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Fathers Count: the Impact of Paternal Risk Factors on Birth Outcomes

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Abstract

Objective—To determine the contribution of paternal factors to the risk of adverse birth outcomes.

Methods—This is a retrospective cross-sectional analysis using birth certificate data from 2004 to 2015 retrieved from the Finger Lakes Regional Perinatal Data System. Primiparous women with singleton pregnancies were analyzed in the study. Two multivariate logistic regression models were conducted to assess potential paternal risk factors including age, race/ethnicity, and education on four birth outcomes, including preterm birth (PTB), low birthweight (LBW), high birthweight (HBW), and small for gestational age (SGA).

Results—A total of 36,731 singleton births were included in the analysis. Less paternal education was significantly related to an elevated risk of PTB, LBW, and SGA, even after adjustment for maternal demographic, medical, and lifestyle factors ($P < 0.05$). Paternal race/ethnicity was also significantly associated with all four birth outcomes ($P < 0.05$) while controlling for maternal factors. Older paternal age was associated with increased odds (OR 1.012, 95% CI 1.003–1.022) of LBW. Maternal race/ethnicity partially mediated the association of paternal race/ethnicity with HBW and SGA. Maternal education partially mediated the relationship between paternal education and SGA.

Conclusion—Paternal factors were important predictors of adverse birth outcomes. Our results support the inclusion of fathers in future studies and clinical programs aimed at reducing adverse birth outcomes.

Keywords

Paternal education; Paternal race/ethnicity; Paternal age; Preterm birth; Low birthweight; High birthweight; Small for gestational age

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Conflict of Interest

The authors declare that they have no conflict of interest.

Introduction

Preterm birth (PTB), low birthweight (LBW) and small-for-gestational age (SGA) are the major causes of infant mortality (Katz et al., 2013; Matthews, MacDorman, & Thoma, 2015). High birthweight (HBW), though not linked to a risk of infant mortality, has been related to long-term health issues, such as obesity and cancers (O'Neill et al., 2015; Thurber, Dobbins, Kirk, Dance, & Banwell, 2015).

Mounting research has been dedicated to identifying maternal risk factors for these adverse birth outcomes. Far less attention has been given to paternal factors that could also predict adverse birth outcomes. Genetically, the father contributes to the newborn's chromosomal make-up. Evidence has shown that the father's genetic components are associated with the quality of the embryo and participate in fetal development (Constantinof, Moisiadis, & Matthews, 2016; Yang et al., 2015; Yuan et al., 2016). In addition, the father is often involved in establishing the quality of the fetal environment. For example, paternal smoking has been linked to LBW (Banderali et al., 2015). Paternal factors have also been associated with maternal anxiety and depression during pregnancy, which have been shown to increase the risk of PTB (Cheng et al., 2016; Liu, Cnattingius, Bergstrom, Ostberg, & Hjern, 2016).

Paternal factors such as age (Alio et al., 2012), height (Myklestad, Vatten, Magnussen, Salvesen, & Romundstad, 2013), weight (Miletic et al., 2007), race/ethnicity (Fulda, Kurian, Balyakina, & Moerbe, 2014), education (Leung, Leung, & Schooling, 2016), and smoking (Mutsaerts et al., 2014), and their relationship to birth outcomes have been investigated. However, previous studies have typically examined only one paternal factor (Alio et al., 2012; Fulda et al., 2014; Myklestad et al., 2013), and few studies have focused solely on paternal influence and assessed multiple paternal factors. The majority of these studies investigated paternal anthropometric parameters (Miletic et al., 2007). Paternal socioeconomic status (SES) (Leung et al., 2016) and paternal lifestyle factors (Mutsaerts et al., 2014) have also been examined.

For the current study the paternal factors of age, race/ethnicity, and educational attainment were assessed. In addition, the association of paternal factors with HBW was evaluated, which to date has not been reported. The purpose of this study was to systematically examine the relationship between potential paternal risk factors and adverse birth outcomes including PTB, LBW, SGA, and HBW in primiparous women. History of previous adverse birth outcomes provides valuable information for subsequent births to health care providers (Fulda et al., 2014; Simonsen et al., 2013). However, there is no prior pregnancy history to assist the prediction of adverse birth outcomes for primiparous women compared to multiparous women (Morken, Kallen, & Jacobsson, 2014). Therefore, the sample was limited to first-time mothers in order to determine whether paternal factors can provide additional value in predicting adverse birth outcomes in primiparous women.

Methods

A cross-sectional analysis was performed using birth certificate data retrieved from the Finger Lakes Regional Perinatal Data System (PDS). The initial dataset consisted of 171,195

de-identified live birth records that were collected in upstate New York from 2004 to 2015. Eligibility criteria for this study included primiparous women with singleton pregnancies. Women who underwent infertility treatment, who were reported to be single mothers, or whose birth records were missing paternal information, were excluded. A total of 36,731 birth records were included for the analysis. The study was conducted in accord with prevailing ethical principles and approved by the Institutional Review Board.

Dependent Variables

The outcomes of interest included PTB, LBW, HBW, and SGA. Clinical estimate of gestational age was categorized into PTB (< 37 weeks) and full term birth (≥37 weeks). The estimate of gestational weeks, per the PDS guidelines, was determined by birth attendants, based on all perinatal factors and assessments such as ultrasounds. Birthweight was categorized into LBW (< 2,500 grams), normal weight (≥ 2,500 grams and ≤ 4,000 grams), and HBW (> 4,000 grams). SGA was classified as a sex-specific and gestational age-specific birthweight falling below the 10^{th} percentile of the national reference (Oken et al., 2003). Due to a lack of reference values, 23 birth records with a gestational age less than 22 weeks were excluded from the analysis of SGA infants.

Independent Variables

All potential paternal risk factors available in the dataset, including age, race/ethnicity, and educational attainment, were included in the analysis. Race/ethnicity was classified into four groups (Non-Hispanic white [NHW], Non-Hispanic black [NHB], Hispanic, and Non-Hispanic others [NHO]). Educational level was divided into three categories (< high school, high school, > high school).

Covariates

The sex of the infant and maternal conditions including maternal employment during pregnancy, prepregnancy and gestational diabetes, prepregnancy and gestational hypertension, other chronic illnesses, smoking, illegal drug use, and financial assistance were all significantly associated with at least one adverse birth outcome ($P<0.05$) and therefore included in all statistical models. Other chronic illnesses, defined according to the PDS guidelines, referred to a chronic illness that required ongoing medical care and carried a significant risk of premature death or disability. Smoking was positive if the women reported smoking during pregnancy or within the three months before pregnancy. Financial assistance was defined as whether the woman received any benefit from government health care programs for low-income families, such as Medicaid and Special Supplemental Nutrition Program for Women Infants and Children (WIC). Maternal variables that corresponded to paternal independent variables, including age, race/ethnicity, and education were included in the fully adjusted statistical models. These maternal variables were categorized in the same manner as the paternal variables.

Statistical Analysis

Descriptive statistics were calculated to summarize the sample characteristics. The crude analyses were conducted on individual paternal independent variables without covariates

using chi-square tests and t tests. Then, two multivariate logistic regression models (partially adjusted and fully adjusted) were performed separately; determined by if the model controlled for the corresponding maternal variables (age, race/ethnicity, and education). Odds ratios (OR) and 95% confidence intervals (CI) were calculated. A *p* value less than 0.05 was considered statistically significant. There was less than 0.5% missing data for all variables. No strong collinearity was detected between independent variables and covariates. All variance inflation factors were less than 4. Statistical analyses were conducted using IBM SPSS version 22.0.

The mediation effects of maternal variables that corresponded to paternal risk factors were evaluated using the PROCESS macro in SPSS for a continuous mediator (Hayes, 2013) (e.g. maternal age), and ldecomp in STATA 13.0 (Buis, 2010) for binary mediators. Maternal and paternal race/ethnicity were dummy coded. Maternal and paternal educational variables were recoded into two categories (high school or less, more than high school) for the purpose of mediation analysis. The 95% CI was estimated with 1000 bootstrapping re-samples.

Results

The characteristics of the sample are presented in Table 1. In total, there were 36,731 singleton infants born to primiparous women in the Finger Lakes Region from January 2004 to December 2015. The rate of PTB, LBW, HBW, and SGA was 8.0%, 6.5%, 9.1% and 9.7% respectively. The age of the fathers ranged from 13 to 74 years old (mean = 28). The majority of fathers were NHW (77.2%) and 57.3% of fathers had more than a high school education at the time of delivery. The age of mothers ranged from 13 to 54 years old (mean = 26). The majority of mothers were NHW (79.7%) and more mothers than fathers had an educational level beyond high school (66.3%). Nearly 19% of mothers smoked during pregnancy or within the 3 months prior to pregnancy. Although the rate of smoking trended down as the pregnancies progressed, 8.5% of mothers continued to smoke during the third trimester. Approximately 40% of mothers received financial assistance from government programs (Medicaid and/or WIC).

Multivariate Regression Analyses

In the crude analysis of paternal factors without covariates, all three paternal factors (age, race/ethnicity, and education) were significantly associated with the four birth outcomes ($P<0.05$), except for a non-significant association between paternal age and PTB (P=0.412). The relationship between paternal factors and birth outcomes was further assessed by taking into consideration maternal factors and the sex of the infant. Two models of logistic regression analyses were conducted. Maternal medical and lifestyle conditions were controlled in the partially adjusted models. Maternal factors that corresponded to the paternal risk factors were added to the fully adjusted models. The results for these two models are presented in Table 2.

Preterm birth—In the partially adjusted logistic model, Hispanic and NHO fathers were associated with lower odds of PTB compared to NHW fathers. If paternal educational level was high school or less there was a significantly greater risk of PTB. When maternal

corresponding variables were added to the full model, the OR of paternal education was slightly reduced ($p < 0.001$). Fathers with less than a high school education had a 34.6% greater odds (95% CI 1.162–1.558) of having premature newborns, and fathers with a high school education had an increased odds of PTB by 22.6% (95% CI 1.107–1.357) compared to fathers with more than a high school education. The rates of PTB were 9.4%, 9.0% and 7.1% in fathers with less than high school, high school, and more than high school education, respectively. The association for NHO fathers persisted in the full model (OR 0.702, 95% CI 0.520–0.948). The rate of PTB in NHO fathers was 5.6% compared to 8.0% in NHW fathers. The maternal corresponding factors were not significantly related to the risk of PTB in the full model. The mediation effect of maternal corresponding factors was assessed but not significant for paternal race/ethnicity and education.

Low birthweight—Increased paternal age, NHB fathers, and fathers having a high school education or less were associated with higher risk of LBW in the partially and fully adjusted models. For each year increase in paternal age the risk of LBW increased by 1.2% in the full model (P=0.014). NHB fathers had higher odds (OR 1.244, 95% CI 1.031–1.501) of having LBW infants than NHW fathers: 9.7% of infants were born LBW in NHB fathers compared to 5.9% in NHW fathers. A less than high school education in fathers increased the odds of LBW by 49.7% (95% CI1.280–1.750), and a high school education in fathers increased the odds of LBW by 26.3% (95% CI 1.128–1.415). The rates of LBW were 9.2%, 7.5%, and 5.4% in fathers with less than high school, high school, and more than high school education, respectively. None of the maternal corresponding factors were significantly associated with the risk of LBW in the full model. Neither did the maternal factors significantly mediate the association of paternal risk factors with LBW.

High birthweight—Hispanic and NHO fathers had a lower chance of having HBW newborns compared to NHW fathers in both the partially and fully adjusted models. NHB fathers was significantly associated with HBW only in the partially adjusted model. NHB mothers mediated 76% of the association of NHB fathers with HBW (95% CI 0.324–1.190, P=0.001). NHO mothers mediated 52% of the association of NHO fathers with HBW (95% CI 0.255–0.795, P<0.001). The mediation effect of Hispanic mothers was not significant (P=0.087). The rate of HBW was 10.2% in NHW fathers, 5.6% in NHB fathers, 6.3% in Hispanic fathers, and 4.5% in NHO fathers.

Small for gestational age—In the partially and fully adjusted models, fathers from race/ethnicities other than NHW, and fathers having less than a high school education were related to an elevated risk of SGA. The risk of SGA increased with paternal age but only in the partially adjusted model. Maternal age significantly mediated the association of paternal age with SGA (P=0.049). The rate of SGA was 8.1% in NHW fathers, 16.1% in NHB fathers, 13.0% in Hispanic fathers, and 15.3% in NHO fathers. Although 42% of the relationship between NHB fathers and SGA was mediated by NHB mothers (95% CI 0.191–0.657, P<0.001), there was still a 38% (95% CI 1.185–1.607) increased risk of SGA for NHB fathers after adjustment for other paternal and maternal risk factors. For Hispanic fathers, the association with SGA was not significantly mediated by Hispanic mothers (P=0.642). The risk of SGA was increased by 22.5% in Hispanic fathers. For NHO fathers,

29% of the relationship with SGA was mediated by NHO mothers (95% CI 0.031–0.0.552, *P*=0.028). The odds of SGA increased by 1.748 times in NHO fathers, independent of the other factors in the model. Paternal education was also related to the risk of SGA. The rates of SGA were 14.0%, 10.7%, and 8.2% in fathers with less than high school, high school, and more than high school education, respectively. Maternal education mediated 38% of the relationship between paternal education and SGA (95% CI 0.053–0.712, *P*=0.023). Being a father with a less than high school education was independently associated with 17% increased odds of SGA.

Discussion

Paternal risk factors of age, race/ethnicity, and educational level were significantly associated with adverse birth outcomes. These paternal risk factors were better predictors for PTB and LBW than the corresponding maternal risk factors. Paternal risk factors also were independently related to the risk of HBW and SGA, even when adjustment was made for maternal corresponding variables and other conditions. These outcomes support the unique association between fathers and neonatal health.

Of all paternal factors, educational attainment was the risk factor that was consistently related to adverse birth outcomes, with the exception of HBW. In this study, an educational level of high school or less was associated with an elevated risk of PTB, LBW, and SGA. A few studies have investigated the association of paternal education with PTB (Mortensen, 2013; Parker, Schoendord, & Kiely, 1994; Shapiro et al., 2017), LBW (Leung et al., 2016; Nicolaidis, Ko, Saha, & Koepsell, 2004; Parker et al., 1994), and SGA (Leung et al., 2016; Mortensen, 2013; Shapiro et al., 2017). The primary focus of these studies was parental socioeconomic factors and other paternal factors were minimally considered. Nicolaidis et al. (2004) included paternal age and race in their study. However, in that study separate analyses by maternal racial group for LBW were conducted as opposed to this study where race was included as a variable in the regression analyses which enable the analysis of the effect of maternal race/ethnic on the relationship between paternal factors and birth outcomes. The association of paternal education with adverse birth outcomes diminished when maternal factors were included in some studies (Leung et al., 2016; Mortensen, 2013; Parker et al., 1994) but not in others (Nicolaidis et al., 2004; Shapiro et al., 2017). In this study, paternal education retained significance even after controlling for paternal age, race/ethnicity, and maternal health and lifestyle conditions. Interestingly, the relationship between paternal education and adverse birth outcomes followed a dose-response relationship: a lower paternal educational level was associated with a higher risk of negative outcomes. A similar trend has been reported in previous studies (Mortensen, 2013; Nicolaidis et al., 2004; Shapiro et al., 2017). The addition of mediation analysis refines findings by demonstrating that maternal education partially mediated the association of paternal education with SGA but not the association with PTB or LBW.

Maternal and/or paternal educational attainment have frequently been used as indicators of socioeconomic status (Galobardes, Shaw, Lawlor, Lynch, & Davey Smith, 2006). The association between education and health outcomes is often classified into two categories: direct and indirect effects (Clark & Royer, 2013; Galobardes et al., 2006). The indirect

effects commonly refer to the relationship between education, income and occupation. For example, adults with a higher educational level may have greater access to health care related resources through their employer. The direct effects are increased accessibility and utilization of health information and technology as a consequence of more education (Yamashita & Kunkel, 2015). Adults with advanced education are more likely to be receptive to and compliant with health instructions. Plus, limited educational levels are related to health risk behaviors, which are associated with adverse birth outcomes. For example, it has been suggested that smoking, which has a negative effect on birthweight, is a mediator between maternal education and birthweight (Mortensen et al. 2009). In the current study, a portion of the relationship between paternal education and SGA was mediated by maternal education. However, factors that might mediate the association of paternal education with PTB and LBW were not determined. Further research is needed to assess if other maternal and paternal health risk behaviors play a role in mediating the association between paternal education and birth outcomes.

Of note, in this study paternal education was consistently associated with birth outcomes, especially PTB and LBW, more so than maternal education. A similar tendency has also been detected in previous studies (Leung et al., 2016; Mortensen, 2013; Parker et al., 1994), which suggests that paternal education may be a better indicator of socioeconomic status for certain birth outcomes than maternal education. The effects of paternal education remained evident even when maternal demographic characteristics, education, and health risk behaviors, such as smoking, were controlled for in the analysis, indicating that other unidentified mechanisms link paternal education with neonatal outcomes.

Paternal race/ethnicity was another factor that was consistently associated with adverse birth outcomes, including PTB, LBW, and SGA, which has also been found in previous studies (Fulda et al., 2014; Ma, 2008; Srinivasjois, Shreya, & Shah, 2012). The results from this study are consistent with prior findings regarding the association of NHB fathers with LBW and SGA, although no association was found with PTB in this study. One difference between this study and previous ones is that the sample in this study was limited to primiparous women with singleton pregnancies, while other studies included multiparous women. To our knowledge, there are no studies that examined the relationship between paternal race/ethnicity and HBW. In this study, fathers being Hispanic and other race/ethnicity had lower odds of having HBW infants. On the other hand, they had higher odds of having SGA infants. These results suggest that the association of paternal race/ethnicity with birthweight is consistent across HBW and SGA, possibly indicating that the mechanism by which paternal race/ethnicity affects HBW is similar to that for SGA.

There have been multiple attempts to explain known racial/ethnic discrepancies in birth outcomes. An advantage of this study is that the analysis on paternal race/ethnicity was conducted while controlling maternal corresponding variables, including age, race/ethnicity, and education. Previous studies paired fathers and mothers by race/ethnicity and investigated multiracial families (Fulda et al., 2014; Srinivasjois, Shreya, & Shah, 2012), an approach which did not allow for evaluation of the role of maternal race/ethnicity on the relationship between paternal race/ethnicity and birth outcomes. In the current study the relationships among maternal and parental race/ethnicity were assessed. Together with the mediation

analysis, paternal racial/ethnic disparities were partially explained by maternal race/ethnicity for HBW and SGA, yet the association between certain paternal race/ethnicity and birth outcomes persisted after adjusting for maternal conditions, which implies there are other potential mediators that link paternal race/ethnicity to adverse birth outcomes. Giscombé and Lobel (2005) suggested that socioeconomic status, health behavior, stress, and biological response to stress could partially clarify the racial/ethnic disparities. There is also the possibility that paternal race exerts a direct effect on gestational age and birthweight through genetic influence. For example, a variation in fetal Catechol-O-methyltransferase (*COMT*) gene, whose activity is different between African-Americans and Caucasians, was associated with PTB only in African-American infants (Thota et al., 2012). Also, methylation status on two PTB related genes has been found to differ between black and non-black fetuses (Salihu et al., 2016).

In this study, paternal age was significantly associated with LBW and SGA. Maternal age significantly mediated the association of paternal age with SGA, but not LBW. Previous studies have investigated the impact of paternal age on PTB, LBW, and SGA. However, the results were inconsistent on all birth outcomes. For example, similar to our findings, Reichman and Teitler (2006) found the odds of LBW increased as paternal age increased. Alio et al. (2010), however, found younger and older age of fathers were associated with a higher risk of LBW. Furthermore, in that study, paternal age between 30 to 45 years was associated with a lower odds of SGA compared to fathers below 30 and over 45 years of age. Yet, Hurley and DeFranco (2017) found that increasing paternal age was related to an elevated risk of SGA. It is possible that the relationship between paternal age and adverse birth outcomes is not linear, and therefore more studies are warranted.

The strengths of this study are the inclusion of multiple paternal factors and adjustment for maternal corresponding factors, medical and lifestyle factors. To our knowledge, this is the first study to evaluate the relationship between paternal risk factors and HBW. We also examined whether corresponding maternal variables mediated the association of paternal factors with birth outcomes. The retrospective design using birth certificate data, which might include recall bias, is a limitation of this study. This is of particular concern, given that paternal factors were reported by mothers and not directly by the fathers. In addition, the perinatal data were collected within the Finger Lakes Region of upstate New York and therefore may not be generalizable to other populations. Racial groups other than NHW and NHB were limited in the dataset, and consequently could not be assessed in a more precise manner. Another limitation of this study is the absence of data on additional paternal and maternal factors, such as family income and paternal smoking status. Future studies using data with additional paternal variables are needed to further elucidate the relationship between paternal risk factors and adverse birth outcomes.

Conclusion

Paternal factors were independently associated with adverse birth outcomes, including PTB, LBW, HBW, and SGA. The majority of existing research, obstetrical care, and policies do not routinely take paternal factors into account. Future studies, programs, and policies targeted at reducing adverse birth outcomes should incorporate paternal characteristics and

engagement of fathers directly into prenatal care. Moreover, the current evidence supports the existence of paternal effects on birth outcomes, but it is uncertain what elements other than the maternal factors assessed in this study could link the paternal factors with birth outcomes. Further research is needed to identify any connections.

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Significance

What is already known on this subject?

Mounting research has been dedicated to identifying maternal risk factors for adverse birth outcomes. Far less attention has been given to the effect of paternal factors on birth outcomes.

What does this study add?

This study systematically assessed the relationship between potential paternal risk factors and four birth outcomes. Paternal age, race/ethnicity, and educational level were significantly related to preterm birth, low birthweight, high birthweight, and small for gestational age after adjusting for maternal factors. Our results support the important contribution of paternal factors to birth outcomes.

Table 1

Demographic and medical characteristics of the sample (n = 36,731) with live births

Characteristic	Total (*n* = 36,731) *n* (%)
Male infant	18,958 (51.6)
Outcomes	
Preterm birth	2,925 (8.0)
Low birthweight	2,382 (6.5)
High birthweight	3,358 (9.1)
SGA[a]	3,546 (9.7)
Paternal factors	
Age, mean (SD)	28 (6.4)
Race/ethnicity	
Non-Hispanic White	28,374 (77.2)
Non-Hispanic Black	3,952 (10.8)
Hispanic	2,749 (7.5)
Non-Hispanic Others	1,656 (4.5)
Education	
< High school	4,884 (13.3)
High school	10,792 (29.4)
> High school	21,055 (57.3)
Maternal covariates	
Age, mean (SD)	26 (5.5)
Race/ethnicity	
Non-Hispanic White	29,273 (79.7)
Non-Hispanic Black	3,185 (8.7)
Hispanic	2,478 (6.7)
Non-Hispanic Others	1,795 (4.9)
Education	
< High school	4,468 (12.2)
High school	7,879 (21.5)
> High school	24,340 (66.3)
Employment during pregnancy (yes)	26,707 (72.7)
Diabetes, prepregnancy	255 (0.7)
Diabetes, gestational	1,542 (4.2)
Hypertension, prepregnancy	591 (1.6)
Hypertension, gestational	3,190 (8.7)
Other Chronic illnesses	3,176 (8.6)
Smoking[b]	6,949 (18.9)
Illegal drug use	1,017 (2.8)
Financial assistance (yes)	14,770 (40.2)

[a]SGA is small for gestational age;;

[b] including smoking within the 3 months before pregnancy.

Table 2

Associations between paternal factors and birth outcomes in the partially adjusted and fully adjusted multivariate logistic regression models.

	Preterm Birth OR (95% CI)		Low Birthweight OR (95% CI)		High Birthweight OR (95% CI)		SGA OR (95% CI)	
	Partially adjusted	**Fully adjusted**	**Partially adjusted**	**Fully adjusted**	**Partially adjusted**	**Fully adjusted**	**Partially adjusted**	**Fully adjusted**
Paternal age[a]	1.005 (0.998–1.012)	1.005 (0.996–1.014)	**1.011**** **(1.004–1.019)**	**1.012*** **(1.003–1.022)**	1.006 (0.999–1.012)	1.007 (0.998–1.016)	**1.009***** **(1.003–1.015)**	1.005 (0.997–1.013)
Paternal race/ethnicity[b]								
Non-Hispanic Black	0.994 (0.874–1.130)	1.074 (0.897–1.286)	**1.326***** **(1.164–1.511)**	**1.244*** **(1.031–1.501)**	**0.584***** **(0.502–0.680)**	0.888 (0.728–1.084)	**1.857***** **(1.670–2.063)**	**1.380***** **(1.185–1.607)**
Hispanic	**0.853*** **(0.730–0.998)**	0.849 (0.708–1.019)	1.048 (0.892–1.231)	1.012 (0.837–1.223)	**0.638***** **(0.539–0.754)**	**0.780*** **(0.645–0.942)**	**1.493***** **(1.314–1.695)**	**1.225**** **(1.052–1.427)**
Non-Hispanic Others	**0.728**** **(0.583–0.909)**	**0.702*** **(0.520–0.948)**	1.065 (0.859–1.321)	1.051 (0.777–1.422)	**0.396***** **(0.311–0.503)**	**0.642**** **(0.470–0.876)**	**2.168***** **(1.872–2.512)**	**1.748***** **(1.402–2.179)**
Non-Hispanic White	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Paternal education[c]								
< High school	**1.408***** **(1.230–1.613)**	**1.346***** **(1.162–1.558)**	**1.541***** **(1.335–1.780)**	**1.497***** **(1.280–1.750)**	0.957 (0.830–1.104)	1.122 (0.963–1.308)	**1.263***** **(1.122–1.421)**	**1.170*** **(1.028–1.332)**
High school	**1.258***** **(1.144–1.383)**	**1.226***** **(1.107–1.357)**	**1.274***** **(1.145–1.417)**	**1.263***** **(1.128–1.415)**	0.945 (0.863–1.035)	0.987 (0.896–1.088)	**1.117*** **(1.022–1.221)**	1.093 (0.993–1.202)
> High school	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Maternal age[a]	-	1.001 (0.989–1.014)		0.998 (0.984–1.012)		0.995 (0.983–1.006)		**1.012*** **(1.000–1.023)**
Maternal race/ethnicity[b]	-							
Non-Hispanic Black		0.863 (0.707–1.055)		1.094 (0.893–1.340)		**0.539***** **(0.426–0.682)**		**1.533***** **(1.303–1.803)**
Hispanic		1.010 (0.839–1.217)		1.05 (0.862–1.279)		**0.707**** **(0.576–0.867)**		**1.402***** **(1.200–1.639)**
Non-Hispanic Others		1.057 (0.802–1.392)		1.028 (0.766–1.379)		**0.497***** **(0.367–0.674)**		**1.366**** **(1.096–1.702)**
Non-Hispanic White		Reference		Reference		Reference		Reference
Maternal education[c]	-							
< High school		1.147 (0.979–1.344)		1.063 (0.899–1.258)		**0.640***** **(0.535–0.764)**		**1.238**** **(1.079–1.419)**
High school		1.083 (0.968–1.213)		1.006 (0.888–1.140)		**0.885*** **(0.792–0.988)**		1.097 (0.988–1.217)

	Preterm Birth OR (95% CI)		Low Birthweight OR (95% CI)		High Birthweight OR (95% CI)		SGA OR (95% CI)	
	Partially adjusted	**Fully adjusted**	**Partially adjusted**	**Fully adjusted**	**Partially adjusted**	**Fully adjusted**	**Partially adjusted**	**Fully adjusted**
> High school	Reference		Reference		Reference		Reference	

P values are indicated by asterisks, with **p* < 0.05; ***p* < 0.01; ****p* < 0.001.

All models (partially adjusted and fully adjusted) include covariates, including sex of the infant, maternal diabetes, hypertension, smoking, illegal drug use, and financial assistance. Fully adjusted models also control for maternal age, race/ethnicity, and education.