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Gestational Weight Gain and Pregnancy Complications in a High-Risk, Racially and Ethnically Diverse Population

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Abstract

Background: Evidence used to guide the current Institute of Medicine (IOM) gestational weight gain (GWG) recommendations is largely derived from studies of European descent, and as such the guidelines are the same for all races and ethnicities. The guidelines are also the same for prepregnancy obesity classes I through III.
Objective: Considering these literature gaps, we aim to (1) determine the association between the IOM GWG guidelines and three common pregnancy complications: preeclampsia, gestational diabetes mellitus, and cesarean delivery in a racially and ethnically diverse population; and (2) assess whether the association between GWG and pregnancy complications differs by race/ethnicity or prepregnancy body mass index (BMI) categories, particularly obesity classes I through III.
Methods: To address these aims, we analyzed data from the Boston Birth Cohort. We calculated GWG using data from electronic medical records or, when missing, from a postdelivery questionnaire. We examined GWG continuously and categorically using the IOM formula.
Results: Of the 5,568 women included, 54.5% met the IOM criterion for excessive GWG. Compared to women who had adequate GWG, women who gained excessive weight had 1.65 (95% confidence interval [CI] 1.27–2.14) times greater odds of preeclampsia; 1.68 (95% CI 1.15–2.46) times greater odds of gestational diabetes; and no significant change in odds of cesarean delivery (odds ratio [OR] = 1.14, 95% CI 0.99–1.31). Associations did not differ by race, ethnicity, or prepregnancy BMI categories including comparisons of obesity class I versus II or III (all p-values for interaction >0.05).
Conclusions: In this racially and ethnically diverse population, excessive GWG was associated with higher odds of preeclampsia, gestational diabetes, and nonsignificantly, with cesarean delivery. Associations did not differ appreciably by race, ethnicity, or prepregnancy BMI categories. Our results support the relevance of the IOM GWG recommendations in racially and ethnically diverse populations, and in women in the higher prepregnancy obesity classes.

Keywords: body mass index, gestational weight gain, obesity, pregnancy, race and ethnicity

Introduction

Approximately 40% of reproductive age women in the United States are obese and 10% have class III obesity, reflecting a significant rise over the last decade.[1] The burden of obesity disproportionately affects women of racial and ethnic minorities.[2,3] The growing number of women entering pregnancy as obese presents a challenge to clinicians who provide recommendations on gestational weight gain (GWG), as inadequate or excess weight gain may have consequences for pregnancy complications.[4–9]

In 2009 the Institute of Medicine (IOM) updated recommendations for GWG based on prepregnancy body mass index (BMI) categories (underweight, normal weight, overweight,

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and obesity).[10,11] The majority of studies evaluating the IOM guidelines' association with pregnancy outcomes were conducted in predominantly white populations,[4–8] despite the greater prevalence of prepregnancy obesity and weight-related pregnancy complications among minority racial and ethnic groups.[12–14] Furthermore, the IOM recommendations do not distinguish women in obesity class I from those in class II or III, in whom weight gain may not be needed.[15]

Considering these literature gaps, our study used a large racially and ethnically diverse U.S. birth cohort to examine the association between the IOMs GWG guidelines and three common pregnancy complications: preeclampsia, gestational diabetes mellitus (GDM), and cesarean delivery. We hypothesized that excessive GWG and inadequate GWG, defined by the IOM guidelines, are associated with these adverse pregnancy outcomes. We further aimed to explore whether the association between GWG and pregnancy complications is modified by race, ethnicity, or prepregnancy BMI categories, including obesity class I and class II/III.

Methods

This study was approved by the Institutional Review Board at Boston University Medical Center and Children's Memorial Hospital.

Study population

We analyzed data from the Boston Birth Cohort. Initiated in 1998, the Boston Birth Cohort is a racially and ethnically diverse (65% black, 11% white, and 25% Hispanic) urban cohort recruited in a single hospital, with electronic medical records (EMRs) in place since 2002. Details about the study design were described previously.[16,17] The cohort excluded pregnancies with multiple gestations, pregnancies conceived *via* assisted reproductive technologies, and pregnancies with less than 23 weeks of gestation. Pertinent epidemiological and clinical data were obtained from both the maternal and infant EMRs and an in-person postdelivery maternal questionnaire (within 1–3 days after index delivery).

For this analysis, we excluded women missing data on prepregnancy BMI, gestational age, GWG, or the pregnancy complications of interest. We excluded women with extreme GWG values (defined as GWG < −9 or ≥45 kg) or women diagnosed with chronic hypertension, type 1 or type 2 diabetes before current pregnancy (Supplementary Fig. S1; Supplementary Data are available online at www.liebertpub.com/jwh).

Outcome variables

Our primary outcomes were preeclampsia, GDM, and cesarean delivery, defined based on the review of maternal EMR. Preeclampsia was defined according to the report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy,[18] as having a systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg on at least two occasions, and proteinuria of at least 1+ by urine dipstick testing on two or more occasions, after 20 weeks of gestation, excluding individuals with preexisting hypertension. In this study, we also included its severe forms, eclampsia and hemolysis, elevated liver enzymes, and low platelets (*i.e.*, HELLP) syndrome in the ''preeclampsia'' definition. GDM diagnosis was based on maternal EMR. As part of clinical care at Boston Medical Center, pregnant women underwent screening for GDM with an oral nonfasting glucose challenge test and/or fasting glucose tolerance test at 24–28 completed weeks of gestation. GDM was defined as mothers ever diagnosed with diabetes mellitus (DM) complicating pregnancy (ICD-9 codes: 648.00 and 648.03) but never diagnosed with DM (250.00–250.93) or self-reported having preexisting DM from prior clinical diagnosis. Mode of delivery, also extracted from EMR, was categorized as cesarean delivery or vaginal delivery. We included operative vaginal deliveries with vaginal deliveries.

Definition of GWG

For the outcomes preeclampsia and cesarean delivery, we calculated total GWG from the EMR prenatal weights as the difference between the earliest recorded weight in first trimester (0–13 weeks) and last recorded weight before delivery. The last prenatal weight had to be measured within 3 weeks of delivery for women with full-term deliveries (≥37 weeks gestation) and within 2 weeks of delivery for preterm deliveries (<37 weeks gestation). Since all the women were universally screened for GDM between 24 and 28 gestational weeks, we decided to use GWG until GDM screening/diagnosis period to examine the association between pregnancy weight gain and GDM.

Given the availability of both self-reported prepregnancy weight and clinically measured prenatal weight (in a subset), we used the following approach to maximize data utility and validity of the analyses. If the last prenatal weight was available from EMR but the first prenatal weight was missing, we used the self-reported prepregnancy weight from the questionnaire for the GWG calculation. The study flow of sample selection and sources of GWG data can be found in the Supplementary Figure S1. The Pearson correlation coefficient between self-reported prepregnancy weight and first prenatal weight from the EMR in first trimester was 0.93, based on 2,439 women having both weights available. When neither the first or last prenatal weight measurements were available from the EMR, we used the self-reported total GWG ($N = 3{,}450$). The Pearson correlation between the EMR-based and self-reported total GWG was 0.70 among 2,293 women with both measurements available. The average rate of GWG per week was 0.36 kg (standard deviation [SD] 0.19) for EMR-based weights ($N = 2{,}118$) and 0.37 kg (SD 0.20) for self-reported total GWG ($N = 3{,}450$). The average difference between gestational week of delivery and gestational week of last weight measurement was 0.60 (SD 0.59) week.

We applied several definitions to characterize GWG. For categorical exposure, we used the IOM GWG categories of recommended total GWG based on prepregnancy BMI.[10] We based our IOM GWG category calculation upon the method published by Gilmore and Redman.[19] This method adjusts for length of gestation, taking into account the 2009 IOM trimester-specific weekly weight gain guidelines. The IOM guidelines assume a gain of 0.5–2 kg in the first trimester for all pregnancy BMI categories and provide weekly incremental weight gain guidelines for the second and third trimesters specific to each prepregnancy BMI category. To enable clinical interpretation and extend the application of our results beyond the IOM guideline, we then explored

continuous measurements of GWG, scaling the total GWG to 5 kg weight gain increments.

Covariates

Maternal age at delivery and parity were obtained from the prenatal EMR. Prepregnancy BMI, race/ethnicity, smoking during pregnancy, drinking alcohol during pregnancy, education level, and marital status were from the maternal questionnaire interview. In the Boston Birth Cohort, gestational age was assessed based on both the first day of the last menstrual period and early (<20 weeks) prenatal ultrasound as recorded in the maternal EMR. This approach has been used in previous publications and in our past and ongoing NIH funded preterm studies in the cohort.[16,17] Prepregnancy BMI was calculated as the weight in kilograms divided by the squared height in meters and applying standard BMI categories: underweight, BMI <18.5 kg/m^2; normal weight 18.5 ≤ BMI <25.0 kg/m^2; overweight, 25.0 ≤ BMI <30.0 kg/m^2; obesity class I: 30.0 ≤ BMI <35.0 kg/m^2; obesity class II/III: BMI ≥35.0 kg/m^2.

Statistical analyses

We used the ANOVA (analysis of variance) and chi-squared tests to examine the differences in baseline characteristics across women in the three GWG groups and to compare GWG across different race and ethnicity groups. For the main analyses, we used multivariable logistic regression to examine the associations of the pregnancy outcomes and GWG categories and continuous GWG. We conducted a test for trend for ordered categorical GWG and further checked for linearity by modeling the relationship between continuous GWG and probability of each outcome using a spline.

We chose to include covariates in our multivariable models based on prior literature and if they were associated with GWG and one of the outcome variables but were not on the causal pathway from clinical and biological perspective.[20] For our primary analyses, we adjusted our models for maternal age, parity, education, marital status, any smoking during pregnancy, and any alcohol drinking during pregnancy. We further adjusted continuous GWG models for gestational week at delivery. Since the GWG categories we derived are specific for gestational age,[19] we did not include gestational age in the categorical GWG analysis models.

We examined effect modification of GWG categories with race and ethnicity categories (non-Hispanic black, non-Hispanic white, Hispanic, and other) and prepregnancy BMI categories (under/normal weight, overweight, obesity I, obesity II/III) on each pregnancy outcome by first stratifying on these covariates and then formally testing multiplicative interaction by including cross-product terms for GWG categories and each covariate in the multivariable model.

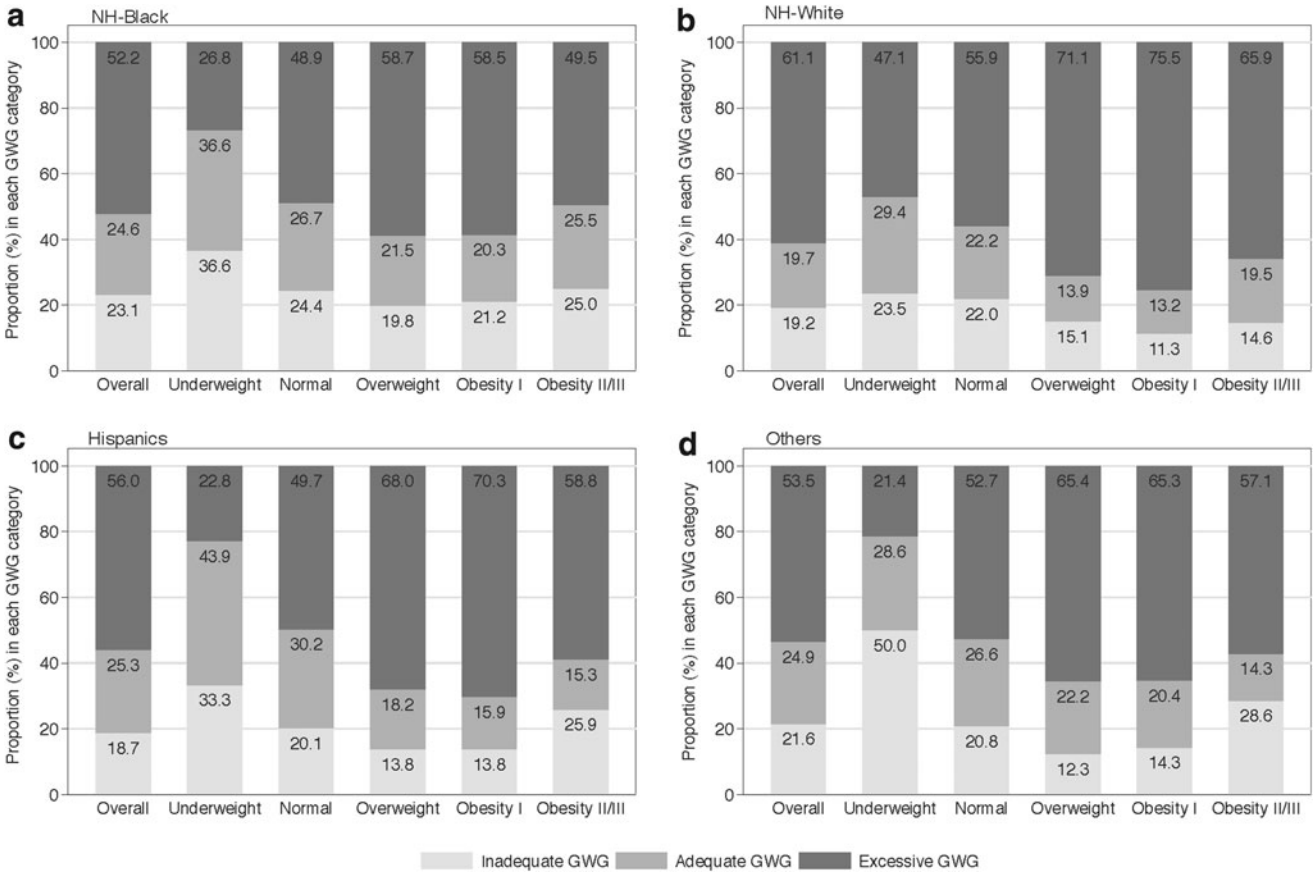

FIG. 1. Proportion (%) of women from the Boston Birth Cohort in each IOM GWG category (x-axis) across prepregnancy BMI categories (y-axis), according to self-identified race and ethnicity. **(a)** Non-Hispanic black women; **(b)** non-Hispanic white women; **(c)** Hispanic women; **(d)** women of other race/ethnicity. BMI, body mass index; GWG, gestational weight gain; IOM, Institute of Medicine.

Given that there were two weight measurements (EMR weight and self-report weight gain) used in this analysis, we performed a sensitivity analysis that was restricted to women with EMR data to test for the robustness of our results.

We performed statistical analyses with Stata 13.0 (StataCorp. 2013, Stata Statistical Software: Release 13; StataCorp LP, College Station, TX). Confidence intervals (CIs) at 95% were presented, and statistical significance was set at two-tailed $p < 0.05$.

Results

We obtained GWG data from 6,620 women, among whom we excluded 32 individuals with extreme GWG outside the predefined range, 574 with missing outcomes or covariates, and 446 with prepregnancy hypertensive disorder or DM before index pregnancy (see Supplementary Fig. S1 for sample selection). We included 5,568 women our analysis, 2,737 (49.2%) were non-Hispanic black, 704 (12.6%) were non-Hispanic white, 404 (25.2%) were Hispanic, and 723 (13.0%) were of another race/ethnicity (mainly Asian). According to the IOM guideline, overall, 24.2% gained an adequate amount of weight during pregnancy, while 54.5% had excessive GWG and 21.3% had inadequate GWG. Black women had the highest proportion of prepregnancy overweight and obesity (Supplementary Fig. S2) and gained less total weight during pregnancy compared with white women, after controlling for other factors (Supplementary Table S1). The mean GWG for non-Hispanic black, non-Hispanic white, Hispanic women, and women of other race/ethnicity were 12.4 kg (27.3 lbs), 14.2 kg (31.3 lbs), 13.5 kg (29.8 lbs), and 13.6 kg (30.0 lbs) respectively.

Figure 1 shows the proportion meeting criteria for the three IOM GWG categories in each prepregnancy BMI group, stratified by race/ethnicity. White women had the highest prevalence of excess GWG (61.1%), while non-Hispanic black women had the highest prevalence of inadequate GWG (23.1%). Women who were overweight or with class I obesity before pregnancy had the highest prevalence of excess GWG across the four race/ethnicity groups (Table 1).

Figure 2 shows the overall associations between the IOM GWG categories and pregnancy complications. Compared to

Table 1. Participant Characteristics by Institute of Medicine Gestational Weight Gain Categories, Mean (Standard Deviation) or *N* (%)

	Overall, N = *5,568*	*Inadequate,* N = *1,187*	*Adequate,* N = *1,348*	*Excessive,* N = *3,033*
Maternal age at delivery, years	27.8 (6.4)	28.1 (6.5)	28.0 (6.4)	27.5 (6.2)
Maternal age >35	791 (14.2)	187 (15.8)	202 (15.0)	402 (13.3)
Race/ethnicity				
Non-Hispanic black	2,737 (49.2)	633 (53.3)	674 (50.0)	1,430 (47.1)
Non-Hispanic white	704 (12.6)	135 (11.4)	139 (10.3)	430 (14.2)
Hispanic	1,404 (25.2)	263 (22.2)	355 (26.3)	786 (25.9)
Other	723 (13.0)	156 (13.1)	180 (13.4)	387 (12.8)
Prepregnancy weight (lbs)	148.2 (35.0)	146.6 (37.6)	143.9 (35.5)	150.7 (33.5)
Prepregnancy BMI, kg/m^2	25.5 (5.7)	25.1 (6.0)	24.7 (5.8)	25.9 (5.5)
Prepregnancy BMI[a]				
Underweight	273 (4.9)	103 (8.7)	96 (7.1)	74 (2.4)
Normal	2,813 (50.5)	629 (53.0)	759 (56.3)	1,425 (47.0)
Overweight	1,509 (27.1)	257 (21.7)	301 (22.3)	951 (31.4)
Obesity I	611 (11.0)	110 (9.3)	114 (8.5)	387 (12.8)
Obesity II/III	362 (6.5)	88 (7.4)	78 (5.8)	196 (6.5)
Currently married	1,929 (34.6)	371 (31.3)	514 (38.1)	1,044 (34.4)
High school or above education	3,891 (69.9)	786 (66.2)	929 (68.9)	2,176 (71.7)
Any smoking during pregnancy	1,145 (20.6)	244 (20.6)	226 (16.8)	675 (22.3)
Any drinking during pregnancy	442 (7.9)	87 (7.3)	91 (6.8)	264 (8.7)
Parity (prior birth ≥1)	3,116 (56.0)	705 (59.4)	786 (58.3)	1,625 (53.6)
Prenatal care onset				
During 1st trimester	2,508 (45.0)	502 (42.3)	621 (46.1)	1,385 (45.7)
After 1st trimester	924 (16.6)	199 (16.8)	206 (15.3)	519 (17.1)
Unknown	2,136 (38.4)	486 (40.9)	521 (38.6)	1,129 (37.2)
GWG, lbs	28.8 (15.4)	11.0 (8.2)	22.7 (5.7)	38.5 (12.6)
Gestational week at delivery, weeks	38.3 (3.0)	38.1 (2.9)	38.3 (3.0)	38.3 (3.0)
Preterm birth	1,291 (23.2)	309 (26.0)	286 (21.2)	696 (22.9)
Male infant	2,761 (49.6)	563 (47.4)	469 (48.15)	1,549 (51.1)
Preeclampsia	425 (7.6)	59 (5.0)	78 (5.8)	288 (9.5)
GDM	364 (6.5)	77 (6.5)	64 (4.7)	223 (7.4)
Cesarean delivery	1,708 (30.7)	291 (24.5)	403 (29.9)	1,014 (33.4)

The range of GWG in the total population is −20 to 96 lbs; the range of GWG in inadequate weight gain group is −20 to 28 lbs; the range of GWG in adequate weight gain group is 6–40 lbs; and the range of GWG in excessive weight gain group is 12–96 lbs.

[a]The cutoffs for the prepregnancy BMI categories follows WHO definition: Underweight: BMI <18.5 kg/m^2; normal weight: BMI 18.5–24.9 kg/m^2; overweight: BMI 25–29.9 kg/m^2; obesity class I: BMI 30–34.9 kg/m^2; obesity class II/III: BMI ≥35 kg/m^2.

BMI, body mass index; GDM, gestational diabetes mellitus; GWG, gestational weight gain.

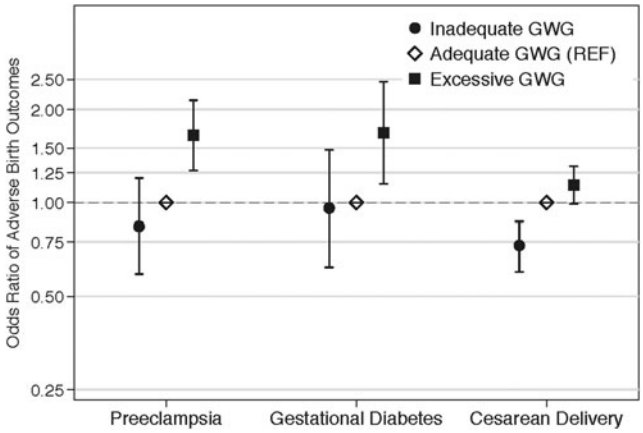

FIG. 2. Associations[a] of IOM GWG categories with pregnancy complications[b] in the Boston Birth Cohort. [a]Logistic regression model was adjusted for age at delivery, race/ethnicity, education level, any smoking during pregnancy, any drinking during pregnancy, marital status, and prepregnancy BMI. [b]Models for preeclampsia and cesarean delivery used data from the whole study population ($N = 5{,}568$); Models for GDM used data from women who had both GWG measurement until GDM diagnosis period and total GWG measurement over pregnancy available ($N = 2{,}544$). GDM, gestational diabetes mellitus.

women with adequate GWG, women who gained excess weight had 1.65 (95% CI 1.27–2.14) times higher odds of preeclampsia, 1.68 (95% CI 1.15–2.46) times higher odds of GDM, and a non-significant 1.14 (95% CI 0.99–1.31) times greater odds of cesarean delivery. Women who gained inadequate weight had 0.73 (95% CI 0.60–0.87) times lower odds of cesarean delivery and a nonsignificant 0.84 (95% CI 0.59–1.20) times lower odds of preeclampsia.

We found evidence of a linear trend of GWG with all three outcomes (trend test p-values were all <0.001). Using a continuous scale for GWG, each 5 kg increase in GWG was associated with 1.38 times greater odds of preeclampsia (95% CI 1.28–1.47), 1.13 times greater odds of GDM (95% CI 0.99–1.28), and 1.17 times greater odds of cesarean delivery (95% CI 1.12–1.23) (Table 2).

In sensitivity analyses, in which we restricted our analytic sample to women with calculated EMR GWG, the categorical and continuous GWG results remain similar as in the main findings, except for the association between excessive GWG and preeclampsia that became nonsignificant (odds ratio [OR] = 1.26, 95% CI 0.84–1.89), which may be a result of the smaller sample size (Supplementary Table S2).

Figure 3 shows the associations between IOM GWG categories and the three pregnancy complications across racial and ethnic groups and prespregnancy BMI categories, including of class I versus class II/III obesity. We observed similar magnitudes in the association between the GWG categories and pregnancy outcomes across the four racial and ethnic groups in our cohort (all p-values for multiplicative interaction >0.05, Fig. 3). We also found that associations between GWG categories and pregnancy outcomes did not differ markedly by prepregnancy BMI groups (all p-values for interaction >0.55, Fig. 3); including when the analysis was restricted to comparisons by obesity class I versus II/III (all p-values for interaction >0.40).

Discussion

In the Boston Birth Cohort—a large cohort of predominantly urban, racially and ethnically diverse women recruited at delivery—we found that 54.5% of women gained excess weight during pregnancy, according to the 2009 IOM recommended GWG categories. Excess GWG was associated with 65% increased odds of preeclampsia, 68% increased odds of GDM, and a nonsignificant 14% increased odds of having cesarean delivery. Stratified analyses and tests for interaction showed that the associations between GWG and pregnancy complications did not differ by race or ethnicity or prepregnancy BMI categories, including when we compared GWG among those with class I versus class II/III obesity.

Overall, the prevalence of excessive GWG in our study (54.5%) was higher than the prevalence reported nationally (47.2%) and in the state of Massachusetts (49.3%) based on birth certificate data.[21] Our findings on GWG and pregnancy complications in our prebirth cohort are largely consistent with previous studies, which showed significant associations between excessive GWG and adverse perinatal outcomes.[5,21–25] The strong association of excessive GWG with

Table 2. Odds Ratios (and 95% Confidence Interval) of Pregnancy Complications Associated with Gestational Weight Gain

	Preeclampsia[a]	*GDM*[b]	*Cesarean delivery*[a]
IOM GWG categories			
Inadequate GWG	0.84 (0.59–1.20)	0.96 (0.62–1.48)	0.73 (0.60–0.87)
Adequate GWG	1 (ref)	1 (ref)	1 (ref)
Excessive GWG	1.65 (1.27–2.14)	1.68 (1.15–2.46)	1.14 (0.99–1.31)
p for trend	<0.001	0.001	<0.001
Continuous GWG			
Per 5 kg weight increase	1.38 (1.28–1.47)	1.13 (0.99–1.28)	1.17 (1.12–1.23)

Logistic regression model adjusted for age at delivery, race/ethnicity, education level, any smoking during pregnancy, any drinking during pregnancy, marital status, and prepregnancy BMI; Continuous GWG analysis additionally adjusted for gestational week at delivery.

[a]The analyses of preeclampsia and cesarean delivery used total GWG over pregnancy ($N = 5{,}568$).

[b]The analysis of GDM used GWG until GDM screening period (24–28 gestational week), which is available among a subpopulation of 2,544 women.

IOM, Institute of Medicine.

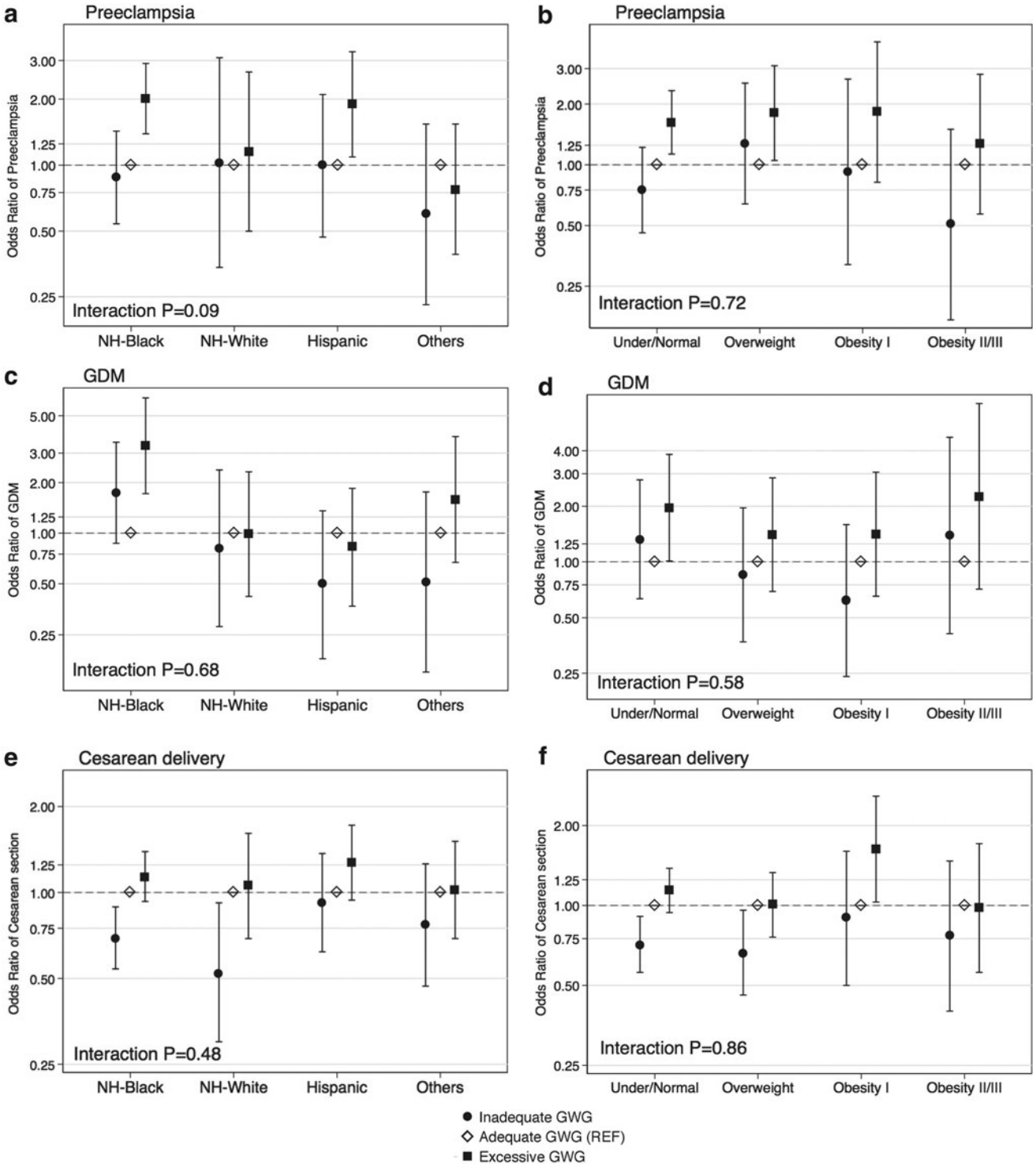

FIG. 3. Associations[a] of IOM GWG categories with preeclampsia,[b] GDM,[c] and cesarean delivery[b] according to prepregnancy BMI categories,[d] and racial and ethnic groups[e] in the Boston Birth Cohort. **(a)** The association of GWG categories with preeclampsia stratified by race/ethnicity; **(b)** the association of GWG categories with preeclampsia stratified by prepregnancy BMI; **(c)** the association of GWG categories with GDM stratified by race/ethnicity; **(d)** the association of GWG categories with GDM stratified prepregnancy BMI; **(e)** The association of GWG categories with cesarean delivery stratified by race/ethnicity; **(f)** the association of GWG categories with cesarean delivery stratified by prepregnancy BMI. [a]In addition to race/ethnicity (prepregnancy BMI) stratification, logistic regression model adjusted for age at delivery, education level, any smoking during pregnancy, any drinking during pregnancy, marital status, and prepregnancy BMI (race/ethnicity). Adequate weight gain is the reference group. [b]Models for preeclampsia and cesarean delivery used data from the whole study population ($N = 5{,}568$); [c]Models for GDM used data from women who had both GWG measurement until GDM diagnosis period and total GWG measurement over pregnancy available ($N = 2{,}544$). [d]The x-axis represents the four prepregnancy BMI categories that are defined as normal/underweight (BMI ≤ 25 kg/m^2), overweight ($25 <$ BMI ≤ 30 kg/m^2), obesity class I ($30 <$ BMI ≤ 35 kg/m^2), and obesity class II/III (BMI > 35 kg/m^2). [e]NH, non-Hispanic.

preeclampsia and GDM observed in our study likely involves mechanisms related to endothelial damage and insulin resistance, respectively.[26–28] Lifestyle intervention trials that control excessive GWG have shown risk reduction for preeclampsia, ranging from 35% to 78%,[29,30] but a less marked, nonsignificant reduction for GDM.[31] Consistent with our findings, a recent meta-analysis of 23 studies[9] ($n = 1{,}309{,}136$ women) evaluating associations between GWG above or below the IOM guidelines and maternal and infant outcomes, showed an excess risk of cesarean delivery associated with excessive GWG (OR = 1.30, 95% CI 1.25–1.35) but not GDM. This meta-analysis also showed an increased risk for preterm birth and small for gestational age infants that was associated with inadequate weight gain, but it did not assess preeclampsia as an outcome.[9]

An important contribution of our study was the examination of GWG in relation to pregnancy complications, by race and ethnicity. Consistent with other studies,[1–3] in our cohort, non-Hispanic black women had higher rates of prepregnancy obesity and gained less gestational weight than other racial/ethnic groups.[1–3,32,33] Despite these differences, the magnitude of the association between GWG and pregnancy complications was fairly consistent across all racial and ethnic groups in our study. Bodnar et al. also showed no difference in the associations of GWG categories with adverse birth outcomes in African American compared with white women.[34] These findings confirm the applicability of the IOM GWG recommendations for all women, regardless of race or ethnicity. Importantly, an implication of our findings will be to support clinicians in counseling all pregnant women using the IOM weight gain goals.

Another important aim of our study was to evaluate the association of IOMs GWG categories with pregnancy outcomes according to prepregnancy BMI categories, particularly class I versus class II or III obesity. As expected, GWG was less among women who started with higher prepregnancy BMI. The proportion of women who met criteria for excessive GWG was similar across prepregnancy BMI groups, as has been shown in previous studies.[21,35,36] Our stratified analyses by prepregnancy BMI stratified did not show differences in the associations between GWG category and pregnancy complications by obesity class I versus obesity class II/III. Our sample size of obese women with gestational weight loss or maintenance was too small ($n = 56$) to draw conclusions about the risk of pregnancy complications in this unique subgroup. However, compared with women with class II/III obesity and adequate GWG, women with class II/III obesity with inadequate GWG had a nonstatistically significant decrease in odds of preeclampsia (OR = 0.51, 95% CI 0.17–1.50). The direction of this association is consistent with other studies that have shown a benefit of restricted weight gain among obese women on the risk of preeclampsia.[15,37,38] Yet, any potential protective association of restricted GWG on preeclampsia among morbidly obese women should be put into the context of excessive risks of preterm birth and small for gestational age infants in women with inadequate weight gain and higher prepregnancy BMI, finding reported in a recent meta-analysis by Goldstein et al.[9]

Although it has many strengths, our study has several limitations worth noting. First, our GWG may not have been precisely measured due to either measurement error or recall bias.[19,39,40] To optimize GWG accuracy, we prioritized the EMR over self-report weight measurements in the GWG calculation. Among those who had both EMR and self-reported GWG measurements we showed the Pearson correlation between self-reported prepregnancy weight and first trimester EMR weight was 0.93 ($n = 2{,}439$). The Pearson correlation between self-reported GWG and GWG abstracted from the EMR was 0.70 ($n = 2{,}293$). To further test the robustness of our results against combined weight measures, we performed a sensitivity analysis restricted to women with GWG abstracted from the EMR. The results of this sensitivity analysis were consistent with the main analyses. We acknowledge that although all the study participants were from a single medical center, the prenatal weights recorded in the EMR may have biases, such as recording errors, problems with scale calibration, and scale differences from time to time. Therefore, caution is needed when generalizing our findings to a different setting and EMR. Second, we did not have the exact timing of the diagnosis of preeclampsia and thus we were not able to examine the association of GWG up to preeclampsia onset. However, we believe that, instead of accelerating, the rate of GWG maintained or decreased after preeclampsia diagnosis, which would have made our estimates more conservative. Future studies should examine GWG up until the exact time of preeclampsia diagnosis to assess whether estimates differ. Finally, due to the nature of observational study design, we cannot determine causality between GWG and pregnancy complications, nor can we exclude the possibility of measured or unmeasured residual confounders, such as physical activity and diet during pregnancy.

In conclusion, we found that over half of the women in our multiethnic U.S. cohort gained excess weight during pregnancy, and this was most marked for non-Hispanic white women. Excessive GWG increased the odds of developing preeclampsia and GDM and, to a lesser non-significant extent, the odds of cesarean delivery. Furthermore, our findings support the applicability and importance of the IOMs recommendations for GWG across all racial and ethnic minority populations and among higher prepregnancy BMI categories (*e.g.*, obesity class II/III). Given the consistency of our findings with others, there is urgent need to develop programs, policies, and interventions to ensure optimal GWG, particularly among minority racial and ethnic groups, with the ultimate aim of reducing the disparities associated with maternal obesity, GWG, and pregnancy complications in this segment of predominantly urban, low income, minority U.S. population.

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Authors' Contributions

X.W. had full access to all the data in the original study and took responsibility for the integrity of the data. N.T.M., Z.Z., W.L.B., and X.W. drafted the article and designed the analysis plan of this study. Z.Z. conducted the data analysis and is responsible for the accuracy of the results. All the authors were involved in writing and revision of the article and had approved this version for submission.

Author Disclosure Statement

No competing financial interests exist.

References

1. Flegal KM, Kruszon-Moran D, Carroll MD, Fryar CD, Ogden CL. Trends in obesity among adults in the United States, 2005 to 2014. JAMA 2016;315:2284–2291.
2. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the united states, 2011–2012. JAMA 2014;311:806–814.
3. Moaddab A, Dildy GA, Brown HL, et al. Health care disparity and state-specific pregnancy-related mortality in the United States, 2005–2014. Obstet Gynecol 2016;128:869–875.
4. Johnson J, Clifton RG, Roberts JM, et al. Pregnancy outcomes with weight gain above or below the 2009 Institute of Medicine guidelines. Obstet Gynecol 2013;121:969–975.
5. Truong YN, Yee LM, Caughey AB, Cheng YW. Weight gain in pregnancy: Does the Institute of Medicine have it right? Am J Obstet Gynecol 2015;212:362.e361–362.e368.
6. Macdonald-Wallis C, Tilling K, Fraser A, Nelson SM, Lawlor DA. Gestational weight gain as a risk factor for hypertensive disorders of pregnancy. Am J Obstet Gynecol 2013;209:327.e321–327.e317.
7. Yee LM, Caughey AB, Cheng YW. Association between gestational weight gain and perinatal outcomes in women with chronic hypertension. Am J Obstet Gynecol 2017;217:348.e1–348.e9.
8. Hedderson MM, Gunderson EP, Ferrara A. Gestational weight gain and risk of gestational diabetes mellitus. Obstet Gynecol 2010;115:597–604.
9. Goldstein RF, Abell SK, Ranasinha S, et al. Association of gestational weight gain with maternal and infant outcomes: A systematic review and meta-analysis. JAMA 2017;317:2207–2225.
10. IOM, NRC. Weight Gain During Pregnancy: Reexamining the Guidelines. Washington, DC: National Academies Press, 2009.
11. Hrolfsdottir L, Rytter D, Olsen SF, et al. Gestational weight gain in normal weight women and offspring cardiometabolic risk factors at 20 years of age. Int J Obes 2015;39:671–676.
12. Wolf M, Shah A, Jimenez-Kimble R, Sauk J, Ecker JL, Thadhani R. Differential risk of hypertensive disorders of pregnancy among Hispanic women. J Am Soc Nephrol 2004;15:1330–1338.
13. Marshall NE, Guild C, Cheng YW, Caughey AB, Halloran DR. Racial disparities in pregnancy outcomes in obese women. J Matern Fetal Neonatal Med 2014;27:122–126.
14. Snowden JM, Mission JF, Marshall NE, et al. The Impact of maternal obesity and race/ethnicity on perinatal outcomes: Independent and joint effects. Obesity (Silver Spring) 2016;24:1590–1598.
15. Kiel DW, Dodson EA, Artal R, Boehmer TK, Leet TL. Gestational weight gain and pregnancy outcomes in obese women: How much is enough? Obstet Gynecol 2007;110:752–758.
16. Wang X, Zuckerman B, Pearson C, et al. Maternal cigarette smoking, metabolic gene polymorphism, and infant birth weight. JAMA 2002;287:195–202.
17. Wang G, Divall S, Radovick S, et al. Preterm birth and random plasma insulin levels at birth and in early childhood. JAMA 2014;311:587–596.
18. NHBPEP. Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy. Am J Obstet Gynecol 2000;183:s1–s22.
19. Gilmore LA, Redman LM. Weight gain in pregnancy and application of the 2009 IOM guidelines: Toward a uniform approach. Obesity (Silver Spring) 2015;23:507–511.
20. Greenland S, Robins JM. Identifiability, exchangeability, and epidemiological confounding. Int J Epidemiol 1986;15:413–419.
21. Deputy NP, Sharma AJ, Kim SY, Hinkle SN. Prevalence and characteristics associated with gestational weight gain adequacy. Obstet Gynecol 2015;125:773–781.
22. Masho SW, Urban P, Cha S, Ramus R. Body mass index, weight gain, and hypertensive disorders in pregnancy. Am J Hypertens 2016;29:763–771.
23. Gibson KS, Waters TP, Catalano PM. Maternal weight gain in women who develop gestational diabetes mellitus. Obstet Gynecol 2012;119:560–565.
24. Xu H, Barnes GT, Yang Q, et al. Chronic inflammation in fat plays a crucial role in the development of obesity-related insulin resistance. J Clin Invest 2003;112:1821–1830.
25. Pantham P, Aye ILMH, Powell TL. Inflammation in maternal obesity and gestational diabetes mellitus. Placenta 2015;36:709–715.
26. Ramsay JE, Ferrell WR, Crawford L, Wallace AM, Greer IA, Sattar N. Maternal obesity is associated with dysregulation of metabolic, vascular, and inflammatory pathways. J Clin Endocrinol Metab 2002;87:4231–4237.
27. Sattar N, Gaw A, Packard CJ, Greer IA. Potential pathogenic roles of aberrant lipoprotein and fatty acid metabolism in pre-eclampsia. Br J Obstet Gynaecol 1996;103:614–620.
28. Hendler I, Blackwell SC, Mehta SH, et al. The levels of leptin, adiponectin, and resistin in normal weight, overweight, and obese pregnant women with and without preeclampsia. Am J Obstet Gynecol 2005;193(3 Suppl):979–983.
29. Barakat R, Pelaez M, Cordero Y, et al. Exercise during pregnancy protects against hypertension and macrosomia: Randomized clinical trial. Am J Obstet Gynecol 2016;214:649.e641–649.e648.
30. Sagedal L, Øverby N, Bere E, et al. Lifestyle intervention to limit gestational weight gain: The Norwegian Fit for

Delivery randomised controlled trial. Br J Obstet Gynaecol 2017;124:97–109.
31. Harrison CL, Lombard CB, Strauss BJ, Teede HJ. Optimizing healthy gestational weight gain in women at high risk of gestational diabetes: A randomized controlled trial. Obesity (Silver Spring) 2013;21:904–909.
32. Chu SY, Callaghan WM, Bish CL, D'Angelo D. Gestational weight gain by body mass index among US women delivering live births, 2004–2005: Fueling future obesity. Am J Obstet Gynecol 2009;200:271.e271–271.e277.
33. Kim SY, Sharma AJ, Sappenfield W, Wilson HG, Salihu HM. Association of maternal body mass index, excessive weight gain, and gestational diabetes mellitus with large-for-gestational-age births. Obstet Gynecol 2014;123:737.
34. Bodnar LM, Hutcheon JA, Platt RW, Himes KP, Simhan HN, Abrams B. Should gestational weight gain recommendations be tailored by maternal characteristics? Am J Epidemiol 2011;174:136–146.
35. Deputy NP, Sharma AJ, Kim SY. Gestational weight gain—United States, 2012 and 2013. MMWR Morb Mortal Wkly Rep 2015;64:1215–1220.
36. Liu L, Hong Z, Zhang L. Associations of prepregnancy body mass index and gestational weight gain with pregnancy outcomes in nulliparous women delivering single live babies. Sci Rep 2015;5:12863.
37. Cedergren M. Effects of gestational weight gain and body mass index on obstetric outcome in Sweden. Int J Gynecol Obstet 2006;93:269–274.
38. Nohr EA, Vaeth M, Baker JL, Sorensen TI, Olsen J, Rasmussen KM. Combined associations of prepregnancy body mass index and gestational weight gain with the outcome of pregnancy. Am J Clin Nutr 2008;87:1750–1759.
39. Wright CS, Weiner M, Localio R, Song L, Chen P, Rubin D. Misreport of gestational weight gain (GWG) in birth certificate data. Matern Child Health J 2012;16:197–202.
40. Bodnar LM, Abrams B, Bertolet M, et al. Validity of birth certificatederived maternal weight data. Paediatr Perinat Epidemiol 2014;28:203–212.

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