Severe obesity, gestational weight gain, and adverse birth outcomes[1–3]

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ABSTRACT

Background: The 2009 Institute of Medicine (IOM) Committee to Reevaluate Gestational Weight Gain Guidelines concluded that there were too few data to inform weight-gain guidelines by obesity severity. Therefore, the committee recommended a single range, 5–9 kg at term, for all obese women.

Objective: We explored associations between gestational weight gain and small-for-gestational-age (SGA) births, large-for-gestational-age (LGA) births, spontaneous preterm births (sPTBs), and medically indicated preterm births (iPTBs) among obese women who were stratified by severity of obesity.

Design: We studied a cohort of singleton, live-born infants without congenital anomalies born to obesity class 1 (prepregnancy body mass index [BMI (in kg/m^2)]: 30–34.9; n = 3254), class 2 (BMI: 35–39.9; n = 1451), and class 3 (BMI: ≥40; n = 845) mothers. We defined the adequacy of gestational weight gain as the ratio of observed weight gain to IOM-recommended gestational weight gain.

Results: The prevalence of excessive gestational weight gain declined, and weight loss increased, as obesity became more severe. Generally, weight loss was associated with an elevated risk of SGA, iPTB, and sPTB, and a high weight gain tended to increase the risk of LGA and iPTB. Weight gains associated with probabilities of SGA and LGA of ≤10% and a minimal risk of iPTB and sPTB were as follows: 9.1–13.5 kg (obesity class 1), 5.0–9 kg (obesity class 2), 2.2 to <5.0 kg (obesity class 3 white women), and <2.2 kg (obesity class 3 black women).

Conclusion: These data suggest that the range of gestational weight gain to balance risks of SGA, LGA, sPTB, and iPTB may vary by severity of obesity. *Am J Clin Nutr* 2010;91:1642–8.

INTRODUCTION

Severe obesity is rising rapidly in the United States (1, 2). Among US women of childbearing age, the rates of class 1 obesity (body mass index [BMI (in kg/m^2)]: 30–34.9) and class 2 obesity (BMI: 35–39.9) have doubled in the past 30 y, whereas the rate of class 3 obesity (BMI ≥40) has tripled (3). Once rare (1.7% of reproductive-aged US women in 1976–1980), severe obesity now affects 6.5% of reproductive-aged US women (3). In pregnant women, health risks increase as obesity becomes more severe. Women classified as severely obese at conception have substantially higher risks of infant mortality, stillbirth, maternal mortality, hypertensive disorders of pregnancy, congenital malformations, gestational diabetes, large-for-gestational-age (LGA) or macrosomic infants, and cesarean delivery than women with class 1 obesity (4–10).

Gestational weight gain may modify the effects of severe obesity on adverse outcomes of pregnancy. However, little is known about the optimal range of weight gain for severely obese women. In May 2009, the Institute of Medicine (IOM) revised weight-gain recommendations for all pregnant women, including those classified as obese according to their prepregnancy BMI (3). In changing the recommendation of total gestational weight gain for obese women from "at least 6.8 kg (15 lb)" for a woman with a pregravid BMI >29 in the 1990 recommendations (11) to "5 to 9 kg (11 to 20 lb) and 0.22 kg/week in the 2nd and 3rd trimesters" for a woman with a pregravid BMI ≥30 in the 2009 guidelines (3), the committee slightly lowered the recommended range of weight gain and placed an upper bound on it. Despite the rising prevalence of severe obesity, the committee lacked sufficient data to inform guidelines specific to each class of obesity. At the time of the committee's deliberation, only one study (12) and work performed by Nohr (3) attempted to identify optimal weight-gain ranges for severely obese women. Each analysis had limitations in terms of the population studied, the sample size of severely obese women, the manner in which weight-gain adequacy was studied, or the outcomes (both maternal and infant) tested. Therefore, the IOM report called for more research on severely obese women to provide a basis for determining optimal gestational weight-gain guidelines. In an effort to fill this gap, we sought to explore associations between gestational weight-gain and birth outcomes among obese women who were stratified by severity of obesity by using a large cohort of deliveries from a large US maternity hospital.

SUBJECTS AND METHODS

Data were collected from the Magee Obstetric Medical and Infant (MOMI) database. The MOMI database, established in 1995, routinely collects comprehensive maternal, fetal, and

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neonatal outcomes from electronic and medical record data on all women delivering at Magee-Womens Hospital, University of Pittsburgh Medical Center, Pittsburgh, PA (13). The database is surveyed periodically to maintain its accuracy by direct comparison at random with patient charts and also by examining frequencies for variables that contain data outliers on download, which, once identified, were verified or corrected by means of a medical chart review. Personal identifying information in the database was eliminated to ensure confidentiality. The University of Pittsburgh Institutional Review Board approved this study.

There were 47,445 deliveries of singleton, live-born infants without congenital anomalies from 20 to 42 wk gestation at Magee-Womens Hospital from 1 January 2003 to 31 December 2008. We excluded deliveries with values for gestational weight gain that were unlikely to be plausible [weight loss ≥18.2 kg (≥40 lb) and weight gain >36.4 kg (>80 lb); *n* = 209] and deliveries with missing data on maternal prepregnancy BMI (*n* = 10,775), weight at delivery (*n* = 3140), or one of the covariates in the final model (*n* = 50). On further inspection into the missing prepregnancy BMI data, we discovered that a systems-level change in the process used to populate prepregnancy weight in the electronic medical records from 2006 to 2008 at Magee-Womens Hospital resulted in 75% of the missing data. Women with missing data compared with women with complete data were more likely to be non-Hispanic blacks (23% compared with 17%), unmarried (43% compared with 35%), have a high school education or less (36% compared with 28%), and to have delivered preterm (13% compared with 9%). There were no differences in parity, age, or smoking status (data not shown).

Exposures

Prepregnancy weight was abstracted from prenatal records on the basis of a self-report at the first prenatal visit. Recalled height was abstracted from the mother's worksheet, a form completed by women immediately after delivery for the birth record. Of the 33,271 births eligible for this study, 4.3% of mothers were underweight (prepregnancy BMI: <18.5), 57.0% of mothers were normal weight (prepregnancy BMI: 18.5–24.9), 22.0% of mothers were overweight (prepregnancy BMI: 25.0–29.9), and 16.7% of mothers were obese (prepregnancy BMI: ≥30) (14). Women whose prepregnancy BMI met the definition of obesity were further divided into grades of obesity: obesity class 1 (30–34.9), class 2 (35–39.9) and class 3 (≥40). For this analysis, we restricted the sample to all obese mothers (*n* = 5550) and used the 18,950 normal-weight women as a comparison group in descriptive analyses.

We defined the adequacy of gestational weight gain as a ratio of observed gestational weight gain to expected (recommended) gestational weight gain at the gestational age of delivery multiplied by 100, as described previously (15, 16). Observed weight gain was calculated as the difference between weight at delivery and the reported prepregnancy weight, where weight at delivery was ascertained from maternal medical records as the last measured weight at or before admission to labor and delivery. Expected gestational weight gain was defined as 100% of the 2009 IOM recommendations at the gestational age of delivery (3). We used the following equation: Expected gestational weight gain = recommended first-trimester total weight gain + (gestational age at weight measurement at or before delivery – 13 wk) × recommended rate of gain in second and third trimesters.

For example, the expected weight gain for an obese woman delivering at 40 wk gestation is 6.4 kg (0.5 kg in the first trimester + 0.22 kg/wk × 27 wk).

We classified the percentage of weight-gain recommendations met as inadequate, adequate, or excessive. In recognition that the IOM recommended a range of total gestational weight gain for each prepregnancy BMI group, we divided the lower and upper limits of the recommended weight-gain range by the expected weight gain at 40 wk gestation for each BMI group and multiplied by 100 to calculate corresponding ranges of recommended percentage of expected weight gain. For example, the range of recommended weight gain for an obese woman is 5–9 kg. Thus, the ranges of recommended weight gain were 5/6.4 = 78% of IOM recommendations and 9/6.4 = 140% of IOM recommendations. We used these ranges as the basis for the following categories of weight-gain adequacy: inadequate (less than the lower cutoff of recommendations), adequate (within recommended range), or excessive (greater than the upper cutoff of recommendations). We also categorized the adequacy of weight gain for obese women more finely into 8 groups that approximated given weight-gain ranges at 40 wk gestation as follows: <0% (weight loss), 0% to <35% (0 to <2.2 kg), 35% to 76% (2.2 to <5.0 kg), 77% to 140% [5.0 to 9.0 kg, which is the range currently recommended by the IOM (3)], 141% to 210% (9.1 to <13.6 kg), 211% to 281% (13.6 to <18.2 kg), 282% to 351% (18.2 to <22.7 kg), and ≥352% (≥22.7 kg).

Outcomes

We studied birth outcomes that have been consistently associated with gestational weight gain in past reports (3, 17): small-for-gestational-age birth (SGA), LGA birth, spontaneous preterm birth (sPTB), and medically indicated preterm birth (iPTB). Data on birth outcomes were obtained from medical records. Gestational age was ascertained from the birth attendant's final estimate of gestational length on the basis of all available perinatal factors and assessments, including ultrasound reports. Although the database did not indicate how gestational age was specifically determined in each patient, 78% of patients that deliver at Magee-Womens Hospital have a dating ultrasound by 20 wk gestation (Magee-Womens Hospital unpublished quality assurance data, 2006). Preterm birth was defined as a delivery occurring at completed 20 to <37 wk gestation. sPTB was defined as a preterm delivery occurring after a preterm labor with intact membranes or a preterm prelabor rupture of the fetal membranes. iPTB were the remainder of preterm deliveries. No preterm deliveries were elected. SGA and LGA were defined as live born infants that were <10th percentile or >90th percentile, respectively, of ultrasound-based intrauterine fetal weight standards (18).

Covariates

At admission or immediately after delivery, mothers self-reported their height, race-ethnicity (non-Hispanic white, non-Hispanic black, or other), education (less than high school or equivalent, high school graduate, some college, or college graduate), marital status (married or unmarried), smoking status during pregnancy (smoker or nonsmoker), and use of the Special

Supplemental Nutrition Program for Women, Infants, and Children (WIC) during pregnancy (yes or no). Data were also abstracted from medical records on age, parity (0 or ≥1), principal source of payment at delivery (private insurance, Medicaid, or other), and type of provider (Magee outpatient resident clinic or Magee-affiliated private practice).

Statistical analyses

The chi-square test and analysis of variance were used to test for differences in maternal characteristics and pregnancy outcomes by BMI category. We fit separate multivariable logistic regression models for each class of obesity by using generalized estimating equations to assess the independent association between gestational weight-gain adequacy and each outcome of interest. Generalized estimating equations were used to account for repeated pregnancies among 578 women in the data set. We specified weight gain as a restricted cubic spline in all models to ensure comparability and to allow for flexibility in estimating curvilinear relations. We chose 100% of the IOM recommended weight gain as the referent value in these models (equivalent to 6.4 kg at 40 wk gestation or 4.7 kg at 32 wk gestation). The contrasting values chosen were 50%, 200%, and 300% of the IOM-recommended weight gain.

Potential confounders were race-ethnicity, age, education, marital status, smoking status, parity, height, WIC participation, payment source, and provider type. Race-ethnicity, smoking, parity, height, and education were the only covariates whose exclusion from any of the models resulted in a >10% change in the weight-gain odds ratio. Therefore, these covariates were retained in all models to ensure comparability. To fill a research gap identified by the 2009 IOM committee on the extent to which optimal weight gain differs by race and parity (3), we studied these variables as effect modifiers in our analysis by using a likelihood ratio test ($\alpha = 0.10$). With the use of the final multivariable logistic model for each outcome and obesity class, we generated confounder-adjusted predicted probabilities and plotted these for each weight-gain category. Stata Software, version 10 (StataCorp LP, College Station, TX) was used for all analyses.

RESULTS

Of all eligible mothers, 9.8% ($n = 3254$) of the women were classified with class 1 obesity, 4.4% ($n = 1451$) of the women were classified with class 2 obesity, and 2.5% ($n = 845$) of the women were classified with class 3 obesity. As obesity became more severe, there was an increase in the prevalence of non-Hispanic black race, low education, older maternal age, and participation in the WIC (**Table 1**). Obese mothers also tended to be unmarried, multiparous, nonsmokers, and Medicaid recipients compared with normal-weight women. Compared with normal-weight women, obese women had a lower prevalence of sPTB and a greater proportion of LGA and iPTB. As obesity became more severe, the mean absolute gestational weight gain declined, and the percentage of women gaining weight inadequately rose (Table 1). Overall, <20% of all obese women gained within the 2009 IOM-recommended weight-gain range.

A majority of women in obesity classes 1 (25%), 2 (22.4%), and 3 (20.8%) gained 141% to <211% of the 2009 IOM recommendations (the equivalent of 9.2 to <13.6 kg at 40 wk; **Figure 1**). As obesity became more severe, excessive weight gains declined and weight loss increased. Among women who lost weight, the median (interquartile range) weight loss was −2.7 kg (−5.5, −1.4 kg) for class 1 obesity, −3.6 kg (−6.8, −1.8 kg) for class 2 obesity, and −3.6 kg (−6.4, −2.3 kg) for class 3 obesity.

The adjusted risk of SGA declined as weight gain increased among class 1 obese mothers, but among class 2 obese mothers, very high weight gain was not protective (**Table 2**). For class 3 obesity, the association between weight gain and risk of SGA was modified by maternal race-ethnicity ($P < 0.0001$). Among non-Hispanic white women in obesity class 3, there was a negative relation between weight-gain adequacy and SGA risk, whereas among non-Hispanic black obese class 3 women, the association was not significant ($P = 0.17$). Neither race-ethnicity nor parity modified any of the other associations studied. In all obesity classes, the risk of LGA increased monotonically with increasing weight gain, and high weight gain was related to an increased risk of iPTB. Low weight gains were associated with an increased risk of sPTB in class 1 and 2 obesity. We could not study sPTB among obesity class 3 women because the sample size of 30 cases produced unstable estimates.

When we plotted the adjusted predicted probabilities for each of the 4 birth outcomes across weight-gain categories for class 1 obesity (**Figure 2**) and class 2 obesity (**Figure 3**) in women, the tradeoffs in birth outcome with varying weight gain were evident. With weight loss, the risk of SGA, iPTB, and sPTB were elevated, and the risk of LGA was low. High weight gain was associated with a lower risk of SGA but a higher prevalence of LGA. At very high weight gains (≥352% of IOM), the risk of iPTB increased for class 1 and 2 women, and the risk of SGA and sPTB increased for class 2 women. For obesity class 3 women, risks of LGA and iPTB were lowest with weight loss and low weight gain (**Figure 4**). However, among white women, weight loss was also associated with an elevated risk of SGA, which declined as weight gain increased.

For obesity class 1 women, a weight gain equivalent to 9.1–13.5 kg was associated with probabilities of SGA and LGA ≤10% and probabilities of sPTB and iPTB that were at or close to the nadir of the risk curve. For these criteria to be met among obesity class 2 mothers and obesity class 3 white mothers, weight gains ranged from 2.2 to 9 kg and 2.2 to <5 kg, respectively. Weight gain <2.2 kg or weight loss for black women was associated with the lowest risk of LGA and iPTB.

DISCUSSION

The 2009 IOM Committee to Reevaluate Gestational Weight Gain Guidelines (3) concluded that there were too few data to inform weight-gain guidelines by obesity severity. Therefore, they recommended a single range, 5–9 kg at term, for all obese women. Our results suggest that gestational weight-gain ranges that balance risks of SGA, LGA, sPTB, and iPTB may vary by severity of obesity. Weight gains that were associated with probabilities of SGA and LGA ≤10% and a minimal risk of PTB were 9.1–13.5 kg and 2.2–9 kg for obesity class 1 and 2 mothers, respectively. For class 3 obese mothers, weight gains that met these criteria were 2.2 to <5 kg for white women and <2.2 kg for black women.

TABLE 1
Characteristics of the population[1]

	Normal weight (n = 18,950)	Obesity class 1 (n = 3254)	Obesity class 2 (n = 1451)	Obesity class 3 (n = 845)
Maternal characteristics				
Maternal race-ethnicity (%)[2]				
Non-Hispanic white	74.1	73.4	71.3	66.0
Non-Hispanic black	15.9	24.6	27.7	32.8
Other	10.0	2.0	1.0	1.2
Maternal education (%)[2]				
Less than high school	14.4	7.7	7.4	7.3
High school or equivalent	25.3	24.8	26.8	28.3
Some college	21.1	29.8	32.0	33.9
College graduate	39.2	37.7	33.8	30.5
Maternal age (%)[2]				
<20 y	11.1	4.4	3.0	2.3
20–29 y	25.9	17.9	18.9	17.8
25–29 y	25.2	28.2	29.2	27.2
30–34 y	24.5	29.3	27.1	29.2
≥35 y	13.3	20.3	21.9	23.6
Marital status (%)[2]				
Unmarried	47.8	38.6	42.4	40.5
Married	52.2	61.4	57.6	59.5
Parity (%)				
0	52.9	38.9	38.5	39.8
≥1	47.1	61.1	61.5	60.2
Smoking status (%)[2]				
Smokers	21.7	14.8	16.2	14.2
Nonsmokers	78.4	85.2	83.8	85.8
Provider (%)[2]				
Magee-affiliated private practice	75.3	79.1	78.2	77.4
Magee resident clinic	24.7	20.9	21.9	22.6
Source of payment (%)[2]				
Medicaid	46.2	39.6	43.5	41.5
Private insurance/self-pay	53.8	60.4	56.5	58.5
Participation in the WIC (%)[2]				
No	64.2	66.3	61.2	59.7
Yes	35.8	33.7	38.8	40.3
Outcomes (%)				
Small-for-gestational-age birth	14.5	9.1	8.0	9.6
Large-for-gestational-age birth[2]	3.6	13.5	13.9	15.3
Indicated preterm birth <37 wk[2]	4.7	6.2	5.6	7.5
Spontaneous preterm birth <37 wk[3]	7.3	5.3	3.6	3.8
Gestational weight gain (kg)[4]	15.4 ± 5.4[5]	12.5 ± 5.4	10.0 ± 8.1	7.7 ± 8.5
Adequacy of gestational weight gain (%)[2]				
Inadequate (<IOM range)	24.4	14.3	24.6	37.9
Adequate (within IOM range)	43.4	13.4	18.8	16.7
Excessive (>IOM range)	32.2	72.3	56.7	45.4

[1] Normal weight, BMI (in kg/m^2) = 18.5–24.9; obesity class 1, BMI = 30–34.9; obesity class 2, BMI = 35–39.9; obesity class 3, BMI = ≥40. IOM, Institute of Medicine; WIC, Special Supplemental Nutrition Program for Women, Infants, and Children.
[2] $P < 0.001$ across groups (chi-square test).
[3] $P < 0.05$ across groups (chi-square test).
[4] $P < 0.001$ across groups (ANOVA).
[5] Mean ± SD (all such values).

Our study was not complete in terms of outcomes related to weight gain. We examined SGA, LGA, iPTB, and sPTB because these outcomes were rigorously defined in our delivery database and were consistently associated with gestational weight gain (3, 17). Maternal postpartum weight retention, childhood obesity, birth complications, and long-term infant cognitive performance are some of the outcomes that require study in other populations. The inclusion of outcomes that have positive, linear associations with gestational weight gain, including maternal postpartum weight retention and childhood obesity (3, 17, 19–21) may drive recommended weight gains down, whereas the inclusion of child cognitive function may drive weight gains upwards. There is concern that fetal brain function may be negatively affected by maternal ketonemia (3, 22, 23), which can result from fasting during pregnancy (24–26) among women attempting weight loss. However, studies are lacking in direct associations between low maternal weight gain or weight loss and child cognition, and research in this area is greatly needed.

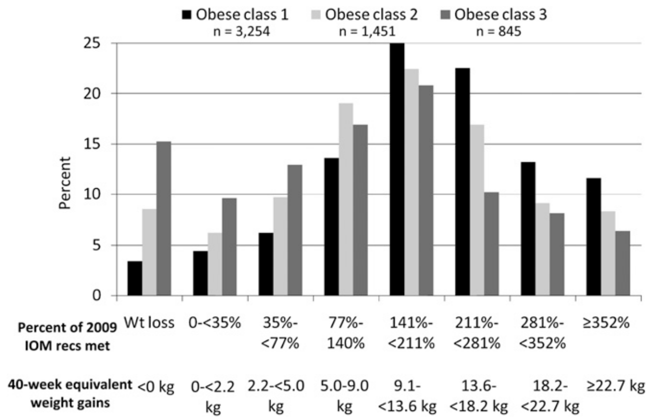

FIGURE 1. Distribution of gestational weight (Wt)-gain categories by severity of obesity. IOM recs, Institute of Medicine recommendations.

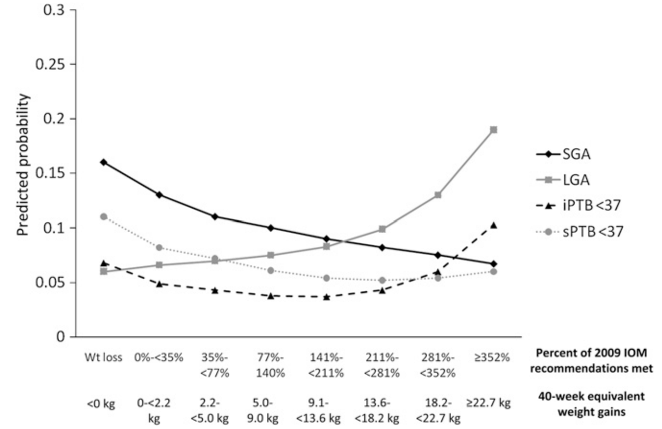

FIGURE 2. Adjusted predicted probabilities for 4 adverse birth outcomes among obesity class 1 women (*n* = 3254). The sample size in each weight (Wt)-gain category was as follows: <0%, *n* = 111; 0% to 35%, *n* = 144; 35% to <77%, *n* = 203; 77% to 140%, *n* = 443; 141% to <211%, *n* = 813; 211% to <281%, *n* = 733; 281% to <352%, *n* = 430; and ≥352%, *n* = 377. The following factors were held constant: non-Hispanic white race-ethnicity, nulliparous, nonsmoker, >12 y education, and height = 64 in. SGA, small-for-gestational-age; LGA, large-for-gestational-age; iPTB <37, medically indicated preterm birth at <37 wk gestation; sPTB <37, spontaneous preterm birth at <37 wk gestation; IOM, Institute of Medicine.

Since the publication of the 2009 IOM report (3), 3 studies were published that simultaneously examined weight gain and several maternal and infant outcomes. Oken et al (20) recommended a weight loss of 0.19 kg/wk (−7.6 kg at 40 wk) for obese women to optimize SGA, LGA, PTB, postpartum weight retention, and child obesity, regardless of how the outcomes were weighted. Studying the same outcomes as Oken et al (20) plus primary cesarean delivery, Margerison Zilko et al (19) suggested gains of <5 kg for obese women. Another group (27) recommended −15–2 kg of weight gain for obese German women to minimize SGA and LGA. However, a small sample size limited most investigators in their ability to stratify by obesity severity.

In 1 (12) of 2 published analyses (3, 12) that stratified by obesity class, Kiel et al (12) used Missouri vital records to study absolute gestational weight gain in relation to preeclampsia, cesarean delivery, SGA, and LGA in 120,170 term, singleton births to obese mothers. They defined optimal weight gain as the gain at which the SGA and LGA risk curves intersected and

TABLE 2

Associations (adjusted odds ratios with 95% CIs) of selected levels of gestational weight gain with adverse birth outcomes by severity of obesity[1]

	SGA	LGA	iPTB	sPTB
Obesity class 1 (*n* = 3254)				
Gestational weight gain adequacy				
50% of IOM recommendations	1.1 (1.1, 1.2)	0.9 (0.8, 1.0)	1.1 (1.0, 1.2)	1.2 (1.1, 1.3)
100% of IOM recommendations	Reference	Reference	Reference	Reference
200% of IOM recommendations	0.8 (0.7, 0.9)	1.2 (1.1, 1.4)	1.0 (0.8, 1.1)	0.8 (0.7, 0.9)
300% of IOM recommendations	0.7 (0.6, 0.9)	1.8 (1.5, 2.1)	1.4 (1.2, 1.8)	0.8 (0.7, 1.0)
Obesity class 2 (*n* = 1451)				
Gestational weight gain adequacy				
50% of IOM recommendations	1.2 (1.1, 1.3)	0.9 (0.8, 0.9)	1.0 (0.9, 1.2)	1.2 (1.1, 1.4)
100% of IOM recommendations	Reference	Reference	Reference	Reference
200% of IOM recommendations	0.8 (0.7, 0.9)	1.3 (1.1, 1.6)	1.0 (0.9, 1.3)	0.8 (0.7, 1.0)
300% of IOM recommendations	0.8 (0.7, 1.2)	1.7 (1.4, 2.2)	1.4 (1.1, 1.9)	1.1 (0.8, 1.6)
Obesity class 3 (*n* = 845)[2]				
Gestational weight gain adequacy				
50% of IOM recommendations	White: 1.2 (1.1, 1.3); black: 0.9 (0.8, 1.1)[3]	0.8 (0.7, 0.9)	1.0 (0.8, 1.2)	—[4]
100% of IOM recommendations	Reference	Reference	Reference	—[4]
200% of IOM recommendations	White: 0.8 (0.7, 0.9); black: 1.2 (0.9, 1.5)[3]	1.5 (1.3, 1.8)	1.2 (1.0, 1.4)	—[4]
300% of IOM recommendations	White: 0.8 (0.7, 1.2); black: 1.4 (0.9, 2.3)[3]	2.1 (1.5, 2.7)	1.8 (1.3, 2.6)	—[4]

[1] SGA, small-for-gestational-age; LGA, large-for-gestational-age; iPTB, medically indicated preterm birth; SPTB, spontaneous preterm birth; IOM, Institute of Medicine. Obesity class 1, BMI (in kg/m^2) = 30–34.9; obesity class 2, BMI = 35–39.9; obesity class 3, BMI = ≥40. Percentages of IOM recommendations are defined as follows: 50%, 3.2 kg at 40 wk; 100%, 6.4 kg at 40 wk; 200%, 12.8 kg at 40 wk; and 300%, 19.2 kg at 40 wk. All models were adjusted for smoking status, race-ethnicity, height, parity, and maternal education, with gestational weight gain adequacy specified as a restricted cubic spline.

[2] White, *n* = 568; black, *n* = 277.

[3] Adjusted odds ratios stratified by maternal race-ethnicity because race-ethnicity was a significant effect modifier ($P < 0.0001$).

[4] Not studied because there were only 30 cases.

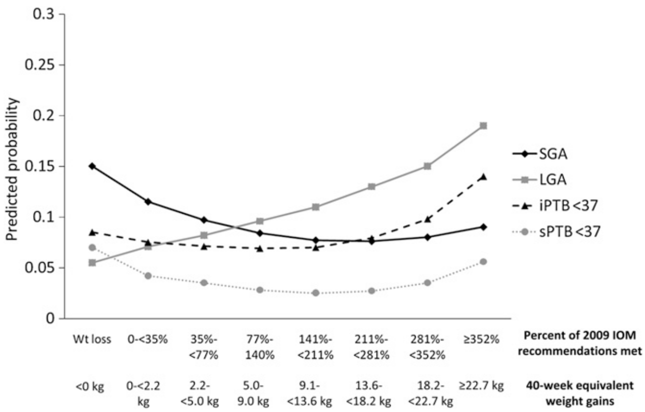

FIGURE 3. Adjusted predicted probabilities for 4 adverse outcomes among obesity class 2 women (n = 1451). The sample size in each weight (Wt)-gain category was as follows: <0%, n = 123; 0% to 35%, n = 90; 35% to <77%, n = 140; 77% to 140%, n = 276; 141% to <211%, n = 325; 211% to <281%, n = 245; 281% to <352%, n = 132; and ≥352%, n = 120. The following factors were held constant: non-Hispanic white race-ethnicity, nulliparous, nonsmoker, >12 y education, and height = 64 in. SGA, small-for-gestational-age; LGA, large-for-gestational-age; iPTB <37, medically indicated preterm birth at <37 wk gestation; sPTB <37, spontaneous preterm birth at <37 wk gestation; IOM, Institute of Medicine.

concluded that weight gains of 4.5–11.4, 0–4.1, and 0–4.1 kg may be ideal for obesity class 1, 2, and 3 mothers, respectively. In work commissioned by the IOM Committee (3), Nohr studied SGA, LGA, emergency cesarean delivery, and postpartum weight retention by weight gain among 3541 class 1 and 1273 class 2 and 3 (BMI: ≥35) obese Danish mothers and reported that weight gain of <5 kg was not associated with deleterious consequences in either group of obese mothers. It is difficult to compare across studies because of the various populations, outcomes, and definitions of gestational weight-gain adequacy. However, to the best of our knowledge, our study is the first that has examined sPTB and iPTB by weight-gain adequacy in severely obese women. Our demonstration that weight gain had different relations with sPTB compared with iPTB highlights the value of studying these outcomes separately in future work.

A striking number of severely obese women in our cohort lost weight during pregnancy. Our database lacked information on diet and physical activity patterns that led to these weight losses: ie, maternal metabolic measures that might explain possible shifts in metabolism that could cause weight loss, socioeconomic, or other stresses that might affect energy needs, food insecurity, and nutrition advice provided during prenatal care. Recent data suggest that 7% of US pregnant women in 2003 attempted weight loss (28), so it is possible that some of the obese women who lost weight in our cohort were doing so intentionally. Future research must explore the mechanisms explaining weight loss and very low weight gains in obese and severely obese mothers. However, we also recognize that there may be some bias in our results relating the severity of obesity to the degree of gestational weight gain or loss because weight change from conception to delivery is correlated to weight at conception (29).

Our study was limited to short-term fetal outcomes. Longer-term infant outcomes such as infant mortality, childhood cognitive function, and childhood obesity, as well as maternal outcomes including postpartum weight retention, are needed in weight-gain studies of severely obese mothers. Our study lacked data on the pattern of weight gain, which would help to determine in what gestational age windows weight gain is most important and also help to address the potential problem of reverse causality (ie, where poor weight gain is a result of adverse antepartum events that lead to SGA or preterm birth). We did not study gestational diabetes or preeclampsia as outcomes in our analysis because total weight gain may be a consequence of the disease, and we lacked data on weight gain up to the time of clinical manifestation of these diseases. Missing data were a concern in

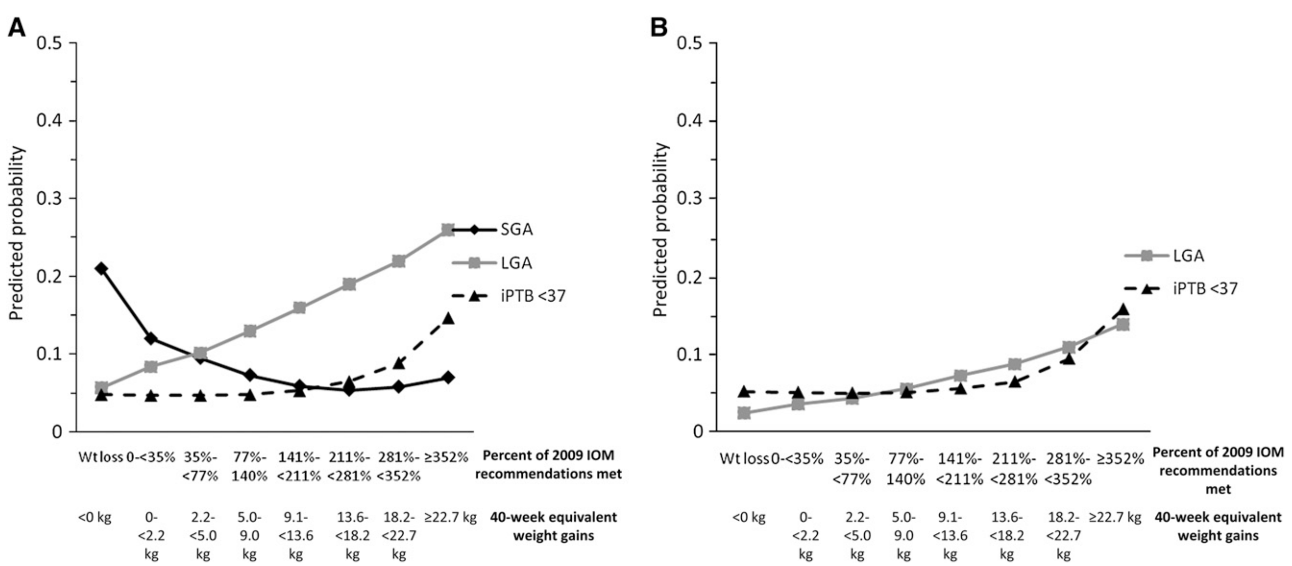

FIGURE 4. Adjusted predicted probabilities for 3 adverse outcomes among obesity class 3 white women (A; n = 568) and obesity class 3 black women (B; n = 277). The sample size in each weight (Wt)-gain category was as follows: <0% (n = 86 white, n = 42 black); 0% to 35% (n = 53 white, n = 28 black); 35% to <77% (n = 74 white, n = 35 black); 77% to 140% (n = 100 white, n = 43 black); 141% to <211% (n = 113 white, n = 63 black); 211% to <281% (n = 67 white, n = 19 black); 281% to <352% (n = 45 white, n = 23 black); and ≥352% (n = 30 white, n = 24 black). The following factors were held constant: nulliparous, nonsmoker, >12 y education, and height = 64 in. We could not study spontaneous preterm birth because there were too few cases. The test of effect modification by race was significant (P < 0.0001). SGA, small-for-gestational-age; LGA, large-for-gestational-age; iPTB <37, medically indicated preterm birth at <37 wk gestation; IOM, Institute of Medicine.

this dataset because women without data differed in some important characteristics, including their higher prevalence of preterm birth. It is possible that our results would be biased if the missing data were related to gestational weight gains and adverse birth outcomes. Similarly, misreporting of prepregnancy weight could have a major effect on calculations of weight-gain adequacy and may also bias findings. Like all observational studies, we are limited in our ability to determine causal relations between gestational weight gain and adverse birth outcomes among obese mothers. Although a small trial (30) proved that interventions could help obese women limit weight gain, its power to detect effects on birth outcomes was limited. Large gestational weight-gain intervention trials are needed to determine whether weight gain is causally linked to adverse birth outcomes.

The current study had many strengths, including its large sample of a socioeconomically and racially diverse population of US women and well-characterized birth outcomes. We used intrauterine-derived fetal weight standards to define SGA and LGA, which avoided some problems with birth-weight-based standards, although only serial ultrasounds can truly capture fetal growth velocity and trajectories. Our simple method of studying gestational weight gain allowed for an evaluation of weight-gain adequacy at any gestational age of delivery. It also overcame limitations of studying the rate of gain [which incorrectly assumes the rate of weight gain is constant across pregnancy (11)].

For minimizing adverse birth outcomes, reducing weight before conception among obese women is likely to have the strongest effect. However, for women who are obese when they become pregnant, optimizing gestational weight gain may also aid in reducing risk. To ensure the safest approach, well-designed observational studies are needed in large populations to allow the study of immediate and long-term maternal and fetal outcomes by grade of maternal obesity and possible differences by race-ethnicity. Ideally, these studies would include data on maternal dietary practices, body composition, physical activity, stress, and biochemical measures of metabolism and energy balance to best understand relations shown in the data. Equally important, large experimental trials are vital to identify how interventions during pregnancy to improve nutrition and weight can help women who are obese when they begin pregnancy achieve the healthiest possible outcomes for themselves and their infants.

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