

Dietary inflammatory index is associated with lipid profile and interleukin-6 in postmenopausal women: A cross-sectional study

Zahra Aslani

Fereydoun Siassi

Mostafa Qorbani

Nitin Shivappa

James R Hébert

Fariba Koohdani

Mehdi Hedayati

Maryam Abshirini

Mahshid Soleymani

Gity Sotoudeh (✉ gsotodeh@tums.ac.ir)

Tehran University of Medical Sciences <https://orcid.org/0000-0001-6541-2581>

Research

Keywords: Diet; Inflammation; Triglycerides; Glucose; Cholesterol; Interleukin-6

Posted Date: February 19th, 2020

DOI: <https://doi.org/10.21203/rs.2.23968/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Abstract

Background: The prevalence of diseases associated with chronic inflammation is higher in postmenopausal women. The dietary inflammatory index (DII[®]) was developed to evaluate the diet-associated inflammation. The aim of this study was to evaluate the association between the DII score and levels of fasting blood sugar (FBS), lipid profile, and inflammatory biomarkers.

Methods: This cross-sectional study was conducted on 175 postmenopausal women referred to the southern health centers and health clinics affiliated to the southern municipality of Tehran, Iran. The DII score was calculated using dietary intakes derived from a validated food frequency questionnaire (FFQ). Concentrations of FBS, triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), high sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), interleukin-1 β (IL-1 β), and tumor necrosis factor- α (TNF- α) were determined. Using linear regression models, we evaluated the association between the DII score and blood biomarkers.

Results: After adjustment for covariates, the highest DII score category was associated with higher TG value ($b_{DII3vs1} = 0.08$, $p=0.03$), TG/HDL-C ratio ($b_{DII3vs1} = 0.10$, $p=0.04$), and IL-6 concentration ($b_{DII3vs1} = 0.06$, $p=0.01$) compared to the first category of the DII score.

Conclusions: We found a positive association between pro-inflammatory diet and unfavorable biomarkers in postmenopausal women.

Background

Inflammation is a tissue response to infections and injuries, resulting in wound healing and the restoration of tissue [1–4]. Chronic inflammation is resulted from repeated stress factors such as obesity and poor diet [5] and it is determined by persistent, enhanced levels of serum inflammatory biomarkers including high sensitivity C-reactive protein (hs-CRP), interleukin (IL)-6 and tumor necrosis factor- α (TNF- α). This state is associated with chronic conditions like type 2 diabetes mellitus, cardiovascular diseases (CVDs), cancer and obesity [6–13]. Several studies have shown that the levels of inflammatory biomarkers increases with aging [14–19]. During menopause, the levels of serum cytokines such as IL-8, monocyte chemoattractant protein-1 (MCP-1), and TNF- α are increased [20]. These changes may adversely affect the function of endothelial cells which in turn leads to CVDs [20]. Furthermore, due to increased levels of inflammation, disturbance in metabolism of glucose such as insulin resistance may occur in postmenopausal women [21].

Nutrition is considered to be an important modulating factor for chronic inflammation during the life cycle [22]. Numerous dietary components affect inflammatory status [23]. For example, high intake of olive oil, plant foods, fish and whole grains and moderate consumption of alcohol, and low intake of red meat, saturated fat and sugar (Mediterranean-style diet) are associated with low levels of plasma inflammatory biomarkers [23–26]. On the contrary, greater adherence to the Western dietary pattern (high consumption of red meat, high-fat dairy product, and refined grains) is associated with increased level of

chronic inflammation [27, 28]. Several dietary factors such as magnesium, β -carotene, vitamin E, vitamin C, omega-3 polyunsaturated fatty acids, and fiber have shown to decrease the level of inflammation [29, 30].

Hebert et al. designed a new index to quantify the diet-associated inflammation. The Dietary inflammatory index (DII®) was developed based on review and scoring of the peer-reviewed literature on the effect of dietary factors (macronutrients, micronutrients, specific food items, spices, energy intake, and flavonoids) on six inflammatory biomarkers [31]. The first construct validation, against hs-CRP, was conducted in the SEASONS study [32]. Subsequently, construct validations have examined the association between DII score and serum levels of hs-CRP, IL-6, and TNF- α in several populations [33–36]. In addition, multiple studies demonstrated the positive link between adherence to pro-inflammatory diet (the higher DII score) and levels of serum glucose, as well as, the association between the DII score and serum levels of lipid profile [37–40]. Previous studies have shown that there is the association between the DII score and different chronic diseases such as obesity [41], metabolic syndrome (MetS) [42], cancers [35, 43–45], and CVDs [46–48]. In Iran, DII has been shown to be associated with various cancers [44, 49–51] and cataract [52] and specifically among Iranian women, a positive association has been observed between higher DII scores and spontaneous abortion [36], gestational diabetes mellitus [53] and decreased bone mineral density among postmenopausal women [54].

Given that there is a high prevalence of cardio-metabolic disorders in postmenopausal women, and absence of any studies investigating the association between the DII score and cardio-metabolic risk factors in Iranian postmenopausal women; therefore, this study aimed to investigate the association between the DII score and fasting blood sugar (FBS), lipid profile, and inflammatory biomarkers levels in these women.

Methods

This cross-sectional study was conducted on enrolled 175 postmenopausal women living in southern regions of Tehran, Iran. The sampling method was as follows: First, two out of six southern regions in Tehran were selected using random sampling. Then 10 urban health centers affiliated to Tehran University of Medical Sciences (TUMS) and 10 health clinics affiliated to the southern municipality of Tehran were randomly chosen from these two regions. The study was conducted on 175 postmenopausal women, aged 40–76 years, who attended urban health centers and health clinics. Participants were postmenopausal women with at least one year elapsed since natural menopause. Postmenopausal women with body mass index (BMI) ≥ 40 kg/m², certain current chronic diseases such as diabetes, cancer, multiple sclerosis, thyroid diseases, and dementia, individuals with hormone replacement therapy for the past six months, and smoking at least once a week were not enrolled into the study. The study was conducted from September 2016 to September 2017. The study protocol was approved by the ethics committee of TUMS. The research objectives were explained to the participants and both verbal and written informed consent were obtained from all participants.

Demographic Information And Socioeconomic Status (ses)

Demographic variables including age, education level, marital status, current chronic diseases (CVDs, endocrine and metabolic, and musculoskeletal diseases), and intake of drugs and dietary supplements were assessed. SES was determined using the number of items of life. These items included personal home or car, freezer, computer, television with flat screen, handmade carpet, dishwasher, villa or additional home, and microwave. Participants with ≤ 3 items were considered low SES; and those with 4 to 6 items were moderate SES; and ≥ 7 items were high SES, respectively [55]. All data were collected using the face-to-face interview.

Anthropometric And Physical Activity (pa) Measurement

Participant's weight was measured using digital scales (Seca725 GmbH & Co. Hamburg, Germany) to the nearest 0.1 kg with light clothing and without shoes. Height was assessed to the nearest 0.5 cm using a non-elastic tape measure while the participants were in a standing position, without shoes, and their shoulders were against the tape measure. BMI was calculated based on weight and height [BMI = weight (kg)/ height (m)²]. Waist circumference (WC) was measured using a non-elastic tape to the nearest 0.5 cm in the middle of the iliac crest and the lowest rib while participants were standing and breathing out (i.e., expiring).

PA was evaluated by International Physical Activity Questionnaire (IPAQ) short form [56]. This questionnaire is used to measure the levels of PA of individuals in the past seven days and includes seven items for assessing walking, vigorous and moderate PA. For appraising the total exercise per week, the frequency of each PA was multiplied by duration of it and then, the obtained value for each activity was multiplied by the metabolic equivalent (MET) of it. Finally, all obtained values were summed to calculate MET-minute per week as the total PA per week [57].

Dietary Intake And Dietary Inflammatory Index (dii®)

Usual dietary intake of participants was measured using a validated 147-item semiquantitative food frequency questionnaire (FFQ) which contained specific portion sizes [58]. The frequency of consumption of each food item in the past year on periods of daily, weekly, monthly, and yearly was asked. The reported frequency for intake of each food substance was changed to daily frequency of consumption for it, and then the amount of daily intake for each food substance was obtained using portion sizes. We applied Nutritionist 4 software (First Databank; Hearst, San Bruno, CA, USA) for extracting of the contents of energy and nutrients of food items comprising the USDA food composition data. Iranian food composition table (IFCT) was used for traditional Iranian food substances analyses that were not contain in this database [59]. Turmeric and thyme were added to FFQ. All FFQs were completed by three trained nutritionists by face-to-face interview.

The DII® is a validated and updated meta-analytic tool that quantifies the inflammatory potential of diet from maximally anti-inflammatory to maximally pro-inflammatory. The procedure used to develop this index has been described in detail elsewhere [31]. Briefly, nearly 2000 relevant papers evaluating the effect of 45 dietary factors on IL-1 β , IL-4, IL-6, IL-10, TNF- α , and CRP were reviewed and scored, with + 1 assigned to dietary factors with pro-inflammatory effects, -1 assigned to anti-inflammatory dietary items, and 0 assigned to dietary factors without any effect on the inflammatory biomarkers.

At first, to measure the DII score for each individual, the amount reported for dietary factors (i.e., macronutrients, micronutrients, food items, spices, and flavonoids) initially was linked to the regionally representative global database that provided a robust estimate of a mean and standard deviation for each of them. To obtain the z-score, we subtracted the value of global mean intake of each dietary factor from reported amount consumed. We then divided this number by world standard deviation. Global daily mean intake and standard deviation for each dietary factor were obtained via datasets of 11 countries (i.e., USA, Australia, Bahrain, Denmark, India, Japan, New Zealand, Taiwan, South Korea, Mexico, and UK). To minimize the effect of 'right skewing', the dietary factor-specific z-scores were converted to a proportion (i.e., with values from 0 to 1). Afterwards, to create a symmetrical distribution with a range of -1 to + 1 and centered on 0, the achieved value for each dietary factor was multiplied by 2 and then 1 was subtracted from it. The resulting value for each dietary factor was multiplied by overall dietary factor-specific inflammatory effect score to achieve dietary factor-specific DII score. Finally, all dietary factor-specific DII scores were summed to calculate the DII score for each participant. The final range of the DII score is from - 8.87 to 7.98, and lower value of the DII score indicates the lower dietary inflammation and higher DII score shows the higher dietary inflammation.

In the current study, we estimated the DII score using 34 dietary factors. The pro-inflammatory constituents contained energy, carbohydrate, protein, total fat, cholesterol, saturated fatty acids (SFA), vitamin B12, and iron and the anti-inflammatory components consisted of monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), n-3 and n-6 fatty acids, fiber, folic acid, niacin, riboflavin, thiamin, vitamin A, vitamin C, vitamin D, vitamin E, vitamin B6, zinc, selenium, magnesium, β -carotene, caffeine, ginger, turmeric, thyme, garlic, onion, pepper and black tea. The other constituents of DII score (i.e., alcohol, eugenol, anthocyanidins, flavan-3-ol, flavones, flavonols, flavonones, isoflavones, trans fat, saffron, and rosemary) were not used in the DII score calculation because the associated data was not available as part of the FFQ. To control the effect of total energy intake, all dietary factors were adjusted for energy via the residual method [60] and then used for the DII calculation. In the current study, none of participants consumed fortified foods and the amount of intake of nutrient supplement was not clear, so supplement intakes of nutrients were not included in the DII score calculation.

Laboratory Measurements

To evaluate blood biomarkers, after 12–14 hours of fasting, 10 ml of venous blood was collected in EDTA free tube by a trained nurse. Blood samples were centrifuged (Behdad Universal Centrifuge, Iran) at

3000 rpm for 10 min. The serum levels of FBS and lipid profile were evaluated on the same day of collecting blood samples and the remaining serum samples were stored in -80 °C for assessment of inflammatory biomarkers. FBS was measured by the enzymatic colorimetric method using glucose oxidase (Pars Azmoon, Tehran, Iran). Triacylglycerol (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) concentrations were evaluated by enzymatic colorimetric method (Pars Azmoon, Tehran, Iran) using cholesterol esterase, cholesterol oxidase, and glycerol phosphate oxidase. FBS and lipid profile were assessed using autoanalyzer (BT 1500, Biotechnica Instruments, Italy). (TG/HDL-C ratio was calculated based on serum levels of TG and HDL-C [TG/HDL-C = TG (mg/dl)/ HDL-C (mg/dl)]).

The serum levels of hs-CRP was determined using German ZellBio kit with intra- and inter-assay coefficient of variation (CVs) of < 9.9% and < 12%, respectively. The concentrations of IL-6, IL-1 β , and TNF- α were quantified using Diaclone kit (made in France). Intra- and inter-assay CVs for IL-6, IL-1 β and TNF- α were 4.2% and 7.7%, 4.5% and 8.7%, and 3.3% and 9.0%, respectively. All inflammatory biomarkers were assessed using enzyme-linked immunosorbent assay (ELISA) method.

Statistical analysis

Initially, normal distribution of variables was examined using Kolmogorov-Smirnov test. TG, TG/HDL-C ratio, IL-1 β and IL-6 did not have normal distribution. Thus, these parameters were logarithm (log) transformed. The DII score was considered as the categorical (tertile) and continuous variables in the analyses. To illustrate quantitative variables according to the tertile of DII variable, means \pm standard deviations (SDs) or median (interquartile range (IQR)) were used for variables with normal and skewed distribution, respectively. Moreover, the qualitative variables were expressed as the number (percentage). Comparisons of mean values of general characteristics, intake of energy, micro and macronutrients, and food groups with normal distribution across the DII tertiles were made using One-way analysis of variance (ANOVA) test. Furthermore, Kruskal-Wallis test was used to compare medians of variables with non-normal distribution. We used χ^2 test to compare the percentage of categorical variables across the DII tertiles. Moreover, linear regression models were used to determine the association between the DII score and FBS, lipid profile and inflammatory biomarkers. To obtain linear trend of blood biomarkers across the DII tertiles, we entered the DII tertile in the regression model as an independent variable. We adjusted no potential covariates in the crude model (model I). Age, educational level, PA, BMI, energy intake, dietary supplement use, lipid-lowering medication, and current chronic diseases for the FBS and lipid profile were adjusted in Model II. Furthermore, for the inflammatory biomarkers in Model II, these covariates were controlled: age, BMI, educational level, PA, energy intake, dietary supplement use, anti-inflammatory and cardiovascular medications, and current chronic diseases. All analyses were performed using SPSS version 20 software package for windows (SPSS Inc., Chicago, USA) and P-value < 0.05 was considered as statistically significant.

Results

This study was conducted on 175 postmenopausal women with mean (\pm SD) age of 56.6 (5.5) years. The DII scores ranged from -3.88 to +3.84 with a mean of +0.35 (SD=1.77). Table 1 shows distribution of the participants' characteristics across the tertiles of DII score. There was no significant trend in the distribution of the participants' characteristics across of the tertiles of DII score except for dietary supplement use ($P=0.01$). With increased adherence of pro-inflammatory diet, the number of participants who consumed dietary supplement decreased.

Table 2 shows the distribution of the intake of energy, micro and macro-nutrients, fiber and food groups across the DII score tertiles. Overall, DII scores were positively associated with higher intake of energy, total fat, SFA, high-fat dairy, hydrogenated fats, carbonated drinks, and refined grains (P for all <0.01) and a lower consumption of carbohydrates, proteins, β -Carotene, vitamin C, and fiber (P for all <0.05).

The association between the DII score and serum levels of FBS and lipid profile is shown in Table 3. The highest tertile of the DII score was associated with higher log of TG concentration compared to participants with anti-inflammatory diet (the lowest tertile of the DII score) in the crude model (Model I) ($b_{DII3vs1}= 0.08$, 95% confidence interval (CI): 0.01, 0.16) and multivariable model (Model II) ($b_{DII3vs1}= 0.08$, 95% CI: 0.008, 0.16). Individuals in the highest DII tertile had a higher log of TG concentration in the crude and multivariable models (P trend for all comparisons <0.05). In addition, individuals in the highest tertile of DII score had a higher log of TG/HDL-C ratio compared with those in the first tertile of DII score in the crude model (Model I) ($b_{DII3vs1}= 0.11$, 95% CI: 0.01, 0.21) and multivariable model (Model II) ($b_{DII3vs1}= 0.10$, 95% CI: 0.003, 0.20). Participants in the highest DII tertile had higher log of TG/HDL-C ratio in the crude and multivariable models (P trend for all comparisons <0.05). We observed no significant association between the DII score and other measured outcomes (FBS, TC, LDL-C, and HDL-C).

The association between the DII score and serum levels of inflammatory biomarkers is shown in Table 4. A significant positive association was observed between the DII score and log of IL-6 concentration in the crude model (Model I) ($b_{DII2vs1}=0.05$, 95% CI: 0.001, 0.11; $b_{DII3vs1}=0.06$, 95% CI: 0.007, 0.11).

Participants in the highest category of the DII score had a greater log of IL-6 value compared to those in the first tertile of the DII after adjustment for potential covariates (Model II) ($b_{DII3vs1}=0.06$, 95% CI: 0.01, 0.12). Individuals in the highest DII tertile had a higher log of IL-6 levels in the crude and multivariable models (P trend for all comparisons <0.05). No significant association was observed between the DII score and other inflammatory biomarkers (hs-CRP, IL-1 β , and TNF- α).

Discussion

This study is the first attempt to assess the relationship between the inflammatory potential of diet (as estimated by the DII score) and FBS, lipid profile, and inflammatory biomarkers in Iranian postmenopausal women. A diet rich in energy, total fat, and SFA (pro-inflammatory dietary factors) and poor in anti-inflammatory dietary factors (β -Carotene, vitamin C, and fiber) can lead to greater DII score which in turn can lead to increased levels of inflammation in individuals as determined by increased

serum levels of IL-6 and higher levels of TG and TG/HDL-C ratio. No significant association was observed between DII score and serum levels of FBS, TC, LDL-C, HDL-C, hs-CRP, IL-1 β , and TNF- α .

Pro-inflammatory diet (i.e., the greater DII score) was associated with higher serum levels of TG. This finding is in line with the study of Neufcourt et al. conducted on 4347 French adults [42]. In addition, an observational study carried out on Iranian adults demonstrated similar findings [37]. Likewise, another research conducted on American adults revealed a direct association between pro-inflammatory diet and the serum levels of TG [39]. These results may be due to an increase in levels of inflammatory biomarkers that stimulate TG production in liver [61-63]. Despite the observed association between the DII score and TG in the mentioned studies, Ren et al failed to find any association between the DII score and serum levels of TG in a national Chinese study conducted on 1,712 participants aged 18-75 years [64]. Similarly, the studies of Naja [65] and Sokol et al. [66] conducted on Lebanese and Polish adults, respectively, did not reveal this association. The absence of the relationship may be due to the smaller number of dietary factors used for the DII score calculation. Moreover, we showed a positive association between the DII score and TG/HDL-C ratio. Higher TG/HDL-C ratio is associated with coronary artery diseases [67]. In line with the current study, the findings of Mazidi study indicated a significant association between the DII score and TG/HDL-C ratio in adults [39].

Our findings were not consistent with our expectation of higher serum levels of TC and LDL-C and lower levels of HDL-C with higher DII scores. Previous studies conducted in Lebanon [65] and China [64] are consistent with our result of HDL-C. An observational study conducted with the aim of determination of association between dietary patterns and serum lipids on Latin America showed a significant inverse association between prudent diet and serum levels of TC and LDL-C [68]. Likewise, another cross-sectional study carried out on Singaporean subjects aged 21 to 94 years showed a significant association between alternative healthy eating index-2010, alternate Mediterranean diet score, and dietary approaches to stop hypertension diet score and selected serum lipids of TC, HDL-C, and LDL-C [69]. Difference in ethnic group and sample size of the studies may explain the different findings from these studies.

TNF- α can cause insulin resistance via several biochemical mechanisms such as increases in circulating free fatty acids [70-72], reduction of glucose transporter (GLUT)-4 transporter in muscle cells [70] and autophosphorylation of insulin receptors [70]. An observational study conducted on adults aged 18 years and older, showed a significant positive association between the increased pro-inflammatory diet and higher serum levels of fasting blood glucose (FBG), which is not in agreement with our results [39], which might be due to the relatively small sample size of the present study. Moreover, in an observational study, Vahid et al. found that individuals in the highest tertile of DII score had higher levels of fasting plasma glucose (FPG) than those in the first tertile [37]. On the contrary, findings of another cross-sectional study showed no significant association between the categorical or continues DII score and FBG [64]. In addition, two studies performed in Lebanon [65] and Poland [66] showed no association between the increased DII score and hyperglycemia. The reported differences between studies may be due to the number of dietary factors applied for the calculation of DII score and differences in sample size as noted.

The synthesis of *17 beta-estradiol* (E2) in ovaries was decreased in menopause [73]. The levels of TNF- α , IL-6 and IL-1 (pro-inflammatory biomarkers) were increased in postmenopausal women following limitation of ovarian function [74]. In addition, the deposition of visceral fat increases in menopause releases the inflammatory cytokines [75]. Various studies demonstrated the higher levels of IL-2, IL-4 and IL-6 in postmenopausal women as compared with premenopausal women [76, 77].

In this study, participants in the highest tertile of DII score had higher serum levels of IL-6 compared to those in the first tertile of DII score. However, we observed no association for hs-CRP. In line with the present study, a cross-sectional study conducted on Belgian adults revealed a significant association between the DII scores and serum levels of IL-6, but not hs-CRP levels [78]. Similarly, Vahid et al. in a case-control study conducted on Iranian women demonstrated a direct association between the DII score and IL-6 levels [36]. In a large observational study carried out with the aim of determining the relationship between the DII score and serum levels of hs-CRP in China, there was a significant association between DII scores and hs-CRP levels, but only in subjects with MetS [64]. Moreover, in a case-control study of gastric cancer in Iran, Vahid et al. showed the positive association between the DII score and hs-CRP [35]. This discrepancy in the findings of the various studies may be due to the observed differences in lifestyles, dietary habits, and races in study participants as well as limitations of sample size with concomitant limitation in statistical power.

The results of the current study on the association between the DII score and serum levels of TNF- α showed no significant relationship in this regard. This lack of association may be due to the relatively small sample size of the current study. The finding is consistent with previous study conducted on the police-officers in Buffalo, New York [40]. On the other hand, two studies conducted on European adolescents [79] and American postmenopausal women [33] showed a positive association between pro-inflammatory diet (as measured by DII score) and serum levels of TNF- α . The discrepancy between the studies may be due to their different sample sizes.

No statistically significant association was addressed between the DII score and serum levels of IL-1 β . By contrast, in an observational study Shivappa et al. showed a significant association between the increased DII score and higher serum levels of IL-1 β [79]. Vahid et al. in a case-control study conducted on subjects with and without gastric cancer found a positive association between the DII score and serum levels of IL-1 β [35]. The discrepancy between results of our study and these studies may be due to difference in participants of the studies.

To the best of our knowledge, this study was the first attempt that investigated the association between the DII score and FBS, lipid profile and inflammatory biomarkers among Iranian postmenopausal women. Using validated dietary and PA assessment tools, the estimation of dietary potential of diet using an updated and validated index, examining the association between the DII score and inflammatory biomarkers applying potential confounders such as the sociodemographic factors and use of anti-inflammatory medications, measuring biochemistry factors of FBS, lipid profile, and inflammatory biomarkers according to standardized procedures are the other strengths of the current study.

The cross-sectional nature of this study is the most important limitation of the current study; therefore, we are unable to elucidate the findings on causality. The population of our study included Iranian postmenopausal women. Thus, we cannot generalize the observed findings to all Iranian people or postmenopausal women in other countries. The FFQ was applied for individual dietary assessment in the current study. So, misclassification and recall bias are prevalent problems existing in most epidemiological studies. Another limitation is the use of an FFQ, known to be subject to a variety of errors, including both random and systematic [80, 81]. Non-availability of 11 dietary factors which may lead to not find any association between the DII score and some biomarkers. However, despite of absence of some dietary factors for the DII score calculation, we could demonstrate some expected associations between the DII score and lipid profile and inflammatory biomarkers.

Conclusion

This study was conducted to investigate the association between the range of inflammation of diet and levels of serum FBS, lipid profile, and inflammatory biomarkers. Our results suggest that higher adherence to anti-inflammatory diet can contribute to modifying inflammatory processes and thus help to improve lipid profile, in form of TG and markers of inflammation, in form of IL-6. Future studies employing prospective designs and meta-analyses combining data across studies to both increase heterogeneity and generalizability and statistical power are recommended.

Abbreviations

ANOVA, Analysis of variance; BMI, Body mass index; CHI, Connecting Health Innovations; CVDs, Cardiovascular diseases; DII, Dietary inflammatory index; E2, 17 beta-estradiol; EDTA, Ethylenediamine tetraacetic acid; ELISA, Enzyme-linked immunosorbent assay; FBG, Fasting blood glucose; FBS, Fasting blood sugar; FFQ, Food frequency questionnaire; FPG, Fasting plasma glucose; GLUT, Glucose transporter; HDL-C, High density lipoprotein-cholesterol; hs-CRP, High sensitivity C-reactive protein; IFCT, Iranian food composition table; IL, Interleukin; IPAQ, International physical activity questionnaire; IQR, Interquartile range; LDL-C, Low density lipoprotein-cholesterol; MCP-1, Monocyte chemoattractant protein-1; MET, Metabolic equivalent; MetS, Metabolic syndrome; MUFA, Monounsaturated fatty acids; PA, Physical activity; PUFA, Polyunsaturated fatty acids; SDs, Standard deviations; SES, Socioeconomic status; SFA, Saturated fatty acids; SPSS, Statistical Package for the Social Sciences; TC, Total cholesterol; TG, Triglyceride; TNF- α , Tumor necrosis factor- α ; TUMS, Tehran University of Medical Sciences; USDA, United States Department of Agriculture; WC, Waist circumference

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee of Tehran University of Medical Sciences. All participants read and signed a written informed consent prior to enrollment.

Consent for publication

Not applicable

Availability of data and materials

Not applicable

Competing interests

None declared

Funding

This study was supported by Tehran University of Medical Sciences (TUMS) under Grants No. 32413-161-02-95 and 32750-61-04-95.

Author Contributions

GS, FS, and FK formulated the research questions and designed the study. ZA, MA and MS carried it out the interviews. MH conducted all the serum biochemistry tests. MQ analyzed the data and ZA, JRH and NS computed the DII score and contributed to writing the article.

Acknowledgment

We express our appreciation to the postmenopausal women for their collaboration in the current study.

Dr. James R. Hébert owns controlling interest in Connecting Health Innovations LLC (CHI), a company planning to license the right to his invention of the dietary inflammatory index (DII) from the University of South Carolina in order to develop computer and smart phone applications for patient counseling and dietary intervention in clinical settings. Dr. Nitin Shivappa is an employee of CHI.

Dr. Shivappa and Dr. Hébert supported the study by grant number R44DK103377 from the United States National Institute of Diabetes and Digestive and Kidney Diseases.

References

1. Keibel A, Singh V, Sharma MC. Inflammation, microenvironment, and the immune system in cancer progression. *Curr Pharm Des.*2009;15:1949-1955. <https://doi.org/10.2174/138161209788453167>.

2. Pan M-H, Lai C-S, Dushenkov S, Ho C-T. Modulation of inflammatory genes by natural dietary bioactive compounds. *J Agric Food Chem*. 2009;57:4467-4477. <https://doi.org/10.1021/jf900612n>.
3. Thun MJ, Henley SJ, Gansler T, editors. *Inflammation and cancer: an epidemiological perspective*, Novartis Foundation symposium. Chichester; New York; John Wiley. 1999
4. Wärnberg J, Gomez-Martinez S, Romeo J, Díaz LE, Marcos A. Nutrition, inflammation, and cognitive function. *Ann N Y Acad Sci*. 2009; 1153:164-175. <https://doi.org/10.1111/j.1749-6632.2008.03985.x>.
5. Libby P. Inflammatory mechanisms: the molecular basis of inflammation and disease. *Nutr Rev* 65:S140-S146. <https://doi.org/10.1301/nr.2007.dec.S140-S146>.
6. Lee, C., Adler, A., Sandhu, M., Sharp, S., Forouhi, N., Erqou, S. et al. Association of C-reactive protein with type 2 diabetes: prospective analysis and meta-analysis. *Diabetologia*, 2009; 52(6), 1040-1047. <https://doi.org/10.1007/s00125-009-1338-3>.
7. Kolb H, Mandrup-Poulsen T. An immune origin of type 2 diabetes?. *Diabetologia*. 2005; 48:1038-1050. <https://doi.org/10.1007/s00125-005-1764-9>.
8. Coussens LM, Werb Z. Inflammation and cancer. *Nature*. 2002; 420:860-867. <https://doi.org/10.1038/nature01322>.
9. Hotamisligil GS. Inflammation and metabolic disorders. *Nature*. 2006; 444:860-867. <https://doi.org/10.1038/nature05485>.
10. Libby P () Inflammation in atherosclerosis. *Nature*. 2002;420:868-874. <https://doi.org/10.1161/ATVBAHA.108.179705>.
11. Mantovani A, Allavena P, Sica A, Balkwill F. Cancer-related inflammation. *Nature*. 2008;454:436-444. <https://doi.org/10.1038/nature07205>.
12. Donath MY, Shoelson SE. Type 2 diabetes as an inflammatory disease. *Nat Rev Immunol*. 2011; 11:98-107. <https://doi.org/10.1038/nri2925>.
13. Gregor MF, Hotamisligil GS. Inflammatory mechanisms in obesity. *Annu Rev Immunol*. 2011; 29:415-445. <https://doi.org/10.1146/annurev-immunol-031210-101322>.
14. Bruunsgaard H, Pedersen M, Pedersen BK. Aging and proinflammatory cytokines. *Curr Opin*. 2001; 8:131-136.
15. Paolisso G, Rizzo MR, Mazziotti G, Tagliamonte MR, Gambardella A, Rotondi M et al Advancing age and insulin resistance: role of plasma tumor necrosis factor- α . *Am J Physiol Endocrinol Metab*. 1998; 275:294-299.
16. Dobbs R, Charlett A, Purkiss A, Dobbs S, Weller C, Peterson D. Association of circulating TNF- α and IL-6 with ageing and parkinsonism. *Acta Neurol Scand*. 1999; 100:34-41.
17. Baggio G, Donazzan S, Monti D, Mari D, Martini S, Gabelli C et al. Lipoprotein (a) and lipoprotein profile in healthy centenarians: a reappraisal of vascular risk factors. *FASEB J*. 1998; 12:433-437.
18. Ballou SP, Lozanski GB, Hodder S, Rzewnicki DL, Mion LC, Sipe JD et al. Quantitative and qualitative alterations of acute-phase proteins in healthy elderly persons. *Age Ageing*. 1996; 25:224-230.

19. Wei J, Xu H, Davies JL, Hemmings GP. Increase of plasma IL-6 concentration with age in healthy subjects. *Life Sci.* 1992;51:1953-1956.
20. Bonetti PO, Lerman LO, Lerman A. Endothelial dysfunction: a marker of atherosclerotic risk. *Arterioscler Thromb Vasc Biol.* 2003; 23:168-175. <https://doi.org/10.1161/01.ATV.0000051384.43104.FC>.
21. Tamakoshi K, Yatsuya H, Wada K, Matsushita K, Otsuka R, Yang PO, et al () The transition to menopause reinforces adiponectin production and its contribution to improvement of insulin-resistant state. *Clin Endocrinol.* 2007; 66:65-71. <https://doi.org/10.1111/j.1365-2265.2006.02687.x>.
22. Calder PC, Albers R, Antoine J-M, Blum S, Bourdet-Sicard R, Ferns G, et al. Inflammatory disease processes and interactions with nutrition. *Br J Nutr.* 2009; 101:1-45. <https://doi.org/10.1017/S0007114509377867>.
23. Estruch R, Martínez-González MA, Corella D, Salas-Salvadó J, Ruiz-Gutiérrez V, Covas MI, et al. Effects of a Mediterranean-style diet on cardiovascular risk factors: a randomized trial. *Ann Intern Med.* 2006;145:1-11. <https://doi.org/10.7326/0003-4819-145-1-200607040-00004>.
24. Ahluwalia N, Andreeva V, Kesse-Guyot E, Hercberg S. Dietary patterns, inflammation and the metabolic syndrome. *Diabetes Metab.* 2013;39:99-110. <https://doi.org/10.1016/j.diabet.2012.08.007>.
25. Casas, R., Sacanella, E., Urpi-Sarda, M., Chiva-Blanch, G., Ros, E., Martínez-González, M.-A, et al. The effects of the mediterranean diet on biomarkers of vascular wall inflammation and plaque vulnerability in subjects with high risk for cardiovascular disease. A randomized trial. *PloS one*, 9. 2014 (<https://doi.org/10.1371/journal.pone.0100084>).
26. Schwingshackl L, Hoffmann G. Mediterranean dietary pattern, inflammation and endothelial function: a systematic review and meta-analysis of intervention trials. *Nutr Metab Cardiovasc Dis.* 2014;24:929-939. <https://doi.org/10.1016/j.numecd.03.003>.
27. Johansson-Persson A, Ulmius M, Cloetens L, Karhu T, Herzig K-H, Önning G. A high intake of dietary fiber influences C-reactive protein and fibrinogen, but not glucose and lipid metabolism, in mildly hypercholesterolemic subjects. *Eur J Nutr.* 2014;53:39-48. <https://doi.org/10.1007/s00394-013-0496-8>.
28. King DE, Egan BM, Geesey ME. Relation of dietary fat and fiber to elevation of C-reactive protein. *Am J Cardiol.* 2003; 92:1335-1339. <https://doi.org/10.1016/j.amjcard.2003.08.020>.
29. Nanri A, Moore MA, Kono S. Impact of C-reactive protein on disease risk and its relation to dietary factors: literature review. *Asian Pac J Cancer Prev.* 2007; 8:167-177.
30. Giugliano D, Ceriello A, Esposito K. The effects of diet on inflammation: emphasis on the metabolic syndrome. *J Am Coll Cardiol.* 2006;48:677-685. <https://doi.org/10.1016/j.jacc.2006.03.052>.
31. 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;17:1689-1696. <https://doi.org/10.1017/S1368980013002115>.

32. Shivappa N, Steck SE, Hurley TG, Hussey JR, Ma Y, Ockene IS et al. 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; 17:1825-1833. <https://doi.org/10.1017/S1368980013002565>.
33. Tabung FK, Steck SE, Zhang J, Ma Y, Liese AD, Agalliu I et al. Construct validation of the dietary inflammatory index among postmenopausal women. *Ann Epidemiol.* 2015; 25:398-405. <https://doi.org/10.1016/j.annepidem.2015.03.009>.
34. Wirth MD, Shivappa N, Davis L, Hurley T, Ortaglia A, Drayton R et al. Construct validation of the dietary inflammatory index among African Americans. *J Nutr Health Aging.* 2017; 21:487-491. <https://doi.org/10.1007/s12603-016-0775-1>.
35. Vahid F, Shivappa N, Faghfoori Z, Khodabakhshi A, Zayeri F, Hebert JR et al. Validation of a dietary inflammatory index (DII) and association with risk of gastric cancer: a case-control study. *Asian Pac J Cancer Prev.* 2018;19:1471-1477. <https://doi.org/10.22034/APJCP.2018.19.6.1471>.
36. Vahid F, Shivappa N, Hekmatdoost A, Hebert JR, Davoodi SH, Sadeghi M. Association between Maternal Dietary Inflammatory Index (DII) and abortion in Iranian women and validation of DII with serum concentration of inflammatory factors: case-control study. *Appl Physiol Nutr Metab.* 2017;42:511-516. <https://doi.org/10.1139/apnm-2016-0274>.
37. Vahid F, Shivappa N, Karamati M, Naeini AJ, Hebert JR, Davoodi SH. Association between Dietary Inflammatory Index (DII) and risk of prediabetes: a case-control study. *Appl Physiol Nutr Metab.* 2016;42:399-404. <https://doi.org/10.1139/apnm-2016-0395>.
38. Phillips C, Shivappa N, Hébert J, Perry I. Dietary Inflammatory Index and Biomarkers of Lipoprotein Metabolism, Inflammation and Glucose Homeostasis in Adults. *Nutrients.* 2018; 10:1033. <https://doi.org/10.3390/nu10081033>.
39. Mazidi M, Shivappa N, Wirth MD, Hebert JR, Mikhailidis DP, Kengne AP et al. Dietary inflammatory index and cardiometabolic risk in US adults. *Atherosclerosis.* 2018;276:23-27. <https://doi.org/10.1016/j.atherosclerosis.2018.02.020>.
40. Wirth M, Burch J, Shivappa N, Violanti JM, Burchfiel CM, Fekedulegn D et al. Association of a dietary inflammatory index with inflammatory indices and the metabolic syndrome among police officers. *J Occup Environ Med.* 2014;56:986-989. <https://doi.org/10.1097/JOM.0000000000000213>.
41. Ruiz-Canela M, Zazpe I, Shivappa N, Hebert JR, Sanchez-Tainta A, Corella D et al. Dietary inflammatory index and anthropometric measures of obesity in a population sample at high cardiovascular risk from the PREDIMED (PREvencion con Dieta MEDiterranea) trial. *Br J Nutr.* 2015;113:984-995. <https://doi.org/10.1017/S0007114514004401>.
42. Neufcourt L, Assmann K, Fezeu L, 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;25:988-996. <https://doi.org/10.1016/j.numecd.2015.09.002>.

43. Shivappa N, Bosetti C, Zucchetto A, Montella M, Serraino D, La Vecchia C et al. Association between dietary inflammatory index and prostate cancer among Italian men. *Br J Nutr*. 2015;113:278-283. <https://doi.org/10.1017/S0007114514003572>.
44. Jalali S, Shivappa N, Hébert JR, Heidari Z, Hekmatdoost A, Rashidkhani B. Dietary Inflammatory Index and Odds of Breast Cancer in a Case-Control Study from Iran. *Nutr Cancer*. 2018;1-9. <https://doi.org/10.1080/01635581.2018.1494843>.
45. Shivappa N, Prizment AE, Blair CK, Jacobs DR, Steck SE, Hébert JR. Dietary inflammatory index and risk of colorectal cancer in the Iowa Women's Health Study. *Cancer Epidemiol Biomarkers Prev*. 2014;23:2383-2392. <https://doi.org/10.1158/1055-9965.EPI-14-0537>.
46. Bodén S, Wennberg M, Van Guelpen B, Johansson I, Lindahl B, Andersson J et al. Dietary inflammatory index and risk of first myocardial infarction; a prospective population-based study. *Nutr J*. 2017;16:21-30. <https://doi.org/10.1186/s12937-017-0243-8>.
47. Garcia-Arellano A, Ramallal R, Ruiz-Canela M, Salas-Salvadó J, Corella D, Shivappa N. Dietary inflammatory index and incidence of cardiovascular disease in the PREDIMED study. *Nutrients*. 2015;7:4124-4138. <https://doi.org/10.3390/nu7064124>.
48. O'Neil A, Shivappa N, Jacka FN, Kotowicz MA, Kibbey K, Hebert JR et al. Pro-inflammatory dietary intake as a risk factor for CVD in men: a 5-year longitudinal study. *Br J Nutr*. 2015;114:2074-2082. <https://doi.org/10.1017/S0007114515003815>.
49. Shivappa N, R Hebert J, E Steck S, Safari A, Sedaghat F, Rashidkhani B. Dietary inflammatory index and odds of colorectal cancer in a case-control study from Iran. *Asian Pac J Cancer Prev*. 2018;19:1999-2006. <https://doi.org/10.22034/APJCP.2018.19.7.1999>.
50. Shivappa N, Hébert JR, Askari F, Kardoust Parizi M, Rashidkhani B. Increased inflammatory potential of diet is associated with increased risk of prostate cancer in Iranian men. *Int J Vitam Nutr Res*. 2017;86:161-168. <https://doi.org/10.1024/0300-9831/a000395>.
51. Shivappa N, Hébert JR, Rashidkhani B. Dietary inflammatory index and risk of esophageal squamous cell cancer in a case-control study from Iran. *Nutr Cancer*. 2015;67:1255-1261. <https://doi.org/10.1080/01635581.2015.1082108>.
52. Shivappa N, Hébert JR, Assadi M, Rashidkhani B, Ghanavati M. Inflammatory Potential of Diet is Associated with Increased Odds of Cataract in a Case-Control Study from Iran. *Int J Vitam Nutr Res*: 2018;1-8. <https://doi.org/10.1024/0300-9831/a000420>.
53. Shivappa N, Hébert JR, Akhoundan M, Mirmiran P, Rashidkhani B. Association between inflammatory potential of diet and odds of gestational diabetes mellitus among Iranian women. *J Matern Fetal Neonatal Med*. 2018;1-7. <https://doi.org/10.1080/14767058.2018.1466275>.
54. Shivappa N, Hébert JR, Karamati M, Shariati-Bafghi S-E, Rashidkhani B. Increased inflammatory potential of diet is associated with bone mineral density among postmenopausal women in Iran. *Eur J Nutr*. 2016;55:561-568. <https://doi.org/10.1007/s00394-015-0875-4>.
55. Daneshi-Maskooni M, Dorosty-Motlagh A, Hosseini SM, Zendehtdel K, Kashani A, Azizi S et al. Prevalence of Food Insecurity and Some Associated Socioeconomic Factors among Patients with

- Upper Gastrointestinal Cancers. Medical journal of mashhad university of medical sciences. 2013;56:56-61.
56. Moghaddam MB, Aghdam FB, Jafarabadi MA, Allahverdipour H, Nikookheslat SD, Safarpour S. The Iranian Version of International Physical Activity Questionnaire (IPAQ) in Iran: content and construct validity, factor structure, internal consistency and stability. World Appl Sci.2012;18:1073-1080. <https://doi.org/10.5829/idosi.wasj.2012.18.08.754>.
57. Committee IR. Guidelines for data processing and analysis of the International Physical Activity Questionnaire (IPAQ)-short and long forms. <http://www.ipaq.ki.se/scoring.pdf>. 2005.
58. Esfahani FH, Asghari G, Mirmiran P, Azizi F. Reproducibility and relative validity of food group intake in a food frequency questionnaire developed for the Tehran Lipid and Glucose Study. J Epidemiol. 2010;20:150-158. <https://doi.org/10.2188/jea.JE20090083>.
59. Azar M, Sarkisian E (1980) Food composition table of Iran: National Nutrition and Food Research Institute, Shaheed Beheshti University, Tehran.
60. Willett WC, Howe GR, Kushi LH. Adjustment for total energy intake in epidemiologic studies. Am J Clin Nutr. 1997;65:1220S-1228S. <https://doi.org/10.1093/ajcn/65.4.1220S>.
61. Feingold KR, Serio MK, Adi S, Moser AH, Grunfeld C. Tumor necrosis factor stimulates hepatic lipid synthesis and secretion. Endocrinology. 1989;124:2336-2342. <https://doi.org/10.1210/endo-124-5-2336>.
62. Feingold KR, Soued M, Serio MK, Moser AH, Dinarello CA, Grunfeld C. Multiple cytokines stimulate hepatic lipid synthesis in vivo. Endocrinology. 1989;125:267-274. <https://doi.org/10.1210/endo-125-1-267>.
63. Grunfeld C, Adi S, Soued M, Moser A, Fiers W, Feingold KR. Search for mediators of the lipogenic effects of tumor necrosis factor: potential role for interleukin 6. Cancer Res. 1990;50:4233-4238.
64. Ren Z, Zhao A, Wang Y, Meng L, Szeto I, Li T et al. Association between Dietary Inflammatory Index, C-Reactive Protein and Metabolic Syndrome: A Cross-Sectional Study. Nutrients. 2018;10:831. <https://doi.org/10.3390/nu10070831>.
65. Naja F, Shivappa N, Nasreddine L, Kharroubi S, Itani L, Hwalla N et al. 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. <https://doi.org/10.1080/09637486.2017.1312297>.
66. Sokol A, Wirth MD, Manczuk M, Shivappa N, Zatonska K, Hurley TG et al. Association between the dietary inflammatory index, waist-to-hip ratio and metabolic syndrome. Nutr Res. 2016;36:1298-1303. <https://doi.org/10.1016/j.nutres.2016.04.004>.
67. da Luz PL, Cesena FH, Favarato D, Cerqueira ES. Comparison of serum lipid values in patients with coronary artery disease at < 50, 50 to 59, 60 to 69, and > 70 years of age. Am J Cardiol. 2005;96:1640-1643. <https://doi.org/10.1016/j.amjcard.2005.07.080>.
68. Poggio R, Elorriaga N, Gutierrez L, Irazola V, Rubinstein A, Danaei G. Associations between dietary patterns and serum lipids, apo and C-reactive protein in an adult population: evidence from a multi-

- city cohort in South America. *Br J Nutr.* 2017;117:548-555.
<https://doi.org/10.1017/S0007114517000514>.
69. Whitton C, Rebello SA, Lee J, Tai ES, van Dam RM. A Healthy Asian A Posteriori Dietary Pattern Correlates with A Priori Dietary Patterns and Is Associated with Cardiovascular Disease Risk Factors in a Multiethnic Asian Population. *J Nutr.* 2018;148:616-623. <https://doi.org/10.1093/jn/nxy016>.
70. Uysal KT, Wiesbrock SM, Marino MW, Hotamisligil GS. Protection from obesity-induced insulin resistance in mice lacking TNF- α function. *Nature.* 1997;389:610-644.
<https://doi.org/10.1038/39335>.
71. Hotamisligil GS, Peraldi P, Budavari A, Ellis R, White MF, Spiegelman BM. IRS-1-mediated inhibition of insulin receptor tyrosine kinase activity in TNF- α -and obesity-induced insulin resistance. *Science.* 1996;271:665-670. <https://doi.org/10.1126/science.271.5249.665>.
72. Van Der Poll T RJ, Endert E, Borm JJJ, Buller HR, Sauerwein HP. Tumor necrosis factor mimics the metabolic response to acute infection in healthy humans. *Am J Physiol.* 1998;24:E457-465.
<https://doi.org/10.1152/ajpendo.1991.261.4.E457>.
73. Pesteei K, Allame M, Amir Khany M, Esmaeel Motlagh M. Clinical guide and executive health program team to provide menopausal services to women 60-45 years. Tehran: Pooneh Publication p. 2008;10-7.
74. Pfeilschifter J, Köditz R, Pfohl M, Schatz H. Changes in proinflammatory cytokine activity after menopause. *Endocr Rev.* 2002;23:90-119. <https://doi.org/10.1210/edrv.23.1.0456>.
75. Kershaw EE, Flier JS (2004) Adipose tissue as an endocrine organ. *J Clin Endocrinol Metab* 89:2548-2556. <https://doi.org/10.1210/jc.2004-0395>.
76. Yasui T, Maegawa M, Tomita J, Miyatani Y, Yamada M, Uemura H et al. Changes in serum cytokine concentrations during the menopausal transition. *Maturitas.* 2007;56:396-403.
<https://doi.org/10.1016/j.maturitas.2006.11.002>.
77. Cioffi M, Esposito K, Vietri MT, Gaggero P, D'Auria A, Ardovino I et al. Cytokine pattern in postmenopause. *Maturitas.* 2002;41:187-192. [https://doi.org/10.1016/S0378-5122\(01\)00286-9](https://doi.org/10.1016/S0378-5122(01)00286-9).
78. 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;113:665-671. <https://doi.org/10.1017/S000711451400395X>.
79. Shivappa N, Hebert JR, Marcos A, Diaz LE, Gomez S, Nova E et al. Association between dietary inflammatory index and inflammatory markers in the HELENA study. *Mol Nutr Food Res.* 2017;61:1600707. <https://doi.org/10.1002/mnfr.201600707>.
80. Hebert JR, Ebbeling CB, Matthews CE, Hurley TG, Yunsheng M, Druker S et al. Systematic errors in middle-aged women's estimates of energy intake: comparing three self-report measures to total energy expenditure from doubly labeled water. *Ann Epidemiol.* 2002;12:577-586.
[https://doi.org/10.1016/S1047-2797\(01\)00297-6](https://doi.org/10.1016/S1047-2797(01)00297-6).
81. Hébert JR, Hurley TG, Steck SE, Miller DR, Tabung FK, Peterson KE et al. Considering the value of dietary assessment data in informing nutrition-related health policy. *Adv Nutr.* 2014;5:447-455.

Tables

Due to technical limitations, Tables 1 - 4 are only available for download from the Supplementary Files section.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Tables14.pdf](#)
- [Tables14.pdf](#)