The Path to Interception in Psoriatic Disease: From Conceptual Clarity to Clinical Translation

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Summary

Psoriatic arthritis (PsA) develops in up to one-third of individuals with psoriasis, typically following a prolonged subclinical phase. Diagnostic delays are common, often exceeding two years, and can result in irreversible joint damage. The growing recognition of this latent period has fuelled interest in earlier identification and interception. However, efforts are hampered by inconsistent definitions of “early” or “subclinical” PsA, limited prognostic tools, and lack of consensus on the outcome for interception studies. This review synthesises a rapidly evolving field, offering a framework organised around four critical questions: i) *What* defines progression from psoriasis to PsA? ii) *Who* is most at risk of transition? iii) *How* can progression be reliably measured using imaging, molecular biomarkers, or digital health technologies? and iv) *When* should preventive intervention be considered? We critically examine new conceptual models, the limitations of existing classification criteria, advances in imaging and biomarker research, and the promise of digital phenotyping. Addressing the current challenges in definitions, risk stratification, measurement, and trial design is essential for the development of biologically grounded, ethically robust interception strategies.

Introduction

Psoriatic disease, encompassing psoriasis and psoriatic arthritis (PsA), are inflammatory conditions of the skin and joints, affecting 2–4% of the population, with substantial physical and psychosocial burden.[1] Around one-third of people with psoriasis develop PsA, with skin symptoms typically preceding joint involvement by several years.[2] This latency offers a "window of opportunity" for early detection and intervention.[3,4] Despite this, up to 50% of patients with new onset PsA experience diagnostic delays, often exceeding two years, contributing to irreversible joint damage and long-term disability.[5–7]

Recognition that PsA often follows a prolonged subclinical phase has driven interest in earlier detection to prevent irreversible damage.[4,8,9] Prevention in psoriatic disease can be conceptualised at three levels: *primary*, reducing the likelihood of PsA or even psoriasis itself; *secondary*, identifying and addressing early or subclinical manifestations to limit or halt progression; and *tertiary*, the current paradigm, minimising joint damage once PsA is established. Within this framework, *disease interception* fits within secondary prevention, with the goal of altering the course of disease before conventional classification or diagnostic criteria are met. This shift aligns with wider moves across other chronic diseases towards secondary prevention through interception. A distinctive feature of psoriatic disease is the presence of a well-defined at-risk population in people with psoriasis, providing a unique foundation for interception strategies.

Disease interception approaches in rheumatoid arthritis, inflammatory bowel disease, and Parkinson's disease offer informative examples, having already begun to tackle similar challenges through staged models of progression, integration of biomarkers and imaging, and structured interception trials.[10–12] A recently proposed framework distilled the key challenges in therapeutic interception trials into four guiding questions: i) *What* defines progression, ii)

Who should be enrolled, iii) *How* to reliably measure progression, and iv) *When* to intervene effectively.[12] Psoriatic disease interception remains in its infancy, and this structured approach offers a pragmatic lens to guide efforts in defining disease stages, selecting high-risk individuals, and developing robust intervention trials. By addressing these questions, we offer a practical roadmap for researchers and clinicians to guide the next phase of research in therapeutic interception in psoriatic disease.

What is the target? Defining the Transition from Psoriasis to PsA

Effective interception of psoriatic arthritis requires a robust definition of what constitutes disease progression. Recent proposals attempt to bridge this gap, conceptualising discrete stages along the psoriasis-to-PsA continuum.[4,8,9,13] However, fundamental disagreements persist on precisely when the disease transitions into overt psoriatic arthritis. Clarifying these definitional uncertainties is essential for effective risk stratification, timely intervention, and robust trial design in therapeutic interception.

Comparing Prominent Proposals

The “2023 EULAR points to consider” outlines three main stages: individuals with long-term risk factors, those with subclinical PsA (defined by arthralgia and/or imaging abnormalities), and those with PsA.[8] This contrasts with earlier, more granular models such as the Scher et al. framework, which describes a stepwise evolution through immune activation, asymptomatic biomarker or imaging abnormalities, and non-specific musculoskeletal symptoms.[4] In parallel, the Psoriasis and Psoriatic Arthritis Clinics Multicenter Advancement Network (PPACMAN) consensus definition provides common terminology for the preclinical and early clinical phases to support research.[9] The EULAR points to consider were formulated to guide both clinical trials and clinical practice in the evolving area of therapeutic interception. By contrast, the PPACMAN definition was developed with a narrower scope to support

clinical trials and translational research in the preclinical and early clinical phases of psoriatic disease (Table 1).[8,9]

A fundamental point of divergence between these proposals is the classification of individuals who have musculoskeletal symptoms (e.g. arthralgia, stiffness) in combination with imaging-confirmed inflammatory changes (e.g. synovitis or enthesitis on ultrasound or MRI). The EULAR framework maintains that this constellation satisfies the definition of "subclinical PsA", unless synovitis is clinically evident.[8] This contrasts with a recent viewpoint from Ciccia et al,[13] which highlights that the co-occurrence of symptoms and inflammation on imaging marks a critical threshold, sufficient to define *"very early PsA"* (Figure 1). In this model, subclinical PsA is reserved for symptomatic individuals without evidence of synovio-entheseal inflammation.[13] The implication is that once inflammation can be objectively verified, the disease has already transitioned biologically, even if conventional classification criteria are not yet satisfied. The distinction between *"subclinical"* and *"very early"* PsA is more than semantic and shapes how researchers define eligibility for trials, how clinicians make decisions around monitoring or initiating therapy, and how patients are counselled about disease risk.

Defining Outcomes: From CASPAR to Synovitis

The Classification Criteria for Psoriatic Arthritis (CASPAR) are widely used for clinical research and regulatory purposes.[14] These criteria demonstrate high sensitivity and specificity in established PsA, relying on features such as dactylitis, radiographic changes, or synovitis. However, their utility is limited in early or subclinical phases of PsA, where such hallmark features may not yet be present.[4,15] Crucially, CASPAR requires the presence of *"inflammatory disease"* at the joints, entheses, or spine as a prerequisite,[16] yet what

constitutes inflammation in the very early or preclinical stages remains inconsistent, as highlighted.

Recent frameworks propose refined outcome definitions to capture earlier stages. Notably, the EULAR proposal recommended clinical synovitis, rather than fulfilment of CASPAR, as the outcome for progression to PsA, specifically in interception trials.[8] The supporting systematic review found that ~80% of incident early PsA presents with peripheral joint swelling, providing a pragmatic, reproducible endpoint for early-phase trials.[15] This enhances reproducibility and diagnostic clarity but risks overlooking patients with early enthesitis or axial involvement, which are less common but recognised presentations in early disease, albeit from studies with small sample sizes.[15,17–19] Reliance on clinical synovitis as a binary threshold may oversimplify the complexity of PsA pathogenesis, since synovitis in early disease may be transient and absent on the day of clinical assessment, potentially delaying intervention in those with imaging-positive, symptomatically evolving disease. Detecting synovitis on clinical exam also depends on clinician skill, risking missed cases of occult inflammation. The EULAR taskforce recognised important limitations in the underpinning evidence for defining transition in early disease, including heterogeneous definitions and outcomes, small cohorts for enthesitis predominant and axial onset disease, and few prospective cohorts or longitudinal imaging studies to characterise transition.[8,15]

Musculoskeletal ultrasound (MSUS) has been investigated as a tool to augment the sensitivity of the CASPAR criteria, particularly in early disease.[20,21] In one study, US-modified CASPAR criteria identified almost twice as many patients as the conventional CASPAR in a cohort of psoriasis patients with and without musculoskeletal pain.[20] These findings lend empirical weight to the Ciccia position that symptomatic individuals with imaging-confirmed inflammation represent a biologically active state best described as *very early PsA*.[13] However, in the current heterogeneous landscape of subclinical and early PsA definitions,

this approach introduces a risk of diagnostic circularity. Within the EULAR framework, such individuals would still be classified as “subclinical”. Their imaging abnormalities, if used to both stratify risk and define the outcome of progression, risks incorporation bias.[22~~22~~] This bias arises when the same test result informs both the predictor and the reference standard, which can inflate associations.[22~~22~~]

Need for unified definitions

Current frameworks reflect growing interest in intercepting in PsA at an early stage, but diverge on when PsA truly begins. Whether symptomatic individuals with imaging-confirmed inflammation are considered “subclinical” or “very early” PsA has real implications: for trial design, for when to intervene, and for how patients are counselled. Until these definitions are harmonised, the field risks inconsistency in research and clinical decision-making.

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Domain	EULAR (2023 'Points to Consider')	PPACMAN	Ciccia et al. (mechanistic model)
Primary purpose	Operational guidance for clinical trial design; not intended for routine clinical diagnosis.	Trial enrichment and stratification of at-risk and subclinical populations.	Immunopathogenic staging; conceptual/mechanistic rather than operational.
Intended scope / audience	Researchers designing early-PsA trials; consensus-based.	Researchers and trialists; focuses on at-risk psoriasis cohorts.	Researchers; theoretical framework, non-consensus.
Terminology note	Defines stages; uses clinical synovitis as trial outcome for progression.	Defines at-risk and subclinical states for recruitment; outcome varies by study.	Posits 'very early PsA' when symptoms plus imaging-confirmed inflammation coexist.
Imaging role	Supportive (US/MRI) for risk and staging; cautions use of US in asymptomatic psoriasis patients.	Often required for entry (e.g., MSUS); enrichment strategy.	Central to staging as biological threshold of inflammation.
Progression outcome (trials)	Clinical synovitis (not necessarily CASPAR) for reproducibility in early disease.	CASPAR criteria	Imaging-confirmed inflammation considered biologic transition; CASPAR viewed as late.
Clinical phenotypes captured at PsA onset	Captures peripheral synovitis; may miss entheseal-only or axial-only onset.	Captures peripheral and entheseal inflammation.	Highlights entheseal and synovial pathways; accommodates axial presentations at index.
Risk enrichment strategy	Symptoms ± imaging; conservative thresholds for progression.	Imaging-heavy enrichment to raise event rates.	Biology-led staging; less operational validation for screening.
Strengths	Simple, reproducible outcome; consensus derived.	Pragmatic for recruitment; aligns with current interception trials (e.g., PAMPA).	Mechanistic clarity; sensitive to early biological change.
Limitations / risks	May miss axial/enthesitis-dominant disease; relies on examiner skill for synovitis.	Risk of incorporation bias if imaging used as both predictor and outcome; heterogeneity across sites.	Non-consensus; operational criteria not standardised for trials.

Ethical / operational implications	Minimises overtreatment risk; may delay therapy in imaging-positive symptomatic patients.	Enables earlier intervention but may broaden eligibility; and false positives.	Supports early biological intervention; risk of overtreatment if applied outside trials.

The Who: Risk Stratification and Biomarkers

Having recognised the challenges in defining early psoriatic disease, the next critical step is identifying which individuals are most likely to transition from psoriasis to PsA. A range of approaches to risk stratification have been proposed, spanning clinical algorithms, genomic profiling, circulating biomarkers, and advanced imaging. In this section, we explore emerging evidence for these approaches and how they might be integrated into a coherent strategy to identify high-risk individuals, both for inclusion in interception trials and enhanced surveillance pathways.

Clinical risk prediction approaches

Clinical risk prediction models aim to identify individuals with psoriasis who are at elevated risk of developing psoriatic arthritis by leveraging large-scale electronic health record (EHR) data,[23–28] or readily available clinical and patient-reported outcome measures.[29] These models combine demographic factors, comorbidities, medications, and healthcare utilisation patterns to estimate PsA risk. Robust evidence from epidemiological studies highlights several clinical features consistently associated with an increased risk of transition from psoriasis to PsA, including psoriasis severity, nail involvement, and obesity.[4,30,31] These factors have been reproduced across cohorts with a prominent example being the recent PRESTO-PsA model, which provides individual-level risk estimates over both short (1-year) and medium-term (5-year) horizons, based on features including psoriasis severity (PASI), nail involvement, fatigue, morning stiffness, pain level, and medication use. Even when all

predictors suggest maximal risk, the absolute predicted probability remains modest at approximately 20% over one year and 40-45% over five years.[29] Psoriasis patients identified as higher risk by PRESTO-PsA might be triaged for further detailed assessments, such as musculoskeletal ultrasound or MRI, enhancing the efficiency of classifying early and subclinical disease, however, the risk thresholds to warrant further investigation have not yet been defined. Furthermore, these model estimates were derived with the outcome set as CASPAR-confirmed PsA. Recalibration of such a model with the outcome set as a clearly defined subclinical phenotype may produce higher point estimates of risk.

In the realm of large-scale EHR-based tools, there has been a rapid expansion across medicine in leveraging readily accessible longitudinal patient records to generate prognostic and diagnostic models and inform clinical practice.[32,33] In principle, these models could offer a scalable strategy to enrich interception trial recruitment and inform targeted screening. However, the low incidence rate of PsA poses fundamental challenges. Even well-calibrated models often yield low positive predictive values in this setting, as the majority of patients flagged as high-risk will not progress to PsA. Furthermore, several biases may affect the development and performance of population-based models utilising EHR data. Differential surveillance bias arises when individuals with psoriasis are subject to more frequent clinical evaluations, leading to higher rates of PsA diagnosis that reflect healthcare utilisation rather than true biological risk.[34,35] Conversely, a patient could present with active PsA for many months or even years before a formal diagnosis.[6] Non-differential misclassification bias occurs when key exposures or outcomes, such as comorbidities, prescriptions, or diagnostic codes are inconsistently or inaccurately recorded across the dataset, can further bias estimates.[36] This bias can also be due to differential access in healthcare due to socioeconomic or participant-level characteristics such as age, sex or ethnicity.[36] Misclassified person-time represents another challenge, where inaccuracies in the timing of

symptom onset, diagnosis, or risk factor exposure can distort the temporal associations that underpin model performance.[37] EHR lack validation and quality control, and likely contain flawed information.

The transition to PsA is a dynamic continuum as the result of evolving immune processes that may accelerate or regress over time. Risk models should reflect this dynamic nature by incorporating longitudinal data, enabling the recalibration of risk estimates as new information becomes available. External validation across diverse populations is critical to ensure model generalisability, and future tools must be transparent, interpretable and clinically meaningful to support robust deployment.[32,33]

Capturing subclinical inflammation: the role of imaging

Imaging has helped to establish that psoriasis patients without musculoskeletal symptoms often harbour subclinical inflammation, supporting the concept of a preclinical phase. Peripheral manifestations such as synovitis, enthesitis, tenosynovitis, peri-tendinous oedema and soft tissue oedema are most frequently observed.[38]

Ultrasound (US) has been central in demonstrating these early changes. In one cohort, up to 40% of psoriasis patients without musculoskeletal symptoms had US-detected synovial inflammation or enthesitis, and were subsequently more likely to develop PsA.[39] A recent prospective study demonstrated that bursitis at the entheses was associated with PsA development, again potentially underlining the link between early entheseal inflammation and disease transition.[40] Ultrasound-detected structural entheseal lesions appear discriminative between psoriasis and PsA.[39] In a 2022 study, enthesophytes were the only significant predictor of subclinical PsA among psoriasis patients however this work was from a small sample, and included imaging in the clinical criterion risking incorporation bias.[39] In a recent cohort of 141 psoriasis patients with mild skin disease, entheseal erosions had a 3.7-

fold higher risk of PsA.[41] However, it is important to note that US is operator dependent, and agreement between readers and across centres is variable, which can limit reproducibility in multicentre studies.[42–44]

High-resolution MRI studies confirm that many psoriasis patients have occult joint inflammation prior to clinical arthritis: in one imaging study, nearly 47% of psoriasis patients without PsA had at least one inflammatory lesion on hand MRI, most commonly synovitis (38% of patients), with smaller proportions showing osteitis or tenosynovitis.[45] The presence of subclinical synovitis on MRI and concurrent hand arthralgia had markedly higher conversion rates to PsA (around 60% at one year) compared with only ~13% in those without inflammation.[45] Emerging work with PET-CT similarly suggests potential for detecting subclinical entheseal and synovial inflammation, though its role remains exploratory.[46,47]

Together, these studies reinforce that imaging can identify a biologically active preclinical phase. A key challenge is a lack of standardisation on imaging protocols for clinical practice and research.[48] In a recent ACR/EULAR joint taskforce on risk stratification for early RA, lack of harmonisation of US variables hampered inclusion into the final prediction model.[49] In psoriatic disease, unifying the data collection with a consistent US acquisition protocol will permit meta-analyses of longitudinal studies. The ongoing GRAPPA Diagnostic Ultrasound Enthesitis Tool (DUET) project aims to develop a data-driven tool for identifying enthesitis at key discriminatory sites across the continuum of psoriatic disease and will be an important complement for longitudinal studies exploring risk of progression.[50] Longitudinal cohorts with harmonised protocols will also be critical to clarify the temporal dynamics between subclinical inflammation and PsA onset, and to account for natural regression of lesions that could otherwise confound risk estimates and trial endpoints. Operator dependency and inter-reader variability reinforce the need for harmonised acquisition, training, and, where possible, central reading. Unanswered questions remain around natural

regression of subclinical inflammation, which may confound risk estimates and trial endpoints. Importantly, progress in standardising imaging for research will ultimately support its implementation in routine clinical practice, benefiting patients through more consistent assessments and earlier identification of those at risk.

Arthralgia as a predictor of imminent transition

Identifying individuals at imminent risk of PsA is a central goal for interception strategies. Unlike background risk, which may remain constant over years, imminent predictors reflect short-term changes that suggest active transition toward overt PsA. These may include new-onset musculoskeletal symptoms or imaging evidence of subclinical inflammation.

Unexplained arthralgia has emerged as one of the most studied indicators of imminent risk and features prominently across multiple definitions of subclinical PsA. However, a standardised definition of arthralgia in the context of PsA transition remains elusive. In a prospective study by Zabotti et al,[51] individuals with psoriasis and arthralgia but without clinical synovitis were found to have a markedly increased risk of developing PsA.[48] The authors defined arthralgia as either inflammatory (presence of morning stiffness lasting >30 minutes, symptoms worse in the morning, and improvement throughout the day), non-inflammatory (absence of these features) or mixed. The annual incidence rate in this enriched cohort was 7.7 per 100 person-years, with cumulative incidence estimates of 9.4 percent at 12 months and 22.7 percent at 36 months.[51] This contrasts with incidence rates in the general psoriasis population estimated at approximately 1.87 per 100 person-years.[52] Notably, most individuals who progressed to PsA initially reported symptoms consistent with non-inflammatory arthralgia, often present for several years before diagnosis. This challenges the assumption that inflammatory symptoms are the primary harbinger of transition and suggests

that subtle or non-specific joint complaints may precede inflammatory disease. These findings, however, must be interpreted with caution. The cohort was selectively enriched, excluding individuals with conditions such as osteoarthritis or fibromyalgia. This approach increases the observed effect size but limits generalisability. In routine clinical care, non-inflammatory musculoskeletal symptoms are common and often benign, meaning that reliance on arthralgia alone may lead to false positives and unnecessary investigations. Furthermore, the progression rate to PsA was still the minority which will have implications for communicating this risk with patients.

Arthralgia remains a subjective and heterogeneous symptom, and distinguishing inflammatory from non-inflammatory causes is a challenge. This underscores the need for validated, structured tools to track musculoskeletal symptoms longitudinally in individuals with psoriasis. The International Dermatology Outcome Measures Musculoskeletal Questionnaire (IDEOM MSK-Q) has recently been validated in a large real-world cohort.[53] Designed for use across the full spectrum of psoriatic disease, it captures musculoskeletal symptom intensity, impact on daily life, and fatigue. Unlike the PsA Impact of Disease (PsAID) questionnaire,[54] which presumes a PsA diagnosis, the IDEOM MSK-Q is disease-stage agnostic and therefore well suited for prospective studies of at-risk individuals.

Genomic and proteomic-based markers

Efforts to enhance PsA risk prediction have increasingly focused on the integration of genomic data.[55] Psoriasis and PsA share substantial genetic overlap, complicating attempts to identify discriminative loci for PsA-specific prediction. Although genome-wide association studies (GWAS) have identified several loci enriched in PsA compared to psoriasis alone, such as variants related to *IL23R* and *HLA-B*;[56] their added predictive value remains modest. HLA-B27 appears to be a marker for a SpA-like endotype within the Psoriatic disease

spectrum. It may also be more than that with previous studies showing that HLA-B27 is associated with early onset, severe PsA and features of enthesitis, dactylitis in addition to symmetric sacroiliitis.[57,58] Uveitis, identified as a predictor of PsA development in psoriasis,[17] is also associated with HLA-B27 but as uveitis is a relatively rare occurrence in PsA (<5%), it is unlikely to be sufficiently sensitive to detect the 30% of patients with psoriasis who will transition to PsA. The lack of limited PsA specific genetic markers is reflected in the comparatively poor performance of polygenic risk scores (PRS) for PsA, showing limited discriminatory capacity when applied to individuals with psoriasis.[59] In cardiovascular disease, PRS can complement clinical models by improving risk stratification.[60,61] The scarcity of disease-specific loci associated with transition from psoriasis to PsA, the shared genetic architecture, and the heterogeneity of clinical phenotypes constrain their utility.

Alongside genomics, proteomic and metabolomic profiling has gained traction as a route to uncover molecular signatures that distinguish PsA from psoriasis alone. Studies have explored serum protein panels, cytokines, lipid mediators, and metabolic profiles, often identifying markers that differ in cross-sectional comparisons. Yet few have demonstrated reproducibility across cohorts, and none have achieved the sensitivity or specificity required for clinical use.[56,62] A recent narrative review of over 30 studies published between 2020 and 2024 highlighted this gap, noting that promising molecular candidates have been undermined by small sample sizes, variation in assay methodology, and inconsistent case definitions[62]. As a result, proteomic and molecular biomarkers have yet to offer reliable differentiation between psoriasis and PsA in real-world settings.

Larger, harmonised datasets are urgently needed to power more reliable genomic discovery and risk prediction efforts. International consortia such as HIPPOCRATES (Health Initiatives in Psoriasis and PsOriatic Arthritis ConsoRTium European States) offer a promising model

for data and sample sharing to overcome the fragmentation and underpowering that have limited prior studies.[63,64] As is hypothesised by the HIPPOCRATES consortium, it is more likely that it will be a combination of clinical and molecular factors, including genetic markers such as HLA-B27, which will successfully identify those patients who will transition from cutaneous psoriasis to PsA.[65] By enabling replication, stratified analyses, and the development of integrated multi-omic risk models, these efforts may ultimately support the identification of individuals at risk with greater precision and clinical relevance. Future risk stratification tools may benefit from multi-modal integration, combining background risk such as genetic susceptibility, with dynamic molecular or clinical features to comprehensively capture risk over time (Figure 2). Background risk reflects fixed, long-term predisposition, while dynamic risk reflects fluctuating signals such as arthralgia, imaging changes, or molecular activity.

How to measure progression in interception trials?

Building on previous discussions about the conceptual clarity needed for defining disease progression in PsA, a pivotal challenge in designing interception trials is how to reliably identify and monitor individuals in the transition phase, and ultimately, how to measure the impact of interventions. At face value, incidence of PsA appears the most robust and critical outcome for any trial exploring preventive interventions. However, this is particularly complex due to the heterogeneous definitions for what constitutes "early" or "subclinical" PsA as well as challenges of classification criteria for PsA in the early stages of the disease, directly impacting the selection and implementation of endpoints. This is further complicated by the relatively low conversion rates observed even in high risk enriched cohorts of psoriasis with arthralgia. In a hypothetical interception trial enrolling individuals with an annual

incidence rate of 7.7 per 100 person-years (as reported by Zabotti et al.),[51] one would expect approximately 15% of participants to transition to PsA over two years of follow-up. The sample size requirements depend heavily on the anticipated effectiveness of the intervention. For a 50% effective intervention (i.e., reducing the 2-year conversion from 15% to 7.5%), approximately 570 participants would be required (285 per arm) to achieve 80% power to detect a statistically significant difference at a two-sided α of 0.05. For a 30% effective intervention (reducing conversion to 10.5%), the required sample size increases substantially to approximately 1,860 participants (930 per arm) under the same conditions.

Given these practical constraints, reliance on PsA diagnosis as the primary outcome presents feasibility challenges for interception trials in psoriatic disease. As a result, there is interest in employing surrogate endpoints that might offer a more sensitive, timely, and pragmatic means of assessing efficacy. While robust blood-based molecular biomarkers for PsA transition remains elusive, MSUS has emerged as a promising tool. The validity of MSUS as a responsive surrogate endpoint is supported by prospective observational data. In an open-label study by Savage et al, treatment of moderate-to-severe psoriasis with the IL-12/23 inhibitor, ustekinumab, resulted in a rapid and sustained reduction of subclinical entheseal inflammation, as measured by serial ultrasound, with mean inflammation scores decreasing by over 40% within the first 24 weeks of treatment.[66,61] In addition, a recent small observational study from Liu et al, has reported regression of subclinical synovitis on ultrasound in subclinical PsA.[67] These findings demonstrate that imaging-defined resolution of subclinical inflammation is both feasible and sensitive to intervention, however, it remains unknown whether this translates into lower rates of progression.

The PAMPA Trial: A First Step Towards Interception

The Preventive Approach to Psoriatic Arthritis (PAMPA) trial is the first randomised controlled trial investigating whether early biologic therapy can alter psoriatic disease progression.[68,69] This multicentre, double-blind trial evaluates guselkumab versus placebo in psoriasis patients with PDUS-detected subclinical inflammation, not meeting CASPAR criteria. Coprimary endpoints are change in PDUS score at 24 weeks and PsA incidence rates at 96 weeks. A parallel observational arm of patients declining biologic therapy will provide valuable natural history data to benchmark intervention effects and inform future trial design.

A particular strength of the PAMPA trial is the inclusion of an observational arm comprising individuals who decline randomisation to biologic therapy but remain under prospective follow-up. This design element will generate critical data on the natural history of musculoskeletal ultrasound abnormalities and the rate of progression to clinical PsA in a real-world, untreated population. These findings, expected to emerge by 2028, will be pivotal in interpreting the true impact of early intervention.

Movement as a Marker: Capturing Arthralgia Through Sensor-based Digital Health Technologies

While MSUS and transition to PsA represent important clinical endpoints for defining transition, their direct relevance to patients' lived experience is limited. Many individuals with psoriasis and arthralgia experience pain, fatigue, and impaired daily function for years before diagnosis.[70] For this group, interception should not only delay PsA onset but also relieve symptoms. Arthralgia, though a frequent marker of imminent risk, is subjective, fluctuating, and overlaps with non-inflammatory complaints. Traditional tools, including clinical history, physical examination, and even imaging, may fail to detect subtle or time-varying manifestations of subclinical disease. These limitations highlight a pressing need for objective, continuous, and ecologically valid methods of monitoring individuals at risk.

Wearable sensor technology offers a promising solution. By passively capturing real-world data on movement, gait, joint function, sleep and activity patterns, wearable devices can provide high-resolution, longitudinal insight into early musculoskeletal changes. Evidence from neurology reinforces this potential. In a recent study by Gupta et al.,[71] wrist-worn accelerometers combined with machine learning were used to track motor decline in individuals with prodromal Parkinson's disease (PD), a population similarly characterised by subtle, progressive changes prior to overt clinical diagnosis.[64] The study showed that movements became slower, smaller, and less variable over time.

This approach has clear parallels in the context of psoriatic disease. Many people with psoriasis report musculoskeletal symptoms for months or years before diagnosis, often described as fatigue, stiffness, or low-grade pain without overt inflammation. Emerging efforts in psoriatic disease are beginning to explore this potential. The Psorcast study is a prospective digital cohort leveraging smartphone-based active tasks to remotely assess upper limb and lower limb function.[72] These tasks generate continuous, quantitative data on range of motion and movement patterns, and early results suggest they can distinguish individuals with joint tenderness or enthesitis from those without.[72] In parallel, the ongoing iPROLEPSIS European consortium represents a comprehensive initiative to define, validate, and apply multimodal digital biomarkers in the health-to-PsA transition.[73] This study incorporates both active and passive sensing via smartphones and wearables to continuously monitor markers of mobility and sleep. These data streams will be integrated with clinical measures, and patient-reported outcomes to train explainable AI models for predicting disease state.[73]

These digital phenotypes could serve two major purposes. First, to enhance risk stratification, distinguishing which individuals with psoriasis and musculoskeletal symptoms are actively progressing toward PsA. Beyond augmenting patient stratification, such measures could serve

as responsive endpoints in interception trials.[74–76] As in neurology, movement is increasingly recognised as the *sixth vital sign*; a dynamic, sensitive indicator of health status that can reveal early, often imperceptible changes.[77] Translating these approaches to psoriatic disease will require careful validation and attention to disease-specific movement signatures. Digital phenotyping could transform how we define and monitor disease transition, however, important considerations around data privacy, interoperability, and the standardisation of sensor-derived measures will be crucial, so as to ensure that digital measures remain patient-centred, and to support scalability and future regulatory acceptance.[76]

When to intervene? Defining the threshold for interception

Determining the optimal timing for interception in psoriatic disease represents perhaps the most ethically and clinically complex question facing this emerging field. Whilst efforts are underway to develop reliable and accurate risk stratification tools, the translation of these tools to the clinic depends fundamentally on understanding the benefit and harm of intervention at an individual patient-level, and in relation to disease stage. Given the uncertainty around the natural progression of psoriatic disease, and the absence of randomised controlled trials testing interceptive strategies, it remains difficult to align hypothetical treatment decisions with specific phases of transition.

The missing voice: patient perspectives on acceptable risk thresholds

Despite increasing interest in the early identification and interception of PsA, the perspectives of those most affected, individuals living with psoriasis who may be at risk, remain largely unexplored. To date, no published qualitative or quantitative studies have systematically investigated how people with psoriasis perceive the idea of interceptive therapy before a formal PsA diagnosis. There is a pressing need to address this research gap given the ethical

complexities of initiating disease-modifying treatment in individuals with non-specific symptoms or imaging changes but no established arthritis.

Insights from patients with RA have shown that willingness to engage in interceptive treatment is highly sensitive to perceived risk, the burden of treatment, and an individual's symptoms during the at-risk window.[78] In PsA, these factors are compounded by the fact that most individuals at risk are already likely managing psoriasis, a chronic inflammatory condition that may itself warrant systemic therapy. The acceptability of interceptive treatments is likely to be influenced by multiple factors, including potential effects on skin disease, psoriasis severity, and individual preferences, but these dynamics need to be assessed empirically.

For individuals with severe psoriasis who already meet indications for systemic treatment, interception may be considered as a potential repurposing opportunity. In this context, the rationale to initiate treatment may also factor in disease transition risk, as well as control of skin disease. Large-scale observational studies using electronic healthcare record data indicate that dermatologists may already take joint symptoms into account when selecting systemic therapy, as PsA diagnoses commonly appear within the first year after systemic treatment initiation in psoriasis.[79] Preliminary data, awaiting publication, presents an early window into patient perspectives of preventative treatment. In a survey of 155 individuals with psoriasis across the UK and the Netherlands, 92% of respondents reported a willingness to consider preventive pharmacological therapy to lower their risk of developing PsA, with the average maximum acceptable post-treatment risk of developing PsA being 45% (IQR 30–50%).[80] Acceptability of hypothetical preventive treatment was higher in participants presented with higher baseline risk scenarios, and lower when side effects were introduced. For mild side effects, participants accepted a median risk of 30% (IQR 20–50%); for moderate side effects, 25% (IQR 10–40%). Notably, two-thirds of participants were willing to

accept a small but non-zero risk of severe side effects (1 in 10,000).[80] However, while these early findings suggest that therapy may be acceptable to many individuals with psoriasis, it is important to recognise that these preferences were elicited using hypothetical risk scenarios. At present, there is no consensus on the absolute level of PsA risk that warrants intervention, nor are there robust, individual-level risk stratification models in clinical use highlighting the pressing need for qualitative research to understand the conditions under which preventive therapy would be acceptable to patients.

Future directions

There is growing momentum for PsA interception, supported by advances in clinical research, imaging, and biomarkers. Yet progress is constrained by uncertainty over when psoriasis transitions to PsA, especially distinguishing “subclinical” from “very early” disease. This ambiguity in definition complicates risk stratification, outcome selection, and comparison across studies. Given the relatively low rates of transition, progress will require large, longitudinal cohorts with harmonised protocols to clarify natural history and regression of subclinical inflammation. Unified definitions will be essential to align research and practice, enabling meta-analyses and reproducible endpoints. Such efforts will underpin refinement of enrichment strategies (who is at risk), outcome definitions (what constitutes transition), and trial endpoints (how to capture change). Finally, defining acceptable risk thresholds, integrating patient preferences, and developing ethical guidance to support decision-making in the pre-arthritis stages will help to define when it is appropriate to intercept (Figure 3).

Conclusion

Progress toward interception in psoriatic disease depends on moving from conceptual models to coordinated, evidence-based practice. Ultimately, coordinated frameworks are needed to translate conceptual advances into a robust evidence base that reflects the heterogeneity of early psoriatic disease and supports the shared goal of therapeutic interception, enabling earlier diagnosis, more effective care, and better outcomes for patients.

Search strategy and selection criteria

To identify relevant articles for this Review, literature searches were conducted on PubMed and Google Scholar for articles published in the English language from 2000 until September 2025. Search terms included: “early psoriatic arthritis”, “subclinical psoriatic arthritis”, “disease interception”, “disease prevention” and “prevention trial”. The final reference list was compiled based on their originality and relevance to this topic.

Contributors

DMcG, conceptualisation, visualisation, writing – original draft, writing – reviewing and editing; AE, writing – original draft, writing – reviewing and editing; TG, writing – reviewing and editing; WW, writing – reviewing and editing; SRP, writing – reviewing and editing, OF, writing – reviewing and editing; LCC, conceptualisation, supervision, writing – reviewing and editing.

Declaration of interest

DMcG reports receiving a research grant from Novartis. AE reports receiving payment for lectures and presentations from Eli Lilly and Boehringer Ingelheim; support for conference attendance from Novartis and Eli Lilly; and reports participating on an AxSpa advisory group with UCB. SRP reports receiving grant funding from the Innovative Medicines Initiative 2 Joint Undertaking (JU) under grant agreement No 101007757; reports a planned serum protein biomarker signature to identify PsA in individuals with psoriasis. OF reports receiving research grant funding from Novartis, UCB, Pfizer, BMS, AbbVie, Janssen and Eli Lilly; spoke at a GRAPPA workshop sponsored by AbbVie; support for conference attendance from Janssen. LCC reports receiving research grants from AbbVie, Amgen, Celgene, Eli Lilly, Janssen, Novartis, Pfizer, and UCB; reports receiving consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Gilead, Galapagos, Janssen, Moonlake, Novartis, Pfizer, and UCB; reports receiving payment for lectures and presentations from AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GSK, Janssen, Medac, Novartis, Pfizer, and UCB; reports being a board member of the BritPACT (British Psoriatic Arthritis Consortium) charity and GRAPPA. All other authors declare no competing interests.

Figure 1. Comparison of existing staging frameworks across psoriatic disease, as proposed by EULAR,[88] PPACMAN,[99] and Ciccia et al,[13~~13~~] respectively.

Figure 2. Integrated multi-modal risk prediction across the psoriasis–to-PsA transition

Risk of progression from psoriasis to psoriatic arthritis (PsA) arises from interacting static and dynamic factors. Baseline genetic susceptibility provides a fixed background risk, while time-varying exposures such as obesity or joint injury change across the life course and can modify risk trajectories. Multi-omic integration (genomic, epigenomic, transcriptomic, proteomic, and metabolomic data) may offer mechanistic insights into evolving risk states. Phenotypic predictors, including fluctuating arthralgia and synovio-entheseal inflammation on imaging, can capture imminent disease transition but may also regress over time. Together,

these layers highlight the heterogeneous and temporally dynamic nature of PsA risk prediction

Figure 3 - Key domains and future research priorities for interception in psoriatic disease

References

FitzGerald O, Ogdie A, Chandran V, *et al.* Psoriatic arthritis. *Nat Rev Dis Primers* 2021; 7: 59.

Gladman DD, Antoni C, Mease P, Clegg DO, Nash P. Psoriatic arthritis: epidemiology, clinical features, course, and outcome. *Ann Rheum Dis* 2005; 64 Suppl 2: ii14-7.

Snoeck Henkemans SVJ, de Jong PHP, Luime JJ, *et al.* Window of opportunity in psoriatic arthritis: the earlier the better? *RMD Open* 2024; 10. DOI:10.1136/rmdopen-2023-004062.

Scher JU, Ogdie A, Merola JF, Ritchlin C. Preventing psoriatic arthritis: focusing on patients with psoriasis at increased risk of transition. *Nat Rev Rheumatol* 2019; 15: 153–66.

Karmacharya P, Wright K, Achenbach SJ, *et al.* Diagnostic Delay in Psoriatic Arthritis: A Population-based Study. *J Rheumatol* 2021; 48: 1410–6.

Haroon M, Gallagher P, FitzGerald O. Diagnostic delay of more than 6 months contributes to poor radiographic and functional outcome in psoriatic arthritis. *Ann Rheum Dis* 2015; 74. DOI:10.1136/annrheumdis-2013-204858.

7 Tillett W, Jadon D, Shaddick G, *et al.* Smoking and delay to diagnosis are associated with poorer functional outcome in psoriatic arthritis. *Ann Rheum Dis* 2013; 72: 1358–61.

8 Zabotti A, De Marco G, Gossec L, *et al.* EULAR points to consider for the definition of clinical and imaging features suspicious for progression from psoriasis to psoriatic arthritis. *Ann Rheum Dis* 2023; 82: 1162–70.

9 Perez-Chada LM, Haberman RH, Chandran V, *et al.* Consensus terminology for preclinical phases of psoriatic arthritis for use in research studies: results from a Delphi consensus study. *Nat Rev Rheumatol* 2021; 17: 238–43.

10 Deane KD, Holers VM, Emery P, *et al.* Therapeutic interception in individuals at risk of rheumatoid arthritis to prevent clinically impactful disease. *Ann Rheum Dis* 2025; 84: 14–28.

11 Honap S, Agrinier N, Torres J, *et al.* Prevention and interception trials in inflammatory bowel disease: an international taskforce assessment on clinical trial design. *Lancet Gastroenterol Hepatol* 2025; 10: 593–604.

12 Crotty GF, Ayer SJ, Schwarzschild MA. Designing the First Trials for Parkinson's Prevention. *J Parkinsons Dis* 2024; 14: S381–93.

13 Ciccia F, Gandolfo S, Caporali R, Scher JU. Understanding the spectrum from preclinical psoriatic arthritis to early diagnosis of the disease. *Lancet Rheumatol* 2025; 7: e208–11.

14 Taylor W, Gladman D, Helliwell P, *et al.* Classification criteria for psoriatic arthritis: development of new criteria from a large international study. *Arthritis Rheum* 2006; 54: 2665–73.

15 De Marco G, Zabotti A, Baraliakos X, *et al.* Characterisation of prodromal and very early psoriatic arthritis: a systematic literature review informing a EULAR taskforce. *RMD Open* 2023; 9. DOI:10.1136/rmdopen-2023-003143.

96 Mease PJ, Garg A, Helliwell PS, Park JJ, Gladman DD. Development of criteria to distinguish inflammatory from noninflammatory arthritis, enthesitis, dactylitis, and spondylitis: a report from the GRAPPA 2013 Annual Meeting. *J Rheumatol* 2014; 41: 1249–51.

97 Eder L, Haddad A, Rosen CF, *et al.* The Incidence and Risk Factors for Psoriatic Arthritis in Patients With Psoriasis: A Prospective Cohort Study. *Arthritis & Rheumatology* 2016; 68: 915–23.

98 Simon D, Tascilar K, Kleyer A, *et al.* Association of Structural Entheseal Lesions With an Increased Risk of Progression From Psoriasis to Psoriatic Arthritis. *Arthritis & Rheumatology* 2022; 74: 253–62.

99 Eder L, Polachek A, Rosen CF, Chandran V, Cook R, Gladman DD. The Development of Psoriatic Arthritis in Patients With Psoriasis Is Preceded by a Period of Nonspecific Musculoskeletal Symptoms: A Prospective Cohort Study. *Arthritis & Rheumatology* 2017; 69: 622–9.

100 Felbo SK, Terslev L, Juul Sørensen I, *et al.* Musculoskeletal pain in psoriasis-relation to inflammation and additional value of ultrasound in psoriatic arthritis classification. *Rheumatology (Oxford)* 2022; 61: 2835–47.

101 Geng Y, Song Z, Zhang X, Deng X, Wang Y, Zhang Z. Improved diagnostic performance of CASPAR criteria with integration of ultrasound. *Front Immunol* 2022; 13: 935132.

102 Schmidt RL, Factor RE. Understanding sources of bias in diagnostic accuracy studies. *Arch Pathol Lab Med* 2013; 137: 558–65.

[13] Rudge A, McHugh N, Tillett W, Smith T. An interpretable machine learning approach for detecting psoriatic arthritis in a UK primary care psoriasis cohort using electronic health records from the Clinical Practice Research Datalink. *Ann Rheum Dis* 2025; 84: 575–83.

[14] Lee LT-J, Yang H-C, Nguyen PA, Muhtar MS, Li Y-CJ. Machine Learning Approaches for Predicting Psoriatic Arthritis Risk Using Electronic Medical Records: Population-Based Study. *J Med Internet Res* 2023; 25: e39972.

[15] Liu P, Kuang Y, Ye L, *et al.* Predicting the Risk of Psoriatic Arthritis in Plaque Psoriasis Patients: Development and Assessment of a New Predictive Nomogram. *Front Immunol* 2021; 12: 740968.

[16] Zhan J, Chen F, Li Y, Huang C. Risk prediction model for psoriatic arthritis: NHANES data and multi-algorithm approach. *Clin Rheumatol* 2025; 44: 277–89.

[17] Wang C, Wang S, Liu L, *et al.* Early detection of psoriatic arthritis in patients with psoriasis: construction of a multifactorial prediction model. *Front Immunol* 2024; 15: 1426127.

[18] Xu J, Ou J, Li C, *et al.* Multi-modality data-driven analysis of diagnosis and treatment of psoriatic arthritis. *NPJ Digit Med* 2023; 6: 13.

[19] Eder L, Lee K-A, Chandran V, *et al.* Derivation of a Multivariable Psoriatic Arthritis Risk Estimation Tool (PRESTO): A Step Towards Prevention. *Arthritis Rheumatol* 2023; published online Aug 9. DOI:10.1002/art.42661.

[20] Zabotti A, De Lucia O, Sakellariou G, *et al.* Predictors, Risk Factors, and Incidence Rates of Psoriatic Arthritis Development in Psoriasis Patients: A Systematic Literature Review and Meta-Analysis. *Rheumatol Ther* 2021; 8: 1519–34.

Piaserico S, Megna M, Bardazzi F, *et al.* TNF-alpha inhibitors reduce the incidence of psoriatic arthritis in patients with psoriasis: a propensity score-matched cohort study. *Rheumatology (Oxford)* 2025; published online July 3. DOI:10.1093/rheumatology/keaf364.

Damen JAAG, Hooft L, Schuit E, *et al.* Prediction models for cardiovascular disease risk in the general population: systematic review. *BMJ* 2016; 353: i2416.

Wynants L, Van Calster B, Collins GS, *et al.* Prediction models for diagnosis and prognosis of covid-19: systematic review and critical appraisal. *BMJ* 2020; 369: m1328.

Agniel D, Kohane IS, Weber GM. Biases in electronic health record data due to processes within the healthcare system: retrospective observational study. *BMJ* 2018; 361: k1479.

Haut ER, Pronovost PJ. Surveillance bias in outcomes reporting. *JAMA* 2011; 305: 2462–3.

Yland JJ, Wesselink AK, Lash TL, Fox MP. Misconceptions About the Direction of Bias From Nondifferential Misclassification. *Am J Epidemiol* 2022; 191: 1485–95.

Funk MJ, Landi SN. Misclassification in administrative claims data: quantifying the impact on treatment effect estimates. *Curr Epidemiol Rep* 2014; 1: 175–85.

Dubash SR, De Marco G, Wakefield RJ, Tan AL, McGonagle D, Marzo-Ortega H. Ultrasound Imaging in Psoriatic Arthritis: What Have We Learnt in the Last Five Years? *Front Med (Lausanne)* 2020; 7: 487.

Elnady B, El Shaarawy NK, Dawoud NM, *et al.* Subclinical synovitis and enthesitis in psoriasis patients and controls by ultrasonography in Saudi Arabia; incidence of psoriatic arthritis during two years. *Clin Rheumatol* 2019; 38: 1627–35.

Azuaga AB, Cuervo A, Reina D, *et al.* Clinical and ultrasound features of a cohort of psoriasis patients without musculoskeletal symptoms: a prospective and multicenter study. *Rheumatology (Oxford)* 2025; published online June 4. DOI:10.1093/rheumatology/keaf307.

43 Zabotti A, Giovannini I, McGonagle D, *et al.* POS0299 RISK ANALYSIS OF CLINICAL PSORIATIC ARTHRITIS DEVELOPMENT IN PATIENTS WITH PSORIASIS AND RECENT-ONSET ARTHRALGIA. *Ann Rheum Dis* 2025; 84: 560–1.

44 Scheel AK, Schmidt WA, Hermann K-GA, *et al.* Interobserver reliability of rheumatologists performing musculoskeletal ultrasonography: results from a EULAR 'Train the trainers' course. *Ann Rheum Dis* 2005; 64: 1043–9.

45 Brulhart L, Ziswiler H-R, Tamborrini G, Zufferey P, SONAR/SCQM programmes. The importance of sonographer experience and machine quality with regards to the role of musculoskeletal ultrasound in routine care of rheumatoid arthritis patients. *Clin Exp Rheumatol* 2015; 33: 98–101.

46 Coates LC, Hodgson R, Conaghan PG, Freeston JE. MRI and ultrasonography for diagnosis and monitoring of psoriatic arthritis. *Best Pract Res Clin Rheumatol* 2012; 26: 805–22.

47 Faustini F, Simon D, Oliveira I, *et al.* Subclinical joint inflammation in patients with psoriasis without concomitant psoriatic arthritis: a cross-sectional and longitudinal analysis. *Ann Rheum Dis* 2016; 75: 2068–74.

48 Corte G, Atzinger A, Temiz SA, *et al.* Anatomical pattern of entheseal and synovial fibroblast activation in patients with psoriasis and its risk of developing psoriatic arthritis. *RMD Open* 2024; 10. DOI:10.1136/rmdopen-2024-004294.

49 Schmidkonz C, Kuwert T, Götz TI, Ramming A, Atzinger A. Recent advances in nuclear medicine and their role in inflammatory arthritis: focus on the emerging role of FAPI PET/CT. *Skeletal Radiol* 2025; 54: 2243–52.

88 Ribeiro AL, Eder L. From Psoriasis to Psoriatic Arthritis: Ultrasound Insights Connecting Psoriasis with Subclinical Musculoskeletal Inflammation and the Path to Psoriatic Arthritis. *Curr Rheumatol Rep* 2024; 26: 235–47.

89 van Steenbergen HW, Doornkamp F, Alivernini S, *et al.* EULAR/American College of Rheumatology Risk Stratification Criteria for Development of Rheumatoid Arthritis in the Risk Stage of Arthralgia. *Arthritis Rheumatol* 2025; published online May 8. DOI:10.1002/art.43218.

90 Eder L, Kaeley GS, Aydin SZ. Development and Validation of a Sonographic Enthesitis Instrument in Psoriatic Arthritis: The GRAPPA Diagnostic Ultrasound Enthesitis Tool (DUET) Project. *J Rheumatol Suppl* 2020; 96: 50–2.

91 Zabotti A, Fagni F, Gossec L, *et al.* Risk of developing psoriatic arthritis in psoriasis cohorts with arthralgia: exploring the subclinical psoriatic arthritis stage. *RMD Open* 2024; 10. DOI:10.1136/rmdopen-2024-004314.

92 Eder L, Chandran V, Shen H, *et al.* Incidence of arthritis in a prospective cohort of psoriasis patients. *Arthritis Care Res (Hoboken)* 2011; 63: 619–22.

93 Perez-Chada LM, Gondo G, Grant C, *et al.* Construct Validity of the IDEOM Musculoskeletal Questionnaire: An Instrument to Measure Musculoskeletal Symptoms in Patients with Psoriatic Disease. *J Invest Dermatol* 2025; 145: 1798-1801.e4.

94 Gossec L, de Wit M, Kiltz U, *et al.* A patient-derived and patient-reported outcome measure for assessing psoriatic arthritis: elaboration and preliminary validation of the Psoriatic Arthritis Impact of Disease (PsAID) questionnaire, a 13-country EULAR initiative. *Ann Rheum Dis* 2014; 73: 1012–9.

[50] Stadler M, Zhao SS, Bowes J. A review of the advances in understanding the genetic basis of spondylarthritis and emerging clinical benefit. *Best Pract Res Clin Rheumatol* 2024; 38: 101982.

[51] Mulder MLM, van Hal TW, Wenink MH, *et al.* Clinical, laboratory, and genetic markers for the development or presence of psoriatic arthritis in psoriasis patients: a systematic review. *Arthritis Res Ther* 2021; 23: 168.

[52] Haroon M, Winchester R, Giles JT, Heffernan E, FitzGerald O. Certain class I HLA alleles and haplotypes implicated in susceptibility play a role in determining specific features of the psoriatic arthritis phenotype. *Ann Rheum Dis* 2016; 75: 155–62.

[53] Haroon M, Winchester R, Giles JT, Heffernan E, FitzGerald O. Clinical and genetic associations of radiographic sacroiliitis and its different patterns in psoriatic arthritis. *Clin Exp Rheumatol* 2017; 35: 270–6.

[54] Soomro M, Stadler M, Dand N, *et al.* Comparative Genetic Analysis of Psoriatic Arthritis and Psoriasis for the Discovery of Genetic Risk Factors and Risk Prediction Modeling. *Arthritis Rheumatol* 2022; 74: 1535–43.

[55] Samani NJ, Beeston E, Greengrass C, *et al.* Polygenic risk score adds to a clinical risk score in the prediction of cardiovascular disease in a clinical setting. *Eur Heart J* 2024; 45: 3152–60.

[56] Fuat A, Adlen E, Monane M, *et al.* A polygenic risk score added to a QRISK®2 cardiovascular disease risk calculator demonstrated robust clinical acceptance and clinical utility in the primary care setting. *Eur J Prev Cardiol* 2024; 31: 716–22.

[57] Grohmann T, Vivekanantham A, Coates LC, Pennington S, FitzGerald O. Clinical, genetic and omics-based biomarkers that might support the identification of the development of

psoriatic arthritis in individuals with psoriasis: a narrative review of the literature. *RMD Open* 2024; 10. DOI:10.1136/rmdopen-2024-004176.

FitzGerald O, Behrens F, Barton A, *et al.* Application of clinical and molecular profiling data to improve patient outcomes in psoriatic arthritis. *Ther Adv Musculoskelet Dis* 2023; 15. DOI:10.1177/1759720X231192315.

FitzGerald O, Pennington SR. HIPPOCRATES: improving diagnosis and outcomes in psoriatic arthritis. *Nat Rev Rheumatol* 2022; 18: 123–4.

Jadon DR, Stober C, Pennington SR, FitzGerald O. Applying precision medicine to unmet clinical needs in psoriatic disease. *Nat Rev Rheumatol* 2020; 16: 609–27.

Savage L, Goodfield M, Horton L, *et al.* Regression of Peripheral Subclinical Enthesopathy in Therapy-Naive Patients Treated With Ustekinumab for Moderate-to-Severe Chronic Plaque Psoriasis: A Fifty-Two-Week, Prospective, Open-Label Feasibility Study. *Arthritis Rheumatol* 2019; 71: 626–31.

Liu Z, Chen J, Zhou K, *et al.* Effectiveness of Biologics on synovitis and enthesitis using Musculoskeletal ultrasound assessment in subclinical psoriatic arthritis: A 12-week observational real-world study. *J Am Acad Dermatol* 2025; published online Sept 8. DOI:10.1016/j.jaad.2025.09.003.

Haberman RH, MacFarlane KA, Catron S, *et al.* Efficacy of guselkumab, a selective IL-23 inhibitor, in Preventing Arthritis in a Multicentre Psoriasis At-Risk cohort (PAMPA): protocol of a randomised, double-blind, placebo controlled multicentre trial. *BMJ Open* 2022; 12: e063650.

Scher J. Multi-Center PAMPA Study (PAMPA). https://www.clinicaltrials.gov/study/NCT05004727. 2022; published online Feb 16.

[48] Ogdie A, Nowell WB, Applegate E, *et al.* Patient perspectives on the pathway to psoriatic arthritis diagnosis: results from a web-based survey of patients in the United States. *BMC Rheumatol* 2020; 4: 2.

[51] Gupta AS, Patel S. Wrist accelerometry and machine learning sensitively capture disease progression in prodromal Parkinson's disease. *NPJ Parkinsons Dis* 2025; 11: 171.

[52] Webster DE, Haberman RH, Chada LMP, *et al.* Clinical validation of digital assessment tools and machine learning models for remote measurement of psoriasis and psoriatic arthritis: a proof-of-concept study. *J Rheumatol* 2024; published online June 15. DOI:10.3899/jrheum.2024-0074.

[53] Hadjileontiadis LJ, Charisis V, Hadjidimitriou S, *et al.* European advances in digital rheumatology: explainable insights and personalized digital health tools for psoriatic arthritis. *EClinicalMedicine* 2025; 84: 103243.

[60] Gonzalez-Robles C, Weil RS, van Wamelen D, *et al.* Outcome Measures for Disease-Modifying Trials in Parkinson's Disease: Consensus Paper by the EJS ACT-PD Multi-Arm Multi-Stage Trial Initiative. *J Parkinsons Dis* 2023; 13: 1011–33.

[63] O'Hanlon CE, Farmer CM, Ryan J, Ernecoff N. Clinical Outcome Assessments and Digital Health Technologies Supporting Clinical Trial Endpoints in Early Parkinson's Disease: Roundtable Proceedings and Roadmap for Research. *Rand Health Q* 2024; 11: 1.

[66] McGagh D, Song K, Yuan H, *et al.* Digital health technologies to strengthen patient-centred outcome assessment in clinical trials in inflammatory arthritis. *Lancet Rheumatol* 2025; 7: e55–63.

[69] Middleton A, Fritz SL, Lusardi M. Walking speed: the functional vital sign. *J Aging Phys Act* 2015; 23: 314–22.

van Steenbergen HW, Cope AP, van der Helm-van Mil AHM. Rheumatoid arthritis prevention in arthralgia: fantasy or reality? *Nat Rev Rheumatol* 2023; 19: 767–77.

Meer E, Merola JF, Fitzsimmons R, *et al.* Does biologic therapy impact the development of PsA among patients with psoriasis? *Ann Rheum Dis* 2022; 81: 80–6.

Groothuizen S, Bolt JW, Veldwijk J, *et al.* AB1643-PARE ASSESSMENT OF PSORIASIS PATIENTS' PREFERENCES FOR INTERVENTIONS TO PREVENT PSORIATIC ARTHRITIS USING A PROBABILISTIC THRESHOLD TECHNIQUE. *Ann Rheum Dis* 2024; 83: 2196–7.

FitzGerald O, Ogdie A, Chandran V, *et al.* Psoriatic arthritis. *Nat Rev Dis Primers* 2021; 7: 59.

Gladman DD, Antoni C, Mease P, Clegg DO, Nash P. Psoriatic arthritis: epidemiology, clinical features, course, and outcome. *Ann Rheum Dis* 2005; 64 Suppl 2: ii14-7.

Snoeck Henkemans SVJ, de Jong PHP, Luime JJ, *et al.* Window of opportunity in psoriatic arthritis: the earlier the better? *RMD Open* 2024; 10. DOI:10.1136/rmdopen-2023-004062.

Scher JU, Ogdie A, Merola JF, Ritchlin C. Preventing psoriatic arthritis: focusing on patients with psoriasis at increased risk of transition. *Nat Rev Rheumatol* 2019; 15: 153–66.

Karmacharya P, Wright K, Achenbach SJ, *et al.* Diagnostic Delay in Psoriatic Arthritis: A Population-based Study. *J Rheumatol* 2021; 48: 1410–6.

Haroon M, Gallagher P, FitzGerald O. Diagnostic delay of more than 6 months contributes to poor radiographic and functional outcome in psoriatic arthritis. *Ann Rheum Dis* 2015; 74. DOI:10.1136/annrheumdis-2013-204858.

Tillett W, Jadon D, Shaddick G, *et al.* Smoking and delay to diagnosis are associated with poorer functional outcome in psoriatic arthritis. *Ann Rheum Dis* 2013; 72: 1358–61.

~~Zabotti A, De Marco G, Gossec L, *et al.* EULAR points to consider for the definition of clinical and imaging features suspicious for progression from psoriasis to psoriatic arthritis. *Ann Rheum Dis* 2023; 82: 1162–70.~~

~~Perez-Chada LM, Haberman RH, Chandran V, *et al.* Consensus terminology for preclinical phases of psoriatic arthritis for use in research studies: results from a Delphi consensus study. *Nat Rev Rheumatol* 2021; 17: 238–43.~~

~~Deane KD, Holers VM, Emery P, *et al.* Therapeutic interception in individuals at risk of rheumatoid arthritis to prevent clinically impactful disease. *Ann Rheum Dis* 2025; 84: 14–28.~~

~~Honap S, Agrinier N, Torres J, *et al.* Prevention and interception trials in inflammatory bowel disease: an international taskforce assessment on clinical trial design. *Lancet Gastroenterol Hepatol* 2025; 10: 593–604.~~

~~Crotty GF, Ayer SJ, Schwarzschild MA. Designing the First Trials for Parkinson's Prevention. *J Parkinsons Dis* 2024; 14: S381–93.~~

~~Ciccia F, Gandolfo S, Caporali R, Scher JU. Understanding the spectrum from preclinical psoriatic arthritis to early diagnosis of the disease. *Lancet Rheumatol* 2025; 7: e208–11.~~

~~Taylor W, Gladman D, Helliwell P, *et al.* Classification criteria for psoriatic arthritis: development of new criteria from a large international study. *Arthritis Rheum* 2006; 54: 2665–73.~~

~~De Marco G, Zabotti A, Baraliakos X, *et al.* Characterisation of prodromal and very early psoriatic arthritis: a systematic literature review informing a EULAR taskforce. *RMD Open* 2023; 9. DOI:10.1136/rmdopen-2023-003143.~~

~~Mease PJ, Garg A, Helliwell PS, Park JJ, Gladman DD. Development of criteria to distinguish inflammatory from noninflammatory arthritis, enthesitis, dactylitis, and~~

spondylitis: a report from the GRAPPA 2013 Annual Meeting. *J Rheumatol* 2014; 41: 1249–51.

17 Eder L, Haddad A, Rosen CF, *et al*. The Incidence and Risk Factors for Psoriatic Arthritis in Patients With Psoriasis: A Prospective Cohort Study. *Arthritis & Rheumatology* 2016; 68: 915–23.

22 Simon D, Tascilar K, Kleyer A, *et al*. Association of Structural Entheseal Lesions With an Increased Risk of Progression From Psoriasis to Psoriatic Arthritis. *Arthritis & Rheumatology* 2022; 74: 253–62.

25 Eder L, Polachek A, Rosen CF, Chandran V, Cook R, Gladman DD. The Development of Psoriatic Arthritis in Patients With Psoriasis Is Preceded by a Period of Nonspecific Musculoskeletal Symptoms: A Prospective Cohort Study. *Arthritis & Rheumatology* 2017; 69: 622–9.

29 Felbo SK, Terslev L, Juul Sørensen I, *et al*. Musculoskeletal pain in psoriasis-relation to inflammation and additional value of ultrasound in psoriatic arthritis classification. *Rheumatology (Oxford)* 2022; 61: 2835–47.

32 Geng Y, Song Z, Zhang X, Deng X, Wang Y, Zhang Z. Improved diagnostic performance of CASPAR criteria with integration of ultrasound. *Front Immunol* 2022; 13: 935132.

34 Schmidt RL, Factor RE. Understanding sources of bias in diagnostic accuracy studies. *Arch Pathol Lab Med* 2013; 137: 558–65.

36 Rudge A, McHugh N, Tillett W, Smith T. An interpretable machine learning approach for detecting psoriatic arthritis in a UK primary care psoriasis cohort using electronic health records from the Clinical Practice Research Datalink. *Ann Rheum Dis* 2025; 84: 575–83.

[39] Lee LT-J, Yang H-C, Nguyen PA, Muhtar MS, Li Y-CJ. Machine Learning Approaches for Predicting Psoriatic Arthritis Risk Using Electronic Medical Records: Population-Based Study. *J Med Internet Res* 2023; 25: e39972.

[42] Liu P, Kuang Y, Ye L, *et al.* Predicting the Risk of Psoriatic Arthritis in Plaque Psoriasis Patients: Development and Assessment of a New Predictive Nomogram. *Front Immunol* 2021; 12: 740968.

[45] Zhan J, Chen F, Li Y, Huang C. Risk prediction model for psoriatic arthritis: NHANES data and multi-algorithm approach. *Clin Rheumatol* 2025; 44: 277–89.

[47] Wang C, Wang S, Liu L, *et al.* Early detection of psoriatic arthritis in patients with psoriasis: construction of a multifactorial prediction model. *Front Immunol* 2024; 15: 1426127.

[49] Xu J, Ou J, Li C, *et al.* Multi-modality data-driven analysis of diagnosis and treatment of psoriatic arthritis. *NPJ Digit Med* 2023; 6: 13.

[51] Eder L, Lee K-A, Chandran V, *et al.* Derivation of a Multivariable Psoriatic Arthritis Risk Estimation Tool (PRESTO): A Step Towards Prevention. *Arthritis Rheumatol* 2023; published online Aug 9. DOI:10.1002/art.42661.

[54] Zabotti A, De Lucia O, Sakellariou G, *et al.* Predictors, Risk Factors, and Incidence Rates of Psoriatic Arthritis Development in Psoriasis Patients: A Systematic Literature Review and Meta-Analysis. *Rheumatol Ther* 2021; 8: 1519–34.

[57] Piaserico S, Megna M, Bardazzi F, *et al.* TNF-alpha inhibitors reduce the incidence of psoriatic arthritis in patients with psoriasis: a propensity score-matched cohort study. *Rheumatology (Oxford)* 2025; published online July 3. DOI:10.1093/rheumatology/keaf364.

[60] Damen JAAG, Hooft L, Schuit E, *et al.* Prediction models for cardiovascular disease risk in the general population: systematic review. *BMJ* 2016; 353: i2416.

Wynants L, Van Calster B, Collins GS, *et al.* Prediction models for diagnosis and prognosis of covid-19: systematic review and critical appraisal. *BMJ* 2020; 369: m1328.

Agniel D, Kohane IS, Weber GM. Biases in electronic health record data due to processes within the healthcare system: retrospective observational study. *BMJ* 2018; 361: k1479.

Haut ER, Pronovost PJ. Surveillance bias in outcomes reporting. *JAMA* 2011; 305: 2462–3.

Yland JJ, Wesselink AK, Lash TL, Fox MP. Misconceptions About the Direction of Bias From Nondifferential Misclassification. *Am J Epidemiol* 2022; 191: 1485–95.

Funk MJ, Landi SN. Misclassification in administrative claims data: quantifying the impact on treatment effect estimates. *Curr Epidemiol Rep* 2014; 1: 175–85.

Dubash SR, De Marco G, Wakefield RJ, Tan AL, McGonagle D, Marzo-Ortega H. Ultrasound Imaging in Psoriatic Arthritis: What Have We Learnt in the Last Five Years? *Front Med (Lausanne)* 2020; 7: 487.

Elnady B, El Shaarawy NK, Dawoud NM, *et al.* Subclinical synovitis and enthesitis in psoriasis patients and controls by ultrasonography in Saudi Arabia; incidence of psoriatic arthritis during two years. *Clin Rheumatol* 2019; 38: 1627–35.

Azuaga AB, Cuervo A, Reina D, *et al.* Clinical and ultrasound features of a cohort of psoriasis patients without musculoskeletal symptoms: a prospective and multicenter study. *Rheumatology (Oxford)* 2025; published online June 4. DOI:10.1093/rheumatology/keaf307.

Zabotti A, Giovannini I, McGonagle D, *et al.* POS0299 RISK ANALYSIS OF CLINICAL PSORIATIC ARTHRITIS DEVELOPMENT IN PATIENTS WITH PSORIASIS AND RECENT-ONSET ARTHRALGIA. *Ann Rheum Dis* 2025; 84: 560–1.

~~Scheel AK, Schmidt WA, Hermann K-GA, *et al*. Interobserver reliability of rheumatologists performing musculoskeletal ultrasonography: results from a EULAR 'Train the trainers' course. *Ann Rheum Dis* 2005; 64: 1043–9.~~

~~Brulhart L, Ziswiler H-R, Tamborrini G, Zufferey P, SONAR/SCQM programmes. The importance of sonographer experience and machine quality with regards to the role of musculoskeletal ultrasound in routine care of rheumatoid arthritis patients. *Clin Exp Rheumatol* 2015; 33: 98–101.~~

~~Coates LC, Hodgson R, Conaghan PG, Freeston JE. MRI and ultrasonography for diagnosis and monitoring of psoriatic arthritis. *Best Pract Res Clin Rheumatol* 2012; 26: 805–22.~~

~~Faustini F, Simon D, Oliveira I, *et al*. Subclinical joint inflammation in patients with psoriasis without concomitant psoriatic arthritis: a cross-sectional and longitudinal analysis. *Ann Rheum Dis* 2016; 75: 2068–74.~~

~~Corte G, Atzinger A, Temiz SA, *et al*. Anatomical pattern of entheseal and synovial fibroblast activation in patients with psoriasis and its risk of developing psoriatic arthritis. *RMD Open* 2024; 10. DOI:10.1136/rmdopen-2024-004294.~~

~~Schmidkonz C, Kuwert T, Götz TI, Ramming A, Atzinger A. Recent advances in nuclear medicine and their role in inflammatory arthritis: focus on the emerging role of FAPI PET/CT. *Skeletal Radiol* 2025; 54: 2243–52.~~

~~Ribeiro AL, Eder L. From Psoriasis to Psoriatic Arthritis: Ultrasound Insights Connecting Psoriasis with Subclinical Musculoskeletal Inflammation and the Path to Psoriatic Arthritis. *Curr Rheumatol Rep* 2024; 26: 235–47.~~

~~van Steenbergen HW, Doornkamp F, Alivernini S, *et al*. EULAR/American College of Rheumatology Risk Stratification Criteria for Development of Rheumatoid Arthritis in the~~

Risk Stage of Arthralgia. *Arthritis Rheumatol* 2025; published online May 8. DOI:10.1002/art.43218.

Eder L, Kaeley GS, Aydin SZ. Development and Validation of a Sonographic Enthesitis Instrument in Psoriatic Arthritis: The GRAPPA Diagnostic Ultrasound Enthesitis Tool (DUET) Project. *J Rheumatol Suppl* 2020; 96: 50–2.

Zabotti A, Fagni F, Gossec L, *et al.* Risk of developing psoriatic arthritis in psoriasis cohorts with arthralgia: exploring the subclinical psoriatic arthritis stage. *RMD Open* 2024; 10. DOI:10.1136/rmdopen-2024-004314.

Eder L, Chandran V, Shen H, *et al.* Incidence of arthritis in a prospective cohort of psoriasis patients. *Arthritis Care Res (Hoboken)* 2011; 63: 619–22.

Perez-Chada LM, Gondo G, Grant C, *et al.* Construct Validity of the IDEOM Musculoskeletal Questionnaire: An Instrument to Measure Musculoskeletal Symptoms in Patients with Psoriatic Disease. *J Invest Dermatol* 2025; 145: 1798-1801.e4.

Gossec L, de Wit M, Kiltz U, *et al.* A patient-derived and patient-reported outcome measure for assessing psoriatic arthritis: elaboration and preliminary validation of the Psoriatic Arthritis Impact of Disease (PsAID) questionnaire, a 13-country EULAR initiative. *Ann Rheum Dis* 2014; 73: 1012–9.

Stadler M, Zhao SS, Bowes J. A review of the advances in understanding the genetic basis of spondylarthritis and emerging clinical benefit. *Best Pract Res Clin Rheumatol* 2024; 38: 101982.

Mulder MLM, van Hal TW, Wenink MH, *et al.* Clinical, laboratory, and genetic markers for the development or presence of psoriatic arthritis in psoriasis patients: a systematic review. *Arthritis Res Ther* 2021; 23: 168.

Haroon M, Winchester R, Giles JT, Heffernan E, FitzGerald O. Certain class I HLA alleles and haplotypes implicated in susceptibility play a role in determining specific features of the psoriatic arthritis phenotype. *Ann Rheum Dis* 2016; 75: 155–62.

Haroon M, Winchester R, Giles JT, Heffernan E, FitzGerald O. Clinical and genetic associations of radiographic sacroiliitis and its different patterns in psoriatic arthritis. *Clin Exp Rheumatol* 2017; 35: 270–6.

Soomro M, Stadler M, Dand N, *et al.* Comparative Genetic Analysis of Psoriatic Arthritis and Psoriasis for the Discovery of Genetic Risk Factors and Risk Prediction Modeling. *Arthritis Rheumatol* 2022; 74: 1535–43.

Samani NJ, Beeston E, Greengrass C, *et al.* Polygenic risk score adds to a clinical risk score in the prediction of cardiovascular disease in a clinical setting. *Eur Heart J* 2024; 45: 3152–60.

Fuat A, Adlen E, Monane M, *et al.* A polygenic risk score added to a QRISK®2 cardiovascular disease risk calculator demonstrated robust clinical acceptance and clinical utility in the primary care setting. *Eur J Prev Cardiol* 2024; 31: 716–22.

Grohmann T, Vivekanantham A, Coates LC, Pennington S, FitzGerald O. Clinical, genetic and omics-based biomarkers that might support the identification of the development of psoriatic arthritis in individuals with psoriasis: a narrative review of the literature. *RMD Open* 2024; 10. DOI:10.1136/rmdopen-2024-004176.

FitzGerald O, Behrens F, Barton A, *et al.* Application of clinical and molecular profiling data to improve patient outcomes in psoriatic arthritis. *Ther Adv Musculoskelet Dis* 2023; 15. DOI:10.1177/1759720X231192315.

54. FitzGerald O, Pennington SR. HIPPOCRATES: improving diagnosis and outcomes in psoriatic arthritis. *Nat Rev Rheumatol* 2022; 18: 123–4.

55. Jadon DR, Stober C, Pennington SR, FitzGerald O. Applying precision medicine to unmet clinical needs in psoriatic disease. *Nat Rev Rheumatol* 2020; 16: 609–27.

56. Savage L, Goodfield M, Horton L, *et al.* Regression of Peripheral Subclinical Enthesopathy in Therapy-Naive Patients Treated With Ustekinumab for Moderate-to-Severe Chronic Plaque Psoriasis: A Fifty-Two-Week, Prospective, Open-Label Feasibility Study. *Arthritis Rheumatol* 2019; 71: 626–31.

57. Liu Z, Chen J, Zhou K, *et al.* Effectiveness of Biologics on synovitis and enthesitis using Musculoskeletal ultrasound assessment in subclinical psoriatic arthritis: A 12-week observational real-world study. *J Am Acad Dermatol* 2025; published online Sept 8. DOI:10.1016/j.jaad.2025.09.003.

58. Haberman RH, MacFarlane KA, Catron S, *et al.* Efficacy of guselkumab, a selective IL-23 inhibitor, in Preventing Arthritis in a Multicentre Psoriasis At-Risk cohort (PAMPA): protocol of a randomised, double-blind, placebo controlled multicentre trial. *BMJ Open* 2022; 12: e063650.

59. Scher J. Multi-Center PAMPA Study (PAMPA). https://www.clinicaltrials.gov/study/NCT05004727. 2022; published online Feb 16.

60. Ogdie A, Nowell WB, Applegate E, *et al.* Patient perspectives on the pathway to psoriatic arthritis diagnosis: results from a web-based survey of patients in the United States. *BMC Rheumatol* 2020; 4: 2.

61. Gupta AS, Patel S. Wrist accelerometry and machine learning sensitively capture disease progression in prodromal Parkinson's disease. *NPJ Parkinsons Dis* 2025; 11: 171.

~~Webster DE, Haberman RH, Chada LMP, *et al.* Clinical validation of digital assessment tools and machine learning models for remote measurement of psoriasis and psoriatic arthritis: a proof-of-concept study. *J Rheumatol* 2024; published online June 15. DOI:10.3899/jrheum.2024-0074.~~

~~Hadjileontiadis LJ, Charisis V, Hadjidimitriou S, *et al.* European advances in digital rheumatology: explainable insights and personalized digital health tools for psoriatic arthritis. *EClinicalMedicine* 2025; 84: 103243.~~

~~Gonzalez-Robles C, Weil RS, van Wamelen D, *et al.* Outcome Measures for Disease-Modifying Trials in Parkinson's Disease: Consensus Paper by the EJS ACT-PD Multi-Arm Multi-Stage Trial Initiative. *J Parkinsons Dis* 2023; 13: 1011–33.~~

~~O'Hanlon CE, Farmer CM, Ryan J, Ernecoff N. Clinical Outcome Assessments and Digital Health Technologies Supporting Clinical Trial Endpoints in Early Parkinson's Disease: Roundtable Proceedings and Roadmap for Research. *Rand Health Q* 2024; 11: 1.~~

~~McGagh D, Song K, Yuan H, *et al.* Digital health technologies to strengthen patient-centred outcome assessment in clinical trials in inflammatory arthritis. *Lancet Rheumatol* 2025; 7: e55–63.~~

~~Middleton A, Fritz SL, Lusardi M. Walking speed: the functional vital sign. *J Aging Phys Act* 2015; 23: 314–22.~~

~~van Steenbergen HW, Cope AP, van der Helm-van Mil AHM. Rheumatoid arthritis prevention in arthralgia: fantasy or reality? *Nat Rev Rheumatol* 2023; 19: 767–77.~~

~~Meer E, Merola JF, Fitzsimmons R, *et al.* Does biologic therapy impact the development of PsA among patients with psoriasis? *Ann Rheum Dis* 2022; 81: 80–6.~~

Groothuizen S, Bolt JW, Veldwijk J, *et al*. AB1643-PARE ASSESSMENT OF PSORIASIS PATIENTS' PREFERENCES FOR INTERVENTIONS TO PREVENT PSORIATIC ARTHRITIS USING A PROBABILISTIC THRESHOLD TECHNIQUE. *Ann Rheum Dis* 2024; 83: 2196–7.

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