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Gestational weight gain, early pregnancy maternal adiposity distribution, and maternal hyperglycemia

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Abstract

Objectives—To estimate the effects of gestational weight gain (GWG), central adiposity and subcutaneous fat on maternal post-load glucose concentration.

Methods—Pregnant women [n = 413, 62% black, 57% with pregravid body mass index (BMI) 25] enrolled in a cohort study at <13 weeks gestation. GWG was abstracted from medical records. In a sub-sample of women (n = 214), waist circumference (WC), and biceps and triceps skinfold thicknesses were measured at enrollment. At 24–28 weeks gestation, post-load glucose concentration was measured using a 50-g 1-hour oral glucose tolerance test.

Results—After adjustment for pre-pregnancy BMI, age, parity, race/ethnicity, smoking, marital status, annual family income, education, family history of diabetes, and gestational age of GDM screening, each 0.3-kg/week increase in weight in the first trimester was associated with a 2.2 (95% CI: 0.1, 4.3)-mg/dl increase in glucose concentration. Each 8.6-mm increase in biceps skinfold thickness and each 11.7-mm increase in triceps skinfold thickness was associated with 4.3 (95% CI: 0.2, 8.5)-mg/dl increase in maternal glucose, independent of BMI and other confounders. Neither GWG in the second trimester nor WC at 13 weeks was significantly associated with glucose concentration after confounder adjustment.

Conclusions—Independent of pre-pregnancy BMI, high early pregnancy GWG and maternal subcutaneous body fat may be positively associated with maternal glucose concentrations at 24–28 weeks.

Keywords

gestational weight gain; skinfold thickness; pregnancy; glucose; gestational diabetes

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INTRODUCTION

In 2008, the Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) study provided compelling evidence that maternal glucose levels below those diagnostic of gestational diabetes mellitus (GDM) have strong positive associations with a variety of adverse pregnancy outcomes previously ascribed solely to overt GDM (1). Nevertheless, the underlying causes of high maternal glucose are not fully understood. A better understanding of the risk factors that lead to high maternal glucose is imperative to improving the health of women and their infants.

There is a strong, consistent positive association between high pre-pregnancy body mass index (BMI) and maternal glucose concentrations (2) and risk of GDM (3). However, prepregnancy BMI only measures general adiposity, and women with similar BMI values may have widely varying distribution of adipose tissue (4). The distribution of adipose tissue may also impact glucose metabolism (5), but has received little attention in the pregnant women (6). Additionally, excessive gestational weight gain, especially in early pregnancy, may influence risk of GDM (6–10).

The objective of our study was to estimate the association between markers of first-trimester maternal central adiposity and subcutaneous fat as well as GWG up to the time of GDM screening on post-load glucose concentrations.

MATERIALS AND METHODS

We analyzed data from the Study of Nutrition and Pregnancy (SNAP), a prospective pregnancy cohort study of women receiving care in the prenatal clinics at Magee-Womens Hospital in Pittsburgh, PA. Eligible women were non-Hispanic white or black based on self-report and had singleton pregnancies with no preexisting conditions, vaginal bleeding, or substance abuse. At enrollment participants provided informed, written consent. The study was approved by the University of Pittsburgh Institutional Review Board.

A total of 724 eligible pregnant women were enrolled at 13 weeks' gestation (mean (SD) gestational age 9.1 (2.9) weeks). At enrollment, women completed a structured interview that included questions on socio-demographic factors and medical history. Of those eligible, we excluded women who had a spontaneous or therapeutic abortion ($n = 85$), implausible or missing weight measurements ($n = 84$), or a first prenatal visit after the first trimester ($n = 53$). Women were also excluded if they did not have a measured weight within 30 days of their GDM screening ($n = 8$). An additional 38 women were excluded because they were missing data on one of the covariates included in the final model. Lastly, 43 women were excluded because their glucose screening was performed at or before 24 weeks' gestation (see details in results section). A total of 413 women were included in the final analysis. Women excluded from our analysis were more likely to be smokers (56% versus 44%, $p < 0.05$) or nulliparous (39% versus 21%, $p < 0.01$) than women included in the analysis. There were no other significant differences in post-load glucose concentrations, pre-pregnancy BMI, GWG, or other maternal characteristics (data not shown). Later in the study, anthropometric measurements were added to the study protocol so that adiposity distribution

could be assessed. Of the women in the final analytic sample, 214 enrolled after waist circumference (WC) and skinfold measurements at 13 weeks gestation [mean (SD) = 8.5 (2.0) weeks gestation] were added.

A 50-g 1-hour oral glucose challenge test was performed as part of routine clinical care at approximately 24–28 weeks gestation to screen for GDM. Post-load glucose values were abstracted from medical records and maternal post-load glucose concentration was used as a continuous outcome variable. Women with post-load glucose values between 135 mg/dL and 180 mg/dL received a subsequent 3-hour 100-g oral glucose tolerance test. Women with 50-g 1-hour post-load glucose concentration values $\geq$ 180 mg/dL, or 2 or more abnormal 3-hour 100-gram oral glucose tolerance results were classified as having GDM (11).

We calculated the first trimester rate of GWG as the difference between a woman's weight measured at the last prenatal visit in the first trimester ($\geq$ 8 and $<$ 14 weeks gestation “first trimester visit”) and her pre-pregnancy weight divided by the weeks of gestation at the first trimester visit. The second trimester rate of GWG was defined as the difference between the weight at GDM screening test and the weight at the first trimester visit divided by the number of weeks between measurements. The total mean rate of GWG up to GDM screening was calculated as the difference between the measured weight at or before GDM screening and the self-reported pre-pregnancy weight, divided by the weeks of gestation at the time of the weight measurement. The difference in time between the measured weight and GDM screening was, on average, less than 1 week [mean (SD) = 0.5 (1.6) weeks]. We defined adequacy of total weight gain using the 2009 IOM recommendations (12). Total GWG up to GDM screening was classified as inadequate, adequate, or excessive, as described previously (13).

For women whose self-reported pre-pregnancy weight was deemed biologically improbable ($n = 45$, $\geq$ 6.8 kg gained or lost between conception and 13 weeks gestation), we imputed pre-pregnancy weight using the rate of weight gain between the first two prenatal visits $\geq$ 16 weeks gestation, similarly to previous work (14). Central adiposity was assessed using WC measured at the smallest point of the waist by a trained research nurse at 13 weeks gestation. Subcutaneous fat was described using biceps and triceps skinfolds, measured by a trained research nurse at 13 weeks. To reduce measurement error, biceps and triceps skinfolds measures were taken three times and the mean was used for analysis. If women had an outlying value, defined as a difference between the lowest and highest of the triplicate that was greater than two standard deviations from the mean of all three measurements (triceps: $n = 10$; biceps: $n = 14$), we calculated the mean of the two remaining values.

Maternal demographics and pre-pregnancy weight were obtained by self-report at enrollment. Family history of diabetes was abstracted from medical records after delivery. Prepregnancy BMI [pre-pregnancy weight (kg)/measured height (m^2)] was categorized as underweight (<18.5); normal weight (18.5–24.9); overweight (25.0–29.9); obese (≥ 30) (15).

Separate multivariable linear regression models were used to assess the association between each independent variable (rate of GWG, GWG adequacy, WC, and skinfolds) and maternal

post-load glucose concentration. The relationship of each independent variable with glucose was assessed by visual inspection of lowess smoothing plots. Because no deviations from linearity were observed, independent variables were specified as continuous. GWG adequacy was categorized to ensure greater interpretability. Potential confounders were maternal age, parity, race/ethnicity, smoking status, marital status, annual family income, education, family history of diabetes and gestational age of glucose challenge test. Pre-pregnancy BMI was included in all models out of convention. To maintain comparability across models, the full model was used for all four of the final GWG models. Because of the smaller sample size, we assessed confounding in the adiposity distribution measurements separately from the GWG models. We defined confounding as a $> 10\%$ change in the adjusted beta coefficient after exclusion of the covariate from the full model. Pre-pregnancy BMI, maternal age, parity, race/ethnicity, maternal education, and family history of diabetes were all found to be confounders in the skinfolds and WC models. Pre-pregnancy BMI and race/ethnicity were evaluated as effect modifiers in each model by including the interaction term in the model and testing for significance using the Wald test ($\alpha < 0.05$), but neither was found to be significant. A $p < 0.05$ was considered significant. We tested the sensitivity of our results to the exclusion of women who were considered by their clinician to be at high risk for GDM (very obese before pregnancy or strong family history of diabetes) and therefore underwent early screening (< 24 weeks gestation); women with a BMI ≥ 35 ; and women whose WC measurements were obtained at 10 to 13 weeks gestation. Data were analyzed using STATA software version 11 (StataCorp, College Station, TX).

RESULTS

Women in our cohort tended to be young, multiparous, African American, unmarried, and high school educated or less (Table 1). Approximately half the women were smokers, low income, and unemployed and more than a third were obese. GDM screening occurred at a mean (SD) of 27.3 (2.3) weeks gestation. The mean post-load glucose concentration was 99.7 (SD: 24.3) mg/dl and the median (interquartile range) was 96 (31) mg/dL. A total of 45 women (9.0%) had elevated post-load glucose concentrations (≥ 135 mg/dl), and of these women, eleven (2.7% of total cohort) were diagnosed with GDM.

On average, women gained approximately 1.4 (SD: 4.1) kg in the first trimester and 6.0 (SD: 4.4) kg in the second trimester. More than half had excessive weight gain up to GDM screening (Table 2). In the unadjusted analysis, first trimester rate of GWG and rate of GWG up to GDM screening were linearly associated with post-load glucose. After adjustment for age, parity, race/ethnicity, smoking status, marital status, income, education, family history of diabetes, gestational age at GDM screening and pre-pregnancy BMI, each 0.3 kg/week of GWG (4 kg total) in the first trimester was associated with nearly a 2-mg/dL increase in maternal blood glucose. In both the unadjusted and adjusted models, weight gain up to GDM screening (rate or adequacy) and second trimester rate of gain were not significantly associated with maternal glucose.

Women in our cohort had a mean (SD) biceps skinfold thickness of 14.6 (8.6) mm, triceps skinfold thickness of 25.9 (11.7) mm, and WC of 99.9 (26.8) cm. The correlation coefficient between biceps and triceps measurements was 0.74. WC measurements had a correlation

coefficient of 0.46 with biceps measurements and 0.49 with triceps measurements. In unadjusted analyses, biceps and triceps skinfolds had positive, linear associations with post-load glucose concentration (Table 3). After adjustment for pre-pregnancy BMI, maternal age, parity, race/ethnicity, maternal education, and family history of diabetes, each standard deviation increase in biceps and triceps skinfold thicknesses was associated with an approximately 4.3 mg/dl increase in maternal glucose. WC at 13 weeks was not associated with maternal glucose after confounder adjustment.

In sensitivity analyses, we found that inclusion of 43 women who underwent early screening did not change our conclusions. However, to ensure that our results were comparable to other studies, these women were excluded from our final analysis. No differences were detected when we excluded women with a BMI ≥ 35 (n=80) from the analysis or when we excluded women whose WC measurements were obtained at 10 to 13 weeks gestation (n=54). No meaningful differences were detected when we excluded 45 women with imputed prepregnancy weights were from our analyses (data not shown).

DISCUSSION

We found that a high first trimester rate of GWG and elevated biceps and triceps skinfold thicknesses were associated with elevated post-load glucose concentrations. Measurements were obtained early in pregnancy and the associations were independent of pre-pregnancy BMI and other relevant covariates. Early-pregnancy WC was not associated with glucose concentrations.

We are unaware of other studies in pregnant mothers to measure markers of both subcutaneous and visceral fat in relation to glucose tolerance. Results from our longitudinal data are consistent with a study that measured a variety of skinfold thicknesses and found that women with overt GDM (n = 56) had higher skinfold thicknesses, including triceps and biceps, than women with normal glucose (n = 42) (6). Skinfold measurements in these prior findings were taken in mid- to late-pregnancy (26–36 weeks' gestation) rather than early pregnancy, and the researchers did not adjust the results for pre-pregnancy BMI. (6) Unlike our study, prior investigations have reported positive associations between WC and post-load glucose (8–10), but used self-reported pre-pregnancy WC (8), which has uncertain validity, or measured WC at mid-pregnancy (9, 10), when it is a less accurate measure of adiposity (16). For example, among 4,981 women in the Nurses' Health Study II, self-reported WC before pregnancy was positively associated with risk for later development of GDM (8), even after adjustment for pregravid BMI. We based WC on measurements taken by a single trained nurse at a mean of 9 weeks gestation, which may partially explain the difference in our results.

In non-pregnant populations, abdominal adiposity is considered to be strongly associated with the development of type 2 diabetes (17). This is contrary to our findings that skinfold thicknesses were associated with glucose, but WC was not. This may be due to changes in adiposity distribution during pregnancy or because our cohort generally had glucose values below the threshold of GDM. However, in a small cohort of non-pregnant individuals, peripheral skinfold thickness (measured using thigh skinfold thickness) was an independent

predictor of both plasma glucose and insulin sensitivity, even after WC was added to the model (18). This suggests that peripheral subcutaneous adiposity may play role in the development of high blood glucose, independent of central adiposity.

Our result that first trimester GWG and maternal glucose are positively associated is well supported by previously studies (7, 19–21). For example, Herring et al. found that weight gain up to 12 weeks' gestation was greater for women with GDM compared to controls (4.0 compared with 2.8 lb, $p < 0.05$). We found no association between total GWG up to the time of GDM screening and maternal post-load glucose concentration, which is consistent with several other studies (21–23). Of the studies that reported a positive association between high total GWG up to screening and maternal glucose, relations were primarily found among obese women (20, 24) or for impaired glucose intolerance, but not overt GDM (19, 25). Other studies did not control for important covariates such as a family history of diabetes (20) or used linear interpolation to calculate GWG (19), although total GWG is not linear (12).

Increases in weight and insulin resistance are normal physiological changes of pregnancy. However, excess fat accumulation can cause high concentrations of free fatty acids, altered adipokine expression, and low-grade inflammation, all of which are thought to increase insulin resistance (26). GWG early in pregnancy is largely fat accumulation, whereas total GWG is not (27), which may explain why we saw an association with first trimester GWG rate, but not total GWG. High subcutaneous fat has been associated with decreased insulin resistance in non-pregnant populations (28), and there is some evidence that maternal diabetes and obesity may cause defects in insulin-signaling pathways in subcutaneous adipose tissue (29).

Because our cohort was generally comprised of younger, single-gestation women, post-load glucose concentrations in our cohort were generally low and only a small proportion of women in our cohort developed GDM. However, our analysis of glucose as a continuous variable allowed us to examine the linearity of the relationship between adiposity and maternal post-load glucose. The demographics of our cohort, although reflective of the population in the clinic in which the women received care, are not reflective of the general obstetric population of the U.S. This reduces the generalizability of our results. The 50-g 1-hour oral glucose challenge test is primarily used as a screening test, and therefore may be affected by time since last meal. Ideally, we would have used a fasting glucose value, but this information was not available for our cohort. As with any study that uses self-reported pre-pregnancy weight, our results may be biased by misreporting. We made our best attempt to mitigate this bias by correcting biologically implausible values as the removal of these women from our analysis reduced the power of our study. Skinfold thicknesses can be altered by the changes in hydration experienced during pregnancy, but our measurement of skinfolds early in pregnancy reduces the likelihood of this biasing our finding. We were unable to measure the contribution of fat versus fat-free mass to GWG. Research in non-pregnant populations has suggested that gains in visceral adipose tissue and intramuscular adipose are associated with type 2 diabetes (30). If this association is also true in pregnancy, than the effect of GWG that we observed actually may be due, in part, to a type of adipose tissue. In a cohort of young, African American and white women, we included assessments

of trimester-specific GWG and adjusted for important medical and socioeconomic covariates, such as pre-pregnancy BMI. Ours is one of very few studies to examine maternal fat distribution early in pregnancy and its association with maternal glucose. Future studies of continuous maternal glucose more rigorous measures of adiposity, and analyses of trimester-specific GWG are warranted.

Our results provide insight into the role that adiposity may play in maternal hyperglycemia. Excessive early-pregnancy weight gain and subcutaneous fat mass, independent of high pre-pregnancy BMI, may be modifiable risk factors. A better understanding of their relationship with glucose concentrations made lead to more effective targeted interventions to reduce the risk of maternal hyperglycemia and improve maternal and fetal outcomes.

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Table 1

Maternal characteristics of SNAP participants, n = 413

Characteristic	
Age at enrollment, mean (SD) Parity, N (%)	24.4 (4.5)
0	88 (21.3)
1–7	325 (78.7)
Race/ethnicity, N (%)	
White	159 (38.5)
African American	254 (61.5)
Smoking status, N (%)	
Smoker	180 (43.6)
Non-smoker	233 (56.4)
Marital status, N (%)	
Married	61 (14.8)
Not married	352 (85.2)
Annual family income, N (%)	
< \$10,000	177 (42.9)
\$10,000	236 (57.1)
Educational status, N (%)	
Greater than a high school degree	49 (11.9)
High school educated	364 (88.1)
Family history of diabetes, N (%)	
History of diabetes	152 (36.8)
No history of diabetes	261 (63.2)
Employment status, N (%)	
Employed	224 (54.2)
Not employed	189 (45.8)
Pre-pregnancy BMI (kg/m ²), mean (SD)	28.0 (7.4)
Underweight (<18.5), N (%)	13 (3.1)
Normal weight (18.5–24.9), N (%)	165 (40.0)
Overweight (25–29.9), N (%)	91 (22.0)
Obese (30+), N (%)	144 (34.9)

BMI, body mass index.

Table 2

Unadjusted and adjusted association between maternal blood glucose (mg/dL) at < 28 weeks gestation and standard deviation changes in maternal GWG, n = 413

	N (%)	Mean (SD) kg/week	Unadjusted β (95% CI)	Adjusted ^I β (95% CI)
1 st trimester average rate of GWG, per 0.3 kg/week increase	--	0.1 (0.3)	2.5 (0.4, 4.6)	2.2 (0.1, 4.3)
2 nd trimester average rate of GWG, per 0.3 kg/week increase	--	0.4 (0.3)	0.1 (-2.5, 2.6)	-0.07 (-2.8, 2.6)
Total rate of GWG up to GDM screening, per 0.2 kg/week increase	--	0.3 (0.2)	2.0 (0.01, 4.0)	1.8 (-0.3, 4.0)
Adequacy of GWG up to GDM screening				
Inadequate	110 (26.6)	--	-3.0 (-10.1, 4.0)	-3.5 (-10.4, 3.4)
Adequate	78 (18.9)	--	ref	ref
Excessive	225 (54.5)	--	1.9 (-4.4, 8.1)	0.3 (-5.8, 6.3)

GWG = gestational weight gain

Missing: 20 women missing 1st and 2nd trimester rates of gain

^I Adjusted for pre-pregnancy BMI, age, parity, race/ethnicity, smoking, marital status, annual family income, education, family history of diabetes, gestational age of GDM screening

Table 3

Association between maternal blood glucose (mg/dL) at < 28 weeks gestation and standard deviation changes in waist circumference, biceps and triceps skinfold thickness at 13 weeks gestation, n = 214

Measurement	Unadjusted β (95% CI)	Adjusted ¹ β (95% CI)
Biceps skin fold (mm), per 8.6 mm increase	5.1 (1.8, 8.3)	4.3 (0.2, 8.5)
Triceps skin fold (mm), per 11.7 mm increase	5.9 (2.6, 9.1)	4.3 (0.2, 8.5)
Waist circumference (cm), per 26.8 cm increase	4.5 (1.3, 7.8)	3.0 (-0.7, 6.8)