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Association of dietary inflammatory index with risk of gestational diabetes mellitus and preeclampsia: a systematic review and meta-analysis

Li Hong[1]†, Liyuan Zhu[2]†, Jinru Zhang[1], Yueqi Fu[1], Xiaoyan Qi[1] and Mei Zhao[1]*

[1]*School of Nursing, Anhui Medical University, Hefei, People's Republic of China*
[2]*Department of Nursing, The First Affiliated Hospital of USTC, Division of Life Sciences and Medicine, University of Science and Technology of China, Hefei, People's Republic of China*

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Abstract

Findings from observational studies have suggested a possible association between dietary inflammatory index (DII) and risk of gestational diabetes mellitus (GDM) and preeclampsia (PE). However, the results of these studies were inconclusive. A systematic review and meta-analysis was carried out to illuminate this association. Systematic literature search was conducted in PubMed, Web of Science, Cochrane Library, EMBASE, Scopus and other databases from inception until January 2023. The qualities of included studies were assessed using the Newcastle–Ottawa scale. Nine studies (seven cohort, two case–control) were included in the meta-analysis, including 11 423 participants from five different countries. The meta-analysis indicated that a 1-unit increase in the DII score, representing pro-inflammatory diet, was associated with 13 % higher risk of GDM (OR = 1·13; 95 % CI 1·02, 1·25, $I^2 = 68{\cdot}4$ %, $P = 0{\cdot}004$) and 24 % higher risk of PE (OR = 1·24; 95 % CI 1·14, 1·35, $I^2 = 52{\cdot}0$ %, $P = 0{\cdot}125$). Subgroup analysis found that this association was evident among studies with Chinese populations (OR = 1·16; 95 % CI 1·06, 1·28) and studies with mid pregnancy (OR = 1·20; 95 % CI 1·07, 1·34). The findings indicate that pro-inflammatory diet can increase the risk of GDM and PE. Considering some limitations in this study, more studies are needed to verify this association.

Keywords: **Inflammation: Dietary inflammatory index: Gestational diabetes mellitus: Preeclampsia: Meta-analysis**

Gestational diabetes mellitus (GDM) refers to abnormal glucose tolerance with onset or first recognition during pregnancy[1]. Study has shown that the prevalence of GDM has significantly increased two to three times in 10 years[2], and the total incidence is 14·8 %[3]. Preeclampsia (PE) is a serious pregnancy complication next only to GDM, with an estimated average incidence of 5 %[4]. GDM and PE in pregnant women are syndromes associated with substantially increased risk of preterm delivery, low birth weight, fetal growth restriction and caesarean section[5–7]. The causes of GDM and PE are multifaceted, involving genetic, physiological and lifestyle-related risk factors. Recently, the role of chronic inflammation has received increasing attention[8,9]. Moreover, studies have shown that diet can regulate the level of inflammation in the body by altering the expression of inflammatory genes and the concentration of inflammatory markers[10,11]. Therefore, recent studies have begun to explore the association between dietary inflammatory potential and risk of GDM and PE.

The dietary inflammatory index (DII) has been developed as the major dietary measurement to evaluate the inflammatory potential of diet, helping to identify the relationship between diet, inflammation and disease. Compared with single food or nutrient intake, DII has been demonstrated to be a reasonable and important indicator for measuring the overall inflammatory potential of diet and an effective tool for studying the relationship between overall diet and diseases[12]. Multiple studies have explored the association between dietary inflammatory potential (evaluated by DII) and the risk of GDM and PE, but the results are inconsistent. A cohort study based on 2639 women in China showed that DII score was positively associated with the risk of GDM[13]. Another case–control study based on 932 women in China suggested that individuals with higher DII score were associated with increased PE risk[14]. However, another Ireland cohort study based on 434 women participants suggested that there was no significant association with GDM or PE[15]. To date, only Gao *et al.*' s meta-analysis[16], which primarily focused on the relationship between diet quality and GDM risk, mentioned the association between DII score and GDM risk. However, their meta-analysis only included four studies and missed some eligible publications. Moreover, no comprehensive and systematic meta-analysis has been performed to evaluate the association between DII score and the risk of PE.

Abbreviations DII, dietary inflammatory index; GDM, gestational diabetes mellitus; PE, preeclampsia.

* **Corresponding author:** Mei Zhao, email zhaomei@ahmu.edu.cn

† These authors have contributed equally to this work and share first authorship.

It is clearly necessary to provide more convincing evidence to clarify the association of DII score with the risk of GDM and PE. Therefore, we performed a systematic review and meta-analysis based on observational studies to explore the association, in order to provide a theoretical basis for maternal diet management.

Methods

This systematic review and meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines[17] and registered in the PROSPERO international prospective register of systematic reviews (CRD42023407877).

Search strategy

Electronic databases, including PubMed, Web of Science, Cochrane Library, EMBASE, Scopus, China National Knowledge Infrastructure, Wan Fang Database and VIP Database, were searched to screen for all relevant published studies on the DII score with risk of GDM and PE from database inception to January 2023, using the following combined keywords: ('dietary inflammatory index' OR 'dietary inflammatory' OR 'DII' OR 'inflammation' OR 'diet') AND ('gestational diabetes mellitus' OR 'gestational diabetes' OR 'GDM' OR 'preeclampsia' OR 'gestational hypertension' OR 'pregnancy-induced hypertension' OR 'pregnancy hypertension' OR 'hypertensive disorder of pregnancy'). Moreover, we also searched the reference lists of pertinent articles for any missing articles.

Inclusion and exclusion criteria

Studies were considered eligible if they met the following criteria: (1) studies reported the association between DII and GDM or PE; (2) studies that reported OR, hazard ratio or risk ratio with 95 % CI and (3) studies with either a cohort study, case–control study or cross-sectional study.

Studies were excluded if they met the following criteria: (1) studies without complete data; (2) studies for which the effect sizes could not be extracted and (3) studies such as systemic reviews, comments, case reports and editorials.

Data extraction

Two researchers independently searched the articles strictly according to the inclusion and exclusion criteria and provided articles with differences to third researcher for analysis to decide whether they should be included. The extracted data included the first author's name, publication year, country, study design, age, pregnancy, sample sizes, DII/DIP/E-DII components, exposure assessment, outcome examined and adjustment for confounders.

Quality assessment

The Newcastle–Ottawa scale was used to evaluate the quality of the literature in all finally included studies[18]. The Newcastle–Ottawa scale consists of three dimensions and eight items, including selection, comparability and outcome (cohort study) or exposure (case–control study). The maximum score for selection, comparability and outcome or exposure was 4, 2 and 3, respectively, for a maximum total score of 9. A total score of 0–3 indicated high risk of bias, 4–6 adequate risk of bias and 7–9 low risk of bias[19]. Studies with a high risk of bias were excluded from the meta-analysis.

Statistical analysis

All statistical analyses were performed by STATA16.0. OR and 95 % CI were selected for the combined effect size; when both crude and adjusted OR were provided, we used the most fully adjusted OR for all studies. Heterogeneity between studies was assessed and quantified with Cochran's Q test and the I^2 statistic, in which $P < 0{\cdot}1$ and $I^2 > 50\,\%$ were defined as statistically significant heterogeneity. When there existed significant heterogeneity, we used a random-effects model; otherwise, a fixed-effects model was used[20]. Subgroup analyses were conducted stratifying by study design, exposure assessment, country, pregnancy, sample size, inflammation assessment tool and energy adjustment. Egger test was conducted to assess the publication bias for each outcome and a sensitivity analysis was performed to examine the robustness of the results.

Results

Study selection and characteristics

A total of 568 articles were retrieved in the database according to the established search strategy, and 405 articles were removed due to duplication. Subsequently, during screening through the title and abstract, 121 irrelevant studies, twenty-three systematic reviews and the other three studies unrelated to GDM were excluded. We then further searched the full text of sixteen articles to assess their eligibility, of which four without complete data, one did not use DII, one study was duplication and one study did not provide useful data were excluded. Eventually, we included nine original studies to investigate the association between DII score with the risk of GDM and PE[13–15,21–26]. The flow of the above-described procedures is shown in Fig. 1.

Nine studies were included in the meta-analysis; there were seven[13,15,22–26] and three[14,15,21] studies evaluating the relationship of DII with GDM and PE. A total of 11 423 participants from various countries were involved, including Finland[26], China[13,14,22,25], Iran[23,24], USA[21] and Ireland[15]. All nine studies were published in the years from 2016 to 2022 inclusively. With regard to study design, there were seven cohort studies and two case–control studies, and the sample sizes ranged from 336 to 4189 participants. All the studies utilised either FFQ or 3-d food diaries as exposure assessment to calculate dietary intake. Continuous DII score was used to evaluate the relationship between DII score with risk of GDM and PE. Adjusted OR was reported in all nine studies and controlled for different types of confounding factors such as age, BMI, energy intake, pregnancy exercise and education level. Detailed information about the nine studies in the analyses is presented in Table 1. According to

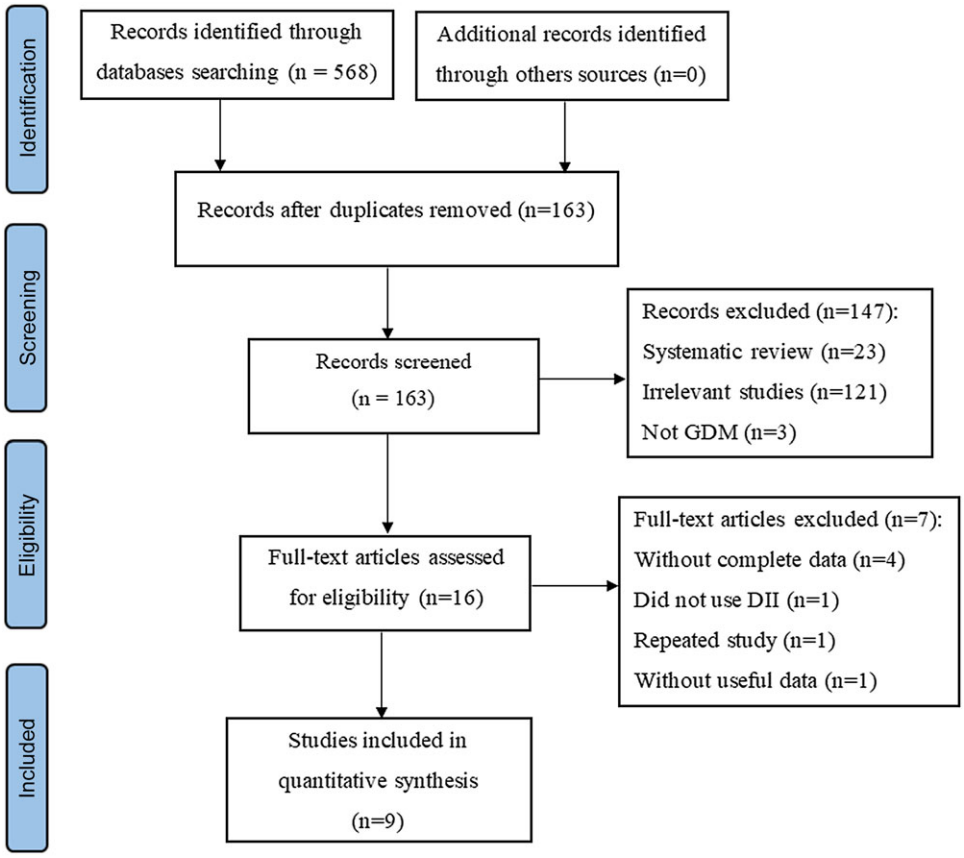

Fig.1. PRISMA flow diagram of study selection process. PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses; GDM, gestational diabetes mellitus; DII, dietary inflammatory index.

the Newcastle–Ottawa scale, three studies scored 6 points, five studies scored 7 points and one study scored 8 points, and the methodological quality of all nine studies was high (Table 2).

Association between dietary inflammatory index and the risk of gestational diabetes mellitus

Seven studies evaluated the association between DII score and the risk of GDM, three studies showed that GDM was associated with DII and the rest were considered unrelated; the total sample sizes were 9149. There was moderate heterogeneity among the studies ($P = 0{\cdot}004$, $I^2 = 68{\cdot}4\,\%$), the meta-analysis result showed that there was a significant association between DII score and risk of GDM (OR = 1·13; 95 % CI 1·02, 1·25) and the difference was statistically significant (Fig. 2).

Association between dietary inflammatory index and the risk of preeclampsia

Three studies evaluated the association between DII score and risk of PE, and one study showed that PE was associated with DII; the total sample sizes were 2708. In the meta-analysis, there was a significant association between DII score and the risk of PE (OR = 1·24; 95 % CI 1·14, 1·35) with heterogeneity ($P = 0{\cdot}125$, $I^2 = 52{\cdot}0\,\%$) (Fig. 3).

Subgroup analysis

Subgroup analysis of PE was not performed because only three studies were included. To explore the sources of heterogeneity between DII score and risk of GDM, a total of seven subgroup variables were selected for meta-analysis (Table 3). Subgroup analysis was conducted for study design and exposure assessment, and the results indicated that both were no significant reduction in heterogeneity. A higher DII score predicted a greater risk of GDM, especially in individuals with mid pregnancy (OR = 1·20; 95 % CI 1·07, 1·34, $I^2 = 36{\cdot}1\,\%$, $P = 0{\cdot}209$). Stratification by country, sample size, inflammation assessment tool and energy adjustment partly reduced the heterogeneity between studies, and the association differed significantly between studies that sample size ≥ 1000 (OR = 1·12; 95 % CI 1·03, 1·20, $I^2 = 0{\cdot}0\,\%$, $P = 0{\cdot}908$) and those that < 1000 (OR = 1·14; 95 % CI 0·96, 1·35, $I^2 = 78{\cdot}3\,\%$, $P = 0{\cdot}001$). Furthermore, stratification by energy adjustment partly reduced the heterogeneity between studies, especially for studies that used covariate method to adjust energy intake ($I^2 = 22{\cdot}2\,\%$) (online Supplementary Fig. S1–S7). In addition to variables in the subgroup analysis, differences in the dietary parameters used to calculate DII score across studies, with a maximum of 35 and a minimum of only 20, were also a possible reason, which may help explain the moderate heterogeneity (online Supplementary Table 1).

Table 1. Characteristics of the included studies

Author, year	Country	Study design	Age (years)	Pregnancy (weeks)	Sample size	Exposure assessment	DII/DIP/E-DII components	Outcome examined	Adjustments
Sen (2016)[21]	USA	Cohort	32·2 ± 5·0	–	1808	FFQ	28	PE	Age, pre-BMI, education, household income, race/ethnicity, smoking during pregnancy, parity
Zhao (2018)[22]	China	Cohort	28·45 ± 3·18	16–20 weeks	336	3-d food diaries	20	GDM	Age, pre-BMI, education level, family income per month, family history of diabetes, hs-CRP, parity, total dietary energy during the second trimester
Shivappa (2019)[23]	Iran	Case–control	18–40	24–28 weeks	388	FFQ	32	GDM	Age, gestational age, BMI, history of diabetes, history of exposure to smoking, exercise, energy, history of supplemental intake
Zhang (2021)[13]	China	Cohort	> 18	8–16 weeks	2639	FFQ	26	GDM	Age, pre-BMI, education level, average personal income, family history of diabetes, smoking and drinking habits, parity, pregnancy exercise, total energy intake, gestational week at FFQ, weight gain before GDM diagnosis, multivitamin supplement use
Soltani (2021)[24]	Iran	Cohort	20–40	8–16 weeks	812	FFQ	29	GDM	Age, baseline-BMI, education, occupation status, physical activity, family number, history of stillbirth, history of preterm delivery, history of caesarean, history of abortion, pregnancy number, maternal weight gain
Yang (2021)[25]	China	Cohort	28·31 ± 3·50	13–28 weeks	4189	FFQ	27	GDM	Age, pre-BMI, education level, average personal income, history of diabetes, smoking, drinking, parity, physical activity, energy, multivitamin/vitamin mineral supplement
Killeen (2021)[15]	Ireland	Cohort	18–45	10–15 weeks	434	3-d food diaries	27	GDM, PE	Age, maternal BMI, ethnicity, economic advantage, smoking, study group
Pajunen (2022)[26]	Finland	Cohort	31·3 ± 4·5	< 18 weeks	351	3-d food diaries	27	GDM	Pre-pregnancy BMI and original trial intervention groups
Liu (2022)[14]	China	Case–control	> 18	> 28 weeks	466	FFQ	35	PE	Age, gestational age, pre-pregnancy BMI, education level, passive smoking, drinking status and dietary supplements used during the 3 months before pregnancy, physical activity and energy intake

DII, dietary inflammatory index; PE, preeclampsia; GDM, gestational diabetes mellitus; DIP, dietary inflammatory potential; E-DII, energy-adjusted dietary inflammatory index.

Table 2. Methodological quality assessment score of the included studies

First author	Selection	Comparability	Outcome/ exposure	Total score
Sen (2016)	3	2	3	8
Zhao (2018)	3	2	2	7
Shivappa (2019)	2	2	2	6
Zhang (2021)	3	2	1	6
Soltani (2021)	3	2	2	7
Yang (2021)	3	2	2	7
Killeen (2021)	3	2	1	6
Pajunen (2022)	3	2	2	7
Liu (2022)	3	2	2	7

Sensitivity analysis

Sensitivity analysis was conducted after removing any one of seven studies, to confirm that our results were not determined by a single study. The results showed that removing most of the studies had no significant effect on our results; the only exceptions were found in the removal of the studies by Zhao[22] and Pajunen[26] changed the relative effect from significant to non-significant in the sensitivity analysis (OR = 1·10; 95 % CI 1·00, 1·20 and OR = 1·11; 95 % CI 1·00, 1·23), as shown in Fig. 4.

Publication bias

Egger test was adopted to evaluate the publication bias included in the study; the Egger test obtained $P = 0{\cdot}202$ for GDM and $P = 0{\cdot}538$ for PE, indicating that there was no significant publication bias in the included studies.

Discussion

This study provides a systematic review and meta-analysis that evaluated all available observational studies on the association between DII score and risk of GDM and PE and provides the most comprehensive evidence in this research field. We included nine studies with 11 423 participants from five different countries. More importantly, we performed a detailed subgroup analysis, including pregnancy, inflammation assessment tools and type of energy adjustment, thus improving statistical power.

The meta-analysis indicated that a 1-unit increase in the DII score was associated with 13 % higher risk of GDM (OR = 1·13; 95 % CI 1·02, 1·25) and 24 % higher risk of PE (OR = 1·24; 95 % CI 1·14, 1·35). Similarly, in a review by Moslehi *et al.*[27], it was concluded that DII was directly associated with increased odds of GDM. In addition, results from our study were in line with other study that reported an association of pro-inflammatory diet indicated by DII, with increased incidence of diabetes mellitus[28]. At present, the relationship between DII score and risk of GDM and PE is controversial, mainly because of the differences of country, sample sizes, energy intake and pregnancy. With regard to country, the pooled results of studies conducted in China[13,14,22,25], including DII score and risk of GDM and PE, showed an association, whereas the other studies indicated no association[15,21,23,24,26]; the differences in country between these studies may partly explain the inconsistent findings. The sample sizes considered ≥ 1000 showed an almost strong association between DII score and GDM risk[13,25]. However, the smaller sample sizes of studies failed to show a significant association between DII score and GDM risk[15,22–24,26]. Therefore, studies assessing the inflammatory potential of diet on GDM risk need larger sample sizes. In addition, among the variables investigated, adjusted for energy intake was critical for achieving significant results, including covariate method[13,22,23,25] and nutrient-density method[15,26], as compared with studies that did not utilise such adjustment[24]. The possible reason is that higher energy intake reflects more food intake, which may be variably distributed among pro-inflammatory and anti-inflammatory related foods. The DII score of women in the mid pregnancy[22,23,25] was more strongly associated with GDM risk than those in the early pregnancy[13,15,24,26], one possible reason is that compared with the mid pregnancy, women in the early pregnancy intake less due to vomiting or other reasons, and are less likely to intake foods with higher DII score.

Diet can regulate the systemic inflammation level through pro-inflammatory or anti-inflammatory mechanisms of foods, nutrients and dietary patterns[29,30]. Studies have shown that higher adherence to the Mediterranean diet was significantly associated with lower levels of inflammation, including reduced levels of CRP (C-Reactive Protein) IL-6 and fibrinogen[31]. The Mediterranean diet is characterised by a high intake of fruits, vegetables, fish and whole grains. It is rich in dietary fibre, vitamin C, vitamin E, folate, carotenoids and PUFA. Previous studies have shown that greater intake of the Mediterranean diet during pregnancy is associated with a reduced risk of GDM and PE[32–35]. The Western diet, characterised by a greater intake of red meat, processed meat and fried foods, is rich in SFA, nitrosamines and other compounds related to oxidative stress and insulin resistance, which may increase the level of serum inflammatory markers[36,37]. Studies have linked a high intake of Western diet and energy from SFA in early pregnancy may contribute to an increased risk of developing GDM and PE[38,39].

From a mechanistic point of view, the role of diet-related inflammation in GDM and PE can be explained by several mechanisms. First, the pro-inflammatory potential of dietary components may led to an imbalance in the level of inflammation by increasing the level of inflammatory factors, such as CRP, TNF-α and IL-6. High level of inflammatory factors induces phosphorylation of insulin receptor substrate-1, leading to its inactivation, and, in turn, inhibits the insulin receptor from phosphorylating this substrate, thus suppressing insulin receptor signalling[40–42]. Inflammatory factors also disrupt the expression of insulin responsive GLUT4, thus reducing insulin-dependent glucose transport and peripheral glucose utilisation[40,41]. Meanwhile, previous studies have shown an association between cytokines and the risk of PE, with women at higher risk of PE when the pro-inflammatory cytokines TNF-α and IL-6 were elevated and the anti-inflammatory cytokines IL-4 and IL-10 were decreased[43,44]. The imbalanced level of inflammation causes activation of associated cellular signalling pathways like Toll-like receptor and NF-κB, and the downstream targets of these pathways may further affect the placental immune

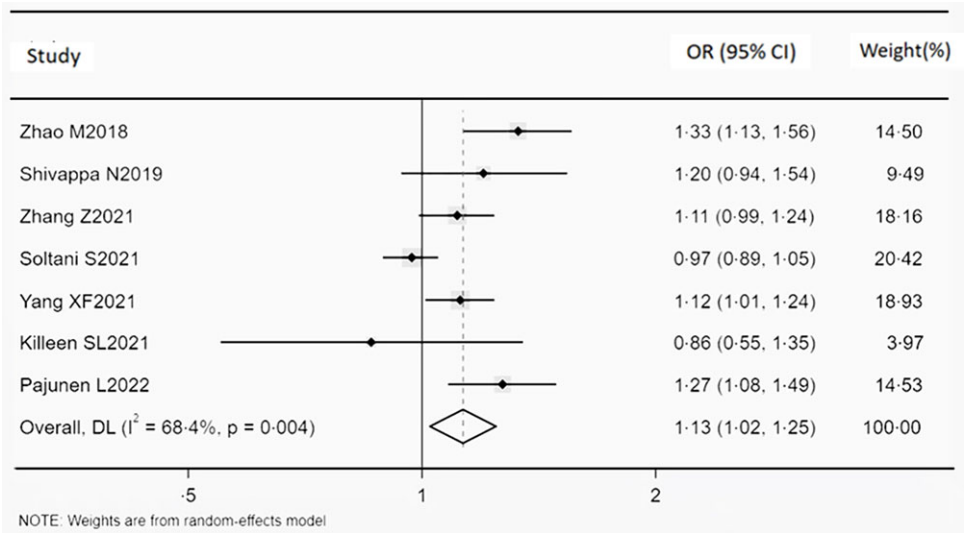

Fig. 2. Forest plot showing the association between the dietary inflammatory index score and the risk of gestational diabetes mellitus.

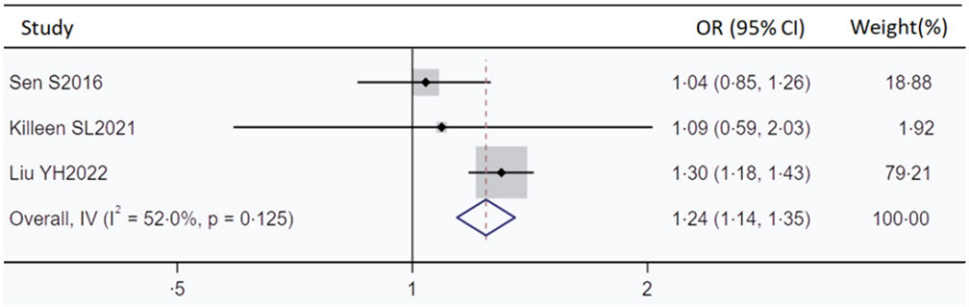

Fig. 3. Forest plot showing the association between the dietary inflammatory index score and the risk of preeclampsia.

Table 3. Subgroup analyses of the association between the DII score and GDM (95 % confidence intervals)

Subgroup	Number of studies	Pooled effect size	95 % CI	*P* (heterogeneity)	I^2 (%)
Total	7	1·13	1·02, 1·25	0·004	68·4
Study design					
Cohort	6	1·12	1·01, 1·25	0·002	72·8
Case–control	1	1·20	0·94, 1·54	–	0·0
Exposure assessment					
FFQ	4	1·07	0·98, 1·18	0·069	57·7
3-d food diaries	3	1·25	1·07, 1·45	0·201	37·7
Country					
China	3	1·16	1·06, 1·28	0·153	46·8
Others	4	1·09	0·91, 1·30	0·014	71·7
Pregnancy					
Early pregnancy	4	1·08	0·94, 1·23	0·014	71·7
Mid pregnancy	3	1·20	1·07, 1·34	0·209	36·1
Sample size					
< 1000	5	1·14	0·96, 1·35	0·001	78·3
≥ 1000	2	1·12	1·03, 1·20	0·908	0·0
Inflammation assessment tool					
DII	4	1·16	1·07, 1·26	0·277	22·2
E-DII	2	1·11	0·77, 1·60	0·109	61·0
DIP	1	0·97	0·89, 1·05	–	0·0
Energy adjustment					
Covariate method	4	1·16	1·07, 1·26	0·277	22·2
Nutrient-density method	2	1·11	0·77, 1·60	0·109	61·0
Non-adjusted	1	0·97	0·89, 1·05	–	0·0

DII, dietary inflammatory index; GDM, gestational diabetes mellitus; E-DII, energy-adjusted dietary inflammatory index; DIP, dietary inflammatory potential.
Early pregnancy, pregnancy < 16 weeks; mid pregnancy, pregnancy ≥ 16 weeks. In addition, we combined the instructions of the authors in the original studies, defined pregnancy < 18 weeks as early pregnancy, 13–28 weeks as mid pregnancy.

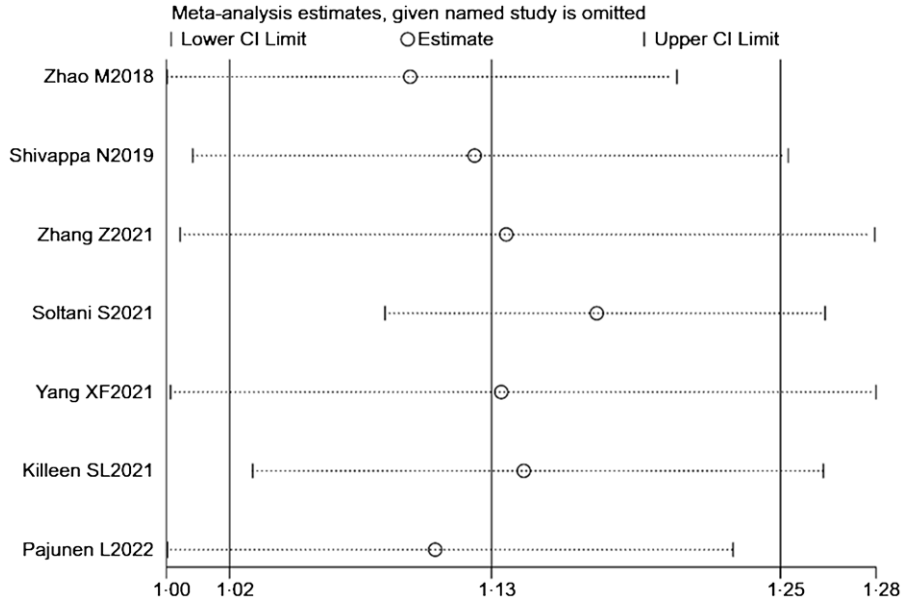

Fig. 4. Sensitivity analysis of dietary inflammatory index and gestational diabetes mellitus.

tolerance by impacting a negative effect on the secretion of trophoblastic microvesicles and endothelial cells disruption, thus leading to several adverse pregnancy complication like PE[43]. Another mechanistic hypothesis relies on the gut microbiota. Gut microbiota are essential modulators of immune homoeostasis, and diet can directly or indirectly affect the composition of gut microbiota, which is associated with gut barrier function[45,46]. For example, a long-term high-fat diet decreases the expression of tight junction proteins in gut epithelial cells and increases the permeability of the gut mucosa, which fails to act as an integral barrier to migrating pathogens; thus, a large amount of bacterial lipopolysaccharide is released into the blood, activating the low-grade chronic inflammation of islets, and inflammation can lead to the structural damage and dysfunction of pancreatic islet B cells, promote B cell apoptosis and cause insufficient insulin secretion, which in turn triggers GDM[46].

However, several limitations of this study should be considered when interpreting the findings of this study. First, the results of this study were based primarily on observational studies, so causality cannot be firmly established. All nine included studies adjusted for confounding factors, but some studies did not adjust for energy intake, gestational weight gain and family history of diabetes, which may be important risk factors for GDM and PE. Most of the included studies were cohort studies, which had the risk of loss to follow-up, but some studies did not describe it. In addition, the substantial heterogeneity across studies was not fully eliminated with subgroup meta-analyses by common study characteristic. Finally, generalisability of our findings to diverse populations should be taken with caution because most of the participants were of Chinese.

We suggest the following recommendations for future studies. Future studies can be carried out in large samples and multi-centre populations, and at the same time, the race, economic status and BMI of the population should be considered in the sampling. The existing studies focused on the association between DII score and risk of GDM and PE, but there were few discussions on the mechanism, which can be studied in the future. Finally, when possible, adequately powered randomised controlled trials should be conducted to support better causal inferences.

Conclusion

In conclusion, this study conducts a systematic review and meta-analysis of nine included studies, and the results show that pro-inflammatory diet estimated by a higher DII score is independently associated with an increased risk of GDM and PE. The finds prompt interventions of GDM and PE patients from the perspective of dietary inflammation. However, more prospective longitudinal studies with improved methodology are warranted to confirm the current findings.

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L. H., L. Y. Z. and M. Z. designed this study. M. Z. obtained the funding. L. H. and L. Y. Z. extracted and confirmed the data. L. H. performed the statistical analysis and drafted the initial manuscript. L. H., L. Y. Z., M. Z., J. R. Z., Y. Q. F. and X. Y. Q. made substantial revisions to the manuscript. All authors read and approved the final manuscript.

There are no conflicts of interest.

Supplementary material

For supplementary material referred to in this article, please visit https://doi.org/10.1017/S0007114523001678

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