Original Article

Maternal and cord blood fatty acid patterns with excessive gestational weight gain and neonatal macrosomia

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Background and Objectives: This study evaluated the association of maternal excessive gestational weight gain with saturated and polyunsaturated fatty acid concentrations in maternal and cord serum. **Methods and Study Design:** We included 77 pairs of women and their newborns and classified them into three groups as follows: mothers with normal gestational weight gain and their babies with normal birth weight in group I (30 pairs), mothers with excessive gestational weight gain and their babies with normal birth weight in group II (30 pairs), and mothers with excessive gestational weight gain and their macrosomic babies in group III (17 pairs). Serum fatty acid concentrations were determined through gas chromatography–mass spectrometry. **Results:** No remarkable difference in maternal dietary intake was observed among the three groups. C16:0, C18:0, eicosapentaenoic acid, and docosahexaenoic acid concentrations were significantly higher in group III mothers than in group I mothers. Compared with group I neonates, total saturated and polyunsaturated fatty acid concentrations were significantly lower but total n-3 polyunsaturated fatty acid and docosahexaenoic acid concentrations were significantly higher in group II neonates (p<0.05). The n-6: n-3 ratio in maternal and cord serum was approximately 10:1 and 1.5:1, respectively. **Conclusion:** Women with excessive gestational weight gain who deliver a macrocosmic neonate tend to have higher total saturated fatty acid concentrations but lower docosahexaenoic acid concentrations in their neonate cord serum. Fatty acid concentrations in maternal and cord serum are not associated with maternal dietary pattern.

Key Words: saturated fatty acid, polyunsaturated fatty acid, gestational weight gain, macrosomia, birth weight

INTRODUCTION

Maternal excessive gestational weight gain (GWG) has been associated with an increased risk of adverse pregnancy outcomes, including fetal macrosomia, childhood obesity, and mental retardation.[1-4] Over the previous two decades, China has been undergoing economic transition and urbanization and has been adopting western dietary culture, which is characterized by a diet higher in total fat concentrations, particularly saturated fatty acids (SFAs) and n-6 fatty acids, and lower in n-3 fatty acid concentrations.[5,6] Therefore, the incidence of overweight pregnant women is increasing in China. From 1992 to 2010, the prevalence of overweight or obesity in women aged 18–44 years increased from 16.8% to 26.4% and from 3.1% to 9.0%, respectively, with a corresponding increase in macrosomia;[7] this implies that China is encountering an increase in macrosomia. A population survey including 594,472 singleton live births from 1994 to 2005 in southeast China showed that macrosomia increased from 6.0% in 1994 to 8.49% in 2000.[8] In addition, a survey in Beijing showed that the average GWG in 16,460 women was 17.1±4.9 kg, that average birth weight of babies was 3406±400 g, and that prevalence of macrosomia was 7.55% (1242/16460).[9] Bao et al reported that the incidence of macrosomia increased from 8.31% in 2001 to 10.5% in 2005 in the city of Harbin.[10] In Shanghai, the incidence of macrosomia increased by 50% between 1989 and 1999.[11]

SFAs might be crucial in modulating the effects of obesity-associated (FTO) gene polymorphisms,[12] which consistently interact with other gene polymorphisms associated with obesity.[13] Because of their tightly packed structure, SFAs increase low-density lipoprotein cholesterol (LDL-C) concentrations. By contrast, unsaturated fatty acids increase high-density lipoprotein cholesterol

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(HDL-C) concentrations by transporting LDL-C to the liver, where it is broken down and eliminated from the body. SFAs can activate toll-like receptor (TLR)-mediated proinflammatory signaling pathways.[14] Several studies have recently linked SFA-induced dysfunction of the endoplasmic reticulum (ER) with lipoapoptosis in hepatocytes.[15,16] Polyunsaturated fatty acids (PUFAs), including n-3 and n-6 PUFA, are crucial in fetal growth and development.[17,18] Evidence has shown that the relative intake of an individual PUFA might influence adipose tissue development. A study reported that maternal dietary intake of n-3:n-6 PUFA ratio is negatively associated with body fat mass, thus suggesting that a high serum n-3 PUFA concentration has beneficial effects on adipogenesis.[19] However, in this study, we investigated only the effect of total dietary fat on body fat mass; the effect of individual fatty acid intake on body fat mass should be further investigated. Little is known about the metabolic effects of maternal nutrition on fetal nutrition, birth weight, and excess adiposity in newborns.[20-22] To the best of our knowledge, few studies have explored the relationship between maternal or cord SFA and PUFA concentrations and macrosomia in women with excessive GWG. Maternal habits, demographic factors, and baby birth weight of Chinese women differ from those of western women.[23] In the present study, we evaluated the association between maternal excessive gestational weight gain and saturated fatty acid and concentration PUFAs in maternal and cord serum concentrations.

MATERIALS AND METHODS

Ethics statement

The study was conducted according to the guidelines of the Declaration of Helsinki, and all procedures were approved by the Ethics Committee of Anhui Medical University (No. 2010011). All parents signed an informed consent form for participation in the study.

Data source and study participants

We conducted a case–control study at the Maternal and Child Hospital in Hefei, the capital city in the Chinese province of Anhui, between October 2010 and June 2011. Maternal demographic and health parameter data were collected through a questionnaire upon entry into the study and through a review of medical records. Self-reported prepregnancy weight and height were noted at the initial visit. Prepregnancy body mass index (BMI) was calculated as [prepregnancy weight (kg)]/[height (m)]^2. GWG was calculated as prepregnancy weight subtracted from the measured weight recorded at the last prenatal visit before delivery. Gestational weeks were estimated from the date of the last menstrual period and were confirmed through ultrasonography. In addition, maternal age, and fasting blood glucose (FBG), serum triglyceride (TG), total cholesterol (TC), HDL-C, and LDL-C concentrations were analyzed upon entry into the study. Also, about dietary intake, mothers were requested by answer a food frequency questionnaire dealing with foods from a variety of groups, such as dairy products, vegetables, meat, fish, eggs or bean products. The inclusive criteria were ≥18 years of age at the expected date of delivery, full-term pregnancy (37–42weeks), singleton pregnancies, and the absence of diabetes and other complications. The information collected for newborns included sex, birth weight, and body length. The participants were classified into three groups according to the criteria based on recommended GWG and neonatal birth weight guidelines from the Institute of Medicine.[24] Finally, mothers with normal GWG and their newborns with normal body weight (NBW) were included in group I (30 pairs), mothers with excessive GWG and their newborns with NBW in group II (30 pairs), and mothers with excessive GWG and their macrosomic newborns (weighing at least 4000 g) in group III (17 pairs).

Gas chromatography–mass spectrometry analysis

Maternal and neonatal cord serums were collected and stored at −80°C. All chemical reagents used were of a chromatographic grade. A commercial fatty acid methyl ester (FAME) standard mixture (FAME Mix 37, Supelco) was purchased from Sigma (St. Louis, MO, USA). The corresponding 37 fatty acid standards were purchased from NU-CHEK Company (Minnesota, USA). The following fatty acids were measured in the serum: C8:0, C10:0, C12:0, C14:0, C15:0, C16:0, C17:0, C18:0, C20:0, C18:2n6, C18:3n6, C20:2n6, C20:3n6, C20:4n6, C18:3n3, C20:3n3, C20:5n3, and C22:6n3. Standard solutions and samples were prepared according to a reported method, and the heptadecanoic (C17:0) fatty acid was used as an internal standard.[25] Fatty acid concentrations were expressed as μmol/L. All gas chromatography–mass spectrometry (GC/MS) analyses were performed at a programmed temperature on an Agilent-7890A gas chromatograph equipped with an Agilent 7683 automated liquid sampler, split–splitless injector, and Agilent-5975C quadrupole mass selective detector. Mass spectra and retention times were acquired on the capillary column DB-23 (60 m, 0.25 mm, and 0.25 mm Agilent). Helium was used as the carrier gases at a flow rate of 1.0 mL/min. One-microliter aliquots were injected with a splitless ratio. Free FAMEs were separated at a constant flow with the following temperature program: (1) 45°C for 2 min, (2) increased to 105°C at 25°C/min, (3) 105°C for 2 min, (4) increased to 190°C at 15°C/min, (5)190°C for 12 min, and (6) increased to 230°C at 1.5°C/min. The injector and detector temperatures were 250°C and 260°C, respectively. The mass spectrometer was operated in the electron impact (EI) mode at 70 eV in the scan range of 50–500 m/z.

Statistical analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences software (SPSS) Version 16.0 (SPSS Inc., Chicago, IL, USA). The contents of fatty acids across the three groups are presented as medians (range, $P_{12.5}$–$P_{87.5}$). Differences in categorical variables were tested using chi-square test or Fisher's exact test. F-Test and t-Test were used to examine differences between demographic parameters of the participants, and Mann–Whitney U test and Kruskal–Wallis H test were employed for comparing fatty acid concentrations between groups. $p<0.05$ was considered statistically significant.

Table 1. Characteristics of pregnant women and their neonates in the three groups

Variable	Group I (n=30)	Group II (n=30)	Group III (n=17)
Maternal Characteristics			
Age (years)†	28.9 (4.83)	26.7 (3.21)*	30.6 (4.00)#
Height (cm)†	161 (3.87)	161 (3.72)	163 (5.32)
Pre-pregnancy weight (kg)†	53.0 (7.28)	53.0 (5.35)	57.5 (9.00)
Pre-pregnancy BMI (kg/m^2)†	20.5 (2.63)	20.3 (1.94)	21.8 (3.12)
Gestational weight gain (kg)†	14.6 (1.81)	20.5 (2.62)*	22.7 (5.94)*#
FBG (mmol/L)†	4.51 (0.53)	4.31 (0.50)	4.26 (0.74)
TC (mmol/L)†	6.60 (1.25)	6.51 (1.05)	6.67 (1.04)
TG (mmol/L)†	2.47 (0.81)	2.71 (1.04)*	3.38 (1.56)*#
HDL (mmol/L)†	2.27 (0.38)	2.26 (0.54)	2.44 (0.70)
LDL (mmol/L)†	3.48 (0.84)	3.40 (0.75)	3.30 (0.54)
Neonatal Characteristics			
Gestational age at delivery (weeks)†	39.0 (0.91)	39.3 (1.11)	39.3 (0.93)
Cesarean delivery‡	18 (60.0)	16 (53.3)	15 (88.2) *#
Birth weight (g)†	3353 (272)	3398 (280)	4144 (229)*#
Length (cm)†	50.5 (0.82)	50.8 (1.29)	52.8 (1.25)*#

BMI: body mass index; FBG: fasting blood glucose; TG: triglyceride; TC: total cholesterol; HDL: high-density lipoprotein; LDL: low-density lipoprotein. Group I: mothers with normal gestational weight gain and their babies with normal birth weight; Group II: mothers with excessive gestational weight gain and their babies with normal birth weight; Group III: mothers with excessive gestational weight gain and their macrosomic babies weighing at least 4,000 g.
Data were expressed as ‡number (percentage) and †mean (standard deviations).
*p<0.05, as compared with Group I, # p<0.05, as compared with Group II.

Table 2. Maternal diet during pregnancy among three groups

Food intake	Group I (n=30)	Group II (n=30)	Group III (n=17)	p value
Vegetables				0.271
<250 g/day	16 (53.3)	17 (56.7)	13 (76.5)	
≥250 g/day	14 (46.7)	13 (43.3)	4 (23.5)	
Fruit				0.266
<250 g/day	15 (50.0)	9 (30.0)	6 (35.3)	
≥250 g/day	15 (50.0)	21 (70.0)	11 (64.7)	
Meat				0.509
<250 g/week	10 (33.3)	6 (20.0)	5 (29.4)	
250-1000 g/week	17 (56.7)	16 (53.3)	9 (52.9)	
≥1000 g/week	3 (10.0)	8 (26.7)	3 (17.6)	
Fish				0.300
<250 g/week	13 (43.3)	8 (26.7)	8 (47.1)	
250-500 g/week	11 (36.7)	9 (30.0)	4 (23.5)	
≥500 g/week	6 (20.0)	13 (43.3)	5 (29.4)	
Eggs				0.410
<7 eggs/week	20 (66.7)	17 (56.7)	8 (47.1)	
≥7 eggs/week	10 (33.3)	13 (43.3)	9 (52.9)	
Milk				0.498
<1 cup/day	10 (33.3)	6 (20.0)	5 (29.4)	
≥1 cup/day	20 (66.7)	24 (80.0)	12 (70.6)	
Bean products				0.648
<250 g/week	10 (33.3)	8 (26.7)	4 (23.5)	
250-500 g/week	13 (43.3)	14 (46.6)	11 (64.7)	
≥500 g/week	7 (23.4)	8 (26.7)	2 (11.8)	

All data were expressed as number (percentage). Group I: mothers with normal gestational weight gain and their babies with normal birth weight, Group II: mothers with excessive gestational weight gain and their babies with normal birth weight; Group III: mothers with excessive gestational weight gain and their macrosomic babies weighing at least 4000 g.

RESULTS

The demographic parameters of 77 pairs of pregnant women and their newborns are listed in Table 1. No significant differences were observed in maternal height, prepregnancy weight, BMI, and FBG, TC, HDL-C, and LDL-C concentrations among the three groups (p>0.05), whereas significant differences were observed in maternal age, TG concentration, and delivery pattern (p<0.05). No significant difference was observed in maternal dietary intake (p>0.05, Table 2).

Seventeen fatty acids (SFAs and PUFAs) were measured in maternal and neonatal serum samples. SFA and PUFA concentrations in maternal serum in all three groups are presented in Table 3. Total SFA and C18:0 concentrations in groups II and III were significantly higher than those in group I, and C16:0 concentrations in

Table 3. Fatty acid status in maternal serum among three groups

Fatty acid	Group I (n=30)	Group II (n=30)	Group III (n=17)
Total SFA	138 (90.5, 186)	193 (121, 240)*	223 (102, 408)*
C8:0	1.52 (0.29, 3.94)	1.01 (0.05, 4.26)	0.73 (0.03, 3.95)
C10:0	0.41 (0.16, 0.89)	0.40 (0.20, 1.36)	0.34 (0.16, 1.45)
C12:0	2.05 (0.86, 4.11)	2.72 (0.90, 4.79)	2.90 (0.90, 5.28)
C14:0	0.60 (0.09, 1.11)	0.42 (0.03, 1.31)	0.23 (0.03, 1.65)
C15:0	0.32 (0.02, 0.81)	0.19 (0.01, 0.36)*	0.29 (0.00, 0.52)
C16:0	110 (73.2, 150)	116 (66.4, 146)	140 (68.8, 250)*
C18:0	17.4 (9.54, 57.6)	72.2 (40.0, 104)*	76.9 (30.16, 132)*
C20:0	0.10 (0.00, 0.31)	0.12 (0.00, 0.29)	0.17 (0.00, 0.29)
Total PUFA	92.3 (39.3, 212)	93.3 (49.2, 215)	88.9 (61.0, 218)
Total n-6 PUFA	84.0 (35.7, 198)	93.2 (42.4, 208)	84.5 (52.8, 105)
C18:2n6	68.2 (30.0, 139)	78.9 (36.8, 133)	65.7 (42.8, 140)
C18:3n6	1.28 (0.68, 1.94)	1.20 (0.89, 3.25)	1.74 (0.90, 3.64)
C20:2n6	6.72 (1.80, 8.58)	5.48 (1.80, 6.89)	7.96 (3.26, 9.64)
C20:3n6	2.13 (0.72, 2.78)	1.87 (1.23, 4.10)	2.27 (1.08, 4.83)
C20:4n6	5.00 (2.37, 8.67)	5.67 (3.28, 11.0)	6.71 (2.59, 13.5)
Total n-3 PUFA	8.35 (3.55, 11.3)	7.04 (3.49, 12.73)	8.98 (4.55, 17.8)
C18:3n3	5.09 (1.49, 6.93)	4.42 (0.88, 6.96)	5.08 (1.60, 11.6)
C20:3n3	0.32 (0.20, 0.42)	0.27 (0.21, 0.35)	0.33 (0.24, 0.45)#
C20:5n3	0.26 (0.18, 0.48)	0.26 (0.15, 1.43)	0.46 (0.17, 0.92)*
C22:6n3	2.69 (1.31, 3.84)	2.68 (1.56, 4.43)	3.58 (1.35, 6.55)*
n-6/n-3	10.4 (5.84, 21.7)	11.9 (5.94, 22.5)	10.1 (5.89, 18.6)

SFA: saturated fatty acid; PUFA: polyunsaturated fatty acid. Group I: mothers with normal gestational weight gain and their babies with normal birth weight, Group II: mothers with excessive gestational weight gain and their babies with normal birth weight, Group III: mothers with excessive gestational weight gain and their macrosomic babies weighing at least 4000 g.
All data were expressed as median ($P_{12.5}$, $P_{87.5}$).
*p<0.05, as compared with Group I, #p<0.05, as compared with Group II.

Table 4. Fatty acid status in cord serum among three groups

Fatty acid	Group I (n=30)	Group II (n=30)	Group III (n=17)
Total SFA	147 (98.6, 252)	109 (62.3, 211)*	139 (70.2, 262)
C8:0	1.36 (0.05, 4.73)	0.53 (0.19, 4.95)	0.65 (0.05, 4.95)
C10:0	0.51 (0.14, 1.03)	0.40 (0.07, 0.81)	0.49 (0.19, 0.75)
C12:0	13.1 (2.66, 27.3)	9.48 (3.97, 20.8)	9.83 (2.63, 19.1)
C14:0	0.14 (0.04, 0.28)	0.10 (0.04, 0.33)	0.16 (0.05, 0.28)
C15:0	0.15 (0.04, 0.31)	0.12 (0.05, 0.31)	0.15 (0.01, 0.31)
C16:0	61.4 (31.4, 104)	49.5 (20.1, 94.2)*	60.5 (28.9, 102)
C18:0	70.7 (38.8, 129)	50.5 (20.9, 93.2)*	69.7 (34.0, 139)
C20:0	0.10 (0.04, 0.20)	0.09 (0.03, 0.13)	0.09 (0.00, 0.19)
Total PUFA	50.9 (25.7, 94.7)	40.9 (15.9, 89.7)*	43.4 (13.2, 61.7)*
Total n-6 PUFA	30.9 (14.9, 51.8)	23.5 (10.3, 81.3)*	27.6 (9.72, 35.8)
C18:2n6	19.7 (8.94, 25.8)	11.7 (6.49, 37.95)	15.5 (5.49, 21.5)
C18:3n6	0.29 (0.17, 0.42)	0.26 (0.16, 0.50)	0.26 (0.17, 0.40)
C20:2n6	1.98 (0.87, 5.44)	3.75 (2.51, 6.81)*	4.12 (2.53, 6.34)*
C20:3n6	2.08 (0.93, 3.69)	1.58 (0.79, 3.44)	1.76 (0.89, 4.12)
C20:4n6	7.27 (3.22, 9.44)	6.77 (1.69, 10.97)	7.03 (2.70, 9.39)
Total n-3 PUFA	20.0 (4.61, 46.1)	17.5 (3.17, 55.0)*	15.8 (3.62, 31.5)*
C18:3n3	8.59 (5.83, 16.0)	7.45 (6.27, 21.0)	7.62 (6.49, 9.26)
C20:3n3	0.34 (0.18, 0.72)	0.38 (0.16, 1.10)	0.42 (0.16, 0.70)
C20:5n3	4.69 (2.55, 8.02)	4.33 (1.80, 8.86)	4.46 (2.65, 8.86)
C22:6n3	5.51 (1.72, 19.0)	4.26 (1.72, 15.6)*	3.96 (1.20, 23.3)#
n-6/n-3	1.48 (0.79, 3.75)	1.55 (0.60, 4.81)	1.74 (0.93, 4.30)

All data were expressed as median ($P_{12.5}$, $P_{87.5}$). SFA: saturated fatty acid; PUFA: polyunsaturated fatty acid. Group I: mothers with normal gestational weight gain and their babies with normal birth weight, Group II: mothers with excessive gestational weight gain and their babies with normal birth weight, Group III: mothers with excessive gestational weight gain and their macrosomic babies weighing at least 4 000 g.
*p<0.05, compared with Group I; #p<0.05, compared with Group II.

group III was significantly higher than that in group I (p<0.05). No significant difference in C18:2n6 concentrations were observed among the three groups. Docosahexaenoic acid (DHA) concentrations in group III were significantly higher than that in group I, and the C20:3n3 concentration in group III was significantly higher than that in group II. C20:5n3 and C22:6n3 concentrations in group III were significantly higher than those in group I.

No significant differences were observed in n-3: n-6 PUFA ratio and total n-3 and n-6 PUFA concentrations.

Fatty acid concentrations in the umbilical cord serum are presented in Table 4. Total SFA and C15:0 concentrations in group II were significantly lower than those in group I ($p<0.05$). Total PUFA concentrations in groups II and III was significantly lower than that in group I. The n-6 PUFA concentrations in group II was significantly lower than that in group I. C20:2n6 concentrations in groups II and III was significantly higher than that in group I. Eicosapentaenoic acid concentrations were similar in all three groups. Total n-3 PUFA concentrations in groups II and III were significantly lower than that in group I ($p<0.05$).

DISSCUSSION

Excessive GWG is one of the most important predictor related to macrosomia. Establishing and implementing programs that prevent excessive GWG is crucial to avoid undesirable fetal outcomes.[26]

A retrospective cohort study reported that excessive GWG is an independent predictor of cesarean delivery. Although macrosomia is a stronger predictor of cesarean delivery than weight gain alone, excessive GWG is much more common than macrosomia is.[27] Maternal dietary fatty acids can play a role in programming the growth of the offspring.[28] In our study, a significant difference was observed in TG concentrations but not in protein intake and FBG concentrations among the three groups, which intrigued us to additionally study the roles of fatty acids. Fetal overgrowth is related to factors such as altered glucose and fatty acid transport and elevated placental hormones, including human placental lactogen, cortisol, and estrogen. Distinguishing the independent effects of obesity and glucose control on fetal growth is very difficult.[29] Several studies have suggested that the placental uptake of maternal fatty acid is altered by maternal over nutrition. The placental activity of mammalian target of rapamycin complex 1 (mTORC1) and eukaryotic translation initiation factor 2a (eIF2a) has been linked to the nutritional, metabolic, and physiological state of the mother. For example, SFA might stimulate mTORC1 activity and decrease eIF2a phosphorylation in the rat placenta, thus up regulating protein synthesis and contributing to placental and fetal overgrowth.[30] The placental transfer of glucose, lipids, and amino acids requires transport across a series of cell membranes by specific membrane transport proteins. Furthermore, excess transfer of glucose and lipids across the placenta promotes fetal adiposity; however, its underlying mechanisms remain unclear. Thus, serum total PUFA and n-6 PUFA concentrations were significantly decreased in the macrosomic cord serum, whereas total SFA, C16:0 and C18:0 concentrations were significantly increased. We did not analyze the maternal dietary intake of fatty acids in their foods and other nutrients throughout pregnancy, which was one of the limitations of the present study. Moreover, the activity of the placental amino acid transporters system L and system A has been reported to be increased with fetal overgrowth in humans and in relevant animal models.[31,32] Placental glucose transporter 1 (GLUT1) is considered the predominant transporter mediating placental glucose transfer in humans, and a relative study has demonstrated that GLUT1 expression was increased in trophoblastic plasma membranes isolated from mice fed a high-fat diet.[31] Once across the fetal side of the basal membrane, glucose and amino acids likely diffuse through endothelial junctions into the fetal circulation; furthermore, fatty acids may require mediated transport across the endothelium.[33] A rodent study has demonstrated that maternal exposure to a high-fat diet causes a significant decrease in DHA and total n-3 PUFA concentrations in fetal plasma. Furthermore, the n-6:n-3 PUFA ratio in fetal plasma was elevated and total SFA, C16:0 and C18:0 concentrations in maternal plasma were significantly increased. Interestingly, Lager et al recently demonstrated that the activation of TLR4 by free fatty acids increases system A transport activity in cultured trophoblastic cells,[34] providing a possible link between maternal hyperlipidemia and increased placental nutrient transport capacity. The results of previous studies are similar to those of our study. There is limited placental transfer of SFAs compared with PUFAs.[35] In addition, lipids, particularly SFAs, can trigger immune responses in the placenta, thus increasing *in situ* inflammation in a manner similar to lipopolysaccharides.[33] An impaired maternal–fetal PUFA transport may occur in pregnancies with complications and/or abnormal placental function. Since the fatty acid patterns in each of cholesterol esters, TG and phospholipids is uniquely different, and the differences in the concentrations of these lipid classes could significantly influence the total serum fatty acid concentration of an individual fatty acid.[36] Thus, future studies should examine the fatty acid concentrations in the major lipid classes of maternal and cord serum. Additional studies should be conducted to study the underlying mechanisms, and a longitudinal study is necessary to evaluate the association between maternal serum fatty acid concentrations and gestational outcomes

A public health promotion strategy for women attending prenatal classes, which emphasizes the importance of correct GWG on the basis of mother's prepregnancy BMI and appropriate energy intakes, should be developed. Comprehensive knowledge of the effect of dietary intake on the placental function during the gestation period is required to alleviate maternal excessive GWG and associated problems. Furthermore, it is crucial to focus on new and ongoing longitudinal studies in this context.

In summary, this Chinese study provides further evidence regarding the benefit of restricting GWG to the appropriate ranges for babies and that of implying crucial policies for optimizing birth outcomes through the modification of dietary fat intake during gestation.

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AUTHOR DISCLOSURES

None of the authors have any conflict of interest or financial disclosures pertaining to this manuscript to report.

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