IRB approval status: Reviewed and approved by IRB (Approval No EC/24).

Data availability statement: Data available on request from the authors.

Ethical approval: The ethical principles for medical research in human subjects outlined in the latest (2013) version of declaration of Helsinki, and by the Committee on Publication Ethics (COPE) were duly followed.

Key words: laser toning; Laser; LPP.

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Conflicts of interest

None disclosed.

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https://doi.org/10.1016/j.jaad.2025.11.074

Improvement of psoriasis following initiation of biologic therapy is associated with lower risk of developing psoriatic arthritis: A single-center, retrospective, cohort study

Psoriasis severity has been identified as a risk factor for psoriatic arthritis (PsA).[1-5] However, it has not been determined whether reduction of psoriasis (PsO) severity in patients on biologic therapy is associated with reduced risk of PsA. This study's objective was to test that hypothesis. This retrospective observational study included consecutive adult patients with PsO who attended the outpatient clinic of the University Hospital of Verona and initiated their first biologic therapy between January 1, 2012, and June 30, 2023. Data collection was performed at the time of biologic initiation, and the diagnosis of PsA was excluded in all the subjects by a rheumatologist during a joint Dermatology-Rheumatology consultation at baseline. If PsA was suspected during follow-up visits based on symptoms, the patient was referred to a rheumatologist. PsA was confirmed by fulfilling CASPAR (Classification criteria for Psoriatic Arthritis) criteria, supported by clinical and ultrasonographic findings. For each patient, the percentage Psoriasis Area Severity Index (PASI) reduction (delta PASI%) from baseline values was calculated. To investigate the association between delta PASI% and PsA development, adjusting for possible confounders, an analysis of covariance on the ranked variable due to the nonnormal distribution of delta PASI% was used; the model was adjusted for baseline PASI, age, sex, statin use, alcohol use, smoking, hypertension, dyslipidemia, diabetes, family history of PsA, and biologic class.

Characteristics of the patients ($n = 622$) are reported in Table I. Of these, 60 (9.6%) developed PsA during follow-up (median time to PsA: 38 months; interquartile range, 24-52 months), whereas 562 (90.4%) did not. Patients with PsO who developed PsA had higher baseline PsO severity, more often reported a family history of PsA, were more frequently tobacco and alcohol users, and demonstrated more arterial hypertension, diabetes, and dyslipidemia. The association between delta PASI% reduction and PsA development is reported in Table II. None of the patients who developed PsA achieved PASI90 or PASI75 compared with 79.0% and 98.4%, respectively, among those who did not develop PsA ($P < .001$). After adjusting for sex, alcohol consumption, family history of PsA, statin use, and tumor necrosis factor-inhibitor use, patients who developed PsA exhibited a significantly lower delta PASI% than those who did not (median: −48.6% vs −98.8%; $P < .001$, from adjusted analysis).

The study limitations include its retrospective and observational design, which do not allow for causal inference. A control arm of untreated patients is missing. Despite multivariate adjustment, confounding by unmeasured confounders cannot be excluded. Additionally, it cannot be determined whether the risk of PsA was reduced due to the degree of PASI reduction or due to use of a biologic that directly impacted development of PsA. Patients with severe PsO that is resistant to medications may

Table I. Demographic, clinical characteristics, class of biological treatment in patients with psoriasis, and overall and stratified by psoriatic arthritis development

	All patients with PsO (n = 622)	Patients not developing PsA (n = 562)	Patients developing PsA (n = 60)	P*
Age, y				
<45	316 (50.8)	286 (50.9)	30 (50.0)	
≥45	306 (49.2)	276 (49.1)	30 (50.0)	.896
Mean ± SD	46.9 ± 12.9	46.9 ± 12.9	47.9 ± 13.5	.519
Sex, female	192 (30.9)	160 (28.5)	28 (46.7)	<.001
BMI, kg/m^2				
≤25	188 (30.2)	176 (31.3)	12 (20.0)	
>25	434 (69.8)	386 (68.7)	48 (80.0)	.070
Mean ± SD	26.6 ± 2.7	26.6 ± 2.8	26.9 ± 2.0	.240
Statin use	216 (34.7)	156 (27.8)	60 (100.0)	<.001
Alcohol use†	465 (74.8)	405 (72.1)	60 (100.0)	<.001
Tobacco use	164 (26.4)	142 (25.3)	22 (36.7)	.057
Hypertension	251 (40.4)	219 (39.0)	32 (53.3)	.031
Dyslipidemia	225 (36.2)	196 (34.9)	29 (48.3)	.039
Diabetes	127 (20.4)	105 (18.7)	22 (36.7)	.001
Family history of PsA	17 (2.7)	8 (1.4)	9 (15.0)	<.001
Class of biologic treatment				
TNF-inhibitors	317 (51.0)	272 (48.4)	45 (75.0)	
IL-17 inhibitors	164 (26.4)	155 (27.6)	9 (15.0)	
IL-23 inhibitors	141 (22.7)	135 (24.0)	6 (10.0)	<.001
Duration of treatment (mo)	38 (24-52)	39 (23—59)	36 (21—60)	.33
Median IQR				
PASI				
≤10	396 (63.7)	379 (67.4)	17 (28.3)	
>10	226 (36.3)	183 (32.6)	43 (71.7)	<.001
Median (q1-q3)	9.0 (8.0-12.0)	9.0 (8.0-11.0)	16.0 (8.5-18.0)	<.001

BMI, Body mass index; *IQR*, interquartile range; *IL*, interleukin; *PASI*, Psoriasis Area Severity Index; *PsA*, psoriatic arthritis; *SD*, standard deviation; *TNF*, tumor necrosis factor.
*From the *t* test or the Wilcoxon-Mann-Whitney test for quantitative variables, or from the Chi-square test for categorical variables.
†More than 2 standard units per day in men and >1 unit per day in women.

Table II. Reduction of Psoriasis Area Severity Index in patients with psoriasis according to psoriatic arthritis development

	PsA		Comparison between groups	
	Yes (n = 60)	No (n = 562)	P from crude analysis	P from adjusted analysis
PASI75, n (%)	0 (0.0)	553 (98.4)	<.001*	NA†
PASI90, n (%)	0 (0.0)	444 (79.0)	<.001*	NA†
% PASI reduction, median (q1-q3)	−48.6 (−41.9 to −51.3)	−98.8 (−90.0 to −100)	<.001‡	<.001§

PASI, Psoriasis Area Severity Index.
*From the χ^2 test.
†The adjusted analysis through logistic regression is not applicable (NA) since 0 cell occurrence. Baseline PASI and the PASI at the time of psoriatic arthritis (PsA) diagnosis and the most recent PASI were considered in patients who developed PsA and did not develop PsA, respectively, for estimating PASI reduction.
‡From the Wilcoxon test.
§From an analysis of covariance model on ranked data, adjusting for baseline PASI, age, sex, statin use, alcohol use, tobacco use, hypertension, dyslipidemia, family history of PsA, diabetes, and class of the first biologic treatment.

be at the highest risk of PsA. Reduction in PASI may be a proxy for having a milder phenotype and lower risk of PsA. The study has some strengths, including a real-world setting, long-term follow-up with rheumatologist-based diagnosis, and multiple adjustments.

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Funding sources: None.

IRB approval status: Reviewed and approved by the Institutional review board.

Key words: psoriasis; psoriatic arthritis; biologic therapy; IL-17; IL-23 inhibitors; TNF.

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Conflicts of interest

Dr Gisondi has been a consultant and/or speaker for AbbVie, Almirall, Amgen, Boehringer Ingelheim, Janssen, Leo pharma, Eli Lilly, Novartis, Pierre Fabre, Sandoz, Sanofi, and UCB. Dr Idolazzi served as consultant and/or speaker for AbbVie, Amgen, Biogen, Merck Sharp & Dohme, Eli Lilly, Novartis, Celgene, and Sandoz. Dr Girolomoni served as consultant and/or speaker for AbbVie, Abiogen, Almirall, Amgen, Biogen, Boehringer Ingelheim, Bristol-Meyers Squibb, Celltrion, Eli Lilly, Genzyme, Leo Pharma, Novartis, OM Pharma, Pfizer, Regeneron, Samsung, Sandoz, and UCB. Dr Zabotti served as consultant and/or speaker for AbbVie, Amgen, Biogen, Eli Lilly, Novartis, UCB, and Jannsen. Dr McGonagle has undertaken research and/or educational program activities with Pfizer, MSD, AbbVie, BMS, UCB, Novartis, Celgene, and J&J. Dr Blauvelt has served as a speaker (received honoraria) for Lilly and UCB; has served as a scientific adviser (received honoraria) for AbbVie, Abcentra, Aclaris, Affibody, Aligos, Almirall, Alumis, Amgen, Anaptysbio, Apogee, Arcutis, Arena, Aslan, Athenex, Bluefin Biomedicine, Boehringer Ingelheim, Bristol Myers Squibb, Cara Therapeutics, Celldex, CTI BioPharma, Dermavant, EcoR1, Escient, Evelo, Evommune, Forte, Galderma, HighlightII Pharma, Incyte, InnoventBio, Janssen, Landos, LEO Pharma, Lilly, Lipidio, Microbion, Merck, Monte Rosa Therapeutics, Nektar, Novartis, Overtone Therapeutics, Paragon, Pfizer, Q32 Bio, Rani, Rapt, Regeneron, Sanofi Genzyme, Spherix Global Insights, Sun Pharma, Takeda, TLL Pharmaceutical, TrialSpark, UCB Pharma, Union, Ventyx, Vibliome, and Xencor; has acted as a clinical study investigator (institution has received clinical study funds) for AbbVie, Acelyrin, Allakos, Almirall, Alumis, Amgen, Arcutis, Athenex, Boehringer Ingelheim, Bristol Myers Squibb, Concert, Dermavant, DermBiont, Evelo, Evommune, Galderma, Incyte, Janssen, LEO Pharma, Lilly, Merck, Novartis, Pfizer, Regeneron, Sanofi, Sun Pharma, Takeda, UCB Pharma, and Ventyx; and owns stock in Lipidio and Oruka. Dr Blauvelt was not compensated for contributing to this manuscript. Drs Bellinato, Galeone, and Turati, have no conflicts of interest to declare.

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https://doi.org/10.1016/j.jaad.2025.11.075

Alopecia areata is associated with adverse pregnancy outcomes in a retrospective cohort study using TriNetX

Autoimmune conditions, including alopecia areata (AA), have been associated with increased risk of adverse pregnancy outcomes, potentially due to immune dysregulation, systemic inflammation, hormone fluctuations, coagulopathy, autoantibodies crossing the placenta, placental dysfunction, and treatments.[1] There is a paucity of studies exploring relationships between AA and adverse pregnancy outcomes. Thus, we analyzed for these potential associations using a retrospective cohort design and a large national database to aid dermatologists in counseling AA patients who are pregnant or planning pregnancy.

On July 21, 2025, TriNetX research network was queried 2004-2025 for all female patients with