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Patterns of gestational weight gain in early pregnancy and risk of gestational diabetes mellitus

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Abstract

Background—Despite a call to study the effect of weight gain pattern on development of gestational diabetes mellitus, few studies have correctly adjusted for independent effects of gain after the first trimester. We used a conditional percentile approach to model the independent association between first and second trimester weight-gain trajectories and development of gestational diabetes.

Methods—We sampled women delivering singleton infants from 1998–2010 at Magee-Womens Hospital in Pittsburgh, PA (n=124,590) using a case-cohort design. We modeled weight-gain trajectories in the first and second trimesters of pregnancy using conditional weight-gain percentiles, and used multivariable logistic regression to assess independent associations of the trajectory with gestational diabetes. We studied associations separately by pre-pregnancy body mass index category.

Results—The final cohort included 806 women with gestational diabetes and 4,819 randomly sampled women who delivered without gestational diabetes. In normal-weight women, every standard deviation increase in weight gain in the first trimester above her predicted gain was associated with a 23% increased odds of gestational diabetes [95% CI: 0.2%, 51%]. Second trimester gain trajectory was not associated with gestational diabetes (OR: 1.1, [95% CI: 0.9, 1.3]) although the direction of effect was positive. This pattern was similar in obese class I and II but not in overweight and obese class III women.

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Computing code is available on request from the corresponding author. Data for replication can be obtained from the corresponding author conditional on appropriate institutional and ethics approval from the Magee-Womens Hospital, Pittsburgh PA.

Conclusions—An upward weight gain trajectory in the first trimester was positively associated with gestational diabetes for women of most pre-pregnancy BMI categories. Second trimester weight gain trajectory was not associated with gestational diabetes for any group.

Keywords

Gestational diabetes mellitus; gestational weight gain; conditional percentiles; pregnancy

Introduction

Gestational diabetes complicates 5%–6% of all pregnancies.¹ Pre-pregnancy obesity is a widely-known risk factor for gestational diabetes,^{2–9} but the role of weight gain during pregnancy is less well understood. In their 2009 recommendations, the National Academies of Sciences/Institute of Medicine (IOM) Committee to Reexamine Pregnancy Weight Guidelines concluded that there was insufficient evidence on the role of gestational weight gain in the development of gestational diabetes.¹⁰ The Committee was concerned that existing studies had analyzed the amount of weight gained during the entire period of pregnancy rather than up until time of diagnosis of gestational diabetes.^{2,11,12} Since treatment for the condition may alter the pattern of weight gain after diagnosis, associations between total pregnancy weight gain and gestational diabetes may reflect reverse causation. In the limited number of studies that have assessed total weight gain before diagnosis of gestational diabetes since the guidelines were published, the findings have been inconsistent, potentially due to differences in the way weight gain was measured and small sample sizes after stratification by body mass index (BMI) category.^{3,5,6,13–18}

In addition to the absolute amount of weight gained, the IOM Committee called for more research into the effect of weight gain *pattern* on adverse obstetric outcomes.¹⁰ To date, studies that have examined the differential effect of weight gain in the first and second trimesters have typically measured gain via the absolute kilograms gained or rate of gain.^{5,6,16,18} However, isolating critical windows in pregnancy for the effect of weight gain must be done more carefully because gain in any one period is correlated with future and total gain. That is, a woman with high weight gain in the second trimester may be at a lower risk for gestational diabetes because the second trimester is not a critical time for development of the condition or because she did not gain much in the first trimester and thus had a low total gain. Similarly, a higher rate of gain in the second trimester may not correlate with higher risk for gestational diabetes simply because the higher rate is compensating for a low rate of gain in the first trimester. To identify the independent effect of weight gain in the second trimester, a methodologically rigorous approach that isolates trajectory in this time window is needed.

Conditional percentiles have been used to examine growth trajectories in the context of pediatric and fetal growth^{19,20} and represent a methodologically rigorous approach by which to address this issue. Conditional percentiles are percentiles fit for an individual's weight measurement at a given point in time, given (conditional on) the individual's weight at a previous time point. In a study of gestational weight gain trajectories, the conditional percentile is a tool to assess the amount of deviation of weight gain in the second trimester

from that which was predicted based on gain since the end of the first trimester. As the percentiles are fit separately among women of the same early weight gain, second trimester gain can be expressed relatively independent of early gain. The purpose of this study was to use such an approach to assess the independent effects of first and, primarily, second trimester weight gain on the development of gestational diabetes in a large, hospital-based cohort.

Methods

The data for this study were collected as part of a larger case-cohort study on maternal weight gain conducted at the Magee-Womens Hospital in Pittsburgh, PA. The study population was drawn from 124,590 eligible women delivering singleton infants from 1998–2010 at Magee-Womens Hospital in Pittsburgh, PA. Detailed data on these deliveries, including maternal characteristics, antepartum events, and labor and delivery outcomes, are maintained in the electronic Magee Obstetrics, Medical, and Infant Database, which has been described in detail elsewhere.²¹

We first classified all eligible women into pre-pregnancy BMI (self-reported weight (kg)/measured height (m)²) categories of underweight (<18.5), normal-weight (18.5–24.9), overweight (25–29.9), obese class I (30.0–34.9), obese class II (35.0–39.9), and obese class III (≥40.0). Within each pre-pregnancy BMI category, we randomly sampled up to 290 women with gestational diabetes to form the case group (the final number was dependent on number of women with gestational diabetes available in each strata) as well as approximately 1,280 women to form a representative sub-cohort for comparisons. The sub-cohort was a representative sample of the study population and included both women with and without gestational diabetes. We then augmented information obtained from the perinatal database with data on serial weight gain measurements and glucose laboratory results via medical record abstraction. We used a case-cohort design because it provides nearly equal statistical efficiency to a cohort study while reducing chart abstraction costs; controls can be selected from the sub-cohort for multiple outcomes; and information from the randomly-selected sub-cohort can be used to estimate the distribution of weight gain and other exposures in the original cohort.²² The study received ethics board approvals by the University of British Columbia and the University of Pittsburgh.

For the present study, we included women who had a gestational diabetes screening test result from 20+0 to 32+0 weeks' gestation and two or more valid antenatal weight measurements before screening. We excluded women with incomplete or implausible data on gestational age or pre-pregnancy BMI, pre-existing diabetes mellitus, delivery before 24+0 weeks' gestation, and missing values for the adjustment variables.

Study measurements

At Magee-Womens Hospital, women are screened for gestational diabetes from 24 to 28 weeks of pregnancy. Screening is conducted via a 50-g glucose challenge test.^{23,24} In the case of an abnormal screening result (≥135 mg/dl), the 100-g glucose tolerance test is typically given to diagnose gestational diabetes. We defined gestational diabetes according to the Carpenter and Coustan criteria.²⁴ We estimated gestational age using the algorithm

endorsed by the American College of Obstetricians and Gynecologists.²⁵ Antenatal visits with improbable dates (e.g., prior to conception) were excluded.

Trained abstractors collected maternal weights and dates from each antenatal visit using an electronic data abstraction tool designed to ensure completion of all data elements and flag potential data entry errors.²⁶ Weight gain (kg) was defined as weight at each visit minus self-reported pre-pregnancy weight. Antenatal visits occur in this population every four weeks until 28 weeks', every two weeks until 36 weeks' and then weekly until delivery.

Implausible pre-pregnancy weights (defined as a weight $>+4$ or <-4 SD based on an internal standard) were replaced by the woman's first prenatal visit weight if a plausible weight <13 weeks was available. Implausible weight trajectories were identified using conditional z-scores, which assessed the plausibility of weight gain at the current visit based on weight gain at the previous visit.^{19,20,27} Weight gain values with conditional z-scores $>+4$ or <-4 SD were flagged and reviewed individually by the study authors (JAH, SCM, LMB).

In our primary analysis, we examined weight gain patterns during two time windows before gestational diabetes screening that we refer to as first- or second-trimester (See weight gain dates for a hypothetical patient in Figure 1a). First trimester weight gain trajectory was weight gain between conception and 13+6 weeks, and was calculated using self-reported pre-pregnancy weight and weight gain at a woman's last antenatal visit up to 13+6 weeks. Second-trimester weight gain trajectory was weight gain between the end of the first trimester and gestational diabetes screening. For the small number of women whose screening occurred after the end of the second trimester (but before 32 weeks), this measurement additionally included some early third trimester weight gain. This weight gain was calculated using the weight identified from the end of the first trimester and the weight measured at the antenatal visit closest to the screening date and no later than three weeks before or one week after the screening date. Screening tests with a missing or implausible date were assigned the median date of screening in the cohort.

In a secondary analysis, we further divided second trimester weight gain (Figure 1b). Early second trimester weight gain trajectory was defined as weight gain between the end of the first trimester and gain within 16+0 to 19+6 weeks. If there was more than one valid weight measurement within 16+0 to 19+6 weeks, the measurement closest to the midpoint of the window (18+0 weeks) was chosen. Late second trimester weight gain trajectory was defined as weight gain between the 16+0 to 19+6 visit and screening for gestational diabetes. Only women with a valid measurement at all three windows (first trimester, 16+0 to 19+6 weeks, and at screening) were included in the secondary analysis.

Statistical Analyses

Conditional Percentiles—We developed an internal reference of pregnancy weight gain trajectories by fitting BMI category-specific multi-level (random-effects) models describing the serial weight gain measurements of women in the sub-cohort as a function of gestational age up to 32 weeks. We specified random effects for the model intercept and slope to allow each woman's amount and rate of weight gain to vary from the population average. We assessed the potential for non-linearity by modeling gestational age using quadratic, cubic,

and fractional polynomials, and restricted cubic splines. Log-transformation of weight gain was investigated to account for potential heteroscedasticity. The final model was selected using the Akaike information criteria, the intraclass correlation coefficient, and visual inspection of the fixed and random residual plots.

The values in the internal reference charts were then used to calculate conditional weight gain z-scores, which summarize weight gain trajectories during different time windows. Conditional weight gain z-scores have been described elsewhere for the assessment of fetal and pediatric growth trajectories.^{19,20} The conditional z-scores reflect the extent to which a woman's current weight gain at a particular gestational age is above or below her expected weight gain for that gestational age, conditional on (given) her weight gain earlier in the pregnancy. A woman whose current weight gain was identical to the weight gain predicted based on her last visit would be assigned a conditional z-score of zero (analogous to being on the 50th percentile of an individualized growth chart). Thus, conditional z-scores above or below zero reflect weight gain greater or less than expected, respectively. Conditional z-scores provide a methodologically rigorous approach to summarize weight gain trajectories because they are able to correctly account for the correlation between a woman's serial weight gain measurements as well as the extent to which women's weight gain trajectories tend to "track" along the same percentiles throughout pregnancy.

As all women have a pregnancy weight gain of zero at conception, the conditional z-score for weight gain at the end of the first trimester will be similar to a population weight gain z-score for gestational age. For simplicity, we used this latter measurement to summarize first trimester gain. In the primary analysis, second trimester weight gain trajectory was expressed as a conditional z-score for weight gain at the time of gestational diabetes screening given (conditional on) weight at the end of the first trimester. In the secondary analysis, late second-trimester weight gain trajectory was expressed as a conditional z-score for weight gain at the time of screening given gain at 16+0 to 19+6 weeks, and early second trimester weight gain trajectory was based on conditional z-score for weight gain at 16+0 to 19+6 weeks given gain at the end of the first trimester. We also calculated an unconditional z-score (e.g., population weight-gain-for-gestational-age z-score) for each woman's total weight gain at the time of gestational diabetes screening.

Modeling—We used weighted logistic regression stratified by pre-pregnancy BMI category to determine the association between weight gain trajectories and gestational diabetes. We first conducted univariable analyses linking gestational diabetes with each of first trimester weight gain trajectory and second trimester weight gain trajectory (Univariable Models). Multivariable regression was then used to co-adjust for first and second trimester weight gain trajectory to assess the relative contributions of weight gain in each of the first and second trimesters (Multivariable Unadjusted). We further adjusted this model for confounders (Multivariable Adjusted). We additionally replicated the methods of previous studies by estimating the effect of total gain at the time of gestational diabetes screening. A clustered sandwich estimator was applied to each model to account for the case-cohort sampling design. Weights were calculated based on the number of sampled women out of the total number of women available to be sampled from each BMI category in the eligible cohort. Adjustment confounders included mother's age (continuous), parity (number of

liveborns: 0, 1–2, 3+), race (non-Hispanic White, non-Hispanic Black, other), within-category BMI (continuous), educational status (< high school, high school or equivalent, some college or associate's degree, college graduate), marital status (married/unmarried), pre-existing hypertension (yes/no), smoking status during pregnancy (yes/no), insurance (Medicaid/private), and year of delivery (continuous).

In a secondary analysis, we fit a multivariable model combining first trimester weight gain trajectory, early second trimester weight gain trajectory, and late second trimester weight gain trajectory to determine the specific contributions of weight gain at each window in gestation (Secondary Unadjusted). This model was further adjusted for the confounders listed above (Secondary Adjusted).

Analyses were conducted in Stata/SE 12.1 (College Station, TX).

Results

Descriptive Characteristics

We sampled 8,997 women (N=1,239 gestational diabetes cases; N=7,758 sub-cohort) for chart abstraction of serial weights (see Figure 2 for complete sampling information by BMI category). As this is an ongoing study, at the time of analysis most, but not all, of the desired number of pregnancies had been abstracted. After exclusions (Figure 3), the final cohort consisted of 5,625 women (N=806 gestational diabetes cases; N=4,819 sub-cohort). Among women in the sub-cohort, 238 were also members of the case group (5%). We could not perform multivariable analyses on underweight women because there were only 20 cases in this group. Descriptive characteristics of women included and excluded from the final cohort are presented in eTable 1. In general, excluded women were more likely to be black, less well educated, insured by Medicaid, non-married, and smokers.

As a result of oversampling women in the obese classes to improve sample size in those classes, the unweighted analysis does not represent the distribution of the descriptive characteristics in the general population of gestational diabetes cases and non-cases. To account for this, we present the characteristics as both unweighted and after applying the sampling weights to represent the distribution in the general population (Table). In the unweighted data, women in the gestational diabetes case series tended to be older than the sub-cohort, and were more likely to be white, married, nulliparous, college graduates, and hold private health care insurance. After applying sampling weights, many of these differences became less apparent. Major differences remaining in the weighted data were that cases were more likely than the sub-cohort to be overweight and obese and were more likely to have pre-existing hypertension. While absolute birth weights were similar, cases tended to be born slightly earlier than the sub-cohort. Indeed, after standardizing by reference gestational age and sex-specific birth weights in the United States,²⁸ infants born to cases had a higher mean z-score than infants born to the sub-cohort. Descriptive characteristics broken down by BMI category are available in eTable 2.

Weight gain trajectories

There was substantial overlap between weight gain trajectories for cases and non-cases (Figure 4). Comparison of multilevel models creating our internal pregnancy weight gain reference in each BMI category determined that a five-knot restricted cubic spline model offered the best fit. Log transformation of weight gain did not improve the model fit. The average weight gain z-scores by month of gestation are presented in eFigure 1. By the end of the first trimester, women with gestational diabetes tended to have slightly higher median weight gain z-scores than non-cases (eTable 3) (e.g. median z-scores in normal-weight women of 0.1 vs. -0.1, respectively), although overlap in interquartile ranges in all instances suggests that the difference was small. There were no meaningful differences in second trimester weight gain trajectory between cases and non-cases (e.g. median conditional z-scores for normal-weight: 0.02 vs. -0.01, respectively).

Primary Analysis

In normal weight women, the odds of gestational diabetes increased by 23% for every standard deviation increase in first trimester weight gain above predicted gain [95% CI: 0.2%, 51%]. For second trimester weight gain the effect was lower (OR: 1.1 [95% CI: 0.9, 1.3]) (Figure 5). Relationships were similar for women in obese classes I and II, however in overweight and obese class III women there was no effect of weight gain at any time of analysis. These patterns were nearly unchanged in the multivariable unadjusted and multivariable adjusted analyses. When examining total weight gain by the time of screening, the odds of gestational diabetes increased by 24% for every one standard deviation increase in total weight gain at screening for the condition [95% CI: 0.9%, 53%] in normal weight women. A similar pattern was evident for women of obese classes I and II.

Secondary Analysis

A total of 5,148 women (N=734 cases, N=4,414 sub-cohort) had a valid weight measurement between 16+0 to 19+6 weeks and so were included in the secondary analysis. After dividing second trimester weight gain trajectory into early and late, we continued to find an increasing association with first trimester weight gain, and little association with both early and late second trimester weight gain trajectory (eTable 4).

Discussion

Overall, we found that normal-weight, obese class I, and obese class II women whose weight gain in the first trimester was higher than predicted had an increased risk of gestational diabetes, but overweight and obese class III women did not. Weight gain trajectory in the second trimester was imprecise but in the positive direction for normal-weight and obese class II women.

A unique benefit of this study was our ability to assess associations separately by BMI categories with large numbers of cases. In contrast to our findings, three studies examining weight gain before screening and risk of gestational diabetes in normal-weight women have found no association.^{5,13,16} Given that the number of normal-weight women in our study is

more than double that in the previous studies, differences in power represent one possible explanation for the lack of association in earlier studies.

Total weight gain at the time of screening was not related to gestational diabetes in overweight women. Four studies have assessed total weight gain at screening and GDM in overweight women specifically.^{3,5,13,16} Of these, three^{3,5,16} reported an association of weight gain and glucose intolerance. However, differences in measures of both exposure (i.e. weight gain ratio,³ exceeding weight gain recommendations,¹³ rate of weight gain,⁵ absolute gain¹⁶) and outcome (i.e. impaired glucose tolerance,³ abnormal glucose tolerance,¹³ gestational diabetes^{5,16}) make comparisons challenging. While our study found no effect of *total* weight gain on risk of gestational diabetes in overweight women, we found that the pattern of first and second trimester gain was similar to that of normal weight and obese class I women, suggesting the mechanism of effect is likely similar. More studies with large sample sizes of overweight women are required to determine whether or not there is a true effect of gestational weight gain and gestational diabetes in this group.

Our study is the first to evaluate the risk of gestational diabetes when stratifying obesity according to three classes of severity. While obese women with gestational diabetes tended to be older and to have heavier babies given their gestational ages, in all three obese classes the relationships with race and cigarette smoking did not follow trends previously reported in the literature.⁴ This was unexpected and the reasons for it are unclear. In terms of total gain at screening, there was a positive association with gestational diabetes for women in obese classes I and II, but not class III women. Tovar and colleagues¹³ performed the only previous study to evaluate weight gain separately by obesity severity on the outcome abnormal glucose tolerance. The authors found that exceeding total weight gain recommendations before screening was not associated with abnormal glucose tolerance in obese class I women, but was a predictor in a combined obese class II/III category. Whether this effect was driven by women in obese class II, as in our findings, or obese class III is difficult to know. A lack of effect for the highly obese has some biologic plausibility. Saldana and colleagues³ have suggested that obese women may already be at a high insulin resistance level or have beta cells closer to a point of exhaustion at the start of their pregnancy, thus limiting the potential effect on glucose intolerance of additional weight gain during pregnancy. Our finding that the association between pregnancy weight gain and risk of gestational diabetes differed in obese class II versus class III women supports the need for studies to differentiate weight gain recommendations by severity of obesity.

The primary purpose of our study was to use conditional percentiles to assess the effects of second trimester weight gain trajectory independent of first trimester gain. While studies have suggested that second trimester weight gain does not increase the risk of gestational diabetes, they were not able to separate out the independent effects of gain in each period.^{5,14–16,18} The results of our study show that even after correcting for the dependency of second trimester gain with earlier weight gain, gain in this period is not a substantial predictor of gestational diabetes. As weight gain in early pregnancy is disproportionately composed of adipose tissue compared to that in later pregnancy,²⁹ one can hypothesize that the increasing fat mass early in pregnancy may play a more central role on glucose

intolerance than gain later. Future studies assessing the association between composition of weight gain and GDM are necessary to confirm this hypothesis.

While our study had a number of strengths including the use of conditional percentiles, large sample sizes, and stratification by obesity severity, there were some limitations. First, in order to assess weight gain at two primary time points during gestation, our study was necessarily restricted to women with at least two prenatal visits in the first and second trimester. Women without at least two prenatal visits before gestational diabetes screening were more likely to have characteristics indicative of lower socioeconomic status, be smokers, and non-white minorities than women with more prenatal visits. However, since we do not expect that the association between weight gain and gestational diabetes is different in women included and excluded from the study after controlling for these factors, this is not expected to cause substantial bias. Second, we did not have data to exclude women with a history of gestational diabetes. Since having the condition in one pregnancy increases the risk in the next pregnancy³⁰ and since women who have had gestational diabetes in the past may be more likely to modify their weight gain so as to prevent its occurrence, the possibility for unmeasured negative confounding cannot be excluded.⁶ However, this bias would likely underestimate associations. Third, we were not able to control for factors related to diet and lifestyle and so explanations related to these pathways could not be explored. Finally, the results of the current report demonstrate associations, but cannot determine causation. While high weight gain in the first trimester is a plausible causal factor for gestational diabetes, other explanations, such as a shared unmeasured common cause of weight gain and gestational diabetes, are possible. We attempted to minimize this possibility by controlling for a large number of known confounders and by assessing weight gain only before gestational diabetes screening (reducing potential for reverse causation).

With rising levels of obesity among women of childbearing age in the United States,³¹ gestational diabetes is likely to remain a prevalent disease of pregnancy and thus measures to reduce its occurrence are critical. Results of our study suggest that interventions to reduce excessive first-trimester weight gain should be evaluated for their ability to reduce risk of gestational diabetes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1. Timeline plot of a hypothetical patient's visit dates

Circles represent visit dates of a single hypothetical patient in the dataset. Black circles represent the chosen visit for the weight gain measurement in each interval. Grey circles represent other visits. Grey boxes represent the time interval for which a visit was eligible to be chosen for a weight measurement.

Figure 2.
Cohort sampling strategy. GDM indicates gestational diabetes mellitus.

Figure 3.
Cohort inclusions and exclusions. GDM indicates gestational diabetes mellitus.

Figure 4. Scatterplot of weight gain versus gestational age for the gestational diabetes mellitus cases (grey circles) and the non-cases (black circles)

Circles represent individual weight gain measurements across gestation. Gray circles are weight gain measurements in cases, and black circles are weight gain measurements in non-cases. Lines represent mean weight gain at each gestational age. The white solid line is mean weight gain for cases, and the black dashed line is mean weight gain for non-cases

Figure 5. Association between trimester-specific weight gain trajectories and gestational diabetes mellitus

Multivariable Adjusted model adjusted for mother's age, parity, race, within-category BMI, educational status, marital status, pre-existing hypertension, smoking status during pregnancy, insurance, and year of delivery. Odds ratios and 95% confidence intervals are presented for a one-standard deviation increase in weight gain trajectory. Black circles represent odds ratios of univariable analyses. Dark grey diamonds represent odds ratios of crude multivariable analyses. Light grey triangles represent odds ratios of adjusted multivariable analyses

Table 1

Descriptive Characteristics of Gestational Diabetes Cases and the Sub-cohort, Magee Womens Hospital 1995–2010.

	Unweighted		Weighted	
	Cases (N=806)	Sub-cohort (N=4,819)	Cases (N=3,373)	Sub-cohort (N=76,239)
Pre-Pregnancy BMI, N(%)				
Underweight	20 (2.5)	542 (11)	41 (1.2)	3,518 (4.6)
Normal-Weight	205 (25)	1,205 (25)	868 (26)	42,818 (56)
Overweight	177 (22)	837 (17)	898 (27)	17,369 (23)
Obese Class I	179 (22)	800 (17)	722 (21)	7,406 (10)
Obese Class II	129 (16)	761 (16)	461 (14)	3,183 (4.2)
Obese Class III	96 (12)	674 (14)	383 (11)	1,945 (2.6)
Mother's Age, year (SD)	31 (5.4)	29 (5.8)	31 (0.2)	30 (0.1)
Gestational age, week (SD)	38 (1.8)	39 (1.7)	38 (0.1)	39 (0.03)
Infant weight, grams (SD)	3,389 (581)	3,379 (554)	3,389 (21)	3,364 (10)
Infant weight z-score (SD) ^a	0.5 (1.1)	0.3 (1.1)	0.5 (0.04)	0.3 (0.02)
Year of Delivery, N(%)				
< 2000	66 (8.2)	437 (9.1)	280 (8.3)	8,758 (11)
2000 – 2004	281 (35)	1,804 (37.)	1,183 (35)	28,943 (38)
2005 – 2009	342 (42)	2,140 (44)	1,420 (42)	32,408 (43)
> 2010	117 (15)	438 (9.1)	490 (15)	6,130 (8)
Parity, N(%)				
0 liveborn	396 (49)	2,134 (44)	1,650 (49)	34,390 (45)
1–2 liveborns	353 (44)	2,352 (49)	1,483 (44)	37,342 (49)
3+ liveborns	57 (7.1)	333 (6.9)	240 (7.1)	4,507 (5.9)
Mother's Race, N(%)				
NH White	655 (81)	3,797 (79)	2,738 (81)	62,236 (82)
NH Black	96 (12)	836 (17)	404 (12)	10,341 (14)
Other	55 (6.8)	186 (3.9)	231 (6.9)	3,662 (4.8)
Mother's Education, N(%)				
< High School	28 (3.5)	298 (6.2)	115 (3.4)	4,309 (5.7)
High School / GED	168 (21)	1,093 (23)	698 (21)	15,245 (20)
Some College/ Assoc.	188 (23)	1,241 (26)	785 (23)	17,220 (23)
College Graduate	422 (52)	2,187 (45)	1,775 (53)	39,464 (52)
Insurance, N(%)				
Private	557 (69)	3,160 (66)	2,336 (69)	52,704 (69)
Medicaid	249 (31)	1,659 (34)	1,037 (31)	23,535 (31)
Married, N(%)	612 (76)	3,236 (67)	2,569 (76)	54,052 (71)
Pre-existing hypertension, N(%)	73 (9.1)	275 (5.7)	298 (8.8)	2,318 (3.0)
Smoker During Pregnancy, N(%)	67 (8.3)	595 (12)	276 (8.2)	8,252 (11)

^aStandardized by gestational age and sex-specific reference birth weights in the United States (Talge et al., 2014)

BMI = Body Mass Index, SD = Standard Deviation, NH =Non-Hispanic, GED = General Educational Development

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