Review

Special site psoriasis and psoriatic arthritis: environmental triggers and disease interplay. A systematic review

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ABSTRACT

Objective. *Psoriatic arthritis (PsA) is a chronic inflammatory musculoskeletal disease affecting approximately 20-30% of patients with psoriasis (PsO). Early identification of PsA risk factors is essential to prevent irreversible joint damage. Psoriasis involving "special sites" such as the nails, scalp, hands, feet and anogenital region has been increasingly linked to elevated risk of development for PsA. This systematic review aimed to assess the evidence for a correlation between special site psoriasis and PsA onset and to evaluate whether this relationship is coincidental or biologically driven.*

Methods. *A comprehensive literature search of PubMed, Cochrane Library and Embase databases through April 2025 identified 26 studies meeting PRISMA criteria. Search terms included "Psoriasis," "Psoriatic Arthritis," and location-specific descriptors (e.g. "Scalp Dermatoses," "Nail," "Foot Dermatosis," "Hand Dermatosis," "Intergluteal Lesions" or "Perianal Lesions"). Eligible studies focused on associations between psoriasis at special sites and PsA development. The certainty of evidence was assessed using the GRADE approach. The review protocol was prospectively registered in the PROSPERO database (registration ID: CRD420251090316).*

Results. *Of the 26 included studies, 19 examined nail involvement, with 17 showing a positive association with PsA. Imaging techniques such as high-frequency ultrasound and capillaroscopy confirmed inflammatory and structural changes in the nail-entheseal complex. Scalp psoriasis was evaluated in nine studies; four supported a correlation with PsA. Five studies assessed genital and intergluteal/perianal involvement, with four showing a significantly increased PsA risk.*

Conclusion. *The involvement of nails shows the strongest association with PsA development, likely due to anatomical and functional continuity between the nail apparatus and the entheseal-joint interface. Scalp psoriasis may reflect or exacerbate systemic inflammation, although available data remains inconsistent. Anogenital psoriasis appears to signify a distinct immunopathological phenotype shaped by its unique microenvironment. Importantly, a range of environmental triggers including mechanical trauma, moisture and occlusion, microbial dysbiosis, chemical and biological irritants, seasonal and climatic influences, smoking- and alcohol-induced immune modulation, UV radiation, and obesity-related metabolic inflammation may modulate immune activation at these sites and contribute to PsA onset. These findings support a biologically driven interplay between site-specific skin inflammation and joint involvement, mediated in part by local environmental influences.*

Introduction

Psoriatic arthritis (PsA) represents one of the most severe manifestations of psoriasis (1). It develops in approximately 30% of patients with cutaneous psoriasis (PsO), leading to chronic inflammation of affected joints and gradually contributes to their destruction (2, 3). The onset of PsA is usually preceded by the occurrence of skin lesions characteristic for PsO and an identification of accessible and reliable predictive markers is of critical importance (4).

Several body regions such as nails, scalp and anogenital areas emerged to be termed "special sites" due to their resistance to both standard and biological treatments as well as frequent concomitant development of PsA (5, 6).

These special sites exhibit distinct characteristics, stemming from unique anatomical and immunological environments, as well as the overarching genetic predisposition underlying psoriasis. The scalp, for instance, due to its susceptibility to microtrauma, rich microflora together with abundant vascular and neural supply, may serve as a local nidus for the initiation of inflammation involving the IL-23/IL-17 axis (7). Microanatomical continuity between the nail unit and the extensor tendon enthesis at the distal phalanx may account for a development of distal interphalangeal (DIP) joint arthritis due to PsA (8, 9). Moisture and friction account for repeated microtrauma which together with an abundant bacterial and fungal flora in the anogenital region may lead to activation of plasmacytoid dendritic cells, elevated type I interferon production and stimulation of the Th17/IL-23 axis (5, 10).

Despite numerous indications suggesting a relationship between the anatomical location of psoriatic lesions and the risk of developing PsA, definitive evidence to establish whether these associations are causal or merely coincidental remains lacking.

The aim of this systematic review was to analyse and critically evaluate associations between the involvement of special regions by PsO and the development of PsA.

Materials and methods

A systematic review of the literature was performed using the PubMed, Cochrane Library and Embase databases. The protocol for this review was prospectively registered in the PROSPERO database (registration ID number: CRD420251090316).

The search strategy incorporated combinations such as "Psoriasis" and "Psoriatic Arthritis," with additional terms added to broaden the scope: ("Scalp Dermatoses" or "Nail" or "Foot Dermatosis" or "Hand Dermatosis") and ("Intergluteal Lesions" or "Perianal Lesions"). Searching was as broad as possible from the inception of the database until April 2025, including EMTREE and MESH approaches, conducted according to the PRISMA guidelines. Eligible studies included randomised controlled trials, prospective and retrospective cohort studies, case series with at least five participants and cross-sectional studies with a high level of evidence, published in English. Exclusion criteria involved narrative reviews, case reports, editorials, abstracts without methodological transparency and studies involving paediatric populations, animals or *in vitro* data. The selection process adhered to PRISMA guidelines, and was carried out independently by four reviewers who screened all titles, abstracts and full texts. Any disagreements were resolved through discussion and consensus to minimise selection bias. All decisions regarding inclusion and exclusion were documented. The certainty of evidence was assessed using the GRADE approach. Data and outcomes were synthesised and presented in structured tables.

Results

A total of 26 clinical studies were included in this review (Fig. 1). Evaluation of nail involvement in patients with PsA was seen in 19 studies, scalp psoriasis in 9 ones and 5 manuscripts explored anogenital involvement. Most studies were observational in design (prospective or retrospective cohort, case-control) and they employed both clinical evaluation as well as various diagnostic methods: ultrasonography, nailfold capillaroscopy (NFC), and magnetic resonance imaging (MRI).

Nail psoriasis and psoriatic arthritis

Nineteen studies included nail involvement, and 17 of them reported a positive correlation between nail lesions and PsA (Table I). The most early two manuscripts (1964 and 1992) failed to demonstrate a statistically significant association between nail psoriasis and development of PsA (11, 12). This lack of correlation was later supported by the study of Wittkowski *et al*. who analysed 180 adult patients with plaque PsO and 55 of them had PsA. Although nail changes were highly prevalent in subjects with PsA, they were poorly correlated with joint disease (13).

While earlier studies showed no significant correlation between nail involvement and PsA, Soy *et al*. observed lesioned nail plates in 91% of patients with PsA, and pitting together with subungual hyperkeratosis were the most common findings (14). Nail involvement emerged as a predictor for the development of PsA in the study of Wilson *et al*. It was associated with a 2.24-fold increase in the risk of development for PsA (15). Nail psoriasis was the strong predictor of PsA development (odds ratio of 6.81) in the observation of Spelman *et al*. (16). The involvement of nail plates was identified by Askin *et al*. to correlate with more severe phenotypes of PsA (3). Dalbeth *et al*. showed baseline nail abnormalities such as onycholysis and hyperkeratosis to be associated with subsequent joint erosions (17). Antony *et al*. found significant associations between onycholysis as well as subungual hyperkeratosis, and erosions at DIP joint level, while nail pitting showed no correlation with joint damage (18). Elkayam *et al*. could see correlation between nail involvement and the severity of joint disease. They found close association between the nail involvement and arthritis affecting DIP joints and the spine (19). The anatomical link between nail dystrophy and the damage of DIP joints was confirmed by ultrasonography which identified thicker nail plates and increased vascular signals in PsA patients (20). Mondal *et al*. and Krajewska-Włodarczyk *et al*. showed also significant associations between nail bed thickness and a progression of PsA disease. Increased nail thickness correlated there with more severe joint involvement, particularly at the DIP joint level (21, 22).

A lack of evident changes in nail plates does not seem to reduce the risk of PsA development. Ultrasonography was shown recently to facilitate nail plate involvement in PsA patients despite the normal appearance of nail plates. A subclinical nail involvement was identified to reflect underlying entheso-

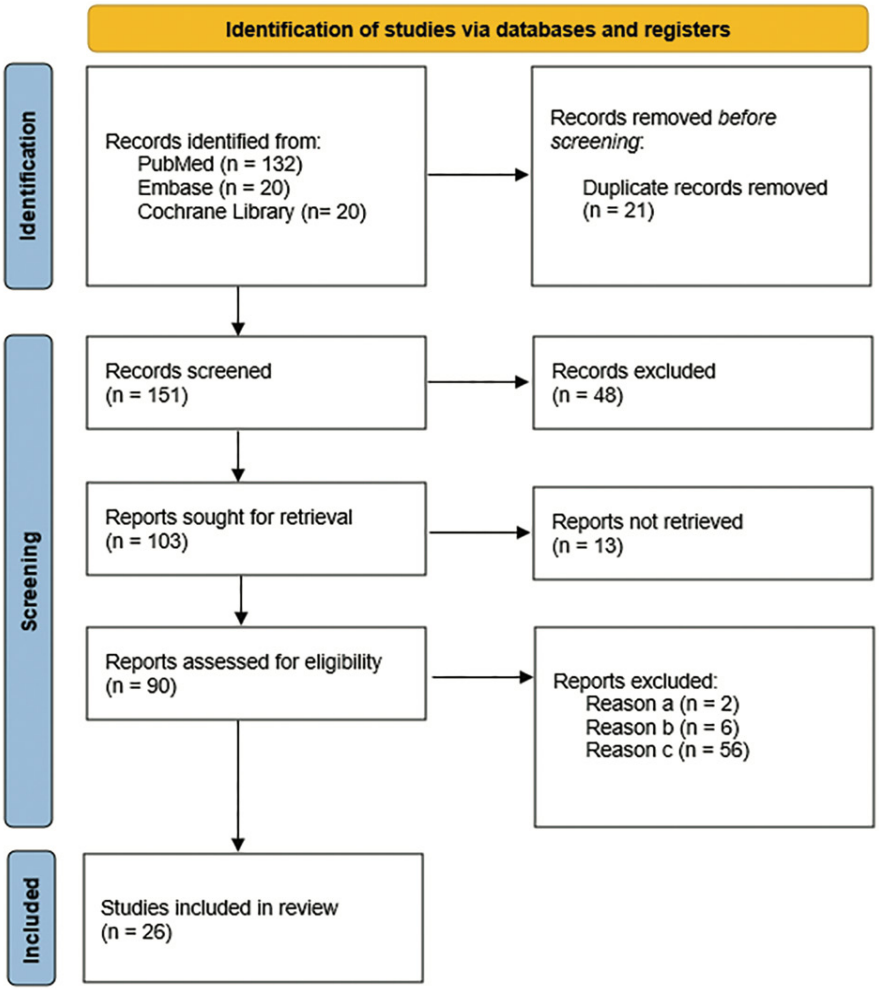

Fig. 1. PRISMA diagram details our search and selection process applied during the overview. a) Studies published in non-English languages were excluded. b) Studies focusing on the paediatric population. c) Ineligible study type for inclusion.

pathic inflammation (23). Earlier studies using high- frequency ultrasound also found subclinical inflammation and morphological changes in the nail unit in patients with PsA but lacking lesioned nails (24, 25). Other studies revealed that only a subtle change in microcirculation of nailbed in clinically normal nails may precede manifestation of PsA (23, 26). In a recent study of Cafaro *et al*. significantly higher number of tortuous capillaries was observed in patients with PsA group but not in subgroups with PsO or suffering from rheumatoid arthritis. Additionally, there were noted lower capillary density and a higher number of dilated capillaries in subjects with PsA (9).

Scalp psoriasis and psoriatic arthritis

Reviewed manuscripts were less indicative for the association of PsA with scalp psoriasis. Only four of nine studies (15, 19, 27, 28) showed a potential link between involvement of the scalp and systemic musculoskeletal inflammation whereas five studies (3, 16, 29-31) identified scalp involvement as a negative or neutral predictor for development of PsA. Scalp involvement had been increasingly recognised as a manifestation of more severe psoriatic disease (3, 16, 30). Elkayam *et al*. reported a significant association between scalp psoriasis and the extent of joint involvement (19). Pavlica *et al*. postulated scalp psoriasis to be linked with more extensive musculoskeletal involvement and increased systemic inflammation (27). The involvement of the scalp by psoriasis was found by Wilson *et al*. to share 3.75-fold (95% CI 2.09–6.71) increased risk of PsA development (15). PET/CT imaging identified subclinical PsA in all patients with scalp disease in the study of Takata *et al*. whereas its prevalence was 67% in other patients with PsO patients (28). However, Langenbruch *et al*. found no significant association between scalp involvement and incidences of PsA (OR 1.07; 95% CI 0.90–1.26; p=0.457) (29), similarly, like Yan *et al*. who observed no significant link in multivariate model after adjusting for confounders (31).

Genital and intergluteal/perianal psoriasis

Four of the five reviewed studies addressing the anogenital involvement by psoriasis showed a significant correlation of lesions in that region with development of PsA (Table III). The work of Wilson *et al*. found increased risk (HR 1.95; 95% CI: 1.07–3.56) of PsA development in case of lesioned anogenital region, and the risk was potentiated when patients had concomitant involvement of more than one "special skin area" (HR 2.24; 95% CI: 1.23– 4.08) (15). The involvement of anogenital region was identified by El-Garf *et al*. as the strongest independent predictor of PsA developing (odds ratio [OR] 12.66; 95% CI: 4.30–37.25; p<0.001) (32). Anogenital involvement was also found to correlate with elevated erythrocyte sedimentation rate and C-reactive protein levels in psoriatic patients sharing joint disease (33). In contrast, multivariate analysis of Yan *et al*. found no meaningful correlation (OR 0.90; p=0.739) between anogenital involvement and the development of PsA, suggesting that psoriasis affecting that region may not independently predict joint disease (31).

Discussion

The strongest association with the development of PsA appears for nail involvement since 17 of 19 papers indicated for such link (3, 9, 11, 14-26, 34), whereas data for other investigated special sites remain less appealing. The nail apparatus forms an integrated "synovio-entheseal complex" with the DIP joint extensor tendon (25), rendering it vulnerable to share inflammatory mechanisms highlighting the importance of the need for early treatment

Table I. Summary of studies investigating the association between nail unit changes and psoriatic arthritis.

Author and year	Study population	Type of the article	GRADE	The applied diagnostic method	Outcome	Existing positive correlation
Baker *et al*. (1964) (19)	53 patients with seronegative PsA	prospective case series	D	- clinical evaluation of psoriatic nail changes - radiographic assessment of hand and foot joints	- nail involvement seen in 83% of patients with PsA at prospective follow-up - severe nail changes were more common in mutilating or sacroiliitis-associated arthritis - lack of association of DIP arthritis with nail changes	YES
Grassi *et al*. (1992) (20)	13 patients with PsA, 25 HC	controlled pilot study	D	- nailfold capillaroscopy - fluorescence videomicroscopy	- lack of association with capillary permeability, dye distribution or other morphodynamic parameters - only the length of capillary loop was significantly greater in PsA patients than in HC (p<0.02)	NO
Elkayam *et al*. (2000) (21)	70 patients with PsA	prospective observational study	D	- clinical evaluation of the skin and joints	- nail involvement was correlated with the number of tender and swollen joints	YES
Soy *et al*. (2008) (22)	40 patients with PsO, 49 patients with PsA	case series	D	- clinical evaluation of the skin and joints	- nail involvement seen in 91% of patients with PsA *vs*. 32% in patients without PsA (p<0.05)	YES
Wilson *et al*. (2009) (23)	1633 patients with PsO (40 patients with already diagnosed PsA, 57 patients developed PsA at follow-up)	longitudinal, retrospective cohort study	B	- clinical evaluation of the skin and joints	- 2.24-fold increased risk of PsA (HR 2.24, 95% CI 1.26–3.98) in case of nail dystrophy	YES
Wittkowski *et al*. (2011) (24)	180 patients with PsO (including 55 with PsA)	observational comparative study	D	- clinical evaluation of the skin and joints	- weak to no correlation between nail involvement and severity of joint involvement - poor association between joint and nail involvement in the same digit	NO
Dalbeth *et al*. (2012) (25)	34 patients with PsA	prospective cohort study	D	- clinical evaluation of nails evaluation of joint involvement with magnetic resonance imaging	- significantly higher rate of bone erosion or proliferation in the distal phalanx in case of onycholysis and hyperkeratosis - baseline bone marrow edema in the distal phalanx was predictive of ongoing development of onycholysis and hyperkeratosis at one-year follow-up - lack of association between nail pitting and pathology of the distal phalanx	YES
Sandobal *et al*. (2014) (26)	35 patients with PsA, 20 patients with PsO, 27 patients with RA, 28 HC	Case-control study	D	- ultrasound evaluation of nails and joints	- subclinical abnormalities of nails in 54% of PsA patients despite of no visible nail involvement - loosening of the borders of the ventral plate in patients with PsA vs focal hyperechoic involvement of the ventral plate without involvement of the dorsal plate in PsO patients	YES
Spelman *et al*. (2015) (27)	424 patients with PsO (37 patients with PsA)	cohort study	D	- clinical evaluation of nails - clinical and radiographic evaluation of joints	- nail psoriasis identified as an independent and strong predictor of PsA (OR: 6.81; 95% CI: 2.09–22.18; p<0.001).	YES
Arbault *et al*. (2015) (28)	27 patients with PsA	prospective cohort study	D	- clinical and ultrasonographic evaluation of nails - clinical and ultrasonographic evaluation of joints	- nail involvement correlated with DIP synovitis and local pain indicating a localised joint-nail link - no correlation with systemic activity scores or enthesitis	YES
Mondal *et al*. (2018) (29)	45 patients with PsA, 45 HC	case-control study	D	- clinical and ultrasonographic evaluation of nails - clinical and ultrasonographic evaluation of joints	- 88% of nails in PsA patients showed ultrasonographic changes, including 75% of clinically normal nails - significantly increased mean nail bed thickness and nail matrix thickness in PsA patients - a moderately correlation between nail matrix thickness and NAPSI (r=0.411)	YES
Krajewska-Włodarczyk *et al*. (2018) (30)	38 patients with PsO, 31 patients with PsA, 30 HC	case-control study	D	- clinical and ultrasonographic evaluation of nails and joints	- a significant correlation between increased nail bed thickness and PsA duration and swollen joint count. - increased power doppler signals in the nail bed significantly more frequent in PsA than PsO	YES

Author and year	Study population	Type of the article	GRADE	The applied diagnostic method	Outcome	Existing positive correlation
Idolazzi *et al*. (2019) (31)	51 patients with PsA, 31 patients with PsO, 37 patients with RA, 34 patients with OA, 50 HC	observational comparative study	D	- clinical and ultrasonographic evaluation of nails and joints	- significantly increased nail plate thickness in patients with PsA and PsO compared to RA and HC - power doppler signal at the enthesis was significantly more frequent in PsA, but rarely seen in PsO, and almost absent in RA and HC	YES
Antony *et al*. (2019) (14)	134 patients with PsA	retrospective cohort study	D	- clinical evaluation of nails - radiographic evaluation of joints	- nail dystrophy seen in 70% of patients with PsA - increased risk of erosions at the DIP joint level in case of onycholysis (OR 1.9) and subungual hyperkeratosis (OR 4.4), but not in case of nail pitting - no similar association was found at non-DIP joints	YES
Wiemann *et al*. (2019) (32)	187 patients with PsA, 31 patients with RA, 149 HC	observational case-control study with validation cohort	D	- indocyanine green enhanced fluorescence optical imaging based on Xiralite® system	- central hypoperfusion surrounded by peripheral hyperfluorescence (the "green nail" sign) observed in 22% of PsA patients in the primary study and 28% at the follow-up cohort - specificity 97% *vs*. RA	YES
Naredo *et al*. (2019) (33)	60 patients with PsA, 21 patients with PsO, 20 HC	prospective cohort study	D	- ultrasonographic evaluation of nails	- significantly higher nail bed thickness and nail plate thickness in both PsA and PsO patients than in HC, including in clinically non-involved nails (subclinical changes) - limited specificity of Doppler-based evaluation due to variability in HC	YES
Askin *et al*. (2024) (3)	763 patients with PsO (including 155 with PsA)	cross-sectional study	D	- clinical evaluation of nails and joints	- nail involvement identified as an independent predictor of PsA in multivariate analysis (OR 2.06, 95% CI 1.293–3.302, p=0.002)	YES
Mahmoud *et al*. (2024) (34)	22 patients with PsA, 21 HC	case-control study	D	- ultrasonographic evaluation of nails	- thickening of nail bed and adjacent skin significantly more common in PsA than in HC - a positively correlation of adjacent skin thickness correlated with tender joint count	YES
Cafaro *et al*. (2025) (11)	18 patients with PsA, 16 patients with PsO, 19 patients with RA, 19 HC	prospective cohort study	D	- ultrasonographic evaluation of nail-enthesis complex - nailfold videocapillaroscopy	- significantly greater severity and type of nail changes in PsA patients compared to HC - significantly more tortuous capillaries in PsA patients compared to HC	YES

PsA: psoriatic arthritis; PsO: skin psoriasis; HC: healthy controls; RA: rheumatoid arthritis; DIP: distal interphalangeal joint; NAPSI: nail psoriasis severity index; OA: osteoarthritis; GRADE scale: A – high quality of evidence, B – moderate quality of evidence, C – low quality of evidence, D – very low quality of evidence.

intervention in case of the involvement of nailplates (21, 22). Onycholysis and subungual hyperkeratosis constitute a potential indicator of enthesopathic processes at DIP joints (14, 17). Both ultrasound and NFC imagining may help to identify subtle subclinical nail and joint involvement (12, 20-24).

The observed association between scalp psoriasis and more severe phenotypes of PsA warrants further investigation into the potential mechanistic link between regional skin involvement and systemic joint disease. The anatomical characteristics of the scalp, particularly its proximity to entheseal (*e.g*., nuchal ligament and occipital bone insertions) make this area susceptible to microtrauma-induced immune activation (19, 27, 28). This localised mechanical stress was postulated to initiate or exacerbate entheseal inflammation through the "deep Koebner phenomenon," wherein cutaneous injury precipitates systemic immunologic consequences (19). The scalp is a unique immunologically active site, characterised by dense vascularisation and a high concentration of antigen-presenting cells, including dermal dendritic cells and Langerhans cells (15). Persistent inflammation in that region may facilitate antigen exposure and amplification of innate and adaptive immune responses, potentially contributing to systemic cytokine release.

The potential link between anogenital psoriasis and PsA has gained increasing attention in recent years. From an immunopathological perspective, the anogenital region is also characterised by unique skin and mucosal immunological environments (31, 33). Repeated exposure to microtrauma together with abundant microbiota were suggested to promote immune activation contributing to musculoskeletal inflammation (15). Recently, anogenital involvement was identified to be associated with significantly greater serum level of lipocalin- 2 in PsO patients, which was not observed in case of nails or hand involvement (35).

Despite these compelling associations, the exact pathophysiological mechanisms remain complex and not fully understood. Most included studies were observational in nature and varied in methodology, diagnostic criteria and definitions of special site involvement,

Table II. Summary of studies investigating the association between scalp psoriasis and psoriatic arthritis.

Author and year	Study population	Type of the article	GRADE	The applied diagnostic method	Outcome	Existing positive correlation
Elkayam *et al*. (2000) (21)	70 patients with PsA	prospective observational study	D	- PASI including scalp score - clinical evaluation of joints	- significant positive correlation between scalp psoriasis and number of swollen/deformed joints, dactylitis, and DIP involvement - the strongest correlation seen in patients with synchronous onset of PsO and PsA.	YES
Pavlica *et al*. (2005) (35)	96 patients with scalp PsO among 162 patients with PsA	retrospective observational study	C	- radiological evaluation of joints - skeletal scintigraphy - HLA typing	- statistically significant correlation between scalp psoriasis and the presence of a severe form of PsA	YES
Wilson *et al*. (2009) (23)	662 patients with scalp PsO among 1,633 patients with PsO (40 patients with already diagnosed PsA, 57 patients developed PsA at follow-up)	longitudinal, retrospective cohort study	B	- retrospective review of medical records from the Rochester Epidemiology Project	- a 3.75-fold increased risk for developing PsA in case of scalp psoriasis	YES
Langenbruch *et al*. (2014) (36)	4,863 patients with PsO (including 1,465 with PsA)	retrospective cross-sectional study	B	- retrospectively analysis of data from three large national cross-sectional studies conducted in Germany (2005, 2007, 2008)	- no increased risk for PsA development in case of scalp involvement (OR 1.07; 95% CI 0.90–1.26; p=0.457)	NO
Spelman *et al*. (2015) (27)	424 patients with PsO	cohort study	D	- clinical, serological and radiographic evaluation of joints	- scalp psoriasis was found to be independent *negative* predictor of PsA, (OR = 0.17, 95% CI: 0.05–0.55; p=0.003),	NO
de Vlam *et al*. (2016) (37)	373 patients with PsA including 273 patients with scalp PsO	randomised, multicentre clinical study	C	- activity of PsA measured with CRP, DAS28, joint counts, entehsitis and dactylitis	- no clear association with increased severity of PsA in case of scalp involvement	NO
Takata *et al*. (2016) (38)	18 patients with PsV (including 6 subclinical patients with PsA), 28 patients with PsA	cross-sectional study	C	- fluorodeoxyglucose positron emission tomography	- 100% prevalence of scalp involvement in subclinical PsA patients vs. 67% with only skin disease	YES
Yan *et al*. (2018) (39)	974 patients with PsO (including 175 patients with tients with PsA)	cross-sectional study	B	- clinical evaluation	- not significant association between scalp psoriasis and development of PsA in multivariate model	NO
Askin *et al*. (2024) (3)	763 patients with PsO (including 155 patients with PsA)	cross sectional study	D	- evaluation of scalp involvement with PSSI.	- scalp involvement showed no significant association with development of PsA - no correlation betwee scalp involvement and activity of PsA	NO

PsA: psoriatic arthritis; PsO: skin psoriasis; PSSI: Psoriasis Scalp Severity Index; GRADE scale: A – high quality of evidence, B – moderate quality of evidence, C – low quality of evidence, D – very low quality of evidence.

which limits the ability to draw firm causal conclusions. Moreover, conflicting results, particularly concerning the involvement of the scalp, highlight the need for standardied, prospective investigations.

Environmental factors modulating the relationship between special site psoriasis and psoriatic arthritis

The reviewed literature highlights several environmental factors contributing to the pathogenesis and clinical manifestations of psoriasis in special areas.

Mechanical trauma and repetitive micro-injury

Mechanical trauma, particularly repetitive micro-injury, is one of the most well-established environmental factors implicated in the pathogenesis of both psoriasis and PsA. Special anatomical locations such as the scalp, nails, intergluteal fold, and plantar/palmar skin are frequently subjected to mechanical friction or pressure, often resulting from clothing, footwear, occupation, or hygiene practices (36). These microtraumas can initiate the Koebner phenomenon, whereby damaged skin becomes the site of new psoriatic lesions.

At the cellular level, trauma stimulates keratinocytes and local immune cells

Table III. Summary of studies investigating the association between other special sites and psoriatic arthritis.

Author and year	Study population	Type of the article	GRADE	The applied diagnostic method	Outcome	Existing positive correlation
Wilson *et al*. (2009) (23)	1,633 patients with PsO (40 patients with already diagnosed PsA, 57 patients developed PsA at follow-up)	longitudinal, retrospective cohort study	B	- retrospective review of medical records from the Rochester Epidemiology Project	- a 1.95-fold increased risk of PsA (HR 1.95, 95% CI 1.07–3.56) in case of psoriasis affecting the intergluteal or perianal areas - a 2.24-fold risk (HR 2.24, 95% CI 1.23–4.08). In case of multiple involvement of special sites	YES
Takata *et al*. (2016) (38)	18 patients with PsV (including 6 subclinial patients with PsA), 28 patients with PsA	cross-sectional study	C	- fluorodeoxyglucose positron emission tomography	- 83% prevalence of scalp involvement in subclinical PsA patients *vs*. 25% with only skin disease	YES
Yan *et al*. (2018) (39)	974 patients with PsO including 175 patients with PsA	cross-sectional study	B	- clinical evaluation and statistical modeling	- no significant association found in either univariate or multivariate analysis (OR 0.90; p=0.739).	NO
El-Garf *et al*. (2021) (40)	200 patients with PsO (known PsA 8, newly diagnosed 52)	cross sectional study	B	- PEST and EARP questionnaires and clinical evaluation	- intergluteal psoriasis identified as the strongest independent predictor of PsA (OR=12.66; CI: 4.30 - 37.25; p<0.001).	YES
Loo *et al*. (2023) (41)	360 patients with PsO including 107 patients with PsA (30 newly diagnosed)	cross-sectional	C	- clinical evaluation - ESR and CRP measurements	- a 2.25-fold increased risk of development for PsA in case of involvement of genital area - higher ESR & CRP levels in those patients with PsA with concomitant involvement of genital area	YES

PsA: psoriatic arthritis; PsO: skin psoriasis; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein erythrocyte sedimentation rate; GRADE scale: A – high quality of evidence, B – moderate quality of evidence, C – low quality of evidence, D – very low quality of evidence.

to initiate innate immune responses through pattern recognition receptors, such as Toll-like receptors (TLRs) (37). This leads to the local release of cytokines such as IL-1β, IL-23, and TNF-α, which promote Th17 polarisation. The IL-17/IL-23 axis then drives keratinocyte proliferation and inflammation, linking localised skin injury to both cutaneous flares and systemic immune activation. Importantly, microtrauma in nail or extensor tendon insertion sites can extend inflammation to the adjacent entheses, forming a direct anatomical and immunological bridge to PsA onset (38).

Moisture and occlusion

Special sites such as the axillae, inframammary folds, groin, and genital area present a distinct microenvironment defined by high moisture, low ventilation, and frequent occlusion. This occlusive environment alters skin barrier function, increases pH, and promotes overgrowth of bacteria and yeasts (*e.g*. *Candida* species). Reduced exposure to ambient air limits keratinocyte differentiation and tight junction integrity, enhancing skin permeability and increasing susceptibility to irritants and pathogens. As a result, there is a predominance of interleukin IL-17-related pathways, along with reduced antimicrobial peptide expression. These immune deviations contribute to sustained local inflammation and may act as triggers for systemic immune dysregulation. Additionally, the hidden nature of lesions in these areas often results in delayed diagnosis and suboptimal treatment, increasing the risk of chronicity and systemic spread (36, 37).

Infectious triggers and microbial dysbiosis

Microbial factors, both pathogenic and symbiotic microbes are increasingly recognised as critical modulators of the psoriasis-PsA axis. Infections with *Streptococcus pyogenes*, *Staphylococcus aureus* or *Candida albicans* have been implicated in triggering or exacerbating psoriasis, especially in the scalp, nails, and flexural areas. These pathogens function as superantigens or disruptors of microbial balance (dysbiosis), enhancing the immunogenicity of skin-resident cells.

Such infections activate pattern recognition receptors (*e.g*. TLRs, dectin-1) on dendritic cells, stimulating IL-23 production and fostering a pathogenic Th17 response. In parallel, dysbiosis in the gut and skin microbiome has been associated with chronic systemic inflammation and altered regulatory T cell function (39, 40). Given the frequent microbial colonisation of nail folds, gluteal cleft, and genital skin, these regions may serve as entry points for immune activation, acting as sentinel sites for subsequent PsA development (37, 41).

Chemical and biological irritants

Patients with psoriasis affecting special sites such as the genital area, perianal region, and skin folds are frequently exposed to various external irritants that may exacerbate local inflammation or hinder healing. Common aggravating factors include biological substances like urine, feces, sweat, and vaginal secretions, as well as chemical irritants such as soaps, detergents, disinfectants, scented hygiene products, and occlusive topical agents (36).

Seasonal and climatic influences

Climatic conditions exert a profound influence on psoriatic disease, particularly in patients with special-site involvement. Cold, dry environments

are associated with increased disease severity, especially in the scalp, palms, and intertriginous areas, which are more prone to microfissuring and mechanical stress during winter months.
In contrast, exposure to moderate sunlight radiation promotes keratinocyte apoptosis, decreases dendritic cell activation, and modulates cytokine profiles toward an anti-inflammatory state. These seasonal variations can determine the immune tone of the cutaneous microenvironment and, by extension, the risk of systemic immune activation leading to PsA (37, 41).

Smoking- and alcohol-induced immune modulation
Cigarette smoking is a significant environmental factor in psoriatic disease. It contributes to oxidative stress, weakens antioxidant defenses, increases blood viscosity, and disrupts endothelial function. Components such as nicotine and reactive oxygen species promote the release of proinflammatory cytokines, including IL-12, IL-2, TNF, IFN-α, and GM-CSF, thereby supporting the development and persistence of inflammation in psoriasis. In patients with established psoriatic arthritis, smoking is consistently associated with worse clinical outcomes, including more active joint disease and reduced response to treatment. Alcohol use, although inconsistently linked to psoriasis onset, is known to stimulate cytokine production and promote proliferation of lymphocytes and keratinocytes, potentially contributing to chronic systemic inflammation and lesion persistence (41, 42).

UV radiation
Ultraviolet (UV) radiation exerts a multifaceted influence on psoriatic disease. Although commonly employed as a treatment modality due to its anti-inflammatory effects, in some individuals UV exposure can unexpectedly lead to symptom aggravation. The exact mechanisms behind this phenomenon remain unclear, but are thought to involve sunburn-induced skin trauma (Koebner phenomenon) or coexisting conditions characterised by increased photosensitivity. A specific subset of patients develops psoriasis flare-ups directly in response to sunlight. This form, referred to as photosensitive psoriasis (PP), typically presents with seasonal worsening and the emergence of new lesions in UV-exposed regions. PP often resembles polymorphic light eruption (PLE) in its clinical presentation and may overlap with it in a considerable number of cases (37, 41).

Obesity and metabolic inflammation
Obesity is both a comorbidity and an independent risk factor that exacerbates psoriasis and psoriatic arthritis (PsA), particularly in special anatomical sites. It contributes through chronic low-grade systemic inflammation and mechanical overload, especially in weight-bearing areas such as the soles, nails, and gluteal cleft. Adipose tissue functions as an active immune organ, releasing proinflammatory adipokines and cytokines (*e.g*, leptin, TNF-α, IL-6), which enhance keratinocyte proliferation and Th17-mediated inflammation. Mechanical stress further aggravates local immune responses at entheses, promoting enthesitis and joint changes characteristic of PsA (41, 42).

Limitations
Although nail involvement demonstrates the most robust statistical association with PsA among the special sites reviewed, it is essential to underscore that causality cannot be inferred from the existing evidence. The majority of studies included in our analysis were observational and scored low on the GRADE quality scale, with most rated as "D" indicating very low certainty. Studies assessing nail involvement were of low methodological quality, often lacking randomisation, standardised diagnostic criteria, or adjustment for confounders. While imaging modalities such as ultrasonography and nailfold capillaroscopy enhance the detection of subclinical disease and provide mechanistic insight into the nail-entheseal complex, the evidence remains insufficient to draw definitive causal inferences. Therefore, although the recurring association between nail psoriasis and PsA across various cohorts may reflect a true pathophysiological relationship, the current body of evidence does not allow for strong prognostic conclusions, and the findings must be interpreted with caution. Future research should prioritise longitudinal, high-quality prospective studies with rigorous methodological design to better establish temporal relationships and causative pathways.
Scalp psoriasis has been proposed as a potential marker of PsA risk due to its immunological and anatomical characteristics but the current evidence remains conflicting and warrants critical appraisal. However, five studies, including large-scale (GRADE B) analyses by Langenbruch *et al*. and Yan *et al*., failed to confirm this association, suggesting that scalp psoriasis may not independently predict PsA when adjusted for confounders such as disease duration or severity. Notably, Spelman *et al*. even reported a negative predictive value. These discrepancies may arise from heterogeneity in diagnostic definitions, patient populations and limited adoption of standardised assessment tools such as the Psoriasis Scalp Severity Index (PSSI), which, despite its availability, is not routinely utilised in clinical practice due to interobserver variability and challenges in accurately scoring lesions obscured by hair density and scalp morphology. Taken together, while the scalp represents a biologically plausible site of PsA initiation due to its exposure to microtrauma and immunological priming, current evidence does not uniformly support a predictive or causal link. The overall quality of scalp-related studies is variable, and inconsistencies highlight the need for longitudinal, multicentre studies employing objective imaging and biomarker-based approaches to elucidate the role of scalp involvement in PsA pathogenesis.
Moreover, the heterogeneity observed among studies investigating anogenital regions, underscores the necessity for greater methodological standardisation. Numerous studies did not adequately account for key confounding variables such as prior or ongoing systemic therapy, duration of cutaneous disease, or the presence of comorbidities, and frequently lacked harmonised

outcome measures. Even in studies utilising advanced imaging modalities, inconsistencies in scoring protocols and interpretative frameworks limited reproducibility and comparability of results.

While recent advances in artificial intelligence (AI) have shown promise in identifying clinical patterns and predicting disease outcomes in PsA, their application in distinguishing patients with special site psoriasis at risk for PsA remains limited. As noted by Esti *et al*. (43) machine learning models have been successfully used to predict minimal disease activity and remission likelihood, as well as to differentiate PsA from other inflammatory arthritides using imaging data. However, these tools are still in early developmental stages and have not yet been widely validated across diverse populations or integrated with site-specific phenotypic data, such as nail or anogenital involvement. Future AI models should aim to incorporate special site variables to improve early diagnostic precision and targeted prevention strategies.

Conclusions

The localisation and the extent of psoriasis, especially when involving special sites such as the nails, scalp and anogenital regions, should be recognised not merely as markers of dermatologic burden but as clinically significant predictors of PsA risk. The involvement of nails shows the strongest association with PsA development due to the connectivity between the nail apparatus and the entheseal-joint interface. Scalp psoriasis may reflect or exacerbate systemic inflammation, but data remains inconsistent. Anogenital psoriasis appears to signify the distinct immunopathological phenotype due to the unique microenvironment. These associations are likely amplified by site-specific environmental factors such as mechanical trauma, microbial colonisation, and occlusion-induced immune deviation, which act as local triggers capable of initiating or perpetuating systemic immune activation. Micro-injury at enthesis-rich regions such as the nail bed or plantar surfaces may serve as a mechanistic conduit linking cutaneous inflammation to articular pathology. Moisture-prone areas like the anogenital region further demonstrate altered barrier function and a shift toward Th17-dominated responses, providing a biologically plausible foundation for PsA development. However, methodological limitations across existing studies, including heterogeneity in diagnostic definitions and insufficient control for confounders, preclude definitive causal inference. Future longitudinal, biomarker-driven research is essential to clarify these pathways and to determine whether targeted intervention in special site psoriasis can modify PsA risk or trajectory.

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