Original Article

The Associate between Energy-Adjusted Dietary Inflammatory Index and Periodontal Disease: A Cross-Sectional Study

Abstract

Background: Previous studies have shown that the dietary inflammatory index (DII) is associated with different health outcomes. However, few studies have investigated the relationship between DII and oral health. We aimed to assess the association between energy-adjusted dietary inflammatory index (E-DII) and periodontal disease. **Methods:** This cross-sectional study was conducted in 2019 on 213 Iranian participants. E-DII was calculated based on the data obtained by the food frequency questionnaire. The plaque control index was measured by the O'Leary method. Gingivitis was evaluated based on the signs of inflammation in the gums. The Decayed, Missing, and Filled Teeth (DMFT) are based on the number of decayed, filled, and missed teeth resulting from caries. **Results:** the score of E-DII among subjects in the last tertile of the E-DII was ≥ -0.33/. Therefore, participants of the present study did not consume a high proinflammatory diet. No significant correlation between E-DII and dental plaque control index (r = 0.046, $P = 0.518$), gingival index (r = 0.001, $P = 0.998$), and DMFT (r = -0.021, $P = 0.762$) was observed. **Conclusions:** In this study, inflammatory diet and plaque and gingival index were not associated; it should also be investigated in prospective studies.

Keywords: *Gingivitis, inflammation, oral health, periodontitis*

Hanie Hosseini[1*], Mohammad Javad Askari[2*], Hossein Rouhani[1], Mohammad Hossein Nikbakht[3], Farinaz Shirban[4], Mohammad Bagherniya[1,5], Gholamreza Askari[1,5]

[1]Nutrition and Food Security Research Center and Department of Community Nutrition, School of Nutrition and Food Science, Isfahan University of Medical Sciences, Isfahan, [2]School of Dentistry, Khorasgan Islamic Azad University, Isfahan, [3]School of Dentistry, Isfahan University of Medical Sciences, Isfahan, [4]Department of Orthodontics, Dental Research Center, Dental Research Institute, School of Dentistry, Isfahan University of Medical Sciences, Isfahan, [5]Anesthesia and Critical Care Research Center, Isfahan University of Medical Sciences, Isfahan, Iran

**Hanie Hosseini and Mohammad Javad Askari have equal contribution as co-first authors*

Address for correspondence:
Dr. Gholamreza Askari, Department of Community Nutrition, School of Nutrition and Food Science, Isfahan University of Medical Sciences, Isfahan, Iran.
E-mail: askari@mui.ac.ir

Access this article online

Website: www.ijpvmjournal.net/www.ijpm.ir

DOI: 10.4103/ijpvm.ijpvm_355_22

Quick Response Code:

Introduction

Gingivitis is a chronic disease of the periodontal tissue that eventually leads to tooth loss.[1,2] The prevalence of gingivitis in adults is 10 to 60%.[3,4] Aging, male gender, low level of education, smoking, and the presence of metabolic disorders are risk factors for gingivitis.[5,6] Additionally, inflammation has an important role in the pathogenesis of gingivitis.[7] A previous study showed that the prevalence of gingivitis among Iranian children aged 6 to 9 years was 97% (in 6–7 years), 98.1% (7-8 years), and 98.5% (in 8-9 years).[8] Periodontal disease is associated with an increase in the production of reactive oxygen species, which, if not sufficiently buffered, will cause damage to the host's cells and tissues.[9] There is a direct association between periodontitis (e.g., gingivitis) and inflammatory biomarkers including C-reactive protein (CRP), interleukin-6 (IL), and tumor necrosis factor-α (TNF-α).[10] Evidence shows that dietary intakes play an important role in regulating chronic inflammation.[11,12] Several nutrients are associated with a reduction in inflammatory biomarkers involved in the periodontal diseases. Antioxidant nutrients, for example, beta-carotene, ascorbic acid (vitamin C) and alpha-tocopherol (vitamin E), important buffers are reactive oxygen species and they are found in vegetables, fruits, seeds, and grains.[8] Dietary Inflammatory Index (DII) is a population-based, literature-derived dietary index developed to predict anti-inflammatory and proinflammatory effects of a diet.[13] Previous studies have shown a significant positive association between DII and inflammatory biomarkers such as CRP, IL-6, and TNF-α, homocysteine, and fibrinogen. Evidence reported that anti-inflammatory dietary components were inversely associated with periodontitis.[13-15] However, proinflammatory nutrients such as saturated fatty acids increase the risk of periodontal disease.[16] Although the association between gingivitis and some individual nutrients was assessed in previous studies, evidence regarding the relationship between gingivitis and DII is limited. Therefore, we aimed to assess the

How to cite this article: Hosseini H, Askari MJ, Rouhani H, Nikbakht MH, Shirban F, Bagherniya M, *et al.* The associate between energy-adjusted dietary inflammatory index and periodontal disease: A cross-sectional study. Int J Prev Med 2024;15:58.

association between DII and indices of gingivitis among healthy adult subjects.

Methods

The present cross-sectional study was conducted on a sample of the Iranian adult population in 2019. Individuals referring to dental clinics in Isfahan were screened for inclusion and exclusion criteria. Adult subjects (≥18 years old) were included. Following exclusion criteria were considered: (1) being pregnant and breastfeeding; (2) using anti-inflammatory drugs or supplements; (3) smoking or alcohol consumption; (4) chronic inflammatory diseases including diabetes, cardiovascular, rheumatoid arthritis; (5) not responding to more than 39 items of dietary questionnaire; and (6) under- or over-reporting of energy intake (less than 800 and more than 4,200).

Sample size

We used the following equation to calculate the required sample size:

$$n = \frac{(1-r)^2 (Z_1 + Z_2)^2}{r^2} + 2$$

Type 1 error was 0.05, and type 2 error was 0.20 ($Z_1 = 1.96$ and $Z_2 = 0.84$). The correlation coefficient between the energy-adjusted dietary inflammatory index (E-DII) and periodontal index was 0.2. Therefore, the required sample size was at least 190 subjects.

Dietary assessment and calculation of E-DII

Usual food intake of individuals was assessed using a validated and reliable semi-quantitative food frequency questionnaire including 168 food items by a face-to-face interview.[17] Subjects were asked to report the frequency of consumption of each food item according to the amount consumed the previous year. Dietary intakes were converted to energy and macro/micro nutrients using Nutritionist 4 software. The E-DII is a novel dietary index estimated from 45 nutrients and dietary components to evaluate the potential effect of a diet on inflammation.[12] In the present study, we calculated DII based on the intake of 35 food components including energy, carbohydrate, dietary fiber, protein, total fat, trans fatty acid, saturated fatty acid, poly-unsaturated fatty acid, mono-unsaturated fatty acid, n-3 fatty acids, n-6 fatty acids, cholesterol, vitamin D, vitamin A, vitamin E, vitamin C, vitamin B12, vitamin B6, niacin, folic acid, thiamin, riboflavin, iron, magnesium, zinc, selenium, beta-carotene, caffeine, garlic, ginger, onion, saffron, turmeric, green/black tea, pepper. To calculate a participant's DII score, daily consumption of the DII components was converted to a Z-score and percentile by a global daily mean intake estimated from a multi-national database. Then, we calculated the participant's DII score as the sum of the components of the centered proportion score and the Overall inflammatory effect score for each of the DII components. The E-DII calculation was based on dietary intake data per 1000 kcal.[12,18]

Anthropometric variables

Height (cm) was measured with tape in a standard position without shoes, with an accuracy of 0.1 cm. Weight (kg) was assessed with light clothing without shoes with an accuracy of 0.1 kg. Body mass index (BMI) was calculated by dividing weight (kg) by squared height (m^2).

Evaluation of dental plaque control index

O'Leary dental plaque control index is the percentage of stained surfaces (plaque finder) with detector pills relative to the total tooth surfaces in an individual. The plaque index, designed by O'Leary, was estimated at the rate (٪) of plaque deposition on the surface of each tooth divided into four parts: mesial, distal, buccal, and lingual surfaces, and a score for plaque deposition.[19]

Evaluation of gingival index

It was determined according to the following criteria[20] shown in Table 1.

Assessment of decayed, missing, and filled teeth (DMFT)

The DMFT was measured by direct clinical examination and observation based on the number of decayed (D), filled (F), and missed (M) teeth resulting from caries. The examination was performed under adequate light with a mirror.[2]

Assessment of other variables

General characteristics including age, sex, education level, smoking, number of brushings, flossing, use of mouthwash and were obtained by completing a general information questionnaire.

Statistical analysis

Normal distribution was checked using the Kolmogorov–Smirnov test. E-DII was reported as tertiles (T1: ≤ -1.46, T2: -1.45, -0.34, and T3: ≥ -0.33). Continuous variables across the tertiles of the E-DII were compared using the analysis of variance (ANOVA) test. The Chi-square test was applied to compare quantitative variables across tertiles of the E-DII. Dietary intakes were adjusted for total energy intake by the analysis of covariance (ANCOVA) test. The association between dental health indices and E-DII was assessed by the Pearson correlation coefficient (for dental plaque control index and DMFT) and Spearman correlation coefficient (for Gingival Index). Analyzes were performed using SPSS software version 16. Mean and standard deviation were used to report quantitative numbers.

Results

We visited 318 individuals, and 213 of them were enrolled in the present study according to the inclusion and exclusion

criteria. General characteristics of the study participants are given in Table 2. There was no significant difference in age (P = 0.333), sex (P = 0.051), height (P = 0.163), weight (P = 0.402), BMI (P = 0.173), frequency of using toothbrush per day (P = 0.758), frequency of using dental floss per day (P = 0.534), frequency of using mouthwash in the week (P = 0.334), smoking (P = 0.476), education (P = 0.253), married (P = 0.480), income (P = 0.085), and frequency of using chewing gum in the week (P = 0.879) across tertiles of E-DII.

Table 3 shows the dietary intakes of the subjects across tertiles of E-DII. Participants in the last tertile of E-DII consumed more amount of potassium (P < 0.001), vitamin A (P = 0.005(, vitamin C (P < 0.001(, vitamin B6 (P = 0.005(, magnesium (P < 0.001(, zinc (P = 0.002), copper (P < 0.001), and fiber (P < 0.001) compared with the first tertiles. Intake of other nutrients had no significant difference across tertiles of E-DII.

Table 4 demonstrates the correlation coefficient between E-DII and dental health indices. No significant correlation between E-DII and dental plaque control index (r = 0.046, P = 0.518), gingival index (r = 0.001, P = 0.998), and DMFT (r = -0.021, P = 0.762) was observed.

Table 1: Determination of gingival index

Property	Score
Normal gingiva	0
Mild inflammation: slight change in the color, slight edema. No bleeding on probing	1
Moderate inflammation; redness, edema, and glazing. Bleeding on probing	2
Severe inflammation; marked redness and edema. Ulceration. Tendency to spontaneous bleeding	3

Discussion

In this cross-sectional study conducted on a sample of 213 Iranian adults, there was no significant association between E-DII, plaque, and gingival index. It is possible that other contributing factors may have a stronger relationship than E-DII in periodontal disease.

The results of some studies were similar to our findings. In a study by Iwasaki *et al.*[21] 1075 Moroccan subjects who consumed an anti-inflammatory diet, the Mediterranean diet, were included to assess the relation between dietary intakes and periodontitis. There was no significant association between the Mediterranean diet and periodontitis in Moroccan youth. Alhassani *et al.*[22] found no association between empirical dietary inflammatory pattern scores and periodontitis in young men. Therefore, inflammatory aspects of the diet may not significantly influence the risk of periodontitis. A long-term prospective cohort study recruited 34,940 men and reported that a "prudent" diet rich in anti-inflammatory components such as fruits and vegetables was not related to the self-reported incidence of periodontitis after a 24-year follow-up period.[23]

Results of some studies were not similar to our findings. Kotsakis, G. A., *et al.*[24] reported that an anti-inflammatory diet was associated with less tooth loss. In another study, participants who consumed a Mediterranean diet, as an anti-inflammatory and healthy diet, had less periodontal bacteria.[25] Machado *et al.*[15] found that

Table 2: General characteristics of the subjects across tertiles of energy-adjusted dietary inflammatory index

Variables	Tertiles of energy-adjusted dietary inflammatory index			*P**
	T1 ≤ -1.46 (*n*=71)	T2-1.45, -0.34 (*n*=71)	T3 ≥ -0.33 (*n*=71)	
Age (y)	35.44±10.47	33.33±8.81	33.37±9.53	0.333
Sex (%)	23.9	21.4	38.6	0.051
Height (cm)	162.70±28.41	165.37±7.73	70.65±13.17	0.163
Weight (kg)	73.05±13.13	70.34±12.72	70.65±13.17	0.402
BMI	26.03±3.96	25.72±4.4	24.81±3.60	0.173
Frequency of using Toothbrush per day	1.46±0.86	1.38±0.66	1.47±0.74	0.758
Frequency of using Dental floss per day	0.90±0.95	1.04±1.15	0.86±0.94	0.534
Frequency of using Mouthwash in the week	0.40±1.19	0.17±0.54	0.34±10.07	0.334
Smoking (%)	4.2	2.9	7.1	0.476
Education (%)				
Diploma and lower	33.9	41.2	35.7	0.253
Associate Degree and bachelor's degree	54.9	51.5	50	
Master of Science or higher	11.3	7.4	14.3	
Married (%)	80.3	74.3	72.9	0.48
Income (%)				
Low	40.9	55.9	54.3	0.085
Middle	33.8	26.5	32.9	
High	25.4	17.6	12.9	
Frequency of using chewing gum in the week	50.7	52.9	48.6	0.879

*P was obtained from ANOVA. BMI: body mass index

Table 3: Dietary intakes of the subjects across tertiles of energy-adjusted dietary inflammatory index

Nutrient	Tertiles of energy-adjusted dietary inflammatory index			*P**
	T1 ≤ -1.46 (*n*=71)	T2-1.45, -0.34 (*n*=71)	T3 ≥ -0.33 (*n*=71)	
Protein (g)	56.70±156.96	56.72±147.14	57.58±142.30	0.301
Carbohydrate (g)	153.87±489.07	153.95±494.16	156.29±491.95	0.981
Fat (g)	55.76±121.35	55.79±121.81	56.64±119.36	0.963
Cholesterol (mg)	375.17±516.72	375.38±494.51	381.08±551.27	0.671
Saturated fatty acids (g)	23.70±38.35	23.71±40.87	24.07±45.36	0.216
Potassium (mg)†	2977.71±7797.38	2979.34±6917.86	3024.59±5476.66	<0.001
Vitamin A (RAE)(µg)††	1137.81±1296.52	1138.45±1017.92	1155.73±657.16	0.005
Vitamin C (mg)†††	174.71±286.38	174.80±209.73	177.45±158.87	<0.001
Calcium (mg)	1529.34±2165.16	1530.18±2080.93	1553.41±1921.59	0.639
Vitamin E (mg)	12.85±19.43	12.86±17.66	13.05±16.08	0.310
Thiamin (mg)	1.28±2.76	1.28±2.86	1.30±2.83	0.889
Riboflavin (ng)	2.16±3.56	2.16±3.53	2.19±3.18	0.519
Niacin (mg)	19.71±41.82	19.74±38.52	20.03±37.86	0.445
VitaminB6 (mg)††	1.20±3.70	1.20±3.42	1.22±3.03	0.005
Folate (total) (µg)	401.69±832	401.92±779.94	408.01±752.55	0.497
Phosphorus (mg)	1144.60±2677.32	1145.22±2623	1162.60±2487.56	0.607
Magnesium (mg)†	177.50±687.58	177.60±632.64	180.30±529.76	<0.001
Zinc (mg)†	6.22±22.67	6.23±21.19	6.32±18.89	0.002
Copper (mg)†	1.12±2.71	1.12±2.48	1.14±1.88	<0.001
Selenium (µg)	67.70±175.09	67.73±172.42	68.77±168.33	0.840
Fiber (total) (g)†	22.13±57.28	22.14±49.47	22.47±36.63	<0.001

*All values were adjusted for total calorie intake. †T3 was significantly different from T1 and T2. ††T1 was significantly different from T3. †††T1 was significantly different from T2 and T3

Table 4: Correlation coefficient between dietary inflammatory index and dental health indices

Dental health index	Correlation Coefficient*	*P*
Dental plaque control index (O'Leary)	0.046	0.518
Gingival index	0.001	0.998
DMFT	-0.021	0.762

DMFT: Decayed, Missing, and Filled Teeth. *All values were Pearson Correlation Coefficient except for gingival index analyzed by Spearman Correlation Coefficient

DII and periodontitis may be linked, and patients with a proinflammatory dietary pattern were more likely to present worse periodontal measures. Some reasons may justify observed differences between our study and previous research: (1) There was a wide difference between dietary items used to calculate DII between studies. In our study, DII was calculated based on the 35 food items. In contrast, another study used 26 dietary items. (2) In the present study, we reported E-DII. In contrast, in other studies, DII was not adjusted for dietary energy intake.

Some reasons may justify the null relationship between E-DII and periodontal disease observed in the present study. According to the findings of the dietary assessment, participants of the present study did not consume a high proinflammatory diet. As reported in the results, the score of E-DII among subjects in the last tertile of the E-DII was ≥ -0.33. It should be kept in mind that a negative score of E-DII represents an anti-inflammatory dietary pattern. It showed that at least some participants in the highest tertile of E-DII had anti-inflammatory dietary intakes. Therefore, the intake of proinflammatory components was not as high as assumed.

Some limitations and strengths should be presented. The present study had a cross-sectional design. Therefore, it could not show a cause-and-effect relationship. Although E-DII should be calculated by 45 dietary items, only 35 items were available for estimating E-DII in the present study. Missing dietary parameters such as alcohol, flavones, flavones, flavanones, and anthocyanins may have a strong effect on periodontal disease. Also, we did not assess the association between E-DII and biomarkers of inflammation. Moreover, the number of individuals in the study is small. Considering a larger sample size might lead to a significant correlation between E-DII and dental-gingival parameters. The most important strength of this study was that DII was energy-adjusted. Most previous studies used unadjusted DII in analysis. Fewer investigations have been performed on the association between DII and periodontal disease.

Conclusions

Findings from the present cross-sectional study showed that there was no significant relationship between DII and plaque and gingival index. It should also be investigated in prospective studies.

Acknowledgments

The authors express their gratitude to the Vice Chancellor for Research at Isfahan University of Medical Sciences for their support. We also thankful all of the participants in this study.

Financial support and sponsorship

The study is financially supported by Isfahan University of Medical Sciences (grant number: 298106).

Conflicts of interest

There are no conflicts of interest.

Received: 26 Oct 22 **Accepted:** 04 Apr 24
Published: 25 Oct 24

References

1. Lang NP, Schätzle MA, Löe H. Gingivitis as a risk factor in periodontal disease. J Clin Periodontol 2009;36(Suppl 10):3-8.
2. Alhassani AA. The Associations Between Dietary Intake and Incidence of Periodontal Disease. Harvard University; 2018.
3. Ketabi M, Tazhibi M, Moheb RS. The prevalance and risk factors of gingivitis among the children referred to Isfahan Islamic Azad University (Khorasgan Branch) Dental School, in Iran. Dent Res J 2006; 3: 1-4.
4. Amiri AA, Maboudi A, Bahar A, Farokhfar A, Daneshvar F, Khoshgoeian HR, *et al.* Relationship between type 2 diabetic retinopathy and periodontal disease in Iranian adults. North Am J Med Sci 2014;6:139-44.
5. Chiapinotto FA, Vargas-Ferreira F, Demarco FF, Corrêa FOB, Masotti AS. Risk factors for gingivitis in a group of B razilian schoolchildren. J Public Health Dent 2013;73:9-17.
6. Khazaei S, Keshteli AH, Feizi A, Savabi O, Adibi P. Epidemiology and risk factors of tooth loss among Iranian adults: Findings from a large community-based study. BioMed Res Int 2013;2013:786462.
7. Cekici A, Kantarci A, Hasturk H, Van Dyke TE. Inflammatory and immune pathways in the pathogenesis of periodontal disease. Periodontology 2000 2014;64:57-80.
8. Jalaleddin H, Ramezani GH. Prevalence of gingivitis among school attendees in Qazvin, Iran. East Afr J Public Health 2009;6:171-4.
9. Moynihan PJ. The role of diet and nutrition in the etiology and prevention of oral diseases. Bull World Health Organ 2005;83:694-9.
10. Kumar S, Shah S, Budhiraja S, Desai K, Shah C, Mehta D. The effect of periodontal treatment on C-reactive protein: A clinical study. J Nat Sci Biol Med 2013;4:379-82.
11. Galland L. Diet and inflammation. Nutr Clin Pract 2010;25:634-40.
12. 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.
13. 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;113:665-71.
14. Woelber JP, Gärtner M, Breuninger L, Anderson A, König D, Hellwig E, *et al.* The influence of an anti-inflammatory diet on gingivitis. A randomized controlled trial. J Clin Periodontol 2019;46:481-90.
15. Machado V, Botelho J, Viana J, Pereira P, Lopes LB, Proença L, *et al.* Association between dietary inflammatory index and periodontitis: A cross-sectional and mediation analysis. Nutrients 2021;13:1194.
16. Iwasaki M, Manz M, Moynihan P, Yoshihara A, Muramatsu K, Watanabe R, *et al.* Relationship between saturated fatty acids and periodontal disease. J Dent Res 2011;90:861-7.
17. 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;20:150-8.
18. Tabung FK, Smith-Warner SA, Chavarro JE, Wu K, Fuchs CS, Hu FB, *et al.* Development and validation of an empirical dietary inflammatory index. J Nutr 2016;146:1560-70.
19. Silberman SL, Jeune RCL, Serio FG, Devidas M, Davidson L, Vernon K. A method for determining patient oral care skills: The University of Mississippi Oral Hygiene Index. J Periodontol 1998;69:1176-80.
20. Löe H. The gingival index, the plaque index and the retention index systems. J Periodontol 1967;38:610-6.
21. Iwasaki M, Ennibi OK, Bouziane A, Erraji S, Lakhdar L, Rhissassi M, *et al.* Association between periodontitis and the Mediterranean diet in young Moroccan individuals. J Periodontal Res 2021;56:408-14.
22. Alhassani AA, Hu FB, Rosner BA, Tabung FK, Willett WC, Joshipura KJ. The relationship between inflammatory dietary pattern and incidence of periodontitis. Br J Nutr 2021;126:1698-708.
23. Alhassani AA, Hu FB, Li Y, Rosner BA, Willett WC, Joshipura KJ. The associations between major dietary patterns and risk of periodontitis. J Clin Periodontol 2021;48:2-14.
24. Kotsakis GA, Chrepa V, Shivappa N, Wirth M, Hébert J, Koyanagi A, *et al.* Diet-borne systemic inflammation is associated with prevalent tooth loss. Clin Nutr 2018;37:1306-12.
25. Laiola M, De Filippis F, Vitaglione P, Ercolini D. A Mediterranean diet intervention reduces the levels of salivary periodontopathogenic bacteria in overweight and obese subjects. Appl Environ Microbiol 2020;86:e00777-20.