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RESEARCH

Evaluation of dietary inflammatory index and dietary antioxidant capacity with maternal inflammatory and oxidative markers in preeclampsia

Iclal Sena Gezer[1] · Ayse Gulcin Bastemur[2] · Atakan Tanacan[2] · Cigdem Karagoz[2] · Burcu Kesikli[3] · Ozgur Kara[2] · Dilek Sahin[2] · Nuray Yazihan[3]

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Abstract

Purpose This study aimed to investigate the potential association between maternal nutritional intakes, Dietary Inflammatory Index (DII), dietary total antioxidant capacity, maternal serum inflammation, and oxidative markers in preeclampsia.

Methods In this prospective case-control study, a total of 53 pregnant women (23 with preeclampsia and 30 normotensive pregnant women as controls) in their third trimester were included. Dietary intake was assessed via dietary recalls, analyzed through standardized databases. Maternal E-DII (Energy-Adjusted Dietary Inflammatory Index), dietary antioxidant capacity (dORAC), serum interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), interleukin-10 (IL-10), total antioxidant status (TAS), and total oxidant status (TOS) were measured.

Results The main findings of this study were significantly elevated E-DII ($p<0.001$) and decreased ORAC ($p<0.01$) levels. Women with preeclampsia had a diet significantly low in protein, plant-derived proteins, overall fat, energy, carbohydrates, fiber, and omega-3 intake. There was a positive correlation between serum TNF-α and E-DII ($p=0.016$, $r=0.329$) and a significant negative correlation between E-DII and dORAC ($r=-.526$, $p<0.001$). Maternal serum TNF-α levels were significantly elevated ($p<0.01$), and IL-10 levels significantly decreased in preeclampsia ($p<0.001$).

Conclusion The findings emphasize the potential role of antioxidants and the inflammatory potential of nutrition on maternal inflammation and oxidative parameters in relation to preeclampsia risk and support the importance of nutritional follow-up during pregnancy.

Keywords Preeclampsia · Dietary Inflammatory Index · Nutrition · Cytokine · TNF-alpha · Pregnancy

✉ Nuray Yazihan
nurayyazihan@yahoo.com; nyazihan@ankara.edu.tr

Iclal Sena Gezer
iclalsenagezer@gmail.com

Ayse Gulcin Bastemur
aysebastemur@gmail.com

Atakan Tanacan
dratakantanacan@gmail.com

Cigdem Karagoz
cigdemkrgz4@gmail.com

Burcu Kesikli
burcukesikli1@gmail.com

Ozgur Kara
drozgurkara@gmail.com

Dilek Sahin
dilekuygur@gmail.com

[1] Interdisciplinary Food, Metabolism and Clinical Nutrition Department, Institute of Health Sciences, Ankara University, Ankara, Turkey

[2] Department of Obstetrics and Gynecology, Division of Perinatology, Turkish Ministry of Health Ankara City Hospital, Ankara, Turkey

[3] Department of Pathophysiology, Faculty of Medicine, Ankara University, Morfoloji Building, Sihhiye, Ankara, Turkey

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Abbreviations

AUC	Area Under Curve
BMI	Body Mass Index
dORAC	Dietary Oxygen Radical Absorbance Capacity
E	DII–Energy–Adjusted Dietary Inflammatory Index
ELISA	Enzyme–Linked Immunosorbent Assay
IL	6–Interleukin–6
IL	10–Interleukin–10
SPSS	Statistical Package for the Social Sciences
TAC	Total Antioxidant Capacity
TAS	Total Antioxidant Status
TNF	α–Tumor Necrosis Factor alpha
TOS	Total Oxidant Status

Introduction

Preeclampsia is recognized as one of the most critical and severe complications of pregnancy worldwide [1]. Characterized by the onset of hypertension following 20 weeks of pregnancy, preeclampsia involves multiple systems and may present with proteinuria, uteroplacental dysfunction, or various forms of maternal organ impairment [2]. Mechanisms underlying the pathogenesis of preeclampsia are altered immune response, endothelial dysfunction, and systemic inflammatory response. Oxidative stress also plays a critical role in preeclampsia pathogenesis, as it can result in elevated reactive oxygen species production and extensive cellular and tissue damage [3]. Key clinical risk factors for pre-eclampsia include a prior history of the condition and chronic hypertension. Additionally, prior obstetric complications such as fetal growth restriction, stillbirth, and placental abruption increase the risk, reflecting shared pathophysiology related to placental dysfunction [4].

Maternal diet plays a significant role in the inflammatory processes underlying preeclampsia. The Dietary Inflammatory Index (DII) is an objective, validated tool that evaluates the inflammatory effect of a diet. A higher DII has been linked to higher levels of serum inflammatory markers in women with preeclampsia [5]. The antioxidant capacity of the diet plays a crucial role in mitigating oxidative stress, which has been linked to the development and severity of preeclampsia [6]. However, the relationship between Dietary Inflammatory Index, dietary antioxidant capacity, maternal serum inflammation, and oxidative markers in preeclampsia remains unclear.

To our knowledge, no previous study has investigated the association of the dietary antioxidant capacity and inflammatory potential of diet with inflammatory and oxidative parameters together in preeclampsia; therefore, this study aimed to reveal the relationship between the levels of maternal E-DII, dietary antioxidant capacity, circulating total oxidant status (TAS/TOS), interleukin-6 (IL-6), interleukin-10 (IL-10), and tumor necrosis factor-alpha (TNF-α) levels in pregnant women diagnosed with preeclampsia.

Materials and methods

Patient population

The present case-control study included pregnant women aged 18–45 in their third trimester (28 to 40 weeks) diagnosed with preeclampsia ($n = 23$), and normotensive ($n = 30$) admitted to the Department of Perinatology, Turkish Ministry of Health Ankara City Hospital between 25 November 2023-10 June 2024. The study complies with the Declaration of Helsinki and was approved by the institutional clinical research ethics committee (Ethical Number: E2-23–4602). All participants provided written informed consent before enrolment. Preeclampsia patients were diagnosed based on the diagnostic criteria outlined in the American College of Obstetricians and Gynecologists (ACOG) Practice Bulletin No. 222: Gestational Hypertension and Preeclampsia and were subsequently monitored until delivery at the perinatology clinic of Ankara Bilkent City Hospital [2]. The median gestational age at sampling was 33.9 ± 2.5 weeks for the preeclampsia and 34.6 ± 1.5 weeks for the control group. Blood samples were collected at admission to the hospital with the diagnosis of preeclampsia and taken without the administration of any medication. Pregnant women with chronic autoimmune diseases, fetal anomalies, infection, membrane ruptures, diabetic pregnant women, and multiple pregnancies were not included in the study. Additionally, women receiving any medication for comorbid conditions were excluded from the study.

Nutritional assessment, energy adjusted dietary inflammatory index (E-DII), and dietary total antioxidant capacity

To ensure data precision, a specialist dietitian (I.S.G) gathered dietary recall information over a three-day period, which comprised two weekdays and one weekend day. This multi-day recall approach is recognized as a reference method due to its high participation rates, reduced subject burden, and independence from literacy levels among diverse populations [7]. This methodology is widely utilized in numerous large-scale nutrition surveys due to its established validity, high participation rates, and practical feasibility [8–11]. Portion sizes were determined based on household measurements, and a photographic atlas was used to measure portion sizes of foods [12]. Daily energy and

nutrient consumption were analyzed using a food composition database (Ebispro for Windows, Stuttgart, Germany; Version (BeBiS9) program, uses data of Bundeslebensmittelschlussel (BLS) 3.01B and USDA-SR).

To calculate the E-DII score, thirty available food parameters were used: energy, protein, carbohydrate, total fat, cholesterol, saturated fats, polyunsaturated fatty acids, monounsaturated fatty acids, n-3 fats, n-6 fats, caffeine, fiber, β-carotene, vitamins A, C, E, D, B6, B12, riboflavin, thiamine, niacin, folate, and zinc, magnesium, iron, selenium, caffeine, alcohol, and black-green tea consumption. To calculate a z-score, a global database of means and SDs for each nutrient or food parameter was subtracted from individual consumption and divided by its SD. The food parameter's DII score was calculated by multiplying the value of each food parameter by its associated overall score. After adjusting the sum of the specific contributions of each food parameter to energy intakes, E-DII scores were obtained [13].

Dietary total antioxidant capacity was determined by the total oxygen radical absorption capacity (dORAC) from dietary recalls. The ORAC assay, which has been proposed as a measure for measuring antioxidant activity in foods, describes the total potential of dietary components to scavenge free radicals and other synergistic antioxidant effects connected to inflammatory complications. The antioxidant contents of foods were updated in the BeBiS database according to calculation using the values determined for 100 g of nutrients based on the respective method [14].

Measurement of serum cytokines, total antioxidant and oxidant status

Blood samples collected from the patients were centrifuged at 3000 rpm. The supernatants were used for evaluation. According to kit manuals, IL-6, TNF-α, and IL-10 were evaluated as triplicate using commercial ELISA kits (e- BioSciences ELISA Ready SET-Go Affymetrix, Thermo Scientific).

Total oxidant status, and total antioxidant status (TAS) were measured using the spectrophotometric methods with commercial kits (Rel Assay Kit Diagnostics, Turkey). A water soluble vitamin E form, Trolox, was used as a TAS calibrator, and hydrogen peroxide as a TOS calibrator [15]. The results are reported in μmol H2O2equiv./lt.

Data analysis

The G Power analysis program was used to calculate the sample size. Statistical analyses were done with IBM SPSS version 26.0 software (IBM, Armonk, NY, USA). Data distributions were assessed with a single sample Kolmogorov-Smirnov test.

The E-DII, dORAC, IL-6, IL-10, TNF-α, TAS/TOS levels, and nutrient intakes were compared between groups. Descriptive statistics with the mean ± SD were used to analyze the participants' baseline characteristics, with demographic data expressed in terms of median and minimum-maximum values. Pearson correlations were used to evaluate relationships between continuous variables, and multivariable binary logistic regression analyses (Enter method) were performed to evaluate the independent associations between dietary intakes, E-DII, ORAC, and preeclampsia. ROC curve was conducted to evaluate the risk factor, and the Youden index (J) was used to determine the optimal cut-off point of E-DII. A two-tailed P value <0.05 was regarded as statistically significant.

Results

Sociodemographic data

No significant age, gravida, parity, or abortus existed between the patient and control groups ($p>0.05$). Pre-pregnancy ($p<0.001$) and prior-to-delivery BMI ($p<0.01$) were higher in the study group, as expected. Gestational age-of-delivery and birthweight were notably higher in controls ($p<0.001$) (Table 1).

Dietary inflammatory index and nutrient intakes

E-DII was found to increase in patients, with a mean level of 2.67 versus −2.19 in controls ($p<0.001$). To address potential scaling issues and adjust for confounding factors, multivariable logistic regression models were constructed. After simultaneously adjusting for pre-pregnancy BMI, maternal age, and total energy intake, higher Dietary Inflammatory Index (DII) scores remained significantly associated with an increased risk of preeclampsia (aOR = 1.46, 95% CI: 1.146–1.879, $p=0.002$). Conversely, a higher total antioxidant capacity of the diet, as measured by ORAC, demonstrated a significant protective effect; each 1000 μmol TE increase in ORAC was associated with an reduced risk of preeclampsia by approximately 23% (aOR = 0.771, 95% CI: 0.603–0.985, $p=0.037$) (Table 2).

Regarding macronutrient distribution, total protein and plant-derived protein intakes showed strong trends toward protective effects independent of the confounders (aOR = 0.558, $p=0.057$ and aOR = 0.504, $p=0.060$, respectively). Carbohydrate, fat, and omega-3 intakes did not reach statistical significance in the fully adjusted models.

Table 1 Demographical characteristics, dietary intakes and inflammatory markers of preeclampsia patients and healthy pregnant women, values are shown as mean ± SD (*0.01 < p < 0.05; **0.001 < p < 0.01; ***p < 0.001 level of significance of effects)

Variables	Preeclampsia (n = 23)	Control (n = 30)	P value
Maternal age (years) (median, IQR)	29 (20–40)	27.5 (20–40)	0.106
Gravida (median, IQR)	1 (1–5)	1 (1–5)	0.344
Parity (median, IQR)	0 (0–3)	0 (0–3)	0.778
Abortus (median, IQR)	0 (0–2)	0 (0–2)	0.813
SBP	146.5±11.7	110.1±6.9	<0.001***
DBP	88.5±9.3	70.2±6.9	<0.001***
Gestational Age of Delivery (week)	35.6±2.1	38.6±1.2	<0.001***
Pre-Pregnancy BMI	27.8±3.8	23.4±3.8	<0.001***
Prior to Delivery BMI	31.5±3	28.3±4	0.001**
Birthweight (gram)	2367.4±782.5	3299.1±399.1	<0.001***
E-DII	2.67±2.8	−2.19 ± 3.9	<0.001***
ORAC (µmol TE)	6109.7±3210	9058.7±4218.8	0.007**
Energy Intake (kcal/day)	1743±287	1864±188	0.036*
Energy intake (kcal/kg)	20±4.5	25.3±4.4	<0.001***
Protein Intake (g/day)	65.3±7	72±8.7	0.003**
Protein Intake (g/kg)	0.75±0.14	0.97±0.2	<0.001***
Protein Intake (%)	15.6±2.7	15.8±1.3	0.887
Plant protein (g/day)	33.7±10.4	41±8.4	0.007**
Fat Intake (g/day)	71.3±16.3	82.6±17.9	0.022*
Fat Intake (g/kg)	0.83±0.2	1.11±0.3	<0.001***
Fat Intake (%)	36.8±7.6	39.6±7.4	0.285
Saturated Fatty Acids (g)	30.4±6.4	30.7±8.9	0.988
Carbonhydrate Intake (g)	204.6±55.5	202.3±39.7	0.801
Carbonhydrate Intake (g/kg)	2.33±0.7	2.76±0.6	0.022*
Carbonhydrate Intake (%)	47.5±8.6	44.5±7.4	0.289
Cholesterol Intake (mg)	395±120	364±159	0.592
Fiber (g/day)	20.1±7.1	26.9±5.9	<0.001***
Omega-3 (g/day)	1.5±0.5	2.1±1.3	0.027*
TAS/TOS	0.8 ±0.2	1±0.9	0.858
TOS µmol/L	10.9±4.2	9.8±3.4	0.247
TAS mmol/L	0.82±0.3	0.78±0.4	0.052
TNF-α pg/mL	171 ±144.8	76.3±59.4	0.001**
IL-10 pg/mL	44.8±23.1	102.3±86.1	<0.001***
IL-6 pg/mL	76.1±162.4	54.8±44.9	0.248
CRP	14.1±17.9	8.2±6.7	0.599

Figure 1 shows the ROC curve of E-DII. The AUC (Area under curve) value was 0.848 (95% CI: 0.739–0.957, p < 0.001) for E-DII. The optimal cutoff value for E-DII was established at 1.61, yielding a sensitivity of 78% and a specificity of 80%. Due to the preliminary nature of this cohort and the lack of external validation, this threshold is intended purely as a hypothesis-generating metric rather than a clinically actionable diagnostic boundary.

Table 2 Multivariable logistic regression analysis of the associations between dietary factors and preeclampsia risk (Adjusted Odds Ratios and 95% Confidence Intervals, n = 53)

Variables	Adjusted OR (aOR)*	95% CI (Lower - Upper)	Sig. (p-value)
Dietary Indices			
E-DII	1.467	1.146–1.879	**0.002**
ORAC (per 1000 µmol TE)	0.771	0.603–0.985	**0.037**
Macronutrients			
Protein intake (g/kg)	0.558	0.306–1.017	0.057
Plant protein (g/kg)	0.504	0.247–1.030	0.060
Fat intake (g/kg)	0.752	0.515–1.097	0.139
Omega 3 intake (g)	0.610	0.250–1.490	0.278
Carbohydrate intake (g/kg)	1.034	0.899–1.190	0.638

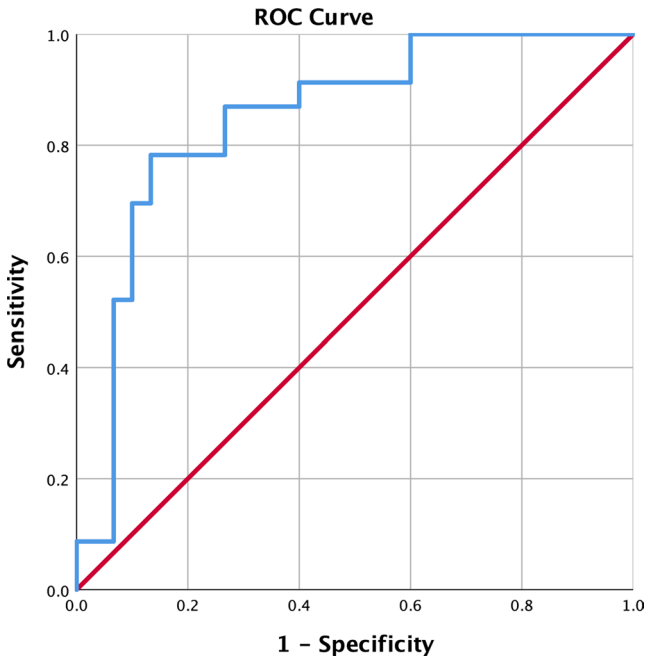


Fig. 1 ROC Curve analysis of E-DII as a risk factor for preeclampsia

Our results showed preeclamptic patients had significantly lower intakes of energy (g/day) (p = 0.04), protein (g/day) (p = 0.003), plant protein (g/day) (p = 0.007), fiber (g/day) (p < 0.001) and omega-3 (p = 0.03) compared to the control group. When calculating per kg intake, fat (p < 0.001) and carbohydrate (p = 0.02) intakes were also found to be lower in preeclamptic patients—conversely, no statistically significant variation in saturated fatty acids and cholesterol intakes (Table 1).

Serum inflammatory and oxidative status

TNF-α levels were elevated ($p<0.001$) and IL-10 levels were significantly decreased in the preeclampsia group ($p<0.001$). The difference was not statistically significant while IL-6 levels were also elevated in the study group ($p=0.248$) (Table 1).

A significant positive association was observed between maternal serum TNF-α levels and E-DII ($r=0.329$, $p=0.016$). Conversely, a negative correlation was identified between E-DII and dORAC ($r=-0.526$, $p<0.001$), while dORAC showed an inverse relationship with TNF-α ($r=-0.289$, $p=0.036$).

Serum total antioxidant status (TAS) and total oxidant status (TOS) levels were higher in the preeclampsia group compared to the control group; however, this difference did not reach statistical significance ($p=0.052$, $p=0.247$, and $p=0.858$, respectively).

Discussion

Our study demonstrated that a lower E-DII is strongly associated with a reduced risk of preeclampsia, and dietary total antioxidant capacity is lower in this condition. Another important finding was that women with preeclampsia had a diet low in protein, plant-derived protein, overall fat, energy and carbohydrates, fiber, and omega-3 intake (Fig. 2).

Healthy pregnancies demonstrate a regulated systemic inflammatory response, with cytokines encouraging spiral arteries' infiltration via the invasion of trophoblast cells. The inflammatory response is impaired in preeclampsia, which leads to aberrant endothelial, neutrophil, and monocyte activation, which causes inflammation in the mother [16]. Nutrition plays a critical function in maintaining inflammation. Recent evidence demonstrated the association between maternal diet, inflammatory markers, and adverse pregnancy outcomes [17].

DII is a review of 1,943 articles that examined the association of 45 food parameters with at least one of six inflammatory factors. A higher value suggests a greater pro-inflammatory impact, whereas a lower value represents a greater anti-inflammatory effect [13].

BMI at the beginning of pregnancy is effective in the development of preeclampsia. In addition, it is observed that preeclamptic pregnant women have higher prior-to-delivery BMI levels. Obesity is linked to maternal hyperinsulinemia, inflammation and oxidative stress is increased, while angiogenic factors and adipokines are altered [18]. Increased dietary inflammatory load and decreased dietary antioxidant capacity of women who have high pre-pregnancy BMI have been indicated in previous studies [19, 20].

In our study, E-DII was significantly higher in the preeclamptic group. The number of studies examining the inflammatory potential of diet in preeclampsia is quite limited. In designing this study, we considered important points from Hong and colleagues' meta-analysis on preeclampsia and DII [21]. Liu et al. (2023) comprehensively demonstrated the effect of Dietary Inflammatory Index on inflammatory biomarkers and preeclampsia. In general, pre-birth BMI was not evaluated, and macronutrient intakes were not adjusted to body weight, which could affect analysis [5]. In our study, we evaluated both parameters.

Our findings align with a study conducted in China in 2023, in which E-DII, calculated from a food frequency questionnaire, was high in preeclamptic pregnant women [5]. In this study, the AUC metric for the E-DII demonstrated a capacity to distinguish between groups, underscoring its prospective value. Nevertheless, the proposed 1.61 cutoff

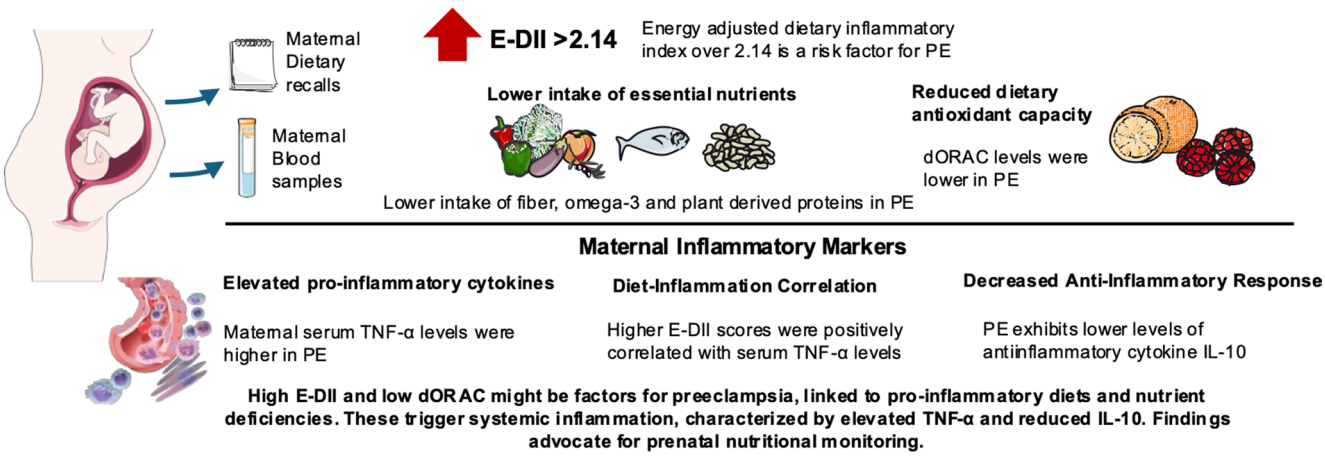


Fig. 2 High E-DII and low dORAC might be risk factors for preeclampsia, linked to pro-inflammatory diets and nutrient deficiencies. These trigger systemic inflammation, characterized by elevated TNF-α and reduced IL-10. Findings advocate for prenatal nutritional monitoring. The figure was modified with text and after adaptation of images from Servier Medical Art by Servier, licensed under a Creative Commons Attribution 4.0 Unported License (https://smart.servier.com)

score remains exploratory and hypothesis-generating. Given our limited sample size and the absence of external validation cohorts, longitudinal, multi-center trials are required to confirm this threshold before any definitive clinical utility or diagnostic recommendations can be established.

Another important finding was that evaluating the nutritional intake, total energy, total protein, plant-based protein, and fiber intake were deficient in the study group, which may relate to the inflammatory and oxidative process. These results match a recent Chinese case-control study on the effects of 'Dietary Approaches to Stop Hypertension (DASH)'-style diet, indicating preeclamptic subjects had lower protein and energy intake [22].

Omega-3 fatty acids are reported to protect from endothelial tissue dysfunction. A Danish cohort research study found that consuming more omega-3 fatty acids was related to reduced risk of preeclampsia [23]. Consistently, this study found that omega-3 fatty acid intake is lower in women with preeclampsia.

In our updated multivariable models, a higher dORAC was associated with decreased risk of preeclampsia. However, despite this lower dietary antioxidant intake, circulating systemic oxidative markers (TAS, TOS, and the formally calculated TAS/TOS ratio) did not show statistically significant differences between the groups. This discrepancy between dietary estimates and systemic biomarkers can be explained by both methodological and biological factors. Methodologically, dORAC reflects cumulative dietary habits, capturing the potential antioxidant capacity entering the body, whereas serum TAS and TOS represent the redox state at a single time point during hospital admission. Biologically, circulating TAS is tightly regulated and comprises not only exogenous antioxidants derived from the diet but also different mechanisms. It is biologically plausible that a reduced dietary antioxidant intake triggers the activation of endogenous compensatory mechanisms to buffer the oxidative burden, maintaining overall redox homeostasis. This adaptive response could lead to a non-significant net change in circulating TAS/TOS despite a pro-oxidant dietary pattern. Therefore, while our data clearly link a deficient dietary antioxidant capacity to an increased risk of preeclampsia, definitive conclusions regarding uncompensated systemic oxidative stress in this cohort should be moderated.

In our study, women with preeclampsia displayed a distinctly altered cytokine profile. Serum TNF-α levels, widely recognized for driving inflammation and endothelial damage in preeclampsia pathogenesis [24], correlated positively with E-DII and inversely with dORAC. While circulating IL-6 was elevated, the variance missed statistical significance likely due to sample size limitations, contrasting with larger trials reporting distinct IL-6 increases [25] alongside studies observing no marked differences [26]. In contrast, we noted a profound decrease in IL-10, an immunomodulatory cytokine that exerts vital anti-inflammatory and vasoprotective actions throughout normal gestation [27]. This systemic shift toward a pro-inflammatory state characterized by high TNF-α and depleted IL-10 tightly mirrors broader meta-analytic evidence [28]. Although TOS and TAS levels were higher in the preeclampsia group compared with controls, these differences did not reach statistical significance. Similarly, CRP levels were almost twice as high in preeclamptic women but also did not reach significance, likely due to sample size limitations. This pattern may indicate the activation of a compensatory antioxidant response aimed at counterbalancing the increased oxidative burden. Such an adaptive mechanism could maintain overall redox homeostasis, leading to a non-significant net change in the TAS/TOS ratio. Similar compensatory regulation of antioxidant capacity in preeclampsia has been described previously [29–31]. Nevertheless, the extent and efficiency of this response likely vary among individuals due to disease heterogeneity, total measurement of oxidative/antioxidative systems, and other pathophysiological factors.

The results of this study highlight the important role that diet's pro-inflammatory potential, as determined by the E-DII, plays in the pathophysiology associated with preeclampsia. Individual nutrient deficiencies may not be as clinically significant as the overall inflammatory burden of the diet, as indicated by the strong correlation between high E-DII scores and an increased risk of preeclampsia. From a clinical perspective, this suggests that evaluating eating habits in pregnancy—more especially, identifying diets deficient in fiber, plant-based proteins, and omega-3 fatty acids—could be a proactive strategy to identify individuals who are at risk. Additionally, a mechanistic basis for dietary patterns is provided by the notable imbalance in serum inflammatory markers (high TNF-α and low IL-10). Therefore, it is important to make nutritional monitoring, aimed at reducing oxidative stress and systemic inflammation in preeclampsia and mitigating diet-induced inflammation, a part of routine prenatal care.

Although this study clearly links pro-inflammatory eating habits, changed serum cytokine profiles, and preeclampsia, there are a number of directions for further investigation. More research is needed because E-DII was found to be a better predictor of risk than dORAC. Future research should investigate the specific synergistic effects of dietary macro and micronutrients to identify which components most effectively modulate systemic inflammation in pregnant populations. To demonstrate a causal relationship between diet-induced inflammation and the onset of preeclampsia, prospective randomized controlled trials are needed, starting before or in early pregnancy.

One of the potential limitations of the present study is the potential confounding effect of socioeconomic status on dietary protein intake and related metabolic markers. Low socioeconomic status is associated with reduced diet quality scores

and limited dietary variety in pregnant populations. Globally, research in low- and middle-income countries indicates that maternal protein intake frequently falls below nationally recommended levels [32]. A previous study conducted in Turkey reported that both preeclamptic and healthy pregnant women had energy, total protein, calcium, and iron intakes below national recommendations [33]. Furthermore, a decrease in dietary diversity was observed as socioeconomic status decreased. Supporting these results, Gokhale and Rao (2022) found an inverse correlation between socioeconomic status and dietary diversity in their study [34]. However, we should note that the control group of pregnant women was comprised of individuals from similar socioeconomic backgrounds who applied to the same hospital under similar conditions.

Some limitations of the present study include the relatively small sample size, lack of longitudinal data, and the self-reported nature of the dietary data. Additionally, although we strategically used separate multivariable models to minimize overfitting, the low events-per-variable ratio—stemming from a cohort of only 23 preeclampsia cases—could be acknowledged as a potential source of model instability in our regression estimates. Furthermore, while dORAC is a widely utilized tool for estimating dietary antioxidant capacity, it is based on in vitro measurements. This methodology does not fully account for human digestion, bioavailability, or the complex in vivo metabolism of these compounds, meaning it serves as a surrogate marker rather than a direct measure of true systemic antioxidant activity. Finally, given the exploratory framework of this research, which aims to identify novel nutritional associations in preeclampsia, we analyzed multiple dietary variables without applying statistical corrections for multiple testing. While conservative adjustments would minimize the risk of false-positive findings (Type I error), they would induce a high Type II error rate in a cohort of this size, potentially obscuring meaningful clinical trends. We acknowledge this calculated statistical trade-off; therefore, our results should be interpreted as hypothesis-generating associations that highlight promising dietary targets for future longitudinal studies. This study has multiple strengths. We calculated dietary intakes using dietary recalls, which has been reported to have relatively high accuracy, and we measured inflammatory and oxidative responses together, which can provide mechanistic insights into the association between dietary patterns [7].

Conclusions

This study provides evidence that a pro-inflammatory maternal diet (reflected by a higher E-DII) and a decreased dietary antioxidant capacity are significantly associated with an elevated systemic inflammatory profile in preeclampsia.

These findings emphasize the potential association between maternal nutrition, inflammation, and oxidative parameters in preeclampsia, supporting the importance of nutritional follow-up during pregnancy. Further research is needed to investigate the underlying mechanisms.

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Author contributions ISG: Conceptualization, Writing – original draft, Methodology, Formal analysis, Data curation. **AGB:** Data curation, Writing - review & editing **. AT:** Formal analysis, Writing - review & editing. **CK:** Data curation, Writing - review & editing. **BK:** Methodology, Writing - review & editing. **OK:** Formal analysis, Writing - review & editing. **DS** : Supervision, Writing - review & editing. **NY:** Conceptualization, Supervision, Writing – original draft.

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Data availability The datasets generated and analyzed during the current study are not publicly available due to ethical and legal regulations in Turkey, but are available from the corresponding author on reasonable request.

Declarations

Ethics approval The study complies with the Declaration of Helsinki. Approval was obtained from the Turkish Ministry of Health Ankara City Hospital ethics committee (Ethical Number: E2-23-4602).

Consent to participate All participants provided written informed consent before enrolment.

Consent to publish There are no circumstances in the study that violate anonymity, and identifying information has been kept confidential. There are no issues regarding its publication.

Conflict of interest The author(s) confirm(s) that there is no conflict of interest.

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