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Inadequate weight gain in overweight and obese pregnant women: what is the effect on fetal growth?

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Abstract

Objective—To evaluate inadequate gestational weight gain and fetal growth among overweight and obese women (O/O).

Study Design—Analysis of prospective singleton term pregnancies in which 1053 O/O gained greater (14.4 ± 6.2 kg) or 188 who either lost or gained ≤ 5 kg (1.1 ± 4.4 kg). Birth weight, fat (FM) and lean mass (LM) were assessed using anthropometry. Small for gestational age (SGA) was defined as $\leq 10^{\text{th}}$ percentile of a standard US population. Univariable and multivariable analysis evaluated the association between weight change and neonatal morphometry.

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Results—There was no significant difference in age, race, smoking, parity, or gestational age between groups. Weight loss or gain ≥ 5 kg was associated with SGA, 18/188 (9.6%) vs. 51/1053 (4.9%); (adjusted OR 2.6, 95% CI 1.4, 4.7; $p=0.003$). Neonates of women who lost or gained ≥ 5 kg had lower birth weight (3258 ± 443 g vs. 3467 ± 492 g, $p<0.0001$), FM (403 ± 175 vs. 471 ± 193 g, $p<0.0001$), LM (2855 ± 321 vs. 2995 ± 347 g, $p<0.0001$) and smaller length, %FM and head circumference (HC). Adjusting for diabetic status, pre-pregnancy BMI, smoking, parity, study site, gestational age and gender; neonates of women who gained ≥ 5 kg had significantly lower birth weight, LBM, FM, %FM, HC and length. There were no significant differences in neonatal outcomes between those who lost weight and those who gained ≥ 5 kg.

Conclusion—In O/O weight loss or gain ≥ 5 kg is associated with increased risk of SGA and decreased neonatal FM, LM and HC.

Keywords

fetal anthropometry; gestational diabetes; gestational weight loss

Introduction

The problem of obesity during pregnancy is now recognized as a major public health concern.¹ In the United States 55.8% of the female population 20–39 years of age is overweight (BMI 25.0–29.9) or obese (BMI ≥ 30), based on World Health Organization (WHO) body mass index criteria (BMI kg/m²).² Because of the increase in obesity there has been an increase in term mean birth weight in developed countries^{3,4} including the United States.⁵ Although there are reports that the increase neonatal weight has reached a plateau or decreased⁶ this may relate more to changes in population demographics such as ethnicity and earlier gestational age at delivery.⁷ Being overweight or obese are significant problems for women and their offspring during pregnancy. In early gestation there is a significant increased risk of early spontaneous abortion and congenital malformations.^{8,9} In later gestation there is a significant increased risk for the metabolic dysfunction associated with insulin resistance presenting as gestational diabetes (GDM) and preeclampsia.¹⁰ At delivery there is an increased risk for cesarean delivery, postpartum wound infection and deep vein thrombophlebitis.¹¹ For the neonate there is an increased risk of fetal macrosomia and more specifically fetal adiposity.¹² Increased adiposity at birth is associated with increased risks of problems such as shoulder dystocia but may also be the harbinger of childhood obesity and metabolic dysfunction.¹³

The Institutes of Medicine (IOM) in 2009 revised the gestational weight gain guidelines, recommending that obese women should have a gestational weight gain of at least 5 kg during pregnancy (5–9 kg), in order to at least meet the obligatory physiologic changes of pregnancy.¹⁴ However some authors have recommended very limited, no weight gain, or even weight loss during pregnancy in order to optimize pregnancy outcomes.^{15,16,17} Hence the purpose of this research was to examine the effect of limited gestational weight gain or weight loss during pregnancy on fetal weight, anthropometry and estimates of neonatal body composition in overweight and obese women.

Materials and Methods

This was a retrospective analysis of prospective multi-center data from 1241 singleton term pregnancies in overweight and obese women examining the effect of limited gestational weight gain or weight loss during pregnancy on fetal growth. The data included 890 patients who were enrolled in the previously reported *Eunice Kennedy Shriver* National Institutes of Child Health and Human Development Maternal-Fetal Medicine Units (MFMU) Network multicenter randomized trial of treatment of mild GDM.¹⁸ Three hundred and fifty-one patients, who were enrolled in another ongoing study of GDM at MetroHealth Medical Center evaluating neonatal growth at delivery, were also included in this analysis. Limited gestational weight gain or loss during pregnancy was estimated as the documented weight at the last prenatal visit minus self-recalled pre-pregnancy weight, and at MetroHealth, when possible, confirmed by a first trimester weight at the first prenatal visit. Height was either measured at the first prenatal visit using a stadiometer or obtained from the prenatal chart. Subjects in this analysis included 395 women with a 50 g glucose challenge test (GCT) < 135 mg/dL, 418 women with a GCT ≥ 135 mg/dL but normal oral glucose tolerance test (OGTT) defined as fasting less than 95 mg/dL, 1 hour < 180 mg/dL, 2 hour < 155 mg/dL and 3 hour < 140 mg/dL, 255 with treated and 173 with untreated GDM (2 or more abnormal values on the aforementioned OGTT). All subjects who required treatment in addition to diet were treated with insulin. There were 36 in the MFMU cohort and 29 in the MetroHealth cohort; total 65 or 5.2% of the 1241 subjects in the entire cohort.

All the subjects participating in the two studies provided written informed consent. The study was approved by the human subjects committee at each of the participating MFMU sites as well as the Institutional Review Board at MetroHealth Medical Center and the Scientific Review Committee of the Clinical Research Unit of the CTSC at Case Western Reserve University. Subjects enrolled in this analysis met the criteria for eligibility as reported elsewhere in the primary report.¹⁸ Subjects were excluded if they met any of the following conditions: an abnormal result on a glucose challenge test at < 24 weeks' gestation or previous GDM; a history of stillbirth in a prior pregnancy, multi-fetal gestation, asthma, or chronic hypertension, received corticosteroid therapy; or were carrying a fetus thought to be anomalous. Similar inclusion and exclusion criteria were used for eligibility for the MetroHealth subjects.

The primary outcome for this study was neonatal morphometry and body composition. Small for gestational age (SGA) was defined as less than 10% for gestational age based on birth weight percentiles from 1994–1996.¹⁹ Birth weight was measured on a calibrated scale and length on a measuring board. Trained research staff performed the anthropometric measures used to estimate neonatal body composition.²⁰ The flank skinfold was measured in the mid-axillary line just above the crest of the ilium. Skinfold measurement was made by lifting the skin with the thumb and index finger with care not to include any underlying tissue. Each skinfold was measured several times until a consistent and stable reading was obtained. The circumference of the head was determined using a tape measure. The coefficient of variation in the anthropometric measures is about 3% and 7% for the skinfolds. Estimates of neonatal body composition were made using the previous validated equation:

fat mass=0.39055 (birth weight kg)+0.0453 (flank skinfold mm)−0.03237 (length cm)+0.54657 . Lean body mass was calculated as birth weight minus fat mass and percent body fat as fat mass/birth weight × 100. The correlation of this anthropometric model with air displacement plethysmography (Pea Pod) estimates of neonatal fat and lean body mass (n=216) is: fat mass (r = 0.83, p < 0.001) and lean body mass (r = 0.94, p < 0.0001) (unpublished data)

We compared baseline characteristics and neonatal outcomes by maternal gestational weight gain less than or equal to 5 kg or greater than 5 kg. Student's t-test and analysis of variance tests were used for continuous variables and chi-square or Fisher's exact test was used for categorical variables. Multiple linear and logistic regression analysis was used to examine the association of neonatal outcomes and weight gain less than or equal to 5 kg or greater than 5 kg, adjusting for potential confounders. The models were adjusted for initial glucose group (mild treated GDM, mild untreated GDM, normal OGTT, normal GCT), pre-pregnancy BMI, smoking status, parity, study site, gestational age at delivery, and gender. The interaction of initial glucose group and weight gain (< 5 kg vs. ≥ 5 kg or more) with each of the neonatal outcomes was examined. Additional regression models examining weight loss/gain classified as weight loss, gain < 5 kg, gain > 5 kg were also run. For these models, the Tukey-Kramer method was used to adjust for multiple comparisons.²¹ Statistical analyses were conducted with SAS software (SAS Institute, Cary, NC).

Results

Of the 1241 subjects in this cohort, 1053 (84.8%) had gestational weight gain greater than 5 kg and 188 (15.2%) had gestational weight gain less than 5 kg. Of 1053 women who gained greater than 5 kg, 691 (65.6%) exceeded IOM gestational weight gain guidelines for their respective pregravid BMI, 331 (31.4%) gained within IOM guidelines and 31 (2.9%) gained below IOM guidelines. Of the women who gained less than 5 kg, 139 (74.0%) were from the MFMU study and 49 (26.1%) were from the MetroHealth study. Women who gained greater than 5 kg had a mean (± SD) gestational weight gain of 14.4 ± 6.2 kg versus 1.1 ± 4.4 kg for the women who gained < 5 kg (p<0.0001). Demographic characteristics are shown in Table 1. Women who gained less than 5 kg during pregnancy had significantly greater pre-pregnancy weight and BMI (p<0.0001). There was significant difference in the glucose tolerance status in women who gained > 5 kg versus < 5 kg (p=0.002). Women who gained > 5 kg had a greater percentage of either a normal GCT or abnormal GCT and normal OGTT, whereas women who gained < 5 kg had a greater percentage of either treated GDM or untreated GDM. There were no other significant differences in any other demographic characteristic between groups.

Table 2 shows the neonatal characteristics of the women who gained less than and more than 5 kg. More neonates of women who gained < 5 kg were SGA versus those whose mothers gained > 5 kg (9.6% vs. 4.9%, p=0.009). More neonates of women who gained > 5 kg were LGA versus those who gained < 5 kg (13.2% vs. 7.5%, p=0.03). Relative to the anthropometry, there was a significantly lower birth weight, length, lean mass, fat mass, percent body fat and head circumference in the neonates of women who gained < 5 kg in contrast to those who gained more than 5 kg. When adjusted for status of glucose tolerance, pre-pregnancy BMI, smoking, parity, study site (MFMU or MetroHealth), gestational age at

delivery and gender, they remained significantly different. After adjustment for the same variables, there remained a significant difference in SGA (adjusted OR 2.6, 95% CI 1.4, 4.7; $p=0.003$). LGA was significantly decreased (adjusted OR 0.42, 95% CI 0.23, 0.77; $p=0.005$). Further there was no significant interaction among the 4 study groups (mild treated GDM, mild untreated GDM, normal OGTT, normal GCT) and weight change for SGA or other outcomes.

Because some authors have suggested that weight loss during pregnancy in overweight and obese women may improve pregnancy outcome, we further analyzed our data in the 46 (3.7%) of women who had a documented loss of weight (-5.0 ± 4.7 kg) versus the 1195 women who gained any weight (13.1 ± 6.9 kg) during pregnancy ($p < 0.0001$). Table 3 shows the neonatal characteristics of women who lost weight, gained 0 to 5 kg and gained greater than 5 kg. There was a significant difference in birth weight, length, lean body mass, fat mass and percent body fat and SGA among the groups. We next adjusted for glucose status, group, prenatal BMI, smoking, parity, study site (MFMU or MetroHealth), gestational age at delivery and male gender in the multiple regression analysis for the effect of change in weight on the anthropometric outcomes making adjustments for multiple comparisons (Table 3). There were significant differences between those who lost weight and those who gained greater than 5 kg in each of the following; birth weight, lean body mass, fat mass, percent body fat, head circumference and the proportion of SGA. There were also significant differences between those who gained ≤ 5 kg and those who gained greater than 5 kg in birth weight, lean body mass, fat mass, percent body fat and neonatal length. It is important to note that a subject's glucose status did not affect SGA results. In women with GDM, whether treated or untreated, there was no significant difference in the risk of SGA in women who lost weight or gained less than 5 kg as compared with women who gained > 5 kg (adjusted OR 1.5, 95% CI 0.5, 4.4; $p=0.43$). Additionally when we eliminated all the GDM subjects, the results for the non-GDM cohort show that this group is similar to the entire cohort (data not shown).

Comment

Overweight and obese women who lose weight or gain less than 5 kg weight during pregnancy have neonates who in addition to an increase in SGA have a significant decrease in lean body mass and fat mass as compared with overweight and obese women who gain greater than 5 kg weight during pregnancy. Some authors have criticized the IOM gestational weight gain recommendations for being too conservative for overweight and obese women and suggested that less gestational weight gain, no weight gain or even weight loss might be more appropriate for this population.^{15,16,17} We observed that weight gain ≤ 5 kg in these women was associated with a significantly lower birth weight, lean body mass, fat mass, percent body fat and length as compared with the neonates of overweight and obese women who gained greater than 5 kg. In these women the potential long term consequences for the offspring of this difference in lean body mass (i.e., decreases in head circumference and length) is unclear but could be associated with further increased metabolic and cognitive risk for the offspring.²² We elected to use the 5 kg weight cutoff for both overweight and obese since our objective was to evaluate inadequate gestational weight gain and fetal growth among overweight and obese women and not base the study

objective on strict IOM criteria. Many clinicians may consider weight gain between 5–7 kg in overweight women as adequate. Hence we elected to choose the lower limit of gestational weight gain at 5 kg rather than using a separate lower limit of gestational weight gain for overweight and obese women.

The results of our study agree with those of Beyerlein et al, who reported that gestational weight loss in overweight and obese class I and II women was associated with an increase in SGA.²³ However, these authors found no increase in SGA in women in class III obesity, which were highly represented in our study cohort. The results of our study differ from recent reports from two Scandinavian groups. Asbjornsdottir et al in a small study evaluated fetal growth in 58 Danish women with type 2 diabetes who were advised to gain 0–5 kg weight during pregnancy.²⁴ Seventeen women (29%) gained less than 5 kg and the remaining 41 gained greater than 5 kg. There was no significant difference in SGA neonates 3 (18%) vs. 4 (10%), $p=0.41$, but there was a significant decrease in birth weight Z score, -0.44 (-3.31 to 1.98) vs. 0.84 (-2.32 to 4.02), $p=0.008$). The authors concluded that in obese women with type 2 diabetes, maternal gestational weight gain less than 5 kg was associated with a more proportionate birth weight and less perinatal morbidity. The differences in results may relate to differences in sample size, type 2 vs. GDM, and ethnicity of the study populations. Bloomberg²⁵ using a Swedish birth cohort reported that women with class II and III obesity who lost weight had an increased risk for SGA which was no longer significant with low weight gain in class III obese women, but persisted in those with class II obesity.

The strength of our study is that in addition to classification of fetal growth using birth weight and characterization of birth weight percentiles for SGA, we were able to measure neonatal anthropometrics and estimate neonatal body composition. All of the measurements were performed using the same standard protocol at each study location. The limitations of our study is that although the data were collected prospectively this was a retrospective analysis. We also used self-reported pre-pregnancy weight and in some patients confirmed with a first trimester weight at the first antenatal visit. As in most other studies reliable pre-pregnancy weight was not readily available. Another limitation of our study is that there were a small number of subjects who lost weight during gestation and we were not able to adjust for all potential confounding variables.

What are the potential mechanisms associated with a decrease in lean body mass as well as fat mass in neonates of overweight and obese women who lose weight during pregnancy? Weight loss in overweight and obese non-pregnant individuals includes both loss of fat mass and lean body mass. A 10 kg weight loss by diet alone would be expected to have a decrease in lean body mass of between 2–3 kg.²⁶ During pregnancy it is very likely that maternal weight loss is associated with a loss of maternal lean body mass as well as fat mass.

The obligatory physiologic weight gain during pregnancy accounts for approximately 8 kg of water and 1 kg of protein plus variable amounts (1–6 kg) of adipose tissue.¹⁴ Since the average weight loss in women who lost weight was -5 kg, plus the obligatory physiologic weight change in pregnancy is approximately 9 kg, this means that the estimated maternal tissue weight loss was on the average of 14 kg or 31 lbs. This decrease of 14 kg most likely

represents loss of both maternal lean body mass and fat mass, particularly since only vigorous exercise during weight loss has been shown to spare considerable loss of lean body mass.²⁷ Vigorous exercise was unlikely in these overweight and obese pregnant women. Hence, we speculate that in overweight and obese women who lose weight during gestation that there is the adaptive protective mechanism to retain lean body mass as well as lose fat mass, thereby decreasing protein availability for fetal growth. This then would result in disproportionate decrease in fetal lean body mass relative to fat mass. Only prospective longitudinal metabolic studies will definitively address and answer these questions.

What are the implications for the neonate because of these significant decreases in lean body mass and fat mass in overweight and obese women who lose weight or gain < 5 kg during pregnancy? While increased body fat is a risk for childhood obesity, the addition of a decrease in lean body mass (including length and head circumference) further increase metabolic and cognitive risk for the offspring.²² The decrease in lean body mass in these neonates may be a forme fruste of metabolic stunting observed in children in developing countries, where there is a dearth of quality protein. The WHO defines stunting as length less than 2 standard deviations below the WHO Multicentre Growth Reference Study Group, 2006.²⁸ However, many children are not classified as stunted until age 2 or 3 because the continuation of poor nutrition in early life, i.e. the first 1000 days of perinatal programming. A decrease in lean body mass in the offspring may increase the risk of metabolic dysfunction in later life because of the decreased ability to store ectopic fat in tissues such as skeletal muscle and liver. The ectopic fat may act as an additional source of inflammation increasing insulin resistance.²⁹ Because the women in this study who lost weight during pregnancy were primarily class III, the poor nutrition in utero the fetus was exposed to may persist in early childhood, despite a surfeit of poor quality of nutritional calories.

In conclusion, weight loss or weight gain less than 5 kg in overweight and obese women during pregnancy increases the risk of SGA, decreases in fat mass and of potentially greater significance, decreases in lean body mass including length and head circumference as compared with those infants whose mothers had a gestational weight gain of greater than 5 kg. These preliminary findings need to be confirmed in prospective studies using more sophisticated methodologies to assess maternal nutrient metabolism and fetal growth. Information concerning the amount and type of energy intake and expenditure available will help determine if the weight loss is a result of decreased intake or increased energy expenditure. If inadequate intake is the primary issue, information on maternal protein metabolism may be critical because of the effect on lean body mass in the fetus. Other factors such as when in gestation the inadequate weight gain occurred may offer insights into the pathophysiology of the changes in body composition of the fetus.

In the interim, although the American Congress of Obstetricians and Gynecologists in a recent Committee Opinion recommends that for the overweight or obese woman who is gaining or wishes to gain less weight than recommended by the IOM care be individualized based on the appropriate growth of the fetus, we urge caution.³⁰ A far safer alternative would be, as women's health care physicians, to recommend a lifestyle approach of attainment of normal body weight through diet and exercise postpartum for all overweight or obese women. This approach may be of specific benefit for those overweight and obese

woman planning another pregnancy as weight loss between pregnancies has been shown to decrease adverse pregnancy outcomes in a subsequent pregnancy.³¹

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Appendix

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CLINICAL IMPLICATIONS

- Inadequate gestational weight gain in overweight and obese women increases the risk of their having infants that are small for gestational age and alterations in fetal composition.
- The long-term implications of inadequate weight gain in infants of overweight and obese women are uncertain and require further examination.
- Ideally, weight reduction in overweight and obese women should occur before a planned pregnancy.

Table 1

Maternal Characteristics

	Gestational Weight loss or gain 5 kg (n = 188)	Gestational Weight gain > 5 kg (n = 1053)	p-value
Age (years)	28.2 ± 5.7	28.1 ± 5.8	0.67
Pre-pregnancy height (cm)	159.3 ± 8.2	160.1 ± 7.5	0.19
Pre-pregnancy weight (kg)	87.4 ± 21.9	78.2 ± 16.1	<0.0001
Pre-pregnancy BMI (kg/m ²)	34.2 ± 7.1	30.4 ± 5.2	<0.0001
25.0 – <30	59 (31.4)	627 (59.5)	
30.0 – 40	97 (51.6)	370 (35.1)	
> 40	32 (17.0)	56 (5.3)	
Tobacco use + (%)	29 (15.4)	120 (11.4)	0.12
Parity (%)			0.09
1	30 (16.0)	231 (21.9)	
2	68 (36.2)	396 (37.6)	
3+	90 (47.9)	426 (40.5)	
Race (%)			0.99
White	62 (33.0%)	345 (32.8%)	
AA/Black	38 (20.2%)	220 (20.9%)	
Hispanic	85 (45.2%)	469 (44.5%)	
Other	3 (1.6%)	19 (1.8%)	
Glucose status (%)			0.002
Normal GCT	52 (27.7%)	343 (32.6%)	
Abnl GCT/NL OGTT	52 (27.7%)	366 (34.8%)	
GDM treated	58 (30.9%)	197 (18.7%)	
GDM untreated	26 (13.8%)	147 (14.0%)	
Weight gain/loss (kg)	1.1 ± 4.4	14.4 ± 6.2	< 0.0001

Data are presented as mean ± SD. Percents are in (%).

Table 2

Neonatal Characteristics

	Gestational Weight loss or gain 5 kg (n = 188)	Gestational Weight gain > 5 kg (n = 1053)	p-value
Gestational age (weeks)	38.8 ± 1.4	38.9 ± 1.4	0.28
Gender (%)			0.32
Male	89 (47.3)	540 (51.3)	
Female	99 (52.7)	540 (51.3)	
Birth weight (g)	3258.4 ± 442.7	3466.8 ± 491.5	<0.0001
Length (cm)	49.3 ± 2.3	50.0 ± 2.8	0.001
Head circumference (cm)	34.2 ± 1.7	34.5 ± 1.7	0.02
Lean Mass (g)	2855.1 ± 321.0	2995.4 ± 346.9	<0.0001
Fat mass (g)	403.4 ± 175.3	471.4 ± 192.7	<0.0001
Body fat (%)	12.0 ± 4.2	13.2 ± 4.3	0.0006
LGA	14 (7.5%)	139 (13.2%)	0.03
SGA	18 (9.6%)	51 (4.9)	0.009

Data are presented as mean ± SD. Percents are in (%).

Table 3

Neonatal Outcomes

	(1) Lost Weight (n=46)	(2) Gained 0 to 5 kg (n=142)	(3) Gained > 5 kg (n=1,053)	P-Value	Adjusted P-Value*
Birth weight (g)	3186.6 ± 458.7	3281.7 ± 436.5	3466.8 ± 491.5	<0.0001	1 vs. 3, <0.0001 2 vs. 3, <0.0001
Length (cm)	49.5 ± 2.4	49.3 ± 2.3	50.0 ± 2.8	0.01	2 vs. 3, 0.017
Head circumference (cm)	34.1 ± 1.5	34.2 ± 1.8	34.5 ± 1.7	0.06	1 vs. 3, 0.04
Lean Mass (g)	2813.9 ± 352.9	2868.4 ± 310.1	2995.4 ± 346.9	<0.0001	1 vs. 3, <0.0001 2 vs. 3, <0.0001
Fat mass (g)	372.7 ± 217.0	413.3 ± 159.1	471.4 ± 192.7	<0.0001	1 vs. 3, 0.0002 2 vs. 3, 0.0003
Body fat (%)	11.3 ± 5.8	12.3 ± 3.5	13.2 ± 4.3	0.001	1 vs. 3, 0.003 2 vs. 3, 0.014
LGA	3 (6.5%)	11 (7.7%)	139 (13.2%)	0.08	ns
SGA	6 (13.0%)	12 (8.5%)	51 (4.8%)	0.02	1 vs. 3, 0.012

* Multiple regression analysis with Tukey-Kramer adjustment for multiple comparisons. Data are presented as mean ± SD. Percents are in (%).

The models were adjusted for initial glucose group (mild treated GDM, mild untreated GDM, normal OGTT, normal GCT), pre-pregnancy BMI, smoking status, parity, study site, gestational age at delivery, and gender.