The Association between Dietary Inflammation Index and Rheumatoid Arthritis in Americans: a cross-sectional study

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Abstract

Background

Studies have shown that personal diet has a certain impact on the development and treatment of rheumatoid arthritis (RA). However, the effect of overall dietary structure on RA is still an exploratory issue at present. Dietary inflammation index (DII) is an index that evaluates the inflammatory potential of the total nutrients consumed by human body. Therefore, we studied the correlation between DII and RA in Americans to explore whether the pro-inflammatory or anti-inflammatory effects of diet contribute to the risk of RA.

Methods

We used data from the 2005–2017 NHANES database for analysis, including 1819 individuals with RA and 8602 individuals without RA. Among them, DII was calculated by collecting the relevant data of Total Nutrient Intakes, First Day in the database. The analysis was carried out through the comparison between the two groups, including logical regression, multiple regression analysis, smooth curve fitting, recursive algorithm, and independent sample T-test.

Results

After adjusting for other confounding factors, we found that there was a positive correlation between DII and RA in Americans ($\beta = 1.068$, 95% CI = 1.026 to 1.111, $P = 0.00121$). In the subgroup analysis, it was found that characteristics of the participants were younger than 50 years old, female, Other Hispanic, $BMI \geq 25$ or federal poverty rate $> 185\%$, there was still a positive association between DII and RA. Compared with non-smokers, the effect between DII and RA of smokers was greater than that of non-smokers. In Other Hispanic, there was an inflection point ($K = 1.195$). When $K > 1.195$, the curve gradient increases rapidly. In addition, the intake of energy, protein, dietary fiber, total saturated fatty acids, vitamin E, vitamin B6, magnesium, zinc, Niacin, Selenium, iron, and alcohol in individuals with RA was significantly lower than that in those without RA.

Conclusions

The inflammatory potential of diet may increase the risk of onset and development of RA. The risk may be different in different individuals, but it may be a feasible way to prevent and alleviate RA by improving dietary structure, reducing pro-inflammatory food, and increasing anti-inflammatory food intake.

Introduction

Rheumatoid arthritis (RA) is an immune-mediated disease characterized by symmetrical polyarthritis. Its main pathological changes are systemic inflammation, synovial cell hyperplasia, and pannus formation. Repeated arthritis leads to the destruction of joint structure, swelling, deformity, and loss of function, finally to progressive disability, other systemic complications, and early death ^[1–2].

RA is the most prevalent inflammatory rheumatic disease. The prevalence rate is 0.5%- 1.1% in North America and northern Europe ^[3–4]. Related genes determine the susceptibility to disease, and environmental factors can induce the occurrence of RA ^[5], such as smoking, low alcohol intake, obesity, low physical activity, etc ^[6–7].

Recent studies have found that personal dietary factors have a significant impact on the risk of developing RA, which may be the “triggers” that leads to the genetic susceptibility. For example, marine oil supplements and the Mediterranean diet had an effective role in relieving RA pain ^[2,8]. The EVOO-polyphenol extract has been shown to reduce the expression of inflammatory cytokines, to alleviate joint edema and cartilage destruction in patients with RA ^[9]. Dietary fiber also affects the development of RA. One of its mechanisms is to promote the production of short-chain fatty acids, improving the intestinal microbiota. A good intestinal microenvironment regulates the T cell phenotype and T cell-mediated immunity ^[10]. As an important trace element, Se is the basic component for the formation of antioxidant enzymes. Its content affects the severity of immune-related diseases ^[11]. The high-calorie intake of red meat, saturated fatty acid, and trans fatty acid increase the risk of RA ^[12], which may lead to obesity and the accumulation of white adipose tissue (WAT), stimulating the release of pro-inflammatory mediators ^[13]. Human cells have a postprandial inflammatory response to food, to regulate the inflammatory state of the human body ^[12]. Therefore, it is possible to prevent and alleviate the onset of RA by regulating the diet model. However, more attention is still focused on the efficacy of a single nutrient at present.

Considering the diversity and complexity of our dietary mix, a single nutrient study does not fully represent the inflammatory effects of an individual diet. Therefore, we are looking for a tool to better describe the effects of food and nutrients on inflammation. Dietary inflammatory index (DII) was developed to evaluate the overall inflammatory potential of individuals' diets ^[14]. According to the effects of specific foods or ingredients on inflammation, it scores the inflammatory effects of 45 dietary parameters with IL-1 β , IL-4, IL-6, IL10, TNF- α , and CRP as markers of inflammation. By calculating the intake of complex pro-inflammatory and anti-inflammatory foods obtain a more standardized assessment of the effects of diet on inflammation ^[15–16]. The higher the DII score, the stronger the pro-inflammatory effect, and vice versa, it suggests that the anti-inflammatory effect is stronger.

Previous studies have shown that DII score is associated with the risk and mortality of a variety of non-communicable diseases. For example, it was reported that DII was positively associated with the risk of colorectal cancer, cancers of the Upper Aerodigestive Tract, gastric cancer, myocardial infarction, obesity, osteoporosis, sarcopenia, and other diseases ^[16–21]. At present, although some studies have shown that there was an association between DII and RA ^[22–23], the ethnicity studied was different, and the results of

the study are different. Here, we report on the American population enrolled onto a cross-sectional study to further explore the relationship between DII and RA.

Methods

Data source and participants

Our analysis was based on data from the National Health and Nutrition Examination Survey (NHANES), which was established to assess the health and nutritional status of adults and children in the United States. Since 1999, investigators have conducted population sample surveys on a two-year cycle. The NHANES study has been approved by the National Center for Health Statistics Institutional Review Board and Ethics Review Board. Therefore, the data used in this study need not be reviewed and approved by the Medicine Institutional Review Board.

The data from 2005 to 2018, which represented seven cycles of the NHANES, were summarized. We extracted the relevant data of Demographic Variables and Sample Weights, Medical Conditions, Diabetes, Smoking-Cigarette Use, Reproductive Health, Physical Activity, Alcohol Use, and Dietary Interview-Total Nutrient Intakes (First Day). Individuals with RA and without RA from 2005 to 2017 were included according to the question "Has the doctor ever said you had arthritis?" and "Which type of arthritis was it?" Individuals under the age of 30, pregnant women, individuals with other arthritis, and those with incomplete data for dietary interviews were excluded ($n = 18022$). To keep the sample size as large as possible, the missing binary variables were separated into a group, and the continuity variables were filled with the average (the data follows normal distribution) or the median (the data did not align with the normal distribution) when we processed the data. Finally, the inclusion of individuals with RA were 1819, and individuals without RA were 8602.

Dietary inflammatory index measurement

DII was created from the Cancer Prevention and Control Program at the University of South Carolina in the United States. It is used to assess the possibility of pro-inflammatory and anti-inflammatory effects in individual food intake. There were 27 kinds of food included in the range of DII parameters, containing energy, protein, carbohydrate, dietary fiber, total fat, total saturated fatty acids, total monounsaturated fatty acids, total polyunsaturated fatty acids, cholesterol, Vitamin E as alpha-tocopherol, Added alpha-tocopherol (Vitamin E), Vitamin A, RAE, beta-carotene, Vitamin B1, Vitamin B2, Niacin, Vitamin B6, Folic acid, Vitamin B12, Added vitamin B12, Vitamin C, magnesium, iron, zinc, Selenium, caffeine, and alcohol.

The specific calculation method of DII refers to the method proposed by Shivappa et al ^[15]. First of all, the average number of dietary ingredients or nutrients consumed by individuals in a day was subtracted from the global average daily intake of the corresponding dietary components or nutrients. The resulting value was divided by the standard deviation of the global per capita daily intake of the diet or nutrient and multiplied by the relevant inflammatory effect index. The result of the above operation was called the Z score, which indicated the individual's exposure to the "standard global average". The Z scores were

converted into percentiles to minimize the effect of right skewing. The results were multiplied by the total inflammation score of each dietary component or nutrient. Finally, the individual DII score was obtained by adding the calculation results of each dietary ingredient or nutrient. The higher the DII score, the stronger the pro-inflammatory effect, and vice versa, it suggests that the anti-inflammatory effect is stronger.

Covariates

The covariates included age, gender, race, whether suffering from anemia, body mass index (BMI), alcohol consumption, marital status, diabetes, physical activity, smoking at least 100 cigarettes in life, and the poverty income ratio. According to BMI, we divided the participants into three groups: underweight, normal range, and overweight (< 18.5, 18.5–24.9, and > 25). The alcohol consumption in the past 12 months was divided into four levels (0, < 48, 48–384, and ≥ 384) and was divided as none, light, moderate and heavy [24]. Physical activity was converted into MET score, and the conversion standard referred to the score recommended by NHANES. Smokers were defined as answering yes to the question “Smoking at least 100 cigarettes in life”.

Statistical Analysis

The statistical method of our study used logistic regression to analyze the measured value of the effect between exposure factors and disease, that was risk ratio (β -coefficients) and 95% confidence interval. To control the effect of other factors on RA and observe the independent effect of DII on RA, we used multiple regression analysis for statistical analysis: model 1 did not adjust any covariables; model 2 adjusted gender, age, and race; model 3 adjusted all covariates. Then, the additive model was used for subgroup analysis to study the effects of DII on RA among different subgroups. At the same time, we used smooth curve fitting to analyze the nonlinear relationship between DII and RA. If the relationship was nonlinear, the recursive algorithm was used to detect the possible threshold effect and saturation effect. All statistical analyses were performed by using the Empower soft.

In addition, we compared the intake of common nutritional parameters between individuals with RA and those without RA through the independent sample T-test. The results were described by mean values and standard deviation (mean $\pm$ SD). P values less than 0.001 were considered statistically significant.

Result

Characterization of the included population

We converted the dietary inflammatory index from a continuous variable to a categorical variable (Quartile). The basic information of each group of participants and other covariates were shown in Table 1. The range of each quartile group was – 4.621 to -0.828, -4.621 to -0.828, 0.418 to 0.419, 1.705 to 4.535. From Quartile 1 to Quartile 4, the proportion of individuals without RA were declining, while the individuals with RA were increasing.

In addition, the average age of the participants was 54.323 ± 14.956 . Except for age and anemia, there were differences in the characteristics of all the groups ($P < 0.001$). Women reported significantly higher rates of RA than men. The proportion of people who are overweight ($BMI \geq 25$) was heavy, accounting for 72.181% of the total participants. The value of MET gradually decreases with the increased score of DII, while the proportion of smokers and non-drinkers continues to increase.

The association between RA and DII scores

The association between DII and RA was shown in Table 2. Model 1 did not adjust any covariates, and there was a positive association between DII and RA ($\beta = 1.142$, 95% CI = 1.108 to 1.177, $P < 0.001$). After adjustment for confounds, there were still a positive association between Model 2 ($\beta = 1.158$, 95% CI = 1.121 to 1.196, $P < 0.001$) and Model 3 ($\beta = 1.068$, 95% CI 1.026 to 1.111, $P < 0.00121$). After adjustment for all confounds, for every unit increase in DII, the risk of RA increased by 6.8%. Using smooth curve fitting found that there was a nonlinear relationship between DII and RA after adjusting all the confounding factors (Fig. 1).

In the subgroup analysis, stratified analysis was conducted according to age, race, sex, BMI, smoking, and poverty income ratio. The results showed that there was a positive association between DII and RA in different age groups. When the age was less than or equal to 50 years old, for each additional unit of DII, the risk of RA increased by 12.6% (95%CI = 1.041 to 1.218, 95%CI = 1.041 to 0.0031); when the age was older than 50 years old, the risk of RA increased by 5.7% (95% CI = 1.010 to 1.107, 0.0167). There was no association between DII and RA in Mexican American, Non-Hispanic White, Non-Hispanic Black, and Other Races. However, in the subgroup of Other Hispanic, there was a strong association between DII and RA ($\beta = 1.265$, 95%, 1.112 to 1.438, $P = 0.0003$). Subsequently, we found that when $BMI \geq 25$, DII had an impact on the risk of RA, and for each additional unit of DII, the disease risk of RA increased by 7.5%. Likewise, when the gender is female, there was an association between DII and RA ($\beta = 1.094$, 95% CI = 1.038 to 1.153, 0.0008), while in the male population, there was no association between the two. Our study also found that smokers increased the effect of DII on the risk of RA ($\beta = 1.074$ to 95% CI = 1.039 to 1.148, Purge 0.0005), which was significantly higher than that of non-smokers ($\beta = 1.092$, 95%CI = 1.016 to 1.136, 0.0113). In the subgroup of poverty income ratio, only when the federal poverty rate was greater than 185%, the RA increased by 1.18% for each unit of DII (95%CI = 1.056 to 1.183, 95%CI = 1.056 to 0.0001).

The association between Race and DII scores in RA patients

We used a recursive algorithm to find the possible threshold effect and saturation effect. Among other Hispanic, there was an "inflection point (K)" between DII and RA, which were fitted by a two-piecewise linear regression model, at 1.195. When $K < 1.195$, the size of the effect was 1.069 (95CI% = 0.867 to 1.317, $P = 0.5336$). When $K > 1.195$, the curve showed a rapid upward trend, and with each unit increase of DII, the risk of RA increases by 67.4% (95%CI = 1.229 to 2.280, $P = 0.0011$).

Nutrient comparison between patients with RA and those without RA

The difference between the intake of common nutrients in individuals with RA and those without RA was showed in Table 5. The results showed that individuals with RA generally consumed fewer nutrients than those without RA. Among the nutrients with significant differences between the two groups, energy, protein, total saturated fatty acids, and iron had pro-inflammatory effects, while other nutrients had anti-inflammatory effects.

Table 1
Baseline characteristics of participants

	Overall	Quartile 1 (n = 2605)	Quartile 2 (n = 2604)	Quartile 3 (n = 2606)	Quartile 4 (n = 2606)	P value
DII	0.425 ± 1.685	-4.621 to -0.828	-0.828 to 0.418	0.419 to 1.705	1.706 to 4.535	
Age (year)	54.323 ± 14.956	54.862 ± 15.372	54.015 ± 14.811	54.589 ± 14.775	53.826 ± 14.841	0.042
Gender(%)						< 0.001
Male	35.256	45.681	38.863	30.967	25.518	
Female	64.744	54.319	61.137	69.033	74.482	
Race/Ethnicity (%)						< 0.001
Mexican American	16.140	16.123	18.241	15.886	14.313	
Other Hispanic	10.412	10.403	10.023	11.819	9.401	
Non-Hispanic White	43.595	47.985	43.817	42.978	39.601	
Non-Hispanic Black	21.927	14.894	20.123	22.371	30.315	
Other Race	7.926	10.595	7.796	6.946	6.370	
Body mass index, kg/m2 (%)						< 0.001
< 18.5	1.324	1.305	1.267	0.921	1.804	
18.5–24.9	26.495	31.631	27.189	23.446	23.715	
≥25	72.181	67.063	71.544	75.633	74.482	
Marital status (%)						< 0.001
Married	54.697	61.382	58.948	54.298	44.167	
Widowed	9.164	7.255	8.180	9.977	11.243	
Divorced	14.883	12.668	13.018	14.658	19.186	
Separated	4.002	2.994	3.379	4.490	5.142	
Never married	10.805	9.904	10.023	10.054	13.239	

*Other represents rejection, unknown or missing data

	Overall	Quartile 1 (n = 2605)	Quartile 2 (n = 2604)	Quartile 3 (n = 2606)	Quartile 4 (n = 2606)	P value
Living with partner	6.420	5.758	6.413	6.485	7.022	
Other	0.029	0.038	0.038	0.038%	0.000	
Physical Activity /MET (mean ± SE)	66.730 ± 113.000	73.353 ± 111.516	69.729 ± 114.650	64.347 ± 114.575	59.496 ± 110.780	< 0.001
Smoking at least 100 cigarettes in life						
Yes	46.848	43.033	44.355	47.583	52.417	
No	52.116	53.282	55.376	52.341	47.467	
Other	1.036	3.685	0.269	0.077	0.115	
Alcohol consumption (%)						< 0.001
none	17.378	12.937	13.249	19.455	23.868	
light	76.826	80.422	82.373	75.096	69.417	
moderate	1.459	2.150	0.960%	1.343	1.381	
heavy	0.240	0.230	0.154	0.269	0.307	
other	4.097	4.261	3.264	3.837	5.027	
Diabetes (%)						< 0.001
Yes	11.432	9.488	11.074	12.347	12.813	
No	86.416	88.203	87.116	85.040	85.309	
Borderline	2.089	2.309	1.811	2.402	1.836	
Other	0.063	0.000	0.000	0.211	0.042	
poverty income ratio (%)						< 0.001
< 100% federal poverty rate	15.901	11.209	13.249	16.999	22.141	
100% – 185% federal poverty rate	22.618	19.463	21.006	23.484	26.516	
> 185% federal poverty rate	61.482	69.328	65.745	59.517	51.343	
*Other represents rejection, unknown or missing data						

	Overall	Quartile 1 (n = 2605)	Quartile 2 (n = 2604)	Quartile 3 (n = 2606)	Quartile 4 (n = 2606)	P value
Anemia (%)						
Yes	4.702	3.877	4.147	4.835	5.948	0.007
No	95.231	96.123	95.776	95.088	93.937	
other	0.067	0.000	0.077	0.077	0.115	
Rheumatoid Arthritis (%)						< 0.001
No	82.545	86.104	84.370	81.274	78.434	
Yes	17.455	13.896	15.630	18.726	21.566	
*Other represents rejection, unknown or missing data						

Table 2
Association of dietary inflammatory index with Rheumatoid Arthritis

Rheumatoid Arthritis	β^1 (95% CI ²), P-value		
	Model 1 ³	Model 2 ⁴	Model 3 ⁵
DII	1.142 (1.108, 1.177) < 0.00001	1.158 (1.121, 1.196) < 0.00001	1.068 (1.026, 1.111) 0.00121
DII categories			
Quartile 1	1.0 (Reference)	1.0 (Reference)	1.0 (Reference)
Quartile 2	1.148 (0.985, 1.338) 0.07798	1.189 (1.015, 1.393) 0.03207	1.144 (0.947, 1.383) 0.16262
Quartile 3	1.428 (1.231, 1.656) < 0.00001	1.476 (1.265, 1.723) < 0.00001	1.216 (1.009, 1.466) 0.04001
Quartile 4	1.704 (1.474, 1.970) < 0.00001	1.792 (1.537, 2.089) < 0.00001	1.314 (1.089, 1.585) 0.00434
In sensitivity analysis, dietary inflammatory index was converted from a continuous variable to a categorical variable (quartiles).			
¹ β : effect sizes;			
² 95% CI: 95% confidence interval;			
³ Model 1: no covariates were adjusted;			
⁴ Model 2: adjusted for age, gender and race;			

⁵Model 3: adjusted for age, gender, race, anemia, BMI, alcohol consumption, marital status, diabetes, physical activity, smoke, poverty income ratio.

Table 3
Subgroup analysis

DII	β ¹ (95% CI ²), P-value	P for interaction
Subgroup analysis stratified by age		
Age ≤ 50 years	1.126 (1.041, 1.218) 0.0031	0.1683
Age > 50 years	1.057 (1.010, 1.107) 0.0167	
Subgroup analysis stratified by race/ethnicity		
Mexican American	1.006 (0.899, 1.125) 0.9222	0.0313
Other Hispanic	1.265 (1.112, 1.438) 0.0003	
Non-Hispanic White	1.063 (0.998, 1.132) 0.0581	
Non-Hispanic Black	1.002 (0.930, 1.079) 0.9612	
Other Races	1.092 (0.908, 1.313) 0.3519	
Subgroup analysis stratified by BMI		
< 18.5	1.035 (0.641, 1.671) 0.8875	0.9777
18.5–24.9	1.067 (0.981, 1.161) 0.1288	
≥25	1.075 (1.028, 1.125) 0.0017	
Subgroup analysis stratified gender		
Male	1.039 (0.976, 1.107) 0.2334	0.2133
Female	1.094 (1.038, 1.153) 0.0008	
Subgroup analysis stratified by smoke		
Yes	1.092 (1.039, 1.148) 0.0005	0.9098
No	1.074 (1.016, 1.136) 0.0113	
Subgroup analysis stratified by poverty income ratio		
< 100% federal poverty rate (%)	1.064 (0.983, 1.151) 0.1269	0.4286
100% – 185% federal poverty rate	1.064 (0.999, 1.133) 0.0543	
> 185% federal poverty rate	1.118 (1.056, 1.183) 0.0001	

The showing results of subgroup analysis were adjusted for age, race, gender, BMI, alcohol consumption, smoke, poverty income ratio, except the effect modifier.

1 β : effect sizes;

295% CI: 95% confidence interval;

Table 4
The association between RA and DII by race

DII	RA
Other Hispanic	
Fitting by the standard linear model	1.265 (1.112, 1.438) 0.0003
Fitting by the two-piecewise linear model	
Inflection point (K)	1.195
<K-segment effect 1	1.069 (0.867, 1.317) 0.5336
> K-segment effect 2	1.674 (1.229, 2.280) 0.0011
The effect difference between 2 and 1	1.567 (0.998, 2.461) 0.0512
The predicted value of the equation at the Inflection point	-1.525 (-1.814, -1.235)
Log-likelihood ratio	0.051
Age, gender, anemia, BMI, alcohol consumption, marital status, diabetes, physical activity, smoke, and poverty income ratio were adjusted.	

Table 5
Difference macronutrient intake for individuals with and without arthritis

	With arthritis Mean(SE)	Without arthritis Mean(SE)	Overall inflammatory effect score	P- value
Energy (kcal)	1906.09	1967.38	0.18	0.010
Protein (gm)	72.52	103.96	0.021	0.000
Carbohydrate(gm)	229.15	234.07	0.097	0.094
Dietary fiber (gm)	15.08	17.78	-0.663	0.000
Total fat (gm)	74.65	76.21	0.298	0.169
Total saturated fatty acids (gm)	23.89	25.53	0.373	0.000
Total monounsaturated fatty acids (gm)	26.77	27.57	-0.009	0.074
Total polyunsaturated fatty acids (gm)	17.22	17.78	-0.377	0.071
Cholesterol (mg)	288.20	280.79	0.11	0.215
Vitamin E as alpha-tocopherol (mg)	7.48	11.88	-0.419	0.000
Vitamin A, RAE (mcg)	587.11	595.71	-0.401	0.602
Beta-carotene (mcg)	2043.56	2253.63	-0.584	0.063
Vitamin B1 (mg)	1.44	18.28	-0.098	0.000
Vitamin B2 (mg)	1.91	1.98	-0.068	0.009
Niacin (mg)	22.23	23.32	-0.246	0.002
Vitamin B6 (mg)	1.81	2.20	-0.365	0.000
Folic acid (mcg)	152.09	164.70	-0.19	0.001
Vitamin B12 (mcg)	4.71	9.24	0.106	0.000
Vitamin C (mg)	76.37	80.41	-0.424	0.081
Magnesium (mg)	268.77	302.53	-0.484	0.000
Iron (mg)	13.46	17.66	0.032	0.000
Zinc (mg)	10.11	10.84	-0.313	0.001
Selenium (mcg)	101.39	143.35	-0.191	0.000
Caffeine (mg)	164.70	161.50	-0.11	0.621

	With arthritis	Without arthritis	Overall inflammatory effect score	P- value
	Mean(SE)	Mean(SE)		
Alcohol (gm)	7.46	11.13	-0.278	0.000

Discussion

Although a large number of studies support the impact of a single diet on RA [25–28], the complexity of the human diet, genetic differences, and the variability of the external environment determines that a single nutrient may not play an important role in the onset and development of RA. Therefore, in this study, DII was used to quantify the inflammatory potential of individual dietary intake, logistic regression was used to study whether there was an association between DII and RA risk, and the degree of association. We also studied the possible interaction of confounding factors on the association between DII and RA through subgroup analysis.

The results of this cross-sectional study based on the American population showed that whether adjusted or not adjusted covariates, the association between DII and RA was positive, and the risk of RA increased with the increase of DII. This is the same result as the previously published study [22–23].

In addition, age, race, BMI, and other covariates that affect the incidence of RA also affect the impact of DII on the risk of RA. Moreover, the direction of the influence is roughly the same. In RA, the prevalence rate of the female is higher than male [29]. Obesity [30] and smokers [31] are all risk factors for RA. The results of our subgroup analysis also showed that DII had a greater impact on the risk of RA if the individuals were female, overweight, or had a smoking habit. It is speculated that DII may have a synergistic effect with the triggering factors of RA.

But in the subgroup of age and poverty income ratio, this rule does not seem to hold. The peak incidence of RA is between the ages of 50 to 60 years [29]. But our research showed that there was a greater association between DII and RA when they are less than 50 years old. It has been reported that the energy, minerals, and vitamin intake of the elderly in the United States were generally lower than Recommended Daily Allowance (RDA), and women were lower than men [32]. Although later studies have shown that the quality of the diet of the elderly was better than the young and middle-aged [33]. The diet quality preference of the elderly, but the food intake may be reduced as the individual gets older, resulting in a decline in overall nutrition intake. As a result, the measured value of the overall effect of individuals over 50 years old may be on the low side.

Genetic susceptibility is the main cause of RA. There is ethnic variation in both the frequency and types of SE-carrying HLA-DRB1 alleles [34]. Races carrying HLA-DRB1 alleles are more likely to lead to the onset of RA [35]. However, studies have shown that Hispanic people were less likely to have HLA-DRB1 alleles. Therefore, one of the reasons for the high incidence of RA in Hispanics may be due to its sensitivity to

pro-inflammatory food. We used a recursive algorithm found that the risk of RA increased rapidly with the increase of DII. An obvious gradient increase curve in Fig. 2 could demonstrate the change. However, the result of the logarithmic likelihood ratio test was 0.051. We consider that the DII data of the second segment of the effect was too little, which leads to the offset of the data operation result.

In our subgroup analysis, it was shown that the poverty income ratio exceeded 185% had a higher risk of individuals with RA. However, this is not consistent with the results of previous studies^[8]. By analyzing the characteristics of the participants included, we found that the population whose poverty income ratio exceeded 185% accounted for 61.482%, but the proportion of those was declining with the increase of DII among the four groups of DII. Therefore, we consider the contradiction in the results is due to the different proportions of the included population in different groups, which leads to the deviation of the data results.

In this study, we also compared the common nutrients in detail and found that individuals with RA had more significant deficiencies in energy, protein, dietary fiber, total saturated fatty acids, vitamin E, vitamin B6, magnesium, zinc, Niacin, Selenium, iron, and alcohol than individuals without RA. The above nutrients mainly come from vegetables, fruits, grains, edible oils, eggs, dairy products, fish, and so on^[37–39]. These foods, combined with drinking a moderate amount of alcohol, are in line with the argument that the Mediterranean diet and the correction of vitamin D are beneficial to the prevention and alleviation of RA.^[10,40] In addition, although energy and protein are pro-inflammatory foods, they are indeed necessary for the human body to maintain weight and energy metabolism. Weight loss as a result of reduced energy intake may be another adverse factor in the development of the disease^[41].

We also found a decrease in total saturated fatty acid intake in individuals with RA. However, most of the current studies have shown that the intake of unsaturated fatty acids was beneficial to RA, which seems not to conform to our results. However, it is impossible to consume unsaturated fatty acids without saturated fatty acids in natural foods^[44]. Studies have shown that dairy products and olive oil contain saturated fatty acids, especially dairy products, which are good for RA. Therefore, we suspect that an excessively low intake of total saturated fatty acids may not reach the amount of fat digestion required by the human body, which is not conducive to the prevention and alleviation of RA.

Nutrition intake is an indispensable activity of the day. This study complements the association between DII and RA in the American, further confirms the role of nutritional factors in the risk and progress of RA which has individualized differences. When the pathogenesis of RA is unknown and the lack of effective cure^[45], reducing anti-inflammatory food intake, increasing the intake of anti-inflammatory foods, and optimizing the dietary nutritional structure of the genetic susceptibility may achieve the effect of prevention and mitigation of RA. However, this doesn't mean a mere intake of anti-inflammatory foods, energy, protein, and saturated fatty acids is still the body's necessity.

The dietary data included in this study were from the participants' daily records, which do not represent their long-term eating habits. Therefore, it is unknown whether the results of this study can be

representative. In addition, due to the possible deviation of the basic characteristics of the included population, such as the impact of the proportion of the poverty income ratio in the above discussion on the results of the study, a slight error in the results may be caused.

Conclusions

Our study revealed a positive association between RA and DII among most Americans. It is more significant in females, other Hispanic, overweight, and smokers. Especially in other Hispanic, there is an inflection point (K) between DII and RA. When $K > 1.195$, the impact of DII on RA risk is significantly increased. Therefore, optimizing the dietary structure and increasing the intake of anti-inflammatory food is helpful to prevent and alleviate the onset and deterioration of RA.

Abbreviations

RA Rheumatoid arthritis

DII Dietary inflammation index

NHANES National Health and Nutrition Examination Survey

BMI Body mass index

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

Not applicable

Availability of data and materials

The datasets generated and/or analyzed during the current study are available in the NAHANSE (<https://www.cdc.gov/nchs/nhanes/index.htm>).

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions statement

SX statistically analyzed data, made the tables and figures and completed the writing of the manuscript.

YW was contributed to the sorting and screening of the data needed for the research, and participating in the writing of the manuscript.

SQ calculated DII score and conception and design of the study.

YJ and JL were contribute to the interpretation of data.

XD and TX contributed to the conception and design of the study.

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Figures

Figure 1

Relationship between dietary inflammatory index and Rheumatoid Arthritis

Figure 2

The association between dietary inflammatory index and Rheumatoid Arthritis by race, age, gender, anemia, BMI, alcohol consumption, marital status, diabetes, physical activity, smoke, and poverty income ratio were adjusted. (1) Mexican American; (2) Other Hispanic; (3) Non-Hispanic White; (4) Non-Hispanic Black; (5) Other Race - Including Multi-Racia