# Dietary inflammatory index and its association with alopecia areata: A cross sectional study.

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## Abstract

**Background: One of the most serious public health issues in the world, Alopecia Areata (AA), is regarded as a chronic inflammatory condition. Chronic inflammation is mainly controlled by diet. The aim of this study was to investigate the relationship between Dietary Inflammatory Index (DII) and Alopecia Areata.**

**Methods: A Food Frequency Questionnaire (FFQ) was used to evaluate food consumption, and the inflammatory diet index (DII) was calculated. The Severity of Alopecia Tool (SALT) score was used to assess the severity of the disease. A chi square test was utilized, and an independent t-test was used to assess quantitative data. Disturbing factors were adjusted in last analysis and $p<0.05$ was considered statistically significant.**

**Results: The mean age of the subjects was 53.84 ± 7.75 years old. The distribution of individuals in terms of SALT score and OPN level was significant on tertile of DII. The odds ratio for Alopecia Areata was increased by increasing DII (P=0.001).**

**Conclusion: Overall, this study suggested that by increasing DII and serum Osteopontin (OPN) level, the odds ratio of Alopecia Areata is increased. Further studies are required to clarify this relationship.**

**Keywords**: Dietary Inflammatory Index, Alopecia areata, Osteopontin, SALT score.

## Introduction

An autoimmune condition known as Alopecia Areata (AA) causes patchy, non-scarring hair loss on the scalp, face, and sometimes other body parts [1]. The onset of the disease often occurs before the age of thirty, men and women are equally affected [2].

Osteopontin (OPN) is an early activator of T lymphocytes that plays an important role it acts in some immunological diseases. Inflammation, tumor metastasis and cellular immunity are examples of situations that can affect the level of OPN [3, 4]. The level of OPN is higher in various inflammatory diseases such as multiple sclerosis, lupus erythematosus and rheumatoid arthritis [5].

There are few reports on the relationship between OPN and AA. Ganzetti et. al showed that in patients suffering from AA, a higher level of OPN can be detected in the blood circulation [6]. Rateb et.al also came to the same conclusion and realized that the level of OPN in tissues is also high [7]. However, Nasiri et.al showed that AA patients have a very low level of OPN compared to the control group [8].

Dietary Inflammatory Index (DII), a score system based on the literature, takes both the function of pro- and anti-inflammatory foods into account [9]. According to previous studies, a number of foods and minerals, including whole grains, fruits, vegetables, vitamin E, and vitamins C, have anti-inflammatory properties [10, 11]. Conversely, consumption of simple sugar, red meat, high-fat dairy products, and refined cereals was linked to an increase in inflammatory markers [12].

The DII calculation takes these dietary elements into account. The fact that DII measures total diet rather than specific nutrients and foods is a plus [13-15]. Additionally, this grading system is based on scientific publications' results rather than population averages or advised intakes [14]. Additionally, the DII was directly linked to colorectal cancer risk and longer hospital stays in individuals with colorectal cancer [16]. Additionally, those with higher DII increased the risk of developing the metabolic syndrome [14]. We hypothesized that DII may be related to alopecia areata. Therefore, the aim of this was to evaluate the dietary inflammatory index and its association with alopecia areata.

## Materials and methods

### *Study design and participants*

This cross-sectional study was performed on 427 Kurdish adults (53.84 ± 7.75 years old) who were referred to multi dermatology centers in Kalar city, Kurdistan regions. Based on another study were done in the Kalar city 440 Individuals was selected to be participated in the study [17]. who had at least one incomplete variable were excluded and finally, 427

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adults remained. Participants with diabetes, thyroid disorders and cancer were excluded from the study. Additionally, those having a total energy intake outside of the range of 800-4200 kcal/day for males and 600-3500 kcal/day for women. The study protocols were approved by the ethical committee of Kalar Technical College and by the Declaration of Helsinki. After informing subjects in detail about the study aims, all of them obtained written informed consent.

### *Demographic data*

A demographic survey was used to collect information on sex, age, marital status and socio economic status.

### *Physical activity*

The degree of physical activity of the individuals was evaluated using the validated International Physical Activity Questionnaire (IPAQ). Based on metabolic equivalents (METs), recorded quantities were displayed and divided into three types (very low: <600, low: 600–3000, and moderate and high > 3000 MET-min/week) [18].

### *Anthropometry*

With an accuracy of 100 gr, the participant's weight was measured using the InBody 770 instrument (Inbody Co, Seoul, Korea) while wearing little clothing and no shoes. With a precision of 0.1 cm, height was measured using an automated stadiometer BSM 370 (Biospace Co., Seoul, Korea) while the subject was standing without shoes.

### *Blood sampling and biochemical assays*

7 mL of blood samples were taken into the clot tubes after the subject had been fasted for eight hours. Serum samples were centrifuged at 4°C for 10-15 minutes after which they were kept at 70°C until bioanalysis. Enzyme-linked immunosorbent assay (ELISA) and EDTA kit (Biokit China) were used to measure OPN serum level.

### *Dietary assessment and DII calculation*

By using a valid and accurate 168-item Food Frequency Questionnaire (FFQ), average dietary consumption was calculated [19] this was conducted by qualified dietitians *via* in-person interviews and contained a list of goods and a standard size of each food item. Each item's frequency of intake on a daily, weekly, monthly, and yearly basis was reported by participants.

Household measurements [20] were used to convert eaten food portion sizes to grams, which were then computed using a customized version of the NUTRITIONIST IV program for Iranian cuisine (version 7.0; N-Squared Computing, Salem, OR, USA).

To determine DII, we used the technique developed by Shivappa et al. [21]. In the current research, information on 21 dietary components including calories, carbohydrate, fat, protein, whole grains, fiber, legumes, cholesterol, dairy, fruits, vegetables, processed meat, nut, sugar, oils, and vitamin (A, D, K, E, B12 and C) was gathered. According to Shivappa et.al published adjusted scores were used to construct the DII score [21]. Each patient's intake was multiplied by the ratings for the overall inflammatory effects. The scores for each dietary item were then added together. The more pro-inflammatory the diet, the higher the DII score. The more anti-inflammatory diets, the more negative values are shown. The following cut points were used to group the DII tertile cut points: Tertile 1 ≤ −1.122; Tertile 2 = −1.123 to 0.651; Tertile 3 ≥ 0.652.

### *SALT Score*

The severity of alopecia tool (SALT) score was used to assess the severity of the disease [22].

Patients with AA were separate into two groups, the moderate to severe AA group with SALT scores ≥25 and the mild AA group with SALT scores <25.

A SALT score of 25 is the point at which steroid minipulse therapy is considered as a treatment option in the AA guidelines published by the Japanese Dermatological Association [23].

### *Statistical analysis*

For data analysis, the Statistical Package for the Social Sciences (SPSS) software version 26.0 was utilized. Data was verified for normality and outliers before to analysis. To compare categorical variables, a chi square test was used while quantitative variables were analyzed using an independent t-test. To determine the relationship between factors and alopecia areata, binary logistic regression was used. The statistical significance level was set at P 0.05.

## Results

A total of 427 males and females aged 42 to 64 years were analysed in this study. The DII score in the Tertiles 3 was about 1.89±0.69. **Table 1** presented the basic characteristics of the study participants according to Dietary inflammatory index Tertiles. Participants included in the highest tertiles of DII scores were significantly younger than those in the lowest quartile (P<0.001). According to DII tertiles, participants with the most intake of anti-inflammatory foods had significantly lower SALT score (P<0.001) and OPN levels (P<0.001).

**Table 2** presented the daily intake of macro-and micronutrients according to DII tertiles. Daily intake of Energy, Lipid, whole grains, cholesterol, processed meat, sugar and oil were significantly higher in the highest tertiles of DII (P<0.001). while daily intake of CHO, protein, fibre, legumes, diary, fruits, vegetables, nuts, Vit (A, K, B12, C, D and E) were significantly higher in the lowest tertiles of DII (P<0.001).

Participants with SALT score ≥25 significantly had a higher serum OPN level and BMI than participants with SALT score <25 (P<0.001).

The mean daily intake of Energy, Lipid, whole grains, cholesterol, processed meat, sugar and oil were significantly higher in the participants with SALT score ≥25 (**Table 3**).

Logistic regression analysis was done to estimate the association of Pro-inflammatory and anti-inflammatory diet with AA (adjusted for BMI) (**Table 4**), and the results revealed a positive association between intake of Pro-inflammatory diet and AA with statistical significance P < 0.001; and a negative association between intake of anti-inflammatory diet with AA (P < 0.001).

***Table 1.*** *Baseline characteristics of the study population according to Dietary inflammatory Index (DII) tertiles (data, N=427).*

| Variable | Dietary Inflammatory Index (DII) | | | P value* |
|---|---|---|---|---|
| | Terties 1 N=142 | Tertiles 2 N=138 | Tertiles 3 N=147 | |
| | Mean± SD / n (%) | | | |
| DII score | -2.63± 0.87 | -0.24±0.53 | 1.89±0.69 | <0.001 |
| Age | 49.81±7.04 | 52.82±6.76 | 58.70±6.64 | <0.001 |
| BMI (kg/m$^2$) | 26.88±5.74 | 27.74±4.90 | 29.52±5.02 | <0.001 |
| WHR | 0.94±0.03 | 0.93±0.03 | 0.94±0.03 | 0.209 |
| SALT score | 11.54±6.76 | 32.72±5.16 | 54.468.27 | <0.001 |
| OPN ng/dL | 19.83±9.21 | 21.82±9.50 | 25.43±11.80 | <0.001 |
| **Gender** | | | | |
| Male | 70(16.4) | 95(22.2) | 115(26.9) | <0.001 |
| Female | 72(16.9) | 43(10.1) | 32(7.5) | |
| **Marital status** | | | | |
| Married | 93(21.8) | 134(31.4) | 77(18) | <0.001 |
| Single | 22(5.2) | 1(0.2) | 62(14.5) | |
| Widowed/Divorced | 27(6.3) | 3(0.7) | 8(1.9) | |
| **Socio-economic status** | | | | |
| 1(lowest) | 49(11.5) | 7(1.6) | 19(4.4) | <0.001 |
| 2 | 63(14.8) | 123(28.8) | 117(27.4) | |
| 3(highest) | 30(7) | 8(1.9) | 11(2.6) | |
| **Physical activity (Met-h/day)** | | | | |
| Light | 20(4.7) | 36(8.4) | 78(18.3) | <0.001 |
| Moderate | 108(25.5) | 29(6.8) | 31(7.3) | |
| High | 13(3.3) | 73(17.1) | 38(8.9) | |

BMI: Body mass index; WHR: Waist hip ratio; OPN: Osteopontin

Tertile 1 ≤ −1.122; Tertile 2 = −1.123 to 0.651; Tertile 3 ≥ 0.652

*Analysis of variance (ANOVA) and Chi square, P< 0.05

***Table 2.*** *Nutrients intake according to the Dietary inflammatory index Tertiles (data, N=427).*

| Food parameters | Dietary inflammatory index (DII) Mean± SD | | | P value* |
|---|---|---|---|---|
| | Terties 1 N=142 | Tertiles 2 N=138 | Tertiles 3 N=147 | |
| Energy intake (kcal/d) | 1511.13±162.02 | 1516.14±172.90 | 2272.82±233.70 | <0.001 |
| Carbohydrate (%E) | 163.89±30.85 | 154.05±28.27 | 134.03±22.24 | <0.001 |
| Lipid (%E) | 69.06±12.01 | 80.94±14.27 | 89.44±15.58 | <0.001 |
| Protein (%E) | 58.48±6.77 | 54.10±6.99 | 48.22±5.59 | <0.001 |
| Whole grains (gr/d) | 300.41±56.81 | 351.37±50.77 | 370.25±53.40 | <0.001 |
| Fibre (gr/d) | 18.86±4.95 | 15.71±4.71 | 10.67±4.13 | <0.001 |
| Legumes (gr/d) | 29.09±12.83 | 23.76±10.77 | 23.94±10.60 | <0.001 |
| Cholesterol (gr/d) | 103.34±3.50 | 115.44±43.11 | 129.72±44.54 | <0.001 |
| Dairy (gr/d) | 440.03±157.80 | 372.33±147.77 | 348.49±136.66 | <0.001 |
| Fruit (gr/d) | 217.12±32.98 | 189.42±32.41 | 156.31±29.77 | <0.001 |
| Vegetables (gr/d) | 317.62±82.21 | 279.85±76.47 | 238.45±176.11 | <0.001 |
| Processed meat (gr/d) | 18.94±7.01 | 26.15±8.32 | 30.22±9.02 | <0.001 |
| Sugar (gr/d) | 39.44±18.17 | 61.95±26.64 | 76.26±32.85 | 0.928 |
| Nuts (gr/d) | 10.80±2.90 | 7.36±2.41 | 4.98±1.72 | <0.001 |
| Oil (gr/d) | 12.14±3.01 | 17.28±3.77 | 22.45±3.81 | <0.001 |
| Vitamin A (mcg) | 738.11±77.65 | 724.80±71.08 | 596.47±38.10 | <0.001 |
| Vitamin K (mg) | 93.53±21.07 | 73.14±18.71 | 61.77±17.02 | <0.001 |
| Vitamin B12 (mcg) | 3.37±1.35 | 1.82±0.74 | 1.08±0.51 | <0.001 |
| Vitamin C (mg) | 116.66±56.32 | 107.63±50.55 | 92.64±48.20 | <0.001 |
| Vitamin D (mcg) | 0.68±0.31 | 0.46±0.17 | 0.10±0.03 | <0.001 |
| Vitamin E (mg) | 6.47±1.54 | 4.16±1.09 | 3.16±1.11 | <0.001 |

Tertile 1 ≤ −1.122; Tertile 2 = −1.123 to 0.651; Tertile 3 ≥ 0.652

*Analysis of variance (ANOVA), P< 0.05

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***Table 3.*** *Comparison of dietary pattern in participants according to SALT score (N=427).*

| Variables | SALT score <25 N=142 | SALT score ≥25 N=285 | P value |
|---|---|---|---|
| | Mean± SD | | |
| OPN (ng/dL) | 19.83±9.21 | 23.68±10.88 | <0.001 |
| BMI (kg/m$^2$) | 26.88±5.74 | 28.66±5.03 | 0.001 |
| **Food Parameters** | | | |
| Energy intake (kcal/d) | 1511.13±162.02 | 1928.22±411.63 | <0.001 |
| Carbohydrate (%E) | 163.89±30.85 | 143.73±27.21 | <0.001 |
| Lipid (%E) | 69.06±12.01 | 85.32±15.53 | <0.001 |
| Protein (%E) | 58.48±6.77 | 51.07±6.95 | <0.001 |
| Whole grains (gr/d) | 300.41±56.81 | 361.11±52.90 | <0.001 |
| Fibre (gr/d) | 18.86±4.95 | 13.11±5.08 | <0.001 |
| Legumes (gr/d) | 28.09±12.83 | 23.85±10.66 | <0.001 |
| Cholesterol (gr/d) | 103.34±37.50 | 122.80±44.36 | <0.001 |
| Dairy (gr/d) | 440.03±157.80 | 360.03±142.39 | <0.001 |
| Fruit (gr/d) | 217.12±32.98 | 172.34±35.17 | <0.001 |
| Vegetables (gr/d) | 317.62±82.21 | 290.07±94.66 | <0.001 |
| Processed meat (gr/d) | 18.94±7.01 | 28.25±9.01 | <0.001 |
| Suger (gr/d) | 39.44±18.17 | 69.33±30.52 | <0.001 |
| Nuts (gr/d) | 10.80±2.90 | 6.13±2.38 | <0.001 |
| Oil (gr/d) | 12.14±3.01 | 19.95±4.32 | <0.001 |
| Vitamin A (mcg) | 783.11±77.66 | 658.61±85.51 | <0.001 |
| Vitamin K (mg) | 93.95±21.07 | 67.28±18.71 | <0.001 |
| Vitamin B12 (mcg) | 3.37±1.35 | 1.44±0.73 | <0.001 |
| Vitamin C (mg) | 116.66±56.32 | 99.90±49.83 | 0.002 |
| Vitamin D (mcg) | 0.68±0.31 | 0.27±0.21 | <0.001 |
| Vitamin E (mg) | 6.47±1.54 | 3.65±1.21 | <0.001 |

*Using t-test, P< 0.05

***Table 4.*** *Association between the dietary inflammatory index (DII) and the alopecia by logistic regression analysis.*

| Variables | Odds ratio | 95% CI | p-value |
|---|---|---|---|
| **Pro-inflammatory** | | | |
| Fat | 1.08 | 1.06 - 1.10 | <0.001 |
| Processed meat | 1.14 | 1.11 – 1.18 | <0.001 |
| Oil | 1.93 | 1.68 – 2.21 | <0.001 |
| Cholesterol | 1.01 | 1.00 – 1.01 | <0.001 |
| Sugar | 1.04 | 1.03 – 1.05 | <0.001 |
| **Anti-inflammatory** | | | |
| Fibre | 0.806 | 0.769 – 0.846 | <0.001 |
| Fruits | 0.963 | 0.956– 0.971 | <0.001 |
| Nuts | 0.549 | 0.491 – 0.614 | <0.001 |

**Note:** adjusted for BMI

## Discussion

To the best of our knowledge, this is the first study which examined association between Dietary inflammatory index and its association with alopecia areata. Participants who had greater DII (matching to diets' heightened proinflammatory potential) were more likely to develop AA than those who had lower DII (corresponding to anti-inflammatory diets). Additionally, there were significant differences in food groups and nutrient consumption amongst DII tertiles. Various studies have shown a strong correlation between DII and serum inflammatory markers [24-26].

AA is an autoimmune disease that causes hair loss due to the effect on hair follicles. Although its exact cause has not been identified yet. An autoimmune condition known as Alopecia Areata (AA) causes patchy, non-scarring hair loss on the scalp, face, and sometimes other body parts [27]. The onset and progression of AA are influenced by a number of hereditary and environmental variables, including nutrition. A person's diet may modify the etiology of AA by affecting their immune system and hair follicles [28]. It was proposed that a Western diet heavy in fat, sugar, and salt and lacking in fiber may encourage the onset and severe autoimmune diseases [29]. Patients in the third tertile of DII ingested more grains, as can be shown in the results section. Similar to our findings, a longitudinal research carried out in Italy revealed that those in the highest quartile of the DII consumed more bread [30]. Higher concentrations of inflammatory markers were linked to intake of refined grains [31, 32]. It follows that this discovery will probably be confirmed in populations who consume large quantities of refined grains.

We noticed that those in the highest tertile of the DII eat more fruits and vegetables. Consumption of fruits and vegetables

had a negative relationship with inflammation [33]. Several nutrients found in abundance in fruits and vegetables, such as dietary fiber have a negative overall inflammatory impact score when calculating the DII [34]. As a result, increased fruit and vegetable consumption was expectable among individuals with lower DII.

We found that dairy consumption was considerably greater in the first tertile of DII than in the top tertile. According to a prior research, following a diet limited in high-fat dairy products reduced the levels of inflammatory markers [35].

The findings showed a slightly significant increase in meat intake among DII participants in the first tertile compared to the last tertile. According to earlier research, eating red meat increased the levels of inflammatory markers [34, 36].

Retinol intake, generally known as vitamin A1, was associated with SALT score. This finding implies that Retinoic Acid (RA), an active retinol metabolite, may worsen AA as a consequence of increased retinol consumption. By triggering the entry of hair follicle stem cells into the anagen phase by signaling *via* the wingless-type mouse mammary tumor virus integration site family, retinoic acid may make hair follicles more vulnerable to assault by NKG2D+ effector cells [37].

In our analysis and another research revealed that a lack of micronutrients, such as vitamins or minerals, may encourage the development of AA. As serum levels of vitamin D, zinc, and folate tended to be lower in patients with AA than in healthy controls [28]. According to certain research, dietary vitamin A levels that are too high or too low may encourage the development of AA [38].

However, OPN is a known factor that can be produced by some immune cells and can be increased in some immunological diseases. The relationship between OPN and AA has been studied, and the results have shown that, like some other inflammatory diseases, OPN serum levels increase in patients with AA. In a case-control study by Ganzetti et al., OPN serum levels were measured before and 12 months after starting immunotherapy with 3 and 2-diphenylcyclopropenone (DPCP) in 40 patients and compared with 20 healthy individuals. The results of this study show that OPN serum level was significantly higher in AA patients; but after using DPCP, there was no significant reduction [6].

In another study conducted by Nasiri et.al, OPN serum level was measured in AA patients and control group. The results of this study showed that OPN serum level in AA patients it was significantly lower than the control group [8].

Rateb and colleagues examined OPN gene expression in patients with AA and compared it with the control group and concluded that OPN tissue level measured by real-time PCR in patients was significantly higher than the control group [7].

In our study, there was a significant statistical relationship between SALT score and OPN serum level. In the study of Rateb et al., there was no significant relationship between OPN mRNA expression and SALT score in patients [7]. Also, in the study of Nasiri et al, there was no relationship between disease severity and OPN serum level [8].

The current study has numerous strengths: first, it is one of the first studies in Iraq to evaluate the association between DII and AA; second, adjusting for several possible confounding variables may offer a strong link between DII and AA. Although there are certain limits, DII may have some limitations linked to dietary regimens. Only 21 FFQ items out of 45 dietary factors were available for computing DII; hence, there may be some missing data on unconsidered products. Finally, since the research was cross-sectional, determining causation is challenging.

## Conclusion

In summary, our findings showed that a strong pro-inflammatory diet was related with an increased risk of AA in the elderly. Our results might help us understand the underlying processes of dietary inflammation and AA. Given the cross-sectional methodology, further prospective cohort studies are required.

## Declarations

### *Ethics approval and consent to participate*

The Ethics Committee of Slemani Polytechnic University, Kalar technical college approved the study. All methods were carried out by relevant guidelines and regulations. All the participants were provided oral and written informed consent. All methods were carried out by relevant guidelines and regulations. This study was conducted by the Declaration of Helsinki.

### *Consent for publication*

Not applicable.

### *Availability of data and materials*

The data analyzed in the study are available from the corresponding author upon reasonable request.

### *Competing interests*

The authors declare no conflicts of interest.

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This research was supported by Slemani Polytechnic University.

### *Author contribution*

The author confirms sole responsibility for the following: study conception and design, data collection, analysis and interpretation of results, and manuscript preparation.

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