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RESEARCH ARTICLE

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Epidemiological analysis of maternal lipid levels during the second trimester in pregnancy and the risk of adverse pregnancy outcome adjusted by pregnancy BMI

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Background: Pregnancy is accompanied by profound changes in lipid metabolism. We aimed to assess whether effects of second trimester body mass index and maternal lipid concentrations are associated with an increased risk of adverse pregnancy outcomes.

Methods: We investigated the serum levels of maternal lipids during the second trimester in pregnancy, and analyzed associations between the lipid levels and the risk of adverse pregnancy outcome. Seven hundred and seventy-four pregnant women were enrolled in this study between February 2016 and June 2016. Multivariate logistic regression analysis was conducted to estimate the relative risk between maternal lipids and adverse pregnancy outcome.

Results: Compared with the control group, during the second trimester of pregnancy, BMI, TG, and Lp(a) were risk factors for gestational diabetes mellitus; middle trimester pregnancy BMI, Lp(a), and APO-B were risk factors for pre-eclampsia; second trimester BMI and TG/HDL-C were risk factors for macrosomia; age and Lp(a) were uterine atony postpartum hemorrhage risk factors, while APO-AI was a protective factor of uterine inertia and postpartum hemorrhage; second trimester BMI, TCH, Lp(a), and TG/HDL-C were risk factors for fetal distress, while parity was a protective factor against fetal distress.

Conclusion: Abnormal blood lipid levels in pregnancy are significantly associated with GDM, pre-eclampsia, and other adverse pregnancy outcomes.

KEYWORDS
adverse pregnancy outcome, risk factors, serum lipid

1 | INTRODUCTION

Pregnancy is accompanied by profound changes in lipid metabolism.[1-3] The early phase of pregnancy can result in increased triglycerides (TG) as a consequence of increased lipogenesis and suppressed lipolysis, whereas the mid-phase of pregnancy can enhance lipolysis and elevate fatty acid concentrations.[4,5] Compared with non-pregnant women, the levels of leptin and insulin are significantly increased in pregnant woman.[6,7] Due to insulin resistance and hormonal control, all serum lipid components gradually increase, with some doubling in concentration. This change in lipid metabolism represents a physiological adaptation in the mother's body that involves switching from glucose metabolism to an increased preference for lipid metabolism to preserve glucose for fetal growth and development, which allows generation of fetal cellular membranes, steroid hormones, and bile acids.

Although hyperlipidemia is the physiological norm during human pregnancy, in complicated pregnancies the mechanisms regulating physiologic hyperlipidemia may malfunction, which has been associated with adverse pregnancy outcomes such as gestational diabetes

mellitus (GDM) or pre-eclampsia (PE).[8,9] Abnormal lipid profiles and species may have a role in the promotion of oxidative stress and vascular dysfunction observed in pre-eclampsia.[10] GDM shares a similar pathophysiology with type 2 diabetes. Patients with insulin resistance and type 2 diabetes tend to have lipid and lipoprotein abnormalities, including elevated TG, lower high-density lipoprotein cholesterol (HDL-C), and higher low-density lipoprotein cholesterol (LDL-C).[8]

There have been some studies that have proposed that lipids change throughout pregnancy according to pre-pregnancy status,[11] and that maternal lipid levels during early pregnancy are related to GDM or pre-eclampsia.[8,9] Middle trimester pregnancy BMI and blood lipids have not been analyzed for their potential impact upon pregnancy outcomes. Considering that maternal weight gain early in pregnancy is disproportionately due to fat deposition, this could influence subsequent maternal insulin resistance.[12] Furthermore, the rate of development of the fetal-placental unit is fastest in the second trimester of pregnancy, where blood lipid levels increase greatly. Therefore, maternal BMI during the second trimester in pregnancy was also included in our study, which has not been reported before. We aimed to assess whether effects of second trimester BMI during pregnancy and maternal lipid concentrations including free fatty acids (FFA) and lipoprotein (a) [Lp(a)], were independently associated with an increased risk of adverse outcomes.

2 | METHODS

2.1 | Subjects

A total of 774 singleton pregnancies women with a live delivery between February 2016 and June 2016 at the Obstetrics & Gynecology Hospital of Fudan University (Shanghai, China) were enrolled in this study. Participants included 456 normal pregnancies in women and pregnancies in women with adverse pregnancy outcome were divided into five groups: gestational diabetes mellitus (GDM) (n = 88), pre-eclampsia (n = 62), uterine inertia and postpartum hemorrhage (n = 42), macrosomia (n = 68), and fetal distress in the uterus (n = 58).

BMI was calculated at weeks 14 through 18 and pre-eclampsia was diagnosed at weeks 34 through 39 of gestation, using the current American College of Obstetricians and Gynecologists (ASOG) guidelines.[13] GDM was diagnosed by universal screening and macrosomia was defined as birthweight >4000 g.

Study exclusion criteria were as follows: established type 1 or type 2 diabetes; established hyperlipidemia, hypertension, cardiovascular diseases, or metabolic syndrome before pregnancy; a history of severe systemic disease such as liver cirrhosis, chronic renal failure, severe anemia, or immune disorders; and untreated endocrinopathies (hyperadrenalism, hypoadrenalism, and hyperthyroidism, or hypothyroidism), age <18 or ≥45 years, or patient had no complete maternal and infant record. None of the participants were taking any form of lipid-lowering drugs, before samples were collected.

This study conformed to the principles of the Declaration of Helsinki. Approval was obtained from the Research Ethics Committee of the Obstetrics & Gynecology Hospital of Fudan University (approval number: Kyy2016-22 on February 29, 2016), and written consent was obtained from all women in this study.

2.2 | Serum samples

Five milliliters of peripheral blood were collected from fasting participants between weeks 24 and 28 of gestation. The peripheral blood was collected in a serum separator tube and samples were allowed to clot for 30 minutes before centrifugation at 1000 × *g* for 5 minutes. All peripheral blood samples were processed within 2 hours of collection.

2.3 | Biochemical analyses

TG, total cholesterol (TCH), HDL-C, LDL-C, apolipoprotein AI (APO-AI), apolipoprotein B (APO-B), FFA, and Lp(a) were analyzed by an automatic biochemical analyzer (Hitachi 7180, WAKO) using commercially available kits. TG and TCH were determined by the HMMPS method (WAKO, Japan), HDL-C and LDL-C were tested by the direct assay method (Shanghai Beijia Biochemistry Reagents Co., Ltd. China), APO-AI and APO-B were tested using the immune transmission turbidity method (Shanghai Beijia Biochemistry Reagents Co., Ltd. China), non-esterified fatty acid was tested using the enzyme POD end-point method (DiaSys Diagnostic, Germany), and lipoprotein (a) was tested by immune turbidimetry of the latex-enhanced method (Shanghai HuaChen Biochemistry Reagents Co., Ltd. China). Interassay coefficients of variation (CV%) were less than 10% for all these assays.

2.4 | Statistical analysis

All statistical analyses were performed using GraphPad Prism version 5.0 for Windows (GraphPad Software, USA). Data are expressed as mean ± SE. For data with normal distribution and homogeneity of variance, an independent-sample *t* test was adopted to compare differences between two groups. For non-parametric data, differences between groups were evaluated using the Mann-Whitney U test. Associations between second trimester maternal lipid level and the risk of adverse pregnancy outcome were tested by multivariate logistic regression analysis. Variable selection in multivariable modeling was based on clinical and statistical significance. $P < .05$ was considered a statistically significant difference.

3 | RESULTS

The clinical data and outcomes for mothers and neonates were obtained from clinical records. All patient characteristics are described in Table 1. The proportion of participants who smoked cigarettes and consumed alcohol was 0%.

Table 1 shows that some adverse outcomes were significantly higher in those who were older during pregnancy (GDM 31.86 ± 4.32,

TABLE 1 Characteristics of mothers and infants

	GDM (n = 88)	PE (n = 62)	Macrosomia (n = 68)	Uterine inertia (n = 42)	Fetal distress (n = 58)	Control (n = 456)
Maternal						
Age (years)	31.86 ± 0.47***	30.98 ± 0.46*	29.94 ± 0.35	32.77 ± 0.72***	30.46 ± 0.33	29.98 ± 0.21
BMI (Kg/m^2)	23.97 ± 0.5***	23.69 ± 0.69**	22.93 ± 0.56**	21.84 ± 0.83	22.18 ± 0.43***	21.22 ± 0.14
Parity	1.24 ± 0.05	1.16 ± 0.05	1.21 ± 0.05	1.38 ± 0.08*	1.04 ± 0.03***	1.19 ± 0.03
Gravidity	1.91 ± 0.099	1.76 ± 0.13	1.68 ± 0.28	2.33 ± 0.25	1.65 ± 0.14	1.46 ± 0.09
Gestational age of BMI	16.61 ± 0.13	17.26 ± 0.19	16.68 ± 0.15	16.67 ± 0.37	16.93 ± 0.15	16.65 ± 0.17
Education level (%)						
University and above	39.8	37.1	38.2	38.1	37.9	39.2
Junior college	37.5	38.7	41.2	33.3	39.7	38.4
High school and under	22.7	24.2	20.6	28.6	22.4	22.4
Monthly Income of family (yuan) (%)						
>8000	54.5	59.7	58.8	59.5	53.7	55.3
4000-8000	28.4	24.2	26.5	26.2	27.6	29.6
<4000	17.1	16.1	14.7	14.3	18.7	15.1
Occupation (%)						
Office staff	42.0	40.3	42.6	40.5	43.1	43.9
Worker	14.8	12.9	11.8	16.7	12.0	14.0
Service personnel	20.5	22.6	20.6	19.0	19.0	20.2
Other	22.7	24.2	25.0	23.8	25.9	21.9
Infant						
Gestational age (weeks)	38.91 ± 0.14	38.31 ± 0.21	39.21 ± 0.57	39.27 ± 0.3	38.73 ± 0.39	39.58 ± 0.12
Birthweight (g)	3348 ± 42.1	3276 ± 70.5	4192 ± 23.9***	3598 ± 149	3201 ± 95.6	3363 ± 39.3

*P < .05,**P < .01, ***P < .001, compared with normal control.

TABLE 2 Maternal lipid levels between weeks 24 and 28 of gestation in normal pregnancy and pregnancy with adverse outcome

	GDM (n = 88)	PE (n = 62)	Macrosomia (n = 68)	Uterine inertia (n = 42)	Fetal distress (n = 58)	Control (n = 456)
TG (mmol/L)	4.49 ± 0.16***	4.78 ± 0.03**	4.44 ± 0.23**	4.1 ± 0.23	5.62 ± 0.47***	3.75 ± 0.09
TCH (mmol/L)	6.2 ± 0.09	6.70 ± 0.13***	6.10 ± 0.14	5.97 ± 0.16	6.97 ± 0.21**	6.17 ± 0.06
FFA (mmol)	0.42 ± 0.02	0.46 ± 0.02*	0.39 ± 0.02	0.40 ± 0.02	0.42 ± 0.03	0.39 ± 0.01
LP(a) (mg/L)	166.6 ± 17.8**	176.3 ± 20.0**	114.3 ± 13.6	156.5 ± 20.16*	169.88 ± 25.94*	111.9 ± 5.7
HDL-C (mmol/L)	1.38 ± 0.02	1.5 ± 0.03***	1.36 ± 0.04	1.33 ± 0.04	1.51 ± 0.03***	1.38 ± 0.01
LDL-C (mmol/L)	3.66 ± 0.08	4.1 ± 0.09***	3.64 ± 0.1	3.38 ± 0.12**	3.97 ± 0.11*	3.7 ± 0.04
LDL-C/HDL-C	2.66 ± 0.04	2.73 ± 0.01	2.61 ± 0.08	2.60 ± 0.07	2.63 ± 0.05	2.68 ± 0.02
APO-AI(g/L)	1.4 ± 0.03	1.40 ± 0.03	1.38 ± 0.03	1.26 ± 0.03***	1.50 ± 0.06	1.4 ± 0.01
APO-B(g/L)	1.3 ± 0.03**	1.42 ± 0.04***	1.27 ± 0.03	1.23 ± 0.04	1.61 ± 0.11**	1.22 ± 0.01
TG/HDL-C	3.25 ± 0.12**	3.19 ± 0.19	3.36 ± 0.17**	3.0 ± 0.21	3.78 ± 0.31**	2.77 ± 0.07

*P < .05,**P < .01,***P < .001, compared with normal control.

PE 30.98 ± 3.61, uterine inertia 32.77 ± 4.48 vs control 29.98 ± 3.16, P < .05). BMI during the second trimester of pregnancy was significantly associated with some adverse outcomes (GDM 23.97 ± 3.3, PE 23.69 ± 3.51, macrosomia 22.93 ± 3.38, fetal distress 22.18 ± 3.07 vs control 21.22 ± 2.08, P < .05) compared to normal pregnant women. Parity in pregnancies was significantly different for uterine inertia or

fetal distress (uterine inertia 1.38 ± 0.49, fetal distress 1.04 ± 0.19 vs control 1.19 ± 0.41, $P < .05$) compared to normal pregnant women.

Table 2 shows that levels of serum TG increased in pregnancy with GDM, PE, macrosomia, and fetal distress; TCH and HDL-C increased in pregnancy with PE and fetal distress; FFA increased in pregnancy with PE; LP(a) increased in pregnancy with GDM, PE, uterine inertia, and fetal distress; APO-AI increased in pregnancy with uterine inertia; APO-B increased in pregnancy with GDM, PE, and fetal distress; and TG/HDL-C increased in pregnancy with GDM, macrosomia, and fetal distress compared with normal pregnant women. The difference in LDL-C/HDL-C between pregnant women with adverse outcomes and controls was not significant.

Factors with significant differences from the univariate analysis were included in the regression analysis. Results for each of the

TABLE 3 Multiple logistic regression analysis for the risk of adverse pregnancy outcome

	β	SE	WaldS	*P*	Odds ratio	95% CI	
						Lower	Upper
GDM							
Age	0.071	0.06	1.24	.27	1.07	0.95	1.22
BMI	0.42	0.08	30.5	.00	1.52	1.31	1.77
TG	1.09	0.47	5.37	.02	2.99	1.18	7.53
LPa	0.89	0.32	7.78	.01	2.43	1.30	4.54
APO-B	0.32	0.39	0.67	.41	0.73	0.34	1.55
TG/HDL-C	0.02	0.40	0.001	.97	1.02	0.46	2.22
PE							
Age	−0.072	0.083	0.74	.39	0.93	0.79	1.10
BMI	0.38	0.10	14.33	.00	1.46	1.20	1.78
TG	0.12	0.64	0.03	.86	1.12	0.32	3.90
TCH	0.01	0.88	0.00	.99	0.99	0.18	5.53
FFA	0.01	0.34	0.001	.97	1.01	0.52	1.97
LPa	0.01	0.002	5.87	.02	1.01	1.0	1.02
HDL-C	0.11	0.89	0.02	.90	1.12	0.20	6.36
LDL-C	0.17	0.68	0.06	.80	1.19	0.31	4.52
APO-B	1.18	0.60	3.84	.05	3.27	1.07	10.67
Macrosomia							
BMI	0.27	0.08	12.16	.00	1.31	1.13	1.53
TG	0.08	0.42	0.03	.86	0.93	0.41	2.12
TG/HDL-C	0.90	0.45	4.12	.04	2.47	1.03	5.90
Uterine inertia							
Age	0.26	0.07	14.72	.00	1.30	1.14	1.49
Parity	0.22	0.47	0.23	.64	1.25	0.50	3.11
APO-AI	−1.16	0.30	14.98	.00	0.31	0.17	0.56
LPa	0.005	0.002	7.31	.01	1.01	1.00	1.02
LDL-C	0.20	0.30	0.43	.51	1.22	0.67	2.21
Fetal distress							
BMI	0.46	0.09	28.56	.00	1.60	1.34	1.88
Parity	−2.75	0.91	9.08	.003	0.06	0.011	0.38
TCH	1.28	0.58	4.92	.03	3.59	1.16	11.10
LPa	0.01	0.002	8.10	.004	1.01	1.00	1.02
TG/HDL-C	0.71	0.33	4.78	.03	2.03	1.08	3.85
TG	0.74	0.53	1.91	.17	2.09	0.73	5.93
HDL-C	−0.38	0.72	0.28	.60	0.68	0.17	2.78
LDL-C	0.38	0.57	0.45	.50	1.46	0.48	4.43
APO-B	0.91	0.53	2.96	.09	0.41	0.14	1.14

Adjusted for age, BMI, parity, gravidity, gestational age of BMI, education, family income, and occupation.

individual lipid markers during pregnancy were divided into 3 groups: low quartile (levels lower than 25th percentile); intermediate (between 25th and 75th percentiles); and upper quartile (levels higher than 75th percentile). Multifactorial logistic regression analysis of each procedure was conducted based on univariate analyses. After adjustment for all confounding factors, compared with the control group, second trimester BMI (OR = 1.52, 95% CI = 1.31-1.77), TG (OR = 2.99, 95% CI = 1.18-7.53), and Lp(a) (OR = 2.43, 95% CI = 1.30-4.54) were risk factors for gestational diabetes mellitus; second trimester BMI (OR = 1.46, 95% CI = 1.20-1.78), Lp(a) (OR = 1.01, 95% CI = 1.0-1.02), and APO-B (OR = 3.27, 95% CI = 1.07-10.67) were risk factors for pre-eclampsia; second trimester BMI (OR = 1.31, 95% CI = 1.13-1.53) and TG/HDL-C (OR = 2.47, 95% CI = 1.03-5.90) were risk factors for macrosomia; age (OR = 1.30, 95% CI = 1.14-1.49) and Lp(a) (OR = 1.01, 95% CI = 1.0-1.02) were uterine atony postpartum hemorrhage risk factors, and APO-AI (OR = 0.31, 95% CI = 0.17-0.56) was a protective epidemiological factor for uterine inertia and postpartum hemorrhage; second trimester BMI (OR = 1.60, 95% CI = 1.34-1.88), TCH (OR = 3.59, 95% CI = 1.16-11.10), Lp(a) (OR = 1.01, 95% CI = 1.0-1.02), and TG/HDL-C (OR = 2.03, 95% CI = 1.08-3.85) were risk factors for fetal distress, while parity (OR=0.06, 95% CI=0.011-0.38) was a protective factor for fetal distress; these differences were statistically significant ($P < .05$) (Table 3).

4 | DISCUSSION

There is a significant rise in serum levels of TG, TCH, APO-AI, APO-B, HDL-C, and LDL-C from the first to second trimester of normal pregnancy.[10] We selected second trimester pregnancy lipids at 20-27 weeks of gestation for study. To date, we believe this is the first study conducted to assess effects of second trimester maternal lipid concentrations on adverse pregnancy outcomes, which were adjusted by second trimester BMI at 14-18 weeks. There were similarities and dissimilarities between our findings and other previously published studies.

4.1 | Pregnancy BMI at 14-18 weeks

Our findings showed that pregnancy BMI measured at the time of 14-18 weeks was significantly increased and independently and significantly associated with the odds of GDM, PE, macrosomia, and fetal distress in the uterus compared to healthy, pregnant women.

Some other studies have also reported that women with excessive gestational weight gain (GWG) had a several-fold higher risk of GDM, pregnancy-induced hypertension, cesarean delivery, large-for-gestational age infant, and macrosomia, compared with women with normal adequate GWG.[14,15]

4.2 | TG and TG/HDL-C

Our findings showed that TG and TG/HDL-C ratios measured at the time of 24-28 weeks were significantly increased in GDM, macrosomia, and fetal distress in uterus, compared to healthy pregnant women, which is consistent with other studies[12,16-18]. Multivariable logistic regression analysis showed that TG was independently and significantly associated with the odds of GDM, while TG/HDL-C was independently and significantly associated with the odds of macrosomia and fetal distress in the uterus, which is a different phenomenon reported in other reports.[17,19]

Similar to our results, high levels of TG have been shown to be associated with increased GDM risk.[16,17] Another study did not observe such positive associations between GDM and any lipid profile changes but in patients with glucose intolerance, decreased TCH and LDL-C concentrations, and increased TG concentrations were detected.[19] One study noted a low HDL-C pattern in women with GDM,[20] whereas another study reported that HDL-C does not change significantly during gestation even in GDM patients.[17] The TG/HDL-C ratio has been demonstrated to be a valuable index for identifying pregnant women with low risk of GDM before 24 weeks of gestation.[18] TG/HDL-C ratios measured and calculated at the time of 24-28 weeks were independently and significantly associated with the odds of GDM.[12]

In our study, GDM and macrosomia had higher TG/HDL-C but the difference in HDL-C between GDM, macrosomia, and controls was not significant in univariate analyses. However, only second trimester BMI, TG, and LP(a) were identified as significant independent predictors of GDM in multivariate logistic regression analysis.

Although maternal circulating TG do not directly cross the placenta, the presence of lipoprotein receptors, fatty acid–binding proteins, and different lipase activities in the placenta allows the efficient transfer of maternal fatty acids to the fetus.[12] Recent studies have reported highly significant positive correlations between fasting maternal TG levels at late gestation and birthweight in both non-diabetic pregnancies and well-controlled GDM pregnancies.[16,21] Furthermore, other investigators have reported that the increase in TG from early to late pregnancy is the most predictive factor for neonatal adiposity.[22]

In our study, macrosomia was associated with higher second trimester BMI, TG, and TG/HDL-C using univariate analyses. However, only second trimester BMI and TG/HDL-C were identified as significant independent predictors of macrosomia using multivariate logistic regression analysis.

4.3 | LP(a)

Our findings showed that LP(a) measured at the time of 24-28 weeks was significantly increased and independently and significantly associated with the odds of GDM, pre-eclampsia, uterine inertia and postpartum hemorrhage, and fetal distress in the uterus compared to healthy pregnant women.

Lp(a) is a subclass of lipoprotein, consisting of a low-density lipoprotein covalently bound via its apolipoprotein B100 portion to apolipoprotein (apo) (a)..[23] The apo(a) element of Lp(a) has a structure similar to plasminogen, allowing Lp(a) particles to reduce the fibrinolytic activity of plasminogen by competitively binding endothelial

plasminogen receptors. Lipoprotein (Lp) (a) can induce thrombosis making it potentially important in the course of normal and complicated pregnancies.[24]

The studies evaluating Lp(a) in pre-eclamptic patients differentially showed an increased, decreased, or equivalent level of Lp(a) in pre-eclamptic patients compared to healthy pregnant controls.[25-30] Three studies showed an increased level of Lp(a) in subjects with pre-eclampsia compared to healthy pregnant controls.[25-28] One of the studies showed a decreased level of Lp(a) in subjects with severe pre-eclampsia. This may be due to more extensive endothelial damage in severe pre-eclampsia and hence higher consumption of Lp(a) as an acute-phase protein.[27] Other studies showed that there was no difference in Lp(a) levels in subjects with pre-eclampsia compared to healthy controls.[29,30] An explanation postulated by Manten et al, who examined women with a history of pre-eclampsia (rather than experiencing pre-eclampsia at the time of blood sampling), is that the rise in Lp(a) may be transient during pregnancy.[29]

4.4 | APO-B

Our findings showed that APO-B measured at the time of 24-28 weeks was significantly increased in GDM, pre-eclampsia, and fetal distress in the uterus, compared to healthy pregnant women. Multivariable logistic regression analysis showed that APO-B was independently and significantly associated with the odds of pre-eclampsia.

Some research studies have shown a pre-eclampsia–dyslipidemic pattern of increased triglycerides, cholesterol, LDL-C, and decreased HDL-C concentrations.[9,10] In the pre-eclampsia group, the LDL-C and APO-B ratio were also significantly reduced during the third trimester, where the reduced ratio was due to an increase in other APO-B containing lipoproteins.[10]

4.5 | APO-AI

Serum APO-AI was increased significantly during pregnancy compared with the non-pregnant state, although interestingly serum HDL-C-cholesterol was not significantly different. APO-AI is the predominant apolipoprotein in HDL-C particles and this would suggest that structural differences occur in the composition of HDL-C particles during pregnancy.[23]

Our findings showed that APO-AI measured at the time of 24-28 weeks was significantly increased in uterine inertia and postpartum hemorrhage, compared to healthy pregnant women. Multivariable logistic regression analysis showed that APO-AI was an epidemiologically protective factor for uterine inertia and postpartum hemorrhage.

4.6 | Parity

Compared with primigravida, the proportion of fetal distress was significantly lower in multigravida.[31] Our findings showed that parity was associated with a significant decrease in fetal distress in the uterus, compared to healthy pregnancy women. Multivariable logistic regression analysis showed that parity was an epidemiological protective factor for fetal distress in the uterus.

4.7 | Limitations

We investigated the effect of second trimester maternal lipid concentrations and adjusted for second trimester BMI. Discordance in the findings among the aforementioned studies may be due to a variety of reasons, including gestational week of specimen sampling, adjustment factors, confounders, sample size, study design, and variations in population characteristics.

Although we adjusted for various potential confounders, we cannot exclude the possible impact of other unmeasured covariates such as genotype, settlements, dietary factors, and ethnicity on lipid profiles as we did not collect these data in this study.

5 | CONCLUSION

The level of abnormal blood lipid moieties in pregnancy is significantly related to the outcome of gestational diabetes, pre-eclampsia, and other adverse pregnancy outcomes. Monitoring and early intervention of body mass index and blood lipid levels in pregnancy could have important clinical significance in reducing the complications of pregnancy while avoiding adverse pregnancy outcomes.

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