Original Article

Food-Based Dietary Inflammatory Index in Relation to Breast Cancer

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Abstract

Background: There is no study on the association between food-based dietary inflammatory index (FDII) and odds of breast cancer. This study was conducted to investigate the association between FDII and odds of breast cancer in Iranian women. **Methods:** In this population-based case-control study, we enrolled 350 pathologically confirmed new cases of breast cancer and 700 controls which were matched with cases in terms of age and socioeconomic status. FDII score was calculated based on participants' dietary intakes. **Results:** A significant positive association was found between FDII and odds of breast cancer (odds ratio [OR]: 1.38, 95% confidence interval [CI]: 1.01–1.88); such that after taking potential confounders into account, women in the highest tertile of FDII score were 1.52 time more likely to have breast cancer compared with those in the lowest tertile (OR: 1.52, 95% CI: 1.05–2.18). Such a significant positive association was also observed among postmenopausal women, either before (OR: 1.53, 95% CI: 1.09–2.14) or after (OR: 1.54, 95% CI: 1.05–2.27) controlling for covariates. The same association was also observed in normal-weight women, and it remained significant even after controlling for potential confounders (OR: 1.58, 95% CI: 1.04–2.41). **Conclusions:** We found a significant positive association between FDII and the odds of breast cancer in women, particularly postmenopausal ones.

Keywords: *Breast cancer, case-control, diet, FDII, food-based dietary inflammatory index*

Introduction

Diet has an important role in the etiology of breast cancer. Previous studies have shown that adherence to different dietary patterns, including Western, Mediterranean, and diverse dietary patterns, affects the incidence of breast cancer. One of the important aspects of diet is its inflammatory potential which affects circulating levels of inflammatory biomarkers. Given the inflammatory nature of breast cancer, dietary inflammatory potential may have a contribution to the incidence of this cancer.

Earlier studies have examined the inflammatory potential of a diet through the nutrient-based dietary inflammatory index (NDII). Some others have applied the food-based dietary inflammatory index (FDII) to examine the inflammatory nature of the diet. Both methods have been validated against circulating inflammatory biomarkers. It must be kept in mind that nutrients are consumed together, not separately. Therefore, they might influence each other, and the interaction among nutrients in the intestinal lumen or their synergistic effects might confine the interpretability of the NDII. On the other hand, dietary recommendations for the general population are based on foods or food groups rather than nutrients. Therefore, the use of FDII can provide more comprehensive information than the NDII.[1]

Although NDII has been used to examine diet-breast cancer associations, no evidence is available linking FDII and breast cancer. In a prospective cohort study on 34,700 United States (US) women, adherence to a pro-inflammatory diet, assessed by the NDII, was associated with an increased risk of breast cancer, particularly in obese postmenopausal women.[2] A similar finding was also reported in a large case-control study in Italy.[3] FDII was associated with the risk of some cancers, including ovarian and colorectal cancers, but no information is available about breast cancer. Therefore, the current study was performed to examine the association between FDII and odds of breast cancer among Iranian women.

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Material and Methods

Study population

This population-based case-control study was conducted on women residing in Isfahan, Iran, who were over 30 years old. In this study, cases were women who were diagnosed with breast cancer by mammography and physical examination during the last 6 months. Between July 2013 and July 2015, patients were selected from those referred to private clinics or hospitals in Isfahan, Iran. Based on the type I error of 5% and the study power of 80%, the sample size was computed. Considering that an unhealthy diet may increase the risk of breast cancer by 1.5 times, and considering the ratio of controls to cases as two and the common ratio of 0.25, almost 350 breast cancer patients and 700 healthy controls were reached. In the present study, we defined breast cancer patients as those who had the primary incident breast tumor with aggressive behavior, and its histology was available from the medical records. Patients who had a history of hormone replacement therapy and a history of neoplastic lesions or cysts (except current breast cancer) were not included in this study. Also, we did not include patients who had a special diet. Age- and socioeconomic status-matched controls were recruited from healthy women, who had no family history of breast cancer or who had no relationship with breast cancer patients. The inclusion criteria of the control group included females, Iranian nationals, having no special diet, no history of any cysts, malignancy, medical disorder, and hormone replacement therapy. By multistage cluster random sampling method, controls were randomly recruited from apparently healthy women. In this study, we selected the control group among women who were not relatives of patients with breast cancer and attended Primary Health Care Centers for their annual personal checkups or attended to receive required information about their children (such as vaccination, growth monitoring, etc.). Among several healthcare centers in Isfahan, two centers were randomly selected, considering the population under coverage, and then, the required sample was recruited based on the attendance of women to these centers. Finally, we included 350 cases and 700 controls in the study. Before starting the study, informed consent was obtained from all participants. This study was approved by the Ethical Committee of Tehran University of Medical Sciences, Tehran, Iran (IR.TUMS.VCR.REC.1398.945).

Assessment of dietary intakes

Using a 106-item Willett-format dish-based semi-quantitative food frequency questionnaire (DS-FFQ), we collected the dietary intakes of participants. This FFQ has been designed and validated for Iranian adults.[4] Information about the design and validity of this questionnaire was reported elsewhere.[5] A trained nutritionist filled out the questionnaires through face-to-face interviews. Based on nine multiple-choice frequency response categories varying from "never or less than once a month" to "12 or more times per day," participants reported their dietary intake of foods and mixed dishes. The frequency response categories for the food list varied from six to nine choices; we omitted the high-frequency categories for foods with low consumption, while for common foods and foods with high consumption, we increased the number of multiple-choice categories. Finally, daily intakes of all food items were computed and then converted to grams per day using household measures.[6] By Nutritionist IV software, which was modified for Iranian foods, the nutrient intake of participants was obtained.

Development of FDII

Our previous dataset on 486 Tehrani females was used for the development of FDII. In that study, we reported food groups and foods contributing to systemic inflammation, which was examined by assessment of serum levels of Interleukin-6 (IL-6), high-sensitivity C-reactive protein (hs-CRP), and tumor necrosis factor-alpha (TNF-alpha).[7] Food groups and foods loaded in the western dietary pattern of that study were considered as pro-inflammatory, and those loaded in the healthy dietary pattern were considered as anti-inflammatory. This was performed due to a positive association we found between western dietary patterns and inflammatory biomarkers and the inverse association between healthy dietary patterns and inflammatory biomarkers.[7] In this study, mean daily intakes of 12 anti-inflammatory food groups (including fish, poultry, low-fat dairy, fruits, fruit juices, cruciferous vegetables, yellow vegetables, other vegetables, tomatoes, legumes, whole grains, and tea) and 13 pro-inflammatory food groups (including red meats, processed meats, eggs, high-fat dairy, butter, potatoes, French fries, refined grains, mayonnaise, hydrogenated fats, vegetable oils, soft drinks, sweets, and desserts) were calculated. Using the residual method, energy-adjusted amounts of these food groups were calculated.[8] Then, average daily intakes were multiplied by their given factor loadings.[7] By summing up the scores of each food group and food, we calculated the overall FDII score for each participant. To reduce the magnitude of the scores, the FDII score was divided by 100. The FDII score ranged between − 34 and 4.19; a more negative FDII score (lower FDII score) demonstrates a less pro-inflammatory diet, and a more positive FDII score (higher FDII score) demonstrates a more pro-inflammatory diet.

Assessment of breast cancer

In the present study, breast cancer patients were Iranian females who were newly diagnosed with breast cancer (stage I-IV). For all patients, the *in situ* or invasive status of breast cancer was confirmed by physical examination and mammography.[9]

Assessment of other variables

Using an interview-based questionnaire, we gathered data on age, education, marital status, region, smoking,

alcohol consumption, family history of breast cancer, disease history, and menopause status. According to family size (≤4, >4 people), acquisition (house ownership or not), and education (academic and non-academic education), we calculated socioeconomic score as an index of socioeconomic status. Details on the calculation of this score were described elsewhere. Using digital scales, we measured participants' weight to the nearest 100 g with minimal clothes while not wearing shoes and by tape meter; while subjects without shoes standing in a normal position, we measured participants' height to the nearest 0.5 cm. We computed body mass index (BMI) for all participants through weight in kilograms divided by height in meters squared. Using the short form of the International Physical Activity Questionnaire (IPAQ), we assessed participants' physical activity. In the present study, all participants were categorized as physically inactive (having <1 h/week of moderate physical activity) or physically active (≥1 h/week of moderate physical activity).

Statistical analysis

To examine differences in general characteristics between cases and controls, we used Chi-square and the independent samples' *t*-tests. Then, based on tertile cut-off points of the FDII score, we categorized participants. To determine differences in continuous variables across tertiles of FDII score, we applied one-way analysis of variance (ANOVA). The Chi-square test was used to examine the distribution of participants in terms of categorical variables across tertiles of FDII score. To examine the association between FDII and breast cancer, we used binary logistic regression in adjusted models. Age and energy intake were adjusted in the first model. In the second model, we made further adjustments for region (urban/rural), education (educated/non-educated), marital status (single/married), socioeconomic score (continuous), family history of breast cancer (no/yes), alcohol consumption (no/yes), smoking (non-smoker/smoker), menopausal status (premenopausal/postmenopausal), and disease history (no/yes). We additionally adjusted BMI in the final model to obtain an obesity-independent association between FDII and breast cancer. To select confounding variables, we considered both the significant contribution of each confounder (i.e., significant *P*-value) and the contribution of variables based on the evidence from previous studies. However, most confounders included in the adjusted models had a *P*-value less than 0.05.

In all analyses, we considered subjects in the first tertile of the FDII score as the reference group. To obtain the overall trend of odds ratios (ORs) across increasing tertiles of FDII score, we considered these categories as an ordinal variable. Also, the same analyses were performed stratified by BMI categories (normal weight/overweight or obese) and menopausal status (premenopausal/postmenopausal). In the current study, we conducted all statistical analyses using SPSS software (version 18). We considered *P*-value <0.05 as a significant level.

Table 1: General characteristics of cases and controls

Variables	Cases (*n*=350)	Controls (*n*=700)	*P**
Age (year)	65.2±11.2	61.0±10.3	<0.001
Social economic score	12.8±5.2	13.4±5.5	0.09
BMI (kg/m^2)	21.8±4.8	25.5±5.0	<0.001
Region (urban) (%)	36.0	36.1	0.96
Marital status (single) (%)	25.1	11.3	<0.001
Education (educated) (%)	17.4	28.9	<0.001
Disease history (yes) (%)	10.3	8.7	0.40
Physical activity (inactive) (%)	31.7	34.4	0.38
Family history of breast cancer (yes) (%)	9.4	3.4	<0.001
Menopausal status (postmenopausal) (%)	88.3	77.4	<0.001
Smoking (smoker) (%)	17.4	13.0	0.05
Alcohol (alcoholic) (%)	4.6	7.4	0.07

Data are presented as mean (SD) or percent. BMI: body mass index. *Obtained from independent sample *t*-test or Chi-square, where appropriate

Results

The mean FDII score ranged from −34.01 to 4.19. General characteristics of cases and controls are shown in Table 1. Compared to controls, cases were older, had lower BMI, and were more likely to be single, postmenopausal, current smokers, have a family history of breast cancer, and less likely to be educated. No other significant difference was found between cases and controls.

The general characteristics of study participants across tertiles of FDII score are presented in Table 2. Participants in the top tertile of the FDII score were less likely to be single compared with those in the bottom tertile. No other significant differences were observed in terms of other variables across categories of FDII.

Dietary intakes of participants across tertiles of FDII score are indicated in Table 3. Individuals in the top tertile of FDII score had higher intakes of energy, protein, refined grains, red meat, fat, saturated fatty acids (SFAs), and lower intakes of carbohydrates, low-fat dairy, fruits, other vegetables, vitamin C, and magnesium compared with those in the bottom tertile.

Multivariable-adjusted ORs and 95% confidence intervals (CIs) for breast cancer across tertiles of FDII score are shown in **T**able 4. A significant positive association was found between FDII and breast cancer (OR: 1.38; 95% CI: 1.01–1.88). However, this association became non-significant after taking age, energy intake, and socio-demographic variables into account. When further adjustment was made for BMI, a significant positive association was observed; such that women in the highest

Table 2: General characteristics of the study participants across tertiles of FDII scores

	T_1	T_2	T_3	*P**
Age (year)	62.29±10.78	62.56±10.49	62.50±11.22	0.94
BMI (kg/m^2)	24.18±5.11	24.18±5.44	24.58±5.32	0.50
Socioeconomic score	13.18±5.86	12.84±5.22	13.62±5.28	0.16
Region (urban) (%)	36.2	38.7	33.7	0.38
Marital Status (single) (%)	14.2	21.4	12.5	0.003
Education (educated) (%)	25.5	24.4	25.2	0.94
Disease history (yes) (%)	9.8	7.7	10.1	0.51
Physical activity (inactive) (%)	35.6	29.5	35.3	0.16
Family history of breast cancer (yes) (%)	5.6	5.4	5.3	0.97
Menopausal status (post) (%)	81.6	81.5	80.1	0.84
Smoking (smoker) (%)	16.9	11.3	15.1	0.10
Alcohol consumption (yes) (%)	6.5	5.1	7.7	0.36

Data are presented as mean (SD) or percent. BMI: body mass index, FDII: food-based dietary inflammatory index. *Obtained from one-way ANOVA or Chi-square, where appropriate

Table 3: Dietary intakes of study participants across tertiles of FDII scores

	T_1	T_2	T_3	*P**
Energy (kcal/d)	2289.45±38.67	2133.87±34.59	2415.67±36.08	<0.001
Carbohydrate (g/d)	5.80±0.14	4.95±0.15	3.91±0.13	<0.001
Protein (g/d)	0.96±0.03	1.07±0.03	1.10±0.03	0.004
Whole grain (g/d)	300.89±7.10	309.95±6.81	296.36±6.90	0.37
Refined grain (g/d)	84.61±3.40	89.92±3.89	108.14±4.09	<0.001
Bean (g/d)	13.32±0.60	14.23±0.58	15.25±0.97	0.19
Low-fat dairy (g/d)	98.39±6.60	93.28±5.19	77.20±3.84	0.01
Fruit (g/d)	204.10±9.35	162.93±6.80	126.31±5.25	<0.001
Red meat (g/d)	14.07±0.87	17.05±1.20	21.98±1.46	<0.001
Yellow vegetables (g/d)	3.70±0.43	4.94±0.65	3.92±0.40	0.19
Cruciferous vegetables (g/d)	3.99±0.42	3.12±0.40	4.02±0.48	0.27
Other vegetables (g/d)	19.90±3.47	8.68±2.21	4.54±2.29	<0.001
SFA (mg/d)	16.46±0.57	18.60±0.57	20.69±0.56	<0.001
Total fat (g/d)	12.77±0.45	14.33±0.46	17.34±0.43	<0.001
Fiber (g/d)	34.33±1.13	33.99±0.98	31.43±0.94	0.08
Vitamin C (mg/d)	22.84±0.72	21.49±0.63	19.51±0.60	0.001
Magnesium (mg/d)	28.35±0.76	24.46±0.72	20.31±0.69	<0.001
Iron (mg/d)	1.55±0.05	1.66±0.04	1.58±0.04	0.29

Data are presented as mean (SE). FDII: food-based dietary inflammatory index, SFA: saturated fatty acid, d: day, g: gram, mg: milligram. *Obtained from one-way ANOVA

tertile of FDII score had 52% greater odds of breast cancer compared with those in the lowest tertile (OR: 1.52; 95% CI: 1.05–2.18).

When we performed stratified analyses based on menopausal status, no significant association was found between FDII and odds of breast cancer in premenopausal women, neither in crude nor in multivariable-adjusted models [Table 5] However, there was a significant positive association between FDII and breast cancer among postmenopausal women (OR: 1.53, 95% CI: 1.09–2.14). After taking potential confounders into account, postmenopausal women in the highest tertile of FDII score were 54% more likely to have breast cancer compared with those in the lowest tertile (OR: 1.54, 95% CI: 1.05–2.27). In stratified analyses based on BMI status, FDII was positively associated with odds of breast cancer in normal-weight women before (OR: 1.48, 95% CI: 1.01–2.18) and after (OR: 1.58, 95%CI: 1.04–2.41) considering potential confounders. However, such a significant association was not observed among women with overweight or obese [Table 6].

Discussion

In the current study, we found that adherence to a pro-inflammatory diet was associated with increased odds of breast cancer. This association remained significant even after taking potential confounders into account. Such a significant positive association was also observed in

Table 4: Multivariable-adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for breast cancer across tertiles of FDII scores

	Total of FDII			*P*-trend*
	T_1	T_2	T_3	
Total				
Crude	1	0.97 (0.74–1.35)	1.38 (1.01–1.88)	0.03
Model 1	1	1.09 (0.77–1.53)	1.28 (0.92–1.78)	0.13
Model 2	1	1.02 (0.71–1.46)	1.34 (0.95–1.88)	0.08
Model 3	1	1.05 (0.71–1.54)	1.52 (1.05–2.18)	0.02

Data are presented as OR and 95% CI. FDII: food-based dietary inflammatory index. Model 1: Adjusted for age and energy. Model 2: Additionally, adjusted for region, marital status, education, disease history, physical activity, family history of breast cancer, menopausal status, smoking, alcohol consumption, and socioeconomic status. Model 3: Additional adjustment for BMI. *Obtained from binary logistic regression

Table 5: Multivariable-adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for breast cancer across tertiles of FDII scores stratified based on menopausal status

	Total of FDII			*P*-trend*
	T_1	T_2	T_3	
Premenopausal				
Crude	1	1.63 (0.72–3.68)	0.73 (0.28–1.89)	0.56
Model 1	1	1.65 (0.71–3.82)	0.66 (0.25–1.74)	0.43
Model 2	1	1.63 (0.65–4.05)	0.79 (0.28–2.22)	0.70
Model 3	1	3.29 (1.01–10.71)	2.33 (0.62–8.72)	0.20
Postmenopausal				
Crude	1	0.88 (0.61–1.27)	1.53 (1.09–2.14)	<0.001
Model 1	1	1.03 (0.70–1.51)	1.41 (0.99–2.01)	0.04
Model 2	1	0.97 (0.65–1.45)	1.47 (1.02–2.11)	0.03
Model 3	1	0.97 (0.63–1.48)	1.54 (1.05–2.27)	0.02

Data are presented as OR and 95% CI. FDII: food-based dietary inflammatory index. Model 1: Adjusted for age and energy. Model 2: Additionally, adjusted for region, marital status, education, disease history, physical activity, family history of breast cancer, menopausal status, smoking, alcohol consumption, and socioeconomic status. Model 3: Additional adjustment for BMI. *Obtained from binary logistic regression

Table 6: Multivariable-adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for breast cancer across tertiles of FDII scores stratified by BMI status

	Total of FDII			*P*-trend*
	T_1	T_2	T_3	
Normal weight				
Crude	1	0.89 (0.59–1.34)	1.48 (1.01–2.18)	0.03
Model 1	1	1.01 (0.66–1.56)	1.44 (0.97–2.15)	0.06
Model 2	1	1.00 (0.63–1.58)	1.58 (1.04–2.41)	0.02
Overweight or obese				
Crude	1	1.20 (0.65–2.24)	1.35 (0.73–2.48)	0.33
Model 1	1	1.32 (0.69–2.53)	1.05 (0.55–2.02)	0.90
Model 2	1	1.15 (0.56–2.33)	1.11 (0.55–2.25)	0.76

Data are presented as OR and 95% CI. FDII: food-based dietary inflammatory index, BMI: body mass index. Model 1: Adjusted for age and energy. Model 2: Additionally, adjusted for region, marital status, education, disease history, physical activity, family history of breast cancer, menopausal status, smoking, alcohol consumption, and socioeconomic status. *Obtained from binary logistic regression

postmenopausal women and also normal-weight ones. To our knowledge, this study was the first to investigate the association between FDII and breast cancer.

Chronic inflammation plays an important role in the development and progression of cancer, especially breast cancer.[10] The contribution of diet on inflammatory biomarkers has been demonstrated in earlier evidence.[11] Although previous studies have investigated the association between dietary factors with pro- and anti-inflammatory properties and breast cancer, little attention has been paid to the inflammatory potential of the diet as a whole. In the current study, we found a positive association between FDII and odds of breast cancer. Previous studies have focused on NDII, and no study assessed FDII about breast cancer. However, our findings were in line with a Swedish cohort study on a large sample size of women,[12] in which nutrient-based inflammatory potential of the diet was positively associated with risk of breast cancer. Also, like our study, in a Swedish cohort study, no significant association was reported between NDII and breast cancer among premenopausal women. In another cohort study on postmenopausal women, a positive association between NDII and breast cancer was observed.[2] Such finding was also reported in a large Italian case-control study.[3] In opposite to our findings, two cohort studies revealed no significant association between NDII and the risk of breast cancer.[13,14] Furthermore, in a case-control study from Germany, researchers failed to find any significant association between NDII score and postmenopausal breast cancer risk.[15] Discrepant findings on the association between DII and breast cancer might be explained by different quality of studies and by including different variables in the adjusted models. Considering different anti- and pro-inflammatory DII components across previous studies could be another reason for the observed discrepancy.

In the current study, the positive association between FDII and breast cancer was observed among postmenopausal women, but not in premenopausal ones. This non-significant association among premenopausal women might be due to the anti-inflammatory properties of internal estrogen interfering with the effects of dietary components with inflammatory properties. In addition, the observed association among postmenopausal women may be explained by the positive association between age-related production of free radicals in the body and breast cancer.[16] In addition, unlike normal-weight women, we found no significant association between FDII and

breast cancer among those with overweight or obesity. This disparity might be due to the adherence of obese women to healthy or anti-inflammatory diets in an attempt to lose their weight.

Consumption of a pro-inflammatory diet rich in food items such as meat and butter increases systemic inflammation by increasing levels of IL-6. This interleukin is involved in the Stat3 pathway, which results in the induction of carboxylic acid (COOH) terminal and increased expression of fascin, both of which play an important role in breast cancer cell migration and invasion.[17] A positive association between FDII and breast cancer might also arise through the activation of insulin-like growth factor (IGF-1R), which initiates a chain of events that leads to the inhibition of apoptosis and increased cell proliferation in breast tissue.[18,19]

Our study had several strengths. Firstly, the association between FDII and breast cancer was examined for the first time. Secondly, a wide range of potential confounders were controlled to obtain an independent association. In this study, we used energy-adjusted scores of FDII, which can help reduce misclassification of study participants. Thirdly, validated questionnaires were used for data collection. However, some limitations should be considered. Our findings are subject to selection and recall bias, which are common in case-control studies. As with all epidemiological studies, misclassification of study participants due to the use of FFQ is inevitable. Despite adjustment for several confounders, the potential effects from residual confounders cannot be excluded. Furthermore, since the dietary intakes of the Middle Eastern population are different from western nations, the generalizability of the findings to other populations should be done with caution.

Conclusions

In conclusion, we found that adherence to a diet with high inflammatory potential was associated with increased risk of breast cancer. Such a significant association was also observed in postmenopausal women, but not in premenopausal ones. Furthermore, unlike women with overweight or obese, we found a significant positive association between FDII and breast cancer among normal-weight women. However, it should be mentioned that we found no large effect size for the significant positive associations, and the lower CIs for the ORs were close to one. Therefore, the positive association between FDII and breast cancer should be interpreted with caution. Further studies are needed to confirm these findings.

Ethical approval and consent to participate

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Also, the current study was approved by the Ethical Committee of Tehran University of Medical Sciences, Tehran, Iran. Before starting the study, informed consent was obtained from all participants.

Availability of data and materials

The datasets analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

AE, SBK, and LA contributed to the conception, design, and data collection. SBK contributed to data preparation. NE, YS, OS, and AE contributed to data analysis and manuscript drafting. OS and GA contributed to the manuscript editing. AE supervised the study. All authors read and approved the final version of the manuscript.

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Conflicts of interest

There are no conflicts of interest.

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