INCIDENCE AND EVOLUTION OF AUTOIMMUNITY AND INFLAMATION MARKERS IN ROMANIAN PATIENTS WITH PSORIATIC DISEASE

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Summary

Psoriatic disease, including psoriasis and psoriatic arthritis, is driven by inflammatory and autoimmune pathways. Biologic agents targeting IL-17 and IL-23 are central to current treatment strategies, but their systemic immunomodulatory impact requires further characte-rization.

***Method:** A prospective study was conducted in 13 biologic-naive patients with moderate-to-severe psoriasis. Systemic inflammation was assessed using C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR), autoimmunity was evaluated by antinuclear antibody (ANA) testing. Measurements were obtained at baseline and after six months of IL-17/IL-23 inhibitor therapy.*

***Result:** At baseline, elevated ANA titers were present in 15.4% of patients, and most demonstrated increased systemic inflammatory markers. Following six months of therapy, ANA positivity was no longer detected, and CRP, ESR, and NLR values decreased significantly.*

***Conclusions:** IL-17/IL-23 blockade in moderate-to-severe psoriasis appears to reduce systemic inflammation and may modulate autoimmune activity. NLR emerges as a simple and informative biomarker of systemic disease burden in this context.*

***Keywords:** psoriasis, psoriatic arthritis, autoimmu-nity, inflammation markers, ANA, CRP, ESR, NLR*

***Abbreviation:** PsO-psoriasis, PsA-psoriatic arthritis.*

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Psoriatic disease is defined by the dual involvement of the skin and joints, as well as by the multitude of comorbidities that result secondarily from the chronic activation of systemic inflammatory mechanisms. The transition from cutaneous psoriasis to psoriatic arthritis occurs in the context of genetic predisposition and environmental factors, including epigenetic influences (1, 2, 3). The pathophysiology of psoriatic disease involves several immune pathways, with participation of both innate and adaptive immunity.

Normal skin is not populated by neutrophils. Neutrophils and T lymphocytes are considered essential in the development and progression of psoriasis, and the presence of neutrophils in the dermis and epidermis represents an early histopathological hallmark of the disease. Under the influence of a trigger, neutrophils are recruited to the skin, perpetuating the inflammatory process in an attempt to limit the initiating aggression. The formation of neutrophil extracellular traps (NETs) is the main mechanism of counteracting pathogens and also contributes

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to the initiation of autoimmunity by releasing large amounts of nucleic acids into the extracellular space, coupled with their inefficient clearance (4).

T lymphocytes, with their plasticity and important role in the additional recruitment of lymphocytes and neutrophils producing IL-17 in inflamed psoriatic lesions, amplify and maintain the inflammatory process. Moreover, certain studies have reported low levels of IL-6 corresponding to a relatively small number of Th17 cells, with IL-6 playing an essential role in Th17 cell differentiation (5).

Rationale for Selecting Inflammatory and Autoimmunity Markers:

NLR

Neutrophil-to-Lymphocyte Ratio (NLR)

NLR is a biomarker derived from the standard complete blood count, with increasing clinical utility, particularly in inflammation, autoimmunity, oncology, and infectious diseases.

Calculation formula:

- NLR = absolute neutrophil count/absolute lymphocyte count
- **Normal range (approximate):** 1–3 in healthy individuals.

Clinical utility of NLR:

1. **Indicator of systemic inflammation** NLR increases in both acute and chronic inflammatory conditions: bacterial infections, autoimmune diseases (including psoriasis, psoriatic arthritis, lupus), metabolic syndromes (obesity, diabetes) (6). Its diagnostic value is considered superior to ESR.
2. **Prognostic indicator in autoimmune and inflammatory diseases**
   - An elevated NLR correlates with higher disease activity, e.g., in psoriasis, NLR >2.5 may indicate moderate-to-severe forms.

Validity and limitations:

Advantages: easy to perform (from CBC), rapid and repeatable, correlates with systemic inflammation.

Limitations: nonspecific and influenced by corticosteroid or immunosuppressive therapy, hematologic malignancies, viral infections (lymphopenia), or bacterial infections (neutrophilia).

Condition	NLR Interval
Healthy individual	1.0 – 3.0
Mild inflammation	3.0 – 4.5
Moderate to severe inflammation	4.5 – 6.0
Severe inflammation/oncologic risk	>6.0

Values may vary slightly depending on laboratory and clinical context.

NLR, derived from the ratio of neutrophils to lymphocytes in peripheral blood, may reflect the balance between innate and adaptive immune responses. Abnormal NLR values are representative of inflammatory states; however, no universally accepted threshold defines its normality. Circulating neutrophil and lymphocyte levels vary between individuals and fluctuate over the course of the disease. In addition, patient medication use influences peripheral leukocyte levels (6,7,14).

Recently, several authors have investigated the diagnostic and prognostic value of NLR in psoriasis. In one observational study including 60 patients with psoriasis and 50 healthy controls, NLR values were significantly higher in the patient group compared with controls. Previous studies also reported that elevated NLR correlates with higher PASI scores. Similar findings were reported in another study, which showed a significant increase in NLR in patients with PASI ≥ 10 compared with those with PASI <10 (6,7,8).

Moreover, NLR has been proposed as a robust predictor of psoriatic arthritis development in patients with psoriasis. NLR has also been reported to predict all-cause mortality and cardiovascular risk, suggesting its potential as a biomarker for evaluating subclinical atherosclerosis in psoriasis patients. Furthermore, NLR is considered a potential predictor of treatment response, as a significant reduction has been observed in patients receiving effective therapies. Although NLR is generally elevated in psoriasis and tends to decrease after treatment initiation, early studies investigating its relationship with

disease severity have yielded inconsistent results (9,10,11).

NLR is an easily accessible index that can help clinicians identify patients at higher risk of developing severe forms of psoriasis (13).

Conclusions:

- NLR is a useful and valid marker for systemic inflammation diagnosis, monitoring, and prognosis.
- Although nonspecific, when correlated with the clinical picture and other markers (CRP, ESR, ferritin, LDH), NLR may guide therapeutic decisions and patient follow-up, particularly during treatment (12,13,14).

Autoimmunity Markers

For the present study, we considered it useful to perform antinuclear antibody (ANA) testing, which is less specific but more sensitive, based on the following considerations:

ANA:

Antinuclear antibodies (ANA) are autoantibodies that bind to components of the cell nucleus. They are primarily used in the diagnosis of systemic autoimmune diseases.

Characteristics of ANA:

1. **Marker of autoimmunity** ANA are used as a screening test for systemic autoimmune diseases, particularly: systemic lupus erythematosus (SLE – almost all patients are ANA positive), systemic scleroderma, juvenile idiopathic arthritis, Sjögren's syndrome, dermatomyositis, other connective tissue diseases, or mixed connective tissue disease (15,16).
2. **Differential diagnosis**. In the case of a positive ANA test, specific antibodies should be tested (anti-dsDNA, anti-Sm, anti-Ro/SSA, anti-La/SSB, anti-Scl70, anti-RNP, etc.), which allow more precise orientation and identification of the autoimmune disease (15,16,17).
3. **Clinical surveillance and prognosis** Although quantitative value does not directly correlate with severity, a high titer and persistent positivity may suggest an increased risk of systemic evolution or organ involvement (18).

Validity and proper interpretation of ANA testing:

1. **Psoriasis vulgaris (PsO):**
   - ANA are typically negative.
   - Psoriasis is a chronic immune-mediated inflammatory disease, but not a classic autoimmune disease.
2. **Psoriatic arthritis (PsA):**
   - ANA can be positive in a small proportion of cases (~10–20%), especially in women or in severe disease.
   - ANA positivity does not define PsA, but may indicate o coexistence of other autoimmune conditions (e.g., lupus, autoimmune thyroiditis)
     - o possible overlap between autoimmune diseases (19, 20).
   - **Clinical validity** ANA may also appear:
     - in the healthy general population (especially in individuals over 65 years old),
     - in viral infections, cancers, or drug exposure,
     - transiently, without clinical significance – therefore retesting is re-quired.
   2. **Sensitivity vs. specificity**. Very sensitive in SLE (>95% ANA positive), but nonspecific.
   3. **Analytical validity**.
      - o Expressed as titers (e.g., 1:160, 1:320). Considered clinically significant when ≥1:160.
      - o Value also depends on the method used:
      - o Indirect immunofluorescence (IIF) is the gold standard.
      - o ELISA and other methods may vary in sensitivity/specificity (19,20).
   4. **Validity over time**
      - o ANA may remain positive for years, even if the patient is in remission, and therefore are not automatically indicators of disease progression/regression.

Conclusion:

ANA testing is a screening tool, not a diagnostic test, and must always be interpreted in a clinical context.

- A positive ANA requires follow up with:
  - specific antibodies,
  - complementary investigations (CRP, complement, proteinuria, ESR, etc.),
  - full history and physical examination.
- They may have predictive value in the course of some autoimmune diseases.

In psoriasis, ANA are usually negative and are not part of the classic disease profile. For this reason, in our study, patients were tested both before and at six months after initiation of biologic therapy (an interval usually considered relevant for their appearance).

1. Psoriasis vulgaris (PsO):

- ANA are usually negative.
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2. Psoriatic arthritis (PsA):

- ANA may be positive in a small percentage (~10–20%), especially in women or in severe disease forms. The presence of ANA does not define PsA but may indicate:
  - coexistence of other autoimmune diseases (e.g., lupus, autoimmune thyroiditis),
  - suspicion of overlap between autoimmune diseases (19,20).

3. Drug-induced ANA:

Some biologic or immunosuppressive therapies used in psoriasis can induce ANA formation:

- **Anti-TNF (e.g., infliximab, etanercept, adalimumab)** – may lead to ANA positivity and even drug-induced lupus. Main mechanisms of autoantibody (ANA, anti-dsDNA) induction during immunomodulatory therapy include interference with immune tolerance regulation, increased apoptosis and release of nuclear antigens, and uncontrolled activation of autoreactive B cells.

1. Anti-TNF (adalimumab, etanercept, infliximab, certolizumab):
   - ANA become positive in 30–50% of patients treated, even if negative at baseline.
   - Anti-dsDNA may appear in ~10–15%, more frequently with infliximab.
   - Generally, ANA positivity is associated with treatment duration but not always with clinical symptoms.

There is also a risk of drug-induced lupus (DIL), most often reversible after discontinuation of therapy.

2. **Anti-IL-17** (secukinumab, ixekizumab, bimekizumab) – lower risk of ANA induction, usually low-titer and clinically irrelevant.
3. **Anti-IL-23** (guselkumab, risankizumab, tildrakizumab) – not associated with significant risk of ANA induction or of other autoimmune diseases (20,21).

Testing is recommended: before initiating biologic therapy, especially for anti-TNF agents, in patients with associated autoimmune pathology (e.g., autoimmune thyroiditis) or family history of autoimmune diseases, if autoimmune symptoms appear during treatment, for monitoring titers and the possible appearance of other autoantibodies (e.g., anti-dsDNA) (19,20,21,22).

Study Design

This prospective, observational study, conducted within the SRD Grant framework, aimed to evaluate the autoimmune and inflammatory profile in patients with moderate-to-severe plaque psoriasis, biologic-naïve (no prior exposure to biologic therapy), who met eligibility criteria for biologic treatment initiation.

Thus, the pre-existing autoimmune status prior to treatment initiation was assessed through the detection of antinuclear antibodies (ANA), while systemic inflammation was evaluated by monitoring the dynamics of inflammatory markers, namely: C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR).

Working Hypothesis

The rationale for selecting these markers in the present study lies in the lack of clear evidence validating autoimmunity markers, as well as the absence of established correlations between autoimmune processes and inflammatory mechanisms in psoriatic disease, with its two main components, cutaneous and articular. Assessing autoimmune status before initiating biologic therapy is important, as the patients' immune

profile may influence both therapeutic choice and subsequent disease evolution.

Methodology

Inclusion period: January 2024 – December 2024 (with a 6-month extension until June 2025).

Testing methods: ANA – ELISA method; CRP – immunoturbidimetric method; ESR – Westergren method; NLR – flow cytometry for complete blood count (neutrophil/lymphocyte ratio)

Normal reference values:

- ESR = 2.00–30.00 mm/1h
- ANA:
  - Negative <20 U/mL
  - Moderately positive: 20–60 U/mL
  - Strongly positive: >60 U/mL
- CRP <=5 mg/L
- Neutrophils = 2.00–8.00 M10H/L
- Leukocytes = 4.00–11.00 M10H/L

Main Objectives

- To evaluate the baseline immune and inflammatory status of the psoriatic patient population.
- To explore the autoimmune potential and assess its role as a mechanism involved in psoriasis pathophysiology.
- To assess the impact of biologic therapies on ANA titres and systemic inflammatory markers (CRP, ESR, NLR) in moderate-to-severe psoriasis, both before treatment initiation and at 24 weeks.

Secondary Objectives

- To compare the evolution of biological parameters between patients with and without psoriatic arthritis (PsA).
- To monitor variations of autoimmunity and inflammation markers according to the type of biologic agent used.
- To establish correlations between disease severity and the evaluated biomarkers.

Patient Characteristics

The study cohort included 13 biologic-naive patients, of whom 8 were male and 5 females, with a mean age of 45.9 ± 14.4 years (median 50 years, range 25–71 years). Among them, 46.2% (n = 6) also had psoriatic arthritis, according to the CASPAR classification criteria (ClASsification criteria for Psoriatic ARthritis).

Inclusion Criteria for Biologic Therapy:

- Patients were eligible according to the Romanian national protocol (Annex 2, Ministry of Health/CNAS Order no. 1206/2022), which requires:
- PASI ≥ 10 or significant involvement of special areas,
- DLQI ≥ 10, indicating major impact on
- failure, intolerance, or contraindication to at least 6 months of conventional systemic therapy (in this study, conventional treatment with methotrexate).

Exclusion Criteria:

- patients with another autoimmune disease (to exclude undiagnosed autoimmune thyroiditis, ATPO testing was also performed),
- other potential causes of inflammation that might alter test results.

Patient Follow-up:

- Patients were evaluated at baseline (T0) and after 6 months of treatment (T6).
- The 6-month re-evaluation period was chosen in accordance with the national monitoring protocol, but also with reference to data from the literature, which consider the interval of 6–12 months after initiating biologic therapy as having the highest risk for autoimmune phenomena (especially antidrug antibodydevelopment or drug-induced autoimmunity) (19,23,26).

The biologics selected for the study did not include TNF-α inhibitors but rather anti-IL-17 and anti-IL-23 agents, thereby minimizing the likelihood of developing antidrug antibodies (23).

Distribution of biologic therapies:

- 6 patients on tildrakizumab,
- 3 on ixekizumab,
- 2 on guselkumab,
- 1 on secukinumab,
- 1 on risankizumab. *(Figure 1)*

Baseline (T0)

- At baseline, only 2 patients (15.4%) had elevated ANA levels (>20 U/mL), while the mean ANA titer for the cohort was 13.29 ± 11.55 U/mL, reflecting predomi-

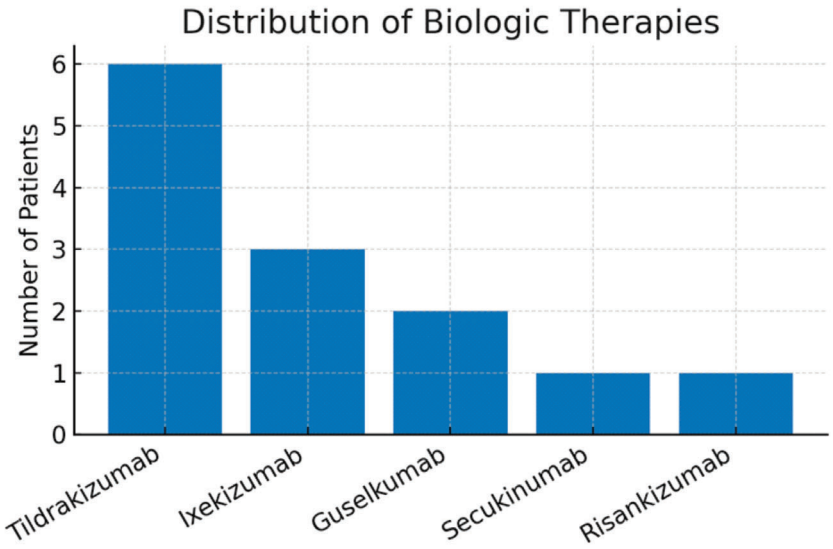

Figure 1 – Distribution of patients by biologic therapy

nantly normal values, though with wide interindividual variability.

- Regarding systemic inflammatory markers: 7 patients had elevated CRP levels, 5 had elevated ESR, and 2 had elevated NLR (>3).

After 6 Months of Treatment (T6)

- **Autoimmunity status at T6:** No patient had ANA >20 U/mL at T6, suggesting a stable and favorable immunologic effect of IL-17/IL-23 inhibitors (Figure 2). Existing studies confirm that ANA changes occur mainly with TNF-α therapies, but are rare or absent with newer agents (IL-17, IL-23), without associated lupus-like manifestations (27,28).
- **Inflammatory status at T6:** Only 5 patients (38.5%) still had elevated CRP, 4 patients (30.8%) had elevated ESR, and only 1 patient (7.7%) had NLR >3.

Observations:

ANA titers:

The most pronounced decrease was observed in patients treated with guselkumab (−15.06 U/mL), followed by ixekizumab (−5.82 U/mL) and tildrakizumab (−2.5 U/mL). Although current literature provides no clear evidence that guselkumab normalizes ANA titers (24), a potential mechanism could involve the role of IL-23 and IL-17 overproduction in autoimmune processes, including autoantibody production, immune complex formation, defective apoptosis, inadequate clearance of dead cells, and loss of self-tolerance (24). The potential of IL-17 and IL-23 inhibitors in reducing autoimmune phenomena has only scarcely been explored (25,26).

Inflammatory markers and correlation with PsA:

- To further explore differences in immune and inflammatory responses by PsA status, patients with PsA (n=6) were compared with those without joint involvement (n=7) after 6 months of biologic therapy (*Figure 3*).
- Mean ANA titers were lower in PsA patients (6.75 ± 1.83 U/mL) compared with non-PsA patients (10.77 ± 3.86 U/mL).
- CRP levels were modestly higher in PsA patients (10.02 ± 9.04 mg/L vs. 7.16 ± 13.54 mg/L), as were ESR values (19.4 mm/h vs. 16.5 mm/h).
- NLR was comparable between groups (1.74 ± 0.53 vs. 1.85 ± 1.12).
- None of these differences reached statistical significance (Mann–Whitney U, $p > 0.05$).

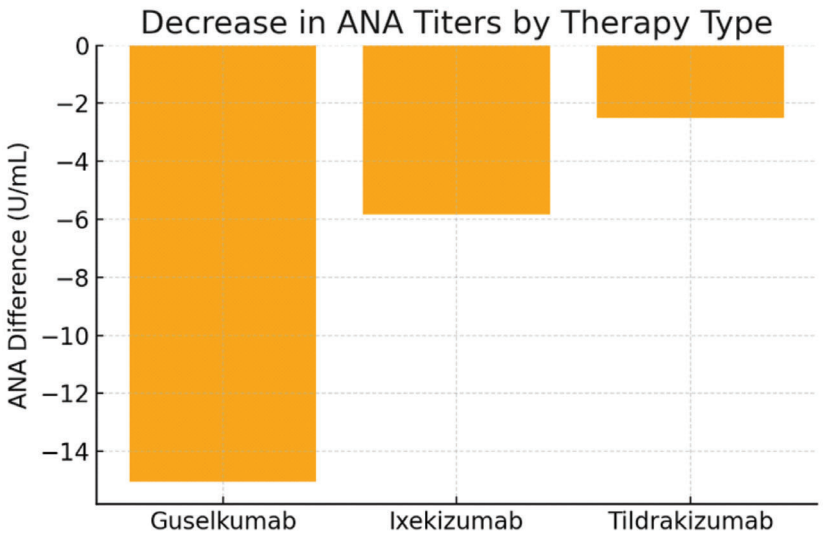

Figure 2 – Evolution of ANA titers after 6 months of treatment, by therapy type: highlights ANA reduction with guselkumab −15.06 U/mL, ixekizumab −5.82 U/mL, tildrakizumab −2.5 U/mL; suggesting a favorable effect of IL-23/IL-17 inhibitors, though the small cohort limits definitive conclusions

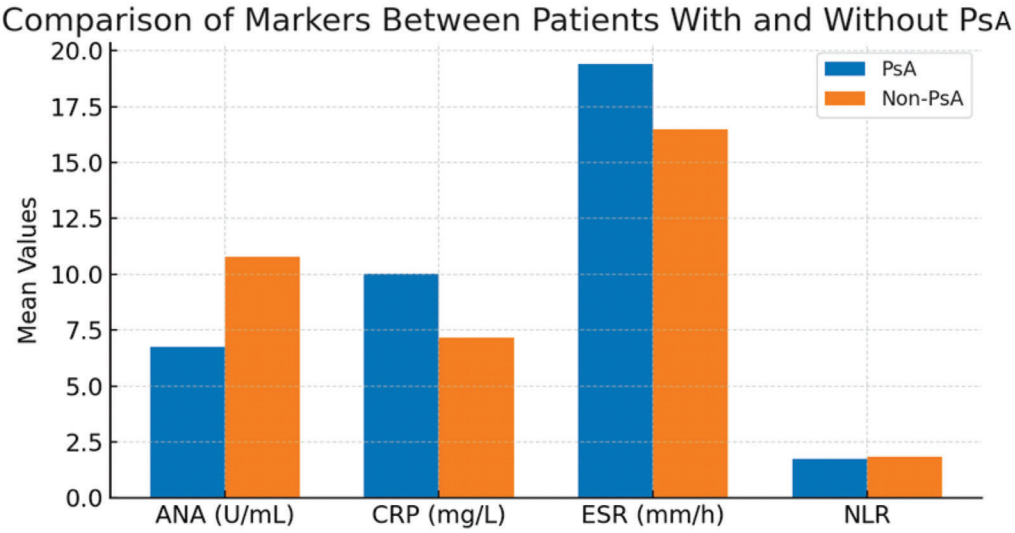

Figure 3 – Comparison of inflammatory and autoimmune markers between PsA and non-PsA patients at T6

Overall, these results suggest that after 6 months of biologic therapy, systemic inflammation and immune parameters are similarly controlled in patients with and without PsA. This may reflect the small sample size, short follow-up, and the strong anti-inflammatory effect of IL-17/IL-23 inhibitors.

Results and Conclusions

- At baseline, a small proportion of patients (15.4%) exhibited ANA positivity. Previous studies have shown that ANA may be detected in a subset of patients with psoriasis, even in the absence of systemic autoimmune disease. Possible explana-

tions include the effect of chronic systemic inflammation on polyclonal B-cell activation and transient autoantibody production (Kutlu et al., 2020; Rodríguez-Jiménez et al., 2017), as well as the impact of prior systemic therapies such as methotrexate or cyclosporine, which have been associated with ANA induction (Rodríguez-Jiménez et al., 2017). Moreover, ANA positivity may also occur as an incidental finding in the general population without clinical significance (Satoh et al., 2012).

- Dynamic monitoring of the studied indicators after 6 months of treatment revealed a general improvement in the inflammatory and autoimmune profile of patients with plaque psoriasis under biologic therapy. ANA, CRP, and NLR all showed meaningful decreases, suggesting a reduction in systemic inflammatory activity. According to literature data, while anti-TNF-α therapies can induce ANA seroconversion, IL-17, IL-23, and IL-12/23 inhibitors have not been associated with increased autoantibody formation; rather, they may even contribute to their reduction. We observed a positive correlation between PASI score and NLR values, consistent with findings from other studies (9).
- Although NLR and ESR can generally be considered statistically significant predictors of psoriatic arthritis (PsA) in psoriasis patients (9), in this study NLR was not associated with the presence or later development of PsA.
- The data obtained do not support a major autoimmune component in psoriasis; however, the finding of increased ANA titers in psoriatic patients more frequently than in the general population remains noteworthy for future research. Extension of the study to a larger cohort is required to validate these observations and to develop objective biomarkers for psoriasis monitoring.
- In our study group, the normalization effects of ANA following biologic therapy (anti-IL-17 and anti-IL-23) were not previously documented, suggesting the involvement of multiple pathophysiological mechanisms at the interface between psoriasis and autoimmunity. Autoimmune disease screening prior to initiating biologic therapy may be considered depending on the patient's profile and the treatment type.

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** The article was prepared by the authors, with only occasional and minimal use of artificial intelligence tools, limited to language support and text formatting.*

** The study was carried out under the SRD Grant*

Dissemination of Results

The results of this study were presented both nationally and internationally, in the form of posters and oral communications at scientific meetings, with the main objective of further exploring autoimmune correlations in systemic diseases:

1. "Inflammation and Autoimmunity Dynamics in Psoriasis" – poster presentation at *The Psoriasis: From Gene to Clinic 10th International Congress*, December 5–7, 2024, London, United Kingdom.
2. "Psoriasis: Between Inflammation and Autoimmunity" – presentation at *FIMSA Advanced Immunology Course 2025, part of the Global Immunology Summit 2025*, New Delhi, India.
3. "Autoimmune and Autoinflammatory Disorders of Early-Onset Disease" – oral pre-sentation at the *World Congress on Immunology*, April 24–25, 2025, Paris, France.
4. Inflammatory markers in psoriatic disease – article in the Romanian Society of Dermatology Journal, in press.

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Conflict of interest
NONE DECLARED

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