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Dietary inflammation influences the prevalence of cardiovascular diseases in prediabetes and diabetes patients: findings from the National Health and Nutrition Examination Survey (NHANES 2001–2018)

Zuheng Liu[1†], Xiujing Wang[1†], Haiyue Liu[2†], Zhijuan Zhang[3], Wei Chin Poh[4], Fang Luo[1], Changqing Sun[1], Rong Tang[1], Qiang Xie[1*] and Wuyang Zheng[1*]

Abstract

Prediabetes is an early phase before diabetes. Diabetes and dietary inflammation are two crucial factors that are strongly associated with cardiovascular diseases (CVDs). Dietary interventions slowed the progression of diabetes and CVD. However, the associations between CVDs and dietary inflammation in different stages of pathoglycaemia have not been investigated. To explore the effect of a proinflammatory diet on CVD incidence at different stages of diabetes, NHANES (2001–2018) data were collected and analysed. A total of 3137 CVD patients with a comparable non-CVD group ($n=3137$) were enrolled after propensity score matching (PSM) analysis. These patients were subsequently categorized into three subgroups: those with diabetes ($n=3043$), those with prediabetes ($n=1099$) and those with normoglycemia ($n=2132$). The DII (Dietary inflammatory index) is a risk factor for CVD, both in overall individuals and in each subgroup of population-based information. In diabetic individuals, the odds ratios (ORs) (95% CIs) of CVD incidence for the DII were 1.10 (1.05, 1.15) and 1.08 (1.03, 1.13) according to the crude and adjusted models, respectively. For individuals with prediabetes, the ORs (95% CIs) of CVD risk for DII were 1.05 (0.97, 1.14) and 1.11 (1.01, 1.22) according to the crude and adjusted models, respectively. After adjusting for population-based information and hypertension status, the DII appeared to have the highest OR for individuals with prediabetes, and no significant association was found between the DII score and CVD risk in the normoglycemia group. Moreover, the OR of CVD for DII in the uncontrolled diabetes group was 1.06 (0.98, 1.16)*. These results

†Zuheng Liu, Xiujing Wang and Haiyue Liu contributed equally to this work.

*Correspondence:
Qiang Xie
arthur2014@sina.com
Wuyang Zheng
zhengwuyang1@163.com

Full list of author information is available at the end of the article

suggest that the DII is more closely associated with the risk of CVDs in prediabetic and diabetic populations, and we should pay more attention to diet control before a person develops diabetes to prevent CVD progression.

Keywords Dietary inflammation, Cardiovascular diseases, Prediabetes, Diabetes, NHANES

Introduction

Cardiovascular disease (CVD), a significant public health concern, increases the mortality and disability of the general population worldwide. Although various hazard factors have been identified in recent years [1], dysglycemia is still a severe risk factor for CVDs. Diabetes has been confirmed to be a significant independent risk factor for CVDs in various studies [2]. However, vascular endothelial cell injury caused by high blood glucose is systemic, as complications are limited not only to cardiovascular diseases (CVDs) but also to the eyes, kidney and peripheral nerves. These complications have increasingly threatened people's lives and health in recent years.

Prediabetes is a disease characterized by impaired fasting glucose or impaired glucose tolerance before diabetes, and it is also predicted to increase the risk and mortality of various CVDs [3]. The link between prediabetes and CVDs has been described in recent years [3]. High blood glucose increases oxidative stress and apoptosis, followed by injury to vascular endothelial and myocardial cells [4, 5]. Prediabetes and diabetes can be managed and postponed by lifestyle control [6], in which diet is one of the essential factors influencing blood glucose. An unhealthy diet can increase inflammation, followed by a series of metabolic diseases [7]. Therefore, when we are concerned about the effects of dietary inflammation on CVDs, it is still indispensable to investigate the diabetic population separately.

The dietary inflammatory index (DII) is a method used to evaluate the inflammatory levels associated with a person's daily intake [8]. A person's dietary consumption influences his or her risk of acquiring prediabetes or diabetes [9]. In addition, various studies have suggested a relationship between diet and CVDs [10, 11]. It is well known that prediabetes or diabetes is a traditional influencing factor of CVD. However, the effects of diet on the risk of CVDs caused by prediabetes or diabetes are unclear. In this study, we investigated the associations between CVDs and dietary inflammatory indices in participants with diabetes or prediabetes by analysing data from the National Health and Nutrition Examination Survey (NHANES).

Materials and methods

Study population and data

The data were collected from the NHANES, a national cross-sectional survey conducted in the United States to determine the health status of local citizens. The NHANES contains variables referring to physical examination and various clinical indices. All participants provided written informed consent, and the survey was approved by the National Center for Health Statistics' Research Ethics Review Board in the U.S., while this study was based on a secondary data analysis, which does not require institutional review. The data from 2001 to 2018 were selected because participants in these years had detailed information on completed 24-h (24 h) diet recalls and the diagnosis of relevant diseases. Participants were divided into two groups, CVD and non-CVD groups, and propensity score matching (PSM) analysis was performed to filter a comparable non-CVD group. PSM is a statistical technique used to create matched samples from two or more groups that differ in observable characteristics but are balanced in terms of the probability of receiving a particular treatment or exposure. We applied PSM to reduce biases and confounding, thereby strengthening the observed relationship between dietary inflammatory index and CVDs in our population. CVDs include heart failure, coronary artery disease, angina, and heart attack. The verification of heart failure, coronary artery disease, angina, or heart attack was based on the MCQ160B, MCQ160C, MCQ160D, and MCQ160E questionnaires, respectively. Accordingly, participants were divided into three subgroups: diabetes, prediabetes and normoglycemia groups. Diabetes was verified by the DIQ010 questionnaire by asking "Doctor told you have diabetes", while prediabetes was obtained from the DIQ160 questionnaire by asking "Ever told you have prediabetes".

Among the diabetic individuals, we defined the "Uncontrolled diabetes" subgroup as those with an HbA1c≥6.5% or a fasting blood glucose≥7.0 mmol/L, and the others were defined as the "Controlled diabetes" subgroup. Among the prediabetic individuals, we defined the "Uncontrolled prediabetes" subgroup as those with an HbA1c>6.0% or fasting blood glucose>6.1 mmol/L, and the others were defined as the "Controlled prediabetes" subgroup. HbA1c and fasting blood glucose can be found as DIQ280 and LBDGLUSI, respectively, in the NHANES database. Detailed information about the NHANES can be found on the following website: http://www.cdc.gov/nchs/nhanes.htm.

Determination of the dietary inflammation index

The dietary inflammation index (DII) was calculated according to reliable 24 h diet recalls introduced by Shivappa, which include three steps [8]. The first step is to obtain a Z score: DII calculations are based on dietary

intake data, which are then linked to a regionally representative world database. This provides a robust estimation of the mean and standard deviation for each parameter. The Z score is utilized to express an individual's exposure in relation to the standard global average. The score is obtained by subtracting the global daily mean intake, dividing by its standard deviation, converting to a percentile score, doubling each percentile score and subtracting 1 for symmetry. The second step is to obtain a "food parameter-specific DII score": The percentile value for each food parameter was first standardized and then multiplied by the corresponding "overall food parameter-specific inflammatory effect score" to derive the "food parameter-specific DII score." The third step is to calculate an "overall DII score", which is calculated by summing each "food parameter-specific DII score". The DII was calculated for a total of twenty-seven nutrients, including alcohol, vitamin B12/B6, b-carotene, caffeine, carbohydrates, cholesterol, energy, total fat, fibre, folic acid, Fe, Mg, monounsaturated fatty acids (MUFAs), niacin, n-3 fatty acids, protein, polyunsaturated fatty acids (PUFAs), riboflavin, saturated fat, Se, thiamine, vitamin A/C/D/E, and Zn.

Statistical analyses

The data were processed by R version 4.3.2 (http://www.R-project.org), and the R packages "MatchIt", "stats" and "missRanger" were used for PSM, logistical analysis and missing data handling, respectively. Student's t test was performed for comparisons of continuous variables. A chi-squared test was used to compare the component ratio of classified variables. Logistic regression was performed to assess the association between CVDs and DII after adjusting for covariates. For PSM, we chose CVD (YES/NO) as the outcome variable and adjusted for age, gender, and race (all complete). We used MatchIt in R (method = "nearest") for PSM analysis. In logistic regression, we fit the model with glm() in R, setting [family=binomial(link = "logit")]. ORs were calculated by [exp(coef(X))], 95%CIs by [exp(cbind(OR=coef(X), confint(X)))]. We used predict() for predictions and residuals() for error assessment to check for overfitting. We imputed missing BMI values (<10%) using missRanger() from the "missRanger" package in R. In all analyses, a p value<0.05 was considered to indicate statistical significance.

Results

A total of 6274 participants with clinical indices and demographic information were included in the present study. Figure 1 shows the process of data collection and the number of participants in each group. Briefly, 12,115 participants were excluded due to a lack of dietetic questionnaires, while participants without diabetes or prediabetes disease information (n=34647) were also excluded. Patients who lacked cardiovascular disease information (n=9227) were also excluded. Finally, 35,362 participants remained; these participants were further divided into CVDs (n=3137) and non-CVDs (n=32225). Age, sex and race were subsequently matched for propensity score matching (PSM) analysis. After PSM analysis, 3137 individuals in the non-CVD group were included. These patients were also categorized as having diabetes (n=3043), prediabetes (n=1099) or hypoglycemia (n=2132). Patients with heart attack, heart failure, coronary artery disease or angina were defined as having CVD. The prevalence of CVDs in the diabetes, prediabetes and normoglycemia groups was 57.15%, 76.80% and 25.98%, respectively.

Baseline characteristics of participants

Table 1 lists the characteristics of 6274 individuals, including 3137 CVD patients and 3137 non-CVD patients, who were matched for age, sex and race, but we can see that CVD patients had a lower educational background (p<0.001), a greater prevalence of hypertension (p<0.001), a greater BMI (30.74±7.33 vs. 29.40±7.57, p<0.001) and a greater DII (0.039±1.74 vs. -0.265±1.68, p<0.001). It is also reasonable that CVD patients had lower incomes; 36.3% of CVD patients had annual family incomes less than 2000 dollars, while 27.5% of non-CVD patients did (p<0.001).

Associations between DII and CVDs

As shown in Fig. 2, the DII was significantly associated with CVDs in the overall population (OR (95% CI)=1.11 (1.08–1.14), p<0.001). Then, we performed subgroup analysis according to sex, age, race, ethnicity, educational level, family income, BMI, diabetes status, hypertension status and blood glucose control conditions in patients with diabetes and prediabetes. The DII was significantly associated with the DII in each group according to sex, age, race, ethnicity, educational level, family income, BMI and hypertension status. Among individuals with diabetes, the DII seems not to be related to CVD risk in individuals with prediabetes. In the blood glucose control subgroup, the DII was significantly associated with the incidence of CVD in individuals with uncontrolled diabetes.

Associations between CVD incidence and DII in the diabetes, prediabetes and normoglycemia groups

To investigate the relationship between CVD incidence and DII at different stages of abnormal glucose metabolism, three logistic regression analyses were performed (Table 2). In diabetic individuals, the crude model showed a statistically significant association between CVDs and DII (OR (95% CI)=1.10 (1.05, 1.15), p<0.001).

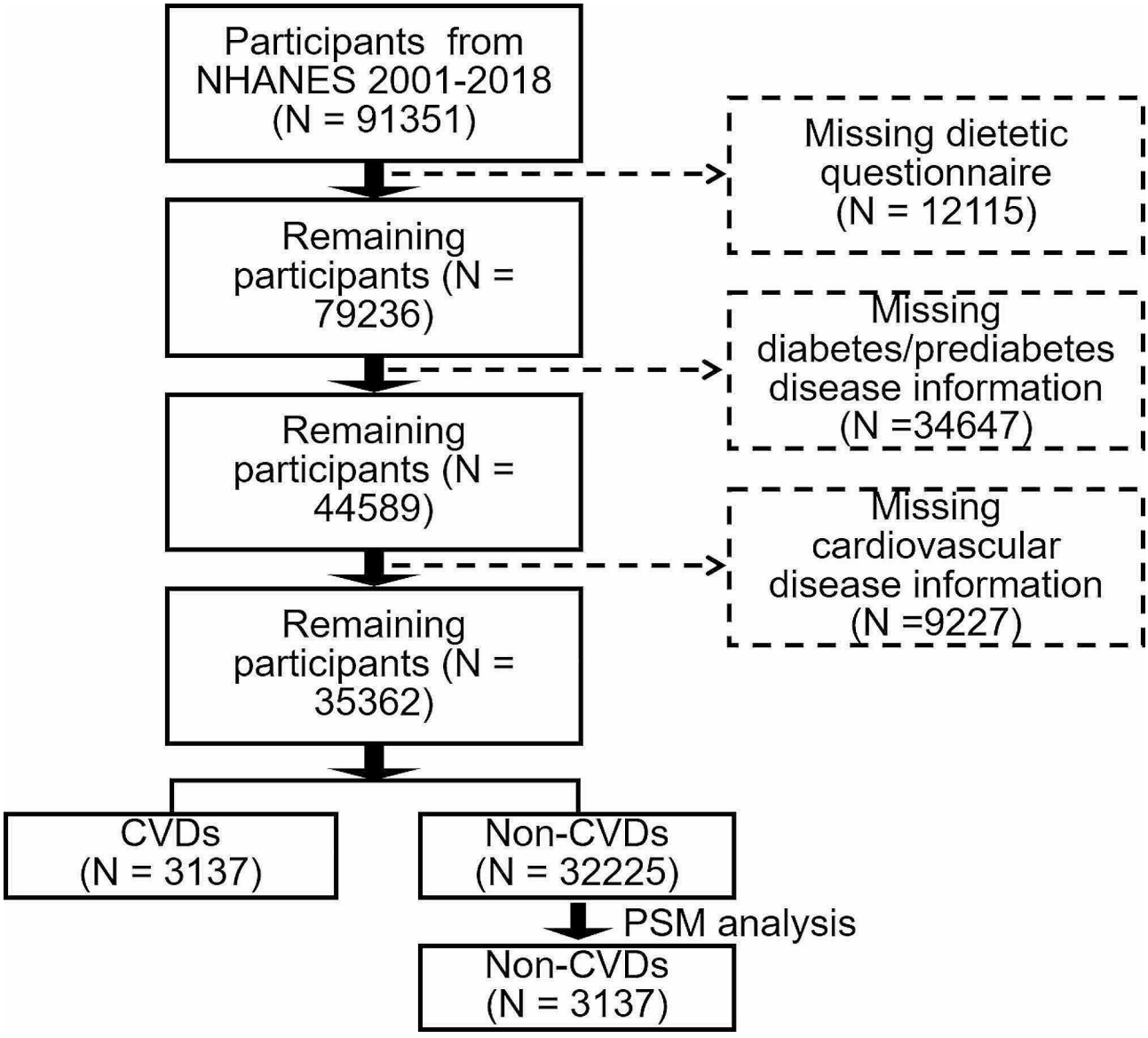

Fig. 1 Flow chart of sample selection criteria from the NHANES 2001–2018

However, the association decreased after adjustment for sex, age, race, ethnicity, education level, family annual income, hypertension status and BMI (OR (95% CI) = 1.08 (1.03, 1.13), $p < 0.01$). In the prediabetes group, the crude model showed no statistically significant association between CVD incidence and DII score (OR (95% CI) = 1.05 (0.97, 1.14)). Moreover, the association became significant after adjustment for sex, age, race, education level, family annual income, hypertension status and BMI (OR (95% CI) = 1.11 (1.01, 1.22), $p < 0.05$). In the normoglycemia group, there was a positive association in the crude model, while the association disappeared in the adjusted model.

Associations between CVDs and DII in control patients and patients with uncontrolled diabetes or prediabetes

From the results of the above analysis, we discovered that the association between DII and CVD risk in normoglycemia patients disappeared in the adjusted model, while the association between DII risk and CVD risk remained significant in the prediabetes and diabetes groups. These results might suggest that dietary control should be started as soon as possible, as the beneficial effect of dietary control can be achieved in the early stage. Thus, to verify the relationship between DII scores and CVD incidence after diabetes or prediabetes were controlled for, we performed a further logistic regression analysis. The "Uncontrolled diabetes" and "Uncontrolled prediabetes" subgroups are defined in the Methods

Table 1 Baseline characteristics of participants in the NHANES 2001–2018

Characteristic	CVDs		*p* value
	No (*N* = 3137)	Yes (*N* = 3137)	
Age, years, Mean ± SD	66.72 ± 12.60	66.73 ± 12.61	0.973[c]
Age, N (%)			1[c]
< 65	1190 (37.9%)	1190 (37.9%)	
≥ 65	1947 (62.1%)	1947 (62.1%)	
Gender			0.898[c]
Male, N (%)	1865 (59.5%)	1860 (59.3%)	
Female, N (%)	1272 (40.5%)	1277 (40.7%)	
BMI, kg/m^2, Mean ± SD	29.40 ± 7.57	30.74 ± 7.33	< 0.001[b]
BMI, N (%)			< 0.001[a]
<25	943 (30.8%)	615 (20.2%)	
≥ 25	2122 (69.2%)	2427 (79.8%)	
Race, N (%)			
Mexican American	330 (10.5%)	328 (10.5%)	1[c]
Other Hispanic	222 (7.1%)	223 (7.1%)	
Non-Hispanic White	1763 (56.2%)	1761 (56.1%)	
Non-Hispanic black	638 (20.3%)	641 (20.4%)	
Other Race or Multi-racial	184 (5.9%)	184 (5.9%)	
Education, N (%)			
< High school	910 (29.0%)	1061 (33.9%)	< 0.001[a]
High school or equivalent	752(24.0%)	763 (24.4%)	
> High school	1472 (47.0%)	1309 (41.8%)	
Missing	3	4	
Annual family income, N (%)			
< 20,000 USD	826 (27.5%)	1083 (36.3%)	< 0.001[a]
≥ 20,000 USD	2182 (72.5%)	1904 (63.7%)	
Missing	129	150	
Current smoking status, N (%)			
Smoking	430 (26.4%)	629 (32.8%)	< 0.001[a]
Non-smoking	1201 (73.6%)	1289 (67.2%)	
Missing	1506	1219	
DII, Mean ± SD	-0.265 ± 1.68	0.039 ± 1.74	< 0.001[b]
Hypertension, N (%)	1917 (61.3%)	2336 (74.6%)	< 0.001[a]

The data are presented as the N% (χ^2 test) and mean ± SD (Student's t test), which are denoted by [a] and [b], respectively. Significant differences are denoted as $^*P<0.05$, $^{**}P<0.01$, and $^{***}P<0.001$. [c]Age, sex and race were matched for propensity score matching (PSM) analysis

section. Surprisingly, logistic regression analysis revealed no significant difference between DII and CVD risk in the adjusted model for "controlled diabetes" (OR (95% CI) = 1.11 (0.93, 1.33)), and there was a significant association between DII risk and CVD risk in the "uncontrolled diabetes" group (OR (95% CI) = 1.06 (0.98, 1.16)*). Additionally, there was no significant association between DII scores and CVD incidence in the "controlled/uncontrolled prediabetes" group. These findings further suggested that the role of dietary control can be fulfilled in the early stage of glucose metabolism dysfunction.

Discussion

Using the data from the NHANES 2001–2018, we explored the associations between dietary inflammation and CVDs in normoglycaemia, prediabetes and diabetes patients. These results suggest that the DII is closely positively associated with the risk of CVDs in prediabetic and diabetic populations but not in normoglycemic populations, which means that dietary control should receive more attention in the early stage of abnormal glycometabolism.

The proinflammatory diet was evaluated by the DII, an important index that is calculated by dozens of dietary elements and a special formula. Previous studies have illustrated the link between DII and various diseases, including heart failure, diabetes, metabolic syndrome, depression and even hormone levels. Indeed, various diseases are related to eating habits. Prediabetes and diabetes are diseases characterized by abnormal glucose metabolism. Generally, the management and optimization of lifestyle habits, including dietary habits, sleep, drinking and cigarette use, are expected to minimize glucose damage to the vascular system [12–14]. Diet is

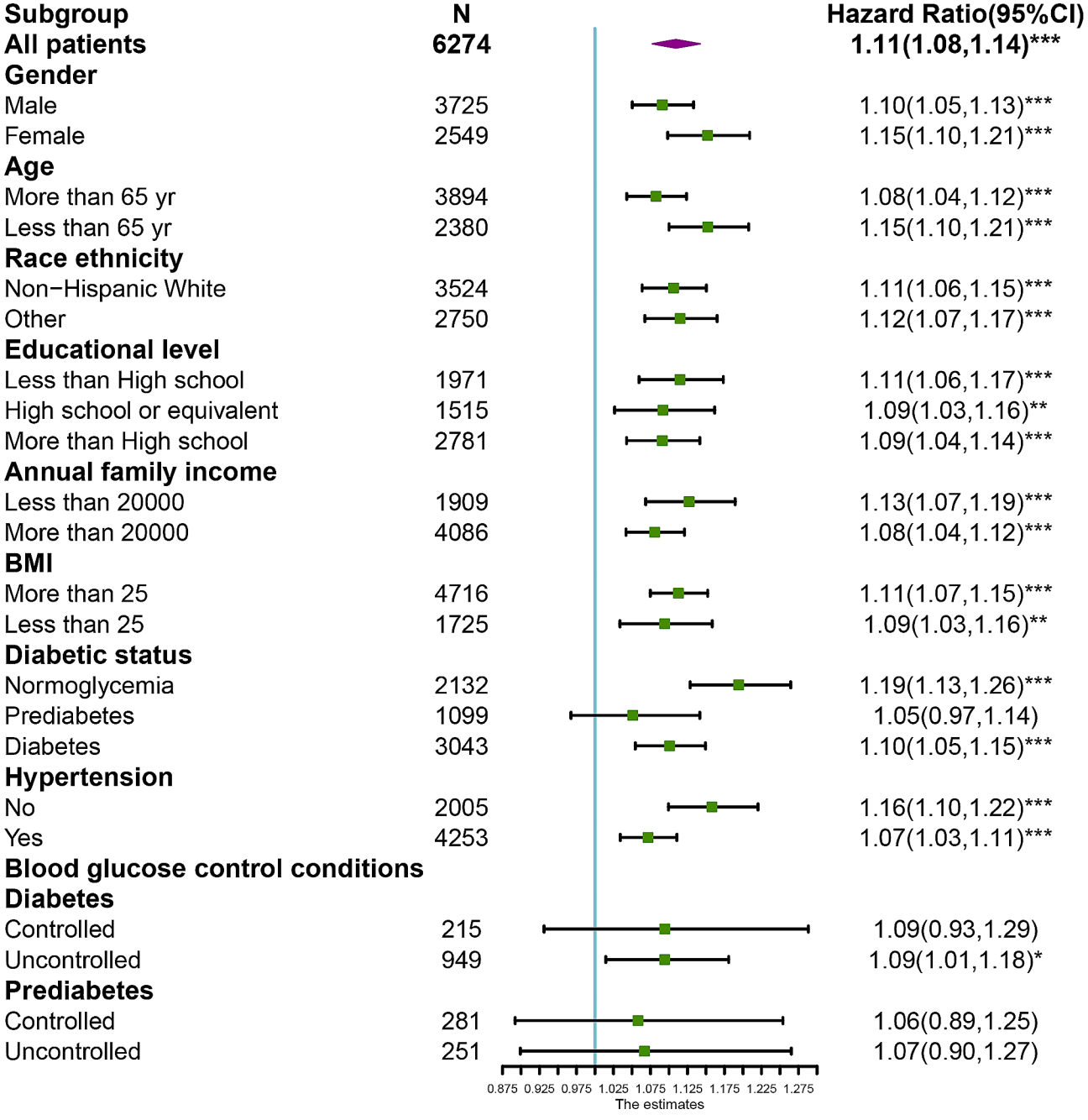

Fig. 2 The association between DII and CVD incidence in different subgroups

an important factor influencing the risk of prediabetes, diabetes, and CVD. Notoriously, people who consumed more proinflammatory foods had higher DIIs. Various studies have suggested that a Western diet including red meat, sweets and fries increases the risk of CVDs [10, 11], while a Western diet is also associated with a higher DII score [15]. A population with a high DII has a greater incidence of CVDs [16]. In contrast, the health intake of vegetables and fresh fruit, such as in the Mediterranean diet, is negatively associated with DII scores [17] and can reduce the risk of CVDs [18].

A previous study indicated that people with higher DIIs have greater risks of death, cancer and CVD [19]. Our research similarly demonstrated that DII scores are positively associated with the risk of CVDs in different groups. In addition, the diabetes group had the highest DII among the three groups. This finding is also consistent with previous findings, where diabetes and prediabetes patients had a greater incidence of CVDs in

Table 2 Association between DII and CVD incidence in different subgroups of different models

Population	Unadjusted	Model I	Model II
Normoglycemia	1.19(1.13,1.26)***	1.12(1.05,1.20)***	1.05(0.97,1.14)
Prediabetes	1.05(0.97,1.14)	1.12(1.02,1.22)*	1.11(1.01,1.22)*
Diabetes	1.10(1.05,1.15)***	1.10(1.05,1.15)***	1.08(1.03,1.13)**
Blood glucose subgroups			
Diabetes			
Controlled	1.09(0.93,1.29)	1.12(0.95,1.33)	1.11(0.93,1.33)
Uncontrolled	1.09(1.01,1.18)*	1.09(1.01,1.18)*	1.06(0.98,1.16)*
Prediabetes			
Controlled	1.06(0.89,1.25)	1.12(0.93,1.33)	1.12(0.92,1.36)
Uncontrolled	1.07(0.90,1.27)	1.16(0.97,1.40)	1.18(0.96,1.46)

The DII was considered a categorical variable. Multiple regression analysis was conducted to assess the associations between DII scores and the incidence of CVD in the diabetes, prediabetes, and normoglycemia subgroups according to different statistical models. In addition, controlled and uncontrolled diabetes and prediabetes subgroup analyses were also conducted

Model I was adjusted for sex, age, and race ethnicity; Model II was adjusted for sex, age, race ethnicity, educational level, family annual income, hypertension and BMI.

*$P<0.05$, **$P<0.01$, ***$P<0.001$. DII, dietary inflammatory index

the present study [20]. In the prediabetes group, the DII score was significantly associated with CVD incidence according to both the crude and adjusted models. In addition, this finding was similar in the normoglycemia subgroup. Although CVD incidence was associated with DII score in the crude model of the diabetes group in our study, this association was not significant in the adjusted model. These inconsistent results among the three subgroups suggested that diet control should be taken into account in the early stage. It is plausible that the influence of diabetes on CVDs exceeds that of dietary inflammation after people are in the diabetes stage. However, the evidence of the association between a proinflammatory diet and prediabetes is still sufficient. Recent research from the same database suggested a positive association between DII score and diabetes-related indices such as fasting plasma glucose (FPG), fasting serum insulin (FSI), and homeostatic model assessment of insulin resistance (HOMA-IR) [21]. In another study, the authors investigated the effect of a proinflammatory diet on the complications of diabetes. They found that the energy-adjusted DII is positively associated with diabetic retinopathy [22].

To support our findings above, we further analysed the relationship between DII and CVDs in controlled and uncontrolled diabetes or prediabetes patients in the present study. We found that the DII is more strongly associated with CVDs in patients with uncontrolled diabetes than in those with controlled diabetes, controlled prediabetes, or uncontrolled prediabetes. These findings suggest that we should pay more attention to the dietary structure of patients with uncontrolled diabetes. However, this does not mean that we can ignore the effect of diet on the general public. Indeed, the DII only contains limited food elements.

However, there are still some limitations of the present study. First, this study is a cross-sectional observational study that lacks follow-up information, necessitating further prospective research. Second, due to the incomplete data of public database, insufficient sample size is also a limitation of the study. Third, excluding participants without complete dietary questionnaire and disease information can potentially introduce selection bias, which is a common drawback when conducting data reanalysis using public databases. Besides, the existence of potential bias in PSM is inevitable. Fourth, although the DII provides a method to evaluate dietary inflammatory potential, the results still require comprehensive analysis and external validation in conjunction with other clinical and epidemiological data. In summary, a greater intake of a pro-inflammatory diet is significantly linked with the risk of CVDs, especially in diabetic and pre-diabetic patients. Thus, dietary control should be performed before the development of diabetes.

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Author contributions
ZHL, WYZ, QX, and HYL conceived the ideas and design of the study. HYL, XJW, ZHL and CQS collected the data from the NEHANES. HYL, XJW and ZHL analysed the data. ZHL and XJW wrote the main manuscript text, and HYL, ZJZ and WCP prepared the figures. WCP, RT, QX and WYZ revised the final version of the manuscript. QX and WYZ supervised the study. All authors reviewed the manuscript and approved the final version of the manuscript for publication.

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Data availability
In this study, we analyzed variables from the NHANES database related to various health conditions and demographics. To verify heart failure, coronary artery disease, angina, or heart attack, we relied on the questionnaires MCQ160B, MCQ160C, MCQ160D, and MCQ160E, which are stored in the

MCQ (Medical Conditions) file. These can be downloaded for specific years, such as 2011–2012, from the following URL: https://wwwn.cdc.gov/Nchs/Nhanes/2011-2012/MCQ_G.XPT.
For diabetes verification, we utilized DIQ010 and DIQ160, while HbA1c was found as DIQ280, all stored in the DIQ (Diabetes) file. Again, these can be downloaded by year from: https://wwwn.cdc.gov/Nchs/Nhanes/2011-2012/DIQ_G.XPT.
Demographic information, including age (RIDAGEYR), gender (RIAGENDR), race (RIDRETH1), education level (DMDEDUC2), and family income (INDFMINC), is stored in the DEMO (Med Demographic Variables & Sample Weights) file. These variables can be downloaded for specific years, for instance, 2011–2012, from: https://wwwn.cdc.gov/Nchs/Nhanes/2011-2012/DEMO_G.XPT.
BMI data (BMXBMI) is stored in the BMX (Body Measures) file and can be downloaded from: https://wwwn.cdc.gov/Nchs/Nhanes/2011-2012/BMX_G.XPT for the year 2011–2012.
Hypertension information (BPQ020) is stored in the BPQ (Blood Pressure & Cholesterol) file and can be accessed via: https://wwwn.cdc.gov/Nchs/Nhanes/2011-2012/BPQ_G.XPT for the specified year.
Smoking status (SMQ040) is recorded in the SMQ (Smoking - Cigarette Use) file, downloadable from: https://wwwn.cdc.gov/Nchs/Nhanes/2011-2012/SMQ_G.XPT for the relevant year.
Lastly, fasting blood glucose levels (LBDGLUSI) are available in the GLU (Plasma Fasting Glucose & Insulin) file, downloadable at: https://wwwn.cdc.gov/Nchs/Nhanes/2011-2012/GLU_G.XPT for 2011–2012.
To locate the download page for any variable across different years, users can search for the variable number in the NHANES variable query section at: https://wwwn.cdc.gov/nchs/nhanes/search/default.aspx.

Declarations

Ethics approval and consent to participate
NHANES, as a long-running research project led by the National Center for Health Statistics (NCHS), has strictly adhered to ethical principles and legal frameworks since its inception. All individuals participating in NHANES surveys voluntarily agreed to participate after being fully informed of the study's purpose, methods, and potential risks, and had the right to withdraw their consent at any time.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Cardiology, Xiamen Key Laboratory of Cardiac Electrophysiology, Xiamen Institute of Cardiovascular Diseases, The First Affiliated Hospital of Xiamen University, School of Medicine, Xiamen University, NO. 55 Zhenhai Road., Xiamen 361003, China
[2]Xiamen Key Laboratory of Genetic Testing, Department of Laboratory Medicine, The First Affiliated Hospital of Xiamen University, School of Medicine, Xiamen University, NO. 55 Zhenhai Road., Xiamen 361003, China
[3]Department of family Medicine and Primary Care, The University of Hong Kong, Hong Kong, China
[4]School of Traditional Chinese Medicine, Xiamen University Malaysia, Sepang, Malaysia

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