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The Impact of Maternal Obesity and Gestational Weight Gain on Early and Mid-Pregnancy Lipid Profiles

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Abstract

Objective—We evaluated the impact of maternal overweight/obesity and excessive weight gain on maternal serum lipids in the first and second trimester of pregnancy.

Design and Methods—Prospective data were collected for 225 women. Maternal serum lipids and fatty acids were measured at <13 weeks and between 24–28 weeks. Analyses were stratified by normal weight versus overweight/obese status and excessive vs. non-excessive weight gain.

Results—Overweight/obese women had higher baseline cholesterol (161.3±29.6 vs 149.4±26.8 mg/dL, p<0.01), LDL (80.0±19.9 vs 72.9 ±18.8 mg/dL, p<0.01) and triglycerides (81.7±47.2 vs 69.7±40.3 mg/dL, p=0.05) when compared to normal weight women, while HDL (43.6 ±10.4 47.6±11.5 mg/dL, p<0.01) was lower. However, cholesterol and LDL increased at a higher weekly rate in normal weight women, resulting in higher total cholesterol in normal weight women (184.1±28.1 vs. 176.0 ±32.1 mg/dL, p=0.05) at 24–28 weeks. Excessive weight gain did not affect the rate of change in lipid profiles in either group. Overweight/obese women had higher levels of arachidonic acid at both time points.

Conclusions—Overweight/obese women have significantly more atherogenic lipid profiles than normal weight women during the period of early pregnancy, delineating one physiologic pathway that could explain differences in pregnancy outcomes between normal weight and overweight/obese women.

Keywords

Pregnancy; Triglycerides; Cholesterol; Fatty Acids; Maternal Obesity; Weight gain

Introduction

Two-thirds of women in the United States are overweight or obese (body mass index (BMI) 25 kg/m² or greater) at the time of conception.^{1–2} Maternal obesity is associated with an increased risk for pregnancy complications including gestational diabetes,³ pre-eclampsia,⁴

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and abnormalities of fetal growth.⁵ In addition, offspring from obese women may be more likely to develop obesity and diabetes in their lifetime.⁶ Excess maternal weight gain has also been linked to an increased risk for adverse pregnancy outcomes including gestational diabetes and fetal macrosomia.⁷ However, the mechanisms underlying these increased risks for pregnancy complications are incompletely understood.

Pregnancy is characterized by progressive increases in serum lipids, and this adaptation is essential to promote normal fetal growth and development.^{8–15} In the non-pregnant state, obesity is associated with an atherogenic phenotype including high serum concentrations of total cholesterol, low density lipoprotein (LDL) and triglycerides as well as a reduction in serum high density lipoprotein (HDL) cholesterol.¹⁶ However, less is known regarding the influence of maternal obesity or excess weight gain on serum lipid profiles during pregnancy.

There are also limited data comparing fatty acid profiles between normal weight and overweight/obese women during pregnancy. Saturated and monounsaturated long-chain fatty acids, n-6 long-chain polyunsaturated fatty acids, and trans fatty acids are important components of the Western diet. These fatty acids are linked to fetal nutrition, membrane function, synthesis of prostaglandins and other eicosanoids, and inflammation, all of which are important to the physiology of pregnancy.

We conducted this analysis to further our understanding of changes in maternal serum lipids and fatty acid profiles in overweight and obese women compared with normal weight women between the first and late second trimester of pregnancy. We also examined the impact of excess weight gain on the rate of change in maternal serum lipid profiles from the first to late second trimester. We hypothesized that changes in maternal serum lipids and fatty acid profiles between the first and second trimester in overweight/obese women may reveal pathophysiology that occurs in maternal obesity or excess weight gain, and that knowledge of these changes may improve our understanding of the increased risks for adverse pregnancy outcomes commonly seen in overweight and obese women.

Methods and Procedures

This was a secondary analysis conducted within a larger prospective cohort study designed to investigate the role of maternal nutritional influences on preterm birth at the University of Pittsburgh (Pittsburgh, PA). The parent study enrolled women from 2004–2011, and this analysis included women enrolled from 2007–2011. Women aged 14–50 years who were carrying singleton infants and planning to deliver at Magee-Womens Hospital of UPMC were eligible. Women with chronic hypertension, diabetes, renal disease, and rheumatologic disorders were excluded from the study. The Institutional Review Board of the University of Pittsburgh approved the protocol prior to study initiation and all participants provided informed consent.

Women were enrolled in the study at less than 13 weeks' gestation, and much of the study enrollment occurred in a clinic providing care for low-income women. Baseline demographic information and medical history were collected via a structured interview, and non-fasting maternal serum was collected at less than 13 weeks' gestation and again at 24–

28 weeks' gestation. Pregnancy outcomes including gestational age at delivery, birthweight, results of gestational diabetes screening, and diagnosis of hypertensive disorders of pregnancy (defined as new onset blood pressure >140/90 on two or more occasions 6 hours apart after 20 weeks' gestation) were recorded from the medical record.

Our primary exposure of interest was maternal pre-pregnancy overweight and obesity, and this was reported as an index of weight-for-height (body mass index, BMI) and defined as the weight in kilograms (kg) divided by the square of height in meters (kg/m²). Pre-pregnancy weight was ascertained by maternal self-report at the first study visit and height was measured via a stadiometer at the baseline visit. Participants were classified as overweight or obese if their BMI was ≥ 25.0 kg/m².¹⁷ Excessive early gestational weight gain was defined as gestational weight gain greater than the upper range of Institute of Medicine 2009 guidelines for each prepregnancy BMI category (underweight, normal weight, overweight, and obese).⁷ Specifically, the cut-off for excessive early gestational weight gain was determined by adding a first-trimester weight gain of 4.4 pounds (2.0 kg) plus second trimester weight gain per week of 1.3 pounds (0.6 kg) for underweight, 1.0 pounds (0.5 kg) for normal weight, 0.7 pounds (0.3 kg) for overweight, and 0.6 pounds (0.3 kg) for obese women.⁷

Study Participants

At the time of our analysis 741 women had been enrolled in the larger cohort study. A total of 225 women (30.4% of the overall cohort of women) who were enrolled in the study from 2007–2011 had serum lipids measured at less than 13 weeks' gestation (8.3 ± 1.9 weeks) and again at 24–28 weeks' gestation (26.0 ± 1.1 weeks) and were included in the analysis involving total cholesterol, HDL, LDL, and triglycerides. Of these patients, 53 (24%) were overweight and 82 (36% were obese). Serum fatty acid profiles were also assessed in 151 women. When compared to all women in the larger cohort, the 225 women who were included in this analysis were slightly younger (23.6 ± 4.5 vs. 24.9 ± 5.0 years, $p < 0.01$), of lower parity (2.7 ± 1.7 vs 3.3 ± 2.0 pregnancies, $p < 0.01$), and of higher body mass index (BMI) (28.7 ± 8.1 vs. 26.6 ± 6.6 kg/m², $p < 0.01$). The 151 women with fatty acids analyzed were similar to the 225 women in the lipid analysis with regards to age (23.6 ± 4.6 vs 23.6 ± 4.4 years, $p = 0.95$), parity (2.5 ± 1.8 vs 2.8 ± 1.6 pregnancies, $p = 0.21$), and BMI (28.8 ± 7.6 vs 28.6 ± 8.4 kg/m², $p = 0.82$).

Lipid Measurement

Serum total cholesterol, HDL cholesterol, LDL cholesterol, and triglyceride concentrations were determined enzymatically using specific reagents from Pointe Scientific (Canton, MI). The inter-assay variability was less than 10% for cholesterol, 10% for LDL cholesterol, and 8% for HDL cholesterol. The inter-assay variability for triglycerides was less than 5%. All samples were analyzed in duplicate and in a blinded fashion.

The following long-chain fatty acid concentrations were ascertained: total long-chain fatty acids, total monounsaturated long-chain fatty acids (MUFA), total long-chain polyunsaturated fatty acids (PUFA, C18_2n6, C18_3n6, C18_3n3, C20_2n6, C20_3n6, C20_4n6, C20_4n3, C20_5n3, C22_4n6, C22_5n3, C22_6n3, C22_5n6), total n-6 long-

chain polyunsaturated fatty acids, total n-3 polyunsaturated fatty acids, arachidonic acid (C20_4n6), docoheaxaenoic acid (DHA, C22_6n3), and eicosapentaenoic acid (EPA, C20_5n3). Lipids were extracted using the technique of Bligh and Dryer,¹⁸ and were measured by gas column chromatography in the Heinz Laboratory at the University of Pittsburgh using a previously described protocol.¹⁹

Statistical Analysis

Statistical analyses were completed using Stata 10 software package Special Edition (StataCorp LP, College Station, TX). Distribution of variables was tested for normality using Shapiro-Wilk *W*-test. Maternal serum lipids were compared between the first and second trimesters using paired t-tests, and differences in maternal serum lipids and other demographic variables were compared between normal weight and overweight/obese women using chi-squared and t-tests as appropriate. Change between the first and second trimester lipid levels was calculated by subtracting the first trimester value from the second trimester value for each study subject. This was normalized to the number of weeks between the first and second trimester by dividing the calculated difference in serum lipid levels by the number of weeks that elapsed between blood draws to calculate a rate of change/week. This calculation assumed that the rate of change per week in serum lipid values was linear. Linear regression was used to identify variables independently associated with each of the serum lipid values as well as the rate of change per week for cholesterol, LDL, HDL, and triglycerides. Variables with a p value <0.10, as well as those with known or suspected biologic importance were then included in multivariable regression models to investigate the relationship among covariates. A p-value <0.05 was considered statistically significant in all analyses, and unadjusted results are shown when no relevant confounding variables were identified.

Results

Table 1 shows the clinical characteristics of normal weight compared to overweight/obese women. Although overweight and obese women had a higher pre-pregnancy BMI, they gained significantly less weight from the beginning of pregnancy to the 24–28 week time period and from the beginning of pregnancy to delivery. Overweight and obese women were also of higher parity and had higher systolic and diastolic blood pressure at the time of their first study visit. Overweight and obese women demonstrated a lower frequency of smoking, although this did not reach statistical significance. Results for the 50 gram glucose challenge test at 24–28 weeks' gestation, gestational age at delivery, rates of preterm birth less than 37 weeks' gestation, birthweight, and hypertensive disorders of pregnancy were similar between overweight/obese and normal weight women. There were only two women diagnosed with gestational diabetes, so we were unable to compare rates of gestational diabetes between groups.

As shown in table 1, total cholesterol, LDL, HDL, and triglycerides increased from <13 weeks to 24–28 weeks' gestation in both normal weight and overweight/obese women. Analysis stratified by normal weight versus overweight/obese status demonstrated that there were marked differences in serum lipids between the two groups. At less than 13 weeks'

gestation overweight/obese women had higher total cholesterol, LDL cholesterol, and triglycerides. In contrast, HDL was lower in overweight/obese women when compared to normal weight women (Table 2). The magnitude and direction of these differences were unchanged when adjusted for smoking, parity, gestational age at blood draw, and maternal race (data not shown).

By the second trimester, total cholesterol was slightly higher in normal weight women when compared to overweight/obese women. There was also a trend towards higher HDL cholesterol in normal weight women and no difference in either LDL cholesterol or triglycerides between normal weight and overweight/obese women (Table 2). Adjustment of these results for parity, weight gain from pre-pregnancy to the time of the 24–28 week blood draw, race, and gestational age at the time of blood draw did not affect the magnitude and direction of these changes (data not shown).

The differences in maternal total cholesterol, LDL, HDL, and triglycerides between normal weight and overweight/obese women in the first and late second trimester suggested that these lipid values have markedly different trajectories between the two groups. Because of this, we sought to examine factors that were associated with the average weekly rate of change for each of these serum lipid values. Univariate analysis of overweight/obese status, smoking, parity, weight gain from pre-pregnancy until the 24–28 week blood draw, and maternal race demonstrated that overweight/obese status was the only variable significantly associated with both the average weekly rate of change in cholesterol (-1.1 , 95% CI $-1.3 - -0.6$, $p < 0.01$) and LDL (-0.6 , 95% CI $-1.0 - -0.2$, $p < 0.01$). Maternal race was associated with the average weekly rate of change in triglycerides (-0.9 , 95% CI $-1.6 - -.2$, $p = 0.02$), but had no significant effect on the relationship between the rate of change in triglycerides and maternal overweight/obese status. Because the relationship between maternal overweight/obese status and the rate of change in lipids were unchanged by any covariates, the results of the unadjusted analysis are shown demonstrating that overweight/obese women had a rate of increase in their total cholesterol and LDL that was significantly lower than the rate of increase in normal weight women (Figure 1). There were no differences in the rate of change in HDL cholesterol or triglycerides between normal weight and overweight/obese women (Figure 1). Furthermore, there was no difference in the weekly rate of change in maternal serum total cholesterol, LDL cholesterol, HDL cholesterol or triglycerides between women with nonexcessive or excessive weight gain in either normal weight or overweight/obese women (Table 3).

Finally, we examined differences in the maternal serum fatty acid profile between normal and overweight/obese women between the first and late second trimester of pregnancy. Total fatty acids increased from <13 weeks to 24–28 weeks in both normal weight and overweight/obese women. Concentrations of MUFA, PUFA, saturated fatty acids, omega-3 fatty acids, omega-6 fatty acids, arachidonic acid and DHA also increased between the first and late second trimesters in both normal weight and overweight/obese women, while EPA was unchanged. Maternal serum arachidonic acid levels were higher in overweight/obese women in both the first and late second trimester, but there were no significant differences in the concentrations of total fatty acids or any of the other subfractions of fatty acids between normal weight and overweight/obese women (table 4). In addition, there was no

difference in the weekly rate of change for each of the fatty acids between normal weight and overweight/obese women (data not shown).

Discussion

Overweight and obese pregnant women demonstrate a more atherogenic lipid profile in early pregnancy when compared to normal weight women. Maternal serum total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides increase in all women from the first to the late second trimester. However, overweight/obese women demonstrate a significantly blunted increase in maternal serum total cholesterol and LDL cholesterol between the first and late second trimesters. This results in slightly higher levels of total cholesterol in normal weight women by the late second trimester when compared to overweight and obese women with no difference between the two groups in LDL cholesterol. These results remained constant even after adjustment for factors such as maternal weight gain. Of note, the rate of change in maternal serum lipids between the first and late second trimester was not different in women with excessive compared to non-excessive weight gain. The incidence of pregnancy complications were similar between the normal weight and overweight/obese women in our study, indicating that the differences seen in overweight and obese women may represent a physiologic response to pregnancy.

Our results complement those recently published by Vahratian, et al, who examined longitudinal changes in maternal serum lipids in normal weight and overweight/obese women²⁰. They also demonstrated that overweight/obese women display marked alterations in the trajectory of maternal serum lipids, including a lower rate of change in LDL cholesterol and total cholesterol in overweight and obese women when compared to normal weight women. However, this cohort was homogenous with regards to race and socioeconomic status, and the authors did not assess the impact of maternal weight gain. We have demonstrated that this lower rate of increase in total and LDL cholesterol in overweight/obese women between the first and late second trimester is not explained by differences in maternal weight gain. Our results also provide unique insight into the similar composition of fatty acid profiles between normal weight and overweight/obese women during pregnancy with the exception of higher arachidonic acid concentrations in overweight/obese women.

Dyslipidemia in early pregnancy is significant because more atherogenic lipid profiles are associated with a variety of pregnancy complications, including spontaneous preterm birth,²¹ gestational diabetes,²² and pre-eclampsia.^{12, 23–24} However, most of the available data is cross sectional, and it is unknown what gestational age or duration of dyslipidemia is necessary to contribute to adverse pregnancy outcomes. The mechanisms linking early pregnancy dyslipidemia to pregnancy complications are incompletely understood. Shallow placentation has been associated with approximately one-third of spontaneous preterm births,^{25–26} and pre-eclampsia is also associated with abnormal implantation.²⁷ Factors such as maternal obesity¹⁴ and hypercholesterolemia^{28–29} may predispose to LDL oxidation, and previous work has demonstrated that oxidized LDL inhibited invasion of extravillous cytotrophoblasts in a concentration-dependent manner.³⁰ These data highlight one possible pathway whereby maternal hyperlipidemia in early pregnancy may influence placentation.

The physiology leading to different trajectories of total cholesterol and LDL cholesterol between normal weight and overweight/obese women are not fully elucidated. Triglycerides and cholesterol increase across normal pregnancy, but they do not differ significantly from non-pregnant controls until approximately 9 weeks' gestation.³¹ Our first trimester samples were measured at a mean of 8.3 weeks' gestation, indicating that these values likely represent pre-pregnancy physiology. In normal pregnancy, lipoprotein alterations appear to be related to changes in sex hormones. Increased plasma prolactin and estrogen are correlated with plasma and VLDL triglyceride concentrations,³² and progesterone is correlated with low-density lipoprotein cholesterol concentrations.³³ Maternal serum lipoprotein lipids and insulin resistance both increase in parallel to maternal plasma hormone levels in normal pregnancy,³²⁻³³ and maternal insulin resistance contributes to plasma lipid perturbations due to an increase in adipose tissue lipolysis. Increased lipolysis provides a surplus of plasma free fatty acid substrate for hepatic triglyceride synthesis and secretion. Additionally, maternal lipoprotein lipase activity declines in liver and adipose tissue, and this leads to decreased peripheral catabolism of VLDL triglycerides.³⁴ These physiologic adaptations may differ significantly between normal weight and overweight/obese individuals, and we speculate that altered first trimester physiology could lead to downstream alterations in placental function and physiologic adaptation in overweight and obese women. Maternal serum lipids changed at the same rate between women with and without excessive weight gain, suggesting that corrections to maternal dyslipidemia may need to occur prior to conception if they could improve outcomes. The majority of overweight and obese women in our study had normal pregnancy outcomes, and more work is needed to understand what are pathologic versus physiologic adaptations in serum lipids among overweight and obese women.

Total fatty acids and levels of MUFA, PUFA, saturated fatty acids, omega-3 fatty acids, omega-6 fatty acids, arachidonic acid, and DHA increased between the first and late second trimester of pregnancy consistent with increasing fetal requirements to ensure normal fetal growth and development.⁸ Fatty acids have important roles as an energy source, membrane building blocks, and regulators of gene expression. They are also the precursors of eicosanoids such as prostaglandins, prostacyclins, thromboxanes, and leukotrienes, and these signaling molecules regulate diverse processes including gene expression, cell differentiation, immunity, and inflammation. Higher levels of arachidonic acid in the first and late second trimester of pregnancy in overweight and obese women were the only significant differences between groups. Arachidonic acid is metabolized to both proinflammatory and anti-inflammatory molecules,³⁵⁻³⁶ and while arachidonic acid supplementation does not appear to have proinflammatory effects in healthy individuals, it may counter the anti-inflammatory effects of omega-3 fatty acid supplementation.³⁷ Our results demonstrate that levels of the majority of fatty acids are constant across a wide range of maternal weights, and the differences in arachidonic acid deserves further attention given the clinical importance of arachidonic acid in inflammatory pathways and fetal neurodevelopment.

One limitation of our study is the relatively small sample size, which hindered our ability to separately analyze women who were overweight and obese. In addition, there were a limited number of women with pregnancy complications such as pre-eclampsia or gestational

diabetes. There was a trend towards higher 50 gram glucose challenge test results and higher rates of preterm delivery in overweight/obese women, and it is possible that these differences would have been significant with a larger sample size. We are unable to comment on the differences in lipid trajectories between normal weight and obese women with pregnancy complications, but this is an important area of future investigation. In addition, we do not have lipid data for the third trimester so we are unable to comment on lipid trajectories over the entire pregnancy. Finally, the use of non-fasting lipids may introduce some variability into our results. For example, non-fasting triglycerides may vary by up to 20%, and LDL by 10%.³⁸ However, serum lipids are associated with disease risk on both the fasting and nonfasting state indicating that non-fasting lipids have clinical relevance.^{39–40}

One strength is that our sample was derived from an economically and racially diverse population, which increases the generalizability of our results. We are also unaware of any previous studies that have compared the maternal serum fatty acid profile between normal weight and overweight/obese women, providing novel insight into the physiologic changes in fatty acids seen in women of different weight categories.

We chose to focus on the first and late second trimester of pregnancy because understanding the physiologic differences that occur in early and mid-pregnancy is crucial for developing tools to identify women at increased risk as well as designing interventions prior to the onset of clinical disease. Our results help define the physiologic changes that occur in overweight and obese women when compared to normal weight women in the first and late second trimester of pregnancy. These findings may have important implications for the design of interventions, particularly with regards to possible adverse effects of early pregnancy lipids on placental implantation and development. These data are relevant in understanding the causes, contributors, prevention, and treatment of pre-eclampsia, disorders of fetal growth, and gestational diabetes mellitus. Future studies are required to determine whether screening for and intervening on early pregnancy dyslipidemia can improve pregnancy or postnatal outcomes.

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All authors participated in the study design. C.S. and J.C. carried out the data analysis and interpretation, and all authors were involved in writing the paper and had final approval of the submitted and published versions.

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What is already known about this subject?

- Maternal serum lipids increase throughout gestation to support normal fetal growth and development
- Obesity in pregnancy is associated with an increased risk for pregnancy complications, but the mechanisms underlying this risk are not completely understood

What this study adds

- To the best of our knowledge this is the first study to examine the impact of excessive maternal weight gain on maternal serum lipids
- There are limited data assessing the impact of maternal obesity on maternal serum lipid profiles
- These data contribute to our understanding of the physiologic adaptations to pregnancy in overweight and obese women

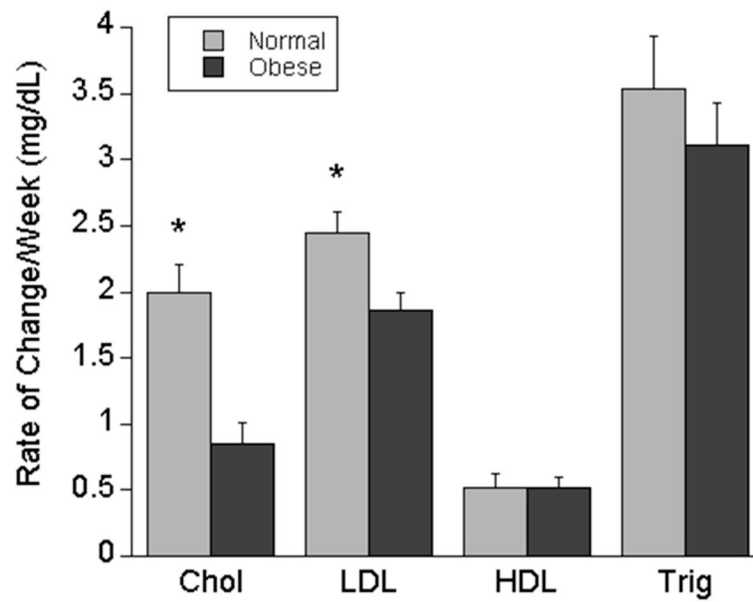


Figure 1.

Average change in maternal serum lipids/week in normal weight compared to overweight/obese women. Legend: * $p < 0.01$ normal weight compared to overweight/obese women. Chol (cholesterol), LDL (Low Density Lipoprotein), HDL (High Density Lipoprotein), Trig (triglycerides) 190×159mm (72 x 72 DPI)

Table 1

Maternal characteristics and pregnancy outcomes by lean vs. overweight/obese status

Demographics	Normal (n=90)	Overweight/Obese (n=135)	P
Age (years)	23.0 (\pm 3.7)	24.0 (\pm 4.9)	0.11
Parity			
1	27 (30)	27 (20)	0.03
2	30 (33)	35 (26)	
3	33 (37)	73 (54)	
Race			
White	34 (37.8)	43 (32.2)	0.18
Black	38 (42.2)	72 (54.1)	
Other	18 (20.0)	18 (13.5)	
Education (years)	12.2 (\pm 1.7)	12.5 (\pm 2.0)	0.15
Yearly Income			
<25,000	65 (77.4)	103 (77.4)	0.19
25,000–75,000	17 (20.2)	30 (22.6)	
>75,000	2 (2.4)	0	
BMI (mean)	21.6 (\pm 2.3)	33.4 (\pm 7.1)	<0.01
Total weight gain (lbs)	38.9 (\pm 16.8)	24.8 (\pm 17.0)	<0.01
Weight gain 24–28 weeks (lbs)	21.9 (\pm 12.3)	13.0 (\pm 14.2)	<0.01
Weight gain >IOM Recommendations at 24–28 weeks	50 (55.6)	66 (49.0)	0.36
Smoking	44 (49.4)	51 (38.1)	0.09
Any alcohol use	6 (6.7)	7 (5.2)	0.65
SBP	108.1 (\pm 10.7)	115.8 (\pm 7.5)	<0.01
DBP	65.2 (\pm 8.1)	70.7 (\pm 6.0)	<0.01
GA at enrollment	8.3 (\pm 2.0)	8.3 (\pm 1.8)	0.98
GA age at 24–28 week visit	26.1 (\pm 1.2)	25.9 (\pm 1.1)	0.17
50 g GCT results (mg/dL)	92.4 (\pm 21.3)	96.7 (\pm 22.1)	0.17
GA at delivery (weeks)	39.2 (\pm 1.7)	38.9 (\pm 2.3)	0.30
Preterm birth (<37 weeks)	5 (5.6%)	12 (8.9%)	0.35
Birthweight (grams)	3215 (\pm 556)	3227 (\pm 594)	0.86
Hypertensive disorders of pregnancy	8 (8.9%)	10 (7.4%)	0.67

All variables presented as mean (\pm standard deviation) or n (percent). BMI (Body Mass Index), lbs (pounds), IOM (Institute of Medicine), SBP (Systolic Blood Pressure), DBP (Diastolic Blood Pressure), GA (gestational age), GCT (glucose challenge test)

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Table 2

Maternal Serum Lipid Values in the 1st and 2nd Trimester

	1 st Trimester		P	2 nd trimester		P	P [^]	P*
	Normal Weight	Overweight/Obese		Normal Weight	Overweight/ Obese			
Total Cholesterol (mg/dL)	149.4 (±26.8)	161.3 (±29.6)	<0.01	184.1 (±28.1)	176.0 (±32.1)	0.05	<0.01	<0.01
LDL Cholesterol (mg/dL)	72.9 (±18.8)	80.0 (±19.9)	<0.01	115.5 (±23.5)	111.0 (±24.2)	0.17	<0.01	<0.01
HDL Cholesterol (mg/dL)	47.6 (±11.5)	43.6 (±10.4)	<0.01	56.9 (±16.0)	53.0 (16.9)	0.08	<0.01	<0.01
Triglycerides (mg/dL)	69.7 (±40.3)	81.7 (±47.2)	0.05	134.0 (±55.6)	138.3 (±59.8)	0.59	<0.01	<0.01

All variables presented as mean (± standard deviation). LDL (Low Density Lipoprotein), HDL (High Density Lipoprotein). P[^] is the p-value comparing first and second trimester lipid results in normal weight women, while P* represents the same comparison in overweight/obese women

Table 3
Weekly Increase in Maternal Serum Lipid Values by Appropriate or Excessive Weight Gain

	Normal Weight		P	Overweight/Obese		P
	Nonexcessive Weight Gain (n=40)	Excessive Weight Gain (n=50)		Nonexcessive Weight Gain (n=68)	Excessive Weight Gain(n=66)	
Cholesterol (mg/dL)	2.1 (\pm 1.6)	1.9 (\pm 2.0)	0.62	0.7 (\pm 2.1)	1.0 (\pm 1.7)	0.33
LDL Cholesterol (mg/dL)	2.4 (\pm 1.5)	2.5 (\pm 1.7)	0.85	1.9 (\pm 1.7)	1.8 (\pm 1.5)	0.79
HDL Cholesterol (mg/dL)	0.5 (\pm 0.9)	0.5 (\pm 0.9)	0.97	0.5 (\pm 0.8)	0.5 (\pm 0.9)	0.77
Triglycerides (mg/dL)	3.9 (\pm 3.5)	3.2 (\pm 4.1)	0.44	3.2 (\pm 3.8)	3.1 (\pm 3.3)	0.88

All variables presented as mean (\pm standard deviation). LDL (Low Density Lipoprotein), HDL (High Density Lipoprotein)

Table 4

Maternal Serum Fatty Acid Values in the 1st and 2nd Trimester

	1 st Trimester			2 nd Trimester			P	P [^]	P*
	Normal (n=62)	Overweight/ Obese (n=89)	P	Normal (n=62)	Overweight/ Obese (n=89)	P			
Total fatty acids (mg/dL)	230.2 (±47.4)	234.2 (±56.7)	0.65	367.1 (±86.4)	356.1 (±80.5)	0.43	<0.01	<0.01	
MUFA (mg/dL)	43.9 (±13.1)	44.1 (±15.2)	0.91	73.0 (±24.4)	70.4 (±21.9)	0.49	<0.01	<0.01	
PUFA (mg/dL)	110.2 (±22.3)	111.0 (±23.6)	0.83	162.7 (±37.8)	158.5 (±33.5)	0.48	<0.01	<0.01	
Saturated fatty acids (mg/dL)	74.9 (±16.0)	78.0 (±20.2)	0.32	129.4 (±31.2)	125.7 (±31.3)	0.48	<0.01	<0.01	
Omega 3 fatty acids (mg/dL)	8.0 (±3.1)	8.2 (±2.3)	0.59	12.6 (±3.7)	12.2 (±3.4)	0.50	<0.01	<0.01	
Omega 6 fatty acids (mg/dL)	102.2 (±20.6)	102.8 (±21.8)	0.87	150.1 (±35.3)	146.3 (±31.1)	0.49	<0.01	<0.01	
Arachidonic Acid (204:n6) (mg/dL)	22.2 (±5.0)	24.3 (±5.5)	0.02	28.3 (±5.9)	30.6 (±7.0)	0.04	<0.01	<0.01	
DHA (226:n3) (mg/dL)	4.5 (±1.9)	4.8 (±1.4)	0.26	7.4 (±2.3)	7.8 (±2.0)	0.24	<0.01	<0.01	
EPA (205:n3) (mg/dL)	0.79 (±0.75)	0.77 (±0.40)	0.83	0.81 (±0.47)	0.73 (±0.46)	0.29	0.79	0.50	

All data presented as mean (±standard deviation). MUFA (monounsaturated fatty acid), PUFA (polyunsaturated fatty acid), DHA (Docosahexaenoic acid), EPA (Eicosapentaenoic acid). P[^] is the p-value comparing first and second trimester fatty acid results in normal weight women, while P* represents the same comparison in overweight/obese women