Original Article

The dietary inflammatory index and metabolic health of population-based Chinese elderly

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Background and Objectives: This study evaluated the relationship between dietary inflammatory index (DII) and metabolic health in the Chinese elderly. **Methods and Study Design:** A total of 6,730 participants from the "Community-based Cardiovascular and Health Promotion Study" (CoCHPS) cohort were included in this study. The DII scores were acquired using a validated 125-item food-frequency questionnaire (FFQ) (ranged −5.84 to 3.90). The correlation of DII with metabolic health indexes was evaluated with logistic regression and multivariable cox regression using SPSS and R software. **Results:** Compared with low DII scores, subjects in the highest DII score quartiles had higher odds of metabolic dysfunction (OR=1.36, 95% CI: 1.07–1.68, *p* trend=0.023). In the subgroup analyses, the effect of a pro-inflammatory diet on metabolic dysfunction was particularly evident among people with hyperglycaemia (HR=1.58, 95% CI: 1.35–2.14), hypertension (HR=1.48, 95% CI: 1.07–2.09), dyslipidemia (HR=1.45, 95% CI: 1.24–1.87), abdominal obesity (HR=2.16, 95% CI: 1.57–2.96), and ≥60 years old (HR=1.32, 95% CI: 1.04–1.56) or who were women (HR=1.35, 95% CI: 1.08–1.67). **Conclusions:** DII score was associated with metabolic health. Further studies are needed to deepen our understanding of dietary parameters and different populations.

Key Words: dietary inflammatory index, elderly, risk, metabolic health, China

INTRODUCTION

Metabolic dysfunction, an intermediate state that ultimately leads to diabetes and cardiovascular disease (CVD), imposes a major burden on the health care system.[1] The prevalence of metabolic dysfunction varies from 13.4% to 70.0% worldwide and appears to be increasing.[2] According to the 2017 National Cardiovascular Disease Report, the prevalence of metabolic dysfunction in China was estimated to be 33.9% in 2010.[3] Several studies have shown that metabolic health is an important factor in the progression of CVD in the elderly in both developing and developed countries.[4,5] The mechanistic pathway of metabolic health can best be explained by the interaction between behavioral, genetic, and environmental changes in contemporary society (Figure 1).[6]

Recent evidence has shown that chronic inflammation, a natural pathophysiological response to tissue damage and environmental stimuli, plays a major role in the development of metabolic dysfunction and CVD.[7] Multiple factors such as residence, smoking, medication history, and diet contribute to the development of inflammation.[8] Diet is a modifiable factor that can be used to regulate inflammatory-related diseases.[7] DII is a dietary index that evaluates the pro- and anti-inflammatory properties of the diet,[9] and has been associated with inflammatory cytokine concentrations.[10] This index was created in 2009 and updated in 2014. To date, there are few articles reporting the association between metabolic health and DII score. A recent study performed in France confirmed that individuals with higher DII scores showed a 39% increased risk of metabolic dysfunction compared to those with lower DII scores (OR: 1.39; 95% CI: 1.01, 1.92).[11]

In this survey, we calculated the DII score using 26 food parameters and assessed the association between DII and the development of metabolic dysfunction and its components in the Chinese elderly.

METHODS

Subjects

The study population included participants from the CoCHPS cohort, which began in 2015 and is currently ongoing. Eligible subjects in the CoCHPS study were 45–74 years old, and the purpose of the study was to analyze the effects of chronic non-communicable diseases in Bengbu (a city in the Anhui Province in China). In the current study, 9,477 participants with available baseline data were included (Figure 2), and the inclusion criteria were: 1) aged 45–74 years; 2) a minimum stay of 6

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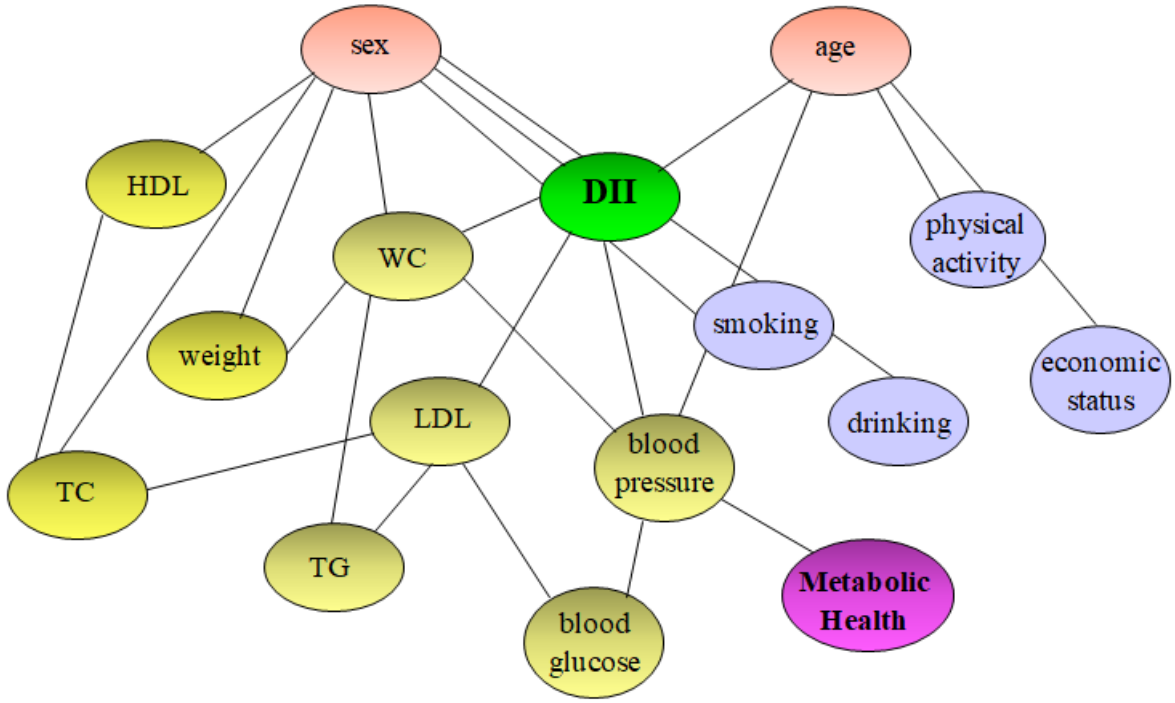

Figure 1. The mechanistic pathway of metabolic health.

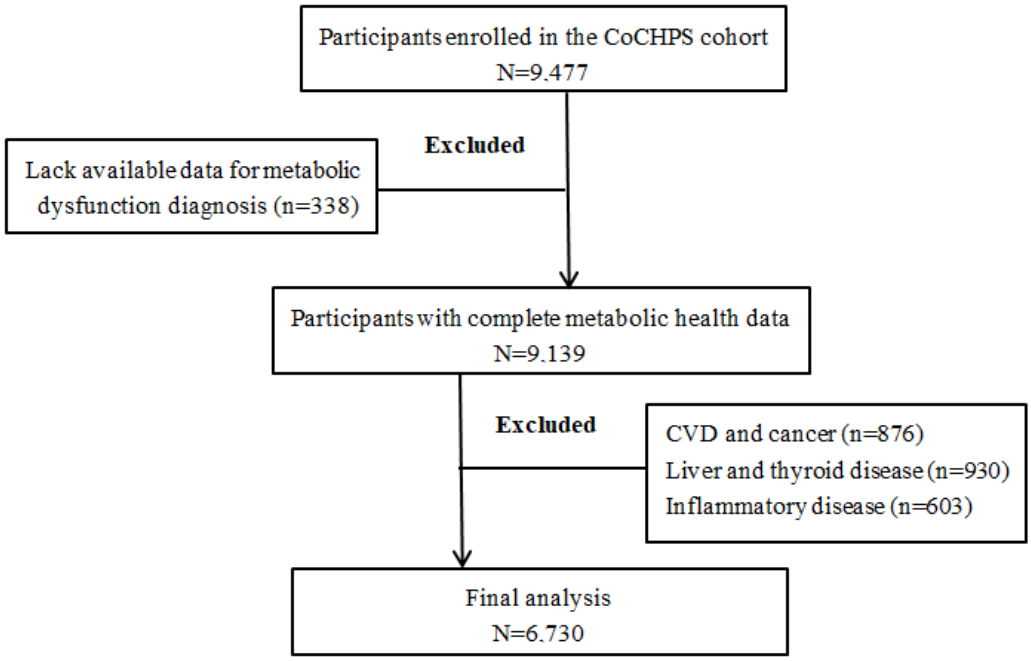

Figure 2. Flowchart of study selection in the CoCHPS Cohort Study.

months in Bengbu; 3) willing to participate in the survey. Oral and written consent was obtained from all participants. This study was conducted according to the Declaration of Helsinki and approved by the Ethics Committee of Bengbu Medical College (No. BBMC-H-2021-098), and written informed consent was obtained from all subjects.

DII

We used a FFQ, which is confirmed to be validated to evaluate food consumption. The FFQ included 125 food items and assessed food intake during the previous 12 months. The food consumption responses ranged from "almost never" to "more than 4–5 times per day." Food intake reported by the participants was estimated with the help of investigators and diet models (Shanghai Gongrong Medical Science and Technology Co., Ltd). The nutrient values were calculated based on reported average food consumption using the Nutrition Calculator software (v2.65).[12]

The DII is a recently developed index used to quantify the inflammatory potential of a daily diet; it was first published in 2009[13] and was updated in 2014.[14] To acquire the original DII, we first obtained dietary intakes of each food item from the FFQ, and for the analyses, 26 parameters out of 45 foods and nutrients were used in the analy-

sis, including energy, protein, carbohydrate, total fat, SFA, MUFA, PUFA, cholesterol, fiber, alcohol, coffee, tea, vitamin A, β-carotene, vitamin D, vitamin E, thiamin, riboflavin, niacin, vitamin B-6, vitamin B-12, vitamin C, iron, zinc, magnesium, and selenium. Then, we used the representative world database to acquire the standardized food parameter intake and converted it to a z-score, which was calculated by subtracting the means from the amount reported by participants and dividing this dietary parameter value by its SD. To reduce the right skewing influence, the z-scores were converted to percentile scores by doubling them and subtracting 1 to obtain a symmetrical distribution centered on 0. Finally, we summed all the DII scores to create the total DII score. The DII score in our study ranged from −5.84 to 3.90.

Covariates

Demographic information was obtained using a self-report questionnaire directed by trained interviewers. The characteristics included age, sex, residence, education level (elementary school or below, secondary school or above), smoking (yes or no), sleep duration (hours per day), and physical activity (METs-min per day).

Body mass index (BMI) was acquired using accurately-measured weight (kg)/height (m^2); we used a cut-off value of 28 kg/m2 to diagnose obesity in China.[15] The metabolic equivalent (MET) value was calculated to acquire the physical activity level of the participants.[16]

Definition of metabolic dysfunction

Metabolic dysfunction was defined as being present when individuals met ≥3 of the NCEP ATP III criteria: 1) waist circumference (WC) ≥90 cm in men and ≥80 cm in women, 2) systolic blood pressure (SBP) ≥130 mmHg or diastolic blood pressure (DBP) ≥85 mmHg, 3) fasting plasma glucose (FPG) ≥110 mg/dL, 4) triglycerides (TG) ≥150 mg/dL, 5) high-density lipoprotein cholesterol (HDL-C) <40 mg/dL in men and <50 mg/dL in women.[17]

Statistical analysis

Statistical analyses were performed with IBM SPSS Statistics ver. 22.0 software and R version 3.4.4 software. We transformed the DII into quartiles (Q), and the participants were divided into four groups based on their DII scores. Continuous variables (age, sleep duration, physical activity, and BMI) were presented as mean ± SD, and categorical variables (sex, residence, smoking, and education) were presented as percentages. Multivariate logistic regression analysis was performed to identify associations between DII quartiles and metabolic health and its components. Subgroup analysis of metabolic dysfunction and the DII score was performed with R 3.4.4 (R package forest plot).

RESULTS

The baseline characteristics of the 6,730 participants (men= 2,774; women=3,956) included in the study are presented in Table 1. The DII score ranged from −5.84 to 3.90. According to the DII score quartile categories, individuals in the highly anti-inflammatory diet group (Q1) had a longer sleep duration (p<0.001), more physical activity (p<0.001), and lower education (p<0.001), and were rural dwellers (p=0.02) compared to subjects with highly pro-inflammatory diets (Q4). Participants with the highest DII score showed a higher prevalence of metabolic dysfunction (p=0.02 in men and p=0.03 in women).

The food parameters that contributed to the calculation of the DII score are shown in Table 2. Participants with the highest quartile of DII score (Q4) reported a higher intake of energy, total fat, MUFA, cholesterol, vitamin D, riboflavin, vitamin B-6, vitamin B-12, iron, meat, poultry, eggs, and alcohol, while they reported a lower intake of fiber, grain, and vegetables than participants with the lowest quartile of DII score (Q1) (p<0.05).

Concerning the components of metabolic dysfunction in the study population, individuals with highly anti-inflammatory diets (Q1) had a lower WC (85.4 vs 88.3 cm, p trend=0.04), lower SBP (125 vs 127 mmHg, p trend=0.02), lower FPG (93.2 vs 98.3 mg/dL, p trend=0.001), lower TG (124 vs 140 mg/dL, p trend <0.001), and higher HDL-C (45.2 vs 42.3 mg/dL, p trend=0.04) compared with individuals with highly pro-inflammatory diets (Q4) (Table 3).

The relationship between overall metabolic health and

Table 1. Basic characteristics of the study subjects[†]

	Quartiles of DII[‡]				p for Trend
	Q1 (n=1683) (-5.84, -1.53)	Q2 (n=1682) (-1.89, -0.23)	Q3 (n=1683) (-0.05, 1.38)	Q4 (n=1682) (1.73, 3.90)	
Men, %	41.8	40.7	39.6	42.8	0.26
Age, y	62.3±7.2	63.6±8.4	62.7±7.5	62.9±8.7	0.12
Residence (urban), %	52.3	49.4	54.8	53.2	0.02
Smoking (current smoker), %	15.3	14.7	13.2	14.2	0.36
Education, %					<0.001
Elementary school/below	58.7	55.3	51.6	49.3	
Secondary school/above	41.3	44.7	48.4	50.7	
Sleep duration, h	8.3±2.5	8.1±2.0	7.8±2.1	7.9±1.9	<0.001
Physical activity, METs-min/d	330±214	317±207	270±183	254±180	<0.001
BMI, kg/m^2	23.8±3.7	24.4±2.6	25.0±2.8	24.3±3.1	0.56
Metabolic dysfunction, %					
Men	13.7	16.6	17.4	19.7	0.02
Women	21.7	23.5	25.7	27.2	0.03

[†]DII is presented by quartiles (Q1 to Q4).
[‡]ANOVA test was used to continuous variables, chi-square test was used to categorical variables.

Table 2. Food intake data according to quartiles of DII score †

Food parameter	Quartiles of DII ‡				*p* for trend‡
	Q_1 (-5.84, -1.53)	Q_2 (-1.89, -0.23)	Q_3 (-0.05, 1.38)	Q_4 (1.73, 3.90)	
Energy (kcal/d)	1896.5±763.4	1942.3±576.2	2014.8±679.2	2086.5±784.5	<0.001
Nutrients					
Protein (g/d)	79.3±16.4	82.2±17.2	76.5±15.9	81.7±16.3	*0.055*
Carbohydrate (g/d)	276.3±72.4	235.8±64.3	221.5±65.7	197.6±59.4	*0.064*
Total fat (g/d)	65.3±19.4	76.8±22.5	81.2±23.2	93.7±28.3	*<0.001*
SFA (g/d)	16.3±6.7	18.4±7.2	21.0±8.3	19.7±7.9	*0.094*
MUFA (g/d)	38.2±14.5	33.1±13.2	45.7±16.8	39.4±17.3	*<0.001*
PUFA (g/d)	11.3±3.4	14.6±4.2	15.7±4.8	14.8±3.5	*0.110*
Cholesterol (mg/d)	267.4±85.3	295.6±96.4	313.2±102.7	347.8±105.6	*0.009*
Fiber (g/d)	23.2±4.6	20.7±3.9	18.4±4.8	19.3±4.3	*0.002*
Vitamin A (μg/d)	660.5±312.6	705.4±387.2	754.1±376.2	760.3±368.5	*0.123*
β-carotene (μg/d)	2305.4±756.2	1876.3±584.2	1543.2±513.6	1669.3±540.2	*0.328*
Vitamin D (μg/d)	3.4±1.3	3.9±1.8	4.7±2.1	5.0±2.5	*<0.001*
Vitamin E (mg/d)	6.4±1.9	7.8±2.5	8.5±3.0	7.6±2.8	*0.155*
Thiamin (mg/d)	1.9±0.9	1.8±0.9	1.9±0.8	1.7±0.9	*0.585*
Riboflavin (mg/d)	1.5±0.5	1.6±0.4	1.9±0.8	1.8±0.7	*0.004*
Niacin (mg/d)	23.4±5.6	26.8±6.5	29.5±6.7	28.6±7.0	*0.141*
Vitamin B-6 (mg/d)	1.6±0.4	1.8±0.5	2.0±0.4	1.8±0.5	*0.009*
Vitamin B-12 (μg/d)	6.8±2.3	7.5±2.9	8.7±3.2	7.6±3.0	*<0.001*
Vitamin C (mg/d)	176.3±45.2	164.7±48.5	129.8±41.6	145.7±51.0	*0.082*
Iron (mg/d)	10.3±2.1	12.4±2.5	13.6±3.7	14.2±3.8	*0.006*
Zinc (mg/d)	8.7±2.3	9.1±2.5	9.8±3.4	9.0±3.2	*0.605*
Magnesium (mg/d)	310.4±41.7	287.6±39.8	277.4±47.6	305.6±50.4	*0.835*
Selenium (μg/d)	67.4±15.3	72.4±20.6	83.4±23.6	76.5±21.7	*0.319*
Daily key foods					
Grain, rice and cereal	421.3±85.7	387.6±89.3	342.5±64.2	317.8±71.3	*0.012*
Vegetables (g/d)	350.5±102.5	307.8±85.7	274.3±96.4	237.2±87.6	*0.001*
Fruit (g/d)	223.4±54.7	206.7±56.8	175.3±47.3	192.6±46.2	*0.089*
Meat (g/d)	45.9±18.3	59.5±18.7	74.0±23.8	109.9±34.3	*<0.001*
Poultry (g/d)	27.8±9.5	36.4±8.6	34.0±10.4	48.9±13.6	*0.005*
Fish (g/d)	44.5±14.2	32.6±12.5	29.3±10.8	49.6±15.7	*0.073*
Eggs (g/d)	46.7±16.7	51.4±17.8	77.9±25.9	63.5±18.4	*0.021*
Dairy (mL/d)	97.0±25.3	75.0±24.2	120.0±38.7	143.0±43.7	*0.074*
Alcohol (mL/d)	15.3±10.6	17.4±12.3	21.2±14.8	27.4±16.9	*<0.001*
Coffee (mL/d)	23.2±15.4	18.9±12.6	20.7±13.5	18.8±13.3	*0.084*
Tea (g/d)	8.7±3.2	10.6±4.1	7.9±3.5	11.4±4.8	*0.195*

†Food intake data were energy-adjusted.
‡*p* values were estimated by ANOVA.

Table 3. Components of metabolic dysfunction characteristics according to quartiles categories of DII score

	Quartiles of DII				*p* for trend
	Q_1 (-5.84, -1.53)	Q_2 (-1.89, -0.23)	Q_3 (-0.05, 1.38)	Q_4 (1.73, 3.90)	
WC, cm	85.4±9.7	86.3±10.2	87.1±11.4	88.3±10.5	0.04
SBP, mmHg	125±10.4	124±9.3	126±8.9	127±9.5	0.02
DBP, mmHg	76.1±7.3	75.3±7.5	76.3±8.4	77.2±8.1	0.22
FPG, mg/dL	93.2±8.9	94.5±9.2	92.7±8.6	98.3±9.5	0.001
TG, mg/dL	124±12.3	133±11.5	130±11.6	140±11.4	<0.001
HDL-C, mg/dL	45.2±5.3	44.5±5.9	44.1±6.6	42.3±5.7	0.04

DII quartile is shown in Table 4. After adjustment for several covariates, the risk of metabolic dysfunction increased by 1.36-fold (OR=1.36, 95% CI: 1.07–1.68, *p* trend=0.023) in individuals with highly pro-inflammatory diets. Moreover, among the components of metabolic health, highly pro-inflammatory diets were associated with hyperglycaemia (OR=1.38, 95% CI: 1.12–1.82, *p* trend=0.019), dyslipidemia (OR=1.32, 95% CI: 1.14–1.67, *p* trend=0.017), and abdominal obesity (OR=2.14, 95% CI: 1.36–4.05, *p* trend <0.001). However, we did not observe a positive association between highly pro-inflammatory diets and hypertension.

The associations between metabolic dysfunction risk and the DII score, which was based on categories of several variables, are shown in Figure 3. The results of the interaction terms indicated that dyslipidemia (*p*=0.024) and abdominal obesity (p=0.013) were statistically significant in the multivariable models. Stronger associations with metabolic dysfunction were evident when comparing DII Q4 to Q1 in participants aged ≥60 years (HR 1.32, 95% CI: 1.04–1.56), women (HR 1.35, 95% CI: 1.08–1.67), hyperglycaemia (HR 1.58, 95% CI: 1.35–2.14), hypertension (HR 1.48, 95% CI: 1.07–2.09), dyslipidemia (HR 1.45, 95% CI: 1.24–1.87), and abdominal obesity (HR 2.16, 95% CI: 1.57–2.96).

DISCUSSION

A paper describing the DII construct process was first published in 2009,[13] and an improved scoring system and comparative nutrient database was produced in 2014.[14]

Table 4. Association of quartiles of the DII score with metabolic dysfunction and its components

	DII				*p* for trend
	Q1 †	Q2	Q3	Q4	
Metabolic dysfunction					
Model I ‡	1.00	1.03 (0.78-1.26)	0.99 (0.78-1.20)	1.05 (0.87-1.28)	0.368
Model II §	1.00	1.21 (1.03-1.49)	1.32 (1.09-1.60)	1.52 (1.20-1.91)	<0.001
Model III ¶	1.00	1.15 (0.92-1.37)	1.24 (1.01-1.47)	1.36 (1.07-1.68)	0.023
Hyperglycaemia					
Model I	1.00	0.96 (0.75-1.27)	1.04 (0.93-1.18)	1.12 (0.97-1.31)	0.274
Model II	1.00	1.10 (0.84-1.43)	1.28 (1.01-1.68)	1.45 (1.13-1.84)	<0.001
Model III	1.00	1.13 (0.89-1.39)	1.18 (0.97-1.54)	1.38 (1.12-1.82)	0.019
Hypertension					
Model I	1.00	1.12 (0.79-1.43)	1.02 (0.85-1.20)	1.10 (0.92-1.29)	0.254
Model II	1.00	1.22 (0.83-1.65)	1.34 (0.93-1.85)	1.65 (1.23-2.37)	0.012
Model III	1.00	1.13 (0.76-1.62)	1.21 (0.81-1.68)	1.47 (0.98-2.14)	0.076
Dyslipidemia					
Model I	1.00	1.06 (0.96-1.18)	1.21 (1.03-1.38)	1.34 (1.12-1.57)	<0.001
Model II	1.00	1.33 (1.07-1.59)	1.28 (1.04-1.73)	1.40 (1.12-1.84)	<0.001
Model III	1.00	1.21 (1.03-1.42)	1.24 (1.09-1.53)	1.32 (1.14-1.67)	0.017
Abdominal obesity					
Model I	1.00	1.13 (0.97-1.35)	0.95 (0.79-1.16)	1.22 (1.01-1.49)	0.031
Model II	1.00	1.17 (1.04-1.43)	1.28 (1.12-1.51)	1.43 (1.24-1.67)	<0.001
Model III	1.00	1.34 (0.87-2.12)	1.57 (1.28-2.23)	2.14 (1.36-3.75)	<0.001

†Referent group.
‡Unadjusted.
§Model II adjustment for sex, age, residence, smoking, education, sleep duration and physical activity.
¶Model III adjustment per model II covariates plus BMI, energy intake.

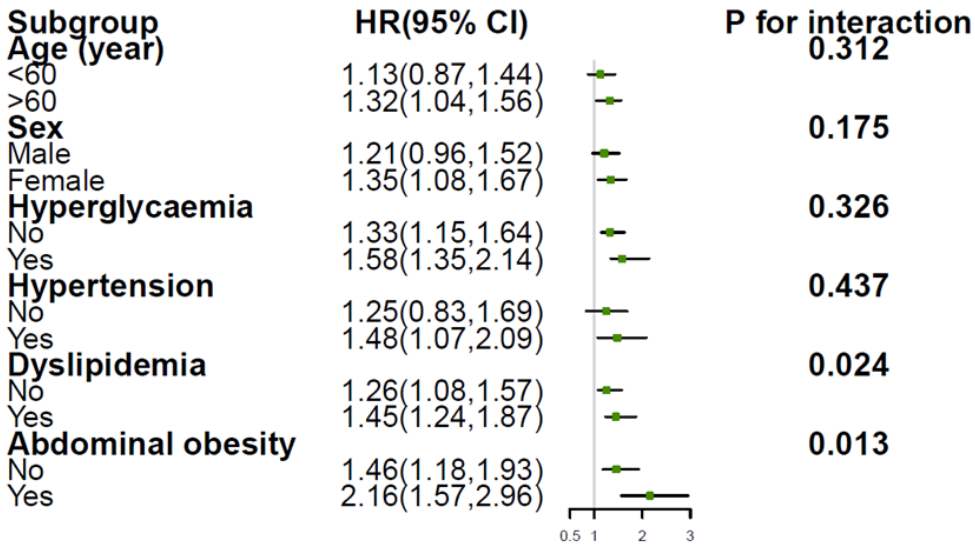

Figure 3. Subgroups analysis. The quartiles of adherence to the DII and metabolic dysfunction. Adjustment for residence, sex, age, smoking, education, sleep duration, physical activity, BMI and energy intake. The interactions were calculated by the log-likelihood ratio test.

The DII is an index used to reflect an individual's anti- and pro-inflammatory dietary potential, and can predict concentrations of inflammatory biomarkers such as IL-6, TNF-α-R2, homocysteine, and WBC in blood lipids.[18-20] Therefore, it can be suggested that the DII score may be a suitable nutritional index to evaluate the immune response and ultimately lead to dietary changes to reduce the risk of chronic non-communicable diseases.[21,22]

This study found that high DII score was associated with the incidence of metabolic dysfunction in participants of the community cohort study from eastern China. Compared with subjects in the most anti-inflammatory diet group, individuals in the most pro-inflammatory diet group had a 36% higher risk of developing metabolic dysfunction. This result is consistent with other prospective cohort studies. A cohort study conducted by Neufcourt et al. found that compared with the lowest DII score group, the highest DII score group had a 39% higher risk of metabolic dysfunction.[23] The Health Workers Cohort Study, conducted by Canto-Osorio F et al. in Mexico, found that subjects in the highest DII quartile were 1.99 times more likely to have developed metabolic dysfunction at the 13-year follow-up.[24]

Our study also showed that low WC, SBP, FPG, and TG and high HDL-C were associated with a low DII score. Increasing numbers of studies have demonstrated the association between components of metabolic health and DII. In Iran, Ariya M et al[25] conducted a cohort study in an Iranian population and found a higher concentration of HDL-C in people with an anti-inflammatory DII

(OR=0.99, 95% CI: 0.98–0.99). A study conducted in China by Ren et al[26] found an association between DII and high BP risk (OR=1.40, 95% CI: 1.03–1.89). In Ireland, Phillips CM et al[27] demonstrated that subjects with higher energy-adjusted DII scores had lower HDL-C, and higher TG and FPG. The authors concluded that the potential mechanism of the inflammatory diet, which influences health, may be that a high-inflammatory diet may increase TG reserves in adipose tissue, and that individuals with higher DII scores consumed more SFA and fewer antioxidants, leading to redox imbalance and a low inflammatory response.[28,29]

In addition, we found that the effect of a pro-inflammatory diet on metabolic health was more evident among people with hyperglycaemia, hypertension, dyslipidemia, abdominal obesity, ≥60 years old, and women. The interaction analysis explained the biological significance of these factors. This result is in accordance with reported cross-sectional studies. A cohort study conducted in China found that a healthy eating pattern was correlated with a reduced risk of metabolic dysfunction.[5] The current study acquired enough information to study the association between DII and metabolic dysfunction. Future studies should examine different population bases such as those with lower socioeconomic status and education level.

Conclusions

In conclusion, components of metabolic dysfunction such as WC, SBP, FPG, TG, and HDL-C showed a significant association with DII, and subjects in the most pro-inflammatory diet group had a 36% higher risk of metabolic dysfunction. This effect was more evident among those with chronic non-communicable diseases. Further studies are needed to deepen our understanding of other dietary parameters and different population bases.

AUTHOR DISCLOSURES

All of the authors declare that they have no conflicts of interest.

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REFERENCES

1. Dalvand S, Niksima SH, Meshkani R, Ghanei Gheshlagh R, Sadegh-Nejadi S, Kooti W, Parizad N, Zahednezhad H, Afrisham R. Prevalence of metabolic syndrome among Iranian population: a systematic review and meta-analysis. Iran J Public Health. 2017;46:456-7.
2. Kaur J. A comprehensive review on metabolic syndrome. Cardiol Res Pract. 2014;2014:943162. doi: 10.1155/2014/943162.
3. Chen WW, Gao RL, Liu LS, Zhu ML, Wang W, Wang YJ et al. China cardiovascular diseases report 2015: a summary. J Geriatr Cardiol. 2017;14:1-10. doi: 10.11909/j.issn.1671-5411.2017.01.012.
4. Saklayen MG. The global epidemic of the metabolic syndrome. Curr Hypertens Rep. 2018;20:12. doi: 10.1007/s11906-018-0812-z.
5. Li R, Zhan W, Huang X, Zhang Z, Zhou M, Bao W, Li Q, Ma Y. Association of dietary inflammatory index and metabolic syndrome in the elderly over 55 years in Northern China. Br J Nutr. 2021;1-8. doi: 10.1017/S0007114521004207.
6. Shin D, Kongpakpaisarn K, Bohra C. Trends in the prevalence of metabolic syndrome and its components in the United States 2007-2014. Int J Cardiol. 2018;259:216-9. doi: 10.1016/j.ijcard.2018.01.139.
7. Minihane AM, Vinoy S, Russell WR, Baka A, Roche HM, Tuohy KM et al. Low-grade inflammation, diet composition and health: current research evidence and its translation. Br J Nutr. 2015;114:999-1012. doi: 10.1017/S0007114515002093.
8. Naja F, Shivappa N, Nasreddine L, Kharroubi S, Itani L, Hwalla N, Mehio Sibai A, Hebert JR. Role of inflammation in the association between the western dietary pattern and metabolic syndrome among Lebanese adults. Int J Food Sci Nutr. 2017;68:997-1004. doi: 10.1080/09637486.2017.1312297.
9. Shivappa N, Godos J, Hébert JR, Wirth MD, Piuri G, Speciani AF, Grosso G. Dietary inflammatory index and cardiovascular risk and mortality-a meta-analysis. Nutrients. 2018;10:200. doi: 10.3390/nu10020200.
10. Laouali N, Mancini FR, Hajji-Louati M, El Fatouhi D, Balkau B, Boutron-Ruault MC, Bonnet F, Fagherazzi G. 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;62:2222-32. doi: 10.1007/s00125-019-04972-0.
11. Neufcourt L, Assmann KE, Fezeu LK, Touvier M, Graffouillère L, Shivappa N et al. Prospective association between the dietary inflammatory index and metabolic syndrome: findings from the SU.VI.MAX study. Nutr Metab Cardiovasc Dis. 2015;25:988-96. doi: 10.1016/j.numecd.2015.09.002.
12. Chinese Nutrition Society. Chinese dietary reference intakes (2013). Beijing: Science Press; 2014.
13. Cavicchia PP, Steck SE, Hurley TG, Hussey JR, Ma Y, Ockene IS, Hébert JR. A new dietary inflammatory index predicts interval changes in serum high-sensitivity C-reactive protein. J Nutr. 2009;139:2365-72. doi: 10.3945/jn.109.114025.
14. 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;17:1689-96. doi: 10.1017/S1368980013002115.
15. National Health and Family Planning Commission of the People's Republic of China Criteria of Weight for Adults, WS/T 428-2013. Beijing: Standards Press of China; 2013.
16. Ainsworth BE, Haskell WL, Whitt MC, Irwin ML, Swartz AM, Strath SJ et al. Compendium of physical activities: an update of activity codes and MET intensities. Med Sci Sport Exerc. 2000;32:S498-504. doi: 10.1097/00005768-200009001-00009.
17. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation. 2009;120:1640-5. doi: 10.1161/ CIRCULATIONAHA.109.192644.
18. Phillips CM, Chen LW, Heude B, Bernard JY, Harvey NC, Duijts L et al. Dietary inflammatory index and non-

communicable disease risk: a narrative review. Nutrients. 2019;11:1873. doi: 10.3390/nu11081873.
19. Adriaensen W, Matheï C, Vaes B, van Pottelbergh G, Wallemacq P, Degryse JM. Interleukin-6 as a first-rated serum inflammatory marker to predict mortality and hospitalization in the oldest old: a regression and CART approach in the BELFRAIL study. Exp Gerontol. 2015;69: 53-61. doi: 10.1016/j.exger.2015.06.005.
20. Shivappa N, Steck SE, Hurley TG, Hussey JR, Ma Y, Ockene IS, Tabung F, Hébert JR. A population-based dietary inflammatory index predicts levels of C-reactive protein in the Seasonal Variation of Blood Cholesterol Study (SEASONS). Public Health Nutr. 2014;17:1825-33. doi: 10.1017/S1368980013002565.
21. Wirth MD, Hébert JR, Shivappa N, Hand GA, Hurley TG, Drenowatz C, McMahon D, Shook RP, Blair SN. Anti-inflammatory dietary inflammatory index scores are associated with healthier scores on other dietary indices. Nutr Res. 2016;36:214-9. doi: 10.1016/j.nutres.2015.11.009.
22. San KMM, Fahmida U, Wijaksono F, Lin H, Zaw KK, Htet MK. Chronic low grade inflammatory measured by dietary inflammatory index and its association with obesity among school teachers in Yangon, Myanmar. Asia Pac J Clin Nutr. 2018;27:92-8. doi: 10.6133/apjcn.042017.06.
23. Neufcourt L, Assmann KE, Fezeu LK, Touvier M, Graffouillère L, Shivappa N et al. Prospective association between the dietary inflammatory index and metabolic syndrome: findings from the SU.VI.MAX study. Nutr Metab Cardiovasc Dis. 2015;25:988-96. doi: 10.1016/j.numecd. 2015.09.002.
24. Canto-Osorio F, Denova-Gutierrez E, Sánchez-Romero LM, Salmerón J, Barrientos-Gutierrez T. Dietary inflammatory index and metabolic syndrome in Mexican adult population. Am J Clin Nutr. 2020;112:373-80. doi: 10.1093/ajcn/nqaa135.
25. Ariya M, Shahraki HR, Farjam M, Ehrampoush E, Bahramali E, Homayounfar R, Shivappa N, Hebert JR. Dietary inflammatory index and metabolic syndrome in Iranian population (Fasa Persian Cohort Study). Sci Rep. 2020;10:16762. doi: 10.1038/s41598-020-73844-0.
26. Ren Z, Zhao A, Wang Y, Meng L, Szeto IM, Li T, Gong H, Tian Z, Zhang Y, Wang P. Association between dietary inflammatory index, C-reactive protein and metabolic syndrome: a cross-sectional study. Nutrients. 2018;10:831. doi: 10.3390/nu10070831.
27. Phillips CM, Shivappa N, Hébert JR, Perry IJ. Dietary inflammatory index and biomarkers of lipoprotein metabolism, inflammation and glucose homeostasis in adults. Nutrients. 2018;10:1033. doi: 10.3390/nu10081033.
28. Santos S, Oliveira A, Lopes C. Systematic review of saturated fatty acids on inflammation and circulating levels of adipokines. Nutr Res. 2013;33:687-95. doi: 10.1016/j.nutres.2013.07.002.
29. Amakye WK, Zhang Z, Wei Y, Shivappa N, Hebert JR, Wang J, Su Y, Mao L. The relationship between dietary inflammatory index (DII) and muscle mass and strength in Chinese children aged 6-9 years. Asia Pac J Clin Nutr. 2018; 27:1315-24. doi: 10.6133/apjcn.201811_27(6).0019.