Association of Pro-inflammatory Diet With Risk of Type 2 Diabetes and Hypertension; Results From RaNCD Cohort Study

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Research

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Abstract

Background: Dietary factors and inflammation are associated with most non-communicable diseases (NCDs). The Dietary Inflammatory Index (DII) is a developed validated assessment tool. This study was conducted to assessed association of DII with the hypertension and type 2 diabetes mellitus (T2DM).

Methods: This cross-sectional analysis was performed on 9,811 participants of 35 to 65 years of the base-line phase data of Ravansar Non-Communicable Diseases (RaNCD) cohort study. The DII was calculated using 31 parameters food of food frequency questionnaire (FFQ). The estimates were performed using univariable and multivariable logistic regression.

Results: The mean DII scores in healthy participants was -2.32± 1.60, in participant with T2DM, hypertension and both were -2.23± 1.59, -2.45± 1.60 and -2.25± 1.60, respectively (P= 0.011). Pre-inflammatory diet was significantly higher in male compared to female (P<0.001). In the most pro-inflammatory diet was significantly higher BMI (body mass index), triglyceride, energy intake, smokers; and was significantly lower socio-economic status (SES), physical activity and HDL-C compared to the most anti-inflammatory diet. Participants with T2DM, hypertension and comorbidity had a significantly higher mean of anthropometry indices (P<0.001) and lipid profile compared to healthy subjects (P<0.001). After adjustment for age, sex and physical activity, the odds of T2DM in the fourth quartile of DII was 1.48 (95% CI: 1.19, 1.85) times higher compared to the first quartile of DII.

Conclusions: Pro-inflammatory diet was weak associations with hypertension. Pro-inflammatory diet was significant associations with increasing T2DM and its related risk factors. Modification of diet and lifestyle is suggested to reduce inflammation.

Introduction

Chronic systemic inflammation is an important factor in the pathogenesis of non-communicable diseases (NCDs) including cardiovascular disease (CVDs), type 2 diabetes mellitus (T2DM), metabolic syndrome (MetS), hypertension and cancer [1–3]. Obesity and overweight, insulin resistance, and overexpression of pro-inflammatory proteins including C-reactive protein (CRP) and cytokines (IL-1β, IL-6, and TNF-α) induce chronic inflammation in type 2 diabetes and hypertension [4]. In addition, environmental, psychosocial, and behavioural factors can cause inflammation during times of stress [5], by modifying some of these factors, inflammation can be reduced. Diet is one of the most important factors related to lifestyle, the modification of which can moderate the inflammatory process [6, 7].

Previous studies have shown that some dietary patterns, including high consumption of fibre, fruits, vegetables, and low consumption of fats reduce levels of inflammatory markers and consequently decrease the risk of NCDs [8, 9]. Inverse, the Western diet (includes processed meats and refined carbohydrates) is associated with increased inflammatory markers and appears to be a risk factor for some of NCDs [10].

Several food and food components affect the concentration of inflammatory markers in the blood. Recently to assess the inflammatory potential of the diet of individuals has been used a relatively new index called the dietary inflammatory index (DII), which is based on 45 food parameters. DII includes a range of from anti to pro-inflammatory and a high DII indicates that a person's diet is inflammatory [11]. Since there are various lifestyles and dietary patterns in different geographical areas and ethnic groups, it is important to evaluate the DII in different populations.

Considering the increasing prevalence of NCDs, its prevention and control is very important. Therefore, identifying and modifying changeable risk factors of NCDs can reduce the burden of diseases in communities. According to reports, hypertension and its complications cause about 9.4 million deaths worldwide every year [12], and also Asia is known as the fastest growing region for type 2 diabetes [13]. Accordingly, using data from about 10,000 adults in the RaNCD on going only Kurdish cohort study provide valuable data. The current study aimed to determine if a pro-inflammatory diet assessed using DII was associated with an increased risk of hypertension and T2DM in a Kurdish population.

Methods And Materials

Study population

In this cross-sectional study, we used the baseline data of RaNCD that is one of the sub-studies of the national Prospective Epidemiological Research Studies in IrAN (PERSIAN) cohort [14]. Ravansar is one of the western cities of Kermanshah Province with a population of about 50,000 in the west of Iran. The details of RaNCD study have already been published [15]. All participants aged 35-65 years in the bassline phase of RaNCD entered this study (10,000 individuals). According to the purpose of the present study, subjects with cancer and pregnant woman were excluded from the study. The final study population included 9,811 adults (Fig 1).

Data collection

Socio-demographic characteristics including age, sex, marital status, residence place and information on personal habits (smoking status and alcohol consumption) was collected face to face using digital questionnaires.

The socio-economic status (SES) was measured, using 18 items (including housing, car based on its price, dishwasher, freezer, washing machine, computer, laptop, internet access, motorcycle, color TV, TV type, bathroom, cell phone, vacuum cleaner, area per capita, room per capita, education level and residence place) by principal component analysis (PCA) method; finally, the SES was classified from the poorest to the richest in five groups [16].

Physical activity questionnaire (including 22 questions) standardized cohort study was used to assess participants' physical activity, based on met/hour per week and divided into three groups (light, moderate, high).

To measure biochemical markers including triglyceride (TG), low-density lipoprotein Cholesterol (LDL-C), high-density lipoprotein Cholesterol (HDL-C), and Total cholesterol (T-C) and fasting blood sugar (FBS); blood samples were collected after a 12 hours fasting.

Height (with 0.1 cm precision) was measured using a BSM 370 (Biospace Co,Seoul, Korea), Weight and other anthropometric indices including body mass index (BMI), body fat mass (BFM) and visceral fat area (VFA), (with 0.5 kg precision) were measured using a Bio Impedance Analyzer BIA (InBody 770 Biospace, Korea). Waist circumference (WC) and waist to hip ratio (WHR) were measured by standard methods. Blood pressure (BP) measured by a manometer cuff and stethoscope after 10 minutes of rest from arm in the seated position.

Assessment of the dietary inflammatory index

The DII scores was calculated using items of Food Frequency Questionnaire (FFQ). Participants responded to questions about amounts and frequencies of consumption of food groups. For help estimate portion sizes the photo in the booklet were shown to them.

Shivappa et al. have reported 45 foods items were associated with one or more of the inflammatory including Interleukin-1b (IL-1b), Interleukin- 6 (IL-6), Tumor Necrosis Factor-a (TNF-a) or C-reactive protein (CRP) or anti-inflammatory markers including Interleukin-4 (IL-4) and Interleukin-10 (IL-10). Based on the Shivappa et al. method, on the basis of mean and standard deviation (SD) of global intake, Zscore was determined for each parameter. In the next step the Z-score became a percentile. The inflammatory score for each of the food parameters was calculated using this method, and finally the inflammatory score of all parameters was summed to calculate the total DII score. The more positive DII scores indicate more pro-inflammatory diets and more negative scores imply more anti-inflammatory diets [17, 18]. DII scores were categorized into four groups (quartile) to assess associations. The first and fourth quartiles had the lowest and highest DII scores, respectively. For the present study out of 45 food parameters, 31 parameters were used to calculate DII, including; carbohydrate, protein, total fat, trans fat, monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), cholesterol, saturated fat, omega-3, omega-6, vitamins of A, B6, B12, C, D, E, selenium, zinc, energy, iron, magnesium, niacin, riboflavin, thiamine, beta-carotene, fiber, folic acid, caffeine, garlic, onion and tea.

Hypertension and type 2 diabetes mellitus assessment

Hypertension was defined as having a systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg, and/or currently taking antihypertensive drugs [19]. T2DM was defined as having an FBS (fasting blood sugar) of ≥126mg/dl and/or being on diabetes medication and/or if the diabetes was confirmed by a health practitioner [20].

Statistical Analysis

Descriptive analysis including mean ± standard deviation and frequency (percentage) by quartiles of DII was done for quantitative and qualitative variables, respectively. In addition, mean ± standard deviation

of anthropometric and biochemical characteristics was compared by one-way ANOVA among four studied groups. Logistic regression model was used to determine the association between DII and hypertension and T2DM. The crude and adjusted odds ratios with 95% confidence interval were reported. All analyses were done with STATA software version 14.2 (Stata Corp, College Station, Tex).

Results

Basic characteristics

A total of 9,811 participants between 35–65 years were included in the present study. Table 1 presented baseline characteristics of the study participants according to DII Quartiles. Pro-inflammatory diet was significantly higher in male compared to female ($P < 0.001$). According to DII score quartiles, subjects with the most pro-inflammatory diet had significantly higher BMI, higher TG, high smokers, and lower SES, lower physical activity, and lower HDL-C. The urban residents ($P < 0.001$) and married subjects ($P < 0.001$) had a significantly higher DII score. The increase in DII scores has been associated with an increase in the percentage of subjects with T2DM ($P = 0.086$) and hypertension ($P = 0.003$). In subjects on a pro-inflammatory diet, daily energy intake ($P < 0.001$) and energy intake from lipids ($P < 0.001$) were significantly higher compared to subjects with anti-inflammatory diet.

Table 1
Baseline characteristics of the participants according to DII Quartiles (RaNCD cohort data, N = 9811).

Variable	Frequency (%), Mean ± SD				P value
	Q1 (Most Anti-Inflammatory)	Q2	Q3	Q4 (Most Pro-Inflammatory)	
Frequency	n = 2,451	n = 2,430	n = 2,463	n = 2,467	
DII range	(-6.18, -3.54)	(-3.54, -2.68)	(-2.69, -1.38)	(-1.38, 4.27)	
Sex					< 0.001
Male	983 (40.11)	1083 (44.57)	1214 (49.29)	1449 (58.74)	
Female	1468 (59.89)	1347 (55.43)	1249 (50.71)	1018 (41.26)	< 0.001
Residence place					
Urban	934 (38.11)	1280 (52.67)	1674 (67.97)	1991 (80.71)	< 0.001
Rural	1517 (61.89)	1150 (47.33)	789 (32.03)	476 (19.29)	
Alcohol use					
No	2373 (96.82)	2334 (96.05)	2344 (95.17)	2278 (92.34)	< 0.001
Yes	78 (3.18)	96 (3.95)	119 (4.83)	189 (7.66)	
Socio-economic status					
1(poorest)	780 (31.84)	499 (20.54)	326 (13.24)	329 (13.35)	< 0.001
2	544 (22.20)	502 (20.67)	470 (19.08)	433 (17.57)	
3	410 (16.73)	510 (21)	519 (21.07)	525 (21.30)	
4	365 (14.90)	475 (19.56)	565 (22.94)	566 (22.96)	
5 (richest)	351 (14.33)	443 (18.24)	583 (23.67)	612 (24.83)	
Marital status					
Married	2114 (86.25)	2193 (90.25)	2245 (91.15)	2294 (92.99)	< 0.001
Single	156 (6.36)	105 (4.32)	90 (3.65)	67 (2.72)	

Variable	Frequency (%), Mean ± SD				P value
	Q1 (Most Anti-Inflammatory)	Q2	Q3	Q4 (Most Pro-Inflammatory)	
Widowed/Divorced	181 (7.39)	132 (5.43)	128 (5.2)	106 (4.3)	
Physical activity (Met-h/week)					
Light	722 (29.46)	788 (32.43)	749 (30.41)	716 (29.02)	< 0.001
Moderate	1242 (50.67)	1138 (46.83)	1233 (50.06)	1135 (46.01)	
High	487 (19.87)	504 (20.74)	481 (19.53)	616 (24.97)	
Smoking status					
No	1962 (80.08)	1962 (80.81)	1960 (79.61)	1892 (76.75)	0.003
Yes	488 (19.92)	466 (19.19)	502 (20.39)	573 (23.25)	
Diabetes					
No	2241 (91.88)	2221 (91.81)	2246 (91.56)	2209 (90.09)	0.086
Yes	198 (8.12)	198 (8.19)	207 (8.44)	243 (9.91)	
Hypertension					
No	2007 (81.88)	2073 (85.31)	2090 (84.86)	2095 (84.92)	0.003
Yes	444 (18.12)	357 (14.69)	373 (15.14)	372 (15.08)	
Age (year)	48.71 ± 8.46	47.58 ± 8.33	46.81 ± 8.16	46.20 ± 7.89	0.004
BMI (kg/m^2)	26.99 ± 4.68	27.40 ± 4.62	27.58 ± 4.60	27.95 ± 4.56	0.668
WHR	0.94 ± 0.06	0.94 ± 0.061	0.94 ± 0.06	0.95 ± 0.06	0.054
TG (mg/dl)	134.20 ± 81.09	134.51 ± 76.98	137.99 ± 82.40	142.95 ± 89.37	< 0.001
HDL-C (mg/dl)	48.03 ± 11.55	46.94 ± 11.31	45.80 ± 11.31	44.49 ± 10.77	< 0.001
LDL-C (mg/dl)	103.40± 25.99	102.24 ± 25.23	101.18± 24.62	101.12 ± 25.73	0.041

Variable	Frequency (%), Mean ± SD				P value
	Q1 (Most Anti-Inflammatory)	Q2	Q3	Q4 (Most Pro-Inflammatory)	
T-C (mg/dl)	187.40 ± 38.69	185.95 ± 37.48	184.06 ± 36.85	183.63 ± 38.20	0.083
Energy intake (kcal/d)	1904.96 ± 691.66	2052.14 ± 720.88	2328.54 ± 822.37	2904.19 ± 1059.81	< 0.001
Carbohydrate (%E)	66.16± 8.69	65.37 ± 7.59	63.89 ± 7.48	62.63 ± 7.95	< 0.001
Protein (%E)	14.97 ± 1.73	15.58 ± 1.95	15.83 ± 1.94	16.56 ± 2.32	< 0.001
Oil/Fat (%E)	18.22 ± 8.47	18.61 ± 7.16	20.08± 7.14	21.03± 7.44	< 0.001

Table 2 presented the anthropometric and biochemical characteristics of the participants in 4 groups. The mean age in subjects with T2DM, hypertension and comorbidity (T2DM and hypertension) was significantly higher compared to healthy subjects ($P < 0.001$). Subjects with T2DM, hypertension and comorbidity had a significantly higher mean of BMI, WHR, WC, BFM and VFA compared to healthy subjects ($P < 0.001$). The mean of lipid profile (LDL-C, TG and T-C) was significantly higher in subjects with T2DM, hypertension and comorbidity ($P < 0.001$).

Table 2
Anthropometric and biochemical characteristics of participant (RaNCD cohort data, N = 9811)

Parameters	Healthy (n = 7708)	Diabetes (n = 571)	HTN (n = 1271)	Diabetes & HTN (n = 276)	P value
Age (year)	45.93 ± 7.88	50.44 ± 7.43	52.83 ± 7.72	54.22 ± 6.85	< 0.001
BMI (kg/m^2)	27.17 ± 4.60	28.88 ± 4.35	28.48 ± 4.68	29.04 ± 4.45	< 0.001
WHR	0.94 ± 0.06	0.96 ± 0.06	0.96 ± 0.06	0.97 ± 0.06	< 0.001
WC (cm)	96.45 ± 10.40	100.58 ± 9.80	99.74 ± 10.67	101.54 ± 9.84	< 0.001
BFM (kg)	24.38 ± 9.47	27.82 ± 9.25	27.20 ± 9.57	28.42 ± 9.25	< 0.001
VFA (cm^2)	117.93 ± 51.02	137.82 ± 49.26	135.55 ± 51.18	143.18 ± 49.65	< 0.001
DII	-2.32 ± 1.60	-2.23 ± 1.59	-2.45 ± 1.60	-2.25 ± 1.60	0.011
TG (mg/dl)	131.03 ± 74.52	184.70 ± 128.79	147.56 ± 89.35	170.32 ± 98.01	< 0.001
HDL-C (mg/dl)	46.60 ± 11.35	43.78 ± 11.25	46.16 ± 11.08	44.30 ± 10.69	< 0.001
LDL-C (mg/dl)	101.31 ± 25.05	104.71 ± 27.73	104.68 ± 25.42	102.33 ± 29.04	< 0.001
T-C (mg/dl)	183.96 ± 36.96	191.74 ± 43.68	189.64 ± 38.15	187.44 ± 44.18	< 0.001
Energy intake (kcal/d)	2328.83 ± 918.87	2285.62 ± 906.03	2146.12 ± 904.61	2198.77 ± 976.80	< 0.001
Carbohydrate (%E)	64.44 ± 8.10	64.05 ± 8.05	64.65 ± 7.89	64.65 ± 7.89	0.021
Protein (%E)	15.66 ± 20.3	16.06 ± 2.16	15.94 ± 2.23	16.28 ± 2.27	< 0.001
Oil/Fat (%E)	19.60 ± 7.72	19.80 ± 7.52	18.76 ± 7.40	19.04 ± 7.10	0.002

Association between dietary inflammatory index and T2DM

The crude model of logistic regression has presented odds of T2DM in subjects with the most pro-inflammatory diet showed 1.24 (95% CI: 1.02, 1.51) times greater compared to subjects with most anti-

inflammatory. After adjusting for age and sex the odds of T2DM in the fourth quartile of DII was 1.54 (95% CI: 1.26, 1.90) times higher compared to the first quartile of DII (Table 3).

Table 3
Logistic regression analysis of association between dietary inflammatory index and T2DM

Diabetes	**Dietary Inflammatory Index**			
	Q 1 (Most Anti-Inflammatory)	**Q 2**	**Q 3**	**Q 4 (Most Pro-Inflammatory)**
	OR	**OR (95% CI)**	**OR (95% CI)**	**OR (95% CI)**
Model I	1	1.01 (0.82, 1.24)	1.04 (0.85, 1.28)	1.24 (1.02, 1.51)
Model II	1	1.10 (0.89, 1.36)	1.22 (0.99, 1.50)	1.54 (1.26, 1.90)
Model III	1	1.08 (0.88, 1.33)	1.19 (0.96, 1.46)	1.48 (1.19, 1.85)

Model I: crude; **Model II**: Adjusted for age and sex; **Model III**: Adjusted for age, sex, BMI, BFM, WHR, carbohydrate (%E), protein (%E) and oil/fat (%E) and physical activity

Association between dietary inflammatory index and hypertension

After adjusting for age and sex, the odds of hypertension, across all DII score quartiles were 0.86 (95% CI: 0.73, 1.0), 0.98 (95% CI: 0.84, 1.15) and 1.06 (95% CI: 0.90, 1.24). After adjusting for another confounder in model III, we observed that in the fourth quartile of DII had approximately 10% higher odds of hypertension compared to the first quartile of DII, which was not statistically significant (Table 4). Figure 2 showed association of DII with risk of T2DM and hypertension in the crude and adjusted models.

Table 4
Logistic regression analysis of association between dietary inflammatory index and hypertension

	Dietary Inflammatory Index			
HTN	Q 1 (Most Anti-Inflammatory)	Q 2	Q 3	Q 4 (Most Pro-Inflammatory)
	OR	OR (95% CI)	OR (95% CI)	OR (95% CI)
Model I	1	0.78 (0.67, 0.91)	0.81 (0.69, 0.94)	0.80 (0.69, 0.93)
Model II	1	0.86 (0.73, 1.0)	0.98 (0.84, 1.15)	1.06 (0.90, 1.24)
Model III	1	0.85 (0.73, 1.01)	0.99 (0.84, 1.16)	1.10 (0.92, 1.31)

Model I: crude; **Model II**: Adjusted for age and sex; **Model III**: Adjusted for age, sex, BMI, BFM, WHR, carbohydrate (%E), protein (%E) and oil/fat (%E) and physical activity

Discussion

The finding of the current study demonstrates odds of T2DM in the fourth quartile of DII (indicating the more pro-inflammatory diet) had significant higher compared to the first quartile of DII (indicating the more anti-inflammatory diet). This association remained after adjustment for confounders including age, sex, physical activity and energy intake. A study by Laouali et al. (2019) on French adults presented that a higher anti-inflammatory diet is associated with a lower risk of T2DM [21]. Denova-Gutiérrez et al. have reported association pro-inflammatory diet with higher odds of T2DM among adult Mexicans [22]. We did not find a significant association between DII and risk of hypertension. Findings of a cohort study on a large population of French show a weak association between DII and the incidence of hypertension [9]. While, a study of Australian women found that having a pro-inflammatory diet was associated with an increased risk of developing hypertension [23]. The cause of this discrepancy may be due to differences in the calculation of DII in different studies. In the present study, the DII score was calculated based on 31 dietary parameters. Some studies have calculated DII using 27 or 25 dietary parameters [22, 23]. Differences in the studied populations, genetic factors and variety dietary patterns in the world - leading to differences in DII range -are the causes of contradiction in the results.

This study demonstrates that male, urban residents and married subjects had a significantly higher DII score. In addition, in the most pro-inflammatory diet were significantly higher BMI, higher TG, and SES less, physical activity less and HDL-C less compared to the most anti-inflammatory diet. These factors are part of the lifestyle; evidence suggests that lifestyle play a role in preventing or causing inflammation. Investigations have showed of association of anti-inflammatory effect of lifestyle changes with T2DM, so that increases in fiber intake and moderate to vigorous leisure time physical activity has predicted decreases in CRP and/or IL-6 level [24, 25]. A longitudinal study in the UK presented a linear association

between CRP levels and weight gain over 9 years [25]. Some researchers has also introduced obesity as a mediator between anti-inflammatory diet and the risk of T2DM and hypertension [9, 21]. Our findings as well as suggest anti-inflammatory diet was positive associated with high BMI that these findings demonstrate the importance of lifestyle on inflammatory markers.

On the other hand adherence to the Mediterranean diet with low inflammation is known as a protective factor for T2DM (49% risk reduction) and lower levels of TNF-α, CRP and IL-6 [26]. Researches with a single-nutrient approach evaluate the polyphenols in grapes and raisins that both of which have shown an effect by reducing plasma TNF- concentrations [27, 28]. Investigation of dietary patterns has shown subjects with a diet high in red meat, low-fiber bread and cereals, dried beans, fried potatoes, tomato vegetables, eggs, cheese, and a low intake of wine had approximately 4.5 times greater risk of T2DM [29]. As well as, a significant association was found between high-salt and high-fat diet and the risk of T2DM [30]. A randomized controlled trial (RCT) has shown CPR in the Mediterranean diet decreased by 37% and adiponectin increased by 43% at 1 year [31]. Moreover, most high-consumption foods in the most pro-inflammatory diet, including red and processed meats, refined grains, and soft drinks, have associated with inflammatory markers and the risk of T2DM [32]. Additionally, CRP concentrations can be predict risk for mortality in adults with T2DM [33]. There are evidence that show, increased production of the IL-1 family cytokines, IL-1β and IL-18 is associated with risk of hypertension [34, 35]. Therefore, it can be concluded that inflammatory markers are involved in the development of T2DM and hypertension, and diet is one of the most important options in increasing or decreasing the level of inflammatory markers; and the DII is a reliable tool to examine these associations.

We used validated dietary questionnaire for calculation of DII, therefore diet was face to face data collection which may have reduced measurement error. However, the diet may change over the time and we are unable to measure the changes; longitudinal studies evaluating these associations are needed to determine causality. Large sample size and using of data from a well-designed cohort study were the advantages of this study and we were able to adjust for potentially confounding variables. In addition, the first study is on a large population of the Kurdish subjects and makes it possible to compare other ethnicities worldwide.

Conclusion

In conclusion, we observed significant associations between pro-inflammatory diet and the risk of T2DM; and weak associations between pro-inflammatory diet and hypertension. Subjects with the most pro-inflammatory diet had significantly higher BMI, TG, and lower SES, physical activity and HDL-C compared to the most anti-inflammatory diet. Therefore, life style modification (including following an anti-inflammatory diet, physical activity and maintaining a normal weight) can be prevented inflammation, ultimately reduces the developing of T2DM and hypertension.

Abbreviations

NCDs: Non-communicable diseases; CVDs: Cardiovascular disease; T2DM: Type 2 diabetes mellitus; MetS: Metabolic syndrome; CRP: C-reactive protein; DII: dietary inflammatory index; RaNCD: Ravansar Non-Communicable Diseases; FFQ: Food frequency questionnaire; SES: Socio-economic status; PERSIAN: Prospective Epidemiological Research Studies in IrAN; PCA: Principal component analysis; TG: Triglyceride; LDL-C: Low-density lipoprotein Cholesterol; HDL-C: High-density lipoprotein Cholesterol; T-C: Total cholesterol; FBS: Fasting blood sugar; BMI: Body mass index; BFM: Body fat mass; VFA: visceral fat area, WC: Waist circumference; WHR: Waist to hip ratio; BP: Blood pressure; DBP : Diastolic Blood Pressure; SBP : Systolic Blood Pressure; Il-1β : Interleukin-1β; Il-4: Interleukin 4; Il-6 :Interleukin 6; Il-10 : Interleukin 10; MUFA: Monounsaturated fatty acids; PUFA: Polyunsaturated fatty acids; RCT: Randomized controlled trial; CI: Confidence interval; SD: Standard deviation

Declarations

Ethics approval and consent to participate

The study was approved by the ethics committee of Kermanshah University of Medical Sciences (KUMS.REC.1399.1050). From all participants was taken oral and written informed consent.

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Consent for publication

Not applicable.

Availability of data and materials

The data sets generated during this study are available from the correspondence author on reasonable request via email.

Competing interests

The authors declare that there is no competing interest.

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Authors' contributions

SAJ Investigation, Writing original draft, Visualization. YP: Supervision, Review & editing. SHR and MD: Conceptualization, Methodology, Validation, Formal analysis, Investigation, Review. FN, BH and ESH:

Review & editing, Funding acquisition. MM, FH and SE: Investigation, review & editing

References

1. Guzik TJ, Touyz RM. Oxidative stress, inflammation, and vascular aging in hypertension. Hypertension. 2017;70(4):660-67. https://doi.org/10.1161/HYPERTENSIONAHA.117.07802.
2. Lontchi-Yimagou E, Sobngwi E, Matsha TE, Kengne AP. Diabetes mellitus and inflammation. Curr Diab Rep. 2013;13(3):435-44. https://doi.org/10.1007/s11892-013-0375-y.
3. Esser N, Legrand-Poels S, Piette J, Scheen AJ, Paquot N. Inflammation as a link between obesity, metabolic syndrome and type 2 diabetes. Diabetes Res Clin Pract. 2014;105(2):141-50.https://doi.org/10.1016/j.diabres.2014.04.006.
4. Duncan BB, Schmidt MI, Pankow JS, Ballantyne CM, Couper D, Vigo A, et al. Low-grade systemic inflammation and the development of type 2 diabetes: the atherosclerosis risk in communities study. Diabetes. 2003;52(7):1799-805.https://doi.org/10.016/10.2337/diabetes.52.7.1799.
5. Kang D-H, Rice M, Park N-J, Turner-Henson A, Downs C. Stress and inflammation: A biobehavioral approach for nursing research. West J Nurs Res. 2010;32(6):730-60.https://doi.org/10.1177/0193945909356556.
6. Smidowicz A, Regula J. Effect of nutritional status and dietary patterns on human serum C-reactive protein and interleukin-6 concentrations. Advanc in nutr. 2015;6(6):738-47.https://doi.org/10.3945/an.115.009415. .
7. Cruz-Teno C, Pérez-Martínez P, Delgado-Lista J, Yubero-Serrano EM, García-Ríos A, Marín C, et al. Dietary fat modifies the postprandial inflammatory state in subjects with metabolic syndrome: the LIPGENE study. Mol Nutr Food Res. 2012;56(6):854-65.https://doi.org/10.1002/mnfr.201200096.
8. Asemi Z, Esmaillzadeh A. DASH diet, insulin resistance, and serum hs-CRP in polycystic ovary syndrome: a randomized controlled clinical trial. Horm Metab Res. 2015;47(03):232-38.https://doi.org/10.1055/s-0034-1376990.
9. MacDonald C-J, Laouali N, Madika A-L, Mancini FR, Boutron-Ruault M-C. Dietary inflammatory index, risk of incident hypertension, and effect modification from BMI. Nutr J. 2020;19(62):1-8.https://doi.org/10.1186/s12937-020-00577-1.
10. Barbaresko J, Koch M, Schulze MB, Nöthlings U. Dietary pattern analysis and biomarkers of low-grade inflammation: a systematic literature review. Nutr Rev. 2013;71(8):511-27. https://doi.org/10.1111/nure.12035. .
11. Shivappa N, Hébert JR, Rietzschel ER, De Buyzere ML, Langlois M, Debruyne E, et al. Associations between dietary inflammatory index and inflammatory markers in the Asklepios Study. Br J Nutr. 2015;113(4):665-71.https://doi.org/10.1017/S000711451400395X. .
12. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in

21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. The lancet. 2012;380(9859):2224-60.https://doi.org/10.1016/S0140-6736(12)61766-8.

13. Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. Nat Rev Endocrinol. 2018;14(2):88.https://doi.org/10.1038/nrendo.2017.151.
14. Poustchi H, Eghtesad S, Kamangar F, Etemadi A, Keshtkar A-A, Hekmatdoost A, et al. Prospective epidemiological research studies in Iran (the PERSIAN Cohort Study): rationale, objectives, and design. Am J Epidemiol. 2018;187(4):647-55.https://doi.org/10.1093/aje/kwx314.
15. Pasdar Y, Najafi F, Moradinazar M, Shakiba E, Karim H, Hamzeh B, et al. Cohort profile: Ravansar Non-Communicable Disease cohort study: the first cohort study in a Kurdish population. Int J Epidemiol. 2019;48(3):682-83.https://doi.org/10.1093/ije/dyy296.
16. Najafi F, Pasdar Y, Hamzeh B, Rezaei S, Nazar MM, Soofi M. Measuring and decomposing socioeconomic inequalities in adult obesity in Western Iran. J Prev Med Public Health. 2018;51(6):289-97.https://doi.org/10.3961/jpmph.18.062. .
17. Shivappa N, Steck SE, Hurley TG, Hussey JR, Hébert JR. Designing and developing a literature-derived, population-based dietary inflammatory index. Public Health Nutr. 2014;17(8):1689-96.
18. Ruiz-Canela M, Bes-Rastrollo M, Martínez-González MA. The role of dietary inflammatory index in cardiovascular disease, metabolic syndrome and mortality. Int J Mol Sci. 2016;17(8):1265.
19. Rajati F, Hamzeh B, Pasdar Y, Safari R, Moradinazar M, Shakiba E, et al. Prevalence, awareness, treatment, and control of hypertension and their determinants: Results from the first cohort of non-communicable diseases in a Kurdish settlement. Sci Rep. 2019;9(1):1-10.https://doi.org/.1038/s41598-019-8232-y.
20. Safari-Faramani R, Rajati F, Tavakol K, Hamzeh B, Pasdar Y, Moradinazar M, et al. Prevalence, awareness, treatment, control, and the associated factors of diabetes in an Iranian Kurdish population. J Diabetes Res. 2019;2019(https://doi.org/10.1155/2019/5869206.).
21. Laouali N, Mancini FR, Hajji-Louati M, El Fatouhi D, Balkau B, Boutron-Ruault M-C, et al. Dietary inflammatory index and type 2 diabetes risk in a prospective cohort of 70,991 women followed for 20 years: the mediating role of BMI. Diabetologia. 2019;62(12):2222-32.https://doi.org/10.1007/s00125-019-4972-0.
22. Denova-Gutiérrez E, Muñoz-Aguirre P, Shivappa N, Hébert JR, Tolentino-Mayo L, Batis C, et al. Dietary inflammatory index and type 2 diabetes mellitus in adults: the diabetes mellitus survey of Mexico City. nutrients. 2018;10(4):385.https://doi.org/10.3390/nu10040385.
23. Vissers LE, Waller M, van der Schouw YT, Hebert JR, Shivappa N, Schoenaker D, et al. A pro-inflammatory diet is associated with increased risk of developing hypertension among middle-aged women. Nutr Metab Cardiovasc Dis. 2017;27(6):564-70.https://doi.org/10.1016/j.numecd.2017.03.005.
24. Herder C, Peltonen M, Koenig W, Sütfels K, Lindström J, Martin S, et al. Anti-inflammatory effect of lifestyle changes in the Finnish Diabetes Prevention Study. Diabetologia. 2009;52(3):433-42.https://doi.org/10.1007/s00125-008-1243-1.

25. Fogarty AW, Glancy C, Jones S, Lewis SA, McKeever TM, Britton JR. A prospective study of weight change and systemic inflammation over 9 y. Am J Clin Nutr 2008;87(1):30-5.https://doi.org/10.1093/ajcn/87.1.30.
26. Koloverou E, Esposito K, Giugliano D, Panagiotakos D. The effect of Mediterranean diet on the development of type 2 diabetes mellitus: a meta-analysis of 10 prospective studies and 136,846 participants. Metabolism. 2014;63(7):903-11.https://doi.org/10.1016/j.metabol.2014.04.010.
27. Zern T, Wood R, Greene C, West K, Liu Y, Aggarwal D, et al. Grape polyphenols lower plasma lipids and apolipoproteins associated with increased risk for cardiovascular disease in pre and post-menopausal women. J Nutr. 2005;135:1911-47.https://doi.org/10.093/jn/135.8.
28. Puglisi MJ, Vaishnav U, Shrestha S, Torres-Gonzalez M, Wood RJ, Volek JS, et al. Raisins and additional walking have distinct effects on plasma lipids and inflammatory cytokines. Lipids Health Dis. 2008;7(1):1-9. https://doi.org/10.1186/476-511X-7-14.
29. Liese AD, Weis KE, Schulz M, Tooze JA. Food intake patterns associated with incident type 2 diabetes: the Insulin Resistance Atherosclerosis Study. Diabetes care. 2009;32(2):263-68.https://doi.org/10.2337/dc08-1325.
30. Fang M, Feng L-J. Association between dietary pattern and the risk of type 2 diabetes mellitus in Zhejiang Province, China: A case-control study. Asia Pac J Clin Nutr. 2020;29(4):821-26.https://doi.org/10.6133/apjcn.202012_29(4).0018.
31. Maiorino MI, Bellastella G, Petrizzo M, Scappaticcio L, Giugliano D, Esposito K. Mediterranean diet cools down the inflammatory milieu in type 2 diabetes: the MÉDITA randomized controlled trial. Endocrine. 2016;54(3):634-41.https://doi.org/10.1007/s12020-016-0881-1.
32. Schulze MB, Hu FB. Primary prevention of diabetes: what can be done and how much can be prevented? Annu Rev Public Health. 2005;26:445-67. https://doi.org/10.1146/annurev.publhealth.26.021304.144532.
33. Cox A, Agarwal S, M Herrington D, Carr J, Freedman B, Bowden D. C-reactive protein concentration predicts mortality in type 2 diabetes: the Diabetes Heart Study. Diabet Med 2012;29(6):767-70. https://doi.org/10.1111/j.464-5491.2011.03560.x.
34. Krishnan SM, Sobey CG, Latz E, Mansell A, Drummond GR. IL-1β and IL-18: inflammatory markers or mediators of hypertension? Br J Pharmacol. 2014;171(24):5589-602. https://doi.org/10.1111/bph.12876.
35. Pioli MR, de Faria AP. Pro-inflammatory cytokines and resistant hypertension: potential for novel treatments? Curr Hypertens Rep. 2019;21(12):1-8. https://doi.org/10.1007/s11906-019-1003-2.

Figures

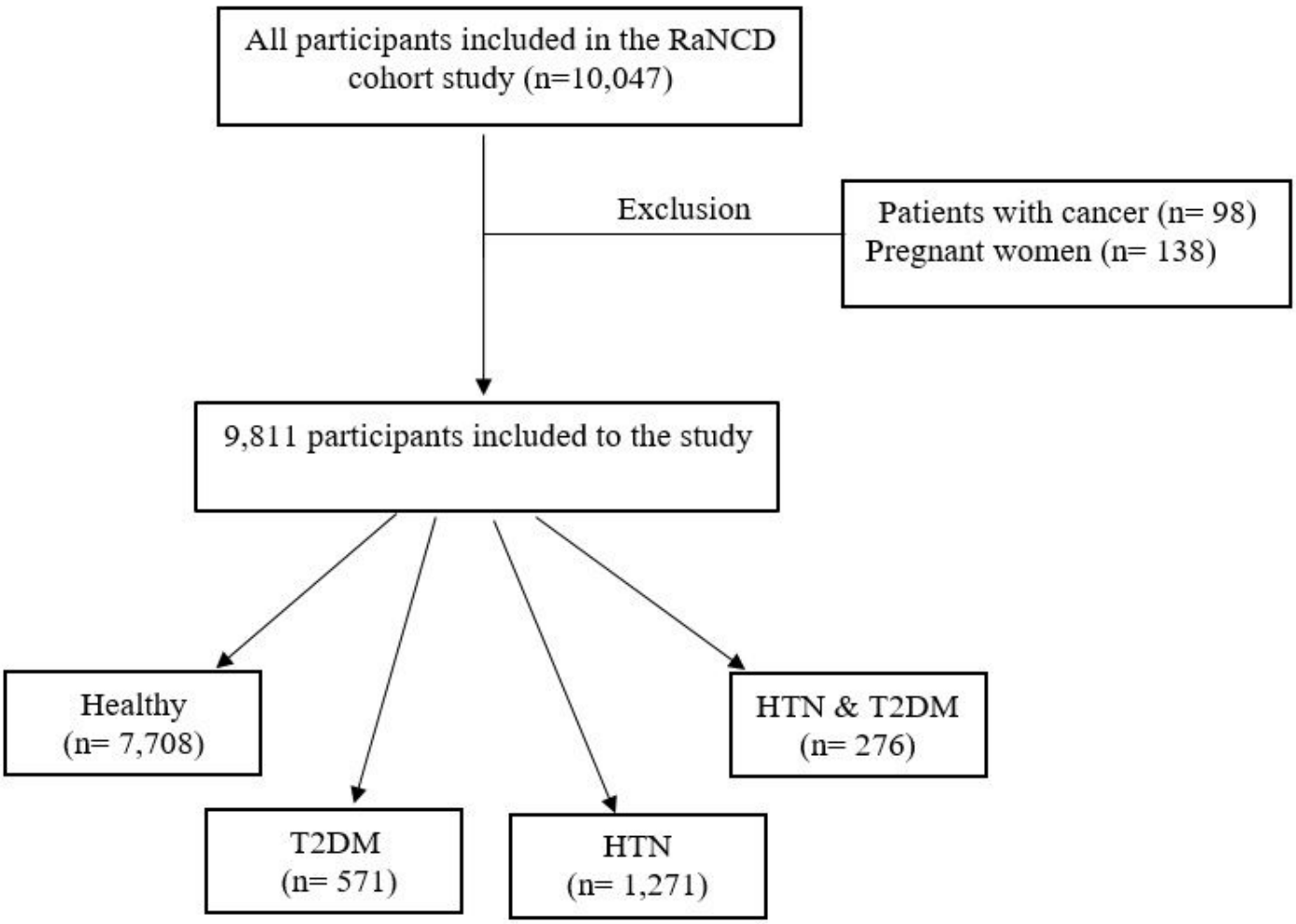

Figure 1

Flow chart of the cross-sectional study in the RaNCD cohort

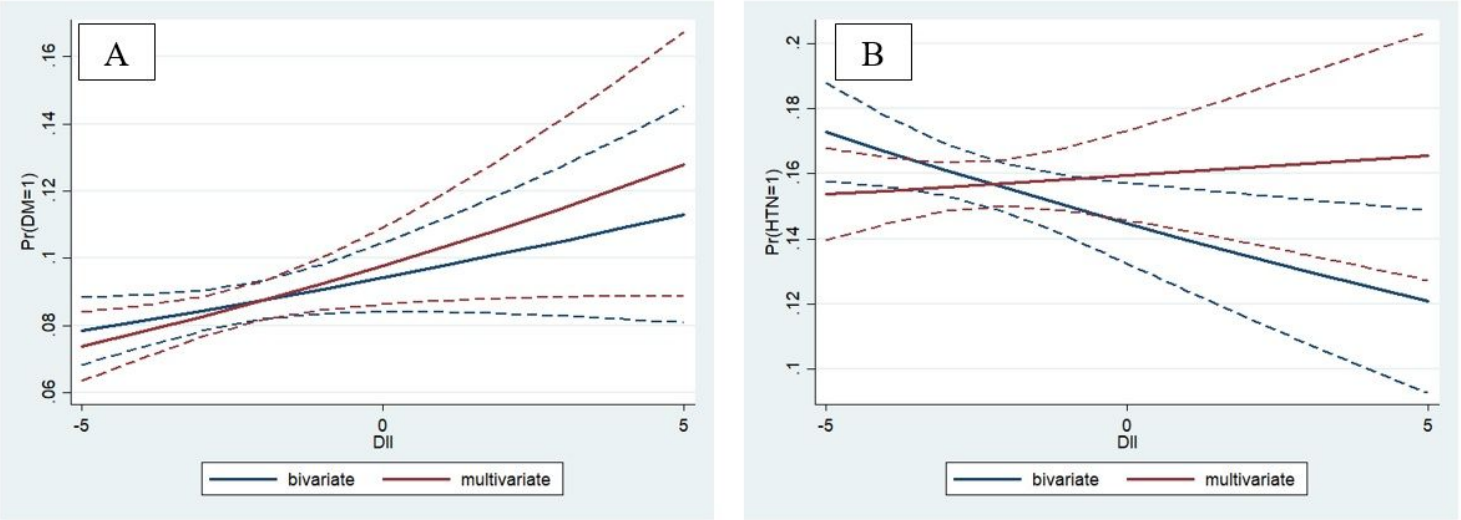

Figure 2

The association between dietary inflammatory index with T2DM (A) and hypertension (B)