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Trabajo Original

Epidemiología y dietética

Association between the dietary inflammatory index and pelvic inflammatory disease – Findings from the NHANES data (2015-2018)

Asociación entre índice de inflamación dietética y enfermedad inflamatoria pélvica: hallazgos de los datos del NHANES (2015-2018)

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Abstract

Background: pelvic inflammatory disease (PID) is a common gynecological condition. The dietary inflammatory index (DII) scoring algorithm is a novel tool for evaluating the inflammatory potential of a diet. However, the association between DII and PID remains unexplored. This study aimed to evaluate and quantify the relationship between DII and the risk for PID.

Material and methods: the present study included two cycles of the National Health and Nutrition Examination Survey (NHANES) conducted between 2015 and 2018. A total of 2769 participants with complete information were enrolled. Weighted univariate and multivariate logistic regression analyses were performed to examine the association between DII and the risk for PID. Subsequently, the association was graphically represented using a restricted cubic spline (RCS).

Results: univariate and multivariate regression analyses revealed a strong correlation between DII and PID occurrence. After adjusting for all covariates, the odds ratio for the effect of DII on PID remained significant (OR = 1.220, 95 % CI: 1.024-1.452). The correlation analysis revealed a linear relationship between DII and the risk for PID.

Conclusions: this study unravels a significant positive correlation between DII and the risk for PID. This finding highlights the potential of anti-inflammatory diet therapy as a novel therapeutic intervention for PID. However, due to the limitations of the study design, further research is needed to explore this relationship in detail.

Keywords:

Dietary inflammatory index. Pelvic inflammatory disease. National Health and Nutrition Examination Survey. Cross-sectional study.

Resumen

Antecedentes: la inflamación pélvica es una enfermedad ginecológica común. El algoritmo de puntuación del índice de inflamación dietética (DII) es una nueva herramienta para evaluar el potencial inflamatorio de la dieta. Sin embargo, el vínculo entre la DII y la inflamación pélvica aún no se ha estudiado. El objetivo de este estudio fue evaluar y cuantificar la relación entre el riesgo de DII y PID.

Materiales y métodos: este estudio incluyó dos rondas de la Encuesta Nacional de Examen de Salud y Nutrición (NHANES) que se llevaron a cabo entre 2015 y 2018. Un total de 2769 participantes con información completa fueron incluidos en el estudio. Se realizaron análisis ponderados de regresión lógica univariable y multivariable para examinar las asociaciones entre el riesgo de DII y PID. Posteriormente, esta asociación se representa con una *spline* cúbica restringida (RCS).

Resultados: los análisis de regresión monovariable y multivariable mostraron una fuerte correlación entre la ocurrencia de DII y PID. Después de ajustar todas las covariables, la relación de ventaja de los efectos de DII sobre PID se mantuvo significativa (OR = 1,220, IC del 95 %: 1,024-1,452, $p = 0,029$). El análisis de correlación reveló una relación lineal entre el riesgo DII y PID.

Conclusiones: este estudio reveló una correlación positiva significativa entre el riesgo de DII y PID. Este hallazgo destaca el potencial de la dieta antiinflamatoria como una nueva intervención terapéutica PID. Sin embargo, debido a las limitaciones del diseño del estudio, se necesita más investigación para explorar esta relación en detalle.

Palabras clave:

Índice de inflamación alimentaria. Enfermedad pélvica. Encuesta Nacional de Salud y Nutrición. Estudios transversales.

Availability of data and materials: the data used in this study are publicly available (https://www.cdc.gov/nchs/nhanes).

Ethics approval and consent to participate: the National Center for Health Statistics Ethics Review Board approved all research (https://www.cdc.gov/nchs/nhanes/irba98.htm)

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BACKGROUND

Pelvic inflammatory disease (PID) is characterized by bacterial infection and inflammation of the upper genital tract (1). PID commonly leads to gynecological issues, including infertility and ectopic pregnancy. In the United States, approximately 1 million people are diagnosed with PID annually. Moreover, PID accounts for over 350,000 annual visits to the emergency department (2). PID can trigger various complications without proper treatment, thus imposing an enormous burden on individuals and healthcare systems (3). Currently, therapies for PID are based on combating the extent of infection and preventing inflammation relapses (4). While exposure to long-term overuse of antibiotics can lead to adverse health effects in patients and induce multidrug resistance in the pathogens. Therefore, strategies based on effective management of inflammation are still required for fighting against PID.

Diet has an important relationship with inflammatory processes in various diseases. Special diets have been demonstrated to have therapeutic potential in treating such diseases, highlighting the importance of studying the relationship between diet and inflammation (5). Excessive consumption of calcium-rich dairy products could increase inflammation and oxidative stress by increasing inflammatory factors TNF-α, reactive oxygen species, and IL-6 (6). Recently, a systematic assessment of the effect of diet on pain in rheumatoid arthritis suggests that anti-inflammatory diets could be effective in subsiding pain and symptoms, possibly via regulation of the inflammatory response (7). Supplementing the diet with foods enriched in vitamin D and trace elements (selenium, magnesium, zinc) is considered to be an effective adjunctive therapy in chronic pain management (8). As a novel disease management strategy, diet therapy is an effective way to counteract PID (9). Avoiding the ingestion of risky diets such as alcohol and coffee might help in alleviating the symptoms of PID (10).

DII is a recently developed assessment tool that allows quantification of the total inflammatory potential of a diet based on the properties of its components such as carbohydrates, proteins, vitamins, and trace elements (11). Based on its effect on inflammatory biomarkers, a diet could be characterized into categories ranging from maximally anti-inflammatory to pro-inflammatory (12). A previous study showed that DII was explicitly correlated with several indicators of inflammation such as increased leucocyte count, C-reactive protein level, interleukin (IL)-6 level, and tumor necrosis factor-α level (13). Several studies have suggested that DII is closely associated with tumor onset in cancers such as ovarian cancer and breast cancer, and higher DII correlates to a greater risk of cancer development (14,15). Moreover, DII might increase cardiovascular disease by enhancing systemic inflammation (16). Therapeutic options based on modulation of the inflammatory response such as decreasing the production of NO and IL-6 have been reported to be effective in treating PID (17). Strategies to effectively control inflammation remain ideal for devising therapeutic interventions for PID. However, few studies have attempted to evaluate the relationship between DII and PID. Hence, studies are necessary to examine the relationship between DII and PID, further quantifying the effect of DII on PID risk.

NHANES was started in the early 1960s to assess the health and nutritional status of the entire population of the United States (18). Taking advantage of the large sample size and comprehensive sampling design, the NHANES database has been widely recommended as a data source in many highly influential studies (19). Therefore, using weighted sampling of the NHANES, we aimed to conduct a systemic evaluation to elucidate the role of DII on the prevalence of PID, thereby providing new ideas for managing inflammation in patients with PID.

MATERIALS AND METHODS

STUDY POPULATION

For each of the surveys in NHANES, participants were interviewed in their homes to collect demographic and health data. This was followed by recording dietary intake recalls and physical measurements at a mobile health examination center (MEC). Data from 2015 to 2018 were used to ensure the objectivity and reliability of the study. According to the PID diagnostic questionnaire guidelines in NHANES, individuals equal to or over 18 years of age were selected as the candidate population. Data on exposure, outcome, and related covariates were collected from all participants, and individuals without complete information were not included in the study.

DIETARY INFLAMMATORY INDEX

Dietary intake information was accessed through face-to-face interviews in the MEC. Each participant was instructed to recall the types and quantities of foods and beverages consumed within 24 hours before the interview. DII reflects the global inflammatory potential of a diet based on the assessment of forty-five pro- and anti-inflammatory food parameters (20). For the intrinsic limitation of NHANES data, only 27 components were incorporated in the DII calculations including carbohydrates; protein; total fat; alcohol; fiber; cholesterol; saturated fat; monounsaturated and polyunsaturated fatty acids; *n*-3 and *n*-6 polyunsaturated fatty acids; niacin; vitamins A, B1, B2, B6, B12, C, D, E; iron; magnesium; zinc; selenium; folic acid; beta carotene; caffeine (21). By subtracting the estimated daily intake, z-scores were computed and transformed into centered proportions for each food parameter, and then summed up to acquire the overall DII scores for individual. Finally, to control for total energy intake, energy-adjusted DII scores were calculated based on the density of food components (intake per 1000 kcal) (22). Specifically, DII contained in the diet was obtained using an inbuilt function in the 'nhanesR' package and used in subsequent analyses. Based on the cut-offs defined by the tertile points, the DII scores were categorized into three groups (DII_Q): Q1, Q2, and Q3, representing anti-inflammatory, marginally pro-inflammatory, and highly pro-inflammatory diets, respectively (23).

PELVIC INFLAMMATORY DISEASE

Participants were screened using the 'rhq078' questionnaire to assess their PID status. Based on their responses to the question 'Ever treated for a pelvic infection/PID?', participants were divided into non-PID and PID groups.

ASSESSMENT OF COVARIATES

Age at menarche and regular menstruation were selected as potential covariates based on their previously reported correlation with the PID outcomes (24). Data for age at menarche were accessed from the responses to the 'Age when first menstrual period occurred?' in the 'rhq010' questionnaire (25). Data regarding the history of regular menstruation were accessed from the responses to the 'Had regular periods in past twelve months?' in the 'rhq031' questionnaire (26).

Data on age, education, marital status, poverty, and race were retrieved from responses to the demographic interview. Data for poverty level were divided into low, medium, and high according to the poverty income ratio (PIR) cut-offs: PIR < 1.35, $1.35 \leq$ PIR ≤ 1.85, and PIR > 1.85 (27). Body mass index (BMI) was measured at the mobile examination center. Based on BMI, participants were stratified into four categories, underweight (BMI < 18.5 kg/m^2), normal weight ($18.5 \leq$ BMI ≤ 24.9 kg/m^2), obese ($25 \leq$ BMI ≤ 29.9), and overweight (BMI ≥ 30) (28).

STATISTICAL ANALYSIS

Considering the complex and multistage sampling design of the NHANES, the data used in this study were weighted according to the sampling guidelines of the NHANES. Given the least common denominator approach, sample weights were calculated by the first-day dietary weight for each two-year cycle in NHANES (29). Descriptive statistics were performed to summarize the non-PID and PID data. For continuous variables, data were summarized using the mean and standard error of the mean (mean ± SE) for each group, and the differences were evaluated using a t-test. For categorical variables, data were expressed as percentages (%), and the differences in proportions were evaluated using the chi-square test.

To probe the relationship between DII and the prevalence of PID, a weighted univariate regression analysis was performed. Subsequently, multivariate regression analysis was conducted to assess the stability of the model. Multiple linear regression models were constructed using the 'svyglm' package in R. Four regression models were established guided by the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology): the crude model was unadjusted; model I was adjusted for age and ethnicity; model 2 was adjusted for covariates that were statistically significant according to univariate regression analysis; model 3 was adjusted for all covariates. The relationship between DII and risk for PID was graphically represented using the 'rms' R package. Interactions test was performed on all variables within DII and other covariates in the multivariable model (model 3). Subgroup analyses were performed to evaluate the stability of these results. All analyses were performed using R (version 4.2.1) and RStudio (version 1.2.5042). Significance was determined using a two-tailed *p*-value threshold of 0.05.

RESULTS

CHARACTERISTICS OF INCLUDED PARTICIPANTS

Based on a detailed selection process, only those with complete information were enrolled in the study (Fig. 1). A total of 2769 participants were analyzed, representing 74,599,932 participants after survey weighting. Of these, 168 had PID and 2601 did not have PID. Participants in the PID group showed higher DII compared to those of the non-PID group, and the mean age of the PID group was older than that of the non-PID group. The covariates 'regular period', 'BMI', 'marital status', and 'PIR' showed significant statistical differences between the non-PID and PID groups (Table I).

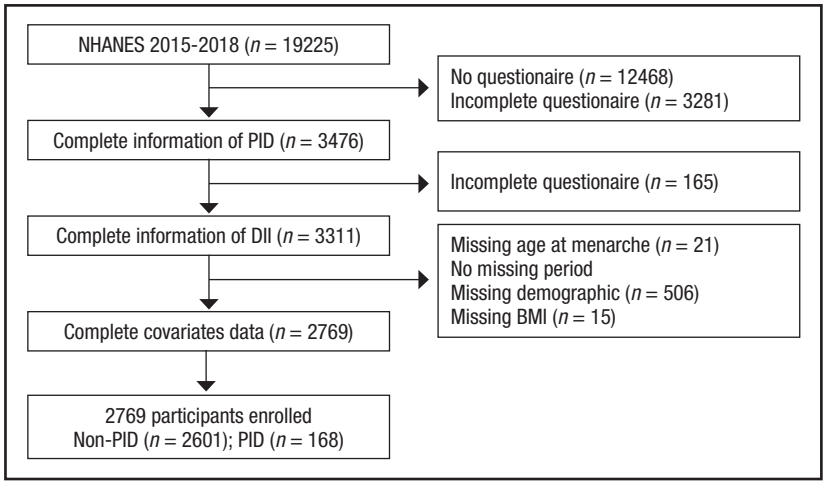

Figure 1.

Flow chart outlining the screening process (data from 2015-2018 NHANES).

Table I. Characteristics of participants (*n* = 2769)

Prevalence of PID	Total	Non-PID	PID	*p*-value
	(*n* = 2769)	**(*n* = 2601)**	**(*n* = 168)**	
DII n (%)	1.655 ± 0.094	1.605 ± 0.094	2.451 ± 0.202	< 0.001
Q1	923 (33.333)	883 (37.396)	40 (20.912)	0.002
Q2	923 (33.333)	869 (33.655)	54 (33.284)	
Q3	923 (33.333)	849 (28.949)	74 (45.804)	
Age, years	39.880 ± 0.391	39.683 ± 0.417	43.008 ± 1.133	0.011
Menarche age, years	12.624 (0.055)	12.642 (0.056)	12.338 (0.172)	0.098
Menstruation, n (%)				0.007
Regular	1900 (68.617)	1809 (65.847)	91 (51.998)	
Irregular	869 (31.383)	792 (34.153)	77 (48.002)	
BMI, n (%)				0.005
Underweight	55 (1.986)	54 (2.464)	1 (0.166)	
Normal weight	760 (27.447)	726 (30.562)	34 (12.585)	
Obese	1269 (45.829)	1178 (42.188)	91 (51.317)	
Overweight	685 (24.738)	643 (24.786)	42 (35.931)	
Education, n (%)				0.164
Less high school	415 (14.987)	386 (9.334)	29 (14.899)	
High school	577 (20.838)	536 (21.554)	41 (28.550)	
College	1777 (64.175)	1679 (69.112)	98 (56.551)	
Marital status, n (%)				0.023
Married	1624 (58.649)	1534 (63.001)	90 (55.794)	
Widowed	57 (2.059)	51 (1.987)	6 (5.247)	
Divorced/separated	426 (15.385)	384 (13.288)	42 (25.313)	
Single	662 (23.908)	632 (21.723)	30 (13.646)	
PIR, n (%)				< 0.001
Low	909 (32.828)	835 (24.132)	74 (42.302)	
Medium	880 (31.78)	821 (26.789)	59 (34.789)	
High	980 (35.392)	945 (49.079)	35 (22.909)	
Race, n (%)				0.371
Non-Hispanic white	900 (32.503)	843 (60.088)	57 (55.221)	
Mexican American	468 (16.901)	449 (10.489)	19 (6.813)	
Non-Hispanic black	635 (22.932)	582 (11.828)	53 (16.314)	
Other	766 (27.663)	727 (17.595)	39 (21.652)	

Significant variables are shown in bold. Continuous variables were presented as mean ± SE and evaluated by t-test. Categorical variables were presented as n *(%) and evaluated by the chi-square test. OR: odds ratio; CI: confidence interval; PID: pelvic inflammatory disease; BMI: body mass index.*

UNIVARIATE WEIGHTED LOGISTIC REGRESSION ANALYSIS

To determine whether DII could be used as a predictor of the occurrence of PID, we performed weighted univariate tests (Table II). For every unit increase in DII, the relative risk of PID increased by 31 % (OR = 1.310, 95 % CI: 1.124-1.527). In comparison with the reference group, univariate analyses of other covariates indicated that 'age' (OR = 1.025, 95 % CI: 1.005-1.045), 'irregular period' (OR = 1.780, 95 % CI: 1.176-2.693), 'divorced or separated' (OR = 2.151, 95 % CI: 1.122-4.123), and BMI were positively associated with the occurrence of PID. In contrast, 'high PIR' (OR = 0.266, 95 % CI: 0.118-0.602) was significantly negatively correlated with the occurrence of PID.

MULTIVARIATE WEIGHTED LOGISTIC REGRESSION ANALYSIS

After adjusting for age and race (model 1), the analysis revealed that a unit increase in the DII corresponds to a 1.307-fold

Table II. Univariate analysis for the prevalence of PID

Covariate	OR (95 % CI)	*p*-value
DII	1.310 (1.124,1.527)	0.001
Age, years	1.025 (1.005,1.045)	0.015
Menarche age, years	0.900 (0.790,1.024)	0.105
Menstruation, n (%)		0.008
Regular	Ref	
Irregular	1.780 (1.176,2.693)	
BMI, n (%)		
Underweight	Ref	
Normal weight	6.106 (0.791, 47.131)	0.08
Obese	18.036 (2.160,150.577)	0.009
Overweight	21.494 (2.722,169.741)	0.005
Education, n (%)		
Less high school	Ref	
High school	0.830 (0.316,2.181)	0.696
College	0.513 (0.223,1.180)	0.112
Marital status, n (%)		
Married	Ref	
Widowed	2.981 (0.614,14.462)	0.167
Divorced or separated	2.151 (1.122, 4.123)	0.023
Single	0.709 (0.417, 1.206)	0.195
PIR, n (%)		
Low	Ref	
Medium	0.741 (0.436,1.260)	0.257
High	0.266 (0.118,0.602)	0.003
Race, n (%)		
Non-Hispanic white	Ref	
Mexican American	0.707 (0.343,1.456)	0.333
Non-Hispanic black	1.501 (0.926,2.433)	0.096
Other	1.339 (0.564,3.178)	0.494

Significant variables are shown in bold. OR: odds ratio; CI: confidence interval; PID: pelvic inflammatory disease; BMI: body mass index.

increase in the risk for PID (OR = 1.307, 95 % CI: 1.116-1.532) (Table III). A similar positive correlation was observed in model 2 (OR = 1.224, 95 % CI: 1.041-1.439) and model 3 (OR = 1.220, 95 % CI: 1.024-1.452). Compared to the anti-inflammatory diets (Q1), the positive correlation between highly pro-inflammatory diets (Q3) and PID persisted in model 1 (OR = 2.797, 95 % CI: 1.573-4.976), model 2 (OR = 2.157, 95 % CI: 1.133-4.106) and model 3 (OR = 2.142, 95 % CI: 1.049-4.375). Moreover, the risk of PID showed a significant trend toward an increased grade of DII in all models ($p < 0.05$).

To present the relationships more intuitively, an RCS was plotted (Fig. 2). The association between DII and the risk for PID could be fitted by a straight line with a positive slope (non-linear *p*-value = 0.560 > 0.05), indicating that increasing DII was correlated with an increased risk of PID.

SUBGROUP ANALYSIS

To test for the robustness of the relationship, we performed a subgroup analysis (Fig. 3). DII values showed the highest odds ratio (OR) in the 'widowed marital' subgroup (OR = 3.004, 95 % CI: 0.602-14.994) and the lowest OR in the 'underweight BMI' subgroup (OR = 0.711, 95 % CI: 0.489-1.034). The test for interaction highlighted a significant interaction of DII with age (p for interaction = 0.011) and BMI (p = 0.009). To eliminate the heterogeneity, the age was then categorized into four subgroups: 20-29, 30-39, 40-49, and 50-59. The results showed that compared to those in other groups, individuals in 40-50 age (OR = 2.016, 95 % CI: 1.284-3.163) may be more sensitive to the DII, thus leading to a greater prevalence of PID. Compared to the 'underweight' group (OR = 0.711, 95 % CI: 0.489-1.034), the 'normal weight' (OR = 1.572, 95 % CI: 1.211-2.040), obese (OR = 1.096, 95 % CI: 0.934-1.287) and 'overweight' (OR = 1.562, 95 % CI: 1.149-2.122) groups had higher ORs. Overall, nearly all OR values in the subgroups were great than one, implying a stable positive association between DII and the prevalence of PID.

Table III. Association between DII and the risk for PID

	Crude	Model 1	Model 2	Model 3
	OR, 95 % CI *p*-value	OR, 95 % CI *p*-value	OR, 95 % CI *p*-value	OR, 95 % CI *p*-value
DII	1.31 (1.124, 1.527) 0.001	1.307 (1.116, 1.532) 0.002	1.224 (1.041, 1.439) 0.017	1.220 (1.024, 1.452) 0.029
Q1	Ref	Ref	Ref	Ref
Q2	1.768 (1.180, 2.651) 0.007	1.831 (1.211, 2.768) 0.006	1.648 (1.069, 2.542) 0.026	1.639 (1.039, 2.587) 0.036
Q3	2.829 (1.621, 4.940) < 0.001	2.797 (1.573, 4.976) 0.001	2.157 (1.133, 4.106) 0.022	2.142 (1.049, 4.375) 0.038
p for trend	0.001	0.002	0.029	0.048

Significant variables are shown in bold. OR: odds ratio; CI: confidence interval; PID: pelvic inflammatory disease; BMI: body mass index. Crude model was unadjusted, model I adjusted for age and ethnicity, model 2 adjusted for covariates found to be of statistical significance in univariate regression analysis, model 3 adjusted for all covariates (age, age at menarche, regular menstruation, BMI, education, marital status, PIR, race).

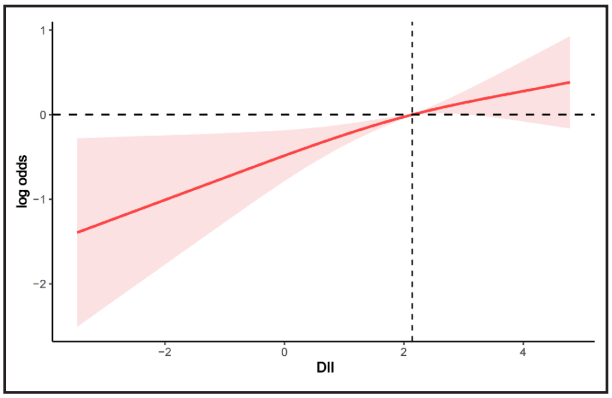

Figure 2.

Association between DII and the prevalence of PID (adjusted for age, age at menarche, regular menstruation, BMI, education, marital status, PIR, and race).

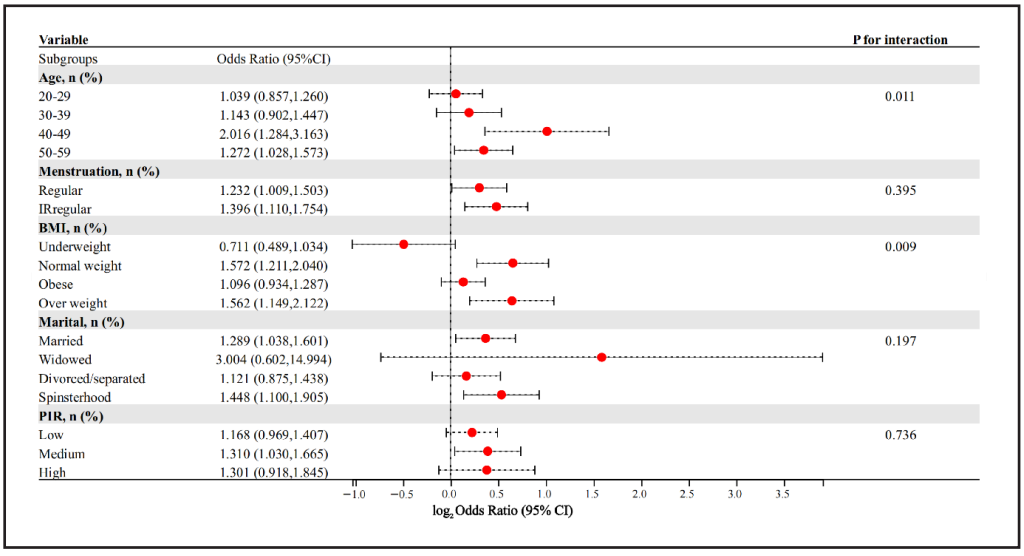

Figure 3.

Results of subgroup analyses of the associations between DII and the risk for PID.

DISCUSSION

In the present study, data from NHANES were processed using weighted approaches to increase the power to detect associations. The correlation between DII and the prevalence of PID was carefully assessed and verified through weighted univariate and multivariate linear regression analyses. The RCS further corroborated the positive linear association. This study confirms the relationship between DII and the risk for PID for the first time.

The overall prevalence of PID in our study was 6.07 %, which was similar to that reported in the prior research (30). While this could be an underestimation since the definitive diagnosis of PID mostly relies on the measurement of clinical symptoms (31). The diet pattern, predominately consisting of fruits and vegetables, can have anti-inflammatory effects by downregulating the immunomodulatory bioactivities in the periphery and in the central nervous system (32). Conversely, intake of high pro-inflammatory components such as processed meats and sugary beverages could induce levels of proinflammatory cytokines, thus increasing the adhesion of leukocytes to the endothelium (33). Regardless of the levels of covariate adjustment, DII consistently emerged as a risk factor for PID. Moreover, this relationship could be verified using the tertile trend of DII, and participants in the 'most pro-inflammatory diet' group had a higher risk for PID than those in the 'anti-inflammatory diet' group. Given the pathogenesis of PID and the regulatory role of DII, it is reasonable to conjecture that diet may affect the occurrence of PID through its effect on the inflammation process. With the help of RCS, this research was able to visualize the positive linear relationship between DII and PID, thus might elicit a preventive and therapeutic effect of DII against PID.

The present study also revealed that irregular menstruation might as a risk factor for PID, which was similar to a previous retrospective study (34). The uterine cavity is protected from bacteria by the cervix and its mucus barrier (35). While during menstrual disorders, the integrity barrier of the cervix was destroyed, thereby potentially assisting the vertical transmission of vaginal bacteria. Under the influence of a pro-inflammatory diet, the participants with irregular menstruation were prone to have a slightly increased prevalence of DII than those in the regular period. Poverty has been recognized as a risk factor for numerous diseases, and high PIR was demonstrated to be a protective factor for the

prevalence of PID. A retrospective clinical study indicated that less poverty condition may contribute to the alleviation of PID in white women (36). . Nevertheless, it's worth noting that the positive association between DII and PID persisted regardless of poverty status, highlighting the important pathogenic role of DII.

When fitting an interaction test for covariates, age, and BMI were the significant variations. The epidemiological analysis shows that the prevalence of PID increases with age (37). Similarly, in our study, age was positively related to the prevalence of PID, and participants in 40-49 years were more affected by DII than those in another age group, implying that more attention should be given and an anti-inflammatory diet was recommended to those participants. Being overweight and obese were identified to be a risk factor for PID, and higher BMI was associated with higher levels of inflammatory cytokines (38). After stratifying the data by BMI, positive correlations between DII and PID were observed in all groups except the underweight group. Notably, the positive correlation between DII and PID was seen in almost all subgroups, further emphasizing the consistency of this relationship.

Collectively, our study is the first to examine the correlation between DII and the risk for PID. With the advantage of the sampling design of NHANES, weighted approaches were used to achieve more definite results. Additionally, after adjustment for various covariates, the risk effects of DII on the prevalence of PID were evaluated and determined by multiple linear regression, thus providing solid evidence for this relationship. Yet, it is essential to note that the findings in this report are subject to several limitations. First, due to the questionnaire format of the 'rhq' and covariates, the analyses presented here might have a recall bias. Second, the sampling method could have introduced a selection bias. Third, the diagnostic criteria of PID were based on the questionnaire, which may have potential deviations. Finally, since this was a cross-sectional study, no causal relationship could be established between the DII and PID.

CONCLUSIONS

In conclusion, DII was tightly correlated with the prevalence of PID. After adjustment for all confounders, a positive linear relationship was presented between DII and the risk for PID. This study indicates that dietary interventions may have a therapeutic role in combating PID.

AUTHORS' CONTRIBUTIONS

Juan Juan Ma and Pan-Wei Hu have contributed equally in this article. MJ and HP collected the data, HP organized the data and wrote the manuscript, and ZQ and PJ designed the research plan. All authors agreed to the final version before submission. All authors read and approved the final manuscript. All authors agreed to the final version prior to submission. All authors read and approved the final manuscript.

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