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Dietary fat quantity and quality in early pregnancy and risk of gestational diabetes mellitus in Chinese women: a prospective cohort study

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Abstract

We aimed to examine the association between the quantity and quality of dietary fat in early pregnancy and gestational diabetes mellitus (GDM) risk. In total, 1477 singleton pregnant women were included from Sichuan Provincial Hospital for Women and Children, Southwest China. Dietary information was collected by a 3-d 24-h dietary recall. GDM was diagnosed based on the results of a 75-g, 2-h oral glucose tolerance test at 24–28 gestational weeks. Log-binomial models were used to estimate relative risks (RR) and 95% CI. The results showed that total fat intake was positively associated with GDM risk (Q4 *v.* Q1: RR = 1·40; 95 % CI 1·11, 1·76; $P_{trend} = 0·001$). This association was also observed for the intakes of animal fat and vegetable fat. After stratified by total fat intake (< 30 %E *v.* ≥ 30 %E), the higher animal fat intake was associated with higher GDM risk in the high-fat group, but the moderate animal fat intake was associated with reduced risk of GDM (T2 *v.* T1: RR = 0·65; 95 % CI 0·45, 0·96) in the normal-fat group. Vegetable fat intake was positively associated with GDM risk in the high-fat group but not in the normal-fat group. No association between fatty acids intakes and GDM risk was found. In conclusion, total fat, animal and vegetable fat intakes were positively associated with GDM risk, respectively. Whereas when total fat intake was not excessive, higher intakes of animal and vegetable fat were likely irrelevant with increased GDM risk, even the moderate animal fat intake could be linked to lower GDM risk.

Key words: Dietary intake: Fatty acids: Fat: Gestational diabetes mellitus

Gestational diabetes mellitus (GDM), a common pregnancy complication, is characterised by hyperglycaemia during pregnancy. In recent decades, the prevalence of GDM has become an imminent health concern globally[1]. According to the International Diabetes Federation[2], GDM occurs in approximately 16·7 % of the population globally in 2021. The China Nutrition and Health Surveys from 2010 to 2013 showed that the prevalence of GDM reached 22·2 %[3]. GDM has been related to substantial short- and long-term adverse health outcomes, such as adverse pregnancy outcomes[4,5] (macrosomia, caesarean section and pre-eclampsia) and an increased risk of developing several metabolic diseases later in life among both women[6] and their offspring[7]. Thus, the early identification of modifiable risk factors for GDM is critical to prevent GDM and its related adverse outcomes.

Dietary factors, as modifiable risk factors, have been associated with GDM risk[8]. Dietary fat, as one of the most important parts of the diet, deserves sustained attention. Previous studies suggested that total fat intake during pregnancy was associated with GDM risk, but the findings have been inconsistent[9–15]. For instance, some studies[12,13,15] have shown that a diet high in fat and low in carbohydrates during pregnancy poses a risk for developing GDM and impaired glucose tolerance. However, two American cohort studies did not identify an association between total fat intake and GDM risk pre-pregnancy[10] or during the first trimester[16]. In Asia, a positive association between total fat intake and GDM risk was observed in three case–control studies[17–19] but was not observed in one cohort study[14]. Therefore, the association between total fat intake during pregnancy and GDM risk requires further research.

In addition to the quantity, the quality (source and composition) of fat is also important; nevertheless, few studies have concentrated on the relationship between the quality of fat and GDM risk. Only two studies[10,14] concerned the source of fat, one[10] of which showed that fat from animal products was positively associated with GDM risk, and neither study observed an association between vegetable fat intake and GDM risk. Studies concentrating on the composition of fat are also sparse. Dietary fat is composed of fatty acids, some of which are bioactive and play a vital role in glucose homoeostasis. Increased plasma NEFA may

Abbreviations: GDM, gestational diabetes mellitus; RR, relative risk; T2DM, type 2 diabetes mellitus.

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contribute to insulin resistance[20], and women with GDM have a distinctive fatty acid profile[21]. However, only three available studies[9,10,14] investigated dietary fatty acid intake and GDM risk, which suggested MUFA may be positively associated with GDM risk, while n-3 PUFA and α-linolenic acid may be negatively associated. However, the above studies were focussed on pre-pregnancy or the second trimester. Thus far, the associations between animal fat, vegetable fat, fatty acid intake and GDM risk during the first trimester remain to be further studied.

Different source of fats (animal/vegetable fat) and fatty acids are positively correlated with total fat intake, and their associations with outcome may change at different levels of total fat intake. For instance, one clinical trial[22] found that substituting MUFA for SFA in the diet improves insulin sensitivity but not in individuals with a high fat intake (> 37E%). Therefore, the influence of the total fat consumption should be taken into consideration when investigating the relationship of the quality of fat with GDM risk.

The present study hypothesised that (1) total fat intake in early pregnancy is positively associated with the risk of GDM and (2) the intakes of animal fat and MUFA are positively associated with the risk of GDM, but these associations disappear in lower total fat intake group. Therefore, using data from a prospective cohort study in China, we aimed to examine the association between the quantity and quality of fat during the first trimester and GDM risk.

Methods

Study population

Participants were drawn from a population-based prospective cohort study conducted in Sichuan Provincial Hospital for Women and Children, Southwest China. From February to July 2017, we recruited 1673 healthy women who met the following inclusion criteria: singleton pregnancy, gestational age ranging from 6 to 14 weeks and no chronic metabolic diseases (diabetes, hypertension and hyperlipidaemia). The study was approved by the Ethics Committee of Sichuan University. All participants provided written informed consent when recruited for the study.

The sample size was calculated based on the GDM incidence from previous research[23] about high-fat and low-carbohydrate diet and GDM risk ($\alpha = 0{\cdot}05$ (two-sided), $1\text{-}\beta = 0{\cdot}90$). The minimum sample size required was 576 participants. We assumed a 20 % dropout rate, resulting in a final included sample size of 720 participants. At recruitment, a total of 1673 participants were invited to join the study; we excluded participants with unfinished dietary surveys (n 8). We also excluded participants with a history of GDM (n 40) because these diagnoses could result in dietary changes in the subsequent pregnancy. Furthermore, we excluded women who had implausible energy intake (< 2092 or > 14644 kJ/d)[24] (n 20) or missing data on the diagnosis of GDM (n 128) (Fig. 1). The final analysis included 1477 women.

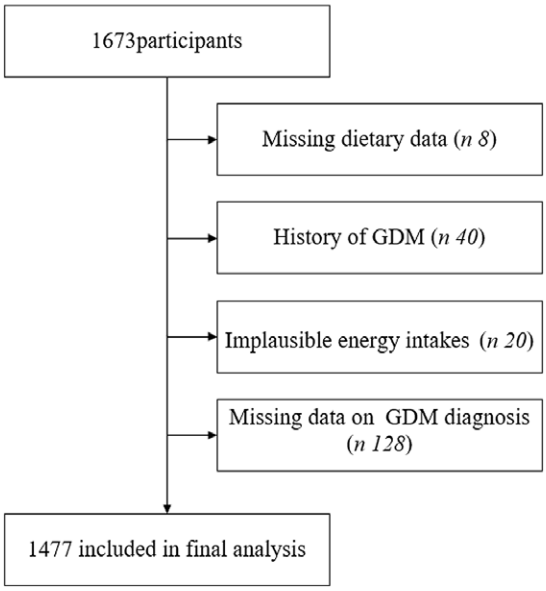

Fig. 1. Flow chart for inclusion and exclusion of the study participants. GDM, gestational diabetes mellitus.

Assessment of dietary intake

Dietary information was assessed by 24-h dietary recalls for three consecutive days, including two weekdays and one weekend day. Information on all types and amounts of food consumed by the participants in the past 24 h was collected by specialised investigators via face-to-face interviews at recruitment (6–14 gestational weeks). To reduce measurement error, standard serving bowls, cups, spoons and illustrative food pictures of various portion sizes[25] were displayed to help the participants estimate their food intake intakes of food. Specialised investigators collected the next 2 d of dietary information through telephone interviews.

Cooking oil intake was calculated as the sum of energy from food and cooking oil consumed by pregnant women multiplied by the ratio of daily cooking oil intake (41·8 g) for the energy of a standard person (2250 kcal/d) used in the data of the China Nutrition and Health Surveys from 2010 to 2013[26]. In this method, the estimate of cooking oil intake is closest to the consumption weighed for three consecutive days[27]. Information regarding the types of cooking oil (animal oil or vegetable oil) mainly consumed in the past month was also collected.

Estimates of the intake of nutrients (including intakes of fat and fatty acids) were calculated mainly using the Chinese Food Composition Tables[28], which are expanded and updated on an ongoing basis and supplemented with the United States Department of Agriculture Nutrient database. The intake of macronutrients was computed as the percentages of total energy intake by the nutrient density method, and other nutrient intakes were adjusted for total energy intake by the residual method[24]. The n-3 PUFA/n-6 PUFA ratio and the PUFA/SFA ratio were calculated.

Ascertainment of gestational diabetes mellitus

Between 24 and 28 weeks of gestation, the participants were routinely screened by the 75-g, 2-h oral glucose tolerance test to diagnose GDM. According to the diagnostic criteria of the International Association of Diabetes Pregnancy Study Group guidelines[29], GDM was diagnosed if any one or more plasma glucose values met or exceeded the following thresholds: fasting plasma glucose ≥ 5·1 mmol/l, 1-h plasma glucose ≥ 10·0 mmol/l or 2-h plasma glucose ≥ 8·5 mmol/l.

Assessment of covariates

Data on baseline characteristics, including maternal age, education level, parity, family history of diabetes mellitus, alcohol drinking status and physical activity level, were collected through a self-designed questionnaire via a face-to-face interview. Pre-pregnancy weight was self-reported, while trained interviewers measured height. Body mass (kg) was divided by height square (m^2) to obtain the BMI. Physical activity (metabolic equivalent of task-h/week) was measured using the Pregnancy Physical Activity Questionnaire[30]. Gestational weight gain before GDM diagnosis was calculated by subtracting self-reported pre-pregnancy weight from the weight measured at the oral glucose tolerance tests.

Maternal age was divided into four categories (≤ 24, 25–29, 30–34 and ≥ 35 years). Pre-pregnancy BMI was categorised as underweight (< 18·5 kg/m^2), normal weight (18·5–23·9 kg/m^2), overweight and obese (≥ 24·0 kg/m^2) according to the Chinese obesity criteria[31]. Educational level was divided into three categories (≤ 12, 13–15 and ≥ 16 years) based on the number of completed years of education. Parity was divided into two categories (primiparity or multiparity). The other covariates, including family history of diabetes and alcohol drinking status, were considered dichotomised variables (yes, no). Alcohol consumption was defined as alcohol consumption during the 6 months before conception or during pregnancy.

Statistical analysis

Distributions of categorical variables are described as frequencies and percentages. Means and standard deviations are used to describe continuous variables with a normal distribution, and medians and interquartile ranges are used to describe continuous variables with a skewed distribution. Comparisons of categorical variables between groups were performed using χ^2 tests. According to their normal or skewed distributions, continuous variables were compared by one-way ANOVA or non-parametric Kruskal–Wallis tests.

Participants were divided into quartiles according to daily dietary fat and fatty acid intake, which were expressed as the nutrient density (percentage of energy content from fat). The use of OR to estimate relative risks (RR) was not appropriate[32] because the GDM incidence in our cohort was > 10 %. Thus, RR and 95 % CI were estimated through log-binomial models[33] with generalised linear regression. When the log-binomial models failed to converge, Poisson regression with robust standard errors was used to fit the models[34]. To test a linear trend, the median values for each quartile of dietary fat and fatty acid intake were assigned and modelled as continuous variables.

In the multivariate analysis, three models were included. Model 1 was adjusted for maternal age, pre-pregnancy BMI, educational level, family history of diabetes, parity, alcohol drinking status, physical activity level and gestational weight gain before GDM diagnosis. Model 2 was adjusted for model 1, and total energy intake, dietary fibre and other fat subtypes or fatty acids were mutually adjusted. For example, when it comes to animal fat, vegetable fat will be adjusted; when it comes to SFA, MUFA and PUFA will be adjusted. To eliminate the influence of the other macronutrients, we additionally and separately adjusted for each macronutrient (carbohydrates, dietary glycaemic load, proteins and animal protein) in model 3.

To evaluate the effects of the substitution of total fat, animal fat and vegetable fat for carbohydrates, continuous nutrient densities were simultaneously included in the multivariate models. By additionally including protein (percentage of energy) concurrently, the coefficients could be interpreted as the effect of exchanging energy from a specific fat for the same amount of energy from carbohydrates.

To reduce the influence of total fat intake, stratified analyses were performed to assess whether the association of the intake of animal fat, vegetable fat and fatty acids and GDM risk was modified by the total fat intake level. Total fat intake was stratified into a normal-fat group (< 30 %E) and a high-fat group (≥ 30 %E), according to the Chinese Dietary Reference Intakes (2013)[35], in which the percentage of energy from fat ≥ 30 %E is excessive. At the same time, stratified analyses were performed to assess whether the association of the intake of total fat, animal fat, vegetable fat and GDM risk was modified by maternal age, pre-pregnancy BMI and family history of diabetes. Interaction tests were conducted in the multivariable models. Considering the small sample size in some groups after stratification, we classified subjects into tertiles according to their intake of fats and fatty acids for analysis in the stratified analyses.

A generalised additive model with a spline smoothing function was applied to examine the relationship between total fat intake and GDM risk and the relationship between animal fat intake and GDM risk at different total fat intake levels, with adjustments for potential confounders.

A two-tailed P value < 0·05 was considered statistically significant. To adjust for multiple testing with regard to the regression analyses, Bonferroni correction was applied to consider the multiple comparisons occurring (for total fat: P values < 0·05; for animal fat and vegetable fat: P values < 0·025, Bonferroni: 0·05/2; for SFA, MUFA and PUFA: P values < 0·017, Bonferroni: 0·05/3). All statistical analyses were performed using Stata version 15.0 (Stata Corp LP) and EmpowerStats software (www.empowerstats.com, X&Y solutions, Inc.).

Results

Baseline characteristics

Of the 1477 women evaluated, 530 (35·9 %) cases of GDM were diagnosed. The mean age of the participants was 28·0 (SD 4·0) years, and the mean pre-pregnancy BMI was 21·0 (SD 2·8) kg/m^2. The median total fat intake was 32·7 (interquartile range 28·9, 36·9) %E, which is above the recommended fat intake during early pregnancy in China (20–30 %E). The intake of animal fat and vegetable fat was 11·0 (interquartile range 7·3, 15·1) %E and 20·8 (interquartile range 18·9, 23·4) %E, respectively. The large majority (98·1 %) mainly consumed vegetable oil in the past month.

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Table 1. Baseline characteristics of participants according to quartiles of the total fat
(Numbers and percentages; medians and interquartile ranges)

	Total fat										
	All participants (*n*)		Q1		Q2		Q3		Q4		
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*P*
Age at enrolment(years)											0·326
≤ 24	383	25·9	95	25·7	91	24·7	99	26·8	98	26·6	
25–29	791	53·6	196	53·1	213	57·7	183	49·5	199	53·9	
30–34	172	11·6	47	12·7	41	11·1	50	13·5	34	9·2	
≥ 35	131	8·9	31	8·4	24	6·5	38	10·3	38	10·3	
Pre-pregnancy BMI (kg/m^2)											0·069
< 18·5	213	14·4	45	12·2	45	12·2	54	14·6	69	18·7	
18·5–23·9	1090	73·8	277	75·1	288	78·0	266	71·9	259	70·2	
≥ 24·0	174	11·8	47	12·7	36	9·8	50	13·5	41	11·1	
Educational level (schooling years)											0·242
≤ 12	340	23·0	88	23·8	89	24·1	91	24·6	72	19·5	
13–15	531	36·0	140	37·9	132	35·8	135	36·5	124	33·6	
≥ 16	606	41·0	141	38·2	148	40·1	144	39·0	173	46·9	
Nulliparous (%)	1090	73·8	255	69·1	273	74·0	269	72·0	293	79·4	0·015
Family history of diabetes (%)	258	17·5	51	13·8	63	17·1	67	18·1	77	20·9	0·089
Alcohol drinking (%)	111	7·5	29	7·9	30	8·1	18	4·9	34	9·2	0·137
Physical activity (MET-h/week^2)*											0·716
Median	103·2		101·9		104·2		101·4		106·3		
Interquartile range	72·3–132·9		72·3–133·5		73·0–135·1		71·0–130·0		74·4–132·3		
Gestational weight gain before GDM diagnosis* (kg)											0·066
Median	6·1		6·0		6·0		6·1		6·5		
Interquartile range	4·1–8·2		3·8–8·0		4·1–8·2		4·0–8·2		4·7–8·7		

MET, metabolic equivalent of task; GDM, gestational diabetes mellitus.
* Data of physical activity and gestational weight gain before GDM diagnosis were described by median and interquartile range.

Women with a higher total fat intake were more often primiparous and consumed more protein, eggs, meat, dairy products, nuts, fish and legumes and less carbohydrates, grains and tubers (tubers mainly included potatoes, sweet potatoes and cassava) (Tables 1 and 2). The baseline characteristics and dietary intakes of the participants according to quartiles of animal fat or vegetable fat are presented in online Supplementary Tables S1–S4.

The correlation coefficients of the energy-adjusted intake of specific types of fat were as follows: 0·74 between total fat intake and animal fat intake; 0·81 between total fat intake and vegetable fat intake and 0·96 between total fat intake and MUFA intake (all $P < 0·05$) (online Supplementary Table S5).

Association between fat and gestational diabetes mellitus risk

Higher intakes of total fat, animal fat and vegetable fat were significantly associated with GDM risk in the fully adjusted models, including both dietary and non-dietary covariates (Table 3). The multivariable-adjusted RR of GDM from the lowest to the highest quartiles of total fat were 1·00 (reference), 0·96 (95 % CI 0·77, 1·19), 1·21 (95 % CI 0·997, 1·48) and 1·24 (95 % CI 1·01, 1·51) ($P = 0·009$ for trend) after adjusting for dietary, socio-demographic and lifestyle factors, respectively (model 2). The smoothing curve showed that after adjusting for confounders, there was a positive linear correlation between the possibility of developing GDM and total fat intake (Fig. 2). The multivariable-adjusted RR of GDM from the lowest to the highest quartiles of animal fat were 1·00 (reference), 1·14 (95 % CI 0·92, 1·41), 1·33 (95 % CI 1·09, 1·63) and 1·28 (95 % CI 1·03, 1·59) ($P = 0·026$ for trend) after adjusting for the same dietary, socio-demographic, lifestyle factors and additional vegetable fat intake (model 2). The multivariable-adjusted RR of GDM from the lowest to the highest quartiles of vegetable fat were 1·00 (reference), 1·16 (95 % CI 0·93, 1·43), 1·31 (95 % CI 1·07, 1·60) and 1·24 (95 % CI 0·995, 1·54) ($P = 0·048$ for trend) in model 2.

To eliminate the influence of the other macronutrients, additional adjustments for carbohydrates, dietary glycaemic load, proteins and animal protein were modelled, respectively. The effect sizes between fat intake (total fat, animal fat and vegetable fat) and GDM risk were increased after adjusting for carbohydrate and dietary glycaemic load but did not change apparently after additionally adjusting for protein or animal protein. Therefore, we only displayed the results after additionally adjusting for glycaemic load (model 3) in Table 3, and other results are displayed in online Supplementary Table S6.

We did not observe a significant association between intakes of SFA, MUFA, PUFA, n-3 PUFA, n-6 PUFA, specific fatty acids and GDM risk in the fully adjusted model (model 2) (Table 4 and online Supplementary Table S7).

These substitution models revealed replacing 5 % of the energy from carbohydrates with total fat increased the risk of GDM by 6·8 % and replacing 3 % of the energy from carbohydrates with animal fat increased the risk of GDM by 4·5 %. However, replacing 3 % of the energy from carbohydrates with vegetable fat was not associated with GDM risk (online Supplementary Table S8).

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Table 2. Dietary intakes of participants according to quartiles of the total fat
(Median values and interquartile ranges)

	All participants(n)		Total fat Q1		Q2		Q3		Q4		
	Median	IQR	Median	IQR	Median	IQR	Median	IQR	Median	IQR	*P*
Total energy (kcal/d)	1797·0	1489·5–2135·6	1720·7	1377·6–2082·0	1764·6	1475·5–2091·5	1858·5	1554·3–2192·4	1876·1	1554·1–2192·4	< 0·001
Protein (%E)	12·1	10·7–13·9	10·8	9·7–12·4	11·9	10·7–13·3	12·6	11·1–14·5	13·2	12·0–15·2	< 0·001
Carbohydrate (%E)	55·7	50·1–60·6	64·3	61·9–67·2	58·2	56·1–59·7	53·3	51·3–55·2	46·9	43·7–49·3	< 0·001
Dietary fibre* (g/d)	11·8	9·4–5·0	12·4	10·1–16·1	11·6	9·2–14·6	11·7	9·2–15·4	11·4	8·9–14·4	0·001
Dietary glycaemic load*	150·6	131·0–170·2	177·1	161·1–194·2	159·9	146·4–174·7	143·4	131·4–158·7	121·9	105·3–135·0	< 0·001
Eggs (g/d)	33·3	12·5–50·0	19·8	0·0–50·0	33·3	16·7–50·0	37·5	16·7–52·1	50·0	25·0–58·3	< 0·001
Grains and tubers (g/d)	266·8	209·1–337·2	304·3	229·8–394·4	285·0	230·6–355·8	261·0	212·5–315·7	227·5	173·4–291·3	< 0·001
Fruit and vegetables (g/d)	553·8	403·7–756·5	564·5	400·5–844·0	554·9	410·1–706·5	550·1	403·9–785·6	549·3	400·5–705·2	0·229
Red meat and poultry (g/d)	96·0	43·0–194·2	38·2	16·0–91·9	81·8	40·7–156·6	112·0	61·2–204·8	169·2	103·3–282·2	< 0·001
Dairy products (g/d)	125·0	0·0–236·1	0·0	0·0–151·4	100·0	0·0–218·1	160·9	39·6–250·0	166·7	83·3–250·0	< 0·001
Nuts (g/d)	5·0	0·0–15·0	0·0	0·0–5·1	4·9	0·0–12·7	8·1	0·0–19·7	11·5	1·2–23·5	< 0·001
Aquatic products (g/d)	0·0	0·0–21·6	0·0	0·0–5·2	0·0	0·0–22·1	0·0	0·0–28·2	0·0	0·0–21·6	< 0·001
Legumes (g/d)	7·6	0·0–19·0	5·4	0·0–16·5	8·2	0·4–20·0	6·9	0·0–20·4	8·9	0·0–20·4	< 0·001
Cooking oil (g/d)	31·2	25·8–37·0	29·9	24·0–36·2	30·6	25·6–36·3	32·1	27·0–37·5	32·6	26·8–37·9	< 0·001

%E, percentage of energy intake; IQR, interquartile range.
* Dietary variables were adjusted for total energy intake by the residual method.

Stratified analyses

The multivariable-adjusted RR of GDM comparing the total fat intake of the high-fat group to the normal-fat group were 1·21 (95 % CI 1·01, 1·44). After stratified by total fat intake (< 30 %E *v.* ≥ 30 %E), a higher intake of animal fat was associated with a higher GDM risk in the high-fat group (> 30 %E). In the normal-fat group (≤ 30 %E), moderate animal fat intake was linked with a reduced risk of GDM. The multivariable-adjusted RR of GDM from the lowest to the highest tertiles of animal fat intake were 1·00 (reference), 0·65 (95 % CI 0·45, 0·96) and 0·79 (95 % CI 0·36, 1·72) ($P = 0{\cdot}067$ for trend) after full adjustment. After adjusting for possible confounders, non-linear relationships between the possibility of developing GDM and animal fat intake in the two groups were observed (Fig. 3). A higher intake of vegetable fat was associated with a higher GDM risk in the high-fat group (> 30 %E); however, the association disappeared in the normal-fat group (≤ 30 %E) (Table 5).

The associations between the intake of total fat, animal fat, vegetable fat and GDM were still consistent in most subgroups after performing stratified analyses. The relationship of total fat intake and GDM risk was positive among the participants in all subgroups stratified by pre-pregnancy BMI, age and no family history of diabetes. The relationship of animal fat intake and GDM risk was positive among the participants in the subgroups of low age (< 35 years), high pre-pregnancy BMI (> 24 kg/m^2) and no family history of diabetes. The relationship between vegetable fat intake and GDM risk was positive among the participants in the subgroups of high age (> 35 years) and high pre-pregnancy BMI (> 24 kg/m^2) (online Supplementary Table S9).

Discussion

We found that higher intakes of total fat, animal fat and vegetables during the first trimester were significantly associated with a higher incidence of GDM. After performing stratified analyses, a higher intake of animal fat was associated with a higher GDM risk in the high-fat group, but moderate animal fat intake was associated with a reduced risk of GDM in the normal-fat group. Vegetable fat intake was positively associated with GDM risk in the high-fat group but not in the normal-fat group. Moreover, we estimated that replacing isoenergetic carbohydrates with total fat and animal fat was associated with a significantly increased risk of GDM. No association between fatty acid intake and GDM risk was found.

Three studies[12,13,15] observed that total fat intake during early and mid-pregnancy was associated with GDM risk, which was generally in line with findings from the present study. Although the precise mechanisms by which high fat intake influences glucose homoeostasis and diabetes risk are unclear, the observed association with GDM risk is biologically plausible. A high-fat diet can reduce the expression of facilitative GLUT, recombinant GLUT2 and glucokinase, thereby impairing glucose-stimulated insulin secretion[36], which is another burden, especially when mothers experience physiologically increased insulin resistance during normal pregnancy[37]. Moreover, a large-scale population (*n* 48 835) study[38] identified the role

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Table 3. Relative risks (RR) of gestational diabetes mellitus (GDM) according to quartiles of total fat, animal fat and vegetable fat (% E) during early pregnancy (Risk ratios and 95 % confidence intervals)

	Quartile							
	Q1	Q2		Q3		Q4		$P_{\text{for trend}}$
		RR	95 % CI	RR	95 % CI	RR	95 % CI	
Total fat								
Median (%E/d)	25·9	30·9		34·6		39·9		
Range	17·2–28·9	28·9–32·6		32·6–36·9		36·9–59·2		
GDM cases/pregnancies (*n*)	113/369	117/369		149/370		151/369		
Model 1*	1·00	0·97	0·78,1·20	1·24	1·02,1·51	1·27	1·04,1·55	0·004
Model 2†	1·00	0·96	0·77,1·19	1·21	0·997, 1·48	1·24	1·01,1·51	0·009
Model 3‡	1·00	0·999	0·80, 1·24	1·31	1·07, 1·61	1·40	1·11, 1·76	0·001
Animal fat								
Median (%TE/d)	4·9	9·2		12·7		18·5		
Range	0·0–7·3	7·3–15·1		11·0–15·1		15·1–46·5		
GDM cases/pregnancies (*n*)	108/370	130/369		153/368		139/370		
Model 1*	1·00	1·13	0·91,1·39	1·31	1·07,1·60	1·22	0·99,1·50	0·042
Model 2† vegetable fat	1·00	1·14	0·92,1·41	1·33	1·09,1·63	1·28	1·03,1·59	0·026
Model 3‡	1·00	1·18	0·96, 1·47	1·41	1·15, 1·74	1·43	1·12, 1·82	0·002
Vegetable fat								
Median (%TE/d)	17·9	19·9		21·9		25·6		
Range	1·2–18·9	18·9–20·8		20·8–23·4		23·4–58·4		
GDM cases/pregnancies (*n*)	116/371	130/369		149/369		135/368		
Model 1*	1·00	1·14	0·92, 1·41	1·29	1·06, 1·58	1·20	0·97,1·48	0·079
Model 2† animal fat	1·00	1·16	0·93, 1·43	1·31	1·07,1·60	1·24	0·995, 1·54	0·048
Model 3‡	1·00	1·16	0·94, 1·44	1·33	1·08, 1·62	1·30	1·04, 1·63	0·016

%E, percentage of energy intake.

* Adjusted for maternal age (≤ 24, 25–29, 30–34 or ≥ 35 years), pre-pregnancy BMI (< 18·5, 18·5–23·9 or ≥ 24·0 kg/m^2), educational level (≤ 12, 13–15 and ≥ 16 years), family history of diabetes (yes, no), parity (primiparity or multiparity), alcohol drinking status (yes, no), physical activity (MET h/week) and gestational weight gain before GDM diagnosis (kg).

† Model 1 plus total energy intake (kJ/d), dietary fibre (g/d) and other fats or fatty acids as listed in the table.

‡ Model 2 plus glycaemic load.

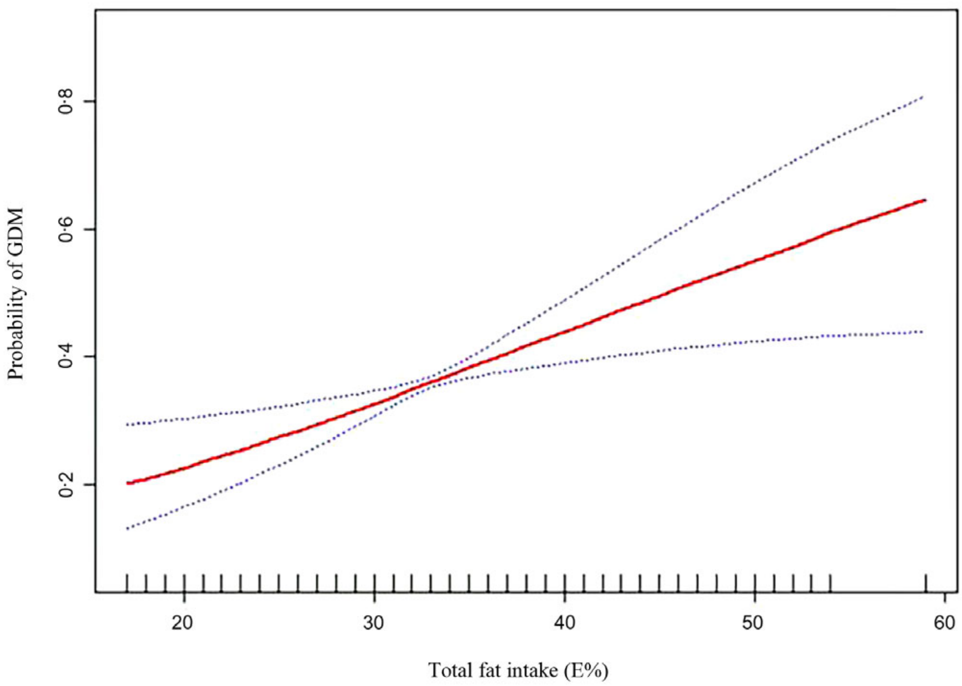

Fig. 2. The relation between total fat intake (E%) and gestational diabetes mellitus (GDM) adjusted for maternal age, pre-pregnancy BMI, educational level, family history of diabetes, parity, alcohol drinking status, physical activity, gestational weight gain before GDM diagnosis, total energy intake, dietary fibre and glycaemic load. In the figure, the solid line indicates the estimated risk of death, and the dotted lines represent point-wise 95 % CI.

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Table 4. Relative risks (RR) of gestational diabetes mellitus (GDM) according to quartiles of fatty acid intake (% E) during early pregnancy (Risk ratios and 95 % confidence intervals)

	Quartile							
	Q1	Q2		Q3		Q4		$P_{for\ trend}$
		RR	95 % CI	RR	95 % CI	RR	95 % CI	
SFA								
Model 1*	1·00	1·17	0·95,1·43	1·10	0·89,1·36	1·18	0·96, 1·45	0·183
Model 2† MUFA, PUFA	1·00	1·08	0·86, 1·35	0·96	0·73,1·25	0·98	0·74, 1·33	0·672
MUFA								
Model 1*	1·00	0·96	0·77,1·18	1·12	0·91, 1·37	1·22	1·01,1·49	0·018
Model 2† SFA, PUFA	1·00	0·99	0·79, 1·25	1·16	0·90, 1·50	1·33	0·97,1·81	0·050
PUFA								
Model 1*	1·00	1·19	0·97,1·46	1·14	0·92,1·41	1·26	1·02,1·54	0·069
Model 2† SFA, MUFA	1·00	1·15	0·94, 1·42	1·06	0·85,1·32	1·16	0·93, 1·46	0·404
n-3 PUFA								
Model 1*	1·00	1·04	0·84,1·29	1·04	0·84,1·28	1·27	1·04,1·54	0·009
Model 2† SFA, MUFA, *n*-6 PUFA	1·00	0·99	0·79, 1·25	0·97	0·76,1·25	1·18	0·87, 1·60	0·098
n-6 PUFA								
Model 1*	1·00	1·17	0·95,1·44	1·14	0·92,1·40	1·26	1·02,1·55	0·053
Model 2† SFA, MUFA, *n*-3 PUFA	1·00	1·11	0·89, 1·38	0·997	0·78,1·28	1·07	0·79,1·44	0·982

%E, percentage of energy intake.
* Adjusted for maternal age (≤ 24, 25–29, 30–34 or ≥ 35 years), pre-pregnancy BMI (< 18·5, 18·5–23·9 or ≥ 24·0 kg/m^2), educational level (≤ 12, 13–15 and ≥ 16 years), family history of diabetes (yes, no), parity (primiparity or multiparity), alcohol drinking status(yes, no), physical activity(MET h/week) and gestational weight gain before GDM diagnosis (kg).
† Model 1 plus total energy intake (kJ/d), dietary fibre (g/d), glycaemic load and other fats or fatty acids as listed in the table.

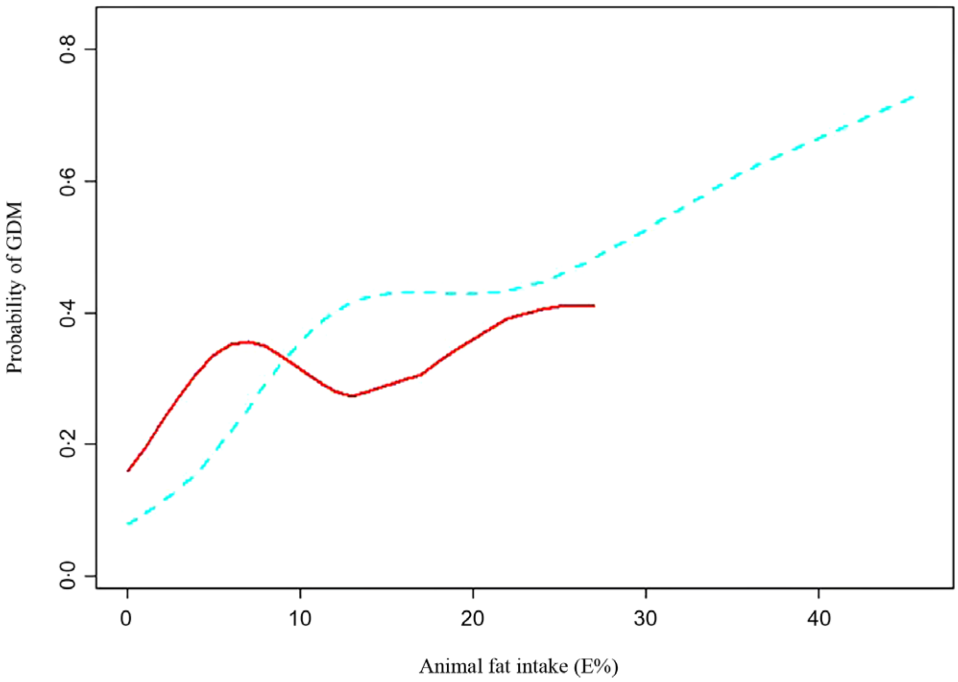

Fig. 3. The relation between animal fat intake (E%) and gestational diabetes mellitus (GDM) in high-fat group (≥30 %E) and normal-fat group (<30 %E), adjusted for maternal age, pre-pregnancy BMI, educational level, family history of diabetes, parity, alcohol drinking status, physical activity, gestational weight gain before GDM diagnosis, total energy intake, dietary fibre, glycaemic load and vegetable fat intake. ——, <30 %E; – – –, ≥30 %E.

of a high-fat diet as an independent risk factor for type 2 diabetes mellitus (T2DM) in women. Similar to GDM, T2DM also results from a mixture of insulin resistance and dysfunctional insulin secretion. However, another Chinese pregnant women cohort study[14] elucidated no significant association between total fat intake in the first trimester and GDM risk, which was not consistent with our results. In the former study, dietary information was collected by a FFQ, and the main source of fat was animal food; while we gathered dietary information by 3-d dietary recalls, the fat in our study mainly came from vegetables. Considering that fats from different sources might be differently associated with the risk of T2DM[39], the aforementioned points could explain the inconsistent results to some extent.

Table 5. Stratified analysis of the association between gestational diabetes mellitus (GDM) and usual fat intake (% E) during pregnancy by the percentage of energy from total fat*
(Risk ratios and 95 % confidence intervals)

	The percentage of energy from total fat											
	≤ 30E% (470)						> 30E% (1007)					
	T1	T2		T3		$P_{\text{for trend}}$	T1	T2		T3		$P_{\text{for trend}}$
		RR	95 % CI	RR	95 % CI			RR	95 % CI	RR	95 % CI	
Animal fat												
Median	5·0	9·8		22·4			6·8	11·4		17·2		
Range	0·0–8·6	8·6–12·9		16·9–26·6			0·0–8·6	8·6–13·5		13·5–46·5		
Model vegetable fat	1·00	0·65	0·45, 0·96	0·79	0·36, 1·72	0·067	1·00	1·55	1·15, 2·07	1·64	1·21, 2·22	0·001
Vegetable fat												
Median	18·3	20·7		23·9			1·8	20·9		24·9		
Range	2·5–19·5	19·5–22·3		22·4–28·2			0·1–19·5	19·5–22·4		22·4–58·4		
Model animal fat	1·00	0·85	0·62, 1·18	0·83	0·55, 1·25	0·419	1·00	1·14	0·91, 1·42	1·29	1·02, 1·63	0·018
SFA												
Median	5·3	7·6		10·2			6·2	8·0		10·5		
Range	2·4–6·8	6·8–8·9		9·1–15·3			3·1–6·8	6·8–8·9		9·0–22·3		
Model MUFA, PUFA	1·00	1·15	0·83, 1·59	1·18	0·63, 2·19	0·450	1·00	0·88	0·67, 1·15	0·87	0·65, 1·18	0·693
MUFA												
Median	10·6	12·5		14·5			11·6	13·0		15·4		
Range	7·6–12·0	12·1–13·9		14·1–16·0			9·2–12·0	12·1–14·1		14·1–28·6		
Model SFA, PUFA	1·00	0·85	0·58, 1·25			0·059	1·00	1·24	0·90, 1·72	1·43	1·01, 2·03	0·020
PUFA												
Median	7·1	8·4		9·9			7·4	8·5		10·5		
Range	2·6–7·8	7·8–9·3		9·4–13·6			2·9–7·8	7·8–9·3		9·3–24·1		
Model SFA, MUFA	1·00	1·21	0·90, 1·63	1·25	0·84, 1·88	0·172	1·00	1·03	0·81, 1·31	1·05	0·83, 1·33	0·536

%E, percentage of energy intake.

* Model was adjusted for maternal age (≤ 24, 25–29, 30–34 or ≥ 35 years), pre-pregnancy BMI (< 18·5, 18·5–23·9 or ≥ 24·0 kg/m^2), educational level (≤ 12, 13–15 and ≥ 16 years), family history of diabetes (yes, no), parity (primiparity or multiparity), alcohol drinking status (yes, no), physical activity (MET h/week), gestational weight gain before GDM diagnosis (kg), total energy intake (kJ/d), dietary fibre (g/d), glycaemic load and other fats or fatty acids as listed in the table.

Indicate that the participants whose MUFA intakes are in T3 and total fat intakes are below 30E% are not enough to analyse.

To eliminate the influence of the other macronutrients, we exploringly took carbohydrates, dietary glycaemic load, protein and animal protein into the model separately (online Supplementary Table S6). The association between total fat intake and GDM risk was increased after adjusting for carbohydrate and dietary glycaemic load, and then we included glycaemic load in the final model. This finding suggested that the observed association may have been partially masked by carbohydrates. It is universally recognised that the dietary glycaemic load may contribute to the onset and progression of diabetes, and a lower glycaemic load is likely to reduce the risk of GDM[40]. A high-fat diet, accompanied by a lower glycaemic load, may offset a part of the hazardous effect of high fat intake. The protein content of the diet tends to remain fairly consistent[41] compared with carbohydrates and fat. Additionally, the effect of proteins, such as carbohydrates, on glucose is not very distinct. This may be why we did not observe apparent changes after additionally adjusting for protein or animal protein.

In the present study, a higher intake of animal fat was associated with an increased risk of GDM, but the association remained only in the high-fat group after stratification by the percentage of energy from fat. The Nurses' Health Study II[10] also observed adverse effects of pre-pregnancy high animal fat intake on GDM risk. In addition, a study in Korea with women having a history of GDM found that participants with high animal fat intake were more likely to develop T2DM[42]. However, these two studies did not exclude the influence of total fat intake on the association between animal fat intake and GDM or T2DM risk. In our study, the influence of total fat intake was partly excluded by the stratified analyses, and we observed that high intake of animal fat increased GDM risk in the high-fat group, and moderate intake (*v.* low intake) of animal fat reduced GDM risk by 38 % in the normal-fat group. Our findings indicated that moderate animal fat intake during pregnancy may be protective against GDM when total fat intake is not excessive, and pregnant women with excessive intake of total fat should preferentially reduce animal fat intake. However, due to the small sample size in the normal-fat group, the results may be unstable. Therefore, this is a point that necessitates further examination in the future.

We also observed a positive association between vegetable fat intake and GDM risk, but it disappeared in the normal-fat group. However, most studies[10,14] did not show an association between vegetable intake and GDM risk; moreover, a review[43] suggested that vegetable fat might be beneficial regarding T2DM prevention. In our cohort, total fat intake was highly correlated with vegetable fat intake (*r* 0·81). Therefore, the association between intake of vegetable fat and GDM risk in the high-fat group can be attributed to the high correlation with total fat intake. Moreover, the replacement of 3 % of energy from carbohydrates with vegetable fat or from animal fat with vegetable fat was not associated with an increased risk of GDM. Thus, high vegetable fat intake during pregnancy may not be an independent risk factor for GDM.

We identified no association between fatty acids intakes and GDM risk. However, in the high-fat group, participants with a higher intake of MUFA had a higher likelihood of having GDM, which is not significant after Bonferroni correction ($P = 0{\cdot}031 > 0{\cdot}017$). The Nurses' Health Study II[10] also observed the adverse effects of higher MUFA intake on GDM risk. However, evidence regarding the association between MUFA intake and diabetes risk is inconsistent[22,44]. And the precise pathological mechanisms underlying the association between MUFA and glucose homoeostasis and diabetes risks are still unclear. In this cohort, the association between intake of MUFA and GDM risk in the high-fat group can still be partly attributed to the high correlation ($r = 0{\cdot}96$) with total fat intake. The correlation between MUFA intake and GDM risk remains to be further studied at different total intake levels. In terms of specific fatty acids, a randomised controlled trial[45] with twenty-eight participants found that palmitic and oleic acids in the diet were related to β-cell function and insulin sensitivity. Another prospective nested case–control study observed that myristic and palmitic and linoleic acids in plasma were correlated with GDM risk among Chinese pregnant women[45,46]. However, we identified no association between specific fatty acids and GDM risk, which could be explained, in part, by the small and opposite effect of fatty acids on GDM. Future studies are suggested to use an index to investigate the balanced effect of fatty acid intake on GDM, which is for diabetes and reflects the protective or hazardous effect of fatty acid intake on insulin resistance or insulin secretion, such as indices of dietary fat quality for CVD[47].

The strengths of our study include the following: First, we observed the positive association between total fat intake and GDM risk and then reduced its influence by stratification in the following analysis, which has not been done in existing studies. Using this approach, we found higher animal fat intake raised the probability of developing GDM but not when total fat intake under the upper limit, which may be more instructive than those simply indicating that higher total fat, animal fat intakes may increase GDM risk. Second, this is the first prospective cohort study to investigate the association between specific fatty acids intakes and GDM risk in China. Third, dietary information was assessed in the first trimester before the diagnosis of GDM in our study, which can enable earlier prevention of GDM.

Nevertheless, some limitations of our study should be acknowledged. First, baseline glycaemia was not collected in our study, which would help to detect unknown diabetes or impaired fasting glucose condition. However, according to the Report on Chinese Residents' Nutrition and Chronic Disease Status in 2020, the prevalence of diabetes among women aged 18–44 years in China was 5 %[48]. In our research, most participants are under age 30, and their prevalence of diabetes may be lower. Therefore, the missing of baseline glycaemia would not affect the results significantly. Second, we did not calculate the intake of *trans*-fatty acids due to a lack of data on *trans*-fatty acids in the updated Chinese Food Composition Tables. High intake of *trans*-fatty acids may increase the risk for T2DM[49]; thus, future studies are suggested to take this into account. Third, as in other observational studies, measurement errors or residual confounding cannot be entirely eliminated, although trained interviewers used the estimation tools to help participants minimise recall bias. Finally, our study population was composed of Chinese pregnant women; therefore, it cannot be extended to other ethnic populations. Our results need further verification in future larger prospective studies and randomised clinical trials.

In conclusion, total fat, animal and vegetable fat intakes were positively associated with GDM risk, respectively. Whereas when total fat intake was not excessive, higher intakes of animal and vegetable fat were likely irrelevant with increased GDM risk, even the moderate animal fat intake could be linked to lower GDM risk. Our findings suggest that compared with quality, the quantity of dietary fat is the priority for nutritional interventions to prevent GDM.

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G. Z. designed the research; Q. F. collected data, completed all the data analysis and wrote the manuscript; H. D., M. Y., Y. Z., X. L. and H. S. critically reviewed and edited the manuscript; S. C., C. C. and D. S. participated in collecting data; G. Z. supervised the research. All authors critically reviewed and approved the final manuscript.

The authors report no potential conflict of interest.

Supplementary material

For supplementary material referred to in this article, please visit https://doi.org/10.1017/S0007114522002422

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