabetes and Women's Health Across the Life Stages: A Public Health Perspective.* Atlanta, Ga: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Division of Diabetes Translation; 2001.

7. El Mallah KO, Narchi H, Kulaylat NA, et al. Gestational and pre-gestational diabetes: comparison of maternal and fetal characteristics and outcome. *Int J Gynaecol Obstet.* 1997;58:203–209.

8. Ray JG, Vermeulen MJ, Shapiro JL, Kenshole AB. Maternal and neonatal outcomes in pregestational and gestational diabetes mellitus, and the influence of maternal obesity and weight gain: the deposit study. *Q J Med.* 2001;94:347–356.

9. Moore LL, Singer MR, Loring Bradlee M, et al. A prospective study of the risk of congenital defects associated with maternal obesity and diabetes mellitus. *Epidemiology.* 2000;11:689–694.

10. Hedderson MM, Ferrara A, Sacks DA. Gestational diabetes mellitus and lesser degrees of pregnancy hyperglycemia: association with increased risk of spontaneous preterm birth. *Obstet Gynecol.* 2003;102: 850–856.

11. Yang X, Hsu-Hage B, Zhang H, et al. Women with impaired glucose tolerance during pregnancy have significantly poor pregnancy outcomes. *Diabetes Care.* 2002;25:1619–1624.

12. Yang X, Hsu-Hage B, Zhang H, et al. Gestational diabetes mellitus in women of single gravidity in Tianjin City, China. *Diabetes Care.* 2002;25:847–851.

13. Kieffer EC, Alexander GR, Kogan MD, et al. Influence of diabetes during pregnancy on gestational age-specific newborn weight among US black and US white infants. *Am J Epidemiol.* 1998;147:1053–1061.

14. Moore TR. Fetal growth in diabetic pregnancy. *Clin Obstet Gynecol.* 1997;40:771–786.

15. Casey BM, Lucas MJ, McIntire DD, et al. Pregnancy outcomes in women with gestational diabetes compared with the general obstetric population. *Obstet Gynecol.* 1997;90:869–873.

16. Catalano PM, Kirwan JP, Haugel-de Mouzon S, et al. Gestational diabetes and insulin resistance: role in short- and long-term implications for mother and fetus. *J Nutr.* 2003;133:1674S–1683S.

17. Gillman MW, Rifas-Shiman S, Berkey CS, et al. Maternal gestational diabetes, birth weight, and adolescent obesity. *Pediatrics.* 2003;111:e221–e226.

18. Sibai BM, Caritis S, Hauth J, et al. Risks of preeclampsia and adverse neonatal outcomes among women with pregestational diabetes mellitus. *Am J Obstet Gynecol.* 2000;182:364–369.

19. Rosenberg TJ, Garbers S, Chavkin W, et al. Prepregnancy weight and adverse perinatal outcomes in an ethnically diverse population. *Obstet Gynecol.* 2003;102:1022–1027.

20. Linne Y. Effects of obesity on women's reproduction and complications during pregnancy. *Obes Rev.* 2004;5:137–143.

21. Weiss JL, Malone FD, Emig D, et al. Obesity, obstetric complication and cesarean delivery rate—a population based screening study. *Am J Obstet Gynecol.* 2004;190:1091–1097.

22. Cunningham FG, Gant NF, Leveno KJ, et al. Hypertensive disorders in pregnancy. In: Cunningham FG, Gant NF, Leveno KJ, et al., eds. *Williams Obstetrics.* 21st ed. New York, NY: McGraw-Hill; 2001:567–618.

23. Cnattingius S, Bergstrom R, Lipworth L, et al. Prepregnancy weight and the risk of adverse pregnancy outcomes. *N Engl J Med.* 1998;338:147–152.

24. Metzger BE, Coustan DR, the Organizing Committee. Summary and recommendations of the Fourth International Workshop-Conference on Gestational Diabetes Mellitus. *Diabetes Care.* 1998;21(suppl 2): B161–B167.

25. Naylor CD, Sermer M, Chen E, et al. Selective screening for gestational diabetes mellitus. *N Engl J Med.* 1997;337:1591–1596.

26. Solomon CG, Willet WC, Carey VJ, et al. A prospective study of pregravid determinants of gestational diabetes mellitus. *JAMA.* 1997;278:1078–1083.

27. Flegal KM, Carroll MD, Ogden CL, et al. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA.* 2002;288:1723–1727.

28. Saldana TM, Siega-Riz AM, Adair LS, et al. The association between impaired glucose tolerance and birth weight among black and white women in central North Carolina. *Diabetes Care.* 2003;26:656–661.

29. Steinfeld JD, Valentine S, Lerer T, et al. Obesity-related complications of pregnancy vary by race. *J Maternal Fetal Med.* 2000;9:238–241.

30. Kieffer EC, Nolan GH, Carman WJ, et al. Glucose tolerance during pregnancy and birth weight in a Hispanic population. *Obstet Gynecol.* 1999;94:741–746.

31. Scholl TO, Chen X, Gaughan C, et al. Influence of maternal glucose level on ethnic differences in birth weight and pregnancy outcome. *Am J Epidemiol.* 2002;156:498–506.

32. Institute of Medicine, Subcommittee on Nutritional Status and Weight Gain During Pregnancy. *Nutrition During Pregnancy. Part I: Weight Gain. Part II: Nutrient Supplements.* Washington, DC: National Academy Press; 1990.

33. Bo S, Signorile A, Bardelli C, et al. Obesity or diabetes: what is worse for the mother and for the baby? *Diabetes Metab.* 2003;29:175–178.

34. Lederman SA, Paxton A. Maternal reporting of prepregnancy weight and birth outcome: consistency and completeness compared with the clinical record. *Maternal Child Health J.* 1998;2:123–126.

35. Roohan PJ, Josberger RE, Acar J, et al. Validation of birth certificate data in New York State. *J Community Health.* 2003;28:335–346.

36. Dobie SA, Baldwin LM, Rosenblatt RA, et al. How well do birth certificates describe the pregnancies they report? The Washington State experience with low-risk pregnancies. *Maternal Child Health J.* 1998;2:145–154.

37. Baeten MJ, Bukusi EA, Lambe M. Pregnancy complications and outcomes among overweight and obese nulliparous women. *Am J Public Health.* 2001; 91:436–440.

38. Brost BC, Goldenberg RL, Mercer BM, et al. The preterm prediction study: association of cesarean delivery with increases in maternal weight and body mass index. *Am J Obstet Gynecol.* 1997;177:333–341.

39. Crane SS, Wojtowycz MA, Dye TD, et al. Association between prepregnancy obesity and the risk of cesarean delivery. *Obstet Gynecol.* 1997;89:213–216.

40. Kaiser PS, Kirby RS. Obesity as a risk factor for cesarean in a low-risk population. *Obstet Gynecol.* 2001;97:39–43.

41. Bianco AT, Smilen SW, Davis Y, et al. Pregnancy outcome and weight gain recommendations for the morbidly obese woman. *Obstet Gynecol.* 1998; 91:97–102.

42. Isaacs JD, Magann ED, Martin RW, et al. Obstetric challenges of massive obesity complicating pregnancy. *J Perinatol.* 1994;14:10–14.

43. Ehrenberg HM, Dierker L, Milluzzi C, et al. Prevalence of maternal obesity in an urban center. *Am J Obstet Gynecol.* 2002;187:1189–1193.

44. Lederman SA, Alfasi G, Deckelbaum RJ. Pregnancy-associated obesity in black women in New York City. *Maternal Child Health J.* 2002;6:37–42.

45. Rooney BL, Schauberger CW. Excess pregnancy weight gain and long-term obesity: one decade later. *Obstet Gynecol.* 2002;100:245–252.

46. Linne Y, Barkeling B, Rossner S. Natural course of gestational diabetes mellitus: long term follow up of women in the spawn study. *BJOG.* 2002;109: 1227–1231.

47. Linne Y, Rossner S. Interrelationships between weight development and weight retention in subsequent pregnancies: the SPAWN study. *Acta Obstet Gynecol Scand.* 2003;82:318–325.

48. Linne Y, Dye L, Barkeling B, et al. Weight development over time in parous women: the SPAWN study—15 years follow-up. *Int J Obes.* 2003;27:1516–1522.

49. Linne Y, Barkeling B, Rossner S. Long-term weight development after pregnancy. *Obes Rev.* 2002;3:75–83.

50. Berkowitz GS, Lapinski RH, Wein R, et al. Race/ethnicity and other factors for gestational diabetes. *Am J Epidemiol.* 1992;135:965–973.

51. Ferrara A, Hedderson MM, Quesenberry CP, et al. Prevalence of gestational diabetes mellitus detected by the national diabetes data group or the Carpenter and Coustan plasma glucose thresholds. *Diabetes Care.* 2002;25:1625–1630.

52. Vangen S, Stoltenberg C, Holan S, et al. Outcome of pregnancy among immigrant women with diabetes. *Diabetes Care.* 2003;26:327–332.

53. McBride CM, Emmons KM, Lipkus IM. Understanding the potential of teachable moments: the case of smoking cessation. *Health Educ Res.* 2003;18: 156–170.

54. Serdula MK, Khan LK, Dietz WH. Weight loss counseling revisited. *JAMA.* 2003;289:1747–1750.