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Relationship between prepregnancy BMI and gestational weight gain(GWG) with preeclampsia: a study based on restricted cubic spline

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Abstract

Objective This study aimed to evaluate the nonlinear associations between prepregnancy body mass index(BMI), gestational weight gain(GWG), and the risk of preeclampsia(PE) using maternal and infant cohort data, thereby providing a scientific foundation for preventive strategies.

Methods Pregnant women with regular obstetric checkups in Urumqi Youai Hospital were selected as study subjects from January 2020 to June 2024. They were divided into the PE group and the non-PE group. Baseline information and pregnancy outcomes were collected, and logistic regression analysis was employed to ascertain the impacts of diverse factors on the odds of developing PE; the restricted cubic spline was used to test the nonlinear relationship between prepregnancy BMI and GWG with PE.

Results 13,294 pregnant women were included in the study, and 559 (4.20%) had PE.The prevalence of PE in underweight, normal-weight, overweight, and obese women was 1.72%, 2.85%, 6.60%, and 16.05%, respectively. However, after adjusting for confounders, only overweight and obesity were significantly associated with elevated PE odds. Logistic regression results showed that the OR was 1.68(95% CI:1.30–2.18) for the comparison between overweight and normal BMI groups, the OR was 3.16(95%CI:2.08–4.79) for the comparison between obesity and normal BMI groups. Restricted cubic spline showed that the association between prepregnancy BMI and the odds of PE showed an inverse L-shaped curve, with an inflection point of 21.5 kg/m^2; the association between GWG and the odds of PE showed a J-shaped curve, with a GWG of 10.94–15.90 kg being at the lowest odds for the development of PE. For pregnant women with prepregnancy underweight, the odds of PE were significantly increased when their GWG exceeded 21.63 kg. Similarly, for those with prepregnancy normal weight, a significant elevation in the odds of PE was observed when their GWG surpassed 15.90 kg.

Conclusion There is a non-linear relationship between prepregnancy BMI, GWG, and PE, and prepregnancy weight management and gestational weight monitoring are important for the prevention of PE.

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Keywords Preeclampsia, Prepregnancy BMI, Gestational weight gain, Restricted cubic spline

Introduction

Preeclampsia(PE) is a pregnancy-specific syndrome that typically manifests with hypertension and proteinuria after the 20th week of gestation. However, in some cases, it can present without proteinuria but with evidence of end-organ dysfunction [1]. Its global prevalence ranges from approximately 2–8%, contributing significantly to maternal and infant mortality and increasing the long-term risk of chronic diseases [2]. The etiology of this complex disease remains elusive due to its multifactorial nature [3]. Numerous studies have demonstrated a strong correlation between a high prepregnancy body mass index(BMI) and the development of preeclampsia [4–7], and excessive gestational weight gain(GWG) may also heighten the risk [8, 9].There may be a complex dose-response relationship between gestational weight gain and preeclampsia, and further research is needed to determine the specific weight gain ranges and odds thresholds.Restricted Cubic Spline(RCS) Modeling allows for a more flexible fit of the data by using cubic splines. By dividing the range of the predictor variable(e.g., gestational weight gain) into intervals and fitting a cubic polynomial within each interval, it can capture the nonlinear trends in the data more accurately. In clinical practice, assessing the impact of gestational weight gain on the development of preeclampsia in individuals with different prepregnancy BMI is more valuable, yet this area has been seldom explored.This study utilized data from a maternal-infant cohort and applied RCS modeling.The primary aim was to precisely quantify the nonlinear associations between gestational weight gain and the odds of preeclampsia among pregnant women stratified by different prepregnancy BMI categories.The study intends to offer an evidence-based, accurate scientific basis for formulating personalized and effective prevention strategies against preeclampsia tailored to women with diverse prepregnancy BMI.

Information and methods

Information

This study enrolled subjects who underwent regular obstetric examinations and were documented at Urumqi Youai Hospital from January 2020 to June 2024. Participants were categorized into PE and non-PE groups based on the occurrence of PE. The inclusion criteria were as follows: (1)Diagnosis of PE by the "Guidelines for the Diagnosis and Treatment of Hypertensive Disorders in Pregnancy (2020)"; (2)Singleton pregnancy; (3)Age range of 18–49 years; (4)Absence of other pregnancy complications and no other concurrent complications or comorbidities during pregnancy. The exclusion criteria included: (1)Prepregnancy neurological disorders, infectious diseases, immunological disorders, and a history of hypertension, hyperlipidemia, other cardiovascular diseases, kidney diseases, and other conditions; (2) Incomplete basic information. Data collected included participants' names, ethnicity, age, diagnosis, height, prepregnancy weight, primiparity, assisted reproductive technology(ART), pre-delivery weight, mode of delivery, newborn gender, newborn weight, one-minute Apgar scores, and contact phone numbers. Ethical approval for this study was granted by the Ethics Committee of Urumqi Youai Hospital(approval number: WLMQY-ALL2022003), and all participants provided written informed consent.

The Prepregnancy BMI and GWG were determined using standard anthropometric calculations based on the pregnant women's prepregnancy height and weight, as well as their weight at the time of delivery. The prepregnancy BMI was calculated by dividing the self-reported prepregnancy weight in kilograms by the square of the height in meters, as measured during the initial prenatal care visit. Gestational weight gain was defined as the difference between the weight at delivery and the prepregnancy weight. According to the recommended value of weight gain of pregnant women(WS/T 801–2022), the prepregnancy BMI was classified into three categories: Underweight: BMI < 18.5 kg/m^2; Normal weight: BMI 18.5–23.9 kg/m^2;Overweight: BMI 24–27.9 kg/m^2; Obesity: BMI ≥ 28 kg/m^2.

Statistical analysis

Data processing and analysis were performed using JMP 14.0 and R version 4.4.0. Data conforming to a normal distribution were presented as mean ± standard deviation, and comparisons between two independent samples were performed using the independent samples *t*-test. For data that did not conform to a normal distribution, results were expressed as median and interquartile ranges (P_{25}, P_{75}), and comparisons between two independent samples were conducted using the nonparametric Mann-Whitney U test. Categorical data were described by the number of cases and the corresponding proportions (n, %), with group comparisons made using the chi-squared (χ^2) test.To explore the nonlinear relationship between prepregnancy BMI, GWG, and the odds of PE, a logistic regression model incorporating restricted cubic splines was employed. The *P* value of < 0.05 was considered statistically significant.

Results

Comparison of baseline characteristics and pregnancy outcomes

A directed acyclic graph (DAG) was constructed to identify potential confounders for PE (Fig. 1). Variables included prepregnancy BMI, GWG, ethnicity, maternal age, primiparity, and ART.

A total of 13,294 pregnant women were enrolled in this study, comprising 559 women with PE(4.20%) and 12,735 women in the non-PE group(95.80%). The incidence of PE varied across different BMI categories:1.72% in women with underweight, 2.85% in women with normal weight, 6.60% in women with overweight, and 16.05% in women with obesity. Significant differences were observed in the distribution of prepregnancy weight, prepregnancy BMI, primiparity, ART, GWG, mode of delivery, one-minute apgar score, and neonatal birth weight across the PE and non-PE groups($P<0.05$). These findings are detailed in Table 1.

Multivariable logistic regression analysis for PE

The multivariable logistic regression model (Table 2) was adjusted for ART, primiparity, prepregnancy BMI, GWG, and cesarean delivery. These variables were selected based on univariate significance($P<0.10$) or established relevance to PE odds in prior literature.The conditional forward selection method was employed to derive the logistic regression equations, and the odds ratios(ORs) along with their 95% confidence intervals(*CIs*) were calculated. The results, presented in a forest plot, indicated the following: Each unit increase in prepregnancy weight was associated with a 4% increase in the odds of PE(OR = 1.04, 95%CI: 1.03–1.05),and each unit increase in GWG was associated with a 7% increase in the odds of PE(OR = 1.07, 95%CI: 1.05–1.08).The OR was 2.12(95% CI: 1.33–3.37) for the comparison between pregnant women using ART and those not using such technology. The OR was 1.92(95% CI: 1.55–2.36) for the comparison between primiparous women and multiparous women. The OR was 1.68(95% CI: 1.30–2.18) for the comparison between overweight pregnant women and normal weight pregnant women.The OR was 3.16(95% CI: 2.08–4.79) for the comparison between obese pregnant women and normal weight pregnant women.Prepregnancy BMI, weight gain, uyghur ethnicity, primiparity, and cesarean section were found to be significantly and positively associated with the odds of PE. Further details are provided in Table 2; Fig. 2.

RCS of prepregnancy BMI and GWG with PE

Employing restricted cubic spline regression analysis with four knots, we modeled the relationship between prepregnancy BMI and the odds of preeclampsia, adjusting for ethnicity, use of assisted reproductive technology, and primiparity.The spline curve revealed an inverse L-shaped association, with an inflection point at a prepregnancy BMI of 21.5 kg/m². Beyond this threshold, there was a significant increase in the odds of preeclampsia for each additional unit increase in BMI (Fig. 3a).

The relationship between GWG and the risk of preeclampsia was J-shaped, indicating that the lowest odds of preeclampsia was observed when GWG ranged from

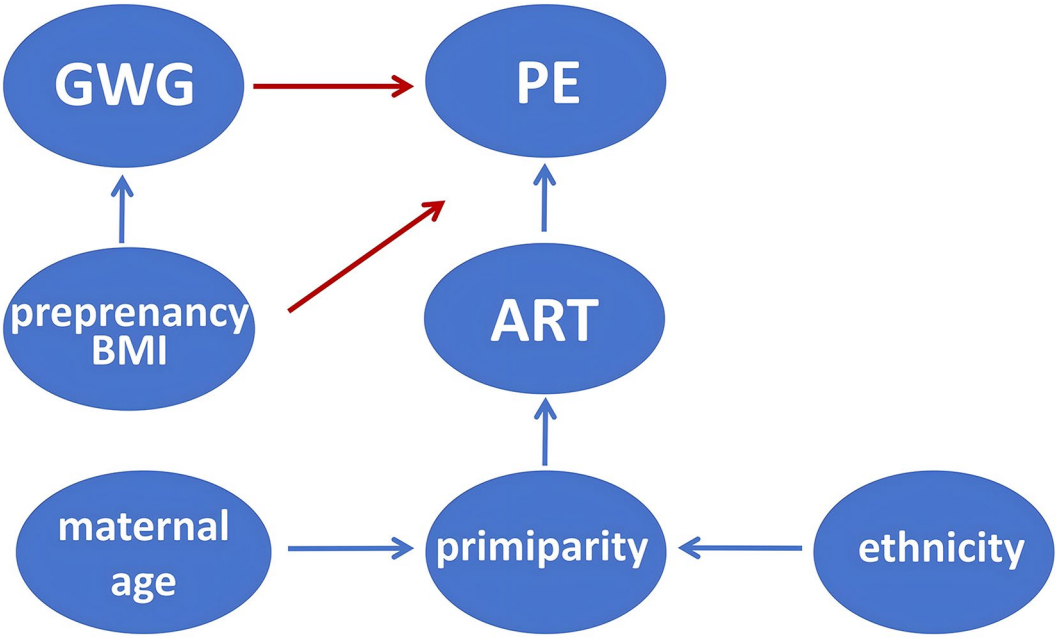

Fig. 1 A directed acyclic graph to identify potential confounders (DAG illustrating causal pathways. Red arrows: direct effect; blue arrows: indirect effect)

Table 1 Comparison of baseline information and pregnancy outcomes in different groups of pregnant women

Variables	Total ($n=13294$)	Non-preeclampsia ($n=12735$)	Preeclampsia($n=559$)	Statistic	*P*
Year, No. (%)				χ^2=9.34	0.053
2020	984 (7.40)	951 (7.47)	33 (5.90)		
2021	2559 (19.25)	2455 (19.28)	104 (18.60)		
2022	4519 (33.99)	4341 (34.09)	178 (31.84)		
2023	3588 (26.99)	3434 (26.97)	154 (27.55)		
2024	1644 (12.37)	1554 (12.20)	90 (16.10)		
Height, Mean ± SD	163.04 ± 5.36	163.04 ± 5.33	163.13 ± 5.93	*t*=-0.36	0.718
Age, M (P_{25}, P_{75})	30.00 (28.00, 33.00)	30.00 (28.00, 32.00)	30.00 (27.00, 33.00)	*Z*=-1.51	0.130
Prepregnancy weight, M (P_{25}, P_{75})	57.00 (52.00, 63.50)	57.00 (52.00, 63.00)	64.00 (57.00, 74.00)	*Z*=-15.00	< 0.001
GWG, M (P_{25}, P_{75})	15.80 (12.50, 19.00)	15.60 (12.50, 19.00)	16.70 (13.00, 21.00)	*Z*=-4.36	< 0.001
Prepregnancy BMI, M (P_{25}, P_{75})	21.48 (19.61, 23.83)	21.40 (19.57, 23.67)	24.09 (21.63, 27.81)	*Z*=-16.64	< 0.001
Prepregnancy BMI, n (%)					
Underweight [BMI < 18.5]	1567 (11.79)	1540 (12.09)	27 (4.83)	χ^2=386.83	< 0.001
Normal [18.5 ≤ BMI < 24]	8558 (64.37)	8314 (65.28)	244 (43.65)		
Overweight [24 ≤ BMI < 28]	2334 (17.56)	2180 (17.12)	154 (27.55)		
Obesity [BMI ≥ 28]	835 (6.28)	701 (5.50)	134 (23.97)		
Assisted reproductive technology, n(%)				χ^2=13.51	< 0.001
yes	249 (1.87)	227 (1.78)	22 (3.94)		
no	13,045 (98.13)	12,508 (98.22)	537 (96.06)		
Primigravida, n(%)				χ^2=27.14	< 0.001
No	4341 (32.65)	4215 (33.10)	126 (22.54)		
Yes	8953 (67.35)	8520 (66.90)	433 (77.46)		
Mode of delivery, n(%)				χ^2=185.94	< 0.001
Cesarean section	6784 (51.03)	6341 (49.79)	443 (79.25)		
Vaginal delivery	6510 (48.97)	6394 (50.21)	116 (20.75)		
Gender, n(%)				χ^2=1.60	0.206
Male	6840 (51.45)	6567 (51.57)	273 (48.84)		
Female	6454 (48.55)	6168 (48.43)	286 (51.16)		
Weight, M(P_{25}, P_{75})	3380.00 (3100.00, 3660.00)	3380.00 (3110.00, 3670.00)	3170.00 (2762.50, 3540.00)	*Z*=-10.53	< 0.001
Score, n(%)				χ^2=12.87	< 0.001
Below 7	135 (1.02)	121 (0.95)	14 (2.50)		
8–10	13,159 (98.98)	12,614 (99.05)	545 (97.50)		

t: *t*-test, *Z*: Mann-Whitney test, χ^2: Chi-square test

10.94 to 15.90 kg (Fig. 3b). For underweight pregnant women(prepregnancy BMI < 18.5 kg/m^2), it is the GWG reaching above 21.63 kg that is associated with an elevated odds of preeclampsia.The odds of PE was significantly increased in normal-weight pregnant women with GWG exceeding 15.90 kg. (Fig. 3c).

Discussion

Despite the global decline in maternal mortality rates, PE continues to be a leading cause of maternal and neonatal mortality. Over the past half-century, there has been a lack of significant advancements in the prevention or treatment of PE [10]. Prepregnancy BMI is acknowledged as a significant risk factor for the development of PE. This study employed restricted cubic spline analysis to explore the impact of prepregnancy BMI and GWG on the odds of PE in the Xinjiang, providing a theoretical basis for the early prediction of PE.

Influencing factors of PE

Women who utilized ART were found to have a significantly increased odds of PE in both univariate(OR = 2.26) and multivariate analyses(OR = 2.12), suggesting that ART may be an independent odds factor for the condition. The Hui Ju Chih study reported a higher relative risk of developing gestational hypertension among singleton pregnant women who used ART compared to those who conceived naturally(OR 1.70; 95%CI 1.60–1.80) [11]. Potential mechanisms underlying this association include epigenetic alterations leading to abnormal placentation, the absence of luteinizing factors, and immune responses to homozygous gametes [12].

Primiparous women also demonstrated significantly increased odds of PE in both univariate and multivariate analyses(OR = 1.70 and 1.92). While studies consistently identify primiparity as a prevalent susceptibility factor for PE, the underlying reasons remain elusive. One hypothesis posits that the immune system of primiparous women, having had limited exposure to paternal antigens from the fetus,

Table 2 Results of single-factor and multifactor logistic regressions

Variables	Single factor analysis					Multifactor analysis				
	β	S.E	Z	*P*	OR (95%CI)	β	S.E	Z	*P*	OR (95%CI)
Ethnic origin										
Han					1.00 (Reference)					1.00 (Reference)
Hui	0.24	0.14	1.73	0.084	1.28 (0.97 ~ 1.68)	0.13	0.15	0.87	0.386	1.14 (0.85 ~ 1.51)
Other ethnic minorities	0.49	0.22	2.28	0.023	1.64 (1.07 ~ 2.50)	0.42	0.22	1.89	0.059	1.53 (0.98 ~ 2.37)
Uyghur	0.89	0.16	5.67	<0.001	2.42 (1.78 ~ 3.29)	0.76	0.16	4.61	<0.001	2.14 (1.55 ~ 2.96)
Assisted reproductive technology										
no					1.00 (Reference)					1.00 (Reference)
yes	0.81	0.23	3.58	<0.001	2.26 (1.45 ~ 3.53)	0.75	0.24	3.17	0.002	2.12 (1.33 ~ 3.37)
Primigravida										
no					1.00 (Reference)					1.00 (Reference)
yes	0.53	0.10	5.15	<0.001	1.70 (1.39 ~ 2.08)	0.65	0.11	6.08	<0.001	1.92 (1.55 ~ 2.36)
Prepregnancy BMI										
Normal					1.00 (Reference)					1.00 (Reference)
Underweight	-0.52	0.20	-2.52	0.012	0.60 (0.40 ~ 0.89)	-0.21	0.22	-0.97	0.333	0.81 (0.53 ~ 1.24)
Overweight	0.88	0.11	8.31	<0.001	2.41 (1.96 ~ 2.96)	0.52	0.13	3.91	<0.001	1.68 (1.30 ~ 2.18)
Obesity	1.87	0.11	16.37	<0.001	6.51 (5.20 ~ 8.15)	1.15	0.21	5.41	<0.001	3.16 (2.08 ~ 4.79)
Prepregnancy weight	0.06	0.00	18.12	<0.001	1.07 (1.06 ~ 1.07)	0.04	0.01	5.96	<0.001	1.04 (1.03 ~ 1.05)
GWG	0.04	0.01	4.62	<0.001	1.04 (1.02 ~ 1.06)	0.06	0.01	7.92	<0.001	1.07 (1.05 ~ 1.08)

OR: Odds Ratio, *CI*: Confidence Interval

Adjusted for ethnicity, ART, primiparity, cesarean delivery, prepregnancy BMI, and GWG

may lack the necessary desensitization, potentially contributing to the pathogenesis of PE [13]. This may relate to primiparous women's adaptation to the physiological changes of pregnancy, placental formation, and maternal vascular adaptations.

The rate of cesarean delivery was markedly higher in the preeclamptic group than in the non-preeclamptic group, possibly due to the increased risk that PE poses to both the mother and fetus. Physicians may opt for cesarean delivery to mitigate complications during labor [14]. Additionally, both birth weight and apgar scores were significantly lower in the PE group compared to the non-PE group, echoing the findings of Melek Büyükeren et al. [15]. The median and interquartile range of birth weight in the PE group were notably lower than those in the control group(1540(960 g, 1920 g) vs. 3135(2850 g, 3440 g)), indicating that PE may adversely affect fetal growth, development, and postnatal health status.

While the incidence of PE was numerically lower in underweight women(1.72%) compared to normal-weight women(2.85%), this difference was not statistically significant in adjusted analyses(OR = 0.81,95%CI:0.53–1.24). This suggests that prepregnancy underweight status, as defined in our cohort, may not independently influence PE odds. Further studies with larger sample sizes are needed to explore this relationship.In contrast, overweight and obese women had significantly increased odds of PE(OR = 1.68, 95%CI:1.30–2.18 and OR = 3.16, 95%CI:2.08–4.79, respectively; $P < 0.001$ for both).Both prepregnancy weight and GWG were positively correlated with the odds of PE. For each unit increase in prepregnancy weight, the odds of PE increased by 4%(OR = 1.04), and for each unit increase in GWG, the odds increased by 7%(OR = 1.07).Other studies also support that prepregnancy overweight and excessive GWG are independently associated with an increased odds of PE [16]. This may be related to metabolic and vascular adaptive changes caused by weight gain. Overweight/obesity is considered a chronic inflammatory disease, which can increase the levels of plasma C-reactive protein and certain inflammatory cytokines [17].The metabolic and biochemical disturbances associated with overweight and obesity lead to an increase in neutrophils, releasing toxic compounds(i.e. reactive oxygen species and myeloperoxidase), capable of attacking and destroying the integrity of vascular endothelial cells. This mechanism ultimately leads to the clinical symptoms of PE [2]. The study emphasizes the importance of prepregnancy weight management in the prevention of PE.

Results of RCS analysis

This study demonstrates that the relationship between prepregnancy BMI and the odds of PE is not a straightforward linear increase or decrease; rather, the odds significantly escalate after reaching a specific BMI threshold. Specifically, prepregnancy BMI < 21.5 kg/m² may appears to offer more protection against PE, suggesting that risk factors may not be significantly activated at this level. Conversely, beyond this threshold, the odds of PE increase markedly with each unit increase in BMI. This phenomenon may be linked to obesity-related physiological changes, such as inflammation, insulin resistance, and endothelial dysfunction, which

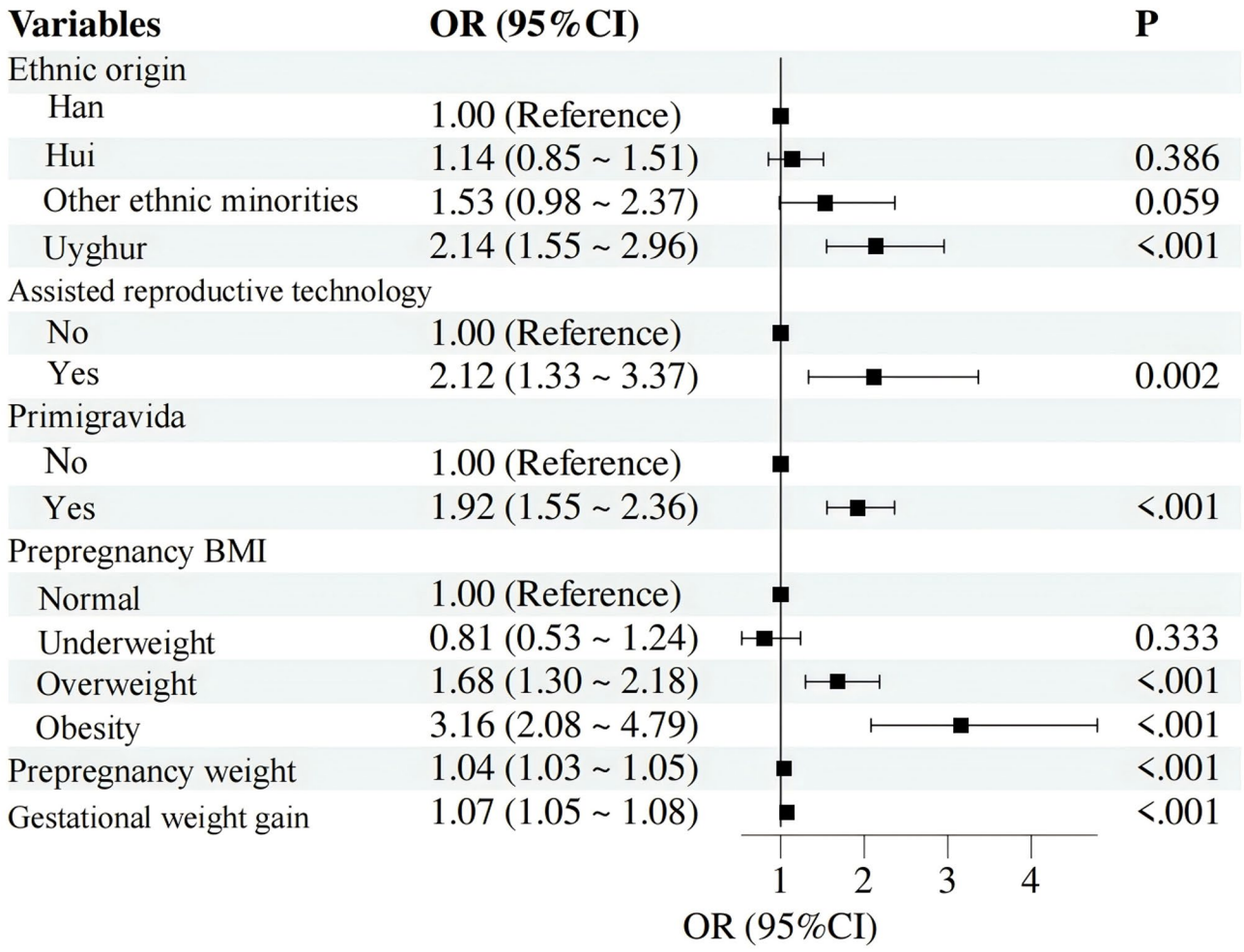

Fig. 2 Forest plot of multifactor logistic regression analysis

can promote the development of PE [18].The inflection point identified in the study by Xiaoli Gong was 24 kg/m², which supports our findings [19]. Women with a prepregnancy BMI above 21.5 kg/m² should adopt more proactive weight management and health interventions to mitigate the odds of PE.

Additionally, the odds of PE was found to be lowest when GWG between 10.94 and 15.90 kg. This range likely reflects a better nutritional balance for both the pregnant woman and the fetus, avoiding both malnutrition and excessive weight gain, thereby reducing the odds of PE. Exceeding this range, particularly with excessive GWG, may heighten the odds of PE. This is potentially due to physiological stresses associated with excessive weight gain, such as increased adipose tissue that releases more inflammatory factors and adversely affects vascular function [20]. A population-based cohort study in Slovenia identified high GWG as a significant risk factor for PE, especially among underweight women, while low GWG served as a protective factor for obese women [9].These findings underscore the critical importance of managing prepregnancy BMI and GWG in the prevention of PE. Effective weight control strategies before and during pregnancy should be considered essential to reduce the odds of developing this condition.

The subgroup analysis examining the impact of GWG in pregnant women across various prepregnancy BMI categories revealed significant findings.For pregnant women with prepregnancy underweight, the odds of PE was significantly increased when their GWG exceeded 21.63 kg. Similarly, for those with prepregnancy normal weight, a significant elevation in the odds of PE was observed when their GWG surpassed 10.94 kg.These thresholds suggest that the interplay between prepregnancy BMI and GWG is crucial in determining the odds of PE.A study from Reunion Island, which tracked 57,000 singleton pregnancies over 18.5 years, demonstrated an independent association between optimal GWG and late-onset PE. This association was characterized by a reduced risk of late-onset PE, with a corrected odds ratio of 0.74, $P=0.004$ [21]. Interestingly, the effect of optimal GWG was more pronounced and linear in overweight and obese patients, indicating a stronger influence of

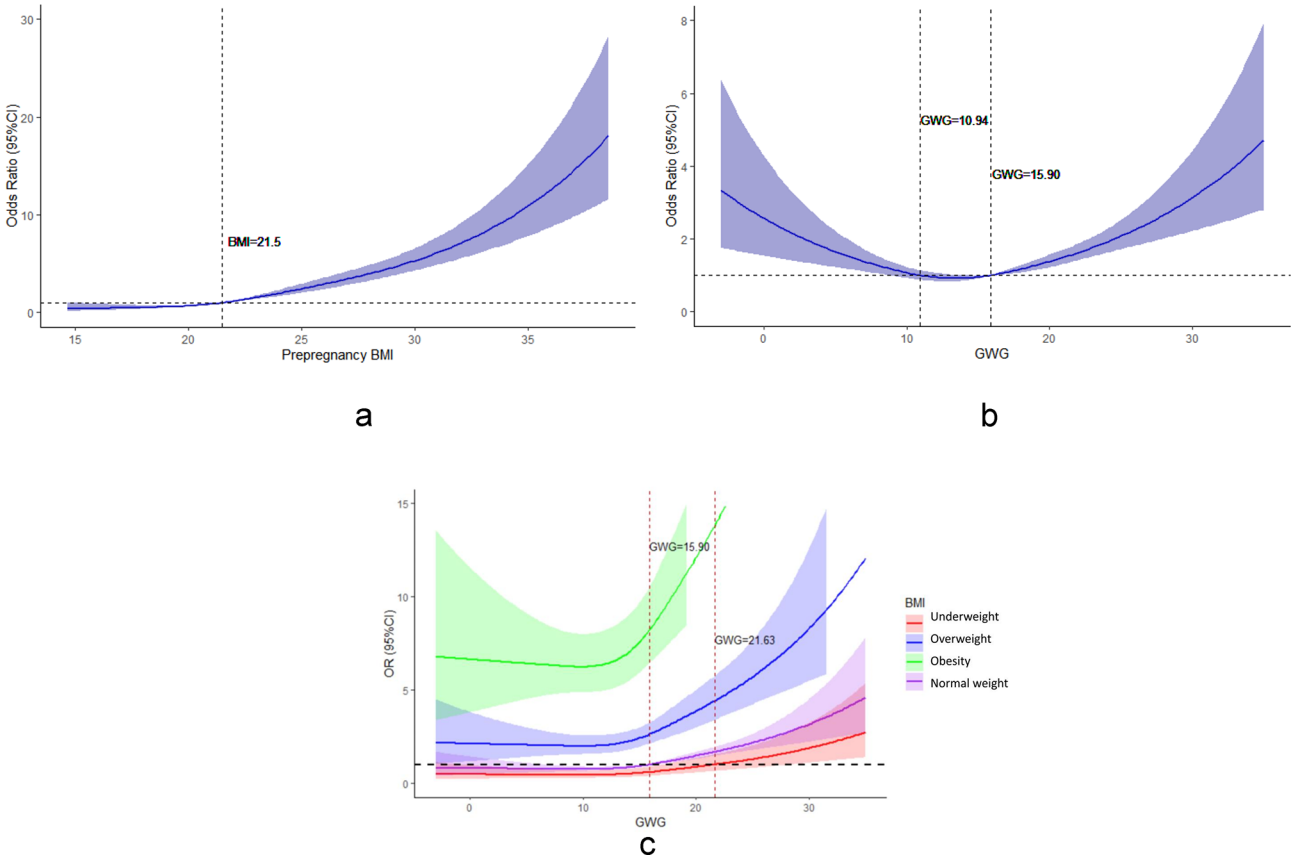

Fig. 3 Restricted cubic spline graphs of prepregnancy BMI (**a**), gestational weight gain (**b**), and gestational weight gain at different prepregnancy body mass index (underweight, normal weight, overweight, and obesity) subgroups (**c**) in relation to the odds of preeclampsia

GWG on PE risk in these groups [22].The metabolic status of mothers before and early in pregnancy has been shown to influence early placental function and gene expression. Alterations in maternal placental function, such as the adaptation of maternal adipose tissue, are dependent on the prior activation of genes controlling adipogenesis and low-grade inflammation in early pregnancy. These metabolic changes precede any observable alterations in maternal phenotype, highlighting the importance of early pregnancy metabolic health in the development of PE.

Conclusion of the study

In conclusion, our study employed logistic regression analysis combined with the RCS function to elucidate the nonlinear relationship between prepregnancy BMI, GWG, and the risk of PE.Our findings complement existing IOM guidelines but propose stricter upper limits for overweight and obese women to account for the nonlinear escalation in PE risk. For underweight women, while our data did not show significant PE risk reduction, adhering to IOM-recommended GWG ranges (12.0–18.0 kg) balances fetal health and maternal safety.Clinicians should prioritize BMI-specific GWG targets—particularly limiting gains to ≤ 9.0 kg in obese women and ≤ 11.0 kg in overweight women—to reduce PE risk. Normal-weight women may safely gain 11.0–15.0 kg, while underweight women should aim for 12.0–18.0 kg.The findings underscore the critical role of prepregnancy weight management and gestational weight monitoring in the prevention of PE.It is essential to reinforce these targets through structured antenatal counseling and regular weight monitoring as part of standardized clinical care. Early and continuous prenatal visits should focus on personalized weight management plans, empowering women to monitor their gestational weight gain while addressing any modifiable risk factors.Future research could focus on developing personalized weight management programs for pregnant women across different prepregnancy BMI categories, emphasizing scalable tools for antenatal education and real-time weight tracking to further reduce the risk of PE.

Study limitations

It is important to acknowledge the limitations of this study. Despite the large sample size, the data were derived from hospitals in a single geographic region, which may introduce selection bias. Additionally, we were unable to collect comprehensive data on factors such as smoking, alcohol consumption, and dietary status of pregnant women, which

could not be adequately controlled for in our analysis. These unmeasured confounding factors might influence the relationship between prepregnancy BMI, GWG, and PE risk. Therefore, future studies should aim to collect more comprehensive demographic information in cohort studies to validate our findings and to better understand the complex interplay of factors contributing to PE.

Author contributions
Luhan Zhang wrote the main manuscript text, Data Analysis.Juan Ding contributed to data collection and interpretation. Jiangling Liu prepared Figs. 2 and 3, Contributed to the discussion. Jing Ma performed statistical analysis, Contributed to the results section. Rui Shi assisted with data interpretation, Reviewed and edited the manuscript. Tian Chen assisted with the literature review, Contributed to the methods section. Guifeng Ding supervised the project, Provided critical feedback on the manuscript.All authors reviewed the manuscript.

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Data availability
The study data could be provided on the request from co-authors.

Declarations

Ethics approval and consent to participate
This study strictly adhered to the Declaration of Helsinki, fulfilling its requirements for participant autonomy, data governance, and protection of vulnerable populations.We ensure that all procedures were performed in compliance with relevant laws and institutional guidelines and have been approved by the appropriate institutional committees.We obtained the necessary ethical approvals (approved by The Ethics Committee of Urumqi Youai Hospital (approval number: WLMQYALL2022003) and ensured informed consent from all participants.The collection and processing of research data are in accordance with privacy protection principles, and all personally identifiable information has been removed or encrypted.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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References

1. Gestational Hypertension and Preeclampsia. ACOG practice bulletin, number 222. Obstet Gynecol. 2020;135(6):e237–60.
2. Rana S, Lemoine E, Granger JP, Karumanchi SA. Preeclampsia: pathophysiology, challenges, and perspectives. Circ Res. 2019;124(7):1094–112.
3. Dimitriadis E, Rolnik DL, Zhou W, Estrada-Gutierrez G, Koga K, Francisco RPV, Whitehead C, Hyett J, da Silva Costa F, Nicolaides K, et al. Pre-eclampsia. Nat Rev Dis Primers. 2023;9(1):8.
4. Mrema D, Lie RT, Østbye T, Mahande MJ, Daltveit AK. The association between pre pregnancy body mass index and risk of preeclampsia: a registry based study from Tanzania. BMC Pregnancy Childbirth. 2018;18(1):56.
5. D'Souza R, Horyn I, Pavalagantharajah S, Zaffar N, Jacob CE. Maternal body mass index and pregnancy outcomes: a systematic review and metaanalysis. Am J Obstet Gynecol MFM. 2019;1(4):100041.
6. Ren QW, Yang FF, Han TB, Guo MZ, Zhao N, Feng YL, Yang HL, Wang SP, Zhang YW, Wu WW. [Relationship between the pre-pregnancy BMI, gestational weight gain, and risk of preeclampsia and its subtypes]. Zhonghua Liu Xing Bing Xue Za Zhi. 2021;42(11):2037–43.
7. Barber E, Ram M, Mor L, Ganor Paz Y, Shmueli A, Bornstein S, Barda G, Schreiber L, Weiner E, Levy M. Pregnancy and placental outcomes according to maternal BMI in women with preeclampsia: a retrospective cohort study. Arch Gynecol Obstet. 2024;309(6):2521–8.
8. Bodnar LM, Himes KP, Abrams B, Parisi SM, Hutcheon JA. Early-pregnancy weight gain and the risk of preeclampsia: A case-cohort study. Pregnancy Hypertens. 2018;14:205–12.
9. Premru-Srsen T, Kocic Z, Fabjan Vodusek V, Geršak K, Verdenik I. Total gestational weight gain and the risk of preeclampsia by pre-pregnancy body mass index categories: a population-based cohort study from 2013 to 2017. J Perinat Med. 2019;47(6):585–91.
10. Chen Z, Qi H. Preeclampsia diagnosis and treatment confusion and reflection. Chin J Obstet Gynecol. 2024;59(01):86–9.
11. Chih HJ, Elias FTS, Gaudet L, Velez MP. Assisted reproductive technology and hypertensive disorders of pregnancy: systematic review and meta-analyses. BMC Pregnancy Childbirth. 2021;21(1):449.
12. Kornfield MS, Gurley SB, Vrooman LA. Increased risk of preeclampsia with assisted reproductive technologies. Curr Hypertens Rep. 2023;25(9):251–61.
13. Di Mascio D, Saccone G, Bellussi F, Vitagliano A, Berghella V. Type of paternal sperm exposure before pregnancy and the risk of preeclampsia: A systematic review. Eur J Obstet Gynecol Reprod Biol. 2020;251:246–53.
14. Adeosun F, Folayan O, Ojo T. Choosing Cesarean section over natural birth: challenges of decision making among pregnant women with pre-eclampsia in Ado-Ekiti. Pregnancy Hypertens. 2022;30:97–102.
15. Büyükeren M, Çelik HT, Örgül G, Yiğit Ş, Beksaç MS, Yurdakök M. Neonatal outcomes of early- and late-onset preeclampsia. Turk J Pediatr. 2020;62(5):812–9.
16. Shao Y, Qiu J, Huang H, Mao B, Dai W, He X, Cui H, Lin X, Lv L, Wang D, et al. Pre-pregnancy BMI, gestational weight gain and risk of preeclampsia: a birth cohort study in Lanzhou, China. BMC Pregnancy Childbirth. 2017;17(1):400.
17. Esposito K, Nicoletti G, Giugliano D. Obesity, cytokines and endothelial dysfunction: a link for the Raised cardiovascular risk associated with visceral obesity. J Endocrinol Invest. 2002;25(7):646–9.
18. Taylor EB. The complex role of adipokines in obesity, inflammation, and autoimmunity. Clin Sci (Lond). 2021;135(6):731–52.
19. Gong X, Li J, Jiang Y, Yuan P, Chen L, Yang Y, Li Y, Sun M, Zhao Y, Shi H, et al. Risk of preeclampsia by gestational weight gain in women with varied pre-pregnancy BMI: A retrospective cohort study. Front Endocrinol (Lausanne). 2022;13:967102.
20. Alston MC, Redman LM, Sones JL. An overview of obesity, cholesterol, and systemic inflammation in preeclampsia. Nutrients 2022, 14(10).
21. Robillard PY, Dekker G, Boukerrou M, Boumahni B, Hulsey T, Scioscia M. Gestational weight gain and rate of late-onset preeclampsia: a retrospective analysis on 57 000 Singleton pregnancies in reunion Island. BMJ Open. 2020;10(7):e036549.
22. Catalano P, deMouzon SH. Maternal obesity and metabolic risk to the offspring: why lifestyle interventions May have not achieved the desired outcomes. Int J Obes (Lond). 2015;39(4):642–9.

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