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# The role of the balance between lipid mediator resolvin D1 and interleukin-17 in the development of psoriasis

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## MAIN POINTS

- Serum IL-17 levels were significantly higher, and Resolvin D1 (RvD1) levels were markedly lower, in patients with psoriasis than in healthy controls, demonstrating a disrupted inflammatory–proresolving balance.
- IL-17 was positively correlated with PASI and NLR, whereas RvD1 was negatively correlated with PASI and NLR, indicating that these biomarkers reflect clinical severity and systemic inflammation.
- Combined ROC analysis of IL-17 and RvD1 increased diagnostic performance (AUC: 0.814), outperforming each marker alone.
- After adjustment for confounders, high IL-17 and low RvD1 levels independently predicted psoriasis risk, suggesting their utility in diagnosis and disease monitoring.

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## ABSTRACT

**Aim:** To determine serum IL-17 and resolvin D1 (RvD1) levels in patients with psoriasis and to assess their relationships with the psoriasis area severity index (PASI) and other inflammatory markers.

**Materials and Methods:** A total of 60 participants, 30 patients diagnosed with psoriasis vulgaris and 30 controls, were included in the study. Psoriasis was diagnosed based on the morphological appearance of the lesions. Psoriasis Area and Severity Index (PASI) scores were calculated for patients with psoriasis. Serum RvD1 and IL-7 levels were measured by ELISA. The predictive performance of IL-17 and RvD1 was assessed using Receiver Operating Characteristic (ROC) curve analysis. Optimal cut-off points were determined using the Youden index. The independent associations of IL-17 and RvD1 with psoriasis were investigated by multiple logistic regression analysis after adjusting for age, BMI, and gender.

**Results:** Serum IL-17 level in the psoriasis group was significantly higher than in the control group [609.32 (436.75-842.13) vs. 354.25 (260.0-659.13), p<0.001], whereas the RvD1 level was significantly lower than in the control group [7.38 (5.32-10.82) vs. 11.3 (8.7-16.1), p=0.004]. IL-17 was positively correlated with PASI and NLR, and negatively correlated with RvD1 and lymphocyte count. RvD1 was negatively correlated with PASI and NLR, and positively correlated with lymphocyte count. Although the diagnostic sensitivities of both markers were similar, their efficiency increased when combined (AUC: 0.814 (0.693-0.903)). Logistic regression analysis revealed that high IL-17 and low RvD1 were associated with psoriasis.

**Conclusion:** After adjusting for potential confounders, high IL-17 and low RvD1 were found to be independent risk factors for the development of psoriasis. Although the diagnostic sensitivity of both molecules was similar for psoriasis, combining the two molecules increased diagnostic yield.

**Keywords:** Psoriasis, IL-17, Resolvin D1, PASI, Inflammation
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## INTRODUCTION

Inflammation is a process that involves prostaglandins, thromboxanes, and leukotrienes , which result from the enzymatic degradation of arachidonic acid and omega-3 fatty acids by cyclooxygenases and lipoxygenases. Although a physiological amount of inflammation is necessary for many cellular functions, chronicity and increased intensity of inflammation can lead to pathological inflammation and cell damage [1,2]. Psoriasis is an immune-mediated, systemic and chronic inflammatory skin disease whose exact etiology has not been clearly established [3]. With a prevalence of 2-3%, psoriasis is considered the most serious non-infectious skin pathology [4]. Sharply demarcated, erythematous, and scaly skin lesions are considered indicators of the inflammatory nature of the disease. Although physiological inflammation is a self-limiting process, resolution of inflammation in psoriasis may

be followed by relapse. Anti-inflammatory and immunomodulatory pro-soluble lipid mediators such as resolvin, protectin, and maresin play a role in the resolution of chronic inflammation associated with psoriasis [5,6].

Resolvin D1 (RvD1) is an omega-3 polyunsaturated fatty acid-derived lipid mediator involved in the resolution of inflammation [7]. RvD1 expression defects in blood and skin lesions of patients with psoriasis have been shown in isolated studies [3]. In particular, the anti-inflammatory lipid mediator RvD1 has been shown to be downregulated in active psoriasis [8]. In contrast to pro-resolvins, increased T cell stimulation triggers inflammation in psoriatic skin lesions, leading to disease progression [9]. The decreased production of anti-inflammatory cytokines such as RvD1 in psoriasis is mediated by T cells activated by interleukin 17 [9]. The remarkable increase in IL-17A and other family members in psoriasis has drawn attention to this molecule in the pathogenesis of the disease [10]. Moreover, IL-17 overexpression contributes significantly to the formation of psoriatic lesions by causing hyperproliferation and differentiation defects of epidermal keratinocytes [4].

While resolvin D1 (RvD1) exerts anti-inflammatory and pro-resolving effects that may counteract the development of psoriasis [3], overexpression of interleukin-17 (IL-17) is known to promote disease progression [9]. Although the pathogenic role of IL-17 in psoriasis has been well established for many years, circulating levels of the pro-resolving lipid mediator RvD1 have been far less extensively investigated. To date, no study has specifically examined the imbalance between a pro-inflammatory cytokine and a pro-resolving lipid mediator in psoriasis. Therefore, the primary novelty of the present study lies not only in evaluating serum RvD1 levels but also in investigating the balance between IL-17 and RvD1 and their relationship with disease severity (assessed by the Psoriasis Area and Severity Index (PASI)) and other inflammatory markers.

## MATERIALS AND METHODS

### *Patients selection*

This case-control study was approved by the Fırat University Non-Interventional Research Ethics Committee (Approval no: 29957, Date: 27.12.2024), after which patient selection and sample collection commenced. A total of 60 participants were included in the study: 30 had psoriasis vulgaris and 30 were controls. Only patients with chronic plaque-type psoriasis (psoriasis vulgaris) were included in the study. Patients with other clinical subtypes of psoriasis (guttate, pustular, and erythrodermic) and those with psoriatic arthritis were excluded to ensure a homogeneous study population. The diagnosis of psoriasis was confirmed by the presence of a symmetrical, sharply demarcated, erythematous plaque covered with silver scales. The diagnosis of psoriasis was based on characteristic clinical findings. Although histopathological confirmation was not routinely performed, this approach reflects standard clinical practice. PASI was calculated by accounting for both the body surface area affected by psoriasis and the severity of redness, swelling, and scaling. A PASI score of 10 or higher was considered to have moderate to severe psoriasis [11]. Participants were selected from patients presenting to the Malatya Gözde Hospital Dermatology Clinic. The control group consisted of age- and BMI-matched individuals without psoriasis, chronic inflammatory or autoimmune diseases, malignancy, or systemic infection. None of the controls were receiving systemic anti-inflammatory or immunomodulatory medications. Controls were recruited from among individuals attending the dermatology outpatient clinic for non-inflammatory, benign conditions. Those using anti-inflammatory drugs (including TNF alpha or IL-17 blockers), those with systemic inflammatory diseases, and those in the acute attack phase of the disease were excluded from the study. Patients receiving systemic anti-inflammatory drugs, TNF-α inhibitors, IL-17 blockers, topical corticosteroids, topical immunomodulators, phototherapy, apremilast, IL-23 inhibitors, or any systemic immunosuppressive or immunomodulatory therapy within the previous 3 months were excluded. Following an overnight fast of 8-10 hours, blood samples were collected from both groups and stored until analysis. All blood samples were collected between 08:00 and 10:00 a.m. after an overnight fast to minimize potential circadian variation in inflammatory mediators. Complete blood count (CBC) parameters (WBC, neutrophil and lymphocyte counts) were measured using the Mindray BC-6000 autohematology analyzer (Mindray Diagnostics, Shenzhen, China). The neutrophil-to-lymphocyte ratio (NLR) was calculated by dividing the neutrophil count by the lymphocyte count, both obtained from the complete blood count. The height and weight of the participants in the psoriasis and control groups were measured, and their BMI values were calculated [weight (kg)/square of height (kg/m$^2$)].

### *Measurement of serum IL-17 and RvD1 with ELISA*

IL-17 and RvD1 levels in serum samples were determined by ELISA method using Human ELISA kits (Catalog no: 201-

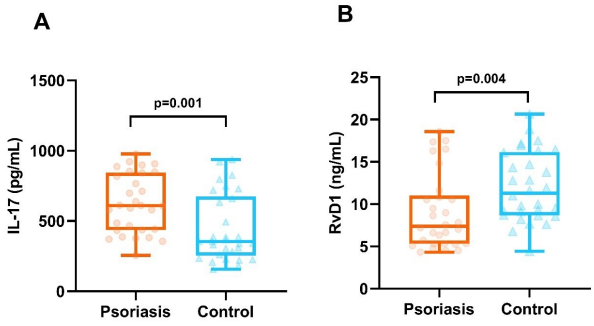


**Figure 1.** Graphical representation of serum values of IL-17 and RvD1 in psoriasis and control groups.


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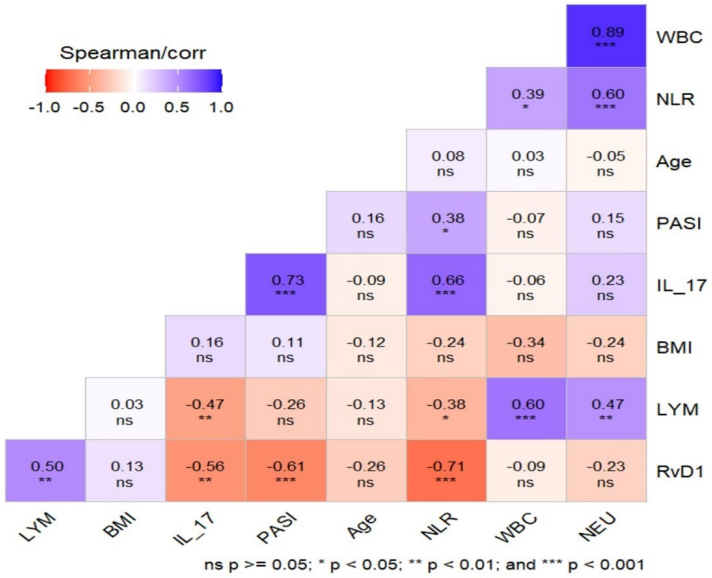


**Figure 2.** Graphical representation of correlation matrices of IL-17, RvD1 and other variables. Blue colors indicate positive correlations and red colors indicate negative correlations.

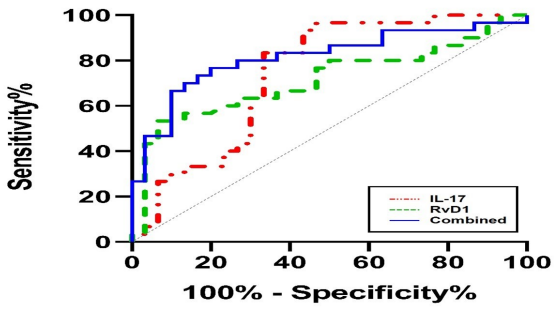


**Figure 3.** ROC curve analysis of IL-17, RvD1 and their combination in the diagnosis of psoriasis.

12-0143, 201-12-9313, Sunred Biotechnology Company, Shanghai, CHINA, respectively). The measurement range of the IL-17 kit was 15 pg/mL – 1000 pg/mL, and the minimum measurable level was 12.03 pg/mL. The measurement range of the RvD1 kit was 0.15–30 ng/mL, and the minimum measurable level was 0.114 ng/mL. The intra-assay CV values for both kits were <10%, while the inter-assay CV valueswere <12%. Absorbances were measured at 450 nm using a Bio-Tek ELx800 (BioTek Instruments, USA) microplate reader. Test results were reported as pg/mL for IL-17 and ng/mL for resolvin-D1, respectively.

### *Statistical analysis*

Statistical analysis of the data was performed using SPSS v. 27 (IBM Corp., Armonk, NY). Graphpad Prism 8.3.0 (GraphPad Software, San Diego, California, USA) software was used for graphical drawing. Normality of the variables was assessed using the Kolmogorov–Smirnov test. Continuous variables were given as mean±standard deviation or median ($25^{th}$ and $75^{th}$ percentile) according to the normality of the distribution. Parameters conforming to a normal distribution were compared using the independent samples t test, whereas those that did not conform were compared using the Mann-Whitney U test. Categorical variables were reported as counts and percentages, and differences between categories were assessed using the Chi-square test. The relationships between the parameters were examined using the Spearman correlation test. The relationships between the parameters were examined using the Spearman correlation test. The predictive performance of the laboratory measurements was assessed using Receiver Operating Characteristic (ROC) curve analysis. For the combined ROC analysis, predicted probabilities derived from the multivariable logistic regression model that included both IL-17 and resolvin D1 were used. Optimal cut-off points were determined by using the Youden index. The independent association between IL-17 and RvD1 with psoriasis was investigated by multiple logistic regression analysis after adjusting for age, BMI and gender. Multicollinearity was assessed using variance inflation factors (VIFs), and all variables had acceptable VIFs (<2.5). Model fit was evaluated using the Hosmer–Lemeshow goodness-of-fit test and Nagelkerke $R^2$. Correlation heatmap was generated via the Metan R package [12]. P<0.05 was considered statistically significant.

## RESULTS

As shown in Table 1, participants in the psoriasis and control groups were similar in mean age, BMI, and gender distribution. Serum IL-17 levels in the psoriasis group were significantly higher than those in the control group [609.32(436.75-842.13) vs. 354.25 (260.0-659.13), p<0.001]. Conversely, serum RvD1 levels in the psoriasis group were significantly lower than those in the control group [7.38 (5.32-10.82) vs.


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**Table 1.** Comparison of demographic characteristics of participants in the psoriasis and control groups as well as RvD1 and IL-17 levels.

| Parameters | Psoriasis (n =30) | Control (n = 30) | p-value |
|---|---|---|---|
| Age (yrs) | 34.87±6.85 | 35.97±7.89 | 0.567[a] |
| Body Mass Index (kg/m2) | 24.18±1.40 | 24.4±2.3 | 0.650[a] |
| Gender (male/female) | 19/11 (63.3%) | 17/13 (56.7%) | 0.792[c] |
| IL-17 (pg/mL) | 609.32 (436.75-842.13) | 354.25 (260.0-659.13) | **0.001[b]** |
| RvD1 (ng/mL) | 7.38 (5.32-10.82) | 11.3 (8.7-16.1) | **0.004[b]** |
| PASI | 4.5 (3.0-9.1) | - | - |
| WBC (109/L) | 8.79±1.95 | 6.51±1.44 | **<0.001[a]** |
| Neutrophil count (109/L) | 5.49±1.41 | 3.67±1.09 | **<0.001[a]** |
| Lymphocyte count (109/L) | 2.48±0.55 | 2.04±0.56 | **0.003[a]** |
| NLR | 2.25±0.49 | 1.89±0.65 | **0.018[a]** |

[a] Student t-test, [b] Mann-Whitney U test, [c] Chi-Square test. IL-17: Interleukin-17; RvD1: Resolvin-D1; PASI: Psoriasis Area Severity Index; WBC: white blood cell count; NLR: Neutrophil to lymphocyte ratio.

**Table 2.** Correlation analysis between IL-17, RvD1 and other variables in psoriasis patients.

| | IL-17 | | RvD1 | |
|---|---|---|---|---|
| | r | p | r | p |
| RvD1 | **-0.559** | **0.001** | - | - |
| Age | -0.086 | 0.652 | -0.259 | 0.168 |
| Body Mass Index | 0.155 | 0.413 | 0.129 | 0.496 |
| PASI | **0.729** | **<0.001** | **-0.605** | **<0.001** |
| WBC | -0.058 | 0.761 | -0.098 | 0.608 |
| Neutrophil count | 0.226 | 0.230 | -0.229 | 0.225 |
| Lymphocyte count | **-0.475** | **0.008** | **0.500** | **0.005** |
| Neutrophil/lymphocyte ratio | **0.656** | **<0.001** | **-0.706** | **<0.001** |

r: Correlation coefficient.

**Table 3.** Cut-off value, sensitivity and specificity of IL-17 and RvD1, individually and when combined, in diagnosing psoriasis.

| | Cut-off | Sensitivity | Specificity | AUC (95.0% CI) | p |
|---|---|---|---|---|---|
| IL-17 | >354.8 | 96.7% | 53.3% | 0.742 (0.613 - 0.847) | **<0.001** |
| RvD1 | ≤7.56 | 53.3% | 93.3% | 0.715 (0.584- 0.824) | **0.002** |
| Combined | - | 80.0% | 76.7% | 0.814 (0.693- 0.903) | **<0.001** |

AUC: Area under ROC curve; CI: Confidence intervals.

**Table 4.** Logistic regression analysis of IL-17 and RvD1 after adjusting for age, body mass index, and gender.

| | Unadjusted | | Adjusted [(1)] | |
|---|---|---|---|---|
| | OR (95% CI) | P | OR (95% CI) | p |
| IL-17 | 1.004 (1.001 -- 1.006) | 0.005 | 1.004 (1.001 -- 1.006) | **0.006** |
| RvD1 | 0.851 (0.749 -- 0.967) | 0.013 | 0.829 (0.721 -- 0.953) | **0.009** |

OR: Odds ratio, CI: Confidence interval, (1) Adjusted with age, body mass index, and gender.

11.3 (8.7-16.1), p=0.004]. Figure 1 shows the distributions of IL-17 and RvD1 across the groups. PASI in psoriasis patients was recorded as 4.5 (3.0-9.1). Neutrophil count, WBC, lymphocyte count, and NLR were significantly higher in psoriasis patients than in the control group (Table 1).

IL-17 was positively correlated with PASI and NLR and negatively correlated with RvD1 and lymphocyte count. RvD1 was negatively correlated with PASI and NLR and positively correlated with lymphocyte count. The correlations between IL-17, RvD1, and other parameters are detailed in both Table 2 and the correlation matrix (Figure 2).

As shown in Table 3 and Figure 3, IL-17 diagnosed psoriasis with 96.7% sensitivity and 53.3% specificity; the AUC (area under the curve) for ROC analysis was 0.742 (0.613 - 0.847) at a cut-off value of >354.8. RvD1 diagnosed psoriasis with 53.3% sensitivity and 93.3% specificity and AUC for ROC analysis was 0.715 (0.584- 0.824) at a cut-off value of ≤ 7.56. When IL-17 and RvD1 were combined, sensitivity and specificity for diagnosing psoriasis were 80% and 76.7%, respectively. When both markers were combined, the AUC reached 0.814 (0.693-0.903). This combination significantly increased diagnostic efficiency.

Multiple logistic regression analysis revealed that high IL-17 and low RvD1 were independently associated with psoria-


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sis (Table 4). After adjusting for age, BMI, and gender, a 1-unit (1 pg/mL) increase in IL-17 levels was significantly associated with a 1.004-fold increase in the odds of having psoriasis (OR: 1.004, 95% CI: 1.001 61.006; p=0.006). After adjusting for age, BMI, and gender, the odds of psoriasis decreased by 17% with each 1-unit (1 ng/mL) increase in serum RvD1 level (OR: 0.829, 95% CI: 0.721–0.953; p=0.009). IL-17 was a risk factor for psoriasis, while RvD1 was a protective factor.

## DISCUSSION

Although IL-17 and RvD1 were studied separately in psoriasis patients, the present study is clinically important because it is the first to study the balance between these two molecules in such patients and to reveal their relationship with PASI. The main finding of our study was that serum IL-17 levels were approximately two-fold higher in the psoriasis group compared to controls, whereas serum RvD1 levels were approximately 1.5-fold lower. This finding supports the concept that psoriasis is a chronic inflammatory process that disrupts the redox balance with a decrease in the expression of anti-inflammatory (RvD1) molecvules and an increase in the expression of inflammatory mediators (IL-17). The decrease in RvD1, a lipid mediator derived from docosahexaenoic acid, in psoriasis may prevent neutrophil phagocytosis and lead to the continuation of psoriasis-related inflammation [13]. Chronic inflammation that does not fully resolve in the presence of low RvD1 may prevent improvement in the clinical and laboratory findings of psoriasis. The role of this molecule in the etiology of the disease can be more clearly demonstrated by studies analyzing RvD1 levels during remission and attack periods in patients with psoriasis.

The fact that systemic indicators of inflammation such as WBC, neutrophil count, lymphocyte count, and neutrophil-to-lymphocyte ratio were increased in the psoriasis group compared to controls suggests that psoriasis activates inflammatory pathways and suppresses production of anti-inflammatory cytokines such as RvD1. The negative correlation between RvD1 and IL-17 strongly supports this idea. In addition, IL-17 has been shown to increase eightfold in psoriatic lesions and to stimulate proinflammatory cytokine production, suggesting that IL-17 is responsible for the increase in systemic inflammation indices [14]. The positive correlation between IL-17 and the neutrophil/lymphocyte ratio and the negative correlation between RvD1 and the neutrophil/lymphocyte ratio support the idea that IL-17 and RvD1 work in opposite ways to maintain the inflammatory balance in psoriasis.

The psoriasis area and severity index (PASI) is the most widely used scoring system as the primary endpoint of applied treatment efficacy [15]. The positive correlation between PASI index and IL-17, and the negative correlation between RvD1 and PASI, suggest that IL-17 increases the attacks and severity of psoriasis, while RvD1 reduces the attacks and severity. Mast cells and neutrophils that accumulate in the lesion area can be considered the main cellular sources of IL-17. However, it is not clear whether the immune and inflammatory cells accumulated in psoriatic lesions are the cause or the result of the disease [16]. The fact that IL-17A inhibitors provide almost complete resolution of skin lesions supports the positive relationship between IL-17 increase and PASI [17]. Resolvin D1 is an omega-3 polyunsaturated fatty acid–derived lipid mediator primarily produced by leukocytes, including macrophages and neutrophils. It promotes the resolution of inflammation by limiting neutrophil recruitment, enhancing macrophage-mediated clearance, and activating specific G-protein–coupled receptors such as ALX/FPR2 and GPR32 [18]. Notably, serum resolvin D1 levels were significantly reduced despite relatively low PASI scores in the psoriasis group. This finding suggests that impairment of pro-resolution pathways may occur even in patients with mild clinical disease activity. In the context of psoriasis, IL-17–driven immune activation may interfere with pro-resolving lipid mediator pathways. IL-17–activated T cells and neutrophils sustain a pro-inflammatory microenvironment that favors continued leukocyte recruitment and cytokine production, potentially suppressing the biosynthesis and bioavailability of pro-resolving mediators such as RvD1. Moreover, persistent IL-17 signaling may impair macrophage polarization toward a pro-resolving phenotype, thereby limiting effective resolution of inflammation. This imbalance between IL-17–mediated inflammation and RvD1-mediated resolution may contribute to the chronic and relapsing nature of psoriasis.

Multiple logistic regression analysis, after adjustment for age, BMI, and gender, revealed that high IL-17 and low RvD1 were independently associated with psoriasis, suggesting that these molecules could be used to diagnose psoriasis and assess its severity. The AUC obtained from ROC analysis of sensitivity and specificity for IL-17 in diagnosing psoriasis was 0.742, whereas the AUC for RvD1 was 0.715. The diagnostic sensitivities of both markers were similar. After the ROC analysis combining both markers, the AUC reached 0.814, and the diagnostic efficiency increased significantly.

### *Limitations*

In the present study, psoriasis was diagnosed based on characteristic clinical findings rather than histopathological confirmation. Although this approach reflects routine clinical practice and is widely accepted in clinical studies, the lack of histopathological verification may have introduced a degree of diagnostic heterogeneity , and this should be considered a limitation. The relatively small sample size and the cross-sectional study design may limit the generalizability of the findings and preclude causal inferences. Although inferential statistical methods were used to evaluate predefined biological associations, the results should be interpreted as exploratory rather than confirmatory. Larger, adequately powered prospective studies are required to validate these findings.


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## CONCLUSION

This is the first study to compare serum levels of interleukin-17 (IL-17), a key driver of proinflammatory cytokine expression, and resolvin D1 (RvD1), an anti-inflammatory lipid mediator, in patients with psoriasis and to relate them to other inflammatory markers and PASI scores. Consistent with their opposing biological roles, IL-17 levels were significantly increased, whereas RvD1 levels were significantly decreased in patients with psoriasis. After adjustment for age, sex, and body mass index, elevated IL-17 and reduced RvD1 remained independently associated with psoriasis, indicating their potential contribution to disease risk in this cohort. Although diagnostic sensitivities of IL-17 and RvD1 were comparable, combined use of the two biomarkers improved overall diagnostic performance, suggesting complementary value in clinical assessment. These findings highlight IL-17 and RvD1 as promising candidates for future diagnostic and disease-monitoring strategies in psoriasis. Finally, therapeutic approaches aimed at restoring the balance between pro-inflammatory and pro-resolving pathways, including modulation of IL-17 and RvD1, may represent a promising avenue for future treatment development, warranting further investigation in larger prospective studies.

**Ethics Committee Approval:** Ethical approval for this study was obtained from the Fırat University Non-Interventional Research Ethics Committee, Elazığ, Turkey (Approval no: 2024/29957, Date: 27.12.2024).

**Informed Consent:** Written informed consent was obtained from all participants prior to their inclusion in the study.

**Peer-review:** Externally peer-reviewed.

**Conflict of Interest:** The authors declare that they have no conflict of interest.

**Author Contributions:** Conception: Ş.G., R.P.S, A.K., Design: Ş.G., R.P.S, A.K., Supervision: Ş.G., R.P.S, Materials: Ş.G., Data Collection and/or Processing: Ş.G., Analysis and/or Interpretation: A.K., Literature Review: Ş.G., R.P.S, A.K., Writing: Ş.G., R.P.S, Critical Review: R.P.S, A.K.

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