Dietary Inflammatory Index in Relation to Psoriasis Risk, Cardiovascular Risk Factors and Clinical Outcomes; A Result from Case-Control Study in Psoriasis Patients

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**Dietary Inflammatory Index in Relation to Psoriasis Risk, Cardiovascular Risk Factors
and Clinical Outcomes; A Result from Case-Control Study in Psoriasis Patients**

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ABSTRACT

Psoriasis is an inflammatory skin disease. Despite the understanding of the disease pathogenesis, the link between diet-induced inflammation and the risk of psoriasis remains underexplored. Therefore, we have examined the capability of literature-derived Energy-adjusted Dietary Inflammatory Index (E-DII) as a predictive tool for inflammation, incidence, and severity of psoriasis (as indexed by The Psoriasis Area Severity Index (PASI)). We have conducted a case-control study consisting of 149 adults ($n = 75$ cases and $n = 74$ controls). The E-DII score was calculated based on the dietary intake that was evaluated by a validated 168 item quantity food-frequency questionnaire. The E-DII tertile cut-offs were categorized based on the following cut points: tertiles 1 ≤ -1.99 ; tertiles 2 $= -2.00 - 0.60$; tertile 3 ≥ 0.61 . Logistic regression models were applied to estimate the multivariable odds ratio (OR) that was adjusted for confounders. Patients with higher pro-inflammatory E-DII had a 3.60-times increased risk of psoriasis relative to patients in tertiles 1 (E-DII_{T3} vs E-DII_{T1}: OR = 3.64; 95% CI 1.51, 8.79, $P = 0.005$). The severity of disease as indexed by PASI remained associated with E-DII (E-DII_{T3} vs E-DII_{T1}: OR = 3.64; 95% CI 1.74, 7.57, $P = 0.015$). For each unit increase in E-DII, the odds of disease severity increased by 3-times. Patients consuming a more pro-inflammatory diet were at greater risk of psoriasis. These patients also demonstrate increased disease severity relative to individuals consuming a more anti-inflammatory diet.

- **A pro-inflammatory diet is associated with higher psoriasis incidence**
- **Subjects with higher DII scores, had higher inflammatory markers levels.**

Key Word: Dietary Inflammatory Index; Psoriasis; Cardiovascular Risk Factors; Clinical Outcomes; Case-Control Study.

51 Introduction

Psoriasis is a chronic inflammatory disease with a global prevalence of 2% (Addolorato et al. 2003). Patients with psoriasis often demonstrate psychological and emotional problems that affect their daily activity and quality of life (QOL) (Albanesi et al. 2010; Camargo et al. 2017). Psoriasis often involves hyper-proliferation of epidermal keratinocytes (Feldman et al. 2016). Metabolic syndrome and obesity are predominant in patients with psoriasis, both of which are metabolic triggers for cardiovascular disease (CVD) (Lowes et al. 2007). Patients with psoriasis are at risk of CVD due to the underlying chronic inflammatory condition (Albanesi and Pastore 2010). Thus, dietary components with pro-inflammatory (e.g., heme, iron, saturated fatty acids,) or anti-inflammatory (e.g., dietary fibers, polyphenols) potentials, and other health beneficial bioactive micronutrients can play a critical role in the promotion or prophylaxis of inflammation associated with psoriasis (Millsop et al. 2014; Phan et al. 2018). Earlier studies have demonstrated the association of a pro-inflammatory diet with several inflammatory conditions such as cardiovascular and inflammatory bowel diseases (Dandona et al. 2007; Moludi et al. 2021). Furthermore, studies have publicized that adherence to a healthy diet (such as the low inflammatory diet) likely decreases the risk of systemic chronic inflammation and thus, reduces the risk of metabolic syndrome, cardiovascular disease, and other chronic inflammatory diseases (Dandona et al. 2007; Giugliano et al. 2006).

52 A diet high in fruits and vegetables, cereals, fish, legumes, and olive oil which are the main
53 components of an anti-inflammatory diet, might lower the inflammation associated with psoriasis
54 (Millsop et al. 2014), whereas pro-inflammatory diets (e.g., red meat) may aggravate the
55 inflammation (Mueller et al. 2010). Thus, it is important to develop a scoring algorithm that is

capable of calculating the dietary influences on disease severity, considering the overall dietary pattern of an individual (Gholizadeh et al. 2018; Shivappa et al. 2014).

Dietary inflammatory index (DII) has been developed to identify the inflammatory potential of an individual's dietary pattern (Cavicchia et al. 2009; Shivappa et al. 2014). In this method, food items were introduced that can potentially decrease or increase the severity of inflammation and thereby, affect the clinical pathogenesis of psoriasis. We have accessed 31 of such food items including onion, garlic, coffee, tea, energy, protein, carbohydrates, fiber, vitamins C, D, A, E, B₆, B₁₂, B₁, B₂, B₃, folate, beta-carotene, saturated fatty acids, total fat, mono and polyunsaturated fatty acids (MUFA, PUFA), omega 3 and 6, cholesterol, Mg, Fe, Se, and Zn. Intake of those food items was linked to world database mean $\pm$ SD, summarizing 11 studies globally (Shivappa et al. 2014). Pro-inflammatory or anti-inflammatory properties of specific food items can have beneficial or harmful effects on the modulators of inflammation including various cytokines like IL (interleukin)-1 β , IL-4, IL-6, IL-10, tumor necrosis factor-alpha (TNF α), and C reactive protein (CRP) (Camargo-Ramos et al. 2017). Several studies have shown that increased consumption of diet associated with a pro-inflammatory phenotype can lead to increased risk of chronic disease including diabetes, cardiovascular diseases (CVDs), cancer, metabolic syndrome, and Alzheimer's diseases (Cavicchia et al. 2009; Ramallal et al. 2015; Shivappa et al. 2014).

As per our knowledge, the association between E-DII and the risk of psoriasis has not been investigated before. Therefore, to illuminate this unexplored, yet critically important association, we have assessed E-DII in relation to inflammatory status, oxidative stress, cardiac risk factors, incidence, and severity of psoriasis using a case-control study design.

79 **Methods**

80 **Study design**

81 The current case-control study was undertaken between December 2018 to January 2019 at
82 Tabriz city, Iran. Subjects with psoriasis ($n = 80$) were chosen by random sampling from patients
83 that were referred by Sheikh Al Rais Clinics. The choice of the selected patients with clinically-
84 defined psoriasis was based on guidelines laid by the American Academy of Dermatology-
85 National Psoriasis Foundation (AAD-NPF) (Martin et al. 2019). We included only new cases and
86 subjects of 20 - 50 years of age in order to lessen the possibility that subjects would change their
87 diet considerably during the study period. In the case of controls, the subjects were referred by
88 the clinic for medicinal problems other than psoriasis (e.g., nose or eye disorders, fractures,
89 trauma, etc.). Exclusion criteria for controls were prior diagnosis of chronic inflammatory
90 diseases such as multiple sclerosis, rheumatoid arthritis, cancer, liver diseases, endocrine, and
91 metabolic disorders, and has a history of weight loss or intake of special diet (e.g., vegan).
92 Matching for sex, age, and BMI between cases and controls was done. Out of the joined cases
93 and controls, 5 psoriasis and 6 controls were excluded because of lacking food frequency
94 questionnaires (FFQs) or other data. The final subjects comprised 75 cases and 74 controls,
95 accounting for a response rate of 92% (**Figure 1**). This study was approved by the ethics board
96 of the Tabriz University of Medical Science (IR.TBZMED.REC.1398.504), and signed written
97 consent was received from all participants.

98 Bodyweight was measured with subjects upright on a digital weighing machine (Soehne, Berlin,
99 Germany), minimally dressed, excluding shoes, and weight was reported with a deviation of 100
100 g. Height measurement was performed using a non-stretch meter tape with close 0.5 cm

accuracy, fixed to a wall, and with subjects wearing no shoes. Body mass index (BMI) was computed by calculating weight-to-height ratio ($\text{BMI} = \text{kg}/\text{m}^2$).

Baseline questionnaires including demographic information, socioeconomic status, physical activities, smoking status, alcohol intake, dietary habits, medical history, and food frequency questionnaire (FFQ) were completed for all participants by face-to-face interview by a trained interviewer.

Dietary intake over the previous years was assessed using a valid and reliable semi-quantitative food frequency questionnaire (FFQ) (Mirmiran et al. 2010; Moradi et al. 2018). The FFQ consisted of a total of 130 food items with typical serving sizes. The subjects were requested to state how often they consumed each of the food listed, in terms of daily intake of the number of times (every day, weekly, once a month, or annually). Nutrient intake was then measured using the Nutritionist IV software (Nutritional Database Manager 4.0.1, First Data Bank, USA). FFQ-derived dietary data were applied to compute E-DII for all participants. Subjects who reported energy intake beyond the normal range of 800-4200 Kcal/day were excluded from the analysis. We obtained dietary information from the questionnaires and subsequently calculated the energy-adjusted E-DII (E-E-DII) scores based on the established method developed by Shivappa et al. (Shivappa et al. 2014). To compute the E-DII scores, we converted all reported intakes of the food parameters to an amount per 1000 kcal of energy intake. In this method, 45 food items were introduced that are known to either decrease or increase inflammatory conditions. Among those food items, we had access to 31 of them including onion, garlic, coffee, tea, energy, protein, carbohydrates, fiber, vitamins C, D, A, E, B₆, B₁₂, B₁, B₂, B₃, folate, beta-carotene, saturated fatty acids, total fat, mono and polyunsaturated fatty acids (MUFA, PUFA), omega 3 and 6, cholesterol, Mg, Fe, Se, and Zn. These food item intakes were linked to the world

database mean $\pm$ SD from 11 studies globally (Shivappa et al. 2014). Z-score was determined for each of the mentioned items and participants by subtracting the standard global mean from participant's reported dietary intake and then was divided by the SD. We transformed this value to a centered percentile score to minimize the right skewing effect. For this purpose, we multiplied the Z score by 2 and subtracted it by one unit. Finally, all food items that were specified for the DII score were summed and were used to generate the DII scores. Higher E-DII was indicative of a pro-inflammatory diet, whereas low DII was indicative of an anti-inflammatory diet. The DII tertile cut-offs were categorized based on specific cut points: tertile 1 ≤ -1.99 ; tertile 2 = $-2.00 - 0.60$; tertile 3 ≥ 0.61

Efficacy assessments

The Psoriasis Symptom Scale (PSS) which is a self-reporting inventory with 4 symptoms that measures the severity of itching, pain, burning, and redness over the previous week was followed. Items are valued on a 5-point gauge ranging from 0 - 4 in terms of severity of the disease. The reported internal consistency of the PSS remains high (Cronbach's alpha = 0.87) (Ghassemzadeh et al. 2005).

Psoriasis Area and Severity Index (PASI) is the best generally employed instrument for quantitative assessment of the psoriasis severity. PASI pools the valuation of the severity of lesions and the area touched into a distinct score in the range from 0 (no disease) to 72 (severe disease) (Langley and Ellis 2004).

Biochemical variables

Blood samples were collected after 12-h overnight fasting. The blood samples were drawn from the antecubital vein. High sensitivity C-reactive protein (hs-CRP) concentrations were assessed

by immunoturbidimetry. The level of interleukin 6 (IL-6) as an inflammatory marker was determined by ELISA using a commercially available kit (Glory Science Co.). The serum concentration of malondialdehyde (Miripour, #28) was assayed as a biomarker of lipid peroxidation which was reflective of oxidative stress. The serum total antioxidant capacity (TAC) as oxidative stress markers was determined by spectrophotometry using a commercially available kit (ZellBio GmbH, Germany) following the instructions of the manufacturer. Serum total triglyceride (TG), total cholesterol (Feldman et al.), and high-density lipoprotein (HDL-C) levels were measured by routine laboratory methods (Pars Azmoon kit, Pars Azmoon Inc., Tehran, Iran). The Friedewald equation was used to calculate the level of low-density lipoprotein (LDL-C).

Statistical analysis

Statistical tests were performed using STATA® 10 software. Continuous variables are presented as mean or median (25th – 75th percentile) for normally distributed and non-normally distributed data median, respectively; qualitative data are presented as frequency (percent). The Kolmogorov-Smirnov test was used to determine the normality of data distribution. Independent samples t-test and Mann–Whitney U test was used for comparing normal and non-normal distributed continuous variables, and chi-square test for categorical variables. Independent samples t-test and Mann–Whitney U test were used to examine the mean differences of nutrient intakes between case and control groups. Odds ratios (OR) and 95% confidence intervals for some related clinical factors including the presence of psoriasis, PASI, and PSS, and also biochemical variables such as HDL, LDL, TG, TC, TAC, MDA, and hs-CRP were calculated by logistic regression after adjusting for confounder factors (age, sex, BMI, activity, smoking,

diabetes, hyperlipidemia, and hypertension) in 3 models. *P* trends were also reported for main outcomes and food parameters across tertiles of E-E-DII. *P* < 0.05 was considered statistically significant for all cases.

Results

Table 1 shows the distribution of cases and controls according to selected variables. No statically significant differences (*P* > 0.05) were detected in terms of age, BMI, sex, and medical history between the two groups. E-DII scores in the current study ranged from -3.3 (extreme anti-inflammatory score) to +3.82 (maximum pro-inflammatory score). Furthermore, the case group had higher values of E-DII as compared to the control groups (0.10 vs -2.14, *P* = 0.03).

Findings of food intake, including dietary fat content, fatty acids, dietary fiber, and micronutrients between case and control groups, are shown in **Table 2**. These data show that there was a statistically significant difference in the mean dietary intake of some nutrients such as fat, MUFA, PUFA, Vitamin A, α -Tocopherol, etc.

Table 3 demonstrates OR (95% CI) for lipid profiles, inflammatory and oxidative stress markers as outcomes. Patients in tertiles 3 of E-DII (more pro-inflammatory) had significantly higher TG (E-DII tertiles 3: OR = 4.77; 95% CI 0.82, 27.56), and lower HDL (E-DII tertiles 3: OR = 0.09; 95% CI 0.01, 0.61, *P* = 0.008) and TAC value (E-DII Tertiles 3: OR = 3.28; 95% CI 0.52, 20.53, *P* = 0.007). For other biomarkers, no significant association was observed. Accordingly, patients in the greatest pro-inflammatory tertiles of E-DII had a greater level of TG and lower levels of HDL and TAC.

Table 4 illustrates OR and 95% confidence intervals for psoriasis status, and severity of disease (PASI, and PSS) as an outcome; After multivariate adjustment, patients in the maximum pro-inflammatory E-DII group had 3.64-times higher odds of developing psoriasis relative to

subjects in tertiles 1 ($E-DII_{T3 \text{ vs } T1}$: OR = 3.64; 95% CI 1.51, 8.79, $P = 0.005$). Similar results were detected for severity of disease as indexed by PASI, ($E-DII_{T3 \text{ vs } T1}$: OR = 3.64; 95% CI 1.74, 7.57, $P = 0.015$). These data essentially indicates that for each unit rise in E-DII, the chances of the severity of disease, which indexed by PASI, increased by 3.64-times. No statically significant relationship was found between E-DII and PSS.

Discussion

In the current study, we aimed to evaluate the association between inflammatory potential of diet measured by E-DII using biomarkers of inflammation, oxidative stress, incidence, and severity of psoriasis. Our data demonstrate that patients with greater E-DII (i.e., signifying a pro-inflammatory diet) were at higher odds of having psoriasis and its severity. These data collectively indicate that increased intake of a diet with pro-inflammatory potential is related to an increased risk of psoriasis. Compared to patients in tertiles 1, those in tertiles 3 were 3.67-times more likely to have psoriasis. We also observed that every 1-unit rise in E-DII score was associated with an increase in PASI score by 3.64-times. This result may support the assumption that the low E-DII score is associated with a lesser amount of severe psoriasis (as demonstrated for PASI)

Intake of food groups such as fruits and vegetables has been revealed to decrease inflammation, whereas dietary patterns that include processed meat may escalate the severity of inflammation (Hurst and Hurst 2012; Ley et al. 2014). Despite these observations, it remains unclear what degree of psoriasis is attributed to diet. While a comprehensive picture of the role of nutrition on psoriasis is far from complete, much development has occurred in this field of research (Phan et

al. 2018). The biggest underexplored aspect includes the assessment of the effect of the whole diet on psoriasis. Indeed, diet appears to exert both beneficial or detrimental effects via mitigating or triggering inflammation (Hurst and Hurst 2012; Shivappa et al. 2014). Evidence suggests that the usage of the E-DII has numerous unique benefits over other dietary measures and in the current study, it was calculated particularly to conclude on the inflammatory potential of diet in relation to psoriasis-associated inflammation (Ramallal et al. 2015). Several studies have revealed that a higher E-DII score is associated with elevated levels of inflammatory markers (e.g., TNF- α , IL-6, IL-1 β), and many diseases including cancer and CVD may develop as a direct or indirect consequence of chronic inflammation (Deng et al. 2017; Giugliano et al. 2006). Our data demonstrated that the pathogenesis of psoriasis could be in part explained by a high E-DII score. This is the first study emphasizing the association between the E-DII and psoriasis.

Although no prior study had specified the association between the E-DII score and the risk of psoriasis, our finding is consistent with previous results for E-DII and abortion in women (Vahid et al. 2017). Effect sizes for abortion risk have generally been within the OR range 1.5 - 2, which is comparable to our results. By comparing the results from this study, we hope to determine the association between E-DII and psoriasis. The E-DII scores also in the current population were comparable to those of earlier studies that had investigated the association of diet with metabolic syndrome, cardiovascular disease, and cancer (Deng et al. 2017; Giugliano et al. 2006; Ramallal et al. 2015; Shivappa et al. 2014).

Chronic inflammation is the underlying association between E-DII and psoriasis, which may be explained through the effect of pro-inflammatory dietary patterns and their effects on disease pathogenesis (Debbaneh et al. 2014; Gottlieb et al. 2005). Previous studies also have indicated

that chronic inflammation plays a critical role in increasing the risk of psoriasis (Reich 2012). Similarly, the anti-inflammatory effects of diet on psoriasis could be explained by beneficial mechanisms mainly exerted by a dietary component such as oleic acid, monounsaturated fatty acids, polyphenols, dietary fibers, anti-oxidant agents, and micronutrients (Katsimbri et al. 2021). Additionally, a particular diet may change the gut microbiota and thus elicit unwanted and aggravated immune function (e.g., modification of the T helper cell, T-regulatory cell, and type 17 and its balance) which can activate systemic autoimmune diseases (Chow and Mazmanian 2009). A popular explanation of the relationship between E-DII and psoriasis is that diet can change the expression of different immunity-related genes through epigenetic modification which in turn could lead to psoriasis (Millsop et al. 2014). Furthermore, in subjects with psoriasis, intestinal permeability is increased, stimulating translocation of gut bacteria-derived products (e.g., endotoxin) into the portal circulation and increasing inflammation (Moludi et al. 2020). It has also been proposed that an anti-inflammatory diet can improve intestinal permeability in patients with psoriasis (Addolorato et al. 2003; Humbert et al. 1991). In the same direction, ω -3 fatty acids, as a component of the anti-inflammatory diet, decreased levels of IFN- γ , IL-17, IL-6, and TNF- α supporting its ability to diminish inflammation (Marinoni et al. 2014). Despite this evidence, we acknowledge the fact that there are considerable discussions among researchers about the etiology of psoriasis which is multifactorial, and no single mechanism is likely to be the key contributing factor for the development of psoriasis. Indeed, we also wanted to compare the E-DII with the inflammatory markers. A recognized low E-DII score has been associated with a minor level of inflammatory biomarkers including hs-CRP. This is consistent with what has been found in previous studies demonstrating that higher E-DII is reflective of a pro-inflammatory milieu or an unhealthier dietary pattern (Ramallal et al. 2015).

Another important finding was that a high E-DII score (i.e., individuals who had the maximum pro-inflammatory diets) was associated with increased severity of psoriasis (as measured by the PASI and PSS scores). A similar conclusion was reached by Barrea et al (Phan et al. 2018), indicating a relationship between the Mediterranean diet and the severity of psoriasis. Since our study is the first to report the association of E-DII with the severity of psoriasis, the implications of the study are likely to be a promising aspect of E-DII. The mechanisms by which E-DII might affect psoriasis remain unclear. One of the possible mechanisms linking E-DII with psoriasis could be through the effect of a pro-inflammatory diet on the levels of various inflammatory mediators, such as hs-CRP, which in turn seem to be significant components in the development and severity of psoriasis (Reich 2012). Diet with high pro-inflammatory properties (e.g., trans fatty acids, processed meat, iron), and low in anti-inflammatory food (e.g., vegetables, fibers,), as indexed by high E-DII, may play an imperative role in the risk of psoriasis development (4, 5). As it is generally agreed that a high E-DII score is related to an increased risk of metabolic syndrome and obesity (Deng et al. 2017; Giugliano et al. 2006), both are associated with severity of psoriasis and a greater risk of CVD (Hamminga et al. 2006). The present work led us to conclude that a higher E-DII score is associated with increased severity of disease in psoriasis patients.

The E-DII has been shown to be related to different inflammatory and oxidative markers including TAC, MDA, and hs-CRP. Diets consisting of high amounts of fruits and vegetables are related to low levels of hs-CRP and higher TAC levels. Certain nutrients such as omega-3 fiber and magnesium are associated with low levels of inflammation. We found that a pro-inflammatory diet (higher E-DII scores) was associated with decreased levels of oxidative stress markers, while no significant associations were found between E-DII and inflammatory markers

including hs-CRP and IL-6. The lack of a significant association in our study likely resulted due to the low detectable levels of these parameters among the participants. This result is consistent with those of previous studies which reported the relationship between diet and inflammatory markers in adults (Neufcourt et al. 2015; Tabung et al. 2015). It is important to note that others have not observed significant results with CRP, IL-4, and IL-6 (Shivappa et al. 2017). Psoriasis is associated with chronic systemic inflammation and an increased risk of cardiovascular diseases (Reich 2012). Moreover, in patients with psoriasis, systemic anti-inflammatory therapy such as methotrexate may reduce the incidence of cardiovascular disease (Martin et al. 2019). In our study, greater adherence to the anti-inflammatory diet (lower E-DII scores) was associated with a lesser severity of psoriasis and lowering of cardiovascular risk factors. These findings suggest a healthy diet with anti-inflammatory capacities can help reduce the risk of cardiovascular disease in subjects with psoriasis.

Finally, we would like to highlight that our study has a number of limitations, which could be addressed through future studies. As with other crosses-sectional studies, it was not possible to discriminate whether the associations were causal or causative. Furthermore, the study population was limited to adult patients referred by the dermatology clinic. Thus, the generalizability of the finding is limited to only adult populations. Some components are missing from the DII computation including γ -eugenol, flavan-3-ol, flavones, and anthocyanidins which are a well-known bioactive constituent of a plant-based diet with anti-inflammatory properties. Therefore, it is probable that the latent effects of these food items may have been compromised even though the DII achieved in the current study was similar to previous studies. A crucial limitation is that the nutritional data was based on the respondents' memory. Thus, we applied a well-trained nutritionist to minimize this recall bias, if any. Additional limiting factors included

the use of a validated FFQ, which is acceptable for a full calculation of main nutrient sources in the diet. Despite these shortcomings, the present study has several major strengths and is the first study to assess and report the association between DII and psoriasis.

Conclusion

In conclusion, our results demonstrate an association between an E-DII score (high in pro-inflammatory diet) and the severity of psoriasis. This relationship was free from demographic and confounding variables. Additionally, these findings provide additional information about chronic inflammation and oxidative stress in the development and severity of psoriasis. If these results are repeated in future research, an adjusted diet shall be one of the multidimensional approaches. Broadly translated, our findings suggest that increased consumption of anti-inflammatory dietary items such as omega-3 fatty acids, β -carotenes, phytochemicals, and plant-based foods rich in fiber, along with decreasing consumption of pro-inflammatory foods such as red and processed meat, trans-fatty or acids saturated fat, may be an effective prophylactic approach for decreasing the risk and severity of psoriasis.

Declaration

Acknowledgments

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Conflict of Interest

The authors declare that they have no conflict of interest.

Role of Funder/Sponsor Statement

funding organization or sponsor no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Author contribution statement

MA, and MJ designed the study, RM and AK conducted the study and collected the data. HJV performed the statistical analysis. MJ, HJV, HL and PD prepared the manuscript. All the authors have read and approved it.

Access to Data and Data Analysis

Data will be available upon request from the corresponding author. Also, Dr. Jalal Moludi as correspondent authors confirmed that full access to all the data in the study upon request and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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442 **Figure legends**

443 **Fig1. Flowchart of study**

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67x65mm (300 x 300 DPI)

Table 1- Characteristics of study subjects ^a

Variable	<i>Case (n=75)</i>	<i>Control (n=74)</i>	<i>P-value</i>
Age (years)^b	56.96 (14.46)	55.30 (13.04)	0.462
Sex			
Male	48 (64.0)	44 (59.5)	0.569
Weight (kg)^b	67.93 (13.05)	68.85 (15.66)	0.699
Height (cm)^b	161.80 (9.53)	160.21 (9.10)	0.304
BMI (kg/m²)^b	25.89 (4.38)	26.89 (6.29)	0.266
Hypertension			
Yes	34 (45.3)	40 (54.1)	0.287
Diabetes			
Yes	25 (33.3)	20 (27.0)	0.402
Hyperlipidemia			
Yes	29 (38.7)	32 (43.2)	0.570
Smoking			
Yes	18 (24.0)	21 (28.4)	0.543
Physical activity			
Low	43 (57.3)	43 (58.1)	0.814
Moderate	26 (34.7)	27 (36.5)	
High	6 (8.0)	4 (5.4)	
E-DII^c	0.10 (-1.59 to 0.83)	-2.14 (-2.96 to 1.00)	0.003

BMI: Body mass index, **E-DII:** Energy-adjusted dietary inflammatory index

a- Values are expressed as frequency (%) and P-value based on Chi-square test

b- Values are expressed as mean (SD) and P-value based on Independent samples t-test

c- Values are expressed as median (percentile 25 to 75) and P-value based on Mann–Whitney U test

Table 2- Comparison of dietary intake in case and control groups ^a

Variable	Case (n=75)	Control (n=74)	MD (95% CI), P-value
Fat (g/day)	119.83 (39.70)	93.75 (39.52)	-26.08 (-38.91 to -13.25), <0.001
SFA (g/day)	24.05 (9.71)	23.79 (8.76)	-0.26 (-3.25 to 2.74), 0.865
MUFA (g/day)	30.45 (11.64)	23.33 (10.14)	-7.12 (-10.69 to -3.55), <0.001
PUFA (g/day)	42.71 (21.13)	34.18 (19.71)	-8.53 (-15.21 to -1.85), 0.013
Linoleic Acids (g/day)	39.51 (21.15)	29.63 (18.96)	-9.89 (-16.97 to -2.80), 0.007
Linolenic Acids (g/day)^b	0.29 (0.14 to 0.48)	0.14 (0.13 to 0.34)	-0.30 (-0.53 to -0.08), 0.004
Dietary Fiber (g/day)	15.25 (5.38)	13.95 (5.12)	-1.30 (-3.01 to 0.39), 0.131
Vitamin A (IU) ^b	822.10 (502.33 to 1416.00)	502.33 (375.67 to 802.67)	-551.58 (-856.38 to -246.78), 0.001
Vitamin D (µg) ^b	1.20 (0.04 to 2.48)	1.05 (0.09 to 2.00)	-0.30 (-0.78 to 0.17), 0.746
Vitamin K (µg) ^b	53.60 (42.16 to 93.57)	48.39 (21.78 to 70.28)	-12.21 (-27.59 to 3.17), 0.103
α-Tocopherol (mg) ^b	12.99 (4.97 to 19.65)	9.61 (5.03 to 12.99)	-2.79 (-4.82 to -0.07), 0.026
Vitamin C (mg) ^b	88.33 (55.67 to 127.73)	66.34 (50.00 to 99.84)	-13.66 (-29.79 to 2.51), 0.085
Calcium (mg/day)	853.33 (241.07)	695.72 (274.63)	-157.61 (-242.23 to -72.98), <0.001
Iron (mg/day)	15.73 (3.38)	13.22 (4.84)	-2.50 (-3.98 to -1.03), 0.001
Zinc (mg/day)	10.26 (3.98)	8.11 (2.70)	-2.15 (-3.35 to -0.95), 0.001
Copper (mg)	1.40 (0.42)	1.13 (0.35)	-0.27 (-0.41 to -0.14), <0.001
Selenium (mg)	0.12 (0.04)	0.11 (0.04)	-0.011 (-0.026 to 0.01), 0.151

MD: Mean difference, **CI:** confidence intervals, **SFA:** Saturated fatty acid, **MUFA:** Mono unsaturated fatty acid, **PUFA:** Poly unsaturated fatty acid

a- Values are expressed as mean (SD) and P-value based on Independent samples t-test

b- Values are expressed as median (percentile 25 to 75) and P-value based on Mann–Whitney U test

Table 3- Odds ratio and confidence intervals for the association between E-DII and biochemical parameters

Outcome	Model 1 ^a		Model 2 ^b		Model 3 ^c	
	<i>Case</i> (<i>n</i> =75)	<i>Control</i> (<i>n</i> =74)	<i>Case</i> (<i>n</i> =75)	<i>Control</i> (<i>n</i> =74)	<i>Case</i> (<i>n</i> =75)	<i>Control</i> (<i>n</i> =74)
	<i>OR (95% CI)</i>		<i>OR (95% CI)</i>		<i>OR (95% CI)</i>	
Triglycerides						
<i>E-DII</i>						
<i>Tertile 1 (<-2.0)</i>	Ref	Ref	Ref	Ref	Ref	Ref
<i>Tertile 2 (-2.0 to 0.6)</i>	1.43 (0.36 to 5.76)	0.70 (0.17 to 2.81)	1.25 (0.28 to 5.59)	0.74 (0.17 to 3.14)	1.19 (0.26 to 5.47)	0.663 (0.15 to 2.90)
<i>Tertile 3 (> 0.6)</i>	3.77 (0.85 to 16.64)	1.58 (0.55 to 4.56)	3.54 (0.69 to 18.24)	1.42 (0.47 to 4.29)	4.77 (0.82 to 27.56)	1.38 (0.45 to 4.23)
<i>P</i> _{for trend} [*]	0.049	0.440	0.072	0.568	0.048	0.620
Total cholesterol						
<i>E-DII</i>						
<i>Tertile 1 (<-2.0)</i>	Ref	Ref	Ref	Ref	Ref	Ref
<i>Tertile 2 (-2.0 to 0.6)</i>	0.73 (0.20 to 2.72)	0.45 (0.11 to 1.84)	0.67 (0.17 to 2.68)	0.45 (0.11 to 1.89)	0.67 (0.16 to 2.70)	0.46 (0.11 to 1.99)
<i>Tertile 3 (> 0.6)</i>	1.48 (0.36 to 6.02)	0.40 (0.13 to 1.20)	1.40 (0.31 to 6.30)	0.39 (0.13 to 1.17)	1.28 (0.28 to 5.90)	0.37 (0.12 to 1.15)
<i>P</i> _{for trend} [*]	0.394	0.091	0.445	0.084	0.571	0.077
LDL-c						
<i>E-DII</i>						
<i>Tertile 1 (<-2.0)</i>	Ref	Ref	Ref	Ref	Ref	Ref
<i>Tertile 2 (-2.0 to 0.6)</i>	0.82 (0.22 to 3.10)	1.88 (0.45 to 7.88)	0.85 (0.21 to 3.37)	1.88 (0.44 to 8.16)	0.83 (0.20 to 3.44)	1.80 (0.41 to 7.95)
<i>Tertile 3 (> 0.6)</i>	0.69 (0.17 to 2.82)	2.16 (0.71 to 6.60)	0.71 (0.16 to 3.17)	2.64 (0.82 to 8.51)	0.73 (0.15 to 3.48)	2.81 (0.84 to 9.37)
<i>P</i> _{for trend} [*]	0.599	0.165	0.635	0.100	0.696	0.090
HDL-c (mg/dL)						
<i>E-DII</i>						
<i>Tertile 1 (<-2.0)</i>	Ref	Ref	Ref	Ref	Ref	Ref

<i>Tertile 2 (-2.0 to 0.6)</i>	0.34 (0.06 to 1.81)	0.90 (0.22 to 3.72)	0.30 (0.05 to 1.67)	0.97 (0.23 to 4.08)	0.31 (0.05 to 1.72)	1.05 (0.24 to 4.60)
<i>Tertile 3 (> 0.6)</i>	0.13 (0.02 to 0.76)	1.12 (0.38 to 3.27)	0.10 (0.02 to 0.63)	1.17 (0.39 to 3.52)	0.09 (0.01 to 0.61)	1.18 (0.39 to 3.61)
<i>P</i> _{for trend} [*]	0.012	0.852	0.007	0.780	0.008	0.771
MDA						
<i>E-DII</i>						
<i>Tertile 1 (<-2.0)</i>	Ref	Ref	Ref	Ref	Ref	Ref
<i>Tertile 2 (-2.0 to 0.6)</i>	0.07 (0.00 to 0.596)	3.14 (0.76 to 12.98)	0.08 (0.00 to 0.73)	3.55 (0.77 to 16.30)	0.04 (0.00 to 0.50)	4.41 (0.88 to 22.18)
<i>Tertile 3 (> 0.6)</i>	0.11 (0.01 to 1.00)	1.25 (0.40 to 3.85)	0.14 (0.01 to 1.40)	0.99 (0.30 to 3.27)	0.09 (0.00 to 1.20)	1.17 (0.34 to 4.06)
<i>P</i> _{for trend} [*]	0.142	0.611	0.297	0.870	0.269	0.674
TAC						
<i>E-DII</i>						
<i>Tertile 1 (<-2.0)</i>	Ref	Ref	Ref	Ref	Ref	Ref
<i>Tertile 2 (-2.0 to 0.6)</i>	3.52 (0.61 to 20.24)	1.44 (0.35 to 5.84)	3.58 (0.59 to 21.87)	1.56 (0.38 to 6.48)	3.28 (0.52 to 20.55)	1.67 (0.39 to 7.10)
<i>Tertile 3 (> 0.6)</i>	9.56 (1.49 to 61.17)	0.55 (0.18 to 1.59)	10.03 (1.42 to 71.08)	0.55 (0.18 to 1.64)	12.87 (1.69 to 98.19)	0.50 (0.16 to 1.55)
<i>P</i> _{for trend} [*]	0.010	0.305	0.013	0.329	0.007	0.280
Hs-CRP						
<i>E-DII</i>						
<i>Tertile 1 (<-2.0)</i>	Ref	Ref	Ref	Ref	Ref	Ref
<i>Tertile 2 (-2.0 to 0.6)</i>	1.26 (0.33 to 4.85)	1.72 (0.42 to 7.08)	1.21 (0.30 to 4.91)	1.70 (0.39 to 7.45)	1.17 (0.27 to 5.00)	1.81 (0.39 to 8.32)
<i>Tertile 3 (> 0.6)</i>	0.81 (0.20 to 3.32)	0.65 (0.21 to 1.99)	0.74 (0.16 to 3.35)	0.76 (0.24 to 2.40)	0.77 (0.15 to 3.74)	0.85 (0.26 to 2.75)
<i>P</i> _{for trend} [*]	0.840	0.832	0.797	0.895	0.827	0.947
IL-6						
<i>E-DII</i>						
<i>Tertile 1 (<-2.0)</i>	Ref	Ref	Ref	Ref	Ref	Ref
<i>Tertile 2 (-2.0 to 0.6)</i>	0.38 (0.09 to 1.50)	1.89 (0.47 to 7.60)	0.23 (0.05 to 1.04)	1.47 (0.34 to 6.48)	0.22 (0.05 to 1.03)	1.53 (0.31 to 7.47)

<i>Tertile 3 (> 0.6)</i>	0.67 (0.16 to 2.91)	1.73 (0.58 to 5.14)	0.39 (0.08 to 1.97)	1.67 (0.53 to 5.28)	0.38 (0.07 to 1.97)	1.50 (0.46 to 4.90)
<i>P</i> for trend [*]	0.908	0.298	0.513	0.370	0.440	0.490

E-DII: Energy-adjusted dietary inflammatory index, **LDL-c:** Low-density lipoprotein cholesterol, **HDL-c:** High-density lipoprotein cholesterol, **TAC:** Total antioxidant capacity, **Hs-CRP:** High-sensitivity C-reactive protein, **IL-6:** Interleukin-6

- **P* for trend based on logistic regression
- a- Adjusted for age and sex
 - b- Adjusted for Model 1+ BMI, activity, smoking
 - c- Adjusted for Model 2+ diabetes, hyperlipidemia and hypertension

Table 4- E-DII and risk of Psoriasis and its severity (indexed by PASI and PSS)

	OR (95% CI)			
Outcome	E-DII Tertile 1 (<-2.0)	E-DII Tertile 2 (-2.0 to 0.6)	E-DII Tertile 3 (> 0.6)	P _{for trend} [*]
Psoriasis				
Model 1 ^a	Ref	10.82 (4.23 to 27.67)	3.68 (1.53 to 8.63)	0.004
Model 2 ^b	Ref	10.72 (4.17 to 27.56)	3.67 (1.53 to 8.76)	0.004
Model 3 ^c	Ref	11.38 (4.32 to 29.96)	3.64 (1.51 to 8.79)	0.005
PSS [†]				
Model 1 ^a	Ref	1.62 (0.42 to 6.20)	1.56 (0.37 to 6.49)	0.616
Model 2 ^b	Ref	1.70 (0.42 to 6.85)	1.70 (0.37 to 7.74)	0.568
Model 3 ^c	Ref	1.75 (0.36 to 8.38)	1.11 (0.19 to 6.49)	0.919
PASI [†]				
Model 1 ^a	Ref	6.54 (1.23 to 34.68)	7.69 (3.84 to 9.05)	0.010
Model 2 ^b	Ref	4.11 (1.26 to 4.20)	5.40 (2.90 to 7.60)	0.011
Model 3 ^c	Ref	2.22 (1.26 to 4.44)	3.64 (1.74 to 7.57)	0.015

E-DII: Energy-adjusted dietary inflammatory index, **PASI:** psoriasis area and severity index, **PSS:** Psoriasis Symptom Scale

^{*}P for trend based on logistic regression

a- Adjusted for age and sex

b- Adjusted for Model 1+ BMI, activity, smoking

c- Adjusted for Model 2+ diabetes, hyperlipidemia and hypertension

[†]Analysis limited to case group (Because these indices are not measured in non-psoriasis patients)