Fifteenth International Congress on Spondyloarthritides

3–5 September 2026
Ghent, Belgium

President

Atul Deodhar

Oregon Health & Science University
Division of Arthritis and Rheumatic Diseases
Portland, USA

Local Organising Committee

Dirk Elewaut
Filip Van den Bosch

Ghent University
Department of Rheumatology
Ghent, Belgium

Abstracts

	Page
Invited Lectures (INV1 – INV20)	1866-1871
Oral Presentations (O1 – O13)	1872-1879
Poster Presentations (P1 – P176)	1880-1996
Author Index	1997-2003

Invited lectures

INV1

COMBINING HUMAN AND ARTIFICIAL INTELLIGENCE FOR NEXT-GENERATION PATHOLOGY IN RHEUMATOLOGY

Saeys Y.[1,2,3]
[1]Dept. of Mathematics, Computer Science and Statistics, Ghent University, Ghent; [2]VIB Center for Inflammation Research, VIB, Ghent; [3]VIB Center for Computational Biology and AI, VIB, Ghent Belgium

High dimensional single cell and spatial technologies now generate data at a scale and complexity that far exceeds the limits of traditional manual interpretation. While classical analytical approaches remain valuable, recent advances in artificial intelligence (AI) provide powerful new avenues for experts to interrogate their data, uncover latent structure, and extract quantitative patterns that may lead to novel biological insights, and ultimately new treatments and drugs.
In this talk I will outline the potential of combining AI with the latest advances in single cell and spatial omics. These technologies have the potential of transforming our understanding of spondyloarthritis (SpA) by revealing not only which cells and molecular pathways are active, but also where they are located within inflamed tissues such as synovium, entheses, bone marrow, and gut. By combining gene expression, protein, and cellular profiling with tissue architecture, spatial omics can map the interactions between immune cells, stromal cells, and bone-forming cells that drive inflammation and pathological bone remodeling.
This approach may help identify previously unrecognized disease mechanisms, define tissue-specific cellular niches, and uncover biomarkers that predict disease progression or treatment response. Ultimately, spatial omics could support more precise patient stratification and accelerate the development of targeted therapies for different SpA disease subtypes and stages.
Looking further ahead, I will showcase how AI assisted exploration and interpretation tools will increasingly function as collaborative scientific partners, transforming the way we will perform science in the future. I will give an example of how next-generation interactive interfaces can be used to navigate the complexity of modern single cell and spatial datasets—ultimately accelerating discovery and deepening biological insight.

INV2

MEASUREMENT OF PATHOGENIC CD8 T CELLS IDENTIFIES HLA-B*27-ASSOCIATED AUTOIMMUNITY

Risch I., Wu Q., Paley M.A.
Dept. of Medicine, Washington University in St. Louis, St. Louis, MO, USA

Introduction. Early recognition of patients with spondyloarthritis is a major unmet need in rheumatology. Prior work in HLA-B*27$^+$ axial spondyloarthritis (axSpA) patients has reported a role for pathogenic CD8 T cells that react to a bacterial peptide antigen from the gastrointestinal tract and cross-react with autoantigens via molecular mimicry. These pathogenic CD8 T cells are defined by their T cell receptor (TCR) sequence, a pairing of TRAV21 with a specific CDR3b motif. We hypothesized that detection of pathogenic TCRs in the blood could serve as a novel approach to distinguish individuals with axSpA from healthy controls.
Methods. We first employed a mutagenesis approach to define the elements of the TCR beta chain that support antigen cross-recognition. We then leveraged a novel high-throughput TCR sequencing platform that allows for computational pairing of bulk TCR alpha and TCR beta sequencing.
Results. Analysis of bulk TCR beta sequencing generated high sensitivity but poor specificity in detection of pathogenic TCRs. By contrast, paired alpha/beta TCR analysis was able to separate axSpA participants from healthy controls with high sensitivity and specificity.
Conclusion. These data provide proof-of-concept that measurement of pathogenic CD8 T cells in the blood has utility in supporting the diagnosis of axial spondyloarthritis. Moreover, paired alpha/beta TCR analysis was critical for high specificity.

INV3

INTERFACE INFLAMMATION, JOINTS AND SPINE

Kuhn K.A.
Division of Rheumatology, Dept. of Medicine, University of Colorado Anschutz, Aurora, Colorado, USA

Introduction. The gut-joint axis is a central paradigm in spondyloarthritis (SpA), supported by the high prevalence of inflammatory bowel disease and subclinical intestinal inflammation in patients with axial disease. Increasing evidence suggests that epithelial barrier tissues, including the intestine and skin, serve as important immunologic interfaces that influence musculoskeletal inflammation. However, whether barrier inflammation directly programs pathogenic immune cells or instead provides a pool of migratory immune cells whose function is determined after tissue egress remains unresolved.
Methods. This invited lecture will review current evidence supporting epithelial interface inflammation as an upstream contributor to SpA pathogenesis, integrating findings from clinical studies, experimental models, and recent work examining intestinal intraepithelial lymphocyte (IEL) trafficking. Particular emphasis will be placed on mechanisms linking barrier tissues with the joints and spine, including constitutive immune cell trafficking, tissue residency, and the influence of local microenvironments on immune cell function.
Results. Recent studies demonstrate that colon-derived CD4$^+$ IELs constitutively traffic from the intestinal epithelium to extraintestinal tissues, including joint-draining lymph nodes and inflamed joints. Rather than acquiring a stable inflammatory phenotype within the intestine, these cells retain features associated with immune regulation despite epithelial injury and inflammation. Single-cell transcriptomic, epigenetic, and T-cell receptor analyses demonstrate that shared IEL clonotypes undergo tissue-specific transcriptional reprogramming after leaving the gut, indicating that local tissue environments, rather than intestinal inflammation alone, determine their functional state. These findings support a model in which epithelial interfaces provide a mobile pool of immunologically experienced cells with functional plasticity that can be reshaped by inflammatory cues within musculoskeletal tissues. Together with evidence from psoriasis and inflammatory bowel disease, these observations suggest that barrier tissues participate in SpA pathogenesis not only through local inflammation but also by supplying migratory immune cells capable of responding to tissue-specific signals.
Conclusion. Epithelial barrier tissues represent critical interfaces linking mucosal and musculoskeletal immunity in SpA. Emerging evidence indicates that immune cell trafficking, rather than fixed inflammatory programming within the gut, may be a key mechanism underlying the gut-joint axis. Understanding how barrier-derived immune cells are subsequently instructed by the joint and spinal microenvironment provides a revised framework for SpA pathogenesis and identifies new opportunities to interrupt disease by targeting both immune cell migration and tissue-specific inflammatory programming.
Acknowledgements. Funding for this work includes grants from the National Institutes of Health (R01 AR075033 and P30 AR079369), the Shear Family Foundation, and the Spondylitis Association of America.

INV4

ENTHESIS BIOLOGY AND THE ROLE OF MECHANICAL STRESS IN THE PATHOGENESIS OF SPA

Gracey E.[1,2]
[1]Molecular Immunology and Inflammation Unit, VIB Centre for Inflammation Research, Ghent; [2]Dept. of Internal Medicine and Pediatrics, Faculty of Medicine and Health Sciences, Ghent University, Ghent, Belgium

The enthesis is the organ that links load bearing tendons and ligaments to bone, allowing for locomotion of the body. Inflammation of the enthesis, enthesitis, is arguably one of the initial locations of inflammation in spondyloarthritis (SpA). Enthesitis results in pain and loss of joint function, and can ultimately result in irreversible structural damage through ectopic ossification. In some joints, such as the sacroiliac joint and the intervertebral joint, the ossification can lead to complete joint fusion. Despite the importance of enthesitis in SpA, its biology remains poorly understood.
Tendinous tissue has a cellular and molecular composition that is distinct from other joint tissues affected by arthritis, such as the synovium. Tendi-

nous tissue is extracellular matrix rich as a result of its load bearing function, and relatively cell poor compared to synovial tissue. Immune cells are relatively infrequent in tendinous tissues. The cell composition of the enthesis in SpA has not been well characterized due to its inaccessibility.
Mechanical loading is a natural phenomenon for joints, and is essential for their homeostasis. To much or too little loading can result in degeneration of tendon/ligament tissues. In the context of spondyloarthritis, mechanical loading has been proposed to act as a trigger for focusing systemic inflammation on the enthesis. The exact mechanisms of how this occurs at the enthesis has yet to be discovered.
In this talk, recent advances in the fields of tendon tissue profiling and mechanobiology will be discussed. How these fields may intersect at the enthesis in the context of enthesitis in SpA will be speculated.

INV5

CASE CLOSED: HAS THE ARTHRITOGENIC PEPTIDES HYPOTHESIS FINALLY WON?

Colbert R.A.
National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS), National Institutes of Health (NIH), Bethesda, Maryland, USA

Since the discovery of the striking association between HLA-B*27 and ankylosing spondylitis (now axial spondyloarthritis [SpA]) and subsequently other forms of SpA, there has been no shortage of hypotheses to explain how HLA-B*27 predisposes to an immune-mediated inflammatory disease that often results in a unique pattern of aberrant bone formation in the axial skeleton. These ideas will be presented in historical perspective focusing on arthritogenic peptides as well as hypotheses linking aberrant features of HLA-B*27 to disease pathogenesis.
The discovery of T cell receptor (TCR)-b (TRBV) complementarity determining region-3 (CDR3) motifs expressed by CD8+ T cells that are expanded in HLA-B*27 positive individuals with SpA and recognize putative HLA-B*27 self-peptides, has provided strong support for the arthritogenic peptide hypothesis. Additional evidence from early clinical trials depleting TRBV9 T cells has supported a pathogenic role for these cells and opened novel therapeutic avenues for HLA-B*27 positive patients with SpA. However, many questions remain including the phenotype of these autoreactive CD8+ T cells, their target cells, and the role they play in the diverse, widespread tissue inflammation seen in axial SpA. It also remains unclear as to why HLA-B*27 among over 5,000 HLA-B alleles across the human population leads to autoimmunity. The hypothesis that it is the only allele that presents self-peptides resembling bacterial peptides remains unproven. Aberrant features of HLA-B*27 have also been linked to pathogenesis. For example, cell surface homodimers of HLA-B*27 can trigger CD4+ Th17 cells and NK cells through specific interactions with the killer immunoglobulin receptor KIR3DL2. This may explain, at least in part, the overproduction of IL-17 which, along with TNF, is an important therapeutic target in axial SpA. Intracellular HLA-B*27 misfolding also generated interest as a potential pathogenic mechanism, linking the allele to an inflammatory disease and more recently, aberrant bone formation. HLA-B*27 misfolding can generate ER stress and cause activation of stress response pathways collectively termed the unfolded protein response (UPR), leading to upregulation of cytokines including IL-23 which promotes Th17 development and activation. However, UPR activation, while prominent in HLA-B*27 transgenic (B27-Tg) rats carrying multiple transgene copies, is not consistently found in studies of human cells. Moreover, recent studies from B27-Tg rats have shown that UPR-mediated overexpression of IL-23 is not required for inflammatory gut disease. In contrast, HLA-B*27 misfolding in patient-derived mesenchymal stromal cells (MSCs) has been shown to activate XBP1 splicing and promote bone mineralization through XBP1-mediated upregulation of alkaline phosphatase (TNAP), providing evidence for a direct role for this allele in aberrant bone formation.
In summary, there is strong evidence that HLA-B*27 contributes to axial SpA pathogenesis in more than one way. There is clear evidence for pathogenic HLA-B*27-restricted $CD8^+$ autoreactive T cells, although the target cells and self-peptides recognized *in vivo* remain to be elucidated. HLA-B*27 homodimers can contribute to inflammation by triggering $CD4^+$ Th17 T cells, and evidence supports a direct role for this allele in pathologic bone formation.
Acknowledgements. The author is supported by the NIH Intramural Research Program (ZO1 AR041184). The views expressed here are those of the author and do not necessarily represent NIAMS, the NIH, or the Department of Health and Human Services of the US Federal Government.

INV6

SPA GENETICS BEYOND MHC CLASS I

Costantino F.[1,2]
[1]Dept. of Rheumatology, Ambroise Paré Hospital, AP-HP, Paris; [2]Infection & Inflammation, UMR 1173, Inserm, Université Versailles Saint Quentin en Yvelines / Paris Saclay, Montigny-Le-Bretonneux, France

Introduction. axial spondyloarthritis (axSpA) exhibits a strong genetic background largely driven by the major histocompatibility (MHC) class I region - notably the HLA-B27 allele. Over the past two decades, numerous non-MHC axSpA-associated loci have been uncovered, though many more remain to be identified. Beyond shedding light on disease pathophysiology, these genetic factors hold significant translational potential for improving disease management.
Results. The advent of genome-wide association studies (GWAS) enabled the identification of the first non-MHC axSpA-associated regions, *ERAP1* and *IL23R,* in 2007. Since then, up to 69 susceptibility loci have been uncovered, explaining approximately 15% of disease heritability, whereas the MHC region accounts for roughly 25%. Several approaches may help uncover the remaining heritability, including larger GWAS, family-based studies, extreme phenotype analyses, and multi-omic approaches. Furthermore, an enhanced understanding of the genetic architecture is gradually enabling the clinical translation of these discoveries, notably through the development of polygenic risk scores for diagnosis, prognostic or theranostic biomarkers, and the identification of novel therapeutic targets, such as ERAP1 inhibitors.
Conclusion. Further genetic discovery and a clearer understanding of axSpA architecture will support the shift toward precision medicine, aiding in earlier diagnosis, refined prognosis, and targeted therapeutic approaches.
Acknowledgements. F. Costantino was supported by grants from FOREUM, Société française de rhumatologie, Fondation pour la recherche médicale (FRM SPE201803005058) and Fondation Bettencourt Schueller.

INV7

POLYGENIC RISK SCORES, WHERE ARE WE NOW?

Brown M.A.
Dept. of Medical and Molecular Genetics, King's College London, London, UK

Introduction. Polygenic risk scores (PRS) represent a significant advance in genetic risk prediction for complex diseases. For axSpA, PRS show high discriminatory capacity – superior to both MRI and HLA-B27 testing including in replication studies. However, their clinical utility and performance across disease subsets and populations remain incompletely characterized. This overview addresses the current state of PRS implementation and identifies key gaps requiring further investigation.
Current Evidence Base. PRS demonstrates excellent discriminatory capacity for AS, outperforming conventional genetic and imaging biomarkers. Data in nr-axSpA are limited, with performance likely reduced due to heterogeneous clinical case classification rather than inherent limitations of PRS methodology. Critically, PRS uniquely identifies individuals at risk *before* disease onset, addressing a major unmet need for early diagnostic or predictive testing in axSpA.
Performance Considerations and Implementation. Test performance depends substantially on prior probability of disease – PRS discriminatory capacity is diminished in lower-prevalence population screening settings compared to diagnostic applications after symptom onset. Large prospective implementation studies (*e.g.* Our Future Health) are now underway to assess PRS performance in general population screening and will define real-world clinical utility. Beyond risk prediction and diagnosis, PRS have broader clinical applications: PRS have been developed to assess risk of anterior uveitis complicating AS, and may help identify other clinically significant AS subsets such as AS complicated by inflammatory bowel disease. PRS may also provide value in pharmacogenomics and healthcare triage, enabling personalized treatment selection and optimized patient prioritization within clinical settings. Combinations of genetic tests – so-called 'integrated risk scores' – may further improve discriminatory capacity across these diverse applications.
Ancestry and Future Directions. Performance in non-European populations has been investigated but remains incomplete, particularly in South Asian populations where axSpA is common but genetically understudied.

Novel approaches combining PRS with identification of AS-associated CD8 T-cell clonotypes may enable pre-clinical intervention and disease prevention, offering transformative potential for axSpA management.

INV9

HOW TO GET THE MOST OUT OF AI TODAY FOR CLINICAL PRACTICE AND RESEARCH

Karmacharya P.
Division of Rheumatology and Immunology, Dept. of Medicine, Vanderbilt University Medical Center, Nashville, TN, USA

Introduction. Generative artificial intelligence (AI) is advancing rapidly, but many clinicians and researchers still use it as a collection of isolated prompts rather than an integrated workspace. The central challenge is no longer access to AI, but translating its capabilities into reliable workflows while preserving privacy, scientific rigor, and human judgment. This hands-on workshop will demonstrate how current general-purpose AI tools can be used for practical clinical and research work.
Workshop Format. The session will be built around live, example task demonstrations in ChatGPT and Claude. Participants will see how to move from a loosely defined task to a usable output by defining the goal, providing context and source files, specifying constraints and the desired format, selecting the appropriate tool, iterating with the model, and verifying the output. Examples will be drawn from representative clinical and research workflows.
Learning Outcomes. Participants will learn to think in complete workflows rather than isolated prompts; distinguish tasks AI can perform reliably from those requiring close expert oversight; provide sufficient context without losing control of the objective; and use AI as an iterative collaborator and execution system. A simple system for dependable AI use will be emphasized: the expert user, an explicit standard operating procedure, and a trusted database or source set.
Conclusion. The most effective use of AI is neither a collection of clever prompts nor full automation. It is the deliberate design of human-supervised workflows that use current tools for what they do well while reserving clinical and scientific judgment for the user. Participants should leave with practical methods they can apply immediately in their own clinical and research work.
Disclosures. P. Karmacharya is the founder of Research Boost, an AI academic writing agent for clinical researchers.

INV10

A DERMATOLOGIST'S PERSPECTIVE OF PSA

Puig L.
Dept. of Dermatology, Hospital de la Santa Creu i Sant Pau, Universitat Autònoma de Barcelona, Barcelona, Spain

Introduction. Psoriatic arthritis (PsA) presents a complex diagnostic and therapeutic challenge at the intersection of dermatology and rheumatology. Up to one-third of patients with psoriasis (PsO) eventually develop PsA, with cutaneous manifestations preceding joint involvement in approximately 80% of cases. This temporal sequence points to a directed spatio-temporal link, where the skin serves as the primary site of immune dysregulation before inflammation spreads to the musculoskeletal system. Understanding the cellular vehicles driving this transition, along with clinical predictors and screening tools, is critical for preventing irreversible joint damage.
Methods. A literature search was conducted focusing on recent publications elucidating the pathophysiological mechanisms of the skin-joint axis in PsA. The search strategy targeted recent breakthrough research on myeloid precursors and tissue-resident memory T cells (Trm) migration the anatomical continuity of the nail-enthesis complex, and the clinical validation of the PURE-4 (Psoriatic arthritis UnclutteRed screening Evaluation) questionnaire.
Results. *Cellular Traffic and Microenvironment Regulation:* A distinct population of $CD2^+MHC\text{-}II^+CCR2^+$ myeloid precursors originates in psoriatic skin and migrates to the joints, differentiating into highly proinflammatory mononuclear phagocytes upon arrival in the synovium. In healthy joints, resident $CD200^+$ regulatory fibroblasts act as gatekeepers, engaging the CD200R1 receptor on these migrating myeloid cells to suppress inflammation. In PsA, a reduction in these regulatory fibroblasts permits these precursors to trigger interleukin-17 (IL-17) expression in local T cells.
Inflammatory Memory and Clonal Sharing: Parallel to myeloid trafficking, $CD8^+$ Trm cells, specifically the type 17 phenotype, drive chronicity in psoriasis and migrate to joints in PsA. Single-cell TCR sequencing confirmed the migration of specific T cells between compartments, identifying 155 shared $CD8^+$ T cell clones between paired skin and joint samples that exhibit a potent cytotoxic and tissue-resident phenotype.
The Skin-Joint vs. Bowel-Joint Axis: This pathway functions as a cutaneous analogue to the bowel-joint axis, where barrier organ dysregulation and IL-23 production prime innate-like lymphocytes (such as ILC3s, γδ T cells, and MAIT cells) to migrate systemically to the synovio-entheseal complex. Just as gut-derived $IL\text{-}17^+$ ILC3s home to the inflamed joints in ankylosing spondylitis, skin-derived Trm and myeloid precursors home to the synovio-entheseal complex, bridging mucosal/cutaneous and articular immunity.
Anatomical Links: Nail psoriasis serves as a powerful predictor of joint disease due to the anatomical continuity of the nail matrix with the distal interphalangeal (DIP) joint enthesis. Inflammation in the nail matrix, mediated by the IL-23/IL-17 axis, can spread via cell and cytokine trafficking directly to the underlying enthesis, leading to subclinical enthesitis and initiating the "cardinal lesion" of PsA/SpA.
Clinical Screening: Given the high rate of underdiagnosis and the risk of irreversible joint damage, early suspicion in the dermatology clinic is paramount. The PURE-4 questionnaire (Psoriatic arthritis UnclutteRed screening Evaluation) is a simple, 4-item tool that evaluates dactylitis, enthesitis, and axial/peripheral involvement. Validation studies in Spain have confirmed that a cutoff score of 32 is optimal for identifying patients at high risk. This tool can detect approximately one in four PsA patients up to a year before official diagnosis, providing a critical window for rheumatological referral and early intervention with biologics, such as IL-23 inhibitors, which may delay or prevent disease progression.
Conclusion. The propagation of inflammation from the skin to the joints is driven by specific migrating myeloid precursors and shared cytotoxic $CD8^+$ T-cell clones, reflecting a functional skin-joint axis. In the clinic, utilizing nail assessments and implementing the simple, validated PURE-4 screening tool in dermatology clinics provides a critical window for early rheumatological referral. Timely intervention with advanced therapies, such as IL-23 inhibitors, offers the potential to alter the disease trajectory and delay or prevent irreversible joint progression.

INV11

AXIAL PSA OR AXSPA WITH A RASH? FACT OR FICTION

Nash P.
School of Medicine, Griffith University, Brisbane, QLD, Australia

The debate continues between the "lumpers" and the "splitters" - is this but differing phenotypes of AxSpA with a rash, or eye inflammation or gut inflammation or are they separate diseases with shared features but also unique triggers, genetics and more importantly differing responses to therapeutic options?
So why even bother to make the distinction?
What are the potential diagnostic pitfalls?
How can the results of the ASAS-GRAPPA co-sanctioned AXIS study (1) inform this debate?
This session hopes to address some of these issues, and to consider a pathway forward.

References

1. TORGUTALP M *et al.*: Frequency and characteristics of axial involvement in psoriatic arthritis: results from the International Multicentre AXIS Study. *Ann Rheum Dis* 2026; 85(6); 1092-109.
2. VAN GAALEN FA, RUDWALEIT M: Challenges in the diagnosis of axial spondyloarthritis. *Best Pract Res Clin Rheumatol* 2023; 37(3): 101871. https://doi: 10.1016/j.berh.2023.101871
3. TSIAMI S, BARALIAKOS X: Pearls and pitfalls in diagnosing non-radiographic axial spondyloarthritis. *Mediterr J Rheumatol*. 2022; 33 (suppl 1): 109-14. https://doi.org/10.31138/mjr.33.1.109

INV12

GLP-1 RECEPTOR AGONISTS IN INFLAMMATORY ARTHRITIS: FROM METABOLIC RISK TO THERAPEUTIC OPPORTUNITY

Karacabeyli D.[1,2]
[1]Division of Rheumatology, Dept. of Medicine, University of British Columbia, Vancouver; [2]Arthritis Research Canada, Vancouver, Canada

Obesity is a pro-inflammatory state that has been consistently associated with higher disease activity and poorer response to treatment in inflammatory arthritides (IA) such as rheumatoid arthritis, psoriatic arthritis (PsA), and axial spondyloarthritis. Glucagon-like peptide-1 (GLP-1)-based therapies have emerged as effective treatments for obesity and type 2 diabetes (T2D). Among adults with T2D and/or obesity, they also reduce the risk of major adverse cardiovascular events, the leading cause of premature death in IA. Observational studies suggest that adults with IA derive similar cardiovascular benefits from GLP-1-based therapies to those reported in broader populations. As such, GLP-1-based therapies may serve as useful tools for improving cardiovascular-kidney-metabolic health in patients with IA and T2D or excess adiposity. Assessing whether GLP-1-based therapies may possess additional, potentially disease-modifying, benefits in IA has become an active area of research. The TOGETHER-PsA trial recently showed that the addition of tirzepatide to ixekizumab resulted in greater disease control than ixekizumab alone in adults with PsA and overweight or obesity. Data in other types of IA are limited and more studies are needed. Preclinical experiments have demonstrated that GLP-1-based therapies have direct, weight loss-independent anti-inflammatory properties. How much of the observed clinical benefits of GLP-1-based therapies are explained by weight loss versus direct (weight-independent) anti-inflammatory effects? This question, among others, remains unanswered, but the potential benefits of this medication class for IA are highly encouraging.

Acknowledgements. D. Karacabeyli is supported by the University of British Columbia Clinician Investigator Program, a Canadian Institutes of Health Research (CIHR) Canada Graduate Research Scholarship-Doctoral Award (FRN: DTA-210343), and a CIHR Project Grant (FRN: PJT-206202).

INV13

TURNING VIRUSES INTO DRUGS

Barone F.[1], Guo Q.[1], Manzanera A.[1], Nichols G.[1], Diers A.R.[1], Tak P.P.[1,2]
[1]Candel Therapeutics, Needham, MA, USA; [2]Faculdade de Medicina, Universidade de Lisboa, Portugal

Introduction. Viral immunotherapies are emerging as locally delivered biological medicines that can couple tumor infection, oncolysis and gene delivery to systemic antitumor immunity. Clinical validation of this concept is provided by Candel Therapeutics' aglatimagene besadenovec (aglatimagene), a replication-defective adenoviral vector encoding herpes simplex virus thymidine kinase, which converts valacyclovir into a cytotoxic metabolite, promotes tumor-cell death and antigen release, and supports $CD8^+$ T-cell-mediated in situ vaccination (DeWeese TL *et al*. *Lancet Oncol* 2026; 27: 673-85), and linoserpaturev, a tumor-selective HSV-1 immunotherapy engineered with Nestin promoter-controlled ICP34.5 expression and ICP6 disruption to support tumor-restricted replication, immunogenic oncolysis and safety (Ling AL *et al*. Nature 2023; 623: 157-166).

Methods. Aglatimagene plus valacyclovir was evaluated in clinical trials in localized prostate cancer, advanced non-small cell lung cancer and borderline resectable pancreatic ductal adenocarcinoma, while single and repeated administrations of linoserpaturev were evaluated in recurrent high-grade glioma. Clinical outcomes, safety, responses in injected and uninjected lesions, and translational biomarkers of local and systemic immune activation were assessed. In the context of our discovery efforts, these clinical outcomes and translational biomarker analyses were integrated with preclinical vector-engineering and payload-selection studies to define design principles for next-generation viral immunotherapies. These principles informed the enLIGHTEN™ Discovery Platform, a systematic, iterative HSV-based platform that uses human biology and advanced analytics to deconvolute tumor microenvironment properties, prioritize multimodal payloads in silico, and integrate optimized payloads into proprietary HSV-1 vectors. Candidate payloads are tested as single-gene minivectors in supervised multiplex assays before multigene-vector construction and candidate nomination.

Results. Across these clinical studies, aglatimagene and linoserpaturev demonstrated complementary evidence of clinical activity and immune activation. In localized prostate cancer, aglatimagene plus valacyclovir added to radiotherapy improved disease-free survival in a randomized phase 3 trial (HR 0.70; p=0.016) without clinically significant added toxicity; prostate cancer-specific disease-free survival and 2-year pathological complete response also favored aglatimagene. In advanced non-small cell lung cancer with inadequate response to immune checkpoint inhibition, aglatimagene was associated with durable survival, shrinkage of uninjected lesions, and biomarker evidence of increased interferon signaling, antigen presentation, myeloid activation and local inflammation, including increased IFNγ, CSF1, CX3CL1 and IL1β expression. In borderline resectable pancreatic ductal adenocarcinoma, aglatimagene plus prodrug combined with neoadjuvant therapy showed median overall survival of 31.4 months versus 12.5 months in the control arm. Linoserpaturev was well tolerated in recurrent high-grade glioma, with updated median overall survival of 11.8 months in arm A and 12.0 months in arm B after a single injection; HSV-1 seropositivity or seroconversion, injected and uninjected lesion responses, and expansion of T-cell repertoire diversity were associated with improved survival and immune activation. Serial multiomics after repeated linoserpaturev administration further demonstrated spatial and temporal tumor microenvironment remodeling, tumor replacement by immune cells, and viral- and tumor-specific T-cell activation.

These clinical observations support the rationale for enLIGHTEN™: viral vectors should be programmable, locally active, immunostimulatory, payload-bearing and biomarker-guided. As proof of mechanism, the Alpha-201 IL-12/IL-15 adaptive immunity stimulator was nominated after machine-learning-driven payload discovery and supervised validation; tumor/PBMC cocultures linked CD8+ T-cell, NK-cell and dendritic-cell activation to killing, and Alpha-201-IL-12/IL-15 suppressed EMT6 tumor growth by 60.0% ± 12.6 while increasing circulating $Ki67^+$ $CD8^+$ T cells, $Ki67^+$ NK cells and cDC2s. A tertiary lymphoid structure (TLS) inducer program was designed from immune checkpoint inhibitor-treated patient datasets to deliver payload combinations promoting lymphoid organization; in preclinical models, multiplexed Alpha-201 vectors induced TLS-like aggregates, mature fibroblastic reticular-cell networks, de novo CCL21 expression, tumor-growth inhibition and enhanced anti-PD-1 activity. A Macro1 innate immune surveillance activator uses Alpha-201 to interfere locally with the CD47/SIRPα "don't eat me" axis, increasing macrophage-mediated phagocytosis and demonstrating antitumor activity *in vivo* while seeking to limit systemic CD47-related toxicity.

Conclusion. Aglatimagene and linoserpaturev demonstrate that engineered viruses can be converted into clinically meaningful, mechanism-driven medicines when local tumor infection is translated into antigen release, immune activation, systemic antitumor effects and measurable biomarkers. enLIGHTEN™ extends these clinical learnings into a discovery engine for rationally designed, next-generation multimodal viral immunotherapies, enabling a pipeline that progresses from clinically validated viral immunotherapy mechanisms to programmable agents tailored for adaptive immunity stimulation, TLS induction and innate immune surveillance activation.

INV14

ENGINEERING THE BIOLOGICS OF THE FUTURE

Parren P.W.H.I.
Leiden University Medical Center, Dept. of Immunology, Leiden, the Netherlands and Gyes BV, Utrecht, The Netherlands

The pathophysiology of autoimmune and inflammatory diseases is highly complex, involving interconnected innate and adaptive immune pathways characterized by extensive crosstalk and functional redundancy. Preclinical and clinical studies indicate that multi-targeting strategies may enhance efficacy, improve safety, and enable broader modulation of disease-driving immune mechanisms. Accordingly, bispecific and multispecific antibodies capable of simultaneously or sequentially engaging multiple targets or pathways are emerging as promising next-generation therapeutics for these conditions.

A key challenge is to achieve greater therapeutic selectivity by directing biological activity toward disease-relevant cell populations while sparing non-target cells. Detailed investigation of antibody biology at the molecular level is revealing design principles that can be exploited to develop therapeutics with greater accuracy.

Building on these insights, we have developed the Multispecific Antibody Platform at Gyes BV. The platform applies novel principles of avidity engi-

neering to generate precision multifunctional antibodies that become conditionally active upon engaging defined combinations of targets co-expressed on selected cell populations. Here, I will discuss our progress in applying this approach to the development of highly selective multispecific antibody therapeutics.

INV15

EMERGING THERAPIES ACROSS THE SPONDYLO-ARTH-RITIS SPECTRUM

Deodhar A.A.
Division of Arthritis & Rheumatic Diseases, Oregon Health & Science University, Portland, Oregon, USA

Introduction. Since the turn of the century, rheumatology as a specialty has made tremendous strides in managing immune-mediated inflammatory arthritis with cytokine inhibitors which have dominated the therapeutic landscape. As of now, axial spondyloarthritis (axSpA) therapeutics have been limited to non-steroidal anti-inflammatory drugs, cytokine inhibitors of TNF, IL-17A, IL-17A+F, and blocking intracellular signaling of cytokines through Janus Kinase inhibitors. For psoriatic arthritis (PsA) in addition to the above, there is a wider selection with additional cytokine inhibitors such as p-40 IL-12/23 inhibitor, p-19 IL-23 inhibitors, as well as intracellular signaling inhibitors of PDE4 and tyrosine kinase (TYK2).
Emerging Therapeutics. With better understanding of multi-system inflammatory diseases pathophysiology, novel CD-19 (B cell) targeting T cell therapies (*e.g.* CAR-T, T-cell engagers) are emerging to treat diseases such as systemic lupus erythematosus, systemic sclerosis, small vessel vasculitides, and inflammatory myositis. Taking a leaf out of these strategies, focus in the treatment of axSpA is shifting from cytokine-based therapeutics to T cell targeting agents. There are several strategies for targeting pathogenic $CD8^+$ T cells in axSpA patients such as using an antibody to V9 antigen on the beta chain of the T cell receptor (TRBV9), using a bispecific antibody that binds to $CD3^+$ T cells and $TRBV9^+$ T cells, and possibly targeting integrins that are expressed on $CD8^+$ T cells. This 'shift in strategy' started with a published proof of concept case report of using a monoclonal antibody against $TRBV9^+$ $CD8^+$ T cells and that was followed by one randomized placebo-controlled trial of this antibody in HLA-B27 positive axSpA patients from Russia (1). Similar approaches being investigated or planned in axSpA include a bispecific antibody targeting TRBV9 $CD8^+$ T cells and IL-17A, and antibody against V21 antigen on the alfa chain of T cell receptor (TRAV21). There is renewed interest in investigating the efficacy of IL-1 inhibitor to treat axSpA after a couple of preliminary studies in the past gave conflicting results. Moving from antibodies to nanobodies (for a theoretical advantage of deep tissue penetration because of its small size), a novel IL-17A plus IL-17F nanobody has completed a 'proof of concept' phase 2 study in axSpA. TNF-like cytokine 1A (TL1A) that drives both inflammation and fibrosis has emerged as a potential target in axSpA, and a phase 2 study is ongoing.
New strategies to treat PsA include combining two cytokine inhibitors (*e.g.* TNF and IL-23 inhibitor) in difficult-to-treat patients. In a randomized controlled trial this combination was found to have a better ACR50 response compared to IL-23 inhibitor alone (2). With the new understanding that obese white adipose tissue is an endocrine organ secreting proinflammatory cytokines, adipocytes are emerging as a valid target in the management of PsA. A combination of IL-17A inhibitor with GLP-1 and GIP agonist was found to be more efficacious in treating musculoskeletal and skin manifestations of PsA compared to IL-17A inhibitor alone in a randomized open label trial (3). The IL-17A+F nanobody mentioned above has completed a successful phase 3 study in PsA. There are at least two new oral cytokine inhibitors (IL-23 inhibitor and IL-17 inhibitor) in development in the treatment of PsA, and eventually the IL-17 oral inhibitor will surely be tried in axSpA.
Conclusion. New therapeutic modalities and targets in the treatment of spondyloarthritis are emerging, and there is a shift from cytokine targets to pathogenic T cell targets in axSpA. Other potential therapeutic targets are IL-1 and TL1A. Psoriatic arthritis management is moving in to combination of biologics, targeting obese adipose tissue, and use of a IL-17A + IL-17F nanobody.

References
1. NASONOV EF, MAZUROV VI, LILA AM *et al.*: Effectiveness and safety of BCD180, anti-$TRBV9^+$ T lymphocyte monoclonal antibody in patients with active radiographic axial spondyloarthritis: 36 weeks results of double-blind randomized placebo-controlled phase II clinical study ELEFTA. *Scientific and Practical Rheumatology* 2024; 62(1): 65-80.
2. SCHER JU, MCINNIS IB, SORIANO ER *et al.*: Combination therapy in participants with active psoriatic arthritis using subcutaneous guselkumab and golimumab: week 24 results from a phase 2a, multicenter, randomized, double-blind, proof-of-concept. *Arthritis Rheumatol* 2026 Mar 25. doi: 10.1002/art.70152.
3. MEROLA JF, MEASE P, KIVITZ A *et al.*: Ixekizumab with tirzepatide achieved greater disease control than ixekizumab alone in adults with psoriatic arthritis and overweight or obesity: results from a randomized clinical Trial. *Arthritis Rheumatol* 2026 Mar 28. doi: 10.1002/art.70134.

INV17

MOLECULAR IMAGING IN SPA

Simon D.[1,2,3]
[1]Dept. of Rheumatology and Clinical Immunology, Charité University Medicine Berlin, Berlin; [2]German Rheumatism Research Centre (DRFZ) Berlin, a Leibnitz Institute, Berlin; [3]Fraunhofer Institute for Translational Medicine and Pharmacology, Allergology and Immunology, Berlin, Germany

Introduction. Conventional imaging captures only selected aspects of spondyloarthritis (SpA). Radiography visualises established structural damage, while magnetic resonance imaging (MRI) detects inflammatory and structural lesions within predefined anatomical regions. Neither directly characterises the cellular processes driving inflammation, tissue remodelling and new bone formation. Molecular imaging using positron emission tomography/computed tomography (PET/CT) may add quantitative, whole-body and biology-specific information in axial SpA and psoriatic arthritis (PsA).
Methods. Current evidence was reviewed for three major tracer concepts: ^{18}F-fluorodeoxyglucose (FDG) as a marker of glycolytic activity, ^{18}F-sodium fluoride (NaF) as a marker of osteoblastic activity and new bone formation, and fibroblast activation protein inhibitor (FAPI) tracers as markers of activated stromal fibroblasts. Their relationships with clinical disease activity, conventional imaging and treatment response were considered.
Results. The available data indicate that these tracers provide complementary rather than interchangeable information. FDG-PET can visualise inflammatory activity across multiple musculoskeletal domains. In PsA, total-body FDG-PET enables simultaneous assessment of joint, entheseal, nail and axial involvement and may detect clinically occult lesions. However, associations between FDG uptake and disease activity appear less consistent in axial SpA.
NaF-PET visualises active bone turnover. NaF-positive lesions have been detected at peripheral joints, entheses, the spine and sacroiliac joints, including asymptomatic sites. Their only partial overlap with MRI and radiographic abnormalities suggests that NaF-PET captures biological activity not fully represented by inflammation or established structural damage. Exploratory longitudinal studies further indicate that osteoblastic activity may change during effective biological therapy.
FAPI-PET provides a stromal perspective. FAP tracer uptake is increased in active arthritis and decreases following successful cytokine inhibition. In PsA, FAPI uptake is most prominent in peripheral joints and correlates with joint counts, composite disease activity and ultrasound findings. In axial SpA, emerging studies report associations between spinal FAPI uptake, ASDAS, C-reactive protein and MRI inflammation.
Conclusion. Molecular imaging does not replace MRI, ultrasound or radiography, but adds a distinct biological dimension. FDG reflects glycolytic inflammatory activity, NaF visualises osteoblastic activity and FAPI identifies activated stromal fibroblasts. Longitudinal multimodal studies are needed to establish whether molecular imaging can support biology-driven patient stratification and treatment selection in SpA.

INV20

NOVEL TRIAL DESIGNS

Coates L.C.
NDORMS, University of Oxford, Oxford, UK

Introduction. Randomised controlled trials (RCTs) remain the gold standard for evaluating the efficacy and safety of new therapies. However, the traditional paradigm of a two-arm, fixed-design trial is increasingly challenged by the complexity of modern clinical research. In spondyloarthritis (SpA), an expanding therapeutic landscape, growing interest in precision medicine, increasing costs of drug development, and the need to evaluate multiple treatment strategies efficiently all highlight the limitations of conventional trial designs. Innovative approaches offer opportunities to generate more informative evidence while maintaining the methodological rigour of randomisation.

This presentation will provide an overview of contemporary trial designs that are reshaping clinical research. It will begin by considering the rationale for moving beyond conventional RCTs and introduce the principles underpinning adaptive and master protocol designs. The talk will cover platform trials, which allow multiple interventions to be evaluated within a single, ongoing trial infrastructure using shared control groups and prespecified adaptive decision rules. Lessons from successful platform trials in other areas of medicine will be discussed, together with their potential application to rheumatology.

The presentation will also describe adaptive trial methodologies, including sample size re-estimation, adaptive enrichment strategies, and Bayesian approaches that allow accumulating evidence to inform pre-planned modifications to trial conduct while preserving statistical validity. These designs have the potential to improve efficiency, reduce participant exposure to ineffective treatments, and accelerate decision-making in both early- and late-phase clinical development.

Finally, the talk will consider how novel trial designs may support the future of precision medicine in SpA through biomarker-enriched populations, master protocols, registry-based randomised trials, and pragmatic studies embedded within routine clinical care. Emphasis will be placed on the opportunities and challenges associated with implementing these designs in rheumatology, including operational complexity, regulatory considerations, and the importance of international collaboration.

The overarching message is that novel trial designs are not a departure from rigorous clinical science, but rather an evolution of the randomised trial that enables researchers to answer more clinically relevant questions, more efficiently, and with greater flexibility. As the therapeutic landscape for spondyloarthritis continues to evolve, these approaches offer an important framework for delivering faster, more informative evidence to improve patient care.

Acknowledgements. Laura Coates is funded as a National Institute for Health and Care Research (NIHR) Research Professor (NIHR304262).

Oral presentations

O1

DECIPHERING THE ROLE OF RUNX3 IN HUMAN γδ T-CELLS

Melchior M.[1], Venturoli D.[1], Azouz A.[1], Donckier De Donceel E.[1], Pedron A.[1], Detavernier A.[1], Szymczak F.[1], Acolty V.[1], Thomas S.[1], Nguyen M.[1], Venken K.[2,3], Elewaut D.[2,3], Vermijlen D.[1,4,5], Goriely S.[1]
[1]ULB Center for Research in Immunology, Brussels; [2]Ghent University, Dept. of Rheumatology, Faculty of Medicine and Health Sciences, Ghent; [3]VIB-UGent Center for Inflammation Research, Unit for Molecular Immunology and Inflammation, Ghent; [4]Dept. of Pharmacotherapy and Pharmaceutics, ULB, Brussels; [5]WELBIO Dept., WEL Research Institute, Wavre, Belgium

Introduction. Genome wide association studies suggested *RUNX3* involvement in spondyloarthritis (SpA) pathogenesis. γδ T-cells are emerging as important drivers of early inflammation and tissue damage in SpA, yet the transcriptional mechanisms governing their differentiation and function remain poorly understood. This study aims to delineate the role of *RUNX3* in human γδ T-cells development and effector function.
Methods. CD3$^+$ cells were isolated from blood of healthy donors, edited using CRISPR-Cas9, cultured with IL-2 then αβCD8$^+$ and γδ T-cells were sorted by flow cytometry and processed for CUT&RUN, bulk-RNA or bulk ATAC sequencing. Humanised immune system (HIS) mice were generated by engrafting WT or *RUNX3*-KO human hematopoietic stem cells into NB-SGW mice. T-cells populations were analysed by flow cytometry 16 weeks post-engraftment.
Results. In both cell types, RUNX3 promotes cytotoxic and TCR-signalling genes expression, while repressing immune checkpoint and cytokine receptors. In γδ T-cell only, RUNX3 promotes *KLRB1* expression that has been associated with type 3 response, alongside repression of cytokine, chemokines receptor and transcription factor involved in naive/effector state regulation (Fig. 1). Most genomic regions with decreased accessibility upon *RUNX3-KO* overlapped with RUNX3 binding sites. Using Binding and Expression Target Analysis (BETA) on these overlapping peaks, we showed direct regulation of cytotoxic and TCR signalling pathways by RUNX3.

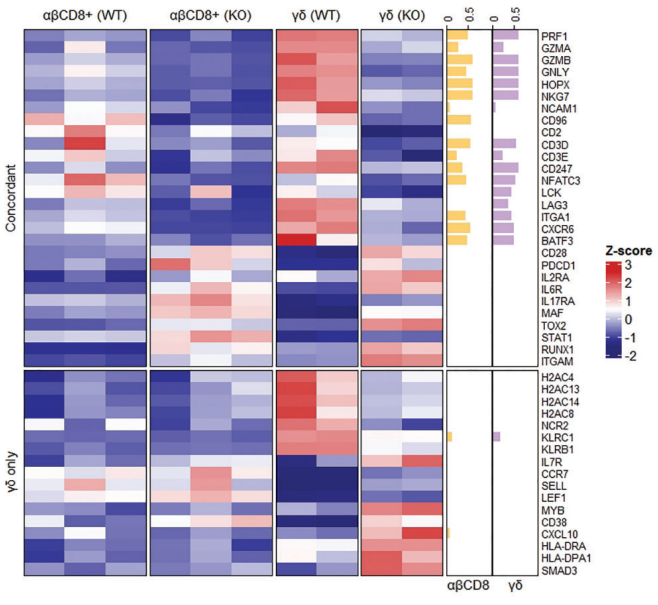


Fig. 1. Heatmap of differentially expressed gene after *RUNX3* knockout in αβCD8$^+$ and γδ T-cells.
From the same healthy donor, we generated 2-3 paired wild-type (WT) or *RUNX3-KO* (KO) technical replicates of αβCD8$^+$ and γδ T-cells depicted in each column. Z-score-normalized expression values for each differentially expressed gene are displayed in raw, with the upper part of the heatmap showing concordant RUNX3 regulation pathways between both cell types and the lower part showing γδ T-cells specific regulation. Evidence of direct regulation by RUNX3 is indicated on the right side of the heatmap for each gene and cell type, based on the highest regulatory potential scores (range: 0–1) assigned by BETA to gene-associated peaks.

In HIS-mice, RUNX3 deficiency resulted in a marked reduction of mature conventional human CD8$^+$ T-cells. In sharp contrast, γδ T-cell frequencies were maintained, although exhibiting impaired cytotoxic function.
Conclusion. *RUNX3* is a central regulator of human γδ T-cell effector programming, with substantial overlap with αβCD8$^+$ T-cells transcriptional network. However, in contrast to αβCD8$^+$ T-cells and *RUNX3-KO* mice studies, *RUNX3* is dispensable for human γδ T-cell development. Together, these findings identify *RUNX3* as a key determinant of γδ T-cell functional identity and support a mechanistic link between *RUNX3* variants and immune dysregulation in rheumatological diseases.
Acknowledgements. This project is carried out with the financial support of the Fonds Erasme for Medical Research and Fund for Scientific Research – FNRS.

O2

STK17B/DRAK2 ENRICHMENT IN T CELLS LINKS TO TREATMENT NON-RESPONSE IN SPONDYLOARTHRITIS

Remalante-Rayco P.[1,2,3], Pacheco A.[1,3], Srinath A.[1,2,3], Qaiyum Z.[1,3], Foroozan Boroojeni S.[1,2,3], Aparnathi M.[1,3], Korshko M.[1,3], Jo S.[4], Kim T.H.[4], Kapoor M.[1,3], Inman R.[1,3,5], Haroon N.[1,3,5]
[1]Schroeder Arthritis Institute, Spondylitis Program, Toronto Western Hospital, University Health Network, Toronto; [2]Institute of Medical Science, Temerty Faculty of Medicine, University of Toronto, Toronto; [3]Krembil Research Institute, University Health Network, Toronto, Canada; [4]Dept. of Orthopedic Surgery, Guri Hospital, Hanyang University College of Medicine, Guri, Republic of Korea; [5]Temerty Faculty of Medicine, University of Toronto, Toronto, Canada

Introduction. T cells play a central role in spondyloarthritis (SpA). DRAK2, a serine-threonine kinase encoded by *STK17B*, modulates T cell activation and survival. In autoimmune models, DRAK2 promotes autoreactive T cell persistence and reduces regulatory T cell abundance (1, 2). We identified *STK17B* upregulation in peripheral blood mononuclear cells (PBMCs) of SpA patients who did not respond to tumor necrosis factor inhibitors (3). To investigate DRAK2 in SpA inflammation and treatment resistance, we compared *STK17B*/DRAK2 expression between SpA patients and healthy controls, and between responders and non-responders to IL-17 inhibitors. We also examined *STK17B*/DRAK2 expression and therapeutic inhibition in the SKG mouse model of SpA.
Methods. PBMCs from SpA patients and controls were sorted by FACS, and *STK17B* expression was measured through qPCR. DRAK2 expression in mature CD4$^+$ and CD8$^+$ T cells was further analyzed using pre-biologic single-cell RNA-sequencing datasets. Protein-level tissue expression was assessed by immunohistochemistry in human spine and mouse ankle, tail, and ileum. Immunofluorescence was used to compare DRAK2 expression in spinal CD4$^+$ versus CD8$^+$ T cells. To evaluate therapeutic potential, curdlan-treated SKG mice received oral AP-204, a DRAK2 inhibitor, for 8 weeks.
Results. DRAK2 expression was significantly higher in SpA PBMCs than controls, particularly in CD4$^+$ T cells (Fig. 1A). Single-cell analyses confirmed higher DRAK2 expression in SpA CD4$^+$ T cells, especially effector and TR1 cells, with only modest increases in CD8$^+$T cells (Fig. 1B-E). DRAK2 expression was higher in SpA spinal tissue, with a CD4$^+$ bias, and in curdlan-SKG mouse tissues (Fig. 2). Pre-biologic CD4$^+$ T cell DRAK2 expression was higher in IL-17 inhibitor non-responders than responders. Importantly, AP-204 reduced clinical arthritis in treated mice compared with untreated controls.
Conclusion. DRAK2 is upregulated in SpA, enriched in CD4$^+$ T cells, and associated with IL-17 inhibitor non-response. Pharmacologic DRAK2 inhibition reduced arthritis severity in a preclinical SpA mouse model, supporting further mechanistic and therapeutic investigation.

References

1. RAMOS S, HERNANDEZ J, GATZKA M, WALSH CM: Enhanced T cell apoptosis within Drak2-deficient mice promotes resistance to autoimmunity. *J Immunol* 2008; 181(11); 7606-16.
2. MANDARANO A, HARRIS TL, CREASY BM *et al*.: DRAK2 contributes to type 1 diabetes by negatively regulating IL-2 sensitivity to alter regulatory T cell development. *Cell Rep* 2023; 42(2); 112106.
3. TALUKDAR A, IBEH N, MACCHAR R *et al*.: Cellular immune activation signatures at baseline predict response to TNF inhibitors in axial spondyloarthritis. [abstract] *Arthritis Rheumatol* 2024; 76 (suppl 9).

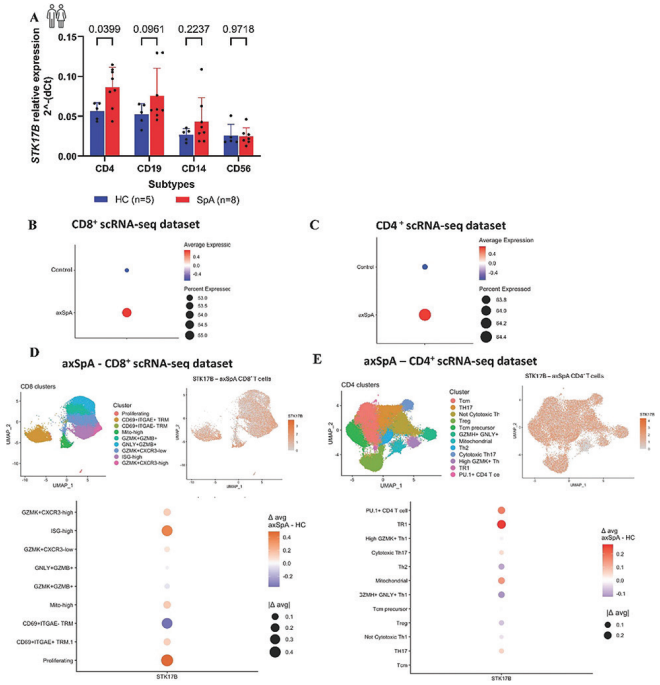


Fig. 1. DRAK2 is enriched in pathogenic immune subsets with a CD4⁺ bias.
A) *STK17B* qPCR in FACS-sorted PBMC subsets from pre-biologic SpA patients (n=8) and healthy control (HC) (n=5). *STK17B* expression in disease versus HC from scRNA-seq of PBMCs in (**B**) CD8⁺ and (**C**) CD4⁺ datasets. Cellular distribution of *STK17B* axSpA PBMCs from scRNA-seq of PBMCs in CD8⁺ (**D**) and CD4⁺ (**E**) datasets.

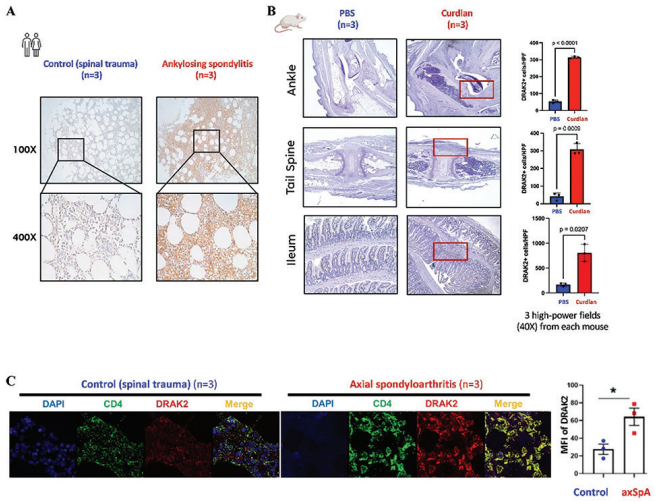


Fig. 2. DRAK2 protein is upregulated in inflamed SpA target tissues.
Histopathology by IHC staining for DRAK2 in **A**) spinal tissue of spinal trauma controls and axial spondyloarthritis (axSpA) patients and **B**) ankle, tail spine, and ileum of SKG mice administered with PBS (control) or curdian (SpA phenotype). **C**) immunofluorescence ofr axSpA spinal tissue versus spinal-trauma controls shows co-localization of DRAK2 with CD4⁺ T cells.

O3

MULTI-OMICS PROFILING OF GUT TISSUE FROM EARLY AXIAL SPONDYLOARTHRITIS PATIENTS REVEALS ENRICHMENT OF PUBLIC TRBV9⁺ CD8⁺ T CELL CLONES WITH A TYPE 17 SIGNATURE

Lukasik Z.[1,2], Vlieghe C.[2], Decruy T.[2], Manuello T.[2], Mortier C.[2], Vandamme N.[3], Roulandt R.[3], De Craemer A.S.[1], Van den Bosch F.[1], Venken K.[1,2], Elewaut D.[1,2]
[1]Faculty of Medicine and Health Sciences, Dept. of Internal Medicine and Pediatrics, Ghent University, Ghent; [2]Molecular Immunology and Inflammation Unit, VIB-UGent Center for Inflammation Research, Zwijnaarde; [3]VIB Single Cell Core, VIB, Ghent/Leuven, Belgium

Introduction. Intestinal inflammation is a major extra-musculoskeletal manifestation of axial spondyloarthritis (axSpA), influencing therapeutic approach and long-term disease outcomes (1). The gastrointestinal tract is a key contributor to immune system development, and emerging evidence suggests that gut-derived cells may migrate to other tissues and contribute to local inflammatory processes (2). We hypothesize that intestinal inflammation plays a role in priming immune pathways that drive chronic joint disease in axSpA.
Methods. We collected paired intestinal biopsies (terminal ileum and colon) and peripheral blood samples from newly diagnosed, treatment-naïve ptsatien with axSpA (n=10) and Crohn's disease (n=6) patients and healthy individuals (n=5). Epithelial, intraepithelial, and lamina propria cells (gut) next to peripheral blood mononuclear cells (blood) were isolated and enriched for specific cell subsets of interest using fluorescence-activated cell sorting; prior to processing for 5´ single-cell RNA sequencing (10x Genomics). The resulting multi-omics dataset was processed using the Cell Ranger pipeline and downstream analyses were performed in R.
Results. We created a comprehensive dataset comprising nearly 800,000 high-quality single-cell profiles representing most relevant epithelial and immune cell subsets. Intestinal inflammation was associated with a marked transcriptional imprint on epithelial cells, which affected the enterocyte-lymphocyte crosstalk. T cell receptor (TCR) profiling revealed the presence of expanded T cell clonotypes, especially among the intraepithelial CD8⁺ lymphocytes. We identified 442 clonotypes shared between study subjects; including condition-specific clonotypes, shared only between SpA patients. Finally, we detected T cells expressing public HLA-B27 associated TRBV9-CDR3-TRBJ2.3 sequences (3). These were found selectively in gut samples from seven SpA patients and one CD patient and expressed high levels of CD8, RORC, GZMA, IL17A and IL22 transcripts, suggestive for a type17 skewed phenotype.
Conclusion. These findings support the hypothesis that specific gut epithelial-immune responses might contribute to immune alterations associated with axSpA.
Acknowledgements. This research was supported by the research Foundation Flanders – FWO (grant ID G0A9824N, 11M8922N).

References

1. DI JIANG C, RAINE C: IBD considerations in spondyloarthritis. *Therapeutic Advances in Musculoskeletal Disease* 2020; 12.
2. GALVÁN-PEÑA S, ZHU Y, BOLA HS *et al.*: A dynamic atlas of immunocyte migration from the gut. *Science Immunology* 2024; 5; 9(91): eadi0672.
3. YANG X, GARNER LI, ZVYAGIN IV *et al.*: Autoimmunity-associated T cell receptors recognize HLA-B*27-bound peptides. *Nature* 2022; 612(7941): 771-77.

O4

EARLY INTERVERTEBRAL BONE FORMATION PRECEDES SYSTEMIC INFLAMMATION IN EXPERIMENTAL ARTHRITIS: EVIDENCE FROM X-RAY PHASE CONTRAST IMAGING

Verhoeven F.[1,2,3], Olivier C.[4], Aubry S.[5], Hecquet S.[2], Viennet C.[2], Totoson P.[2], Romand X.[3], Baillet A.[3], Broche L.[4], Wendling D.[1], Demougeot C.[2], Prati C.[1,2], Brun E.[4]

[1]Service de Rhumatologie, CHU de Besançon, Besançon; [2]UMR 1098 RIGHT, Université Marie et Louis Pasteur-EFS-INSERM Besançon; [3]UMR 5525- TIMC, Université Grenoble Alpes-CNRS, Grenoble; [4]UA7 STROBE, Université Grenoble Alpes-Inserm, Grenoble; [5]Service de Radiologie Ostéoarticulaire, CHU Besancon, Besançon, France

Introduction. The aim of this study was to demonstrate, using x-ray phase-contrast imaging, that ossification occurs upstream of both systemic and local inflammation in a model of reactive arthritis, adjuvant-induced arthritis (AIA).

Methods. Arthritis was induced in 6-week-old male Lewis rats by subcutaneous injection of *Mycobacterium butyricum*. Analyses were performed in control rats and at three key stages of peripheral arthritis development in AIA rats: preclinical phase (day 4 post-induction), onset phase (day 11), and acute phase (day 30). A 2-cm tissue sample from the proximal tail and a terminal blood collection were performed. X-ray phase-contrast tomography of the tail sample was performed at the European Synchrotron Radiation Facility (ESRF, Grenoble). For each time point, automated analyses assessed bone mineral density, microarchitecture (bone volume, connectivity, degree of anisotropy, trabecular thickness), new bone formation, as well as the presence of tenosynovitis. Plasma levels of iFABP, P1NP, IL-1β, and TNF-α were measured by ELISA to evaluate systemic inflammation, bone formation, and intestinal barrier disruption.

Results. A total of five rats per group were included. A decrease in bone mineral density was observed as early as day 11, concomitant with increased TNF-α and IL-1β levels. Microarchitectural changes (Fig. 1) were evident at day 11, including a significant decrease in trabecular thickness and connectivity ($p<0.01$). New bone formation was detected as early as day 4 at the periosteal surface of the metaphysis ($p<0.001$), in parallel with increased levels of bone formation markers (P1NP; $p<0.05$) and intestinal barrier damage markers (iFABP; $p<0.05$). Tenosynovitis was observed from day 11 onwards.

Conclusion. This study demonstrates that, in a model of reactive arthritis, early bone alterations and initiation of intervertebral ossification occur as early as day 4, preceding both systemic and local inflammation, in parallel with intestinal barrier disruption.

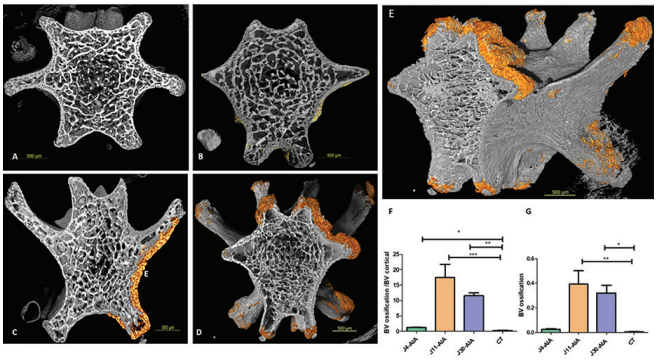


Fig. 1. Subligamentous and perisoteal ossification over time:
Subligamentous and perisoteal ossification (in orange) in controls (**A**) and 4 days (**B**), 11 days (**C**) and 30 days (**D** and **E**) after the induction of the arthritis and comparison of the volume of the ossification in the different time point (E, F); * $p<0.05$; ** $p<0.01$.

O5

DRUG-FREE REMISSION IN EARLY PERIPHERAL SPONDYLOARTHRITIS: 10-YEAR FOLLOW-UP FROM THE CRESPA-TRIAL

Damen C.[1], De Craemer A.S.[1,2], Webers C.[3,4], Elewaut D.[1,2], Varkas G.[1,2], Van den Bosch F.[1,2], Carron P.H.[1,2]

[1]Dept. of Rheumatology, Ghent University Hospital, Ghent; [2]VIB Inflammation Research Center, Ghent University, Ghent, Belgium; [3]Dept. of Rheumatology, Maastricht University Medical Centre, Maastricht; [4]Care and Public Health Research Institute (CAPHRI), Maastricht University, Maastricht, The Netherlands

Introduction. In the CRESPA-trial[1] very early pSpA patients (symptom duration ≤12 weeks), fulfilling the ASAS-classification criteria for pSpA, were randomized to golimumab (n=40) or placebo (n=20). Placebo-treated patients switched to golimumab at week 24 if no sustained clinical remission was achieved (primary end point) (1). In patients achieving sustained remission, treatment was stopped to assess the possibility of drug-free remission, while those not meeting the primary endpoint at week 48 continued golimumab (2).

Methods. All participants from the original CRESPA study were invited for a standardized evaluation 10 years after inclusion. Our aims were to assess the proportion of patients in (drug-free) clinical remission and identify baseline predictors of achieving (drug-free) remission at 10-year follow-up. Clinical remission was defined as the absence of swollen joints, enthesitis or dactylitis.

Results. As reported previously, sustained clinical remission was achieved in 82% (49/60) of patients after an induction regimen, 15 placebo treated patients needed golimumab (Fig. 1) (1). One patient was excluded due to progression to seronegative rheumatoid arthritis.

Of those 60 patients originally included in the CRESPA study, we were able to evaluate 49 patients within an average of 10.5 years (SD 0.87 years) of follow-up. Clinical remission was observed in 82% (40 of 49) patients (Fig. 2) including 30.6% (15/49) drug-free remission. 21 patients remained on golimumab, without unexpected serious adverse effects. Among the 9 patients not in remission, 7 had enthesitis, 1 arthritis, and 1 both.

There were no baseline differences regarding sex, age, HLA-B27 positivity, psoriasis, or initial treatment allocation between patients in remission and those not. However, drug-free remission was significantly less frequent in patients with psoriasis (OR = 0.11, 95% CI: 0.02–0.55, p=0.005).

Conclusion. After 10 years, most patients achieved clinical remission, with nearly one-third in drug-free remission. Baseline psoriasis reduced the likelihood of drug-free remission.

References

1. CARRON P, VARKAS G, CYPERS H *et al*.: Anti-TNF-induced remission in very early peripheral spondyloarthritis: the CRESPA study. *Ann Rheum Dis* 2017; 76(8): 1389-95.
2. CARRON P, VARKAS G, RENSON T *et al*.: High rate of drug free remission after induction therapy with golimumab in early peripheral spondyloarthritis. *Arthritis Rheumato*l 2018; 70(11): 1769-77.

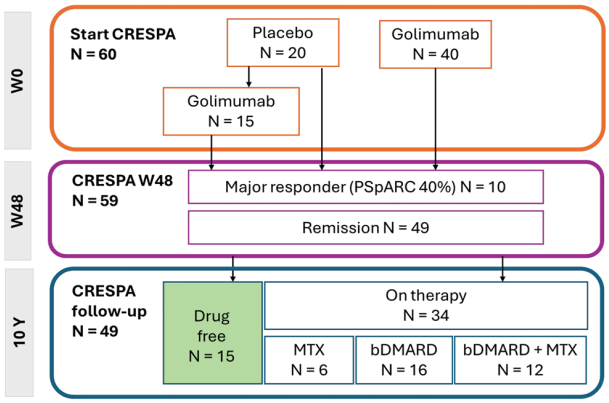


Fig. 1. Flowchart of the possible trajectories and number of patients in the different phases during the CRESPA study and 10 years after initiation.

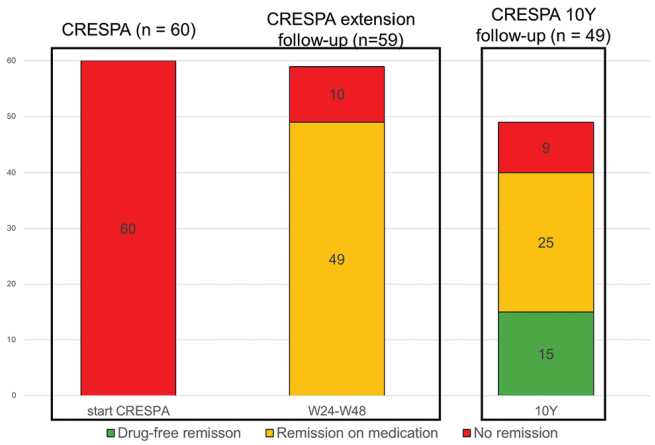


Fig. 2. Proportion of patients who did or did not achieve remission during the CRESPA trial and after 10 years of follow-up.

O6

NO SUPERIORITY OF IL-17A INHIBITORS OVER TNF INHIBITOR CYCLING AFTER FIRST TNF INHIBITOR FAILURE IN AXIAL SPONDYLOARTHRITIS: THE ROC-SPA RANDOMIZED TRIAL

Dalix E.[1], Goupille P.[2], Wendling D.[3], Roux C.[4], Samon J.H.[5], Brocq O.[6], Tournadre A.[7], Constantin A.[8], Breban M.[9], Cormier G.[10], Pascart T.[11], Lequerre T.[12], Claudepierre P.[13], Lespessailles E.[14], Le Goff B.[15], Sellam J.[16], Baillet A.[17], Schaeverbeke T.[18], Pavy S.[19], Devauchelle-Pensec V.[20], Gervais E.[21], Gossec L.[22], Miceli C.[23], Pers Y.M.[24], Lukas C.[25], Felten R.[26], Bouvard B.[27], Denis A.[28], Dernis E.[28], Presles E.[1], Rancon F.[1], Marotte H.[1]

[1]Université Jean Monnet, CHU Saint-Étienne, CIC 1408, INSERM, Saint-Étienne; [2]Dept. of Rheumatology, University Hospital of Tours, Tours; [3]Dept. of Rheumatology, CHU Besançon, Besançon; [4]Dept. of Rheumatology, CHU Nice, Université Côte d'Azur, Nice; [5]Dept. of Rheumatology, CHU Reims, Université de Reims Champagne-Ardenne, Reims, France; [6]Dept. of Rheumatology, CHPG Monaco, Monaco; [7]Dept. of Rheumatology, CHU Clermont-Ferrand, Clermont-Ferrand; [8]Dept. of Rheumatology, Toulouse University Hospital, Toulouse; [9]INSERM UMR1173, Ambroise Paré Hospital (AP-HP), Boulogne-Billancourt; [10]Dept. of Rheumatology, Centre Hospitalier Départemental Vendée, La Roche-sur-Yon; [11]Dept. of Rheumatology, Hôpital Saint-Philibert, Lille; [12]Dept. of Rheumatology, CHU Rouen, Inserm U1234, Rouen; [13]Dept. of Rheumatology, Henri-Mondor Hospital (AP-HP), Université Paris-Est Créteil, Créteil; [14]Translational Medicine Research Platform, CHU Orléans, Orléans; [15]Nantes Université, CHU Nantes, INSERM UMR1229, Nantes; [16]Dept. of Rheumatology, Sorbonne Université, AP-HP Saint-Antoine Hospital, Paris; [17]Université Grenoble Alpes, Grenoble; [18]Dept. of Rheumatology, Hôpital Pellegrin, CHU Bordeaux, Bordeaux; [19]Dept. of Rheumatology, Hôpital Bicêtre (AP-HP), Le Kremlin-Bicêtre; [20]Dept. of Rheumatology, CHU Brest, Université de Bretagne Occidentale, Brest; [21]CHU Poitiers, Université de Poitiers, Poitiers; [22]Sorbonne Université, INSERM, AP-HP Pitié-Salpêtrière Hospital, Paris; [23]Institut Pasteur, Cochin Hospital (AP-HP), Paris; [24]University of Montpellier, Inserm U1183, CHU Montpellier, Montpellier; [25]Dept. of Rheumatology, CHU Montpellier, Montpellier; [26]Dept. of Rheumatology, CHU Strasbourg, Strasbourg; [27]Dept. of Rheumatology, CHU Angers, Angers; CH Le Mans, Le Mans, France

Introduction. Axial spondyloarthritis (axSpA) is initially treated with non-steroidal anti-inflammatory drugs, followed by biological DMARDs in case of inadequate response. After failure of a first TNF inhibitor (TNFi), current recommendations allow either cycling to another TNFi or switching to a different mechanism such as IL-17A inhibition (IL-17Ai), although no randomized head-to-head comparison has been available to guide this choice (1).

Methods. ROC-SpA is a prospective, randomized, multicenter, open-label, phase IV superiority trial conducted in 31 centers in France and Monaco. Patients with active axSpA and inadequate response to a first TNFi were randomized (1:1) to receive either an IL-17Ai or a second TNFi. The primary endpoint was ASAS40 response at week 24. Analyses were performed according to the intention-to-treat principle.

Table I. Main characteristics of the patients at baseline.

	IL-17 inhibitors (N=158)	Anti-TNF inhibitors (N=142)	All (N=300)
Age, mean (SD), years	46.2 (12.4)	44.4 (12.7)	45.4 (12.6)
Male, n (%)	72 (45.6%)	76 (53.5%)	148 (49.3%)
HLA-B27 positive, n (%)	88 (59.1%)	78 (59.1%)	166 (59.1%)
Time between diagnosis and randomization, mean (SD), years	7.8 (8.1)	7.0 (8.4)	7.4 (8.2)
Smoking status, n (%)			
Former	45 (28.5%)	31 (21.8%)	76 (25.3%)
Current	54 (34.2%)	50 (35.2%)	104 (34.7%)
ax-Spa subtypes			
nr-axSpa	65 (40.4%)	61 (43.0%)	126 (42%)
AS	93 (59.6%)	81 (57.0%)	174 (58%)
BASDAI, mean (SD)	6.2 (1.5)	6.1 (1.4)	6.1 (1.4)
ASDAS, mean (SD)	3.2 (0.8)	3.2 (0.8)	3.2 (0.8)
BASFI, mean (SD)	4.3 (2.3)	4.5 (2.3)	4.4 (2.3)
CRP, mean (SD), mg/L	7.9 (13.5)	8.8 (16.0)	8.3 (14.7)
BMI, mean (SD), kg/m²	26.5 (5.3)	25.8 (5.1)	26.2 (5.2)
First anti-TNF duration, mean (SD), months	33.8 (45.9)	31.1 (42.7)	32.5 (44.3)
Causes of failure to the first anti-TNF, n (%)			
Primary nonresponse	47 (29.7%)	38 (26.8%)	85 (28.3%)
Secondary nonresponse	99 (62.7%)	93 (65.5%)	192 (64.0%)
Side effect	12 (7.6%)	11 (7.7%)	23 (7.7%)
Previous anti-TNF, n (%)			
Adalimumab	83 (52.5%)	71 (50.0%)	154 (51.3%)
Etanercept	34 (21.5%)	33 (23.2%)	67 (22.3%)
Golimumab	23 (14.6%)	18 (12.7%)	41 (13.7%)
Certolizumab	10 (6.3%)	11 (7.7%)	21 (7.0%)
Infliximab	8 (5.1%)	9 (6.3%)	17 (5.7%)

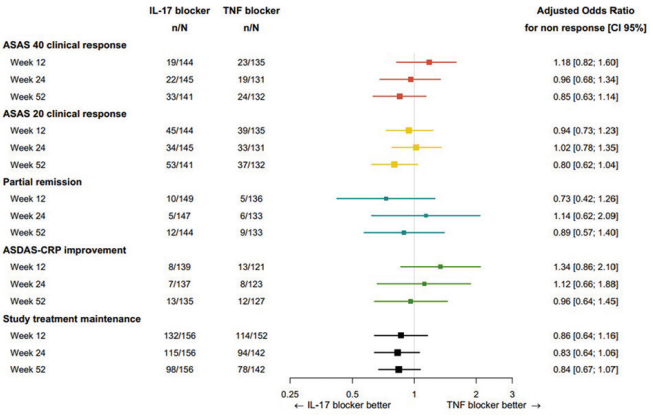


Fig. 1. Clinical responses with adjusted odds ratio in IL-17i and TNFi groups at weeks 12, 24, and 52.

Results. A total of 300 patients were included. Baseline characteristics are shown in Table I. ASAS40 response at week 24 was similar between groups (15.2% IL-17Ai vs. 14.5% TNFi), with no evidence of superiority of IL-17Ai (Fig. 1). No significant differences were observed for secondary outcomes (Fig. 1).

ASDAS decreased similarly in both groups over time. Treatment maintenance at week 54 was 62.8% with IL-17Ai and 54.9% with TNFi. Safety profiles were comparable. Exploratory analyses suggested trends favoring IL-17Ai in primary non-responders and specific subgroups, whereas TNFi cycling appeared more favorable after discontinuation due to adverse events.

Conclusion. Switching to an IL-17Ai is not superior to TNFi cycling after a first TNFi failure in axSpA.

Treatment selection should be tailored to each patient based on their profile and the reason for discontinuing TNFi.

Acknowledgements. This study was funded by a grant from the French Ministry of Health (PHRC-N 2016-0642).

Reference

1. DALIX E *et al*.: Rotation or change of biotherapy after TNF blocker treatment failure for axial spondyloarthritis. *BMJ Open* 2024;1 4: e087872.

O7

DEFINING AN MRI INDICATIVE OF AXIAL SPONDYLOARTHRITIS IN THE SACROILIAC JOINTS OF PATIENTS WITH AXIAL SPONDYLOARTHRITIS: A DATA-DRIVEN AND CONSENSUS-BASED FRAMEWORK OF RECOMMENDATIONS BY THE SPONDYLOARTHRITIS RESEARCH AND TREATMENT NETWORK AND THE SOCIETY OF SKELETAL RADIOLOGY

Maksymowych W.P.[1], Chalian M.[2], Bittar M.[3], Dubreuil M.[4], Jurik A.G.[5], Weber U.[6], Bayindir Tsechelidis O.[7], Bernard S.[8], Bird P.[9], Caplan L.[10], Carrino J.A.[11], Danve A.[12], Deodhar A.[3], Gensler L.S.[13], Grainger A.J.[14], Herregods N.[15], Horbal N.[1,16], Jans L.[17], Jaremko J.L.[18], Karmacharya P.[19], Magrey M.[20], Mayer A.[21], O'Neill J.[22], Paschke J.[23], Pedersen S.J.[24], Pezeshk P.[25], Rennie W.[26], Reveille J.D.[27], Salonen D.[28], Serfaty A.[29], Stovall R.[30], Tuite M.[31], Wichuk S.[1], Griffith J.[32], Østergaard M.[24], Lambert R.G.W.[18]

[1]University of Alberta, Medicine, Edmonton, Canada, [2]University of Washington, Radiology, Seattle; [3]Oregon Health and Science University, Medicine, Portland; [4]Boston University School of Medicine, Medicine, Boston, USA; [5]Aarhus University Hospital, Radiology, Aarhus, Denmark; [6]Medical Centre Zenit, Rheumatology, Schaffhausen, Switzerland; [7]Dept. of Medicine, University of Ottawa, Ottawa, Canada; [8]Brooke Army Medical Center, Radiology, Fort Sam, USA; [9]University of New South Wales, Medicine, Sydney, Australia; [10]University of Colorado School of Medicine, Medicine, Aurora; [11]Hospital for Special Surgery, Radiology and Imaging, New York; [12]Yale University School of Medicine, Medicine, New Haven; [13]University of California, San Francisco, USA; [14]Cambridge University Hospitals NHS Foundation Trust, Radiology, Cambridge, UK; [15]Ghent University Hospital, Radiology and Nuclear Medicine, Ghent, Belgium; [16]Danylo Halytsky Lviv National Medical University, Medicine, Lviv, Ukraine; [17]Dept. of Radiology, Ghent University Hospital, Ghent, Belgium; [18]University of Alberta, Radiology and Diagnostic Imaging, Edmonton, Canada; [19]Vanderbilt University Medical Center, Medicine, Nashville; [20]Case Western Reserve University School of Medicine, Medicine, Cleveland; [21]Hackensack University Medical Center, Adult and Pediatric Rheumatology, Nutley, USA; [22]McMaster University, Radiology, Hamilton; [23]CARE ARTHRITIS Limited, Edmonton, Canada; [24]Copenhagen Center for Arthritis Research (COPECARE), Copenhagen, Denmark; [25]UT Southwestern Medical Center, Radiology, Dallas, USA; [26]University Hospitals of Leicester NHS Trust, Radiology, Leicester, UK; [27]McGovern School of Medicine at the University of Texas Health Science Center at Houston, Medicine, Houston, USA; [28]University of Toronto, Medical Imaging, Toronto, Canada; [29]Medscape Universidade Federal do Estado do Rio de Janeiro - UNIRIO, Radiology, Rio De Janeiro, Brazil; [30]University of Washington, Medicine, Seattle; [31]University of Wisconsin School of Medicine and Public Health, Radiology, Madison, USA; [32]The Chinese University of Hong Kong, Dept. of Imaging and Interventional Radiology, Hong Kong, China

Background/Objective The ASAS-SPARTAN 2025 revised classification criteria for axial spondyloarthritis (axSpA) include a new MRI variable that requires assessment of both inflammatory and/or structural lesions in the sacroiliac joints (SIJ). SPARTAN and the Society of Skeletal Radiology (SSR) have collaborated to develop guidance for a definition of a positive MRI.

Methods. MRI scans from three cohorts (two inception, one case-control) and 1050 cases were analyzed for MRI lesions typical of axSpA. We calculated sensitivity and specificity for the number of SIJ quadrants with BME, erosion, fat lesion, or sclerosis or SIJ halves with backfill or ankylosis when central readers considered the MRI was indicative of axSpA was the reference criterion. Sensitivity analyses included 'rheumatologist diagnosis of axSpA' as the reference endpoint. These analyses and evidence from the SLR were discussed by the working group, which drafted overarching principles and recommendations followed by rounds of Delphi consensus voting.

Results. Consensus was attained on 8 overarching principles and 10 recommendations which focus on demographic and clinical context in MRI interpretation. Consensus was attained on prespecified targets ≥90% specificity and ≥70% sensitivity for the quantitative component. MRI indicative of axSpA by central readers was recorded in 53.4% CLASSIC, 60.5% SASPIC, 88.9% IMAX-SPICE cases. A new quantitative component addressing the spectrum of axSpA was based on BME ≥3 consecutive slices OR any two of the following 3 features (BME, Erosion, Fat lesion) each lesion being present in ≥3 SIJ quadrants OR ankylosis in ≥3 SIJ halves. This attained sensitivity/specificity of 88%/90%, 81%/98%, 96%/95% in the 3 cohorts, respectively, where 'MRI indicative of axSpA' by central readers was the reference (Fig. 1). Prespecified target specificity ≥90% was attained in all sensitivity analyses (Fig. 2).

Conclusion. The SPARTAN-SSR recommendations provide comprehensive guidance on what constitutes an MRI indicative of axSpA based on qualitative and quantitative considerations.

Performance of Rule of '3s': Central reader designation 'MRI indicative of axSpA' is gold standard

Cohort	MRI indicative of axSpA=Yes N (%)	MRI indicative of axSpA=No N (%)	Sensitivity (95% CI)	Specificity (95% CI)	LR+	LR-
	Yes=85	No=416				
CLASSIC (n=501)	78 (91.8)	34 (8.2)	91.8 (83.8 - 96.6)	91.8 (88.8 - 94.3)	11.2	0.1
	Yes=77	No=233				
SASPIC (n=310)	60 (77.9)	5 (2.1)	77.9 (67.0 - 86.6)	97.9 (95.1 - 99.3)	36.3	0.2
	Yes=147	No=92				
IMAX-SPICE (n=239)	139 (94.6%)	4 (4.3%)	94.6 (90.9-98.2)	95.7 (91.5-99.8)	21.8	0.1

Fig. 1.

Performance of Rule of '3s': Local Rheumatologist diagnosis of axSpA is gold standard

Cohort	Diagnosis of axSpA=Yes N (%)	Diagnosis of axSpA=No N (%)	Sensitivity (95% CI)	Specificity (95% CI)	LR+	LR-
	Yes=148	No=353				
CLASSIC (n=501)	87 (58.8)	25 (7.1)	58.8 (50.4 - 66.8)	92.9 (89.7 - 95.4)	8.3	0.4
	Yes=124	No=185				
SASPIC (n=309)	60 (48.4)	5 (2.7)	48.4 (39.3 - 57.5)	97.3 (93.8 - 99.1)	17.9	0.5
	Yes=161	No=78				
IMAX-SPICE (n=239)	136 (84.5)	7 (9.0)	84.5 (78.9-90.1)	91.0 (84.7-97.4)	9.4	0.2

Performance of Rule of '3s': Local reader designation 'MRI indicative of axSpA' is gold standard

Cohort	MRI indicative of axSpA=Yes N (%) N=150	MRI indicative of axSpA=No N (%) N=351	Sensitivity (95% CI)	Specificity (95% CI)	LR+	LR-
CLASSIC* (n=501)	90 (60)	22 (6.3)	60 (51.7 - 67.9)	93.7 (90.7 - 96.0)	9.6	0.4

*Local reader assessment 'MRI is indicative of axSpA' only available in CLASSIC

Fig. 2.

O8

EARLY DIAGNOSIS OF AXIAL SPONDYLOARTHRITIS IS ASSOCIATED WITH LESS LONG-TERM SPINAL STRUCTURAL DAMAGE: A LONGITUDINAL ANALYSIS OF THE SWISS CLINICAL QUALITY MANAGEMENT IN RHEUMATIC DISEASES REGISTRY

Steimer A.[1], Brändli J.[2], Bachman M.[1], Götschi A.[2], Rodrigues C.L.[2], Nissen M.J.[3], Möller B.[4], Bürki K.[1], Wendiggensen M.[5,6], Grosswiler S.[7], Andor M.[8], Dan D.[9], Förger F.[10], Adler S.[11], Kyburz D.[12], Distler O.[1], Micheroli R.[1], Ramiro S.[13,14], Ciurea A.[1]

[1]Dept. of Rheumatology, Zurich University Hospital, University of Zurich, Zurich; [2]Swiss Clinical Quality Management in Rheumatic Diseases Foundation, Zurich; [3]Dept. of Rheumatology, Geneva University Hospital, Geneva; [4]Dept. of Rheumatology and Immunology, Inselspital, Bern University Hospital, University of Bern, Switzerland; [5]Johns Hopkins University School of Advanced International Studies, Washington DC, USA; [6]ETH Zurich, Zurich; [7]Swiss Ankylosing Spondylitis Association, Zurich; [8]Rheumatologie im Zürcher Oberland, Uster; [9]Dept. of Rheumatology, Lausanne University Hospital, University of Lausanne, Lausanne; [10]Dept. of Rheumatology, Cantonal Hospital St. Gallen, St. Gallen, Switzerland; [11]Dept. of Rheumatology, Cantonal Hospital Aarau, Aarau; [12]Dept. of Rheumatology, University Hospital Basel, University of Basel, Basel, Switzer-

land; [13]Dept. of Rheumatology, Leiden University Medical Centre, Leiden; [14]Dept. of Rheumatology, Zuyderland Medical Centre, Heerlen, The Netherlands

Introduction. Early axial spondyloarthritis (axSpA) was defined as axial symptoms ≤2 years. Spinal structural damage over time in early axSpA was compared with established axSpA using the modified Stoke Ankylosing Spondylitis Spinal Score (mSASSS).
Methods. We included patients from the Swiss Clinical Quality Management in Rheumatic Diseases (SCQM) registry who had ³2 available radiographs between 2004 and 2024. Lateral cervical and lumbar radiographs were scored by two readers blinded to clinical data, but not to chronology. Patients were classified at diagnosis as early (≤2 years) or established (>2 years) axSpA. We applied a four-parameter logistic model to assess longitudinal spinal structural damage (Fig. 1A). In this model, the starting mSASSS value was fixed at zero (L), while the remaining three estimated parameters describe the progression over time: the time to median progression (M), the maximum slope (S), and the upper asymptote (U), representing long-term spinal structural damage. To account for between-patient variability, random effects were included on M and U. In addition, a second model was fitted with adjustment for mSASSS at the first radiograph.

Table I. Characteristics of patients with early and established axSpA.

Variable	Early axSpA	Established axSpA	*p*-value
N	138	238	
Male sex, N (%)	89 (64.5)	139 (58.4)	0.27
HLA-B27 positive, N (%)	83 (67.5)	146 (70.2)	0.62
	At diagnosis		
Age at diagnosis, years	34.9 (11.8)	36.2 (9.9)	0.13
Smokers at diagnosis, N (%)	54 (39.4)	94 (40.3)	0.93
	At the time of first radiograph		
Diagnosis to first radiograph, years	5.4 (6.5)	6.6 (8.5)	0.62
ASDAS at first radiograph	2.5 (1.1)	2.5 (1.1)	0.83
CRP (mg/l) at first radiograph	8.2 (13.2)	6.5 (8.5)	0.89
mNYC status at first radiograph, N (%)	79 (60.8)	176 (78.9)	< 0.001
mSASSS at first radiograph	4.1 (11.4)	6.8 (15.6)	0.01
N syndesmophytes at first radiograph	1.6 (4.2)	2.5 (5.6)	0.09
	At the time of last radiograph		
mSASSS at last radiograph	5.1 (12.6)	8.5 (17.4)	0.01
N syndesmophytes at last radiograph	1.9 (4.7)	3.1 (6.2)	0.05
Follow-up time from diagnosis to last radiograph, years	10.4 (7.1)	12.2 (9.3)	0.16
Time between first and last radiograph, years	5.0 (3.3)	5.6 (3.7)	0.21

Values are reported as mean and standard deviation. ASDAS, Axial Spondyloarthritis Disease Activity Score; axSpA, axial spondyloarthritis; CRP, C-reactive protein; HLA-B27, Human Leucocyte Antigen B27; mNYC, modified New York Criteria; mSASSS, modified Stoke Ankylosing Spondylitis Spine Score.

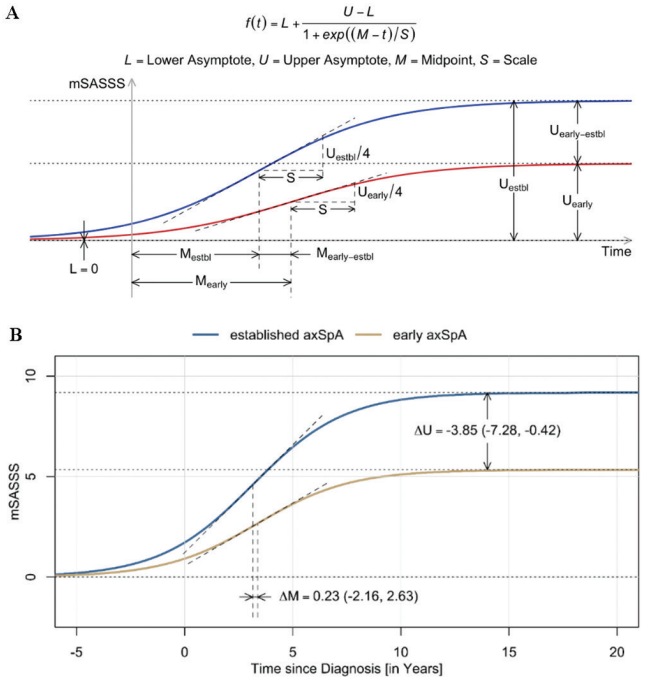


Fig. 1. A. Four-parameter logistic model to compare radiographic progression over time between early and established axSpA: schematic representation.
B. Estimated differences in long-term spinal structural damage and time to median progression between early and established axSpA (model-based estimates using SCQM data). axSpA, axial spondyloarthritis; mSASSS, modified Stoke Ankylosing Spondylitis Spine Score.

Results. In total, 376 patients were included (138 early and 238 established axSpA) with 1181 radiographic sets. The smallest detectable change was 2.12 and the intraclass-correlation coefficient for the mSASSS between the two scorers was excellent with 0.94. The characteristics of early and established axSpA were comparable (Table I). We found a statistically significant lower long-term mSASSS in early compared to established axSpA (difference in parameter ΔU=-3.85 mSASSS points (95%CI -7.28 to -0.42) (Fig. 1B). This difference became apparent once both curves approached a plateau, which occurred 10-15 years after diagnosis and was statistically significant after adjustment for radiographic damage at first radiograph (ΔU=-5.70 mSASSS points, 95%CI -9.31 to -2.09).
Conclusion. Early diagnosis of axSpA is associated with clinically relevant lower long-term spinal structural damage.

O9

2026 UPDATE OF THE ASAS MRI DEFINITIONS OF SACROILIAC JOINT INVOLVEMENT FOR CLASSIFICATION OF AXIAL SPONDYLOARTHRITIS: TESTING OF THE CANDIDATE LESIONS IN THE CLASSIC COHORT

Protopopov M.[1], Meghnathi B.[2], Ramiro S.[3], Sepriano A.[4], van Lunteren M.[3], Rudwaleit M.[5], Poddubnyy D.[6], Baraliakos X.[7]
[1]Medical Clinic for Gastroenterology, Infectious Diseases and Rheumatology CBF, Charité – Universitätsmedizin Berlin, Berlin, Germany; [2]Dept. of Clinical Immunology and Rheumatology, Smt. NHL Municipal Medical College, Ahmedabad, Gujarat; RheumaCARE, Ahmedabad, Gujarat; Marengo CIMS Hospital, Ahmedabad, India; [3]Leiden University Medical Center, Leiden, The Netherlands; [4]NOVA Medical School, Universidade Nova de Lisboa and Dept. of Rheumatology, Hospital de Egas Moniz - ULSO, Lisbon, Portugal; [5]Bielefeld University, Medical School and University Medical Center OWL, Klinikum Bielefeld, Dept. of Internal Medicine and Rheumatology, Bielefeld, Germany; [6]Division of Rheumatology, Dept. of Medicine, University of Toronto and Schroeder Arthritis Institute, University Health Network, Toronto, Canada; [7]Rheumazentrum Ruhrgebiet Herne, Ruhr Universität Bochum, Bochum, Germany

Introduction/Objective. Candidate MRI lesions for defining sacroiliac joint (SIJ) involvement in axial spondyloarthritis (axSpA) were identified in a systematic literature review (1). We tested their diagnostic performance in the ASAS part of the CLASSIC cohort.
Materials and methods. CLASSIC included consecutive patients with undiagnosed back pain lasting ≥3 months and onset ≤45 years (ASAS part: n=514). The reference standard was the final (stage 5) diagnosis by the treating rheumatologist after central imaging evaluation. Thirty-five SLR-derived and 18 expert-proposed lesions were assessed in a random 50% testing dataset. A lesion was present if reported by two central readers; local-reader assessment was also tested. Sensitivity (Sn), specificity (Sp), and positive (LR+) and negative (LR-) likelihood ratios were calculated.
Results. The testing dataset comprised 243 patients (axSpA prevalence 42%; mean symptom duration 8 years). Twenty-seven SLR-derived lesions were testable. Individual inflammatory lesions had moderate Sn (29.7-53.5%) and high Sp (87.3-98.6%); structural lesions had lower Sn (9.9-41.6%) but high Sp (91.5-100%) (Table I). Combinations of inflammatory or structural lesions increased Sn (29.7-69.3%) while retaining high Sp (Table II). The best performance was observed for central-reader global assessment of axSpA-typical structural and inflammatory MRI lesions (Sn 52.5%, Sp 98.6%, LR+ 37.26), although local readings performed worse (Sn 52%, Sp 92%, LR+ 6.7). Among quantitative definitions, the ASAS active sacroiliitis definition combined with Leiden structural lesions showed Sn 53.5%, Sp 96.5% and LR+ 15.18; combined with the ASAS MRI Group structural definition, it showed Sn 54.5%, Sp 95.1% and LR+ 11.05.
Conclusion. Several SIJ MRI lesions performed well in CLASSIC. Combinations of active or structural lesions performed particularly well. These data will inform the upcoming 2026 revision of the ASAS definition of SIJ MRI involvement in axSpA.

Reference

1. PROTOPOPOV M, MEGHNATHI B, SEPRIANO A *et al.*: 2026 update of the ASAS MRI definitions of sacroiliac joint and spinal involvement for classification of axial spondyloarthritis: a systematic literature review. Submitted to 15th International *Congress* on Spondyloarthritides 2026.

O9 - Table I. Performance of individual inflammatory/structural MRI lesions in CLASSIC.

Lesion	Sn, % (95% CI)	Sp, % (95% CI)	LR+ (95% CI)	LR- (95% CI)
BME ≥ 4 SIJ quadrants	29.7 (21.0–39.6)	98.6 (95.0–99.8)	21.09 (5.16-86.25)	0.71 (0.63-0.81)
BME in ≥ 3 consecutive slices	43.6 (33.7–53.8)	93.7 (88.3–97.1)	6.87 (3.52–13.43)	0.60 (0.50–0.72)
BME in ≥ 4 consecutive slices	36.6 (27.3–46.8)	97.9 (94.0–99.6)	17.34 (5.50–54.68)	0.65 (0.56–0.75)
BME in ≥ 2 SIJ quadrants OR in ≥ 3 consecutive slices	53.5 (43.3–63.5)	87.3 (80.7–92.3)	4.22 (2.64–6.74)	0.53 (0.43–0.66)
BME in ≥ 3 SIJ quadrants OR in ≥ 3 consecutive slices	48.5 (38.4–58.7)	93.0 (87.4–96.6)	6.89 (3.67–12.94)	0.55 (0.46–0.67)
BME in ≥ 4 SIJ quadrants OR in ≥ 3 consecutive slices	46.5 (36.5–56.7)	93.7 (88.3–97.1)	7.34 (3.77–14.29)	0.57 (0.47–0.69)
Fat lesions in ≥ 4 SIJ quadrants	25.7 (17.6–35.4)	98.6 (95.0–99.8)	18.28 (4.44–75.27)	0.75 (0.67–0.85)
Fat lesion in ≥ 2 consecutive slices	35.6 (26.4–45.8)	96.5 (92.0–98.8)	10.12 (4.12–24.90)	0.67 (0.57–0.77)
Erosions ≥ 2 SIJ quadrants	41.6 (31.9–51.8)	98.6 (95.0–99.8)	29.52 (7.31–119.18)	0.59 (0.50–0.70)
Erosions in ≥ 3 consecutive slices	30.7 (21.9–40.7)	100.0 (97.4–100.0)	-	0.69 (0.61–0.79)
Erosions in ≥ 4 consecutive slices	22.8 (15.0–32.2)	100.0 (97.4–100.0)	-	0.77 (0.69–0.86)
Backfill (any)	10.9 (5.6–18.7)	100.0 (97.4–100.0)	-	0.89 (0.83–0.95)
Ankylosis (any)	9.9 (4.9–17.5)	99.3 (96.1–100.0)	14.06 (1.83–108.10)	0.91 (0.85–0.97)
Fat lesion OR erosion ≥ 1 quadrant	55.4 (45.2–65.3)	91.5 (85.7–95.6)	6.56 (3.71–11.59)	0.49 (0.39–0.61)
Fat lesion OR erosion ≥ 4 quadrants	42.6 (32.8–52.8)	98.6 (95.0–99.8)	30.23 (7.49–121.93)	0.58 (0.49–0.69)
Fat lesion ≥ 1 AND erosion ≥ 1 quadrants	29.7 (21.0–39.6)	100.0 (97.4–100.0)	-	0.70 (0.62–0.80)
Deep fat lesion (>1 cm) ≥ 1 OR erosion ≥ 2 quadrants	47.5 (37.5–57.7)	95.8 (91.0–98.4)	11.25 (5.01–25.27)	0.55 (0.45–0.66)
Deep fat lesion (>1 cm) ≥ 1 OR erosion ≥ 3 quadrants	43.6 (33.7–53.8)	97.2 (92.9–99.2)	15.47 (5.74–41.67)	0.58 (0.49–0.69)
Ankylosis OR bone bud	14.9 (8.6–23.3)	99.3 (96.1–100.0)	21.09 (2.83–157.10)	0.86 (0.79–0.93)

BME: bone marrow edema; LR+: positive likelihood ratio; LR– : negative likelihood ratio; n/a: not possible to calculate because the false positive rate is zero; Sn: Sensitivity (Sn); Sp: Specificity; in blue: candidate lesions added after working group discussion.

O9 - Table II. Performance of combinations of inflammatory and structural MRI lesions in CLASSIC.

Lesion	Sn, % (95% CI)	Sp, % (95% CI)	LR+ (95% CI)	LR– (95% CI)
BME ≥1 quadrant AND erosion (scored similarly to ASAS definition)	35.6 (26.4–45.8)	98.6 (95.0–99.8)	25.31 (6.24–102.71)	0.65 (0.56–0.76)
BME ≥1 quadrant AND fat lesion ≥1 quadrant	29.7 (21.0–39.6)	97.2 (92.9–99.2)	10.54 (3.83–29.00)	0.72 (0.64–0.82)
BME ≥2 quadrants OR fat lesion ≥1 quadrant	66.3 (56.2–75.4)	83.8 (76.7–89.4)	4.10 (2.75–6.10)	0.40 (0.30–0.53)
BME ≥2 quadrants OR fat lesion ≥2 quadrants	65.3 (55.2–74.5)	85.2 (78.3–90.6)	4.42 (2.90–6.72)	0.41 (0.31–0.54)
BME ≥3 quadrants OR fat lesion ≥1 quadrant	59.4 (49.2–69.1)	91.5 (85.7–95.6)	7.03 (4.00–12.37)	0.44 (0.35–0.56)
BME ≥3 quadrants OR fat lesion ≥2 quadrants	57.4 (47.2–67.2)	93.0 (87.4–96.6)	8.15 (4.38–15.17)	0.46 (0.36–0.58)
AxSpA-typical structural AND inflammatory MRI lesions (global assessment)	52.5 (42.3–62.5)	98.6 (95.0–99.8)	37.26 (9.29–149.37)	0.48 (0.39–0.59)
Active sacroiliitis (ASAS definition) OR structural lesions (Leiden definition)	53.5 (43.3–63.5)	96.5 (92.0–98.8)	15.18 (6.30–36.61)	0.48 (0.39–0.60)
Active sacroiliitis (ASAS MRI Group definition) OR structural lesions (Leiden definition)	63.4 (53.2–72.7)	90.8 (84.9–95.0)	6.92 (4.04–11.86)	0.40 (0.31–0.52)
Active sacroiliitis (ASAS definition) OR structural lesions (ASAS MRI Group definition)	54.5 (44.2–64.4)	95.1 (90.1–98.0)	11.05 (5.25–23.25)	0.48 (0.39–0.59)
Active sacroiliitis (ASAS MRI Group definition) OR structural lesions (ASAS MRI Group definition)	66.3 (56.2–75.4)	89.4 (83.2–94.0)	6.28 (3.82–10.34)	0.38 (0.28–0.50)
BME in ≥ 2 SIJ quadrants OR in ≥ 3 consecutive slices OR Leiden definition of structural changes	68.3 (58.3–77.2)	85.2 (78.3–90.6)	4.62 (3.05–7.01)	0.37 (0.28–0.50)
BME in ≥ 3 SIJ quadrants OR in ≥ 3 consecutive slices OR Leiden definition of structural changes	65.3 (55.2–74.5)	90.8 (84.9–95.0)	7.14 (4.17–12.21)	0.38 (0.29–0.50)
ASAS definition of active sacroiliitis OR Leiden definition of structural changes OR backfill OR ankylosis/bone bud	56.4 (46.2–66.3)	96.5 (92.0–98.8)	16.03 (6.66–38.56)	0.45 (0.36–0.57)
ASAS definition of active sacroiliitis OR ASAS MRI Group definition of chronic changes OR backfill OR ankylosis/bone bud	57.4 (47.2–67.2)	95.1 (90.1–98.0)	11.65 (5.55–24.45)	0.45 (0.36–0.56)
ASAS MRI Group definition of active sacroiliitis OR chronic changes OR backfill OR ankylosis/bone bud	69.3 (59.3–78.1)	89.4 (83.2–94.0)	6.56 (4.00–10.77)	0.34 (0.25–0.46)
BME in ≥ 3 consecutive slices OR ≥ 2 of 3 lesions (BME, erosions, fat lesions) each present in ≥ 3 SIJ quadrants OR Ankylosis ≥ 3 SIJ halves	60.4 (50.2–70.0)	93.0 (87.4–96.6)	8.58 (4.62–15.91)	0.43 (0.33–0.54)

BME: bone marrow edema; LR+ : positive likelihood ratio; LR– : negative likelihood ratio; Sn: Sensitivity (Sn); Sp: Specificity; ASAS definition of active sacroiliitis: BME in 2 consecutive slices or 2 lesions on one slice, highly suggestive of axSpA; ASAS MRI group definitions for active sacroiliitis: BME in ≥4 SIJ quadrants in any location or BME in the same location in ≥3 consecutive slices; Leiden definition of structural lesions: ≥3 erosions, ≥3 fat lesions or ≥5 fat lesions and/or erosions; ASAS MRI group definition of structural lesions: erosions in ≥3 quadrants or erosion at the same location in ≥2 consecutive slices, or fat lesions in ≥5 SIJ quadrants or fat lesions in the same location in ≥3 consecutive slices; in blue – candidate lesions added after working group discussion.

O10

IMPACT OF A TRAINING PROGRAM ON THE DIAGNOSTIC SUSPICION OF SPONDYLOARTHRITIS AND PSORIATIC ARTHRITIS AMONG SPANISH A&E MEDICAL STAFF: THE URGESPAR EXPERIENCE

Guillén-Astete C.A.[1], Ahijón-Lana M.[2], Valero-Expósito M.[1], Nieto-González J.C.[3], Castrejón I.[3], Lois-Iglesias A.[4], González-Martín J.J.[5], Penedo-Alonso R.[6], Domínguez J.[1], Vázquez M.[1]

[1]Rheumatology Dept., Ramón y Cajal University Hospital, Madrid; [2]Rheumatology Dept., La Princesa University Hospital IIS-Princesa, Madrid; [3]Rheumatology Dept., Gregorio Marañón University Hospital, Madrid; [4]Rheumatology Dept., Complejo Hospitalario Universitario de A Coruña, A Coruña; [5]Rheumatology Dept., HM Sanchinarro University Hospital, Madrid; [6]A&E Dept. Ramón y Cajal University Hospital, Madrid, Spain

Introduction. Around 30% of patients who are eventually diagnosed with a form of spondyloarthritis in Spain have visited a hospital A&E Dept. due to the very symptoms that ultimately led to their diagnosis (1). The aim of this study is to conduct a training programme for A&E doctors (URGESPAR) and assess its impact on patient referrals to rheumatology Departments.

Methods. A panel of experts in spondyloarthritis, advised by an emergency physician with extensive teaching experience, developed a 12-hour online training programme aimed at emergency physicians. Twenty-two hospital A&E Departments, representative of the whole country, were selected. URGESPAR was promoted through the Spanish Society of Emergency and Urgent Medicine (SEMES), and the heads of each A&E Dept. were contacted to ensure that over 50% of the permanent staff in each unit participated. The results of a diagnostic skills test were measured before and after the training, as were the referral frequencies to the corresponding rheumatology Departments before and 4 months after UGESPAR.

Results. Six hundred and ten doctors took part in URGESPAR, of whom 104 were permanent staff at the selected A&E Departments (mean age 40.5 years). The mean (SD) number of patients seen per month for non-traumatic musculoskeletal conditions was 57.4(11.3). Self-assessment of diagnostic ability for spondyloarthritis and psoriatic arthritis (scale from 1-10) increased from 4.7 to 7.2 and from 4.5 to 7.1, respectively, following the training ($p<0.01$). The failure to recognise risk factors fell from 68% to 40% and from 60% to 38%, respectively. The mean number of quarterly referrals increased from 3.4(1.2) to 7.6(3.1) and from 2.9(2.2) to 6.9(2.3) patients, respectively.

Conclusion. The URGESPAR training initiative has directly improved the diagnostic skills of participating A&E doctors and increased the frequency of referrals of suspected cases to the corresponding rheumatology Departments. We believe that focusing on this group of doctors will help to address the problem of delayed diagnosis in spondyloarthritis.

Acknowledgements. Sociedad de Reumatología de la Comunidad de Madrid, Sociedad Española de Medicina de Urgencias y Emergencias, Abbvie.

Reference

1. GUILLEN-ASTETE CA, GAITE-GONZALEZ I, ZURITA-PRADA PA, URREGO C: Delay and diagnostic pathway of patients with psoriatic arthritis in Spain. *Reumatol Clin* 2021; 17(9): 525-29.

O11

A RARE VARIANT IN THE *EXTL3* GENE IS ASSOCIATED WITH ALTERED WNT SIGNALING PATHWAY IN A NOVEL MOUSE MODEL OF AXIAL SPONDYLOARTHRITIS

Hilliquin S.[1,2,3], Ibrahim K.[1], Fogel O.[2], Do Cruzeiro M.[4], Albanese P.[5], Houard X.[6], Bardet C.[1], Miceli-Richard C.[2,3]

[1]Université Paris Cité, Institut des Maladies Musculo-squelettiques, Laboratory Orofacial Pathologies, Imaging and Biotherapies URP2496 and FHU-DDS-Net, Dental School, and Plateforme d'Imagerie du Vivant (PIV), Montrouge; [2]Dépt. de Rhumatologie, Hôpital Cochin, Assistance publique des Hôpitaux de Paris, Université Paris-Cité, Paris; [3]Institut Pasteur, Université Paris Cité, Immunoregulation Unit, Paris; [4]Plateformes PDA et MouseT'IC, Institut Cochin, Université Paris Cité, Paris; [5]Laboratoire Gly-CREET, Université Paris-Est Créteil Val de Marne (UPEC) Faculté des sciences et technologies; [6]Centre de Recherche Saint-Antoine, UMR_S938 INSERM /Sorbonne Université, Paris, France

**Co-first authors*

Background. Ossification in ankylosing spondylitis (AS) remains poorly understood. Human and animal studies implicate dysregulated Wnt signaling in the excessive endochondral ossification characteristic of AS. Using linkage analysis, we identified a rare *EXTL3* variant in a family with severe familial AS. *EXTL3* belongs to the exostosin family and is required for heparan sulfate biosynthesis. Extl3$^{mut/+}$ mice develop osteoarticular lesions of the sacroiliac joint (SIJ) resembling axial spondyloarthritis, together with altered trabecular bone structure in the spine and SIJ, accelerated chondrocyte differentiation, disrupted cartilage architecture, and impaired osteoblast differentiation and mineralization.

Objective. To investigate Wnt signaling abnormalities and the effects of Wnt modulation in this mouse model.

Methods. Tcf1 immunohistochemistry was performed on SIJs from 6-month-old Extl3$^{mut/+}$ and wild-type littermates. Primary osteoblast and chondrocyte cultures were generated at different differentiation stages. RNA sequencing, quantitative PCR, and Western blot analyses were conducted at multiple time points. To assess Wnt pathway involvement, three inhibitors targeting canonical and non-canonical pathways (Dkk-1, sFRP2, and PKF118-310) were applied to osteoblast and chondrocyte cultures, followed by repeated molecular analyses.
Results. Tcf1 staining was markedly increased in chondrocyte columns and subchondral bone from Extl3$^{mut/+}$ SIJs. RNA sequencing showed reduced expression of Wnt inhibitors Dkk-1 and sclerostin at late stages of osteoblast and chondrocyte differentiation. Quantitative PCR demonstrated overexpression of β-catenin, Tcf7, and Wnt ligands including Wnt3, Wnt4, Wnt5a, and Wnt5b, supporting enhanced Wnt activation and accelerated chondrogenesis. Western blot confirmed Tcf1 overexpression in mutant osteoblasts at terminal differentiation. Wnt inhibition, particularly with Dkk-1, partially reversed delayed osteoblast differentiation and accelerated chondrocyte maturation.
Conclusion. *EXTL3* mutation induces abnormal Wnt activation in osteoblasts and chondrocytes, supporting a central role for Wnt signaling in pathological ossification in AS and identifying potential therapeutic targets. These results suggest that it would be of interest to treat Extl3$^{mut/+}$ mice with a Wnt inhibitor *in vivo* in order to validate its protective effect on the osteoarticular SIJ abnormalities observed on microCT.

O12

MULTIOMIC AND KINETIC MAPPING IDENTIFIES CIRCULATING MONOCYTES COMMITTED TO OSTEOCLAST FATE AND SYNOVIAL TISSUE HOMING

Hutton J.[1,2,3,§], Saha A.[1], Li Q.[1], Farahi N.[3], Leek F.[4], McDonnell S.[5], Ade-Ojo B.[4], Gillett D.[4], Shenker N.[2], Jadon D.[2], Laddach A.[6], Summers C.[3,§], McGovern N.[1,§]
[1]Dept. of Pathology, University of Cambridge; [2]Dept. of Rheumatology, Cambridge University Hospitals NHS Foundation Trust, Cambridge; [3]Victor Phillip Dahdaleh Heart & Lung Research Institute, Cambridge Biomedical Campus, University of Cambridge; [4]Dept. of Nuclear Medicine, Cambridge University Hospitals NHS Foundation Trust, Cambridge; [5]Dept. of Orthopaedics, Cambridge University Hospitals NHS Foundation Trust, Cambridge; [6]The Francis Crick Institute, London, UK

Introduction. Psoriatic arthritis (PsA) is a heterogeneous immune-mediated disease where osteoclasts drive bone destruction. Osteoclasts derive from monocyte precursors, yet their circulating identity remains unclear. Abnormal leukocyte infiltration into synovium is a PsA hallmark, but the mechanisms remain poorly understood.
Methods. To investigate tissue homing, CD14^{+} monocytes were Tc99m-labelled, reinfused into 2 healthy controls (HC) and 3 active PsA patients, and tracked by SPECT imaging. PBMCs were collected from treatment-naïve PsA, psoriasis without arthritis (PsO), and HC. Monocyte subsets from PsA (n=5) and HC (n=4) were profiled by bulk RNA-seq. Gene enrichment guided development of a myeloid mass cytometry panel, applied to PBMCs from PsA (n=10), PsO (n=10), and HC (n=10). A pre-osteoclast (preOC) gating strategy was established; preOCs and monocytes were isolated for scRNAseq from PsA (n=4) and HC (n=2). PreOC identity was validated via *in vitro* differentiation datasets and functional osteoclastogenesis assays. PreOCs were mapped in synovial scRNAseq datasets (PsA, undifferentiated arthritis, RA, OA, HC) and spatial sequencing of PsA synovium. Cells were fate-mapped from blood to tissue using Xenium spatial sequencing and imaging mass cytometry.
Results. Tc99m-tracking revealed selective monocyte recruitment to inflamed PsA joints. A distinct preOC state was identified, derived from classical monocytes but enriched for *NFATC1*, transcriptionally distinct from monocytes, and resembling *in vitro* monocyte-osteoclast intermediates. PreOCs generated multinucleated osteoclasts *in vitro* without exogenous RANKL. Circulating preOCs were expanded in PsA versus PsO/HC and enriched for synovial homing signatures. scRNAseq confirmed tissue preOC expansion in PsA, and labelled preOCs demonstrated preferential synovial recruitment.
Conclusion. These findings provide mechanistic insight into how abnormal preOC homing fuels PsA structural damage, highlight systemic immune priming in disease pathophysiology, and reveal a novel cellular therapeutic target at the bone-joint-immune interface.
Acknowledgements. This work is supported by the Medical Research Council (Clinical Research Training fellowship award to JPH) and Addenbrookes Charitable Trust and Evelyn Trust awards. NMcG is funded by the Wellcome Trust and Royal Society (G112870) and ERC award funded by UKRI (G122519). This work was also supported by the UKRI-Medical Research Council (MR/S035753/1) and the NIHR Cambridge Biomedical Research Centre (BRC-1215-20014). We also acknowledge the support of the National Institute for Health and Care Research Clinical Research Network (NIHR CRN). C.S. was funded by the UKRI-Medical Research Council (MR/P502091/1 and MR/X005070/1) and the National Institute for Health and Care Research (NIHR133788). This research was also supported by the NIHR Cambridge Biomedical Research Centre (NIHR203312). The views expressed are those of the authors and not necessarily those of the MRC, the Addenbrookes Charitable Trust, the Evelyn Trust, NIHR or the Dept. of Health and Social Care.

O13

EXTL3 MUTATION PROMOTES PATHOLOGICAL OSSIFICATION IN A CHRONIC INFLAMMATORY CONTEXT IN ANKYLOSING SPONDYLITIS

Calot A.[1], Al Monla R.[1], Casel B.[1], Garteiser P.[2], Doblas S.[2], Bardet C.[1], Miceli-Richard C.[1], Hilliquin S.[1]
[1]Université Paris Cité and Sorbonne Paris Nord, Inserm UMR_S1333 Oral Health, Montrouge; [2]Imaging Biomarkers Laboratory, Inflammation Research Center, Inserm UMR1149, Université Paris Cité, Paris, France

Spondyloarthritis is a chronic inflammatory rheumatic disease characterized, in severe forms, by spinal ankylosis associated with pathological ossification. This pathological ossification, linked to increased endochondral ossification, may represent a secondary reparative response to chronic inflammation, as suggested by several studies (Sieper *et al*., *Arthritis & Rheumatology*, 2008). However, the mechanisms leading to new bone formation remain poorly understood.
Whole-exome sequencing in a family with severe familial ankylosing spondylitis identified a rare missense variant in EXTL3 (c.803C>T, p.Thr268Met), a gene encoding a glycosyltransferase involved in heparan sulfate chain elongation. While the heterozygous Extl3$^{mut/+}$ mouse model alone recapitulates early sacroiliac and vertebral lesions, it does not reproduce the severe ankylosis observed in humans, suggesting that genetic predisposition alone is insufficient to induce a full pathological ossification phenotype.
We hypothesized that pathological ossification results from the interaction between this genetic susceptibility and a chronic inflammatory environment. To test this hypothesis, we developed a murine model combining the *Extl3* mutation with overexpression of transmembrane TNF (TNFtm TgA86). Longitudinal phenotypic characterization was performed at 1, 2, and 3 months in four genotype groups (Wt, Extl3$^{mut/+}$, TNFtm, Extl3$^{mut/+}$-TNFtm) using micro-CT of the sacroiliac joints and spine, and a novel preclinical MRI protocol developed for this study, including MGE sequences (T1 equivalent) and MSME sequences (T2 equivalent).
Extl3$^{mut/+}$-TNFtm mice rapidly developed a severe phenotype characterized by tail deformity, peripheral arthritis, and early lameness. MRI detected signal abnormalities as early as 1 month, with hypointensity at the sacroiliac joints consistent with early inflammatory infiltration and corresponding hyperintensity on MGE sequences, evolving toward a hyperintensity pattern with increased signal heterogeneity at 2 months, suggesting active bone remodeling. Micro-CT revealed progressive alteration of trabecular bone microarchitecture in Extl3$^{mut/+}$-TNFtm group.
These preliminaries results suggest that the EXTL3 mutation potentiates osteoarticular damage in an inflammatory context, supporting the hypothesis that genetic predisposition and chronic inflammation cooperate in the pathogenesis of ankylosing spondylitis. The multimodal imaging approach combining MRI and micro-CT represents a particularly innovative aspect of this work and provides new insights into the mechanisms underlying pathological ossification, opening new therapeutic perspective.

Poster Presentations

P1

INTERMITTENT FASTING ATTENUATES JOINT INFLAMMATION AND REMODELING IN EXPERIMENTAL SPONDYLOARTHRITIS ALONGSIDE METABOLIC TRANSCRIPTIONAL REPROGRAMMING

González-Chávez S.A.[1], Cuevas-Martínez R.[1], Salas-Leiva J.S.[2], Pacheco-Tena C.[1]
[1]PABIOM Laboratory, Faculty of Medicine and Biomedical Sciences, Autonomous University of Chihuahua, Chihuahua; [2]The Center for Research in Advanced Materials, Chihuahua, Mexico

Introduction. Dietary strategies that modulate inflammation and the gut microbiota are of growing interest in immune-mediated diseases; however, the effects of intermittent fasting in spondyloarthritis (SpA) remain poorly defined. We evaluated whether intermittent fasting attenuates joint inflammation and pathological bone/cartilage remodeling in experimental SpA and explored associated changes in joint RNA expression and fecal microbiota.
Methods. Sixteen male DBA/1 mice with spontaneous SpA were assigned to control or intermittent fasting for 4 weeks (n=8/group). Tarsal joints were evaluated histologically. Joint RNA expression changes were assessed by DNA microarray and functional enrichment analysis. Fecal microbiota was analyzed by 16S rRNA sequencing.
Results. Intermittent fasting was associated with lower histological disease severity, including reduced inflammatory infiltrate, enthesitis, cartilage damage, bone erosion, chondral and osseous neoformation, and joint space narrowing (Fig. 1). Transcriptomic analysis identified 151 upregulated and 516 downregulated annotated genes in fasting mice. At the RNA expression level, downregulated genes were mainly enriched in metabolic, bioenergetic, and structural/contractile terms, including oxidative energy metabolism, precursor metabolite generation, cellular respiration, TCA cycle/respiratory electron transport, carbon metabolism, pyruvate metabolic process, muscle system process, and ion homeostasis (Fig. 2A). This profile was not dominated by canonical interleukin or osteogenic pathway signatures. In parallel, fecal microbiota analysis suggested a selective rather than global effect of fasting. Alpha diversity did not differ between groups, and Bray-Curtis PCoA showed no significant global separation. However, genus-level differential abundance showed enrichment of *Lactobacillus* and *Alloprevotella*, with depletion of *Lachnospiraceae*_UCG_001 and Bacteroidia-related taxa in fasting mice (Fig. 2B).
Conclusion. Intermittent fasting was associated with reduced joint inflammation and pathological remodeling in experimental SpA. These changes were accompanied by RNA-expression changes mainly involving metabolic, bioenergetic and structural/contractile pathways, together with selective fecal microbiota shifts. These findings support further evaluation of fasting-responsive immunometabolic changes in the gut-joint axis of SpA.

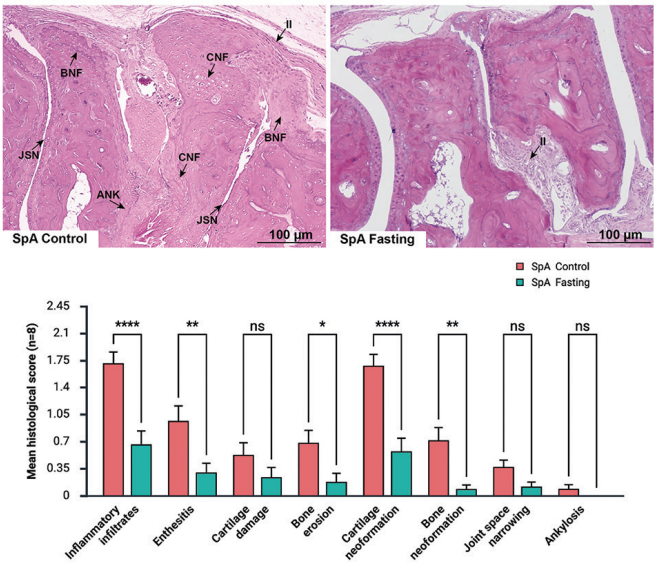


Fig. 1. Intermittent fasting reduces histological joint pathology in experimental spondyloarthritis.
Representative H&E-stained tarsal joint sections from SpA control and SpA fasting mice are shown. Histological assessment showed lower inflammatory infiltrates, enthesitis, cartilage damage, bone erosion, chondral neoformation, bone neoformation, and joint space narrowing in fasting mice. Bars represent the mean histological score; error bars indicate SEM.
BNF: bone neoformation; CNF: cartilage neoformation; JSN: joint space narrowing; ANK: ankylosis; II: inflammatory infiltrate; ns: not significant. *p<0.05, **p<0.01, ****p<0.0001.

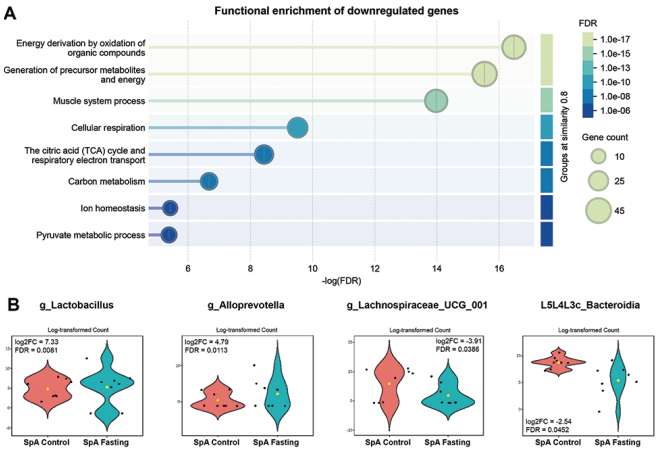


Fig. 2. Joint RNA-expression and fecal microbiota changes associated with intermittent fasting.
A. Functional enrichment of genes downregulated in tarsal joints from fasting mice, showing selected metabolic, bioenergetic, and structural/contractile terms. The x-axis represents –log(FDR), dot size represents gene count, and colour indicates FDR.
B. Genus-level fecal microbiota differences shown as log-transformed counts. Fasting mice showed enrichment of *Lactobacillus* and *Alloprevotella*, with lower abundance of *Lachnospiraceae_UCG_001* and Bacteroidia-related taxa.

P2

OSTEOCHONDRAL NEOFORMATION SITES DISPLAY INFLAMMATORY-OSTEOGENIC STROMAL ACTIVATION IN DBA/1 SPONTANEOUS ARTHRITIS

González-Chávez S.A., Moreno-Soltero M.F., Romero-Huerta R., Ortiz-Domínguez V.M., Sausameda-García J., Flores-Adame I.D., Arrieta-Martínez P.I., Ruizesparza-Hinojos D.A., Prieto-Carrasco R., Loya-Rivera M., Chaparro-Barrera E., Pacheco-Tena C.
PABIOM Laboratory, Faculty of Medicine and Biomedical Sciences, Autonomous University of Chihuahua, Chihuahua, Mexico

Introduction. Pathological new bone formation in spondyloarthritis (SpA) is usually interpreted through immune and osteogenic pathways, but the spatial and cellular organization of these signals within osteochondral neoformation sites remains insufficiently defined. We aimed to characterize whether pathological ossification in DBA/1 spontaneous arthritis (SpAD) is associated with tissue-specific inflammatory–osteogenic stromal activation.
Methods. Hind paws and spine from DBA/1 SpAD mice and controls were analyzed by transcriptomic enrichment and RT-qPCR. Histological sections were evaluated for ectopic bone/cartilage formation. Immunohistochemistry optical density was compared between normal bone and ectopic/new bone formation sites for IL-17, IL-23, OCN, OPN, OSX and PTH-rP. IL-17/IL-22 staining was assessed in chondro-osteogenic areas. Cultured fibroblast-like synoviocytes (FLS) from SpAD and BALB/c mice were analyzed by qPCR. Human tarsitis samples were evaluated for osteogenic remodeling markers.
Results. Hind paws and spine showed distinct over-regulated transcriptional programs. Hind paws were enriched in tissue remodeling, chondro-osteogenic differentiation, adhesion/cytoskeletal and MAPK/JAK-STAT pathways, whereas spine showed metabolic, organelle-stress, tight-junction, Wnt and Hippo-related signatures (Fig. 1A). RT-qPCR showed stronger inflammatory cytokine induction in hind paws than in spine (Fig. 1B). Histology identified ectopic bone formation, chondro-osseous remodeling, ankylosis and joint-space narrowing (Fig. 2A). Compared with normal bone, ec-

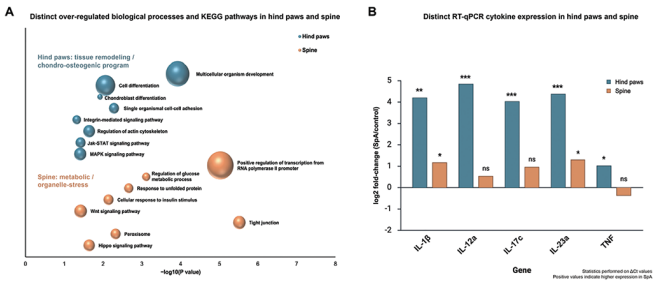


Fig. 1. Tissue-specific molecular and cytokine signatures in DBA/1 SpAD.
A. Bubble plot of selected over-regulated biological processes and KEGG pathways in hind paws and spine. Bubble size represents the number of genes associated with each term, and the x-axis indicates –log10 (*p*-value).
B. RT-qPCR analysis of inflammatory cytokines in hind paws and spine, shown as log2 fold-change relative to control tissues. Statistical comparisons were performed on ΔCt values.

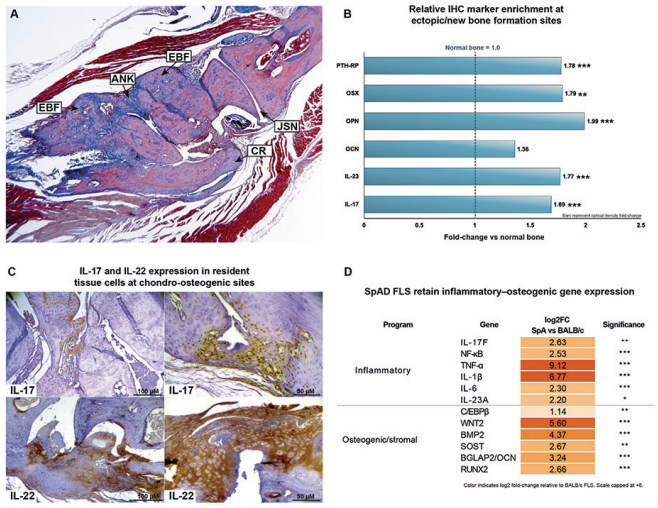


Fig. 2. Inflammatory-osteogenic stromal activation at osteochondral neoformation sites in DBA/1 SpAD.
A. Representative Masson's trichrome staining showing ectopic bone formation (EBF), chondro-osseous remodeling (CR), ankylosis (ANK), and joint-space narrowing (JSN).
B. Relative IHC marker enrichment in ectopic/new bone formation sites compared with normal bone.
C. Representative IL-17 and IL-22 staining in resident tissue cells within chondro-osteogenic regions.
D. qPCR heatmap showing inflammatory and osteogenic/stromal gene expression in cultured SpAD FLS compared with BALB/c FLS.

topic/new bone formation sites showed increased IL-17, IL-23, OPN, OSX and PTH-rP, while OCN showed a non-significant increase (Fig. 2B). IL-17 and IL-22 staining was observed in resident tissue cells within chondro-osteogenic regions (Fig. 2C). SpAD FLS retained increased inflammatory and osteogenic/stromal gene expression compared with BALB/c FLS (Fig. 2D). In human tarsitis, Wnt-2, β-catenin, BMP-2 and OCN were increased in entheses and/or bone matrix compared with healthy controls.
Conclusion. Pathological osteochondral neoformation in SpAD is associated with site-specific inflammatory–osteogenic stromal activation, suggesting that resident tissue cells and FLS participate in the abnormal remodeling microenvironment beyond inflammatory cell infiltration alone.

P3

INHIBITION OF MITOCHONDRIAL FISSION ATTENUATES INFLAMMATORY RESPONSE AND IMPROVES MITOCHONDRIAL FUNCTION IN FIBROBLAST-LIKE SYNOVIOCYTES OF SPONDYLOARTHRITIS

González-Chávez S.A.[1], Prieto-Carrasco R.[1], Loya-Rivera M.[1], Cuevas-López B.[2], García-Arroyo F.E.[2], Aparicio-Trejo O.A.[2], Pacheco-Tena C.[1]
[1]PABIOM Laboratory, Faculty of Medicine and Biomedical Sciences, Autonomous University of Chihuahua, Chihuahua; [2]Dept. of Cardio-Renal Pathophysiology, National Institute of Cardiology "Ignacio Chávez", Mexico City, Mexico

Introduction. Mitochondrial dynamics are essential for maintaining cellular homeostasis. We previously demonstrated that spondyloarthritis (SpA) is associated with mitochondrial dysfunction, characterized by increased mitochondrial fission, impaired mitochondrial turnover, oxidative stress, and reduced ATP production (1). Therefore, this study investigated whether inhibition of Drp1 mediated mitochondrial fission could restore mitochondrial homeostasis and attenuate inflammatory response in fibroblast-like synoviocytes (FLS) from a murine model of SpA.
Methods. FLS isolated from the joints of mice with established SpA were treated with the Drp1 inhibitor Mdivi-1. Cell viability, gene and protein expression of mitochondrial dynamics, mitochondrial turnover, and inflammatory markers were evaluated.
Results. Drp1 inhibition increased cell viability and promoted mitochondrial homeostasis by upregulating the mitochondrial fusion marker *MFN2* and mitochondrial biogenesis marker *PPARGC1A* (PGC-1α), while downregulating *DNM1L* (Drp1) and the mitophagy regulator *PINK1* (Fig. 1). These changes were accompanied by increased protein expression of Mfn1/2, PGC-1α, and VDAC, a marker of mitochondrial mass, together with reduced expression of Drp1, Fis1, and Pink1 (Fig. 2). Furthermore, mitochondrial fission inhibition attenuated the inflammatory response, as evidenced by decreased gene and protein expression of the pro-inflammatory cytokines IL-6, TNF-α and IL-1β (Fig. 1 and 2).
Discussion. The observed shift toward mitochondrial fusion and biogenesis following Drp1 inhibition was associated with reduced mitophagy and at-

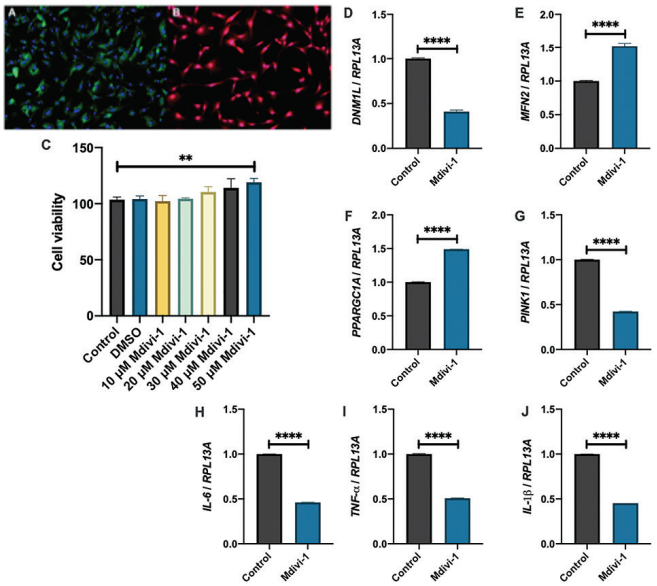


Fig. 1. Characterization of fibroblast-like synoviocytes (FLS) and effects of Drp1 inhibition on mitochondrial dynamics, mitochondrial turnover, and inflammatory geneexpression.
A. Representative immunofluorescence staining of Cadherin-11 (Cdh11, green) with DAPI nuclear counterstaining (blue).
B. Representative immunofluorescence staining of the fibroblast marker ER-TR7 (red) with DAPI nuclear counterstaining (blue).
C. Cell viability assessed by MTT assay in FLS treated with Mdivi-1 compared with untreated controls.
D-J. Relative mRNA expression of genes involved in mitochondrial dynamics and turnover, including *DNM1L* (**D**), *MFN2* (**E**), *PPARGC1A* (**F**), and *PINK1* (**G**), as well as the pro-inflammatory cytokines *IL-6* (**H**), *TNF*-α (**I**), and *IL-1*β (**J**), determined by RT-qPCR.
Data are presented as mean ± SEM from at least two independent experiments. Statistical significance was determined using unpaired Student's t-test (**p*<0.05, ***p*<0.01, ****p*<0.001, *****p*<0.0001).

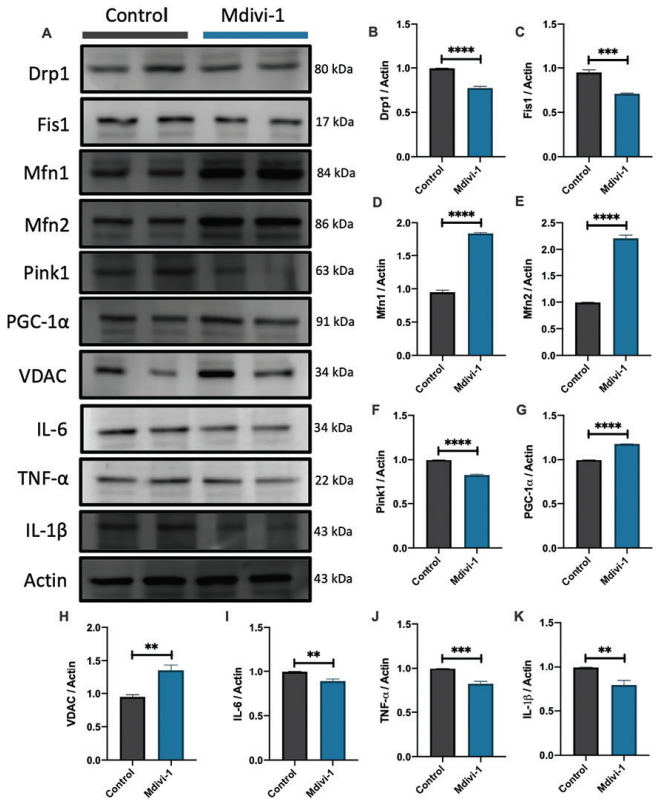


Fig. 2. Effects of Drp1 inhibition on mitochondrial dynamics, mitochondrial turnover, and inflammatory protein expression in fibroblast-like synoviocytes (FLS).
(**A**) Representative Western blots. (**B-K**) Quantification of protein expression levels of Drp1 (**B**), Fis1 (**C**), Mfn1 (**D**), Mfn2 (**E**), Pink1 (**F**), PGC-1α (**G**), VDAC (**H**), IL-6 (**I**), TNF-α (**J**), and IL1β (**K**), normalized to actin. Data are presented as mean ± SEM from at least two independent experiments. Statistical significance was determined using unpaired Student's t-test (*p<0.05, **p<0.01, ***p<0.001, ****p<0.0001.

tenuation of inflammatory cytokine expression, suggesting that mitochondrial dynamics directly influence inflammatory response in FLS. These findings indicate that Drp driven mitochondrial fragmentation may contribute to the pathogenic phenotype of FLS in SpA, highlighting mitochondrial fission as a promising therapeutic target.

Conclusion. Our findings demonstrate that Drp1 inhibition restores mitochondrial homeostasis by promoting mitochondrial fusion and biogenesis while attenuating inflammatory signaling in FLS. These results highlight the central role of mitochondrial dynamics in the pathogenesis of SpA and identify mitochondrial fission as a promising therapeutic target for controlling synovial inflammation.

Reference

1. PRIETO-CARRASCO R, GONZÁLEZ-CHÁVEZ SA, CHAPARRO-BARRERA E *et al*.: Mitochondrial dysfunction drives oxidative stress and energy imbalance in a murine model of spondyloarthritis. *Cell Biochem Funct*. 2025; 43(12): e70151. doi: 10.1002/cbf.70151. PMID: 41410024.

P4

SPONDYLOARTHRITIS PROMOTES CARDIOVASCULAR INFLAMMATION THROUGH NON-CANONICAL WNT SIGNALING IN AN HLA-B27 TRANSGENIC RAT MODEL

Kwon S.R., Kim C.R., Lim M.J., Song H.S., Yoon J.Y.
Division of Rheumatology, Dept. of Internal Medicine, College of Medicine, Inha University, Incheon, Republic of Korea

Introduction. Research on spondyloarthritis (SpA) has primarily focused on axial SpA, cardiovascular (CV) inflammation, pro-inflammatory cytokines (such as TNF-α, IL-17, and IL-23), and Wnt signaling pathways. However, these studies often focused on individual components, resulting in a lack of comprehensive approaches. Furthermore, no experimental studies have thoroughly demonstrated the interactions between these components. The present study utilized an HLA-B27 transgenic rat (TG) model, which closely resembles human SpA, to investigate the associations between SpA, CV inflammation, cytokines, and Wnt signaling.
Materials and methods. Six-week-old HLA-B27 TG F1 rats were injected with heat-inactivated *Mycobacterium tuberculosis*, whereas Lewis control rats received phosphate-buffered saline. One week after injection, TG (F1 G0) and control (Ctrl G0) rats were euthanized before the onset of clinical arthritis. When TG rats reached an arthritis score of grade 6 (F1 G6), they were euthanized together with age-matched control rats (Ctrl G6). Aortic expression of inflammatory and Wnt signaling proteins was analyzed, and tail spine samples were histologically evaluated for inflammation, bone destruction, and osteoproliferation.
Results. All F1 G6 rats developed clinical arthritis, and 60% exhibited spondylitis. A significant correlation was observed between spinal inflammation and osteoproliferation (r^2=0.7992, p=0.041). In the aortas of F1 G6 rats, expression of WNT5 and RUNX2, key mediators of non-canonical Wnt signaling, as well as TNF-α and IL-17, was significantly increased compared with controls. Furthermore, WNT3, IL-17, and IL-23 levels were elevated in F1 G6 rats compared with F1 G0 rats, indicating progression of CV inflammation. Notably, IL-17 and IL-23 were already elevated in F1 G0 rats compared with Ctrl G0 rats, suggesting early vascular immune activation before arthritis onset.
Conclusion. SpA induces CV alterations through inflammatory and Wnt signaling pathways, particularly non-canonical Wnt signaling. These pathways may represent therapeutic targets for preventing CV complications in SpA.
Acknowledgements. This work was supported by the National Research Foundation of Korea (NRF) grant funded by the Korea government (MSIT) (NRF-2020R1A2C1101992). The authors thank Dr Joel D. Taurog and the Rat Resource and Research Center for providing the HLA-B27 TG rats.

P5

ABSENCE OF SEX DIFFERENCES AND ANTI-IL-17 RESPONSE IN A MOUSE MODEL OF ENTHESITIS AND PAIN

Vegt D.E.[1], Blaney Davidson E.N.[1], Blom A.B.[1], Braunhofer L.[1], Leijten E.F.A.[1,2], van der Horst-Bruinsma I.E.[1], Koenders M.I.[1]
[1]Dept. of Rheumatology, Radboud University Medical Centre, Nijmegen; [2]Dept. of Rheumatology, Sint-Maartenskliniek, Nijmegen, The Netherlands

Introduction. The IL-17 pathway is critical in the pathogenesis of spondyloarthritis (SpA). Intriguingly, men might have better responses to anti-IL-17 treatment than women (1, 2). To untangle whether sex-specific anti-IL-17 therapy responses have its roots in altered inflammatory or pain responses, we assessed anti-IL-17 therapy responses in a mouse model with unilateral enthesitis and investigated sex-related differences in pain and inflammation.
Methods. Unilateral enthesitis was induced in male and female mice by injection of collagenase into the right knee joint. Animals were treated with anti-IL-17 antibodies or isotype control. Gait and mechanical pain were measured by Catwalk and Pressure Application Measurement (PAM) throughout the experiment. Mice were sacrificed at days 4 or 56 for histology of the enthesis and subsequent enthesophyte formation. We isolated the dorsal root ganglia (DRG) for single cell RNA sequencing (scRNAseq).
Results. Enthesitis on day 4 (including NIMP.R14 expression on histology (Fig. 1A)) and subsequent enthesophyte formation on day 56 was equal be-

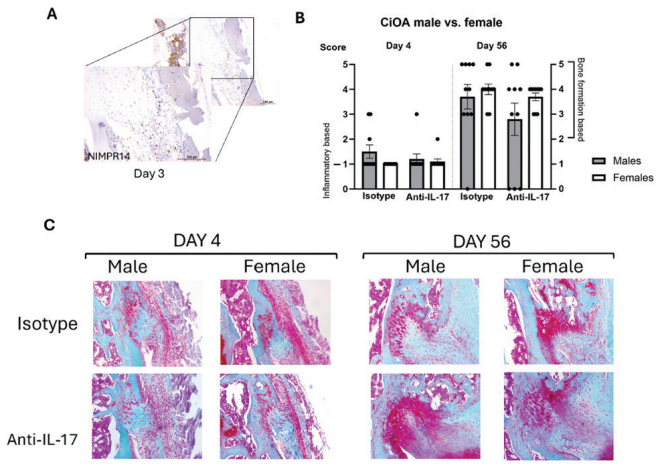


Fig. 1. Enthesitis presents similarly in male and female mice.
A. Presence of neutrophils in the collateral enthesis by NIMP.R14 staining using immunohistochemistry.
B. SafO/Fast Green stained sections were used to evaluate the enthesis of the collateral ligament of the right knee. Enthesitis on day 4 and day 56 showed no sex-differences in both enthesitis (score 1) and endochondral bone formation (score 2-5).
C. Representative histological imaging of enthesitis in male/female mice with anti-IL-17 treatment or isotype control on days 4 and 56 with[a] SafO/Fast Green staining.

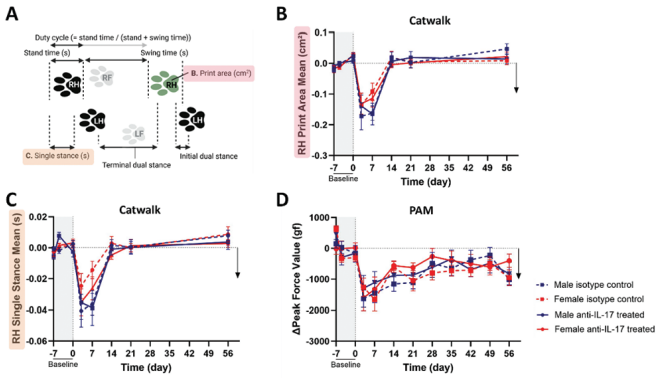


Fig. 2. Selection of gait analyses (**A**,**B**,**C**) and PAM (**D**) showed no differences between anti-IL-17-treated and isotype control mice. Gait was analysed using CatWalk XT with baseline measures on days –7, -5 and 0. A) CatWalk XT parameter overview, B) RH print area, C) RH single stance and D) Pressure tolerance of the affected right knee, measured with pain application measurement. All values are corrected for individual baseline and expressed as mean with SEM of n=10/group. Arrows indicate the direction of change in value as a proxy of pain. Significance was tested using Two-way ANOVA with multiple comparisons. # significant difference anti-IL-17-treated and isotype control.
RH: right hind; LH: left hind; gf: gram-force; g: gram; WT: Wild type; SEM: standard error of the mean; s: second.

tween male and female mice and unaffected by blocking of IL-17 (Fig. 1B). Pain clearly increased during early inflammation, but were not significantly different between sexes. IL-17 blockage did not alter pain, in either sex (Fig. 2). We previously showed that pain-associated expression patterns differed between sexes. Therefore, we analysed DRG gene profiling in male versus female mice without IL-17 blocking. Rough preliminary screening did not reveal differences in immune cell numbers, but gene expression patterns of immune cells themselves were different between sexes.

Conclusion. We demonstrated that enthesitis and pain were not different between male and female mice in a mouse model induced by collagenase-injection leading to SpA enthesitis. Moreover, given sex-disparities in IL-17 treatment responses in SpA patients, we set out to investigate whether anti-IL-17 treatment changed enthesitis and/or pain. However, after anti-IL-17 treatment, pathology and pain were not differentially affected between sexes.

Acknowledgements. This research was conducted with the support of Eli Lilly (IIR-75361), and our laboratory technicians M.A. Helsen, E.L. Vitters, B. Walgreen and H. van Beuningen.

References

1. VAN DER HORST-BRUINSMA IE, DE VLAM K, WALSH JA *et al*.: Baseline characteristics and treatment response to ixekizumab categorised by sex in radiographic and non-radiographic axial spondylarthritis through 52 weeks: data from three phase III randomised controlled trials. *Adv Ther* 2022; 39(6): 2806-19.
2. MOLICA COLELLA F, ZIZZO G, PARRINO V *et al*.: Effectiveness and safety of secukinumab in ankylosing spondylitis and psoriatic arthritis: a 52-week real-life study in an Italian cohort. A*dv Rheumatol* 2023; 63: 15. doi: 10.1186/s42358-023-00295-2.

P6

EFFECT OF PLASMA FROM PATIENTS WITH SPONDYLOARTHRITIS ON IL-22 RECEPTOR EXPRESSION AND EXTRACELLULAR MATRIX FORMATION IN HFOB 1.19 OSTEOBLASTS

Cerón-Cruz E.[1,2], Ortega-Mejia I.I.[2,3], Domínguez-López M.L.[3], Medina-Reyes I.[4], Chirino-López Y.I.[4], Reyes-Maldonado E.[1], Sánchez-Pérez S.[2,3], García-Hernández A.L.[6], Casasola-Vargas J.C.[7], Romero-López J.P.[1,2]
[1]Postgraduate Studies in Biological Sciences, Postgraduate Unit, University City, Mexico City; [2]Immunopathogenesis laboratory 4 B, Facultad de Estudios Superiores Iztacala, UNAM, Tlalnepantla de Baz, State of Mexico; [3]Laboratory of Immunochemistry 1, Escuela Nacional de Ciencias Biológicas IPN, Mexico City; [4]UBIMED, Facultad de Estudios Superiores Iztacala, UNAM, Tlalnepantla de Baz, State of Mexico; [5]Laboratory of Morphophysiology, Escuela Nacional de Ciencias Biológicas IPN, Mexico City; [6]Laboratory of Dental Research, CUSI Almaraz, Facultad de Estudios Superiores Iztacala, UNAM, Tlalnepantla de Baz, State of Mexico; [7]Rheumatology, General Hospital of Mexico Dr. Eduardo Liceaga, Mexico City, Mexico

Introduction. Spondyloarthritis (SpA) is a heterogeneous group of chronic, inflammatory, immune-mediated diseases that affect joints and entheses. Its multifactorial pathogenesis involves complex interactions among genetic and environmental factors, immune dysregulation, alterations in the gut microbiota, and biomechanical stress responses. The IL-23/IL-17 and IL-22 axis has been proposed as a key pathway linking enthesitis to the ectopic new bone formation characteristic of SpA. IL-22 is a pleiotropic cytokine that has recently gained attention for its capacity to modulate osteogenesis in SpA. Therefore, this study assessed the effect of plasma from patients with SpA, a highly pro-inflammatory stimulus, on IL-22 receptor (IL-22R) expression and mineralized extracellular matrix formation in the human osteoblast cell line hFOB 1.19.
Methods. A cross-sectional study was conducted, including 8 patients with SpA. Clinical variables were recorded, and plasma was obtained from peripheral blood. The hFOB 1.19 osteoblasts were stimulated with SpA patient plasma diluted at 5% and 10%. IL-22R expression was assessed after 8 hours using secondary immunofluorescence, and mineralized matrix formation was evaluated after 7 days of stimulation using alizarin red staining and quantification. Cell morphology and viability were assessed by optical microscopy. Results were compared with healthy-subjects plasma and correlated with disease activity scores.
Results. We found that the SpA plasma significantly reduced osteoblast viability compared to controls (p<0.0001). IL-22R expression was markedly increased in osteoblasts stimulated with SpA plasma (p<0.0001), accompanied by receptor internalization into the nucleus. Furthermore, patient plasma enhanced mineralized matrix formation (p<0.0001), evidenced by increased calcium deposits and a morphology consistent with differentiated osteoblasts.
Conclusion. Plasma from SpA patients contains pro-inflammatory factors that influence the viability, morphology, and function of hFOB 1.19 osteoblasts, as well as their IL-22R expression and potential signaling via non-canonical pathways, mechanisms relevant to pathological bone formation in SpA.

P7

EPIGENETIC LANDSCAPE OF OSTEOPROGENITORS IN ANKYLOSING SPONDYLITIS: FOXF2 METHYLATION AS A KEY DETERMINANT OF PATHOLOGICAL BONE FORMATION

Jo S.[1], Wang S.E.[2], Kim T.H.[3]
[1]Soonchunhyang University, Asan; [2]College of Medicine, Hallym University, Chuncheon; [3]Hanyang University Hospital for Rheumatic Diseases, Republic of Korea

Introduction. Ankylosing spondylitis (AS) is characterized by pathological bone formation. While abnormal differentiation of osteoprogenitors is central to AS pathogenesis, the underlying epigenetic mechanisms remain largely elusive. This study aimed to identify specific DNA methylation patterns in AS-derived osteoprogenitors to uncover novel regulatory factors.
Materials and methods. We isolated osteoprogenitors from 3 healthy control and 5 AS and conducted both DNA methylarray and bulk RNA-seq to profile epigenome and transcriptome on collected osteoprogenitors. We analyzed the identified hyper- and hypo-methylated regions with their corresponding transcriptome to evaluate epigenetic alterations on gene expression. To investigate the functional role of FOXF2, a known negative regulator of Wnt molecule, we utilized the CRISPR/Cas9 system to generate FOXF2-knockout (KO) Saos-2 cell lines. Additionally, osteoprogenitors were treated with a DNA methyltransferase (DNMT) inhibitor to assess the effect of DNA methylation on osteogenic potential.
Results. AS-osteoprogenitors exhibited a distinct epigenetic landscape, characterized by significant hypermethylation at the FOXF2 locus compared to controls. In consistently, FOXF2 was found to be downregulated in AS-osteoprogenitors. Overexpression of FOXF2 in osteoprogenitors significantly suppressed bone-forming activity and expression of differentiation markers. Conversely, CRISPR-mediated knockout of FOXF2 in Saos-2 led to increased osteoblast differentiation, as evaluated by matrix mineralization. Moreover, treatment with DNMT inhibitor dramatically decreased the bone-forming activity of AS-osteoprogenitors during osteoblast differentiation.
Conclusion. Our findings demonstrate that FOXF2 is a critical epigenetic target in AS. The promotion of osteogenesis through FOXF2 knockout, combined with the inhibitory effect of DNMT inhibitors, suggests that the epigenetic modulation of FOXF2 is key driver of pathological bone formation in AS.
Funding. This work was supported by the grant RS-2025-25441283, RS-2025-23523847 (to S.J), and 2021R1A6A1A03038899, RS-202400351695 (to T-H. K) funded by the Ministry of science and ICT through the National Research Foundation of Korea (NRF).

P8

OSTEOCLAST-DERIVED DEL1 PROMOTES PATHOLOGICAL BONE FORMATION OF AS VIA REGULATING RUNX2 EXPRESSION IN OSTEOBLAST

Lee S.H.[1], Lee S.[2], Jeon C.[1], Kim D.[1], Jo S.[3], Kim T.H.[4]
[1]Hanyang University Institute for Rheumatology Research (HYIRR), Hanyang University, Seoul; [2]Dept. of Radiology, Hanyang University Hospital for Rheumatic Diseases, Seoul; [3]Dept. of Biology, College of Natural Sciences, Soonchunhyang University, Asan; [4]Dept. of Rheumatology, Hanyang University Hospital for Rheumatic Diseases, Seoul, Republic of Korea

Introduction. Ankylosing spondylitis (AS) is a chronic inflammatory disease characterized by ectopic bone formation. We investigated *in vitro* and *in vivo* the role of developmental endothelial locus-1 (DEL1) on new bone formation and determined the association between DEL1 and spinal progression in AS.
Methods. DEL1 levels were measured in plasma and facet joint tissues from patients with AS, and in osteoclast-derived medium. Human osteoblast precursor cells were treated with recombinant DEL1 protein and cilengitide trifluoroacetate, an αvβ3 integrin inhibitor, to evaluate their effects on osteoblast differentiation markers. A curdlan-injected SKG mouse model was used to mimic AS pathogenesis. Three weeks after curdlan injection, recombinant DEL1 protein or cilengitide was administered, and the mice's ankle thickness was assessed. The mice were sacrificed after six weeks, and micro-CT and histological analyses were performed.
Results. DEL1 expression significantly increased during osteoclast differentiation, peaked at the terminal stage, and correlated with disease progression in AS mice. Systemic plasma DEL1 levels were not different between patients with AS and the control group but were positively correlated with structural damage (mSASSS; R=0.3433, p=0.0195). DEL1 pro-osteogenic effects were neutralized by cilengitide, which attenuated DEL1-induced RUNX2 expression and matrix mineralization.
Conclusion. Our findings establish DEL1 as a key osteoclast-derived coupling factor that drives new bone formation in AS. DEL1 promotes pathological bone formation and osteoblast differentiation by signaling through the integrin αVβ3-RUNX2 axis; targeting this pathway may offer a novel approach to prevent structural damage in patients with AS.

P9

BLOCKADE OF IL-17RA HIGHLIGHTS ALTERNATIVE OSTEOGENIC PATHWAYS DRIVEN BY IL-17F VIA BMP/WNT SIGNALLING

Paternina Martinez D.[1], Brown S.[1], Shah M.[2], Roberts S.J.[1]
[1]Skeletal Biology Group, Dept. of Comparative Biomedical Sciences, The Royal Veterinary College, London; [2]Division of Surgery and Interventional Science, University College London, London, UK

Introduction. Radiographic axial spondyloarthritis (r-axSpA) is a chronic inflammatory disease within the spondylarthritides family. Although its molecular mechanisms are not fully elucidated, TNF-α and IL-17 are implicated in pathological bone formation (PBF), particularly at periosteal-entheseal sites.
Methods. A human periosteum-derived cell (hPDC) model of osteogenic differentiation (OD) was used. Cells were stimulated with IL-17A or IL-17F (±TNF-α) with mineralisation and gene expression assessed by Alizarin Red staining and qPCR, respectively. Receptor blockade was achieved using neutralising antibodies, and transcriptomics by RNA sequencing with subsequent pathway enrichment analysis.
Results. IL-17A increased hPDC mineralisation in a dose-dependent manner across 1-20ng/ml (8-16-fold change (p≤0.01) vs. control). In contrast, IL-17F induced a similar increase (10-12-fold change (p≤0.01) without a clear dose-response trend. TNF-α (500 pg/ml) synergistically enhanced hPDC OD in combination with both IL-17A and IL-17F (p≤0.01). Conversely, 10ng/ml TNF-α reduced expression of key osteogenic markers (*RUNX2*, *BMP2*, *PHOSPHO1*), indicating a concentration-dependent effect. Blockade of IL-17RA (receptor for both IL-17F and IL-17A) with brodalumab decreased IL-17A-mediated OD to baseline (p≤0.001), but only partially reduced IL-17F-driven OD (p≤0.001). RNA sequencing and STRING analysis revealed that IL-17A preferentially upregulated genes linked to osteoclastogenesis and Th17 pathways, whilst IL-17F upregulated genes associated with osteogenesis (e.g. *BMP2*). Additionally, IL-17RA blockade attenuated TNF-α/IL-6 signalling-related gene expression in IL-17A-treated hPDCs. Interestingly, IL-6R blockade with tocilizumab reduced OD from both cytokines. Notably, IL-17F maintained a BMP/Wnt-associated gene signature (including *WNT9A*, *SFRP1* and *TMEM119*) despite blockade of IL-17RA, suggesting at least a partial receptor independence vs. IL-17A.
Conclusion. These findings support the hypothesis that IL-17F drives PBF through alternate pathways to IL-17A, potentially involving BMP/Wnt signalling. This is consistent with previous evidence that IL-17F (but not IL-17A) upregulates *RUNX2*. Further research is required to clarify the clinical implications of these results.
Acknowledgment. This research was funded by UCB Pharma.

P10

MATRIXMETALLOPROTINASE (MMP)-13 AS A POTENTIAL BIOMARKER OF STRUCTURAL CHANGES IN SPONDYLOARTHRITIS

Roman I.[1,2], Ney L.[1,2], Lalazi B.[1,2], Tsiami S.[1,2], Kiltz U.[1,2], Baraliakos X.[1,2], Schütz C.[1,2]
[1]Ruhr-Universität Bochumy; [2]Rheumazentrum Ruhrgebiet, Herne, Germany

Introduction/Objective. axSpA(r-/nr-axSpA) and PsA are inflammatory rheumatic musculoskeletal diseases (I-RMDs) characterized by distinct patterns of skeletal involvement: predominantly axial (axSpA) or peripheral (PsA). Structural changes (SC) are detected by imaging techniques, and no biomarker is currently established for their detection in clinical practice. MMP-13, a mediator of inflammation-driven structural remodeling, is highly expressed in axSpA entheseal tissue. The aim was to investigate whether MMP-13 is associated with the presence of SC.
Materials and methods. 163 donors (PsA, axSpA, healthy controls (HC)) were analyzed. Patients' data were extracted from hospital-records. MMP-13 was quantified by ELISA. R/nr-axSpA was defined using mNY-Criteria, whereas r/nr-PsA was defined based on the presence of SC on X-rays of peripheral joints.
Results. MMP-13 was significantly elevated in nr-axSpA vs. r-axSpA (p<0.01), and in HC vs. r-axSpA (p<0.0001), whereas no significant differences were detected between all other groups. In a multiple regression model analysis, MMP-13 together with disease duration substantially improved the discriminative power between nr- and r-axSpA (AUC 0.85; p<0.001), when compared to clinical parameters (AUC 0.78; p=0.001).
Conclusion. MMP-13 in combination with disease duration were able to predict radiographic status according to mNY-Criteria in axSpA. Despite comparable MMP-13 expression patterns (axSpA, PsA), there was no additive value for the prediction of SC in PsA.
Discussion. MMP-13 is associated with SC in axSpA, but not in PsA, which might be related to the different pattern of skeletal involvement in both diseases. MMP-13 in combination with clinical parameters substantially improved predictive power in axSpA only, supporting a potential role as an adjunct biomarker in I-RMDs with axial manifestation. Further experiments are needed to confirm MMP-13 as pathological actor and its possible clinical utility in axSpA in delimitation to peripheral SpA and axial PsA.

P11

A DYSREGULATED IL-22/IL-22BP AXIS IN SPONDYLOARTHRITIS IS RELATED TO IL-22-INDUCED OSTEOBLAST PROLIFERATION AND MINERALIZATION

Ortega-Mejía I.I.[1,2], Cerón-Cruz E.[2], Domínguez-López M.L.[1], García-Hernández A.L.[2], Alvarez-Rodríguez C.[2], Reyes-Maldonado E.[1], Casasola-Vargas J.C.[3], Vega-Salcedo A.V.[2], Bernal-Alferes B.[1], Romero-López J.P.[2]
[1]Posgrado en Ciencias Biológicas, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Mexico City; [2]Facultad de Estudios Superiores Iztacala, Universidad Nacional Autónoma de México, Tlalnepantla; [3]Departamento de Reumatología, Hospital General de México Dr. Eduardo Liceaga, Mexico City, Mexico

Introduction/Objective. Spondyloarthritis (SpA) is characterized by chronic inflammation and pathological bone formation. IL-22 has been implicated in tissue remodeling, whereas IL-22BP acts as its soluble antagonist. We aimed to evaluate whether dysregulation of the IL-22/IL-22BP axis contributes to osteoblast proliferation and mineralization *in vitro*.
Materials and methods. hFOB 1.19 osteoblasts were cultured and stimulated with IL-6, IL-23 and TGF-β to evaluate IL-22R expression by flow cytometry. Osteoblasts were subsequently stimulated with rIL-22 (50, 250 and 500 ng/mL) and rIL-22BP (1, 10 and 100 ng/mL) to assess cell proliferation. Bone matrix formation was evaluated by Alizarin Red staining in two-dimensional and three-dimensional *in vitro* models. Also, IL-22BP concentration was measured in blood and synovial fluid of SpA patients.
Results. hFOB 1.19 osteoblasts expressed IL-22R at basal levels, and receptor expression increased after stimulation with pro-inflammatory cytokines. IL-22 promoted osteoblast proliferation and extracellular matrix formation in both two-dimensional and three-dimensional culture systems. In contrast, IL-22BP reduced the IL-22-induced proliferative and mineralization responses. In patient samples, serum concentration of IL-22BP was reduced.
Conclusion. These preliminary findings support a dysregulated IL-22/IL-22BP axis in SpA and suggest that IL-22 may directly promote osteoblast proliferation and mineralization. IL-22BP may counterbalance this effect, representing a potential regulatory mechanism and therapeutic target for pathological osteoproliferation in SpA.

P12

SELF-MANAGEMENT NEEDS AND DIGITAL TOOLS IN AXIAL SPONDYLOARTHRITIS – A QUALITATIVE STUDY THROUGH THE LENS OF SELF-DETERMINATION THEORY

Barnett R.[1,2], Clarke C.[3], Sengupta R.[2], Rouse P.C.[1]
[1]Dept. for Health, University of Bath, Bath; [2]Royal National Hospital for Rheumatic Diseases, Royal United Hospitals Bath NHS Foundation Trust, Bath; [3]Dept. of Computer Science, University of Bath, Bath, UK

Introduction. Digital tools may support and empower people living with axial spondyloarthritis (axSpA) to self-manage between rheumatology appointments. Self-determination theory (SDT) and the Motivation, Engagement and Thriving in User Experience model (METUX) can be leveraged to gain a deeper understanding of the digital self-management needs of people living with axSpA.
Methods. Online qualitative surveys were distributed via UK patient and healthcare professional networks. Responses were analysed using reflexive thematic analysis, guided by a constructivist epistemology. Inductive (semantic/latent) and deductive (through the lens of SDT/METUX) codes were generated. Themes were defined by the interdisciplinary research team. None of the researchers have axSpA.
Results. Thirty patients, 10 physiotherapists, and one consultant rheumatologist completed the survey. Four themes were reflexively interpreted from participant responses (Fig. 1): 1) Effective long-term self-management is personalised, flexible, and holistic; 2) Making the invisible, visible: people

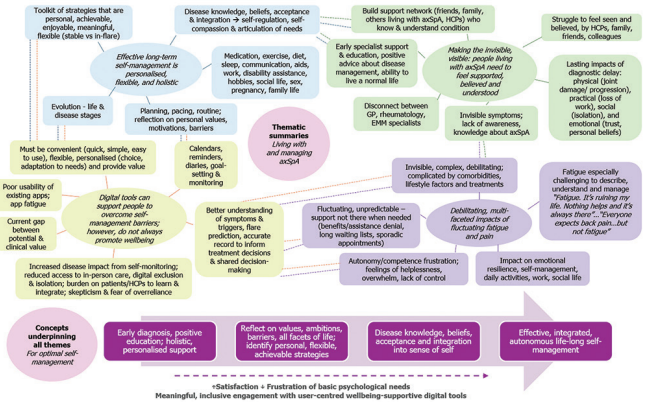


Fig. 1. Thematic map, theme summaries, and proposed key concepts underpinning all themes.

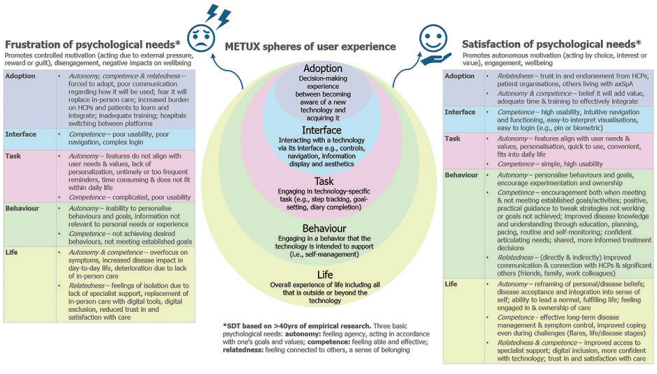


Fig. 2. Satisfaction and frustration of basic psychological needs across METUX spheres (theme 4).

living with axSpA need to feel supported, believed and understood; 3) Debilitating, multi-faceted impacts of fluctuating fatigue and pain; 4) Digital tools can support people to overcome self-management barriers, however do not always promote wellbeing. Optimal self-management requires early diagnosis, positive education and holistic, personalised support; to address disease beliefs, personal barriers and fluctuating symptoms, across life/disease stages. Digital self-management tools can satisfy and frustrate basic psychological needs across METUX spheres of user experience (Fig. 2, theme 4). There is an unmet need for effective, convenient, integrated digital self-management tools for axSpA.

Conclusion. SDT can inform the design of more effective digital self-management tools, that support (and not thwart or frustrate) the needs and wellbeing of people living with axSpA. Self-management interventions should target fatigue, pain, disease knowledge and beliefs, and support patients to reflect on all facets of their life. To enable patients to gain a deep understanding of their condition, and integrate achievable, flexible self-care strategies that work for them.

P13

ACHIEVING LOW DISEASE ACTIVITY AT 6 MONTHS IN SPONDYLOARTHRITIS: REAL-WORLD PREDICTORS

Boni V.[1], Patti D.G.[1], Niedda F.[1], Cristiano F.[1], Gavotti C.[1], Masnata R.[1], Moroli M.[1], De Piano R.[1], Fano M.[1], Natalello G.[1,2], De Lorenzis E.[1,2], D'Agostino M.A.[1,2], Ortolan A.[1,2]

[1]Catholic University of Sacred Heart, Rome; [2]Fondazione Policlinico Universitario Agostino Gemelli IRCCS, Rheumatology Division, Rome, Italy

Introduction. Remission or low disease activity (LDA) is recommended target in the treatment of axSpA, but frequency of LDA in real-life are under-investigated. Our aim is to evaluate the frequency of LDA achievement in a real-life in axSpA population starting bDMARD treatment at 6 months and to find potential predictors of LDA.

Material and methods. A longitudinal study enrolling consecutive patients with diagnosis of axSpA by the rheumatologist, who started a bDMARD treatment between 06/2023-09/2025 was performed. Patients were followed up at 6 and 12-months. Characteristics of patients and disease, activity indices including ASDAS, therapy adherence via Compliance Questionnaire for Rheumatology (CQR5)], comorbidities and contextual factors were compared between patients achieving ASDAS-LDA (ASDAS <2.1) at 6 months (sustained ASDAS-LDA) and those who did not, using Chi-square and Mann-Whitney tests. A survival analysis was performed to predict first LDA achievement (outcome) in every patient. Factors potentially influencing ASDAS-LDA (according to literature and own hypothesis) were first tested in univariable analysis, then retained as independent variables in the multivariable analysis if $p<0.2$ at univariable analysis. Results were expressed as hazard ratio (95% confidence interval).

Results. A total of 153 patients were enrolled, 50% males, mean age 49.9±13.9 years, disease duration 9.8±8.0 years,42% were HLA-B27+, 44% had peripheral arthritis, 28% enthesitis. Most patients were treated with TNF-inhibitors (58%), followed by IL-17 inhibitors (35%) and JAK-inhibitors (7%). At 6 months, 70/149 (47%) achieved LDA, and at 63/137 (46%). Patients achieving LDA at 6 m were more frequently male and, university-educated, and less frequently had peripheral arthritis (36% vs. 53%, $p=0.040$) or psoriasis (17% vs. 36%, $p=0.010$) as compared to those not reaching LDA (Table I). They also showed lower baseline disease activity across all clinical indices (Table I). In the multivariable Cox regression model, university education was the only contextual factor that remained independently associated with LDA achievement, whereas fibromyalgia was negatively associated with LDA. [(Table II)]

Conclusion. ASDAS-LDA is an achievable target, although certain patient-related and disease-related factors may make it more difficult to reach. Understanding the factors associated with failure to achieve LDA will also allow us to take measures to promote positive outcomes, such as managing comorbidities or educating patients to improve their understanding of their condition and their coping strategies.

Table I. Baseline characteristics by achievement of low disease activity (LDA) at V1.

Variable	Non-LDA (n=78)	LDA (n=70)	p-value
Demographic characteristics			
Male sex, n (%)	30 (38%)	43 (61%)	0.005
Age, years	49.9 ± 12.6 51.5 (43–59)	50.0 ± 15.3 49.5 (36–61)	0.894
Disease duration, years	9.9 ± 8.2 7.5 (5–11)	9.6 ± 8.1 7.0 (4–13)	0.633
University education, n (%) †	11 (14%)	26 (37%)	0.019
Living alone, n (%) ‡	13 (16%)	18 (25%)	0.323
Disease characteristics			
Sacroiliitis on X-ray, n (%)	71 (91%)	68 (97%)	0.120
HLA-B27 positive, n (%) †	33 (43%)	28 (40%)	0.753
Radiographic form (raxSpA), n (%) †	35 (44%)	38 (54%)	0.269
IBP (ASAS criteria), n (%)	75 (96%)	67 (96%)	0.892
Peripheral arthritis, n (%)	41 (53%)	25 (36%)	0.040
Enthesitis, n (%)	27 (35%)	16 (23%)	0.116
Dactylitis, n (%)	5 (6%)	5 (7%)	0.859
Psoriasis, n (%)	28 (36%)	12 (17%)	0.010
Uveitis, n (%)	9 (12%)	7 (10%)	0.743
IBD, n (%)	5 (6%)	8 (11%)	0.282
Family history of SpA (ASAS), n (%)	2 (3%)	6 (9%)	0.107
Good NSAID response, n (%)	42 (54%)	44 (62%)	0.299
Elevated CRP at diagnosis, n (%)	13 (16%)	11 (16%)	0.935
Disease activity indices			
VAS pain (0–10)	7.1 ± 1.3 7 (6–8)	5.7 ± 2.0 6 (5–7)	<0.001
VAS patient global activity (0–10)	7.0 ± 1.7 7 (6–8)	6.0 ± 2.0 7 (5–7)	0.002
VAS physician global activity (0–10)	5.6 ± 1.4 5 (5–7)	4.9 ± 1.7 5 (4–6)	0.029
BASDAI	5.2 ± 1.6 5.2 (4.2–6.0)	3.5 ± 1.6 3.6 (2.4–4.8)	<0.001
ASDAS-CRP	3.2 ± 0.6 3.1 (2.8–3.6)	2.6 ± 0.7 2.6 (2.2–3.2)	<0.001
Swollen joint count (66)	0.9 ± 2.0 0 (0–1)	0.3 ± 0.9 0 (0–0)	0.005
Tender joint count (68)	1.9 ± 2.9 1 (0–3)	0.9 ± 1.6 0 (0–1)	0.012
CRP, mg/L	5.1 ± 6.8 2 (2–5)	3.9 ± 3.5 2 (2–4)	0.714
Comorbidities and BMI			
BMI, kg/m²	25.4 ± 4.0 25.2 (23.0–27.0)	23.8 ± 5.6 24.3 (21.5–26.2)	0.108
Fibromyalgia, n (%)	32 (41%)	8 (11%)	<0.001
Spinal osteoarthritis, n (%) †	34 (43%)	25 (36%)	0.407
RDCI	0.8 ± 1.1 0 (0–2)	0.9 ± 1.1 0 (0–2)	0.964
Treatment			
Biological class, n (%)			0.643
TNFi	44 (56%)	41 (59%)	
IL-17i	27 (35%)	24 (34%)	
IL-23i	0 (0%)	1 (1%)	
JAKi	7 (9%)	4 (6%)	
Biological line, n (%)			0.077
First line (naive)	34 (44%)	43 (61%)	
Second line	27 (35%)	22 (31%)	
Third line or beyond	17 (22%)	5 (7%)	
Concomitant NSAIDs, n (%)	47 (60%)	43 (61%)	0.884
Concomitant csDMARDs, n (%)	20 (26%)	10 (14%)	0.086

LDA: low disease activity (ASDAS-CRP <2.1). *p*-values from Mann-Whitney U-test for continuous variables and Pearson chi-squared test for categorical variables.
Continuous variables presented as mean ± SD and median (IQR).

Table II. Multivariable Cox regression analysis for predictors of low disease activity (LDA).

Variable	HR	95% CI	p-value
Male sex	1.36	0.85–2.18	0.195
ASDAS-CRP at baseline	0.77	0.55–1.08	0.129
Naive (first-line biological)	1.10	0.67–1.80	0.707
University education	**1.72**	**1.04–2.86**	**0.036**
BMI, kg/m²	0.98	0.94–1.03	0.434
Fibromyalgia	**0.51**	**0.26–0.98**	**0.043**
Peripheral involvement *	0.92	0.57–1.49	0.725
CQR5 score (continuous) ¶	1.07	0.95–1.20	0.258

HR: hazard ratio; CI: confidence interval. LDA defined as ASDAS-CRP <2.1.
* Peripheral involvement = arthritis, enthesitis, or dactylitis. ¶

P14

EXPERIENCE WITH COMBINED B/TSDMARD THERAPY IN REFRACTORY INFLAMMATORY ARTHRITIS: A MULTICENTER MULTIDISCIPLINARY STUDY

De la Morena Barrio I.[1], Aguilar Zamora M.[2], Ochoa de Aranda B.[3], Charca Mamani L.[1], Sanmartin J.[1], Huaylla Quispe A.[4], Robustillo Villarino M.[5], García Martos A.[6], Palma Sánchez D.[7], Trenor Larraz P.[1]
[1]Hospital Clínico Universitario de Valencia, Valencia; [2]Hospital de Sagunto, Sagunto; [3]Unidad de Reumatología INCLIVA, Valencia; [4]Hospital Francesc de Borja, Gandía; [5]Hospital La Plana, Castellón; [6]Hospital Universitario del Tajo, Aranjuez; [7]Hospital Rafael Méndez, Lorca, Spain

Introduction. Achieving clinical remission remains an unmet need for a significant proportion of patients with inflammatory arthritis. Recent consensus by EULAR and ASAS has defined "Difficult-to-Treat" Rheumatoid Arthritis (D2T-RA) and "Difficult-to-Manage" axial Spondyloarthritis (D2M-axSpA) as states where conventional strategies fail after at least two advanced therapy lines. Combined biological (bDMARD) or targeted synthetic (tsDMARD) therapy is an emerging strategy for these highly refractory cases or complex overlaps like Spondyloarthritis associated with Inflammatory Bowel Disease (IBD).
Methods. This is a multicenter, retrospective, observational study involving 6 Spanish hospitals. We included 36 patients requiring dual b/tsDMARD therapy due to therapeutic refractoriness or disease overlap. We analyzed demographics, prior treatment mechanisms, combination types, safety, and clinical activity (BASDAI, CDAI, DAPSA) alongside steroid requirements at baseline, 6, and 12 months.
Results. The cohort included 36 patients (55.5% female; mean age 50.6 years) with a mean disease duration of 8.7 years. Patients had failed a mean of 4.4 prior bDMARDs and 3 different mechanisms of action (range 1–7). The most frequent diagnosis was SpA-IBD (52.8%) (Fig. 1). Upadacitinib was the most used drug for combinations (n=18). Steroid requirements dropped significantly: 80% of baseline users successfully discontinued prednisone by month 12. Clinical activity scores showed a marked improvement across all subtypes (Table I). Persistence was 83.3% at the end of follow-up (mean 15.7 months). One major adverse event (lacunar stroke) was reported.

Table I. Baseline characteristics and combinations (n=36).

Characteristic	Value / Frequency
Age (years)	50.6 ± 9.5
Prior bDMARDs failed (mean)	4.4 (Range 1–15)
Prior mechanisms of action failed (mean)	3.0 (Range 1–7)
Preferred Combinations (Top 3)	iJAK + antiTNF (n=6), iJAK + RTX (n=5), iTNF + iIL23 (n=5)

Table II. Clinical evolution and steroid tapering.

Parameter	Baseline	6 Months	12 Months
Mean Prednisone (mg/day)	6.5 ± 8.4	4.4 ± 7.6	1.9 ± 3.5
BASDAI (Axial SpA)	6.7	5.7	4.8
CDAI (Peripheral arthritis)	16.3	10.7	5.2
DAPSA (PsA)	7.0	11.0*	6.6

Conclusion. Combined b/tsDMARD therapy is a viable strategy for D2T-RA and D2M-axSpA, showing high persistence (83.3%) and efficacy despite multiple prior failures. It achieves a profound steroid-sparing effect, allowing 80% of users to discontinue prednisone. This synergistic approach addresses unmet needs in highly refractory cases and complex comorbidities like IBD, providing a successful multidisciplinary alternative when standard sequential treatments fail.

P15

THE IMPACT OF MULTIPLE GENDER DIMENSIONS ON HEALTH-RELATED QUALITY OF LIFE IN PEOPLE WITH RHEUMATIC DISEASES: A CROSS-SECTIONAL SURVEY STUDY (GENDER-Q-ARISE)

Hamamou A.[1], Mulder M.L.M.[2], van der Horst-Bruinsma I.[2], van Leuven S.I.[2], Vonk M.C.[2], Popa-Diaconu D.A.[2], Göttgens I.[1], Oertelt-Prigione S.[1,3]
[1]Radboud University Medical Center (Radboudumc), Dept. of Primary and Community Care, Unit Gender in Primary and Transmural Care, Nijmegen; [2]Radboud University Medical Center (Radboudumc), Dept. of Rheumatology, Nijmegen, The Netherlands; [3]Dept. of Sex- and Gender-Sensitive Medicine, Medical Faculty OWL, University of Bielefeld, Bielefeld, Germany

Introduction. Axial spondyloarthritis (AxSpA), psoriatic arthritis (PsA), systemic sclerosis (SSc) and systemic lupus erythematosus (SLE) are chronic inflammatory rheumatic diseases associated with impaired health-related quality of life (HRQoL). Women with spondyloarthritis often report poorer HRQoL than men, but binary sex/gender comparisons overlook gender as a multidimensional sociocultural construct, including gender identity, expression, roles/norms and relations. We examined and ranked associations between these gender dimensions and HRQoL, overall and by disease group (AxSpA/PsA; SSc/SLE).
Methods. Adults with AxSpA, PsA, SSc or SLE completed an online survey assessing HRQoL (SF36_total, Physical Component Summary (PCS), Mental Component Summary (MCS)), gender identity, sex assigned at birth, gender expression, BSRI gender role orientation and private-domain gender relations. Linear regression models assessed associations with SF36_total, adjusted for age, disease duration, education and diagnosis.
Results. In total 587 participants completed the survey (Table I). Mean SF36_total was 56.1±18.4, PCS 33.7±10.6 and MCS 50.4±10.2. Gender relations showed the strongest association with HRQoL, ranking first among all (Table II). Changes in household task division, income contribution and paid work since diagnosis were associated with lower SF36_total overall and in both disease groups. Higher household involvement was associated with higher SF36_total overall and in AxSpA/PsA. Gender role orientation ranked second: masculine BSRI profiles were associated with higher HRQoL overall, while feminine profiles were associated with lower HRQoL. Gender identity and expression showed smaller positive associations, mainly overall and in AxSpA/PsA.

Table I. Sample characteristics and HRQoL**.**

Characteristic	All	AxSpA/PsA	SSc/SLE
Sample size	643	396	247
Complete SF-36 outcomes, n (%)	587 (91.3%)	356 (89.9%)	231 (93.5%)
Age in years, mean (SD)	53.1 (13.6)	51.0 (13.3)	56.3 (13.3)
Disease duration in years, mean (SD)	15.0 (14.7)	16.2 (15.9)	13.1 (12.5)
Gender identity: woman / man	454 (77.3%) / 133 (22.7%)	278 (78.1%) / 78 (21.9%)	176 (76.2%) / 55 (23.8%)
SF36_total, mean (SD)	56.1 (18.4)	55.3 (18.1)	57.2 (18.9)
PCS, mean (SD)	33.7 (10.6)	33.6 (10.3)	33.9 (11.0)
MCS, mean (SD)	50.4 (10.2)	50.0 (10.6)	51.1 (9.5)

Demographics are reported for participants with complete SF-36 outcomes.
SF-36: 36-Item Short Form Survey; SF36_total: average of eight SF-36 domain scores; PCS: Physical Component Summary; MCS: Mental Component Summary. Higher SF-36 scores indicate better HRQoL.

Table II. Gender dimensions ranked by impact on SF36 Sumscore.

Impact rank	Gender dimension	Predictor / contrast	All adj. β (95% CI)	AxSpA/PsA adj. β (95% CI)	SSc/SLE adj. β (95% CI)
1	Gender relations (Private-domain)	Household task division changed yes vs. no since diagnosis	-15.2 (-18.4, -12.0)***	-13.6 (-17.7, -9.6)***	-16.5 (-21.9, -11.1)***
		Income contribution changed yes vs. no since diagnosis	-12.4 (-15.9, -9.0)***	-13.3 (-17.4, -9.1)***	-12.1 (-18.2, -6.0)***
		Paid work changed yes vs. no since diagnosis	-11.7 (-15.0, -8.4)***	-13.2 (-17.2, -9.2)***	-10.9 (-16.5, -5.2)***
		Household involvement score per 1-unit increase	+7.8 (+3.6, +12.0)***	+8.9 (+3.8, +14.1)***	+6.5 (-0.9, +13.9)
2	Gender roles/norms BSRI type	Masculine high M, low F	+6.0 (+2.6, +9.4)***	+4.0 (-0.3, +8.2)	+9.0 (+3.5, +14.6)**
		Feminine high F, low M	-3.3 (-6.6, +0.1)	-4.8 (-9.0, -0.7)*	-1.3 (-6.8, +4.3)
		Androgynous high M, high F	-1.0 (-4.4, +2.4)	-0.6 (-4.7, +3.6)	-3.6 (-9.4, +2.2)
		Undifferentiated low M, low F	-1.7 (-5.2, +1.8)	+1.4 (-3.2, +6.1)	-4.2 (-9.5, +1.1)
3	Gender identity	Man vs. woman woman = reference	+6.0 (+2.4, +9.7)**	+7.2 (+2.4, +11.9)**	+3.5 (-2.3, +9.4)
4	Gender expression 1=feminine, 7=masculine	Self-perceived expression per +1 toward masculine	+1.1 (+0.4, +1.8)**	+1.0 (+0.1, +1.9)*	+1.0 (-0.1, +2.0)
		Others' perceived expression per +1 toward masculine	+1.0 (+0.4, +1.7)**	+1.1 (+0.2, +2.0)*	+0.8 (-0.3, +1.9)

Cells show adjusted β (95% CI) for SF36_total; + = higher HRQoL and - = lower HRQoL. Rank is based on the pooled sample. Gender relations restricted to participants with a partner. BSRI: Bem Sex Role Inventory; M/F: masculine/feminine traits. For BSRI, β represents each category compared with the other BSRI categories combined. *p<0.05, **p<0.01, ***p<0.001.

Conclusion. Gender-related differences in HRQoL cannot be fully understood through gender identity or binary sex/gender comparisons alone. Gender relations displayed the largest impact on HRQoL, particularly changes in domestic and economic roles since diagnosis, followed by gender role orientation, identity and expression. These findings support multidimensional gender operationalisation in rheumatology and integration of relational and role-based gender dimensions into gender-sensitive rheumatology care.
Acknowledgements. Supported by a Radboudumc Incentive Grant.

P16

UPDATE OF THE MSQUASH MANUAL AND SYNTAX: VALID AND RELIABLE PHYSICAL ACTIVITY QUESTIONNAIRE ALIGNING WITH RECENT INTERNATIONAL GUIDELINES

Harmsen D.H.[1], Paap D.[1,2], van der Kraan Y.M.[1], Carbo M.J.G.[1], Wink F.R.[3], Gensler L.S.[4], Spoorenberg J.P.L.[1], Arends S.[1]
[1]Rheumatology and Clinical Immunology, University Medical Center Groningen, University of Groningen, Groningen; [2]Physiotherapy, Saxion University of Applied Sciences, Enschede; [3]Rheumatology, Medical Center Leeuwarden, Leeuwarden, The Netherlands; [4]Medicine/Rheumatology, University of California San Francisco, San Francisco, California, USA

Introduction. The modified Short QUestionnaire to ASses Health-enhancing physical activity (mSQUASH) is a widely used patient-reported instrument validated in axial spondyloarthritis (axSpA) (1, 2). However, based on mSQUASH analyses in different rheumatic diseases, evolving international physical activity guidelines and recent update to Ainsworth's Compendium of physical activities, revision is necessary.
Objective. To update the mSQUASH manual and syntax and to assess potential impact of these adaptations on previous clinical validation data in axSpA patients.
Methods. Intensity classification, demand factors (based on MET values and perceived physical demand), sport/activity codes, handling of missing data and manual checks were reassessed. Patients from the GLAS cohort and San Francisco (UCSF) were combined for clinical validation (1, 2). Classification accuracy (n=40) was analyzed descriptively. Criterion validity (n=40) and construct validity (n=212) were assessed using Spearman correlations (ϱ). Test-retest reliability (n=102) was tested with ICC. Responsiveness (n=132) was assessed using SRM.
Results. Intensity classification was adapted to 1.6-2.9 METs, 3.0-5.9 METs and ≥6.0 METs for light, moderate and vigorous intensity respectively, in accordance with international guidelines. Demand factors were adapted according to this classification. Sport/activity codes were updated according to 2024 Compendium. Handling of missing data was adapted by imputing minutes per day. Manual checks were adapted by lowering limits for excessive hours.

Table I. Classification accuracy of the mSQUASH minutes per week compared to accelerometer in patients with axSpA (n=40) showing less overestimation of intensity.

	Light intensity	Moderate intensity	Vigorous intensity
mSQUASH	1860 (716-2471)	978 (500-1328)	0 (0-23)
Accelerometer	1766 (1455-2071)	196 (118-270)	0 (0-5)

Data presented as median (IQR 25th-75th percentile).

Table II. Test-retest reliability of the mSQUASH total activity scores and underlying domains in patients with axSpA (n=102).

	Baseline	2 weeks	ICC	95% CI
Total activity score	8685 (5715-12304)	7925 (5623-10553)	0.78	0.68-0.84
Commute	600 (230-1388)	660 (236-1203)	0.43	0.26-0.57
Work or school	3840 (0-4890)	3840 (0-5280)	0.92	0.88-0.94
Household activities	803 (420-1995)	795 (345-1875)	0.70	0.59-0.79
Leisure activities, sports and exercise	2745 (1211-4095)	2565 (1201-4144)	0.77	0.67-0.84

Data presented as median (IQR 25th-75th percentile). ICC: intraclass correlation coefficients; CI: confidence interval.

Tri-axial accelerometer and mSQUASH classified both most minutes as light intense (Table I). Moderate correlation between mSQUASH total activity score and accelerometer was found (ϱ=0.53). Fair associations between mSQUASH total activity score and other outcomes were found (ASDAS: ϱ=-0.23, BASDAI: ϱ=-0.21, BASFI: ϱ=-0.27, ASAS-HI: ϱ=-0.23). The mSQUASH showed good test-retest reliability (ICC=0.78) (Table II). Responsiveness showed small to moderate SRM, in correspondence with self-reported changes in physical activity after three months.
Conclusion. The updated mSQUASH aligns with current international physical activity guidelines and remains a valid and reliable questionnaire to assess daily physical activity in axSpA.

References

1. CARBO MJG, PAAP D, MAAS F, BARON AJ *et al*.: The mSQUASH; a valid, reliable and responsive questionnaire for daily physical activity in patients with axial spondyloarthritis. *Semin Arthritis Rheum* 2021; 51: 719-27.
2. VAN DER KRAAN YM, GENSLER L, PAAP D, THOVMASYAN E *et al*.: The mSQUASH is a feasible and valid measurement tool to uniformly assess daily physical activity in patients with rheumatic diseases. *RMD Open* 2024; 10:e004696.

P17

PHYSICAL ACTIVITY OVER TIME IN RELATION TO PATIENT AND DISEASE CHARACTERISTICS IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS

He Z.[1], Harmsen D.H.[1], Paap D.[1,2], Carbo M.J.G.[1], Kieskamp S.C.[1], van der Kraan Y.M.[1], Wink F.[1,3], Arends S.[1], Spoorenberg J.P.L.[1]
[1]Dept. of Rheumatology and Clinical Immunology, University Medical Center Groningen, University of Groningen, Groningen; [2]Dept. of physiotherapy, Saxion University of Applied Sciences, Enschede; [3]Dept. of Rheumatology, Frisius Medical Center, Leeuwarden, The Netherlands

Introduction. Physical activity (PA) is a cornerstone of axial spondyloarthritis (axSpA) management, improving quality of life, symptoms and inflammation. However, most data are cross-sectional with limiting longitudinal insight. The modified Short QUestionnaire to ASsess Health-enhancing PA (mSQUASH) is validated in axSpA with work, commute, household and leisure/sport domains. This study explored the longitudinal relationships between PA, disease-related outcomes and coping strategies in axSpA patients.
Methods. Patients from the Groningen Leeuwarden axSpA (GLAS) cohort completing the mSQUASH twice (between 18–48 months apart) were included (1, 2). Other assessments included BASDAI, ASDAS, patient GDA, BASFI, ASQoL and CORS. Baseline mSQUASH total scores, domain scores and minutes were divided into tertiles. Clinically relevant PA changes were defined using limits of agreement from previous reliability studies by Bland-Altman analysis (1, 3). As leisure/sport domain is most beneficial to health, baseline disease-related outcomes were compared according to longitudinal changes of mSQUASH total scores and leisure/sport scores.
Results. 131 axSpA patients were included with mean duration of 35±7 months between completing mSQUASH. PA was mostly performed during work. Females reported more household and less leisure/sports activities than males. Patients in the lowest mSQUASH PA tertiles were older and had

Table I. Baseline comparison of patient characteristics and disease-related assessments for PA level stratified in tertiles.

		mSQUASH total score 7740 (5010-11100)				mSQUASH total minutes 2745 (1440-3390)			
	All patients	Low PA range 600-6000 (n=44)	Intermediate PA range 6290-9800 (n=44)	High PA range 9830-28110 (n=43)	p-value	Low PA range 225-2070 (n=44)	Intermediate PA range 2120-3200 (n=44)	High PA range 3210-5730 (n=43)	p-value
Age, years	47.3±13.9	51.2±15.3 *	43.7±12.6	47.0±13.0	**0.040**	51.4±15.3 *	44.6±12.8	45.8±12.9	**0.049**
Male	73 (55.7%)	23 (52.3%)	23 (52.3%)	27 (62.8%)	0.523	24 (54.5%)	21 (47.7%)	28 (65.1%)	0.259
R-axSpA	91 (69.5%)	29 (65.9%)	32 (72.7%)	30 (69.8%)	0.785	29 (65.9%)	32 (72.7%)	30 (69.8%)	0.785
BMI	27.1±6.5	30.2±6.2 *†	26.8±4.8	25.7±4.5	**<0.001**	30.4±5.7 *†	25.2±7.7	25.5±4.1	**<0.001**
BASDA	3.8±2.2	4.4±2.3 *†	3.5±1.9	3.5±2.2	**0.007**	4.6±2.1 *†	3.2±2.1	3.6±2.1	**0.007**
BASDAI≥4	54 (41.9%)	23 (52.3%)	15 (34.1%)	16 (37.2%)	0.180	26 (59.1%) *†	12 (27.3%)	16 (37.2%)	**0.008**
BASDAI fatigue	5.1±2.5	5.6±2.5	5.2±2.2	4.6±2.7	0.164	5.7±2.5	4.7±2.2	5.0±2.7	0.184
BASDAI axial pain	4.5±2.7	4.9±2.7	4.5±2.7	4.3±2.7	0.610	5.2±2.7	4.0±2.6	4.4±2.6	0.113
BASDAI peripheral pain	2.9±2.9	3.5±2.8	2.6±2.9	2.7±3.1	0.331	3.9±2.7†	2.3±3.1	2.7±2.8	**0.034**
ASDAS	2.2±0.9	2.4±0.9	2.1±0.9	2.2±0.9	0.271	2.5±0.9 *	2.0±0.9	2.2±0.9	**0.020**
ASDAS≥2.1	64 (50.8%)	22 (50.0%)	18 (40.9%)	19 (44.2%)	0.686	25 (56.8%)	15 (34.1%)	19 (44.2%)	0.100
CRP, mg/L	2 (1-5)	2 (1-5)	3 (1-7)	2 (1-4)	0.392	2 (1-5)	3 (2-6)	2 (1-4)	0.323
CRP≥5	27(22.1%)	10 (22.7%)	11 (25.0%)	6 (14.0%)	0.406	9 (20.5%)	11 (25.0%)	7 (16.3%)	0.603
Patient GDA	4.1±2.7	4.7±2.6	3.6±2.6	4.2±2.8	0.191	4.9±2.5 *†	3.3±2.8	4.2±2.6	**0.022**
BASFI	3.3±2.2	3.8±2.4	3.0±2.2	3.1±2.1	0.264	4.3±2.3 *†	2.7±1.9	3.0±2.3	**0.004**
ASQoL	5.6±4.8	7.3±5.4	4.7±4.3	5.1±4.4	0.090	7.8±5.0 *†	4.0±4.0	5.2±4.7	**0.006**
CORS									
Coping with pain									
· Comforting cognitions	27.3±4.2	26.9±3.2	27.2±4.6	27.6±4.6	0.808	27.0±3.3	27.3±4.5	27.4±4.7	0.941
· Decreasing activities	19.7±4.5	20.8±3.8	19.9±4.4	18.5±5.2	0.156	21.1±3.8	19.3±4.2	18.8±5.3	0.130
· Diverting attention	20.4±4.5	19.3±3.2	21.0±4.2	21.0±5.6	0.252	20.1±3.5	19.9±4.8	21.1±5.0	0.541
Coping with limitations									
· Optimism	15.7±3.2	16.0±2.8	15.5±3.5	15.8±3.4	0.806	15.9±2.6	15.8±3.1	15.5±3.8	0.864
· Pacing	26.8±6.9	28.2±5.4	26.6±6.4	25.6±8.3	0.363	28.7±5.7	25.8±5.0	25.8±8.9	0.187
· Creative solution seeking	22.1±5.0	22.0±3.7	21.9±5.2	22.4±5.9	0.918	22.3±3.8	21.9±4.1	22.1±6.6	0.965
Coping with dependence									
· Accepting dependence	14.3±4.5	14.8±4.1	14.1±4.9	14.0±4.4	0.781	15.1±4.0	13.6±4.5	14.1±4.8	0.420
· Showing consideration	19.3±4.0	19.0±2.9	19.8±4.7	19.2±4.3	0.694	19.3±3.2	19.6±3.9	19.0±4.9	0.861

*p-values ≤0.05 for highest PA group compared to lowest PA group. †p-values ≤0.05 for intermediate PA group compared to lowest PA group.

Table II. Baseline comparison of patient characteristics and disease-related assessments stratified for change in PA level over time.

	mSQUASH total score change				Leisure sport score change			
	Decrease ΔPA 6390-13690 (n=15)	Stable ΔPA (-4860)-5870 (n=88)	Increase ΔPA (-18690)-(-5330) (n=28)	p-value	Decrease ΔPA 3210-8280 (n=10)	Stable ΔPA(-2550)-3000 (n=104)	Increase ΔPA(-16320)-(-2700) (n=17)	p-value
Age	55.0 (45.0-60.0)	49.5 (34.0-57.8)	44.5 (29.3-51.8)	**0.018**	57.5 (41.5-65.0)	49.0 (34.0-56.8)	48.0 (35.0-56.0)	0.373
Male	6 (40.0%)	50 (56.8%)	17 (60.7%)	0.401	6 (60.0%)	55 (52.9%)	12 (70.6%)	0.380
R-axSpA	11 (73.3%)	60 (68.2%)	20 (71.4%)	0.894	8 (80.0%)	68 (65.4%)	15 (88.2%)	0.125
BMI	27.5 (23.6-29.2)	26.5 (23.7-31.1)	27.1 (24.4-29.0)	0.452	27.3 (22.8-30.3)	26.9 (24.2-30.8)	25.0 (21.9-28.0)	0.313
BASDAI	3.6 (1.6-5.4)	3.6 (2.2-5.6)	3.3 (1.6-5.9)	0.781	3.4 (1.4-5.2)	3.6 (2.2-5.8)	2.8 (1.3-4.4)	0.702
BASDAI≥4	6 (40.0%)	38 (44.2%)	10 (35.7%)	0.724	4 (40.0%)	44 (43.1%)	6 (35.3%)	0.825
BASDAI fatigue	6.0 (4.0-8.0)	5.0 (3.0-7.0)	5.0 (3.0-7.0)	0.346	5.0 (3.5-8.0)	5.0 (2.0-7.0)	4.0 (2.5-5.5)	0.190
BASDAI acial pain	3.0 (2.0-7.0)	4.0 (2.0-7.0)	5.0 (2.0-7.0)	0.834	3.0 (1.8-6.0)	5.0 (2.0-7.0)	3.0 (1.0-7.0)	1.000
BASDAI peripheral pain	1.0 (0.0-6.0)	2.0 (0.0-6.0)	2.0 (0.0-5.0)	0.791	1.0 (0.0-6.0)	2.5 (1.6-5.0)	0.0 (0.0-4.5)	0.347
ASDAS	2.5 (1.5-2.7)	2.1 (1.5-2.8)	2.1 (1.5-3.3)	0.641	2.0 (1.5-2.7)	2.2 (1.6-3.0)	1.9 (1.2-2.8)	0.419
ASDAS≥2.1	8 (53.3%)	38 (49.4%)	13 (50.0%)	0.961	4 (40.0%)	48 (52.2%)	7 (43.8%)	0.662
CRP, mg/L	2 (1-4)	2 (1-5)	3 (2-8)	0.123	3 (1-16)	2 (1-4)	2 (1-4)	0.460
CRP≥5	0 (0%)	19 (23.5%)	8 (30.8%)	0.065	3 (30.0%)	21 (21.9%)	3 (18.8%)	0.791
Patient GDA	5.0 (1.8-7.0)	4.0 (2.0-6.0)	4.0 (1.8-7.0)	0.949	4.0 (0.3-6.0)	5.0 (2.0-7.0)	3.5 (1.3-5.0)	0.835
BASFI	4.0 (1.3-5.3)	2.8 (1.5-5.6)	2.1 (1.0-5.1)	0.414	6.2 (0.9-6.7)	2.8 (1.5-5.3)	1.4 (1.0-3.7)	**0.035**
ASQOL	6.0 (3.0-8.0)	4.0 (2.0-9.8)	5.0 (0.0-9.0)	0.674	4.0 (1.0-8.0)	5.0 (1.5-5.3)	3.5 (0.3-8.5)	0.995
CORS								
Coping with pain								
· Comforting cognitions	25.0 (23.0-32.0)	28.0 (24.3-29.0)	28.0 (26.0-30.5)	0.571	30.5 (24.5-32.0)	27.5 (24.0-29.0)	28.0 (26.0-34.5)	0.956
· Decreasing activities	20.0 (17.0-26.0)	21.0 (17.0-23.0)	18.0 (17.0-20.5)	0.498	21.5 (17.0-27.0)	21.0 (17.0-23.3)	17.0 (14.5-19.5)	**0.049**
· Diverting attention	19.0 (17.0-26.0)	20.0 (17.3-23.0)	20.5 (19.0-23.3)	0.430	21.5 (18.0-25.5)	20.0 (13.0-18.0)	20.0 (16.5-27.5)	0.989
Coping with limitations								
· Optimism	15.0 (13.0-19.0)	16.0 (14.0-18.0)	15.5 (14.0-18.3)	0.819	17.5 (15.3-19.0)	15.0 (13.0-18.0)	17.0 (14.5-20.0)	0.805
· Pacing	28.0 (23.0-34.0)	28.0 (23.0-31.0)	27.0 (20.8-30.5)	0.321	32.5 (26.0-37.3)	28.0 (23.0-31.5)	24.0 (18.5-30.0)	**0.040**
· Creative solution	22.0 (19.0-26.0)	23.0 (19.0-25.0)	22.0 (19.5-25.0)	0.780	30.5 (23.0-31.3)	22.0 (18.5-25.0)	22.0 (19.0-26.5)	**0.045**
Coping with dependence								
· Accepting dependence	17.0 (9.8-18.0)	14.5 (11.0-17.8)	13.5 (9.8-17.3)	0.530	14.0 (11.0-18.0)	15.0 (11.0-18.0)	13.0 (10.5-17.0)	0.744
· Showing consideration	19.0 (12.8-21.8)	20.0 (17.0-23.0)	17.5 (15.8-21.0)	0.895	23.0 (22.0-24.0)	19.0 (16.8-22.0)	19.0 (16.0-21.5)	0.061

"Δ" refers to the difference between baseline and the second timepoint. Comparisons were only conducted between increase and decrease PA groups.

higher BMI, BASDAI, ASDAS, patient GDA, BASFI and ASQoL (Table I). Similar patterns were observed across domains. Most patients were stable in PA over time: 67.2% by total mSQUASH and 79.4% by leisure/sport domain scores; even more patients remained PA-stable in total minutes. Patients with decreased PA were older, whereas no other significant differences were found between patients with decreased and increased mSQUASH total scores. Patients with decreased leisure/sport scores had higher BASFI and more passive coping strategies at baseline (Table II).

Conclusion. PA patterns differed by sex and coping strategies influenced PA trends. Overall, PA levels generally remained stable over time in axSpA patients without targeted intervention.

References

1. CARBO MJG *et al*.: The mSQUASH; a valid, reliable and responsive questionnaire for daily physical activity in patients with axialspondyloarthritis. *Semin Arthritis Rheum* 2021; 51(4): 719-27.
2. KIESKAMP SC *et al*.: Interrelationships of disease activity, central sensitization, psychosocial and lifestyle factors in axial spondyloarthritis. *Rheumatology* (Oxford) 2025; 64(6): 3547-55.
3. VAN DER KRAAN YM *et al*.: Interrelationships of disease activity, central sensitization, psychosocial and lifestyle factors in axial spondyloarthritis. *RMD Open* 2024; 10(4).

P18

AXIAL SPONDYLOARTHRITIS POPULATION PERSPECTIVE OF VIRTUAL REALITY REHABILITATION: A QUALITATIVE INTERVIEW STUDY

Kattan A.[1], Cazzola D.[1], Rouse P.[1], Wade L., Sengupta R.[2]
[1]Dept. for Health, University of Bath, Bath; [2]Royal National Hospital for Rheumatic Diseases, Bath, UK

Introduction. Axial spondyloarthritis (axSpA) is a chronic inflammatory disease causing pain, stiffness and reduced spinal mobility. During flares, increased pain, fatigue and fear of movement (kinesiophobia) can limit physical activity. Although exercise is recommended, staying active during flares is difficult due to concerns about symptom exacerbation. Virtual reality (VR) can support pain management through distraction and immersion mechanism, but its feasibility and acceptability in axSpA are unclear. This study explored axSpA patients' perceptions of VR exercise during flares.

Methods. Fifteen people with axSpA completed an online questionnaire (demographic/clinical information and Tampa Scale for Kinesiophobia (TSK) during flares) and took part in semi-structured online interviews via Microsoft Teams. After a short presentation introducing VR exercise, interviews explored feasibility and acceptability. Transcripts were analysed using thematic analysis.

Results. All participants reported high kinesiophobia (TSK 39–50; ≥37 indicates high kinesiophobia). Three themes were identified. In the feasibility theme most participants showed willingness to try VR during flares, valuing its potential for immersive distraction and viewing it as a non-pharmacological tool for pain management to maintain physical activity during flare. in acceptability theme Participants expressed curiosity and openness to VR as a way to make exercise more enjoyable and motivating. Most participants preferred purposeful, sport-like immersive activities (*e.g*., tennis, golf) and home-based use for flexibility. In the concerns theme, participants worried about symptom exacerbation due to headset weight, neck pain, session duration, and limited ability to remain standing, and highlighted the need for seated options. They also raised concerns about home use, including balance loss and risk of injury, and noted that limited familiarity with VR would require training and safety guidance.

Conclusion. Participants expressed acceptance and openness to experience VR as a rehabilitation method during flares. This creates opportunities for future feasibility and pilot studies, while accommodating participants' concerns within study design.

Acknowledgements. This research sponsored by King Abdullah Medical City, Makkah, via the Saudi Ministry of Education through the Saudi Cultural Bureau in London as part of the author's PhD studentship, and hosted at the University of Bath, UK.

P19

SLC39A14-DEPENDENT IRON UPTAKE IN FIBROBLASTS DRIVES PATHOLOGICAL NEW BONE FORMATION IN SPONDYLOARTHRITIS

Zhuo D., Zu L., Wang Y., Tang K., Wang J., Liu J.
Fudan University, Shanghai, China

Introduction. Radiographic axial spondyloarthritis (r-axSpA) is characterized by chronic inflammation, fibrosis and pathological new bone formation. Although systemic iron dysregulation has been reported in inflammatory diseases, whether local iron accumulation directly drives fibroblast activation, extracellular matrix (ECM) remodeling and osteogenic niche formation remains unclear. This study investigated how inflammation-induced iron dysregulation contributes to pathological ossification in r-axSpA, with a focus on SLC39A14-mediated iron influx in fibroblasts.

Methods. Blood and tissue samples from r-axSpA patients and healthy controls were analyzed for iron-related parameters and their association with disease activity. Local iron deposition was assessed by Prussian blue staining, and ferritin colocalization with fibroblasts and macrophages was examined by immunofluorescence. Single-cell RNA sequencing was performed to identify iron-responsive cell populations and ECM-related transcriptional programs. *In vitro*, fibroblasts and tendon-derived cells were exposed to iron overload, and ECM gene expression was evaluated by qRT-PCR and Western blotting. SLC39A14 was genetically disrupted to determine its functional role. *In vivo*, iron overload and SLC39A14 conditional knockout models were used to assess ECM accumulation and osteophyte formation.

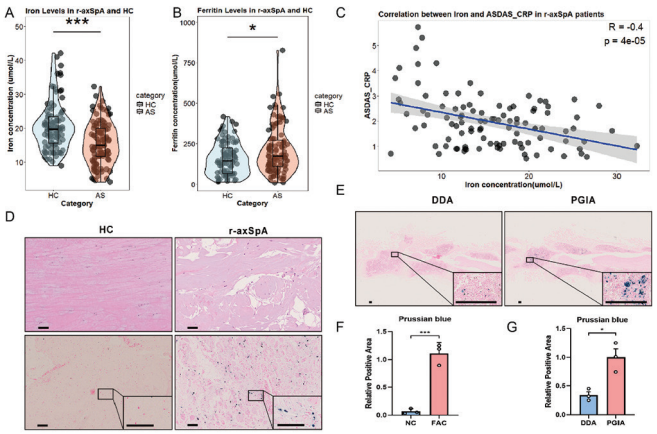


Fig. 1. Iron dysregulation in r-axSpA and its correlation with disease activity.
Single-cell analysis identified fibroblasts as major iron-responsive cells and revealed enrichment of iron-handling, ECM remodeling and osteogenic pathways (Fig. 2). Iron overload promoted fibroblast ECM production, whereas SLC39A14 deletion reduced ECM accumulation and osteophyte formation *in vivo*.

Results. r-axSpA patients showed reduced serum iron and increased ferritin, and serum iron was negatively correlated with ASDAS-CRP. Local iron deposition was increased in r-axSpA lesions and inflammatory arthritis models (Fig. 1).

Conclusion. Inflammation-driven SLC39A14-mediated iron influx activates fibroblasts, promotes pathological matrix deposition and supports osteogenic niche formation in r-axSpA. Targeting fibroblast iron handling may represent a therapeutic strategy for inflammatory pathological ossification.

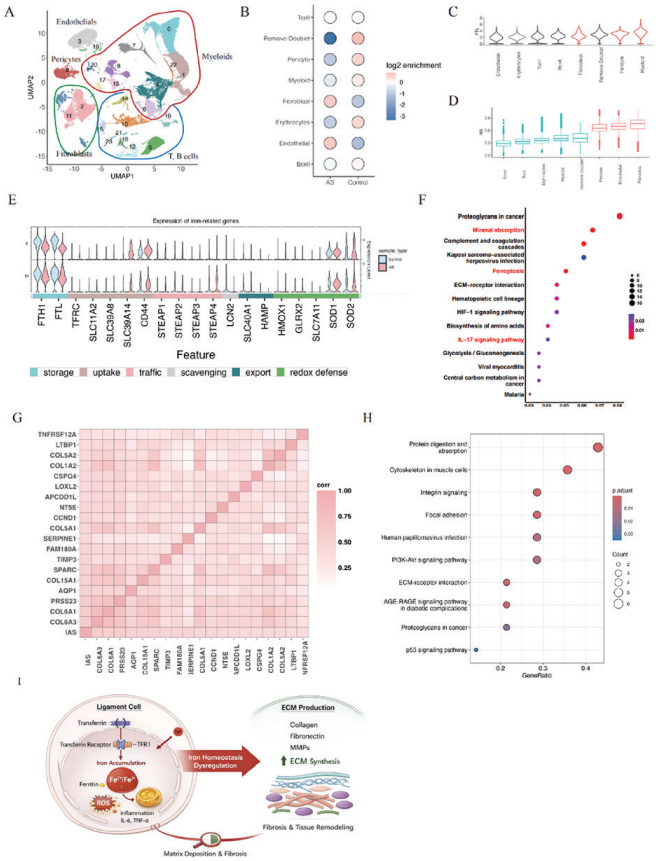


Fig. 2. Single-cell analysis reveals iron-related gene expression and pathways in r-axSpA.

P20

INSTRUMENT SELECTION FOR THE ASSESSMENT OF PERIPHERAL ARTHRITIS IN AXIAL AND PERIPHERAL SPONDYLOARTHRITIS: RESULTS FROM ASAS-SPARADISE

Ramiro S.[1,2], Capelusnik D.[3,4], López-Medina C.[5], Ortolan A.[6,7], Webers C.[3,8], Baraliakos X.[9], Bautista-Molano W.[10], Boonen A.[3,8], Van den Bosch F.[11], Carron P.[11], van Dam M.[12], van Gaalen F.[1], Gensler L.S.[13], Gossec L.[14,15], Hmamouchi I.[16], Kiltz U.[9], Kishimoto M.[17], Lowe J.[18], Machado P.M.[19], Navarro-Compán V.[20], Nikiphorou E.[21,22,23], Pina Vegas L.[24,25], Poddubnyy D.[26,27], van de Sande M.[28,29], Sepriano A.[30], Sieper J.[27], Varkas G.[11], Mease P.[31], Landewé R.[2,28], van der Heijde D.[1], Moltó A.[32]

[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden; [2]Dept. of Rheumatology, Zuyderland Medical Center, Heerlen; [3]Care and Public Health Research Institute (CAPHRI), Maastricht University, Maastricht, The Netherlands; [4]Dept. of Rheumatology, Tel Aviv Sourasky Medical Center, Tel Aviv, Israel; [5]Dept. Rheumatology, University of Cordoba, Reina Sofia University Hospital, Maimonides Biomedical Research Institute of Cordoba (IMIBIC), Cordoba, Spain; [6]Dept. of Rheumatology, Fondazione Policlinico Universitario Agostino Gemelli IRCSS, Rome; [7]Catholic University of the Sacred Heart, Rome, Italy; [8]Dept. of Rheumatology, Maastricht University Medical Centre, Maastricht, The Netherlands; [9]Ruhr-Universität Bochum, Germany and Rheumazentrum Ruhrgebiet, Herne, Germany; [10]Dept. of Rheumatology, University Hospital Fundacion Santa Fe de Bogota, School of Medicine Universidad El Bosque, Bogota, Colombia; [11]VIB-UGent Center for Inflammation Research, Dept. of Internal Medicine and Pediatrics, Ghent University and University Hospital, Dept. of Rheumatology, Ghent, Belgium; [12]Founder Axial Spondyloarthritis Association of South Africa, Patient Research Partner, Patient, South Africa; [13]Dept. of Medicine/Rheumatology, University of California, San Francisco, San Francisco, USA; [14]Sorbonne Université, INSERM, Institut Pierre Louis d'Epidémiologie et de Santé Publique, Team Pepites, Paris; [15]AP-HP, Pitié-Salpêtrière Hospital, Rheumatology Dept., Paris, France; [16]Health Sciences Research Center (CReSS), Faculty of Medicine, International University of Rabat (UIR), Rabat, Morocco; [17]Dept. of Nephrology and Rheumatology, Kyorin University School of Medicine, Tokyo, Japan; [18]Axial Spondyloarthritis International Federation (ASIF), London; [19]Dept. of Neuromuscular Diseases, University College London, London; National Institute for Health Research (NIHR) University College London Hospitals Biomedical Research Centre, University College London Hospitals NHS Foundation Trust, London; Dept. of Rheumatology, Northwick Park Hospital, London North West University Healthcare NHS Trust, London, UK; [20]Dept. of Rheumatology. University Hospital La Paz, IdiPaz, Madrid, Spain; [21]Centre for Rheumatic Diseases, Faculty of Life Sciences and Medicine, King's College London, London; [22]Rheumatology Dept., King's College Hospital NHS Foundation Trust, London; [23]Centre for Education, Faculty of Life Sciences and Medicine, King's College London, London, UK; [24]Dept. of Rheumatology, Henri Mondor Hospital, Créteil; [25]EPILOGY - Inserm, Université Paris-Est Créteil (UPEC), Créteil, France; [26]Division of Rheumatology, Dept. of Medicine, University of Toronto and Schroeder Arthritis Institute, University Health Network, Toronto, Canada; [27]Dept. of Gastroenterology, Infectiology and Rheumatology (including Nutrition Medicine), Charité – Universitätsmedizin Berlin, corporate member of Freie Universität Berlin and Humboldt-Universität zu Berlin, Berlin, Germany; [28]Amsterdam UMC, University of Amsterdam, Dept. of Rheumatology & Clinical Immunology and Dept. of Experimental Immunology, Amsterdam Institute for Immunology & Infectious Diseases, Amsterdam; [29]Amsterdam Rheumatology Immunology Center, Reade and Amsterdam UMC, Amsterdam, The Netherlands; [30]NOVA Medical School, Universidade Nova de Lisboa and Dept. of Rheumatology, Hospital de Egas Moniz, ULSLO, Lisboa, Portugal; [31]Dept. of Rheumatology, Swedish Medical Center, Providence St. Joseph Health and University of Washington, Seattle, WA, USA; [32]Université Paris Cité and Université Sorbonne Paris Nord, INSERM, INRAE, Centre for Research in Epidemiology and Statistics, Paris and Rheumatology Dept., Bichat Hospital, APHP, Paris, France

Introduction/Objective. To identify the most appropriate instrument for assessing disease activity related to peripheral arthritis in axial (axSpA) and peripheral spondyloarthritis (pSpA) in clinical research.

Materials and methods. The ASAS-SPARADISE project followed the OMERACT framework for instrument selection. Candidate instruments were first identified through a systematic literature review (SLR), followed by assessment of domain match and feasibility by an international working group including clinicians and patient research partners. A second SLR evaluated measurement properties (construct validity, reliability, responsiveness, clinical trial discrimination, and thresholds of meaning). To address evidence gaps, additional analyses were performed using individual patient data from 13 randomised controlled trials (RCTs). Evidence was synthesised in Summary of Measurement Properties (SOMP) tables and discussed by the working group, followed by consensus voting at the ASAS annual meeting.

Instruments	Axial spondyloarthritis with peripheral arthritis
PGA	Not endorsed
SJC44	Not endorsed
TJC44	Not endorsed
SJC66	Not endorsed
TJC68	Not endorsed
CRP	Endorsed
BASDAI	Endorsed
ASDAS	Endorsed
DAPSA	Not endorsed
DAPSA44	Endorsed
DAS44	Provisionally endorsed

Colour code: greenendorsed; amberprovisionally endorsed; rednot endorsed.
PGA, Patient Global Assessment; SJC44/66, Swollen Joint Count with 44/66 joint count; TJC44/68, Tender Joint Count with 44/68 joint count; CRP, C-reactive protein; BASDAI, Bath Ankylosing Spondylitis Disease Activity Score; ASDAS, Axial Spondyloarthritis Disease Activity Score; DAPSA, Disease Activity in Psoriatic Arthritis; DAPSA44, Disease Activity in Psoriatic Arthritis with 44 joint count; DAS44, Disease Activity Score with 44 joint count.

Instruments	Peripheral Spondyloarthritis
PGA	Not endorsed
SJC44	Not endorsed
TJC44	Provisionally endorsed
SJC66	Provisionally endorsed*
TJC68	Provisionally endorsed
CRP	Not endorsed
BASDAI	Provisionally endorsed*
ASDAS	Provisionally endorsed*
DAPSA	Provisionally endorsed*
DAPSA44	Provisionally endorsed*
DAS44	Provisionally endorsed*

Colour code: greenendorsed; amberprovisionally endorsed; rednot endorsed
*The *"provisionally endorsed"* status were due to the scarcity of data, with only one study (showing good performance) for some measurement properties, which therefore resulted in an AMBER.
PGA, Patient Global Assessment; SJC44/66, Swollen Joint Count with 44/66 joint count; TJC44/68, Tender Joint Count with 44/68 joint count; CRP, C-reactive protein; BASDAI, Bath Ankylosing Spondylitis Disease Activity Score; ASDAS, Axial Spondyloarthritis Disease Activity Score; DAPSA, Disease Activity in Psoriatic Arthritis; DAPSA44, Disease Activity in Psoriatic Arthritis with 44 joint count; DAS44, Disease Activity Score with 44 joint count.

Fig. 1. Final result of the Summary of measurement properties table of each instrument measuring peripheral arthritis, separately for axial spondyloarthritis with peripheral arthritis and for peripheral spondyloarthritis.

Results. Composite disease activity instruments consistently outperformed single-item measures such as joint counts. Evidence from published studies was limited, particularly in axSpA with peripheral arthritis, but was strengthened by RCT analyses. Across both axSpA and pSpA, composite instruments – including BASDAI, ASDAS, and DAPSA44 – demonstrated superior construct validity, responsiveness, and clinical trial discrimination compared with single-item instruments. Clinical trial discrimination was particularly better for DAPSA44, making it the best-performing instrument across all measurement properties (Fig. 1). Following ASAS voting, DAPSA44 was endorsed as the preferred instrument, with 92% agreement for axSpA and 97% for pSpA (Fig. 2).
Conclusion. DAPSA44 demonstrated the most adequate measurement performance for assessing disease activity related to peripheral arthritis across axSpA and pSpA. It is now recommended by ASAS as the preferred instrument for use in clinical research. Future clinical trials and research studies should report DAPSA44 when analysing disease activity related to peripheral arthritis in SpA.

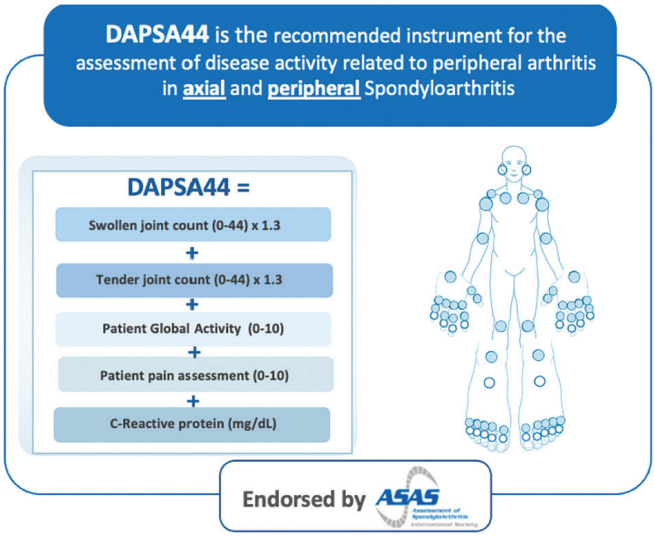


Fig. 2. ASAS recommendation for the assessment of disease activity related to peripheral arthritis in clinical research.
ASAS: Assessment of Spondyloarthritis international Society; SpA: spondyloarthritis; DAPSA44: Disease Activity in Psoriatic Arthritis with 44 joint count.

Acknowledgements. We would like to thank patients who contributed to the study, patient research partners from ASIF involved and ASAS members.
Funding. The ASAS-SPARADISE project was funded by the Assessment of Spondyloarthritis international Society (ASAS).

P21

METABOLIC AND PROTEOMIC ALTERATIONS ASSOCIATED WITH HEPATIC STEATOSIS AND FIBROSIS IN PSORIATIC ARTHRITIS

Ruiz-Ponce M.[1], Martín-Salazar J.E.[1], Arias-de la Rosa I.[1,2], Barranco A.M.[1], López Montilla M.D.[1], Romero Zurita L.[1,3], Pérez-Sánchez C.[3,4], Abalos-Aguilera M.C.[1], Ruiz-Vilchez D.[1], Ortiz-Buitrago P.[1], López-Pedrera C.[1], Collantes-Estévez E.[1], López-Medina C.[1], Escudero-Contreras A.[1], Barbarroja N.[1,3]
[1]Rheumatology service/Dept. of Medical and Surgical Sciences, Maimonides Institute for Research in Biomedicine of Cordoba (IMIBIC)/ University of Cordoba/ Reina Sofia University Hospital, Córdoba, Spain, Córdoba; [2]Dept. of Gastroenterology. Hospital General de Tomelloso, Spain/ Health Research Institute of Castilla-La Mancha (IDISCAM), Toledo & Biomedical Research Networking Center for Liver and Digestive Diseases (CIBERehd), Madrid; [3]Cobiomic Bioscience S.L. Cordoba; [4]Dept. of Cell Biology, Physiology and Immunology, Maimonides Institute of Biomedical Research of Cordoba (IMIBIC), Reina Sofia University Hospital, University of Córdoba, Córdoba, Spain

Introduction. Psoriatic arthritis (PsA) is associated with increased cardiometabolic risk and a higher prevalence of metabolic liver disease. However, the molecular mechanisms underlying hepatic involvement in PsA remain poorly characterized. This study aimed to evaluate hepatic steatosis and fibrosis in non-obese PsA patients and analyze their association with serum metabolomic-lipidomic and proteomic profiles.
Methods. Cross-sectional study including 42 non-obese PsA patients. Clinical, biochemical, and hepatic risk parameters were assessed. Liver elastography (FibroScan®) was performed to evaluate fibrosis using the controlled attenuation parameter (CAP) and hepatic steatosis. Serum metabolomic-lipidomic profiling quantified 250 metabolites, while serum proteomics analyzed 276 proteins using three Olink® panels (cardiovascular, inflammation, and organ damage).
Results. Mean age was 56.2±13.8 years, with moderate disease activity (DAPSA 11.2±6.8). Despite generally normal conventional metabolic parameters, liver indices suggested increased subclinical hepatic risk. Hepatic steatosis was detected in 46.3% and fibrosis in 12.2% of patients. Unsupervised metabolomic analysis identified two distinct patient clusters. Cluster 1 showed higher triglycerides, ApoB, LDL cholesterol, TyG, FLI, and CAP values, together with lower HDL cholesterol and marked alterations in lipoprotein subfractions. Proteomic analysis revealed increased expression of proteins related to vascular inflammation, tissue remodeling, and cardiometabolic dysfunction. Comparison of PsA patients with versus without steatosis revealed distinct metabolic and proteomic signatures, with 37 metabolites associated with steatosis and 19 with fibrosis. Steatosis was characterized by triglyceride-rich VLDL particles, HDL alterations, elevated branched-chain amino acids, and insulin resistance-related changes. Fibrosis was associated with VLDL/LDL remnant alterations, fatty acid remodeling, and increased sphingomyelins. Proteomic enrichment analysis linked fibrosis to inflammatory and immune-related pathways, whereas steatosis was primarily associated with metabolic and vascular signaling pathways.
Conclusion. Non-obese PsA patients exhibit distinct metabolomic and proteomic signatures associated with subclinical hepatic steatosis and fibrosis, suggesting potential utility for early hepatic and cardiometabolic risk stratification.
Acknowledgements. Supported by the Minister of Science, Innovation and Universities (PID2023-152503OB-I00).

P22

ANXIETY, DEPRESSION, AND FIBROMYALGIA COMPROMISE TARGET ATTAINMENT IN AXIAL SPONDYLOARTHRITIS REGARDLESS OF INFLAMMATORY ACTIVITY

Vieira V.[1], Bassara-Macedo R.[1], Resende G.G.[2], Marques C.[3], Pinheiro M.M.[4], Marinho A.[5], Soares A.M.[6], Albuquerque C.[7], Rodrigues D.[5], Castro G.[8], Bulbol G.[9], Carneiro J.[10], Fernandes J.M.[11], Orchtrop M.[12], Gavi M.B.[13], Yazbek M.[14], Cavalcanti N.[3], Machado N.[15], Malheiro O.[2], Vieira R.[16], Lage R.[2], Menin R.C.[17], Golebiovski R.[4], Ribeiro S.[9], Oliveira T.[4], Diniz V.[13], Sampaio-Barros P.[1], Saad C.G.S.[1]
[1]Faculdade de Medicina da Universidade de São Paulo (USP) – SP; [2]Hospital das Clínicas da Universidade Federal de Minas Gerais (UFMG) – MG; [3]Hospital das Clínicas da Universidade Federal de Pernambuco (UFPE) – PE; [4]Escola Paulista de Medicina (EPM) - Universidade Federal de São Paulo (UNIFESP) – SP; [5]Fundação Hospital do Acre – AC; [6]Hospital Universitário da Universidade Federal de Santa Catarina (UFSC) – SC; [7]Hospital Universitário de Brasilia; PPG em Ciências Médicas; FM/UnB – DF; [8]Hospital Governador Celso Ramos (HCR) – SC; [9]Faculdade de Medicina da Universidade Federal do Amazonas (UFAM) – AM; [10]Hospital de Base do Distrito Federal (HBDF) – DF; [11]Universidade Federal do Maranhão (UFMA) – MA; [12]Hospital Universitário Pedro Ernesto / UERJ – RJ; [13]Universidade Federal do Espírito Santo (UFES) – ES; [14]Universidade Estadual de Campinas (UNICAMP) – SP; [15]Universidade Federal do Paraná (UFPR); [16]Universidade Estadual do Ceará (UECE) – CE; [17]Faculdade de Medicina de São José do Rio Preto (FAMERP) – SP, Brazil

Introduction. Treat-to-target in axial spondyloarthritis (axSpA) relies on composite scores (ASDAS, BASDAI) that incorporate patient-reported outcomes. Coexisting mental health and central sensitization conditions (e.g. depression, anxiety and fibromyalgia), may increase these scores independently of inflammation. However, their impact on target attainment in real-world axSpA patients is poorly defined.
Methods. Cross-sectional analysis of the Brazilian Registry of Spondyloarthritis (RBE), including 19 centers nationwide. All patients were diagnosed with axSpA by a rheumatologist. Anxiety, depression, and fibromyalgia were recorded as present/absent and collectively analyzed as "Coexisting factors". Outcomes were BASDAI, ASDAS-CRP, and failure to reach remission (ASDAS<1.3) or low disease activity (ASDAS<2.1; BASDAI<4).

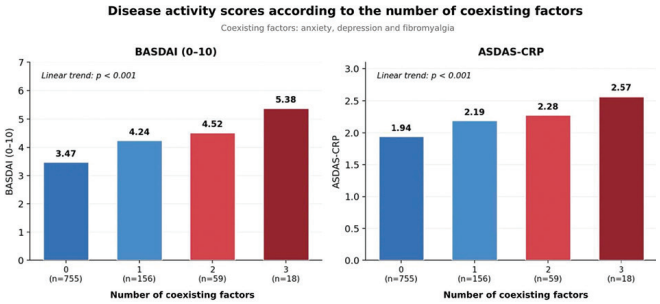


Fig. 1.

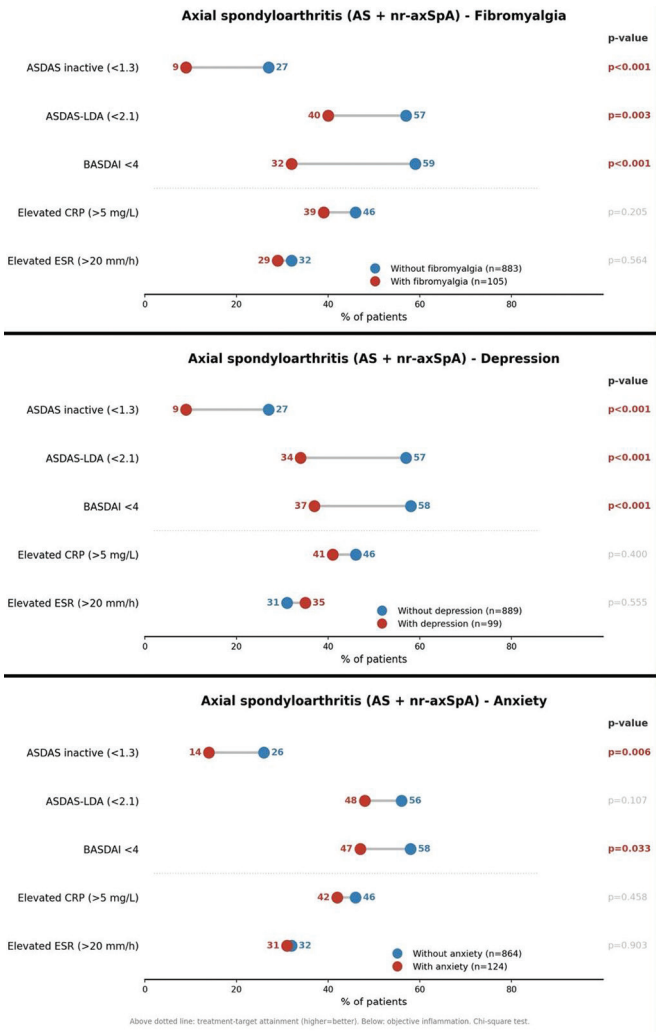


Fig. 2.

Separate linear and logistic regression models for each factor were adjusted for sex, age, disease duration, BMI, CRP, and ESR. A predefined normal-CRP subgroup was analyzed separately. Cumulative affective burden was modeled as the number of factors (0–3).

Results. A total of 988 patients were analyzed. Coexisting factors were present in 23.6% (anxiety-12.6%, depression-10.0%, fibromyalgia-10.6%). Mean BASDAI was higher with anxiety(p=0.005), depression (p<0.001), and fibromyalgia (p<0.001); mean ASDAS-CRP was also more elevated in such conditions (anxiety, p=0.033; depression, p<0.001; fibromyalgia, p<0.001). CRP and ESR did not differ between groups. In adjusted linear models, fibromyalgia (BASDAI, p<0.001; ASDAS-CRP, p=0.004) and depression (BASDAI, p=0.005; ASDAS-CRP, p=0.008) independently predicted higher scores. Each additional coexisting factor increased BASDAI by 0.48 and ASDAS-CRP by 0.16 (both p<0.001) (Fig. 1). Overall, remission was not achieved by 75.2%, while 45.2% failed ASDAS-LDA and 43.9% failed BASDAI<4. Coexisting factors increased adjusted odds of not achieving remission: fibromyalgia OR4.70, depression OR4.43, anxiety OR2.29; all p<0.01(Fig. 2). In the normal-CRP subgroup(n=513), fibromyalgia remained independently associated with higher BASDAI (p<0.001) and ASDAS-CRP (p<0.001).

Conclusion. Anxiety, depression, and fibromyalgia are associated with higher patient-reported disease activity and reduced attainment of therapeutic targets in axSpA, independently of objective inflammation. As important contributors to a difficult-to-manage disease state, these conditions should be actively identified and addressed in patients with persistent symptoms.

P23

CLINICAL CHARACTERISTICS AND DRUG SURVIVAL IN A COHORT OF PATIENTS WITH LATE-ONSET SPONDYLOARTHRITIS

Alcántara Álvarez I.[1], Grau García E.[1,2], Muñoz-Martínez P.F.[1], Torrat Noves A.M.[1], Ramos Castro D.[1], Villanueva Mañes B.[1], Simeó Vinaixa, M.[1], Pérez Hurtado A.[1], Díaz Mora B.[1], Antón Enguidanos P.[1], Román Ivorra J.A.[1]
[1]Rheumatology Dept., Hospital Universitari i Politècnic La Fe, València; [2]Institut d'Investigació Sanitària La Fe, València, Spain

Introduction. Late-onset spondyloarthritis (LO-SpA) is defined in patients presenting with the disease after being 45 years-old. It accounts for 3.5–14% of all cases, mainly affects women, and is characterized by lower HLA-B27 positivity, greater peripheral involvement, and a higher prevalence of psoriasis. Regarding response and drug survival to advanced therapy, the literature shows conflicting evidence.

Methods. Single-center, retrospective, longitudinal observational study of SpA patients treated with at least one advanced therapy between January 2010 and June 2023. Clinical, demographic, and therapeutic data were extracted from electronic health records.

Results. A total of 180 patients (64.6% male) with 259 treatment lines were included; 28.3% were classified as LO-SpA. In the LO-SpA group, 76.5% had a radiographic phenotype, and 41.3% were HLA-B27+ (different from non–LO-SpA, p=0.007).

Among LO-SpA patients, 29.4% presented extra-musculoskeletal manifestations (EMMs), mainly uveitis (15.7%), followed by inflammatory bowel disease (13.7%), and with psoriasis being the least frequent (5.9%). The LO-SpA group showed fewer peripheral manifestations, especially arthritis (p=0.013). No differences between groups were observed regarding acute-phase reactants. In addition, within the LO-SpA population we observed a higher prevalence of hypertension (p=0.001), dyslipidemia (p<0.001), osteoarthritis (p<0.001), and osteoporosis (p=0.006).

The main therapy used in LO-SpA patients was anti-TNF agents (56.9%), followed by IL-17 inhibitors (33.3%). A higher proportion of patients requiring more than two treatment families was observed in the LO-SpA

Table I.

Variable	Non LO-SpA n=129	LO-SpA n=51
Age at symptom onset, years	30.87 (8.41)	52.86 (8.15)
Age at diagnosis, years	34.57 (9.51)	54.18 (7.39)
Months from symptom onset to diagnosis	4.11 (5.32)	1.81 (2.15)
Months from diagnosis to treatment	28.41 (38.72)	16.88 (21.26)
CRP	12.16 (16.79)	10.6 (17.2)
ESR	20.08 (16.25)	24.86 (21.12)
Sex (male)	88 (68.22%)	29 (56.86%)
Phenotype – non-radiographic axSpA	31 (24.6%)	12 (23.53%)
Phenotype – radiographic axSpA	95 (75.4%)	39 (76.47%)
Patients with ≥3 therapeutic families	5 (3.88%)	6 (11.76%)
Patients with ≥2 treatments	26 (20.16%)	15 (29.41%)
Therapeutic target – anti-TNF	88 (68.22%)	29 (56.86%)
Therapeutic target – IL-17	39 (30.23%)	17 (33.33%)
Therapeutic target – JAK inhibitor	2 (1.55%)	1 (1.96%)
Therapeutic target – others	0 (0%)	4 (7.84%)
HLA-B27 positivity	83 (68.6%)	19 (41.3%)
Family history	10 (11.9%)	0 (0%)
EMMs	36 (27.91%)	15 (29.41%)
Osteoporosis	2 (1.55%)	7 (14%)
Hypertension	14 (10.85%)	20 (40%)
Diabetes	3 (2.33%)	3 (6%)
Dyslipidemia	26 (20.16%)	22 (44%)
Obesity	16 (14.04%)	8 (17.39%)
Osteoarthritis	7 (5.51%)	14 (28%)

group (p=0.0382). No differences in drug survival were observed in the LO-SpA population.

Conclusion. In our cohort of LO-SpA patients, similar rates of psoriasis and a low prevalence of peripheral involvement, particularly arthritis, were observed. This group presented more comorbidities, with statistically significant differences in hypertension, dyslipidemia, osteoarthritis, and osteoporosis. No differences were observed in acute-phase reactants or in drug survival across treatment lines; however, LO-SpA patients showed a higher likelihood of requiring ≥3 lines of therapy.

P24

IMPACT OF HLA-B27 ON CLINICAL PRESENTATION AND IMAGING IN AXIAL SPONDYLOARTHRITIS IN LATIN AMERICA: INSIGHTS FROM THE ESPALDA-PANLAR REGISTRY

Bautista-Molano W.[1], Garcia-Salinas R.[1], Casasola J.C.[2], Sommerfleck F.[3], Guaracha Basañez G.A.[4], Londono J.[5], Gamboa-Cardenas R.V.[6], Fernández-Ávila D.G.[7], Vila D.[8], Mestanza Peralta M.[9], Then J.[10], Dufau E.[11], Bermudez Bermejo P.E.[12], Macias Oviedo L.[13], Duran Barragan S.[14], Valdez Donelli F.[15], Barcia Cansino A.[16], Medina G.[17], Casalla L.[18], Castillo Ortiz A.[19], Chinchay L.[20], Caballero L.[21], Benegas M.[3], Medina Bornachera D.[22], Vega C.[23], Melgarejo P.[21], Belén Gómez A.[24], Ferreyra Garrot L.G.[25], Candia Zuniga D.L.[26], Gil-Calderon D.[27], Rivera Terán V.[28], Eguez Del Pozo M.F.[29], Sanchez Prado E.[30], Avellanal V.[24], Brance M.L.[31], Marin Zucaro N.[31], on Behalf of ESPALDA-PANLAR

[1]Fundación Santa Fé, Bogota, Colombia; [2]Hospital Italiano de La Plata, La Plata, Argentina; [3]Hospital General de Mexico, Mexico, Mexico; [4]Instituto Nacional de Ciencias Médicas y Nutrición "Salvador Zubirán", Mexico, Mexico; [5]Hospital Militar Central, Universidad de La Sabana, Bogota; [6]Hospital Nacional Guillermo Almenara Irigoyen, Lima, Peru; [7]Hospital San Ignacio, Bogota, Colombia; [8]Hospital Virgen del Carmen, Zarate, Argentina; [9]Hospital Metropolitano, Quito, Ecuador; [10]Hospital Metropolitano de Santiago, Santiago de los Caballeros, Dominican Republic; [11]Sanatorio Guemes, Buenos Aires, Argentina; [12]Centro Medico ABC, Mexico, Mexico; [13] Consultorio reumaart.ec, Guayaquil, Ecuador; [14]Clínica de Investigación en Reumatología y Obesidad S.C., Guadalajara, Mexico; [15]IREP, Buenos Aires, Argentina; [16]Hospital General Portoviejo del Instituto Ecuatoriano de Seguridad Social (IESS), Portoviejo, Ecuador; [17]Hospital de Clínicas José de San Martín, Buenos Aires; [18]Genea Centro Médico, Buenos Aires, Argentina; [19]Hospital Faro del Mayab Grupo Christus Muguerza, Merida, Mexico; [20]Hospital Guillermo Kaelin de La Fuente, Piura, Peru; [21]Instituto de Previsión Social, Asunción, Paraguay; [22]Red de Investigación Colaborativa del Caribe Colombiano (RICER), Barranquilla, Colombia; [23]Hospital General ISSSTE Tacuba, Mexico, Mexico; [24]Instituto Nacional de Reumatología del Uruguay, Montevideo, Uruguay; [25]Hospital Italiano de Buenos Aires, Buenos Aires, Argentina; [26]Hospital Regional Primero de Octubre ISSSTE, Fuerzas Armadas, Mexico, Mexico; [27]Artemedica SAS, Bogota, Colombia; [28]Instituto Nacional de Rehabilitación, Mexico, Mexico; [29]Altius Medical, Guayaquil; [30]Hospital del IESS Los Ceibos, Guayaquil, Ecuador; [31]UI PANLAR, Rosario, Argentina

Background/Objective. HLA-B27 is a major genetic determinant of axial spondyloarthritis (axSpA), associated with disease susceptibility and clinical heterogeneity. Most phenotype–genotype evidence derives from Europe and North America. Understanding HLA-B27-associated features in Latin America may improve risk stratification and disease characterization to determine the prevalence of HLA-B27 positivity in ESPALDA and to compare demographic, clinical, inflammatory, and imaging features between HLA-B27-positive and HLA-B27-negative patients, as well as to identify independent predictors of HLA-B27 positivity.

Methods. ESPALDA is a multicenter Latin American registry of patients with axSpA recruited from referral centers. Baseline data included demographics, patient-reported outcomes, inflammatory markers (CRP and ESR), and imaging variables, including radiographic sacroiliitis and MRI findings such as bone marrow edema and structural lesions. Patients were stratified according to HLA-B27 status (positive vs. negative), and group comparisons were performed using appropriate tests for categorical and continuous variables. A multivariable logistic regression model was used to identify variables independently associated with HLA-B27 positivity, with particular attention to demographic characteristics, imaging involvement, and extra-musculoskeletal manifestations.

Results. Among 740 patients, 61.8% were HLA-B27 positive. Compared with HLA-B27-negative patients, HLA-B27-positive individuals were younger at enrollment and had an earlier onset of low back pain. Overall clinical activity indices were similar between groups; however, HLA-B27-positive patients showed a trend toward a higher inflammatory burden and

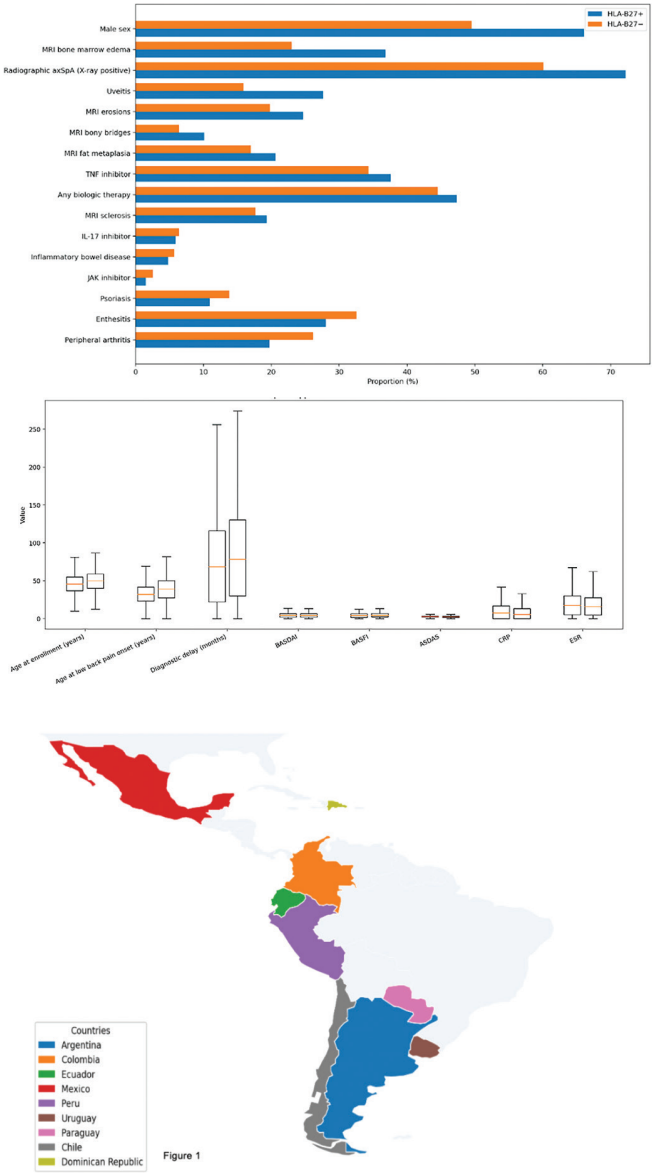


Figure 1

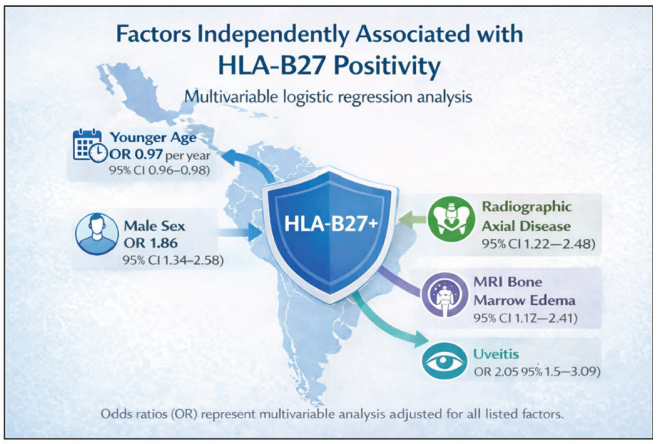


Fig. 1.

a more axial-dominant imaging phenotype. Radiographic axSpA, defined by sacroiliitis on plain radiographs, was more frequent in the HLA-B27-positive group, and MRI inflammatory lesions, particularly bone marrow edema, were also more common, supporting a stronger axial inflammatory signature. In contrast, peripheral arthritis and enthesitis showed limited differences between groups, suggesting that peripheral involvement remains relevant across genotypes. Among extra-musculoskeletal manifestations, uveitis was notably more frequent in HLA-B27-positive patients, consistent with a genotype-associated ocular phenotype. In multivariable analysis, younger age, male sex, radiographic axial disease, MRI inflammation, and uveitis remained independently associated with HLA-B27 positivity.

Conclusion. HLA-B27 positivity was common in ESPALDA and was associated with younger age and earlier symptom onset, supporting a distinct demographic profile. HLA-B27-positive patients also showed a higher frequency of axial imaging involvement, including radiographic sacroiliitis and MRI inflammation, suggesting an imaging-enriched axial phenotype. Uveitis was more frequent in HLA-B27-positive patients and remained independently associated with HLA-B27 positivity, reinforcing its clinical relevance in this subgroup. Peripheral manifestations were observed across both strata, supporting continued multidomain assessment regardless of HLA-B27 status.

Table I. Baseline characteristics of included axSpA participants.

Variable	AxSpA (n=83)
Age, years (mean/SD)	43.3 (12.9)
Female sex (N/%)	42 (50.6%)
White (N/%)	68 (81.9%)
BMI, kg/m^2 (mean/SD)	29.7 (8.54)
Current smokers (N/%)	8 (9.6%)
Current alcohol use (N/%)	36 (43.4%)
Current drug use (N/%)	14 (16.9%)
Anxiety/depression (N/%)	53 (63.9%)
Hx of fibromyalgia (N/%)	10 (12.0%)
Hx of peripheral arthritis (N/%)	15 (18.1%)
Hx of uveitis (N/%)	29 (34.9%)
Hx of IBD (N/%)	8 (9.6%)
Hx of psoriasis (N/%)	12 (14.5%)
Disease duration, years (mean/SD)	19.4 (13.9)
Delay in diagnosis, years (mean)	8.9
HLA-B27 positivity (N/%)	67 (80.7%)
BASDAI (mean/SD)	3.58 (2.19)
ASDAS-CRP (mean/SD)	1.99 (1.03)
CRP elevation (N/%)	8 (9.6%)
b/tsDMARD use (N/%)	69 (83.1%)
TNFi use (N/%)	53 (63.9%)
IL17i use (N/%)	10 (12.0%)
JAKi use (N/%)	5 (6.0%)
Neuropathic med use (N/%)	25 (30.1%)
Opioid use (N/%)	7 (8.4%)
Nr-axSpA (N/%)	42 (50.6%)
Global inflammation Y/N (N/%)	14 (16.9%)
FSQ (mean/SD)	10.3 (5.79)
NRS, 0-10 (mean/SD)	3.72 (2.43)

SD: standard deviation, BMI: body mass index, Hx: history, IBD: inflammatory bowel disease, BASDAI: bath ankylosing spondylitis disease activity index, ASDAS-CRP: axial spondyloarthritis disease activity score, CRP: c-reactive protein, b/tsDMARD: biologic/targeted synthetic disease modifying antirheumatic drugs, TNFi: tumor necrosis factor inhibitor, IL17i: interleukin-17 inhibitor, JAKi: Janus kinase inhibitor, nr-axSpA: non-radiographic axial spondyloarthritis, FSQ: fibromyalgia survey questionnaire, NRS: numeric rating scale.

P25

CHARACTERIZING NOCIPLASTIC PAIN IN AXIAL SPONDYLOARTHRITIS

Bittar M.[1], Choi D.[2], Engel A.[1], Melz I.[1], Gaydish C.[1], Deodhar A.[1], Lee Y.C.[3]
[1]Division of Arthritis & Rheumatic Diseases, Oregon Health & Science University, Portland, OR; [2]Dept. of Biostatistics, Oregon Health & Science University, Portland, OR; [3]Division of Rheumatology, Northwestern University, Chicago, IL, USA

Introduction/Objective. Chronic pain is one of the most common causes of disability in axial spondyloarthritis (axSpA). Its mechanism is not well understood, hindering the development of efficacious therapeutics. This study aims to estimate the prevalence of nociplastic pain in a well-phenotyped axSpA cohort and to examine associations between symptom burden, disease activity, and quantitative sensory testing (QST).

Materials and methods. This is a cohort study enrolling adults with axSpA who meet the 2009 ASAS classification criteria. Participants complete validated questionnaires including the Fibromyalgia Survey Questionnaire (FSQ), which includes the Widespread Pain Index and Symptom Severity Scale. Patients undergo QST, including pain pressure threshold (PPT), temporal summation (TS), and conditioned pain modulation (CPM). Participants with pain undergo C-reactive protein (CRP) and MRI of the sacroiliac joint testing to assess inflammation. FSQ≥13 suggests nociplastic pain features. In a cross-sectional approach, we performed Pearson correlation analysis between QST, FSQ and axSpA disease activity score (ASDAS-CRP), as well as linear regression analysis to study the difference in QSTs between participants with and without pain, adjusting for several factors including age, sex, and inflammation status.

Results. Eighty-three participants are enrolled to date: 51% female, 82% White, with a mean age of 43 years. Features consistent with nociplastic pain were present in 32.5%. FSQ is strongly correlated with PPTs and right trapezius TS, but not with left TS and CPM. FSQ showed strong correlations with ASDAS-CRP. In adjusted analyses, participants with nociplastic pain features (FSQ≥13) had significantly lower PPTs compared to those without. There was no statistical difference in TS and CPM. Female sex was strongly associated with FSQ≥13(OR 14, *p*-value 0.02).

Conclusion. Features consistent with nociplastic pain phenotype are common in axSpA and are associated with lower PPTs. ASDAS-CRP correlates with nociplastic symptom burden, suggesting that patient-reported components may reflect non-inflammatory pain mechanisms. Utilizing FSQ and QSTs in clinical studies may help better characterize pain heterogeneity in axSpA.

Table II. Differences in QSTs between axSpA adults with nociplastic pain features (FSQ≥13) and those without (FSQ<13).

QSTs by FSQ	Marginal test			Regression analysis		
Difference	Estimate	Std Error	p-value	Estimate	Std Error	p-value
PPT RT TRAP	-2.1661	0.5160	0.0001	-1.8452	0.6147	**0.0038**
PPT LT TRAP	-2.1584	0.5119	0.0001	-1.8907	0.5773	**0.0017**
PPT RT SIJ	-2.8885	0.5761	0.0000	-1.9500	0.6495	**0.0038**
PPT LT SIJ	-2.9856	0.5626	0.0000	-2.1384	0.6378	**0.0013**
PPT RT KNEE	-2.6005	0.5536	0.0000	-1.8571	0.6354	**0.0048**
PPT LT KNEE	-2.1957	0.6123	0.0006	-1.3018	0.7145	0.0730
RT TS	0.6768	0.3702	0.0712	0.2669	0.4403	0.5464
LT TS	0.5022	0.4115	0.2258	0.0021	0.4930	0.9966
CPM	0.0522	0.0593	0.3816	0.0785	0.0733	0.2878

QST: quantitative sensory testing; FSQ: fibromyalgia survey questionnaire; std error: standard error; PPT: pain pressure threshold; rt: right; trap: trapezius; lt: left; SIJ: sacroiliac joint; TS: temporal summation; CPM: conditioned pain modulation.

P26

ALTERED BRAIN CONNECTIVITY IS ASSOCIATED WITH PAIN AND DISEASE BURDEN IN AXIAL SPONDYLOARTHRITIS

Bittar M.[1], Smith D.[2], Sridhar A.[3,4], Kohno M.[5], Deodhar A.[1], Clauw D.[6], Harris R.[3,4]
[1]Division of Arthritis and Rheumatic Diseases, Oregon Health & Science University, Portland, OR; [2]Dept. of Behavioral Neuroscience, Oregon Health and Science University, Portland OR; [3]Susan Samueli Integrative Health Institute, School of Medicine, University of California, Irvine; [4]Dept. of Anesthesiology and Perioperative Care, School of Medicine, University of California, Irvine; [5]Dept. of Psychiatry, Oregon Health & Science University, Portland, OR; [6]Dept.s of Anesthesiology, Medicine (Rheumatology) and Psychiatry, University of Michigan, Ann Arborn, MI, USA

Introduction. Altered brain connectivity has been shown to play a role in chronic pain conditions. The aim of this study is to examine associations between symptom burden/pain, disease activity, and resting state connectivity between the brain's default mode network (DMN) and insula in axial spondyloarthritis (axSpA).
Methods. Patients who met the 2009 ASAS classification criteria were invited to complete questionnaires including the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), and the Widespread Pain Index(WPI). Patients underwent resting-state functional brain imaging(rsfMRI). ROI-to-ROI analysis was performed which generated Fisher-Z transformed correlation coefficients for each subject that represent the resting-state functional connectivity(rsFC) between the DMN and insula. Multiple linear regressions were then performed to assess the relationship between clinical measures (BASDAI, WPI, current pain, PROMIS29) and DMN-insula rsFC after adjusting for sex. Lastly, differences between those with pain (based on current pain and/or BASDAI Q#2 ≥4) or without were assessed via welches two-sample t-tests or chi-squared tests.
Results. Thirty-one participants are enrolled to date: 61% female, 77% White, with a mean age of 39 years.
Bilateral anterior insula-DMN connectivity is positively correlated with BASDAI (adjusted β-estimate 2.99, *p*-0.04), WPI (adjusted β-estimate 5.34, *p*-0.03), and current pain (adjusted β-estimate 3.75, *p*-0.03), after adjusting for sex (Fig. 1). Patients with pain had significantly stronger left and right anterior insula-DMN connectivity compared to patients without pain (Table I). Notably, 84% (26/31) of the patients did not have evidence of active inflammation, and BASDAI was driven by non-inflammatory components of axSpA. Similarly, higher DMN-insula connectivity was seen with worse depression (adjusted β-estimate 0.13, *p*-0.03) and PROMIS29 (adjusted β-estimate 0.038, *p*-0.04).
Conclusion. Altered brain connectivity (DMN-anterior insula) is associated with higher disease burden and comorbid pain and depression, independent of inflammatory activity. These findings suggest that altered central brain processing plays a role in driving worse disease outcomes in axSpA.

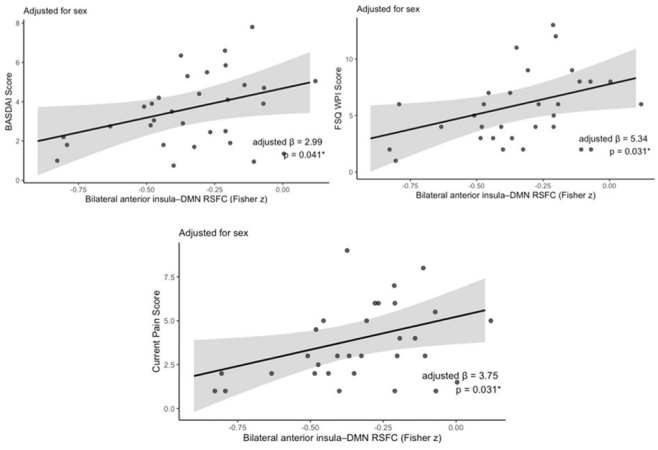


Fig. 1. Bilateral anterior insula-DMN connectivity is significantly correlated with BASDAI scores, WPI scores, and current pain scores, after adjusting for sex.
BASDAI: bath ankylosing spondylitis disease activity index; FSQ WPI: fibromyalgia survey questionnaire widespread pain index; DMN: default-mode-network; RSFC: resting state functional connectivity.

Table I. Differences in clinical scores and insula-DMN connectivity between axSpA patients with and without pain.

Characteristic	No_Pain N = 15[1]	Pain N = 16[1]	Overall N = 31[1]	p-value[2]
Age (years)	34.0 (7.6)	43.4 (17.0)	38.8 (13.9)	0.058
Sex (# male)	5 (33%)	7 (44%)	12 (39%)	0.8
BMI (kg/m^2)	25.5 (6.7)	29.4 (5.5)	27.5 (6.3)	0.089
Anxiety Score	7.9 (3.6)	8.6 (4.1)	8.3 (3.8)	0.7
Depression Score	6.1 (2.5)	9.0 (3.7)	7.6 (3.5)	**0.015**
Fatigue Score	10.7 (4.0)	12.9 (4.5)	11.8 (4.3)	0.2
BASDAI Score	2.2 (0.9)	4.8 (1.5)	3.5 (1.8)	**<0.001**
FSQ Score	8.1 (2.6)	13.8 (5.2)	11.0 (5.0)	**<0.001**
FSQ_WPI	3.7 (1.9)	7.3 (3.0)	5.5 (3.1)	**<0.001**
PROMIS Score	84.5 (10.9)	89.5 (12.1)	86.7 (11.5)	0.3
Insula-DMN RSFC (Fisher Z)	-0.5 (0.3)	-0.3 (0.2)	-0.4 (0.2)	0.13
Ant-Insula-DMN Fisher Z RSFC (Fisher Z)	-0.5 (0.2)	-0.2 (0.2)	-0.3 (0.2)	**0.005**
L-Insula-DMN Fisher Z RSFC (Fisher Z)	-0.4 (0.3)	-0.3 (0.2)	-0.3 (0.2)	0.10
R-Insula-DMN Fisher Z RSFC (Fisher Z)	-0.4 (0.2)	-0.3 (0.2)	-0.4 (0.2)	0.3
R-Ant-Insula-DMN Fisher Z RSFC (Fisher Z)	-0.4 (0.2)	-0.2 (0.1)	-0.3 (0.2)	**0.010**
L-Ant-Insula-DMN Fisher Z RSFC (Fisher Z)	-0.4 (0.2)	-0.2 (0.2)	-0.3 (0.2)	**0.011**

[1] Mean (SD); n (%)
[2] Welch Two Sample t-test; Pearson's Chi-squared test

BMI: body mass index; BASDAI: bath ankylosing spondylitis disease activity index; FSQ: fibromyalgia survey questionnaire; WPI: widespread pain index; PROMIS: Patient-Reported Outcomes Measurement Information System; DMN: default-mode-network; RSFC: resting state functional connectivity.

P27

SEX DIFFERENCES IN INFLAMMATORY MARKERS IN AXIAL SPONDYLOARTHRITIS (AXSPA)

Juditzki M.[1], Andreica I.[2], Lalazi B.[2], Baraliakos X.[2], Capellino S.[1]
[1]Dept. of Immunology, Research Group of Neuroimmunology, IfADo – Leibniz Research Centre for Working Environment and Human Factors, Dortmund; [2]Rheumazentrum Ruhrgebiet Herne, Ruhr Universität Bochum, Germany

Introduction. Female and male axial Spondyloarthritis (axSpA) patients show differently pronounced disease progression, treatment outcomes and biomarker levels, suggesting that there are sex-specific differences in the pathophysiology of axSpA. Nevertheless, knowledge of sex-specific inflammatory pathways in axSpA pathology is still limited and treatment response is worse in female patients. The aim of this study is therefore to delineate sex-specific differences in axSpA-related biomarkers in biologically naïve non-radiologic (nr-axSpA) and radiologic axSpA (r-axSpA) patients.
Methods. Plasma samples from biologically naïve nr-axSpA (males n=35, females n=19), r-axSpA (males n=49, females n=16) and healthy controls (males n=19, females n=20) were analyzed for levels of cytokines by multiplex analysis. PBMC subsets were investigated by flow cytometry utilizing specific extracellular markers.
Results. HGF (hepatocyte growth factor) and VEGF (vascular endothelial growth factor) are significantly higher in axSpA patients compared to healthy controls, independently of biological sex. TNF (tumor necrosis factor) and IL-23 (interleukin-23) are differently modulated in female and male axSpA patients: TNF is significantly higher in female r-axSpA compared to male r-axSpA patients, whereas IL-23 is significantly lower in male axSpA compared to the respective HC and no differences were observed in female patients. Other cytokines show a sex-specific rather than axSpA-speific pathway, with MMP-3 (matrix metalloproteinase-3) showing significantly higher levels in male and leptin in female.
Conclusion. This study confirms sex-specific inflammatory pathway activation, and also highlights specific differences between female and male axSpA patients. These results demonstrate the importance and clinical relevance of sex-specific analysis to understand disease pathophysiology and to identify sex-specific therapeutic targets to improve personalized treatment response in axSpA.

P28

HIGH SPECIFICITY BUT MODEST SENSITIVITY OF THE 2025 ASSESSMENT OF SPONDYLOARTHRITIS INTERNATIONAL SOCIETY (ASAS) AND SPONDYLOARTHRITIS RESEARCH AND TREATMENT NETWORK (SPARTAN) REVISED AXIAL SPONDYLOARTHRITIS CLASSIFICATION CRITERIA IN EARLY AXIAL SPONDYLOARTHRITIS: DATA FROM THE SPONDYLOARTHRITIS CAUGHT EARLY (SPACE) COHORT

de Bruin L.J.E.[1], Ramiro S.[1,2], van Lunteren M.[1], Marques M.L.[1,3], Bento da Silva A.[1], Ayan G.[1,4], de Hooge M.[5], Berg I.J.[6], Exarchou S.[7], Ramonda R.[8], Landewé R.[2,9], van der Heijde D.[1], van Gaalen F.A.[1]

[1]Rheumatology, Leiden University Medical Center, Leiden; [2]Rheumatology, Zuyderland Medical Center, Heerlen, The Netherlands; [3]Rheumatology, Coimbra Local Health Unit, Coimbra, Portugal; [4]Ankara Research and Training Hospital, Ankara, Türkiye; [5]Rheumatology, Ghent University Hospital, Ghent, Belgium; [6]Center for Treatment of Rheumatic and Musculoskeletal Diseases (REMEDY), Diakonhjemmet Hospital, Oslo, Norway; [7]Dept. of Clinical Sciences Malmö (Rheumatology), Lund University, Malmö, Sweden; [8]Rheumatology Unit, Dept. of Medicine-DIMED, University of Padova, Padova, Italy; [9]The Dept. of Rheumatology and Clinical Immunology, Amsterdam University Medical Center, Amsterdam, The Netherlands

Introduction. An ASAS-SPARTAN initiative revised the 2009 ASAS classification criteria (2009-criteria) for axial spondyloarthritis (axSpA), developing the ASAS-SPARTAN 2025 revised classification criteria (2025-criteria) (1). In early axSpA, their performance was tested and compared with that of the 2009-criteria.

Methods. Patients with chronic back pain from the SPondyloArthritis Caught Early (SPACE) cohort received a rheumatologist's diagnosis (axSpA/non-axSpA) after 2-year follow-up. Baseline data were included for analysis. Local and blinded central readers assessed active and structural lesions on MRI and radiography of the sacroiliac joints. Both criteria sets classify an imaging or clinical arm, based on present SpA features. The 2009-criteria apply the ASAS MRI-SIJ positive (ASAS-MRI-SIJ+) definition. The 2025-criteria apply a global assessment for MRI for active and structural lesions, weighted SpA features and thresholds to the total feature score. In SPACE, local readers globally assessed MRI. Additionally, three central readers assessed the ASAS-MRI-SIJ+ and Leiden structural lesion definitions (2). For each set, patients were classified. Prevalence of the classifications (incl. arms; local reading), sensitivity and specificity for axSpA (each reader and central reader consensus) were calculated. Patient overlap between sets was assessed in axSpA.

A

	axSpA				Non-axSpA				Total +
	Total+	Both arms	Imaging only	Clinical only	Total+	Both arms	Imaging only	Clinical only	
2009 ASAS classification criteria	291	133	83	75	51	0	13	38	342
2025 ASAS-SPARTAN revised classification criteria	212	2	205	5	8	0	8	0	220

B

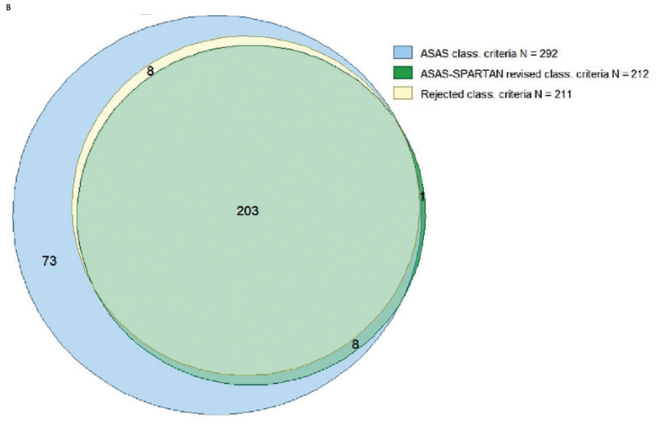


Fig. 1. Prevalence of classification criteria sets in the SPACE cohort.
A: The prevalence and separate classification arms according to local reading of imaging in 669 patients with chonic back pain included in the SPACE cohort.
B: Euler diagram of the axial spondyloarthriris patient (N=293) that do and do not overlap between different classification sets, classified with use of the scores of the local reading. ASAS: Assessment of SpondyloArthritis international Society; axSpA: axials spondyloarthritis; Class: classification; SPACE: SpondyloArthritis Caught Early cohort; SPARTAN: SpondyloArthritis Research and Treatment Network; Total +: total of patients that fulfilled the classification critaria set.

Table I. Performance of the ASAS classification criteria and the two candidate criteria sets in 669 patients with chronic back pain included in the SPACE cohort

	Sensitivity %	Specificity %
2009 ASAS classification criteria		
Local reader	82.7	83.9
Central reader 1 - ASAS-MRI-SIJ+	70.4	85.2
Central reader 2 - ASAS-MRI-SIJ+	74.1	85.8
Central reader 3 - ASAS-MRI-SIJ+	76.7	76.7
Consensus ≥2 of 3 central readers - ASAS-MRI-SIJ+	71.8	85.8
Central reader 1 - ASAS-MRI-SIJ+ and Leiden structural lesions	71.8	84.2
Central reader 2 - ASAS-MRI-SIJ+ and Leiden structural lesions	76.9	84.9
Central reader 3 - ASAS-MRI-SIJ+ and Leiden structural lesions	78.1	76.3
Consensus ≥2 of 3 central readers - ASAS-MRI-SIJ+ and Leiden structural lesions	73.2	85.8
2025 ASAS-SPARTAN revised classification criteria		
Local reader	60.2	97.5
Central reader 1 - ASAS-MRI-SIJ+	34.7	98.4
Central reader 2 - ASAS-MRI-SIJ+	43.2	97.8
Central reader 3 - ASAS-MRI-SIJ+	45.7	95.0
Consensus ≥2 of 3 central readers - ASAS-MRI-SIJ+	39.2	98.4
Central reader 1 - ASAS-MRI-SIJ+ and Leiden structural lesions	39.2	98.4
Central reader 2 - ASAS-MRI-SIJ+ and Leiden structural lesions	49.1	96.8
Central reader 3 - ASAS-MRI-SIJ+ and Leiden structural lesions	50.6	94.3
Consensus ≥2 of 3 central readers - ASAS-MRI-SIJ+ and Leiden structural lesions	44.0	98.4

The Leiden structural lesions definition is defined by having ≥3 erosions, ≥3 fat lesions, or ≥5 fat lesions and/or erosions.[2] ASAS: Assessment of SpondyloArthritis international Society; ASAS-MRI-SIJ+: a positive MRI according to the ASAS definition; SPACE: SPondyloArthritis Caught Early; SPARTAN: SpondyloArthritis Research and Treatment Network.

Results. In 669 patients (age 30 (SD 8) years; 39% male; 53% axSpA), the 2025-criteria classified 27% fewer axSpA patients than the 2009-criteria, with 93% less patients in the clinical arm only, reflecting imaging-prioritised classification (97% imaging-only) (Fig. 1). The 2025-criteria showed higher specificity with lower sensitivity than the 2009-criteria (specificity: 94-98% vs. 76-86%; sensitivity: 35-60% vs. 70-83%) (Table I).

Conclusion. The highly specific 2025-criteria excluded 27% of early axSpA patients classified by the 2009-criteria. Their reduced sensitivity reflects fewer classifications by clinical features alone, highlighting the predominance of imaging. Local reading sufficed to achieve high specificity of the 2025-criteria.

References

1. MAKSYMOWYCH *et al.*: *ACR* 2025
2. DE BRUIN *et al.*: *RMD Open* 2026

P29

INCIDENCE, PREDICTORS AND BURDEN OF DIFFICULT-TO-MANAGE AXIAL SPONDYLOARTHRITIS: A 10-YEAR ANALYSIS OF THE DESIR COHORT

Fakih O.[1], Moltó A.[2,3], Verhoeven F.[1], Prati C.[1], Wendling D.[1]

[1]Université Marie et Louis Pasteur, service de rhumatologie, CHU de Besançon; [2]Université Paris Cité and Université Sorbonne Paris Nord, INSERM, INRAE, Centre for Research in Epidemiology and Statistics, Paris; [3]Rheumatology Dept., Bichat Hospital, AP-HP, Paris, France

Introduction. Difficult-to-treat inflammatory arthritis refers to persistent disease activity despite multiple targeted therapies. ASAS has proposed a consensus definition of difficult-to-manage (D2M-axSpA) and treatment-refractory axial spondyloarthritis (TR-axSpA), encompassing broad challenging disease management and, in select cases, objective persistent inflammation. Most studies on D2M-axSpA remain cross-sectional, leaving the longitudinal incidence and long-term outcomes unexplored.

Objective. To determine the cumulative incidence of D2M-axSpA and TR-axSpA in an early axSpA inception cohort, identify baseline factors, and characterise long-term outcomes.

Methods. Patients from the French prospective DESIR cohort fulfilling the ASAS criteria for axSpA and exposed to at least one biologic DMARD were included. D2M-axSpA and TR-axSpA patients were classified according both to the ASAS definition but also an extended definition adapted to historical treatment availability (failure of ≥3 b/tsDMARDs regardless of mechanism of action). Cox models were used to identify baseline factors associated with D2M-axSpA. Clinical, imaging and socio-economic outcomes were analysed over 10 years of follow-up.

Results. Among 177 patients, the cumulative incidence of D2M-axSpA was 6.8% (95% CI 2.4–11.0) using the ASAS definition and 14.4% (8.3–20.2) using the extended definition, while TR-axSpA remained rare (0.7% and

Table I. Baseline characteristics of all patients, then according to difficult-to-manage axial spondyloarthritis (D2M-axSpA) status according to ASAS definitions and then extended.

Characteristic[$]	All patients N=177	D2M (ASAS) patients n=9	Non-D2M (ASAS) patients n=168	D2M (extended) patients n=20	Non-D2M (extended) patients n=157	p-value*
Age (years)	31.2 (7.8)	31.7 (10.1)	31.2 (7.7)	32.3 (8.3)	31.1 (7.7)	0.48
Female sex	87 (49.2%)	7 (77.8%)	80 (47.6%)	**16 (80.0%)**	**71 (45.2%)**	**0.003**
Follow-up duration (years)	8.9 (2.4)	10.0 (0.3)	8.9 (2.5)	9.5 (1.8)	8.8 (10.0)	0.53
HLA-B27	152 (85.9%)	7 (77.8%)	145 (86.3%)	16 (80.0%)	136 (86.6%)	0.42
BMI (kg/m^2)	24.0 (3.8)	22.1 (4.1)	24.1 (3.8)	24.1 (4.8)	23.9 (3.7)	0.79
Family history of spondyloarthritis	84 (50.0%)	3 (33.3%)	81 (50.9%)	**5 (27.8%)**	**79 (52.7%)**	**0.047**
Smoking	79 (44.6%)	4 (44.4%)	75 (44.6%)	10 (50.0%)	69 (43.9%)	0.61
Hypertension	4 (2.5%)	0 (0%)	4 (2.3%)	0 (0%)	4 (2.3%)	1
Fibromyalgia	29 (16.4%)	1 (11.1%)	28 (16.7%)	4 (20%)	25 (15.9%)	0.75
Symptom duration (years)	1.6 (0.8)	2.0 (1.0)	1.6 (0.8)	1.7 (1.0)	1.6 (0.8)	0.53
Symptom duration before 1st DMARD (years)	1.6 (0.9)	2.1 (1.0)	1.6 (0.9)	1.7 (1.0)	1.6 (0.9)	0.41
Diagnostic delay	1.2 (1.0)	1.0 (1.0)	1.2 (1.0)	1.0 (0.8)	1.2 (1.0)	0.33
Triggering factor (all)	49 (27.7%)	6 (66.7%)	43 (25.6%)	**10 (50.0%)**	**39 (24.8%)**	**0.02**
Peripheral involvement	103 (58.2%)	3 (33.3%)	71 (42.3%)	7 (35.0%)	67 (42.7%)	0.51
Uveitis	14 (26.4%)	0 (0%)	14 (26.9%)	1 (25.0%)	13 (26.5%)	0.95
Psoriasis	32 (18.1%)	1 (11.1%)	31 (18.5%)	3 (15%)	29 (18.5%)	0.70
Inflammatory bowel disease	10 (5.6%)	0 (0%)	10 (6.0%)	0 (0%)	10 (6.4%)	0.61
Patient global assessment	5.8 (2.5)	6.1 (2.1)	5.8 (2.6)	**6.8 (2.0)**	**5.6 (2.6)**	**0.047**
Physician global assessment	5.2 (2.2)	5.6 (1.7)	5.2 (2.2)	**6.3 (1.5)**	**5.0 (2.3)**	**0.02**
ASDAS-CRP	3.1 (0.9)	3.4 (0.9)	3.1 (0.9)	**3.6 (0.9)**	**3.0 (0.9)**	**0.004**
BASDAI	5.0 (1.9)	5.9 (2.4)	4.9 (1.9)	**6.2 (1.4)**	**4.8 (1.9)**	**<0.001**
BASFI	37.5 (22.2)	52.1 (28.7)	36.7 (21.7)	**53.8 (22.8)**	**35.4 (21.4)**	**<0.001**
ASQoL	10.9 (4.5)	13.7 (2.7)	10.8 (4.5)	**14.3 (2.4)**	**10.5 (4.5)**	**<0.001**
CRP (mg/L)	13.4 (18.4)	13.2 (17.2)	13.4 (18.5)	17.0 (20.6)	12.8 (18.1)	0.57
Radiographic sacro-iliitis	44 (25.3%)	2 (22.2%)	42 (25.5%)	6 (30.0%)	38 (24.7%)	0.61
mSASSS	0.51 (1.85)	0.04 (0.11)	0.54 (1.90)	0.51 (1.6)	0.51 (1.9)	0.63
Number of syndesmophytes	0.2 (0.7)	0 (0)	0.2 (0.8)	0.39 (1.1)	0.20 (0.7)	0.67
SPARCC MRI SIJ score	5.5 (8.6)	5.0 (10.1)	5.5 (8.6)	4.7 (9.1)	5.6 (8.6)	0.12
SPARCC MRI Spine score	5.4 (13.7)	1.8 (4.1)	5.6 (14.2)	2.0 (4.2)	5.8 (14.4)	0.22

[$]Values are expressed as mean (standard deviation) for quantitative variables and numbers (percentages) for qualitative variables.
* *p*-values correspond to comparisons between D2M and non-D2M-axSpA patients according to the extended definition.

3.7%, respectively). Female sex and higher baseline disease activity were predictors of D2M-axSpA development (Table I), confirmed in multivariate analysis. During follow-up (Table II), D2M-axSpA patients had a higher prevalence of fibromyalgia (60% vs. 22.9%, p<0.001), greater healthcare resource utilisation (cumulative number of medical visits 110 vs. 60, p<0.001) and opioid use (100% vs. 72%, p=0.004).

Conclusion. In this early axSpA inception cohort with long-term follow-up, D2M-axSpA was rare, rarely associated with true treatment refractoriness, and predominantly driven by a persistent disease burden carrying a substantial socio-economic impact, highlighting the need for a broader, patient-centred approach to its management.

Table II. Characteristics during the 10-year follow-up of all patients, then according to difficult-to-manage axial spondyloarthritis (D2M-axSpA) status according to ASAS definitions and then extended.

Characteristic[$]	All patients N=177	D2M (ASAS) patients n=9	Non-D2M (ASAS) patients n=168	D2M (extended) patients n=20	Non-D2M (extended) patients n=157	p-value*
Obesity	50 (28.4%)	2 (22.2%)	48 (28.7%)	7 (35.0%)	43 (27.6%)	0.49
Smoking	102 (57.6%)	5 (55.6%)	97 (57.7%)	13 (65.0%)	89 (56.7%)	0.48
Hypertension	15 (8.5%)	1 (11.1%)	14 (8.3%)	2 (10.0%)	13 (8.3%)	1
Dyslipidemia	32 (18%)	3 (33%)	29 (17%)	6 (30%)	26 (17%)	0.21
Diabetes	3 (2%)	0 (0%)	3 (2%)	0 (0%)	3 (2%)	N/A
Major adverse cardiovascular events	4 (2%)	0 (0%)	4 (2%)	0 (0%)	4 (3%)	N/A
Fibromyalgia	48 (27.1%)	6 (66.7%)	42 (25.0%)	**12 (60%)**	**36 (22.9%)**	**<0.001**
Peripheral involvement	157 (88.7%)	9 (100%)	148 (88.1%)	20 (100%)	137 (87.3%)	0.09
Uveitis	36 (50.0%)	3 (75%)	33 (48.5%)	5 (71.4%)	31 (47.7%)	0.43
Psoriasis	69 (39.0%)	5 (55.6%)	64 (38.1%)	9 (45.0%)	60 (38.2%)	0.56
Inflammatory bowel disease	24 (13.6%)	0 (0%)	24 (14.3%)	2 (10.0%)	22 (14.0%)	1
mSASSS progression	49 (54.4%)	2 (28.6%)	47 (56.6%)	6 (54.5%)	43 (54.4%)	1
At least 3 fatty lesions on MRI at any point	35 (59.3%)	2 (28.6%)	47 (56.6%)	3 (60%)	32 (59.3%)	1
SPARCC SIJ inflammatory score at 10 years	1.89 (4.95)	0.27 (0.60)	1.98 (5.08)	0.15 (0.44)	2.08 (5.18)	0.07
SPARCC Spine inflammatory score at 10 years	3.75 (6.84)	0.43 (0.74)	4.04 (7.06)	2.67 (5.29)	3.91 (7.05)	0.43
Mean ASAS NSAID score over follow-up	33.9 (24.9)	37.2 (24.2)	33.7 (25.0)	47.9 (32.7)	32.1 (23.3)	0.05
Max ASAS NSAID score over follow-up	91.08 (41.23)	108.36 (53.46)	90.15 (40.47)	**114.94 (55.40)**	**88.04 (38.23)**	**0.010**
Any opioid use	133 (75%)	9 (100%)	124 (74%)	**20 (100%)**	**113 (72%)**	**0.004**
Opioid prescription ≥6 months	80 (45%)	5 (56%)	75 (45%)	**14 (70%)**	**66 (42%)**	**0.03**
Any strong opioid use	25 (14%)	2 (22%)	23 (14%)	**7 (35%)**	**18 (11%)**	**0.01**
Cumulative number of sick leave days	74.97 (128.18)	99.61 (69.37)	73.65 (130.58)	**169.80 (149.48)**	**62.89 (120.48)**	**<0.001**
Long-term sick leave	21 (12%)	3 (33%)	18 (11%)	4 (20%)	17 (11%)	0.27
Disability pension related to SpA	27 (15%)	5 (56%)	22 (13%)	**9 (45%)**	**18 (11%)**	**<0.001**
Rheumatologist visits/year	2.6 (2.1)	3.0 (2.1)	2.5 (2.1)	**4.1 (3.5)**	**2.4 (1.8)**	**0.009**
General practitioner visits/year	5.64 (5.1)	8.52 (5.2)	5.49 (5.1)	**9.07 (8.1)**	**5.20 (4.4)**	**0.01**
Cumulative medical visits	65.0 (47.6)	107.1 (53.1)	62.7 (46.4)	**109.6 (73.8)**	**59.3 (40.1)**	**<0.001**
Physiotherapy visits/year	13.7 (20.0)	24.1 (28.9)	13.2 (19.4)	**21.0 (25.2)**	**12.8 (19.2)**	**0.04**
Cumulative ED visits	1.4 (1.5)	3.2 (1.3)	1.3 (1.5)	**2.2 (1.7)**	**1.3 (1.5)**	**0.02**
At least one ED visit	116 (66%)	9 (100%)	107 (64%)	15 (75%)	101 (64%)	0.46
Cumulative hospitalisation days	6.2 (13.1)	7.3 (8.4)	6.1 (13.3)	**11.8 (23.4)**	**5.5 (11.1)**	**0.04**
At least one hospitalisation	133 (75%)	8 (89%)	125 (74%)	**19 (95%)**	**114 (73%)**	**0.03**

[$]Values are expressed as mean (standard deviation) for quantitative variables and numbers (percentages) for qualitative variables.
*p-values correspond to comparisons between D2M and non-D2M-axSpA patients according to the extended definition.

P30

SEX DIFFERENCES IN AXIAL SPONDYLOARTHRITIS ARE NOT ATTENUATED BY HLA-B27 POSITIVITY: A 10-YEAR ANALYSIS OF THE DESIR COHORT

Fakih O.[1,2], Harrison S.R.[1], Moltó A.[3,4], Prati C.[2], Ramiro S.[5], Gossec L.[6,7], Marzo-Ortega H.[1]
[1]NIHR Leeds Biomedical Research Centre, The Leeds Teaching Hospitals NHS Trust and Leeds Institute of Rheumatic and Musculoskeletal Medicine, University of Leeds, Leeds, UK; [2]Université Marie et Louis Pasteur, Service de Rhumatologie, CHU de Besançon; [3]Université Paris Cité and Université Sorbonne Paris Nord, INSERM, INRAE, Centre for Research in Epidemiology and Statistics, Paris; [4]Rheumatology Dept., Bichat Hospital, APHP, Paris, France; [5]Leiden University Medical Center, Leiden and Zuyderland Medical Center, Heerlen, The Netherlands; [6]INSERM, Institut Pierre Louis d'Epidémiologie et de Santé Publique, Team Pepites, Sorbonne Université, Paris; [7]Rheumatology Dept., AP-HP, Pitié Salpêtrière Hospital, Paris, France

Introduction. Females with axial spondyloarthritis (axSpA) report a greater disease burden than males despite similar or lower CRP. HLA-B27 positivity is associated with greater objective inflammation and radiographic progression and is less prevalent in females. Whether HLA-B27+ attenuates sex differences in axSpA has rarely been explored (1). We compared disease activity, function, quality of life (QoL), structural progression, extra-musculoskeletal manifestations (EMMs), comorbidities, and treatment retention

Table I. Comparison of baseline characteristics between females and males in the whole cohort, and according to HLA-B27 status.

Characteristic	All (N=689)	All females (n=371)	All Males (n=318)	p	All HLA-B27+ (N=403)	HLA-B27+ Females (n=193)	HLA-B27+ Males (n=209)	p	All HLA-B27- (N=286)	HLA-B27- Females (n=177)	HLA-B27- Males (n=109)	p
Age (years)	**33.7 (8.6)**	**34.6 (8.9)**	**32.6 (8.2)**	**0.004**	**32.4 (8.4)**	**33.6 (8.4)**	**31.3 (8.3)**	**0.006**	35.5 (8.6)	35.7 (9.3)	35.2 (7.4)	0.555
BMI (kg/m²)	**23.9 (4.0)**	**23.8 (4.4)**	**24.1 (3.6)**	**0.022**	**23.6 (3.9)**	**23.3 (4.2)**	**23.9 (3.5)**	**0.012**	24.4 (4.2)	24.3 (4.6)	24.5 (3.6)	0.307
Symptom duration (years)	1.5 (0.9)	1.5 (0.9)	1.5 (0.9)	0.385	1.5 (0.9)	1.5 (0.9)	1.5 (0.9)	0.851	1.5 (0.8)	1.6 (0.8)	1.4 (0.9)	0.229
Diagnostic delay (years)	1.2 (1.0)	1.3 (1.1)	1.1 (0.9)	0.060	1.2 (1.0)	1.3 (1.1)	1.1 (0.9)	0.105	1.6 (1.2)	1.7 (1.3)	1.4 (1.1)	0.368
Smoking	254 (37.2%)	125 (33.8%)	129 (41.2%)	0.055	158 (39.3%)	68 (35.1%)	90 (43.3%)	0.113	96 (34.2%)	57 (32.4%)	39 (37.1%)	0.494
Family history of spondyloarthritis	**283 (41.1%)**	**172 (46.4%)**	**111 (34.9%)**	**0.003**	**182 (45.2%)**	**101 (52.1%)**	**81 (38.8%)**	**0.010**	**101 (35.3%)**	**71 (40.1%)**	**30 (27.5%)**	**0.042**
Peripheral arthritis	188 (27.3%)	98 (26.4%)	90 (28.3%)	0.639	111 (27.5%)	50 (25.8%)	61 (29.2%)	0.513	77 (26.9%)	48 (27.1%)	29 (26.6%)	1.000
Enthesitis	387 (56.2%)	219 (59.0%)	168 (52.8%)	0.119	214 (53.1%)	107 (55.2%)	107 (51.2%)	0.487	173 (60.5%)	112 (63.3%)	61 (56.0%)	0.270
Dactylitis	95 (13.8%)	52 (14.0%)	43 (13.5%)	0.939	57 (14.1%)	29 (14.9%)	28 (13.4%)	0.762	38 (13.3%)	23 (13.0%)	15 (13.8%)	0.995
Uveitis	64 (9.3%)	38 (10.2%)	26 (8.2%)	0.424	41 (10.2%)	22 (11.3%)	19 (9.1%)	0.561	23 (8.0%)	16 (9.0%)	7 (6.4%)	0.571
Psoriasis	115 (16.7%)	62 (16.7%)	53 (16.7%)	1.000	61 (15.1%)	27 (13.9%)	34 (16.3%)	0.604	54 (18.9%)	35 (19.8%)	19 (17.4%)	0.737
Inflammatory bowel disease	35 (5.1%)	22 (5.9%)	13 (4.1%)	0.356	14 (3.5%)	9 (4.6%)	5 (2.4%)	0.338	21 (7.3%)	13 (7.3%)	8 (7.3%)	1.000
ASDAS	2.7 (0.9)	2.7 (0.9)	2.6 (1.0)	0.091	2.6 (1.0)	2.6 (0.9)	2.5 (1.0)	0.118	2.8 (0.9)	2.8 (0.8)	2.8 (1.0)	0.997
BASDAI	**44.5 (20.0)**	**47.4 (19.4)**	**41.2 (20.3)**	**<0.001**	**42.0 (20.6)**	**46.2 (20.2)**	**38.1 (20.1)**	**<0.001**	48.1 (18.7)	48.6 (18.4)	47.2 (19.2)	0.477
BASFI	**30.4 (22.8)**	**33.2 (23.4)**	**27.1 (21.6)**	**<0.001**	**28.1 (22.3)**	**31.5 (23.4)**	**24.9 (20.9)**	**0.005**	33.6 (23.1)	35.1 (23.4)	31.2 (22.4)	0.191
PtGA	**9.3 (5.0)**	**10.3 (4.7)**	**8.2 (5.0)**	**<0.001**	**8.8 (5.1)**	**10.1 (5.0)**	**7.6 (5.0)**	**<0.001**	10.1 (4.6)	10.5 (4.4)	9.4 (4.9)	0.077
PGA	**41.4 (8.9)**	**40.2 (8.8)**	**42.8 (8.9)**	**<0.001**	**42.3 (8.9)**	**40.8 (8.8)**	**43.6 (8.8)**	**0.002**	40.1 (8.8)	39.5 (8.8)	41.2 (8.8)	0.101
ASQOL	**39.1 (11.5)**	**38.0 (11.4)**	**40.4 (11.5)**	**0.007**	**39.8 (11.5)**	**37.9 (11.5)**	**41.5 (11.3)**	**0.003**	38.2 (11.4)	38.2 (11.4)	38.2 (11.5)	0.815
SF-36 PCS	**33.7 (8.6)**	**34.6 (8.9)**	**32.6 (8.2)**	**0.004**	**32.4 (8.4)**	**33.6 (8.4)**	**31.3 (8.3)**	**0.006**	35.5 (8.6)	35.7 (9.3)	35.2 (7.4)	0.555
SF-36 MCS	**23.9 (4.0)**	**23.8 (4.4)**	**24.1 (3.6)**	**0.022**	**23.6 (3.9)**	**23.3 (4.2)**	**23.9 (3.5)**	**0.012**	24.4 (4.2)	24.3 (4.6)	24.5 (3.6)	0.307
NSAID score in the last 6 months	43.6 (36.1)	43.0 (35.9)	44.2 (36.3)	0.759	47.8 (37.4)	48.3 (37.2)	47.3 (37.7)	0.647	38.8 (34.0)	38.2 (34.2)	39.7 (33.8)	0.629
CRP (mg/L)	8.1 (13.7)	6.2 (9.4)	10.2 (17.2)	0.109	**8.4 (13.9)**	**6.2 (9.5)**	**10.5 (16.8)**	**0.030**	7.6 (13.5)	6.3 (9.4)	9.7 (18.0)	0.747
ASAS positive sacroiliitis on MRI	**95 (30.4%)**	**36 (21.7%)**	**59 (40.1%)**	**<0.001**	**71 (34.8%)**	**23 (24.0%)**	**48 (44.4%)**	**0.004**	24 (22.0%)	13 (18.6%)	11 (28.2%)	0.356
Radiographic sacroiliitis (modified New York criteria)	**47 (14.7%)**	**17 (10.1%)**	**30 (19.9%)**	**0.022**	41 (19.5%)	14 (14.0%)	27 (24.5%)	0.080	6 (5.5%)	3 (4.4%)	3 (7.3%)	0.670
mSASSS	0.3 (1.3)	0.1 (0.3)	0.6 (1.9)	0.166	0.3 (1.5)	0.1 (0.2)	0.5 (2.0)	0.764	**0.4 (0.9)**	**0.1 (0.4)**	**0.7 (1.4)**	**0.027**

ASDAS: Ankylosing Spondylitis Disease Activity Score; ASQoL: Ankylosing Spondylitis Quality of Life; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index; MCS: Mental Component Summary; mSASSS: modified Stoke Ankylosing Spondylitis Spine Score; MRI: magnetic resonance imaging; NSAID: non-steroidal anti-inflammatory drug; PCS: Physical Component Summary; PGA: physician global assessment; PtGA: patient global assessment; SF-36: Short Form 36.

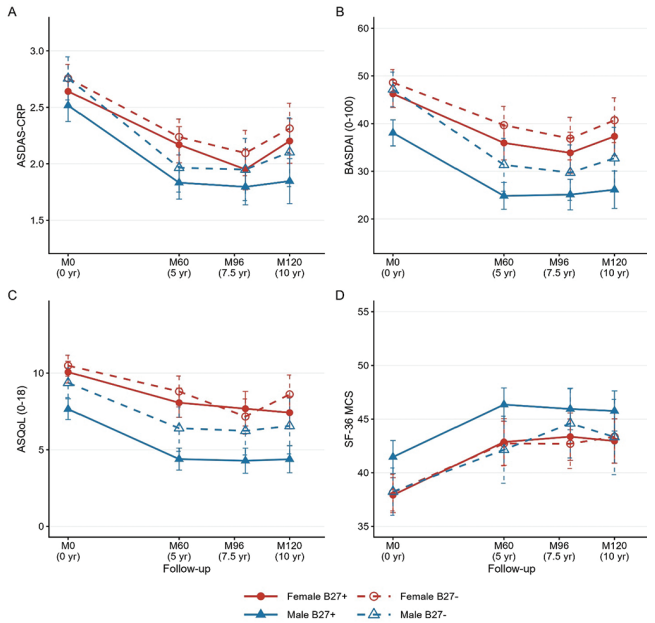


Fig. 1. Ten-year evolution of ASDAS-CRP (**A**), BASDAI (**B**), ASQoL (**C**), and SF-36 MCS (**D**) according to sex and HLA-B27 status in the DESIR cohort (observed means ± 95% confidence intervals).

between sexes according to their HLA-B27 status.

Methods. Patients with a confirmed clinical diagnosis of axSpA from the French inception cohort DESIR were included (2). Baseline characteristics were compared according to sex, overall, and by HLA-B27 status (B27+/B27-). Disease activity (ASDAS, BASDAI), function (BASFI), QoL (ASQOL, SF-36), and CRP were compared at baseline and over 10 years using linear mixed-effects models adjusted for age, symptom duration, and BMI. Time to first bDMARD and its retention were analysed using Cox models. Sex-HLA-B27 interaction was tested for all multivariable analyses.

Results. Among 689 patients (53.8% females), females were older, had lower BMI, more family history of SpA, higher patient-reported outcomes, and less radiographic and MRI-detected sacroiliitis. These differences were present in the HLA-B27+ subset but not in the HLA-B27- subset, except family history (Table I). Longitudinal analyses showed similar results over 10 years of follow-up (Fig. 1), with a significant sex-HLA-B27 interaction on BASDAI (p=0.01), ASQOL (p=0.02) and SF-36 mental component (p=0.03), sex differences being larger among HLA-B27+ patients. At 10 years, EMM prevalence was similar, but fibromyalgia was more frequent in females in both HLA-B27+ (20.9 vs. 10.2%, p=0.01) and B27- (24.5 vs. 9.1%, p=0.02) subsets. Male sex was independently associated with longer drug survival (HR 0.60 [0.43-0.83], p=0.002), HLA-B27 status was not, and sex-B27 interaction was not significant.

Conclusion. Sex differences in disease activity, patient-reported outcomes, objective inflammation, and treatment retention in axSpA are not neutralised by HLA-B27 positivity.

References

1. GUERRA O, HARRISON SR, COSTINAS G *et al.*: POS0755 Exploring the differential effects of HLA-B27 and biological sex on clinical characteristics, inflammatory burden, and service utilisation in Axspa to improve service delivery and reduce delay to diagnosis in axSpA. *Ann Rheum Dis* 2025; 84(1); 917-18.
2. DOUGADOS M, D'AGOSTINO MA, BENESSIANO J *et al.*: The DESIR cohort: a 10-year follow-up of early inflammatory back pain in France: study design and baseline characteristics of the 708 recruited patients. *Joint Bone Spine* 2011; 78(6); 598-603.

P31

CAN CERULOPLASMIN SERVE AS AN INFLAMMATORY BIOMARKER IN SPONDYLOARTHRITIS? A CROSS-SECTIONAL STUDY

Fenniche I., Ben Ayed H., Ben Ammar L., Jebri R., Dhahri R., Gharsallah I.
Dept. of Rheumatology, Military Hospital of Tunis, Tunis, Tunisia

Introduction. Ceruloplasmin is an acute-phase inflammation protein. Despite its recognized role, its use as an inflammatory biomarker remains limited due to moderate sensitivity and hormonal interferences.
However, data suggest potential utility in assessing inflammation and activity in spondyloarthritis (SpA).
This study aimed to explore the relationship between ceruloplasmin levels, inflammatory markers, and disease activity parameters in SpA patients.

Patients and methods. A cross-sectional study over four months included SpA patients per 2009 ASAS criteria. Inflammatory markers analyzed were C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR). Systemic inflammation index (SII) and ratios neutrophil/lymphocyte (NLR), platelet/lymphocyte (PLR), monocyte/lymphocyte (MLR) were calculated from complete blood counts.
Disease activity was assessed via Ankylosing Spondylitis Disease Activity Score using CRP (ASDAS-CRP), ESR (ASDAS-ESR), and Bath Ankylosing Spondylitis Disease Activity Index (BASDAI).
Serum ceruloplasmin was measured in all patients (normal range: 0.20–0.60 g/L).

Results. The study included 101 patients (sex ratio M/F 4.9; 84 men, 17 women). Mean age was 40.5±11.4 years, onset age 35.1±10.7 years, disease duration 5.7±6.5 years.
Mean CRP was 10.2±16.8 mg/L; ESR 28.2±25.1 mm/h. Mean SII 660,278±516,767; NLR 2.2±1.3; MLR 0.26±0.12; PLR 129.9±67.1. Mean ASDAS-CRP 2.06±1.16; BASDAI 2.39±1.68 (12.8% with BASDAI >4, n=13). Mean ceruloplasmin was 0.26 0.04 g/L [0.17–0.49]. Ninety-six patients (95.05%) had normal levels; 5 (4.95%) low (<0.20 g/L). Significant positive correlations emerged between ceruloplasmin and CRP (r=0.541, p<0.001), ESR (r=0.516, p<0.001), SII (r=0.404, p<0.001), PLR (r=0.334, p=0.001), NLR (r=0.339, p=0.001), and ASDAS-CRP (r=0.487, p<0.001). Mean ceruloplasmin was higher in BASDAI >4 patients but not significantly (0.28±0.04 vs. 0.26±0.05 g/L, p=0.208).
Multivariate linear regression showed ceruloplasmin significantly associated with CRP (β=0.001, p<0.0001), ASDAS-CRP (β=0.021, p=0.004), and BASDAI (β=0.015, p=0.001).

Conclusion. Serum ceruloplasmin significantly correlated with key inflammatory markers and SpA activity scores. These findings suggest ceruloplasmin could aid in jointly assessing inflammatory status and disease activity in SpA, though further studies are needed to confirm clinical value.

P32

COMPARATIVE PERFORMANCE OF ASAS-SPARTAN-2025 AND ASAS-2009 CLASSIFICATION CRITERIA IN AXIAL SPONDYLOARTHRITIS: INSIGHTS FROM THE REUMA-CHECK COHORT

Garcia Salinas R., Mejia-Maggi N., Reyes-Jara G., Solis-Mayor J., Bardini C., Apter D., Ruta A.O., Magri S.J.
Hospital Italiano de La Plata, La Plata, Argentina

Introduction/Objective. The ASAS-SPARTAN-2025 criteria incorporate weighted inflammatory and structural MRI lesions to refine objective classification in axSpA, whereas ASAS-2009 captures broader phenotypes through MRI and HLA-B27 arms. We aimed to compare both systems in the structured Reuma-Check early axSpA pathway and characterize overlapping and discordant phenotypes.

Materials and methods. Reuma-Check includes standardized clinical assessment, laboratory markers, pelvic radiography and sacroiliac MRI. Patients were classified according to ASAS-SPARTAN-2025 (cut-off >11 points) and ASAS-2009. Four analytic groups were defined: SPARTAN+, ASAS-MRI+, ASAS-HLA-B27+ and ASAS-general+. Binary variables were compared using chi-square tests and continuous variables using ANOVA. MRI assessment included bone marrow edema, erosions, fat metaplasia, sclerosis and ankylosis.

Results. Among 128 patients, 78.9% fulfilled ASAS-SPARTAN-2025 and 89.1% fulfilled ASAS-2009. Classification overlap identified three discordant phenotypes: SPARTAN+/ASAS+ (72.7%), SPARTAN-/ASAS+ (16.4%) and SPARTAN+/ASAS- (6.3%); 4.7% were negative for both. SPARTAN+ patients had greater imaging burden than SPARTAN-/ASAS+ patients, with universal MRI sacroiliitis (100% vs. 47.6%) and more bone marrow edema (74.2% vs. 47.6%), erosions (59.1% vs. 28.6%) and fat metaplasia (37.6% vs. 19.0%). Clinical manifestations, including inflammatory back pain, peripheral arthritis and enthesitis, were similar across discordant groups. HLA-B27 positivity was comparable in SPARTAN+ and SPARTAN-/ASAS+ patients (49% vs. 52%). Despite imaging differences, ASDAS-CRP (3.23 vs. 3.11) and BASDAI (4.66 vs. 4.43) were similar, whereas CRP and ESR were modestly higher in SPARTAN+ patients. The ASAS-MRI+ group resembled SPARTAN+; the ASAS-HLA-B27+ group showed a genetically driven, imaging-attenuated phenotype (Table I).

Table I. Key classification patterns in Reuma-Check.

Classification finding	Value/interpretation
ASAS-SPARTAN-2025 positive	78.9%
ASAS-2009 positive	89.1%
SPARTAN-/ASAS+ discordant group	16.4%
SPARTAN+/ASAS- discordant group	6.3%
Main SPARTAN+ feature	Higher MRI inflammatory and structural burden

Conclusion. ASAS-SPARTAN-2025 preferentially identifies an imaging-defined axSpA phenotype with higher objective inflammatory and structural burden, while ASAS-2009 captures broader clinical and HLA-driven presentations. Understanding classification divergence is essential for cohort enrichment and future harmonization.

P33

HLA-B27 IN AXIAL SPONDYLOARTHRITIS: CLINICAL AND IMAGING FEATURES FROM THE LATIN AMERICAN ESPALDA REGISTRY

Bautista-Molano W.[2], Garcia Salinas R.[1], Sommerfleck F.[3], Casasola J.C.[4], Guaracha Basanez G.A.[5], Londono J.[6], Gamboa-Cardenas R.V.[7], Fernandez-Avila D.G.[8], Vila D.[9], Mestanza Peralta M.[10], Then J.[11], Dufau E.[12], Bermudez Bermejo P.E.[13], Duran Barragan S.[14], Macias Oviedo L.[15], Valdez Donelli F.[16], Barcia Cansino A.[17], Medina G.[18], Casallas L.[19], Castillo Ortiz A.[20], Chinchay L.[21], Caballero L.[22], Benegas M.[3], Medina Bornachera D.[23], Vega C.[24], Melgarejo P.[22], Gomez A.B.[25], Ferreyra Garrot L.G.[26], Candia Zuniga D.L.[27], Gil-Calderon D.[28], Rivera Teran V.[29], Eguez Del Pozo M.F.[30], Sanchez Prado E.[31], Avellanal V.[25], Marin Zucaro N.[32], Brance M.L.[32] on behalf of ESPALDA-PANLAR
[1]Hospital Italiano de La Plata, La Plata, Argentina; [2]Fundacion Santa Fe, Bogota, Colombia; [3]Sanatorio Mendez, Buenos Aires, Argentina; [4]Hospital General de Mexico, Mexico City, Mexico; [5]Instituto Nacional de Ciencias Medicas y Nutricion Salvador Zubiran, Mexico City, Mexico; [6]Hospital Militar Central, Universidad de La Sabana, Bogota, Colombia; [7]Hospital Nacional Guillermo Almenara Irigoyen, Lima, Peru; [8]Hospital San Ignacio, Bogota, Colombia; [9]Hospital Virgen del Carmen, Campana, Argentina; [10]Hospital Metropolitano, Quito, Ecuador; [11]Hospital Metropolitano de Santiago, Santiago de los Caballeros, Dominican Republic; [12]Sanatorio Guemes, Buenos Aires, Argentina; [13]Centro Medico ABC, Mexico City, Mexico; [14]Clinica de Investigacion en Reumatologia y Obesidad S.C., Guadalajara, Mexico; [15]Consultorio reumaart.ec, Guayaquil, Ecuador; [16]IREP, Buenos Aires, Argentina; [17]Hospital General Portoviejo del Instituto Ecuatoriano de Seguridad Social, Portoviejo, Ecuador; [18]Hospital de Clinicas Jose de San Martin, Buenos Aires, Argentina; [19]Genea Centro Medico, Buenos Aires, Argentina; [20]Hospital Faro del Mayab, Merida, Mexico; [21]Hospital Guillermo Kaelin de La Fuente, Piura, Peru; [22]Instituto de Prevision Social, Asuncion, Paraguay; [23]Red de Investigacion Colaborativa del Caribe Colombiano, Barranquilla, Colombia; [24]Hospital General ISSSTE Tacuba, Mexico City, Mexico; [25]Instituto Nacional de Reumatologia del Uruguay, Montevideo, Uruguay; [26]Hospital Italiano de Buenos Aires, Buenos Aires, Argentina; [27]Hospital Regional Primero de Octubre ISSSTE, Mexico City, Mexico; [28]Artmedica SAS, Bogota, Colombia; [29]Instituto Nacional de Rehabilitacion, Mexico City, Mexico; [30]Altius Medical, Guayaquil, Ecuador; [31]Hospital del IESS los Ceibos, Guayaquil, Ecuador; [32]UI-PANLAR, Rosario, Argentina

Introduction/Objective. HLA-B27 is a key genetic marker in axSpA, yet its relationship with clinical expression and imaging findings has been insufficiently characterized in Latin American populations. We aimed to describe the prevalence of HLA-B27 positivity and its association with clinical, inflammatory, imaging and extra-musculoskeletal features in the ESPALDA registry.

Materials and methods. ESPALDA is a multicentre Latin American registry including patients with axSpA from referral centres. Baseline demographic, clinical, laboratory and imaging data were analysed. Patients were compared according to HLA-B27 status. Multivariable logistic regression was performed to identify factors independently associated with HLA-B27 positivity.

Results. Among 740 patients with available data, 457 (61.8%) were HLA-B27 positive. Compared with HLA-B27-negative patients, HLA-B27-positive patients were younger, more frequently male and had earlier axial symptom onset. They also showed higher inflammatory burden, with higher ASDAS and C-reactive protein levels. Axial imaging involvement was more frequent in HLA-B27-positive patients, including radiographic axSpA (72.2% vs. 60.1%, p=0.001), MRI bone marrow edema (36.8% vs. 23.0%, p<0.001) and MRI bony bridges (10.1% vs. 6.4%, p=0.04). Uveitis was also more frequent (27.6% vs. 15.9%, p<0.001). Peripheral arthritis, enthesitis, psoriasis and IBD did not show consistent clinically relevant differences. In multivariable analysis, younger age (OR 0.97 per year), male sex (OR 1.86), radiographic axial disease (OR 1.74), MRI bone marrow edema (OR 1.68) and uveitis (OR 2.05) were independently associated with HLA-B27 positivity (Table I).

Table I. Factors independently associated with HLA-B27 positivity.

Factor	OR (95% CI)
Age, per year	0.97 (0.96-0.98)
Male sex	1.86 (1.34-2.58)
Radiographic axial disease	1.74 (1.22-2.48)
MRI bone marrow edema	1.68 (1.17-2.41)
Uveitis	2.05 (1.36-3.09)

Conclusion. In ESPALDA, HLA-B27 positivity identified a distinct axial-inflammatory phenotype characterized by earlier onset, male predominance, greater axial imaging involvement and higher prevalence of uveitis. These region-specific data support HLA-B27 as a central marker of axial disease expression in Latin American axSpA.

P34

PREVALENCE AND ONE-YEAR INCIDENCE OF MUSCULOSKELETAL AND EXTRA-MUSCULOSKELETAL MANIFESTATIONS IN AXIAL SPONDYLOARTHRITIS: RESULTS FROM THE LATIN AMERICAN ESPALDA REGISTRY

Garcia Salinas R.[1], Casasola J.C.[4], Sommerfleck F.[3], Guaracha Basanez G.A.[5], Londono J.[6], Gamboa-Cardenas R.V.[7], Fernandez-Avila D.G.[8], Vila D.[9], Mestanza Peralta M.[10], Then J.[11], Dufau E.[12], Bermudez Bermejo P.E.[13], Macias Oviedo L.[15], Duran Barragan S.[14], Valdez Donelli F.[16], Barcia Cansino A.[17], Medina G.[18], Casallas L.[19], Castillo Ortiz A.[20], Chinchay L.[21], Caballero L.[22], Benegas M.[3], Medina Bornachera D.[23], Vega C.[24], Melgarejo P.[22], Gomez A.B.[25], Ferreyra Garrot L.G.[26], Candia Zuniga D.L.[27], Gil-Calderon D.[28], Rivera Teran V.[29], Eguez Del Pozo M.F.[30], Sanchez Prado E.[31], Avellanal V.[25], Brance M.L.[32], Marin Zucaro N.[32], Bautista-Molano W.[2] on behalf of ESPALDA-PANLAR

[1]Hospital Italiano de La Plata, La Plata; [2]Fundacion Santa Fe, Bogota, Colombia; [3]Sanatorio Mendez, Buenos Aires, Argentina; [4]Hospital General de Mexico, Mexico City; [5]Instituto Nacional de Ciencias Medicas y Nutricion Salvador Zubiran, Mexico City, Mexico; [6]Hospital Militar Central, Universidad de La Sabana, Bogota, Colombia; [7]Hospital Nacional Guillermo Almenara Irigoyen, Lima, Peru; [8]Hospital San Ignacio, Bogota, Colombia; [9]Hospital Virgen del Carmen, Campana, Argentina; [10]Hospital Metropolitano, Quito, Ecuador; [11]Hospital Metropolitano de Santiago, Santiago de los Caballeros, Dominican Republic; [12]Sanatorio Guemes, Buenos Aires, Argentina; [13]Centro Medico ABC, Mexico City; [14]Clinica de Investigacion en Reumatologia y Obesidad S.C., Guadalajara, Mexico; [15]Consultorio reumaart.ec, Guayaquil, Ecuador; [16]IREP, Buenos Aires, Argentina; [17]Hospital General Portoviejo del Instituto Ecuatoriano de Seguridad Social, Portoviejo, Ecuador; [18]Hospital de Clinicas Jose de San Martin, Buenos Aires; [19]Genea Centro Medico, Buenos Aires, Argentina; [20]Hospital Faro del Mayab, Merida, Mexico; [21]Hospital Guillermo Kaelin de La Fuente, Piura, Peru; [22]Instituto de Prevision Social, Asuncion, Paraguay; [23]Red de Investigacion Colaborativa del Caribe Colombiano, Barranquilla, Colombia; [24]Hospital General ISSSTE Tacuba, Mexico City, Mexico; [25]Instituto Nacional de Reumatologia del Uruguay, Montevideo, Uruguay; [26]Hospital Italiano de Buenos Aires, Buenos Aires, Argentina; [27]Hospital Regional Primero de Octubre ISSSTE, Mexico City, Mexico; [28]Artmedica SAS, Bogota, Colombia; [29]Instituto Nacional de Rehabilitacion, Mexico City, Mexico; [30]Altius Medical, Guayaquil, Ecuador; [31]Hospital del IESS los Ceibos, Guayaquil, Ecuador; [32]UI-PANLAR, Rosario, Argentina

Introduction/Objective. Musculoskeletal (MSK) and extra-musculoskeletal manifestations are major determinants of burden, prognosis and treatment selection in axSpA, but longitudinal data from Latin America remain limited. We aimed to describe baseline prevalence and one-year incidence of MSK and extra-musculoskeletal manifestations in ESPALDA, the first PANLAR-supported Latin American axSpA registry.

Materials and methods. ESPALDA is a multicentre registry including patients with axSpA from referral centres in Argentina, Colombia, Ecuador, Mexico, Peru, Uruguay, Paraguay, Costa Rica, Honduras and the Dominican Republic. Baseline prevalence was assessed in 741 patients. Incidence analyses included 165 patients with complete one-year follow-up. MSK manifestations were peripheral arthritis and enthesitis; extra-musculoskeletal manifestations were uveitis, inflammatory bowel disease (IBD) and psoriasis. Incidence rates were expressed per 100 patient-years (py) with 95% confidence intervals. Exploratory multivariable analyses were adjusted for sex, HLA-B27 and imaging findings.

Results. At baseline, peripheral arthritis and enthesitis were present in 22.2% and 29.7% of patients, while uveitis, IBD and psoriasis were present in 23.1%, 5.1% and 12.0%, respectively. During follow-up, any incident MSK manifestation occurred at 18.9/100 py; peripheral arthritis at 11.6/100 py and enthesitis at 12.8/100 py. Any incident extra-musculoskeletal manifestation occurred at 7.3/100 py; uveitis was most frequent (4.1/100 py), followed by psoriasis (1.6/100 py) and IBD (1.1/100 py) (Table I). Higher baseline ASDAS showed a trend toward incident MSK manifestations, but sex, HLA-B27 and inflammatory imaging findings were not independently associated with incident events.

Table I. Baseline prevalence and one-year incidence of selected manifestations.

Manifestation	Baseline prevalence	One-year incidence
Peripheral arthritis	22.2%	11.6/100 py
Enthesitis	29.7%	12.8/100 py
Uveitis	23.1%	4.1/100 py
IBD	5.1%	1.1/100 py
Psoriasis	12.0%	1.6/100 py

Conclusion. In this large Latin American registry, MSK and extra-musculoskeletal manifestations were frequent at baseline and new events occurred within one year. These findings support systematic longitudinal assessment of peripheral and extra-musculoskeletal domains in axSpA.

P35

AN INTERDISCIPLINARY MODEL TO IMPROVE ACCESS TO RHEUMATOLOGY CARE AND PROMOTE EARLY IDENTIFICATION FOR AXIAL SPONDYLOARTHRITIS: PRELIMINARY RESULTS OF THE FASTRAX STUDY

Passalent L.[1,2], Gendron E.[9], Chim T.[1], Chow N.[1], MacLeod A.[5], Correale M.[2,6], Sabido Sauri R.[7,8], Fidler W.[5], Pirson C.[10], Pus L.[10], Rampersaud R.[2,6], Aydin S.[7,8], Haroon N.[1,2], Inman R.D.[1,2]

[1]Schroeder Arthritis Institute; [2]University Health Network; [3]Krembil Research Institute; [4]University of Toronto; [5]Northern Ontario School of Medicine University, Thunder Bay; [6]Rapid Access Clinic – Low Back Pain Program; [7]The Ottawa Hospital; [8]University of Ottawa; [9]Centre de l'ostéoporose et de rhumatologie de Québec (Québec), Canada; [10]UCB, Belgium

Introduction/Objective. FASTRAX is a multicentre Canadian initiative using Advanced Clinician Practitioners in Arthritis Care (ACPAC)-trained extended scope practitioners (ESP) to facilitate early axSpA detection (1). We evaluated access to care, geographic reach, screening performance, and number needed to screen (NNS).

Methods. Adults (≥18 years) with low back pain (LBP) >3 months duration, onset age <50 years were referred by their PCP, physician specialist or through the provincial LBP program to their nearest FASTRAX study site for axSpA screening. Screeners performed standard of care assessment. All patients screened were reviewed by a rheumatologist with expertise in SpA. Screeners and attending rheumatologists indicated risk of axSpA on an 11-point scale (-5, high confidence not axSpA to +5, high confidence axSpA). The rheumatologist provided final diagnosis for all patients. Descriptive analyses were performed.

Results. From May 2024 to December 2025, 179 patients completed the screening process across the 3 FASTRAX study sites (mean (SD) age of 39.5 (9.6) years; 42.4% male; mean (SD) duration of back pain 10.9 (10.6) years). Median wait time to assessment was 28 (IQR 17-61) days. Geographic reach of referring providers spanned 208, 600 km^2, with referrals from rural and remote regions accounting for 2.8% (Toronto), 14.7% (Ottawa) and 25.6% (Thunder Bay). Across all sites, 41 patients were diagnosed with axSpA, for a study prevalence of 22.9%. Mean (SD) time to diagnosis was 12.1 (12.7) years for patients diagnosed with axSpA. Overall SE and SP of the screener's assignment of axSpA risk was 90.2% (95% CI: 85.9% to 94.6%) and 76.6% (95% CI: 68.7% to 84.6%). Overall NNS was 5 patients with chronic back pain to identify one patient with axSpA.

Conclusion. The FASTRAX model uses ACPAC-trained ESPs to efficiently screen patients with chronic low back pain for axSpA, providing timely access to care with referral wait times below the provincial median (2).

References

1. PASSALENT L, SUNDARARAJAN K, PERRUCCIO AV *et al*.: Bridging the gap between symptom onset and diagnosis in axial spondyloarthritis. *Arthritis Care Res* (Hoboken). 2022; 74(6): 997-1005
2. WIDDIFIELD J, BERNATSKY S, THORNE JC *et al*.: Wait times to rheumatology care for patients with rheumatic diseases: a data linkage study of primary care electronic medical records and administrative data. *CMAJ Open* 2016; 4(2): E205-12

P36

THE 5-YEAR FOLLOW-UP OF THE DUTCH PRE-SPA COHORT

Groothuizen S.[1], Ez-Zaitouni Z.[2], De Jong H.M.Y.[3], Van der Horst-Bruinsma I.E.[4], Weel A.E.A.M.[5], Van Gaalen F.A.[6], Van Tubergen A.M.[7], Landewé R.[1], Van de Sande M.G.H.[1]

[1]Dept. of Rheumatology and Clinical Immunology, Amsterdam UMC, Amsterdam; [2]Dept. of Rheumatology, Reade, Amsterdam; [3]Dept. of Radiology, UMC Utrecht, Utrecht; [4]Dept. of Rheumatology, Radboud UMC, Nijmegen; [5]Dept. of Rheumatology, Erasmus UMC, Rotterdam; [6]Dept. of Rheumatology, LUMC, Leiden; [7]Dept. of Rheumatology, Maastricht UMC, Maastricht, The Netherlands

Introduction. Early diagnosis in axial spondyloarthritis (axSpA) remains challenging and is often delayed by several years. Previously, we reported SpA features and subclinical inflammation on imaging in first-degree relatives (FDRs) of HLA-B27-positive axSpA patients, with a 1-year SpA incidence of 6%. Here, we extended follow-up to five years to investigate the development of clinically manifest SpA and associated factors.

Methods. The Pre-SpA cohort is a 5-year prospective inception cohort (1). FDRs of HLA-B27-positive axSpA patients were followed for 5 years to investigate incident SpA. Main exclusion criteria were a SpA diagnosis at baseline and chronic back pain with a non-rheumatic diagnosis. Clinical data and biological samples were collected annually, and imaging was performed at baseline (sacroiliac joints MRI and spine radiographs) and at 5-year follow-up (spine radiographs). In case of suspected SpA during follow-up, participants were referred to a rheumatologist for assessment and diagnosis. Baseline characteristics were compared between FDRs with and without incident SpA using chi-square tests for categorical variables and Mann-Whitney U-tests for continuous variables. A Cox proportional hazards model was used to assess associations between baseline HLA-B27 status, age and time to SpA diagnosis; $p<0.05$ was considered statistically significant. Imaging scores are currently being finalized and will be incorporated in future analyses.

Results. A total of 239 FDRs were included between 2013 and 2023; 204 (85%) completed 5-year follow-up, and 19 (7.9%) were diagnosed with SpA. Baseline characteristics of FDRs with and without incident SpA are shown in Table I. In the Cox regression model, HLA-B27 positivity was associated with incident SpA (HR 5.0).

Table I. Baseline characteristics.

	FDR with SpA diagnosis (n=19)	FDR without SpA diagnosis (n=220)	p-value
Age (year)	26 (24–30)	27 (22–31)	
Female (n)	8 (42%)	127 (58%)	
White (n)	18 (95%)	200 (91%)	
Axial disease			
Back pain: chronic	5 (26%)	26 (12%)	
Back pain: intermittent	4 (21%)	85 (39%)	
Peripheral disease			
Arthralgia, current (n)	5 (26%)	71 (32%)	
Arthritis, ever (n)	2 (11%)	5 (2%)	
Enthesitis, ever (n)	3 (16%)	7 (3%)	0.030
Dactylitis, ever (n)	0 (0%)	1 (1%)	
Extraarticular disease			
Psoriasis	1 (5%)	6 (3%)	
Inflammatory bowel disease	1 (5%)	1 (1%)	
Uveitis (ever)	0 (0%)	3 (1%)	
Disease activity measures			
BASDAI (0-10)	1.8 (0.3–3.5)	1.1 (0.5–2.4)	
BASFI (0-10)	0.0 (0.0–1.0)	0.1 (0.0–0.8)	
Patient global health scale (mm)	26 (0–37)	2 (0–21)	
Total back pain scale (mm)	20 (0–43)	9 (0–28)	
Physician disease activity scale (mm)	3 (0–23)	0 (0–7)	
Clinical findings			
Tender joint count >0 (n)	6 (32%)	25 (11%)	
Swollen joint count >0 (n)	0 (0%)	2 (1%)	
MASES >0 (n)	6 (32%)	44 (20%)	
LEI >0 (n)	3 (16%)	19 (9%)	
Laboratory tests			
HLA-B27 positive (n)	16 (89%)	116 (53%)	0.003
CRP (mg/L)	2.4 (1.0–4.1)	1.4 (0.6–2.8)	
ESR (mm/hr)	5.0 (2.0–10.0)	5.0 (2.0–8.0)	

Categorical variables are described as n with percentage, continuous variables are described as median with IQR.
FDR: first-degree relative, SpA: spondyloarthritis, BASDAI: Bath Ankylosing Spondylitis Disease Activity Index, BASFI: Bath Ankylosing Spondylitis Functional Index, MASES: Maastricht Ankylosing Spondylitis Enthesitis Score, LEI: Leeds Enthesitis Index, ESR: erythrocyte sedimentation rate.

Conclusion. In this Dutch inception cohort of 239 FDRs of HLA-B27–positive axSpA patients, 19 participants (7.9%) developed SpA during up to 5 years of follow-up. HLA-B27 positivity was associated with incident disease, highlighting its importance in SpA risk.

Reference

1. doi: 10.1002/acr.24743

P37

SEX-SPECIFIC ASSOCIATIONS BETWEEN PRO- INFLAMMATORY BIOMARKERS AND PAIN-RELATED OUTCOMES IN AXIAL SPONDYLOARTHRITIS

He Z., Littmann N.S.[#1], Arends S.[1], Paap D.[1,2], van der Kraan Y.M.[1], Wink F.[1,3], Verstappen G.[1], Spoorenberg A.[1]

[1]Dept. of Rheumatology and Clinical Immunology, University Medical Center Groningen, University of Groningen, Groningen; [2]Dept. of physiotherapy, Saxion University of Applied Sciences, Enschede; [3]Dept. of Rheumatology, Frisius Medical Center, Leeuwarden, The Netherlands
[#]Contributed equally

Introduction. Chronic pain, a main symptom of axial spondyloarthritis (axSpA) is reported differently by men and women (1). Both immunological (*e.g.* pro-inflammatory cytokines) and non-immunological factors may contribute to chronic pain in axSpA. In rheumatoid arthritis, the pro-inflammatory adipokine leptin is associated with chronic pain (2). This study explored the associations between pro-inflammatory biomarkers and pain-related outcomes in axSpA patients accounting for sex.

Methods. Explorative cross-sectional study from the Groningen Leeuwarden axSpA (GLAS) cohort. Patients who performed quantitative sensory tests (pain thresholds (PPTs), temporal summation (TS), conditioned pain modulation (CPM)) and had serum stored at the index visit were included (3). Patient and disease characteristics including Central Sensitization Inventory (CSI) and treatment were assessed according to the GLAS protocol. Serum levels of IL-6, IL-1β, TNF-α, IL-17A, IL-12/23p40 and leptin were measured using custom U-PLEX multiplex immunoassays. Univariable and multivariable regression analyses explored associations between the biomarkers and pain-related outcomes. Secukinumab and etanercept users were excluded in IL-17A and TNF-α analyses, respectively, due to essay interference.

Results. 167 patients were included (63.5% male, mean age 51±14, symptom duration 23.2±13.3 years, ASDAS 2.2±0.9, BASDAI 3.9±2.2, median CRP 2(1-5), 42.5% used TNF-a inhibitors and 7.2% secukinumab). In univariable analyses, only logIL-17A was significantly associated with higher ASDAS, BASDAI and more axial and peripheral pain (BASDAI-Q2,3). In multivariable analyses, logIL-17A was only significantly associated with more peripheral pain after adjustment for sex. In sex-stratified analyses, logIL-17A was significantly associated with higher ASDAS and more peripheral pain in females, whereas in males it was associated with higher CPM percentage (Table I). No significant associations were found between other biomarkers and pain-related outcomes.

Table I. Univariable and multivariable regression analyses exploring the association of pro-inflammatory biomarkers and clinical outcomes.

Biomarkers	Outcomes	Whole group		Male		Female	
		β (95%CI)	P-value	β (95%CI)	P-value	β (95%CI)	P-value
logIL-17A	ASDAS	0.292 (0.030-0.554)	0.029	-0.030 (-0.446-0.386)	0.887	0.381 (0.049-0.713)	0.025
logIL-17A	BASDAI	0.702 (0.095-1.309)	0.024	0.272 (-0.695-1.238)	0.578	0.708 (-0.079-1.496)	0.077
logIL-17A	BASDAI axial pain (Q2)	0.911 (0.125-1.697)	0.023	0.313 (-0.914-1.539)	0.614	0.930 (-0.131-1.992)	0.084
logIL-17A	BASDAI peripheral pain (Q3)	1.415 (0.620-2.209)	<0.001	0.826 (-0.385-2.038)	0.179	1.579 (0.428-2.729)	0.008
logIL-17A	CRP	0.010 (-0.109-0.129)	0.866	-0.099 (-0.279-0.081)	0.279	0.011 (-0.150-0.171)	0.894
logIL-17A	CSI (total score)	3.854 (-0.120-7.829)	0.057	1.398 (-4.535-7.332)	0.641	2.717 (-2.607-8.041)	0.311
logIL-17A	CPM percentage	2.304 (-3.368-7.977)	0.423	9.505 (0.088-18.923)	0.048	-2.155 (-9.177-4.867)	0.541
logIL-17A	TS (mean)	-0.232 (-0.579-0.115)	0.188	-0.122 (-0.674-0.430)	0.661	-0.400 (-0.873-0.072)	0.095
logIL-17A	PPT (mean)	-3.866 (-8.728-0.956)	0.115	-1.649 (-9.867-6.569)	0.691	-1.908 (-6.713-2.897)	0.429
logIL-17A	Number of tender enthesis	0.107 (-0.057-0.271)	0.198	-0.042 (-0.357-0.274)	0.788	0.100 (-0.108-0.308)	0.333

Skewed continuous variables were log-transformed before entering the regression models.

Conclusion. In axSpA, potential sex-specific associations between IL-17A and pain-related outcomes were identified, whereas other systemic inflammation-related biomarkers showed no clear associations.

References

1. VAN DER KRAAN YM *et al*.: Sex differences in pain-related biopsychosocial assessments in patients with axial spondyloarthritis. *Arthritis Res Ther* 2026; 28(1): 27.
2. ARONSSON M *et al*.: High leptin levels in blood are associated with chronic widespread pain in rheumatoid arthritis. *Arthritis Res Ther* 2024; 26(1): 228
3. VAN DER KRAAN YM *et al*.: Value of the central sensitisation inventory in patients with axial spondyloarthritis. *RMD Open*. 2024; 10(4)

P38

PERIPHERAL BLOOD TRANSCRIPTOMIC PROGRAMS DISTINGUISH RADIOGRAPHIC PROGRESSION TRAJECTORIES IN AXIAL SPONDYLOARTHRITIS INDEPENDENT OF DISEASE ACTIVITY AND TREATMENT

Hwang M.C.[1], Wang X.[2], Brown M.A.[3], Weisman M.H.[4], Ishimori M.L.[5], Gensler L.S.[6], Ward M.M.[7], Reveille J.D.[1], Assassi S.[1]

[1]Division of Rheumatology, McGovern Medical School, University of Texas Health Science Center at Houston, Houston, TX; [2]Baylor Institute for Immunology Research, Baylor Health Care System, Dallas, TX, USA; [3]NIHR Biomedical Research Centre, King's College London, London, UK; [4]Division of Rheumatology, Stanford University, Palo Alto, CA; [5]Division of Rheumatology, Cedars-Sinai Medical Center, Los Angeles, CA; [6]Division of Rheumatology, University of California San Francisco, San Francisco, CA; [7]Verier Outcomes Research, Rockville, MD, USA

Introduction. Structural progression in axial spondyloarthritis (axSpA) is heterogeneous and incompletely captured by conventional clinical measures. We evaluated whether baseline peripheral blood transcriptional modules distinguish long-term radiographic progression trajectories in ankylosing spondylitis independent of demographics, disease activity, inflammation, and treatment.

Methods. Baseline whole-blood RNA-seq from 218 PSOAS participants with radiographic axSpA and RIN >7.0 was analyzed. Patients were classified using predefined 15-year mSASSS trajectories as non-progressors (n=114) or progressors (late, early, or rapid; n=104; Table I) (1). Differential gene expression was assessed with DESeq2. Gene set enrichment analysis used 62 curated whole-blood transcriptional modules from the modular transcriptional repertoire framework (2). Models adjusted for age, sex, NSAID use, TNF inhibitor use, BASDAI, and CRP. Module results are reported as normalized enrichment scores (NES) with Benjamini-Hochberg FDR correction.

Results. Progressors were older and had higher baseline mSASSS than non-progressors, while NSAID and TNF inhibitor use were similar (Table I). In both demographics-adjusted and fully adjusted models, progressors showed increased enrichment of myeloid-lineage (M5.7, M3.2), inflammatory (M5.1, M4.13), neutrophil/granulocyte (M4.9, M6.20), and interferon-response modules (M3.4, M5.12; Fig. 1). Conversely, progressors showed decreased enrichment of protein synthesis (M4.3, M5.9, M3.5), mitochondria/proteasome (M5.10), plasmablast (M4.11), B-cell (M4.10), lymphoid-lineage (M6.12), and T-cell modules (M4.1). These module-level signals remained directionally consistent after adjustment for BASDAI, CRP, and treatment exposure.

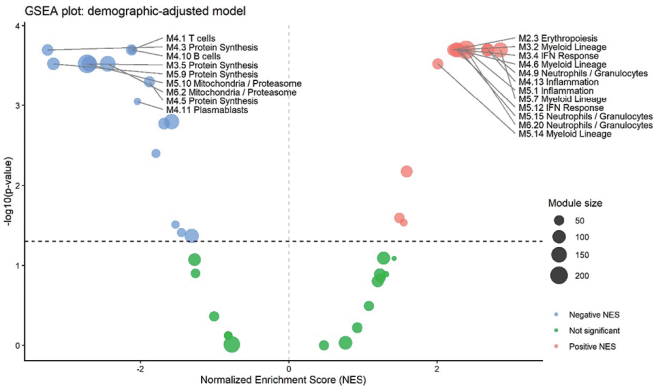
Conclusion. Radiographic progression trajectories in axSpA are associated with a peripheral blood transcriptional architecture characterized by increased innate/myeloid and interferon-associated signals and lower adaptive immune and metabolic module enrichment. These findings suggest that structural progression reflects biologically distinct immune programs not fully explained by clinical disease activity, systemic CRP, NSAID use, or TNF inhibitor exposure, supporting further evaluation of transcriptional modules as progression biomarkers.

Acknowledgements. This work is supported by the National Institute of Arthritis and Musculoskeletal and Skin Diseases/National Institutes of Health (NIAMS/NIH) K23 Program (5K23AR083506).

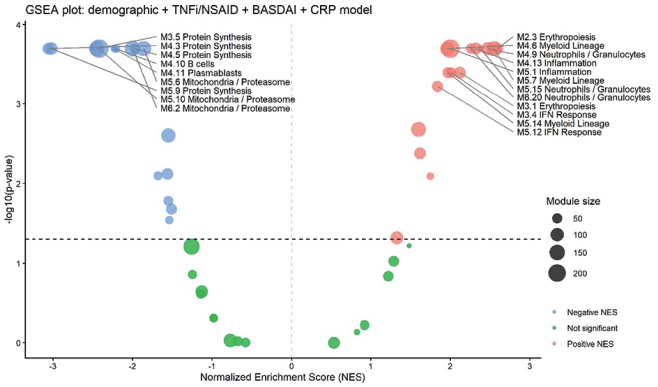
Table I. Demographics stratified by progressor status (non-progressors vs. progressors, n=218)*.

Characteristic	Overall N=218	Non-progressor N=114	Progressor N=104	p-value
Group				<0.001
1 (Non-progressors)	114 (52.3%)	114 (100%)	0 (0%)	
2 (Late-progressors)	20 (9.2%)	0 (0%)	20 (19.2%)	
3 (Early-progressors)	35 (16.1%)	0 (0%)	35 (33.7%)	
4 (Rapid-progressors)	49 (22.5%)	0 (0%)	49 (47.1%)	
NSAID use	142 (65.1%)	77 (67.5%)	65 (62.5%)	0.62
TNFi use	128 (58.7%)	67 (58.8%)	61 (58.7%)	0.98
Gender				0.52
Female	61 (28.0%)	35 (30.7%)	26 (25.0%)	
Male	157 (72.0%)	79 (69.3%)	78 (75.0%)	
Ethnicity				>0.99
Hispanic	6 (2.8%)	3 (2.6%)	3 (2.9%)	
Non-Hispanic	212 (97.2%)	111 (97.4%)	101 (97.1%)	
Race				>0.99
Caucasian	191 (87.6%)	99 (86.8%)	92 (88.5%)	
African-American	4 (1.8%)	2 (1.8%)	2 (1.9%)	
Asian	18 (8.3%)	10 (8.8%)	8 (7.7%)	
Native American/Alaskan Native	3 (1.4%)	2 (1.8%)	1 (1.0%)	
Multi-racial	2 (0.9%)	1 (0.9%)	1 (1.0%)	
Age, mean (SD)	39.35 (13.05)	33.86 (10.37)	45.38 (12.78)	<0.001
BASDAI, mean (SD)	3.14 (2.25)	2.86 (2.10)	3.46 (2.39)	0.054
mSASSS, mean (SD)	17.48 (23.14)	0.68 (2.84)	35.87 (21.36)	<0.001

**All PSOAS RNA patient samples had RIN >7.0.*



a. GSEA demographics-adjusted model comparing rapid, early, and late progressors (n=104) with non-progressors (n=114). Dot size represents number of genes per module.



b. GSEA fully adjusted model including demographics, medications, disease activity, and C-reactive protein comparing progressors with non-progressors. Dot size represents number of genes per module.

Fig. 1. Volcano-style plots of gene-set enrichment analyses comparing axial spondyloarthritis radiographic trajectories (progressors *vs*. non-progressors).

References

1. HWANG, MC, LEE M, GENSLER LS *et al*.: Identifying trajectories of radiographic spinal disease in ankylosing spondylitis: a 15-year follow-up study of the PSOAS cohort. *Rheumatology* (Oxford) 2022; 61: 2079-87. 10.1093/rheumatology/keab661
2. CHAUSSABEL D, BALDWIN N: Democratizing systems immunology with modular transcriptional repertoire analyses. *Nat Rev Immunol* 2014; 14: 271-80. 10.1038/nri3642

P39

BEYOND DYSLIPIDEMIA: APOLIPOPROTEINS AS MARKERS OF DISEASE ACTIVITY IN PSORIATIC ARTHRITIS

Ishchenko A.[1], Van Mechelen M.[2], Neerinckx B.[3,4], Verschueren P.[3,4], Lories R.[3,4], de Vlam K.[3,4]
[1]Dept. of Rheumatology, Ziekenhuis Netwerk Antwerpen (ZNA), Antwerp; [2]Dept. of Immunology, Allergology and Rheumatology, University of Antwerp, Antwerp University Hospital, Antwerp; [3]Division of Rheumatology, University Hospitals Leuven, Leuven; [4]Skeletal Biology and Engineering Research Centre, Dept. of Development and Regeneration, KU Leuven, Leuven, Belgium
Corresponding author: Prof. Kurt de Vlam, Reumatologie, UZ Leuven, Belgium.
ORCID ID: 0000-0003-3848-5122

Introduction/Objective. Early PsA patients have a pro-atherogenic lipid profile with low ApoA1 and high ApoB levels, distinct from early RA (1). We suggest that apolipoprotein changes might be linked to psoriatic arthritis at a pathophysiological level and reflect the disease activity in PsA.
Materials and methods. Adult patients with newly diagnosed, DMARD-naive PsA patients were recruited. Patients with early RA were used as a comparative arthritis population. ApoA1 and ApoB levels were measured (ELISA) at baseline and at 1-year follow-up.
Results. PsA patients (n=75) had mean age of 47.8 years, 66.7% male, median symptom duration 0.6 years. RA patients (n=50), had mean age comparable to PsA of 51.4 years, were predominantly female (72%), had median symptom duration of 0.5 years[2].
The ApoA1 levels correlated with TJC68 and SJC68 (ϱ=0.35 and ϱ=0.341 accordingly, $p<0.001$) but not with inflammation markers: CRP, S100A8/9 and C3 (Fig. 1). In PsA, ApoA1 correlated with baseline PASI (ϱ=0.33, $p=0.46$), despite moderate skin involvement (median: 1.2).
For 52 PsA and 50 RA patents 1-year follow-up data was available. At baseline, PsA patients had mean±SD DAPSA of 19.5±10.8 (MDA). After 1 year, 44 patients (85%) achieved DAPSA <15 (LDA, mean DAPSA 8.3±10.2). In these patients, ApoA1 levels increased at 1 year as compared to baseline (t=2.5; $p=0.016$); this was not the case in patients with active disease or in RA. In PsA, the ApoB levels remained comparable to baseline despite improvement of disease activity (Fig. 2).
Conclusion. The data suggests that ApoA1 in Apo A1 levels correlate with joint involvement and disease activity in early PsA. Decrease in disease activity improves ApoA1 levels in PsA, which is not the case in RA. In conclusion, changes of ApoA1 levels are specific to PsA and are linked to disease activity, underlining the role of lipid changes in early disease.

	Age	BMI	TJC68	SJC66	N_big_joints	CRP, mg/L	C3_g/L	ApoA1, µg/ml	ApoB, µg/ml/ml	S100A8/A9, µg/ml
Age	1,000	0,266	0,18	0,191	0,025	0,149	0,126	0,088	0,020	0,132
BMI	0,266	1,000	0,218	0,213	0,162	0,297	0,398	-0,15	-0,001	0,185
TJC68	0,18	0,218	1,000	0,931	0,314	0,505	0,409	0,305	-0,454	0,454
SJC66	0,191	0,213	0,931	1,000	0,327	0,521	0,407	0,341	-0,457	0,499
N_big_joints	0,025	0,162	0,314	0,327	1,000	0,347	0,358	-0,306	-0,120	0,281
CRP, mg/L	0,149	0,297	0,505	0,521	0,347	1,000	0,643	0,004	-0,175	0,574
C3_g/L	0,126	0,398	0,409	0,407	0,358	0,643	1,000	-0,019	-0,128	0,433
ApoA1, µg/ml	0,088	-0,15	0,305	0,341	-0,306	0,004	-0,019	1,000	-0,144	-0,005
ApoB, µg/ml/ml	0,020	-0,001	-0,454	-0,457	-0,121	-0,175	-0,128	-0,144	1,000	-0,224
S100A8/A9, µg/ml	0,132	0,185	0,454	0,499	0,281	0,574	0,433	-0,005	-0,224	1,000

Fig 1. Spearman correlation heatmap showing association between apolipoproteins and disease activity parameters.
Spearman correlation analysis: the cells indicate Spearman's ϱ, significant results are noted in bold. Weak correlation: Spaerman's ϱ 0.2-0.39; moderate: 0.4-0.59, strong: 0.6-0.79. Blue represents negative correlations (ϱ = −1), white represents no correlation (ϱ = 0) and red represents positive correlations (ϱ = +1). Increasing color intensity corresponds to increasing correlation strength.
BMI: body mass index; TJC68: 68 tender joint count; SJC66: 66 swollen joint count; ApoA1: apolipoprotein A1; ApoB: apolipoprotein B; S100A8/9: serum calprotectin S100 A8/9 heterodimer.

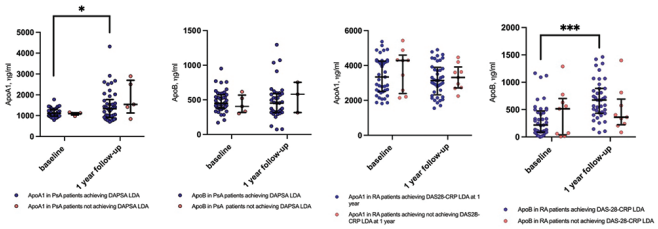


Fig 2. Apolipoprotein levels at baseline and at 1-year follow-up in patients with PsA and RA, grouped by disease activity.
A. In PsA, the levels of ApoA1 (µg/ml, mean (±SD)) increased at 1-year follow-up as compared to baseline in patients achieving DAPSA LDA (baseline: 1160±217, 1 year: 1336±543; $t^1=2.5$; $p=0.016$) and remained comparable to baseline in patients not-achieving DAPSA-LDA (baseline: 1052±67, 1-year: 1264±375; t=-2.01, $p=0.106$).
B. The ApoB levels (µg/ml, mean(±SD)) were comparable at baseline and at 1-year in patients achieving DAPSA LDA (baseline: 486±162, 1 year: 482±258; t=0.114; $p=0.910$) and not achieving DAPSA LDA (baseline: 442±160; 1-year: 551±220; t=-0.904, $p=0.461$).
C. In RA, the levels of ApoA1 remained comparable at baseline and at 1-year follow-up in patients achieving DAS-28-CRP LDA (baseline:3436±1052, 1 year 3201±941; $t^1=0.93$; $p=0.357$) and not-achieving DAS-28-CRP-LDA (baseline: 3734±1195, 1-year: 3319±731; t=0.846, $p=0.422$).
D. In RA, the ApoB levels were increased at 1 year as compared to baseline in patients achieving DAS-28-CRP LDA (baseline: 312±306, 1 year 685±359; t=-5.39, $p<0.001$). In patients not achieving DAS-28-CRP LDA, 1-year levels were comparable to baseline (baseline: 436±439, 1 year: 491±402; t=-0.251, $p=0.808$).
1 – paired samples t-test was used to compare baseline and 1-year follow-up data
PsA: psoriatic arthritis; RA: rheumatoid arthritis; DAPSA: disease activity in psoriatic arthritis; DAS-28: Disease activity in 28 joints; LDA: low disease activity; ApoA1: apolipoprotein A1; ApoB: apolipoprotein B.

References
1. ISHCHENKO A, VAN MECHELEN M, STORMS L *et al*.: Low apolipoprotein A1 and high apolipoprotein B levels indicate specific lipid changes in treatment naïve early psoriatic arthritis. *RMD Open* 2025.
2. ISHCHENKO A, PAZMINO S, NEERINCKX B, LORIES R, DE VLAM K: Comorbidities in early psoriatic arthritis: data from the Metabolic Disturbances in Psoriatic Arthritis Cohort Study. *Arthritis Care Res* (Hoboken) 2024; 76(2): 231-40.

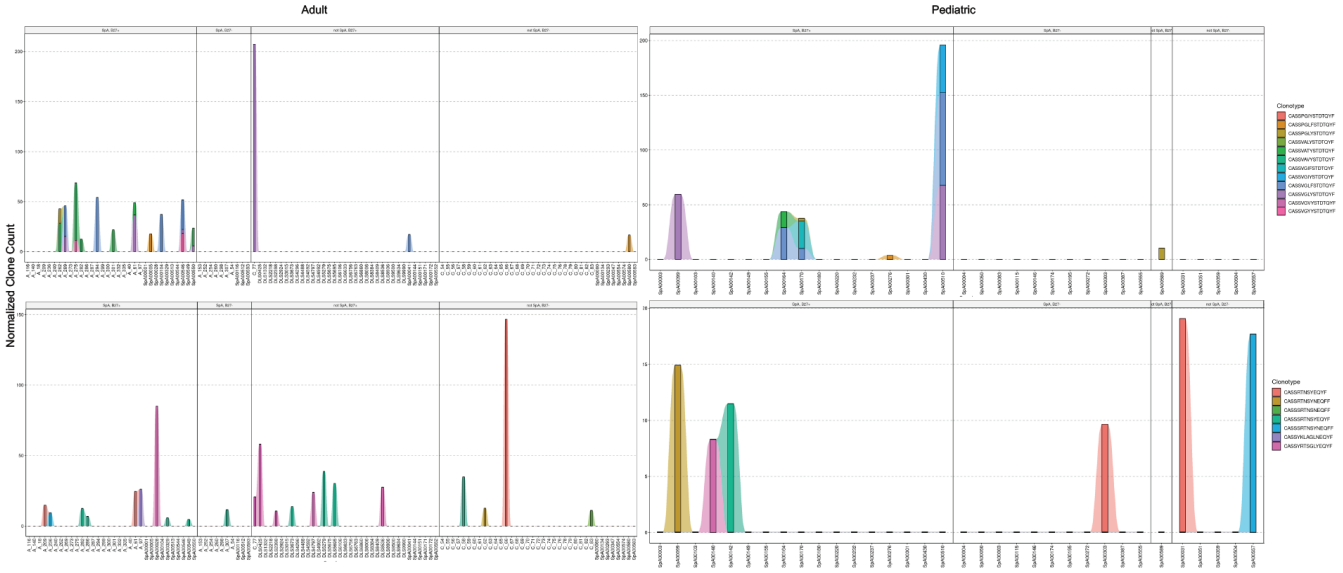
P40

CLONAL T CELLS IN JUVENILE AND ADULT SPONDYLOARTHRITIS

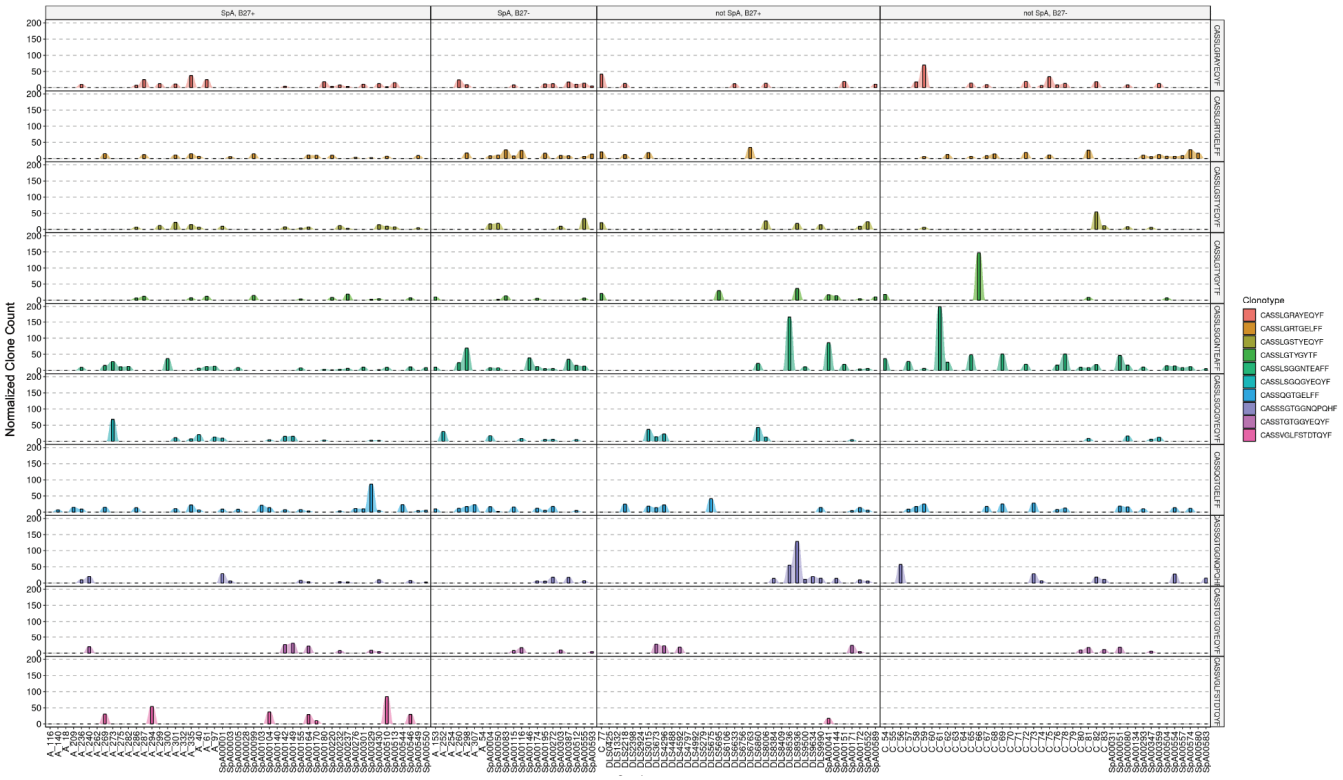
Lotfi-Emran S.[1], Kumar D.[1], Paul S.[2], Schaughency P.[2], Lack J.[2], Islam S.[3], Dell'Orso S.[3], Gensler L.S.[4], Navid F.[1], Colbert R.A.[1]
[1]Pediatric Translational Research Branch, NIAMS; [2]Integrated Data Sciences Section, NIAID, [3]Genomic Technology Section, NIAMS, [4]University of California, San Francisco, USA

Introduction/Objective. Recent demonstration that Axial Spondyloarthritis (AxSpA)-associated, clonal CD8+ T cell receptors (TCRs), identified by their complementarity determining region 3 (CDR3) motif Y/FSTDTQ, recognize HLA-B27 bound self and microbial peptides has reinvigorated this arthritogenic peptide hypothesis. This study aims to evaluate our AxSpA cohort, which includes both children and adults, for the presence of previously reported CDR3 motifs.
Materials/methods. Samples were collected from an ongoing, prospective observational cohort at NIAMS. HLA-B27 status was determined by clinical testing (CLIA certified lab) for samples labelled "SpAxxxxx" and "DLSxxxxx" and otherwise inferred from bulk RNA sequencing by ArcasHLA. RNA was isolated from peripheral blood PAXgene tubes, assessed for integrity, globulin mRNA and rRNA were removed with NEBNext kit, and samples sequenced using NEBNext Immune Sequencing Kit. Data were analyzed in Immcantation, as described by NEB, which aligns reads against germline segments and assembles clonotypes. Data was screened for clonal sequences previously reported by Faham *et al*. (1) and Hanson (2) *et al*.
Results. The Y/FSTDTQ motif *family* is identified in 33% of HLA-B27+ AxSpA, 0% of B27- AxSpA, 8% of B27+ controls, and 2% of B27- controls, and in children and adults. The CASSYRTSGLYEQ sequence (Fig. 1A) is identified in 4% of B27+ AxSpA and 14% of B27+ controls but not in B27- individuals, with or without AxSpA. (Fig. 1B) Only the Y/FSTDTQ sequence reported by Hanson et al. was associated with either HLA-B27 carriage and AxSpA. (Fig. 2)
Conclusion. The Y/FSTDTQ CDR3 motif can be identified in children and adults who are HLA-B27+ and more prominently for those with AxSpA. The CASSYRTSGLYEQ CDR3 sequence is associated with HLA-B27 carriage but its association with AxSpA remains unclear. An optimized approach to generating CDR3 motif families is likely required to improve detection of clonal TCRs to enable discovery of new motifs, particularly those associated with HLA-B27- AxSpA.

References
1. FAHAM M, CARLTON V, MOORHEAD M *et al*.: Discovery of T cell receptor β motifs specific to HLA-B27-positive ankylosing spondylitis by deep repertoire sequence analysis. *Arthritis Rheumatol* 2017; 69(4): 774-84. doi: 10.1002/art.40028. PMID: 28002888.
2. HANSON AL, NEL HJ, BRADBURY L *et al*.: Altered repertoire diversity and disease-associated clonal expansions revealed by T cell receptor immunosequencing in ankylosing spondylitis patients. *Arthritis Rheumatol* 2020; 72(8): 1289-302. doi: 10.1002/art.41252. Epub 2020 Jul 13. PMID: 32162785.



P40 - Fig. 1. Normalized clone counts for clonal motifs identified by Faham *et al*. can be identified in children as well as adults with AxSpA.
A. Y/FSTDTQ *family* of motifs are confirmed to be associated with carriage of HLA-B27, moreso in HLA-B27+ AxSpA.
B. The remainder of Faham *et al*. *individual sequences* are shown demostrating CASSYRTSGLYEQ association with HLA-B27 carriage.



P40 - Fig. 2. Normalized clone counts for clonal sequences identified by Hanson *et al*. demonstrating lone association of reported Y/FSTDTQ with HLA-B27+ AxSpA.

P41

ANTI-CD74 IGA AUTOANTIBODIES IN AXIAL SPONDYLOARTHRITIS

Elbana N.K.[1], Gharbia O.M.[1], El Arman M.M.[2], Machaly S.A.[1]
[1]Dept. of Rheumatology, Faculty of Medicine- Mansoura University, Mansoura; [2]Dept. of Clinical Pathology, Faculty of Medicine- Mansoura University, Mansoura, Egypt

Introduction. CD74 is a molecule involved in antigen presentation and immune activation. It is also the receptor for macrophage migration inhibitory factor (MIF), which promotes inflammation. In patients with axial spondyloarthritis, the immune system appears to produce IgA autoantibodies against CD74. Accordingly, anti-CD74 IgA has been proposed as a promising biomarker for axial spondyloarthritis, although its diagnostic and prognostic significance is still being evaluated.
Aim. The study aimed to assess the diagnostic value of anti-CD74 IgA antibodies for axSpA and its association with structural damage.
Patients and methods. Anti-CD74 IgA was analyzed by ELISA in the serum of 45 patients with axSpA who met the ASAS criteria for axial spondyloarthritis and 45 age- and sex-matched patients of chronic back pain (CBP) served as controls. BASDAI, ASDAS, BASFI and BASMI were assessed. Spinal and sacroiliac radiographs were scored with mSASSS and modified New York criteria for sacroilitis respectively.
Results. This cross-sectional case-control study revealed high serum Anti-CD74 IgA in axSpA patients compared with controls with specificity 77.78% and sensitivity 80.0%. Anti-CD74 IgA levels showed a significant positive correlation with mSASSS for spines, modified New York criteria for sacroilitis, BASMI and BASFI (p<0.001). Anti-CD74 IgA levels had no significant correlation with inflammatory markers (ESR and CRP), BASDAI, ASDAS ESR and ASDAS CRP (p=0.797, p=0.956, p=0.079, p=0.155 and p=0.247).
Conclusion. Anti-CD74 IgA is a useful tool for identifying axSpA with high sensitivity and specificity, also associated with structural damage development in the sacroiliac joints and spines.

P42

PERFORMANCE OF THE 2025 ASAS-SPARTAN REVISED CLASSIFICATION CRITERIA FOR AXIAL SPONDYLOARTHRITIS IN A MULTICENTER INCEPTION COHORT WITH PSORIASIS, UVEITIS, OR COLITIS PRESENTING WITH UNDIAGNOSED BACK PAIN

Wichuk S.[1], Horbal N.[1], Carmona R.[3], Weber U.[4], Aydin S.Z.[5], Yeung J.[6], Reis J.[7], Masetto A.[8], Rohekar S.[9], Mosher D.[10], Zouzina O.[10], Martin L.[10], Keeling S.O.[1], Paschke J.[11], Dadashova R.[11], Carapellucci A.[11], Lambert R.G.W.[12], Chan J.[13], Maksymowych W.P.[1]
[1]University of Alberta, Medicine, Edmonton, Canada; [2]Danylo Halytsky Lviv National Medical University, Medicine, Lviv, Ukraine; [3]McMaster University, Medicine, Hamilton, Canada; [4]Medical Centre Zenit, Rheumatology, Schaffhausen, Switzerland; [5]University of Ottawa, Medicine, Ottawa; [6]Richmond Health Sciences Centre, Richmond; [7]College of Medicine, University of Saskatchewan, Medicine, Saskatoon; [8]Université de Sherbrooke, Medicine, Sherbrooke; [9]Western University, Medicine, London; [10]University of Calgary, Medicine, Calgary; [11]CARE ARTHRITIS Limited, Edmonton; [12]University of Alberta, Radiology and Diagnostic Imaging, Edmonton; [13]University of British Columbia, Medicine, Vancouver, Canada

Background. Analysis of the Classification in Axial Spondyloarthritis Inception Cohort (CLASSIC) generated the 2025 ASAS-SPARTAN revised criteria with sensitivity/specificity of 79.5%/90.4%. Performance of the revised criteria should be examined in additional cohorts.
Methods. The multicenter Screening for AxSpA in Psoriasis, Iritis, and Colitis (SASPIC) Study was aimed at early detection of axSpA in consecutive patients presenting with undiagnosed back pain. Consecutive patients ≤45 years of age with ≥3 months undiagnosed back pain with psoriasis, acute anterior uveitis (AAU), or colitis had routine clinical evaluation by a rheumatologist for axial SpA. The design of the SASPIC study was similar to the CLASSIC study. Final diagnosis by the rheumatologist was used as gold standard to test the performance of the 2009 and 2025 criteria according to sensitivity and specificity [95%CI]. We conducted regression to determine which SpA variables were independently associated with the final rheumatologist diagnosis.
Results. 410 patients were recruited to the SASPIC cohorts, axSpA being diagnosed in 124 (40.1%). For the entire cohort, sensitivity/specificity of the 2009 criteria were 74.2%/80.5%, with variable performance across the 3 subgroups (Table I), while sensitivity/specificity of the 2025 criteria were

P42 - Table I. Performance of the 2009 ASAS versus the 2025 ASAS SPARTAN classification criteria in the SASPIC cohort.

Cases	ASAS 2009 Overall		ASAS 2009 Imaging Arm		ASAS 2009 Clinical Arm		ASAS-SPARTAN 2025 Overall		ASAS-SPARTAN 2025 Imaging arm		ASAS-SPARTAN 2025Clinical Arm	
	Sensitivity 95%CI	Specificity 95%CI	Sensitivity 95%CI	Specificity 95%CI	Sensitivity 95%CI	Specificity 95%CI	Sensitivity 95%CI	Specificity 95%CI	Sensitivity 95%CI	Specificity 95%CI	Sensitivity 95%CI	Specificity 95%CI
All SASPIC N=309	74.2 (66.5-81.9)	80.5 (74.8-86.2)	54.8 (46.1-63.6)	98.4 (96.6-100.2)	54.8 (46.1-63.6)	81.6 (76-87.2)	56.5 (47.3-65.3)	98.9 (96.1-99.9)	56.5 (47.3-65.3)	98.9 (96.1-99.9)	0	100
SASPIC-PsO N=84	57.7 (38.7-76.7)	89.7 (81.8-97.5)	50 (30.8-69.2)	98.3 (94.9-101.6)	26.9 (9.9-44)	91.4 (84.2-98.6)	53.9 (33.4-73.4)	98.3 (90.8-100.)	53.9 (33.4-73.4)	98.3 (90.8-100.0)	0	100
SASPIC-AAU N=103	88.7 (80.8-96.6)	53.7 (38.4-68.9)	61.3 (49.2-73.4)	100 (100-100)	77.4 (67-87.8)	53.7 (38.4-68.9)	59.7 (46.4-71.9)	97.6 (87.1-100)	59.7 (46.4-71.9)	97.6 (87.1-99.9)	0	100
SASPIC-IBD N=122	61.1 (45.2-77)	87.2 (80.2-94.3)	47.2 (30.9-63.5)	97.7 (94.5-100.9)	36.1 (20.4-51.8)	88.4 (81.6-95.1)	52.8 (35.5-69.6)	100.0 (95.8-100)	52.8 (35.5-69.6)	100.0 (95.8-100.0)	0	100

P42 - Table II. Univariable and multivariable regression analyses, the dependent variable being final axSpA local rheumatologist diagnosis.

SpA features	Univariable OR (95% CI)	Multivariable OR (95% CI)		
		Model 1	Model 2	Model 3
MRI-SIJ indicative of axSpA	**140.1 (33.2-590.7)**	NI	NI	**62.1(13.1 to 294.7)**
Radiographic sacroiliitis (mNY)*	**8.7E+009**	**4.8E+009**	NI	**157E+006**
HLA-B27	**4.4 (2.7-7.2)**	**2.9 (1.5 to 5.7)**	**3.4 (1.8 to 6.4)**	**2.4 (1.1 to 5.2)**
IBP clinician global	**2.5 (1.4-4.4)**	**2.9 (1.4 to 6.3)**	**2.4 (1.2 to 4.7)**	**3.8 (1.1 to 5.2)**
IBD	**0.5 (0.3-0.9)**	1.0 (0.2 to 4.3)	1.4 (0.4 to 4.7)	0.9 (0.1 to 5.9)
Peripheral arthritis	0.9 (0.4-2.1)	0.8 (0.3 to 2.5)	0.7 (0.2 to 2.3)	1.1 (0.3 to 3.9)
Heel enthesitis	**0.3 (0.1-0.7)**	0.5 (0.2 to 1.2)	0.4 (0.1 to 1.0)	0.6 (0.2 to 1.7)
AAU	**3.6 (2.2-5.9)**	2.1 (0.5 to 9.2)	3.3 (0.9 to 11.2)	1.7 (0.3 to 10.9)
Dactylitis, current	2.5 (0.6-10.9)	3.0 (0.4 to 20.1)	3.0 (0.5 to 20.0)	1.6 (0.1 to 20.8)
Psoriasis	0.6 (0.4-1.0)	1.0 (0.2 to 4.4)	1.6 (0.5 to 5.6)	0.9 (0.2 to 5.3)
Good response to NSAIDs	1.4 (0.9-2.3)	1.2 (0.6 to 2.1)	1.3 (0.7 to 2.3)	1.0 (0.5 to 2.0)
Family history for SpA (ASAS definition)	0.6 (0.4-1.0)	0.5 (0.3 to 1.0)	**0.5 (0.3 to 1.0)**	0.5 (0.2 to 1.1)
Elevated CRP, current (>5 mg/L)	**3.0 (1.8-5.0)**	**2.4 (1.3 to 4.7)**	**3.0 (1.6 to 5.6)**	2.1 (1.0 to 4.4)
Male gender	**3.1 (2.0-5.0)**	**2.8 (1.6 to 5.1)**	**3.2 (1.8 to 5.6)**	**2.2 (1.1 to 4.3)**

NI: not included *n=307 cases where mNY data available, all cases with radiographic sacroiliitis were diagnosed with axSpA which makes OR estimates unreliable with logistic regression.

56.5% and 98.9%, performance being consistent among the 3 subgroups. No patients were classified with axSpA if both radiography and MRI were considered negative for axSpA. Variables associated with diagnosis of axSpA in univariable regression were almost the same as those selected by regression analyses of CLASSIC leading to the 2025 revised criteria (Table II). In multivariate regression, the highest odds ratios were noted for MRI indicative of axSpA, radiographic sacroiliitis, HLA-B27, clinician global inflammatory back pain, male gender, and elevated CRP.
Conclusion. The 2025 ASAS SPARTAN revised criteria for axSpA perform with consistently higher specificity than the 2009 criteria in an inception cohort with back pain and psoriasis, AAU, or IBD.

P43

ANTI-14-3-3ETA AUTOANTIBODIES SUPPORT DIFFERENTIAL DIAGNOSIS OF AXIAL SPONDYLOARTHRITIS FROM MECHANICAL AND NON-AXSPA BACK PAIN

Marotta A.[1], Sengupta R.[2], Maksymowych W.P.[3], Peng J.[1], Cavill C.[2], Gui Y.[1], Bleakley S.[1], Wichuk S.[3], Biln N.[1]
[1]Augurex Life Sciences Corp., Vancouver, Canada; [2]National Hospital for Rheumatic Diseases, Bath, UK; [3]University of Alberta, Division of Rheumatology, Edmonton, Canada

Introduction. Patients with axial spondyloarthritis (axSpA) frequently experience prolonged diagnostic delay, driven by symptom overlap between inflammatory back pain and mechanical back pain (MBP) or other musculoskeletal conditions, and by the lack of objective serological markers to support differential diagnosis. Earlier, more confident identification of axSpA has direct implications for timely access to appropriate therapy. This study evaluated the diagnostic performance of anti-14-3-3eta autoantibodies, alone and in combination with age and sex, to discriminate axSpA from MBP and disease controls representing conditions with overlapping symptomatology.
Methods. Diagnostic performance of anti-14-3-3eta autoantibodies, alone and combined with age and sex, was evaluated in patients with axSpA (n=109) versus MBP (n=51) and other non-axSpA conditions (n=375). Non-axSpA conditions included autoimmune and inflammatory conditions (rheumatoid arthritis, psoriatic arthritis/psoriasis, Crohn's disease, ulcerative colitis) and non-inflammatory joint conditions (osteoarthritis, gout). Sensitivity, specificity, and AUC were assessed across comparator populations.
Results. When comparing axSpA to MBP, the anti-14-3-3eta model achieved an AUC of 0.77 (95% CI: 0.69-0.84), 90% specificity, and 42% sensitivity. Incorporating age and sex improved performance, with AUC increasing to 0.88 (95% CI: 0.83-0.93), sensitivity to 67%, and DxOR to 18.4; see Table I. In the broader non-axSpA comparator cohort (n=375), the model achieved an AUC of 0.63 (95% CI: 0.56-0.69) with 90% specificity and 23% sensitivity, with comparable performance after addition of age and sex (AUC 0.64, 95% CI: 0.57-0.70; sensitivity 22%); see Table II.
Conclusion. Anti-14-3-3eta autoantibodies demonstrated consistent discrimination of axSpA from MBP and non-axSpA conditions at high specificity, with meaningful improvement when combined with readily available clinical variables. These findings support the role of anti-14-3-3eta as a practical adjunct to help address the diagnostic gap facing patients with suspected axSpA.

Table I. Anti-14-3-3eta autoantibody diagnostic performance: axSpA *vs*. MBP (n=51).

Model (95% CI)	14-3-3eta AAbs	14-3-3eta AAbs + Age, Sex
AUC	0.77 (0.69–0.84)	0.88 (0.83–0.93)
SP	90% (78%–96%)	90% (78%–96%
SN	42% (33%–51%)	67% (58%–75%)
PLR	4.20 (1.79–9.85)	6.70 (2.9–15.42)
NLR	0.64 (0.53–0.77)	0.37 (0.28–0.49)
DxOR	6.5 (2.7–15.6)	18.4 (6.74–50.2)

Table II. Anti-14-3-3eta autoantibody diagnostic performance: axSpA *vs*. non-axSpA comparators (n=375).

Model (95% CI)	14-3-3eta AAbs	14-3-3eta AAbs + Age, Sex
AUC	0.63 (0.56–0.69)	0.64 (0.57–0.70)
SP	90% (87%–93%)	90% (87%–93%)
SN	23% (16%–32%)	22% (15%–31%)
PLR	2.31 (1.46–3.66)	2.20 (1.38–3.51)
NLR	0.86 (0.77–0.95)	0.87 (0.78–0.96)
DxOR	2.7 (1.6–4.8)	2.3 (1.5–4.6)

P44

BEYOND DISEASE ACTIVITY: CLINICAL AND CONTEXTUAL DETERMINANTS OF DIFFICULT-TO-MANAGE AXIAL SPONDYLOARTHRITIS

Masnata R.[1,2], De Lorenzis E.[1,2], Massaro A.[2], Boni V.[1,2], Natalello G.[1,2], Peluso G.[1], Calabrese G.[1], Cristiano F.[1], D'Agostino M.A.[1,2], Ortolan A.[1,2]
[1]Dept. of Rheumatology, Fondazione Policlinico Universitario Agostino Gemelli IRCCS, Rome; [2]Catholic University of the Sacred Heart, Rome, Italy

Background. Failure to achieve or maintain the therapeutic target identifies the difficult-to-manage (D2M) phenotype of axial spondyloarthritis (axSpA), resulting from persistent inflammation and/or contextual factors (CF).
Objective. To describe D2M axSpA patients and identify associated factors.
Methods. Cross-sectional study including consecutive patients with rheumatologist-diagnosed axSpA attending our tertiary-level clinic between 01/25-05/26. Demographic, clinical, therapeutic data and CF were collected. Disease activity was assessed by ASDAS. CF included Rheumatic Disease Comorbidity Index (RDCI), Hospital Anxiety and Depression Scale (HADS-A/HADS-D), International Physical Activity Questionnaire (IPAQ), and Caranci Deprivation Index (CDI), a census-based index including education, employment, housing, overcrowding, linked to Italian demographic data, and extensively validated (the higher, the more deprived). D2M status was defined according to Assessment of axSpA international society (ASAS) definition. Variables were compared between D2M vs non-D2M using Chi-square / Mann-Whitney tests. LASSO regression was performed with D2M status as outcome. Two models were run: unadjusted for current disease activity -to avoid circular reasoning- and, ASDAS-adjusted.
Results. 156 patients were included 26 (17%) fulfilled D2M definition.

Table I. Comparison between not-D2M and D2M patients.

Variables	Non-D2M N=130	D2M N=26	p-value
Male, N (%)	64 (49)	7 (27)	0.037
Age, years, mean (SD)	49.1 (12.0)	53.3 (8.9)	0.14
Disease duration, years, mean (SD)	10.2 ±9.3	7.7 ±4.7	0.60
HLA-B27 positive (N=143), N (%)	48 (41)	10 (40)	0.95
Sacroileitis on RM, ASAS criteria (N=147), N (%)	68 (56)	15 (58)	0.86
Sacroileitis on XR, mNY, N (%)	58 (46)	11 (42)	0.73
Arthritis, (ASAS def), N (%)	54 (42)	14 (54)	0.25
Enthesitis (ASAS def), N (%)	33 (25)	12 (46)	**0.033**
Dactylitis (ASAS def), N (%)	8 (6)	3 (12)	0.33
Psoriasis, N (%)	35 (27)	7 (27)	1.00
Uveitis, N (%)	26 (20)	4 (15)	0.59
IBD, N (%)	7 (5)	0 (0)	0.23
Previous infection, N (%)	4 (3)	1 (4)	0.84
Good response to NSAID, N (%)	77 (61)	9 (35)	**0.013**
CRP elevation > 5 mg/L at diagnosis, N (%)	39 (31)	3 (12)	**0.042**
Inflammatory back pain (ASAS criteria), N (%)	21 (16)	14 (54)	**<0.001**
Family history of SpA (def ASAS), N (%)	18 (14)	2 (8)	0.39
RDCI, mean (SD)	0.9 (1.2)	1.0 (1.2)	0.28
BMI, mean (SD)	25.1 (4.0)	25.2 (4.8)	0.78
Physical activity IPAQ (N=130), N (%) Inactive Sufficiently active Active/very active	 39 (36) 33 (30) 38 (35)	 12 (57) 5 (24) 4 (19)	0.17
Sedentary behaviour (N=138), N (%) Low Moderate High	 63 (55) 41 (36) 11 (10)	 19 (83) 4 (17) 0 (0)	**0.036**
HADS-A, mean (SD)	6.1 ± 4.1	8.9 ± 4.5	**0.007**
HADS-D, mean (SD)	5.5 ± 3.9	8.1 ± 4.2	**0.006**
University degree (N=125), N (%)	37 (36)	7 (33)	0.84
Lives alone (N=125), N (%)	11 (11)	4 (19)	0.28
Caranci Deprivation Index (continuous, national reference) (N=152) mean (SD)	0.8 ±2.6	−0.3 ±1.6	0.07
National quintiles of Caranci Deprivation Index, (N=152) N(%) Q1 (Least deprived) Q2 Q3 Q4 Q5 (most deprived)	 12 (9) 24 (19) 29 (23) 33 (26) 29 (23)	 8 (32) 4 (16) 2 (8) 8 (32) 3 (12)	**0.018**

D2M: difficult-to-manage; HLA-B27: Human Leukocyte Antigen B27; MRI: magnetic resonance imaging; X-ray: conventional radiography; ASAS: Assessment of SpondyloArthritis International Society; RDCI: Rheumatic Disease Comorbidity Index; BMI: Body Mass Index; IPAQ: International Physical Activity Questionnaire; HADS-A: Hospital Anxiety and Depression Scale – Anxiety subscale; HADS-D: Hospital Anxiety and Depression Scale - Depression subscale; EF: Environmental Factors; SD: standard deviation; N: number of patients. The Caranci Deprivation Index is reported as a continuous variable according to the national reference and as national quintiles, with Q1 indicating lower deprivation and Q5 higher deprivation. *p*-values <0.05 were considered statistically significant.

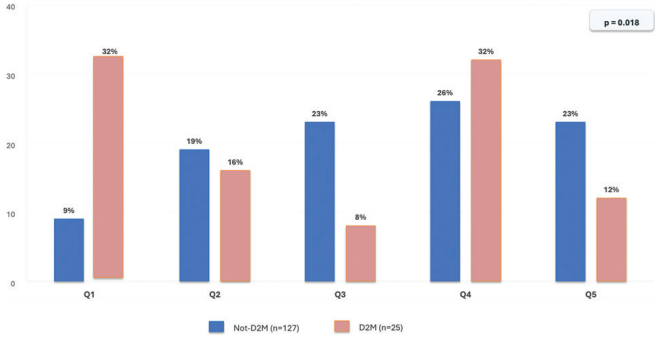


Fig. 1. Distribution of Caranci deprivation quintiles in not-D2M and D2M patients
Q1: least deprived; Q5: most deprived. Percentages are calculated among patients with available Caranci index data.D2M: difficult-to-manage.

D2M patients were predominantly female and showed higher prevalence of enthesitis, and less frequent NSAID response, CRP elevation at diagnosis, inflammatory back pain. D2M patients hadhigher HADS-A, HADS-D and more sedentary lifestyle (Table I). CDI was lower in D2M patients (Fig. 1). In unadjusted LASSO, HADS-D, peripheral involvement and lower CDI were associated with D2M status (λ=0.036). In ASDAS-adjusted LASSO, ASDAS, peripheral involvement and lower IDI were associated with the outcome (λ=0.040).

Discussion. D2M axSpA is a complex phenotype at the intersection of biology, psychology and social context.

The unexpected association between lower deprivation and D2M status, robust to multivariable penalization, suggests that D2M criteria may capture patients who succeed in reaching e remaining within specialists care.

Conclusion. Recognising the social architecture of refractoriness is fundamental for equitable SpA management and requires studies beyond tertiary level centres.

P45

HIGH VERSUS NORMAL CRP SPONDYLOARTHRITIS: STUDYING DEMOGRAPHICS, DISEASE ACTIVITY, FUNCTIONING, QUALITY OF LIFE, AND SCORE DISCORDANCE IMPACT ON TREATMENT DECISIONS

Shah U.[1], Meghnathi B.[1,2,3], Pandya S.[1,2], Sumra A.[2], Shah S.[2]
[1]Smt. NHL Municipal Medical College, Ahmedabad; [2]RheumaCARE, Ahmedabad; [3]Marengo CIMS Hospital, Ahmedabad, India

Introduction. In spondyloarthritis (SpA), approximately 40% of patients have normal C-reactive protein (CRP) despite active disease. ASAS endorses ASDAS-CRP over ASDAS-ESR, yet CRP can act independently of disease activity due to gene polymorphisms (Claushuis *et al.*), and Indian data on CRP-stratified phenotypes is absent. We aimed to compare elevated versus normal CRP SpA patients based on scores and examine BASDAI/ASDAS-CRP discordance and its impact on treatment decisions.

Methods. Prospective cross-sectional study was conducted at a single tertiary centre in India. Adult SpA patients (ASAS 2009 criteria) were enrolled; those with fibromyalgia (2016 ACR criteria) and psychological comorbidities were excluded. CRP grouping was based on values over the preceding 12 weeks; elevation even once classified patients as elevated CRP. Groups were compared on BASDAI, ASDAS-CRP, BASFI, and ASQoL.

Results. Of 154 enrolled, 17 were excluded, yielding n=137 (elevated CRP n=97, normal CRP n=40). Demographics, disease duration, HLA-B27, imaging, and extra-articular features were comparable. Elevated CRP patients had significantly higher BASDAI (4.2 vs. 1.5, $p<0.001$), BASFI (3.2 vs. 1.1, $p<0.001$), ASQoL (7.0 vs. 3.0, $p<0.001$), and ASDAS-CRP (2.9 vs. 1.3, $p<0.001$). In Scenario 1 (BASDAI inactive, ASDAS-CRP active; n=25), rheumatologists aligned with BASDAI in 76% of cases. In Scenario 2 (both scores inactive, CRP elevated; n=24), alignment rose to 95.8%, with majority maintaining or reducing therapy. Notably, 3 patients had active infection and 10 had mildly elevated CRP (<3x ULN). Full comparative data and scenarios are presented in Table I and II respectively.

Table I. Comparison of clinical and demographic characteristics between elevated and normal CRP groups in spondyloarthritis patients.

Characteristic (statistic)	Elevated CRP (n=97)	Normal CRP (n=40)	p value
Demographics			
Age, years (Median (IQR))	33.0 (28.0–45.0)	37.0 (28.8–50.2)	0.479
Male sex (n (%))	70/97 (72.2%)	30/40 (75.0%)	0.898
Body mass index, kg/m² (Median (IQR))	25.8 (22.3–30.5)	25.5 (23.9–28.2)	0.731
Disease duration, years (Median (IQR))	7.5 (4.0–15.0)	7.0 (4.0–12.5)	0.791
Disease activity			
BASDAI (Median (IQR))	4.2 (2.6–6.0)	1.5 (1.0–2.3)	**<0.001***
ASDAS-CRP (Median (IQR))	2.9 (2.1–3.8)	1.3 (1.0–1.6)	**<0.001***
BASDAI Active (n (%))	56/97 (57.7%)	8/40 (20.0%)	**<0.001***
BASDAI Inactive (n (%))	41/97 (42.3%)	32/40 (80.0%)	**<0.001***
ASDAS-CRP Active (n (%))	82/97 (84.5%)	9/40 (22.5%)	**<0.001***
ASDAS-CRP Inactive (n (%))	15/97 (15.5%)	31/40 (77.5%)	**<0.001***
C-reactive protein, mg/L (Median (IQR))	9.0 (5.3–19.3)	1.5 (1.0–2.8)	**<0.001***
Physical function			
BASFI (Median (IQR))	3.2 (1.4–5.3)	1.1 (0.5–1.6)	**<0.001***
Quality of Life			
ASQoL (Median (IQR))	7.0 (4.0–12.0)	3.0 (1.0–5.2)	**<0.001***
Imaging and genetics			
X-ray sacroiliac joints positive (of tested) (n (%))	63/75 (84.0%)	25/33 (75.8%)	0.455
MRI sacroiliac joints positive (of tested) (n (%))	48/58 (82.8%)	23/25 (92.0%)	0.332
HLA-B27 positive (of tested) (n (%))	43/53 (81.1%)	18/25 (72.0%)	0.537
Extra-articular manifestations			
Peripheral arthritis (n (%))	4/97 (4.1%)	0/40 (0.0%)	0.321
Enthesitis (n (%))	8/97 (8.2%)	7/40 (17.5%)	0.202
Uveitis (n (%))	14/97 (14.4%)	6/40 (15.0%)	1.000
Dactylitis (n (%))	4/97 (4.1%)	0/40 (0.0%)	0.321
Psoriasis (n (%))	10/97 (10.3%)	4/40 (10.0%)	1.000
Inflammatory bowel disease (n (%))	7/97 (7.2%)	0/40 (0.0%)	0.106
Comorbidities			
Hypertension (n (%))	15/97 (15.5%)	11/40 (27.5%)	0.163
Diabetes mellitus (n (%))	2/97 (2.1%)	7/40 (17.5%)	**0.003***
Tobacco/substance use (n (%))	14/97 (14.4%)	3/40 (7.5%)	0.394

* $p<0.05$. Continuous variables: independent t-test (normally distributed) or Mann-Whitney U-test (non-normally distributed; Shapiro-Wilk applied). Categorical variables: Chi-square or Fisher's exact test (when expected cell count <5). For imaging and HLA-B27, only patients who underwent the respective investigation are included in the denominator. CRP group classification was based on whether CRP was elevated at any point during the 12-week period.

Table II. Rheumatologist treatment decisions in discordant disease activity scenarios in spondyloarthritis.

Scenario / Rheumatologist Action	n	%
Scenario 1 — BASDAI Inactive + ASDAS-CRP Active (n = 25)		
BASDAI <4 (clinically inactive) but ASDAS-CRP ≥2.1 (objectively active). CRP elevated in all patients.		
No change	12	48.0%
Dose reduced	7	28.0%
Dose increased of same treatment	3	12.0%
Escalated treatment	3	12.0%
Rheumatologist aligned with BASDAI score (maintained or reduced treatment)	**19**	**76.0%**
Scenario 2 — Both Scores Inactive + CRP Elevated (n = 24)		
BASDAI <4 and ASDAS-CRP <2.1 (both inactive); CRP elevated, suggesting subclinical or residual inflammation.		
No change	17	70.8%
Dose reduced	6	25.0%
Escalated treatment	1	4.2%
Rheumatologist aligned with both scores (maintained or reduced treatment)	**23**	**95.8%**

BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; ASDAS-CRP: Ankylosing Spondylitis Disease Activity Score using CRP; CRP: C-reactive protein. Active disease defined as BASDAI ≥ 4 and/or ASDAS-CRP ≥ 2.1; inactive as BASDAI < 4 and ASDAS-CRP < 2.1.
Rheumatologist action: No change: same treatment maintained; Dose reduced: within-drug de-escalation; Dose increased: within-drug escalation; Escalated/switched: step-up to or change of biologic/targeted therapy. Rheumatologist aligned: sum of No change and Dose reduced, reflecting conservative management consistent with score-guided assessment.

Conclusion. Elevated CRP in SpA identifies a clinically distinct subgroup with substantially greater disease burden. In discordant scenarios, rheumatologists favoured patient-reported symptoms over biochemical markers, reflecting that CRP elevation may arise from non-SpA causes. These findings are among the first Indian data on CRP-stratified SpA phenotypes underscore the importance of history-taking and clinical examination when interpreting CRP in SpA.

P46

PRESENCE AND IMPACT OF FIBROMYALGIA ON DISEASE ACTIVITY, FUNCTIONING, AND QUALITY OF LIFE IN SPONDYLOARTHRITIS PATIENTS ALONG WITH VALIDATION OF FIBROMYALGIA RAPID SCREENING TOOL (FIRST) AGAINST ACR FIBROMYALGIA DIAGNOSTIC CRITERIA OF 2016

Shah U.[1], Meghnathi B.[1,2,3], Pandya S.[1,2], Sumra A.[2], Shah S.[2]
[1]Smt. NHL Municipal Medical College, Ahmedabad; [2]RheumaCARE, Ahmedabad; [3]Marengo CIMS Hospital, Ahmedabad, India

Introduction. Spondyloarthritis (SpA) frequently coexists with fibromyalgia (FM), complicating disease activity assessment; FM prevalence in SpA reaches 4-21%, yet Indian data is virtually absent. We aimed to determine FM prevalence in SpA, evaluate its impact on disease activity, functioning, and quality of life, and validate the Fibromyalgia Rapid Screening Tool (FiRST) against 2016 ACR FM diagnostic criteria.
Methods. Prospective cross-sectional study was conducted at a single tertiary rheumatology centre in India. Adult SpA patients (ASAS 2009 criteria) were enrolled; FM was diagnosed using 2016 ACR criteria. Sample size of 151 (5% margin of error) was calculated based on FM prevalence of 11.1% in axSpA as per a 2020-SLR. Groups were compared using BASDAI, ASDAS-CRP, BASFI, and ASQoL. FiRST and rheumatologist diagnosis were evaluated against ACR criteria as gold standard.
Results. Of 154 screened, 3 were excluded (psychological comorbidities), yielding 151 patients. Fourteen (9.3%) fulfilled ACR FM criteria. The SpA+FM group was predominantly female (64.3% vs. 27.0%, p=0.011) with significantly higher BASDAI (5.40 vs. 3.10, p=0.001), BASFI (4.40 vs. 2.00, p=0.026), ASDAS-CRP (3.70 vs. 2.20, p=0.003), and ASQoL (12.00 vs. 6.00, p=0.003). FiRST and rheumatologist diagnosis achieved sensitivities of 85.7% and 100%, specificities of 88.3% and 94.2%, PPVs of 42.9% and 63.6%, NPVs of 98.4% and 100%, and accuracies of 88.1% and 94.7%, respectively (κ=0.511 vs. 0.749). Rest comparative data are presented in Table I and II.
Conclusion. FM was present as a comorbidity in approximately 1 in 10 SpA patients with significantly worsened disease activity, functioning, and quality of life. To our knowledge, this is the first Indian data on FM prevalence in SpA. Both rheumatologist diagnosis and FiRST performed well against ACR criteria, with FiRST proving a valid, practical screening tool. Routine FM screening in SpA is essential especially in peripheral involvement and to prevent unnecessary treatment escalation.

Table I. Baseline characteristics, clinical features and outcomes of the study cohort (N=151).

Variable	Full cohort (N=151)	SpA + FM (n=14)	SpA without FM (n=137)	p-value
FM positive (2016 ACR FM criteria): 14 (9.3%) | FM negative: 137 (90.7%)				
Demographics				
Age, years	35.0 (28–47)	41.5 (38–47)	35.0 (28–47)	0.215
Female sex	46 (30.5%)	9 (64.3%)	37 (27.0%)	**0.011***
BMI, kg/m²	25.8 (23–29)	27.3 (25–28)	25.8 (23–29)	0.606
Disease duration, years	7.0 (4–14)	6.5 (3–10)	7.0 (4–14)	0.440
CRP, mg/L	6.5 (2.5–17.3)	15.7 (7–31)	6.1 (2–16)	0.051
SpA classification				
HLA-B27 positive [a]	67/87 (77.0%)	6/9 (66.7%)	61/78 (78.2%)	0.424
X-ray SI joints positive [b]	96/120 (80.0%)	8/12 (66.7%)	88/108 (81.5%)	0.255
MRI SI joints positive [c]	78/92 (84.8%)	7/9 (77.8%)	71/83 (85.5%)	0.622
Axial SpA [d]	70 (46.4%)	2 (14.3%)	68 (49.6%)	**0.012***
Axial + peripheral SpA [d]	81 (53.6%)	12 (85.7%)	69 (50.4%)	**0.028***
Extra-articular features				
Enthesitis	19 (12.6%)	4 (28.6%)	15 (10.9%)	0.079
Uveitis	24 (15.9%)	4 (28.6%)	20 (14.6%)	0.240
Dactylitis	6 (4.0%)	2 (14.3%)	4 (2.9%)	0.097
Psoriasis	14 (9.3%)	0 (0.0%)	14 (10.2%)	0.365
Crohn's / Colitis	7 (4.6%)	0 (0.0%)	7 (5.1%)	1.000
Comorbidities				
Hypertension	27 (17.9%)	1 (7.1%)	26 (19.0%)	0.466
Diabetes mellitus	9 (6.0%)	0 (0.0%)	9 (6.6%)	1.000
Thyroid disease [e]	10 (6.6%)	0 (0.0%)	10 (7.3%)	0.602
Addiction / tobacco use	28 (18.5%)	0 (0.0%)	28 (20.4%)	0.074
Disease activity, functioning and Quality of life				
FiRST score	2.0 (1–4)	5.0 (5.0–5.0)	2.0 (1–3)	**<0.001***
BASDAI	3.4 (1.8–5.5)	5.40 (4.70–6.20)	3.10 (1.60–5.40)	**0.001***
BASFI	2.1 (1.2–4.8)	4.40 (3.58–5.38)	2.00 (1.10–4.70)	**0.026***
ASDAS-CRP	2.2 (1.6–3.6)	3.70 (2.58–4.30)	2.20 (1.40–3.41)	**0.003***
ASQoL	6.0 (3.0–12.0)	12.00 (7.50–14.75)	6.00 (2.00–11.00)	**0.003***

Continuous variables: median (IQR); categorical: n (%). SpA+FM vs. SpA without FM compared by Mann-Whitney-U (continuous) and Fisher's exact test (categorical). IQR: interquartile range. *p<0.05. FM: fibromyalgia. All continuous variables non-normally distributed (Shapiro-Wilk p<0.05); median (IQR) reported; Mann-Whitney U-test used for between-group comparisons, confirmed by permutation test (10,000 iterations). Fisher's exact test used for all categorical variables.
[a]HLA-B27 tested in 87/151 patients; percentage of those tested. [b]X-ray SI joints performed in 120/151 patients; percentage of those tested. [c]MRI SI joints performed in 92/151 patients; percentage of those tested. [d]No patient had purely peripheral SpA; all were either axial or combined axial+peripheral. [e]All thyroid disease cases were hypothyroidism.

Table II. Diagnostic accuracy of FiRST and rheumatologist diagnosis *vs*. 2016 ACR FM criteria (gold standard). 2016 ACR FM criteria used as gold standard. FM prevalence in cohort = 9.3% (14/151).

FiRST (cut off ≥ 5) vs. ACR 2016 FiRST+ = 28 | ACR FM+ = 14 TP = 12 · FP = 16 · FN = 2 · TN = 121		**Rheumatologist diagnosis vs. ACR 2016** Rheum FM+ = 22 | ACR FM+ = 14 TP = 14 · FP = 8 · FN = 0 · TN = 129	
Metric	**Value**	**Metric**	**Value**
Sensitivity	85.7%	Sensitivity	100.0%
Specificity	88.3%	Specificity	94.2%
PPV	42.9%	PPV	63.6%
NPV	98.4%	NPV	100.0%
LR+	7.34	LR+	17.12
LR−	0.162	LR−	0.000
Overall accuracy	88.1%	Overall accuracy	94.7%
Cohen's κ	0.511 (moderate)	Cohen's κ	0.749 (substantial)

TP: true positive; FP: false positive; FN: false negative; TN: true negative. LR+: positive likelihood ratio; LR−: negative likelihood ratio. PPV: positive predictive value; NPV: negative predictive value. κ: Cohen's Kappa (0.41–0.60: moderate; 0.61–0.80: substantial). FiRST: Fibromyalgia Rapid Screening Tool (score 0–6; positive ≥ 5). The rheumatologist over-diagnosed FM in 8 patients not fulfilling 2016 ACR criteria (FP: 8). 95% CI (Clopper-Pearson, bootstrap 5,000 iterations) FiRST: sensitivity 57.2–98.2%, specificity 81.7–93.2%, PPV 24.5–62.8%, NPV 94.2–99.8%; Rheumatologist: sensitivity 76.8–100.0%, specificity 88.8–97.4%, PPV 40.7–82.8%, NPV 97.2–100.0%.

P47

SYMPTOM PHENOTYPE, MISDIAGNOSIS PATHWAYS, AND STAGE-SPECIFIC DIAGNOSTIC DELAY IN A REAL-WORLD SPONDYLOARTHRITIS COHORT

Mohith Kumar Reddy P., Veeravalli S.C.M.
Dept. of Rheumatology, KIMS hospitals, Secunderabad, India

Introduction. Diagnostic delay in spondyloarthritis (SpA) is usually attributed to delayed healthcare seeking. We evaluated whether delay was instead driven by symptom phenotype, first-contact specialty and initial diagnostic labelling.
Methods. We studied 274 patients with SpA, classified as axial SpA, psoriatic arthritis (PsA) or enthesitis-predominant SpA. Diagnostic intervals were T1, symptom onset to first healthcare contact; T2, first healthcare to rheumatology contact; and T3, rheumatology contact to definitive diagnosis. Initial labels were classified as SpA/PsA or misdiagnosis. Multivariable logistic regression assessed determinants of total delay >2 years, T2 >180 days and T3 >90 days.
Results. Final phenotypes were axial SpA in 208, PsA in 38 and enthesitis-predominant SpA in 24 patients. Females constituted 48.5%; HLA-B27 positivity was 84/274 (30.7%). Misdiagnosis occurred in 201/274 (73.4%), most often as undifferentiated arthritis/arthralgia. Median total delay was 760 days (IQR 1782), with median T1 30 days, T2 730 days and T3 0 days. Axial symptoms were the commonest first presentation (121/274, 44.2%), followed by peripheral arthritis (95/274, 34.7%) and enthesitis-first disease (27/274, 9.9%). The overall diagnostic pathway is shown in Figure 1. Enthesitis-first presentations had the highest symptom-group misdiagnosis rate (85.2%) and median total delay of 1126 days. Enthesitis-predominant SpA had the highest phenotype-level misdiagnosis burden (20/24, 83.3%), longest median total delay (1633 days) and persistent T3 delay (median 120 days). Total delay >2 years was associated with mechanical/degenerative/OA labels, undifferentiated arthritis/arthralgia, RA, other/non-specific diagnoses and HLA-B27 negativity. Stage-specific determinants of delay are shown in Figure 2.
Conclusion. Diagnostic delay was driven mainly by missed recognition after early healthcare contact, rather than late presentation. Non-SpA labels delayed referral, while enthesitis-predominant SpA remained vulnerable even after rheumatology contact.

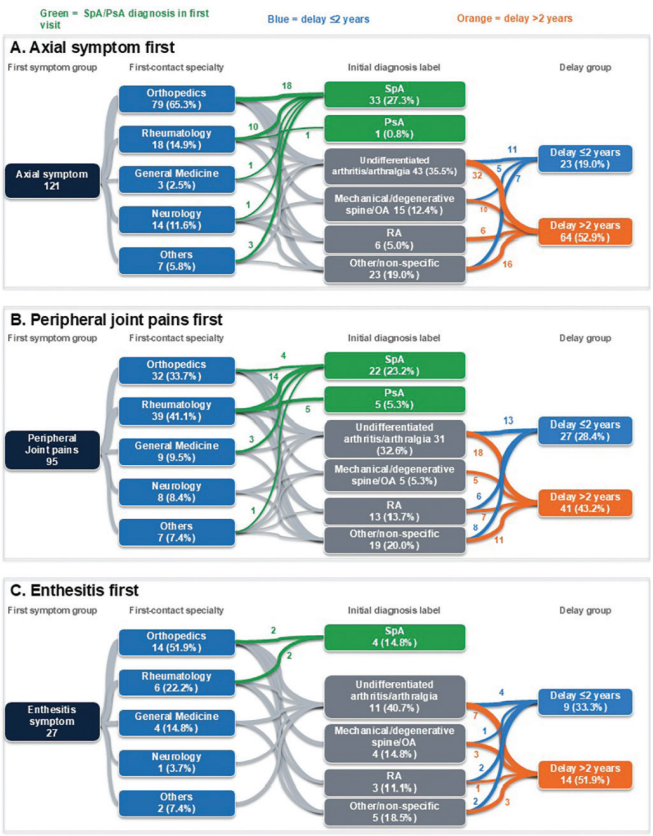


Fig. 1. Symptom-entry diagnostic pathways in SpA.
1. Undifferentiated arthritis/arthralgia includes - arthalgia, arthritis, polyarthralgia, joint pains, and earty arthritis.
2. Other/non-specific/alternative diagnosis includes - bone TB, gout, fibromyalgia, procedure-based arthritis, post-trauma joint pains, and no specific diagnosis given.
3. Others departments includes - Ayurvedic, Homeopathy, Dermatology, Sports medicine, Neurosurgery, Ophthalmology.

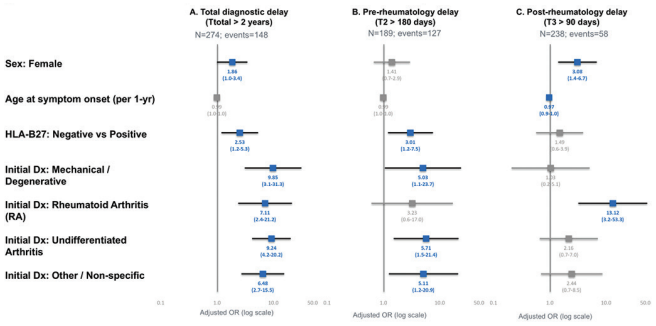


Fig. 2. Determinants of stage-specific diagnostic delay.
Adjusted odds ratios with 95% confidence intervals; vertical reference line indicates OR = 1. Blue markers indicate $p<0.05$.
OR: odds ratio; CI: confidence interval; RA: rheumatoid arthritis; SpA: spondyloarthritis. Initial diagnosis categories are compared against initial SpA/PsA diagnosis.

P48

IMPACT OF DIAGNOSTIC DELAY ON LONG-TERM RADIOGRAPHIC STRUCTURAL OUTCOMES IN TNF INHIBITOR-TREATED RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS

Nam B.[1], Kim Y.[1], Lee S.[2], Shin J.H.[1], Kim T. [3], Kim T.H.[1]
[1]Rheumatology, Hanyang University Hospital for Rheumatic Diseases; [2]Radiology, Hanyang University Hospital for Rheumatic Diseases; [3]Rheumatology, Chonnam National University Medical School and Hospital, Korea

Background/Objective. Diagnostic delay remains common in axial spondyloarthritis (axSpA) and has been associated with increased structural damage. Although tumor necrosis factor inhibitors (TNFi) effectively suppress inflammation and improve disease activity, structural progression may continue in a subset of patients. This study aimed to examine whether diagnostic delay is associated with long-term radiographic structural outcomes in patients with radiographic axial spondyloarthritis (r-axSpA) treated with TNFi.
Methods. This observational study retrospectively analyzed data from the Korean Spondyloarthropathy Registry, including patients with r-axSpA who received TNFi therapy. Diagnostic delay was defined as the interval between symptom onset and diagnosis and categorized as <2 years, 2–5 years, or >5 years. Long-term radiographic structural damage was assessed using the modified Stoke Ankylosing Spondylitis Spine Score (mSASSS) at follow-up. Univariable and multivariable linear regression analyses were performed to evaluate the association between diagnostic delay and radiographic structural outcomes, adjusting for relevant covariates.
Results. A total of 277 TNFi-treated patients with r-axSpA were included (89.9% male; median age at diagnosis, 26.0 years). The duration from diagnosis to the final mSASSS assessment was a median of 14.3 (10.9–18.7) years. Diagnostic delay was <2 years in 75 patients, 2–5 years in 70 patients, and >5 years in 132 patients, with no meaningful differences in age at diagnosis, follow-up duration, or time to TNFi initiation across groups. In multivariable analyses, a diagnostic delay of >5 years remained associated with worse long-term radiographic structural outcomes compared with a delay of <2 years (adjusted β 9.49, 95% CI 4.34–14.64, $p<0.001$). Male sex, absence of peripheral arthritis, and presence of uveitis were also associated with long-term radiographic structural damage.
Conclusion. Despite TNFi therapy, prolonged diagnostic delay was associated with worse long-term radiographic structural outcomes in r-axSpA. These findings underscore the importance of early diagnosis to limit long-term structural damage, even in the era of biologic treatment.

P49

SINGLE CELL SEQUENCING ANALYSIS OF TUMOR NECROSIS FACTOR INHIBITOR DRUG RESPONSE SUGGESTS ENRICHMENT OF PRO-INFLAMMATORY PATHWAY IN NON-RESPONDERS AND AMINO ACID METABOLIC PATHWAYS IN RESPONDERS

Talukdar A., Nguyen P., Ibeke N., Machhar R., Aparnathi M., Keshavarazi S., Srinath A., Nakamura A., Inman R., Jurisica I., Rahman P., Haroon N.
Division of Rheumatology, Toronto Western Hospital, University Health Network, Toronto, ON, Canada

Introduction/ Objective. Ankylosing Spondylitis (AS) is an inflammatory arthritis affecting the spine and peripheral joints. Although tumor necrosis factor inhibitors (TNFi) substantially improve symptoms, 40% fail to respond. Predictors of treatment response could help make well-informed choices about TNFi treatment, elucidate mechanism of resistance and explore potential alternative therapeutic targets.
Materials and methods. PBMCs derived from 7 biologic naive AS patients (4 responders, 3 non-responders to TNFi treatment) were profiled using single cell RNA sequencing technology. Statistical analysis was carried out using Seurat R package (v5.2.0). The results were then validated using Nanostring nCounter gene expression assay. Serum cytokines levels were validated using ELISA. Protein expressions of CXCR4 and CD74 were determined using flow cytometry.
Results. Non-responders had a higher number of B cells, dendritic cells, and monocytes but a lower T cells number. 5020 differentially expressed genes were identified. CXCR4 and CD74 were found to be upregulated in the mono-

cytes of non-responders. Pathway enrichment analysis revealed that regulation of osteoclast function, innate immune response, and regulation of T cell activation pathways are among the most enriched pathways in responders. Interferon regulated genes (IRGs) expression and MHC class II regulation pathways were upregulated in non-responders. Upregulation of CD74 in non-responders suggests that monocytes in these patients are activated before the start of treatment. This has been further confirmed by assessing inflammatory cytokines levels in the serum of patients before and after TNFi treatment. We observed that after TNFi treatment, there is a decrease in the levels of CXCL10, CCL4, IL-1b, TNFa and MIF in responders, but not in non-responders.

Conclusion. Enhanced expression of inflammatory cytokines like MIF, TNF, IL1b and chemokines like CXCL8, CXCL10 and their receptors (CXCR4 and CD74) in non-responders may be the crucial factors responsible for modulating the immune system towards TNFi treatment response in AS patients.

P50

DIFFICULT-TO-MANAGE PSORIATIC ARTHRITIS: PREVALENCE, PHENOTYPE, AND CLINICAL BURDEN IN REAL-WORLD PRACTICE: INSIGHTS FROM THE SCQM REGISTRY

Elsehrawy G.G.[1,2], Courvoisier D.S.[1], Brülhart-Bletsas L.[3], Dan D.[4], Lazarou I.[5], Micheroli R.[6], Ciurea A.[6], Scholz G.A.[7], Möller B.[7], Nissen M.J.[1]

[1]Dept. of Rheumatology, Geneva University Hospital, University of Geneva, Geneva, Switzerland; [2]Dept. of Physical Medicine, Rheumatology and Rehabilitation, faculty of medicine, Suez Canal University, Ismailia, Egypt; [3]Dept. of Rheumatology, Neuchâtel Hospital, La Chaux-de-Fonds; [4]Dept. of Rheumatology, University Hospital of Lausanne, University of Lausanne, Lausanne; [5]Dept. of Rheumatology, La Tour Hospital, Geneva; [6]Dept. of Rheumatology, University Hospital of Zurich, University of Zurich, Zurich; [7]Dept. of Rheumatology and Immunology, Inselspital, Bern University Hospital, University of Bern, Switzerland

Objective. To apply consensus-based, difficult-to-treat definitions of psoriatic arthritis (PsA) and to investigate the demographics and clinical phenotypes of difficult-to-manage (D2M)/complex-to-manage (C2M) and treatment-refractory (TR) PsA.

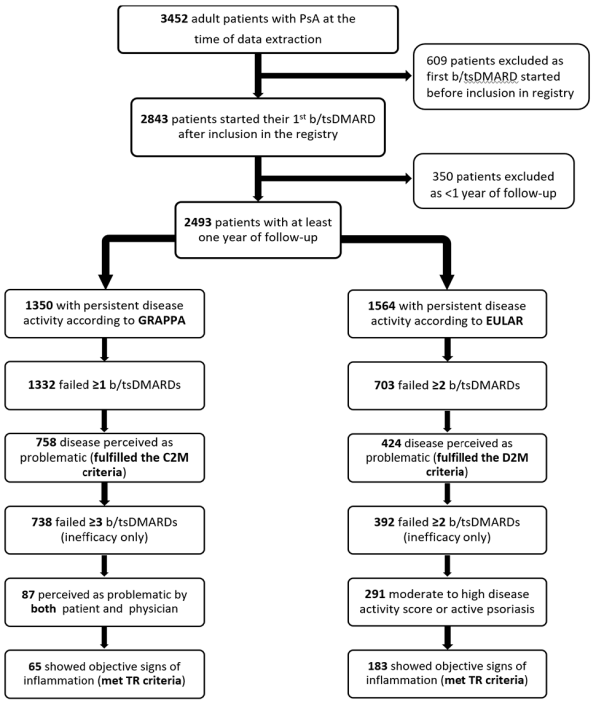


Fig. 1. Flow chart of study subject selection and criteria fulfilment according to GRAPPA and EULAR definitions.
b/tsDMARDs: biological/targeted synthetic disease-modifying anti-rheumatic drug; PsA: psoriatic arthritis; D2M: difficult-to-manage; C2M: complex-to-manage; GRAPPA: Group for Research and Assessment of Psoriasis and Psoriatic Arthritis; EULAR: European Alliance of Associations for Rheumatology; TR: treatment refractory.

Table I. Baseline demographic and disease characteristics comparison between C2M/D2M and non-C2M/non-D2M according to the GRAPPA and EULAR criteria.

Variables - GRAPPA	Level	nC2M, n=1733	C2M, n=758	p-value
Age		51.10 (12.61)	52.31 (12.22)	**0.026**
Gender n (%)	Female	825 (47.6)	455 (60.0)	**<0.001**
BMI		27.26 (5.18)	28.00 (5.70)	**0.004**
HLA-B27 n (%) positive		164 (18.3)	84 (21.3)	0.238
Smoking n (%)	Non-smoker	607 (45.7)	249 (38.2)	
	Former	451 (34.0)	237 (36.4)	**0.003**
	Current	270 (20.3)	165 (25.3)	
CRP		4.61 (7.75)	6.54 (10.78)	**<0.001**
ESR		10.96 (11.19)	13.74 (14.07)	**<0.001**
Skin involvement n (%)	None/almost none	888 (64.4)	339 (48.6)	
	Minor	388 (28.2)	204 (29.2)	**<0.001**
	moderate/severe	102 (7.4)	155 (22.2)	
Spinal involvement n (%)		597 (40.6)	344 (53.5)	**<0.001**
Enthesitis n (%)		862 (58.0)	443 (70.3)	**<0.001**
DAS-28 CRP		2.08 (0.82)	2.83 (1.16)	**<0.001**
DAPSA (median (IQR))		9.20 [5.0, 15.00]	23.00 [14.0, 32.0]	**<0.001**
DLQI (median (IQR))		1.00 [0.0, 3.00]	3.00 [1.00, 8.00]	**<0.001**
BASDAI		2.27 (1.78)	5.14 (2.31)	**<0.001**
EQ-5D		0.80 (0.16)	0.62 (0.21)	**<0.001**
HAQ		0.36 (0.47)	0.87 (0.65)	**<0.001**
NSAIDs n (%)		572 (33.0)	361 (47.6)	**<0.001**
Opiates n (%)		35 (2.0)	59 (7.8)	**<0.001**
Variables - EULAR	**Level**	**nD2M, n=2069**	**D2M, n=424**	**p-value**
Age		51.43 (12.62)	51.64 (11.91)	0.752
Gender n (%)	Female	1015 (49.1)	265 (62.5)	**<0.001**
BMI		27.32 (5.20)	28.30 (6.02)	**0.002**
Smoking n (%)	Non-smoker	717 (44.3)	140 (38.5)	**0.044**
	Former	560 (34.6)	128 (35.2)	
	Current	340 (21.0)	96 (26.4)	
CRP		4.96 (8.02)	6.44 (11.95)	**0.002**
ESR		11.34 (11.67)	14.13 (14.40)	**<0.001**
Skin involvement n (%)	None/almost none	1030 (60.9)	198 (51.2)	**<0.001**
	Minor	496 (29.3)	97 (25.1)	
	Moderate/severe	165 (9.8)	92 (23.8)	
Spinal involvement n (%)		754 (42.9)	188 (52.5)	**0.001**
Enthesitis n (%)		1063 (60.2)	243 (69.4)	**0.001**
DAS-28 CRP		2.20 (0.91)	2.88 (1.19)	**<0.001**
DAPSA (median (IQR))		11.6 [6.0, 20.0]	24.0[14.8,33.2]	**<0.001**
BASDAI		3.20 (2.39)	4.84 (2.43)	**0.001**
EQ-5D		0.77 (0.19)	0.62 (0.21)	**<0.001**
HAQ		0.45 (0.55)	0.89 (0.64)	**<0.001**

All variables are expressed as mean (± standard deviation) unless otherwise indicated.
BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BMI: body mass index; C2M: Complex to Manage; CRP: C-reactive protein; DAPSA: Disease Activity index for Psoriatic Arthritis; DAS: Disease Activity Score; DLQI: Dermatology Life Quality Index; D2M: difficult to manage, EQ-5D: EuroQol 5-Dimension Questionnaire; ESR: erythrocyte sedimentation rate; HAQ: Health Assessment Questionnaire; HLA-B27: human leukocyte antigen B27; IQR: interquartile range; n: number; NSAIDs: nonsteroidal anti-inflammatory drugs; PsA: psoriatic arthritis.

Methods. Data were derived from the prospective, multicentre, longitudinal Swiss registry. Patients were eligible if they initiated their advanced therapy after inclusion in the registry and had ≥12 months of follow-up. Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) and European Alliance of Associations for Rheumatology (EULAR) definitions were applied to identify cases of C2M/D2M and TR (Fig. 1).

Results. From 2493 patients, 30.4% / 2.6% fulfilled the GRAPPA criteria for C2M/TR disease, while 17% / 7.3% met the D2M/TR EULAR criteria, respectively. At baseline, C2M/D2M patients were more frequently women (p<0.001), smokers, had higher body mass index (BMI), and had more comorbidities (fibromyalgia, depression, anxiety, and osteoarthritis, p<0.001). They also demonstrated higher levels of inflammatory markers, a higher prevalence of enthesitis and axial involvement, more severe psoriasis, and worse disease activity scores than non-C2M/non-D2M patients (p≤0.001). Use of analgesics and opiates was higher across C2M/D2M patients (p<0.001) (Table I). However, there were no significant differences regarding the prevalence of HLA-B27, dactylitis, nail disease, or other extra-musculoskeletal manifestations. Across both criteria, the TR subgroup showed higher counts of swollen joints and tender entheses. When applying the EULAR criteria, TR patients showed higher levels of inflammatory markers, quality of life, disability, and disease-activity indices, compared with non-TR D2M patients (p<0.05).

Conclusion. This exploratory analysis, using a large national database of patients with PsA, demonstrated that the prevalence of C2M/D2M PsA in a real-world setting ranged from 17% to 30%, depending on the criteria employed. The consistent association with obesity, smoking, and comorbidities highlights the need for a more holistic management approach for PsA, beyond pharmacological treatment alone.

P51

AGREEMENT BETWEEN ASAS AND REVISED ASAS-SPARTAN CLASSIFICATION CRITERIA IN AXIAL SPONDYLOARTHRITIS

Patela M.[1], Rua C.[1], Silva C.[1], Fernandes M.A.[1], Vieira R.[1,2], Meirinhos T.[1], Pinto A.S.[1]
[1]Dept. of Rheumatology, Unidade Local de Saúde Gaia e Espinho, Gaia; [2]Dept. of Medical Sciences, University of Aveiro, Aveiro, Portugal

Introduction. Classification of axial spondyloarthritis (axSpA) relies on different criteria sets, including the ASAS criteria and the recently revised ASAS-SPARTAN criteria. This study aimed to assess the agreement between these criteria.
Methods. Patients diagnosed with axSpa followed at our center were cross-sectionally evaluated. Based on both classification criteria agreement, patients were stratified into four groups; clinical, laboratory and imaging features were compared. Agreement was assessed using Cohen's kappa. A multivariable logistic regression model including statistically significant variables across concordant and discordant groups was performed.
Results. Among 232 patients, 167 (71.9%) were classified concordantly and 65 (28.1%) discordantly. Agreement between ASAS and revised ASAS–SPARTAN criteria was slight (κ=0.192). Revised ASAS-SPARTAN sensitivity was 68.5%, lower than in the CLASSIC-SPARTAN cohort (79.5%), while ASAS sensitivity was 82.3%, like the original ASAS criteria (83%). Significant differences across the four classification groups (Table I) were observed for inflammatory back pain, radiographic sacroiliitis, HLA-B27 positivity, and positive MRISJ. In the concordance analysis, HLA-B27 positivity and inflammatory back pain were the strongest factors associated with agreement between the two criteria sets. In multivariable concordance analysis, HLA-B27 positivity showed the highest odds of agreement (OR 14.12, 95% CI 5.20–38.33), followed by inflammatory back pain (OR 8.49, 95% CI 2.88–25.04) and uveitis (OR 5.28, 95% CI 1.08–25.96) (Table II).
Conclusion. Agreement between the ASAS and revised ASAS-SPARTAN criteria was slight. Although the four classification groups differed in HLA-B27 status, inflammatory back pain, radiographic sacroiliitis, and positive MRISJ, concordant classification was independently associated with HLA-B27 positivity, inflammatory back pain, and uveitis. These findings suggest that these features are the main predictors of agreement between the two criteria sets, whereas imaging findings help distinguish classification patterns.

Table I. Clinical characteristics according to ASAS/SPARTAN classification groups.

Variable	ASAS+/SPARTAN + (n=153)	ASAS+/SPARTAN − (n=59)	ASAS−/SPARTAN + (n=6)	ASAS−/SPARTAN− (n=14)	p-value
Male gender	96/153 (62.7%) 159	38/59 (64.4%)	4/6 (66.7%)	4 /14 (28.6%)	0.080
Inflammatory back pain	118/153 (77.1%)	20/59 (33.9%)	4 /6 (66.7%)	6/14 (42.9%)	< 0.001
HLA-B27 Positive	106/142 (74.6%)	4 /48 (8.3%)	3/5 (60.0%)	3/12 (25.0%)	< 0.001
Radiographic Sacroiliitis	129 /152 (84.9%)	44 /59 (74.6%)	6/6 (100.0%)	3/14 (21.4%)	< 0.001
MRISJ positive	60/68 (88.2%)	21 /24 (87.5%)	2/2 (100.0%)	3/6 (50.0%)	0.034
Positive CRP	19/84 (22.6%)	10/29 (34.6%)	1/ 5 (20.0%)	2/7 (28.6%)	0.635
Uveitis (history)	26/152 (17.1%)	6/59 (10.2%)	2/6 (33.3%)	3/14 (21.4%)	0.351
Enthesitis (history)	20 /153 (13.1%)	8/59 (13.6%)	1/6 (16.7%)	1/14 (7.1%)	0.916
Dactylitis (history)	6 /152 (3.9%)	1/59 (1.7%)	0/6 (0.0%)	0/14 (0.0%)	0.701
Psoriasis (history)	10 (6.5%)	1 (1.7%)	0 (0.0%)	1 (5.2%)	0.482
Peripheral arthritis (history or current)	32/153 (20.9%)	12/59 (20.3%)	2/5 (40.0%)	4/14 (28.9%)	0.747
Inflammatory bowel disease	16/152 (10.5%)	5/58 (86.2%)	0/6 (0%)	4/14 (28.6%)	0.134

Table II. Factors associated with agreement between ASAS and ASAS-SPARTAN revised criteria.

Variable	Concordant (n=167)	Discordant (n=65)	p-value	OR (95% CI) of concordance with 95%
Male gender	100 (59.9%)	42 (64.6%)	0.518	
HLA-B27 Positive	109 /149 (73.2%)	7 /58 (12.1%)	**< 0.001**	14.119 (5.200-38.334)
Inflammatory back pain	118/161 (73.3%)	30/71 (42.3%)	**< 0.001**	8.489(2.878-25.040)
Uveitis	29 /160 (18.1%)	8 /71 (11.3%)	**0.041**	5.282 (1.075-25.963)
Psoriasis	11/160 (6.9%)	1 /70 (1.4%)	0.087	-
Radiographic Sacroiliitis	129/160 (70.9%)	31/71 (63.3%)	0.305	-
MRISJ positive	59/70 (86.6%)	27(30 (84.4%)	0.748	-
Positive CRP	21 /88 (23.9%)	11 /37 (29.7%)	0.493	-
Enthesitis	21/161 (12.6%)	9 /71 (13.8%)	0.803	-
Dactylitis	6 /160 (3.8%)	1 /71 (1.5%)	0.338	-
Peripheral arthritis (history or current)	35/97 (36.1%)	14/43 (32.6%)	0.848	-
Inflammatory bowel disease	20/160 (12.5%)	5/70 (7.1%)	0.230	-

P52

REAL-WORLD PERFORMANCE OF THE REVISED ASAS-SPARTAN CRITERIA IN PATIENTS WITH BACK PAIN EVALUATED IN A RHEUMATOLOGY UNIT

Patela M.[1], Rua C.[1], Silva C.[1], Fernandes M.A.[1], Vieira, R.[1,2], Meirinhos T.[1], Pinto A.S.[1]
[1]Dept. of Rheumatology, Unidade Local de Saúde Gaia e Espinho, Gaia; [2]Dept. of Medical Sciences, University of Aveiro, Aveiro, Portugal

Introduction. The revised ASAS-SPARTAN criteria were recently proposed to improve axial spondyloarthritis (axSp) classification, with higher specificity as a key objective. We aimed to assess their performance in a real-world cohort of patients referred to Rheumatology unit for back pain.
Methods. We retrospectively evaluated patients assigned to Rheumatology outpatient clinic for back pain (ICD-10 code) between 2022 and 2026. Patients who did not fulffill the entry criteria of chronic back pain for over 3 months with onset before age 45 years were excluded. The revised ASAS-SPARTAN criteria were applied. Performance measures were calculated using the rheumatologist's clinical diagnosis as the gold standard.
Results. Of 313 patients identified, 105 were excluded due to not fulfilling the entry criteria or because their diagnosis had not yet been clarified,

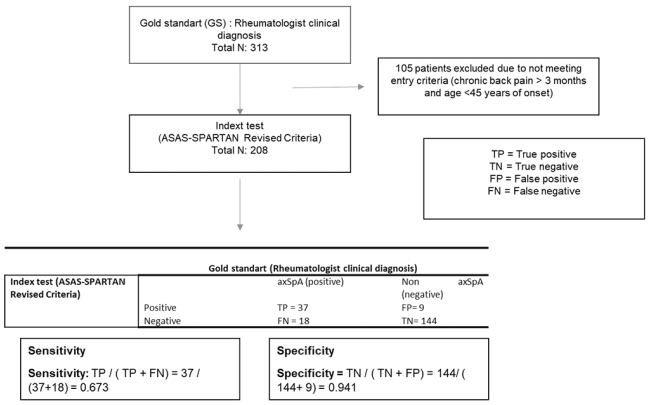


Fig. 1. Diagnostic performance measures of the revised ASAS-SPARTAN criteria.

Table I. Patient and disease characteristics according to revised ASAS-SPARTAN classification.

	ASAS-SPARTAN positive clinical diagnosed AxSpA	ASAS-SPARTAN negative clinical diagnosed AxSpA	ASAS-SPARTAN positive non-AxSpA	ASAS-SPARTAN negative non-AxSpA	p-value
Patients, n	37	18	9	144	
Rheumatologist-diagnosed axSpA, n (%)	37	18	-	-	
Inflammatory back pain, n/N (%)	36/37 (97.3%)	9/18 (50.0%)	9/9 (100.0%)	36/144 (25.0%)	**<0.001**
HLA-B27 positive, n/N (%)	20/34 (58.8%)	5/16 (31.2%)	0/8 (0.0%)	14/120 (11.7%)	**<0.001**
CRP > 0.5mg/dL n/N (%)	4/20 (20.0%)	2/14 (14.3%)	3/9 (33.3%)	9/139 (6.5%)	**<0.002**
MRI positivity, n/N (%)	27/30 (90.0%)	4/7 (57.1%)	8/8 (100.0%)	3/66 (4.5%)*	**<0.001**
Radiographic sacroiliitis, n/N (%)	19/36 (52.8%)	5/18 (27.8%)	1/9 (11.1%)	1/140 (0.7%)	**<0.001**
Uveitis, n/N (%)	6/37 (16.2%)	1/18 (5.6%)	0/9 (0.0%)	1/144 (0.7%)	**0.001**
Psoriasis, n/N (%)	4/37 (10.8%)	0/18 (0.0%)	2/9 (22.2%)	9/144 (6.2%)	0.054
IBD, n/N (%)	2/37 (5.4%)	2/18 (11.1%)	0/9 (0.0%)	10/144 (6.9%)	0.862
Peripheral arthritis, n/N (%)	3/37 (8.1%)	1/18 (5.6%)	0/9 (0.0%)	2/144 (1.4%)	0.111
Enthesitis, n/N (%)	1/37 (2.7%)	3/18 (16.7%)	0/9 (0.0%)	4/144 (2.8%)	0.083
Dactylitis	1/37 (2.7%)	2/18 (11.1%)	0/9 (0.0%)	0/144 (0.0%)	**0.012**

leaving 208 for analysis. Rheumatologist-diagnosed axSpa was present in 55 patients (26.4%). Sensitivity was 67.3%, specificity was 94.1%, positive predictive value was 80.4%, negative predictive value was 88.9%, and it showed an excellent accuracy of 87.0% (Fig. 1). Agreement between the revised criteria and clinical diagnosis of axSpA was substantial, with a Cohen's kappa of 0.648 (p<0.001). ASAS-SPARTAN-positive patients more frequently had inflammatory back pain, were HLA-B27 positive, MRI sacroilitis, radiographic sacroiliitis, and uveitis compared with ASAS-SPARTAN-negative patients (Table I).

Conclusion. Compared to the CLASSIC validation dataset, which reported 79.5% sensitivity and 90.4% specificity for the revised ASAS SPARTAN criteria, our real-world back pain referral cohort demonstrated lower sensitivity but higher specificity, while maintaining strong overall accuracy and substantial agreement with rheumatologist diagnosis.

P53

DIFFICULT-TO-MANAGE AXIAL SPONDYLOARTHRITIS: A TERTIARY CENTRE EXPERIENCE

Rua C.[1], Silva C.[1], Patela M.[1], Fernandes M.[1], Meirinhos T.[1,2], Vieira R.[1,3], Pinto A.S.[1]
[1]Rheumatology Dept., ULS Gaia Espinho, Vila Nova de Gaia; [2]Faculty of Medicine, University of Porto, Porto; [3]Dept. of Medical Sciences, University of Aveiro, Aveiro, Portugal

Introduction. In 2025, the Assessment of SpondyloArthritis International Society (ASAS) proposed a definition for difficult-to-manage (D2M) axial spondyloarthritis (axSpA), including treatment-refractory (TR). Data on D2M/TR-axSpA remain limited. This study aimed to assess the prevalence, clinical characteristics, and associated factors of D2M/TR-axSpA in a real-world cohort.

Methods. We conducted a retrospective single-centre study including adult axSpA patients treated with at least one biologic or targeted synthetic DMARD. D2M and TR axSpA were defined according to the ASAS 2025 criteria. Demographic, clinical, treatment, and disease activity data were collected retrospectively. Clinical factors associated with D2M were explored using univariate logistic regression.

Results. Among 336 patients with axSpA, 140 treated with ≥1 b/tsDMARD were included. Median age was 46.5 years, 60% were female, and median follow-up was 5.8 years. D2M-axSpA was identified in 12 patients (8.6%), of whom 50% also fulfilled criteria for TR-axSpA. Among D2M patients, the most frequent domain was insufficient disease control (61.4%), followed by problematic management (38.6%) and treatment failure (13.6%) (Fig. 1). Factors associated with D2M included absence of paid work, diagnostic delay, inflammatory bowel disease, fibromyalgia, glucocorticoid and NSAID use, exposure to IL-17 inhibitors or tsDMARDs, and higher ASDAS, BASDAI, BASFI, CRP, patient global and physician global assessment scores (Table I). Multivariable analysis was not performed due to the limited number of events.

Conclusion. D2M-axSpA affected nearly one in twelve b/tsDMARD-treated patients in our cohort. Like previous reports, D2M disease was associated with higher disease burden, poorer functional status, and comorbidities such as fibromyalgia and inflammatory bowel disease. The application of the ASAS D2M/TR-axSpA criteria in routine clinical practice proved challenging. Although limited by the small number of D2M cases and missing data for some domains, these findings support the clinical relevance of the ASAS D2M definition and highlight the need for further multicentre studies.

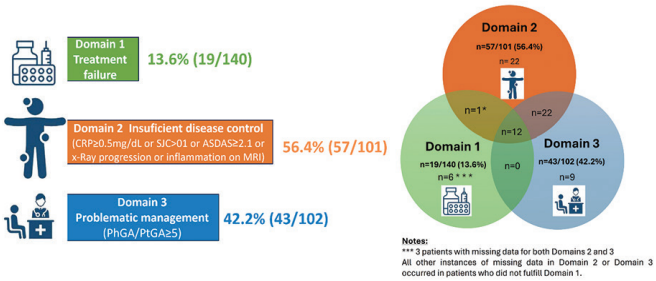


Fig. 1. Distribution and overlap of D2M-axSpA domains according to the ASAS 2025 definition. D2M-axSpA: difficult-to-manage axial spondyloarthritis; ASAS: Assessment of Spondyloarthritis international Society, CRP: C-reactive protein; SIJ: sacroiliac Joint; ASDAS: Ankylosing Spondylitis Disease Activity Score; MRI: magnetic resonance imaging; PhGA: physician global assessment; PtGA: patient global assessment.

Table I. Baseline clinical features of D2M vs. non-D2M axSpA, statistical comparison for model inclusion, and results of univariate analysis.

Variable	Total (N=140)	Non-D2M (N=128)	D2M (N=12)	p-value (Group comparison)	p-value (Univariate analysis)
Female sex, N (%)	56 (60.0)	50 (39.1)	6.0 (50.0)	0.543	-
Age, years	36.6±1.05	47 (24-75)	43 (25-62)	0.815	-
Obesity, N(%)	25 (17.9)	23 (18.0)	2 (16.7)	0.167*	0.461
Higher education, N (%)	35 (28.5)	32 (28.8)	3 (25.0)	0.539	-
No paid work, N (%)	8 (5.9%)	4 (3.2)	4 (33.3)	0.002*	0.001 (OR=15.0) *
Current smoking, N (%)	42 (33.9)	40 (31.3)	2 (16.7)	0.335	-
Symptom duration, months	144 (18-720)	144 (0-720)	168 (48-420)	0.674	-
Diagnostic delay, months	36.3(12.0-82.8)	35.3(1.0-720)	79.4 (1.0-720)	0.027*	0.014*
HLA-B27 positive, N (%)	70 (50.0)	65 (55.6)	5 (41.7)	0.602	-
Psoriasis, N (%)	10 (7.10)	10 (7.8)	0 (0.0)	0.601	-
IBD, N (%)	22 (15.7)	19 (14.8)	3 (25.0)	0.001*	0.049 (OR=4.10)
Uveitis, N (%)	23 (16.4)	22 (17.2)	0 (0.0)	0.213	-
Depression, N (%)	20 (14.3)	18 (14.1)	2(16.7)	0.681	-
Fibromyalgia, N (%)	10 (7.10)	7 (5.5)	3 (25)	0.041*	0.023 (OR=5.76)*
History of peripheral manifestations[a], N (%)	51 (36.4)	41 (32.0%)	7 (58.3)	0.541	-
NSAID (last visit), N (%)	42(30.9)	35 (27.3)	7 (58.3)	0.047*	0.04 (OR=3.56)*
csDMARD, N (%)	24 (17.5)	19 (15.2)	4 (33.3)	0.194*	0.088
bDMARD: TNFI, N (%)	112 (81.8)	104 (81.3)	8 (66.7)	0.224	-
bDMARD: IL-17I, N (%)	8 (5.80)	4 (3.2)	4 (33.3)	0.002*	0.001 (OR=15)**
bDMARD: other, N (%)	3 (2.10)	1 (0.80)	2 (16.7)	0.019*	0.011 (OR=30)**
tsDMARD, N (%)	6 (4.40)	2 (15.2)	4 (33.3)	0.001*	0.001 (OR=30)**
Glucocorticoid use (last visit), N (%)	9 (6.60)	6 (4.80)	3 (25.0)	0.024*	0.018 (OR=6.44)*
Analgesic use (last visit), N (%)	15 (10.9)	12 (9.60)	3 (25.0)	0.246	-
ASDAS (last visit)	1.95 (0.60-5.00)	1.8 (0.6-5)	3.1 (2.3-4.6)	<0.001*	<0.001* (OR=3.48)**
BASDAI (last visit), 0–10	2.48 (0.0-8.6)	3.0 (0.0-8.5)	6.1 (0.0-8.5)	<0.001*	<0.001*
BASFI (last visit), 0-10	2.79 (0-8.9)	1.8 (0.0-8.9)	5.15 (0.0-8.8)	0.023*	0.021 (OR=1.33)*
CRP (last visit), MG/L	0.15 (0.03-6.80)	0.14 (0.04- 6.9)	0.64 (0.04-4.0)	0.033*	0.033*
PTGA (last visit), 0–10	3.00 (0.50-10.0)	2 (0-10)	6.5 (4-10)	<0.001*	<0.001*
PHGA (last visit), 0–10	1.0(0-9.0)	1 (0-6)	5.3 (0-9)	0.040*	0.004

Comparison of demographic, clinical, and treatment-related characteristics between patients with D2M-axSpA (n=12) and non-D2M axSpA (n=128), among those treated with ≥1 b/tsDMARD (n=140). Values are presented as median (range) or n (%), as appropriate. Between-group comparisons used chi-square or Fisher's exact test for categorical variables, and t-test or Mann-Whitney U for continuous variables. Variables with p<0.20 in these comparisons were considered for univariate logistic regression (D2M as outcome); *p<0.05 was considered statistically significant. **expected value due to definition of D2M-axSpA.
OR: odds ratio; CI: confidence interval; ASDAS, BASDAI, BASFI: activity/functional indices; CRP: C-reactive protein; bDMARD: biological DMARD; tsDMARD: targeted synthetic DMARD; csDMARD: conventional synthetic DMARD; IL-17i: interleukin-17 inhibitor; TNFi: TNF inhibitor; NSAID: non-steroidal anti-inflammatory drug; IBD: inflammatory bowel disease; PtGA/PhGA: patient/physician global assessment.

P54

WHO GETS LEFT BEHIND? PATIENTS CLASSIFIED BY ASAS 2009 BUT EXCLUDED BY ASAS-SPARTAN 2025 IN A REAL-WORLD SPA COHORT

Resende G.G.[1], Saad C.G.S.[2], Marques C.[3], Marinho A.[4], Soares A.M.[5], Paiva B.E.[6], Rodrigues D.[4], Souza E.C.R.[7], Castro G.[8], Bulbol G.[6], Carneiro J.[9], Fernandes J.M.[10], Orchtrop M.[11], Gavi M.B.[12], Yazbek M.[13], Cavalcanti N.[3], Machado N.[14], Malheiro O.[1], Bassara-Macedo R.[2], Vieira R.[15], Lage R.[1], Menin R.C.[16], Golebiovski R.[17], Ribeiro S.[6], Oliveira T.[17], Diniz V.[12], Carvalho T.R.A.F.[11], Pinheiro M.M.[17], Sampaio-Barros P.[2]

[1]Hospital das Clínicas da Universidade Federal de Minas Gerais (UFMG) – MG; [2]Faculdade de Medicina da Universidade de São Paulo (USP) – SP; [3]Hospital das Clínicas da Universidade Federal de Pernambuco (UFPE) – PE; [4]Fundação Hospital do Acre – AC; [5]Hospital Universitário da Universidade Federal de Santa Catarina (UFSC) – SC; [6]Faculdade de Medicina da Universidade Federal do Amazonas (UFAM) – AM; [7]Hospital de Clínicas da Universidade Federal de Uberlândia (UFU) – MG; [8]Hospital Governador Celso Ramos (HCR) – SC; [9]Hospital Universitário de Brasilia; PPG em Ciências Médicas; FM/UnB – DF; [10]Universidade Federal do Maranhão (UFMA) – MA; [11]Hospital Universitário Pedro Ernesto / UERJ – RJ; [12]Universidade Federal do Espírito Santo (UFES) – ES; [13]Universidade Estadual de Campinas (UNICAMP) – SP; [14]Universidade Federal do Paraná (UFPR); [15]Universidade Estadual do Ceará (UECE) – CE; [16]Faculdade de Medicina de São José do Rio Preto (FAMERP) – SP; [17]Escola Paulista de Medicina (EPM) – Universidade Federal de São Paulo (UNIFESP) – SP, Brazil

Introduction. The ASAS-SPARTAN 2025 classification criteria for axial spondyloarthritis (axSpA) were developed to improve specificity compared with the ASAS 2009 criteria. This study aimed to compare the performance of the two criteria in a large real-world SpA cohort and to characterize discordant patients.

Methods. We analyzed 1,602 patients from the Brazilian Registry of Spondyloarthritis (RBE), including 1,200 investigator-defined axSpA patients and 402 peripheral SpA (pSpA) patients. Positive percent agreement (PPA) and negative percent agreement (NPA) were calculated. Agreement between the criteria sets was assessed using Cohen's kappa. Discriminative performance for axSpA versus pSpA was evaluated by ROC analysis. Logistic regression analyses were performed to identify factors independently associated with discordance.

Results. Among axSpA patients, 1,095 (91.2%) fulfilled ASAS 2009 criteria, and 1,021 (85.1%) fulfilled ASAS-SPARTAN 2025 criteria. Among pSpA patients, 58 (14.4%) fulfilled ASAS 2009 criteria versus 23 (5.7%) fulfilling ASAS-SPARTAN 2025 criteria, corresponding to higher operational NPA (used here as a proxy for specificity) for the 2025 criteria (94.3% vs 85.6%). Transition from ASAS 2009 to ASAS-SPARTAN 2025 exclusively reduced classification, excluding 74 previously classified patients and adding no new cases. In univariate analysis, imaging was the main driver of discordance: only 39.2% of patients fulfilling ASAS 2009 but not ASAS-SPARTAN 2025 criteria had sacroiliitis on X-ray or MRI, compared with 99.6% of concordant patients (Table I).

Table I. Clinical characteristics and univariable predictors of discordance between ASAS 2009 and ASAS-SPARTAN 2025 criteria among axial SpA patients.

Variable	Concordant n=1021	Discordant n=74	OR (95% CI)	p value
Any sacroiliitis imaging	1017 (99.6%)	29 (39.2%)	0.00 (0.00–0.01)	<0.0001
Radiographic sacroiliitis	905 (88.6%)	27 (36.5%)	0.07 (0.04–0.12)	<0.0001
Inflammatory back pain	980 (96.2%)	53 (71.6%)	0.11 (0.06–0.19)	<0.0001
Elevated CRP	717 (70.4%)	24 (32.4%)	0.20 (0.12–0.33)	<0.0001
Psoriasis	37 (3.6%)	12 (16.2%)	5.15 (2.47–10.11)	<0.0001
Heel enthesitis	384 (37.7%)	7 (9.5%)	0.17 (0.07–0.36)	<0.0001
MRI sacroiliitis	293 (28.8%)	2 (2.7%)	0.07 (0.01–0.22)	<0.001
Age at onset in years, mean (SD)	27.9 (11.1)	31.5 (11.5)	1.03 (1.01–1.05) per year	<0.01
IBD (Crohn/colitis)	46 (4.5%)	8 (10.8%)	2.57 (1.09–5.39)	0.02
Peripheral Arthritis	619 (60.6%)	35 (47.3%)	0.58 (0.36–0.94)	0.02
Female sex	303 (29.7%)	31 (41.9%)	1.70 (1.05–2.75)	0.03
HLA-B27 positivity	727 (71.4%)	45 (60.8%)	0.62 (0.38–1.02)	0.05
Dactylitis	68 (6.7%)	9 (12.2%)	1.94 (0.87–3.88)	0.08
Diagnostic delay in years, mean (SD)	8.7 (9.3)	6.8 (8.9)	0.97 (0.94–1) per year	0.11
Fibromyalgia	98 (9.6%)	11 (14.9%)	1.64 (0.8–3.11)	0.15
Good response to NSAIDs	489 (47.9%)	29 (39.2%)	0.7 (0.43–1.13)	0.15
Disease duration in years, mean (SD)	19.6 (11.9)	17.5 (11.7)	0.98 (0.96–1.01) per year	0.15
Age at inclusion in years, mean (SD)	47 (12.4)	48.6 (13.5)	1.01 (0.99–1.03) per year	0.28
Depression	92 (9%)	4 (5.4%)	0.58 (0.17–1.43)	0.29
Anxiety	119 (11.7%)	6 (8.1%)	0.67 (0.25–1.46)	0.35
Uveitis	348 (34.1%)	25 (33.8%)	0.99 (0.59–1.61)	0.96
Family history of SpA	251 (24.6%)	18 (24.3%)	0.99 (0.55–1.68)	0.96

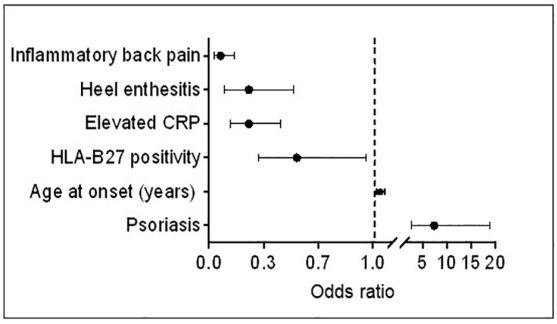


Fig. 1.

In the multivariable analysis, imaging variables produced quasi-complete separation. Consequently, a secondary model excluding imaging variables was fitted to identify other factors associated with discordance: inflammatory back pain (OR=0.07), elevated CRP (OR=0.24), heel enthesitis (OR=0.24), and HLA-B27 positivity (OR=0.53) remained independently associated with lower odds of discordance, whereas psoriasis (OR=7.39) and older age at symptom onset (OR=1.03 per year) were associated with higher odds of discordance (Fig. 1).

Conclusion. Compared with the ASAS 2009 classification criteria, the ASAS-SPARTAN 2025 criteria demonstrated higher operational specificity in a real-world SpA cohort and preferentially classified patients with a more objective inflammatory, classically axial phenotype.

P55

CARDIOVASCULAR RISK FACTORS BURDEN AND DISEASE ACTIVITY IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS

Silva C.[1], Rua C.[1], Patela M.[1], Fernandes M.A.[1], Vieira R.[1,2], Meirinhos T.[1], Pinto A.S.[1]

[1]Rheumatology Dept., Unidade Local de Saúde de Gaia/Espinho, Vila Nova de Gaia; [2]Dept. of Medical Sciences, University of Aveiro, Aveiro, Portugal

Introduction. Patients with axial spondyloarthritis (axSpA) have increased cardiovascular morbidity related to both systemic inflammation and traditional cardiovascular risk factors (CVRF). This study aimed to evaluate the association between CVRF burden and disease-related outcomes in axSpA patients.

Methods. A cross-sectional study including axSpA patients followed at our rheumatology centre and registered in Reuma.pt was conducted. Patients were assessed for five traditional CVRF (hypertension, dyslipidemia, diabetes mellitus, obesity, and active smoking) and grouped according to CVRF burden (0, 1, or ≥2 CVRF). A multivariable logistic regression model including clinically relevant and statistically significant variables across CVRF burden groups was performed to identify predictors of higher CVRF burden (≥2 CVRF).

Results. A total of 233 axSpA patients were included. While 36.1% (n=84) had no CVRF, 33.0% (n=77) and 31.0% (n=72) presented 1 and ≥2 CVRF, respectively (Table I). Statistically significant differences across CVRF burden groups were observed regarding age, sex, body mass index, ischemic heart disease, ASDAS-CRP, BASDAI and BASFI. In multivariable logistic regression analysis (Table II), older age (OR 1.08, 95%CI 1.04–1.12, p<0.001), male sex (OR 2.57, 95%CI 1.08–6.13, p=0.033), and higher ASDAS-CRP (OR 1.70, 95%CI 1.11–2.59, p=0.014) remained independently associated with the presence of ≥2 CVRF.

Conclusion. Patients with higher CVRF burden presented higher disease activity and worse functional status. After multivariable adjustment, older age, male sex and higher ASDAS-CRP remained independently associated with ≥2 CVRF, supporting a potential relationship between inflammatory burden and cardiovascular comorbidity in axSpA. These findings reinforce the importance of systematic CV risk assessment in axSpA patients.

Table I. Clinical characteristics according to cardiovascular risk factor burden.

Variable	Total (N=233)	0 CVRF (N=83)	1 CVRF (N=77)	≥2 CVRF (N=72)	p-value
Male	142 (60.9)	47 (56.0)	42 (54.5)	53 (73.6)	0.029
Age, years	48 (40-59)	42 (33-49)	46 (40-56)	58 (50-67)	<0.001
Cardiovascular data					
Hypertension	61 (26.2)	0 (0)	10 (13.0)	51 (70.8)	<0.001
Dyslipidemia	68 (29.2)	0 (0)	13 (16.9)	55 (76.4)	<0.001
Diabetes mellitus	27 (11.6)	0 (0)	1 (1.3)	26 (36.1)	<0.001
Body mass index	25.4 (22.9-28.3)	24.2 (22.5-26.0)	24.8 (22.7-27.0)	28.7 (25.4-32.0)	<0.001
Obesity	46 (19.7)	0 (0)	15 (19.5)	31 (43.1)	<0.001
Active smoking	65 (27.9)	0 (0)	38 (49.4)	27 (37.5)	<0.001
Number of CVRF - 0 - 1 - 2 - 3 - 4	 84 (36.1) 77 (33.0) 39 (16.7) 20 (8.6) 13 (5.6)	-	-	-	
Ischemic heart disease	8 (3.4)	0 (0)	0 (0)	8 (11.1)	<0.001
Stroke	5 (2.1)	1 (1.2)	2 (2.6)	2 (2.8)	0.741
Disease-related data					
Median disease duration, years	11 (6-22) (N=226)	10 (5-20) (N=82)	12 (6-20) (N=75)	16 (7-27) (N=68)	0.051
Radiographic axSpA	180 (77.9) (N=231)	63 (75.9) (N=83)	55 (71.4) (N=77)	62 (87.3) (N=71)	0.057
HLA-B27 positivity	116 (56.0) (N=207)	46 (60.5) (N=76)	36 (52.2) (N=69)	34 (54.8) (N=62)	0.606
Peripheral arthritis	50 (21.5)	24 (28.6)	12 (15.6)	14 (19.4)	0.114
Enthesitis	30 (12.9)	11 (13.1)	9 (11.7)	10 (13.9)	0.940
Dactylitis	7 (3.0)	3 (3.6)	2 (2.6)	2 (2.8)	1.000
Psoriasis	12 (5.2) (N=235)	6 (7.1)	3 (3.9)	3 (4.2)	0.613
Uveitis	39 (16.7)	15 (17.9)	13 (16.9)	11 (15.3)	0.928
Inflammatory bowel disease	26 (11.2) (N=235)	11 (13.1)	10 (13.0)	5 (6.9)	0.411
Anti-TNFα therapy	111 (48.3)	46 (54.8)	36 (46.8)	29 (40.3)	0.198
IL-17 inhibitor therapy	8 (3.4)	3 (3.6)	4 (5.2)	1 (1.4)	0.493
Janus kinase inhibitors therapy	6 (2.6)	3 (3.6)	3 (3.9)	0 (0)	0.287
Disease activity and functional status measures					
CRP, mg/dL	0.20 (0.09-0.54) (N=231)	0.16 (0.06-0.38) (N=83)	0.22 (0.08-0.60) (N=77)	0.27 (0.11-0.66) (N=71)	0.074
ASDAS-CRP	1.8 (1.2-2.7) (N=152)	1.6 (0.9-2.3) (N=56)	1.8 (1.2-2.7) (N=51)	2.1 (1.7-2.9) (N=45)	0.001
BASDAI	2.7 (1.0-4.8) (N = 154)	1.8 (0.6-3.7) (N=58)	3.0 (1.2-5.3) (N=51)	3.0 (2.3-5.1) (N=45)	0.006
BASFI	1.8 (0-4.9) (N=142)	0.6 (0-3.6) (N = 53)	1.7 (0-5.8) (N=49)	3.7 (1.7-5.7) (N=39)	0.001

Values are expressed as n (%) or median (IQR). N represents the number of patients with available data. Categorical variables were compared using Chi-square or Fisher's exact test, as appropriate. Continuous variables were compared using the Kruskal–Wallis test. A p-value <0.05 was considered statistically significant. Legend: ASDAS-CRP: Ankylosing Spondylitis Disease Activity Score using C-reactive protein; axSpA: axial spondyloarthritis; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index; CRP: C-reactive protein; CVRF: cardiovascular risk factors; HLA-B27: human leukocyte antigen B27; IL-17: interleukin-17; IQR: interquartile range; TNF: tumour necrosis factor.

Table II. Multivariable logistic regression analyses evaluating factors independently associated with the presence of ≥2 cardiovascular risk factors.

Variable	OR	95% CI	p-value
Age	1.08	1.04–1.12	<0.001
Male sex	2.57	1.08–6.13	0.033
ASDAS-CRP	1.70	1.11–2.59	0.014

A *p*-value <0.05 was considered statistically significant.
ASDAS-CRP: Ankylosing Spondylitis Disease Activity Score using C-reactive protein.

P56

ASSOCIATION BETWEEN AIR POLLUTION AND THE RISK OF FLARES IN AXIAL SPONDYLOARTHRITIS – A CASE CROSS-OVER ENVIRONMENTAL STUDY

Pujol S.[1], Bernard N.[2], Vauchy C.[3], Martin B.[1], Fossi Djembi L.[1], Prati C.[4], Mauny F.[1], Toussirot E.[3]
[1]CHU de Besançon Franche-Comté, Unité de Méthodologie uMETh, Centre d'Investigation Clinique INSERM CIC-1431, Besançon, France; [2]Université Marie et Louis Pasteur, UMR Chrono-environnement 6249 et UMR ThéMA 6049 CNRS, Besançon, France; [3]CHU de Besançon Franche-Comté, Centre d'Investigation Clinique INSERM CIC-1431, Besançon, France; [4]CHU de Besançon Franche-Comté, Rhumatologie, Besançon, France

Introduction. Environmental factors including air pollutants are involved in the pathophysiology of different inflammatory rheumatic diseases. They may influence disease activity and trigger flares. The relationships between ambient air pollution and disease activity in patients with axial spondyloarthritis (axSpA) have not been determined.
Methods. This was an environmental case cross-over study. Patients with axSpA came from a prospective clinical cohort of patients with inflammatory rheumatic diseases. Residential exposure to four pollutants (PM10, PM2.5, NO_2 and O_3) was evaluated using the patients' home addresses and daily modeling concentrations. For each patient, one case period was considered corresponding to the timeframe preceding a flare, along with four control periods that were temporally distant from the flare. We determined the odds ratios (ORs) associated with an increase of 10 μg/m^3 in air pollutant concentration.
Results. The study included 378 patients (242 radiographic axSpA; 58% male; 77% HLA B27+) for whom 182 flares were retained. The daily mean atmospheric concentrations of NO_2, O_3, PM2.5 and PM10 were 7.9, 58.2, 13.0 and 7.6 μg/m^3, respectively. The highest ORs associated with the occurrence of a flare were observed six weeks before a flare for PM10 (1.46 [1.09–1.94]), PM2.5 (1.72 [1.16–2.55]) and NO_2 (1.48 [0.92–2.38]). For O_3, the highest OR (1.06 [0.95–1.18]) was related to week 2 before a flare. A stratified analysis suggested the existence of a potential critical window one week before a flare for PM2.5 in patients who were female, had non radiographic axSpA, had extra-articular manifestations, were nonactive smokers, did not have HLA-B27, were taking bDMARDs or were younger than 48 years.
Conclusion. Exposure to air pollutants may trigger disease flares in patients with axSpA, especially when the patients are exposed to high concentrations of PM10, PM2.5 and NO_2 six weeks before the event.
Acknowledgements. This study was supported by a grant from the University Hospital of Besançon Franche Comté, Besancon, France (registration number P/2023/811).

P57

DO COPING STRATEGIES IMPACT HEALTH OUTCOME DOMAINS OVER TIME IN PATIENTS WITH LONGSTANDING AXSPA?

Van Essen M.D.[1,2,3], Webers C.J.P.[1,2,3], Capelusnik D.[1,4], Van der Heijde D.[5], Landewé R.B.M.[6,7], Van Tubergen A.[1,2], Boonen A.[1,2], Ramiro S.[5,7]
[1]Care and Public Health Research Institute (CAPHRI), Maastricht University, Maastricht; [2]Dept. of Rheumatology, Maastricht University Medical Center, Maastricht; [3]Dept. of Epidemiology, Maastricht University, Maastricht, The Netherlands; [4]Dept. of Rheumatology, Tel Aviv Sourasky Medical Center, Tel Aviv, Israel; [5]Dept. of Rheumatology, Leiden University Medical Center, Leiden; [6]Dept. of Rheumatology & Clinical Immunology, Amsterdam University Medical Centers, University of Amsterdam, Amsterdam; [7]Dept. of Rheumatology, Zuyderland Medical Center, Heerlen, The Netherlands

Objective. To investigate the impact of coping strategies on various health outcomes in longstanding axSpA over eight years of follow-up.
Methods. Data from the Outcome in AS International Study (OASIS) were used. Coping was assessed with the COping with Rheumatic Stressors (CORS) questionnaire (8 strategies to cope with pain, limitations, and dependence; score 1–4) at years 4 and 6. Health outcomes were assessed every other year up to year 12. Associations over time were determined using multivariable Generalized Estimating Equations (GEE) adjusted for confounders. Model 1 used year-4 coping as a time-fixed predictor across 8 years (baseline predictive model). Model 2 was time-lagged autoregressive, including the prior timepoint outcome as covariate.
Results. 116 patients [72% male, mean age 48 (SD: 12) years] had CORS data at ≥1 timepoint. Patients most frequently used *optimism* (mean: 3.1 (SD: 0.5)) and *comforting cognitions* (3.0 (0.5)) as coping and made the least frequent use of *accepting dependence* (2.3 (0.7)). In model 1 (Table I), three baseline coping strategies were relevant for future outcomes. *Optimism* was a significant predictor of less fatigue, less morning stiffness, better SF36-MCS and lower patient global assessment. C*reative solution seeking* was associated with worse SF36-PCS. *Accepting dependence* was associated with higher ASDAS, more fatigue and higher pain. In model 2 (time-lagged autoregressive) (Table II), again *optimism* and two other coping strategies were significant predictors for outcomes. More *optimism* was associated with lower ASDAS, less fatigue, less pain, less morning stiffness, better SF36-MCS and lower patient global assessment. Using more *comforting cognitions* or *showing consideration* was associated with more morning stiffness or higher BASFI scores, respectively.
Conclusion. Coping strategies show longitudinal independent associations with health outcomes. Optimism was most widely beneficial and the strongest coping-related predictor suggesting that interventions promoting an optimistic mindset may be valuable in longstanding axSpA.

Table I. Multivariable associations between year four time-fixed coping strategies (range 1-4) and health outcome domains over eight years (Baseline predictive model, Model 1).

Coping strategies, (Mean (SD))	ASDAS [a] (n = 87) β (95% CI)	BASFI [b], 0-10 (n = 103) β (95% CI)	BASMI [c], 0-10 (n = 95) β (95% CI)	Fatigue [d], 0-10 (n = 88) β (95% CI)	Back pain [e], 0-10 (n = 107) β (95% CI)	Morning stiffness [f], 0-10 (n = 109) β (95% CI)	SF36 MCS [g], 0-100 (n = 88) β (95% CI)	SF36 PCS [h], 0-100 (n = 96) β (95% CI)	Patient global assessment [i], 0-10 (n = 106) β (95% CI)
Comforting cognitions, (3.0 (0.5))	0.23 (-0.28 to 0.73)	-0.15 (-0.92 to 0.62)	0.03 (-0.98 to 1.04)	0.29 (-0.62 to 1.20)	0.14 (-0.60 to 0.88)	0.67 (-0.63 to 1.96)	0.60 (-3.49 to 4.69)	-1.25 (-4.29 to 1.80)	0.31 (-0.72 to 1.34)
Decreasing activities, (2.4 (0.5))	0.31 (-0.08 to 0.69)	0.17 (-0.52 to 0.86)	-0.43 (-1.16 to 0.30)	0.34 (-0.43 to 1.11)	0.09 (-0.57 to 0.74)	0.32 (-0.61 to 1.25)	-0.38 (-3.75 to 3.00)	1.08 (-1.81 to 3.98)	0.16 (-0.73 to 1.05)
Diverting attention, (2.4 (0.5))	-0.12 (-0.55 to 0.31)	-0.60 (-1.31 to 0.11)	-0.34 (-0.97 to 0.29)	0.56 (-0.28 to 1.39)	0.34 (-0.24 to 0.93)	-0.17 (-1.14 to 0.80)	-3.62 (-7.49 to 0.26)	0.64 (-1.67 to 2.95)	0.30 (-0.66 to 1.26)
Optimism, (3.1 (0.5))	-0.31 (-0.85 to 0.23)	0.33 (-0.35 to 1.00)	-0.27 (-1.12 to 0.57)	**-0.87 (-1.66 to -0.08)**	-0.24 (-1.14 to 0.66)	**-1.38 (-2.60 to -0.16)**	**6.65 (2.74 to 10.56)**	2.48 (-0.13 to 5.09)	**-1.24 (-2.17 to -0.30)**
Pacing, (2.7 (0.6))	-0.11 (-0.62 to 0.40)	-0.24 (-1.12 to 0.63)	0.07 (-0.78 to 0.93)	0.19 (-0.74 to 1.13)	-0.07 (-0.83 to 0.69)	0.33 (-0.72 to 1.37)	-1.38 (-5.60 to 2.84)	-2.03 (-4.64 to 0.58)	0.18 (-0.93 to 1.28)
Creative solution seeking, (2.6 (0.6))	0.11 (-0.38 to 0.59)	0.34 (-0.48 to 1.16)	0.48 (-0.34 to 1.30)	-0.44 (-1.39 to 0.51)	0.09 (-0.61 to 0.79)	0.03 (-0.96 to 1.02)	3.03 (-1.62 to 7.68)	**-2.70 (-5.18 to -0.21)**	-0.08 (-1.15 to 0.99)
Accepting dependence, (2.3 (0.7))	**0.27 (0.01 to 0.52)**	0.38 (-0.10 to 0.85)	0.41 (-0.09 to 0.90)	**0.58 (0.08 to 1.09)**	**0.69 (0.23 to 1.16)**	0.55 (-0.14 to 1.24)	-0.23 (-2.91 to 2.46)	-0.44 (-2.47 to 1.59)	0.57 (-0.01 to 1.15)
Showing consideration, (2.8 (0.4))	0.05 (-0.49 to 0.59)	0.59 (-0.15 to 1.33)	-0.14 (-0.97 to 0.68)	0.17 (-0.69 to 1.03)	-0.19 (-0.87 to 0.48)	0.26 (-0.91 to 1.42)	-3.68 (-8.30 to 0.93)	-1.91 (-4.80 to 0.97)	0.25 (-0.79 to 1.28)

[a] Model adjusted for age, sex, education level, employment status, BMI, ASAS NSAID score and bDMARDs;
[b] Model adjusted for sex, education level, employment status, comorbidities, ASDAS, BASMI, fatigue (BASDAI Q1) and bDMARDs;
[c] Model adjusted for age, sex, education level, employment status, comorbidities and ASDAS;
[d] Model adjusted for age, BMI, comorbidities, ASDAS and bDMARDs. Fatigue was measured using fatigue (BASDAI Q1);
[e] Model adjusted for age, education level, employment status, morning stiffness (BASDAI Q5/6) and ASAS NSAID score. Back pain was measured using BASDAI Q2;
[f] Model adjusted for sex, education level, comorbidities, ASAS NSAID score and bDMARDs. Morning stiffness was measured using BASDAI Q5/6;
[g] Model adjusted for sex, education level, BMI, comorbidities, ASDAS, BASMI, fatigue (BASDAI Q1) and ASAS NSAID score;
[h] Model adjusted for sex, comorbidities, ASDAS, BASFI and fatigue (BASDAI Q1);
[i] Model adjusted for education level, employment status, comorbidities, ASAS NSAID score, bDMARDs and csDMARDs.
Bold indicates significance (p<0.05).
BMI: Body Mass Index; SF36 MCS: Short Form (36) Mental Component Summary score; SF36 PCS: Short Form (36) Physical component score; ASDAS: Axial Spondyloarthritis Disease Activity Score; BASMI: Bath ankylosing spondylitis mobility index; BASFI: Bath ankylosing spondylitis functional index; BASDAI: Bath ankylosing spondylitis disease activity index; ASAS: Assessment of SpondyloArthritis international Society; NSAID: nonsteroidal anti-inflammatory drug; bDMARDs: biological disease-modifying anti-rheumatic drugs; csDMARDs: conventional synthetic disease-modifying anti-rheumatic drugs.

Table II. Time-lagged autoregressive multivariable associations between coping strategies (range 1-4) and health outcome domains over four years (Time-lagged autoregressive, Model 2).

Coping strategies, (Mean (SD))	ASDAS [a] (n=51) β (95% CI)	BASFI [b], 0-10 (n=62) β (95% CI)	BASMI [c], 0-10 (n=78) β (95% CI)	Fatigue [d], 0-10 (n=55) β (95% CI)	Back pain [e], 0-10 (n=78) β (95% CI)	Morning stiffness [f], 0-10 (n=77) β (95% CI)	SF36 MCS [g], 0-100 (n=53) β (95% CI)	SF36 PCS [h], 0-100 (n=64) β (95% CI)	Patient global assessment [i], 0-10 (n=78) β (95% CI)
Comforting cognitions, (3.0 (0.5))	0.12 (-0.24 to 0.47)	0.22 (-0.32 to 0.77)	-0.23 (-0.47 to 0.01)	0.53 (-0.21 to 1.27)	0.17 (-0.63 to 0.96)	**0.68 (0.03 to 1.34)**	1.62 (-3.04 to 6.28)	-2.79 (-5.67 to 0.09)	0.49 (-0.21 to 1.19)
Decreasing activities, (2.4 (0.5))	0.13 (-0.29 to 0.55)	0.15 (-0.31 to 0.62)	0.03 (-0.28 to 0.33)	-0.21 (-1.08 to 0.65)	0.26 (-0.51 to 1.03)	0.14 (-0.42 to 0.70)	2.55 (-3.02 to 8.12)	-0.98 (-4.71 to 2.76)	0.28 (-0.58 to 1.13)
Diverting attention, (2.4 (0.5))	0.24 (-0.15 to 0.62)	0.03 (-0.42 to 0.49)	0.22 (-0.04 to 0.33)	0.19 (-0.51 to 0.89)	0.59 (-0.03 to 1.21)	-0.21 (-0.80 to 0.37)	-1.82 (-5.97 to 2.33)	0.55 (-1.50 to 2.60)	0.06 (-0.68 to 0.80)
Optimism, (3.1 (0.5))	**-0.65 (-1.08 to -0.22)**	-0.27 (-0.81 to 0.27)	0.24 (-0.03 to 0.52)	**-1.40 (-2.50 to -0.29)**	**-0.98 (-1.82 to -0.14)**	**-1.01 (-1.90 to -0.13)**	**6.74 (1.83 to 11.66)**	1.79 (-1.08 to 4.67)	**-1.45 (-2.28 to -0.62)**
Pacing, (2.7 (0.6))	0.05 (-0.37 to 0.47)	-0.08 (-0.61 to 0.46)	0.07 (-0.18 to 0.32)	0.78 (-0.09 to 1.66)	-0.06 (-0.79 to 0.68)	0.02 (-0.56 to 0.61)	-1.58 (-7.49 to 4.33)	-1.13 (-4.30 to 2.03)	0.01 (-0.73 to 0.75)
Creative solution seeking, (2.6 (0.6))	-0.26 (-0.60 to 0.08)	0.10 (-0.45 to 0.65)	-0.11 (-0.37 to 0.15)	-0.41 (-1.20 to 0.39)	-0.04 (-0.68 to 0.60)	0.42 (-0.21 to 1.05)	0.36 (-4.67 to 5.39)	-1.14 (-3.55 to 1.27)	0.33 (-0.42 to 1.08)
Accepting dependence, (2.3 (0.7))	0.10 (-0.13 to 0.33)	0.18 (-0.14 to 0.49)	0.05 (-0.10 to 0.21)	0.25 (-0.28 to 0.79)	0.21 (-0.28 to 0.70)	0.05 (-0.35 to 0.45)	-0.39 (-3.20 to 2.43)	-1.13 (-3.13 to 0.88)	0.36 (-0.14 to 0.85)
Showing consideration, (2.8 (0.4))	0.19 (-0.18 to 0.56)	**0.53 (0.05 to 1.01)**	0.14 (-0.17 to 0.44)	0.73 (-0.04 to 1.49)	0.28 (-0.40 to 0.96)	0.04 (-0.76 to 0.83)	1.15 (-3.05 to 5.35)	-2.30 (-5.33 to 0.73)	0.30 (-0.54 to 1.13)

[a] Model adjusted for age, sex, education level, employment status, BMI, ASAS NSAID score and bDMARDs;
[b] Model adjusted for sex, education level, employment status, comorbidities, ASDAS, BASMI, fatigue (BASDAI Q1) and bDMARDs;
[c] Model adjusted for age, sex, education level, employment status, comorbidities and ASDAS;
[d] Model adjusted for age, BMI, comorbidities, ASDAS and bDMARDs. Fatigue was measured using fatigue (BASDAI Q1);
[e] Model adjusted for age, education level, employment status, morning stiffness (BASDAI Q5/6) and ASAS NSAID score. Back pain was measured using BASDAI Q2;
[f] Model adjusted for sex, education level, comorbidities, ASAS NSAID score and bDMARDs. Morning stiffness was measured using BASDAI Q5/6;
[g] Model adjusted for sex, education level, BMI, comorbidities, ASDAS, BASMI, fatigue (BASDAI Q1) and ASAS NSAID score;
[h] Model adjusted for sex, comorbidities, ASDAS, BASFI and fatigue (BASDAI Q1);
[i] Model adjusted for education level, employment status, comorbidities, ASAS NSAID score, bDMARDs and csDMARDs.
Bold indicates significance (p<0.05).
BMI: Body Mass Index; SF36 MCS: Short Form (36) Mental Component Summary score; SF36 PCS: Short Form (36) Physical component score; ASDAS: Axial Spondyloarthritis Disease Activity Score; BASMI: Bath ankylosing spondylitis mobility index; BASFI: Bath ankylosing spondylitis functional index; BASDAI: Bath ankylosing spondylitis disease activity index; ASAS: Assessment of SpondyloArthritis international Society; NSAID: nonsteroidal anti-inflammatory drug; bDMARDs: biological disease-modifying anti-rheumatic drugs; csDMARDs: conventional synthetic disease-modifying anti-rheumatic drugs.

P58

GROUP-LEVEL STABILITY IN COPING USE MASKS INDIVIDUAL CHANGES IN AXIAL SPONDYLOARTHRITIS: A LONGITUDINAL ANALYSIS

Van Essen M.D.[1,2], Ramiro S.[3,4], Landewé R.B.M.[4,5], Van Tubergen, A.[1,2], Boonen A.[1,2]
[1]Care and Public Health Research Institute (CAPHRI), Maastricht University, Maastricht; [2]Dept. of Rheumatology, Maastricht University Medical Center, Maastricht; [3]Dept. of Rheumatology, Leiden University Medical Center, Leiden; [4]Dept. of Rheumatology, Zuyderland Medical Center, Heerlen; [5]Dept. of Rheumatology & Clinical Immunology, Amsterdam University Medical Centers, University of Amsterdam, Amsterdam, The Netherlands

Objective. To determine occurrence and co-occurrence of individual changes in the use coping strategies in axSpA, and to explore which patients exhibit changes.
Methods. Data from the Outcome in AS International Study (OASIS) were used. Coping was assessed with the COping with Rheumatic Stressors (CORS) questionnaire (eight strategies to cope with pain, limitations, and dependence; score 1–4) at years 4 and 6. Smallest Detectable Changes (SDC) were calculated for all eight subscales. Change was categorised as increased, stable, or decreased use beyond the SDC, and a predominant change direction was determined. Co-occurrence of predominant change across strategies was examined. Multivariable logistic regression, containing patient characteristics and changes in health outcome scores (between years 2 and 4), was used to predict changes in coping (between years 4 and 6).
Results. 102 patients [73% male, mean age 50 (SD: 11) years] had CORS data at both timepoints. SDCs ranged from 0.78 (*showing consideration*) to 1.14 (*comforting cognitions*) (Table I). Individual change beyond the SDC occurred in 16%–37% of patients depending on the strategy, with clear predominant directions. Four strategies showed predominant increases and four predominant decreases. Only 17% of patients showed no change in any coping strategy. Co-occurrence of change across coping strategies happened infrequently (0–15%) and did not align with the stressor-specific grouping of strategies. No consistent associations were found between patient demographics or change in health outcomes and subsequent individual coping changes (Table II).
Conclusion. Change in coping use beyond measurement error was not uncommon and followed clear predominant directions, with co-occurrence of change not aligning with the stressor-specific coping structure. No clear determinants of change were identified. Although findings should be replicated in larger samples, results highlight that individual coping trajectories are more dynamic and broadly used than group-level data suggest.

Table I. Distribution of change in coping strategy scores between years 4 and 6 relative to the smallest detectable change (n=102).

Disease stressor	Coping strategy	SDC*	Change beyond SDC, n (%)**		Change 1-4, mean (SD)
Pain	Comforting cognitions	1.14	Decrease	0 (0.0%)	-
			Stable	69 (67.6%)	0.21 (0.56)
			Increase	**33 (33.7%)**	1.60 (0.35)
	Decreasing activities	0.90	**Decrease**	**19 (18.6%)**	-1.37 (0.36)
			Stable	81 (79.4%)	-0.21 (0.43)
			Increase	2 (2.0%)	1.11 (0.03)
	Diverting attention	0.91	**Decrease**	**38 (37.3%)**	-1.35 (0.33)
			Stable	63 (61.8%)	-0.30 (0.42)
			Increase	0 (0.0%)	-
Limitations	Optimism	1.09	Decrease	1 (1.0%)	-1.38 (n.a.)
			Stable	70 (68.6%)	0.30 (0.50)
			Increase	**31 (33.3%)**	1.60 (0.43)
	Pacing	0.99	Decrease	2 (2.0%)	-1.29 (0.41)
			Stable	81 (79.4%)	0.12 (0.47)
			Increase	**19 (19.2%)**	1.41 (0.35)
	Creative solution seeking	1.01	**Decrease**	**16 (15.7%)**	-1.64 (0.54)
			Stable	84 (82.4%)	-0.31 (0.46)
			Increase	2 (2.0%)	1.48 (0.30)
Dependence	Accepting dependence	0.99	Decrease	3 (3.0%)	-1.36 (0.33)
			Stable	81 (79.4%)	-0.15 (0.51)
			Increase	**18 (18.0%)**	1.15 (0.07)
	Showing consideration	0.78	**Decrease**	**22 (21.6%)**	-1.04 (0.08)
			Stable	78 (76.5%)	0.10 (0.40)
			Increase	2 (2.0%)	1.04 (0.22)

* SDC was calculated according to Bruynesteyn *et al*. (2005): $SDC = \pm 1.96 \times SD_{\Delta \text{change scores}} / (\sqrt{2} \times \sqrt{k})$;
** Percentage change adjusted for floor and ceiling values;
Predominant change direction for each coping strategy is shown in bold.
SDC: smallest detectable change.

Table II. Association between patient demographics, change in health outcomes (years 2-4) and predominant change in each copying strategy (years 4-6) – multivariable logistic regression.

	Coping with pain			Coping with limitations			Coping with dependence	
	Increasing comforting cognitions use, (n=93) OR (95% CI)	Decreasing decreasing activities use, (n=83) OR (95% CI)	Decreasing diverting attention use, (n=81) OR (95% CI)	Increasing optimism use, (n=83) OR (95% CI)	Increasing pacing use, (n=81) OR (95% CI)	Decreasing creative solution seeking use, (n=80) OR (95% CI)	Increasing accepting dependence use, (n=81) OR (95% CI)	Decreasing showing consideration use, (n=79) OR (95% CI)
Age	0.99 (0.95 – 1.04)	1.00 (0.96 – 1.06)	0.99 (0.94 – 1.03)	1.02 (0.98 – 1.07)	1.03 (0.98 – 1.09)	0.96 (0.91 – 1.02)	0.98 (0.93 – 1.03)	1.03 (0.98 – 1.08)
Sex	1.11 (0.38 – 3.21)	0.60 (0.19 – 1.96)	0.37 (0.13 – 1.02)	1.20 (0.39 – 3.71)	0.53 (0.15 – 1.87)	0.59 (0.15 – 2.33)	1.67 (0.41 – 6.72)	0.72 (0.22 – 2.33)
Lower vs. higher education	0.89 (0.27 to 2.99)	1.81 (0.47 – 6.92)	2.43 (0.76 – 7.77)	1.99 (0.59 – 6.69)	2.52 (0.67 – 9.46)	1.03 (0.19 – 5.60)	0.78 (0.18 – 3.35)	1.04 (0.25 – 4.42)
Change in CRP level	0.98 (0.93 – 1.02)	0.99 (0.95 – 1.03)	1.02 (0.99 – 1.04)	1.01 (0.98 – 1.03)	0.98 (0.92 – 1.03)	0.98 (0.92 – 1.04)	0.99 (0.95 – 1.03)	1.00 (0.98 – 1.03)
Change in back pain, 0-10	1.14 (0.91 – 1.43)	0.91 (0.70 – 1.19)	1.03 (0.82 – 1.29)	- [a]	- [a]	- [a]	- [a]	- [a]
Change in BASFI, 0-10	- [a]	- [a]	- [a]	0.65 (0.40 – 1.06)	**1.78 (1.05 – 3.04)**	1.00 (0.56 – 1.77)	1.45 (0.91 – 2.32)	0.70 (0.41 – 1.20)

[a] Variable not considered as an explanatory factor in this model;
Bold indicates significance ($p<0.05$). Change is modelled as differences between years 2 and 4; Separate models explored ASDAS, back pain and BASFI (cross-sectional, non-change score values) as explanatory factors but showed non-significant associations with coping change.
ASDAS: Axial Spondyloarthritis Disease Activity Score; BASFI: Bath Ankylosing Spondylitis Functional Index; CRP: C-reactive protein; SF36 MCS: Short Form (36) Mental Component Summary score.

P59

AXIAL SPONDYLOARTHRITIS ACROSS ARAB COUNTRIES: A MULTINATIONAL CHARACTERISATION OF OVER 1,000 PATIENTS FROM 15 NATIONS

Ziadé N.[1], Abou Abdallah F.[1], Tekaya R.[2], Alchama N.[3], Maroof A.[4], Haouichat C.[5], Ayoub L.[6], Allat R.[5], Rahmouni S.[7], Saad S.[8], Al Khater N.R.[8], Aljazwi L.M.[6], Bahiri R.[9], Djennane M.[10], Khalifa D.[11], Zemrani S.[12], Alnaimat F.[13], Hannawi S.[14], Hamdi W.[15], Taha Z.I.[16], Mahmoud I.[2], Abdellaoui S.[17], Abdulateef N.[18], Redha A.A.[18], Al Rayes H.[19], Aouad K.[20], Atawneh S.[21], Dahou Makhloufi C.[22], Gamal S.M.[23], Gorial F.I.[18], Awadh N.I.[18], Ismailoustafa N.M.[24], Ben Abdelghani K.[25], Boussaa H.[25], Heddi F.Z.Y.[5], Dougados M.[26], Hmamouchi I.[27]
[1]Saint Joseph University, Hotel-Dieu de France Hospital, Beirut, Lebanon; [2]Tunia El Manar University, Charles Nicolle Hospital, Tunis, Tunisia; [3]Ibn-Alnafis Hospital, Damascus, Syria; [4]University of Kurdistan Hawler, School of Medicine, Erbil, Iraq; [5]Saad Dahlab Blida University, Douera University Hospital, Algiers, Algeria; [6]Tripoli Medical University, Tripoli Central Hospital, Tripoli, Libya; [7]Tunis Manar University, Rabta Hospital, Tunis, Tunisia; [8]King Hamad University Hospital, Bahrain, Bahrain; [9]El Ayachi Hospital, Rabat University Hospital, Rabat, Morocco; [10]Rheumatology Dept., University Hospital, Faculty of Medicine Mouloud Mammeri, Tizi Ouzou, Algeria; [11]Sousse University, Farhat Hached University Hospital, Sousse, Tunisia; [12]Dept. of Rheumatology A, El Ayachi Hospital, Ibn Sina University Hospital, Rabat-Salé, Morocco; [13]Dept. of Internal Medicine, Division of Rheumatology, School of Medicine, The University of Jordan, Amman, Jordan; [14]Alkuwait Hospital-Dubai, Emirates Health Services, Dubai, UAE; [15]Tunis el Manar University, Faculty of Medicine of Tunis, Kassab Institute, Tunis, Tunisia; [16]University of Bahri, Khartoum, Sudan; [17]Issaad Hassani Ben Messous Hospital, Algiers, Algeria; [18]Rheumatology Unit, Dept. of Medicine, University of Baghdad, College of Medicine, Baghdad, Iraq; [19]Prince Sultan Military Medical City, Princess Nourah Bint Abdulrahman University, Riyadh, Saudi Arabia; [20]Saint George University of Beirut, Saint George University Medical Center, Beirut, Lebanon; [21]Dept. of Internal Medicine, Al-Ahli Hospital, Hebron, Palestine; [22]Mohamed Lamine Debaghine University Hospital, Bab el Oued, Algiers, Algeria; [23]Cairo University, Cairo, Egypt; [24]Suez Canal University, Egypt; Asterclinics, Abu Dhabi, UAE; [25]Dept. of Rheumatology, Mongi Slim Hospital, La Marsa, Faculty of Medicine of Tunis, University of Tunis El Manar, Tunis, Tunisia; [26]Dept. of Rheumatology, Hôpital Cochin, Université Paris Cité, Paris, France; [27]International University of Rabat, Rabat, Morocco

Introduction. Axial spondyloarthritis (axSpA) is a chronic inflammatory disease with heterogeneous manifestations and a substantial comorbidity burden. Data from Arab countries remains limited, and large standardised multinational registries are needed to better characterise axSpA phenotype and management across Arab populations.
Methods. This cross-sectional ArLAR Cross-sectional cohort multinational study enrolled consecutive eligible adults with rheumatologist-confirmed axSpA from 26 tertiary centres across 15 Arab countries between February and November 2025. A standardised case report form captured physician-reported clinical data and validated patient-reported outcomes. Descriptive and regional comparative analyses were performed across North Africa, the Levant, and the Gulf.
Results. Among 1,112 patients (North Africa 54%, Levant 32%, Gulf 14%), mean age was 42.5 years, 64% were male, median disease duration was 10 years, and median diagnostic delay was 3 years. Subtypes included radiographic axSpA (62%), non-radiographic axSpA (17%), axial psoriatic arthritis (12%), IBD-associated axSpA (5%), and unclassified axSpA (4%); HLA-B27 was positive in 52%. Peripheral arthritis (46%), enthesitis (29%), psoriasis (17%), uveitis (17%), and IBD (9%) were common extra-articular features. Comorbidities included smoking (36%), obesity (27%), and fibromyalgia (22%). Mean BASDAI was 3.8±1.9 and BASFI 3.9±2.1; bDMARD ever-use reached 70%, reflecting the tertiary-centre setting. Significant regional differences were observed: North Africa had higher HLA-B27 positivity, more radiographic damage, and worse functional indices; Gulf patients were more often female with more metabolic comorbidities but better global health. The Levant showed higher rates of enthesitis and dactylitis.
Conclusion. This first standardised ArLAR multinational cohort demonstrates a high disease burden with substantial regional heterogeneity in damage, comorbidities, and function across Arab countries, though tertiary-centre sampling may limit generalisability. These findings support the need for region-adapted, patient-centred care strategies.

Table I. Key regional differences in patients' demographics, disease phenotype, comorbidities, and treatments in 1112 patients with axial spondyloarthritis from 15 Arab countries.

	N*	All	North Africa	Levant	Gulf	p-value
N (%)		1112	599 (54)	354 (32)	159 (14)	
Age, mean (SD)	956	42.5 (12.2)	43.4 (12.5)	41.1 (12.4)	42.5 (12.2)	0.034
Male Sex, n (%)	1112	710 (63.8)	411 (68.6)	236 (66.7)	63 (39.6)	<0.001
Active occupation, n (%)	992	640 (64.5)	336 (61.3)	222 (74.0)	82 (56.9)	<0.001
axSpA disease, n (%)	1107					<0.001
- r-axSpA		690 (62.3)	456 (76.7)	182 (51.9)	50 (31.4)	
- nr-axSpA		187 (16.9)	42 (7.0)	85 (84.2)	60 (37.7)	
- axPsA		129 (11.7)	57 (9.5)	42 (12.0)	30 (18.9)	
- IBD-SpA		52 (4.7)	34 (5.7)	10 (2.8)	8 (5.0)	
Disease duration, years, median (IQR)	1104	10.0 (5-18)	13.0 (7.0-20)	7.0 (4.0-13.0)	7.0 (4.0-13.0)	<0.001
Diagnostic delay, years, median (IQR)	1110	3.0 (1-8)	4.0 (1-9)	3.0 (1-7)	1.0 (0-3)	<0.001
Arthritis, n (%)	1112	508 (45.7)	306 (51.1)	135 (38.1)	67 (42.1)25	<0.001
Enthesitis, n (%)	1112	320 (28.8)	147 (24.5)	121 (34.2)	52 (32.7)	0.003
Dactylitis, n (%)	1112	60 (5.4)	27 (4.5)	29 (8.2)	4 (2.5)	0.011
Psoriasis, n (%)	1112	193 (17.4)	76 (12.7)	72 (20.3)	45 (28.3)	<0.001
Uveitis, n (%)	1112	186 (16.7)	123 (20.5)	49 (13.8)	14 (8.8)	<0.001
IBD, n (%)	1112	95 (8.5)	61 (10.2)	20 (5.6)	14 (8.8)	0.053
BASMI score, mean (SD)	674	2.9 (1.9)	3.8 (1.7)	2.4 (1.3)	1.1 (1.6)	<0.001
Spine structural lesions, n (%)	883	478 (48.7)	347 (66.0)	99 (32.0)	32 (21.9)	<0.001
- Syndesmophytes		293 (26.3)	221 (36.9)	55 (15.5)	17 (10.7)	<0.001
Radiographic sacroiliitis, n (%)	1000	758 (68.2)	475 (79.3)	195 (55.1)	88 (55.3)	<0.001
Coxitis, n (%)	1017	323 (35.0)	245 (47.9)	62 (23.7)	16 (10.7)	<0.001
Shoulder arthritis, n (%)	1026	129 (13.5)	61 (12.2)	58 (19.0)	10 (6.7)	<0.001
Elevated CRP, n (%)	1035	810 (76.8)	444 (77.9)	241 (73.7)	125 (79.1)	0.422
Positive HLA-B27, n (%)	615	317 (51.5)	176 (62.9)	101 (48.8)	40 (31.3)	<0.001
Surgery related to axSpA, n (%)	1025	71 (6.9)	60 (11.2)	10 (3.0)	1 (0.6)	<0.001
NSAIDs, n (%)	1108	1013 (91.4)	547 (91.6)	325 (92.1)	141 (89.2)	0.555
- Non-selective		765 (68.9)	473 (79.2)	198 (55.9)	94 (59.1)	<0.001
- COX-2 inhibitors		449 (40.5)	204 (34.2)	152 (42.9)	93 (58.5)	<0.001
Corticosteroid therapy, n (%)	1103	155 (14.1)	96 (16.1)	43 (12.3)	16 (10.1)	0.083
csDMARDs, n (%)	1107	472 (42.6)	288 (48.3)	126 (35.7)	58 (36.7)	<0.001
bDMARDs, n (%)	1107	776 (70.1)	370 (62.1)	278 (78.8)	128 (81.0)	<0.001
- TNFi		712 (64.0)	358 (59.8)	252 (71.2)	102 (64.2)	0.002
- IL17i		97 (8.7)	25 (4.2)	35 (9.9)	37 (23.3)	<0.001

axSpA: axial spondyloarthritis, BASMI: Bath Ankylosing Spondylitis Metrology Index, bDMARDs: biologic disease-modifying anti-rheumatic drugs, csDMARDs: conventional synthetic disease-modifying anti-rheumatic drugs, CRP: C-reactive protein, HLA-B27: Human Leucocyte Antigen B27, IBD: inflammatory bowel disease, IL17i: Interleukin 17 inhibitors, IQR: interquartile range, nr-axSpA: non-radiographic axSpA, NSAIDs: non-steroidal anti-inflammatory drugs, r-axSpA: radiographic axSpA, SD: standard deviation, TNFi: tumor necrosis factor inhibitors
*Number of available data

Table II. Key regional differences in comorbidities and patient-reported outcomes in 1112 patients with axial spondyloarthritis from 15 Arab countries.

	N	All	North Africa	Levant	Gulf	p-value
N (%)		1112	599 (54)	354 (32)	159 (14)	
Smoking (ever), n (%)	1013	369 (36.4)	227 (41.1)	122 (39.7)	20 (13.0)	<0.001
BMI m/kg^2, mean (SD)	1080	28.6 (10.8)	25.9 (5.7)	32.8 (16.3)	29.1 (5.7)	<0.001
Hypertension, n (%)	1101	199 (17.9)	96 (16.0)	80 (22.6)	23 (14.5)	0.025
Diabetes, n (%)	1101	93 (8.4)	53 (8.8)	21 (5.9)	19 (11.9)	0.090
Dyslipidemia, n (%)	1102	166 (15.1)	72 (12.1)	55 (15.8)	39 (24.7)	<0.001
Osteoporosis, n (%)	963	96 (10.0)	60 (11.4)	25 (8.9)	11 (7.1)	0.211
Fibromyalgia, diagnosed by the physician, n (%)	1101	129 (11.7)	53 (8.9)	61 (17.5)	15 (9.5)	<0.001
Elevated FiRST score, n (%)	1024	230 (22.5)	116 (19.9)	77 (24.8)	37 (28.5)	0.051
Severe infections, n (%)	1102	52 (4.7)	26 (4.4)	25 (7.2)	1 (0.6)	0.005
Tuberculosis (ever), n (%)	1022	53 (5.2)	40 (7.4)	7 (2.2)	6 (3.8)	0.003
History of cancer, n (%)	1110	30 (2.7)	10 (1.7)	17 (4.8)	3 (1.9)	0.016
BASDAI, mean (SD)	1031	3.8 (2.4)	3.8 (2.4)	3.7 (2.4)	3.8 (2.5)	0.560
ASDAS-CRP, mean (SD)	1011	2.9 (0.9)	3.0 (1.0)	2.8 (1.0)	2.9 (0.8)	0.152
ASAS-HI, mean (SD)	1029	6.7 (4.4)	6.9 (4.1)	6.8 (4.7)	5.4 (4.5)	<0.001
BASFI, mean (SD)	1029	3.9 (3.7)	4.6 (4.2)	3.1 (2.6)	2.8 (2.2)	<0.001
GPH, mean (SD)	1023	12.2 (3.1)	11.9 (3.0)	12.6 (3.1)	12.9 (3.0)	<0.001
GMH, mean (SD)	1023	12.2 (3.5)	11.9 (3.5)	12.5 (3.5)	13.2 (3.2)	<0.001
PHQ4, mean (SD)	1023	3.1 (2.9)	3.3 (3.1)	2.9 (2.7)	2.4 (2.8)	0.003
IPAQ category	1031					<0.001
- Inactive, n (%)		398 (38.6)	229 (39.1)	104 (33.5)	65 (47.8)	
- Moderate		360 (34.9)	218 (37.3)	93 (30.0)	49 (36.0)	
- Active		273 (26.5)	138 (23.6)	113 (36.5)	22 (16.2)	

ASAS-HI: Assessment of Spondyloarthritis international Society Health Index, ASDAS: Assessment of Spondyloarthritis international Society Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index; FiRST: Fibromyalgia Screening Tool; GMH: Global Mental Health; GPH: Global Physical Health; IPAQ: International Physical Activity Questionnaire; PHQ4: Patient Health Questionnaire.

P60

FROM GENETIC RISK TO THERAPEUTIC TARGET: THE MYELOID CARD9 REGULATORY AXIS IN AXIAL SPONDYLOARTHRITIS

Frison E.[1], Gervaise P.[1], Brown A.C.[2,3], Cohen C.[2,3], Jacoutot M.[1], Migliorini G.[2,3], Said-Nahal R.[1,4], Scozzafava G.[2,3], Bowness P.[2,5], Wordsworth B.P.[2,5], Garchon H.J.[1,6], Glatigny S.[1], Breban M.[1,4], Knight J.C.[2,5], Costantino F.[1,4]
[1]UMR1173, Université de Versailles St Quentin en Yvelines/Inserm, Montigny-le-Bretonneux, France; [2]NIHR Oxford Biomedical Research Centre, Oxford; [3]Centre for Human Genetics, University of Oxford, Oxford, UK; [4]Rheumatology Dept., Ambroise Paré Hospital (AP-HP), Boulogne-Billancourt, France; [5]NDORMS, University of Oxford, Oxford, UK; [6]Genetics Dept., Ambroise Paré Hospital (AP-HP), Boulogne-Billancourt, France

Introduction/Objective. The *CARD9* locus is a major non-MHC susceptibility region shared by axial spondyloarthritis (axSpA), inflammatory bowel diseases (IBD), and IgA nephropathy (IgAN). We aimed to identify the causal variants at this locus and assess their functional and therapeutic relevance.
Materials and methods. We integrated Bayesian fine-mapping with eQTL and caQTL datasets. Chromatin accessibility profiling and nucleosome positioning were investigated in primary human monocytes from SpA patients and HC. Functional consequences of high basal CARD9 expression were assessed by single-cell RNA sequencing of peripheral blood mononuclear cells before and after *Candida albicans* stimulation in healthy controls. Pharmacological CARD9 inhibition (BRD5529) was tested in LPS or anti-CD3/CD28-stimulated PBMCs from three axSpA patients via multiplex cytokine assays.
Results. Fine-mapping identified a shared causal signal across axSpA, IBD, and IgAN within a 32-variant credible set (H4-PP: 0.86; lead SNP rs11793497). This pleiotropic signal colocalized with a myeloid-specific distal enhancer regulating *CARD9*, which contains three credible set variants, including rs11793497. The risk haplotype induced a 55-bp local nucleosomal shift, increasing chromatin accessibility and basal *CARD9* expression in monocytes (Fig. 1). High basal CARD9 levels primed monocytes toward an exaggerated inflammatory response upon *C. albicans* stimulation, amplifying downstream CD4+ and CD8+ T-cell activation signatures (Fig. 2). BRD5529-mediated inhibition significantly reduced LPS-induced IL-23 (-38%), TNF (–66%), and CCL2 (-99%) at 18h, while decreasing IL-17A release (-52%) following anti-CD3/CD28 stimulation (74h). Paradoxically, CARD9 blockade increased inflammasome-related outputs (IL-1beta and IL-18 production).
Conclusion. Genetically driven CARD9 overexpression programs a myeloid-primed inflammatory state shared across axSpA and related immune-mediated diseases. Targeting the CARD9 regulatory axis suppresses key IL-23/IL-17 pathway outputs, positioning CARD9 as a novel upstream therapeutic target in axSpA. *In vivo* validation in the SKG-curdlan SpA mouse model is underway.

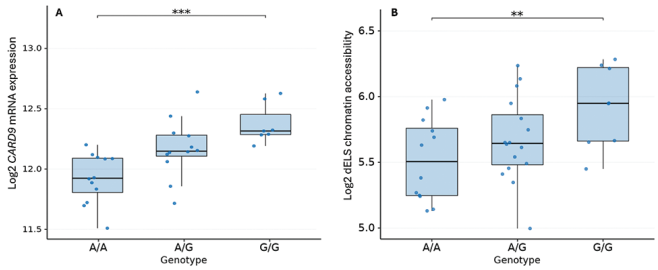


Fig. 1. *CARD9* cromatin accessibility and expression by rs11793497 genotype.
A. *CARD9* mRNA expression in primary human monocytes according to rs11793497 genotype, with the G allele denoting susceptibility.
B. Chromatin accessibility at the *CARD9* distal enhancer (dELS) by genotype. Data are presented as boxplots including 16 axSpA patients and 19 healthy controls. Horizontal lines indicate medians; whiskers represent the interquartile range. Statistical significance determined by linear regression; $p<0.01$, *** $p<0.001$.

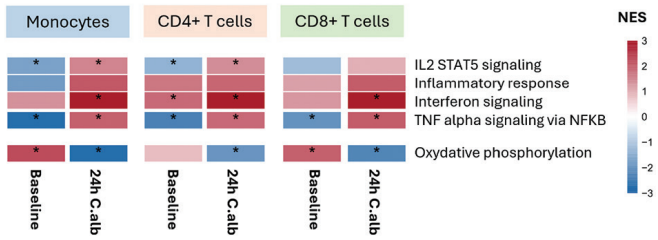


Fig. 2. Transcriptomic profiling of PBMCs based on baseline monocyte *CARD9* expression levels.
Heatmap of Gene Set Enrichment Analysis (GSEA) Normalized Enrichment Scores (NES) for selected hallmark pathways in monocytes, CD4+, and CD8+ T cells, stratified by baseline monocyte *CARD9* expression levels. Data are shown for two conditions: Baseline and 24h post-*C. albicans* (*C. alb*) stimulation. The colour scale represents the NES, with red indicating positive enrichment and blue indicating negative enrichment. * False discovery rate <0.05.

P61

PLASMA EXOSOMES INDUCE REGULATORY T CELL DYSFUNCTION IN ANKYLOSING SPONDYLITIS

Tavasolian F.[1,2], Lively S.[2,3,4,5], Pastrello C.[2,5,8], Tang M.[1,2], Lim M.[1,2], Pacheco A.[1,2], Qaiyum Z.[1,2], Yau E.[1,2], Baskurt Z.[7], Jurisica I.[2,5,8], Kapoor M.[2,3,4,5], Inman R.D.[2,9,10]
[1]Schroeder Arthritis Institute, Toronto Western Hospital, University Health Network, Toronto, ON; [2]Krembil Research Institute, University Health Network, Toronto, ON; [3]Osteoarthritis Research Program, Division of Orthopaedics, Schroeder Arthritis Institute, University Health Network, Toronto, ON; [4]Dept. of Medicine and Pathobiology, University of Toronto, Toronto, ON; [5]Dept. of Surgery, Division of Orthopaedic Surgery, University of Toronto, Toronto, ON; [6]Osteoarthritis Research Program, Division of Orthopedic Surgery, Schroeder Arthritis Institute, and Data Science Discovery Centre for Chronic Diseases; [7]Dept. of Biostatistics, University Health Network, Toronto, ON, Canada; [8]Depts. of Medical Biophysics and Computer Science, and Faculty of Dentistry, University of Toronto, Canada; and Institute of Neuroimmunology, Slovak Academy of Sciences, Bratislava, Slovakia; [9]Spondylitis Program, Division of Rheumatology, Schroeder Arthritis Institute, University Health Network, Toronto, ON; [10]Depts. of Medicine and Immunology, University of Toronto, Toronto, Ontario, Canada
Correspondence to: Robert.Inman@uhn.ca

Background. Regulatory T cells (Tregs) are present but functionally impaired in ankylosing spondylitis (AS). The upstream mechanisms driving this dysfunction remain unclear. We investigated whether plasma-derived exosomes contribute to Treg instability through coordinated molecular and signaling pathways.
Methods. Exosomes were isolated from platelet-poor plasma of AS patients and healthy controls using ultracentrifugation and size-exclusion chromatography, with standard quality controls (CD9/CD63/CD81 profiling, nanoparticle tracking). Primary human CD4+CD25^high^CD127^low^ Tregs from healthy donors were exposed to exosomes and assessed by flow cytometry, suppression assays, cytokine measurement, and DNA methylation analysis. Exosomal cargo was profiled using miRNA sequencing and cytokine assays. Single-cell RNA-sequencing datasets of AS peripheral blood and synovial fluid Tregs were reanalyzed to validate *in vitro* findings.
Results. AS-derived Tregs exhibited epigenetic and transcriptional repression of key regulatory genes, including FOXP3, IL2RA, and IRF4, accompanied by reduced IL-10 expression. DNA methylation analysis confirmed hypermethylation at FOXP3 and IRF4 loci. Tregs also showed upregulation of IGF1R and increased STAT3 signaling.
Plasma-derived exosomes from AS patients were enriched in miR-30c-5p, miR-130a-5p, and inflammatory mediators including IL-6, IL-18, and CRP. Exposure of healthy donor Tregs to AS exosomes reproduced disease-associated features, including:

- Reduced FOXP3+IRF4+ Treg frequency
- Decreased IRF4, MAF, and IL-10 expression
- Increased BHLHE40 expression
- Impaired suppressive function in co-culture assays

Mechanistically, exosomal miR-30c-5p was associated with IRF4 repression, disrupting the IRF4-MAF-IL-10 regulatory axis. In parallel, exosome-associated cytokines induced STAT3 activation, promoting Th17-like transcriptional features. These effects were amplified in the context of elevated IGF1R expression.
Reanalysis of single-cell RNA-seq data confirmed reduced IL-10 and MAF and increased BHLHE40 and STAT3 in synovial fluid Tregs, particularly within inflammation-adapted and cytotoxic-like subsets, which also expressed exhaustion markers (PD-1, TIM3, LAG3).

Conclusion. AS plasma exosomes carry coordinated miRNA and cytokine signals that reprogram Tregs through two converging mechanisms: 1) IRF4-centered transcriptional repression and 2) STAT3-driven inflammatory plasticity. These pathways induce stable epigenetic and functional impairment of Tregs and are recapitulated by exosome exposure *in vitro* and observed in patient-derived single-cell datasets.
Impact. These findings identify circulating exosomes as active mediators of immune dysregulation in AS and define molecular signatures that may serve as mechanistic biomarkers and therapeutic targets.

P62

EXPLORATORY COHORT STUDY OF ASSOCIATIONS BETWEEN JAK-STAT PATHWAY POLYMORPHISMS AND ANXIETY/DEPRESSION IN PATIENTS WITH ANKYLOSING SPONDYLITIS

Michelsen B.[1,2,3], Roberts R.[1], Stamp L.[4], Harrison A.[5], White D.[6,7], Dalbeth N.[7], Ching D.[8], Stebbings S.[1]
[1]Dunedin School of Medicine, University of Otago, Dunedin, New Zealand; [2]Diakonhjemmet Hospital, Oslo; [3]Research Unit, Sørlandet Hospital, Kristiansand, Norway; [4]Dept. of Medicine, University of Otago, Christchurch; [5]Dept. of Medicine, Wellington School of Medicine, University of Otago, Wellington; [6]Dept. of Rheumatology, Waikato Hospital, Hamilton; [7]Faculty of Medical and Health Sciences, University of Auckland, Auckland; [8]Timaru Medical Specialists Limited, Timaru, New Zealand

Introduction/Objective. Anxiety and depression are common in patients with ankylosing spondylitis (AS). Evidence suggests JAK-STAT signaling may be involved in the pathophysiology of mood disorders. The aim of this study was to explore associations between SNPs within JAK-STAT signaling pathways and anxiety/depression in AS.
Methods. We included participants from the observational New Zealand Spondyloarthritis Genetics and Environment (SAGE) study who fulfilled ASAS criteria for axSpA, had self-reported anxiety/depression on the EuroQol-5D-3L questionnaire, and had available SNP data within JAK-STAT-related inflammatory pathways. Genotype frequencies were compared between patients with and without anxiety/depression using chi-square tests and logistic regression analyses adjusted for age, sex, and ethnicity. For SNPs showing significant associations, dominant, recessive, and heterozygote models were explored. Disease activity (ASDAS) was included in secondary analyses.
Results. A total of 314 outpatients were included of which 92 (29.3%) reported moderate or extreme anxiety/depression. In patients without/with anxiety/depression mean (SD) age was 43 (13)/42 (12) years, 157 (70.7%)/59 (64.1%) were men, median (IQR) disease duration 7 (1-16)/5 (1-14) years, ASDAS 2.4 (1.6-3.1)/3.3 (2.5-4.2), and 6.8/14.1% were Asian, 11.3/13.0% European, 7.7/2.2% Māori (mixed ethnicity included), 67.6/57.6% New Zealand European, 5.4/12.0% mixed New Zealand European, and 1.4/1.1% of other ethnicities, respectively. Of the SNPs examined, only JAK2 rs10758669 (A<C) was associated with anxiety/depression (Table I), consistently in both unadjusted and adjusted analyses. Genetic model analyses identified a heterozygote effect, with AC carriers having higher odds of anxiety/depression compared with homozygotes (adjusted OR 1.90 (95%CI 1.16-3.11; Table II)). The association was consistent after additional adjustment for ethnicity (OR 2.03 (1.22-3.38)), and ASDAS (OR 1.99 (1.17, 3.38)).
Conclusion. In this exploratory AS cohort study, heterozygous carriers of JAK2 rs10758669 had increased odds of self-reported anxiety/depression. Further studies in larger cohorts are needed to confirm these findings.

Table I. Comparison of genetic markers between patients with ankylosing spondylitis with and without anxiety/depression.

	Not anxious/ depressed N=222	Moderately or extremely anxious/depressed N=92	p-value, unadjusted	p-value, adjusted for age and sex	p-value, adjusted for age, sex, and ethnicity
JAK2 rs10758669, n (%)					
AA	101 (45.5)	33 (35.9)	0.035	0.033	0.023
CC	35 (15.8)	9 (9.8)			
AC	86 (38.7)	50 (54.3)			
IL12B rs10045431, n (%)					
AA	11 (5.0)	10 (10.9)	0.067	0.089	0.096
CC	114 (51.8)	52 (56.5)			
AC	95 (43.2)	30 (32.6)			
STAT3 rs744166, n (%)					
CC	42 (19.1)	16 (17.6)	0.820	0.798	0.714
TT	73 (33.2)	28 (30.8)			
CT	105 (47.7)	47 (51.6)			
IL23R rs1343151, n (%)					
CC	107 (48.2)	56 (60.9)	0.110	0.113	0.161
TT	27 (12.2)	7 (7.6)			
CT	88 (39.6)	29 (31.5)			
IL23R rs10489630, n (%)					
GG	40 (18.0)	15 (16.3)	0.238	0.233	0.352
TT	72 (32.4)	39 (42.4)			
GT	110 (49.5)	38 (41.3)			
IL23R rs11209026, n (%)					
AA	1 (0.5)	0 (0.0)	0.741	0.958	0.884
GG	207 (93.2)	84 (92.3)			
AG	14 (6.3)	7 (7.7)			

p-value, unadjusted: Chi-Square or Fisher's exact test, as appropriate; *p*-value, adjusted; logistic regression analysis, adjusted for variables as stated above.

Table II. Association between JAK2 rs10758669 and anxiety/depression in patients with ankylosing spondylitis according to genetic model.

Genetic model	Comparison	Crude OR (95%CI), p-value N=314	Adj. for age and sex, OR (95%CI), p-value N=314	Adj. for age, sex, ethnicity OR (95%CI), p-value N=314	Secondary analysis: adj. for age, sex, ethnicity, and ASDAS, OR (95%CI) p-value, N=303
Dominant	AA vs. AC + CC	0.67 (0.41-1.11), p=0.118	0.67 (0.41-1.11), p=0.119	0.62 (0.37-1.05), p=0.074	0.69 (0.40-1.18), p=0.176
Recessive	CC vs. AA + AC	0.58 (0.27-1.26), p=0.168	0.57 (0.26-1.24), p=0.154	0.58 (0.26-1.29), p=0.179	0.48 (0.21-1.12), p=0.090
Heterozygote	AC vs. AA + CC	1.88 (1.15-3.08), p=0.012	1.90 (1.16-3.11), p=0.011	2.03 (1.22-3.38), p=0.007	1.99 (1.17-3.38), p=0.011

P63

ASSOCIATION BETWEEN PERSISTENT INFLAMMATION AND LUMBAR SYNDESMOPHYTES IN BIOLOGIC-TREATED AXIAL SPONDYLOARTHRITIS

Coskun Sagirkaya S., Akyazi Sener E., Armagan B.
Dept. of Rheumatology, Ankara Bilkent City Hospital, Ankara, Turkey

Introduction/Objective. The relationship between persistent systemic inflammation and structural damage in axial spondyloarthritis (axSpA) remains controversial, particularly in the biologic era. Whether cumulative inflammatory burden contributes to syndesmophyte formation despite biologic disease-modifying antirheumatic drug (bDMARD) treatment is unclear. This study aimed to investigate the association between persistent C-reactive protein (CRP) elevation and lumbar syndesmophyte presence in biologic-treated axSpA patients.
Materials and methods. Clinical data and lumbar radiographs of axSpA patients receiving bDMARDs who attended an adult rheumatology clinic within the previous six months were retrospectively analyzed. The proportion of follow-up days with elevated CRP was calculated by dividing the number of days with CRP levels above the upper limit of normal by the total number of follow-up days. Persistent CRP elevation, defined as elevated CRP during ≥50% of follow-up days, was considered the primary inflammatory exposure. The proportion of follow-up days with elevated CRP was also analyzed as a continuous measure of inflammatory burden. Patients were classified according to the presence or absence of lumbar syndesmophytes.
Results. Among 194 eligible patients, 184 with available lumbar radiographs were included in the analysis. Lumbar syndesmophytes were identified in 79 patients (42.9%). Persistent CRP elevation was observed in 29 patients (36.7%) with lumbar syndesmophytes and 35 patients (33.3%) without lumbar syndesmophytes (p=0.608) (Table I). Similarly, the median proportion of follow-up days with elevated CRP was numerically higher among patients with lumbar syndesmophytes than among those without syndesmophytes [48.6% vs. 44.7%; p=0.162] (Fig. 1). Patients with lumbar syndesmophytes were more frequently male and had longer disease duration.

Table I. Clinical characteristics according to lumbar syndesmophyte status.

Variable	No Lumbar Syndesmophytes (n=105)	Lumbar Syndesmophytes (n=79)	p value
Persistent CRP elevation ≥50%, n (%)	35 (33.3)	29 (36.7)	0.608
Proportion of follow-up days with elevated CRP, median (IQR)	44.7 (88.2)	48.6 (86.6)	0.162
Male sex, n (%)	52 (49.5)	63 (79.7)	<0.001
Disease duration, months, median (IQR)	132 (81)	180 (117)	<0.001

CRP: C-reactive protein; IQR: interquartile range.

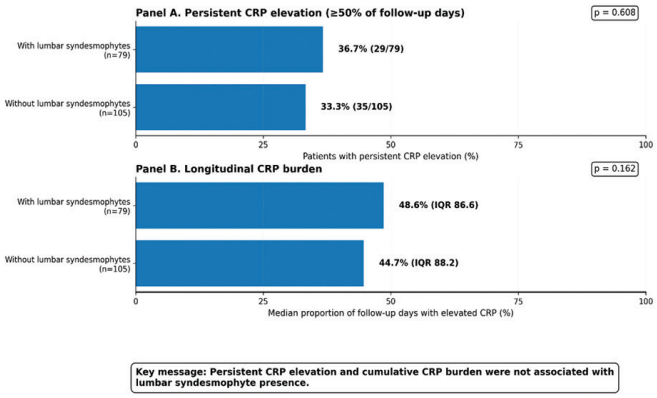


Fig. 1. Inflammatory burden according to lumbar syndesmophyte status.

Conclusion. Persistent CRP elevation and longitudinal inflammatory burden were not associated with lumbar syndesmophyte presence in biologic-treated axSpA patients. These findings suggest that mechanisms beyond measurable systemic inflammation may contribute to structural damage in the biologic era.

P64

LOW-DOSE CT, MRI OR RADIOGRAPHS: WHICH IMAGING MODALITY BEST CAPTURES SPINAL STRUCTURAL PROGRESSION IN AXIAL SPONDYLOARTHRITIS

Ayan G.[1,2], de Hooge M.[1,3], de Bruin L.[1], Bento da Silva A.[1], van Lunteren M.[1], Marques M.L.[4], Reijnierse M.[5], Landewé R.[6,7], Berg I.J.[8], Ramonda R.[9], Exarchou S.[10], van Gaalen F.[1], Ramiro S.[1,6]

[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden, The Netherlands; [2]Dept. of Rheumatology, Ankara Research and Training Hospital, Ankara, Türkiye; [3]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [4]Dept. of Rheumatology, Coimbra Local Health Unit, Coimbra, Portugal; [5]Dept. of Radiology, Leiden University Medical Center, Leiden; [6]Amsterdam University Medical Center, Amsterdam; [7]Zuyderland Medical Center, Heerlen, The Netherlands; [8]Center for Treatment of Rheumatic and Musculoskeletal Diseases (REMEDY), Diakonhjemmet Hospital, Oslo, Norway; [9]Dept. of Medicine-DIMED, University of Padova, Padova, Italy; [10]Dept. of Clinical Sciences Malmö, Lund University, Malmö, Sweden

Introduction/Objective. Spinal structural progression in axial spondyloarthritis (axSpA) is minimal on radiographs and magnetic resonance imaging (MRI), with MRI not showing superior sensitivity to change. Low-dose CT (ldCT) may better detect structural progression. We evaluated the sensitivity to change of ldCT compared with MRI and radiographs.

Methods. Patients with axSpA from the SPondyloArthritis Caught Early cohort with ldCT at two time points (TP1–TP2, 2 years apart) and ≥1 additional imaging modality were included. ldCT was scored using Computed Tomography Syndesmophyte Score (CTSS) (0–552), MRI using a modified Canada-Denmark system (0–322), and radiographs using modified stoke ankylosing spondylitis spinal score (mSASSS) (0–72), all scored in blinded chronological order. Scores were averaged across readers to derive status and change scores.

Outcomes were standardised to enable cross-modality comparison and analysed using multilevel generalised estimating equations models to estimate standardised yearly rates of change (SROC). Relative rates were calculated using mSASSS as reference, with sensitivity analyses in cases with all imaging modalities available.

Results. Sixty-seven patients were included (mean (SD) age 35 (8) years; symptom duration 6 (3) years; 57% male; 84% HLA-B27+). MRI was available in all and radiographs in 96%. Status and change scores are reported in the Table I. Both the CTSS and the number of syndesmophytes on ldCT showed the highest SROC and therefore RSROC corresponding to 2.333 and 2.722 (Table I and example in Figure 1). MRI bone spurs showed higher RSROC (2.111), fat lesions were comparable (1.055), while erosions showed a negative change. Ankylosis could not be analysed due to no progression. Sensitivity analyses in 64 (96%) patients showed comparable findings.

Conclusion. Structural progression in axSpA is minimal across modalities, but ldCT demonstrates superior sensitivity to change compared with MRI and radiographs, supporting its use for assessing structural progression in axSpA.

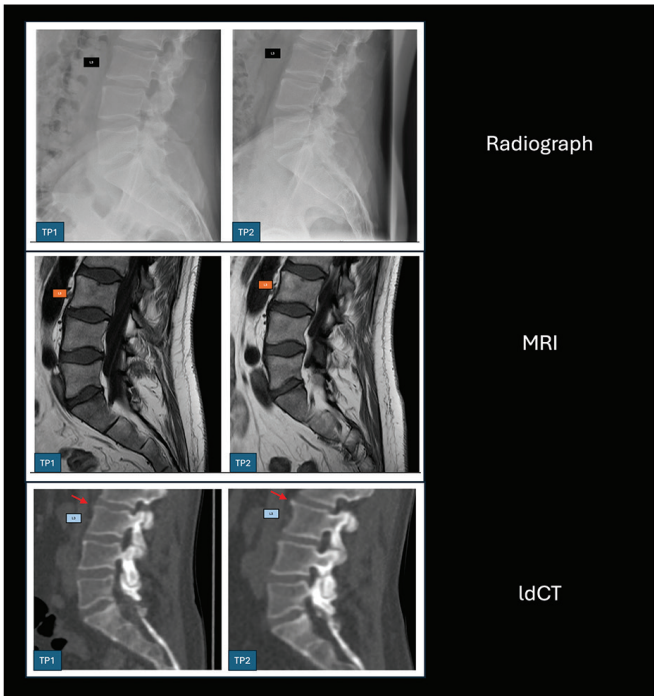


Fig. 1. Detection of a new syndesmophyte on ldCT at L3 not visible on radiographs or MRI. A new syndesmophyte at the upper anterior vertebral corner of L3 was identified on ldCT at TP2 (by all 3 readers), while undetected on either radiographs or MRI (none of the readers). L3: third lumbar vertebra; ldCT: Low-dose computed tomography; MRI: magnetic resonance imaging; TP1: time point 1; TP2: time point 2.

Table I. Status scores, change scores, standardised and relative standardised rates of structural change across imaging modalities.

		All patients (n=67)				Patients with available imaging at 2TP (n=64)		
		Baseline value*	Change scores *	Standardised rate of change per year (95% CI)	Relative standardised rate of change	% of patients with a change score>0	Standardised rate of change per year (95% CI)	Relative standardised rate of change
ldCT	CTSS (0-552) Number of syndesmophytes	9.6 (18.5) 5.1 (8.1)	1.6 (4.5) 0.9 (2.1)	0.042 (0.02;0.07) 0.049 (0.02;0.08)	2.3 2.7	34 (53.1) 31 (48.4)	0.043 (0.02;0.07) 0.052 (0.02; 0.08)	2.4 2.9
Radiographs	mSASSS (0-72) Number of syndesmophytes	0.78 (1.7) 0.27 (0.7)	0.06 (0.6) 0.03 (0.3)	0.018 (-0.020;0.057) 0.024 (-0.019;0.068)	1 (reference) 1.33	7 (10.4) 4 (6.3)	0.018 (-0.020;0.057) 0.024 (-0.019;0.068)	1(reference) 1.3
MRI	Erosions (0-92) Fat lesions (0-92) Bone spurs (corresponding to syndesmophytes) (0-92) Ankylosis (0-46) Number of structural lesions (0-322)	0.16 (0.49) 1.3 (3) 0.04 (0.3) 0 1.5 (3.3)	-0.04 (0.2) 0.1 (0.8) 0.03 (0.2) NA 0.1 (0.8)	-0.038 (-0.09;0.01) 0.019 (-0.01;0.05) 0.039 (-0.01:0.09) NA 0.015 (-0.01;0.04)	-2.1 1.1 2.1 NA 0.8	1 (1.6) 13 (20.3) 1 (1.6) 0 12 (18.8)	-0.021 (-0.06;0.017) 0.015 (-0.01-0.04) 0.047 (-0.04-0.14) NA 0.013 (-0.01-0.04)	-1.2 0.8 2.6 NA 0.7

* All scores are given in mean (SD)

ldCT; low-dose computed tomography; CTSS; CT Syndesmophyte score; MRI; magnetic resonance imaging; CI: confidence interval; mSASSS: modified stoke ankylosing spondylitis spinal score; TP; Time point; NA; not available

P65

EVALUATION OF SACROILIAC RADIOGRAPHIC PROGRESSION IN PATIENTS WITH SPONDYLOARTHRITIS UNDERGOING BIOLOGICAL THERAPY USING MNY CRITERIA AND PHYSICIAN DECISION

Kalyoncu U., Ayan G., Kiliç L.
Dept. of Internal Medicine, Division of Rheumatology, Hacettepe University, Ankara, Turkey

Objective. We aimed to investigate long-term sacroiliac radiographic changes in patients with Spondyloarthritis (SpA) followed in a real-world bDMARD registry using mNY criteria and physician assessment.
Methods. The HUR-BIO registry was updated in 2025 (version 3), with repeated clinical and radiographic evaluations. Among the first 500 SpA patients, 174 had at least two pelvic radiographs and 157 had an interval of ≥2 years between scans and were included. Pelvic radiographs were independently evaluated by three readers according to mNY criteria and additionally classified as "no change," "progression," or "regression" based on physician judgement. Progression was defined as an increase of ≥1 in mNY score or physician-defined progression. Final decisions were based on majority agreement among readers. Demographic and clinical characteristics were compared between patients with and without progression.
Results. A total of 157 patients (52% male) were analysed. Mean (SD) age at first radiograph was 37.5 (11) years and mean disease duration was 3.2 (5.7) years. At baseline, 116/157 (74%) patients fulfilled mNY criteria. During follow-up, progression was detected in 35% (n=41) of baseline mNY-positive patients, while 44% (n=18) of baseline mNY-negative patients demonstrated progression. Regression from mNY-positive to mNY-negative status was observed in 3% of patients, although only 1.5% were considered regression by physician assessment. Patients with progression had earlier symptom onset (26±9 vs. 31.5±10 years, p=0.003), younger age at diagnosis (31±10 vs. 36±11 years, p=0.004), and higher HLA-B27 positivity (67.5% vs. 46.2%, p=0.03). Male sex was more frequent among progressors (61% vs. 46%). Time between radiographs was longer in progressing patients (7.6±4.0 vs. 6.2±3.8 years, p=0.02).
Conclusion. Radiographic progression persisted in approximately one-third of mNY-positive patients despite long-term bDMARD follow-up. Nearly half of baseline mNY-negative patients also developed progression. Earlier disease onset and HLA-B27 positivity were associated with radiographic progression, while radiographic regression was uncommon.

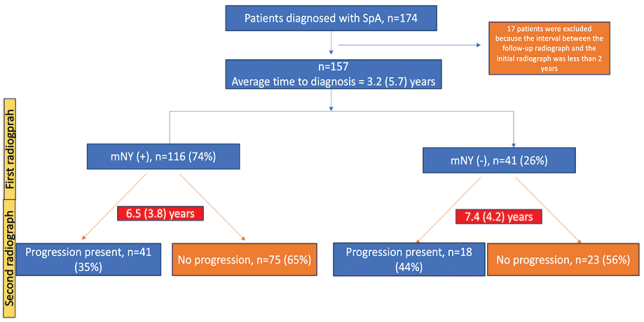


Fig. 1. Progression rates in initial and follow-up radiographs according to the mNY criterion and physician assessment.

Table I. Demographic and clinical characteristics of patients according to radiographic change category.

	All patients n=157	Progression present (n=59)	No progression (n=98)	p-value
Male gender, n (%)	81 (51.6)	36 (61)	45 (46)	0.07
Age at first radiograph release, (years) mean (SD)	37.5 (11)	35.5 (10.7)	38.7 (11)	0.07
Age at symptom onset, (years) mean (SD)	29.5 (10)	26 (9)	31.5 (10)	**0.003**
Age at diagnosis (years) mean (SD)	34 (11)	31 (10)	36 (11)	**0.004**
Interval between radiographs, (years) mean (SD)	6.7 (3.9)	7.6 (4)	6.2 (3.8)	**0.02**
HLA-B27, n (%)	54.2	67.5	46.2	**0.03**
Education, secondary school and above, n (%)	106 (67.5)	40 (68)	66 (70)	0.95
Smoking (at any time), n (%)	93 (59.2)	39 (66)	54 (55)	0.17
Family history of SpA, n (%)	47 (30)	19 (32.8)	28 (28.6)	0.58
Body mass index, mean (SD)	27.8 (5.2)	26.5 (4.5)	28.7 (5.5)	**0.01**
Psoriasis presence*, n (%)	13 (8.3)	5 (8.5)	8 (8.2)	0.94
Presence of inflammatory bowel disease*, n (%)	8 (5)	5 (8.5)	3 (3)	0.13
Presence of enthesitis according to LEI*, n (%)	31 (27.7)	10 (23.2)	21 (30.4)	0.41
Presence of peripheral arthritis*, n (%)	31 (38.8)	12 (34.3)	19 (42.2)	0.47
FMF presence, n (%)	13 (8.3)	5 (8.5)	8 (8.2)	0.95
Presence of dysplasia*, n (%)	7 (4.9)	2 (4)	5 (6)	0.66
Presence of uveitis*, n (%)	18 (11.5)	7 (11.9)	11 (11.2)	0.90
Elevated CRP*^, n (%)	101 (94.4)	41 (97.6)	60 (92.3)	0.22
Final BASDAI, mean (SD)	36.5 (24.4)	33.8 (24.1)	38.2 (24.5)	0.23
Final BASFI, mean (SD)	30.9 (27)	29.4 (26.6)	31.9 (27.3)	0.68
bDMARD change*, n (%)	66 (45)	28 (50)	38 (42)	0.32
Diabetes mellitus, n (%)	11 (7)	1 (1.6)	10 (10)	0.06
Hypertension*, n (%)	35 (22.4)	11 (18)	24 (25)	0.27
Hyperlipidaemia*, n (%)	32 (21.1)	13 (23)	19 (20)	0.93

*Evaluated as presence at any time, ^Elevated CRP was defined as 5 mg/l or higher
LEI: Leeds Enthesitis Index; CRP: C-reactive protein; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index; bDMARD: biologic disease-modifying antirheumatic drug; FMF: Familial Mediterranean Fever; SpA: spondyloarthritis; SD: standard deviation

P66

A COMBINED SCORE OF SYNDESMOPHYTES AND FACET JOINT ANKYLOSIS IMPROVES SENSITIVITY TO SPINAL STRUCTURAL DAMAGE PROGRESSION IN AXIAL SPONDYLOARTHRITIS

Bento da Silva A.[1], van Gaalen F.[1], Landewé R.[2,3], de Hooge M.[1,4], Marques M.L.[1,5], van Lunteren M.[1], de Bruin L.[1], Ayan G.[1,6], Baraliakos X.[7,8], Reijnierse M.[9], Braun J.[8,10], van der Heijde D.[1], Ramiro S.[1,3]
[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden; [2]Dept. of Rheumatology, Amsterdam University Medical Center, Amsterdam; [3]Dept. of Rheumatology, Zuyderland Medical Center, Heerlen, The Netherlands; [4]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [5]Dept. of Rheumatology, Coimbra Local Health Unit, Coimbra, Portugal; [6]Dept. of Rheumatology, Ankara Research and Training Hospital, Ankara, Turkey; [7]Rheumazentrum Ruhrgebiet Herne; [8]Ruhr-University, Bochum, Germany; [9]Dept. of Radiology, Leiden University Medical Center, Leiden, The Netherlands; [10]Rheumatologisches Versorgungszentrum (RVZ) Steglitz, Berlin, Germany

Introduction/Objective. To investigate whether a score combining syndesmophytes and facet joint ankylosis (FJA) is more sensitive than the computed tomography (CT) syndesmophyte score (CTSS) for assessing spinal structural damage progression in axial spondyloarthritis (axSpA).
Methods. Data from patients with axSpA participating in the Sensitive Imaging in Ankylosing Spondylitis cohort were used. Baseline and 2-year (2Y) spinal radiography and low-dose CT (ldCT) were scored by 3 (radiography) or 2 (ldCT) readers. Syndesmophytes were assessed by the mSASSS (radiography) and CTSS (ldCT), and FJA by the facet ankylosis score (FAS) on ldCT (Fig. 1). Three versions of the CT facet ankylosis syndesmophyte spine score (CTFASSS) were computed by adding the CTSS and FAS while giving equal weight to both components (CTFASSS-A), 50% (CTFASSS-B)

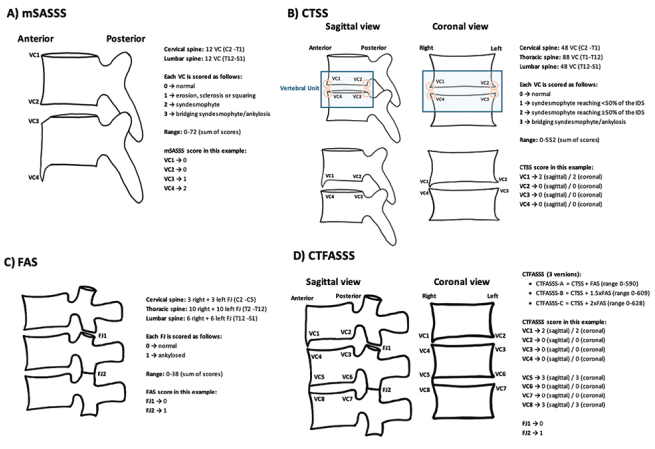


Fig. 1. Schematic representation to guide the calculation of the mSASSS (**A**), CTSS (**B**), FAS (**C**) and CTFASSS (**D**).
CTFASSS: Computed Tomography Facet Ankylosis Syndesmophyte Spine Score; CTSS: Computed Tomography Syndesmophyte Score; FJ: facet joint; FAS: Facet Ankylosis Score; IDS: intervertebral disc space; mSASSS: modified Stoke Ankylosing Spondylitis Spine Score; VC: vertebral corner.

Table I. Number of patients with axSpA with spinal structural damage progression on low-dose computed tomography (n=48).

A. According to change in spinal structural damage scores

	mSASSS*	CTSS	FAS	CTFASSS-A (CTSS + FAS)	CTFASSS-B (CTSS + 1.5 x FAS)	CTFASSS-C (CTSS + 2 x FAS)
Change >0, n (%)						
Positive	24 (65)	43 (90)	27 (56)	47 (98)	47 (98)	47 (98)
Negative	7 (20)	3 (6.3)	4 (8.3)	1 (2.1)	1 (2.1)	1 (2.1)
Net progression[$]	17 (46)	40 (83)	23 (48)	46 (96)	46 (96)	46 (96)
Change >0.5, n (%)						
Positive	22 (59)	43 (90)	23 (48)	45 (94)	47 (98)	47 (98)
Negative	6 (16)	3 (6.3)	2 (4.2)	1 (2.1)	1 (2.1)	1 (2.1)
Net progression[$]	16 (43)	40 (83)	21 (44)	44 (92)	46 (96)	46 (96)

B. According to syndesmophyte and facet joint ankylosis development

	Total number of new lesions[&]	Mean (SD)	Range	≥1, n(%)[#]	≥2, n(%)[#]	≥3, n(%)[#]
New syndesmophytes	327	6.8 (7.0)	0-33	35 (73)	31 (65)	22 (46)
Growing syndesmophytes	145	3.0 (2.5)	0-8	25 (52)	19 (40)	14 (29)
New or growing syndesmophytes	472	9.8 (8.3)	0-37	41 (85)	36 (75)	28 (58)
New facet joint ankylosis	73	1.5 (2.0)	0-8	11 (23)	10 (21)	6 (13)
New/growing syndesmophytes and FJA	545	11.3 (9.0)	0-42	41 (85)	38 (79)	33 (69)

*Retrieved from. de Koning A et al. *Ann Rheum Dis* 2018;77:293-299 (n=37).
[$]Net progression: number of patients with positive progression minus number of patients with negative progression.
[&]Total number of new lesions (out of 48 patients) is the average across both readers.
[#]Agreement between both readers was required on the presence of ≥1, ≥2 and 13 lesions.
axSpA: axial spondyloarthritis; CTFASSS: Computed Tomography Facet Ankylosis Syndesmophyte Spine Score; CTSS: Computed Tomography Syndesmophyte Score, FAS: Facvet Ankylosis Score; mSASSS: modified Stoke Ankylosis Spondylitis Spine Score.

or 100% (CTFASSS-C) more weight to FAS. Change scores were calculated per reader and then averaged. The number of patients with positive, negative and net change >0 and >0.5 were calculated for each score. The total number of new or growing syndesmophytes and new FJA were determined.
Results. Forty-eight patients were included: mean (SD) age 49 (9.7) years, 85% male. Between baseline and 2Y, progression of spinal damage was observed: mean (SD) mSASSS 18.3 (14.3) *vs*. 20.4 (15.1), CTSS 161.6 (127.1) *vs*. 179.5 (132.3), FAS 8.2 (9.8) *vs*. 9.4 (10.2). CTFASSS-A changed from 169.8 (134.5) at baseline to 188.9 (140.0) at 2Y. After 2Y, all CTFASSS detected any net change in a higher percentage (96%) of patients compared to the remaining scores (*e.g*. 83% with CTSS) (Table IA). Moreover, all CTFASSS captured 1.15 times more new bone formation than CTSS (545 *vs*. 472 new lesions) (Table IB).
Conclusion. CTFASSS captures more spinal damage progression in axSpA than assessing syndesmophytes alone. All CTFASSS versions showed similar sensitivity to change, with CTFASSS-A being the most straightforward approach.
Acknowledgements. *FCT* (grant 2024.01498.BD).

P67

A COMBINED SCORE OF FACET JOINT ANKYLOSIS AND SYNDESMOPHYTE DEVELOPMENT CORRELATES WELL WITH SPINAL MOBILITY IN AXIAL SPONDYLOARTHRITIS

Bento da Silva A.[1], van Gaalen F.[1], Landewé R.[2,3], de Hooge M.[1,4], Marques M.L.[1,5], van Lunteren M.[1], de Bruin L.[1], Ayan G.[1,6], Baraliakos X.[7,8], Reijnierse M.[9], Braun J.[8,10], van der Heijde D.[1], Ramiro S.[1,3]
[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden; [2]Dept. of Rheumatology, Amsterdam University Medical Center, Amsterdam; [3]Dept. of Rheumatology, Zuyderland Medical Center, Heerlen, The Netherlands; [4]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [5]Dept. of Rheumatology, Coimbra Local Health Unit, Coimbra, Portugal; [6]Dept. of Rheumatology, Ankara Research and Training Hospital, Ankara, Turkey; [7]Rheumazentrum Ruhrgebiet Herne; [8]Ruhr-University, Bochum, Germany; [9]Dept. of Radiology, Leiden University Medical Center, Leiden, The Netherlands; [10]Rheumatologisches Versorgungszentrum (RVZ) Steglitz, Berlin, Germany

Introduction/Objective. To assess whether a combined score of syndesmophyte and facet joint ankylosis (FJA) retains good correlation with spinal mobility in patients with axial spondyloarthritis (axSpA) compared to current measurement instruments.
Methods. Data from patients with axSpA included in the Sensitive Imaging in Ankylosing Spondylitis cohort were used. Spinal radiography and low-dose computed tomography (ldCT) were performed at baseline and scored by 3 (radiography) or 2 (ldCT) readers. Syndesmophytes were assessed by the mSASSS on radiography and the CT syndesmophyte score (CTSS) on ldCT, and FJA by the facet ankylosis score (FAS) on ldCT. Three versions

Table I. Spearman's correlation between spinal structural damage scores and spinal mobility (at baseline, n=46)*

	mSASSS r_s	CTSS r_s	FAS r_s	CTFASSS-A (CTSS + FAS) r_s	CTFASSS-B (CTSS + 1.5 x FAS) r_s	CTFASSS-C (CTSS + 2 x FAS) r_s
Spinal mobility						
Occiput-to-wall distance	0.50	0.64	0.64	0.65	0.66	0.66
Lateral spinal flexion	-0.61	-0.72	-0.53	-0.72	-0.72	-0.73
Modified Schober test	-0.41	-0.50	-0.40	-0.50	-0.50	-0.50
Cervical rotation	-0.49	-0.58	-0.62	-0.58	-0.59	-0.59
Chest expansion	-0.32	-0.49	-0.31	-0.49	-0.49	-0.48
Composite measure						
BASMI	0.60	0.71	0.65	0.71	0.72	0.72

* Aggregate scores (average across readers) were used. Higher r_s values mean higher strength of the association; the following cutoffs were applied (Castro MP *et al*, *Clin Rheumatol* 2016;35(7):1777-87): Low: 0.00-0.20; Fair: 0.21-0.40; Moderate: 0.41-0.60; Substantial: 0.61-0.80; Excellent: 0.81-1.00.
BASMI: Bath Ankylosing Spondylitis Metrology Index; **CTFASSS:** Computed Tomography Facet Ankylosis Syndesmophyte Spine Score; **CTSS:** Computed Tomography Syndesmophyte Score; **FAS:** Facet Ankylosis Score; **mSASSS:** modified Stoke Ankylosis Spondylitis Spine Score; r_s: Spearman's rho.

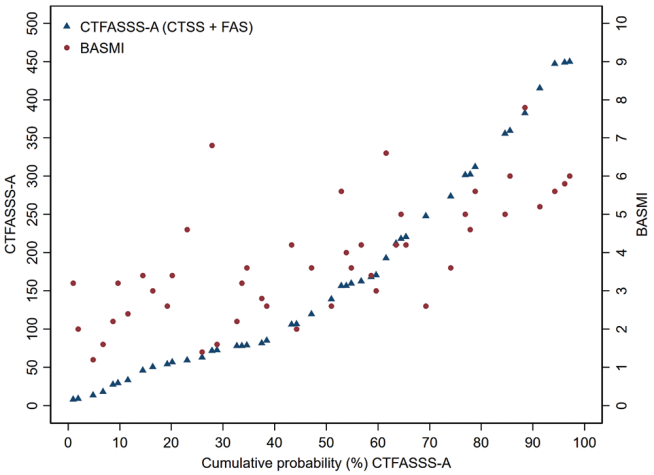


Fig. 1. Cumulative probability plot representing the relationship between BASMI and CTFASSS-A.
BASMI: Bath Ankylosing Spondylitis Metrology Index; CTFASSS-A: Computed Tomography Facet Ankylosis Syndesmophyte Spine Score version A; CTSS: Computed Tomography Syndesmophyte Score; FAS: Facet Ankylosis Score.

of the CT facet ankylosis syndesmophyte spine score (CTFASSS) were computed by adding the CTSS and FAS while giving equal weight to both components (CTFASSS-A), 50% (CTFASSS-B) or 100% (CTFASSS-C) more weight to FAS. Spinal mobility was evaluated by: occiput-to-wall distance (OWD), lateral spinal flexion (LSF), modified Schober test, cervical rotation, and chest expansion. The Bath Ankylosing Spondylitis Metrology Index (BASMI) was calculated. Correlations between spinal structural damage and mobility were assessed with Spearman's rank correlation.
Results. Forty-six patients were included: mean (SD) age 47 (9.6) years, 87% male. All CTFASSS versions showed substantial correlations with OWD (r_s 0.65; 0.66) and LSF (-0.72; -0.73), and moderate correlations with the other measures, similarly to CTSS, but better than mSASSS (Table I). Compared to mSASSS, FAS correlated better with OWD (0.64 *vs* 0.50) and cervical rotation (-0.62 *vs* -0.49). However, no additional benefit was obtained with a CTFASSS attributing more weight to FAS compared to CTFASSS-A. Although substantial correlations were observed between all scores and BASMI, CTSS and CTFASSS showed better results (Fig. 1).
Conclusion. CTFASSS correlates with spinal mobility similarly well as CTSS but better than mSASSS, supporting the validity of the combined score in assessing spinal damage progression in axSpA.
Acknowledgements. *FCT* (grant 2024.01498.BD).

P68

STRUCTURAL DAMAGE OF THE SPINE HAS A MODEST EFFECT ON SPINAL MOBILITY OVER 2 YEARS IN AXIAL SPONDYLOARTHRITIS

Bento da Silva A.[1], Webers C.[2], de Hooge M.[1,3], Ayan G.[1,4], Marques M.L.[1,5], de Bruin L.[1], van Lunteren M.[1], Landewé R.[6,7], Reijnierse M.[8], Baraliakos X.[9,10], Braun J.[10,11], van der Heijde D.[1], van Gaalen F.[1], Ramiro S.[1,7]

[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden; [2]Dept. of Epidemiology, Maastricht University, Maastricht, The Netherlands; [3]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [4]Dept. of Rheumatology, Ankara Research and Training Hospital, Ankara, Turkey; [5]Dept. of Rheumatology, Coimbra Local Health Unit, Coimbra, Portugal; [6]Dept. of Rheumatology, Amsterdam University Medical Center, Amsterdam; [7]Dept. of Rheumatology, Zuyderland Medical Center, Heerlen; [8]Dept. of Radiology, Leiden University Medical Center, Leiden, The Netherlands; [9]Rheumazentrum Ruhrgebiet Herne; [10]Ruhr-University, Bochum, Germany; [11]Rheumatologisches Versorgungszentrum (RVZ) Steglitz, Berlin, Germany

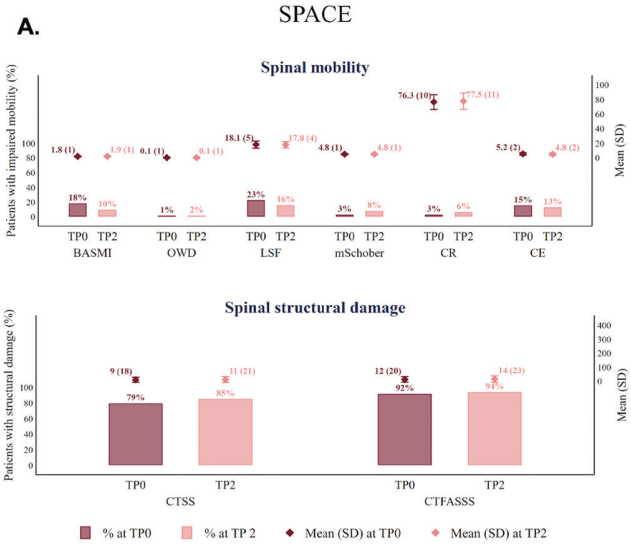
Introduction. To assess the relationship between spinal structural damage and spinal mobility over 2 years (2Y) in axSpA.

Methods. Patients with axSpA from the Sensitive Imaging in Ankylosing Spondylitis (SIAS) and SPondyloArthritis Caught Early (SPACE) cohorts were included. Spinal structural damage was assessed on low-dose CT at 2 time points with a 2Y interval (TP0/TP2) with the CT Syndesmophyte Score (CTSS) and CT Facet Ankylosis Syndesmophyte Spine Score (CTFASSS) by 2-3 readers. Spinal mobility was evaluated using occiput-to-wall distance (OWD), lateral spinal flexion (LSF), modified Schober test, cervical rotation, chest expansion and BASMI. Confounder-adjusted standard and time-lagged autoregressive generalized estimating equation (GEE) models were used to investigate the relationship between structural damage and spinal mobility over 2Y, separately in each cohort. To compare the effect across measures, analyses were repeated using standardized variables.

Results. Sixty patients from SIAS (mean age 48 [SD 10] years, 85% males) and 73 from SPACE (age 35 [8] years, 59% males) were included. At TP0, in SIAS, all patients had spinal structural damage by design and 87% had ³1 impaired mobility measure (Fig. 1). In SPACE, impairments were rare. In SIAS, although weak, associations were observed between structural damage and all mobility measures except cervical rotation over 2Y (*e.g.* 10-unit increase in CTSS associated with 0.04-unit increase in BASMI) (Fig. 2, standard-GEE). Stronger associations were seen with LSF and OWD (standardized b: LSF -0.46, OWD 0.45, other measures -0.32 to 0.40). However, prior damage was not associated with a decrease in mobility after 2Y, except for OWD (autoregressive-GEE). In SPACE, only weak associations with chest expansion were observed.

Conclusion. In advanced axSpA, but not in patients with little prior damage, structural damage has modest effects on spinal mobility over 2Y, particularly thoracic damage, as reflected by stronger associations with LSF and OWD.

Acknowledgements. *FCT* (2024.01498.BD).



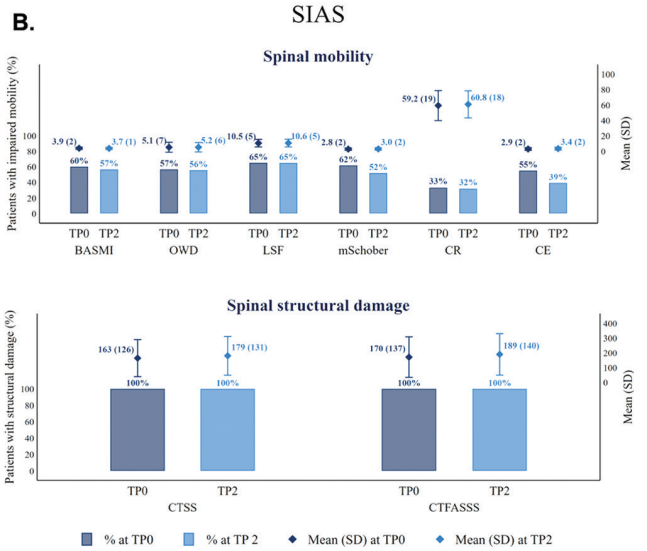


Fig. 1. Descriptive overview of spinal mobility and spinal structural damage of included patients from SPACE (**A**) and SIAS (**B**).
Definition of impared spinal mobility: <2.5th percentile (>97.5th for BASMI; <0cm for occiput-to-wall distance) based on age-specific percentile curves derived from healthy individuals (Ramiro S *et al*. ARD 2015;74(6). Definition of structuraldamage:CTSS (orCTFASSS) <0 units; average scores across allreaders were used.
BASMI: Bath Ankylosing Spondylitis Metrology Index; CE: chest expansion; CTFASSS: Computed Tomography Facet Ankylosing Syndesmophyte Spine Score; CTSS: Computed Tomography Syndesmophyte Score; LSF: lateral spinal flexion; mSchober: modofied Schober test; OWD: occiput-to-wall distance; SD: standard deviation, SIAS: Sensitive Imaging in Ankylosing Spondylitis; SPACE: SpondyloArthritis Caught Early; TP: time point.

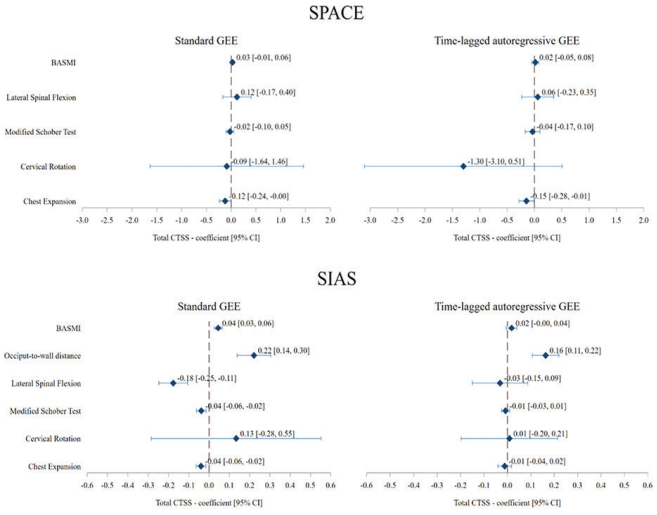


Fig. 2. Association between structural damage of the whole spine and spinal mobility over 2 years in axSpA.
Coefficients (95% CI) frepresent change in spinal mobility per 10-unit increase in CTSS: For BASMI and occiput-to-wall distance, a positive coefficient indicate more impairment, white for the other measures this is refected by a negative coefficient. Models adjusted foe age, sex,disease activity (ADAS), spinal inflammation on MRI-spine (SPARCC score), HLA-B27 positivity, NSAIDs use, and bDMARDs used (time-lagged as appropriate for the time-lagged model), as well as time-lagged outcome from previous time point (time-lagged autoregressive model only). In SPACE, as there were no impairments in occiput-to-wall distance, analyses could not be conducted for this measure.
ASDAS: Axial Spondyloarthritis Diseases Activity Score; BASMI: Bath Ankylosing Spondylitis Metrology Index; bDMARDs: biologic disease-modifying antirheumatic drugs; CI: confidence interval; CTSS: Computed Tomography Syndesmophyte Score; GEE: generalized estimating equations; HLA-B27: Human Leukocytes Antigen B27; MRI spine: magnetic resonance imaging of the spine; NSAIDs: non-steroidal anti-inflammatory drugs; SIAS: Sensitive Imaging in Ankylosing Spondylitis; SPACE: SpondyloArthritis Caught Early; SPARCC: Spondyloarthritis Research Consortium of Canada.

P69

STRUCTURAL LESIONS OF THE SACROILIAC JOINTS IN EARLY AXSPA VERSUS NON-AXSPA CHRONIC BACK PAIN: 2-YEAR RESULTS FROM THE SPACE COHORT

Cinakli H.[1,2], de Bruin L.[1], Ayan G.[1,3], Bento da Silva A.[1], de Hooge M.[1,4], van Lunteren M.[1], Reijnierse M.[5], van de Sande M.[6], Berg I.J.[7], Ramonda R.[8], Exarchou S.[9], van Gaalen F.[1], Ramiro S.[1,10]

[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden, The Netherlands; [2]Dept. of Rheumatology, Manisa City Hospital, Manisa; [3]Dept. of Rheumatology, Ankara Research and Training Hospital, Ankara, Turkey; [4]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [5]Dept. of Radiology, Leiden University Medical Center, Leiden; [6]Dept. of Rheumatology and Clinical Immunology, Amsterdam University Medical Center, Amsterdam, The Netherlands; [7]Center for Treatment of Rheumatic and Musculoskeletal Diseases (REMEDY), Diakonhjemmet Hospital, Oslo, Norway; [8]Rheumatology Unit, Dept. of Medicine-DIMED, University of Padova, Padova, Italy; [9]Dept. of Clinical Sciences Malmö, Lund University, Malmö, Sweden; [10]Dept. of Rheumatology, Zuyderland Medical Center, Heerlen, The Netherlands

Introduction. Imaging findings play an important role in diagnosing axial spondyloarthritis (axSpA) early. However, the presence and evolution of sacroiliac joint (SIJ) structural lesions in early axSpA versus non-axSpA chronic back pain (CBP) remain unclear. We compared SIJ structural lesions on radiography and magnetic resonance imaging (MRI) at baseline (BL) and over 2 years (2Y) between early axSpA and non-axSpA CBP.

Methods. Patients with axSpA or non-axSpA CBP from the SPACE cohort with available radiography or MRI of the SIJ at BL and 2Y were included.

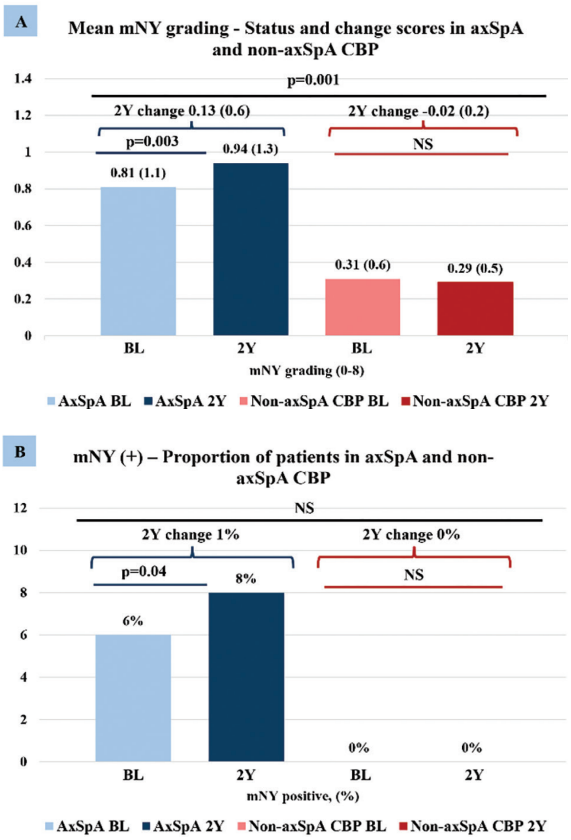


Fig. 1. A. Status and change scores of modified New York (mNY) grading in patients with axSpA and with non-axSpA chronic back pain (CBP).
B. Patients fulfilling mNY criteria (mNY (+)) in axSpA and non-axSpA CBP.
The lines between the bars represent *p*-values for changes from BL to 2Y within the axSpA and non-axSpA groups. Above these, the mean (SD) 2Y change scores are presented and connected by curly brackets, and the line above indicates the *p*-value for the difference in change between the two groups. Note: change scores do not necessarily reflect the difference between BL and 2Y status scores, as change scores are calculated as the mean of the change scores of the three individual readers for continuous variables, and by agreement of ≥2/3 readers for dichotomous variables. A *p*-value <0.05 was considered statistically significant. AxSpA; axial spondyloarthritis; BL, baseline; CBP, chronic back pain; mNY, modified New York criteria; mNY(+), positive mNY; NS, non-significant; 2Y, 2 years.

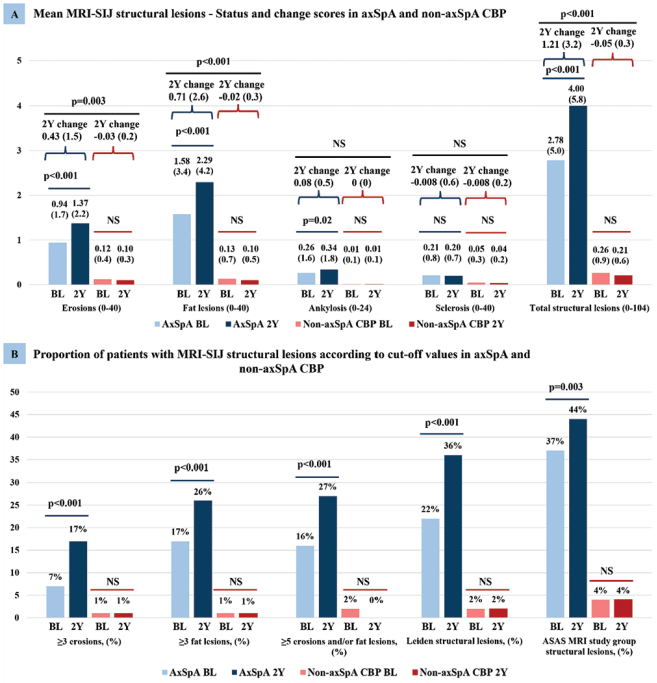


Fig. 2. A. Status and change scores of structural lesions on magnetic resonance imaging of the sacroiliac joints (MRI-SIJ) in axSpA and non-axSpA chronic back pain (CBP).
B. Different cut-off values of MRI-SIJ structural lesions in axSpA and non-axSpA CBP.
The lines between the bars represent *p*-values for changes from BL to 2Y within the axSpA and non-axSpA groups.
Panel A: above these lines, the mean (SD) 2Y change scores are presented and connected by curly brackets, and the line above indicates the p-value for the difference in change between the two groups. The Y-axis represents the mean MRI-SIJ structural lesions status score. Total structural lesions were calculated as the sum of erosions, fat lesions and ankylosis.
Panel B: Leiden structural lesions are defined by having ≥3 erosions or ≥3 fat lesions or ≥5 erosions and/or fat lesions (each individual lesion being defined as present on 2 consecutive slices). ASAS-MRI study group structural lesions are defined as the presence of erosion in ≥3 SIJ quadrants, or in ≥2 consecutive SIJ slices, or the presence of fat lesion in ≥5 SIJ quadrants, or in ≥3 consecutive SIJ slices. Note: Change scores do not necessarily reflect the difference between BL and 2Y status scores, as change scores are calculated as the mean of the change scores of the three individual readers. A *p*-value <0.05 was considered statistically significant. AxSpA, axial spondyloarthritis; BL, baseline; CBP, chronic back pain; MRI, magnetic resonance imaging; NS, non-significant; SIJ, sacroiliac joints. 2Y, 2 years.

SIJ structural lesions were assessed by three central readers, and averages (continuous scores) and ≥2/3 reader agreements (dichotomous scores) of their totals and change scores were calculated. Generalized estimating equation (GEE) models were used to evaluate 2Y progression, stratified for diagnosis (if timeXdiagnosis interaction *p*-value <0.15) and were adjusted for confounders and readers.

Results. Radiography and MRI data were available for 366 (68% axSpA) and 363 patients (69% axSpA), respectively, with a mean age of 30 (SD 8) years, and 45% were male. On radiographs, modified New York (mNY) grading was higher in axSpA than non-axSpA at both timepoints. In non-axSpA, no changes were observed on any modality. In axSpA, minimal progression of the mNY grading was detected with radiography (mean [SD] mNY change: 0.13 [0.6]; Fig. 1A), and on MRI, significant increases were observed over 2Y for erosions, fat lesions, ankylosis, and total structural lesions (Fig. 2A-B). Adjusted (GEE) progression rates in axSpA were 0.24 units/year for erosions, 0.40 for fat lesions, and 0.68 for total structural lesions, with minimal ankylosis progression and unchanged sclerosis.

Conclusion. Over 2Y, in axSpA, SIJ structural progression was minimal on radiographs, but MRI showed significant increases in erosions, fat lesions and total structural lesions. No progression was shown in non-axSpA on either radiographs or MRI.

P70

RISK FACTORS FOR PROGRESSION OF SACROILIAC JOINT STRUCTURAL LESIONS ON MRI IN EARLY AXIAL SPONDYLOARTHRITIS: 2-YEAR RESULTS FROM THE SPACE COHORT

Cinakli H.[1,2], de Bruin L.[1], Ayan G.[1,3], Bento da Silva A.[1], de Hooge M.[1,4], van Lunteren M.[1], Reijnierse M.[5], van de Sande M.[6], Berg I.J.[7], Ramonda R.[8], Exarchou S.[9], van Gaalen F.[1], Ramiro S.[1,10]

[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden, The Netherlands; [2]Dept. of Rheumatology, Manisa City Hospital, Manisa; [3]Dept. of Rheumatology, Ankara Research and Training Hospital, Ankara, Turkey; [4]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [5]Dept. of Radiology, Leiden University Medical Center, Leiden; [6]Dept. of Rheumatology and Clinical Immunology, Amsterdam University Medical Center, Amsterdam, The Netherlands; [7]Center for Treatment of Rheumatic and Musculoskeletal Diseases (REMEDY), Diakonhjemmet Hospital, Oslo, Norway; [8]Rheumatology Unit, Dept. of Medicine-DIMED, University of Padova, Padova, Italy; [9]Dept. of Clinical Sciences Malmö, Lund University, Malmö, Sweden; [10]Dept. of Rheumatology, Zuyderland Medical Center, Heerlen, The Netherlands

Introduction. Structural lesions of the sacroiliac joints (SIJ) may be present in early axial spondyloarthritis (axSpA), but data on risk factors are limited. We assessed SIJ structural lesion progression on radiographs and magnetic resonance imaging (MRI) according to potential risk factors.

Methods. Patients with axSpA from the SPACE cohort with baseline (BL) and 2-year (2Y) imaging of the SIJ were included. Central readers assessed SIJ structural and active lesions on radiographs and MRI blinded. BL and 2Y change scores were calculated (average across readers) per potential risk factor. Generalized estimating equation (GEE) calculated progression, with time as the main independent variable, adjusting for age, sex, NSAID-use, and bDMARD-use, and interactions with time per potential risk factors were investigated (Table I); if relevant, models were stratified for the risk factor.

Results. Data of 249 patients with radiography and 251 with MRI were used (mean age 30 (SD 8) years; 54% males for both subgroups). On MRI, mean (SD) 2Y-change in total structural lesions was 1.21 (3.2), with higher progression seen in males, and mNY+, ASAS-MRI-SIJ+, or HLA-B27+ patients (Table I). GEE models showed statistically significant interactions with time in erosion progression for the risk factors mNY (mNY+ 0.58; mNY- 0.21 units/year), ASAS-MRI-SIJ (ASAS-MRI-SIJ+ 0.34; ASAS-MRI-SIJ- 0.06 units/year), and HLA-B27 (HLA-B27+ 0.32; HLA-B27- 0.01 units/year). In the change of fat lesions, this was observed for ASAS-MRI-SIJ (ASAS-MRI-SIJ+ 0.92; ASAS-MRI-SIJ- -0.10 units/year). In progression of total structural lesions, interactions with time were observed for ASAS MRI-SIJ and HLA-B27 (Fig. 1).

Conclusion. Over 2Y, progression was observed in erosions, fat lesions, and total structural lesions on MRI. ASAS-MRI-SIJ+ showed the strongest association with progression across lesion types. Progression in erosions and total structural lesions was more pronounced in HLA-B27+ patients, highlighting the prognostic value of combining imaging and genetic/clinical markers in early axSpA.

Table I. Baseline and 2-year change score of MRI-SIJ structural lesions according to the presence of potential risk factors in patients with early axSpA.

Risk factors	Number (%) of patients (BL)	Erosion scores (BL)	Change scores of erosions (BL-2Y)	Fat lesions scores (BL)	Change scores of fat lesions (BL-2Y)	Total structural lesions (BL)	Change scores of total structural lesions (BL-2Y)
All patients	251 (100)	0.9 (1.7)	0.43 (1.5)	1.6 (3.4)	0.71 (2.6)	2.8 (5.0)	1.21 (3.2)
mNY positive	12 (5)	2.8 (2.4)	1.70 (2.4)	5.3 (7.8)	1.78 (5.1)	11.4 (11.7)	4.04 (6.2)
mNY negative	226 (90)	0.9 (1.7)	0.34 (1.5)	1.4 (2.9)	0.65 (2.3)	2.3 (4.0)	1.03 (2.8)
ASAS-MRI-SIJ positive	106 (42)	1.8 (2.2)	0.71 (1.9)	2.3 (3.6)	1.50 (3.2)	4.3 (4.8)	2.34 (3.8)
ASAS-MRI-SIJ negative	143 (57)	0.4 (0.8)	0.11 (0.9)	1.1 (3.2)	-0.20 (1.2)	1.7 (4.9)	-0.07 (1.6)
CRP (>5 mg/L)	81 (32)	1.4 (2.4)	0.43 (1.9)	1.6 (2.8)	0.71 (2.0)	3.3 (4.3)	1.32 (2.7)
CRP (≤5 mg/L)	167 (66)	0.7 (1.2)	0.43 (1.5)	1.6 (3.6)	0.73 (2.8)	2.6 (5.3)	1.22 (3.3)
Males	136 (54)	1.0 (1.6)	0.51 (1.7)	2.1 (4.0)	0.88 (2.9)	3.5 (5.9)	1.45 (3.6)
Females	115 (46)	0.9 (1.8)	0.33 (1.3)	1.0 (2.4)	0.52 (2.2)	2.0 (3.5)	0.94 (2.7)
HLA-B27 positive	182 (73)	1.1 (1.8)	0.59 (1.7)	2.0 (3.8)	0.74 (2.7)	3.4 (5.6)	1.42 (3.5)
HLA-B27 negative	69 (27)	0.6 (1.4)	0.01 (0.8)	0.4 (1.2)	0.63 (2.3)	1.2 (2.4)	0.68 (2.4)
ASDAS ≥2.1	62 (25)	1.0 (2.0)	0.42 (1.5)	1.5 (3.2)	0.40 (1.6)	2.8 (4.4)	0.91 (2.5)
ASDAS <2.1	154 (61)	0.8 (1.2)	0.47 (1.6)	1.7 (3.8)	0.91 (3.1)	2.9 (6.0)	1.48 (3.7)
Current smoker positive	41 (16)	0.9 (1.8)	0.24 (1.1)	1.1 (2.1)	0.51 (1.3)	2.1 (3.5)	0.90 (2.1)
Current smoker negative	201 (80)	0.9 (1.7)	0.37 (1.2)	1.7 (3.6)	0.85 (2.6)	2.9 (5.2)	1.31 (3.1)
Ever smoker positive	97 (39)	1.0 (1.8)	0.35 (1.2)	1.5 (3.1)	0.61 (1.9)	2.6 (4.1)	1.09 (2.4)
Ever smoker negative	145 (58)	0.9 (1.7)	0.35 (1.3)	1.7 (3.6)	0.92 (2.8)	2.9 (5.5)	1.35 (3.2)

All values are presented as mean (SD). Total structural lesions were calculated as the sum of erosions, fat lesions and ankylosis. Due to the minimal progression in ankylosis, it was not analysed separately.

ASAS: Assessment of SpondyloArthritis international Society; ASAS-MRI-SIJ: a positive MRI-SIJ according to the ASAS definition; ASDAS: axial spondyloarthritis disease activity score; axSpA: axial spondyloarthritis; BL: baseline; CRP: C-reactive protein; HLA-B27: human leukocyte antigen-B27; mNY: modified New York criteria; MRI: magnetic resonance imaging; SD: standard deviation; SIJ: sacroiliac joints; 2Y: 2 years.

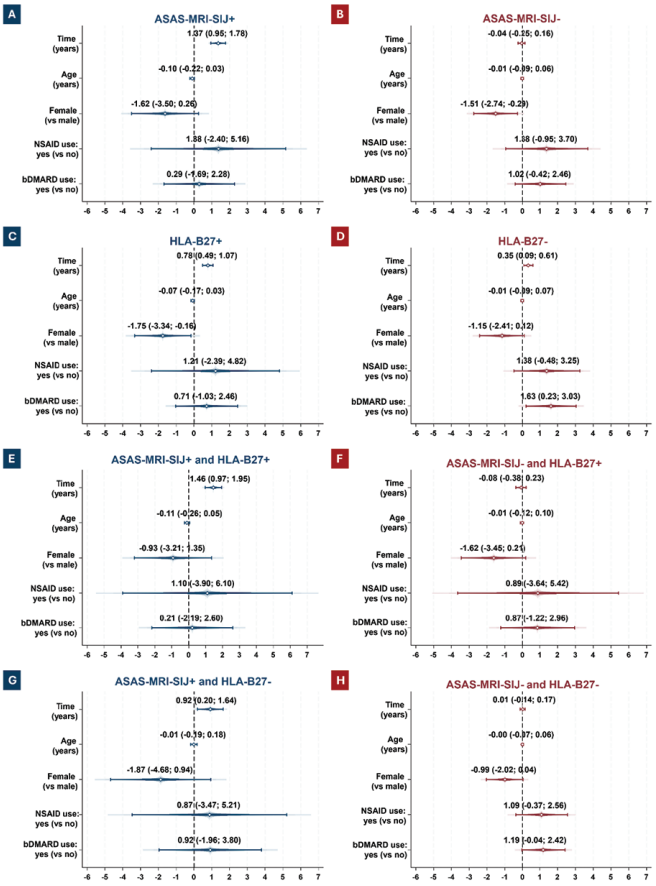


Fig. 1. Yearly change of total structural lesions according to risk factors in early axSpA. Models stratified by each risk factor due to a significant interaction between time and the corresponding risk factor. Panels A and B show the yearly change of total structural lesions in ASAS-MRI-SIJ+ and ASAS-MRI-SIJ- patients, respectively. Panels C and D show the yearly change in HLA-B27+ and HLA-B27- patients, respectively. Panels E-H show the yearly change in the four subgroups defined by ASAS-MRI-SIJ+ and HLA-B27+, following stratification due to a significant three-way interaction between time, ASAS-MRI-SIJ status, and HLA-B27. Results derived from GEE multilevel multivariable models, taking the effect of the reader into account. Coefficients with 95% confidence intervals are shown for time (reflecting yearly progression) and for covariates included in the model: age (in years), sex (female vs. male), NSAID 'ever use' (yes vs. no), and bDMARD 'ever use' (yes vs. no). Total structural lesions were calculated as the sum of erosions, fat lesions and ankylosis.

ASAS: Assessment of SpondyloArthritis international Society; ASAS-MRI-SIJ: a positive MRI-SIJ according to the ASAS definition; axSpA: axial spondyloarthritis; bDMARD: biologic disease-modifying anti-rheumatic drug; GEE: generalized estimating equation; HLA-B27: human leukocyte antigen-B27; MRI: magnetic resonance imaging; NSAID: non-steroidal anti-inflammatory drug; SIJ: sacroiliac joints.

P71

ASSOCIATIONS OF MUSCLE MASS, ADIPOSE TISSUE MASS, AND SKELETAL MUSCLE RADIODENSITY WITH DISEASE ACTIVITY IN AXIAL SPONDYLOARTHRITIS

Cui R.[1], Song X.Y.[2], Tao Y.L.[1], Wei X.E.[2], Dai S.M.[1]
[1]Dept. of Rheumatology and Immunology, Shanghai Sixth People's Hospital Affiliated to Shanghai Jiao Tong University School of Medicine, Shanghai; [2]Institute of Diagnostic and Interventional Radiology, Shanghai Sixth People's Hospital Affiliated to Shanghai Jiao Tong University School of Medicine, Shanghai, China

Background. Body composition is increasingly recognized as an important modulator of chronic inflammation. However, the association between body composition parameters and disease activity in axial spondyloarthritis (axSpA) remains to be clearly established.
Objective. We aimed to investigate the association between muscle, subcutaneous fat composition assessed via computed tomography (CT) imaging, and disease activity in patients with axSpA.
Design. This is a retrospective data registry observational study.
Methods. This study analyzed 215 patients with axSpA. Body composition parameters were measured from pelvic CT scans. Parameters included the total adipose tissue volume to total skeletal muscle volume ratio (TATV/TSMV), a single-slice subcutaneous adipose tissue to skeletal muscle area ratio (SAT/SM), and skeletal muscle (SM) radiodensity. Disease activity was evaluated using the Ankylosing Spondylitis Disease Activity Score (ASDAS). Associations were analyzed using multivariate linear regression and restricted cubic splines (RCS). The probability of achieving major improvement (MI, ΔASDAS 2.0) was compared using Kaplan-Meier analysis.
Results. After adjusting for age, sex, and disease duration, a higher TATV/TSMV ratio, rather than the single-slice SAT/SM ratio, was independently associated with increased ASDAS-C-reactive protein (β = 0.722, 95% confidence interval (CI): 0.215-1.231, p=0.004). SM radiodensity showed an inverse, nonsignificant association (p=0.48). RCS analysis identified a threshold effect of the TATV/TSMV ratio on disease activity at 0.65 (nonlinear p=0.045). Above this threshold, a higher ratio was significantly associated with elevated ASDAS-CRP (hazard ratio (HR) = 3.64, 95% CI: 1.13-11.71, p=0.029). Patients with a ratio <0.65 had a significantly higher probability of achieving MI (HR = 1.64, 95% CI: 1.04-2.59, log-rank p=0.04).
Conclusion. The pelvic TATV/TSMV ratio is an independent imaging biomarker associated with higher disease activity and lower odds of clinical improvement in patients with axSpA. A cut-off value of 0.65 may be a promising tool for risk stratification in clinical practice.

P72

WHICH FACTORS ARE ASSOCIATED WITH CT SYNDESMOPHYTE SCORE PROGRESSION OVER 2 YEARS IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS FROM THE SPACE COHORT?

de Hooge M.[1,2], Ayan G.[1,4], Marques M.L.[1,3], van Lunteren M.[1], de Bruin L.[1], Reijnierse M.[5], Ramiro S.[1,6], van Gaalen F.[1]
[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden, The Netherlands; [2]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [3]Dept. of Rheumatology, Coimbra Local Health Unit, Coimbra, Portugal; [4]Ankara Research and Training Hospital, Ankara, Turkey; [5]Dept. of Radiology, Leiden University Medical Center, Leiden; [6]Zuyderland Medical Center, Heerlen, The Netherlands

Introduction/Objective. To investigate Computed Tomography Syndesmophyte Score (CTSS) progression over time and whether clinical, laboratory, and imaging features modify this progression in patients with axSpA.
Materials and methods. Sixty-seven patients with axSpA from the SPACE-cohort underwent initial (T1; 0-10 year post-inclusion) and follow-up (T2; 2year) low-dose CT (ldCT) scans. Three readers evaluated images using the CTSS at vertebral corner level (23 vertebral units, sagittal/coronal planes), with averaged reader scores used for analyses. CTSS progression over time was examined using generalized estimating equations (GEE). Interactions between time and clinical (bDMARD use, high disease activity [ASDAS≥2.1]), laboratory (elevated CRP [≥5 mg/L]) and imaging features (radiographic syndesmophytes, modified New York [mNY] criteria, ASAS MRI-SIJ/spine positivity) were analysed. Features were defined as "once positive, always positive". If interactions were significant, models were stratified. Multivariable GEE models were adjusted for sex, symptom duration, HLA-B27 status, and CTSS at T1.
Results. In 67 patients, mean age at back pain onset was 28 (7) years, symptom duration 6 (3) years, 57% were male, 84% HLA-B27 positive, and 12% had early axSpA. Mean CTSS increased 0.8 unit/year (95%confidence interval 0.3-1.3). Significant interactions with time were observed for radiographic syndesmophytes (p=0.001) and elevated CRP (p=0.039), while no significant interactions were found for mNY status, ASAS MRI positivity (SIJ/spine), sex, bDMARD use, or ASDAS (Table I). The table also shows progression rates of stratified analyses.
Conclusion. Structural damage on ldCT increased significantly over time in axSpA, even though absolute progression was modest (0.8 CTSS unit/year). Nevertheless, even in this cohort with limited disease duration, ldCT was sensitive enough to detect structural progression over time as well as differential progression according to baseline risk factors. Progression is higher in patients with pre-existing radiographic syndesmophytes and elevated CRP, supporting the role of prior structural damage and systemic inflammation as key drivers of spinal progression.

Table I. Longitudinal CTSS progression and interactions between time and various demographic, clinical and imaging features.

Variable/subgroup[#]	Progression of CTSS per year β (95%CI)	p-value*
CTSS progression over time	0.8 (0.3-1.3)	
Time x Syndesmophytes [a]		**0.001**
Progression rate if radiographic syndesmophytes present (n=17)	1.4 (0.5 to 2.3)	
Progression rate if radiographic syndesmophytes absent (n=52)	0.2 (-0.27–0.69)	
Time x mNY status [b]		0.053
Time x ASAS MRI-SIJ positivity [c]		0.57
Time x ASAS MRI-spine positivity [d]		0.362
Time x Elevated CRP		**0.039**
Progression rate if CRP ≥5 (n=12)	1.2 (0.1–2.2)	
Progression rate if CRP <5 (n=55)	0.7 (0.1–1.2)	
Time x Sex		0.377
Time x bDMARD use		0.361
Time x ASDAS≥2.1		0.533

[#] Features were defined according to a "once positive, always positive" approach, meaning that a feature was considered present if positive either at inclusion in the SPACE cohort or at T1
*p-values are only shown for interactions between time and risk factors. When a significant interaction was observed, results were stratified and subgroup-specific progression rates are presented as regression coefficients
[a] According to the modified Stoke Ankylosing Spondylitis Spine Score (mSASSS)
[b] According to modified New York criteria; pelvic radiograph missing in one patient at time of initial CT scan
[c] According to Assessment in SpondyloArthritis international Society (ASAS) definition of a positive MRI-SIJ
[d] According to ASAS definition of a positive MRI-spine
CTSS: computed tomography syndesmophyte score; CI: confidence interval; mNY: modified New York criteria; ASAS: Assessment of SpondyloArthritis international Society; MRI-SIJ: magnetic resonance imaging of sacroiliac joints; CRP: C-reactive protein; bDMARD: biological disease-modifying antirheumatic drug; ASDAS: axial spondyloarthritis disease activity score

P73

COMPLEMENTARY DETECTION OF SYNDESMOPHYTES BY SAGITTAL AND CORONAL LOW-DOSE CT IN AXIAL SPONDYLOARTHRITIS: SPACE COHORT ANALYSIS

de Hooge M.[1,2], Ayan G[1,4], Marques M.L.[1,3], van Lunteren M.[1], de Bruin L.J.E.[1], Reijnierse M.[5], Ramiro S.[1,6], van Gaalen F.[1]
[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden, The Netherlands; [2]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [3]Dept. of Rheumatology, Coimbra Local Health Unit, Coimbra, Portugal; [4]Ankara Research and Training Hospital, Ankara, Turkey; [5]Dept. of Radiology, Leiden University Medical Center, Leiden; [6]Zuyderland Medical Center, Heerlen, The Netherlands

Introduction/Objective. To assess plane-specific inter-reader reliability and spinal damage progression using low-dose (ld)CT in patients with axSpA.

Materials and methods. In 67 axSpA patients from the SPACE-cohort, ldCT scans at T1 (0-10y post-inclusion) and follow-up T2 (2y) were evaluated. Using the CT Syndesmophyte Score (CTSS), syndesmophytes were assessed at vertebral corner level across 23 vertebral units (VU) in sagittal and coronal planes. Inter-reader reliability of CTSS progression was assessed in sagittal and coronal planes by spinal segment. Continuous outcomes = mean reader scores, dichotomous outcomes = ≥2/3 reader consensus. Missed lesion-positive units using single-plane assessment were evaluated per reader. Quadrant-level comparisons between sagittal and coronal reconstructions were not feasible due to non-corresponding anatomical projections across planes (*e.g.* an anterior syndesmophyte in sagittal image may appear in right or left coronal quadrant).
Results. In sagittal plane, ICCs on progression scores were 0.78 (whole spine), 0.64 (cervical), 0.80 (thoracic), 0.89 (lumbar) spine. In coronal plane; 0.64 (whole spine), 0.51 (cervical), 0.68 (thoracic), 0.88 (lumbar). Table I shows CTSS and progression.
When using a single imaging plane, a substantial proportion of lesion-positive units was missed. Coronal-only missed 42–45% of lesions, while sagittal-only missed 28–31%: Per reader (R): R1 45%/31%, R2 42%/30%, R3 44%/28% (coronal/sagittal).
VU-level progression across imaging planes shown in Figure 1.
Conclusion. Sagittal and coronal ldCT reconstructions scoring had moderate-to-good inter-reader reliability, with higher agreement in sagittal plane. However, a substantial proportion of syndesmophytes are detected exclusively in one imaging plane. Thus, sagittal and coronal views provide complementary information, and reliance on a single plane seems to result in systematic underestimation of structural damage and progression. Although limited degree of progression restricts robust quantification of added value of combining planes, the consistent pattern of lesions that otherwise remained undetected supports a dual-plane (sagittal and coronal) approach for structural damage assessment in axSpA.

Table I. Plane-specific mean CTSS across three readers at initial low-dose CT scan (T1) and 2-year follow-up (T2), including overall and segmental progression.

	Sagittal view				Coronal view			
Spinal segment	**T1**	**T2**	**CTSS progression**	**CTSS progression distribution***	**T1**	**T2**	**CTSS progression**	**CTSS progression distribution***
Total spine	4.1 (8.1)	4.7 (9.1)	1.2 (3.8)	100%	5.5 (10.8)	6.4 (12.1)	1.6 (4.4)	100%
Cervical	0.9 (4.2)	0.9 (3.8)	0.5 (1.9)	42%	1.3 (5.3)	1.6 (6.9)	1.0 (2.9)	62.5%
Thoracic	2.6 (5.6)	3.2 (6.3)	0.6 (1.7)	50%	3.3 (6.8)	3.7 (7.3)	0.4 (1.7)	25%
Lumbar	0.6 (1.6)	0.7 (1.7)	0.1 (0.9)	8%	0.9 (2.9)	1.1 (3.1)	0.2 (0.8)	12.5%

Numbers in mean with standard deviation, unless stated otherwise.
*Percentage contribution of each spinal segment to total spine CTSS progression.
T1: initial time point of low dose CT; T2: follow-up timepoint (2 years); CTSS: computer tomography syndesmophytes score

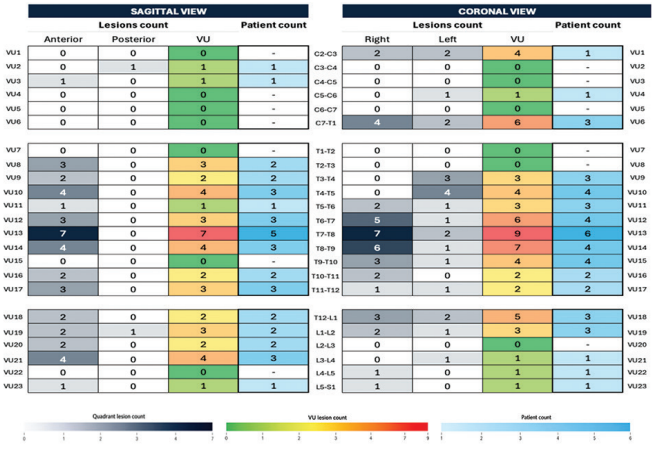


Fig. 1. Distribution of new and growing syndesmophytes by quadrant (anterior/posterior in the sagittal plane; right/left in the coronal plane), vertebral unit (VU1-23) and patients with the number of patients exhibiting CTSS progression per level.

P74

POSTERIOR ELEMENT LESIONS IN AXIAL SPONDYLOARTHRITIS: 10-YEAR FOLLOW-UP FROM THE DESIR COHORT

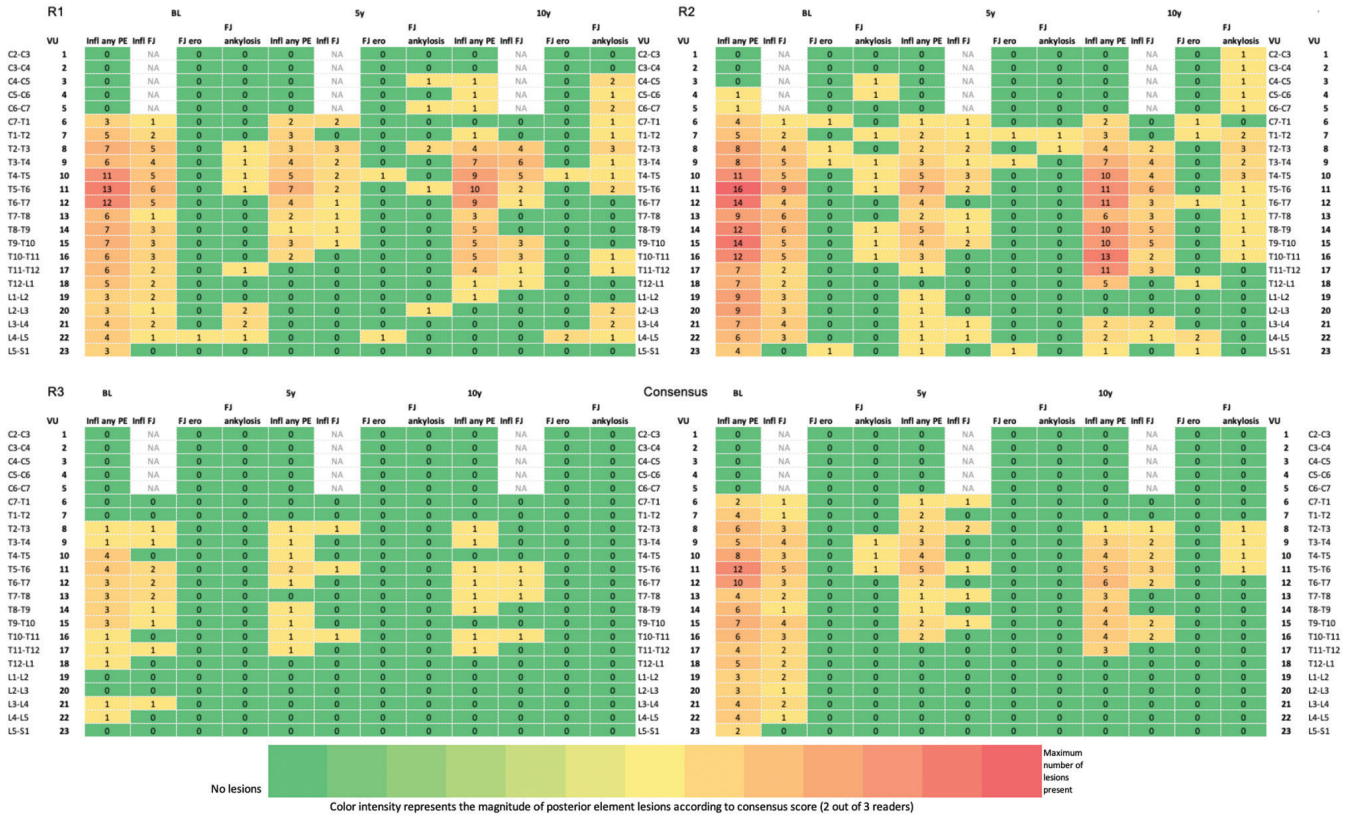
de Hooge M.[1,2], van Lunteren M.[1], Molto A.[3,4], Reijnierse M.[5], Lopez-Medina C.[6], Saraux A.[7], van Gaalen F.[1], Ramiro S.[1,8]
[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden, The Netherlands; [2]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [3]Université Paris Cité and Université Sorbonne Paris Nord, Inserm, INRAE, Centre for Research in Epidemiology and Statistics (CRESS), Paris; [4]Dept. of Rheumatology, Bichat Hospital, APHP Nord, Paris, France; [5]Dept. of Radiology, Leiden University Medical Center, Leiden, The Netherlands; [6]Reina Sofía University Hospital, IMIBIC, University of Córdoba, Córdoba, Spain; [7]Université de Bretagne Occidentale (Univ Brest), Dept. of Rheumatology, CHU Brest, INSERM (U1227), LabEx IGO, Brest, France; [8]Zuyderland Medical Center, Heerlen, The Netherlands

Introduction/Objective. To assess prevalence and 10-year evolution of posterior element (PE) inflammation and facet joint (FJ) damage, and estimate probability of developing FJ ankylosis conditional on baseline inflammation in patients with axSpA.
Materials and methods. Patients from DESIR cohort with 10-year spinal MRI and ≥1 additional timepoint were included. MRI-spine at baseline/5/10 years were independently scored by three blinded central readers for PE and FJ inflammation, FJ erosions and ankylosis, dichotomously per vertebral unit. Aggregated scores (≥2/3 readers) were used to estimate the probability of developing FJ ankylosis on 5-/10-year time intervals, conditional on the presence/absence of baseline inflammation in all PE [P(FJA | PE inflammation)] or in FJ only.
Results. Total of 320 patients (mean age 34.6(8.6) years; 54% female) were included. At baseline, PE inflammation was present in 5% (n=15), FJ inflammation in 4% (n=12), and FJ ankylosis in 0.3% (n=1). No consensus was reached for FJ erosions. The thoracic spine was most frequently affected (Fig. 1).
Absence of PE inflammation over time was observed in 92%-98% of patients across readers, while new inflammation developed in <1%-5%. For FJ inflammation, 91%-98% remained persistently negative, with new lesions in <1%-3%. FJ erosions were rare, with 0–2 new lesions per reader, but no consensus. FJ ankylosis remained absent in 99%-100%, with 1% developing new ankylosis. No new FJ erosions or ankylosis were identified by consensus at 5 years. Baseline PE and FJ inflammation were associated with a higher 10-year probability of developing new FJ ankylosis (Table I).
Conclusion. PE and FJ lesions were uncommon and remained largely stable over 10 years. FJ ankylosis was rare, occurring predominantly in patients with baseline inflammation, supporting a pathophysiological link between inflammation and structural progression. Lesions are mainly located in the thoracic spine, highlighting this preferential site of involvement in axSpA.

Table I. Probability of developing facet joint ankylosis depending on the presence or absence of posterior element inflammation 10 year prior, in patients with axial spondyloarthritis.

Inflammation in any part of the posterior elements			
Posterior element inflammation	New facet joint ankylosis after 10 years	N*	P(FJ ankylosis | PE inflammation)
0	0	6151	P(FJ ankylosis | 0) = 2/6153 = 0.00033
0	1	2	
1	0	92	P(FJ ankylosis | 1) =2/94 = 0.02128
1	1	2	
Inflammation only in the facet joint			
Facet joint inflammation	New facet joint ankylosis after 10 years	N#	P(FJ ankylosis | FJ inflammation)
0	0	4899	P(FJ ankylosis | 0) = 3/4902 = 0.00064
0	1	3	
1	0	38	P(FJ ankylosis | 1) =1/39 = 0.0256
1	1	1	

Based on the consensus defined as 2 out of 3 readers.
*Number of vertebral unit levels with inflammation in at least one part of the posterior elements (pedicle, facet joints, processes spinosi, soft tissue).
#Number of facet joints; P: probability (scale 0–1); FJ: facet joint; PE: posterior elements



P74 - Fig. 1. Posterior element lesions on spinal MRI over 10 years in patients with axial spondyloarthritis: heatmaps of individual reader scores (R1–R3) and consensus defined as 2 out of 3 readers. R1: reader 1; R2: reader 2; R3: reader 3; BL: baseline;[a] 5y: 5-year follow-up; 10y: 10-year follow-up; Infl: inflammation; FJ: facet joint; VU: vertebral unit; ero: erosion

P75

ENHANCED DETECTION OF BONE FORMATION ON SYNTHETIC CT VERSUS CONVENTIONAL MRI IN AXIAL SPONDYLOARTHRITIS: DATA FROM THE GO-GUT STUDY

de Hooge M.[1], Lukasik Z.[1,2], Pardaens L.[1], Varkas G.[1], De Craemer A.S.[1,2], Jans L.[3], Carron P.[1], Elewaut D.[1,2], Van den Bosch F.[1,2]

[1]Dept. of Rheumatology, Ghent University Hospital, Ghent; [2]Dept. of Rheumatology and Molecular Immunology and Inflammation Unit, VIB-UGent Center for Inflammation Research, Ghent University, Ghent; [3]Dept. of Radiology, Ghent University Hospital, Ghent, Belgium

Objective. To compare detection and extent of structural lesions in sacroiliac joints (SIJ)/spine assessed by conventional MRI and synthetic CT (sCT) in patients with early axial spondyloarthritis (axSpA) at a 5-year post-inclusion in the GO-GUT cohort.

Methods. Twenty-four patients from a cohort of 58 treatment-naïve early axSpA patients underwent SIJ and spinal imaging with conventional MRI and sCT at 5 years after inclusion. Imaging was independently evaluated by two blinded readers with consensus scoring. SIJ lesions included erosions, sclerosis, and ankylosis, analysed using normalized scores. Spinal lesions included erosions and syndesmophytes and analysed using raw scores. Differences between modalities were assessed using the Wilcoxon signed-rank test.

Results. Of 24 patients with MRI and sCT data available, 10 (42%) were female, mean age was 33 (SD 6) years, and 92% HLA-B27 positive. Mean disease duration was 5 (SD 4) years.

Table I summarizes structural lesions in SIJ/spine detected by conventional MRI and sCT. Figure 1 shows proportion of patients with SIJ structural lesions detected on both conventional MRI and sCT, on sCT only, or on neither modality. Spinal erosions were rare, observed in one patient on both MRI and sCT. Syndesmophytes were more frequently detected on sCT than MRI (mean counts 0.6 vs. 0.1). Seven patients (29%) had syndesmophytes on sCT, compared with one (4%) on MRI (and sCT). Sixteen patients (67%) had no syndesmophytes on either modality.

Conclusion. In patients with early axSpA at a 5-year post-inclusion time point, sCT provides increased detection of sclerosis (SIJ) and bone formation (SIJ/spine) compared with conventional MRI, while showing comparable detection of erosions. These findings suggest a complementary role for sCT in structural assessment, particularly for bone formation lesions, which may be clinically relevant because they reflect structural remodeling and are linked to disease progression. In contrast, the added value of detecting additional sclerosis or small erosions is less clear.

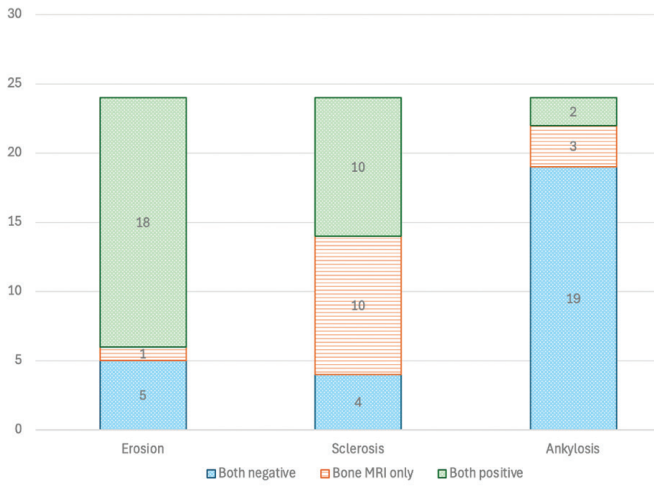


Fig. 1. Comparison of sacroiliac joint structural lesions detected by conventional MRI and sCT, showing the proportion of patients with lesions detected on both modalities, sCT only, or neither modality.

Table I. Structural lesions detected by conventional MRI and sCT in the sacroiliac joints and spine of patients with early axial spondyloarthritis.

Sacroiliac joints	Modality	Raw lesion count, mean (SD)	Maximum observed lesion count	Normalized score, mean (SD)	p-value†
Erosions	MRI*	6.3 (5.5)	18	13.2 (11.4)	0.472
	SCT**	3.3 (3.0)	13	13.9 (12.4)	
Sclerosis	MRI*	1.5 (2.0)	6	3.0 (4.2)	<0.001
	SCT**	2.7 (2.4)	9	11.3 (9.8)	
Ankylosis	MRI*	0.4 (1.3)	6	0.8 (2.8)	0.063
	SCT***	0.4 (0.8)	3	3.1 (6.9)	
Spine	**Modality**	**Mean lesion count (SD)**	**Maximum lesion count**	**Normalized score, mean (SD)**	**p-value**
Erosions‡	MRI	0.1 (0.4)	2	—	0.500
	SCT	0.1 (0.4)	2	—	
Syndesmophytes‡	MRI	0.04 (0.2)	2~	—	0.008
	SCT	0.6 (1.0)	3	—	

* MRI SIJ erosion/sclerosis scoring range: 0–48; ** SCT SIJ erosion/sclerosis scoring range: 0–24; *** SCT SIJ ankylosis scoring range: 0–12; ‡ Spine lesion scoring range: 0–96; † p-values compare normalized scores between MRI and sCT;
~ Bridging syndesmophyte; assigned as a score of 2.
MRI: magnetic resonance imaging; SD: standard deviation

P76

RADIOGRAPHIC PROGRESSION IN SACROILIAC JOINTS IN FIVE YEARS FOLLOW UP IN NON-RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS PATIENTS AND THE PREDICTIVE FACTORS

Erpek E.[1], Kocaayan H.[1], Ediboğlu E.[1], Tosun Ö.[2], Solmaz D.[1], Akar S.[1]
[1]Dept. of Internal Medicine, Division of Rheumatology, Izmir Katip Celebi University, Izmir; [2]Dept. of Radiology, Izmir Katip Celebi University, Izmir, Turkey

Introduction. Non-radiographic axial spondyloarthritis (nr-axSpA) represents an early disease stage without definite radiographic sacroiliitis, yet a substantial proportion of patients progress to radiographic axSpA (r-axSpA) over time. This study aimed to assess sacroiliac joint progression rates and identify predictive factors.
Methods. Patients with nr-axSpA and at least one follow-up pelvic radiograph were included. Radiographs were independently evaluated by two blinded readers according to the modified New York criteria, with discrepancies resolved by a third rheumatologist. Progression to r-axSpA was assessed at two and five years. Factors associated with five-year progression were analyzed using Generalized Estimating Equations (GEE), and variables with $p<0.10$ were included in multivariate analysis.
Results. Among patients with available two-year radiographic follow-up (n=194), progression to r-axSpA was observed in 17.5%. At five years, among patients with available follow-up (n=116), the progression rate increased to 24.1%. Baseline demographic and clinical characteristics are summarized in Table I. In univariate analysis, symptom duration, education level, body mass index (BMI), BASFI score, C-reactive protein (CRP) levels, BASMI scores, and family history of SpA were associated with radiographic progression. However, in the multivariate model, only elevated CRP levels and higher BMI remained significant predictors of progression to r-axSpA (Table II).
Conclusion. The 5-year progression rate was 24.1%, within the range reported in previous studies (2–43.5%) (1). The PROOF study reported a lower rate (16%) (2), possibly due to shorter symptom duration at inclusion. Our higher rate may reflect longer baseline disease duration (Table I). Notably, BMI emerged as an independent predictor, suggesting a role for metabolic and mechanical factors in structural progression. Elevated CRP was also confirmed as a key predictor. These findings highlight the importance of integrating clinical, laboratory, and imaging parameters to improve prediction of progression in nr-axSpA.

References

1. PROTOPOPOV M, PODDUBNYY D: Radiographic progression in non-radiographic axial spondyloarthritis. *Expert Rev Clin Immunol* 2018; 14(6): 525-33.
2. PODDUBNYY D *et al*.: Radiographic progression in sacroiliac joints in patients with axial spondyloarthritis: results from a five-year international observational study. *ACR Open Rheumatol* 2024; 6(2): 103-10.

Table I. Baseline demographic, disease related clinical, and laboratory characteristics of non-radiographic axSpA patients.

Characteristics (N=194)	
Male sex n (%)	84/194 (43.3)
Age, years mean ± SD	44.6 ± 10.3
Symptom duration, yıl mean ± SD	16 ± 8.8
Time since diagnosis, years, mean ± SD	10.1 ± 5.5
Education duration, years mean ± SD	10.8 ± 4
Current smoking n (%)	74/181 (40.9)
BMI kg/m2 mean ± SD	28.3 ± 21.1
HLA-B27 positivity n (%)	78/159 (49.1)
C-reactive protein mg/dl mean ± SD	9.5 ± 17.9
BASDAI mean ± SD	4.3 ± 2.3
BASFI mean ± SD	3.1 ± 2.6
ASDAS-CRP mean ± SD	2.6 ± 1.1
BASMI mean ± SD	2.8 ± 1.3
ASQOL mean ± SD	8.7 ± 5.1
HAQs mean ± SD	0.8 ± 0.6
Dactylitis, n (%)	5/184 (2.7)
Peripheral arthritis n (%)	58/177 (32.8)
Heel pain n (%)	89/170 (52.4)
Radiographic hip involvement, n (%)	8/194 (4.1)
Psoriasis n (%)	8/147 (5.4)
Inflammatory bowel disease n (%)	15/166 (9)
Uveitis n (%)	19/184 (10.3)
NSAID use n (%)	140/171 (81.9)
Biologic treatment use, n (%)	88/193 (45.6)
Positive ASAS family history,n (%)	54/168 (32.1)

Table II. Univariable and multivariable longitudinal analysis results of factors influencing progression from nr-axSpA to r-axSpA.

	Univariable analysis				Multivariable analysis			
	B	%95 CI		p	B	%95 CI		p
Symptom duration	0.033	-0.002	0.067	0.065	0.031	-0.008	0.115	0.088
Education level	-0.052	-0.222	-0.014	**0.026**	-0.052	-0.159	0.056	0.347
Body Mass Index	0.016	-0.002	0.034	0.075	0.014	0.000	0.028	**0.043**
BASFI score	0.169	0.031	0.307	**0.016**	0.035	-0.169	0.239	0.735
C-reaktive protein	0.023	0.003	0.044	**0.027**	0.019	0.806	1.680	**0.038**
BASMI score	0.329	-0.049	0.708	0.088	0.372	-0.240	0.983	0.195
ASAS family history for SpA	-0.099	-1.913	-0.80	**0.033**	-0.715	-1.619	0.189	0.121

P77

PREVALENCE AND CLINICAL ASSOCIATIONS OF SACROILIAC JOINT MRI LESIONS IN PATIENTS REFERRED FOR SUSPECTED AXIAL SPONDYLOARTHRITIS: REAL-WORLD DATA FROM THE FASTRAX COHORT

Aydin S.[7,8], Sabido Sauri R.[7,8], Gendron E.[9], Chim T.[1], Chow N.[1], MacLeod A.[5], Correale M.[2,6], Fidler W.[5], Pirson C.[10], Pus L.[10], Rampersaud R.[2,6], Haroon N.[1,2], Inman R.D.[1,2], Passalent L.[1,2]
[1]Schroeder Arthritis Institute, [2]University Health Network, [3]Krembil Research Institute, [4]University of Toronto, [5]Community Rheumatology (Thunder Bay), [6]Rapid Access Clinic – Low Back Pain Program, [7]The Ottawa Hospital, [8]University of Ottawa, [9]Centre de l'ostéoporose et de rhumatologie de Québec (Québec), Canada; [10]UCB

Introduction/Objective. To assess the prevalence and clinical associations of SIJ MRI lesions in patients with back pain referred for suspected axSpA.
Methods. We used baseline data from the FASTRAX cohort from May 7, 2024 to May 27, 2026, across Ontario (Toronto, Thunder Bay, and Ottawa). Adults (≥18 years) with low back pain >3 months duration, onset age <50 years were referred by their primary care provider, physician specialist or through the provincial LBP program to their nearest FASTRAX site for axSpA screening. Patients underwent standard-of-care assessment, including imaging when indicated. All screened patients were reviewed by the rheumatologist, who provided the final diagnosis. MRI inflammatory and structural lesions were evaluated individually and in combination, and their associations with clinical findings, sex, HLA-B27 status, and extra-musculoskeletal manifestations (EMM) were analyzed using logistic regression analyses.

Results. Of 313 patients, 180 patients had SIJ MRIs (61.7% female; 26.7% HLA-B27 present; 26.7% axSpA). SIJ MRI changes were identified (26.7% BME; 17.2% erosions; 10% fat lesions). 32.2% of patients had inflammatory and/or structural SIJ MRI lesions, including 5.6% with isolated structural lesions and 10.6% with isolated BME. Across MRI-positive patients, 11.4% did not receive a clinical diagnosis of axSpA whereas 10.4% of patients with no MRI findings on the SIJs were diagnosed with axSpA, all of which demonstrated BME exclusively in the spine but not the SIJs. Within individual SIJ MRI findings, the presence of erosions had the highest odds of being diagnosed as axSpA (OR: 60.2, 95%CI: 16.7-216.6), followed by BME (OR: 30, 95%CI: 12.4-72.5) and fatty lesions (OR: 7, 95%CI: 2.5-20) (Table I). Only male sex was associated with MRI findings, including increased BME and erosions (Table II).

Conclusion. SIJ MRI lesions were common in patients referred for suspected axSpA. Structural lesions, especially erosions, were most strongly associated with axSpA diagnosis and may provide additional diagnostic value.

Table I. The prevalence of individual MRI lesions.

	axSpA (n=48)			Non-axSpA (n=132)		
	All groups	Male (n=19)	Female (n=23)	All groups	Male (n=45)	Female (n=80)
BME*	36 (75%)	16 (84.2%)	15 (65.2%)	12 (9.1%)	6 (13.3%)	6 (7.5%)
Erosion	28 (58.3%)	12 (63.2%)	12 (52.2%)	3 (2.3%)	3 (6.7%)	0 (0%)
Fatty lesion	12 (25%)	6 (31.6%)	5 (21.7%)	6 (4.5%)	2 (4.4%)	4 (5%)

Variables are presented in n (%), *BME: bone marrow edema

Table II. The prevalence of individual MRI lesions according to sexes.

	Male (n=64)	Female (n=103)	p-value
BME*	22 (34.4%)	21 (20.4%)	0.048
Erosion	15 (23.4%)	12 (11.7%)	0.053
Fatty lesion	8 (12.5%)	9 (8.7%)	0.442

Variables are presented in n (%), *BME: bone marrow edema

P78

COSTOTRANSVERSE JOINT INVOLVEMENT ON NA[^{18}F]F PET/CT IN FIRST-DEGREE RELATIVES OF HLA-B27-POSITIVE AXIAL SPONDYLOARTHRITIS PATIENTS

Groothuizen S.[1], Van der Heijden W.R.P.[1], Doorschodt T.C.[2], Knol R.J.J.[3], Zwezerijnen G.J.C.[4], Van der Laken C.J.[1], Van de Sande M.G.H.[1]

[1]Dept. of Rheumatology and Clinical Immunology, Amsterdam UMC, Amsterdam; [2]Dept. of Radiology, OLVG, Amsterdam; [3]Dept. of Nuclear Medicine, Northwest Clinics, Alkmaar; [4]Dept. of Radiology and Nuclear Medicine, Amsterdam UMC, Amsterdam, The Netherlands

Introduction. A substantial portion of seemingly healthy first-degree relatives (FDR) of axSpA patients show signs of subclinical inflammation (1). We aimed to investigate whether new bone formation can also be detected using ^{18}F-sodium fluoride (Na[^{18}F]F) PET/CT.

Methods. The Pre-SpA cohort is a 5-year prospective inception cohort of seemingly healthy FDRs of HLA-B27-positive axSpA patients, aged 18–40 years. In a substudy, whole-body Na[^{18}F]F PET/CT was performed within eight weeks of inclusion, to evaluate metabolic new bone formation. Using a previously published semi-quantitative scoring method, PET/CT scans were assessed for PET-positive lesions in the axial skeleton (2). PET/CT scans of sex-matched oncology patients without SpA served as controls. Group differences were analyzed using the Mann-Whitney U-test; $p<0.05$ was considered statistically significant.

Results. Nine FDRs (18–33 years) and 19 non-SpA controls (33–47 years) were included. All were female, and 6 FDRs were HLA-B27–positive. The median number of PET-positive lesions was 8 (range 2–22) in FDRs and 2 (range 0-11) in controls; $p<0.01$. Notably, the FDRs showed more frequent costotransverse joint uptake compared with controls ($p<0.0001$; Fig. 1A and Fig. 2), while the number of facet and costovertebral lesions did not differ significantly. Higher scores were mostly seen in HLA-B27–positive FDRs, although this was not statistically significant (Fig. 1B). Longitudinal follow-up of the Pre-SpA cohort is still ongoing; to date, one FDR has developed non-radiographic axSpA after 4 years.

Conclusion. In this exploratory study, FDRs at risk of SpA showed a higher frequency of Na[^{18}F]F PET/CT-positive lesions; in particular costotransverse lesions, which were almost absent in controls. These findings suggest that costotransverse joint uptake may reflect preclinical bone involvement in at-risk individuals, but further follow-up and confirmation in larger cohorts are needed to investigate if this is associated with development of SpA.

References

1. doi: 10.1002/acr.24743
2. doi: 10.1186/s40658-025-00734-7

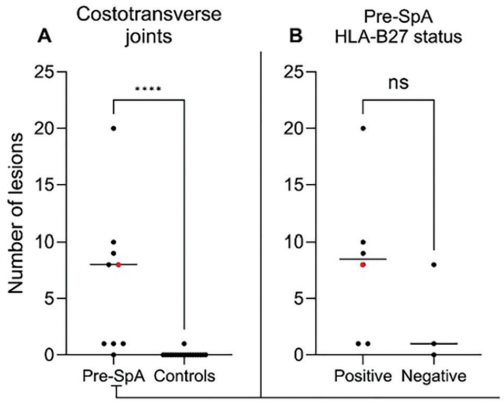


Fig. 1. Frequency of costotransverse lesions on Na[^{18}F]F PET/CT.
A. In Pre-SpA participants vs. non-SpA controls and **B.** in HLA-B27-positive vs. -negative Pre-SpA participants. The red dot is the Pre-SpA participant that was diagnosed with nr-axSpA.

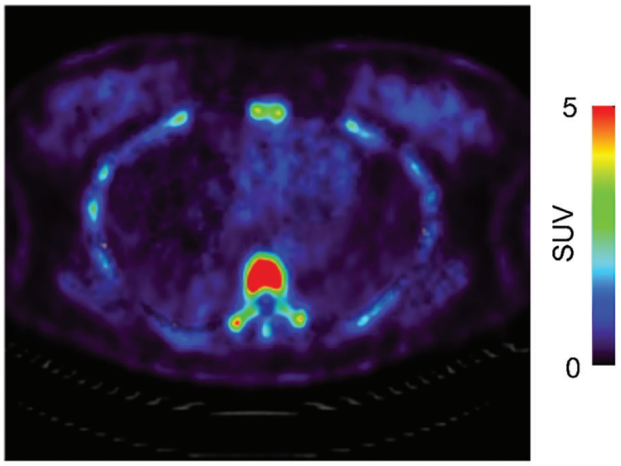


Fig. 2. Costotransverse lesions on Na[^{18}F]F PET/CT.
The figure shows a merged PET/CT image of bilateral costotransverse lesions in a Pre-SpA participant

P79

HEAD-TO-HEAD COMPARISON OF THE RATINGEN SCORE AND SHARP-VAN DER HEIJDE METHOD FOR RADIOGRAPHIC ASSESSMENT IN PSORIATIC ARTHRITIS

Kulyk M.[1], Scriffignano S.[2], Steinfeld S.[3], Nzeusseu Toukap A.[4], Van den Bosch F.[5,6], Joos R.[7], Geusens P.[8], Lories R.[1,9], De Vlam K.[1,9], and BEPAS Study Investigators

[1]Skeletal Biology and Engineering Research Center, KU Leuven, Leuven, Belgium; [2]Internal Medicine Unit, Hospital G. Giglio, Cefalù, Italy; [3]Dept. of Rheumatology, Hospital St. Jean, Brussels; [4]Dept. of Rheumatology, Cliniques Universitaires St. Luc, Brussels; [5]Dept. of Rheumatology, UZ Gent, Gent; [6]Molecular Immunology and Inflammation Unit, VIB-UGent Center for Inflammation Research, Zwijnaarde; [7]Dept. of Rheumatology, ZAS, Antwerpen; [8]ReumaClinic, Genk; [9]Dept. of Rheumatology, University Hospitals Leuven, Leuven, Belgium

Introduction/Objective. The modified Sharp-van der Heijde method (SVDH) quantifies erosions and joint space narrowing, whereas the Psoriatic Arthritis Ratingen Score (PARS) also captures proliferative bone changes. We compared the psychometric performance of PARS and SVDH in the Belgian Psoriatic Arthritis (BePas) cohort.

Materials and methods. Standard radiographs of hands and feet were obtained at baseline (T0), 1 year (T1), and 2 years (T2). Images were anonymized and independently scored by two trained, blinded readers using SVDH (maximum score 528) and PARS (maximum score 360). Inter-rater reliability was assessed using intraclass correlation coefficients (ICC). Agreement was evaluated with Bland-Altman analysis. Responsiveness was quantified using the standardized response mean (SRM) and measurement error using the smallest detectable change (SDC). Two-reader average analyses were also performed.

Results. Radiographs were available for 287 patients at T0, 168 at T1, and 121 at T2; 101 patients had complete radiographic data at all three time points. Both methods detected positive mean radiographic change, mainly between T0 and T1. Inter-rater reliability for absolute scores was high for both instruments, with baseline ICC values of 0.906 for PARS and 0.888 for SVDH. Reliability for cumulative 2-year change was low and nearly identical: ICC 0.485 for PARS and 0.483 for SVDH. Responsiveness over two years was moderate and slightly higher for PARS than SVDH, with two-reader average SRM values of 0.571 and 0.531, respectively. Mean cumulative progression remained below corresponding SDC values for both methods. Bland–Altman analysis showed narrower absolute limits of agreement for PARS, whereas SVDH showed narrower relative limits after normalization.

Conclusion. PARS and modified SVDH showed broadly comparable psychometric performance. Both were reliable for cross-sectional damage assessment but limited for detecting small cumulative change in this low-progression cohort. Neither method demonstrated clear overall superiority.

P80

WHOLE-SPINE MRI PREDICTS 5-YEAR RADIOGRAPHIC PROGRESSION IN AXIAL SPONDYLOARTHRITIS

Oh S.H.[1], Lee B.W.[1], Jung J.Y.[2], Park H.W.[1], Yang J.W.[1], Kim J.G.[3], Kwok S.K.[1], Ju J.H.[1], Park S.H.[1], Kim W.U.[1], Lee J.J.[1]

[1]Division of Rheumatology, Dept. of Internal Medicine, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul; [2]Dept. of Radiology, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul; [3]Division of Rheumatology, Dept. of Internal Medicine, Inje University Ilsan Paik Hospital, Goyang, Republic of Korea

Introduction. We aimed evaluate whether baseline whole-spine MRI predicts 5-year spinal radiographic progression in axial spondyloarthritis (axSpA), and to assess their prognostic value beyond other clinical factors.

Methods. We retrospectively analyzed 104 patients with axSpA who underwent baseline whole-spine MRI and spinal radiography, with repeat spinal radiography after 5 years. Radiographic progression was defined as an increase in the modified Stoke Ankylosing Spondylitis Spine Score (ΔmSASSS) of ≥2. Baseline MRI indices included the SPARCC spine score, fatty metaplasia count, facet joint inflammation count, and total inflammatory and structural lesion counts. Clinical variables included age, sex, HLA-B27 status, smoking, CRP, sacroiliac joint grade, and biologic initiation during follow-up. Group comparisons, correlation analyses, and prespecified multivariable linear regression models were performed.

Results. Among 104 patients, 59 (56.7%) showed radiographic progression over 5 years. Progressors were older and had higher baseline mSASSS, higher sacroiliac joint grade, higher baseline and time-averaged CRP, and greater MRI inflammatory and structural lesion burden than non-progressors. Five-year ΔmSASSS correlated with baseline mSASSS (r=0.59), SPARCC spine score (r=0.47), and fatty metaplasia count (r=0.48). In multivariable analysis, a model including baseline mSASSS explained 37.8% of the variance in progression. Addition of SPARCC spine score and fatty metaplasia modestly improved model fit (R^2=0.426; ΔR^2=0.048; p=0.025), although neither variable remained independently significant after adjustment for baseline mSASSS. A model including total inflammatory and total structural lesion counts assessed by whole-spine MRI, without baseline mSASSS, showed the best overall fit (R^2=0.444) and both lesion counts were independently associated with greater progression. In subgroup analysis, patients with both SPARCC ≥2 and fatty metaplasia had the highest 5-year progression.

Conclusion. Baseline whole-spine MRI lesion burden predicts 5-year spinal radiographic progression in axSpA and provides clinically relevant prognostic information beyond conventional factors. Whole-spine MRI may improve early risk stratification, particularly in patients with minimal or absent baseline radiographic damage.

P81

WHAT MRI CUT-OFFS DEFINE A POSITIVE MRI FOR INFLAMMATION IN THE SACROILIAC JOINTS WHEN AN MRI IS GLOBALLY CONSIDERED INDICATIVE OF AXSPA? AN ANALYSIS FROM THE SPONDYLOARTHRITIS RESEARCH AND TREATMENT NETWORK CLASSIFICATION IN AXSPA INCEPTION COHORT

Horbal N.[1,2], Caplan L.[3], Aydin S.[4], Bernard S.[5], Burgos-Vargas R.[6], Carrino J.A.[7], Chan J.[8], Danve A.[9], Haroon N.[10], Magrey M.[11], Mease P.J.[12], O'Neill J.[13], Reveille J.D.[14], Tuite M.[15], Wang R.[16], Wichuk S.[2], Deodhar A.[17], Lambert R.G.W.[18], Maksymowych W.P.[2]

[1]Danylo Halytsky Lviv National Medical University, Medicine, Lviv, Ukraine; [2]University of Alberta, Medicine, Edmonton, Canada; [3]University of Colorado School of Medicine, Medicine, Aurora, USA; [4]University of Ottawa, Medicine, Ottawa, Canada; [5]Brooke Army Medical Center, Radiology, Fort Sam, USA; [6]National Autonomous University of Mexico, Mexico City, Mexico; [7]Hospital for Special Surgery, Radiology and Imaging, New York, USA; [8]University of British Columbia, Medicine, Vancouver, Canada; [9]Yale University School of Medicine, Medicine, New Haven, USA; [10]University Health Network, Medicine, Toronto, Canada; [11]Case Western Reserve University School of Medicine, Medicine, Cleveland; [12]Swedish Medical Center, Medicine, Seattle, USA; [13]McMaster University, Radiology, Hamilton, Canada; [14]McGovern School of Medicine at the University of Texas Health Science Center at Houston, Medicine, Houston; [15]University of Wisconsin School of Medicine and Public Health, Radiology, Madison;[16]Cooperman Barnabas Medical Center, Union; [17]Oregon Health and Science University, Medicine, Portland, USA; [18]University of Alberta, Radiology and Diagnostic Imaging, Edmonton, Canada

Background/Objective. There is a new MRI variable in the 2025 ASAS-SPARTAN Revised Classification Criteria for axSpA termed 'MRI indicative of axSpA in the sacroiliac joints (SIJ)' based on global evaluation of all lesions. However, there may be research settings that still require defining a positive MRI for inflammation in a standardized manner. We tested the performance of MRI cut-offs for bone marrow edema (BME) in the SPARTAN CLASSIC inception cohort.

Methods. The study recruited patients from North America presenting with undiagnosed back pain. Scans were assessed globally for 'MRI indicative of axSpA' by central and, independently, by local readers. Subsequently, detailed assessment of all MRI lesions in the SIJ was conducted. We calculated sensitivity and specificity for the number of SIJ quadrants (SIJQ) or consecutive semicoronal slices with BME where 'MRI indicative of axSpA' was reference criterion. Sensitivity analyses included rheumatologist diagnosis of axSpA as reference criterion. We prespecified a requirement for ≥90% specificity for MRI cut-offs.

Results. 501 patients were recruited, 148 (29.5%) were diagnosed with axSpA, 85 (57.4%) had 'MRI indicative of axSpA' by central readers, 61/85 (71.8%) had both structural lesions and BME, 1/85 (1.2%) had only BME. For local reader assessments, 129 of 187 (69.0%) diagnosed with axSpA had 'MRI indicative of axSpA', 72 (55.8%) had both structural lesions and

Table I. Central or local reader assignment of 'MRI indicative of axSpA' is the reference criterion for testing MRI cut-offs.

Central Reader 'MRI indicative of axSpA'

MRI variable SIJ quadrants or consecutive slices	MRI indicative of axSpA=Yes N (%) N=85	MRI indicative of axSpA=No N (%) N=416	Sensitivity (95% CI)	Specificity (95% CI)	LR+	LR-
BME≥2 qds	65 (76.5)	105 (25.2)	76.5 (66.0 - 85.0)	74.8 (70.3 - 78.9)	3	0.3
BME≥3 qds	62 (72.9)	70 (16.8)	72.9 (62.2 - 82.0)	83.2 (79.2 - 86.6)	4.3	0.3
BME≥4 qds	61 (71.8)	42 (10.1)	71.8 (61.0 - 81.0)	89.9 (86.6 - 92.6)	7.1	0.3
BME≥5 qds	58 (68.2)	32 (7.7)	68.2 (57.2 - 77.9)	92.6 (89.6 - 94.9)	9.2	0.3
BME≥2 slices	56 (65.9)	61 (14.7)	65.9 (54.8 - 75.8)	85.3 (81.6 - 88.6)	4.5	0.4
BME≥3 slices	46 (54.1)	27 (6.5)	54.1 (43.0 - 65.0)	93.5 (90.7 - 95.7)	8.3	0.5
BME≥4 slices	40 (47.1)	8 (1.9)	47.1 (36.1 - 58.2)	98.1 (96.2 - 99.2)	24.5	0.5

Local Reader 'MRI indicative of axSpA'

MRI variable SIJ quadrants or consecutive slices	MRI indicative of axSpA=Yes N (%) N=150	MRI indicative of axSpA=No N (%) N=351	Sensitivity (95% CI)	Specificity (95% CI)	LR+	LR-
BME≥2 qds	92 (61.3)	78 (22.2)	61.3 (53.0-69.2)	77.8 (73.1-82.0)	2.8	0.5
BME≥3 qds	82 (54.7)	50 (14.2)	54.7 (46.3-62.8)	85.8 (81.7-89.2)	3.8	0.5
BME≥4 qds	71 (47.3)	32 (9.1)	47.3 (39.1-55.6)	90.9 (87.4-93.7)	5.2	0.6
BME≥5 qds	65 (43.3)	24 (6.8)	43.3 (35.3-51.7)	93.2 (90.0-95.6)	6.3	0.6
BME≥2 slices	73 (48.7)	44 (12.5)	48.7 (40.4-57.0)	87.5 (83.5-90.7)	3.9	0.6
BME≥3 slices	54 (36)	19 (5.4)	36.0 (28.3-44.2)	94.6 (91.7-96.7)	6.7	0.7
BME≥4 slices	41 (27.3)	7 (2)	27.3 (20.4-35.2)	98.0 (95.9-99.2)	13.7	0.7

Table II. Local rheumatologist diagnosis of axSpA is the reference criterion for testing MRI cut-offs.

MRI variable SIJ quadrants or consecutive slices	axSpA=Yes N (%) N=148	axSpA=No N (%) N=353	Sensitivity (95% CI)	Specificity (95% CI)	LR+	LR-
BME≥2 qds	85 (57.4)	85 (24.1)	57.4 (49.0 - 65.5)	75.9 (71.1 - 80.3)	2.4	0.6
BME≥3 qds	76 (51.4)	56 (15.9)	51.4 (43.0 - 59.6)	84.1 (79.9 - 87.8)	3.2	0.6
BME≥4 qds	70 (47.3)	33 (9.3)	47.3 (39.0 - 55.7)	90.7 (87.1 - 93.5)	5.1	0.6
BME≥5 qds	64 (43.2)	25 (7.1)	43.2 (35.1 - 51.6)	92.9 (89.7 - 95.4)	6.1	0.6
BME≥2 slices	69 (46.6)	48 (13.6)	46.6 (38.4 - 55.0)	86.4 (82.4 - 89.8)	3.4	0.6
BME≥3 slices	55 (37.2)	18 (5.1)	37.2 (29.4 - 45.5)	94.9 (92.1 - 97.0)	7.3	0.7
BME≥4 slices	42 (28.4)	6 (1.7)	28.4 (21.3 - 36.4)	98.3 (96.3 - 99.4)	16.7	0.7

BME, 15 (11.6%) had only BME. MRI cut-offs for BME ≥4 SIJQ or ≥3 SIJ consecutive slices were consistently required to attain ≥90% specificity for 'MRI indicative of axSpA' per central and local readers (Table I), and when rheumatologist diagnosis of axSpA was the reference criterion (Table II). Specificity of cut-offs was improved when the anterior 2 slices comprising the load zone were not analyzed.

Conclusion. BME in ≥4 SIJQ or ≥3 consecutive slices reflects a positive MRI for inflammation with consistently high specificity for axSpA.

P82

PERFORMANCE OF STRUCTURAL LESION MRI CUT-OFFS FOR AN MRI INDICATIVE OF AXIAL SPONDYLOARTHRITIS IN THE SPONDYLOARTHRITIS RESEARCH AND TREATMENT NETWORK CLASSIFICATION OF AXSPA INCEPTION COHORT STUDY

Maksymowych W.P.[1], Caplan L.[2], Aydin S.[3], Bernard S.[4], Burgos-Vargas R.[5], Carrino J.A.[6], Chan J.[7], Danve A.[8], Gensler L.S.[9], Haroon N.[10], Horbal N.[1,11], Magrey M.[12], Mease P.J.[13], O'Neill J.[14], Reveille J.D.[15], Tuite M.[16], Wang R.[17], Wichuk S.[1], Deodhar A.[18], Lambert R.G.W.[19]

[1]University of Alberta, Medicine, Edmonton, Canada; [2]University of Colorado School of Medicine, Medicine, Aurora, USA; [3]University of Ottawa, Medicine, Ottawa, Canada; [4]Brooke Army Medical Center, Radiology, Fort Sam, USA; [5]National Autonomous University of Mexico, Mexico City, Mexico; [6]Hospital for Special Surgery, Radiology and Imaging, New York, USA; [7]University of British Columbia, Medicine, Vancouver, Canada; [8]Yale University School of Medicine, Medicine, New Haven, USA; [9]University Health Network, Medicine, Toronto, Canada; [10]University of San Francisco, Medicine, San Francisco, USA; [11]Danylo Halytsky Lviv National Medical University, Medicine, Lviv, Ukraine; [12]Case Western Reserve University School of Medicine, Medicine, Cleveland, USA; [13]Swedish Medical Center, Medicine, Seattle, USA; [14]McMaster University, Radiology, Hamilton, Canada; [15]McGovern School of Medicine at the University of Texas Health Science Center at Houston, Medicine, Houston, USA; [16]University of Wisconsin School of Medicine and Public Health, Radiology, Madison, USA; [17]Cooperman Barnabas Medical Center, Union, USA; [18]Oregon Health and Science University, Medicine, Portland, USA; [19]University of Alberta, Radiology and Diagnostic Imaging, Edmonton, Canada

Background/Objective. The most highly weighted SpA variable in the 2025 ASAS-SPARTAN Revised Classification Criteria for axSpA is a new MRI variable termed 'MRI indicative of axSpA in the sacroiliac joints (SIJ)' and is based on a global evaluation of both inflammatory and structural lesions. However, the contribution of different structural lesions is unclear. We assessed the performance of structural lesion MRI cut-offs for 'MRI indicative of axSpA' in the multicenter SPARTAN CLASSIC inception cohort.

Methods. Patients were recruited from North America presenting with undiagnosed back pain and symptom onset less than 45 years. Clinical and imaging assessments were conducted locally. Central readers first assessed scans globally for 'MRI indicative of axSpA', followed by detailed assessment of all lesions. We calculated sensitivity and specificity for the number of SIJ quadrants with BME, erosion, fat lesion, or sclerosis or SIJ halves with backfill or ankylosis when central reader 'MRI indicative of axSpA' was th e reference criterion. Target performance was prespecified at ≥70% sensitivity and ≥90% specificity.

Results. 501 patients were recruited, 148 (29.5%) were diagnosed with axSpA, 85 (57.4%) of axSpA cases had 'MRI indicative of axSpA' (22 (25.8%) had only structural lesions, 61 (71.8%) had both structural and inflammatory lesions). Among individual lesion MRI cut-offs, only erosion achieved prespecified target cut-offs for sensitivity ≥70%/specificity ≥90% (Table I). Fat lesion, backfill, and ankylosis cut-offs were all highly specific, even at ≥2 SIJ quadrants, but lacked sensitivity. Combinations of structural lesions with or without BME were highly specific (Table II), with the 'BME and erosion' combination achieving target sensitivity/specificity of ≥70%/≥90%.

Conclusion. Most structural lesions are highly specific for 'MRI indicative of axSpA' but erosion contributes most to its ascertainment. Combinations of different structural lesions, and with BME, are highly contributory emphasizing the importance of contextual interpretation of MRI.

Table I. Performance of single lesion MRI cut-offs (sacroiliac joint quadrants or halves) versus central reader designated 'MRI indicative of axSpA' as reference criterion.

MRI assessed in SIJ quadrants (SIJQ) or halves (SIJH) or consecutive slices	MRI indicative of axSpA=Yes N (%)	MRI indicative of axSpA=No N (%)	Sensitivity (95% CI)	Specificity (95% CI)	LR+	LR-
Erosion≥2 SIJQ	75 (88.2)	26 (6.3)	88.2 (79.4 - 94.2)	93.8 (91.0 - 95.9)	14.1	0.1
Erosion≥3 SIJQ	72 (84.7)	20 (4.8)	84.7 (75.3 - 91.6)	95.2 (92.7 - 97.0)	17.6	0.2
Erosion≥4 SIJQ	69 (81.2)	17 (4.1)	81.2 (71.2 - 88.8)	95.9 (93.5 - 97.6)	19.9	0.2
Erosion ≥2 slices	60 (70.6)	7 (1.7)	70.6 (59.7 - 80.0)	98.3 (96.6 - 99.3)	42.0	0.3
Erosion ≥3 slices	44 (51.8)	1 (0.2)	51.8 (40.7 - 62.7)	99.8 (98.7 - 100.0)	215.3	0.5
Fat≥2 SIJQ	49 (57.6)	25 (6)	57.7 (46.4 - 68.3)	94 (91.3 - 96.1)	9.6	0.5
Fat≥3 SIJQ	46 (54.1)	19 (4.6)	54.1 (43.0 - 65.0)	95.4 (93.0 - 97.2)	11.9	0.5
Fat≥4 SIJQ	41 (48.2)	14 (3.4)	48.2 (37.3 - 59.3)	96.6 (94.4 - 98.1)	14.3	0.5
Fat ≥2 slices	30 (35.3)	8 (1.9)	35.3 (25.2 - 46.4)	98.1 (96.2 - 99.2)	18.4	0.7
Fat≥3 slices	26 (30.6)	5 (1.2)	30.6 (21.0 - 41.5)	98.8 (97.2 - 99.6)	25.5	0.7
Backfill≥2 SIJH	26 (30.6)	3 (0.7)	30.6 (21.0 - 41.5)	99.3 (97.9 - 99.9)	42.4	0.7
Backfill≥3 SIJH	20 (23.5)	2 (0.5)	23.5 (15.0 - 34.0)	99.5 (98.3 - 99.9)	48.9	0.8
Backfill≥4 SIJH	17 (20)	1 (0.2)	20 (12.1 - 30.1)	99.8 (98.7 - 100.0)	83.2	0.8
Sclerosis≥2 SIJQ	56 (65.9)	136 (32.7)	65.9 (54.8 - 75.8)	67.3 (62.6 - 71.8)	2.0	0.5
Sclerosis≥3 SIJQ	52 (61.2)	137 (32.7)	61.2 (50.0 - 71.6)	74.5 (70.0 - 78.6)	2.4	0.5
Sclerosis≥4 SIJQ	49 (57.6)	87 (20.9)	57.7 (46.4 - 68.3)	79.1 (74.9 - 82.9)	2.8	0.5
Ankylosis≥2 SIJH	13 (15.3)	4 (1)	15.3 (8.4 - 24.7)	99 (97.6 - 99.7)	15.9	0.9
Ankylosis≥3 SIJH	12 (14.1)	3 (0.7)	14.1 (7.5 - 23.4)	99.3 (97.9 - 99.9)	19.6	0.9
Ankylosis≥4 SIJH	11 (12.9)	1 (0.2)	12.9 (6.6 - 22.0)	99.8 (98.7 - 100.0)	53.8	0.9

Table II. Performance of combinations of MRI lesion cut-offs versus central reader designated 'MRI indicative of axSpA' as reference criterion.

MRI lesion assessed in SIJ quadrants (SIJQ)	MRI indicative of axSpA=Yes N (%)	MRI indicative of axSpA=No N (%)	Sensitivity (95% CI)	Specificity (95% CI)	LR+	LR-
Erosion ≥ 1 AND BME ≥ 1	67 (78.8)	27 (6.5)	78.8 (68.6 - 86.9)	93.5 (90.7 - 95.7)	12.1	0.2
Erosion ≥ 1 AND BME ≥ 2	62 (72.9)	20 (4.8)	72.9 (62.2 - 82.0)	95.2 (92.7 - 97.0)	15.2	0.3
Erosion ≥ 2 AND BME ≥ 1	66 (77.6)	17 (4.1)	77.7 (67.3 - 86.0)	95.9 (93.5 - 97.6)	19.0	0.2
Erosion ≥ 2 AND BME ≥ 2	61 (71.8)	13 (3.1)	71.8 (61.0 - 81.0)	96.9 (94.7 - 98.3)	23.0	0.3
Erosion ≥ 3 AND BME ≥ 1	64 (75.3)	14 (3.4)	75.3 (64.7 - 84.0)	96.6 (94.4 - 98.1)	22.4	0.3
Fat ≥ 1 AND BME ≥ 1	42 (49.4)	27 (6.5)	49.4 (38.4 - 60.5)	93.5 (90.7 - 95.7)	7.6	0.5
Fat ≥ 1 AND BME ≥ 2	37 (43.5)	15 (3.6)	43.5 (32.8 - 54.7)	96.4 (94.1 - 98.0)	12.1	0.6
Fat ≥ 1 AND BME ≥ 3	35 (41.2)	10 (2.4)	41.2 (30.6 - 52.4)	97.6 (95.6 - 98.8)	17.1	0.6
Fat ≥ 2 AND BME ≥ 1	39 (45.9)	17 (4.1)	45.9 (35.0 - 57.0)	95.9 (93.5 - 97.6)	11.2	0.6
Fat ≥ 2 AND BME ≥ 2	34 (40.0)	9 (2.2)	40 (29.5 - 51.2)	97.8 (95.9 - 99.0)	18.5	0.6
Fat ≥ 1 AND Erosion ≥ 1	46 (54.1)	9 (2.2)	54.1 (43.0 - 65.0)	97.8 (95.9 - 99.0)	25.0	0.5
Fat ≥ 1 AND Erosion ≥ 2	45 (52.9)	6 (1.4)	52.9 (41.8 - 63.9)	98.6 (96.9 - 99.5)	36.7	0.5
Fat ≥ 1 AND Erosion ≥ 3	44 (51.8)	3 (0.7)	51.8 (40.7 - 62.7)	99.3 (97.9 - 99.9)	71.8	0.5
Fat ≥ 2 AND Erosion ≥ 1	43 (50.6)	6 (1.4)	50.6 (39.5 - 61.6)	98.6 (96.9 - 99.5)	35.1	0.5
Fat ≥ 2 AND Erosion ≥ 2	42 (49.4)	5 (1.2)	49.4 (38.4 - 60.5)	98.8 (97.2 - 99.6)	41.1	0.5

P83

DISCORDANCE BETWEEN SYMPTOM BURDEN AND INFLAMMATORY ACTIVITY IN AXIAL SPONDYLOARTHRITIS: CONTRIBUTIONS OF FIBROMYALGIA AND MRI-DEFINED INFLAMMATION

Maroof A.[1,2], Kader S.H.[1]
[1]College of Medicine, Hawler Medical University, Erbil; [2]School of Medicine, University of Kurdistan Hawlêr, Erbil, Iraq

Introduction. Discordance between symptom-based and inflammation-based disease activity measures is frequently observed in axial spondyloarthritis (axSpA), yet the mechanisms underlying this phenomenon remain insufficiently understood.
Objective. To investigate clinical and mechanistic determinants of discordance between BASDAI and ASDAS-CRP, with particular focus on fibromyalgia and MRI-defined inflammation.
Methods. In this multicenter cross-sectional study, 118 patients fulfilling ASAS classification criteria for axSpA were categorized into four phenotypes according to BASDAI (<4/≥4) and ASDAS-CRP (<2.1/≥2.1). Fibromyalgia was defined according to the American College of Rheumatology criteria. MRI-defined inflammation was determined according to ASAS-positive MRI criteria based on bone marrow edema lesions. Clinical characteristics and outcomes were compared across phenotypes. Multivariable logistic regression identified predictors of inflammation-dominant discordance.
Results. BASDAI and ASDAS-CRP correlated strongly (r=0.78, $p<0.001$), but agreement for active disease was only moderate (κ=0.39). Inflammation-dominant discordance (low BASDAI/high ASDAS-CRP) occurred in 38/118 patients (32.2%), whereas symptom-dominant discordance was rare (1/118, 0.8%). Fibromyalgia prevalence increased with symptom burden, from 13.6% (6/44) in concordant low disease activity to 40.0% (14/35) in concordant high disease activity ($p=0.018$). MRI-defined inflammation was markedly enriched in ASDAS-driven phenotypes, particularly inflammation-dominant discordance (92.1%) and concordant high disease activity (97.1%; $p=0.009$). In multivariable analysis, MRI inflammation (OR 3.80, 95% CI 1.60-9.20, $p=0.002$) and fibromyalgia (OR 2.30, 95% CI 1.20-4.50, $p=0.010$) independently predicted discordance.
Conclusion. Inflammation-dominant discordance is common in axSpA, indicating that clinically relevant inflammatory activity may persist despite low symptom burden. MRI-defined inflammation appears to be a major driver of this phenotype, whereas fibromyalgia contributes to symptom amplification. These findings support multidimensional disease assessment integrating both objective inflammatory measures and patient-reported outcomes.
Acknowledgements. The authors thank the participating centers and patients for their contributions to this study.

References

1. RUDWALEIT M, VAN DER HEIJDE D, LANDEWE R *et al*.: The development of Assessment of SpondyloArthritis international Society classification criteria for axial spondyloarthritis (part II): validation and final selection. *Ann Rheum Dis* 2009; 68(6): 777-83.
2. MACHADO P, LANDEWÉ R, LIE E *et al*.: Ankylosing Spondylitis Disease Activity Score (ASDAS): defining cut-off values for disease activity states and improvement scores. *Ann Rheum Dis* 2011; 70(1): 47-53. doi:10.1136/ard.2010.138594
3. WOLFE F, CLAUW DJ, FITZCHARLES MA *et al*.: The American College of Rheumatology preliminary diagnostic criteria for fibromyalgia and measurement of symptom severity. *Arthritis Care Res* (Hoboken) 2010; 62(5): 600-10. doi:10.1002/acr.20140
4. LAMBERT RG, BAKKER PA, VAN DER HEIJDE D *et al*.: Defining active sacroiliitis on MRI for classification of axial spondyloarthritis: update by the ASAS MRI working group. *Ann Rheum Dis* 2016; 75(11): 1958-63.
5. MAKSYMOWYCH WP, LAMBERT RG, BARALIAKOS X *et al*.: Data-driven definitions for active and structural MRI lesions in the sacroiliac joint in spondyloarthritis and their predictive utility. *Rheumatology* (Oxford) 2021; 60(10): 4778-89. https://doi.org/10.1093/rheumatology/keab099

P84

ASSESSMENT OF ENTHESITIS IN PSORIATIC ARTHRITIS USING CLINICAL EXAMINATION, ULTRASOUND AND SHEAR WAVE ELASTOGRAPHY

Montvydaite M., Butrimiene I., Silale G.
Clinic of Rheumatology, Orthopedics Traumatology, and Reconstructive Surgery, Institute of Clinical Medicine, Vilnius University, Faculty of Medicine, Vilnius, Lithuania

Introduction. Enthesitis is a hallmark feature of psoriatic arthritis (PsA). Clinical examination and musculoskeletal ultrasound (US) may identify different aspects of entheseal involvement. We aimed to evaluate the agreement between clinical tenderness and ultrasonographic enthesitis and to explore shear wave elastography (SWE) findings in PsA entheses.
Methods. The study was conducted at the Dept. of Rheumatology, Vilnius University Hospital Santaros Klinikos. Twenty patients with PsA aged 22–67 years and without fibromyalgia symptoms underwent clinical and ultrasonographic assessment of six entheseal sites at the non-dominant upper and lower limb (120 entheses). Ultrasound examination was performed using a CANON Aplio i800 system. Enthesitis was defined according to the 2018 OMERACT ultrasound criteria. SWE measurements were expressed as shear wave velocity (m/s). Agreement between clinical tenderness and US findings was assessed using Cohen's kappa coefficient. Group comparisons of SWE measurements were performed using the Wilcoxon rank-sum test.
Results. Clinical-ultrasonographic classification and SWE findings across individual enthesis sites are summarized in Table I. Overall, 24 of 120 entheses (20.0%) demonstrated subclinical enthesitis and 21 (17.5%) were painful without ultrasonographic abnormalities. Agreement between clinical tenderness and US findings was poor (κ=0.115). Median shear wave velocity varied across anatomical locations. At the patellar tendon enthesis, median shear wave velocity was lower in US-positive than in US-negative sites (2.86 vs. 3.93 m/s; p=0.037).

Table I.

Enthesis	Subclinical enthesitis, n (%)	Pain only, n (%)	SWE US− (M/S)	SWE US+ (M/S)	p-value
Medial epicondyle	2 (10)	2 (10)	3.19	3.64	0.315
Lateral epicondyle	2 (10)	6 (30)	4.03	2.80	0.118
Quadriceps tendon	5 (25)	4 (20)	4.19	5.42	0.201
Patellar tendon	3 (15)	7 (35)	3.93	2.86	**0.037**
Achilles tendon	5 (25)	2 (10)	3.97	3.48	0.384
Plantar fascia	7 (35)	0 (0)	3.81	4.44	1.000

Conclusion. Clinical examination and ultrasound demonstrated poor agreement in the assessment of enthesitis in PsA. A substantial proportion of ultrasonographically detected enthesitis was clinically silent. SWE findings were heterogeneous across enthesis sites.

P85

DEVELOPMENT AND VALIDATION OF A NEW ULTRASOUND SCORE (SONAR-7) FOR SYNOVITIS IN AXIAL SPONDYLOARTHRITIS AND PSORIATIC ARTHRITIS

Elsehrawy G.G.[1,2], Courvoisier D.S.[3], Deman E.[4], Dan D.[5], Micheroli R.[6], Ziswiler H.[7], Zufferey P.[8], Brulhart L.[9], Nissen M.J.[3]
[1]Dept. of Rheumatology, Geneva University Hospital, University of Geneva, Geneva, Switzerland; [2]Dept. of Physical Medicine, Rheumatology and Rehabilitation, Faculty of Medicine, Suez Canal University, Ismailia, Egypt; [3]Dept. of Rheumatology, Geneva University Hospital, University of Geneva, Geneva; [4]Dept. of Rheumatology, University Hospital of Basel, University of Basel, Basel; [5]Dept. of Rheumatology, University Hospital of Lausanne, University of Lausanne, Lausanne; [6]Dept. of Rheumatology, University Hospital of Zurich, University of Zurich, Zurich; [7]OsteoRheuma, Bern; [8]Dept. of Rheumatology, Fribourg Hospitals, Fribourg; [9]Dept. of Rheumatology, Neuchâtel Hospital, La Chaux-de-Fonds, Switzerland

Objective. Development of a new ultrasound (US) synovitis score (SONography in Arthritis and Rheumatism (SONAR)-7) with evaluation of its diagnostic performance in a cohort of patients with axial spondyloarthritis (axSpA) and psoriatic arthritis (PsA), in comparison with healthy controls (HC).
Methods. We included 121 participants: 41 patients with PsA and 39 patients with axSpA, from six Swiss hospitals and 41 HC. All participants

underwent a clinical examination of joints (68/66 tender joint count/swollen joint count (SJC)), followed by a detailed musculoskeletal US examination of 22 joints ("PsA-Son22" score) with semi-quantitative scoring of grey-scale (GS) and power Doppler (PD) synovitis in adherence with the definitions established by the OMERACT group, as well as evaluation of erosions and new bone formation (NBF).

Results. The 'SONAR-7' score incorporated GS and PD lesions across 14 joints (all demonstrating significance (or a trend, $p<0.08$) in distinguishing between SpA and HC in regression analyses (Table I)), including four metacarpophalangeal joints, four distal interphalangeal joints of the hands, two metatarsophalangeal joints, knees and wrists (Fig. 1). The score demonstrated an area under the receiver operating characteristic curve of 0.831 (95% CI 0.76 to 0.90), with excellent specificity (95.1%) and moderate sensitivity (44%), comparable to existing US scores including more joints. The inflammatory components of the SONAR-7 correlated significantly with clinical measures of inflammation and disease activity (correlation: 0.37 for SJC-28, 0.33 for DAPSA and 0.48 for US enthesitis). PsA and axSpA subgroups demonstrated comparable inflammatory joint US features (B-mode and Doppler). NBF with SONAR-7 was associated with the Health Assessment Questionnaire ($p=0.035$) and Bath Ankylosing Spondylitis Metrology Index ($p=0.016$).

Conclusion. The SONAR-7 score represents a fast and effective US-based tool for the evaluation of inflammatory and structural joint involvement in patients with PsA and axSpA, with high specificity and meaningful associations with clinical and quality-of-life outcomes.

Acknowledgements. This study was funded by the Warnery Prize from a Swiss non-profit organization and Dr Elsehrawy was supported by a post-doctoral Swiss Government Excellence Scholarship provided by the Swiss Confederation.

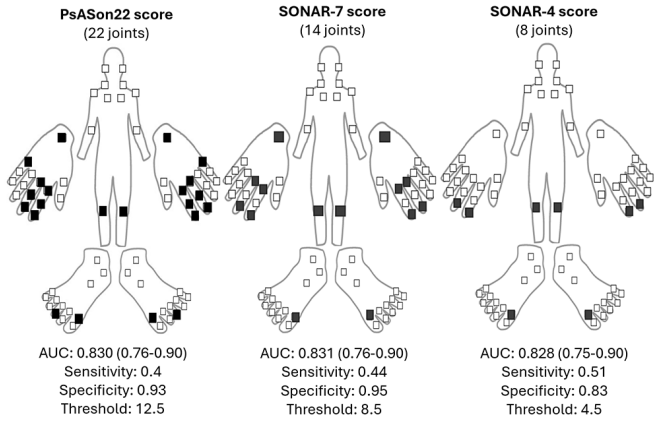


Fig. 1. Evaluated joints, AUC values, threshold, sensitivity, and specificity of the evaluated scores.
AUC: area under the curve; SONAR: Swiss Sonography in Arthritis and Rheumatism group. SONAR-4 score with 4 joints bilaterally. FICJAN A *et al*.: Ultrasound composite scores for the assessment of inflammatory and structural pathologies in Psoriatic Arthritis (PsASon-Score). *Arthritis Res Ther* 2014; 16(5): 476.

Table I. Association between the inflammatory score (GS and PD synovitis) for each joint region and a diagnosis of SpA.

	Univariable			**Multivariable***			**Multivariable****		
	OR	95% CI	p-value	OR	95% CI	p-value	OR	95% CI	p-value
Joint region									
Wrist	1.43	1.02 - 2.1	*0.052*						
Hand MCP 2	1.43	1.0 - 2.2	*0.077*						
Hand MCP 3	1.56	0.9 - 2.6	*0.073*						
Hand MCP 5	1.40	0.8 - 2.5	0.22						
Hand PIP 2	1.19	0.7 - 2.0	0.48						
Hand PIP 3	0.95	0.6 - 1.4	0.83						
Hand DIP 2	2.33	1.4 - 4.3	**0.002**	1.99	0.96 to 4.57	*0.07*	2.49	1.39 to 5.05	**0.004**
Hand DIP 3	2.03	1.2 - 3.4	**0.004**	1.37	0.72 to 2.84	0.34			
Knee	3.30	1.5 - 12.0	**0.019**	3.23	1.34 to 12.4	**0.02**	3.43	1.41 to 13.50	**0.025**
Foot MTP 1	1.24	1.02 - 1.5	**0.046**	1.0	0.78 to 1.30	0.94			
Foot PIP 3	1.38	0.6 - 3.1	0.38						
Demographics									
Age				0.98	0.94 to 1.03	0.59	0.98	0.94 to 1.03	0.65
BMI				1.25	1.10 to 1.46	**0.001**	1.25	1.10 to 1.45	**0.001**

* Included only the sites with p-value <0.05 in the univariable analysis. **Included the sites that were associated with the diagnosis of SpA in the LASSO model. Multivariable analyses were adjusted for age and BMI. GS: grayscale; SpA: Spondyloarthritis; BMI: body mass index; MCP: metacarpophalangeal, PIP: proximal interphalangeal, PD: power Doppler; DIP: distal interphalangeal, MTP: metatarsophalangeal joints.

References
1. FICJAN A, HUSIC R, GRETLER J *et al*.: Ultrasound composite scores for the assessment of inflammatory and structural pathologies in Psoriatic Arthritis (PsASon-Score). *Arthritis Res Ther* 2014; 16(5): 476.
2. D'AGOSTINO MA, TERSLEV L, AEGERTER P *et al*.: Scoring ultrasound synovitis in rheumatoid arthritis: a EULAR-OMERACT ultrasound taskforce – Part 1: definition and development of a standardised, consensus-based scoring system. *RMD Open* 2017; 3(1): e000428.

P86

MISSED IMAGING OPPORTUNITIES IN AXIAL SPONDYLOARTHRITIS: A POTENTIAL CONTRIBUTOR TO DIAGNOSTIC DELAY

Placido F.[1], Martini F.[2], Vitale A.[1], Lacarpia A.[1], Martini N.[2], Caggiano V.[1], Sbalchiero J.[1], Di Meglio N.[2], Frediani B.[1], Mazzei M.A.[2], Cantarini L.[1]
[1]Rheumatology Unit, Dept. of Medical, Surgical and Neuro Sciences, Azienda Ospedaliero-Universitaria Senese, University of Siena, Siena; [2]Unit of Diagnostic Imaging, Dept. of Medical Sciences, Azienda Ospedaliero-Universitaria Senese, University of Siena, Siena, Italy

Introduction/Objective. Diagnostic delay remains a major challenge in axial spondyloarthritis (axSpA) despite increasing awareness and wider availability of advanced imaging techniques. We investigated the role of imaging misinterpretation on axial-SpA diagnostic delay.

Materials and methods. We retrospectively evaluated 176 axSpA patients with documented delayed diagnosis. Initial imaging examinations had been interpreted as negative or degenerative. Imaging modality, anatomical coverage, contrast enhancement, and inflammatory and structural lesions involving the sacroiliac joints (SIJ) and spine were compared with the examination leading to diagnosis.

Results. Initial imaging was reported as negative in 147/176 patients (83.5%) and degenerative in 29/176 (16.5%). Subsequent imaging identified sacroiliitis in 173 patients (98.3%) and inflammatory spinal involvement in 155 (88.1%), with combined SIJ and spinal disease in 153 cases (86.9%). Magnetic resonance imaging accounted for the most frequent radiologic exam used at diagnosis ($p<0.0001$), especially using contrast enhancement ($p<0.0001$). Complete evaluation of both spine and pelvis was performed in 92.0% of diagnostic examinations compared with 29.0% of initial studies ($p<0.0001$). Retrospective reassessment demonstrated that inflammatory and structural lesions were already detectable on initial imaging, including bone marrow oedema (54.0%), facet joint capsulitis (44.3%), microerosions (40.9%), erosions (38.1%) and interspinous enthesitis (36.9%) (Table I). Based on clinical data available in 98 patients, imaging abnormalities were associated with classification of axSpA according to ASAS criteria in 94.9% of patients when at least one anatomical site was involved and in 95.5% when both spine and SIJs were affected. Anti-tumor necrosis factor therapy (used in 53 patients) induced improvement in clinimetric scores in 64.2% of cases, while CRP normalized in 76.2% of patients with elevated baseline levels.

Conclusion. Delayed diagnosis is also associated with radiologic misinterpretation. Based on the high rate of clinical confirmation and objective therapeutic response observed in this study, the radiologic delay may significantly affect the correct axSpA management.

Table I. Frequency of inflammatory and structural lesions at initial and diagnostic imaging examinations.

Lesion	**Initial exam n (%)**	**Diagnostic exam n (%)**
Bone marrow edema	95 (54.0)	162 (92.0)
Facet joint capsulitis	78 (44.3)	122 (69.3)
Microerosions	72 (40.9)	112 (63.6)
Erosions	67 (38.1)	114 (64.8)
Interspinous enthesitis	65 (36.9)	103 (58.5)
Fat metaplasia	62 (35.2)	106 (60.2)

P87

2026 UPDATE OF THE ASAS MRI DEFINITIONS OF SACROILIAC JOINT AND SPINAL INVOLVEMENT FOR CLASSIFICATION OF AXIAL SPONDYLOARTHRITIS – A SYSTEMATIC LITERATURE REVIEW

Protopopov M.[1], Meghnathi B.[2], Sepriano A.[3], Diekhoff T.[4], Eshed I.[5], Machado P.M.[6], Poddubnyy D.[7], Baraliakos X.[8], Ramiro S.[9]
[1]Medical Clinic for Gastroenterology, Infectious Diseases and Rheumatology CBF, Charité – Universitätsmedizin Berlin, Berlin, Germany; [2]Smt. NHL Municipal Medical College, RheumaCARE, Marengo CIMS Hospital, Ahmedabad, India; [3]NOVA Medical School, Universidade Nova de Lisboa and Dept. of Rheumatology, Hospital de Egas Moniz - ULSO, Lisbon, Portugal; [4]Brandenburg Medical School Theodor Fontane, Dept. of Radiology, Rüdersdorf, Germany; [5]Dept. of Diagnostic Imaging, Sheba Medical Centre, Tel-Aviv University, Tel Aviv, Israel; [6]Dept. of Neuromuscular Diseases, University College London, London; NIHR University College London Hospitals Biomedical Research Centre, University College London Hospitals NHS Foundation Trust, London; Dept. of Rheumatology, Northwick Park Hospital, London North West University Healthcare NHS Trust, London, UK; [7]Division of Rheumatology, Dept. of Medicine, University of Toronto and Schroeder Arthritis Institute, University Health Network, Toronto, Canada; [8]Rheumazentrum Ruhrgebiet Herne, Ruhr Universität Bochum, Bochum, Germany; [9]Leiden University Medical Center, Leiden, The Netherlands

Introduction. To review evidence on diagnostic performance of MRI lesions informing the 2026 update of ASAS definitions of sacroiliac joint (SIJ) and spinal involvement in axial spondyloarthritis (axSpA).
Methods. We performed a systematic literature review of SIJ and spinal MRI lesions in suspected axSpA, using expert clinical diagnosis as the main reference standard; fulfilment of classification criteria was also accepted. Databases were searched from inception to 30 March 2026, and major rheumatology and radiology conference abstracts from 2024-2025 were screened. Two reviewers performed screening, data extraction and QUADAS-2 risk-of-bias assessment. Data for inflammatory, structural and combined lesions were extracted or calculated as two-by-two tables. Where at least one study was available per lesion, pooled sensitivity, specificity and likelihood ratios were estimated. Lesions from studies at low or unclear risk of bias were selected if LR+ was >6.0 and sensitivity >25%.
Results. The search identified 4,961 full-text studies and 68 abstracts. Overall, 88 unique studies contributed 327 candidate lesions (SIJ: 267; spine: 60). Thirty-six studies at low or unclear risk of bias provided 152 single lesions or combinations. Finally, 37 candidate lesions from 21 studies met thresholds; 35 (95%) concerned the SIJ. Individual inflammatory and structural lesions (BME, erosions, fat lesions backfill) were highly specific (0.93-1.00) with acceptable sensitivity (0.26-0.51); sclerosis and ankylosis did not fulfil thresholds, and sensitivity decreased with higher quantitative thresholds (Table I). Combined lesions were also highly specific (0.92-1.00), especially when presence of either inflammatory or structural lesions was considered (Table II). More combined than individual lesions had LR+ >10 (10/14 vs. 6/11). Only two spinal lesions met thresholds.
Conclusion. Several candidate MRI lesions showed good rule-in performance, driven by high specificity. Combinations of inflammatory and structural lesions performed particularly well. These findings will inform the 2026 update of ASAS MRI definitions for axSpA.

Table I. Performance of individual inflammatory and structural lesions (meta-analysis).

Lesion	n. of studies	n. of patients	% of axSpA	Sen (95% CI)	Sp (95% CI)	LR+ (95% CI)	LR- (95% CI)
BME ≥ 4 SIJ quadrants	4	906	54%	0.37 [0.19, 0.60]	0.97 [0.84, 1.00]	12.82 [3.05, 53.92]	0.65 [0.48, 0.88]
BME in ≥3 consecutive slices	2	729	55%	0.30 [0.26, 0.35]	0.99 [0.98, 1.00]	49.47 [12.33, 198.53]	0.70 [0.66, 0.75]
BME in ≥4 consecutive slices	1	86	80%	0.26 [0.16, 0.38]	1.00 [0.80, 1.00]	9.51 [0.60, 150.45]	0.76 [0.64, 0.89]
BME ≥4 SIJ quad or ≥3 consecutive slices	1	643	52%	0.31 [0.26, 0.36]	0.99 [0.98, 1.00]	47.35 [11.79, 190.24]	0.70 [0.65, 0.75]
Fat lesions in ≥ 4 SIJ quad	2	161	77%	0.51 [0.29, 0.73]	0.95 [0.81, 0.99]	9.46 [2.28, 39.27]	0.52 [0.32, 0.84]
Fat lesion in ≥2 consecutive slices	1	79	80%	0.41 [0.29, 0.54]	0.94 [0.70, 1.00]	6.60 [0.97, 45.06]	0.63 [0.49, 0.80]
Presence of homogenous fat lesion	3	239	61%	0.49 [0.36, 0.62]	0.95 [0.86, 0.98]	9.02 [3.16, 25.70]	0.54 [0.41, 0.70]
Erosions ≥2 SIJ quad	4	454	60%	0.50 [0.38, 0.61]	0.93 [0.88, 0.96]	7.30 [3.44, 15.52]	0.54 [0.42, 0.70]
Erosions in ≥3 consecutive slices	1	79	80%	0.37 [0.25, 0.50]	1.00 [0.79, 1.00]	12.48 [0.80, 195.21]	0.65 [0.53, 0.80]
Erosions in ≥4 consecutive slices	1	79	80%	0.30 [0.19, 0.43]	1.00 [0.79, 1.00]	10.36 [0.66, 162.98]	0.72 [0.60, 0.86]
Presence of backfill	4	965	64%	0.27 [0.13, 0.49]	0.99 [0.97, 1.00]	29.45 [12.12, 71.56]	0.74 [0.58, 0.94]

axSpA: axial spondyloarthritis; BME: bone marrow edema; CI: confidence interval; quad: quadrants; SIJ: sacroiliac joint; Sn: sensitivity; Sp: specificity

Table II. Performance of combinations of inflammatory and structural lesions (meta-analysis).

Lesion	n. of studies	n. of patients	% of axSpA	Sen (95% CI)	Sp (95% CI)	LR+ (95% CI)	LR- (95% CI)
BME AND erosion (similar to ASAS 2009 definition)	4	906	54%	0.37 [0.19, 0.60]	0.97 [0.84, 1.00]	12.82 [3.05, 53.92]	0.65 [0.48, 0.88]
BME ≥1 quad AND fat lesion ≥1 quad	2	729	55%	0.30 [0.26, 0.35]	0.99 [0.98, 1.00]	49.47 [12.33, 198.53]	0.70 [0.66, 0.75]
BME ≥2 quad AND/OR fat lesion ≥1 quad	1	86	80%	0.26 [0.16, 0.38]	1.00 [0.80, 1.00]	9.51 [0.60, 150.45]	0.76 [0.64, 0.89]
BME ≥2 quad AND/OR fat lesion ≥2 quad	1	643	52%	0.31 [0.26, 0.36]	0.99 [0.98, 1.00]	47.35 [11.79, 190.24]	0.70 [0.65, 0.75]
BME ≥3 quad AND/OR fat lesion ≥1 quad	2	161	77%	0.51 [0.29, 0.73]	0.95 [0.81, 0.99]	9.46 [2.28, 39.27]	0.52 [0.32, 0.84]
BME ≥3 quad AND/OR fat lesion ≥2 quad	1	79	80%	0.41 [0.29, 0.54]	0.94 [0.70, 1.00]	6.60 [0.97, 45.06]	0.63 [0.49, 0.80]
BME ≥2 quad AND/OR deep fat lesion (>1 cm) ≥1 quad	3	239	61%	0.49 [0.36, 0.62]	0.95 [0.86, 0.98]	9.02 [3.16, 25.70]	0.54 [0.41, 0.70]
BME AND homogenous fat lesion	4	454	60%	0.50 [0.38, 0.61]	0.93 [0.88, 0.96]	7.30 [3.44, 15.52]	0.54 [0.42, 0.70]
BME AND Fat lesion*	1	79	80%	0.37 [0.25, 0.50]	1.00 [0.79, 1.00]	12.48 [0.80, 195.21]	0.65 [0.53, 0.80]
AxSpA-typical structural AND inflammatory MRI lesions	7	1332	38%	0.60 [0.52, 0.69]	0.93 [0.87, 0.96]	8.92 [4.96, 16.04]	0.42 [0.35, 0.52]
2009 ASAS active OR Leiden structural	1	643	52%	0.46 [0.40, 0.51]	0.98 [0.96, 0.99]	23.44 [10.53, 52.22]	0.55 [0.50, 0.61]
ASAS MRI group active AND Leiden structural	1	643	52%	0.08 [0.05, 0.12]	1.00 [0.98, 1.00]	24.82 [3.39, 181.59]	0.92 [0.89, 0.95]
2009 ASAS active OR ASAS MRI Group chronic	1	643	52%	0.48 [0.42, 0.53]	0.97 [0.94, 0.98]	14.71 [7.91, 27.34]	0.54 [0.49, 0.60]
ASAS MRI group active AND chronic changes	1	643	52%	0.13 [0.09, 0.17]	0.99 [0.98, 1.00]	19.31 [4.71, 79.09]	0.88 [0.84, 0.92]

*subchondral OR homogenous OR distinct border – 2 out of 3
ASAS: The Assessment of SpondyloArthritis international Society; axSpA: axial spondyloarthritis; BME: bone marrow edema; CI: confidence interval; quad: quadrant; SIJ: sacroiliac joint; Sn: sensitivity; Sp: specificity.
2009 ASAS definition of active sacroiliitis: BME in 2 consecutive slices or 2 lesions on one slice, highly suggestive of axSpA.
ASAS MRI group definitions for active sacroiliitis: BME in ≥4 SIJ quadrants in any location or BME in the same location in ≥3 consecutive slices.
Leiden definition of structural changes: ≥3 erosions, ≥3 fat lesions or ≥5 fat lesions and/or erosions.
ASAS MRI group definition of chronic changes: erosions in ≥3 quadrants or erosion at the same location in ≥2 consecutive slices, or fat lesions in ≥5 SIJ quadrants or fat lesions in the same location in ≥3 consecutive slices.

P88

PREDICTORS OF PREVALENT AND INCIDENT RADIOGRAPHIC VERTEBRAL FRACTURES IN AXIAL SPONDYLOARTHRITIS

Richter G.[1], Maas F.[1], Wink F.[2], Bos R.[2], Spoorenberg A.[1], Arends S.[1]
[1]Dept. of Rheumatology and Clinical Immunology, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands; [2]Dept. of Rheumatology, Frisius Medical Center Leeuwarden, The Netherlands

Introduction/Objective. While most attention is paid to spinal radiographic progression, axial spondyloarthritis (axSpA) is also associated with an increased risk of vertebral fractures (VF). We investigated demographic, clinical and bone-related predictors of prevalent and incident radiographic VF over 2 years.
Methods. All consecutive axSpA patients from the GLAS cohort with available baseline radiographs between 2004 and 2012 were included. Patient and disease characteristics were part of the standardized GLAS protocol. Radiographs at baseline and 2-year follow-up were scored for VF (Genant method) and spinal radiographic damage (mSASSS) by two independent trained readers. Analysis of incident VF combined new VF and worsening of existing VF. BMD of the hip (total proximal femur) and lumbar spine (AP/lateral L1-L4) was measured using DXA.
Results. Of 433 patients, 66% were male, mean age 43.1±12.5 years, 91% radiographic axSpA, median disease duration 14.5 (7–24.6) years, mean ASDAS 3.15±1.08, 76% used NSAIDs and 56% started TNFi.
Prevalent radiographic VF were present in 97/433 patients (22%). The presence of VF was associated with older age, male sex, higher BMI, larger occiput-to-wall distance (OWD), lower hip BMD T-score, higher mSASSS and higher syndesmophyte count (Table I). In multivariable analysis, higher BMI, larger OWD and less frequent NSAID use were independent predictors of prevalent VF, while male sex and lower hip BMD T-score showed trends (Table I).
Incident VF occurred in 25/314 patients (8%) and was associated with older age, larger OWD, lower baseline lumbar (AP/lateral) BMD T-scores and less frequent NSAID use (Table II).
Conclusion. In our prospective observational cohort of axSpA patients, radiographic VF were frequently present. New or worsening VF occurred within only 2 years. In addition to the known risk factors, VF were associated with less frequent NSAID use at baseline, suggesting a potential protective effect.

Table I. Univariable and multivariable logistic regression of baseline characteristics associated with prevalent radiographic vertebral fracture.

Predictor	Univariable OR (95% CI)	p	Multivariable OR (95% CI)	p
Age, per year	1.03 (1.01–1.05)	**<0.001**	1.00 (0.98–1.02)	0.991
Male sex	2.03 (1.20–3.43)	**0.008**	1.80 (0.95–3.70)	0.073
BMI, per kg/m²	1.06 (1.01–1.12)	**0.015**	1.07 (1.01–1.15)	**0.033**
Disease duration	1.01 (0.99–1.03)	0.315		
HLA-B27 positive	0.70 (0.41–1.20)	0.197		
ASDAS	1.08 (0.87–1.34)	0.471		
Occiput-to-wall, per cm	1.08 (1.04–1.13)	**<0.001**	1.06 (1.02–1.11)	**0.006**
Hip BMD T-score, per unit	0.73 (0.57–0.93)	**0.011**	0.76 (0.56–1.03)	0.073
LS BMD AP T-score, per unit	0.97 (0.83–1.14)	0.775		
LS BMD lat T-score, per unit‡	0.90 (0.72–1.14)	0.397		
mSASSS total, per 10 pts	1.19 (1.05–1.34)	**0.006**		
mSASSS 1–9 vs no damage	2.32 (0.99–5.44)	0.053		
mSASSS ≥10 vs no damage	4.84 (2.04–11.47)	**0.001**		
Syndesmophyte count, per 2 pts	1.10 (1.02–1.18)	**0.007**		
NSAID use	0.74 (0.44–1.22)	0.235	0.54 (0.30–0.96)	**0.037**

OR: odds ratio; CI: confidence interval; LS: lumbar spine; mSASSS: modified Stoke Ankylosing Spondylitis Spine Score; OWD: occiput-to-wall distance. The multivariable column shows the model (n=372, 85 events; Nagelkerke R^2=0.14, AUC 0.70) including age, sex, BMI, NSAID use, occiput-to-wall distance and hip BMD T-score; OWD was entered as the measure of spinal damage instead of mSASSS, as both capture structural damage and cannot be entered together. Predictors with univariable p>0.3 were not entered; mSASSS and the overlapping syndesmophyte count were therefore not included. ‡LS lateral DXA only performed in Frisius MC Leeuwarden.

Table II. Univariable logistic regression of baseline predictors for incident VF during 2 years.

Predictor	Univariable OR (95% CI)	p
Age, per year	1.04 (1.01–1.07)	**0.023**
Male sex	1.53 (0.59–3.95)	0.383
BMI, per kg/m²	1.02 (0.92–1.12)	0.754
Disease duration	0.98 (0.94–1.03)	0.400
HLA-B27 positive	0.58 (0.23–1.47)	0.251
ASDAS	1.27 (0.86–1.88)	0.230
Occiput-to-wall, per cm	1.06 (1.01–1.11)	**0.020**
Hip BMD T-score, per unit	0.67 (0.43–1.05)	0.078
LS BMD AP T-score, per unit	0.70 (0.50–0.98)	**0.039**
LS BMD lat T-score, per unit‡	0.56 (0.34–0.92)	**0.022**
mSASSS total, per 10 pts	1.05 (0.83–1.33)	0.675
Syndesmophyte count, per 2 pts	1.02 (0.89–1.17)	0.771
NSAID use	0.35 (0.15–0.83)	**0.017**

OR: odds ratio; CI: confidence interval; LS: lumbar spine; OWD: occiput-to-wall distance. Incident VF (n=25/314, 8%) = new VF or worsening of VF grade. ‡LS lateral DXA only performed in Frisius MC Leeuwarden.

P89

MRI-ASSESSED STRUCTURAL DAMAGE PROGRESSION IN SACROILIAC JOINTS OF EUROPEAN ROUTINE CARE PATIENTS WITH AXIAL SPONDYLOARTHRITIS RECEIVING BIOLOGICAL OR CONVENTIONAL THERAPY

Steimer A.[1], Brändli J.[2], Micheroli R.[1], Hadsbjerg A.E.F.[3,4], Krabbe S.[3], Vladimirova N.[3,4], Eshed I.[5], Diekhoff T.[6], Lambert R.G.W.[7], Pedersen S.J.[3,4], de Hooge M.[17], Bubová K.[9], Gregová M.[9], Nissen M.J.[10], Möller B.[11], Rodrigues C.L.[2], Závada J.[9], Snoj Z.[12], Pintaric K.[12], Gudbjörnsson B.[13], Rotar Z.[14,15], Sudol-Szopinska I.[16], Gosvig K.[17], Møller J.M.[17], Wetterslev M.[3], Wichuk S.[18], Hetland M.L.[3,4], Ørnbjerg L.M.[3], Østergaard M.[3,4], Ciurea A.[1]

[1]Dept. of Rheumatology, University Hospital of Zurich, University of Zurich, Zurich; [2]Swiss Clinical Quality Management in Rheumatic Diseases Foundation, Zurich, Switzerland; [3]Copenhagen Center for Arthritis Research, Center for Rheumatology and Spine Diseases, Centre for Head and Orthopaedics, Rigshospitalet, Glostrup; [4]Dept. of Internal Medicine, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen, Denmark; [5]Dept. of Diagnostic Imaging, Sheba Medical Center, Tel Hashomer affiliated with The Gray School of Medicine, Tel Aviv University, Tel Aviv, Israel; [6]Dept. of Radiology, Immanuel Clinic Ruedersdorf, University Hospital of Brandenburg Medical School, Ruedersdorf bei Berlin, Brandenburg, Germany; [7]Dept. of Radiology and Diagnostic Imaging, University of Alberta, Edmonton, Canada; [8]Dept. of Rheumatology, Ghent University, Ghent, Belgium; [9]Institute of Rheumatology and Dept. of Rheumatology, First Faculty of Medicine Charles University, Prague, Czech Republic; [10]Rheumatology Dept., Geneva University Hospital, Geneva; [11]Dept. of Rheumatology and Immunology, Inselspital, University Hospital Bern, Bern, Switzerland; [12]Institute of Radiology, UMC Ljubljana, Ljubljana, Slovenia; [13]Centre for Rheumatology Research, Landspitali University Hospital and Faculty of Medicine, University of Iceland, Reykjavik, Iceland; [14]Dept. of Rheumatology, University Medical Centre Ljubljana, Ljubljana; [15]Faculty of Medicine, University of Ljubljana, Ljubljana, Slovenia; [16]Dept. of Radiology, National Institute of Geriatrics, Rheumatology and Rehabilitation, Warsaw, Poland; [17]Dept. of Radiology, Copenhagen University Hospital Herlev-Gentofte, Herlev, Denmark; [18]Division of Rheumatology, University of Alberta, Edmonton, Canada

Introduction. We aimed to evaluate progression of MRI structural sacroiliac joint (SIJ) lesions during routine clinical care in patients with axial spondyloarthritis (axSpA).

Methods. Bionaive patients diagnosed with axSpA from five European countries participating in the EuroSpA Research Collaboration Network were included if a follow-up SIJ-MRI was available 1-4 years after a first MRI. SIJ-MRIs were scored centrally by two trained readers blinded to chronology, using the Spondyloarthritis Research Consortium of Canada SIJ inflammation index and structural score. Additionally, readers indicated whether each MRI was indicative of axSpA. During follow-up, patients received either biologic disease-modifying antirheumatic drugs (bDMARDs) or conventional therapy (*e.g.* NSAIDs, physical therapy).

Results. Among 179 axSpA patients relevant structural changes were observed exclusively in those with MRIs indicative of axSpA (N=112). Baseline scores for bone marrow oedema (BME), fat lesions, and erosions were higher in patients initiating a bDMARD (N=53) compared with those receiving conventional therapy (N=59) (Table I). In bDMARD-treated patients, mean (SD) changes in scores over a 2-year follow-up were -14.7 (17.4) for BME, 1.2 (3.6) for fat lesions, -2.4 (5.1) for erosions, 1.6 (3.4) for backfill, and 0.2 (1.2) for ankylosis. Corresponding changes in scores in conventionally treated patients were -3.0 (14.6), 0.03 (3.1), -0.04 (3.5), 0.9 (2.6), and 0.7 (2.1), respectively. Net ankylosis progression (SPARCC score change ≥2) occurred in 9% of conventionally treated patients, compared with 6% of patients receiving bDMARD therapy. Baseline backfill and ankylosis correlated with progression of ankylosis in conventionally treated patients (Spearman's rank correlation coefficients R=0.38 and R=0.42, respectively), but not in bDMARD-treated patients (R=-0.17 and R=0.09) (Fig. 1).

Conclusion. Baseline backfill was associated with ankylosis progression in conventionally but not bDMARD-treated patients, suggesting a potential dissociation between backfill and ankylosis under biological therapy. Further studies are needed to establish potential causality.

Table I. Baseline characteristics at first MRI of the sacroiliac joints in bDMARD-naive patients, stratified by type of treatment during follow-up.

	Baseline MRI indicative of axSpA n=112				
	Conventional treatment n=59		bDMARD treatment n=53		p-value
Time interval between MRIs, years	59	2.3 (0.8)	53	1.8 (0.8)	<0.001
Male sex, N (%)	59	43 (72.9)	53	37 (69.8)	0.83
Age, years	59	34.9 (9.0)	53	38.1 (11.1)	0.13
Symptom duration, years	46	7.5 (8.4)	41	9.1 (8.3)	0.18
HLA-B27 positive, N (%)	58	46 (79.3)	52	41 (78.8)	1.00
ASDAS	25	2.5 (1.0)	33	3.8 (0.8)	<0.001
C-reactive protein, mg/l	30	8.0 (6.6)	35	17.6 (21.9)	0.06
Current smoking, N (%)	58	17 (29.3)	53	19 (35.8)	0.77
Body mass index	36	24.1 (4.0)	51	25.5 (4.3)	0.13
ASAS-positive MRI (active sacroiliitis), N (%)	59	46 (78.0)	53	41 (77.4)	1.00
Radiographic axSpA, N (%)	27	8 (29.6)	22	9 (40.9)	0.75
SPARCC BME score (range 0-72), median (IQR)	59	12.5 (4.3; 24)	53	17.0 (9.0; 34.5)	0.047
SPARCC sacroiliac joints structural score					
• Erosion (range 0-40), median (IQR)	59	4.5 (1.5; 8.3)	53	6.5 (3.5; 12.5)	0.04
• Fat lesion (range 0-40), median (IQR)	59	2.0 (0.0; 6.5)	53	4.0 (0.5; 10.0)	0.048
• Backfill (range 0-20), median (IQR)	59	0.0 (0.0; 1.3)	53	0.0 (0.0; 2.5)	0.73
• Ankylosis (range 0-20), median (IQR)	59	0.0 (0.0; 0.3)	53	0.0 (0.0; 0.0)	0.33

Except where indicated otherwise, values are the mean (SD). Readers defined if an MRI was indicative of axSpA. In case of disagreement, an adjudication was performed by an expert radiologist.
ASDAS: Ankylosing Spondylitis Disease Activity Score; axSpA: axial spondyloarthritis; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; bDMARD: biologic disease-modifying antirheumatic drug; BME: bone marrow oedema; EMM: extra-musculoskeletal manifestations; FU: follow-up (MRI interval); HLA-B27: human leucocyte antigen B27; MRI: magnetic resonance imaging.

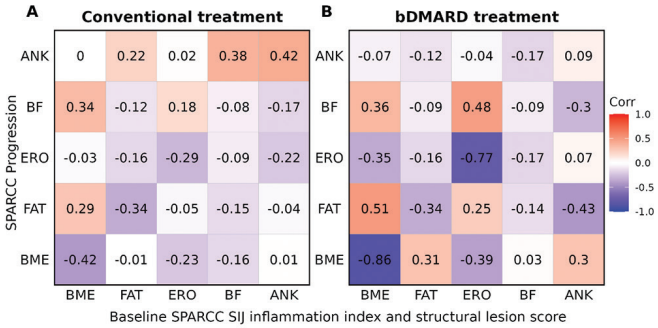


Fig. 1. Correlation matrix demonstrating associations SPARCC sacroiliac inflammation index and structural lesion score at baseline on the x-axis and the progression of individual sacroiliac joint lesions on the y-axis in patients with baseline MRI indicative of axSpA.
A: Correlation in bionaive patients on conventional treatment during the MRI interval.
B: Correlations in patients treated with a first bDMARD during the MRI interval. Spearman's rank correlation is used with the following interpretation of coefficients: 0 to 0.19 very weak association; 0.2 to 0.39 weak association; 0.4 to 0.59 moderate association; 0.6 to 0.79 strong association; 0.8 to 1 very strong association. ANK: ankylosis; axSpA: axial spondyloarthritis; bDMARD: biologic disease-modifying antirheumatic drug; BF: backfill; BME: bone marrow oedema; ERO: erosions; FAT: fat lesions; SCL: sclerosis; SPARCC: Spondyloarthritis Research Consortium of Canada.

P90

NAILFOLD CAPILLAROSCOPY REVEALS A DISTINCT MICROVASCULAR SIGNATURE IN PSORIATIC ARTHRITIS COMPARED TO PSORIASIS: A CASE-CONTROL STUDY

Teixeira P.M.[1,3], Vilafanha C.[1,3], Loureiro M.[2], Eugénio G.[1,3], Barcelos A.[1,3,4]
[1]Rheumatology Dept., Unidade Local de Saúde da Região de Aveiro, Aveiro; [2]Dermatology Dept., Unidade Local de Saúde da Região de Aveiro, Aveiro; [3]Centro de Investigação em Reumatologia de Aveiro, Centro Académico Clínico Egas Moniz Health Alliance, Aveiro; [4]EpiDoc Unit, NOVA Medical School, NOVA University Lisbon, Lisbon, Portugal

Introduction. Psoriatic disease is associated with systemic inflammation and microvascular dysfunction. Nailfold capillaroscopy (NC) non-invasively assesses microcirculation using standardized EULAR definitions. Still, data on psoriatic disease are scarce, and previous reviews have been unable to reliably distinguish psoriatic arthritis (PsA) from psoriasis without arthritis (PsO). We aimed to compare NC findings in PsA, PsO, and healthy controls and identify factors independently associated with NC abnormalities.
Materials and methods. We conducted an observational case-control study comprising three groups: PsA (CASPAR criteria), PsO (dermatologist-diagnosed, without arthritis), and healthy controls. NC was assessed with EULAR definitions (enlarged and giant capillaries, microhaemorrhages, capillary loss, disorganization, ramified/neoangiogenic capillaries). Demographics, comorbidities, disease duration, and treatment were recorded. PsA patients were compared with healthy controls and PsO; logistic regression identified factors independently associated with any NC abnormality.
Results. We included 46 PsA, 27 PsO, and 25 controls (n=98). Age (≈58 years) and comorbidities were comparable between groups, except for obesity (Table I). Any NC abnormality was more frequent in PsA (67%) than in controls (32%, p=0.006) and PsO (22%, p<0.001), with more abnormalities per patient (p<0.001). Enlarged capillaries (41% vs. 15%, p=0.021) and microhaemorrhages (30% vs. 7%, p=0.038) were more frequent in PsA than PsO; PsO did not differ from controls. In multivariable analysis, PsA (adjusted OR 5.0 [1.5–16.9], p=0.009) and hypertension (4.2 [1.3–13.0], p=0.014) were independently associated with any NC abnormality.
Conclusion. NC revealed a distinct microvascular signature in PsA - more frequent enlarged capillaries, microhaemorrhages, and overall abnormality burden, which was absent in PsO. PsA and hypertension were independently associated with NC abnormalities. Future large-scale longitudinal studies are required to confirm these findings and further elucidate the prognostic significance of NC in disease assessment and progression.

Table I. Demographics, disease characteristics, treatment and nailfold capillaroscopy findings across groups.

	Controls (n=25)	PsO (n=27)	PsA (n=46)	Controls vs. PsA	PsO vs. PsA
Demographics					
Age, years (mean±SD)	57.7±17.0	57.3±13.2	58.5±12.9	0.83	0.71
Female, n (%)	16 (64)	4 (15)	17 (37)	0.046	0.061
BMI, kg/m² (m ean±SD)	24.5±4.5	28.4±7.4	30.5±5.1	<0.001	0.41
Smoking, ever, n (%)	4 (16)	12 (44)	15 (41)	0.052	0.80
Alcohol, n (%)	0 (0)	9 (33)	9 (22)	0.010	0.40
Comorbidities, n (%)					
Obesity	3 (12)	3 (11)	26 (57)	<0.001	<0.001
Hypertension	5 (20)	9 (33)	15 (33)	0.29	1.00
Dyslipidemia	7 (28)	13 (48)	20 (43)	0.31	0.81
Diabetes	1 (4)	4 (15)	4 (9)	0.65	0.46
Disease characteristics and treatment					
Psoriasis duration, years	—	24.4±16.3	24.9±12.4	—	0.53
Arthritis duration, years	—	—	17.9±8.7	—	—
Nail involvement, n (%)	—	7 (26)	18 (39)	—	0.31
csDMARD, n (%)	—	2 (7)	29 (63)	—	<0.001
b/tsDMARD, n (%)	—	12 (44)	18 (39)	—	0.81
Nailfold capillaroscopy, n (%)					
Enlarged capillaries (>20 µm)	**4 (16)**	**4 (15)**	**19 (41)**	**0.036**	**0.021**
Giant capillaries (>50 µm)	1 (4)	1 (4)	1 (2)	1.00	1.00
Microhaemorrhages	**3 (12)**	**2 (7)**	**14 (30)**	**0.144**	**0.038**
Capillary loss (density <7/mm)	3 (12)	2 (7)	9 (20)	0.52	0.20
Architectural disorganization	2 (8)	2 (7)	9 (20)	0.31	0.20
Neoangiogenic	1 (4)	0 (0)	0 (0)	0.36	1.00
Any abnormality	**8 (32)**	**6 (22)**	**31 (67)**	**0.006**	**<0.001**
Number of abnormalities (mean±SD)	**0.56±1.04**	**0.41±0.97**	**1.13±1.00**	**0.007**	**<0.001**

BMI: body mass index; b/tsDMARD: biologic/targeted-synthetic disease-modifying antirheumatic drug; csDMARD: conventional synthetic disease-modifying antirheumatic drug; PsA: psoriatic arthritis; PsO: psoriasis; SD: standard deviation.

P91

DIFFERENCE IN INFLAMMATORY LESIONS ON MRI OF THE SACROILIAC JOINTS AT BASELINE AND AFTER 2 YEARS BETWEEN PATIENTS WITH EARLY AXSPA AND NON-AXSPA CHRONIC BACK PAIN

Tekeoglu S.[1,2], Ayan G.[1,3], de Hooge M.[1,4], de Bruin L.[1], van Lunteren M.[1], Bento da Silva A.[1], Reijnierse M.[5], Landewé R.[6,7], Berg I.J.[8], Turesson C.[9], Ramonda R.[10], van Gaalen F.[1], Ramiro S.[1,7]
[1]Leiden University Medical Center, Dept. of Rheumatology, Leiden, The Netherlands; [2]Biruni University Hospital, Dept. of Rheumatology, Istanbul; [3]Ankara Research and Training Hospital, Ankara, Turkey; [4]Ghent University Hospital, Dept. of Rheumatology, Ghent, Belgium; [5]Leiden University Medical Center, Dept. of Radiology, Leiden; [6]Amsterdam University Medical Center, Dept. of Rheumatology, Amsterdam; [7]Zuyderland Medical Center, Heerlen, The Netherlands; [8]Diakonhjemmet Hospital, Center for Treatment of Rheumatic and Musculoskeletal Diseases (REMEDY), Oslo, Norway; [9]Lund University, Rheumatology, Dept. of Clinical Sciences, Malmö, Sweden; [10]University of Padova, Rheumatology Unit, Dept. of Medicine-DIMED, Padova, Italy

Introduction. Detection of bone marrow oedema (BME) on MRI-SIJ is crucial for the early diagnosis of axSpA. However, BME may also occur in non-axSpA chronic back pain (CBP), limiting specificity. We investigated differences in MRI-SIJ locations of BME and inflammatory lesion scores at baseline and over 2 years (2Y) between axSpA and non-axSpA CBP.
Methods. Patients from the SPACE cohort (CBP ≥3 months and ≤2 years, starting <45 years) were diagnosed as axSpA or non-axSpA CBP by their rheumatologist at 2-year follow-up. MRI-SIJ were centrally assessed by three blinded readers using the Spondyloarthritis Research Consortium of Canada (SPARCC) method. The BME location was compared at baseline and 2Y. Change of BME over time was assessed with Generalized Estimating Equations (GEE), stratified for diagnosis (if time-by-diagnosis interaction p-value <0.15), and adjusted for confounders and readers.
Results. Baseline MRI-SIJ was available for 447 patients (age 30 [8] years; 45% male; 55% HLA-B27 positive; 296 axSpA, 151 non-axSpA); 372 had 2Y MRI-SIJ, and 359 had both. The BME location differed between groups at both time points. In axSpA, lesions were broadly distributed, whereas in non-axSpA, BME was rare and mainly confined to the ventral and middle

superior sacral regions (Fig. 1a). Patients with axSpA had higher SPARCC-SIJ scores at both time points, and scores decreased over 2 years only in axSpA (Table I). In adjusted GEE analysis, axSpA diagnosis was associated with higher SPARCC-SIJ scores (β 5.6, 95%CI 4.48;6.71) vs. non-axSpA (reference), with a significant time-by-diagnosis interaction indicating a larger decline in axSpA over time (β -1.74/year, 95%CI -2.27;-1.21) than in non-axSpA (β 0.02/year, 95%CI -0.05;0.10) (Fig. 1b).

Conclusion. MRI-based lesion location and longitudinal change differentiate axSpA from non-axSpA CBP. In axSpA, BME is more frequent, more extensive, and decreases over time; in non-axSpA CBP, it is infrequent, anatomically limited, and stable.

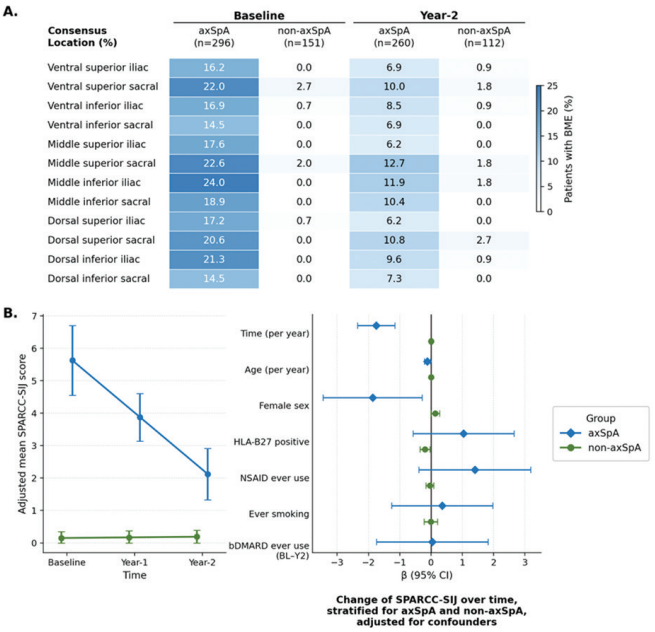


Fig. 1. A. Location of BME on MRI-SIJ in patients with axSpA and non-axSpA chronic back pain at baseline and 2 years.
Values represent the percentage of patients with BME in each sacroiliac joint region based on the consensus assessment (≥2 of 3 readers).
B. Adjusted mean SPARCC-SIJ scores over time (left) and stratified fully-adjusted GEE models (right). Two-year change scores were calculated only in patients with both scans available. Predicted margins were derived from the reader adjusted GEE model.
axSpA: axial spondyloarthritis; bDMARD: biologic disease-modifying antirheumatic drug; BL: baseline; BME: bone marrow edema; GEE: generalised estimating equation, HLA-B27: human leukocyte antigen; MRI-SIJ: magnetic resonance imaging of the sacroiliac joints; NSAID: non-steroidal anti-inflammatory drug; SPARCC-SIJ: Spondyloarthritis Research Consortium of Canada method score for sacroiliac joints; Y2: year 2.

Table I. MRI-SIJ inflammatory lesion status scores at baseline and at 2 years, and 2-year change scores, in patients with axSpA and non-axSpA chronic back pain. Values represent the average for continuous variables and consensus (≥2 of 3 readers) for dichotomous variables.

	Baseline			Year-2			Baseline – Year-2		
	axSpA (N=296)	non-axSpA (N = 151)	p-value	axSpA (N=260)	non-axSpA (N = 112)	p-value	axSpA (N=248)	non-axSpA (N = 111)	p-value
SPARCC-SIJ total (0–72)	5.6 (9.2)	0.2 (0.6)	<0.001	2.4 (4.9)	0.2 (0.9)	<0.001	↓3.4 (8.5)	↑0.05 (0.9)	<0.001
Quadrants with BME (0–48)	4.7 (7.2)	0.2 (0.6)	<0.001	2.2 (4.1)	0.2 (0.9)	<0.001	↓2.7 (6.6)	↑ 0.04 (0.8)	<0.001
Quadrants with deep/intense BME (0–24)	1.0 (2.5)	0 (0.0)	<0.001	0.3 (1.1)	0 (0.1)	0.003	↓0.7 (2.3)	↑0.01 (0.1)	0.004
Any BME present (yes/no)	138 (46.6)	6 (4.0)	<0.001	90 (34.6)	7 (6.3)	<0.001	–	–	–
Any deep/intense BME present (yes/no)	54 (18.2)	0 (0.0)	<0.001	21 (8.0)	0 (0.0)	0.001	–	–	–
Positive MRI-SIJ (ASAS) (yes/no)	124 (42.0)	4 (2.7)	<0.001	81 (31.2)	3 (2.7)	<0.001	–	–	–

Continuous variables are presented as mean (SD); dichotomous variables are presented as n (%).
ASAS: Assessment of Spondyloarthritis International Society; axSpA: axial spondyloarthritis; BME: bone marrow edema; MRI-SIJ: magnetic resonance imaging of the spine; SPARCC-Spine: Spondyloarthritis Research Consortium of Canada method score for sacroiliac joints.

P92

DIFFERENCE IN INFLAMMATORY LESIONS ON MRI OF THE SPINE AT BASELINE AND AFTER 2 YEARS BETWEEN PATIENTS WITH EARLY AXSPA AND NON-AXSPA CHRONIC BACK PAIN

Tekeoglu S.[1,2], Ayan G.[1,3], de Hooge M.[1,4], de Bruin L.[1], van Lunteren M.[1], Bento da Silva A.[1], Reijnierse M.[5], Landewé R.[6,7], Berg I.J.[8], Turesson C.[9], Ramonda R.[10], van Gaalen F.[1], Ramiro S.[1,7]
[1]Leiden University Medical Center, Dept. of Rheumatology, Leiden, The Netherlands; [2] Biruni University Hospital, Dept. of Rheumatology, Istanbul, Turkey; [3]Ankara Research and Training Hospital, Ankara, Turkey; [4]Ghent University Hospital, Dept. of Rheumatology, Ghent, Belgium; [5]Leiden University Medical Center, Dept. of Radiology, Leiden; [6]Amsterdam University Medical Center, Dept. of Rheumatology, Amsterdam; [7]Zuyderland Medical Center, Heerlen, The Netherlands; [8]Diakonhjemmet Hospital, Center for Treatment of Rheumatic and Musculoskeletal Diseases (REMEDY), Oslo, Norway; [9]Lund University, Rheumatology, Dept. of Clinical Sciences, Malmö, Sweden; [10]University of Padova, Rheumatology Unit, Dept. of Medicine-DIMED, Padova, Italy

Introduction. Bone marrow oedema (BME) on MRI-spine may provide additional information on inflammation in early axSpA but may also occur in non-axSpA chronic back pain (CBP), limiting specificity. We investigated differences in locations with BME and inflammatory lesion scores on MRI-spine at baseline and over 2 years (2Y) between early axSpA and non-axSpA CBP.
Methods. Patients from the SPACE cohort (CBP ≥3 months and ≤2 years, starting <45 years) were diagnosed with axSpA or non-axSpA CBP by their rheumatologist after 2-year follow-up. AxSpA-suspected inflammatory MRI-spine lesions were centrally assessed by two readers, blinded to image chronology and clinical data, using the Spondyloarthritis Research Consortium of Canada (SPARCC) method. The BME location was compared at baseline and 2Y. Change of BME over time was assessed with Generalized Estimating Equations (GEE), stratified for diagnosis (if time-by-diagnosis interaction *p*-value <0.15), and adjusted for confounders and readers.
Results. Baseline MRI-spine was available for 448 patients (age 30 [8] years; 45% male; 55% HLA-B27+; 296 axSpA, 152 non-axSpA); at 2Y, 372 had MRI-spine and 358 had both. BME was more frequent in axSpA than in non-axSpA at both time points. In axSpA, lesions were predominantly located in the anterior thoracic and upper lumbar regions; in non-axSpA, BME was rare (Fig. 1a). AxSpA patients had higher SPARCC-spine scores at baseline and 2Y, and scores decreased over 2Y only in axSpA (Table I). In adjusted GEE analysis, axSpA diagnosis was associated with higher SPARCC-spine scores (β 2.09, 95%CI 1.33;2.85) vs. non-axSpA (reference), with a significant time-by-diagnosis interaction indicating a larger decline over time in axSpA (β -0.54/year, [-0.90;-0.19]) than non-axSpA (β 0.08/year, [-0.08;0.24]).
Conclusion. In this early CBP cohort, MRI-spine findings differentiate axSpA from non-axSpA. In axSpA, BME is more frequent, predominantly anterior thoracic and upper lumbar, and decreases over time, whereas in non-axSpA, it is rare and stable over time.

Table I. MRI-Spine inflammatory lesion status scores at baseline and at 2 years, and 2-year change scores, in patients with axSpA and non-axSpA chronic back pain. Values represent the average for continuous variables and consensus for dichotomous variables. *

	Baseline			Year-2			Baseline – Year-2		
	axSpA (N=296)	non-axSpA (N = 152)	p-value	axSpA (N=260)	non-axSpA (N = 112)	p-value	axSpA (N=248)	non-axSpA (N = 110)	p-value
SPARCC-Spine total (0–414)	2.9 (6.5)	0.5 (1.1)	<0.001	1.7 (4.8)	0.7 (1.5)	0.03	↓0.91 (5.0)	↑0.15 (1.7)	0.03
Quadrants with BME (0–276)	2.5 (5.7)	0.5 (1.1)	<0.001	1.5 (4.3)	0.6 (1.2)	0.02	↓0.80 (4.5)	↑0.06 (1.4)	0.05
Quadrants with deep/intense BME (0–138)	0.3 (1.2)	0.02 (0.2)	0.001	0.2 (0.7)	0.1 (0.4)	0.34	↓0.11(0.9)	↑0.09 (0.5)	0.04
Any BME present (yes/no)	70.0 (23.6	8.0 (5.3)	<0.001	39.0 (15.0)	8.0 (7.1)	0.04	–	–	–
Any deep/intense BME present (yes/no)	11.0 (3.7)	1.0 (0.7)	0.07	7.0 (2.7)	2.0 (1.8)	0.73	–	–	–

Continuous variables are presented as mean (SD); dichotomous variables are presented as n (%).
*A positive consensus required agreement of both readers; absence was assigned if at least one reader scored the lesion as absent, whereas cases with one positive and one missing score were treated as missing.
axSpA: axial spondyloarthritis; BME: bone marrow edema; MRI-Spine: magnetic resonance imaging of the spine; SPARCC-Spine: Spondyloarthritis Research Consortium of Canada method score for spine.

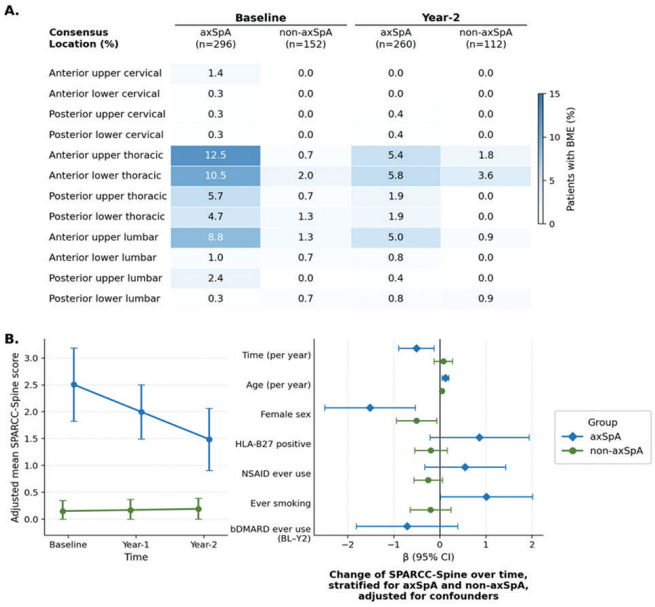


Fig. 1. A. Location of BME on MRI-Spine in patients with axSpA and non-axSpA chronic back pain at baseline and 2 years.
Values represent the percentage of patients with BME in each spine region based on the consensus assessment. A positive consensus required agreement of both readers; absence was assigned if at least one reader scored the lesion as absent, whereas cases with on epositive and one missing score were treated as missing.
B. Adjusted mean SPARCC-Spine scores over time (left) and stratified fully-adjusted GEE models (right). Two-year change scores were calculated only in patients with both scans available. Predicted margins were derived from the reader adjusted GEE model.
axSpA: axial spondyloarthritis; bDMARD: biologic disease-modifying antirheumatic drug; BL: baseline; BME: bone marrow edema; GEE: generalised estimating equation, HLA-B27: human leukocyte antigen; MRI-Spine: magnetic resonance imaging of the spine; NSAID: non-steroidal anti-inflammatory drug; SPARCC-SIJ: Spondyloarthritis Research Consortium of Canada method score for sacroiliac joints; Y2: year 2.

P93

SACROILIAC JOINT ANATOMY AS A DIAGNOSTIC MIMIC: BUTTOCK PAIN ASSOCIATIONS WITHOUT HIGHER QUANTITATIVE MRI LESION BURDEN IN AXSPA

Aksoy Ö.T.[1], Aysin İ.K.[2], Kocaayan H.[1], Yoğurtçu Ö.[1], Tosun Ö.[3], Solmaz D.[1], Akar S.[1]
[1]Dept. of Internal Medicine, Division of Rheumatology, İzmir Katip Çelebi University Faculty of Medicine, İzmir; [2]Rheumatology Clinic, Balıkesir State Hospital, Balikesir; [3]Dept. of Radiology, İzmir Katip Çelebi University Faculty of Medicine, İzmir, Turkey

Objective. To determine whether sacroiliac joint (SIJ) anatomical variants and lumbosacral transitional vertebrae (LSTV) are associated with buttock pain and increased quantitative SIJ MRI lesion burden in axial spondyloarthritis (axSpA), and to explore a diagnostic mimic phenotype in non-SpA.
Methods. We analysed 172 consecutive adults undergoing SIJ MRI: radiographic axSpA (r-axSpA; n=80), non-radiographic axSpA (nr-axSpA; n=38) and non-SpA (n=54). SIJ variants were classified on MRI by two blinded readers; a CT-MRI sub-study (n=67) confirmed excellent agreement for MRI-based variant assessment (κ=0.964). In axSpA, SPARCC inflammation and Berlin/SPARCC structural scores were applied. Associations with buttock pain were tested using multivariable logistic regression adjusted for age, sex, HLA-B27 status, axSpA subtype, and SPARCC inflammation. Non-SpA status was confirmed after at least 12 months of follow-up.
Results. In axSpA (n=118), dysmorphic SIJ morphology was independently associated with buttock pain (aOR 3.36, 95% CI 1.23–9.17; p=0.018), but not with higher quantitative inflammatory or structural lesion burden (Table I). Sensitivity analyses showed no ipsilateral lesion excess in unilateral variants and no mid/posterior compartment score differences by dysmorphic morphology. In non-SpA, dysmorphic morphology was the dominant correlate of buttock pain (aOR 37.20, 95% CI 4.48–309.23; p<0.001; Table I).
Accessory SIJ was associated with bone marrow oedema prevalence but not independently with buttock pain after adjustment; no non-SpA participant fulfilled ASAS MRI criteria for active sacroiliitis. Representative MRI appearances are shown in Figure 1.
Conclusion. Dysmorphic SIJ morphology is associated with buttock pain more consistently than with quantitative MRI lesion burden in axSpA and may contribute to diagnostic ambiguity in non-SpA. Recognising this anatomy-driven mimic pattern may help avoid overattribution of buttock pain or minor MRI lesions to inflammatory sacroiliitis.

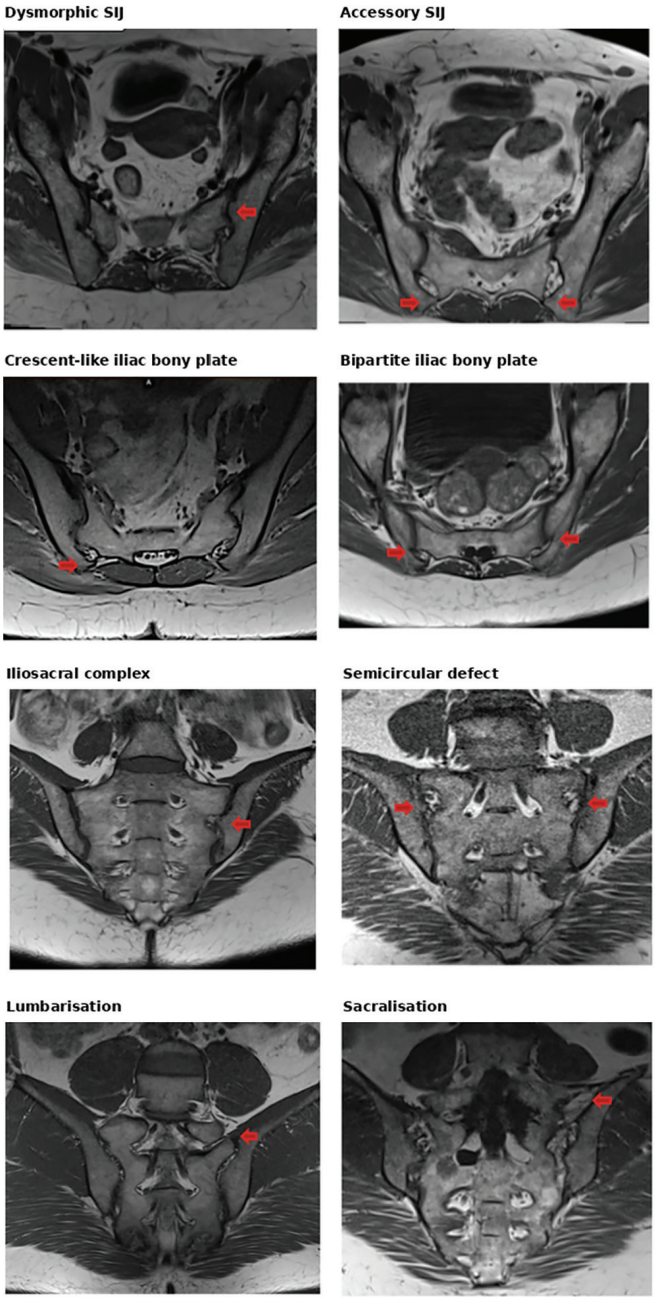


Fig. 1. Representative MRI examples of SIJ anatomical variants and LSTV.
Red arrows indicate the relevant anatomical feature. Dysmorphic SIJ, accessory SIJ, crescent-like iliac bony plate and bipartite iliac bony plate were assessed on semi-axial images.

Table I. Key adjusted associations with buttock pain and MRI sensitivity analyses.

Analysis	Endpoint or comparison	Result
axSpA adjusted model	Dysmorphic SIJ and buttock pain	aOR 3.36 (1.23–9.17); p=0.018
non-SpA adjusted model	Dysmorphic SIJ and buttock pain	aOR 37.20 (4.48–309.23); p<0.001
non-SpA adjusted model	Accessory SIJ and buttock pain	aOR 6.32 (0.90–44.15); p=0.062
MRI sensitivity, axSpA	Ipsilateral vs. contralateral SPARCC inflammation in unilateral variants	p=0.453
MRI sensitivity, axSpA	Dysmorphic present vs absent: Berlin mid/posterior compartment scores	p≥0.087

Values are aOR (95% CI) unless otherwise specified. The axSpA model was adjusted for age, sex, HLA-B27 status, axSpA subtype and SPARCC inflammation; the non-SpA model included age, sex, dysmorphic SIJ and accessory SIJ.
aOR: adjusted odds ratio; CI: confidence interval; SIJ: sacroiliac joint; SPARCC: Spondyloarthritis Research Consortium of Canada.

P94

PERFORMANCE OF MRI-BASED SYNTHETIC CT IN DETECTING SPINAL NEW BONE FORMATION: A COMPREHENSIVE COMPARISON WITH T1-WEIGHTED MRI, LOW-DOSE CT, AND RADIOGRAPHY

Willesen S.T.[1,2], Møller J.M.[3], Gosvig K.[3], Pedersen S.J.[1,2], Hadsbjerg A.E.F.[1,2], Georgiadis S.[1], Østergaard M.[1,2]
[1]Copenhagen Center for Arthritis Research, Center for Rheumatology and Spine Diseases, Rigshospitalet, Glostrup; [2]Dept. of Clinical Medicine, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen; [3]Dept. of Radiology, Copenhagen University Hospital at Herlev Gentofte, Copenhagen, Denmark

Introduction. Assessment of spinal new bone formation (NBF) in spondyloarthritis is limited by current imaging methods. MRI-based synthetic computed tomography (sCT) provides radiation-free, CT-like bone imaging (Fig. 1) but requires further validation. This study aims to compare sCT, T1-weighted MRI (T1wMRI), low-dose CT (ldCT), and radiography for detecting spinal NBF in participants with axial spondyloarthritis (axSpA), psoriatic arthritis (PsA), rheumatoid arthritis (RA), and healthy controls (HC).

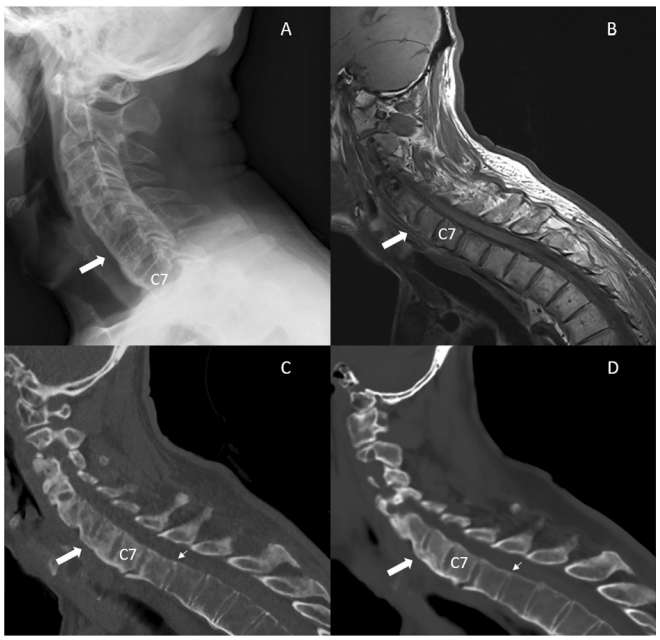


Fig. 1. Radiography (**A**), T1-weighted MRI (T1wMRI, **B**), low-dose CT (**C**), and MRI-based synthetic CT (**D**) of the cervical and upper thoracic spine in a 61-year-old male with axial spondyloarthritis.
New bone formation is present in all four images on several levels (*e.g.* on level C5/C6, large arrows); however, it is not visible below the C7 level on radiography and appears much more subtle on T1wMRI (B) compared with low-dose CT (C) and MRI-based synthetic CT (D). In the latter two modalities, new bone formation is more clearly depicted (*e.g.* posterior ankylosis at level T1/T2, short arrows), allowing visualization of different types and morphologies, which are difficult to characterize on T1wMRI and radiography. Note: The slice position differs slightly between modalities.

Table I. Performance measures for detecting new bone formation – including occurrence, sensitivity and specificity (with low-dose CT as the reference standard), inter-reader and intra-reader reliability – across low-dose CT, MRI-based synthetic CT, T1-weighted MRI, and radiography in patients with inflammatory arthritis and healthy controls.

Means, all readers	All participants, n=69	Axial spondyloarthritis, n=30	Psoriatic arthritis, n=19	Rheumatoid arthritis, n=10	Healthy controls, n=10
Total cervical spinal segment/thoracic spinal segment/lumbar spinal segment					
Low-dose CT					
Occurrence of NBF (mean per participant)	25.8 5.3/15.1/5.4	35.6 8.0/20.1/7.4	16.5 3.2/10.1/3.2	23.2 4.4/13.9/4.8	17.0 2.2/10.6/4.2
Inter-reader reliability (ICC)	0.94 0.86/0.93/0.96	0.93 0.85/0.92/0.97	0.97 0.85/0.98/0.97	0.88 0.89/0.88/0.59	0.91 0.66/0.92/0.92
Inter-reader reliability (Kappa)	0.69 0.64/0.69/0.72	0.66 0.66/0.63/0.74	0.71 0.65/0.72/0.75	0.69 0.50/0.77/0.64	0.74 0.63/0.79/0.68
MRI-based synthetic CT					
Occurrence of NBF (mean per participant)	24.0 5.9/13.4/4.7	33.7 8.6/18.8/6.4	16.0 4.2/8.9/2.9	21.0 4.4/11.6/5.0	13.3 2.2/7.8/3.2
Sensitivity (%) vs. low-dose CT	70 74/68/72	74 80/70/78	68 64/70/67	66 68/63/74	61 58/64/56
Specificity (%) vs. low-dose CT	96 96/96/97	94 94/93/97	97 96/98/98	96 96/96/97	98 98/99/98
Inter-reader reliability (ICC)	0.85 0.85/0.79/0.76	0.81 0.85/0.73/0.70	0.94 0.80/0.94/0.93	0.91 0.91/0.88/0.67	0.93 0.65/0.97/0.92
Inter-reader reliability (Kappa)	0.70 0.69/0.71/0.71	0.66 0.67/0.65/0.69	0.77 0.70/0.81/0.72	0.69 0.70/0.70/0.66	0.79 0.67/0.82/0.79
T1-weighted MRI					
Occurrence of NBF (mean per participant)	9.3 1.8/4.8/2.7	13.9 2.9/7.0/4.1	6.4 1.2/3.1/2.1	6.4 1.4/3.4/1.6	3.5 0.2/2.7/0.6
Sensitivity (%) vs. low-dose CT	28 27/26/34	31 29/29/37	29 27/26/42	23 26/19/33	17 10/21/12
Specificity (%) vs. low-dose CT	99 99/99/98	98 99/98/96	99 99/99/98	99 99/99/100	100 100/100/100
Inter-reader reliability (ICC)	0.66 0.75/0.65/0.60	0.60 0.76/0.65/0.55	0.77 0.44/0.45/0.67	0.64 0.72/0.53/0.74	0.88 0.21/0.92/0.25
Inter-reader reliability (Kappa)	0.45 0.44/0.45/0.46	0.44 0.42/0.44/0.44	0.43 0.50/0.37/0.47	0.51 0.43/0.47/0.65	0.48 0.40/0.57/0.17
Radiography					
Occurrence of NBF (mean per participant)	6.9 3.4/NA/3.5	9.8 5.1/NA/4.8	3.4 1.4/NA/2.0	4.8 2.4/NA/2.5	NA
Sensitivity (%) vs. low-dose CT	50* (21**) 44/NA/55	55* (24**) 52/NA/59	45* (19**) 32/NA/57	34* (14**) 31/NA/37	NA
Specificity (%) vs. low-dose CT	98* (0.99**) 97/NA/98	97* (98**) 96/NA/98	99* (100**) 99/NA/99	97* (98**) 97/NA/97	NA
Inter-reader reliability (ICC)	0.72 0.60/NA/0.85	0.68 0.58/NA/0.86	0.84 0.60/NA/0.88	0.46 0.43/NA/0.32	NA
Inter-reader reliability (Kappa)	0.62 0.57/NA/0.67	0.62 0.58/NA/0.67	0.71 0.63/NA/0.76	0.45 0.41/NA/0.49	NA

NBF: new bone formation; ICC: intraclass correlation coefficient; vs: versus; NA: not applicable.
* Values when only investigating levels included in the modified Stoke Ankylosing Spondylitis Spinal Score (C2/C3-C7/Th1 and Th12/L1-L5/S1).
** Values when investigating the entire spine.

Methods. A prospective study (the SpACT-study) was performed from July 2023 to August 2024. All participants underwent whole-spine ldCT and MRI in addition to cervical and lumbar spine radiography (not HCs). sCT was reconstructed using BoneMRI. Images were independently assessed for NBF by two readers blinded to imaging and clinical information. One reader repeated evaluation of ldCT and sCT after 6 months. Sensitivity and specificity were calculated using ldCT as reference standard. Inter- and intra-reader reliability and inter-modality agreement was assessed using intraclass correlation coefficient (ICC) and Cohen's kappa.
Results. Sixty-nine participants (mean age 51.4 years, 36 males) were included: 30 axSpA, 19 PsA, 10 RA, and 10 HCs. The mean number of NBFs per participant was 25.8 on ldCT, 24.0 on sCT, 9.3 on T1wMRI, and 6.9 on radiography (Table I). Sensitivity for NBF detection was highest for sCT (70%), followed by radiography (50% cervical/lumbar spine, 21% whole-spine) and T1wMRI (28%); specificity was ≥93% for all modalities. Inter-reader reliability was high for sCT and ldCT (excellent ICCs, substantial kappas), while radiography and T1wMRI showed good ICCs with substantial and moderate kappas. Intra-reader reliability and inter-modality agreement for sCT and ldCT were excellent/substantial (ICCs/kappas).
Conclusion. Using ldCT as reference standard, sCT demonstrated substantially higher sensitivity than T1wMRI and radiography. Intra- and inter-reader reliability and inter-modality agreement between ldCT and sCT were comparable, suggesting that discrepancies in lesion detection primarily reflect reader variability rather than inter-modality differences.

P95

ONE-YEAR CHANGES OF SPINAL NEW BONE FORMATION IN AXIAL SPONDYLOARTHRITIS ASSESSED BY LOW-DOSE CT AND MRI-BASED SYNTHETIC CT

Willesen S.T.[1,2], Møller J.M.[3], Gosvig K.[3], Pedersen S.J.[1,2], Georgiadis S.[1], Østergaard M.[1,2]
[1]Copenhagen Center for Arthritis Research, Center for Rheumatology and Spine Diseases, Rigshospitalet, Glostrup; [2]Dept. of Clinical Medicine, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen; [3]Dept. of Radiology, Copenhagen University Hospital at Herlev Gentofte, Copenhagen, Denmark

Objective. To evaluate low-dose computed tomography (ldCT) and MRI-based synthetic CT (sCT) for detecting spinal new bone formation (NBF) at baseline and NBF-progression over 1-year in patients with axial spondyloarthritis (axSpA).
Methods. Participants underwent whole-spine ldCT and MRI at baseline and at 1-year. sCT images were reconstructed using BoneMRI v1.8(MRIGuidance, Utrecht, NL). One reader assessed NBF (marginal and non-marginal syndesmophytes, osteophytes) at all vertebral levels, blinded to other imaging and clinical data but unblinded to chronology. Lesions were scored as 1 if present and non-bridging and 3 if bridging between vertebral units. Non-bridging lesions judged larger at one timepoint were registered. Progression was defined as new or enlarging lesions; regression as the reverse. Sensitivity, specificity, and accuracy of sCT were calculated using ldCT as reference standard. Inter-modality agreement was assessed with intraclass correlation coefficients (ICCs).
Results. Twenty-five patients with axSpA were included (16 males, mean age 52.7 years). Mean NBF scores increased over 1-year on both modalities (Table I and Fig. 1). Progression of marginal syndesmophytes was observed in 7 patients on ldCT and 21 on sCT; regression in 2 and 13, respectively. Similar ldCT/sCT differences were seen for the other NBF-types, with most changes in the thoracic spine.
Using ldCT as reference standard, the sensitivity of synthetic CT for marginal syndesmophytes was 0.81/0.81 (baseline/1-year); for non-marginal syndesmophytes, 0.54/0.54; and for osteophytes, 0.41/0.42. Specificity and accuracy were ≥0.91.
ICCs between ldCT and sCT were 0.90/0.87 (baseline/1-year) for marginal syndesmophytes, 0.79/0.81 for non-marginal syndesmophytes, and 0.50/0.58 for osteophytes.
Conclusion. Both ldCT and sCT detected 1-year spinal NBF-progression. More patients showed progression on sCT than on ldCT, but with the overall magnitude of change being comparable. Given the radiation-free nature of sCT and the excellent specificity and accuracy, this method, together with ldCT, holds promise for longitudinal assessment of spinal structural damage in axSpA.

Table I. Status and 1-year change scores of different types of spinal new bone formation in patients with axial spondyloarthritis, including the number of patients showing progression and/or regression on low-dose CT and MRI-based synthetic CT.

AxSpA, n=25	Low-dose CT					MRI-based synthetic CT				
Spinal segment	No. of NBF scored (mean per patient)			n. of patients showing progression	n. of patients showing regression	No. of NBF scored (mean per patient)			n. of patients showing progression	n. of patients showing regression
	Baseline	Year 1	Change (Δ)			Baseline	Year 1	Change (Δ)		
	Marginal syndesmophytes					*Marginal syndesmophytes*				
Total spine	21.7	22.7	1.0	7	2	29.6	32.0	2.4	21	13
Cervical	5.2	5.2	0.0	1	1	5.8	5.9	0.1	7	3
Thoracic	11.6	12.5	0.9	6	1	17.8	19.4	1.6	15	10
Lumbar	4.9	5.0	0.1	2	0	6.0	6.8	0.8	12	1
	Non-marginal syndesmophytes					*Non-marginal syndesmophytes*				
Total spine	6.2	6.5	0.3	6	1	4.8	5.5	0.7	8	5
Cervical	1.6	1.6	0.0	1	0	1.4	1.7	0.3	5	2
Thoracic	2.4	2.5	0.1	2	1	1.4	1.7	0.3	3	2
Lumbar	2.3	2.4	0.1	3	0	2.0	2.0	0.0	3	1
	Osteophytes					*Osteophytes*				
Total spine	10.3	11.0	0.7	9	2	5.7	6.5	0.8	13	5
Cervical	2.2	2.5	0.3	4	0	1.3	1.4	0.1	4	2
Thoracic	6.2	6.8	0.6	4	2	3.2	3.7	0.5	12	3
Lumbar	1.8	1.8	0.0	1	0	1.2	1.4	0.2	3	0

AxSpA: axial spondyloarthritis, n.: number; NBF: new bone formation

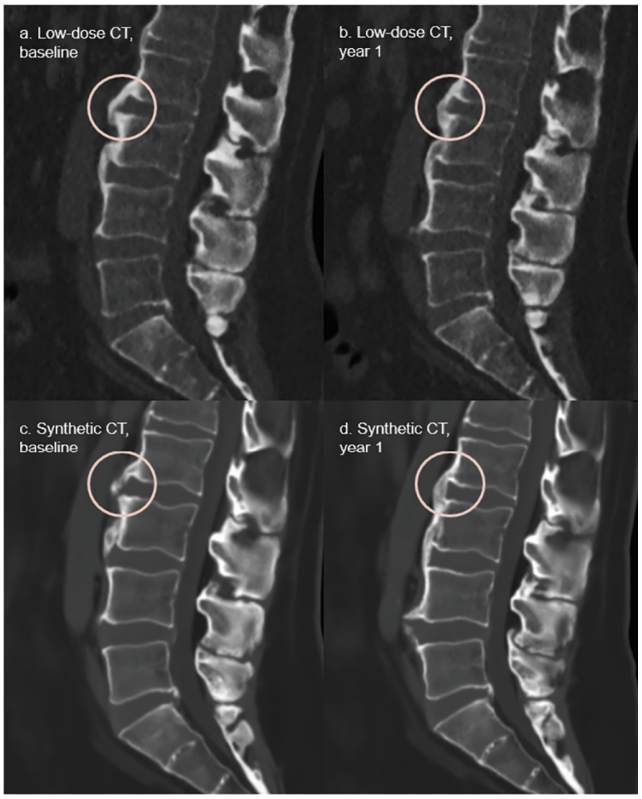


Fig. 1. Low-dose computed tomography (ldCT) and MRI-based synthetic computed tomography (sCT) images of the lumbar spine from the same patient at baseline (**a**, **c**) and at 1-year follow-up (**b**, **d**). Circled regions demonstrate progression of new bone formation on both imaging modalities, with a non-bridging lesion at baseline (**a**, **c**) evolving into a bridging lesion at follow-up (**b**, **d**).

P96

IDENTIFYING SUBCLINICAL BOWEL INFLAMMATION IN SPONDYLOARTHRITIS (SUBOWSPA): AN MRI FEASIBILITY STUDY

Weddell J.[1,2], Hoad C.[3], Tolan D.[4], Bray T.[5], Hensor E.M.A.[1,2], Selinger C.[6], McGonagle D.[1,2], Marzo-Ortega H.[1,2]
[1]Dept. of Rheumatology, Leeds Biomedical Research Centre, Leeds; [2]Leeds Institute of Rheumatic and Musculoskeletal Medicine, University of Leeds, Leeds; [3]Sir Peter Mansfield Imaging Centre, University of Nottingham, Nottingham; [4]Dept. of Gastroenterology Radiology, Leeds Teaching Hospitals NHS Trust, Leeds; [5]Dept. of Radiology, University College London Hospitals NHS Foundation Trust, London; [6]Dept. of Gastroenterology, Leeds Teaching Hospitals NHS Trust, Leeds, UK

Introduction. Up to 15% of people with axial spondyloarthritis (axSpA) have inflammatory bowel disease (IBD) and nearly 50% may have subclinical bowel involvement. Conversely, 10-20% of IBD patients may develop axSpA and up to 50% have sacroiliitis on imaging. MRI is used in both axSpA and IBD to assess inflammation in spinal and bowel tissues. Here we present the results of a feasibility study combining MRI of the spine/sacroiliac joints (SIJs) with MRI of the small bowel in a single study visit in patients with axSpA or IBD, with the aim of visualising the full extent of inflammatory burden across spine and bowel.
Methods. Biologic naïve patients with a diagnosis of IBD (n=15) or axSpA (n=15) underwent combined MRI spine/SIJ + MR enterography in one single visit, after providing informed written consent (REC 25/SW/0094). T1/STIR sequences of the lumbar spine/SIJs were first obtained followed by consumption of 1L of oral mannitol solution over 1 hour, before bowel MRI (T2weighted sequences + fat saturation, motility sequences, quantitative T2 and diffusion weighted sequences.) IV buscopan was used to minimise bowel peristalsis. Total time of study procedures were 3 hours, after which participants completed a questionnaire exploring acceptability.

Results. Overall, 27/30 participants completed the full MRI protocol (Table I shows demographic information and Table II feasibility). Feasibility analysis showed nearly all (29/30) either agreed or strongly agreed that the proposed MRI protocol is acceptable for routine care and all (27/27) who completed it were willing to repeat it, if required for their care. Improvement priority areas highlighted were scan length (16/30), followed by cannulation/IV medication (5/30).

Conclusion. An imaging protocol combining MRI of the spine/SIJ with MRI of the small bowel is safe, well tolerated and acceptable to patients. Further work analysing data obtained and optimising the MRI protocol is ongoing.

Table I. Demographics of participants enrolled into the SUBOWSPA study at baseline.

	Axial spondyloarthritis (n=15)	**Inflammatory bowel disease** (n=15)
Age (median[IQR])	39 (32-46)	43 (34-52)
Sex (n[%])	6 (40%)	6 (40%)
BMI (mean [SD])	29.3 (7.1)	28.3 (5.4)
Current or ever smoked (n[%])	0 (0%)	2 (13.3%)
Crohn's disease (n[%])	NA	9 (60%)
Ulcerative colitis (n[%])	NA	6 (40%)
Radiographic axSpA (n[%])	5 (33.3%)	NA
History of Psoriasis (n[%])	1 (6.7%)	0 (0%)
History of Uveitis (n[%])	2 (13.3%)	0 (0%)
Peripheral arthritis (n[%])	0 (0%)	NA
CRP raised >5 (n[%])	2 (13.3%)	5 (33%)
Fecal calprotectin raised >50 (n[%])	2 (17.5%)- 3 missing	4 (40%) – 5 missing

BMI: Body Mass Index; axSpA: axial spondyloarthritis; ASAS: Assessment of Spondyloarthritis International Society; CRP: C-reactive protein.

Table II. Results of acceptability and feasibility questionnaire and adverse events.

	Overall (n=30)
"Did you complete the full MRI protocol? "	
• Yes (n[%])	27 (90%)
• No(n[%])	3 (10%)
Reasons participants were unable to complete full MRI protocol	
• gastrointestinal side effects from buscopan/mannitol (n[%])	1 (3.3%)
• unable to comply with breath hold requirements (n[%])	1 (3.3%)
• delay to study visit due to scanner malfunction (n[%])	1 (3.3%)
"Would you be willing to undertake the combined MRI protocol in future?"	
Yes - if required for my clinical care	27 (100%)
No- I would not be willing to undertake the MRI again	0 (0%)
"The MRI protocol I experienced today is acceptable for routine care"	
• strongly agree (n[%])	17 (56.7%)
• agree (n[%])	12 (40%)
• undecided (n[%])	1 (3.3%)
"Which is the single most important priority for research to improve the MRI protocol?"	
• length of scan (n[%])	16 (53.3%)
• requirement for IV cannulation/medication (n[%])	5 (16.7%)
• length of lead up	2 (6.7%)
• oral contrast	1 (3.3%)
• requirement to stop NSAIDs	1 (3.3%)
Adverse events (n[%])	4 (13.3%)
• Vomiting (n[%])	2 (6.7%)
• Nausea (n[%])	1 (3.3%)
• Pre-syncope during cannulation (n[%])	1 (3.3%)

P97

NON-RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS IN THE ARAB WORLD: PREVALENCE, CLASSIFICATION DISCORDANCE, AND PHENOTYPE HETEROGENEITY: INSIGHTS FROM THE PROSPA STUDY

Zemrani S.[1,2], Amine B.[1,2], Hmamouchi I.[3], Ben Abdelghani K.[4], Tekaya R.[5,6], Boussaa H.[4], Hamdi W.[5,7], Mahmoud I.[5,6], Al Khater N.R.[8], Alnaimat F.[9], Ziade N.[10,11], Bahiri R.[1,2]

[1]Rheumatology Dept., El Ayachi Hospital, Ibn Sina University Hospital Center, Rabat-Salé; [2]Faculty of Medicine and Pharmacy, Mohammed V University, Rabat; , [3]Faculty of Medicine, Health Science Research Center, International University of Rabat, Rabat, Morocco, [4]Rheumatology Dept., Mongi Slim Hospital, Faculty of Medicine of Tunis, Tunis; [5]Tunis El Manar University, Tunis; [6]Rheumatology Dept., Charles Nicolle Hospital, Tunis; [7]Rheumatology Dept., Kassab Institute, Faculty of Medicine of Tunis, Tunis, Tunisia; [8]Rheumatology Dept., King Hamad University Hospital, Bahrain; [9]Dept. of Internal Medicine, Division of Rheumatology, School of Medicine, The University of Jordan, Amman, Jordan; [10]Rheumatology Dept., Saint Joseph University, Beirut; [11]Rheumatology Dept., Hotel-Dieu de France Hospital, Beirut, Lebanon

Introduction. The prevalence and significance of non-radiographic axial spondyloarthritis (nr-axSpA) may vary according to rheumatologist opinion or imaging findings. Using PROSPA (Profile of axial Spondyloarthritis in Arab countries) cohort data, we aimed to assess the prevalence of nr-axSpA across classification approaches, evaluate agreement between definitions, and characterize borderline phenotypes.

Methods. This ancillary PROSPA analysis included adults with axSpA from 15 Arab countries. The prevalence of nr-axSpA was determined according to three classifications: rheumatologist-defined, mNY-negative (absence of radiographic sacroiliitis), and imaging-based (absence of any structural lesions on X-rays of the sacroiliac joints and spine). Agreement between definitions was assessed using Cohen's kappa coefficient. Borderline phenotypes, defined as mNY-negative patients not classified as nr-axSpA by the rheumatologist, were further characterized.

Results. 1,112 patients were enrolled. The mean age was 42.5±12.2 years, 64% were male. The median diagnostic delay was 3 [1–8] years The prevalence of nr-axSpA according to rheumatologist, mNY-based, and imaging-based classifications was 16.9%, 25.3%, and 19.4%, respectively, with significant regional differences across all approaches (p<0.001) (Fig. 1). Across classifications, nr-axSpA patients were more frequently female and had shorter diagnostic delay, fewer radiographic structural lesions, and better functional status (ASAS-HI) (all p<0.001). According to the imaging-based and rheumatologist-defined classifications, they were also younger (p<0.001 and p=0.005, respectively), had less peripheral involvement (both p<0.001), less coxitis (both p<0.001), and lower disease activity (ASDAS-CRP) (p=0.008 and p=0.01, respectively). Agreement was highest between the mNY-based and imaging-based classifications ($\varkappa$=0.61; 87%) and lowest between rheumatologist and mNY-based classifications (κ=0.48; 83%) (Table I). Borderline phenotypes accounted for 52% of mNY-negative patients and were characterized by more peripheral involvement (p=0.002), psoriasis (p<0.001), IBD (p=0.003), and radiographic structural spinal damage (p<0.001).

Conclusion. The prevalence of nr-axSpA varied across approaches, with moderate agreement between them. The identification of borderline phenotype further highlights the complexity and heterogeneity of nr-axSpA.

Table I. Agreement between rheumatologist, modified New-York* criteria, and imaging-based definitions** of non-radiographic axial spondyloarthritis.

Definition	**Agreement (%)**	**Cohen's Kappa**	**p-value**
Rheumatologist definition vs. Modified New York criteria definition*	83	0.48	<0.001
Rheumatologist definition vs. Imaging-based definition*	87	0.57	<0.001
Modified New York criteria definition* vs. Imaging-based definition**	87	0.61	<0.001

*Patients without radiographic sacroiliitis according to the modified New York criteria.
**Absence of structural lesions in the sacroiliac joint and spine.

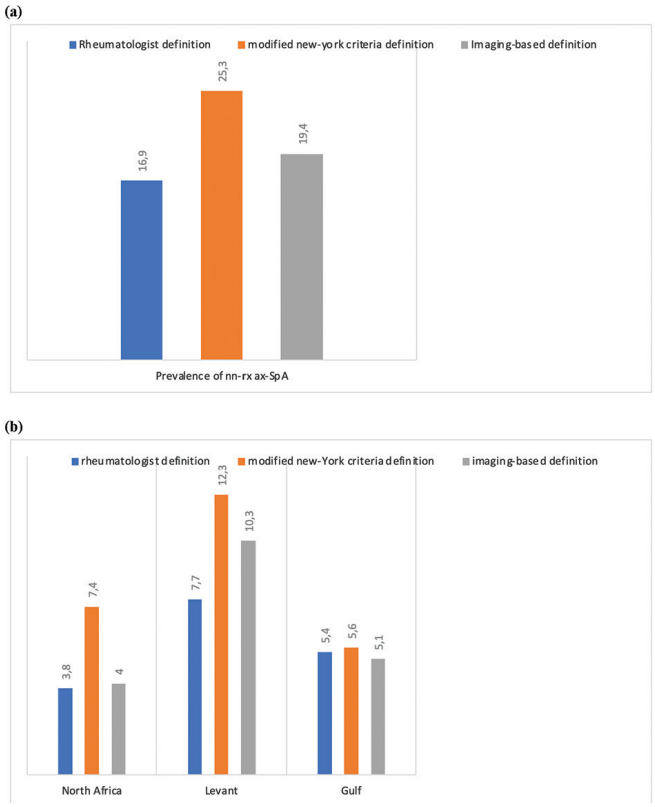


Fig. 1. Prevalence of non-radiographic axial spondyloarthritis according to three definitions: overall (**a**) and across regions (**b**).

P98

A MULTIOMIC SINGLE-CELL ATLAS OF HLA-B27-ASSOCIATED SPONDYLOARTHRITIS

Jin L.[1], Simone D.[1], Penkava F.[1], Hernandes M.C.[1], Baird T.[1], Attar M.[1], Rademacher J.[2], Teh J.[3], Sharma S.[4], McGonagle D.[5], Poddubnyy D.[6], Bowness P.[1], Sansom S.[1]

[1]Nuffield Dept. of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Oxford, UK; [2]Dept. of Gastroenterology, Infectiology and Rheumatology, Charité – Universitätsmedizin Berlin, Berlin, Germany; [3]Radiology Dept., Nuffield Orthopaedic Centre, Oxford University Hospitals NHS Foundation Trust, Oxford; [4]Oxford Eye Hospital, John Radcliffe Hospital; Oxford; [5]Rheumatology, University of Leeds, Leeds, UK; [6]Division of Rheumatology, Dept. of Medicine, University of Toronto and Schroeder Arthritis Institute, Krembil Research Institute, University Health Network, Toronto, Canada

Introduction. The cellular pathogenesis of Axial Spondyloarthritis (AxSpA) and related HLA-B*27-associated inflammatory mediated diseases remains only partially understood. We set out to profile, at the single cell level, a range of tissues including the blood, synovial fluid, sacroiliac joint, spinal enthesis and uveal fluid. As controls we studied Rheumatoid arthritis (RA) synovial fluid and blood and healthy control blood.

Materials and methods. We carried out multimodal gene-expression, T cell receptor (TCR) sequencing and surface protein (CITE-seq) profiling using 10x Genomics single-cell RNA sequencing. After quality control, removal of ambient RNA and cell doublets, datasets were integrated using scVI, before clustering and annotation via the Cellhub pipeline. Compositional changes were assessed with EdgeR, transcriptional differences using pseudobulk DESeq2 and GSEA, and genetic association analysis with scDRS. TCR analysis was used to evaluate clonal expansion and public AxSpA clone motifs. We then performed differential expression, GWAS-based disease relevance and clonal expansion analyses.

Results. We have established a single-cell atlas comprising approximately 10^6 cells from 129 samples across AxSpA, RA, and healthy controls. In AxSpA synovial fluid, we identified increased frequencies of $CD8^+$ T cell effector memory and TEMRA populations, and an increased monocyte subpopulation. Analysis of $CD8^+$ populations is ongoing and shows evidence of clonal expansions, including "private" expansions, likely virus-reactive clones and TCR sequences matching the public TRBV9+ CDR3b sequence recently described.

Conclusion. Our atlas has revealed AxSpA-specific changes in immune cell composition in affected tissues, and shows evidence of CD8 T cell clonal expansions in AxSpA affected tissue. When fully curated, the atlas will constitute an important resource for the research community and will also assist with the identification of potential targets for AxSpA-specific therapies.

Acknowledgements. Arthritis UK (grant 22252). National Institute for Health and Care Research (NIHR) Oxford Biomedical Research Centre (BRC).

P99

A T CELL-DRIVEN *CCL20*$^+$ MACROPHAGE CIRCUIT DRIVES TNF PATHOLOGY AND TH17 CELL RECRUITMENT IN AXIAL SPONDYLOARTHRITIS SYNOVIUM

Zhao J.[1*], Liu F.[1*], Li J.[1], Shi H.[1], Worth C.[1,3], Jiang L.[4,5], Simone D.[1], Penkava F.[1], Fanf H.[2], Chan A.[3], Duan L.[4,5 **], Bowness P.[1 **], Chen L.[1 **]

[1]Botnar Research Centre, Nuffield Dept. of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Oxford, UK; [2]Shanghai Institute of Hematology, State Key Laboratory of Medical Genomics, National Research Center for Translational Medicine at Shanghai, Ruijin Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, China; [3]Dept. of Rheumatology, Royal Berkshire NHS Foundation Trust, Reading, UK; [4]Dept. of Rheumatology and Clinical Immunology, Jiangxi Provincial People's Hospital, Medical College of Nanchang University, Nanchang; [5]Dept. of Rheumatology and Clinical Immunology, Jiangxi Provincial People's Hospital, The First Affiliated Hospital of Nanchang Medical College, Nanchang, China

[*]Contributed equally; [**]Jointly supervised this work

Introduction/Objective. Tumor necrosis factor (TNF) and interleukin (IL)-17 are central drivers of axial spondyloarthritis (AxSpA) pathology. We recently identified activated $CD4^+$ tissue-resident memory Th17 (TRM17) cells as the major source of IL17A/F within AxSpA synovium. This study aimed to identify the cellular sources of TNF and the factors driving the $CCL20^+$ myeloid signature in the joint and blood.

Methods. Single-cell RNA sequencing (scRNA-seq) was performed on synovial tissue from eight AxSpA patients. To model synovial interactions, healthy control monocyte-derived macrophages (MDMs) were stimulated with activated autologous $CD4^+$ T cells. Blocking antibodies and cytokine screens identified drivers of the $CCL20^+$ macrophage phenotype. ScRNA-seq and functional assays characterized circulating monocyte subsets in AxSpA blood.

Results. scRNA-seq identified a macrophage subset as the predominant sources of TNF in AxSpA synovium, expressing high levels of CCL20 known to recruit Th17 via CCR6. Activated $CD4^+$ T cells induced a $CCL20^+$ MDM phenotype mirroring synovial macrophages; this was driven by CD40L, IL-1 and TNF-α. Furthermore, $CCL20^+CD14^+$ monocytes - the peripheral equivalent of synovial CCL20+ macrophages - were expanded in AxSpA blood, hyper-responsive to stimulation and could be induced *in vitro* from healthy monocytes by IL-1β and TNF-α.

Conclusion. We identify CCL20+ macrophages as the key source of TNF in AxSpA synovium. Their induction by activated T cells suggests an inflammatory circuit where CCL20 production likely recruits circulating Th17 cells to replenish the synovial TRM17 pool. These findings identify synovial CCL20+ macrophages as a potential therapeutic target and circulating CCL20+ monocytes as a candidate biomarker for AxSpA.

P100

A UNIVERSAL TCR LIBRARY ENABLES SYSTEMATIC ANTIGEN DEORPHANIZATION IN HLA-B27-RESTRICTED AXIAL SPONDYLOARTHRITIS

Reitars M.H., Soundararajan J., Larsen I., Singh N., Holec P.
Fletcher Biosciences, Cambridge, USA

Introduction/Objective. HLA-B27 is the strongest genetic risk factor for radiographic axial spondyloarthritis (r-axSpA), implicating antigen-specific CD8⁺ T cells in disease pathogenesis. However, the peptide targets recognized by disease-associated T cell receptors (TCRs) remain incompletely defined, limiting mechanistic understanding of how HLA-B27 shapes pathogenic immunity. We aimed to systematically map HLA-B27-restricted peptide reactivity across axSpA-associated TCRs and validate these specificities in patient-derived samples.
Materials and methods. We aggregated paired TCR sequences reported across published r-axSpA single-cell studies and synthesized them into a pooled library of TCR+ cell lines. Using a proprietary antigen screening platform, we profiled peptide-HLA reactivity across candidate disease-relevant peptides. Candidate reactivities were then evaluated in an internal cohort of r-axSpA peripheral blood samples.
Results. The integrated TCR library comprised 45,236 TCR+ cell lines derived from 8 published single-cell sequencing studies of r-axSpA donors. Antigen screening identified TCR reactivity to more than 12 distinct HLA-B27-presented targets. A conserved cluster of TCRs demonstrated shared recognition across peptide panels and was observed across independent studies, including both peripheral blood and synovial fluid datasets. These peptide-associated TCR signatures were detected in HLA-B27⁺ r-axSpA peripheral samples (n=8) and were absent from HLA-B27- r-axSpA controls (n=2). Cross-reactivity analysis identified shared clonotypes recognizing multiple antigens, consistent with molecular mimicry and convergent TCR recognition among disease-associated CD8⁺ T cell responses.
Conclusion. To our knowledge, this represents the largest cross-study analysis of TCR reactivity in HLA-B27⁺ axial spondyloarthritis to date. By integrating published TCR repertoires into a synthesized cell-line library and pairing this resource with high-throughput antigen screening, we systematically mapped disease-associated peptide-HLA reactivities and validated conserved signatures in patient samples. These findings support a model in which shared and cross-reactive CD8⁺ T cell responses contribute to axSpA pathogenesis, while nominating defined peptide-TCR interactions for future mechanistic and therapeutic investigation.

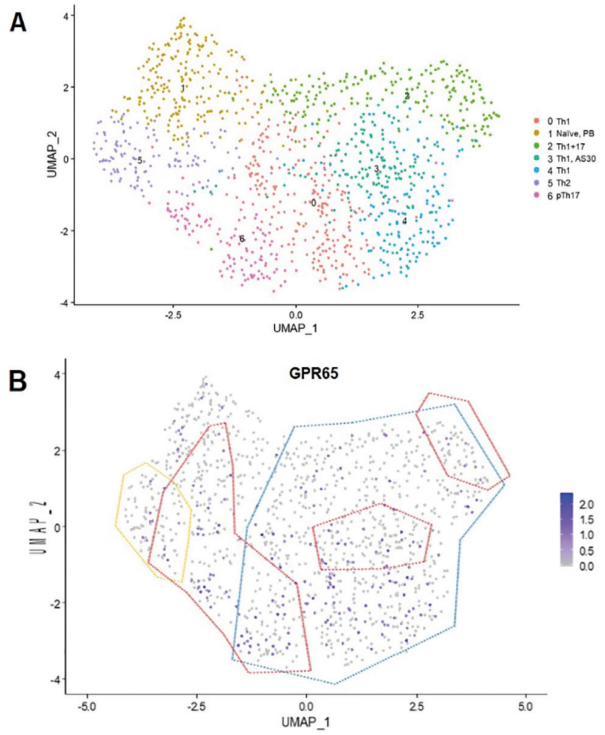


Fig. 1. Characterization of T-cell subsets and GPR65 expression in r-axSpA via CITE-seq analysis.
A. UMAP plot demonstrating the clustering and color-coded identification of distinct T-cell subpopulations, including Th1, naive PB, Th1+17, Th2, and pathogenic Th17 (pTh17) cells.
B. UMAP plot illustrating GPR65 transcript and surface protein expression density across the identified clusters, highlighted in shades of blue with preferential enrichment within the pTh17 subset.

P101

SINGLE-CELL TRANSCRIPTOMICS AND HISTOLOGICAL PROFILING REVEAL GPR65 UPREGULATION IN PATHOGENIC TH17 CELLS AND INFLAMED SPINAL TISSUES OF RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS

Kim T.J.[1], Lee Y.J.[1], Kim M.J.[1], Yu S.M.[1], Jo S.[2], Lee J.S.[3], Lee E.Y.[4], Kim T.H.[5,6]
[1]Dept. of Rheumatology, Chonnam National University Medical School, Gwangju; [2]Soonchunhyang University, Asan, Republic of Korea; [3]Inocras, Inc., San Diego, California, USA; [4]Division of Rheumatology, Dept. of Internal Medicine, Seoul National University College of Medicine, Seoul; [5]Hanyang University Institute for Rheumatology Research, Seoul; [6]Dept. of Rheumatology, Hanyang University Hospital for Rheumatic Diseases, Seoul, Republic of Korea

Introduction/Objective. Radiographic-axial spondyloarthritis (r-axSpA) is a chronic inflammatory spine disease driven by pathogenic Th17 (pTh17) cells. Identifying specific therapeutic targets for pTh17 cells could lead to novel treatment strategies. Recent single-cell RNA-sequencing (scRNA-seq) studies found GPR65 highly upregulated in pTh17 cells, suggesting its potential as a therapeutic target. This study aimed to characterize GPR65 expression across T-cell subsets and validate its histological relevance in r-axSpA target tissues.
Materials and methods. Peripheral blood mononuclear cells (PBMCs) and synovial fluid mononuclear cells (SFMCs) from r-axSpA patients were isolated for scRNA-seq and CITE-seq to profile T-cell subsets. Spinal tissue samples from AS patients and healthy controls (HCs) were examined using immunohistochemistry (IHC) to assess GPR65 expression. Immunofluores-

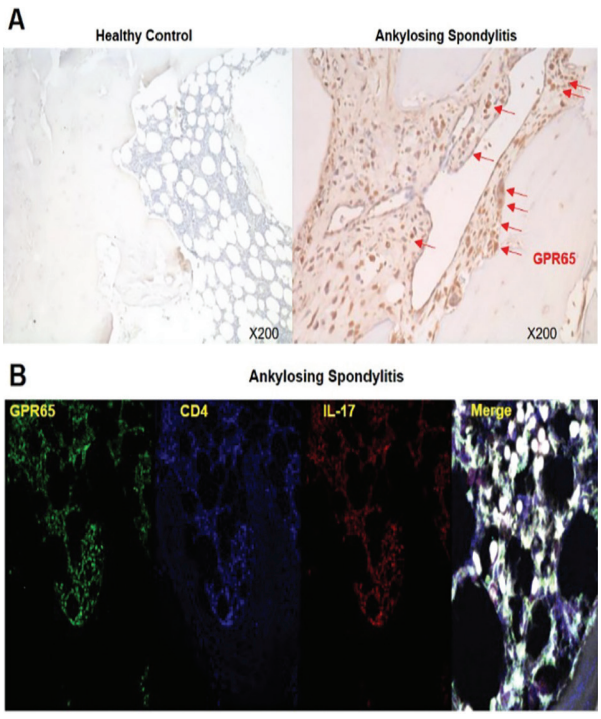


Fig. 2. Elevated GPR65 expression in pathogenic Th17 cells and inflamed spinal lesions of r-axSpA.
A. Immunohistochemical staining of human spinal tissue sections from a healthy control (left) and an r-axSpA patient (right) at 200x magnification, with red arrows indicating enhanced GPR65 immunoreactivity within the r-axSpA lesion.
B. Confocal immunofluorescence staining of AS spinal tissue demonstrating the spatial co-localization of GPR65 (green), CD4 (blue), and IL-17 (red). The merged image confirms GPR65 enrichment specifically within IL-17-producing CD4⁺ pathogenic T cells.

cence (IF) staining for GPR65, CD4, and IL-17 was performed on spinal tissues to confirm GPR65 localization in Th17 cells.
Results. CITE-seq analysis revealed distinct T cell subsets with GPR65 significantly upregulated in the pTh17 cluster, compared to conventional T-cell lineages, marking its pathogenic potential (Fig. 1). IHC indicated significantly higher GPR65 expression in r-axSpA spinal tissues than in HCs, particularly in hypercellular inflammatory regions. Multiplexed IF confirmed GPR65 co-localization with IL-17-producing CD4$^+$ T cells, directly linking its expression to r-axSpA -related tissue inflammation (Fig. 2).
Conclusion. GPR65 is highly expressed in both circulating/synovial pTh17 cells and inflamed spinal regions in r-axSpA. Its distinct enrichment in IL-17-producing CD4$^+$ T cells supports its role as a promising therapeutic target for managing r-axSpA progression.

References

1. GHORESCHI K, LAURENCE A, YANG XP *et al*.: Generation of pathogenic T(H)17 cells in the absence of TGF-β signalling. *Nature* 2010; 467: 967-71.
2. BASDEO SA, MORAN B, CLUXTON D *et al*.: Polyfunctional, pathogenic CD161+ Th17 lineage cells are resistant to regulatory T cell-mediated suppression in the context of autoimmunity. *J Immunol* 2015; 195: 528-40.
3. YI K, JO S, SONG W *et al*.: Analysis of single-cell transcriptome and surface protein expression in ankylosing spondylitis identifies ox40-positive and glucocorticoid-induced tumor necrosis factor receptor-positive pathogenic Th17 cells. *Arthritis Rheumatol* 2023; 75(7): 1176-86.

P102

MICROENVIRONMENTAL HYPOXIA AND HIF-1A ACTIVITY SHAPE COMPARTMENT-SPECIFIC CD8 T-CELL ADAPTATION IN PSORIATIC DISEASE

Mauro D.[1], Raimondo M.G.[2], Ciancio A.[1], Forte G.[1], Mohammadian H.[2], Gandolfo S.[1], Ilardi G.[3], Giacca R.[1], Raimondi M.[1], Salzillo A.[1], Ali K.[1], Martinelli E.[1], Cincinelli G.[5], Parafioriti A.[4], Caporali R.F.[5], Ramming A.[2], Ciccia F.[1]
[1]Dept. of Precision Medicine, University of Campania L. Vanvitelli, Naples, Italy; [2]Dept. of Medicine 3 – Rheumatology and Immunology, Friedrich-Alexander-Universität Erlangen-Nürnberg and Universitätsklinikum Erlangen, Erlangen, Germany; [3]Dept. of Advanced Biomedical Sciences, University of Naples Federico II, Naples; [4]Unit of Pathology, ASST Gaetano Pini-CTO, Milan; [5]Dept. of Clinical Sciences and Community Health, University of Milan, Milan, Italy

Introduction. Psoriasis (PsO) and psoriatic arthritis (PsA) share inflammatory circuits but respond differently across domains, suggesting tissue-specific pathogenic constraints and skin-to-joint propagation.[2] Because skin, enthesis and synovium differ in oxygen architecture and an epithelial HIF-1α–type-17 axis is established,[3] we asked whether hypoxia/HIF-1α aligns with CD8 exhaustion-like (Tex) adaptation across psoriatic microenvironments.
Methods. IHC/IF assessed HIF-1α localisation and TIGIT$^+$CD8$^+$ infiltration in PsO skin and PsA synovium (n=10 each). Bulk skin and synovial transcriptomics were interrogated with a 236-gene CD8 Tex module (GSEA, PCA, PERMANOVA). Public scRNA-seq of paired PsA tissues (1) informed the HIF1A gradient; STCAT-annotated skin/synovial scRNA-seq supported Tex and HC/OA comparisons; entheseal biopsies (PsA n=10) extended the interface. SKG mice received topical IMQ × 5 days; dermal TIGIT$^+$/TIGIT$^+$PD-1$^+$ CD8 T cells were quantified by flow cytometry.
Results. Pseudobulk HIF1A followed a gradient skin > synovial fluid > PBMC > synovium (Kruskal-Wallis p<0.001). HIF-1α protein was nuclear in lesional skin and cytoplasmic in PsA synovium (Fig. 1–2). PsO skin showed stronger Tex features (Fig. 1): increased TIGIT$^+$CD8$^+$ infiltration and higher HIF1A in Tex (both p<0.001), with bulk-level Tex module enrichment versus p synovium (NES=1.9986; FDR=5.48×10^{-8}; PERMANOVA R^2=0.6865, p=0.008; Fig. 2B). Skin scRNA-seq confirmed CD4/CD8 Tex expansion, whereas PsA synovium was Tex-poor despite broad T-cell heterogeneity (Fig. 2). In entheseal biopsies, CD8 Tex ranked among the top upregulated CD8 programmes. Despite Tex annotation, these cells remained polyfunctional and inflammatory, producing IL17A/F, IFNG, IL26, CXCL13 and CCL-family chemokines. In IMQ-treated SKG mice, dermal TIGIT$^+$ CD8 cells expanded (TIGIT$^+$PD-1$^+$ trend), supporting a cutaneous TIGIT$^+$CD8 phenotype.
Conclusion. These data support tissue-level oxygen sensing as a compartmental organizer of psoriatic inflammation. HIF-1α-active skin/enthesis favours inflammatory Tex-like CD8 adaptation, whereas PsA synovium remains Tex-attenuated. This framework shifts PsA pathogenesis from a uniform cytokine model toward tissue-contextualized immune programming with domain-specific consequences.

References

1. DURHAM LE, HUMBY F, NG N *et al*.: Linking skin and joint inflammation in psoriatic arthritis through shared CD8$^+$ T cell clones. *Arthritis Rheumatol* 2026; 78: 152-65. doi: 10.1002/art.43286.
2. RAIMONDO MG, MOHAMMADIAN H, SCHETT G *et al*.: Skin-derived myeloid precursors and joint-resident fibroblasts spread psoriatic disease from skin to joints. *Nat Immunol* 2026; 27: 35-47.
3. SUBUDHI I, KONIECZNY P, PRYSTUPA A *et al*.: Metabolic coordination between skin epithelium and type 17 immunity sustains chronic skin inflammation. *Immunity* 2024; 57: 1665-80.

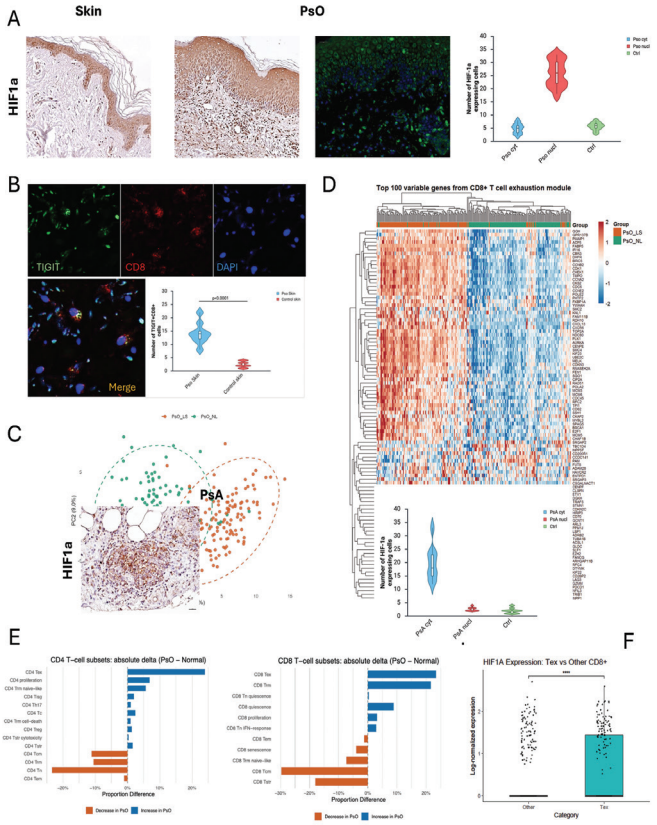
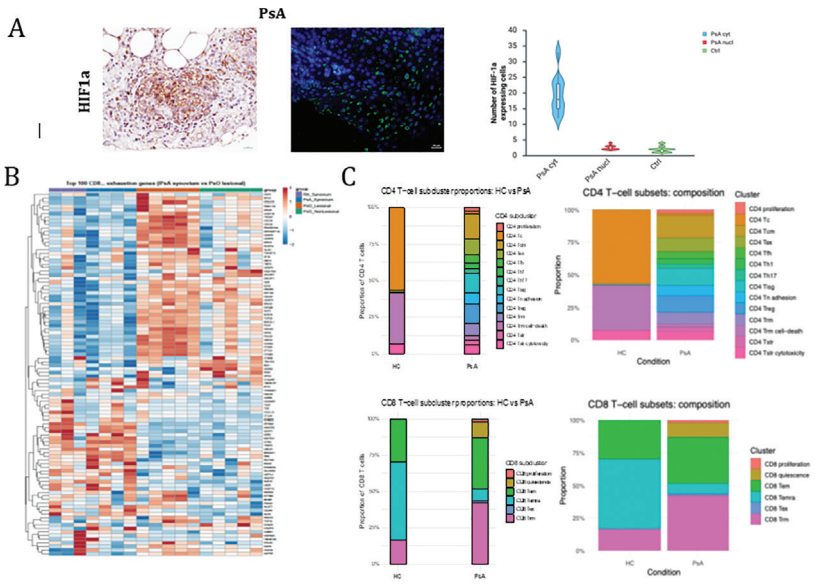


Fig. 1. Hypoxia/HIF-1α activation and Tex-associated CD8 immunity in psoriatic skin.
A. Representative HIF-1α immunohistochemistry (IHC) in control skin and psoriatic lesional skin (PsO), with representative immunofluorescence (IF). Right: quantification of HIF-1α–positive cells stratified by subcellular localisation (PsO cytoplasmic *vs*. PsO nuclear) and controls (Ctrl).
B. Multiplex IF showing TIGIT (green), CD8 (red) and nuclei (DAPI, blue), with merged image. Right: quantification of TIGIT$^+$CD8$^+$ cells in PsO skin *vs*. control skin (p<0.0001 as indicated).
C. Principal component analysis (PCA) based on the CD8 exhaustion-module expression separates psoriatic lesional (PsO_LS) and non-lesional (PsO_NL) samples;
D. Heatmap of the top 100 most variable genes within the CD8 exhaustion module across PsO_LS and PsO_NL;
E. Absolute changes in the relative abundance of annotated CD4 and CD8 T-cell states in PsO vs. normal skin (Δ proportion); blue indicates increased and orange decreased fractions in PsO.
F. HIF1A expression in Tex *vs*. other CD8$^+$ T-cell states (log-normalised expression); significance annotated on the plot.



P102 - Fig. 2. Compartmental divergence in PsA synovium: HIF-1α localisation, Tex-module expression, and T-cell composition.
A. Representative HIF-1α IHC and IF in PsA synovial tissue. Right: quantification of HIF-1α–positive cells stratified by cytoplasmic (PsA cyt) *vs*. nuclear (PsA nucl) localisation compared with controls (Ctrl).
B. Heatmap of the top 100 CD8 exhaustion-module genes across biopsies spanning synovial tissue (PsA and RA) and psoriatic skin compartments (lesional and non-lesional), showing scaled expression and unsupervised clustering.
C. Stacked bar plots summarising CD4 and CD8 T-cell subcluster proportions/composition in healthy controls (HC) *vs*. PsA synovium, using STCAT atlas-based state annotation (including Tex and other memory/effector states); bars represent relative proportions per condition.

P103

DISEASE-SPECIFIC REGULATION OF THE IMMUNE CHECKPOINT VISTA IN INFLAMMATORY RHEUMATIC MUSCULOSKELETAL DISORDERS (I-RMDS)

Roman I.[1,2], Ney L.[1,2], Lalazi B.[1,2], Tsiami S.[1,2], Kiltz U.[1,2], Baraliakos X.[1,2], Schütz C.[1,2]
[1]Ruhr-Universität Bochum; [2]Rheumazentrum Ruhrgebiet, Herne, Germany

Introduction/Objective. Rheumatoid arthritis (RA), psoriatic arthritis (PsA), and axial spondyloarthritis (axSpA) are among the most prevalent I-RMDs. These disorders are characterized by inflammation of the synovium and/or the axial skeleton, which is driven by dysregulated interactions between the innate and adaptive immune systems. V-domain-Ig-suppressor-of-T-cell-activation (VISTA) is an inhibitory checkpoint that functions as both a receptor and a ligand. It is highly expressed on antigen-presenting cells and T cells, which place it at the interface between innate and adaptive immunity. However, its expression in I-RMDs remains poorly defined. The objective of this study was to map VISTA expression in the peripheral blood of patients with RA, PsA, and axSpA.
Methods. Sex-matched patients with RA (n=49), PsA (n=53), axSpA (n=61), as well as healthy controls (HC,n=49) were included. VISTA mRNA expression was quantified in PBMC using qPCR. Expression of VISTA and its ligands (VSIG-8, PSGL-1) on immune cells was assessed by flow cytometry. Soluble VISTA (sVISTA) serum concentrations were quantified using ELISA.
Results. mRNA expression was significantly elevated in RA (p<0.001) and PsA (p<0.05) vs. in axSpA, whereas axSpA patients showed levels comparable to HCs. Differences in cell surface VISTA expression were observed on CD4$^+$ T cells, with statistical significance when comparing RA and axSpA. Preliminary data on VSIG-8 and PSGL-1 surface expression suggest an IRMD-specific pattern across monocytes and T-cells. sVISTA concentrations were significantly elevated in both RA and axSpA compared to HCs, whereas PsA showed no significant differences in levels relative to HCs.
Conclusion. VISTA exhibits distinct mRNA, cell surface, and soluble expression patterns across different I-RMDs, indicating specific regulation at the intersection of innate and adaptive immunity.
Discussion. These findings suggest that VISTA may modulate inflammatory pathways differently in RA, PsA and axSpA, warranting further studies to investigate its functional role and its potential as a biomarker of specific disease phenotypes.

P104

TRAINED-LIKE IMMUNE SIGNATURES IN γδ T CELLS AND NK CELLS FROM PATIENTS WITH SPONDYLO-ARTHRITIS

Romero-García H.A.[1,2], Sánchez-Pérez S.[1,2], Romero-López N.[2], Ortega-Mejía I.I.[1,2], Prieto-Chávez J.L.[3], Casasola-Vargas J.C.[4], Domínguez-López M.L.[1], Burgos-Vargas R.[4], Romero-López J.P.[1,2]
[1]Laboratorio de Inmunoquímica 1, Posgrado en Ciencias Biológicas, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Mexico City; [2]Laboratorio de Patogénesis Molecular, Red MEDICI, Carrera de Médico Cirujano, Facultad de Estudios Superiores Iztacala, Universidad Nacional Autónoma de México, Tlalnepantla; [3]Laboratorio de Citometría, Centro de Instrumentos - CIS IMSS, Mexico City; [4]Departamento de Reumatología, Hospital General de México Dr. Eduardo Liceaga, Mexico City, Mexico

Introduction/Objective. Spondyloarthritis (SpA) is a chronic inflammatory disease in which intestinal dysbiosis, barrier dysfunction, and mechanical stress may promote abnormal activation and tissue migration of innate-like lymphocytes. We aimed to evaluate whether γδ T cells and NK cells from patients with SpA display markers compatible with trained immunity, intestinal homing, and IL-17/IL-22 production.
Materials and methods. Peripheral blood and synovial fluid samples from patients with SpA and blood from healthy controls were analyzed by spectral flow cytometry. We evaluated CD45RO, CD161, CD27, α4β7, IL-17, and IL-22 expression in αβ T cells, γδ T cells, NK cells, and NKT cells. Manual gating was complemented with t-SNE and FlowSOM analyses.
Results. Seven patients with SpA and six healthy controls were included. All patients were HLA-B27 positive, with disease duration of 9±7.1 years, ASDAS-CRP of 3.4±1.7, CRP of 19.7±9.8 and ESR of 36.8±35.5. Compared with controls, patients showed higher frequencies of total IL-17+ γδ T cells (p=0.0453), CD161+ γδ T cells (p<0.0001), α4β7+IL-17+ γδ T cells (p=0.0411), α4β7+CD161+ γδ T cells (p<0.0001), α4β7+CD27− γδ T cells (p=0.0260), α4β7+CD45RO− γδ T cells (p=0.0431), CD161+ αβ T cells (p=0.0416), CD161+ NK cells (p=0.0167) and α4β7+CD161+ NK cells (p=0.056). Unsupervised analysis identified rare cell clusters not evident by conventional manual gating.
Conclusion. Patients with SpA show expanded γδ T-cell and NK-cell subsets expressing CD161, α4β7, and IL-17, supporting the presence of intestinal-homing, trained-like innate immune signatures that may contribute to SpA inflammation.

P105

HUMAN ENDOGENOUS RETROVIRUS-DERIVED PEPTIDES FROM AN HLA-B27 REGION VARIANT (RS13202464) ARE PRESENTED BY HLA-B27: A CANDIDATE MECHANISM IN ANKYLOSING SPONDYLITIS

Marston J.L.[1], Romero-López J.P.[2], Casado-Lima B.[1], Bowness P.[3], De Mulder-Rougvie M.[4], Reyes-Gopar H.[1], Nixon D.F.[1]
[1]Institute of Translational Research, Feinstein Institutes for Medical Research, New York, USA; [2]Facultad de Estudios Superiores Iztacala, Universidad Nacional Autónoma de México, Tlalnepantla, Mexico; [3]Nuffield Dept. of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Oxford, UK; [4]Dept. of Genetic Medicine, Weill Cornell Medicine, New York, USA

Introduction/Objective. The etiology of ankylosing spondylitis (AS), an autoimmune condition strongly associated with the HLA-B27 genotype, remains incompletely understood. We investigated a novel mechanism involving single nucleotide variations (SNVs) within human endogenous retroviral (HERV) sequences in the HLA-B27 genomic region and their potential impact on AS pathogenesis. We specifically explored how the SNV rs13202464 alters the binding affinity of the HLA-B27 molecule to self and microbial peptides, hypothesizing that this missense mutation (methionine to threonine) may drive differential T cell selection during immune development, whereby HSV-derived peptides that mimic self-proteins are insufficiently tolerated.
Materials and methods. We compared predicted peptide binding to HLA-B27 between reference and variant sequences using predictive modeling with the Immune Epitope Database (IEDB) analysis suite, evaluating artificial neural network (ANN)-predicted IC50 values for self- and microbial-derived peptides. We further used T2 cell binding assays to test whether peptides derived from translation of the variant are presented by HLA-B27.
Results. Peptide binding to HLA-B27 was reduced in carriers of the variant, reflected by an approximately ten-fold increase in predicted ANN IC50 values for HLA-B27-positive individuals carrying the SNV. T2 assays confirmed that these variant-derived peptides are presented by HLA-B27, stabilizing the molecule at the cell surface.
Discussion. This reduced binding could initiate AS pathogenesis through either altered thymic selection or molecular mimicry arising from sequence similarity between HSV- and HERV-derived peptides.
Conclusion. Our findings underscore the need for further investigation into the interplay between HERV sequences, viral mimicry, and immune tolerance, with the goal of identifying novel therapeutic strategies targeting AS at its genetic and immunological roots.
Acknowledgements. Supported by an MSTP grant from the NIH (T32GM152349) to the Weill Cornell/Rockefeller/Sloan Kettering Tri-Institutional MD-PhD Program, and an F30 Predoctoral Fellowship from the NCI (5F30CA278653-02).

P106

EXHAUSTION ESCAPE IN AXIAL SPONDYLOARTHRITIS: A TRANSCRIPTIONALLY DISTINCT CHECKPOINT-EXPRESSING CTL SUBSET RETAINS CYTOTOXIC EFFECTOR POTENTIAL

Tang M.[1], Qaiyum Z.[1], Lim M.[1], Inman R.D.[1,2]
[1]Schroeder Arthritis Institute, University Health Network, Toronto; [2]Dept. of Immunology, University of Toronto, Toronto, Canada

Introduction. Persistent chronic inflammation is a hallmark of axial spondyloarthritis (axSpA), with cytotoxic T cells (CTLs) increasingly implicated in its pathogenesis. Ordinarily, T cell exhaustion follows sustained T cell receptor (TCR) activation under high antigen load to restrain collateral tissue damage. We previously identified a CTL subset (PD-1$^+$TIGIT$^+$LAG-3$^+$CD127$^+$) enriched in axSpA synovial fluid that may evade exhaustion despite high immune checkpoint expression. Here, we further characterize the transcriptomic profile of these CTLs and test whether HLA-B27:05's interaction with its associated TCR predisposes CTLs toward exhaustion escape.
Methods. We performed single-cell RNA and TCR sequencing (scRNAseq) of paired PBMCs and SFMCs to comprehensively characterize the CTL transcriptome at single-cell resolution in axSpA patients. Pseudo-time trajectory analyses using Monocle3 were performed to map developmental trajectories across CTL subsets.
Results. Our dataset comprised 31,184 scRNAseq profiles of mature CTLs from synovial fluid and peripheral blood of axSpA patients and age- and sex-matched healthy controls. Seurat-based clustering resolved 12 distinct CTL clusters, 6 of which expressed varying levels of cytotoxic effector genes including *GZMA*, *GZMB*, *GZMK*, *PRF1*, and *NKG7*. We identified a clonally expanded synovial CTL subset from axSpA patients co-expressing cytotoxic effector genes and canonical checkpoint markers. Despite this, these cells downregulated expression of exhaustion-associated transcripts (*CD244*, *HAVCR2*, *ENTPD1*, *NT5E*) and notably showed no transcriptional or protein expression of TOX, a key regulator of T cell exhaustion. *In silico* trajectory analyses suggest this PD-1$^+$TIGIT$^+$LAG-3$^+$ subset may differentiate into potent effector CTLs.
Conclusion. Our findings confirm a checkpoint-expressing CTL population that evades exhaustion and retains an effector phenotype, potentially contributing to autoinflammation in axSpA patients. Ongoing work using CTL lines from a K562-based artificial antigen-presenting cell co-culture system will investigate whether the interaction of HLA-B27:05 and its cognate TCR drives exhaustion escape. We hypothesize that structural constraints on HLA-B27 epitope presentation generate high-avidity CTLs that resist exhaustion.
Acknowledgements. Supported by The Arthritis Society of Canada (Strategic Operating Grant) & Canadian Institute of Health Research (Project grant).

P107

TARGETING NAD$^+$ METABOLISM IN SPONDYLOARTHRITIS: IMPACT OF ANTI-TNF THERAPY AND NAD$^+$ SUPPLEMENTATION

Vellón-García B.[1], García-Delgado G.M.[1], López-Medina C.[2,3], Llamas-Urbano A.[2], Romero Zurita L.[2,3], Hanaee Y.[2], Ortiz-Buitrago P.[3], Merlo-Ruiz C.[3], Abalos-Aguilera M.C.[3], Martínez-Moreno J.M.[2], Puche-Larrubia M.A.[3], Arias-de la Rosa I.[3], López Montilla M.D.[1,3], Ortega-Castro R.[3], Calvo-Gutiérrez J.[3], Ladehesa-Pineda M.L.[3], López-Pedrera C.[3], Escudero-Contreras A.[3], Villalba J.M.[1], Barbarroja N.[2,3], Collantes-Estevez E.[3], Pérez-Sánchez C.[1,2,3]
[1]Dept. of Cell Biology, Physiology and Immunology, Maimonides Institute of Biomedical Research of Cordoba (IMIBIC), Reina Sofia University Hospital, University of Cordoba, Cordoba; [2]Cobiomic Bioscience SL. EBT University of Cordoba/IMIBIC, Cordoba; [3]Rheumatology Service/Maimonides Institute for Research in Biomedicine of Cordoba (IMIBIC)/University of Cordoba/ Reina Sofia University Hospital, Córdoba, Spain

Introduction. NAD$^+$ is a central metabolic cofactor that plays a key role in the regulation of immune responses.
Objective. To characterize the NAD$^+$ metabolome in spondyloarthritis by analyzing key pathway metabolites and their association with clinical traits.
1. Assess the impact of 6-month anti-TNF therapy on NAD$^+$ metabolome alterations.
2. Evaluate the preclinical potential of several NAD$^+$ boosters to restore NAD$^+$ pathway alterations.
Methods. Whole blood and clinical data were collected from 70 healthy donors (HDs), 80 spondyloarthritis (SpA), 80 psoriatic arthritis (PsA), and 80 rheumatoid arthritis (RA) patients. NAD$^+$ pathway metabolites (NAD+, NADH, NAM, NMN, NR, NADP and Tryp) were analyzed using DOSY-NMR technology. A longitudinal analysis in 90 patients evaluated NAD$^+$ metabolome changes before and after 6 months of anti-TNF therapy. PBMCs from 45 patients were treated *ex-vivo* with NAD$^+$ boosters (NAM, NR, NMN, NRT, NRH, and NAR) for 24 hours. Intracellular NAD$^+$/NADH levels, inflammatory mediators (Olink T96) and transcriptomic changes (RNAseq) were assessed.
Results. All diseases showed a reduced NAD$^+$/NADH ratio and elevated NAM and NMN compared to HDs. PsA and SpA displayed increased NADH, while RA exhibited decreased NAD+ levels; SpA also presented reduced NADP and elevated tryptophan levels. These alterations correlated with distinct clinical features, including CRP, ESR, uveitis, DAS28, joint counts, among others.
Anti-TNF therapy partially normalized NAD$^+$ metabolites in a disease-specific manner. All NAD$^+$ boosters increased intracellular NAD$^+$ levels and reduced inflammatory mediator secretion, with NRH showing the strongest effect. RNA-seq analysis of PBMCs treated with NAD+ boosters further revealed a disease-specific modulation of inflammatory and metabolic pathways.
Conclusion. 1. Spondyloarthritis patients display disease-specific NAD$^+$ metabolome disturbances linked to clinical activity. 2. Anti-TNF therapy partially restores NAD$^+$ metabolic balance. 3. NAD$^+$ boosters enhance intracellular NAD$^+$ and exert anti-inflammatory effects, supporting their therapeutic potential.
Support. RYC2021-033828-I; PID2022-141500OA-I00; DIN2022-012766; PI22/00539; PMP21/00119; PID2023-152503OB; UE.

P108

DYNAMIC ORCHESTRATION OF TH17, TRM, AND MAIT CELLS LINKS THE GUT-JOINT AXIS DURING SPONDYLOARTHRITIS DEVELOPMENT IN THE SKG MODEL

Verhoeven F.[1,2,3], Guet L.[3], Favier B.[3], Romand X.[3], Demougeot C.[2], Wendling D.[1], Prati C.[1,2], Baillet A.[3], Hannani D.[3]
[1]Service de Rhumatologie - CHU Besancon, Besançon; [2]Université Marie et Louis Pasteur, UFR Santé, UMR1098, EFS, INSERM, Besançon; [3]CNRS, UMR 5525, T-RAIG, TIMC, Université Grenoble Alpes, Grenoble, France

Introduction. This study aimed to characterize, in the SKG murine model, the temporal dynamics of the main IL-17A-producing immune cell populations, namely Th17, MAIT, and TRM cells, and their cytokine production profiles at different stages of SpA development across three key compartments: the spleen, ileum, and synovium.
Methods. Arthritis was induced in 12-week-old SKG mice by intraperitoneal injection of curdlan. Three disease stages were analyzed (n=6 mice/group): day 4 (D4, preclinical phase), day 11 (D11, arthritis onset), and day 21 (D21, established arthritis). Ileum, spleen and synovium from all four limbs, were harvested and dissociated by mechanical (spleen) or enzymatic digestion. Immune cell populations were analyzed by flow cytometry and defined as follows: Th17 ($CD3^+CD4^+IL\text{-}17A^+$), MAIT ($CD3^+TCRV\beta6\text{-}8^+MR1\text{–}5\text{-}OP\text{-}RU^+$), and TRM ($CD45^+CD3^+CD4^+$ or $CD8^+CD69^+CD103^+$). Intracellular production of IL-17A and IFN-γ was assessed. Plasma levels of TNF, IL-22, IL-17A, IL-17F, IL-23, IL-33, soluble ST2 (IL-33 receptor), and zonulin were quantified by ELISA.
Results. At day 4 a significant decrease in ileal MAIT and Th17 cells was observed with an increase in highly inflammatory $IL\text{-}17A^+/IFN\text{-}\gamma^+$ Th17 cells and increased plasmatic TNF, IL-17F, IL-23 and zonulin levels.
At day 11, accumulation of $CD4^+$ and $CD8^+$ TRM was detected in the spleen and joints. Synovial $CD4^+$ TRM exhibited an $IL\text{-}17^+$ TRM phenotype, whereas $CD8^+$ TRM displayed a highly inflammatory $IL\text{-}17A^+/IFN\text{-}\gamma^+$ profile. Th17 cells were detected in the synovium. In plasma, IL-17F, IL-22, and IL-33 levels were increased, with a decrease in ST2L.
At day 21, while synovial Th17 and $IL\text{-}17^+$ TRM accumulation persisted, a marked emergence of IL-17A-producing MAIT cells was observed in the synovium, with reduction of IFN-γ production in the ileum and spleen. In plasma, IL-17A, IL-17F, and IL-22 levels were elevated.
Conclusion. Th17 cells and TRM17 are early drivers of the immune response, whereas MAIT cells emerge at later stages in a context of increased intestinal permeability.

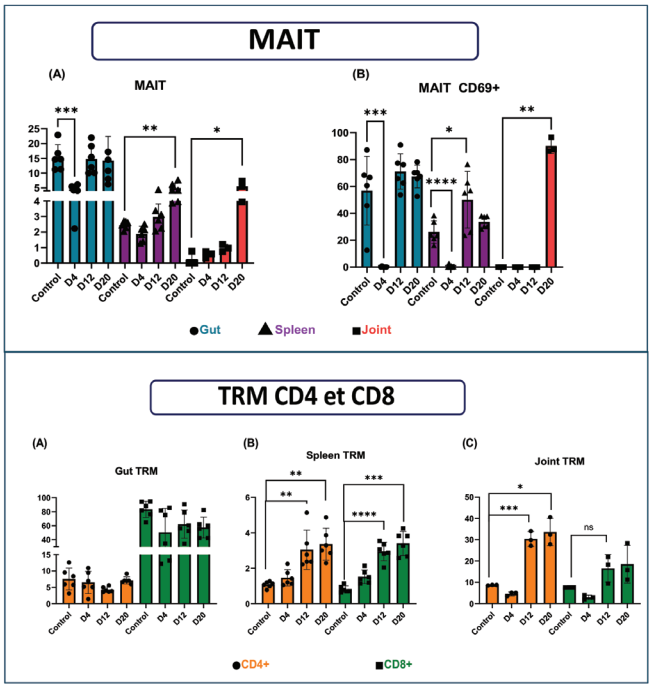


Fig. 1. Temporal evolution of IL-17A-secreting immune cell populations in the gut, spleen, and synovium during arthritis development.

P109

PROINFLAMMATORY STRAINS OF *MEDITERRANEIBACTER GNAVUS* ARE ENRICHED DURING SPONDYLOARTHRITIS AND BOLSTER DISEASE SEVERITY IN ANIMAL MODEL

Jacoutot M.[1,2], Dias Munhoz D.[1,3], Beaufrère M.[1,4], Boukamel A.[1], Gautreau G.[5], Bergot A.S.[6], Said-Nahal R.[1,4], Robert V.[7], Karpman P.[1], Frison E.[1], Ouahbi D.[8], Lamarque D.[1,9], Bazin T.[1,10], Manuello T.[2], Dumas E.[2], Maraquin P.[1], Chatel J.M.[7], Langella P.[7], Elewaut D.[2], Thomas R.[6], Breban M.[1,4*], Glatigny S.[1*]
[1]UMR1173, Université Paris Saclay, Université de Versailles St Quentin en Yvelines, Inserm, Infection et Inflammation, Montigny le Btx, France; [2]VIB Center for Inflammation Research, VIB, Ghent, Belgium and Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [3]Experimental Biology Laboratory Prof. Dr Geraldo Medeiros-Neto, Hospital israelita Albert Einstein, Sao Paulo, Brazil; [4]Rheumatology Division, Ambroise Paré Hospital (AP-HP), Boulogne-Billancourt; [5]Université Paris-Saclay, INRAE, MaIAGE, Jouy-en-Josas, France; [6]Frazer Institute, The University of Queensland, Woolloongabba, Queensland, Australia; [7]Université Paris Saclay, INRAE, AgroParisTech, UMR1319 MICALIS, Jouy-en-Josas; [8]Université Paris-Saclay, INRAE, MGP, Jouy-en-Josas; [9]Gastroenterology Division, Ambroise Paré Hospital (AP-HP), Boulogne-Billancourt; [10]Gastro-enterology Division, Beaujon Hospital (AP-HP), Clichy, France
**Contributed equally.*

Introduction. Spondylarthritis (SpA) is a chronic inflammatory rheumatism with osteoarticular and extra-articular manifestations. Intestinal dysbiosis has been implicated in SpA pathogenesis, notably the expansion of *Mediterraneibacter gnavus* (*MG*), a Gram-positive *coccus* associated with several inflammatory disorders. We aimed to determine whether distinct *MG* strains were associated with SpA and might contribute to disease pathogenesis.
Methods. *MG* strains from SpA patients and healthy controls (HCs) were identified based on metagenomic analysis, isolated from stools and whole-genome sequenced. Pathogenicity of selected strains was assessed by monocolonizing germ-free (GF) SpA-resistant SKG mice. Putative strain selection under inflammatory pressure was examined by culturing *MG* in the presence of nitric oxide (NO). Functional effects were assessed by exposing monocytes from SpA patients to intact *MG* strains.
Results. Phylogenetic analysis separated *MG* strains into three main clades/subclades, using both metagenomic data and sequencing 45 *MG* isolates (38 from SpA and 7 from HCs). SpA-derived strains were significantly enriched in two clades/subclades, whereas the last one was dominated by strains from HCs, suggesting clade-specificity concerning disease association. Monocolonization of GF SKG mice with *MG* restored SpA susceptibility, with stronger colonic inflammation and arthritis observed with SpA-derived strains. Consistently, those strains displayed enhanced resistance to NO. *In vitro* assays further revealed an enrichment of *MG* strains with toxic and proinflammatory properties in SpA.
Conclusion. SpA is associated with a distinct repertoire of MG strains characterized by increased adaptability to inflammatory stress and heightened proinflammatory and toxic potential. These findings establish a link between strain-level microbial diversity and disease susceptibility.

P110

CHARACTERIZATION OF THE ARTICULAR MICROBIOME IN EXPERIMENTAL SPONDYLOARTHRITIS

González-Chávez S.A., Prieto-Carrasco R., Muñoz-Muñoz V., Pacheco-Silva C., Varela-Rodríguez H., Pacheco-Tena C.
PABIOM Laboratory, Faculty of Medicine and Biomedical Sciences, Autonomous University of Chihuahua, Chihuahua, Mexico

Introduction/Objective. Intestinal dysbiosis has been implicated in the gut-joint axis of spondyloarthritis (SpA); however, the articular microbiome remains insufficiently characterized. We characterized knee-associated bacterial communities in DBA/1 mice with spontaneous SpA-like arthritis (DBA/1-SpAD) compared with BALB/c controls, and evaluated overlap with gut, lung and liver microbiomes, as well as viable cultivable bacteria.
Methods. Twenty DBA/1-SpAD and twenty BALB/c mice were included. For microbiome profiling, knee, stool, lung and liver DNA from 10 mice/group were analyzed by 16S rRNA V3 amplicon sequencing. Alpha and beta diversity, taxonomic composition and shared genera were evaluated. Joint tissues were analyzed histologically for bacterial structures/compo-

nents. Bacterial viability was assessed in an independent subset of 10 mice/group using aerobic and anaerobic cultures from knee samples, followed by presumptive identification through biochemical testing.

Results. Knee samples showed detectable bacterial profiles in both groups. Shannon diversity differed among compartments/groups (Kruskal-Wallis $p=4.3\times10^{-4}$), with lower diversity in knee than stool/lung samples. Weighted UniFrac PCoA showed separation across tissue/strain groups (PERMANOVA $R^2=0.48$, $p=0.001$; Fig. 1). Compared with BALB/c controls, DBA/1-SpAD knee samples showed a distinct taxonomic composition, including comparatively higher representation of Aerococcaceae/Aerococcus in descriptive analyses. Other genera detected in knee samples included Staphylococcus and Jeotgalicoccus. Several knee-associated genera were also detected in stool and/or lung, whereas liver samples contained genera shared with knee, gut and lung. Histological analysis identified bacterial components in joint structures. Culture-dependent assays recovered viable bacteria from knee samples, with presumptive biochemical identification of Rodentibacter, Lysinibacillus, Bacillus, Sphingomonas and Staphylococcus taxa (Fig. 2).

Conclusion. DBA/1-SpAD mice exhibit a detectable and compositionally distinct articular microbiome compared with BALB/c controls. Joint bacterial components, genus-level overlap across compartments and cultivable knee-associated bacteria indicate that the articular microbiome warrants further evaluation as a potential contributor to SpA pathogenesis.

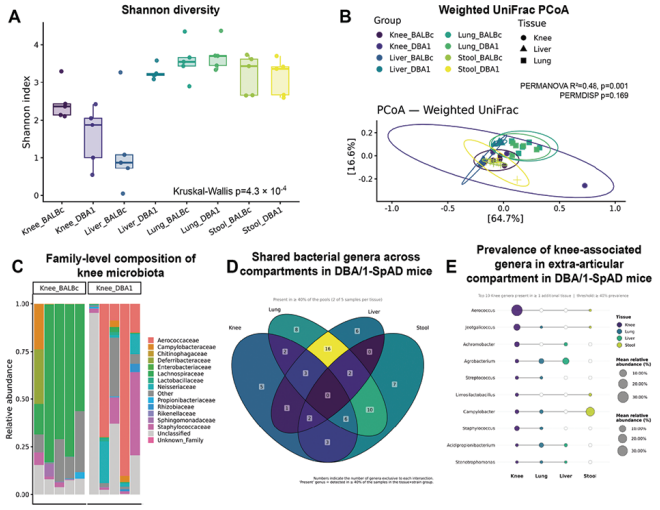


Fig. 1. Articular microbiome composition and inter-compartment bacterial overlap in experimental SpA.
A. Shannon diversity index across knee, liver, lung and stool samples from BALB/c and DBA/1-SpAD mice.
B. Weighted UniFrac PCoA showing beta-diversity separation among tissue/strain groups.
C. Family-level relative abundance of knee microbiota in BALB/c and DBA/1-SpAD mice.
D. Venn diagram showing shared bacterial genera across knee, lung, liver and stool compartments in DBA/1-SpAD mice.
E. Prevalence and mean relative abundance of knee-associated genera detected in extra-articular compartments. Each bar or point represents pooled sequencing libraries. Statistical values are indicated within panels.

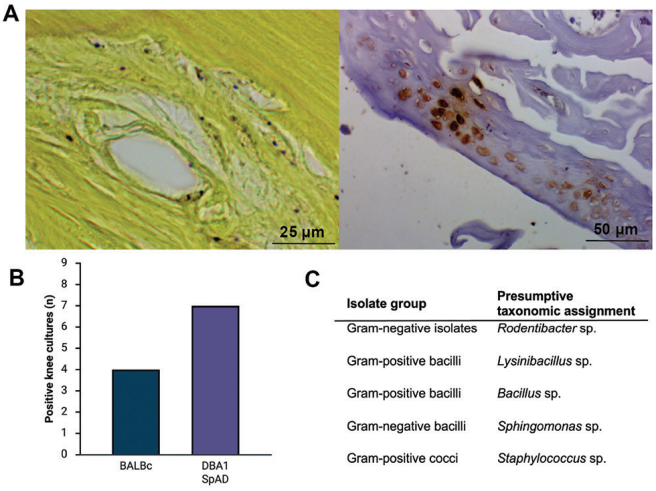


Fig. 2. Histological detection and culture-based recovery of knee-associated bacteria.
A. Representative knee joint images showing bacterial structures by modified Gram staining and lipoteichoic acid (LTA) immunohistochemistry. Scale bars are indicated.
B. Frequency of culture-positive knee samples in BALB/c and DBA/1-SpAD mice; bars indicate positive cultures per group. Fisher's exact test p=0.370.
C. Presumptive biochemical identification of bacterial isolates recovered from knee samples. Molecular confirmation is ongoing.

P111

DONOR VERSUS PLACEBO FAECAL MICROBIOTA TRANSPLANTATION FOR AXIAL SPONDYLOARTHRITIS: PROTOCOL FOR A DOUBLE-BLIND, PLACEBO-CONTROLLED, RANDOMISED TRIAL

Raanes S.S.[1], Johnsen P.H.[2,3], Goll R.[3,4], Østgård R.D.[5], Hansson P.[3], Elvenes J.[6], Hansen I.M.[7], Njøs H.[8], Heiberg M.[9], Hagfors J.[10], Bakland G.[1,3]
[1]Dept. of Rheumatology, University Hospital of North Norway (UNN), Tromsø; [2]Dept. of Medicine, University Hospital of North Norway (UNN), Harstad; [3]Faculty of Health Science, UiT The Arctic University of Norway, Tromsø; [4]Dept. of Gastroenterology, University Hospital of North Norway (UNN), Tromsø, Norway; [5]Regional Hospital Goedstrup, Hernin, Denmark; [6]Dept. of Rheumatology, University Hospital of North Norway (UNN), Harstad; [7]Dept. of Rheumatology, Helgeland Hospital, Mo I Rana; [8]Dept. of Rheumatology, Nordland Hospital, Bodø; [9]Centre for treatment of Rheumatic and Musculoskeletal Diseases (REMEDY), Diakonhjemmet Hospital, Oslo; [10]Bekhterev Norge, Norway

Introduction/Objective. Axial spondyloarthritis (axSpA) is an inflammatory rheumatic disease affecting the spine, large joints and entheses. Altered gut microbiota may contribute to disease pathogenesis by disrupting the intestinal barrier and activating inflammatory pathways (1). Faecal microbiota transplantation (FMT) has emerged as a potential strategy to modify dysbiosis. Its therapeutic role in axSpA remains unclear. This study's primary aim is to evaluate the efficacy and safety of FMT in patients with active axSpA. Secondary aims are to identify biomarkers associated with treatment response and disease activity using blood and faecal samples collected before and after FMT.

Materials and methods. This study comprises two phase II, single-centre clinical trials (RCT1 and RCT2) with a parallel-group, randomised, double-blind, placebo-controlled design. In each trial, 99 participants with active axSpA, defined as ASDAS-CRP ≥2.1 with either elevated CRP (≥3 mg/L) or active inflammation on MRI within the previous 3 months, are randomised 2:1 to receive a single enema dose of FMT from one of three donors or placebo. Follow-up is blinded to Day 90, followed by an open-label period to Day 365. RCT1 includes participants receiving stable immunomodulatory treatment (TNF, IL-17, or JAK inhibitors), whereas RCT2 includes participants without immunomodulatory treatment. The primary endpoint is the proportion of participants achieving minimal clinically important improvement (MCII), defined as a decrease of ≥1.1 in ASDAS-CRP at Day 90. Secondary endpoints include adverse events, donor microbiota engraftment assessed in stool samples at Day 90, and changes in BASDAI, ASAS40, SF-36/RAND, and other patient-reported outcomes from baseline to Day 90.

Results. Recruitment is ongoing.
Conclusion. These trials are designed to evaluate the efficacy and safety of FMT in patients with and without concomitant immunomodulatory therapy, and to assess whether clinical improvement is associated with changes in the gut microbiota. Trial registration: ClinicalTrials.gov (NCT06451588).
Acknowledgements. We would like to thank Bekhterev Norge – the Norwegian axial spondyloarthritis patient organisation and Harstad Revmatikerforening for financial support as well as Northern Norway Regional Health Authority (Helse Nord RHF) for a public funding grant.

Reference

1. MAURO D, CAI B, CIANCIO A *et al.*: The role of the gut and intestinal dysbiosis in the pathogenesis of spondyloarthritis. *Joint Bone Spine* 2025; 92(6): 105923. doi: 10.1016/j.jbspin.2025.105923

P112

RESPONSE-DEPENDENT GUT MICROBIOME TRAJECTORIES DURING BIOLOGIC THERAPY IN RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS: 2-YEAR RESULTS FROM GESPIC

Rios Rodriguez V.[1], Essex M.[2,3,4], Torgutalp M.[1,5], Proft F.[1], Haibel H.[1], Protopopov M.[1], Rademacher J.[1], Siegmund B.[1], Forslund S.K.[2,3], Poddubnyy D.[1,6,7]
[1]Dept. of Gastroenterology, Infectiology and Rheumatology, Charité – Universitätsmedizin Berlin, Berlin; [2]Charité Experimental and Clinical Research Center (ECRC), Berlin; [3]Charité – Universitätsmedizin Berlin, Berlin, Germany; [4]Leiden University Center for Infectious Diseases (LUCID), LUMC, Leiden, The Netherlands; [5]Fraunhofer Institute for Translational Medicine and Pharmacology ITMP, Berlin, Germany; [6]Division of Rheumatology, Temerty Faculty of Medicine, University of Toronto, Toronto; [7]Division of Rheumatology, Schroeder Arthritis Institute, University Health Network, Toronto, Canada

Introduction/Objective. The gut microbiome is increasingly implicated in the pathogenesis of axial spondyloarthritis (axSpA). However, evidence linking microbiome signatures to clinical response to biological disease-modifying antirheumatic drugs (bDMARDs) remains limited. We investigated baseline microbiome signatures associated with future treatment response and longitudinal microbiome trajectories during two years of bDMARD therapy.
Materials and methods. A total of 62 patients with radiographic (r)-axSpA from the GESPIC-AS subcohort initiating bDMARDs and 62 controls with chronic back pain were included. Stool samples were collected at baseline, year 1 and year 2 (247 samples total). Shotgun metagenomic sequencing was performed and taxonomic and functional profiling were generated using the CHAMP pipeline. Clinical response was defined as ASDAS clinically important improvement (ASDAS-CII). Variance partitioning, linear mixed-effects models, and response-stratified interaction analyses were applied.
Results. Patients had mean(SD) age 38.7(10.7) years, 62.3% male, 83.6% HLA-B27 positive, mean ASDAS 3.44(0.78) and 81.9% were biologic-naive. ASDAS-CII was achieved by 67.2% at year 1 and 72.1% at year 2. Alpha diversity remained stable over time, whereas Bray-Curtis profiles differed across clinical groups, with non-responders showing the most distinct composition before and after treatment (PERMANOVA p=0.049 overall; p=0.009 among patients) -Fig1. Shared post-treatment shifts included depletion of *Veillonella rogosae* and enrichment of *Prevotella rara*, *Copromonas* and Clostridia-related CAG lineages. Functional remodelling involved increased histamine synthesis and reduced propionate-production potential. Baseline responder/non-responder differences were modest and involved selected taxa and metabolic pathways. In contrast, longitudinal analyses revealed broader restructuring in non-responders, including TANB77/CAG-508/*Copromonas* enrichment and divergent DOPAC, glycerol-degradation and 4-aminobutyrate trajectories.
Conclusion. Biologic therapy in r-axSpA was accompanied by selective, response-dependent microbiome remodelling. Rather than a static microbial profile, non-response was associated with distinct taxonomic and functional trajectories during treatment, linking clinical heterogeneity to gut microbial pathways involved in SCFA, histamine and neuroactive metabolite metabolism.

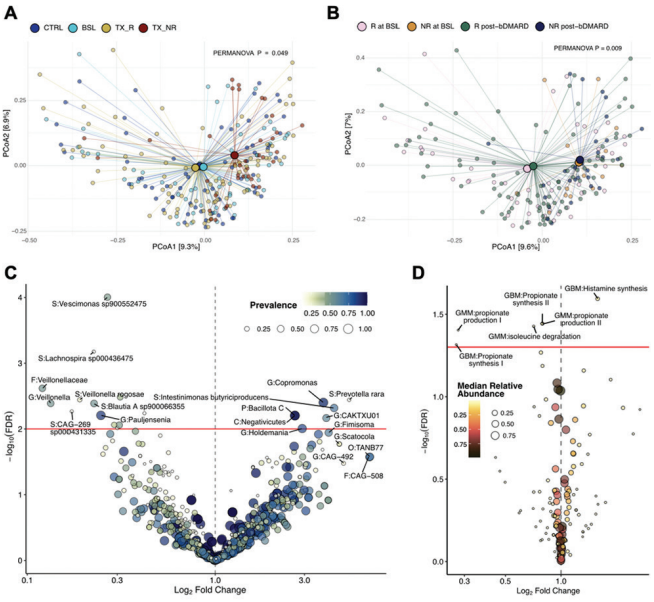


Fig. 1. Community-level diversity and shared microbiome shifts following bDMARD initiation in r-axSpA.
A. Principal coordinates analysis (PCoA) of Bray-Curtis dissimilarities comparing controls and patients with r-axSpA at baseline and after treatment.
B. PCoA restricted to patients, comparing responders and non-responders before and after treatment. Overall group differences were assessed by PERMANOVA.
C. Differentially abundant microbial species following bDMARD initiation.
D. Differentially abundant functional modules following bDMARD initiation. Volcano plots show estimated log2 fold changes versus statistical significance; labelled features represent the most strongly associated taxa or modules.
CRTL: controls; BSL: baseline; TX: treatment; R: responder, NR: non-responder; bDMARD: biological disease-modifying anti-rheumatic drug

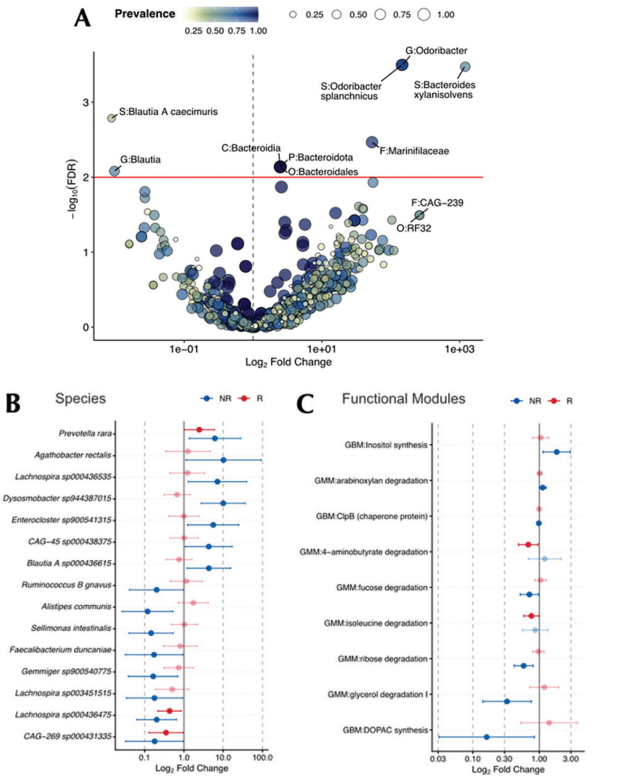


Fig. 2. Gut microbial features associated with clinical response to biologic therapy in r-axSpA.
A. Baseline taxonomic differences between future responders (R) and non-responders (NR).
B. Species showing distinct longitudinal trajectories according to clinical response after bDMARD initiation.
C. Functional modules showing distinct longitudinal trajectories according to clinical response. Points represent effect estimates from response-stratified mixed-effects models; error bars indicate 95% confidence intervals.

P113

BACTERIAL TRANSLOCATION IN SPONDYLOARTHRITIS: EVALUATION BEFORE AND 3 MONTHS AFTER INITIATION OF BIOLOGIC DMARDS (TEARE BIO STUDY)

Verhoeven F.[1,2], Miceli-Richard C.[3], Ramon A.[2,4], Vauchy C.[2,5], Meyer F.[1,2], Pais De Barros J.P.[6], Saas P.[7], Prati C.[1,2], Desmarets M.[5,*], Wendling D.[1,*]
[1]Rhumatologie, CHU de Besancon, Besançon; [2]Université Marie et Louis Pasteur, EFS, INSERM UMR 1098 RIGHT, Besançon; [3]Service de Rhumatologie, Hôpital Cochin, Paris; [4]Service de Rhumatologie, CHU Dijon, Dijon; [5]INSERM Centre InvestigationClinique CIC-1431, CHU de Besancon, Besançon; [6]Plateforme Diviomics, US 58 BioSanD, Université Bourgogne Europe, Dijon; [7]Univ. Grenoble-Alpes, INSERM U1209, CNRS UMR5309, EFS AuRA, Institute for Advanced Biosciences, Grenoble, France
**equal contribution*

Introduction. The objective of this study is to evaluate the impact of biologic therapies targeting TNF or IL-17 on serum levels of LPS and its subtypes and the impact of the level of LPS on the BASDAI 50 responses.
Methods. This multicentre controlled study (NCT05244109), included biologic-naive patients with axSpA fulfilling the 2009 ASAS criteria. Patients with inflammatory bowel disease (IBD) or suspected IBD were excluded. Three groups were defined: first-line anti-TNF, first-line anti-IL-17, and NSAIDs alone. Assessments were performed at baseline and at 3 months. Serum levels of total LPS and LPS subtypes (3OHC10, 12, 14, 16, and 18) were measured using liquid chromatography coupled with mass spectrometry. Clinical outcomes (ASDAS-CRP, BASDAI, BASDAI 50 response) and demographic data were collected.
Results. A total of 77 patients were included. Patient characteristics are presented in Table I.
At baseline, total LPS levels did not differ significantly between groups or according to disease activity. In contrast, C10 levels (3-hydroxydecanoic acid) were lower in the NSAID group, both at baseline and at 3 months ($p < 0.05$). At 3 months, changes in BASDAI, ASDAS-CRP, and BASDAI 50 response rates were comparable between anti-TNF and anti-IL-17 groups. A trend toward increased LPS levels was observed with anti-TNF (Table II). Among LPS subtypes, only an inverse correlation between changes in C10 levels and changes in BASDAI/ASDAS-CRP was observed ($p<0.05$). LPS levels and subtypes did not differ according to BASDAI 50 response.

Table I. Patients characteristics at baseline.

Characteristic	NSAIDs n=9	ANTI IL-17 n=38	ANTI-TNF n=28	*p*-value
Age Median (Q1, Q3)	39.0 (32.0, 46.0)	43.5 (34.0, 50.0)	35.5 (31.5, 55.5)	0.87
Sex (% male)	3 (33.3%)	22 (57.9%)	15 (53.6%)	0.44
smoking (PA)	5.0 (2.0, 10.0)	5.0 (3.0, 16.0)	5.0 (2.0, 15.0)	0.66
Alcohol intake	0 (0.0%)	16 (42.1%)	11 (39.3%)	0.012
Uveitis story	0 (0.0%)	2 (5.3%)	7 (25.0%)	0.062
Psoriasis story	1 (11.1%)	4 (10.5%)	4 (14.3%)	0.83
Dactylitis story	2 (22.2%)	1 (2.6%)	6 (21.4%)	0.032
SpA in the family	3 (33.3%)	6 (15.8%)	8 (28.6%)	0.33
Rx Sacroiliitis (NY)	3 (33.3%)	22 (57.9%)	16 (57.1%)	0.44
Syndesmophyte	1 (12.5%)	7 (19.4%)	6 (23.1%)	0.45
HLA B27	7 (77.8%)	24 (63.2%)	20 (71.4%)	0.63
Disease duration since diagnosis	9.0 (2.0, 10.0)	2.4 (0.5, 10.4)	7.2 (1.0, 12.1)	0.26
ESR	15.0 (6.0, 29.5)	8.0 (5.0, 18.0)	13.0 (8.0, 54.0)	0.10
CRP	1.0 (0.6, 2.1)	2.8 (1.0, 8.3)	14.0 (2.0, 24.5)	0.003
BMI	21.1 (18.0, 25.2)	24.0 (21.7, 26.8)	24.4 (22.2, 27.6)	0.28
BASDAI V1	2.4 (1.7, 2.6)	5.1 (3.6, 6.6)	5.4 (3.6, 6.7)	0.003
ASDAS CRP V1	1.1 (0.8, 1.6)	2.9 (2.4, 3.5)	3.1 (2.8, 3.9)	<0.001
ASAS NSAIDs V1	75.0 (50.0, 100.0)	47.6 (12.8, 83.3)	58.4 (48.4, 74.3)	0.70
BASFI V1	0.0 (0.0, 0.3)	2.8 (1.5, 4.0)	3.4 (2.3, 6.0)	0.004

Table II. Evolution of the LPS and its different sub types in relation with the treatment.

Difference between V1 and V2	AINS n=9[1]	ANTI IL-17 n=38[1]	ANTI-TNF n=28[1]	*p*-value[2]
Δ LPS total	9.4 (224.2)	19.0 (190.7)	94.9 (220.4)	0.29
Δ c10	-13.9 (40.5)	-1.3 (54.7)	22.7 (66.7)	0.14
Δ C12	-0.8 (13.4)	2.8 (15.2)	1.6 (19.4)	0.83
Δ C14	5.3 (27.6)	3.9 (21.0)	2.6 (32.3)	0.96
Δ C16	1.1 (95.4)	5.2 (77.3)	26.0 (78.5)	0.53
Δ C18	17.8 (78.7)	8.3 (66.7)	42.0 (82.3)	0.19

[1]Mean (SD), [2]One-way analysis of means,* $p<0.05$; **$p<0.01$.

Conclusion. In patients with axSpA, total serum LPS levels are not associated with disease activity. C10 levels were higher in patients requiring biologic therapy but did not differ according to the biologic agent used and did not predict clinical response. These findings suggest that LPS measurement is not a relevant biomarker for monitoring disease activity or response to bDMARDs in axSpA.

P114

TREATMENT PERSISTENCE OF ORIGINAL VERSUS BIOSIMILAR TNF-ALPHA INHIBITORS IN SPONDYLOARTHRITIS: A PORTUGUESE MULTICENTRE REAL-WORLD STUDY

Almeida I.[1,*], Saraiva L.[1,*], Henriques C.[2,3], Matos A.[2,4], Almeida S.[5], Cunha A.[5], Rodrigues S.[6], Tremoceiro J.[6], Natal M.[7], Bernardes M.[7], Raimundo D.[8], Martins C.[8], Rodrigues M.M.[9], Carones A.[9], Reynolds L.[10], Miguel C.[10], Sushko U.[11], Novais T.[12], Matias C.[12], Oliveira D.[13], Fonseca R.[13], Teixeira P.M.[14], Samões B.[15], Silva L.[15], Rua C.[16], Pacheco M.S.[17], Nero P.[18], Carneiro H.[19], Silva-Dinis J.[20], Dias J.M.[21], Gonçalves H.[22], Marona J.[23], Tavares-Costa J.[24], Araújo F.[25], Manica S.[26], Mateus M.[27], Garcia J.[28], Couto M.[1]
[1]Dept. of Rheumatology, Unidade Local de Saúde Viseu Dão-Lafões, Viseu; [2]School of Technology and Management, Polytechnic Institute of Viseu, Viseu; [3]Centre for Mathematics of the University of Coimbra - CMUC, Coimbra; [4]Health Sciences Research Unit: Nursing - UICISA: E, Coimbra; [5]Dept. of Rheumatology, Unidade Local de Saúde do Alto Minho, Ponte de Lima; [6]Dept. of Rheumatology, Unidade Local de Saúde de Lisboa Ocidental, Lisboa; [7]Dept. of Rheumatology, Unidade Local de Saúde de São João, Porto; [8]Dept. of Rheumatology, Unidade Local de Saúde Santa Maria, Lisboa; [9]Dept. of Rheumatology, Unidade Local de Saúde de Coimbra, Coimbra; [10]Dept. of Rheumatology, Instituto Português de Reumatologia, Lisboa; [11]Dept. of Rheumatology, Hospital do Divino Espírito Santo, Ponta Delgada; [12]Dept. of Rheumatology, Unidade Local de Saúde Almada-Seixal, Almada; [13]Dept. of Rheumatology, Unidade Local de Saúde Entre Douro e Vouga, Santa Maria da Feira; [14]Dept. of Rheumatology, Unidade Local de Saúde Região de Aveiro, Aveiro; [15]Dept. of Rheumatology, Unidade Local de Saúde Trás-os-Montes e Alto Douro, Vila Real; [16]Dept. of Rheumatology, Unidade Local de Saúde de Gaia Espinho, Vila Nova de Gaia; [17]Dept. of Rheumatology, Unidade Local de Saúde da Cova da Beira, Covilhã; [18]Dept. of Rheumatology, Hospital CUF Descobertas, Lisboa; [19]Dept. of Rheumatology, Hospital CUF Tejo, Lisboa; [20]Dept. of Rheumatology, Unidade Local de Saúde São José, Lisboa; [21]Dept. of Rheumatology, Unidade Local de Saúde do Médio Tejo, Torres Novas; [22]Dept. of Rheumatology, Unidade Local de Saúde de Braga, Braga; [23]Dept. of Rheumatology, Unidade Local de Saúde da Lezíria, Santarém; [24]Dept. of Rheumatology, Hospital Lusíadas Braga, Braga; [25]Rheumatology and Osteoporosis Unit, Hospital de Sant'Ana, Cascais; [26]Dept. of Rheumatology, Montepio Rainha D. Leonor, Caldas da Rainha; [27]Dept. of Rheumatology, Clínica CUF Almada, Almada; [28]Dept. of Rheumatology, Unidade Local de Saúde do Médio Tejo, Tomar, Portugal
**These authors contributed equally and share first authorship.*

Objective. To compare 36-month treatment persistence between original and biosimilar tumour necrosis factor-alpha inhibitors (TNFi) in a Portuguese multicentre cohort of patients with spondyloarthritis (SpA) as a real-world measure of treatment effectiveness and safety. To evaluate clinical response outcomes.
Materials and methods. A retrospective multicentre study using data from the Rheumatic Diseases Portuguese Registry (Reuma.pt) was conducted. Adult patients with axial or peripheral SpA initiating original TNFi (Humira®, Enbrel®, Simponi®) or biosimilars (Amgevita®, Hulio®, Hyrimoz®, Idacio®, Imraldi®, Yuflima®, Benepali®, Erelzi®) were included. Treatment persistence was analysed using multivariable Cox regression and inverse probability of treatment weighting (IPTW). Analyses were performed in the overall and post-2017 cohorts. Clinical outcomes were assessed using multivariable logistic regression models.
Results. A total of 2121 patients were included: 1583 (74.6%) received original TNFi and 538 (25.4%) biosimilars. Baseline differences included age, smoking status, HLA-B27 positivity, concomitant cDMARD use, concomitant corticosteroid use, and treatment era (Table I). At 36 months, treatment discontinuation occurred in 21.1% and 25.3% of original- and biosimilar-treated patients, respectively, with no significant differences in treatment persistence after multivariable adjustment (adjusted HR 1.05, 95%CI 0.82–1.35, $p=0.690$; Fig. 1) or IPTW analysis (HR 1.08, 95%CI 0.83–1.40, $p=0.554$). Similar findings were observed in the post-2017 restricted cohort and in subgroup analyses comparing Enbrel® versus Benepali®, Humira® versus Amgevita®, and Humira® versus Imraldi® (Table I). Regarding clinical outcomes, after multivariable adjustment, biosimilar treatment was associated with lower odds of ASDAS response at 6 months (adjusted OR 0.61, 95%CI 0.41–0.92, $p=0.018$), with no differences for other outcomes or later timepoints.
Conclusion. No significant differences in long-term treatment persistence were observed between original and biosimilar TNFi after adjustment for baseline imbalance. Differences in clinical response were limited to ASDAS response at 6 months. These findings suggest comparable real-world effectiveness of biosimilar TNFi in SpA.

Table I. Baseline characteristics and adjusted treatment persistence analyses in original- and biosimilar-treated patients with spondyloarthritis.

	Original (n=1583)	Biosimilar (n=538)	p-value
Male, n (%)	887 (56.0)	281 (52.2)	0.126
Age (years), mean (SD)	42.6 (12.0)	44.6 (12.6)	0.001
Smoking status, n (%) Never smoker Former smoker Current smoker	 630 (54.4) 242 (20.9) 286 (24.7)	 255 (63.4) 61 (15.2) 86 (21.4)	0.005
HLA-B27 positivity, n (%)	952 (77.8)	309 (71.0)	0.004
Disease duration, mean (SD)	12.9 (10.3)	13.3 (11.3)	0.962
bDMARD naive, n (%)	1464 (92.5)	487 (90.5)	0.148
Comorbidities, n (%) Hypertension Dyslipidaemia Diabetes Hypothyroidism Solid tumour	 181 (11.4) 138 (8.7) 53 (3.3) 16 (1.0) 29 (1.8)	 60 (11.2) 59 (11.0) 19 (3.5) 11 (2.0) 7 (1.3)	 0.859 0.121 0.839 0.065 0.410
Concomitant treatment NSAIDs, n (%) csDMARDs, n (%) Glucocorticoids, n (%)	 986 (62.3) 748 (47.3) 346 (21.9)	 341 (63.4) 221 (41.1) 96 (17.8)	 0.650 0.013 0.048
Treatment initiation before 2017, n (%)	1105 (69.8)	2 (0.4)	<0.001
Treatment persistence analyses			
	Adjusted_HR	IPTW_HR	p-value
Original vs. biosimilar TNFi in the overall cohort	1.05 (0.82–1.35)	1.08 (0.83–1.40)	0.690 / 0.554
Original vs. biosimilar TNFi in the post-2017 cohort	1.05 (0.81–1.35)	1.03 (0.80–1.33)	0.728 / 0.798
Enbrel® vs. Benepali®	1.80 (0.97–3.32)	1.80 (0.89–3.67)	0.060 / 0.103
Humira® vs. Amgevita®	0.762 (0.47–1.23)	0.741 (0.46–1.19)	0.266 / 0.219
Humira® vs. Imraldi®	1.23 (0.83–1.82)	1.33 (0.91–1.97)	0.297 / 0.145

csDMARDs: conventional synthetic disease-modifying anti-rheumatic drugs; HR: hazard ratio; IPTW: inverse probability of treatment weighting; NSAIDs: non-steroid anti-inflammatory drugs; SD: standard deviation; TNFi: tumour necrosis factor-alpha inhibitors.

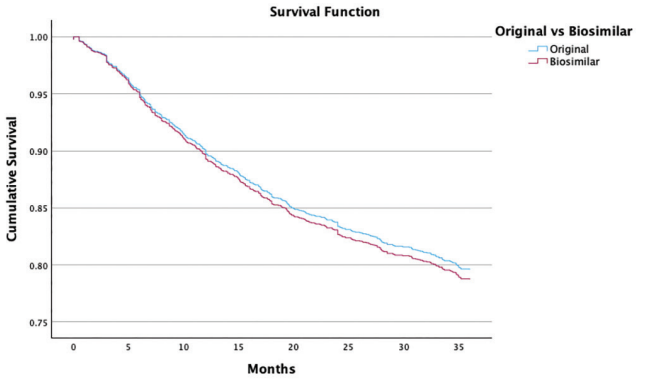


Fig. 1. Persistence in treatment with original and biosimilar TNF-alpha inhibitors in patients with spondyloarthritis.

P115

TWENTY YEARS OF REAL-WORLD BIOLOGIC TREATMENT TRAJECTORIES IN SPONDYLOARTHRITIS: DRUG SURVIVAL, SWITCHING PATTERNS, AND REMISSION OUTCOMES FROM THE HUR-BIO REGISTRY

Ayan G., Kiliç L., Kalyoncu U.
Dept. of Internal Medicine, Division of Rheumatology, Hacettepe University, Ankara, Turkey

Objective. To evaluate treatment patterns, switching dynamics, treatment persistence, and remission outcomes among patients with SpA receiving biologic and targeted synthetic therapies in routine clinical practice.
Methods. This retrospective registry-based study included 3,623 patients with SpA treated with biologic or targeted synthetic therapies in the HUR-BIO database, initiated in 2005. Treatment utilization was analyzed across sequential treatment lines. Drug survival was calculated from treatment initiation to treatment switch, with censoring at the last follow-up visit for patients without switching. Remission analyses were based on BASDAI low disease activity/remission (<4) assessed at 3–6, 6–12, and 12–24 months.
Results. Overall, 13 treatment lines were observed. TNF inhibitors (TNFi) dominated treatment utilization, with adalimumab representing 41.3%, followed by etanercept (22.1%) and infliximab (15.3%) as 1st line, while IL-17i and JAKi were mainly used in later treatment lines (Table I). Etanercept demonstrated the longest median drug survival during first-line therapy (73.2 months, followed by certolizumab (63.3 months) and adalimumab (56.6 months), while treatment persistence progressively decreased across subsequent treatment lines. BASDAI remission rates at 3–6 months ranged from 52.4% to 60.8% across most first-line TNFi therapies, highest for certolizumab. At 6–12 months, remission rates remained relatively stable (48.0–57.0%), while sustained remission at 12–24 months ranged from 47.3% to 63.6%, highest for golimumab and certolizumab. Treatment trajectories and sequencing strategies across biologic lines were additionally visualized using Sankey analyses in Figure 1.
Conclusion. This large real-world registry spanning nearly two decades demonstrates the central role of TNFi therapies in SpA management, with favorable long-term persistence and sustained remission outcomes in earlier treatment lines. Increasing switch rates and declining treatment persistence across subsequent lines highlight the challenges associated with refractory disease in routine clinical practice.

Table I. Distribution of biologic and targeted synthetic therapies across sequential treatment lines in patients with spondyloarthritis.

Drug molecule	Line 1	Line 2	Line 3	Line 4	Line 5	Line 6	Line 7	Line 8	Line 9	Line 10	Line 11	Line 12	Line 13	Total
Adalimumab	1495 (41.26%)	416 (27.42%)	134 (19.62%)	56 (15.60%)	25 (13.74%)	11 (12.09%)	3 (7.50%)	3 (14.29%)	2 (14.29%)	0 (0%)	1 (16.67%)	0 (0%)	0 (0%)	2146 (32.77%)
Certolizumab pegol	376 (10.38%)	219 (14.44%)	137 (20.06%)	65 (18.11%)	34 (18.68%)	19 (20.88%)	6 (15%)	3 (14.29%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	859 (13.12%)
Etanercept	802 (22.14%)	380 (25.05%)	113 (16.54%)	62 (17.27%)	22 (12.09%)	6 (6.59%)	5 (12.50%)	1 (4.76%)	0 (0%)	1 (11.11%)	0 (0%)	0 (0%)	0 (0%)	1392 (21.26%)
Golimumab	308 (8.50%)	89 (5.87%)	48 (7.03%)	25 (6.96%)	10 (5.49%)	5 (5.49%)	1 (2.50%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	486 (7.42%)
Infliximab	555 (15.32%)	251 (16.55%)	109 (15.96%)	54 (15.04%)	35 (19.23%)	12 (13.19%)	8 (20%)	6 (28.57%)	3 (21.43%)	2 (22.22%)	2 (33.33%)	2 (66.67%)	1 (100%)	1040 (15.88%)
Ixekizumab	0 (0%)	0 (0%)	2 (0.29%)	5 (1.39%)	0 (0%)	1 (1.10%)	0 (0%)	2 (9.52%)	0 (0%)	0 (0%)	0 (0%)	1 (33.33%)	0 (0%)	11 (0.17%)
Secukinumab	82 (2.26%)	128 (8.44%)	107 (15.67%)	70 (19.50%)	35 (19.23%)	17 (18.68%)	14 (35%)	3 (14.29%)	1 (7.14%)	1 (11.11%)	0 (0%)	0 (0%)	0 (0%)	458 (6.99%)
Tofacitinib	5 (0.14%)	22 (1.45%)	12 (1.76%)	5 (1.39%)	6 (3.30%)	6 (6.59%)	0 (0%)	2 (9.52%)	6 (42.86%)	1 (11.11%)	1 (16.67%)	0 (0%)	0 (0%)	66 (1.01%)
Upadacitinib	0 (0%)	12 (0.79%)	21 (3.07%)	17 (4.74%)	15 (8.24%)	14 (15.38%)	3 (7.50%)	1 (4.76%)	2 (14.29%)	4 (44.44%)	2 (33.33%)	0 (0%)	0 (0%)	91 (1.39%)
Total	3623 (100%)	1517 (100%)	683 (100%)	359 (100%)	182 (100%)	91 (100%)	40 (100%)	21 (100%)	14 (100%)	9 (100%)	6 (100%)	3 (100%)	1 (100%)	6549 (100%)

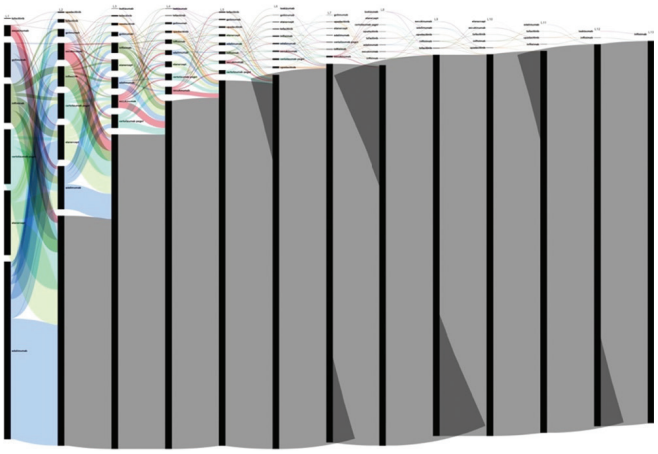


Fig. 1. Treatment trajectories and switching patterns across sequential biologic and targeted synthetic therapy lines in spondyloarthritis patients visualized by Sankey analysis.

P116

FOUR DISTINCT TARGETED THERAPY RESPONSE TRAJECTORIES IN AXIAL SPONDYLOARTHRITIS: EVIDENCE FOR EARLIER TREATMENT INITIATION

Burrows D.[1], Ehrhardt M.J.[2], Barnett R.[3], Sengupta R.[3]
[1]Institute for Mathematical Innovation, University of Bath, Bath; [2]Dept. of Mathematical Sciences, University of Bath, Bath; [3]Dept. of Rheumatology, Royal United Hospitals Bath NHS Foundation Trust, Bath, UK

Introduction. Longitudinal axSpA datasets offer critical insights into disease progression, treatment effects, and flare dynamics. Patient subgroups sharing similar disease trends may exist, offering a pathway into personalised treatment, yet require data analysis tools for their recovery.
Methods. A generalisation of K-means clustering was developed, tailored to longitudinal patient data. Patient subgroups are identified, enabling exploratory analysis of disease trajectories including targeted therapy sequencing effects. AxSpA patient data from the Bath SpA Biobank at the Royal National Hospital for Rheumatic Diseases, Bath was analysed. Non-smoking targeted therapy-treated patients were clustered into subgroups using joint BASFI, BASDAI, BASMI, and FACIT-F trajectories, time-anchored to first targeted therapy initiation. AsQol trajectories were computed post-hoc to assess quality-of-life effects.
Results. Four distinct patient subgroups were identified. Clusters 3 and 4 exhibit the most significant improvement in BASDAI, BASFI, and FACIT-F from targeted therapy treatment, highlighting these subpopulations as those benefitting most from the intervention. Across all clusters, BASMI did not improve following the intervention, aligning with the expectation that biologics rarely impact spinal mobility. The age demographics in Figure 2 correlate with the treatment benefits in Figure 1, with younger patients with shorter diagnosis times mapping to more positive targeted therapy responses. The clusters with the two youngest subpopulations also exhibited the steepest improvement in AsQol, suggesting that younger patients experience the greatest improvement in quality-of-life following targeted therapy interventions.

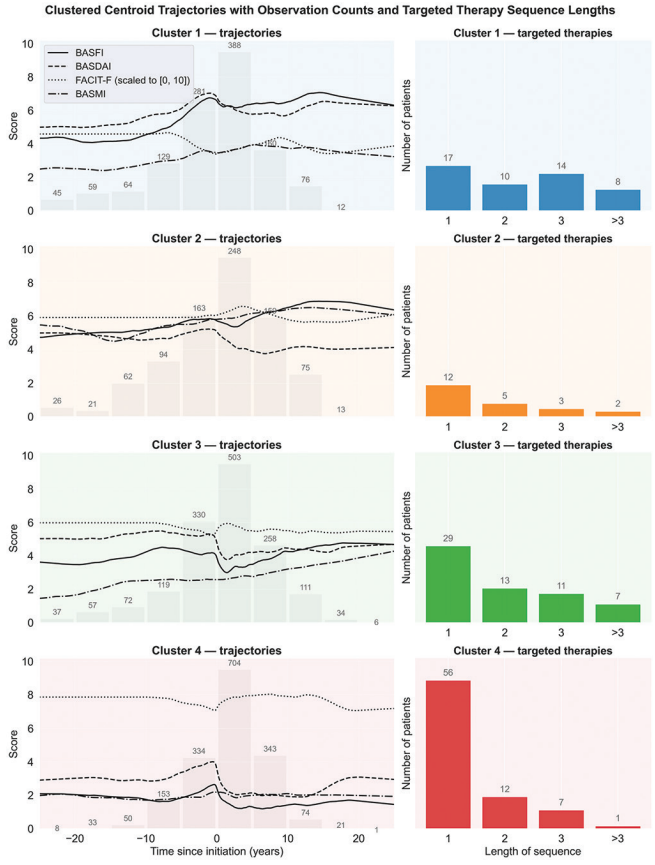


Fig. 1.

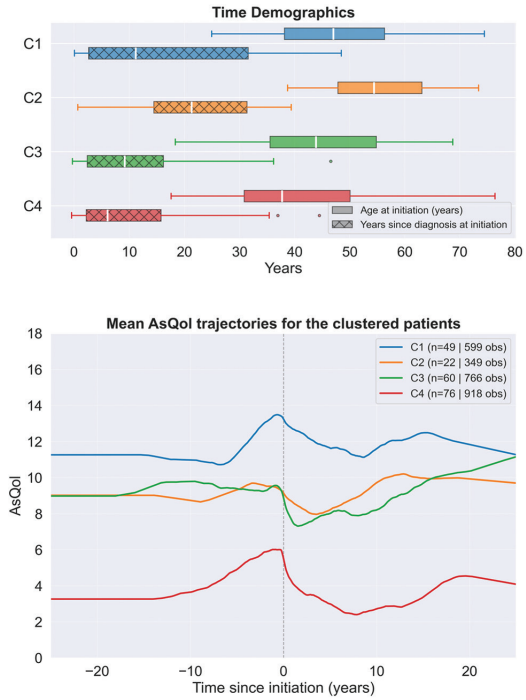


Fig. 2.

Discussion. These findings suggest that targeted therapy interventions are most effective when patients are young and/or earlier in their disease development. For the older subpopulations in Clusters 1 and 2, alternative interventions may be more beneficial. Future work will disentangle age, time-since-diagnosis and other candidate variables as predictors and assess the impact of alternative interventions in older subpopulations. These findings support earlier targeted therapy initiation as a treatment strategy in axSpA.

P117

INFLIXIMAB VS ADALIMUMAB FOR UVEITIS PREVENTION IN ANKYLOSING SPONDYLITIS: A META-ANALYSIS

Chen S., Zhang Y., Gu J.
Rheumatology immunology Dept., Third Affiliated Hospital of Sun Yat-sen University, Guangzhou, China

Introduction. Uveitis is the most common extra-articular manifestation of ankylosing spondylitis (AS), and the prevention effects may be different among different tumor necrosis factor inhibitors (TNFi). The purpose of this study was to systematically compare the efficacy of infliximab (IFX) and adalimumab (ADA) for the prevention of uveitis in patients with AS through a head-to-head real-world cohort study.
Methods. PubMed, Embase, Cochrane Library and Web of Science databases established until April 2026 were systematically searched. Inclusion criteria were cohort studies or randomized controlled trials that directly compared IFX to ADA and reported outcomes for uveitis. The primary outcome was the risk of uveitis with IFX versus ADA after subgroup analyses based on the history of uveitis. A random-effect model was used for Meta-analysis, and the effect amount was determined by risk ratio (HR) and 95% confidence interval (CI). Heterogeneity was assessed by i-statistics.
Results. A total of 11669 patients were included in 7 cohort studies. Meta-analysis shows:
Figure 1 In a patient without a history of uveitis (preventing a first episode), the risk ratio (HR) between IFX and ADA was 1.16 (95% CI 0.92–1.47) and there was no significant difference.
Figure 2 shows a statistically significant difference in HR between IFX and ADA of 1.44 (95% CI 1.05–1.97) in patients with a history of uveitis (relapse prevention), suggesting that the risk of relapse was approximately 44% higher in the IFX group than in the ADA group.
In the overall population without history differentiation, the combined HR

was 1.23 (95% CI 0.89–1.69). Heterogeneity was low to moderate in all analyses.

Conclusion. BASed on the evidence of existing real-world cohort studies, infliximab is as effective as adalimumab in preventing new-onset uveitis in patients with AS. However, patients with a history of uveitis may have a higher risk of relapse after infliximab treatment. This finding provides an important evidence-based reference for clinicians in the selection of TNFi drugs, especially for the high-risk population for relapse.

Protocol registration. PROSPERO (CRD420261389123)

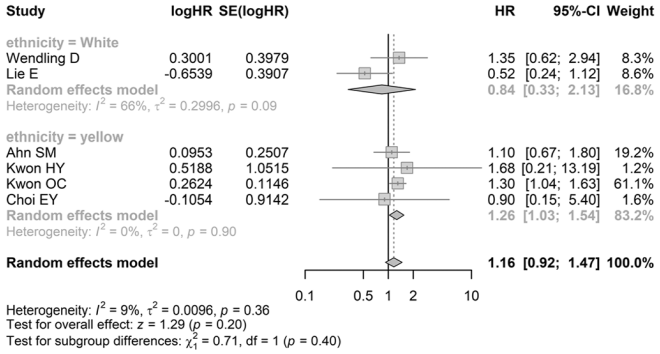


Fig. 1.

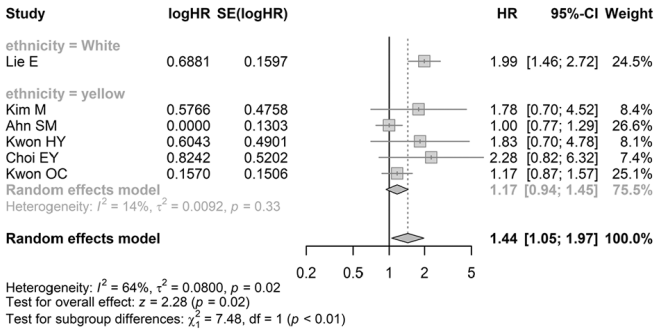


Fig. 2.

P118

EFFICACY OF UPADACITINIB IN NORMALIZING FAECAL CALPROTECTIN IN PATIENTS WITH SPONDYLOARTHRITIS AND PSORIATIC ARTHRITIS

de la Torre-Rubio N., Merino-Argúmanez C., García-Magallón B., Godoy-Tundidor H., Nuño-Nuño L., Martín-Bescos A., Coronado-González M., Calvo-Sparks C., Mazo P., Sanz-Sanz J.

Rheumatology Dept., Hospital Universitario Puerta de Hierro Majadahonda, Majadahonda, Spain

Introduction. Faecal calprotectin (FC) is elevated in approximately 50% of patients with axial spondyloarthritis (SpA) and psoriatic arthritis (PsA), reflecting subclinical intestinal inflammation. This exploratory study evaluates whether upadacitinib (UPA) reduces FC below 80 µg/g (Quantum Blue®, EISER threshold[1]), indicating improvement in intestinal inflammation. The secondary aim was to compare post-treatment FC with baseline levels, when available.

Methods. Real-world retrospective descriptive study. Data collected included age, sex, months since UPA initiation, comorbidities, and diagnosis (axial/peripheral spondyloarthritis and PsA) based on clinical criteria.

Results. Twenty-eight patients were enrolled. Mean age was 55.0 [52.8–62.5] years; 8 (28.6%) males and 20 (71.4%) females. Mean duration from UPA initiation was 18.5 [9.4–26.5] months. Diagnoses comprised spondyloarthritis in 17/28 (60.7%) and PsA in 11/28 (39.3%). Prior biologic therapies included TNF inhibitors 10 (35.7%), IL-17 inhibitors 8 (28.6%), JAK inhibitors 4 (14.3%), IL-23 inhibitors 2 (7.1%), treatment-free ≥6 months 2 (7.1%), and biologic-naive 1 (3.6%). Following UPA initiation, 24/28 (85.7%) achieved FC <80 µg/g versus 4/28 (14.3%) with elevated FC. Baseline FC was available in 14/28 (50%): 3/14 (21.4%) had FC ≥80, all 3 (100%) normalized to <80; 9/11 (81.8%) maintained negative FC and 2/11 (18.2%) became positive; one of the patients is currently being evaluated to rule out inflammatory bowel disease, and the other experienced an episode of anemia and melena at the time the FC sample was collected. Other baseline patient characteristics are summarized in Table I.

Conclusion. We observed that 85% of UPA-treated patients had a CF score <80 µg/g, suggesting effective control of subclinical intestinal inflammation as a surrogate marker of therapeutic response.

Funding. No funding was received for this study.

Reference

1. SANZ-SANZ J *et al*.; EISER Collaborative Group: Prevalence of undiagnosed inflammatory bowel disease in spondyloarthritis patients. *J Clin Med* 2025; 14(13): 4569.

Table I. Baseline patient characteristics.

Characteristics	**n=28**	
Prior biologic therapies		
TNF inhibitors	26	92.9%
IL-17 inhibitors	23	82.1%
JAK inhibitors	7	25.0%
IL-23 inhibitors	3	10.7%
Comorbidities		
Fibromyalgia	4	14.3%
Dyslipidemia	4	14.3%
Crohn's disease	3	10.7%
Asthma	3	10.7%
Sleep apnea syndrome	2	7.1%
Depression	2	7.1%
Multiple sclerosis	2	7.1%
Hypertension	2	7.1%
Hypothyroidism	2	7.1%
Current smokers	2	7.1%
Disease activity measures in last visit		
VAS pain, median [IQR] (n=13)	4.0	[1.0–7.0]
Patient global assessment, median [IQR] (n=13)	3.0	[1.0–5.0]
BASDAI, median [IQR] (n=10)	2.2	[1.6–4.5]
DAPSA, median [IQR] (n=4)	4.8	[1.5–10.6]
MDA, median [IQR] (n=2)	7.0	[7.0–7.0]

BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; DAPSA: Disease Activity index for Psoriatic Arthritis; IL: interleukin; IQR: interquartile range; JAK: Janus kinase; MDA: minimal disease activity; TNF: tumour necrosis factor; VAS: visual analogue scale.

P119

IMPACT OF FILGOTINIB ON MRI INFLAMMATORY AND STRUCTURAL LESIONS IN THE SPINE OF PATIENTS WITH AXIAL SPONDYLOARTHRITIS: 52-WEEK RESULTS FROM THE PHASE 3 OLINGUITO TRIAL

Maksymowych W.P.[1], Navarro-Compán V.[2], Machado P.M.[3,4,5], Gheyle L.[6], Mangili A.[7], Le Brun F.-O.[7], Muller K.[6], de Hooge M.[8], Lambert R.G.W.[9], Landewé R.[10,11], Østergaard M.[12,13], Baraliakos X.[14]

[1]Dept. of Medicine, University of Alberta, Edmonton, AB, Canada; [2]La Paz University Hospital, Hospital La Paz Institute for Health Research (IdiPAZ), Madrid, Spain; [3]Dept. of Neuromuscular Diseases, University College London, London; [4]NIHR University College London Hospitals Biomedical Research Centre, University College London, Hospitals NHS Foundation Trust, London; [5]Dept. of Rheumatology, Northwick Park Hospital, London North West University Healthcare NHS Trust, London, UK; [6]Alfasigma Belgium BV, Mechelen, Belgium; [7]Alfasigma Schweiz AG, Zofingen, Switzerland; [8]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [9]Dept. of Radiology and Diagnostic Imaging, University of Alberta, Edmonton, AB, Canada; [10]Dept. of Clinical Immunology and Rheumatology, Amsterdam University Medical Center, Amsterdam; [11]Dept. of Rheumatology, Zuyderland Medical Center, Heerlen, The Netherlands; [12]Dept. of Clinical Medicine, University of Copenhagen, Copenhagen; [13]Copenhagen Center for Arthritis Research (COPECARE), Center for Rheumatology and Spine Diseases, Rigshospitalet, Glostrup, Denmark; [14]Rheumazentrum Ruhrgebiet Herne, Ruhr University Bochum, Bochum, Germany

Introduction. The Phase 3 OLINGUITO trial (NCT05785611) evaluated the efficacy and safety of filgotinib in patients with axial spondyloarthritis

(axSpA). We report the effect of filgotinib on spinal inflammation and structural damage progression.

Methods. OLINGUITO comprises two, randomized, double-blind, placebo-controlled studies in patients with radiographic or nonradiographic axSpA, with inadequate response or intolerance to conventional therapy. Patients were randomized to receive once daily filgotinib (200 mg) or placebo to Week (W) 16, then filgotinib (200 mg) only through W52 in the open-label period (patients ≥65 years and/or with prespecified risk factors received response-based dosing). MRIs were completed at baseline, W16 and W52, and evaluated using SPARCC and CANDEN scores. Change from baseline (CFB) in SPARCC/CANDEN MRI scores of the spine were evaluated.

Results. 258 patients with r-axSpA and 237 with nr-axSpA were enrolled. Baseline SPARCC/CANDEN scores were similar across treatment groups (Table I). Across studies, reductions in spinal inflammatory MRI lesions were observed with filgotinib versus placebo at W16, with improvements maintained or further improved at W52 (Fig. 1). In both studies, at W16, increases in CANDEN spine fat scores were observed with filgotinib versus placebo (mean CFB [95% CI]: r-axSpA, 1.7 [1.0, 2.4] vs. 0.0 [–0.4, 0.3]; nr-axSpA, 0.9 [0.4, 1.4] vs. –0.1 [–0.2, 0.1]). Further increases in fat score were minimal in patients who continued to receive filgotinib through W52, while increases were observed in patients who switched from placebo to filgotinib (mean CFB [95% CI] at W52: r-axSpA, 1.5 [0.7, 2.2]; nr-axSpA, 0.9 [0.4, 1.4]). No increases were observed in spinal bone erosion or new bone formation up to W52.

Conclusion. In patients with axSpA, filgotinib was associated with reductions in spinal inflammation and post-inflammatory tissue changes, indicating a consistent anti-inflammatory profile of filgotinib, with no or minimal structural progression over 52 weeks.

Study funding. Alfasigma S.p.A. (Bologna, Italy)

Table I. Baseline disease characteristics (MRI findings).

Characteristic	r-axSpA			nr-axSpA		
	FIL 200 mg (n=129)	PBO (n=129)	All patients (N=258)	FIL 200 mg (n=119)	PBO (n=118)	All patients (N=237)
SPARCC score: spine inflammation (range: 0–108)						
Mean (SD)	12.6 (17.9)	9.4 (15.0)	11.0 (16.6)	3.7 (7.4)	3.8 (10.7)	3.8 (9.2)
Median (min, max)	4.5 (0.0, 115.5)	3.0 (0.0, 78.0)	4.0 (0.0, 115.5)	0.0 (0.0, 34.0)	0.0 (0.0, 102.5)	0.0 (0.0, 102.5)
CANDEN score: spine inflammation (range: 0–614)						
Mean (SD)	13.9 (20.7)	9.9 (15.7)	11.9 (18.4)	5.7 (10.2)	5.0 (12.4)	5.4 (11.4)
Median (min, max)	6.0 (0.0, 130.5)	4.0 (0.0, 106.0)	5.0 (0.0, 130.5)	1.0 (0.0, 47.5)	1.8 (0.0, 123.0)	1.5 (0.0, 123.0)
CANDEN score: spine fat (range: 0–480)						
Mean (SD)	17.6 (28.2)	11.1 (19.1)	14.4 (24.3)	6.2 (14.1)	4.7 (8.7)	5.5 (11.7)
Median (min, max)	6.5 (0.0, 157.5)	3.5 (0.0, 143.0)	5.5 (0.0, 157.5)	1.5 (0.0, 118.5)	1.0 (0.0, 55.0)	1.5 (0.0, 118.5)
CANDEN score: spine bone erosion (range: 0–208)						
Mean (SD)	0.2 (0.5)	0.1 (0.5)	0.1 (0.5)	0.1 (0.3)	0.1 (0.3)	0.1 (0.3)
Median (min, max)	0.0 (0.0, 5.0)	0.0 (0.0, 3.0)	0.0 (0.0, 5.0)	0.0 (0.0, 2.0)	0.0 (0.0, 2.0)	0.0 (0.0, 2.0)
CANDEN score: spine new bone formation (range: 0–460)						
Mean (SD)	16.6 (28.0)	18.7 (36.9)	17.7 (32.7)	5.2 (16.4)	2.5 (4.9)	3.8 (12.2)
Median (min, max)	4.0 (0.0, 158.0)	2.5 (0.0, 218.5)	3.0 (0.0, 218.5)	0.0 (0.0, 131.5)	0.0 (0.0, 28.0)	0.0 (0.0, 131.5)

CANDEN: Canada-Denmark; FIL: filgotinib; MRI: magnetic resonance imaging; nr-axSpA: nonradiographic axial spondyloarthritis; PBO: placebo; r-axSpA: radiographic axial spondyloarthritis; SD: standard deviation; SPARCC: Spondyloarthritis Research Consortium of Canada.

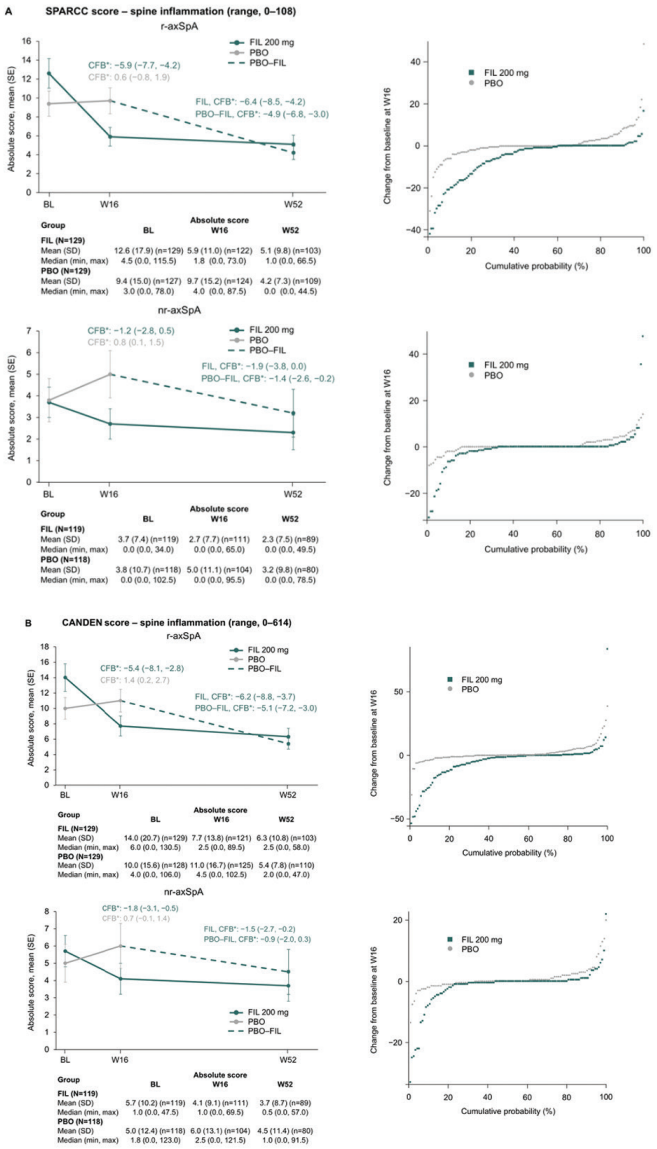


Fig. 1. MRI scores of the spine in patients with r-axSpA and nr-axSpA: SPARCC spine inflammation (**A**) and CANDEN spine inflammation (**B**).
Imputation was not applied; data reflect only those patients who remained on treatment and hence maybe subject to selection bias. Due to the large number of low or zero values at baseline and in changes from baseline at W16, empirical cumulative distribution functions were used to assess changes from baseline. *Data presented for CFB are mean (95% CI).
BL: baseline; CANDEN; Canada-Denmark; CFB: change from baseline; CI: confidence interval; FIL: filgotinib; MRI: magnetic resonance imaging; nr-axSpA: nonradiographic axial spondyloarthritis; PBO: placebo; r-axSpA, radiographic axial spondyloarthritis; SD: standard deviation; SE: standard error; SPARCC: Spondyloarthritis Research Consortium of Canada; W: Week.

P120

IMPACT OF FILGOTINIB ON MRI INFLAMMATORY AND STRUCTURAL LESIONS IN THE SACROILIAC JOINT OF PATIENTS WITH AXIAL SPONDYLOARTHRITIS: 52-WEEK RESULTS FROM THE PHASE 3 OLINGUITO TRIAL

Baraliakos X.[1], Navarro-Compán V.[2], Machado P.M.[3,4,5], Gheyle L.[6], Mangili A.[7], Le Brun F.-O.[7], Muller K.[6], de Hooge M.[8], Lambert R.G.W.[9], Landewé R.[10,11], Østergaard M.[12,13], Maksymowych W.P.[14]

[1]Rheumazentrum Ruhrgebiet Herne, Ruhr University Bochum, Bochum, Germany; [2]La Paz University Hospital, Hospital La Paz Institute for Health Research (IdiPAZ), Madrid, Spain; [3]Dept. of Neuromuscular Diseases, University College London, London; [4]NIHR University College London Hospitals Biomedical Research Centre, University College London Hospitals NHS Foundation Trust, London; [5]Dept. of Rheumatology, Northwick Park Hospital, London North West University Healthcare NHS Trust, London, UK; [6]Alfasigma Belgium BV, Mechelen, Belgium; [7]Alfasigma Schweiz AG, Zofingen, Switzerland; [8]Dept. of Rheumatology, Ghent University Hospital, Ghent, Belgium; [9]Dept. of Radiology and Diagnostic Imaging, University of Alberta, Edmonton, AB, Canada; [10]Dept. of Clinical Immunology and Rheumatology, Amsterdam University Medical Center, Amsterdam; [11]Dept. of Rheumatology, Zuyderland Medical Center, Heerlen, The Netherlands; [12]Dept. of Clinical Medicine, University of Copenhagen, Copenhagen; [13]Copenhagen Center for Arthritis Research (COPECARE), Center for Rheumatology and Spine Diseases, Rigshospitalet, Glostrup, Denmark; [14]Dept. of Medicine, University of Alberta, Edmonton, AB, Canada

Introduction. The Phase 3 OLINGUITO trial (NCT05785611) evaluated the efficacy and safety of filgotinib in patients with axial spondyloarthritis (axSpA). We report the effect of filgotinib on sacroiliac joint (SIJ) inflammation and structural damage progression.

Methods. OLINGUITO comprises two, randomized, double-blind, placebo-controlled studies in patients with radiographic or non-radiographic axSpA, with inadequate response or intolerance to conventional therapy. Patients were randomized (1:1) to receive once daily filgotinib (200 mg) or placebo through Week (W) 16. After W16, all patients entered an open-label period and received filgotinib through W52, with adjustment to the lowest effective dose in patients aged ≥65 years and/or with predefined risk factors. MRIs were completed at baseline, W16 and W52, and evaluated using the SPARCC score. Change from baseline (CFB) at W16 (secondary endpoint) and W52 (exploratory endpoint) were evaluated.

Results. 258 patients with r-axSpA and 237 with nr-axSpA were enrolled. Baseline SIJ inflammation and structural scores were similar across treatment groups (Table I). In both studies, SIJ inflammation reduced significantly with filgotinib at W16 (least squares mean CFB [95% CI]: r-axSpA, –2.69 [–3.60, –1.77] vs. –0.62 [–1.53, 0.28], $p<0.001$; nr-axSpA, –1.88 [–3.00, –0.77] vs. 1.39 [0.26, 2.51], $p<0.001$), with reductions maintained at W52 (Fig. 1A). Reductions in bone erosion mean scores were comparable between treatment groups; however, cumulative distribution analyses indicated a shift towards greater improvement with filgotinib, and further reductions were observed with filgotinib at W52 (Fig. 1B). SIJ fat lesion and backfill scores increased with filgotinib at W16 and were maintained at W52. Minimal or no changes were observed in ankylosis with filgotinib at W52.

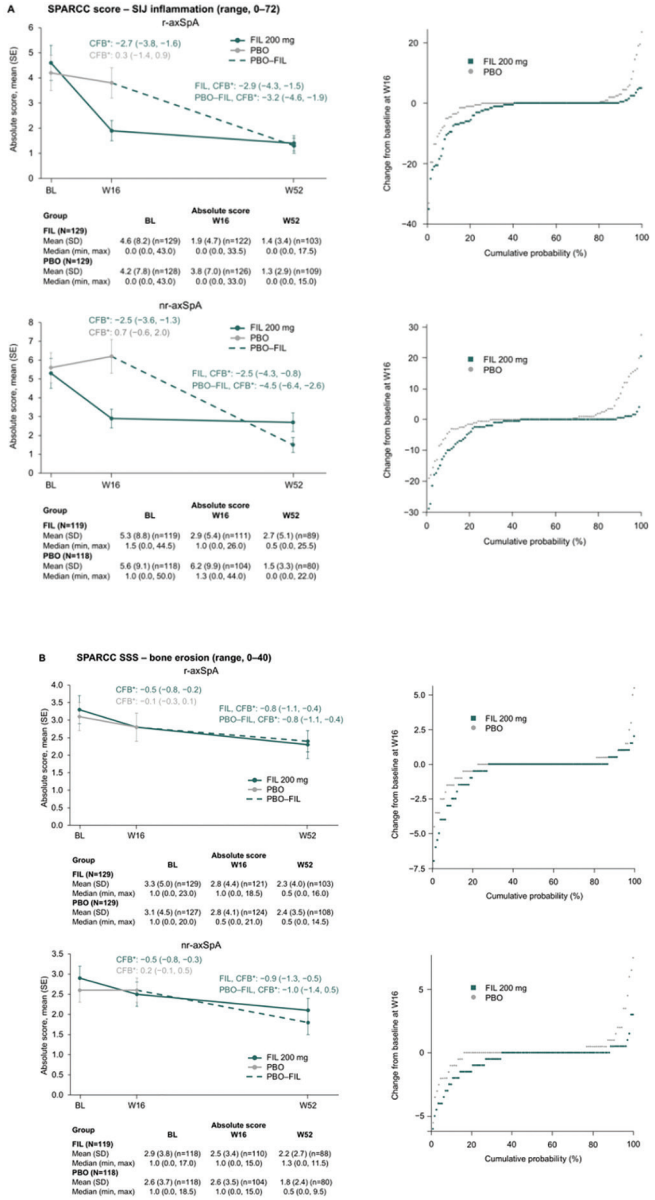


Fig. 1. MRI scores of the SIJ in patients with r-axSpA and nr-axSpA: Inflammation (**A**) and bone erosion (**B**).
Imputation was not applied; data reflect only those patients who remained on treatment and hence may be subject to selection bias. Due to the large number of low or zero values at baseline and in changes from baseline at W16, empirical cumulative distribution functions were used to assess changes from baseline.
*Data presented for CFB are mean (95% CI) (exploratory analyses).
BL: baseline; CFB: change from baseline; CI: confidence interval; FIL: filgotinib; MRI: magnetic resonance imaging; nr-axSpA: nonradiographic axial spondyloarthritis; PBO: placebo; r-axSpA: radiographic axial spondyloarthritis; SD: standard deviation; SE: standard error; SIJ: sacroiliac joint; SPARCC: Spondyloarthritis Research Consortium of Canada; SSS: SIJ Structural Score; W: Week.

Table I. Baseline disease characteristics (MRI findings).

Characteristic	r-axSpA			nr-axSpA		
	FIL 200 mg (n=129)	PBO (n=129)	All patients (N=258)	FIL 200 mg (n=119)	PBO (n=118)	All patients (N=237)
SPARCC score: SIJ inflammation (range, 0–72)						
Mean (SD)	4.6 (8.2)	4.2 (7.8)	4.4 (8.0)	5.3 (8.8)	5.6 (9.1)	5.5 (9.0)
Median (min, max)	0.0 (0.0, 43.0)	0.0 (0.0, 43.0)	0.0 (0.0, 43.0)	1.5 (0.0, 44.5)	1.0 (0.0, 50.0)	1.5 (0.0, 50.0)
SPARCC SSS: bone erosion (range, 0–40)						
Mean (SD)	3.3 (5.0)	3.1 (4.5)	3.2 (4.8)	2.9 (3.8)	2.6 (3.7)	2.8 (3.7)
Median (min, max)	1.0 (0.0, 23.0)	1.0 (0.0, 20.0)	1.0 (0.0, 23.0)	1.0 (0.0, 17.0)	1.0 (0.0, 18.5)	1.0 (0.0, 18.5)
SPARCC SSS: fat lesion (range, 0–40)						
Mean (SD)	5.6 (7.6)	4.6 (6.0)	5.1 (6.9)	2.0 (4.9)	1.3 (3.6)	1.6 (4.3)
Median (min, max)	1.5 (0.0, 35.5)	1.5 (0.0, 26.5)	1.5 (0.0, 35.5)	0.0 (0.0, 38.0)	0.0 (0.0, 22.5)	0.0 (0.0, 38.0)
SPARCC SSS: backfill (range, 0–20)						
Mean (SD)	1.6 (2.5)	1.2 (2.1)	1.4 (2.3)	0.8 (1.9)	0.7 (1.8)	0.7 (1.9)
Median (min, max)	0.0 (0.0, 12.0)	0.0 (0.0, 11.0)	0.0 (0.0, 12.0)	0.0 (0.0, 12.5)	0.0 (0.0, 13.0)	0.0 (0.0, 13.0)
SPARCC SSS: ankylosis (range, 0–20)						
Mean (SD)	7.6 (8.2)	7.3 (8.4)	7.5 (8.3)	0.6 (3.0)	0.2 (1.5)	0.4 (2.4)
Median (min, max)	4.0 (0.0, 20.0)	0.5 (0.0, 20.0)	3.0 (0.0, 20.0)	0.0 (0.0, 19.5)	0.0 (0.0, 15.0)	0.0 (0.0, 19.5)

FIL: filgotinib; MRI: magnetic resonance imaging; nr-axSpA: nonradiographic axial spondyloarthritis; PBO: placebo; r-axSpA: radiographic axial spondyloarthritis; SD: standard deviation; SIJ: sacroiliac joint; SPARCC: Spondyloarthritis Research Consortium of Canada; SSS: SIJ Structural Score.

Conclusion. In patients with axSpA, filgotinib reduced SIJ inflammation at W16, and these results were maintained until W52. In parallel, erosions were reduced and fat and backfill were increased. Progression of ankylosis over 52 weeks was not observed.
Study funding. Alfasigma S.p.A. (Bologna, Italy)

P121

MULTIDRUG EXPOSURE MAY TRIGGER IATROGENIC TREATMENT FAILURE IN SPONDYLOARTHRITIS: INSIGHTS FROM HISTORIC REAL-WORLD DATA OF THE LEEDS SPECIALIST SPONDYLOARTHRITIS SERVICE

Fakih O.[1,2], Weddell J.[1], Meridor K.[1], Barr A.[1], Freeston J.E.[1], Vandevelde C.[1], Marzo-Ortega H.[1]
[1]NIHR Leeds Biomedical Research Centre, The Leeds Teaching Hospitals NHS Trust and Leeds Institute of Rheumatic and Musculoskeletal Medicine, University of Leeds, Leeds, UK; [2]Université Marie et Louis Pasteur, Service de Rhumatologie, CHU de Besançon, France

Introduction. Treatment retention is a common proxy for b/tsDMARD effectiveness in axial spondyloarthritis (axSpA) and psoriatic arthritis (PsA), but the reasons for discontinuation and the subsequent within-class cycling and/or mode of action switching strategy are rarely analysed across successive lines.
Objective. To evaluate first-line b/tsDMARD retention in axSpA and PsA, identify factors associated with the absence of first-line discontinuation, and compare characteristics associated with cyclers and switchers.
Methods. Retrospective analysis of historic (2000-2020), routinely collected real-world data from a tertiary centre including treatment history with reasons for change. After a descriptive analysis, drug survival was assessed from treatment initiation to first treatment change using Cox models.

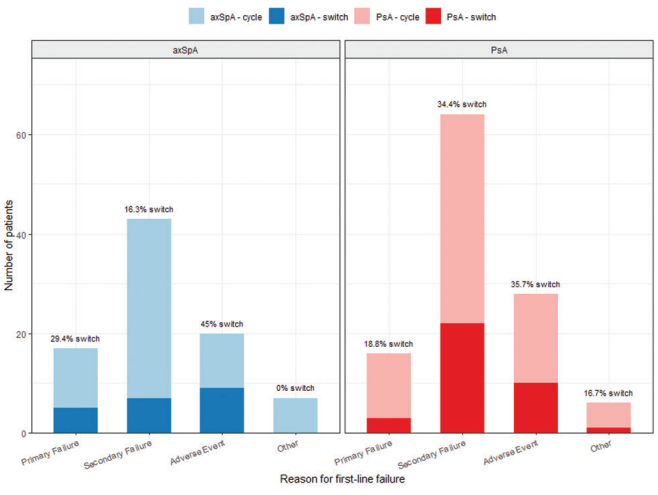


Fig. 1. Reasons for first-line bDMARD failure and proportion of treatment switching between different modes of action according to the disease phenotype.

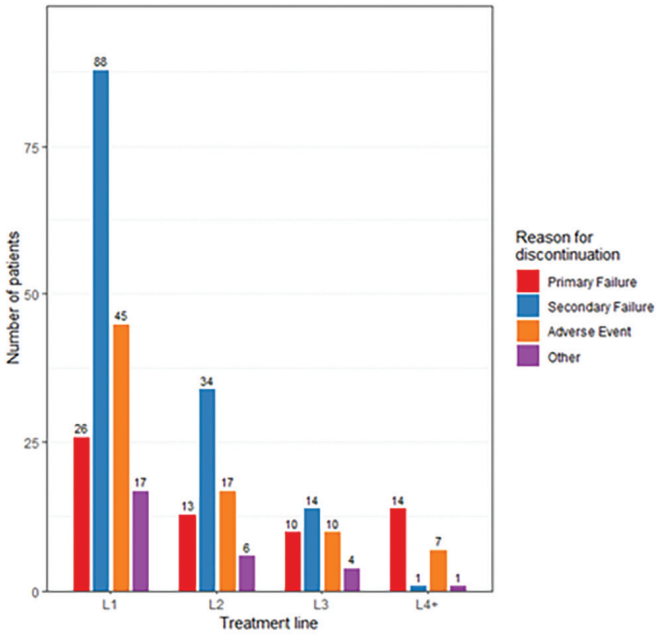


Fig. 2. Reasons for b/tsDMARD discontinuation by treatment line.

Results. A total of 396 patients were included (211 PsA, 185 axSpA, 42.5% female) with a mean follow-up duration of 7.2±5.7 years. Mean age at first-line initiation was 46.4 years with 86.4% initiating a TNF inhibitor. First-line retention was observed in 165 patients (41.7%), while 164 (41.4%) underwent cycling and 67 (16.9%) underwent switching. Bivariate analysis showed lower retention in PsA (HR: 1.47 [1.08 – 1.99], p=0.01) and females (HR: 1.67 [1.23 – 2.25], p<0.001). The strongest predictor was calendar year of first-line initiation (HR 1.15 per year, 1.11–1.19), indicating shorter retention in more recent years. Concordantly, among patients initiating L1 in 2000–2004 no primary failures were recorded, rising to 13%, 19% and 24% across successive 5-year periods (p=0.01), while secondary failure declined from 63% to 40%. In both axSpA and PsA, secondary non-response was the leading reason for first-line treatment discontinuation (56% and 62% respectively) (Fig. 1). However, the proportion of primary non-response increased with each subsequent line of treatment (Fig. 2).
Conclusion. These patterns suggest a contemporary lower tolerance to imperfect response and the possible iatrogenic generation of successive failures, underscoring the need to explore reasons for treatment failure, not retention alone.

P122

DIFFICULT-TO-MANAGE AXIAL SPONDYLOARTHRITIS: CLINICAL PROFILE, ASSOCIATED FACTORS AND TREATMENT TRAJECTORIES FROM THE ESPALDA REGISTRY

Sommerfleck F.[3], Garcia Salinas R.[1], Casasola Vargas J.C.[4], Guaracha Basanez G.A.[5], Londono J.[6], Gamboa R.[7], Fernandez Avila D.[8], Vila D.[9], Mestanza Peralta M.[10], Then Baez J.[11], Dufau E.[12], Bermudez Bermejo P.[13], Macias Oviedo L.[15], Duran Barragan S.[14], Valdez F.[16], Barcia Cansino M.A.[17], Medina G.[18], Casallas L.[19], Castillo Ortiz A.A.[20], Chinchay L.[21], Caballero L.[22], Benegas M.[3], Medina D.[23], Vega C.[24], Melgarejo P.[22], Gomez A.B.[25], Ferreyra Garrot L.G.[26], Candia D.L.[27], Gil Calderon D.R.[28], Rivera V.[29], Eguez Del Pozo M.F.[30], Sanchez Prado E.[31], Avellanal V.[25], Marin Zucaro N.[32], Brance L.[32], Bautista-Molano W.[2] on behalf of ESPALDA-PANLAR
[1]Hospital Italiano de La Plata, La Plata, Argentina; [2]Fundacion Santa Fe, Bogota, Colombia; [3]Sanatorio Mendez, Buenos Aires, Argentina; [4]Hospital General de Mexico, Mexico City; [5]Instituto Nacional de Ciencias Medicas y Nutricion Salvador Zubiran, Mexico City, Mexico; [6]Hospital Militar Central, Universidad de La Sabana, Bogota, Colombia; [7]Hospital Nacional Guillermo Almenara Irigoyen, Lima, Peru; [8]Hospital San Ignacio, Bogota, Colombia; [9]Hospital Virgen del Carmen, Campana, Argentina; [10]Hospital Metropolitano, Quito, Ecuador; [11]Hospital Metropolitano de Santiago, Santiago de los Caballeros, Dominican Republic; [12]Sanatorio Guemes, Buenos Aires, Argentina; [13]Centro Medico ABC, Mexico City; [14]Clinica de Investigacion en Reumatologia y Obesidad S.C., Guadalajara, Mexico; [15]Consultorio reumaart.ec, Guayaquil, Ecuador; [16]IREP, Buenos Aires, Argentina; [17]Hospital General Portoviejo del Instituto Ecuatoriano de Seguridad Social, Portoviejo, Ecuador; [18]Hospital de Clinicas Jose de San Martin, Buenos Aires; [19]Genea Centro Medico, Buenos Aires, Argentina; [20]Hospital Faro del Mayab, Merida, Mexico; [21]Hospital Guillermo Kaelin de La Fuente, Piura, Peru; [22]Instituto de Prevision Social, Asuncion, Paraguay; [23]Red de Investigacion Colaborativa del Caribe Colombiano, Barranquilla, Colombia; [24]Hospital General ISSSTE Tacuba, Mexico City, Mexico; [25]Instituto Nacional de Reumatologia del Uruguay, Montevideo, Uruguay; [26]Hospital Italiano de Buenos Aires, Buenos Aires, Argentina; [27]Hospital Regional Primero de Octubre ISSSTE, Mexico City, Mexico; [28]Artmedica SAS, Bogota, Colombia; [29]Instituto Nacional de Rehabilitacion, Mexico City, Mexico; [30]Altius Medical, Guayaquil, Ecuador; [31]Hospital del IESS los Ceibos, Guayaquil, Ecuador; [32]UI-PANLAR, Rosario, Argentina

Introduction/Objective. Difficult-to-manage (D2M) axSpA is an emerging challenge, but data on its prevalence, associated phenotype and treatment trajectories in Latin America are scarce. We aimed to estimate D2M prevalence, identify associated factors and describe real-world treatment sequences by mechanism of action in ESPALDA.
Materials and methods. ESPALDA is a multicentre Latin American axSpA registry. Baseline demographic, clinical, laboratory, imaging and treatment data were analysed. D2M axSpA was defined according to the ASAS definition. Patients with and without D2M were compared using univariate analyses, and multivariable logistic regression was used to identify independent associations. Treatment trajectories were described across therapeutic lines.
Results. Among 741 patients, 83 fulfilled D2M criteria, yielding a prevalence of 11.2%. Compared with non-D2M patients, D2M patients had higher BASDAI (4.74 vs. 4.19, p=0.013), BASFI (4.63 vs. 4.22, p=0.036) and MASES (3.12 vs. 2.28, p=0.007). Peripheral arthritis (43.4% vs. 19.5%, p<0.001), enthesitis (49.4% vs. 27.2%, p<0.001) and psoriasis (20.5% vs. 10.9%, p=0.02) were more frequent. Sex, HLA-B27 status, radiographic axSpA and MRI inflammatory or structural lesions did not differ significantly. In multivariable analysis, peripheral arthritis (OR 2.87; 95% CI 1.69-4.87) and enthesitis (OR 2.45; 95% CI 1.46-4.12) were independently associated with D2M (Table I). D2M patients had more treatment lines, with frequent switching from TNF inhibitors to IL-17 inhibitors and later JAK inhibitors.
Conclusion. D2M axSpA affected approximately one in ten Latin American patients and was mainly associated with peripheral MSK involvement rather than axial imaging severity. Early recognition of this phenotype may support more comprehensive management beyond axial inflammation.

Table I. Key differences between D2M and non-D2M axSpA.

Variable	D2M	Non-D2M	p value
Prevalence	83/741 (11.2%)	-	-
Peripheral arthritis	43.4%	19.5%	<0.001
Enthesitis	49.4%	27.2%	<0.001
Any biologic therapy	75.9%	42.4%	<0.001
>=2 biologic lines	47.0%	14.3%	<0.001

P123

EFFECTIVENESS OF ADALIMUMAB AND SECUKINUMAB WITH AND WITHOUT CONCOMITANT CSDMARD USE IN PATIENTS WITH PSORIATIC ARTHRITIS – A RETROSPECTIVE COHORT STUDY

Gerritsen S.M.[1], Mahler E.A.M.[2], Vriezekolk J.E.[1], van Herwaarden N.[2], den Broeder A.A.[2,3], van der Horst-Bruinsma I.E.[3], den Broeder N.[1], Leijten E.F.A.[2,3]
[1]Dept. of Research, Sint Maartenskliniek, Nijmegen; [2]Dept. of Rheumatology, Sint Maartenskliniek, Nijmegen; [3]Dept. of Rheumatology, Radboudumc, Nijmegen, The Netherlands

Introduction. Conventional synthetic disease-modifying antirheumatic drugs (csDMARDs), including methotrexate and leflunomide, are first-line therapy in psoriatic arthritis (PsA). Biological disease-modifying antirheumatic drugs (bDMARDs), including tumour necrosis factor inhibitors (TNFi, like adalimumab) and interleukin-17 inhibitors (IL-17i, like secukinumab), are recommended after inadequate response. Whether csDMARDs should be continued when initiating bDMARDs remains debated. Observational data suggest improved retention of TNFi with csDMARDs, whereas

evidence for IL-17i is inconsistent. We investigated the effectiveness of adalimumab and secukinumab with and without concomitant csDMARD use in terms of bDMARD drug retention in PsA patients.

Methods. PsA patients starting first-line adalimumab and/or second-line secukinumab between November 2019-March 2025 were included and categorized as bDMARD monotherapy or combination therapy (bDMARD with concomitant csDMARD). Patients ineligible for combination therapy (csDMARD contraindications or side effects) were excluded. bDMARD treatment discontinuations because of side effects or ineffectiveness were considered events. Individual patients could contribute data to both adalimumab and secukinumab analyses. Primary outcome was bDMARD drug retention, analyzed using Kaplan-Meier curves and multivariable Cox regression adjusted for age and sex.

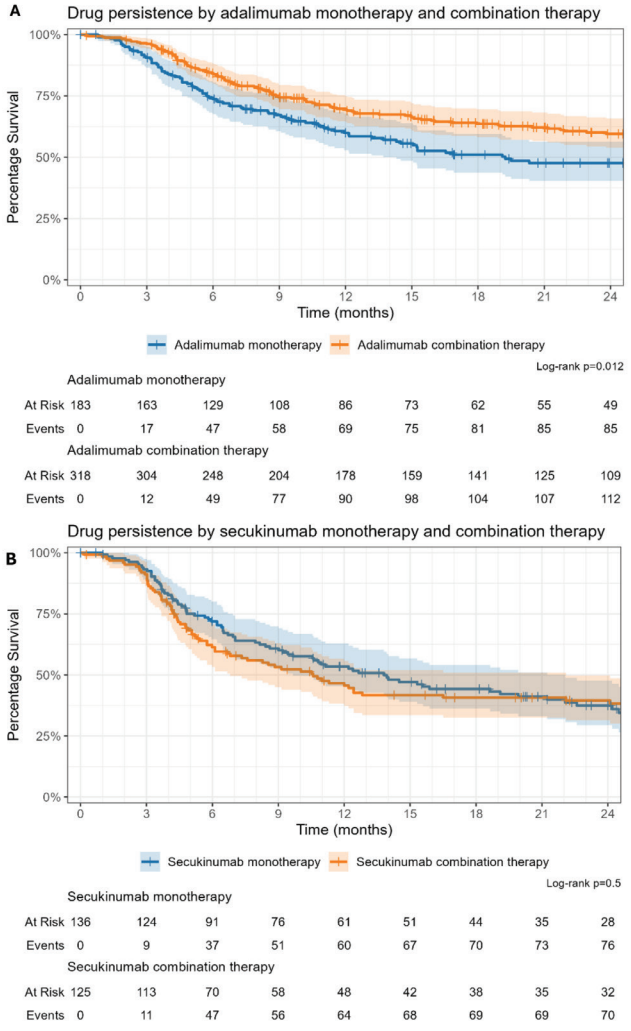


Fig. 1. Drug retention in patients treated with (**A**) adalimumab monotherapy versus combination therapy and (**B**) secukinumab monotherapy versus combination therapy. Kaplan-Meier curves illustrate drug retention for each treatment strategy, with shaded areas representing 95% confidence intervals. Numbers at risk and cumulative events are shown below the plots.

Results. 635 PsA patients were included (Table I). Adalimumab combination therapy showed significantly lower discontinuation risk in unadjusted (median retention 11.0 vs. 15.0 months, HR 0.71, p=0.012) and adjusted analyses (adjusted HR 0.67, p=0.003) compared to adalimumab monotherapy (Fig. 1A). In secukinumab, no significant difference was observed for monotherapy versus combination therapy (median retention 10.6 vs. 7.3 months, HR 1.11, p=0.50 and adjusted HR 1.14, p=0.42) (Fig. 1B). The effect of csDMARD use was significantly greater in adalimumab compared to secukinumab (interaction: p=0.028). Per-protocol analyses also demonstrated prolonged retention for adalimumab combination therapy (adjusted HR 0.62, p=0.002) but not for secukinumab (adjusted HR 1.22, p=0.25).

Conclusion. Concomitant csDMARDs prolonged drug retention for adalimumab but not for secukinumab. csDMARDs should be continued with adalimumab, but consider discontinuation when starting secukinumab.

Table I. Baseline characteristics.

Variable	Adalimumab monotherapy n=183	Adalimumab combination therapy n=318	Secukinumab monotherapy n=136	Secukinumab combination therapy n=125
Age (years), median (Q1, Q3)	48 (34, 58)	50 (39, 59)	52 (37, 60)	54 (47, 62)
Sex (female), N (%)	94 (51%)	166 (52%)	78 (57%)	74 (59%)
Disease duration (years), median (Q1, Q3)	1.0 (0.0, 3.0)	1.0 (0.0, 4.0)	3.0 (1.0, 9.0)	5.0 (2.0, 11.0)
Anti-citrullinated protein, n (%)				
Positive	1 (0.8%)	3 (1.4%)	0 (0%)	1 (1.1%)
Rheumatoid factor, n (%)				
Positive	5 (4.3%)	16 (7.7%)	3 (3.2%)	6 (7.1%)
DAS28-CRP, median (Q1, Q3)[1]	3.2 (2.3, 3.9)[a]	3.0 (2.4, 3.7)[b]	3.1 (2.3, 3.8)[c]	3.3 (2.4, 4.3)[d]
Concomitant csDMARD, N (%)				
Leflunomide		66 (21%)		37 (30%)
Leflunomide + methotrexate		33 (10%)		2 (1.6%)
Leflunomide + sulfasalazine		0 (0%)		1 (0.8%)
Methotrexate		212 (67%)		81 (65%)
Methotrexate + sulfasalazine		7 (2.2%)		4 (3.2%)
Number of previously used b/tsDMARDs, n (%)				
0				
1	177 (97%)	312 (98%)	12 (8.8%)	5 (4.0%)
2 or more	6 (3.3%)	6 (1.9%)	114 (84%)	114 (91%)
	0 (0%)	0 (0%)	10 (7.3%)	6 (4.8%)
Number of previously and/or currently used csDMARDs, n (%)				
0	20 (11%)	2 (0.6%)	13 (96%)	0 (0%)
1	93 (51%)	166 (52%)	58 (43%)	43 (34%)
2	64 (35%)	135 (42%)	50 (37%)	60 (48%)
3 or more	6 (3.3%)	15 (4.7%)	15 (11%)	22 (18%)

[1]Number of available data: a = 65, b = 116, c = 45 and d = 50.

P124

CLINICAL OUTCOMES FOLLOWING WITHDRAWAL OF UPADACITINIB IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS WHO ACHIEVED REMISSION: FINDINGS FROM THE PHASE 3, PLACEBO-CONTROLLED, DOUBLE-BLIND SELECT-AXIS 2 TRIAL

Deodhar A.[1], Kishimoto M.[2], Poddubnyy D.[3], Maksymowych W.P.[4], Lagunes-Galindo I.[5], Kato K.[5], Parikh B.[5], Carter K.[5], Philips C.[5], Baraliakos X.[6]

[1]Division of Arthritis and Rheumatic Diseases, Oregon Health & Science University, Portland, OR, USA; [2]Dept. of Nephrology and Rheumatology, Kyorin University School of Medicine, Tokyo, Japan; [3]Division of Rheumatology, Dept. of Medicine, University of Toronto, Toronto; [4]Dept. of Medicine, University of Alberta, Edmonton, Alberta, Canada; [5]AbbVie Inc., North Chicago, IL, USA; [6]Rheumazentrum Ruhrgebiet Herne, Ruhr-University Bochum, Germany

Introduction/Objective. To assess maintenance of disease control following withdrawal and subsequent retreatment with upadacitinib in patients with axSpA who experience a disease flare after achieving clinical remission.

Materials and methods. SELECT-AXIS 2 (NCT04169373) enrolled patients with r-axSpA who had an inadequate response/were intolerant to biologic DMARDs (bDMARD-IR) and patients with nr-axSpA. Patients received upadacitinib 15mg or placebo. At week 104, those in clinical remission (ASDAS <2.1 at week 88 and ASDAS <1.3 at week 104) entered an open label treatment withdrawal period. In the event of a disease flare (ASDAS (CRP) ≥2.1 at two visits at least two weeks apart, or ASDAS >3.5 at one visit), patients re-initiated upadacitinib 15 mg daily for 24 weeks.

Results. Of 734 patients in SELECT-AXIS 2, 194 patients (26.4%) were in remission at week 104 and included in this analysis (r-axSpA bDMARD-IR, n=114; nr-axSpA, n=80). Following 48 weeks of upadacitinib withdrawal, 22.2% maintained pre-defined clinical remission; median time to flare was 3.1 months (r-axSpA bDMARD-IR: 2.8 months, nr-axSpA: 5.0 months). (Fig. 1A). Upon re-initiation of upadacitinib therapy, 72.2% (96/133) achieved ASDAS LDA (<2.1) by week 4 and 88.9% (104/117) by week 24 (Fig. 1B). Additionally, ASDAS ID (<1.3) was achieved by 35.3% (47/133) by week 4 and by 48.7% (57/117) by week 24 after re-treatment (Fig. 1C). Consistent with previous reports, upadacitinib was generally well-tolerated during re-treatment and in the long-term, with no new safety concerns or significant risks identified (Fig. 2).

Conclusion. Continuous treatment with upadacitinib is necessary for maintaining disease control in axSpA. However, only about 1/4 of patients may achieve long-term drug-free remission. Most patients who experience a disease flare after discontinuation regain at least ASDAS LDA within 24 weeks of reinitiation. These findings are crucial in guiding treatment decisions when therapy interruptions are necessary.

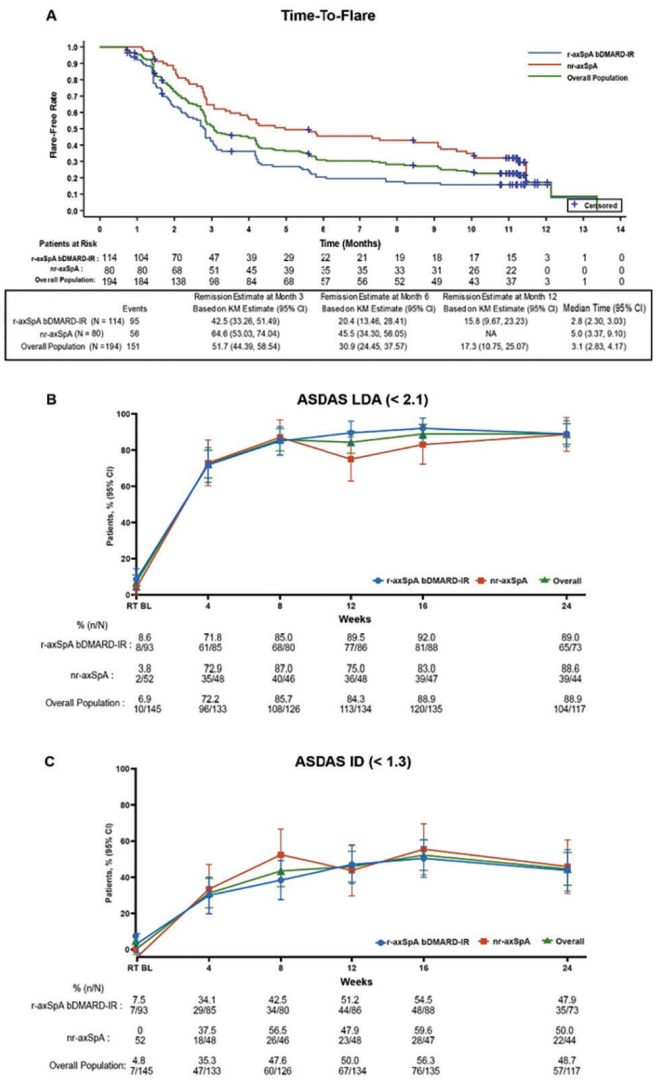


Fig. 1. Outcomes assessed during withdrawal and retreatment period with Upadacitinib in the SELECT-AXIS 2 study.
BL: baseline, CI: confidence interval; ID: inactive disease, KM: Kaplan-Meier method; LDA: low disease activity; RT: retreatment. All data are as observed.

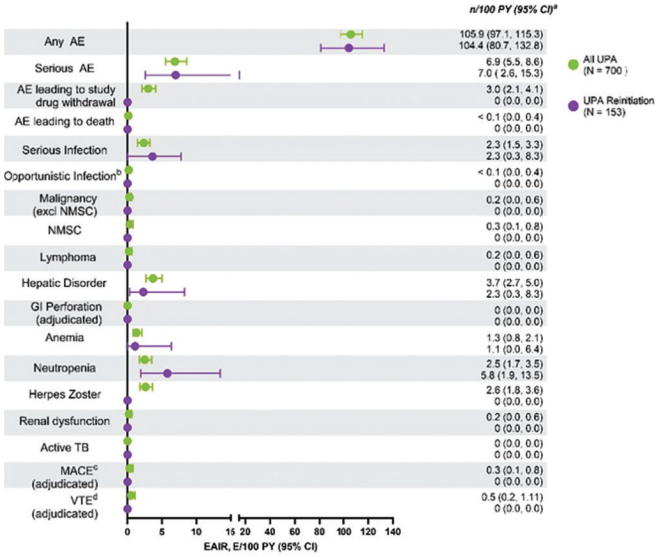


Fig. 2. Exposure-adjusted incidence of treatment-emergent events during Upadacitinib re-initiation in the SELECT-AXIS 2 study. AE: adverse events; MACE: major adverse cardiovascular events; NMSC: non-melanoma skin cancer, UPA: upadacitinib; VTE: Venous thromboembolic events.
[a] Data are presented as EAIRs (defined as n/100 PY][(95% CI])s, patients were censored at the time of the first event.
[b] Opportunistic infection excluding tubercolosis and herpes zoster.
[c] Mace is defined as cardiovascular death, non-fatal myocardial infarction and non-fatal stroke.
[d] VTE includes pulmonary embolism and venous thrombosis.
Note: A treatment emergent adverse event is defined as an AE with an onset after the first dose of study drug, and no more than 30 days after the last dose of study drug. Events that occur during treatment withdrawal and are more than 30 days past the last dose of upadacitinib will be excluded from all UPA. All UPA includes all TEAEs occurring during the study.

P125

IMPACT OF TREATMENT WITH UPADACITINIB ON NON-NOCICEPTIVE PAIN AND ITS RELEVANCE FOR THE PRESENCE OF RESIDUAL SYMPTOMS IN AXIAL SPONDYLOARTHRITIS: RESULTS FROM A MULTI-COUNTRY OBSERVATIONAL STUDY

Poddubnyy D.[1,2], Navarro-Compán V.[3], Basu N.[4], Naffaa M.E.[5,6], Gao T.[7], Urbanik J.R.[7], Saffore C.D.[7], Parikh B.K.[7], Taylor P.C.[8], Mease P.J.[9]
[1]Division of Rheumatology, University of Toronto and University Health Network, Toronto, Canada; [2]Dept. of Gastroenterology, Infectious Diseases and Rheumatology, Charité - Universitätsmedizin Berlin, Berlin, Germany; [3]Dept. of Rheumatology, Hospital Universitario La Paz, IdiPAZ, Madrid, Spain; [4]Institute of Infection, Immunity & Inflammation, University of Glasgow, Glasgow, UK; [5]The Rheumatology Unit, Galilee Medical Center, Naharyia; [6]The Azrieli's Faculty of Medicine, Bar-Ilan University, Safed, Israel; [7]AbbVie Inc., North Chicago, USA; [8]Botnar Research Centre, Nuffield Dept. of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Oxford, UK; [9]University of Washington School of Medicine, Seattle, USA

Introduction/Objective. This post hoc analysis examined the real-world impact of upadacitinib (UPA) treatment on non-nociceptive pain and the association of non-nociceptive pain with residual symptoms, sleep disturbance, anxiety, and depression in patients with axSpA.
Materials and methods. UPSTAND was a 12-month, multicountry, non-interventional, observational study evaluating the effectiveness of UPA on pain in adult patients with axSpA. This analysis used interim data collected after all patients completed at least week 12. Patients were grouped by severity of neuropathic pain, as captured by painDETECT (<13, 13-18, and >18) or severity of nociplastic pain, as captured by widespread pain index (WPI: <4, 4-6, and >6). While patients demonstrating active symptoms of fibromyalgia (FM) per clinical diagnosis were excluded from UPSTAND, WPI and symptom severity score (SSS) were used together to assess the presence of nociplastic pain fulfilling FM criteria (as per Wolfe et al. *Semin Arthritis Rheum*. 2016; 46: 319-29). WPI 4-6 and SSS ≥9 or WPI ≥7 and SSS ≥5.[1] All other scores (absence of FM) were grouped together.

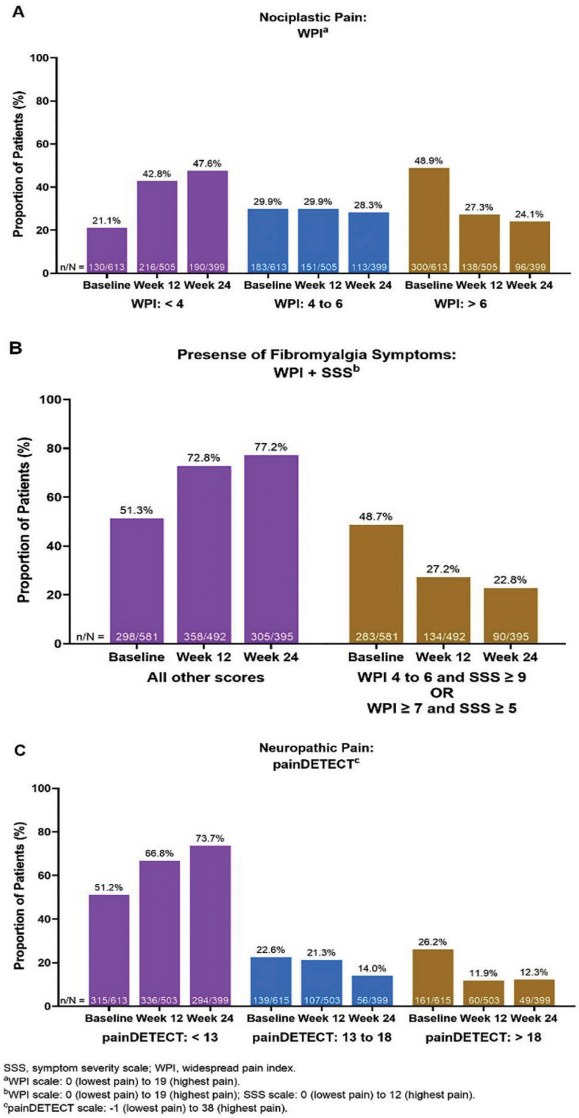


Fig. 1. Proportion of patients in each non-nociceptive pain severity group at baseline, Week 12, and Week 24.

Results. Overall, UPA treatment was associated with a reduction in the burden of nociplastic and neuropathic pain over time (Fig. 1). The proportion of patients in the least severe pain groups increased from BL over time; the proportion of patients in the more severe pain groups or meeting criteria for FM decreased over time. The presence of non-nociceptive pain was associated with smaller improvements in total back pain, nocturnal back pain, fatigue, and BASFI from BL to wk 12 (Fig. 2). Non-nociceptive pain at wk 12 was associated with residual symptoms, anxiety/depression, and poor sleep (Fig. 2).

Conclusion. In patients with axSpA, treatment with UPA was associated with improvements in non-nociceptive pain. Non-nociceptive pain was associated with residual symptoms, sleep disturbance, signs of anxiety/ depression, and fatigue.

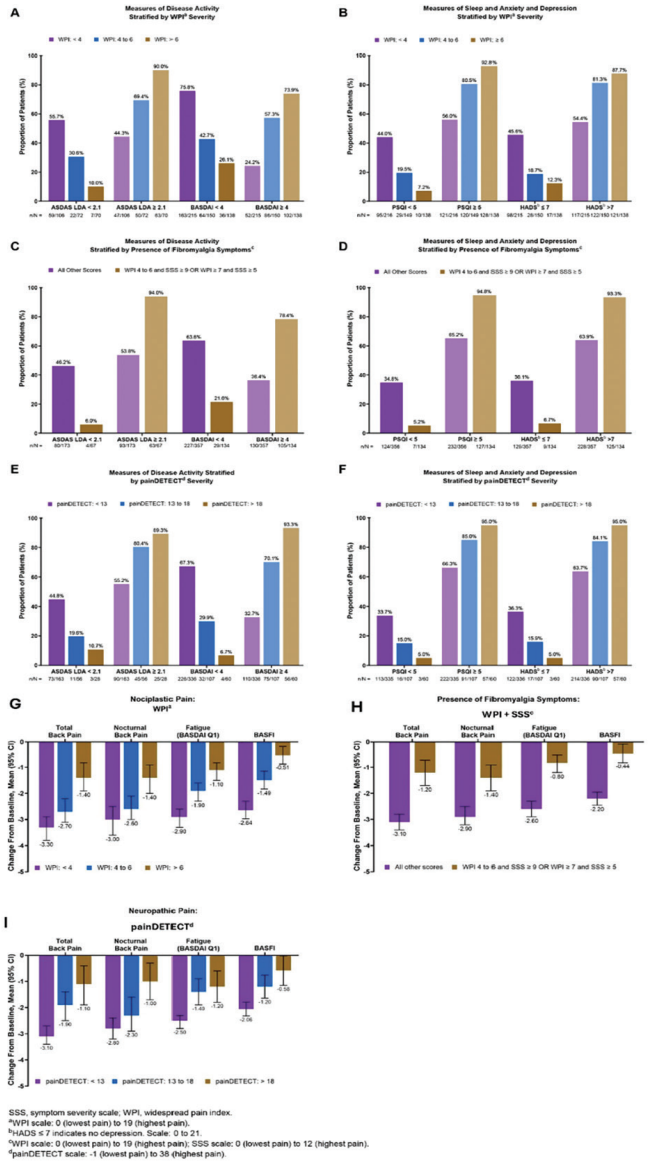


Fig. 2. Achievement of response or change from baseline at Week 12 stratified by non-nociceptive pain severity at Week 12.

P126

REAL WORLD DATA COMPARING THE SAFETY AND ADVERSE EVENT PROFILES OF BIOLOGIC THERAPIES AND TARGETED SYNTHETIC DMARDS IN ANKYLOSING SPONDYLITIS

Heydari-Kamjani M.[1], Murphy J.[2], Krupinski L.D.[1], Magrey M.N.[1]
[1]Dept. of Rheumatology, University Hospitals Cleveland Medical Center/Case Western Reserve University, Cleveland; [2]Dept. of Center for Clinical Research, University Hospitals Cleveland Medical Center, Cleveland, USA

Introduction. Biologic and targeted therapies, including TNF inhibitors (TNFi), IL-17 inhibitors (IL-17i), and Janus kinase inhibitors (JAKi), have transformed ankylosing spondylitis (AS) treatment, but comparative real-world safety data remain limited. This study used the TriNetX network to compare adverse events across these therapies.

Methods. A retrospective cohort study using TriNetX included adults with AS treated with TNFi, IL-17i, or JAKi for ≥90 days who were medication-naive and received a repeat prescription within 90 days. Patients with pre-existing adverse events or infections were excluded. Propensity score

matching (1:1 nearest neighbor) addressed baseline differences for TNFi vs. IL-17i and TNFi vs. JAKi. New-onset adverse events from 3 months to 5 years were assessed using odds ratios (ORs), and major adverse cardiovascular events (MACE) were evaluated with Kaplan-Meier and Cox proportional hazards models.

Results. 11,269 TNFi, 822 IL-17i, and 245 JAKi patients were identified (Table I). After matching, groups were balanced for age, sex, race, and inflammatory markers. All patients received monotherapy. IL-17i was associated with lower MACE risk versus TNFi (6.1% vs. 9.3%; OR 0.64, 95% CI 0.43–0.97) (Fig. 1A), though time-to-event analysis was non-significant (HR 0.83, 95% CI 0.56–1.23) (Fig. 1B). No significant differences were seen for mortality, malignancy, depression, herpes zoster, or infection (Fig. 1A). JAKi was associated with higher MACE risk versus TNFi (OR 2.40, 95% CI 1.14–5.06) (Fig. 1A) and increased hazard over time (HR 3.48, 95% CI 1.69–7.20) (Fig. 1B), with no significant differences were seen for mortality, malignancy, depression, herpes zoster, or infection (Fig. 1A).

Conclusion. In AS, IL-17i were associated with lower odds of MACE versus TNFi, while JAKi were associated with increased of MACE. No significant differences were observed in mortality, malignancy, or infection outcomes across treatment classes.

Table I. Baseline characteristics in adult AS patients treated with a TNF inhibitor (TNFi), IL-17 inhibitor (IL-17i), or JAK inhibitor (JAKi) for at least ≥90 days after diagnosis.

	Before Matching			After Matching			
	Ref. Group	Comparison 1	Comparison 2	Comparison 1		Comparison 2	
	TNFi	IL-17i	JAKi	TNFi	IL-17i	TNFi	JAKi
	N = 11,269	N = 822	N = 245[1]	N = 1,120[1]	N = 1,120[1]	N = 244[1]	N = 244[1]
Age at index	43.8 ± 14.5	48.9 ± 13.9*	51.9± 15.5*	48.8 ± 13.9	48.86 ± 13.9	51.8 ± 15.2	51.8 ± 15.4
Sex							
Male	4765 (42.3)	416 (50.6)*	177 (72.2)*	418 (50.9)	415 (50.5)	176 (72.1)	176 (72.1)
Female	6500 (57.7)	406 (49.4)*	68 (27.8)*	403 (49.1)	406 (49.5)	68 (27.9)	68 (27.9)
Race							
American Indian or Alaska Native	108 (1)	10 (1.2)	10 (4.1)*	10 (1.2)	10 (1.2)	10 (4.1)	10 (4.1)
Asian	801 (7.1)	51 (6.2)	21 (8.6)	56 (6.8)	51 (6.2)	21 (8.6)	21 (8.6)
Black or African American	733 (6.5)	51 (6.2)	12 (4.9)	41 (5)	51 (6.2)	11 (4.5)	12 (4.9)
Other Race	438 (3.9)	28 (3.4)	10 (4.1)	26 (3.2)	28 (3.4)	10 (4.1)	10 (4.1)
Unknown Race	1323 (11.7)	114 (13.9)	26 (10.6)	116 (14.1)	113 (13.8)	26 (10.7)	25 (10.2)
White	7843 (69.6)	572 (69.6)	178 (72.7)	580 (70.6)	572 (69.7)	180 (73.8)	178 (73)
Ethnicity							
Hispanic or Latino	638 (5.7)	40 (4.9)	20 (8.2)	35 (4.3)	40 (4.9)	17 (7)	19 (7.8)
Non-Hispanic	8095 (71.8)	563 (68.5)*	178 (72.7)	567 (69.1)	563 (68.6)	177 (72.5)	178 (73)
Unknown ethnicity	2536 (22.5)	219 (26.6)*	47 (19.2)	219 (26.7)	218 (26.6)	50 (20.5)	47 (19.3)
CRP (mg/L)	14 ± 27.1	14.67 ± 26.1	14.2 ± 31.2	15.9 ± 32.5	14.3 ± 27.2	14.8 ± 22.9	14.16 ± 31.2
Medications							
Adalimumab	6510 (57.8)	0 (0)	0 (0)	475 (57.9)	0 (0)	162 (66.4)	0 (0)
Certolizumab pegol	530 (4.7)	0 (0)	0 (0)	24 (2.9)	0 (0)	10 (4.1)	0 (0)
Etanercept	2465 (21.9)	0 (0)	0 (0)	169 (20.6)	0 (0)	32 (13.1)	0 (0)
Golimumab	692 (6.1)	0 (0)	0 (0)	52 (6.3)	0 (0)	25 (10.2)	0 (0)
Infliximab	1114 (9.9)	0 (0)	0 (0)	102 (12.4)	0 (0)	20 (8.2)	0 (0)
Secukinumab	0 (0)	662 (80.5)	0 (0)	0 (0)	661 (80.5)	0 (0)	0 (0)
Ixekizumab	0 (0)	156 (19)	0 (0)	0 (0)	156 (19)	0 (0)	0 (0)
Bimekizumab	0 (0)	10 (1.2)	0 (0)	0 (0)	10 (1.2)	0 (0)	0 (0)
Tofacitinib	0 (0)	0 (0)	132 (53.9)	0 (0)	0 (0)	0 (0)	131 (53.7)
Upadacitinib	0 (0)	0 (0)	114 (46.5)	0 (0)	0 (0)	0 (0)	114 (46.7)

[1]Mean ± SD; n (%)
*p<0.05 based on Welch Two Sample t-test or Pearson's Chi-squared test between TNFi *vs*. IL-17i and TNFi *vs*. JAKI

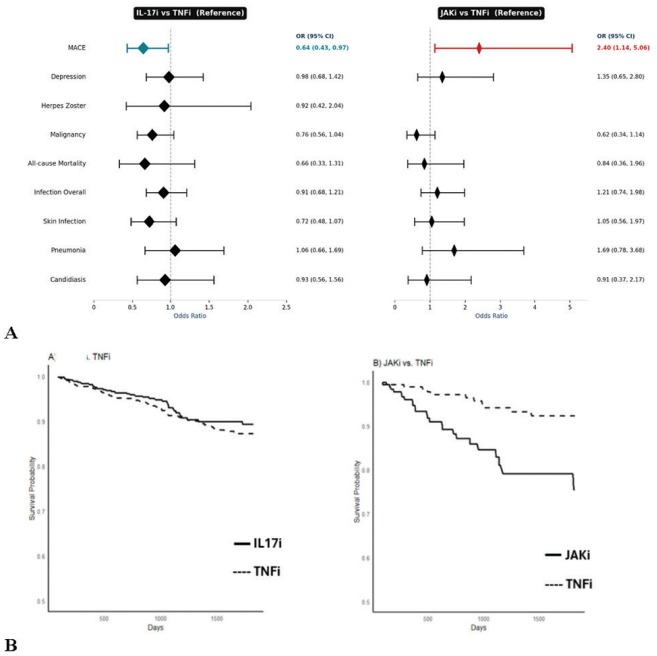


Fig. 1. A. New onset adverse symptoms within 3 months to 5 years between IL-17i *vs*. TNFi and JAKi *vs*. TNFi, after propensity score matching.
B. Kaplan-Meier curves depicting time to major adverse cardiovascular events (MACE) among patients treated within A) IL-17i *vs*. TNFi, and B) JAKi *vs*. TNFi within 3-months to 5-years of treatment initiation, after propensity score matching.

P127

DISTINCT PERIPHERAL BLOOD TRANSCRIPTOMIC SIGNATURES ASSOCIATED WITH DISEASE ACTIVITY IN AXIAL SPONDYLOARTHRITIS

Heydari-Kamjani M.[1], Richardson B.[2], Krupinski L.D.[1], Tamilselvan B.[2], Frumker L.[1], Cameron M.[2], Cameron C.[2], Magrey M.N.[1]
[1]Dept. of Rheumatology, University Hospitals Cleveland Medical Center/Case Western Reserve University, Cleveland; [2]Dept. of Nutrition, Case Western Reserve University, Cleveland, USA

Introduction. Axial spondyloarthritis (axSpA) is a chronic inflammatory disease with heterogeneous disease activity, yet its underlying molecular mechanisms remain incompletely understood. We hypothesized that peripheral blood mononuclear cell (PBMC) transcriptomic profiling would reveal distinct gene expression signatures associated with inflammatory burden in high versus low disease activity.

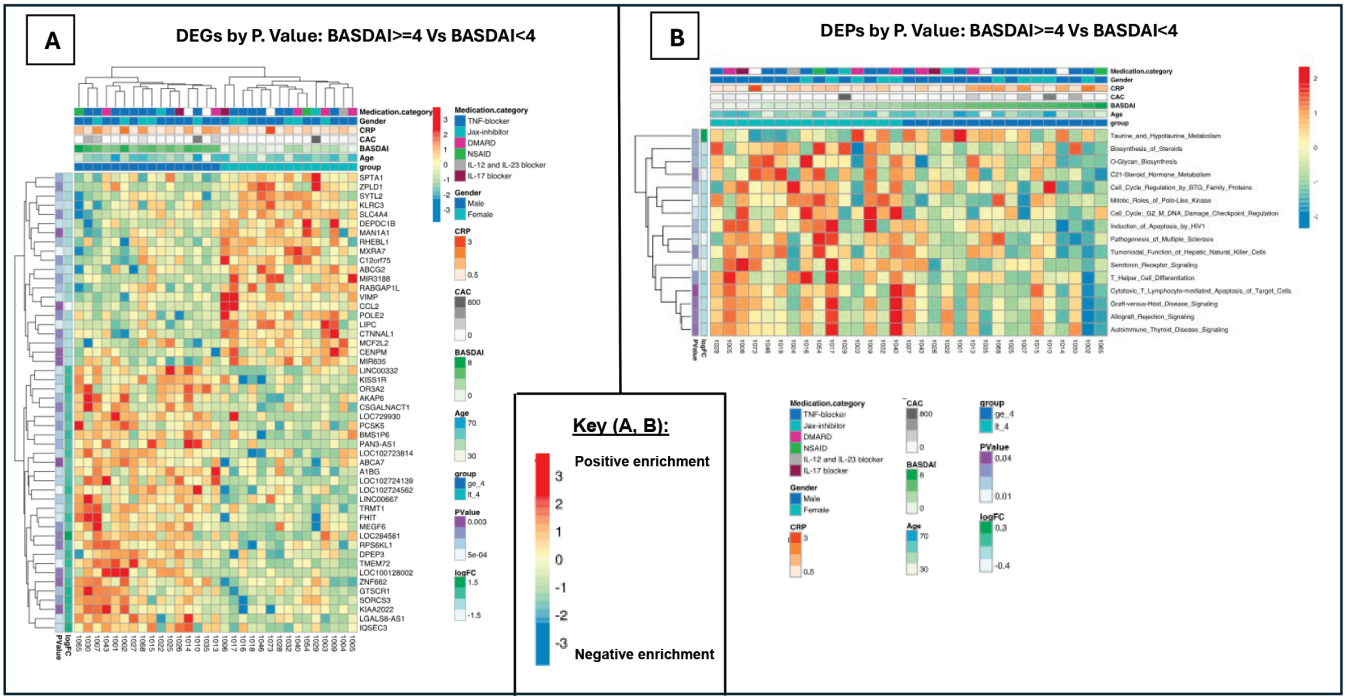
Methods. AxSpA patients were prospectively recruited from the Arthritis clinic and fulfilled 2009 ASAS classification criteria. Demographics, disease activity with Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) and treatment were collected. Categorical and continuous variables were analyzed using chi-square and Mann-Whitney U-tests. PBMCs isolated by Ficoll density-gradient centrifugation were cryopreserved and stored in RNA stabilization buffer prior to RNA extraction, library preparation, and bulk RNA sequencing. Patients were stratified into high (BASDAI ≥4) and low (BASDAI <4) disease activity group. Differentially expressed genes (DEGs) and pathways (DEPs) were identified through bioinformatic analyses and visualized using enrichment plots and heat maps.

Results. 31 axSpA patients were stratified into low (BASDAI <4, n=15) and high (BASDAI ≥4, n=16) groups (Table I). Patients were predominantly Caucasian (77%), male (67%), with a median age of 48 years, and TNFi was the most prevalent treatment (55%). Baseline demographics, treatment type, and CRP levels did not differ significantly between groups (all p>0.05). Comparison of BASDAI ≥4 versus <4 identified 871 DEGs and many significantly enriched DEPs from multiple databases (both p<0.05) (Fig. 1A/B). Pathway analysis revealed negative enrichment of serotonin receptor signaling (p=0.00535), C21 steroid hormone metabolism (p=0.0355), and multiple sclerosis pathogenesis (p=0.0141), with positive enrichment of taurine/hypotaurine metabolism (p=0.0262) (Fig. 1B).

Conclusion. Patients with high disease activity in axSpA demonstrate distinct PBMC transcriptomic signatures involving neurotransmitter signaling, steroid metabolism, and immune-related pathways. These findings provide insight into biological mechanisms associated with disease severity and warrant further validation to identify key genes driving these molecular differences.

Table I. Baseline characteristics of patients with axial spondyloarthritis stratified by BASDAI score (<4 vs. ≥4).

	BASDAI < 4	BASDAI > 4	TOTAL	P VALUE
COORT SIZE	15	16	31	-
MEDIAN AGE (IQR)	50 (41-59)	48 (38-60)	48 (39-60)	0.99
SEX (MALE %)	10 (66%)	11 (68%)	21 (67%)	0.9
RACE (%)				0.45
CAUCASIAN	10 (66%)	14 (88%)	24 (77%)	
AFRICAN AMERICAN	1 (7%)	1 (6%)	2 (6%)	
ASIAN	1 (7%)	0 (0%)	1 (3%)	
OTHER	3 (20%)	1 (6%)	4 (14%)	
TREATMENT TYPE (%)				0.96
NAÏVE	1 (7%)	2 (12%)	3 (10%)	
NSAID	1 (7%)	1 (6%)	2 (6%)	
TNFI	8 (53%)	9 (56%)	17 (55%)	
DMARD	3 (20%)	2 (12%))	5 (16%)	
OTHER BIOLOGIC	2 (13%)	2 (12%)	4 (13%)	



P127 - Fig. 1. Differential gene and pathway expression by disease activity in axial spondyloarthritis.
A. Heatmap of the top 50 differentially expressed genes (DEGs; $p<0.05$) stratified by BASDAI score (≥4 *vs*. <4). Columns represent individual patients; rows represent genes.
B. Heatmap of the top 16 differentially enriched pathways (DEPs; $p<0.05$) against increasing BASDAI scores (0-8). Columns represent individual patients; rows represent pathways.

P128

CAN A MULTIDISCIPLINARY MODEL IMPROVE THE MANAGEMENT OF COMPLEX IBD-SPA CASES? INSIGHTS FROM GASTROENTEROLOGY-RHEUMATOLOGY-PHARMACY MULTIDISCIPLINARY MEETINGS

Juárez-García M.[1], Navarro-Compán V.[1], Suárez-Ferrer C.[2], Amiama C.[2], Bohórquez C.[1], Tornero C.[1], Martín-Arranz E.[2], Peiteado D.[1], Poza J.[2], Rueda J.L.[2], Sánchez Azofra M.[2], Monjo I.[1], González M.A.[3], Castillo I.[3], Noci J.[2], Villalba A.[1], Bonilla G.[1], de Miguel E.[1], Martín-Arranz M.D.[2]*, Plasencia-Rodríguez C.[1]*
[1]Rheumatology Dept., Hospital Universitario La Paz, IdiPAZ, Madrid; [2]Inflammatory Bowel Disease Unit, Gastroenterology Dept., Hospital Universitario La Paz, IdiPAZ, Madrid; [3]Pharmacy Dept., Hospital Universitario La Paz, Madrid, Spain
**Contributed equally.*

Introduction. Spondyloarthritis (SpA) and inflammatory bowel disease (IBD) are immune-mediated inflammatory diseases with overlapping pathogenic and therapeutic pathways. In concomitant disease, multidisciplinary management is recommended, although its optimal real-world format remains unclear.
Objective. To describe cases discussed in Gastroenterology-Rheumatology-Pharmacy multidisciplinary meetings (MDMs) and assess their impact.
Methods. All cases discussed in MDMs from their implementation (October 2019) to July 2025 were retrospectively reviewed. Referral reasons were categorised as diagnostic evaluation, assessment of inflammatory activity by the counterpart specialty, or therapeutic decision. Impact was assessed according to referral category. Descriptive statistics are reported.
Results. Overall, 226 cases were discussed in 49 MDMs, corresponding to 155 patients (Table I); annual distribution is shown in Figure 1A. Most cases were discussed once (69.7%), 50.4% were women and mean age was 54.3±13.5 years. Gastroenterological diagnoses were mainly Crohn's disease (67.3%), while rheumatological diagnoses were predominantly axial, peripheral or axial/peripheral SpA. Cases had a median of 2 IMIDs and 29.2% had received ≥3 b/tsDMARDs. At MDM discussion, 58.8% were receiving a bDMARD, 9.7% a tsDMARD and 2.7% combination b/tsDMARD therapy.
Therapeutic decision was the most common referral reason (153/226, 67.7%), mainly b/tsDMARD switch or initiation (Fig. 1B). Decisions were implemented in 128 cases (83.7%), of which 95 (74.2%) were maintained at 6 months; face-to-face visits were avoided in 85 (55.5%). Assessment referrals accounted for 66 cases (29.2%): 58 (87.9%) were assessed, with median time to assessment of 29 days (IQR 13.0–57.0). Diagnostic referrals accounted for 19 cases (8.4%) and led to 6 new diagnoses (4 axSpA, 2 Crohn's disease), with median time to diagnosis of 44.5 days (IQR 14.3–77.3).
Conclusion. Gastroenterology-Rheumatology-Pharmacy MDMs are feasible and improve coordination in complex patients with concomitant SpA and IBD. They enabled effective therapeutic decisions, reduced face-to-face visits and facilitated cross-specialty assessment and diagnosis.

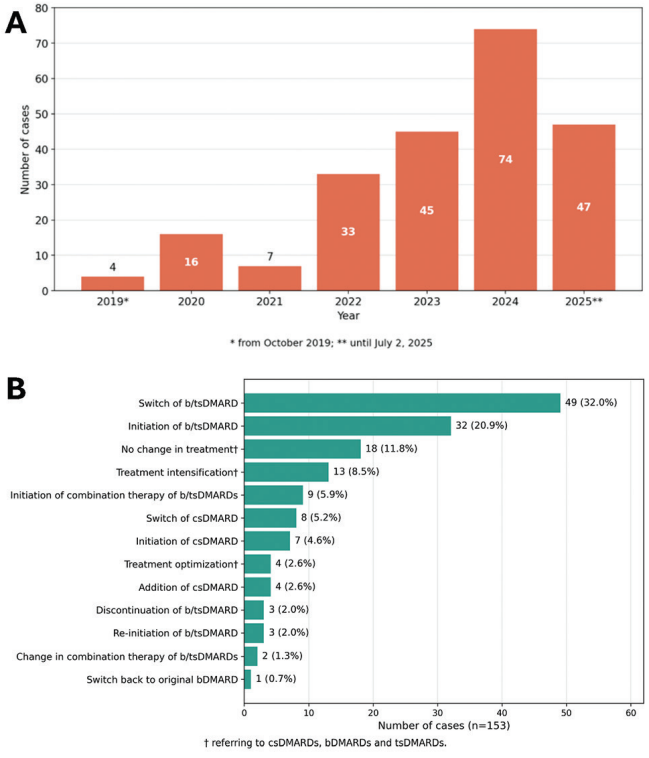


Fig. 1. Number of cases discussed per year (**A**) and distribution of therapeutic decisions (**B**).

P128 - Table I. Baseline characteristics of cases discussed.

		Value
Age (years), mean±SD		54.3±13.5
Sex (female), n (%)		114 (50.4)
Referring specialty, n (%)	Rheumatology	85 (37.6)
	Gastroenterology	135 (59.7)
	Both	6 (2.7)
Rheumatologic primary diagnosis, n (%)	axSpA	53 (23.5)
	pSpA	52 (23.0)
	axSpA&pSpA	48 (21.2)
	Psoriatic arthritis	10 (4.4)
	Rheumatoid arthritis	8 (3.5)
	Other	24 (10.6)
	No diagnosis	31 (13.7)
Gastroenterologic primary diagnosis, n (%)	Crohn's disease	152 (67.3)
	Ulcerative colitis	51 (22.6)
	Other	12 (5.3)
	No diagnosis	11 (4.9)
Number of IMIDs, n (%)	0	0 (0.0)
	1	35 (15.5)
	2	108 (47.8)
	≥ 3	83 (36.7)
IMIDs, n (%)	SpA	163 (72.1)
	IBD	209 (92.5)
	Uveitis	51 (22.6)
	Psoriasis	50 (22.1)
	Rheumatoid arthritis	8 (3.5)
	Erythema nodosum	12 (5.3)
	Hidradenitis suppurativa	2 (0.9)
	Sarcoidosis	3 (1.3)
	Behçet's disease	3 (1.3)
HLA-B27 positivity, n/N (%)		49/203 (24.1)
Number of previous b/tsDMARD, n (%)	0	52 (23.0)
	1	71 (31.4)
	2	37 (16.4)
	≥ 3	66 (29.2)
Baseline csDMARD or inmunomodulators, n/N (%)*	Any	144/226 (63.7)
	Methotrexate	47/144 (32.6)
	Sulfasalazine	37/144 (25.7)
	Mesalazine	42/144 (29.2)
	Azathioprine	18/144 (12.5)
	Other	13/144 (9.0)
	Combination	13/144 (9.0)
Baseline bDMARD mechanism, n/N (%)*	Any	133/226 (58.8)
	TNFi	81/133 (60.9)
	IL-12/23i	26/133 (19.5)
	IL-23i	5/133 (3.8)
	Integrin inhibitors	22/133 (16.5)
	IL-17i	1/133 (0.8)
	Other	2/133 (1.5)
Baseline tsDMARD, n/N (%)	Any	22/226 (9.7)
	Upadacitinib	16/22 (72.7)
	Tofacitinib	2/22 (9.1)
	Filgotinib	2/22 (9.1)
	Apremilast	1/22 (4.5)
	Ozanimod	1/22 (4.5)
Combination therapy with…, n (%)	2 bDMARDs	4 (1.8)
	bDMARD+tsDMARD	2 (0.9)

*Percentages may exceed 100% due to combination therapy. n=226 unless otherwise indicated. SD: standard deviation; IQR: interquartile range; SpA: spondyloarthritis (ax: axial, p: peripheral); IMIDs: immune-mediated inflammatory diseases; IBD: inflammatory bowel disease; DMARDs: disease-modifying antirheumatic drugs (cs: conventional synthetic, b: biologic, ts: targeted synthetic); TNFi: tumour necrosis factor inhibitor; IL-17i: interleukin-17 inhibitor; IL-12/23i: interleukin-12/23 inhibitor; IL-23i: interleukin-23 inhibitor.

P129

REASONS FOR TREATMENT DISCONTINUATION AND ADVERSE EVENT PATTERNS IN A TWO-DECADE SPONDYLOARTHRITIS BIOLOGIC REGISTRY

Kart Bayram G.S.[1], Ayan G.[1,2], Firlatan Yazgan B.[1,2], Bulat B.[1], Kustas A.A.[1], Donmez D.[1], Bulut S.[1], Akdoğan A.[1], Karadag O.[1], Apras Bilgen S.[1], Ertenli A.İ.[1], Kiraz S.[1], Kilic L.[1], Kalyoncu U.[1]

[1]Division of Rheumatology, Dept. of Internal Medicine, Hacettepe University Faculty of Medicine, Ankara; [2]Division of Rheumatology, Dept. of Internal Medicine, Ankara Training and Research Hospital, Ankara, Turkey

Background. Treatment discontinuation in spondyloarthritis (SpA) reflects not only treatment effectiveness but also adverse events (AEs) and therapy-specific tolerability. Understanding these patterns may provide clinically relevant insights from routine practice.

Methods. This retrospective registry-based study included 2,912 biologic and targeted synthetic treatment courses discontinued for any reason and recorded in the HUR-BIO database over a 20-year period. Discontinuation reasons were evaluated for TNF inhibitors (adalimumab, etanercept, infliximab, golimumab, certolizumab pegol), IL-17 inhibitors (secukinumab, ixekizumab), and JAK inhibitors (tofacitinib, upadacitinib). Reasons were categorized as primary inefficacy, secondary inefficacy, AEs, physician decision, patient decision, pregnancy, and other causes. AE-related discontinuations were further classified into predefined AE categories.

Results. Among 2,912 discontinued treatment courses, the leading reasons for discontinuation were primary inefficacy (31.9%) and secondary inefficacy (25.6%), followed by AEs (19.9%). Primary inefficacy was particularly frequent with secukinumab (51.2%), tofacitinib (56.5%), and upadacitinib (68.0%), whereas secondary inefficacy was most prominent with golimumab (35.3%). AE-related discontinuation was most frequently observed with etanercept (24.8%) and infliximab (22.7%). Infliximab also showed a distinct pattern, with physician decision accounting for 24.4% of discontinuations. AE profiles differed across therapies: allergic/infusion reactions predominated with infliximab, dermatologic events with golimumab and certolizumab pegol, and gastrointestinal events with IL-17 and JAK inhibitors, whereas infectious AEs were observed across all treatment groups.Detailed discontinuation patterns according to individual therapies are presented in Table I, while class-specific discontinuation profiles and AE signatures are summarized in Figure 1.

Conclusion. Treatment discontinuation patterns varied substantially across biologic and targeted therapies in real-world SpA practice. Although inefficacy remained the leading cause of discontinuation, distinct therapy-specific AE profiles also contributed to treatment cessation, highlighting the importance of both effectiveness and tolerability in long-term treatment outcomes.

Table I. Reasons for treatment discontinuation across biologic and targeted therapies in spondyloarthritis.

Therapy	Primary inefficacy	Secondary inefficacy	Adverse Events	Physician Decision	Patient decision	Pregnancy	Other	Total
TNF inhibitors								
Adalimumab	286(35.5)	230(28.6)	162(20.1)	23(2.8)	38(4.7)	7(0.8)	58(7.2)	804(100)
Certolizumabpegol	145(42.4)	89(26)	64(18.7)	16(4.6)	23(6.7)	1(0.2)	4(1.1)	342(100)
Etanercept	158(25.6)	166(26.9)	153(24.8)	23(3.7)	35(5.6)	14(2.2)	68(11)	617(100)
Golimumab	102(38.3)	94(35.3)	23(8.6)	16(6)	13(4.8)	7(2.6)	11(4.1)	266(100)
Infliximab	99(15.9)	116(18.6)	141(22.7)	152(24.4)	26(4.1)	3(0.4)	84(13.5)	621(100)
IL-17 inhibitors								
Ixekizumab	3(42.8)	1(14.2)	2(28.5)	1(14.2)	0	0	0	7(100)
Secukinumab	106(51.2)	51(24.6)	29(14)	5(2.4)	9(4.3)	3(1.4)	4(1.9)	207(100)
JAK inhibitors								
Tofacitinib	13(56.5)	1(4.3)	4(17.3)	3(13)	1(4.3)	0	1(4.3)	23(100)
Upadacitinib	17(68)	0	4(16)	3(12)	1(4)	0	0	25(100)
Total	929(31.9)	748(25.6)	582(19.9)	242(8.3)	146(5)	35(1.2)	230(7.8)	2912(100)

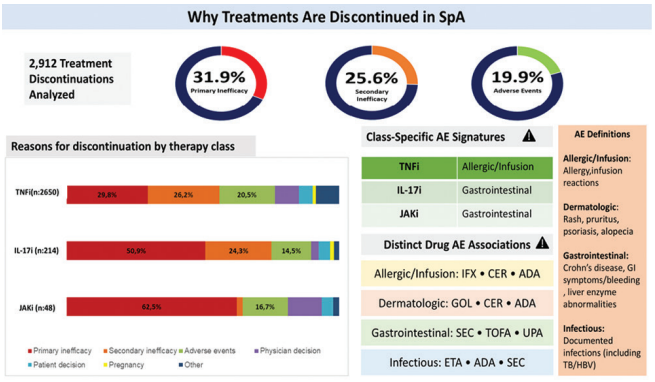


Fig. 1. Overview of treatment discontinuation patterns and class-specific adverse event signatures in SpA.

P130

DIGITAL BEHAVIORAL THERAPY IMPROVES OUTCOME IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS AND PERSISTENT PAIN: RESULTS FROM A RANDOMIZED CONTROLLED TRIAL

Kiefer D.[1,2,5], Sonkaya Y.[1,2], Krause D.[1,3], Voglau M.[4], Mintrop B.[1,3], Redeker I.[1,2], Baraliakos X.[1,2], Kiltz U.[1,2]
[1]Ruhr-Universität Bochum; [2]Rheumazentrum Ruhrgebiet, Herne; [3]Private rheumatology practice, Gladbeck; [4]Private rheumatology practice, Oldenburg; [5]Private rheumatology practice, Hattingen, Germany

Background/Objective. Axial spondyloarthritis (axSpA) is often associated with persistent pain despite effective anti-inflammatory treatment, substantially impacting patients' quality of life. Digital health applications (DHA) provide an innovative approach to address multidimensional aspects of chronic pain through psychological and behavioral strategies.
We aimed to assess the impact of a DHA utilizing the acceptance and commitment therapy (ACT) on disease outcomes, including the West Haven-Yale Multidimensional Pain Inventory (MPI), in axSpA patients experiencing chronic back pain despite stable pharmacological therapy.
Methods. This unblinded, monocentric, randomized controlled trial (1:1) compared an intervention group receiving the ACT-app with a standard of care (SOC) group. The ACT-app provided behavioral therapy in seven interactive lessons. Patients were followed for 12 weeks. The primary outcome was MPI pain-related life interference; secondary outcomes included pain severity, affective distress and other patient reported outcomes (PRO). Acceptance of the DHA was assessed using the Net promoter score (NRS 0-10). Linear models estimating the effect of the ACT-app on the change of MPI pain-related life interference (model 1) and affective distress (model 2), with adjustments made for their respective baseline values, were calculated.

Table I. Baseline characteristics stratified by group and user activity.

Characteristics		Intervention group total (n=73)	Active App Users (n=44)	Inactive App Users (n=29)	SOC patients (n=63)
Age (years)		46.8 (13)	47.3 (12)	46.2 (12.4)	50.9 (11,4)
Gender, female, n (%)		36 (49.3)	23 (52.3)	13 (44.8)	27 (42.9)
Time (years) since axSpA diagnosis		13.3 (13)	13.2 (13.7)	13.6 (12.4)	15.2 (12.7)
No. of patients on bDMARD, n (%)		47 (64.4)	29 (65.9)	18 (64.3)	42 (66.6)
No. of patients on tsDMARD, n (%)		5 (6.8)	4 (9.1)	1 (3.4)	3 (4.8)
No. of previous b-DMARDs		1.6 (1.4)	1.7 (1.5)	1.5 (1.6)	1.4 (1.1)
Physician global assessment (PhGA), 0-10		3 (1.5)	2.7 (1.6)	3.3 (1.3)	2.9 (1.2)
ASDAS, 0-10		2.7 (0.8)	2.6 (0.8)	2.9 (0.7)	2.9 (0.8)
BASFI, 0-10		4.6 (2.4)	4.5 (2.5)	4.8 (2.1)	4.9 (2.3)
BASMI		3 (1.4)	3.3 (1.3)	2.6 (1.4)	3.4 (1.6)
ASAS Health Index, 0-17		8.1 (3.5)	7.9 (3.7)	8.3 (3.3)	8 (3.8)
PainDETECT >18, n (%)		13 (29.5)	13 (29.5)	6 (21.4)	22 (36.0)
MPI	Pain severity (0-6)	3.4 (1.1)	3.1 (1.2)	3.8 (0.9)	3.84 (1.2)
	Pain-related life interference (0-6)	3.4 (1.1)	3.3 (1.3)	3.7 (0.6)	3.6 (1)
	Affective distress (0-6)	3.2 (1.2)	3.1 (1.3)	3.4 (1.1)	3.2 (1.2)
	Social support (0-6)	3.4 (1.9)	3.3 (2.0)	3.5 (1.7)	3.8 (1.5)
	Life control (0-6)	3.8 (1.2)	4.0 (1.2)	3.2 (1.1)	3.5 (1)
HADS-D, 0-21		7.9 (4.3)	7.6 (4.5)	8.3 (3.9)	9 (5.3)
HADS-A, 0-21		8.5 (4.4)	8.3 (4.6)	9.0 (4.1)	9 (4.3)
SF-36 (PCS), 0-100		33.5 (8.3)	33.5 (9.6)	33.5 (6.1)	32,6 (7.4)
SF-36 (MCS), 0-100		41.7 (12.2)	42.6 (12.2)	40.4 (12.2)	42,1 (13.1)

Values are presented as mean (standard deviation), unless otherwise indicated; ASA: Assesment of Spondyloarthritis International Society; ASDAS: Ankylosing Spondylitis Disease Activity Score, BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index; BASMI: Bath Ankylosing Spondylitis Metrology Index; CRP: C-reactive protein; FIQ: Fibromyalgia Impact Questionnaire; HADS: Hospital Anxiety and Depression Scale; MPI (D): Multidimensional Pain Inventory; NRS: Numerical rating scale; NSAID: non-steroidal anti-inflammatory drug; PAM 13: Patient Activation Measure-13; SF-36: Short Form 36; PCS: Physical component summary (PCS); MCS: Mental component summary.

Results. A total of 136 patients were randomized to the intervention (n=73) with ACT-app and SOC group (n=63) without ACT-app (Table I). Among intervention patients, 44 actively used ACT-app, while 29 either did not receive the app code by their health insurance company or did not use the ACT-app at all. At week 12, all lessons in the ACT-app were fully completed by 19 (43%) patients in the intervention group. Baseline characteristics, including MPI scores, were comparable between groups (Table I). Differences in changes of the two groups are shown in Figure 1. The improvements in pain-related life interference (model1: -0.36, 95% CI: -0.73 to 0.01) and affective distress related to the disease (model 2: -0.4, 95% CI: -0.84,0.003) were greater compared to SOC patients. Overall acceptance was moderate (Net Promoter Score 5.8 (4.3)).
Discussion. The ACT-app demonstrated a meaningful reduction in pain-related interference, supporting that the use of DHAs might become a supplementary tool in managing pain for axSpA patients. However, a substantial number of patients experienced challenges to use the app actively.

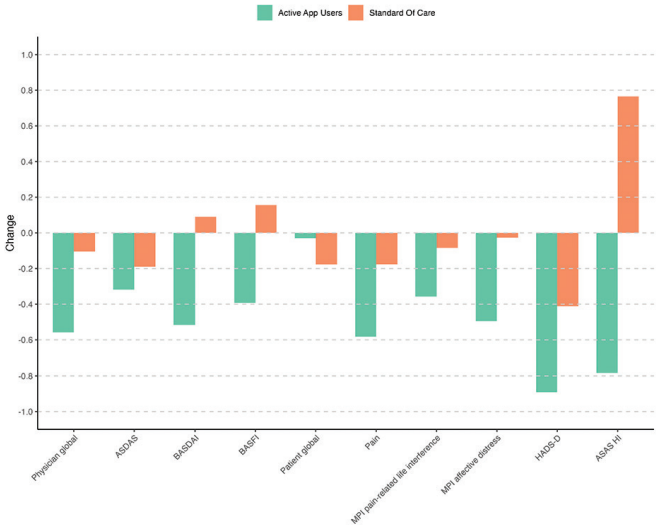


Fig. 1. Change of clinical characteristics stratified by group.

P131

ACCELERATING ACCESS TO BIOLOGIC THERAPY AND REHABILITATION IN AXIAL SPONDYLOARTHRITIS: A RETROSPECTIVE ANALYSIS OF MULTI-DISCIPLINARY AXSPA CLINIC

Bembridge L., Sooriyaarachchi D., Das S., Laxminarayan R.
Dept. of Rheumatology, Queen's Hospital, University Hospitals of Derby and Burton NHS Foundation Trust, Burton on Trent, UK

Background. Significant diagnostic and therapeutic delays remain a profound clinical challenge in axial spondyloarthritis (axSpA) (1). In addition, there is significant delays to see allied health professionals (clinical nurse specialist and specialist physiotherapist) (2). This study evaluates the operational and clinical impact of a multidisciplinary team (MDT) clinic designed to expedite specialist care and biologic initiation.
Methods. We are running a dedicated axSpA MDT comprising a rheumatologist, specialist physiotherapist and nurse specialist at our centre. A retrospective review of service delivery metrics and clinical outcomes across two cohorts: the conventional referral pathway (n=105) and the physiotherapy-led MDT pathway (n=53). Primary measures included wait times to see specialist allied health professionals, disease activity using Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), and functional capacity using Bath Ankylosing Spondylitis Functional Index (BASFI).
Results. Following the implementation of the physiotherapy-led MDT pathway, the average time to clinical nurse specialist evaluation decreased by 28% (42 to 30 days), physiotherapy review wait times dropped by 28% (75 to 54 days), and biologic treatment initiation accelerated by 62% (797 to 300 days). The physiotherapy-led MDT pathway achieved a significantly greater mean BASDAI reduction (Δ = 1.9, baseline 6.2 to 4.3) compared to the conventional referral pathway (Δ= 1.1, baseline 5.0 to 3.9; p=0.0037), exceeding the Minimal Clinically Important Difference (MCID).
Regarding functional outcomes, the conventional referral pathway showed a statistically significant BASFI reduction (3.9 to 3.2; p=0.035). Conversely, patients in the physiotherapy-led MDT pathway presented with a significantly higher baseline functional burden (5.3) that dropped to 4.3 at follow-up; while this absolute reduction was larger (Δ = -1.0), it was a non-significant trend (p=0.065). At follow-up, the physiotherapy-led MDT group maintained higher functional impairment (p=0.0055).

Conclusion. Transitioning to an MDT clinic pathway significantly reduces critical service delays and accelerates access to biologics. While this integrated model drives superior, highly predictable reductions in clinical disease activity, it captures a more complex patient population with a persistent functional burden, requiring intensive, long-term therapeutic strategies.

References
1. Diagnostic delay is common for patients with axial spondyloarthritis: results from National Early Inflammatory Arthritis Audit. doi: 10.1093/rheumatology/keab428
2. Improving standards of care for axial spondyloarthritis through project CATALYST: Experience from two UK centres. doi: 10.1002/msc.70073

P132

PREDICTORS OF A POSITIVE RESPONSE TO NSAIDS AND PHYSICAL EXERCISE IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS IN SUB-SAHARAN AFRICA

Lebughe P.L., Mavinga A.N., Ndaie G.M., Mbuyi J., Mulumba C.B., Mbuyi-Muamba J.M.
Dept. of Internal Medicine, Rheumatology Unit, University Hospital of Kinshasa, Democratic Republic of the Congo

Introduction. Axial spondyloarthritis (axSpA) is a chronic inflammatory rheumatic disease affecting mainly the sacroiliac joints and spine. Data on axSpA epidemiology, clinical features, and treatment outcomes in Sub-Saharan Africa are limited. This study aimed to identify predictors of treatment response to accessible therapies (NSAIDs and physical exercise) in Congolese patients with axSpA.
Methods. A prospective 24-week study was conducted at the University Hospital of Kinshasa from March to August 2024. Eighty-six axSpA patients received standardized NSAID treatment alongside a supervised exercise program targeting spinal mobility, posture, and physical function. Adherence was monitored through follow-ups. Treatment response was evaluated at 3 and 6 months using BASDAI, ASDAS-CRP, and BASFI scores. Variables analyzed included age, sex, disease duration, baseline severity, nocturnal pain, functional status, inflammatory markers (CRP, ESR), and exercise adherence. Multivariate logistic regression identified independent predictors of clinical improvement.
Results. Among 86 patients (53.5% male, mean age 43.2±18.4 years), 67.4% showed significant improvement in BASDAI and BASFI at 6 months. Positive response predictors were age under 40 (p=0.02), disease duration less than 2 years (p=0.01), baseline CRP under 5 mg/L (p=0.03), high exercise adherence (p<0.001), and absence of comorbidities such as renal failure, gastroduodenal ulcer, or obesity. Severe nocturnal pain and marked initial functional impairment correlated with poorer responses (p=0.04).
Conclusion. In Congolese axSpA patients, younger age, early disease stage, moderate inflammation, and strong adherence to physical exercise predict better outcomes with NSAID therapy combined with exercise. These findings highlight the need for early diagnosis and education to improve treatment efficacy in low-resource settings.

P133

PREDICTORS OF TREATMENT RESPONSE TO IL-17 INHIBITORS IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS: A MULTICENTER REAL-WORLD LONGITUDINAL STUDY

Oh S.[1], Yang J.[2], Park H.W.[2], Lee B.W.[2], Park Y.[2], Kwok S.K.[2], Ju J.H.[2], Kim W.U.[2], Park S.H.[2], Lee J.J.[2]
[1]Dept. of Internal Medicine, College of Medicine, The Catholic University of Korea, Seoul; [2]Division of Rheumatology, Dept. of Internal Medicine, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, Republic of Korea

Introduction. In patients with axial spondyloarthritis (axSpA) receiving biologics, several predictors of favorable response have been identified for TNF inhibitors, including elevated baseline C-reactive protein (CRP). It has been proposed that response to IL-17 inhibitors may be less dependent on baseline inflammatory status compared with TNF inhibitors. However, whether baseline inflammation has limited relevance in predicting IL-17 inhibitor response remains uncertain. This study aimed to identify predictors of treatment response to IL-17 inhibitors in patients with axSpA using multicenter real-world longitudinal data.
Methods. A total of 266 axSpA cases from the Korean College of Rheumatology Biologics and Targeted Therapy (KOBIO) registry and Seoul St. Mary's Hospital receiving IL-17 inhibitors were analyzed. Univariable and multivariable mixed-effects logistic regression analyses were performed to identify predictors for achieving Assessment of SpondyloArthritis International Society 40% response (ASAS40) and Ankylosing Spondylitis Disease Activity Score-Low Disease Activity (ASDAS-LDA) at 1, 2, and 3 years of treatment.

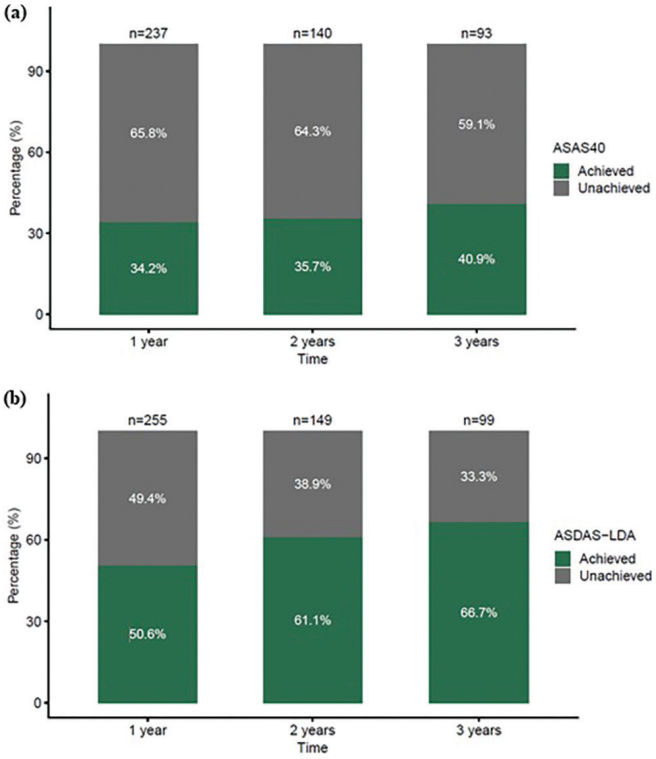


Fig. 1. Baseline characteristics stratified by group and user activity.

Table I. Multivariable mixed-effects logistic regression analysis of predictors for achieving ASAS40 and ASDAS-LDA at 1 and 3 years.

Variable	ASAS40				ASDAS-LDA			
	1 year		3 year		1 year		3 year	
	Multivariable OR (95% CI)	*P*-value	Multivariable OR (95% CI)	*P*-value	Multivariable OR (95% CI)	*P*-value	Multivariable OR (95% CI)	*P*-value
Age	0.97 (0.95–1.00)	**0.032***	0.96 (0.91–1.01)	0.114	1.00 (0.98–1.02)	0.907	0.99 (0.94–1.04)	0.628
Male gender	0.88 (0.46–1.70)	0.710	1.46 (0.49–4.34)	0.493	1.65 (0.86–3.15)	0.129	5.41 (1.12–26.2)	**0.036***
HLA-B27 positivity	N/A	N/A	N/A	N/A	N/A	N/A	3.43 (0.69–17.1)	0.132
Disease duration	N/A	N/A	N/A	N/A	1.00 (0.99–1.00)	0.161	N/A	N/A
BMI	0.97 (0.89–1.05)	0.464	0.85 (0.71–1.01)	0.072	0.94 (0.87–1.02)	0.141	0.78 (0.62–0.98)	**0.029***
Number of previous biologics	N/A	N/A	N/A	N/A	0.86 (0.68–1.09)	0.215	N/A	N/A
NSAID use	N/A	N/A	4.11 (0.81–20.9)	0.089	N/A	N/A	N/A	N/A
Baseline ASDAS-CRP	1.80 (1.27–2.54)	**<0.001****	2.54 (1.27–5.07)	**0.008****	0.75 (0.56–0.99)	**0.046***	0.84 (0.47–1.50)	0.557
BASFI	1.05 (0.91–1.21)	0.500	N/A	N/A	0.92 (0.80–1.05)	0.201	N/A	N/A
Psoriasis	N/A	N/A	N/A	N/A	N/A	N/A	3.84 (0.48–30.6)	0.204

*p<0.05, **p<0.01. OR: odds ratio; CI: confidence interval; N/A: not applicable.

Results. Achievement rates for ASAS40 at 1, 2, and 3 years were 34.2%, 35.7%, and 40.9%, and ASDAS-LDA achievement rates were 50.6%, 61.1%, and 66.7%, respectively (Fig. 1). Multivariable analysis showed that achieving ASAS40 was significantly predicted by higher baseline ASDAS-CRP (1-year OR 1.80, 2-year OR 1.93, 3-year OR 2.54, all p<0.01), younger age (1-year OR 0.97, p=0.032), and lower BMI (2-year OR 0.86, p=0.033).

In contrast, ASDAS-LDA achievement was associated with lower baseline ASDAS-CRP (1-year OR 0.75, p=0.046; 2-year OR 0.49, p=0.011), NSAID use (2-year OR 3.59, p=0.032), male gender (3-year OR 5.41, p=0.036), and lower BMI (3-year OR 0.78, p=0.029) (Table I).

Conclusion. Higher baseline ASDAS-CRP predicted achievement of clinical improvement (ASAS40), whereas lower baseline ASDAS-CRP was associated with achieving target status (ASDAS-LDA). Younger age, male sex, lower BMI, and NSAID use were additionally associated with favorable outcomes.

P134

WHO BENEFITS MOST FROM INTENSIVE TREATMENT IN EARLY PSORIATIC ARTHRITIS? INSIGHTS FROM AN IPD META-ANALYSIS AND CAUSAL MACHINE LEARNING

Lema Martinez J.M.[1,2], Jiang Y.[1], Kang J.H.[1], De Marco G.[3,4,5], den Broeder A.A.[6], van Herwaarden N.[6], Koc G.H.[7], Vis M.[7], Kok M.R.[8], Luime J.J.[7], Marzo-Ortega H.[3,4], Mulder M.L.M.[6], Tchetverikov I.[9], Massa S.[1,2], Coates L.C.[1]

[1]NDORMS, University of Oxford, Oxford; [2]Oxford Clinical Trials Research Unit, University of Oxford, Oxford; [3]NIHR Leeds Biomedical Research Centre, University of Leeds, Leeds; [4]Leeds Institute of Rheumatic and Musculoskeletal Medicine, University of Leeds, Leeds; [5]Dept. of Rheumatology, Mid Yorkshire Teaching NHS Trust, Wakefield, UK; [6]Dept. of Rheumatology, Sint Maartensskliniek, Nijmegen; [7]Dept. of Rheumatology, Erasmus MC, Rotterdam; [8]Dept. of Rheumatology and Clinical Immunology, Maasstad Hospital, Rotterdam; [9]Dept. of Rheumatology, Albert Schweitzer Hospital, Dordrecht, The Netherlands

Introduction/Objective. Several RCTs have examined intensive versus standard treatment in early or DMARD-naive psoriatic arthritis (PsA). While COMPLETE-PsA and SPEED trials demonstrated superior disease control with combination csDMARDs/biologic induction over standard step-up care, GOLMePsA and STAMP showed only small benefits of early biologics over methotrexate (MTX)-based treat-to-target care, suggesting intensive strategies may benefit specific subgroups. We aimed to estimate the overall treatment effect using individual participant data meta-analysis (IPD-MA), and to identify subgroups most likely to benefit using causal machine learning.

Methods. IPD from four RCTs (n=440) were pooled. The primary outcome was PASDAS at week 16/24. A one-stage IPD-MA used linear mixed models (LMM) with random trial intercepts, adjusted for baseline covariates. Robustness was assessed via Bayesian LMM, leave-one-out cross-validation, two-stage meta-analysis, and multiple imputation. Treatment effect heterogeneity was evaluated using causal forests with shallow policy trees (depth-2 and depth-3) to derive clinically interpretable treatment decision rules.

Results. The complete case analysis included 349 participants. Intensive therapy demonstrated a significant improvement over standard care (PASDAS mean difference: -0.62; 95% CI: -0.91 to -0.33), approaching the minimal clinically important difference of 0.8. Results were consistent across sensitivity analyses; exclusion of SPEED attenuated the effect (Fig. 1). Causal forest analyses revealed substantial heterogeneity in individual treatment effects. Age (18.9%), baseline PASDAS (16.9%), and BMI (16.8%) were the leading drivers. Depth-2 tree identified benefit from intensive therapy for younger patients with lower BMI and older patients; the depth-3 tree further refined recommendations using baseline PASDAS and sex (Fig. 2), with approximately three-quarters of participants recommended intensive treatment.

Conclusion. Intensive treatment in early PsA provides a clinically meaningful improvement in disease activity over standard care. Causal machine learning reveals substantial treatment effect heterogeneity and unveils subgroups most likely to benefit, demonstrating the potential of these techniques to inform personalised treatment allocation in early PsA.

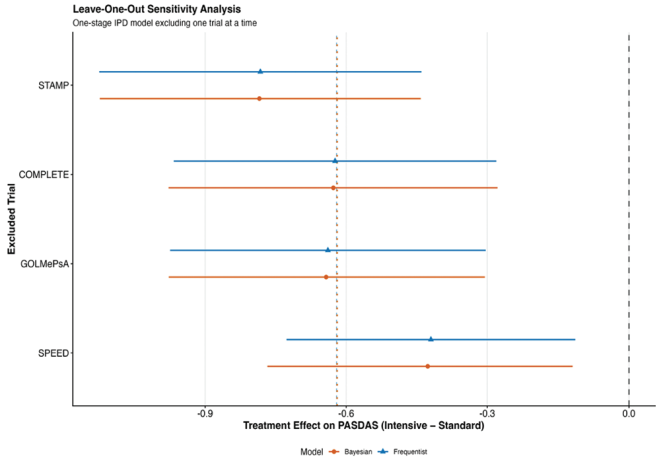


Fig. 1. Comparison of the adjusted treatment effect when iteratively excluding one trial from the analysis. The Frequentist estimates and 95% confidence intervals are depicted using blue lines and triangles, and Bayesian posterior means and 95% credible intervals are shown using orange lines and circles. The vertical dashed line represents the pooled estimate from the full dataset (= 0.0.62). The vertical grey dashed line indicates the reference at 0.

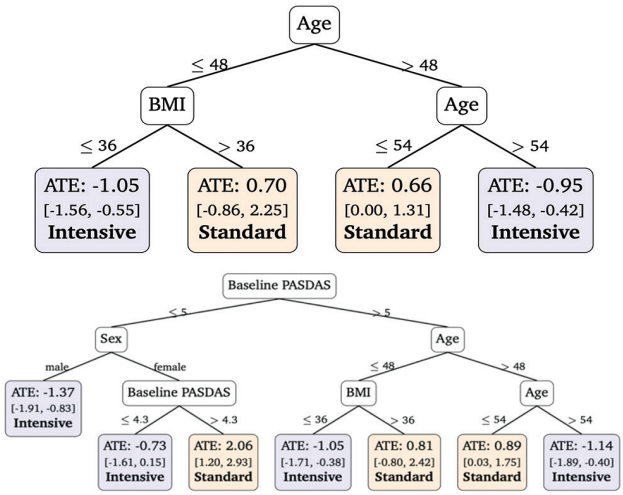


Fig. 2. (Top) Depth-2 policy tree showing the most interpretable treatment decision rule derived from causal forest estimates. Age was identified as the primary splitting variable, with subsequent stratification by body mass index (BMI), generating patient subgroups for which intensive therapy or standard care was estimated to provide greater benefit. (Bottom) Depth-3 policy tree providing a more granular representation of treatment effect heterogeneity. Additional splits incorporate baseline disease activity and demographic variables, yielding more refined subgroup definitions but with increased model complexity.
Terminal nodes display the estimated conditional average treatment effect and corresponding confidence intervals for intensive versus standard therapy. The average treatment effect (ATE) represents the estimated difference in outcome between intensive and standard therapy for patients within that subgroup – a negative ATE indicates that intensive therapy is estimated to produce lower disease activity scores than standard care and therefore suggests greater benefit from intensive therapy. Internal nodes display splitting variables; edges display decision rules; terminal nodes display leaf-level average treatment effect with 95% confidence intervals and recommended treatment. Outcomes are PASDAS units at week 16/24.

P135

DURABILITY OF INHIBITION OF STRUCTURAL DAMAGE PROGRESSION AND IMPROVEMENTS IN JOINT DISEASE ACTIVITY WITH GUSELKUMAB IN ACTIVE AND EROSIVE PSORIATIC ARTHRITIS: WEEK 48 RESULTS FROM APEX

Ritchlin C.T.[1], Mease P.J.[2,3], Coates L.C.[4], Kollmeier A.P.[5], Zhou B.[6], Jiang Y.[6], Bensley K.[6], Im K.[7], Batra R.[8], Lozenski K.[9], Fakharzadeh S.[9,10], Chakravarty S.D.[9,11], Rahman P.[12,*], van der Heijde D.[13]

[1]Dept. of Medicine, Allergy/Immunology and Rheumatology, University of Rochester Medical Center, Rochester; [2]Rheumatology Research, Providence Swedish Medical Center, Seattle; [3]University of Washington School of Medicine, Seattle, USA; [4]Nuffield Dept. of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Botnar Research Centre, Oxford, UK; [5]Johnson & Johnson, San Diego; [6]Johnson & Johnson, Spring House; [7]Johnson & Johnson, Cambridge, USA; [8]Johnson & Johnson, Toronto, Canada; [9]Johnson & Johnson, Horsham; [10]Dept. of Dermatology, Perelman School of Medicine University of Pennsylvania, Philadelphia; [11]Drexel University College of Medicine, Philadelphia, USA; [12]Craig L Dobbin Genetics Research Centre, Faculty of Medicine, Division of Rheumatology, Memorial University of Newfoundland, St. Johns, Canada; [13]Leiden University Medical Center, Leiden, The Netherlands

Introduction. APEX (NCT04882098) evaluates guselkumab (GUS), a fully-human mAb that binds CD64 and selectively inhibits the IL-23p19-subunit, in participants with active and erosive PsA. At Week (W)24, GUS demonstrated significantly higher ACR20 rates (primary endpoint) and radiographic progression inhibition vs placebo (PBO; major secondary endpoint). Here we report W48 findings.
Methods. APEX enrolled adults with active PsA (≥3 tender, ≥3 swollen joints; CRP ≥0.3 mg/dL) and ≥2 erosive joints on radiographs of hands/feet, despite previous non-biologic therapy. The modified full analysis set comprised 1020 randomized participants (273 GUS 100mg Q4W; 371 GUS 100mg at W0/W4 then Q8W; 376 PBO-to-GUS Q4W at W24). Key endpoints through W48 included ACR20/ACR50 rates and least-squares mean (LSM) change in PsA-modified van der Heijde-Sharp (vdH-S) score per reading session 2 (W0/24/48 radiographs). Exposure-adjusted incidence rates of adverse events (AEs) per 100 participant-years (100PY) [95% CI] are reported through W48.
Results. GUS Q4W/Q8W ACR20 rates increased from W24 (67%/68% vs. 47% PBO; both $p<0.001$) to W48 (71%/74%). GUS ACR50 rates increased from W24 (41%/42% vs. 20% PBO; both nominal-$p<0.001$) to W48 (51%/56%). At W48, 71% and 48% of PBO-to-GUS Q4W participants achieved ACR20 and ACR50, respectively. Reading session 2 results indicated continued suppression of radiographic progression with GUS Q4W/Q8W from W0-24 (LSM changes in PsA-modified vdH-S score: 0.36/0.46) throughout W24-48 (0.24/0.32). Radiographic progression in the PBO group from W0-24 (0.96) was curtailed by GUS W24-48 (0.41). Through W24, AE incidence rates with GUS Q4W/Q8W (168 [146-191]/163 [145-183]) and PBO (174 [155-195]) were similar. Incidence rates did not increase with continued GUS (W0-48:147 [132-163]/148 [136-162]), or after PBO-to-GUS transition (W24-48:156 [138-176]).
Conclusion. In biologic-naive adults with active and erosive PsA, inhibition of radiographic progression and joint disease activity improvements with GUS were durable through W48 without increased AE incidence, further substantiating GUS benefit for preserving joint health.
Disclosures. CTR received grant/research support from AbbVie, Amgen, UCB and consulting fees from AbbVie, Amgen, Eli Lilly, Gilead, Johnson & Johnson, Novartis, Pfizer, UCB. **PJM** consultant for AbbVie, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Inmagene, Johnson & Johnson, Merck, Moonlake, Novartis, Pfizer, Spyre, SUN Pharma, Takeda, and UCB; received grants from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, Sana, and UCB; speaker for AbbVie, Amgen, Eli Lilly, Johnson & Johnson, and UCB. **LCC** has received grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Johnson & Johnson, Novartis, Pfizer and UCB; worked as a paid consultant for AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Gilead, Galapagos, Johnson & Johnson, Moonlake, Novartis, Pfizer, and UCB; and has been paid as a speaker for AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GlaxoSmithKline, Johnson & Johnson, Medac, Novartis, Pfizer, and UCB**. APK, BZ, YJ, KB, KI, RB, SF, and SDC** are employees of Johnson & Johnson; employees may own stock/stock options in Johnson & Johnson. **KL** is an employee of Johnson & Johnson and owns stock/stock options in Bristol Myers Squibb and Johnson & Johnson. **PR** received consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Johnson & Johnson, Merck, Novartis, Pfizer, and UCB; meeting attendance/travel support from Johnson & Johnson; and research grants from Johnson & Johnson, and Novartis. **DvdH** received consulting fees from AbbVie, Alfasigma, ArgenX, Bristol Myers Squibb, Elly-Lilly, Grey-Wolf Therapeutics, Johnson & Johnson, Novartis, Pfizer, Takeda, UCB Pharma and is associate editor of Annals Rheumatic Diseases, and is an editorial board member Journal of Rheumatology, and director of Imaging Rheumatology BV.

P136

INTEGRATED PROTEOMICS AND MACHINE LEARNING REVEAL PREDICTORS OF TNFI RESPONSE IN PSORIATIC ARTHRITIS

Martin-Salazar J.E.[1*], Arias-de la Rosa I.[1,2], López-Montilla M.D.[1], Ortiz-Buitrago P.[1], Puche-Larrubia M.A.[1], Ruiz-Ponce M.[1], Pérez-Sánchez C.[1,3,4], Barranco A.M.[1], Ábalos-Aguilera M.C.[1], Romero-Zurita L.[1], Moreno-Caño E.[1], Escudero-Contreras A.[1], Collantes-Estévez E.[1], López-Medina C.[1*], Barbarroja N.[1,3*]
[1]Rheumatology service/Dept. of Medical and Surgical Sciences, Maimonides Institute for Research in Biomedicine of Cordoba (IMIBIC)/ /University of Cordoba/ Reina Sofia University Hospital, Córdoba; [2]Dept. of Gastroenterology, Hospital General de Tomelloso, Tomelloso; Instituto de Investigación Sanitaria de Castilla-La Mancha (IDISCAM), Toledo; [3]Cobiomic Bioscience S.L, Córdoba; [4]Dept. of Cell Biology, Physiology and Immunology, Maimonides Institute of Biomedical Research of Cordoba (IMIBIC), Reina Sofia University Hospital, University of Córdoba, Spain

Introduction. Psoriatic arthritis is a heterogeneous immune-mediated disease in which up to 40% of patients fail to respond adequately to TNF inhibitors. Currently, no validated biomarkers are available to predict therapeutic response, leading to empirical treatment strategies. The aim of this study was to identify predictive biomarkers of TNFi response in PsA using high-throughput proteomics and machine learning approaches, and to evaluate longitudinal molecular changes associated with treatment.
Methods. A prospective study including 47 PsA patients initiating TNFi therapy was conducted. PBMCs were analyzed using Olink® proteomics technology assessing 384 inflammatory-related proteins. Clinical response was defined as ≥50% reduction in DAPSA after 6 months. Machine learning models integrating clinical and molecular variables were developed using logistic regression and cross-validation. An independent cohort of 26 patients was used for validation. Longitudinal analyses were performed in 20 patients. *In vitro* PBMC assays using responder and non-responder sera assessed functional differences.
Results. Nine proteins were differentially expressed between responders and non-responders at baseline. A predictive model combining ESR, C1QA, and CD200 demonstrated the best discriminatory performance (AUC=0.84), outperforming isolated clinical or molecular models. External validation confirmed the robustness of the model (AUC=0.80). Longitudinal analyses revealed significant reductions in DAPSA, CRP, ESR, and neutrophil-associated pathways after TNFi treatment, particularly in responders. Conversely, proteins related to B-cell activation and immune regulation were enriched after therapy. Both CD200 and C1QA expression significantly increased only in responders during follow-up. *In vitro* experiments demonstrated differential modulation of key biological pathways between responder and non-responder sera, including cell adhesion, proliferation, and inflammatory signaling pathways.
Conclusion. An integrated biomarker signature based on ESR, C1QA, and CD200 accurately predicts TNFi response in PsA and may potentially contribute to precision medicine strategies. TNFi response appears associated with coordinated modulation of innate and adaptive immune pathways, supported by functional *in vitro* validation.
Acknowledgements. Funded by the Instituto de Salud Carlos III (PI22/00539 and RICOR RD21/0002/0033), co-funded by the European Union, and by the Junta de Andalucía (PI-0243-2022).

P137

BIMEKIZUMAB TREATMENT IN ROUTINE CLINICAL CARE FOR AXIAL SPONDYLOARTHRITIS: INTERIM OBSERVATIONAL EVIDENCE OF PATIENT-REPORTED OUTCOME IMPROVEMENTS FROM THE EUROPEAN, MULTICENTRE SPEAK STUDY

Baraliakos X.[1], Kiltz U.[1], de Vlam K.[2], Ruyssen-Witrand A.[3], Ramírez-García J.[4], Walsh J.A.[5], Poddubnyy D.[6,7,8], Nicholls D.[9], Oortgiesen M.[10], Castagna F.[11], Bonny M.A.[12], Korjagina M.[13], Bajracharya R.[14], Öhagen P.[15], de la Loge C.[12], Ganz F.[16], Tillett W.[17,18]
[1]Rheumazentrum Ruhrgebiet Herne, Ruhr-University Bochum, Germany; [2]Dept. of Rheumatology, UZ Leuven, Leuven, Belgium; [3]CHU of Toulouse – Pierre-Paul Riquet Hospital (Inflammation, Infection, Immunology and Locomotor Center – Rheumatology Service), Toulouse, France; [4]Rheumatology Dept., Hospital Clinic and IDIBAPS of Barcelona, Barcelona, Spain; [5]Division of Rheumatology,

Salt Lake City Veterans Affairs Health and University of Utah Health, Salt Lake City, USA; [6]Division of Rheumatology, Dept. of Medicine, University of Toronto, Toronto; [7]University Health Network, Schroeder Arthritis Institute, Toronto, Canada; [8]Dept. of Gastroenterology, Infectious Diseases and Rheumatology, Charité – Universitätsmedizin Berlin, Berlin, Germany; [9]Clinical Trials Unit, University of the Sunshine Coast, Queensland, Australia; [10]UCB, Raleigh, USA; [11]UCB SpA, Milano, Italy; [12]UCB, Brussels, Belgium; [13]UCB, Breda, The Netherlands; [14]UCB, Slough, UK; [15]UCB, Uppsala, Sweden; [16]UCB, Bulle, Switzerland; [17]Royal National Hospital of Rheumatic Diseases, Bath; [18]Dept. of Life Sciences, Centre for Therapeutic Innovation, University of Bath, Bath, UK

Introduction/Objective. Axial spondyloarthritis (axSpA) puts significant burden on patients' health-related quality of life (HRQoL). Bimekizumab selectively inhibits interleukin (IL)-17F/IL-17A, and has demonstrated sustained efficacy in patient-reported outcomes (PROs) to 3-years in patients with non-radiographic(nr-)/radiographic(r-)axSpA in clinical trials. SPEAK is the first prospective, ongoing 52-week (wk), multicentre observational study evaluating bimekizumab effectiveness on HRQoL/PROs in adult patients with axSpA or PsA in European routine clinical practice. We report planned interim data from patients with axSpA in SPEAK, including patient characteristics, safety and effectiveness to 24wks, by prior treatment history.
Methods. Enrolment began in September 2023, data-cut at April 2025 when ~50% completed Wk24. Baseline characteristics and change from baseline (CfB) are reported to 24wks, stratified by prior b/tsDMARD exposure: ASAS-HI total score (0–17; primary outcome was Wk52 CfB), PGADA (0–10 [rescaled to derive ASDAS]) and SF-36 PCS. Adverse events (AEs) were collected/reported following pharmacovigilance reporting guidelines.
Results. By data-cut, baseline characteristics were available for 197 patients (nr-axSpA:78/r-axSpA:119; Table I). Improvements in ASAS-HI/PGADA/SF-36 PCS were observed regardless of treatment line (Fig. 1). For ASAS-HI and SF-36 PCS, improvements were numerically greater in b/tsDMARD-naive patients versus those with prior exposure.

Table I. Baseline characteristics of patients with axSpA in the SPEAK study.

Characteristic, mean (SD) [n] unless otherwise stated	Prior b/tsDMARD exposure			Total N=197
	b/tsDMARD naïve n=38	1 b/tsDMARD n=56	≥2 b/tsDMARDs[a] n=103	
Age, years	38.8 (10.7)	46.6 (13.0)	47.4 (12.4)	45.5 (12.7)
Sex, male, n (%)	20 (52.6)	27 (48.2)	45 (43.7)	92 (46.7)
BMI, kg/m^2	28.6 (5.9)	28.8 (8.9) [54]	27.8 (6.0) [102]	28.2 (6.9) [194]
nr-axSpA, n (%) **r-axSpA**, n (%)	18 (47.4) 20 (52.6)	23 (41.1) 33 (58.9)	37 (35.9) 66 (64.1)	78 (39.6) 119 (60.4)
Time since first symptoms, years	9.1 (7.5)	13.1 (10.6)	16.1 (11.9) [99]	13.9 (11.1) [193]
Time since diagnosis, years	3.7 (5.6)	7.6 (8.8)	11.0 (9.6) [101]	8.6 (9.1) [195]
History of uveitis, n (%)	3 (7.9)	12 (21.4)	16 (15.5)	31 (15.7)
History of IBD, n (%)	0 (0)	0 (0)	3 (2.9)	3 (1.5)
History of peripheral arthritis, n (%)	11 (28.9)	24 (42.9)	41 (39.8)	76 (38.6)
CRP, mg/L, median (min–max)	5.3 (0.3–62.0) [30]	4.0 (0.5–93.0) [40]	3.9 (0.0–90.0) [90]	4.0 (0.0–93.0) [160]
ASAS-HI total score	7.7 (3.5)	8.8 (3.9) [46]	9.6 (3.7) [92]	9.0 (3.8) [176]
PGADA score[b]	5.4 (1.8)	5.9 (2.0) [46]	6.3 (1.9) [92]	6.0 (1.9) [176]
SF-36 PCS score[c] **SF-36 PCS <45**, n (%)	37.5 (7.1) [37] 30 (78.9)[d]	35.7 (6.6) [45] 41 (73.2)[e]	33.7 (7.3) [84] 77 (74.8)[f]	35.1 (7.2) [166] 148 (75.1)[g]
SF-36 MCS score[c] **SF-36 MCS <45**, n (%)	43.1 (11.8) [37] 20 (52.6)[d]	40.9 (12.2) [45] 30 (53.6)[e]	41.5 (12.2) [84] 52 (50.5)[f]	41.7 (12.1) [166] 102 (51.8)[g]
ASDAS	3.2 (1.0) [29]	3.0 (0.9) [31]	3.2 (0.8) [79]	3.1 (0.9) [139]
BASDAI total score	5.5 (1.9) [37]	5.7 (1.7) [46]	6.0 (1.8) [92]	5.8 (1.8) [175]

Safety analysis set (includes all patients enrolled into the study). Where the number of patients with available data for a specific baseline characteristic differed from the overall population size, the number of patients with available data has been given in square brackets [n].
[a] Of 103 patients with ≥2 prior b/tsDMARDs, 12.6% received ≥7 prior b/tsDMARDs. Of 77 patients previously treated with ≥2 b/tsDMARDs with distinct mechanisms of action, 63 received prior TNFi and IL-17Ai therapy.
[b] PGADA in this study was assessed using a 0–100 VAS. Data presented here are the rescaled score used to derive ASDAS and which ranges from 0 to 10.
[c] SF-36 PCS and MCS are norm-based T-scores, where higher scores reflect better HRQoL.
[d] 1 patient (2.6%) had a missing score at baseline.
[e] 11 patients (19.6%) had missing scores at baseline.
[f] 19 patients (18.4%) had missing scores at baseline.
[g] 31 patients (15.7%) had missing scores at baseline.
ASAS-HI: Assessment of SpondyloArthritis international Society Health Index; ASDAS: Ankylosing Spondylitis Disease Activity Score; axSpA: axial spondyloarthritis; b/tsDMARD: biological/targeted-synthetic disease modifying anti-rheumatic drug; BASDAI: Bath Ankylosing Disease Activity Index; BMI: body-mass index; CRP: C-Reactive protein; HRQoL: health-related quality of life; IBD: inflammatory bowel disease; MCS: Mental Component Summary; nr-axSpA: non-radiographic axSpA; PCS: Physical Component Summary; PGADA: Patient Global Assessment of Disease Activity; r-axSpA: radiographic axSpA; SD: standard deviation; SF-36: 36-Item Short Form Survey; VAS: visual analogue score.

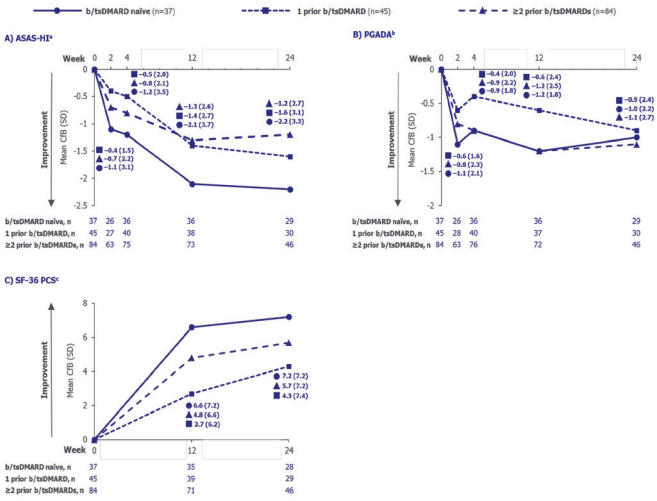


Fig. 1. Change from baseline in (**A**) ASAS-HI, (**B**) PGADA and (**C**) SF-36 PCS scores in patients with axSpA stratified by prior treatment, to Week 24 (OC).
Full analysis set. Values were rounded to 1 decimal place. Dates are approximate depending on how close to baseline the first dose was received. Data are reported as observed case.
[a] ASAS-HI total score ranges from 0–17 (higher scores indicate worse status).
[b] PGADA in this study was assessed using a 0–100 VAS (higher scores indicate higher disease activity); data presented here are the rescaled scores used to derive ASDAS and which range from 0 to 10.
[c] The SF-36 PCS is a norm-based T-score, where higher scores reflect better HRQoL. ASAS-HI: Assessment of SpondyloArthritis international Society Health Index; ASDAS: Ankylosing Spondylitis Disease Activity Score; axSpA: axial spondyloarthritis; b/tsDMARD: biological/targeted-synthetic disease modifying anti-rheumatic drug; CfB: change from baseline; HRQoL: health-related quality of life; OC: observed case; PCS: Physical Component Summary; PGADA: Patient Global Assessment of Disease Activity; SD: standard deviation; SF-36: 36-Item Short Form Survey; VAS: visual analogue score.

AEs were reported in 62.9%; 33.0% drug-related. Three uveitis cases in three patients (1.5%), two with prior uveitis history. Sixteen *Candida* infections in fifteen patients (7.6%); twelve oral. Eight (4.1%) reported serious infections. One (0.5%) reported IBD; one (0.5%) reported MACE (myocardial infarction; not drug-related). No cases of venous thromboembolism/serious hypersensitivity/anaphylaxis/suicidal ideation or behaviour.
Conclusion. Patients with axSpA commencing bimekizumab showed early and sustained PRO improvements to Wk24. Improvements were observed across all prior treatments, greater in first-line bimekizumab patients. These data support the effectiveness and safety of bimekizumab in patients with axSpA in real-world clinical practice, providing early evidence that bimekizumab treatment benefits in clinical trials may extend to routine practice.
Acknowledgements. Funded by UCB. Medical writing and editorial support provided by Costello Medical and funded by UCB.
Disclosures. XB: Speakers bureau for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, J&J, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; paid instructor for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, J&J, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; consultant for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, J&J, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; grant/research support from AbbVie, Celltrion, Janssen, MoonLake and Novartis; **UK:** Grant and research support and consultancy fees from AbbVie, Amgen, Biocad, Biogen, BMS, Chugai, Eli Lilly, Fresenius, GBA, Gilead, Grünenthal, GSK, Hexal, Janssen, MSD, Novartis, onkowissen.de, Pfizer, Roche, UCB and Viatris; **KdV:** Speakers bureau for AbbVie, Alfasigma, Amgen, BMS, Galapagos, J&J, Lilly, Novartis and UCB; consulting for AbbVie, Alfasigma, Amgen, BMS, Galapagos, J&J, Lilly, MoonLake, Novartis, Roche and Sandoz; **ARW:** Speaker fees from AbbVie, Amgen, Biogen, Biocon, BMS, Fresenius-Kabi, J&J, Lilly, Novartis, Nordic-Pharma, Pfizer, Chugai, UCB and Viatris; consultant for AbbVie, Celltrion, J&J, Lilly, Novartis, Nordic Pharma, Pfizer, Sandoz and UCB; **JRG:** Speaker for AbbVie, Eli Lilly, Johnson and Johnson, Novartis and UCB; consultant for AbbVie, Johnson and Johnson, Novartis and UCB; grant/research support from Novartis, Johnson and Johnson and Pfizer; **JAW:** Consultant for/grant support from AbbVie, Amgen, Eli Lilly, Janssen, Merck, Novartis, Pfizer, Spyre and UCB; **DP:** Speaker for AbbVie, BMS, Eli Lilly, MSD, Novartis, Pfizer and UCB; consultant for AbbVie, Biocad, Eli Lilly,

Gilead, GSK, MSD, MoonLake, Novartis, Pfizer, Samsung Bioepis and UCB; grant/research support from AbbVie, Eli Lilly, MSD, Novartis and Pfizer; **DN:** Research support from AbbVie, BMS, Eli Lilly, Gilead, Incannex, Janssen, Novartis, Pfizer, Servatus, Sun Pharma and UCB; advisory board or speaker fees from AbbVie, AstraZeneca, Janssen, Novartis, Pfizer, Sandoz and UCB; **MO, FC, MAB, RB, FG:** Employees and stockholders of UCB; **MK, PO:** Employees of UCB; **CdlL:** Consultant to UCB; **WT:** Research grants, consulting fees, speaking fees, and/or honoraria from AbbVie, Amgen, BMS, Celgene, Eli Lilly and Company, GSK, Janssen, MSD, Novartis, Ono Pharma, Pfizer, Takeda and UCB.

P138

BIMEKIZUMAB DEMONSTRATED REMISSION AND LOW DISEASE ACTIVITY TO THREE YEARS IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS: EXPLORING DIFFERENT DEFINITIONS OF REMISSION AND LOW DISEASE ACTIVITY USING RESULTS FROM TWO PHASE 3 STUDIES

Ramiro S.[1,2], Gossec L.[3], Nikiphorou E.[4,5,6], Proft F.[7], Dubreuil M.[8], Elmoufti S.[9], de la Loge C.[10], Ganz F.[11], de Peyrecave N.[10], Marten A.[12], Boonen A.[13]

[1]Dept. of Rheumatology, Leiden University Medical Center, Leiden; [2]Zuyderland Medical Center, Heerlen, The Netherlands; [3]Sorbonne Universite and Pitié-Salpêtrière Hospital, Paris, France; [4]Centre for Rheumatic Diseases, Faculty of Life Sciences and Medicine, King's College London, London; [5]Rheumatology Dept., King's College Hospital NHS Foundation Trust, London; [6]Centre for Education, Faculty of Life Sciences and Medicine, King's College London, London, UK; [7]Dept. of Gastroenterology, Infectiology and Rheumatology (including Nutrition Medicine), Charité – Universitätsmedizin Berlin, corporate member of Freie Universität Berlin and Humboldt-Universität zu Berlin, Berlin, Germany; [8]Dept. of Rheumatology, Boston University School of Medicine, Boston; [9]UCB, Morrisville, USA; [10]UCB, Brussels, Belgium; [11]UCB, Bulle, Switzerland; [12]UCB, Monheim am Rhein, Germany; [13]Dept. of Rheumatology, Maastricht University Medical Center; Care and Public Health Research Institute (Caphri), Maastricht University, Maastricht, The Netherlands

Introduction/Objective. Multiple definitions of remission and low disease activity (LDA) exist in axial spondyloarthritis (axSpA), capturing different components and stringencies of disease control. We explored how remission and LDA rates over three years vary by definition in bimekizumab-treated patients with non-radiographic (nr-) and radiographic (r-) axSpA.

Methods. Patients randomised in BE MOBILE 1 (nr-axSpA; NCT03928704) and 2 (r-axSpA; NCT03928743) received bimekizumab or placebo; all received subcutaneous bimekizumab 160 mg every 4 weeks from Week (Wk)16. Eligible patients completing Wk52 could enter the open-label extension (NCT04436640).

We explored validated and candidate definitions of remission (clinical and MRI) and LDA (see Table I for a complete list of definitions). We report remission and LDA rates with bimekizumab treatment to Wk164 (observed case).

Results. Overall, 586 patients were enrolled in BE MOBILE 1 (n=254) and 2 (n=332).

Remission/LDA rates generally increased from Wk16 to Wk164 across all definitions, varying based on definition stringency (Table I, Fig. 1). Wk164 clinical remission rates ranged from 35.6% (ASDAS inactive disease [ID; <1.3]; most stringent core definition) to 48.6% (BASDAI <2; least stringent). High MRI remission rates were observed at Wk164 (SPARCC SIJ <2: 59.4% [19/32]; Berlin spine ≤2: 77.8% [28/36]), though these were assessed in small, pre-specified sub-study populations. Wk164 LDA rates were 65.1% (ASDAS <2.1 [ID + LDA]) and 75.4% (BASDAI <4). Remission/LDA rates were numerically lower when combined with swollen joint count =0.

Conclusion. Bimekizumab-treated patients with axSpA achieved remission and LDA across multiple definitions by Wk16, with response rates generally increasing to Wk164. Remission/LDA rates were definition-dependent; ASDAS ID yielded the most stringent remission estimate.

This analysis is the first to examine these definitions and helps contextualise how definition choice influences interpretation of disease control in axSpA clinical settings. Assessing predictive validity of these definitions is required to better inform definition selection.

Acknowledgements. Funded by UCB. Medical writing support provided by Costello Medical and funded by UCB.

Disclosures. SR: Consultant for AbbVie, Alfasigma, Eli Lilly, Johnson & Johnson, MSD, Novartis, Pfizer, Sanofi, Takeda and UCB; grants from AbbVie, Alfasigma, Eli Lilly, MSD, Novartis, Pfizer and UCB. **LG:** Research grants from AbbVie, Lilly, Novartis and UCB; consulting fees from AbbVie, AlfaSigma, Amgen, BMS, Celltrion, J&J, Lilly, Moonlake, Novartis, Nordic Pharma, Oruka, Pfizer, Stada, Takeda and UCB; non-financial support from AbbVie, Amgen, Biogen, BMS, Celltrion, J&J, Lilly, Pfizer and UCB. **EN:** Speaker honoraria from/participated in advisory boards for AbbVie, Alfasigma, Eli Lilly, Fresenius, Gilead, Galapagos, Novartis, Pfizer and UCB; research grants from Eli Lilly and Pfizer. **FP:** Grant/research support from Eli Lilly and Company, Novartis and UCB; consultancy fees and speakers bureau from AbbVie, Amgen, BMS, Celgene, Eli Lilly and Company, Galapagos, Hexal, Janssen, Medscape, Moonlake Pharma, MSD, Novartis, Pfizer, Roche and UCB. **MD:** Educational Grant from Pfizer paid to institution; consulting fees (*e.g.* advisory boards) from Amgen, Hillstar Bio and UCB; funding from Rheumatology Research Foundation, Boston University School of Medicine, Dept. of Medicine and Boston Medical Center. **SE, FG, AM:** Employee and shareholder of UCB. **CdlL:** Consultant to UCB. **NdP:** Employee of UCB. **AB:** Research grants from AbbVie and Eli Lilly; speaker honoraria and consultation fees from AbbVie, Galapagos, Pfizer, Novartis and UCB.

Table I. Achievement of remission or LDA across different definitions to Week 164 (OC).

Definitions	Patients achieving remission or LDA (% [n/N])			
	Week 16	Week 52	Week 104	Week 164
Remission core definition				
ASDAS ID (<1.3)	12.7 (71/557)	29.1 (145/499)	35.8 (165/461)	35.6 (148/416)
ASAS partial remission	18.3 (103/564)	35.6 (183/514)	40.6 (189/466)	43.0 (181/421)
BASDAI <2	20.9 (118/564)	37.5 (193/515)	47.9 (223/466)	48.6 (205/422)
BASDAI <2 with CRP normalisation[a]	15.1 (84/557)	31.7 (158/499)	40.8 (188/461)	39.2 (163/416)
MRI SPARCC SIJ <2[b]	–	53.1 (17/32)	53.1 (17/32)	59.4 (19/32)
MRI Berlin spine ≤2[c]	–	77.8 (28/36)	72.2 (26/36)	77.8 (28/36)
Remission combined with SJC=0[d]				
ASDAS ID (<1.3) + SJC=0	5.4 (8/147)	14.4 (19/132)	24.8 (30/121)	25.0 (27/108)
ASAS partial remission + SJC=0	6.7 (10/149)	19.5 (26/133)	30.1 (37/123)	36.1 (39/108)
BASDAI <2 + SJC=0	8.7 (13/149)	20.3 (27/133)	39.0 (48/123)	37.0 (40/108)
BASDAI <2 with CRP normalisation + SJC=0[a]	6.8 (10/147)	16.7 (22/132)	33.1 (40/121)	27.8 (30/108)
LDA core definition				
ASDAS <2.1[e]	35.2 (196/557)	61.7 (308/499)	66.6 (307/461)	65.1 (271/416)
BASDAI <4	48.9 (276/564)	71.1 (366/515)	76.6 (357/466)	75.4 (318/422)
LDA combined with SJC=0[d]				
ASDAS <2.1 + SJC=0[e]	17.7 (26/147)	43.2 (57/132)	57.0 (69/121)	55.6 (60/108)
BASDAI <4 + SJC=0	26.2 (39/149)	48.1 (64/133)	65.9 (81/123)	65.7 (71/108)

Pooled randomised set. Data reported for patients with non-missing measurements at the given week, with percentages calculated accordingly. Week 16 MRI remission outcomes were reviewed in a different reading session from Week 52, Week 104 and Week 164 outcomes, and are therefore not reported in this analysis.

[a] CRP normalisation defined as CRP <5 mg/L.

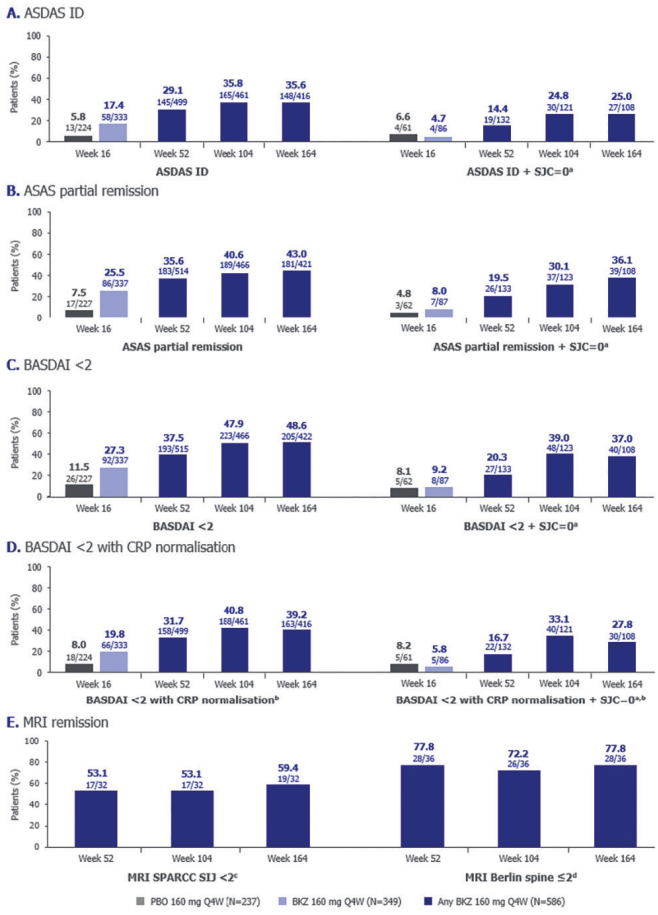
[b] In patients enrolled in the MRI sub-study in BE MOBILE 1 with MRI SPARCC SIJ score ≥2 at baseline and non-missing data at baseline, Week 52, Week 104 and Week 164 (nr-axSpA only; n=32).

[c] In patients enrolled in the MRI sub-study in BE MOBILE 2 with MRI Berlin spine score >2 at baseline and non-missing data at baseline, Week 52, Week 104 and Week 164 (r-axSpA only; n=36).

[d] In patients with peripheral arthritis (SJC >0) at baseline (n=154).

[e] Includes patients who achieved ASDAS ID.

ASAS: Assessment of SpondyloArthritis international Society; ASDAS: Axial Spondyloarthritis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; CRP: C-reactive protein; ID: inactive disease; LDA: low disease activity; MRI: magnetic resonance imaging; OC: observed case; SIJ: sacroiliac joint; SJC: swollen joint count; SPARCC: Spondyloarthritis Research Consortium of Canada.



P138 - Fig. 1. Achievement of remission based on different definitions to week 164 (OC). Pooled randomised set. 'Any BKZ' includes patients who were randomised to placebo and switched to bimekizumab at Week 16. Data reported for patients with non-missing measurements at the given week, with percentages calculated accordingly. Week 16 MRI remission outcomes were reviewed in a different reading session from Week 52, Week 104 and Week 164 outcomes, and are therefore not reported in this analysis.
[a] In patients with peripheral arthritis (SJC >0) at baseline (n=154).
[b] CRP normalisation defined as CRP <5 mg/L.
[c] In patients enrolled in the MRI sub-study in BE MOBILE 1 with MRI SPARCC SIJ score ≥2 at baseline and non-missing data at baseline, Week 52, Week 104 and Week 164 (nr-axSpA only; n=32).
[d] In patients enrolled in the MRI sub-study in BE MOBILE 2 with MRI Berlin spine score >2 at baseline and non-missing data at baseline, Week 52, Week 104 and Week 164 (r-axSpA only; n=36).
ASAS: Assessment of SpondyloArthritis international Society; ASDAS: Axial Spondyloarthritis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BKZ: bimekizumab; CRP: C-reactive protein; ID: inactive disease; MRI: magnetic resonance imaging; OC: observed case; PBO: placebo; Q4W: every 4 weeks; SIJ: sacroiliac joint; SJC: swollen joint count; SPARCC: Spondyloarthritis Research Consortium of Canada.

P139

IMPACT OF BIMEKIZUMAB ON MRI INFLAMMATORY AND STRUCTURAL LESIONS IN THE SACROILIAC JOINTS OF PATIENTS WITH NON-RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS: 2-YEAR RESULTS FROM A PHASE 3 STUDY AND ITS OPEN-LABEL EXTENSION

Maksymowych W.P.[1], Ramiro S.[2], Poddubnyy D.[3,4,5], Gensler L.S.[6], Lambert R.G.W.[7], Prajapati C.[8], Marten A.[9], Østergaard M.[10,11], Baraliakos X.[12]
[1]Dept. of Medicine, University of Alberta, Edmonton, Canada; [2]Dept. of Rheumatology, Leiden University Medical Center, Leiden and Zuyderland Medical Center, Heerlen, The Netherlands; [3]Division of Rheumatology, Dept. of Medicine, University of Toronto, Toronto; [4]University Health Network, Schroeder Arthritis Institute, Toronto, Canada; [5]Dept. of Gastroenterology, Infectious Diseases and Rheumatology, Charité – Universitätsmedizin Berlin, Berlin, Germany; [6]Dept. of Medicine/Rheumatology, University of California, San Francisco, USA; [7]Dept. of Radiology & Diagnostic Imaging, University of Alberta, Edmonton, Canada; [8]UCB, Slough, UK; [9]UCB, Monheim am Rhein, Germany; [10]Dept. of Clinical Medicine, University of Copenhagen, Copenhagen; [11]Copenhagen Center for Arthritis Research, Center for Rheumatology and Spine Diseases, Rigshospitalet, Glostrup, Denmark; [12]Rheumazentrum Ruhrgebiet Herne, Ruhr-University Bochum, Germany

Introduction/Objective. Bimekizumab (BKZ), a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, was efficacious and well tolerated over 3 years (yrs) in the phase 3 studies, BE MOBILE (BM)1 (non-radiographic [nr-]axSpA), BM2 (radiographic axSpA) and their combined open-label extension (OLE).
We evaluated MRI SIJ inflammatory and structural lesions in patients in BM1 and the OLE with 2yrs of BKZ treatment.
Methods. BM1 (NCT03928704) comprised 16-wk double-blind and 36-wk maintenance periods. Patients were randomised to BKZ 160mg every 4wks or PBO; all received BKZ from Wk16. After Wk52, patients could enter

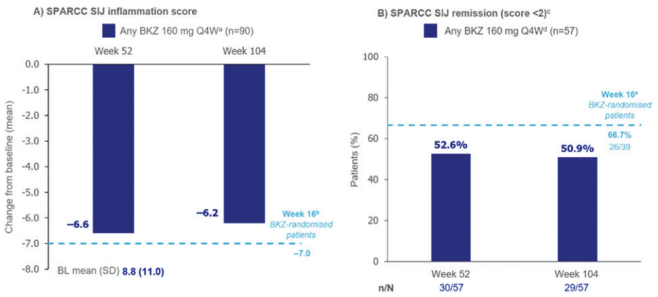


Fig. 1. Change from baseline in MRI SPARCC SIJ inflammation scores and achievement of SPARCC SIJ remission at Week 52 and Week 104 (OC).
Includes patients in the MRI sub-study of BE MOBILE 1 with SPARCC SIJ assessments available at baseline, Week 52 and Week 104.
[a] Includes patients originally randomised to placebo who switched to BKZ at Week 16.
[b] Previously published BE MOBILE 1 Week 16 data, scored at Week 52; includes BKZ-randomised patients with SPARCC SIJ assessments available at baseline, Week 16 and Week 52 (n=64).
[c] MRI remission was assessed in patients with SPARCC SIJ ≥2 at baseline.
[d] Includes patients originally randomised to placebo who switched to BKZ at Week 16 and had SPARCC SIJ ≥2 at baseline.
[e] Previously published BE MOBILE 1 Week 16 data, scored at Week 52; includes BKZ-randomised patients with SPARCC SIJ assessments available at baseline, Week 16, and Week 52, with SPARCC SIJ inflammation score ≥2 at baseline.
BKZ: bimekizumab; BL: baseline; Q4W: every 4 weeks; MRI: magnetic resonance imaging; OC: observed case; SIJ: sacroiliac joint; SD: standard deviation; SPARCC: Spondyloarthritis Research Consortium of Canada.

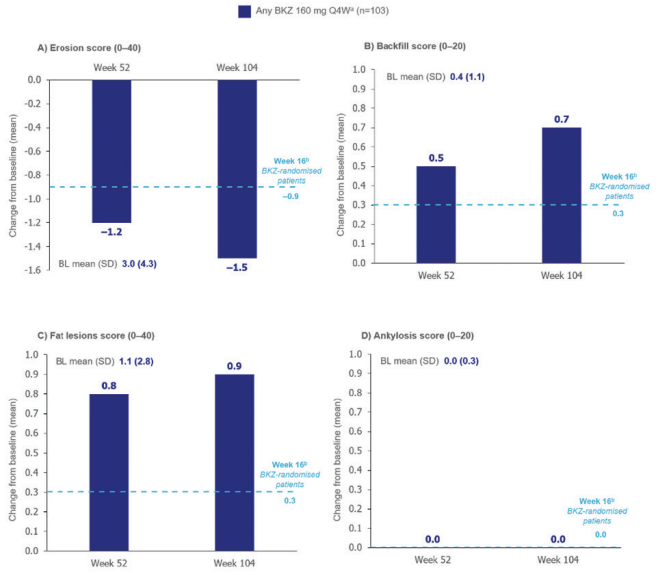


Fig. 2. Change from baseline in MRI SPARCC SSS components at Week 52 and Week 104 (OC).
Includes patients in the MRI sub-study of BE MOBILE 1 with SPARCC SSS assessments available at baseline, Week 52 and Week 104.
[a] Includes patients originally randomised to placebo who switched to BKZ at Week 16.
[b] Previously published BE MOBILE 1 Week 16 data, scored at Week 52; includes BKZ-randomised patients with SPARCC SSS assessments available at baseline, Week 16, and Week 52 (n=69). BKZ: bimekizumab; BL: baseline; MRI: magnetic resonance imaging; OC: observed case; Q4W: every 4 weeks; SD: standard deviation; SIJ: sacroiliac joint; SPARCC SSS: Spondyloarthritis Research Consortium of Canada SIJ Structural Score.

the OLE to continue BKZ for 112wks. Assessments included patients in the MRI sub-study with evaluable MRIs at baseline, Wk52 and Wk104.
At Wk52 and Wk104, we report mean change from baseline (CfB) in SPARCC SIJ inflammation score, proportions achieving MRI remission (SPARCC SIJ <2) in those with baseline SPARCC SIJ ≥2, and mean CfB in SPARCC SIJ Structural Scores (SSS; erosion, backfill, fat lesions, ankylosis).
Results. The BM1 MRI sub-study included 152/254 (59.8%) patients. 90/152 (59.2%) had SPARCC SIJ and 103/152 (67.8%) had SPARCC SSS assessments at all three timepoints.
SPARCC SIJ scores reduced substantially from baseline: Wk52 (CfB: –6.6)/ Wk104 (CfB: –6.2; Fig. 1). In patients with baseline SPARCC SIJ score ≥2, 30/57 (52.6%) at Wk52 and 29/57 (50.9%) at Wk104 achieved MRI remission. Erosions reduced from baseline at Wk52 (CfB: –1.2) and Wk104 (CfB: –1.5; Fig. 2). Backfill and fat lesions increased from baseline at Wk52 (backfill CfB: 0.5; fat lesions CfB: 0.8) and Wk104 (backfill CfB: 0.7; fat lesions CfB: 0.9). No ankylosis was observed.
Conclusion. In patients with nr-axSpA, treatment with BKZ led to substantial reductions in MRI SIJ inflammation at Wk52 and Wk104, indicating sustained control of inflammation and potential tissue repair with dual IL-17A and IL-17F inhibition.
Acknowledgements. Funded by UCB. Medical writing and editorial support provided by Costello Medical and funded by UCB.
Disclosures. WPM: Honoraria/consulting fees from AbbVie, BMS, Boehringer-Ingelheim, Celgene, Eli Lilly, Galapagos, Janssen, Novartis, Pfizer and UCB; research grants from AbbVie, BMS, Eli Lilly, Galapagos, Pfizer and UCB; educational grants from AbbVie, Novartis and Pfizer; Chief Medical Officer for CARE ARTHRITIS; **SR:** Consultant for AbbVie, Alfasigma, Eli Lilly, Johnson & Johnson, MSD, Novartis, Pfizer, Sanofi, Takeda and UCB; grants from AbbVie, Alfasigma, Eli Lilly, MSD, Novartis, Pfizer and UCB; **DP:** Speaker for AbbVie, BMS, Eli Lilly, MSD, Novartis, Pfizer and UCB; consultant for AbbVie, Biocad, Eli Lilly, Gilead, GSK, MSD, Moonlake, Novartis, Pfizer, Samsung Bioepis and UCB; grant/research support from: AbbVie, Lilly, MSD, Novartis and Pfizer; **LSG:** Grants from UCB paid to institution; consulting fees from Acelyrin, Eli Lilly, Janssen, Novartis, Pfizer and UCB; **RGWL:** Consultant for AbbVie, CARE Arthritis and Image Analysis Group; **CP:** Contractor for UCB and employee of Veramed; **AM:** Employee and shareholder of UCB; **MØ:** Research grants from AbbVie, Amgen, BMS, Celgene, Eli Lilly, Merck, Novartis and UCB; speaker fees from AbbVie, BMS, Celgene, Eli Lilly, Galapagos, Gilead, Janssen, MEDAC, Merck, Novartis, Pfizer, Sandoz and UCB; consultancy fees from AbbVie, BMS, Celgene, Eli Lilly, Galapagos, Gilead, Janssen, Merck, Novartis, Pfizer and UCB; **XB:** Speakers bureau for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, J&J, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; paid instructor for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, J&J, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; consultant for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, J&J, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; grant/research support from AbbVie, Celltrion, Janssen, MoonLake and Novartis.

P140

BIMEKIZUMAB DEMONSTRATED SUSTAINED IMPROVEMENTS IN PAIN AND FATIGUE FOLLOWING EARLY CONTROL OF INFLAMMATION IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS OVER 3 YEARS: RESULTS FROM 2 PHASE 3 STUDIES AND THEIR OPEN-LABEL EXTENSION

Navarro-Compán V.[1], Ramiro S.[2,3], Kiltz U.[4], Dubreuil M.[5], Poddubnyy D.[6,7,8], Magrey M.[9], Deodhar A.[10], Elmoufti S.[11], de la Loge C.[12], Baraliakos X.[4]
[1]Dept. of Rheumatology, La Paz University Hospital, IdiPaz, Madrid, Spain; [2]Dept. of Rheumatology, Leiden University Medical Center, Leiden, The Netherlands; [3]Zuyderland Medical Center, Heerlen, The Netherlands; [4]Rheumazentrum Ruhrgebiet Herne, Ruhr-University Bochum, Germany; [5]Section of Rheumatology, Boston University School of Medicine, Boston, USA; [6]University of Toronto, Division of Rheumatology, Dept. of Medicine, Toronto; [7]University Health Network, Schroeder Arthritis Institute, Toronto, Canada; [8]Charité – Universitätsmedizin Berlin, Dept. of Gastroenterology, Infectious Diseases and Rheumatology, Berlin, Germany; [9]Case Western Reserve University, University Hospitals, Cleveland; [10]Division of Arthritis and Rheumatic Diseases, Oregon Health & Science University, Portland; [11]UCB, Morrisville, USA; [12]UCB, Brussels, Belgium

Introduction/Objective. Bimekizumab (BKZ), a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F/IL-17A, demonstrated sustained efficacy to 3 years in patients with non-radiographic (nr-)axSpA and radiographic (r-)axSpA.
We report whether reduction in active inflammation at Wk16, as measured by ASDAS or MRI, is associated with sustained, long-term improvements in pain and fatigue over 3 years.
Methods. BE MOBILE 1 (nr-axSpA) and 2 (r-axSpA) comprised a 16-wk double-blind, placebo-controlled and 36-wk maintenance period. From Wk16, all received subcutaneous BKZ 160mg Q4W. At Wk52, eligible patients could enrol in the OLE.
Pain/fatigue were measured by total spinal pain (TSP)/BASDAI Q1, respectively. Both used a 0–10 point rating scale with higher scores reflecting greater pain/fatigue. Achievement of mild pain (TSP <4) or fatigue (BASDAI Q1 <4) are presented to Wk164 for BKZ-randomised patients who achieved ASDAS <2.1 or MRI remission (SPARCC SIJ <2/Berlin spine ≤2 in the MRI sub-studies) at Wk16.

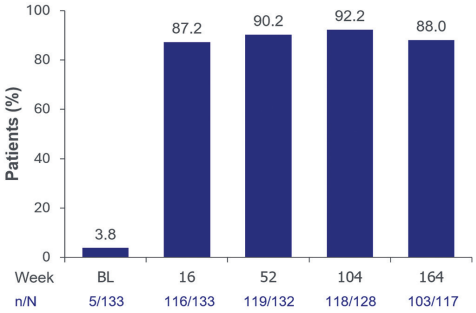


Fig. 1. Achievement of total spinal pain <4 (mild pain) to Week 164 in BKZ-randomised patients who achieved ASDAS <2.1 at Week 16 (OC).
Only patients randomised to BKZ and entering the OLE were included in the analysis. All data are reported as observed case. Data are presented as pooled across BE MOBILE 1 and BE MOBILE 2. At Week 16, 133/293 (45.4%) patients achieved ASDAS <2.1. ASDAS: Axial Spondyloarthritis Disease Activity Score; BKZ: bimekizumab; BL: baseline; OC: observed case; OLE: open-label extension; TSP: total spinal pain.

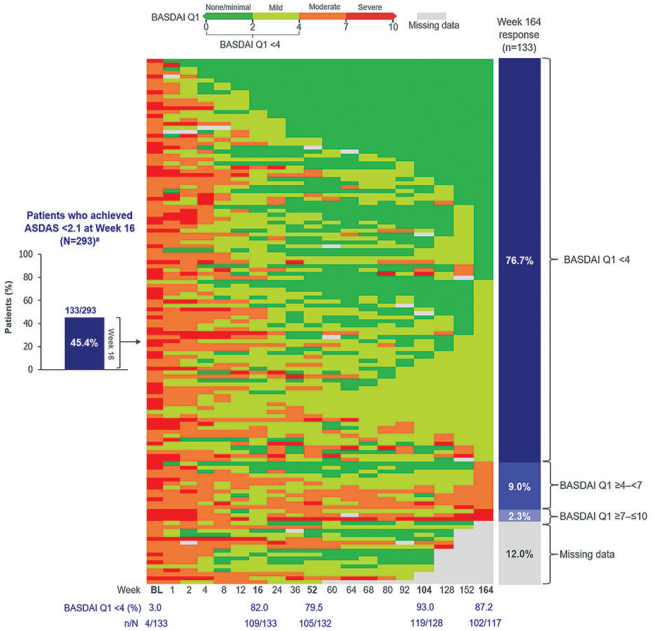


Fig. 2. BASDAI Q1 (fatigue) score over time for BKZ-randomised patients who achieved ASDAS <2.1 at Week 16.
Only patients randomised to BKZ and entering the OLE were included in the analysis. Data are presented as pooled across BE MOBILE 1 and BE MOBILE 2. n/N data are presented as observed case. BASDAI Q1 scores (one row per patient) are presented as observed at each timepoint (one column per assessed visit). [a] At Week 16, 133/293 (45.4%) patients achieved ASDAS <2.1. ASDAS: Axial Spondyloarthritis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BKZ: bimekizumab; BL: baseline; OLE: open-label extension.

Results. In total, 349 patients were randomised to BKZ (nr-axSpA:128; r-axSpA: 221). Of 293 who entered the OLE, 133 (45.4%) achieved ASDAS <2.1 at Wk16. Most who achieved ASDAS <2.1 at Wk16 achieved mild pain (TSP <4; Fig. 1) and mild fatigue (BASDAI Q1 <4; Fig. 2) to Wk164. For nr-axSpA, 65.0% patients achieved SPARCC SIJ <2 at Wk16, of those with MRI remission status available at Wk16 (n=40); ≥50% had no/mild pain/fatigue at Wks16/52/104/164 (TSP <4: 50.0%/72.0%/70.8%/76.2%; BASDAI Q1 <4: 57.7%/69.2%/70.8%/61.9%).
For r-axSpA, 77.8% patients achieved MRI Berlin spine ≤2 at Wk16, of those with MRI remission status available at Wk16 (n=27); 38.1% and 42.9% of these achieved no/mild pain and fatigue, respectively, at Wk16, which increased at Wks52/104/164 (TSP <4: 61.9%/57.1%/52.4%; BASDAI Q1 <4: 47.6%/61.9%/57.1%).
Conclusion. Improvements in pain/fatigue were observed with BKZ through Wk164 in patients with axSpA who achieved control of inflammation at Wk16.
Acknowledgements. Funded by UCB. Medical writing and editorial support provided by Costello Medical and funded by UCB.
Disclosures. VNC: Speakers bureau for AbbVie, Alfasigma, Eli Lilly and Company, Fresenius Kabi, Janssen, MSD, Novartis, Pfizer and UCB; consultant for AbbVie, Alfasigma, Eli Lilly and Company, Galapagos, MoonLake, MSD, Novartis, Pfizer and UCB; grant/research support from AbbVie and Novartis; **SR:** Consultant for AbbVie, Alfasigma, Eli Lilly, Johnson & Johnson, MSD, Novartis, Pfizer, Sanofi, Takeda and UCB; grants from AbbVie, Alfasigma, Eli Lilly, MSD, Novartis, Pfizer and UCB; **UK:** Grant and research support and consultancy fees from AbbVie, Amgen, Biocad, Biogen, BMS, Chugai, Eli Lilly, Fresenius, GBA, Gilead, Grünenthal, GSK, Hexal, Janssen, MSD, Novartis, onkowissen.de, Pfizer, Roche, UCB and Viatris; **MD:** Educational grant from Pfizer paid to institution; consulting fees (e.g. advisory boards) from Amgen and UCB; funding from Rheumatology Research Foundation, Boston University School of Medicine, Dept. of Medicine and Boston Medical Center; **DP:** Speaker for AbbVie, BMS, Eli Lilly, MSD, Novartis, Pfizer and UCB; consultant for AbbVie, Biocad, Eli Lilly, Gilead, GSK, MSD, Moonlake, Novartis, Pfizer, Samsung Bioepis and UCB; grant/research support from: AbbVie, Lilly, MSD, Novartis and Pfizer; **MM:** Consultancy fees from AbbVie, BMS, Eli Lilly, Novartis, Pfizer and UCB; research grants from AbbVie, BMS and UCB; **AD:** Speaker for Eli Lilly, J&J, Novartis, Pfizer and UCB; consultant for BMS, Eli Lilly, J&J, MoonLake, Novartis, Pfizer and UCB; grant/research support from BMS, Eli Lilly, J&J, Novartis, Pfizer and UCB; **SE:** Employee and shareholder of UCB; **CdlL:** Consultant to UCB; **XB:** Speakers bureau for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, J&J, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; paid instructor for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, J&J, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; consultant for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, J&J, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; grant/research support from AbbVie, Celltrion, Janssen, MoonLake and Novartis.

P141

SECUKINUMAB AND IXEKIZUMAB IN SPONDYLOARTHRITIS: REAL-WORLD RETENTION DATA AT 24 MONTHS

Moreno Morales J.[1], Gonzalez Ponce C.M.[2]
[1]Servicio de Reumatología, Hospital Universitario Virgen de La Arrixaca, Murcia; [2]Servicio de Farmacia, Hospital Universitario Virgen de La Arrixaca, Murcia, Spain

Introduction. The objective of this study was to determine the retention rate of two interleukin-17A (IL-17A) inhibitors (secukinumab and ixekizumab) in a cohort of patients with spondyloarthritis. We also sought to identify predictive factors for discontinuation.
Methods. A retrospective observational study was conducted on a cohort of 137 patients. Baseline demographic and clinical variables were analysed. Drug survival was estimated using Kaplan-Meier curves, and comparisons were performed using the log-rank test. A Cox proportional hazards regression model was used to identify independent predictors of treatment discontinuation (age, sex, diagnosis, and line of treatment).
Results. The overall mean age was 54.6 years. Treatment distribution was 77.4% for secukinumab and 22.6% for ixekizumab. In the predominant psoriatic arthritis (PsA) subgroup, the proportion of females was 58.7% for secukinumab and 40.7% for ixekizumab. Heterogeneity in prior biologic exposure was observed. In the secukinumab group, 30.7% of PsA patients were on their third or subsequent line of treatment, compared to 48.1% in the ixekizumab group. Overall retention rates were: Secukinumab at 12 months: 86.4% (95% CI: 79.5–93.3), 24 months: 76.2% (95% CI: 67.3–85.2). Ixekizumab at 12 months: 79.4% (95% CI: 63.3–95.5), 24 months: 75.0% (95% CI: 57.6–92.4). Diagnostic subgroups showed overlapping survival curves. The adjusted Cox regression model did not identify statistically significant independent predictors (p>0.05) for treatment discontinuation. There were no significant differences between secukinumab and ixekizumab (Fig. I).

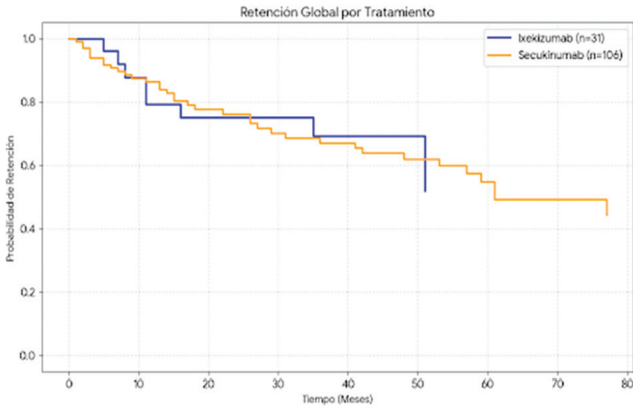


Fig. 1.

Conclusion. In this real-world cohort, secukinumab and ixekizumab demonstrated high retention rates at 12 and 24 months. Treatment persistence was robust regardless of age, sex, diagnostic subgroup, or line of treatment. These data suggest that both IL-17A inhibitors are sustainable medium- and long-term therapeutic options in the management of spondyloarthritis.

P142

BASELINE CLINICAL PROFILE OF PATIENTS WITH PSORIATIC ARTHRITIS WITH AXIAL INVOLVEMENT TREATED WITH SECUKINUMAB IN REAL CLINICAL PRACTICE IN SPAIN

Moreno Martínez M.J.[1], León Torralba P.[2], Arévalo Ruales K.[3], Aguilar M.[3], Eyre-Olmos C.[4], Moreno M.[4], Almodóvar R.[5], Fernández Prada M.[6], de la Morena I.[7], Castillo Dayer P.V.[8], Castillo A.C.[17], de la Torre N.[9], Marín J.[10], Villaverde V.[11], Senabre Gallego J.M.[12], González Ferrández J.A.[13], Candelas G.[14], Urruticoechea Arana A.[15], Martínez-Prada C.[14], Montolio Chiva L.[16], Macia-Villa C.[17], Moreno Ramos M.J.[18]
[1]Hospital General Universitario Rafael Méndez de Lorca, Murcia; [2]Hospital Universitario Doctor Peset, Valencia; [3]Hospital Universitario de Sagunto, Sagunto; [4]Hospital Universitario Parc Taulí, Sabadell; [5]Hospital Universitario Fundación Alcorcón, Alcorcón; [6]Hospital Universitario de Guadalajara, Guadalajara; [7]Hospital Clínico Universitario de Valencia, Valencia; [8]Hospital General Universitario Santa Lucía, Cartagena; [9]Hospital Universitario Puerta de Hierro, Majadahonda; [10]Hospital Universitario Rey Juan Carlos, Móstoles; [11]Hospital Universitario de Móstoles, Móstoles; [12]Hospital Marina Baixa, Villajoyosa; [13]Hospital General Universitario de Elche, Elche; [14]Hospital Clínico San Carlos, Madrid; [15]Hospital Universitario Son Espases, Palma de Mallorca; [16]Hospital General Universitario de Castellón, Castellón; [17]Hospital Ramón y Cajal, Madrid; [18]Hospital Clínico Universitario Virgen de la Arrixaca, El Palmar, Murcia, Spain

Introduction. Psoriatic arthritis (PsA) is a heterogeneous chronic inflammatory disease that can affect both the peripheral and axial skeleton. Axial involvement (axPsA) is associated with a higher inflammatory burden, poorer quality of life, and greater functional disability. Secukinumab, an anti-IL-17A monoclonal antibody, is the only drug that has demonstrated efficacy in PsA and axial manifestations in the MAXIMISE clinical trial; however, real-world data from Spain remain limited.
Objective. To evaluate treatment persistence with secukinumab in patients

Table I. Baseline disease characteristics.

Variable	n	Mean ± SD	%
Duration of symptoms (years)	94	7 ± 5.5	
CRP (mg/dL)	95	2.96 ± 9	
BMI	79	28.4 ± 5.9	
Tender joint count	84	4 ± 5	
Swollen joint count	83	2 ± 3	
Nocturnal low back pain (VAS)	66	5.3 ± 3	
Patient global VAS (0–10)	74	6.3 ± 2	
Pain VAS (0–10)	70	6.3 ± 2	
BASDAI	52	5 ± 2	
DAPSA	92	17 ± 15	
ASDAS	91	2.4 ± 1.2	
Enthesitis	92		39
Dactylitis	93		17

SD: standard deviation; CRP: C-reactive protein; BMI: body mass index; VAS: visual analogue scale; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; DAPSA: Disease Activity in PSoriatic Arthritis; ASDAS: Ankylosing Spondylitis Disease Activity Score.

Table II. Radiological findings. MRI: magnetic resonance imaging.

Variable	n	%
Syndesmophytes	78	10.2
Sacroiliac joint involvement	81	75.3
MRI involvement	62	56.4

Table III. Extra-articular manifestations and comorbidities.

Variable	n	%
Uveitis	92	3.2
IBD	83	2.4
Arterial hypertension	94	30.8
Dyslipidaemia	93	35.4
Diabetes	93	15
Hepatic steatosis	91	15
Depression	92	22.8
Prior malignancies	93	5.3
MACEs	82	1.2

IBD: inflammatory bowel disease; MACEs: major adverse cardiovascular events.

with PsA and axial involvement in real clinical practice at 12 months, and to analyse clinical effectiveness, safety, and reasons for discontinuation. We present a baseline analysis of the enrolled patients.

Materials and methods. Longitudinal, ambispective, observational, multicentre study conducted in Spanish hospitals. Patients aged ≥18 years with a clinical diagnosis of PsA and axial manifestations who received at least one dose of secukinumab from January 2015 onwards were included. Demographic, clinical, laboratory, radiological, disease activity, persistence, and safety data were collected at baseline and follow-up visits according to standard clinical practice.

Statistical plan: A descriptive analysis of baseline patient variables was performed, reporting mean and standard deviation for quantitative variables, and percentages for qualitative variables.

Results. Baseline data from 106 patients (56% male) were analysed. Of these, 78% had mixed (axial and peripheral) disease involvement, with a mean age of 51.2 ± 10.1 years. Active psoriasis was present in 63% of patients, with nail involvement in 26%. Table I shows the remaining disease characteristics.

Regarding the number of prior biologics (n=73):

- 25% of patients were biologic-naive.
- 41% had received 1 prior biologic.
- 29% had received 2 prior biologics.
- 5% had received ≥3 prior biologics.

Secukinumab was prescribed at a dose of 300 mg in 60% of patients.

At the time of inclusion, 73% of patients presented with inflammatory low back pain ≥4, as well as BASDAI ≥4; 53% had ASDAS >2.1, and 49% had DAPSA ≥15. HLA-B27 was analysed in 83 patients and was positive in 22.9%. Radiological findings are shown in Table II. Comorbidities and baseline extra-articular manifestations are shown in Table III.

Conclusion. In real clinical practice, patients with axPsA predominantly present with mixed disease involvement and a high baseline disease burden, both symptomatic and inflammatory. A relevant proportion of patients showed moderate baseline disease activity (BASDAI ≥4, ASDAS >2.1, and DAPSA ≥15). The high prevalence of comorbidities and prior biologic treatments reflects the clinical complexity of these patients.

These findings provide valuable information on the baseline profile of patients with axPsA treated with secukinumab in Spain and lay the groundwork for analysing treatment effectiveness and persistence.

P143

GUSELKUMAB RESPONSE AND INHIBITION OF STRUCTURAL DAMAGE PROGRESSION IN ACTIVE PSORIATIC ARTHRITIS ACROSS APEX PARTICIPANT SUBGROUPS

Mease P.J.[1,2], Ritchlin C.T.[3], Coates L.C.[4], Kollmeier A.P.[5], Zhou B.[6], Jiang Y.[6], Bensley K.[6], Im K.[7], Batra R.[8], Lozenski K.[9], Chakravarty S.D.[9,10], Rahman P.[11], van der Heijde D.[12]

[1]Rheumatology Research, Providence Swedish Medical Center, Seattle; [2]University of Washington School of Medicine, Seattle; [3]Dept. of Medicine, Allergy/Immunology and Rheumatology, University of Rochester Medical Center, Rochester, USA; [4]Nuffield Dept. of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Botnar Research Centre, Oxford, UK; [5]Johnson & Johnson, San Diego; [6]Johnson & Johnson, Spring House; [7]Johnson & Johnson, Cambridge, USA; [8]Johnson & Johnson, Toronto, Canada; [9]Johnson & Johnson, Horsham; [10]Drexel University College of Medicine, Philadelphia, USA; [11]Craig L Dobbin Genetics Research Centre, Faculty of Medicine, Division of Rheumatology, Memorial University of Newfoundland, St. Johns, Canada; [12]Leiden University Medical Center, Leiden, The Netherlands

Introduction. Guselkumab (GUS), a fully-human/dual-acting mAb, selectively inhibits IL-23p19-subunit and is indicated for moderate-to-severe plaque psoriasis/active PsA/moderately-to-severely active Crohn's disease/ulcerative colitis. The ongoing phase-3b/randomized/double-blind/placebo (PBO)-controlled APEX (NCT04882098) study is evaluating GUS effects on clinical/radiographic progression outcomes in participants with active and erosive PsA. GUS demonstrated significantly higher ACR20 response rates and significant inhibition of structural damage progression vs PBO through Week (W)24. Subgroup analyses evaluated treatment consistency across subgroups defined by participant characteristics.

Methods. APEX evaluated biologic-naive adults with active PsA (SJC≥3/TJC≥3/CRP≥0.3 mg/dL) and ≥2 erosive joints on radiographs of hands/feet, despite previous non-biologic treatment. Participants were randomized to GUS Q4W; GUS at W0/W4 then Q8W; or PBO Q4W. The multiplicity-controlled primary (ACR20) and major secondary (PsA-modified van der Heijde-Sharp [vdH-S] score change from baseline) endpoints at W24 were evaluated in predefined subgroups based on baseline clinical/laboratory/radiographic features.

Results. At W24, 67%/68% of GUS Q4W-/Q8W-treated participants achieved ACR20 response, vs. 47% PBO (both p<0.001). GUS effects on improving joint disease activity were consistent across subgroups, including those defined by sex/BMI/joint involvement/concomitant MTX use (Fig. 1). GUS Q4W-/Q8W-treated participants exhibited significantly lower mean changes in vdH-S score (0.55/0.54 vs. 1.35 PBO; p=0.002/<0.001). GUS effects on inhibiting radiographic progression were generally consistent across subgroups defined by baseline characteristics/radiographic features (Fig. 2). PBO-treated participants with SJC>15 and CRP≥2 mg/dL exhibited notably higher rates of radiographic progression, leading to robust GUS effects. GUS Q8W showed greater effects vs PBO with TJC>15 and erosion score>6.0.

Conclusion. GUS-treated biologic-naive participants with active/erosive PsA demonstrated significantly greater clinical improvement and significant inhibition of structural damage progression vs PBO at W24. GUS effects were generally consistent across diverse subgroups of participants defined by baseline characteristics/radiographic features of interest. Findings spe-

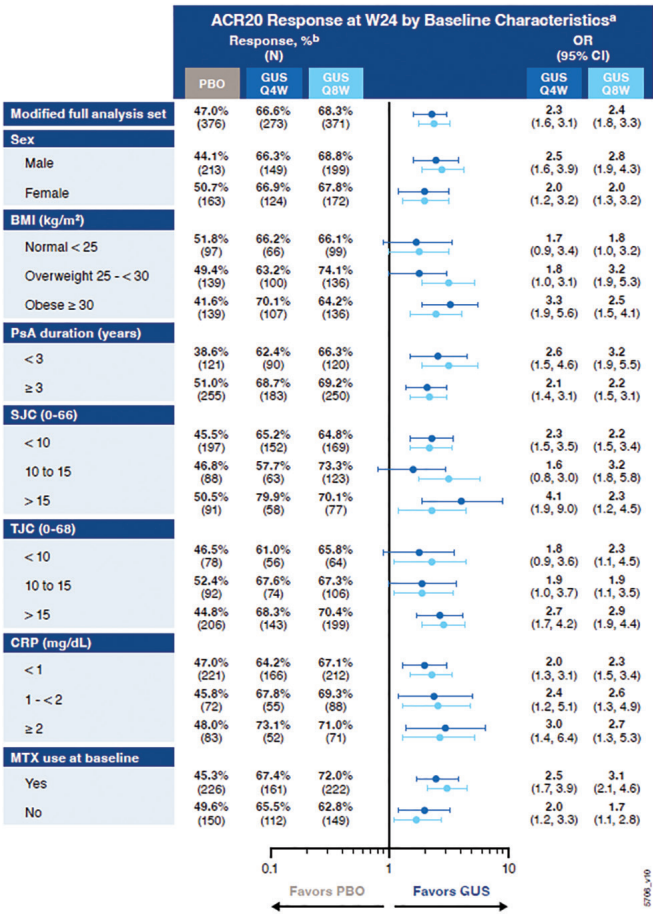


Fig. 1. ACR20 Response at W24 with GUS vs. PBO in APEX participant subgroups defined by baseline characteristics.
[a] Shown are a portion of the predefined subgroups to evaluate treatment consistency over baseline demographics (n=7), disease characteristics (n=12), and medication use (n=5).
[b]Average proportion achieving ACR20 response over 200 multiple imputation datasets. The modified full analysis set (N=1020) comprises all randomized pts except those from Ukraine sites rendered unable to support key study operations due to major disruptions. Pts meeting treatment failure criteria prior to W24 and pts with data missing for reasons other than natural disaster/major disruption were considered nonresponses.
ACR20: American College of Rheumatology ≥20% Improvement; BMI: body mass index; CI: confidence interval; CRP: C-reactive protein; GUS: guselkumab; MTX: methotrexate; OR: odds ratio; PBO: placebo; PsA: psoriatic arthritis; pts: participants; Q4W: every 4 weeks; Q8W: every 8 weeks; SJC: swollen joint count; TJC: tender joint count; W24: Week 24.

cific to PBO participants with the highest degree of inflammation/erosions confirmed these are important risk factors for radiographic progression.

Disclosures. PJM received grants from AbbVie, Acelyrin, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, and UCB; consulting fees from AbbVie, Acelyrin, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Inmagene, Johnson & Johnson, Novartis, Pfizer, Spyre, Takeda, and UCB; and speaker fees from AbbVie, Amgen, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, and UCB. **CTR** received grant/research support from AbbVie, Amgen, UCB and consulting fees from AbbVie, Amgen, Eli Lilly, Gilead, Johnson & Johnson, Novartis, Pfizer, UCB. **LCC** has received grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Johnson & Johnson, Novartis, Pfizer and UCB; worked as a paid consultant for AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Gilead, Galapagos, Johnson & Johnson, Moonlake, Novartis, Pfizer, and UCB; and has been paid as a speaker for AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GlaxoSmithKline, Johnson & Johnson, Medac, Novartis, Pfizer, and UCB. **APK**, **BZ**, **YJ**, **KB**, **KI**, **RB**, and **SDC** are employees of Johnson & Johnson; employees may own stock/stock options in Johnson & Johnson. **KL** is an employee of Johnson & Johnson and owns stock/stock options in Bristol Myers Squibb and Johnson & Johnson. **PR** received consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Johnson & Johnson, Merck, Novartis, Pfizer, and UCB; meeting attendance/travel support from Johnson & Johnson; and research grants from Johnson & Johnson, and Novartis. **DvdH** received consulting fees from AbbVie, Alfasigma, ArgenX, Bristol Myers Squibb, Elly-Lilly,

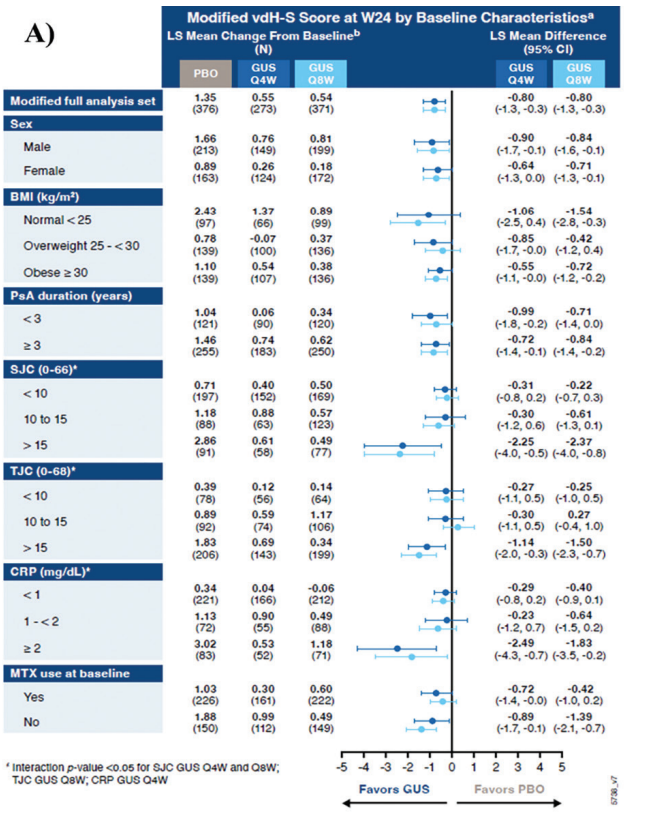


Fig. 2. A. Change in total PsA-modified vdH-S score from baseline to W24 with GUS *vs.* PBO in APEX participant subgroups defined by baseline characteristics.
[a] Shown are a portion of the predefined subgroups to evaluate treatment consistency over baseline demographics (n=7), disease characteristics (n=12), and medication use (n=5).
[b]Average LS mean change in vdH-S over 200 multiple imputation datasets, calculated for each based on an ANCOVA model (explanatory variables: baseline vdH-S score, treatment group, and randomization stratification level). The modified full analysis set (N=1020) comprises all randomized pts except those from Ukraine sites rendered unable to support key study operations due to major disruptions.
BMI: body mass index; CI: confidence interval; CRP: C-reactive protein; GUS: guselkumab; LS: least squares; MTX: methotrexate; PBO: placebo; PsA: psoriatic arthritis; pts: participants; Q4W: every 4 weeks; Q8W: every 8 weeks; SJC: swollen joint count; TJC: tender joint count; vdH-S: van der Heljde-Sharp; W24: Week 24.

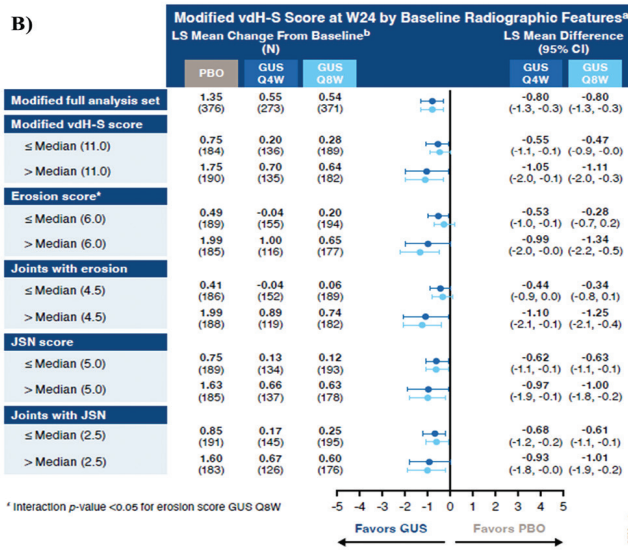


Fig. 2. B. Change in total PsA-modified vdH-S score from baseline to W24 with GUS *vs.* PBO in APEX participant subgroups defined by baseline radiographic features.
[a] Shown are all predefined subgroups to evaluate treatment consistency over baseline demographics features (n=5). [b] Average LS mean change in vdH-S over 200 multiple imputation datasets, calculated for each based on an ANCOVA model (explanatory variables: baseline vdH-S score, treatment group, and randomization stratification level). The modified full analysis set (N=1020) comprises all randomized pts except those from Ukraine sites rendered unable to support key study operations due to major disruptions. BMI: body mass index; CI: confidence interval; GUS: guselkumab; JSN: joint space narrowing: LS: least squares; PBO: placebo; pts: participants; Q4W: every 4 weeks; Q8W: every 8 weeks; vdH-S: van der Heljde-Sharp; W24: Week 24.

Grey-Wolf Therapeutics, Johnson & Johnson, Novartis, Pfizer, Takeda, UCB Pharma and is associate editor of Annals Rheumatic Diseases, and is an editorial board member Journal of Rheumatology, and director of Imaging Rheumatology BV.

P144

EFFECT OF GUSELKUMAB AND GOLIMUMAB COMBINATION THERAPY ON MAGNETIC RESONANCE IMAGING-DETECTED INFLAMMATION AND DAMAGE IN PHALANGEAL JOINTS OF THE HANDS AND FEET AND ENTHESES OF THE HEELS AMONG PARTICIPANTS WITH ACTIVE PSORIATIC ARTHRITIS: FINDINGS FROM THE PHASE 2A AFFINITY STUDY

Wellen J.[1], Li M.[2], Wang T.[2], Cheng E.[2], Zhou B.[2], Reynoso E.[2], Seridi L.[2], Lozenski K.[3], Scher J.U.[4], Ritchlin C.T.[5], Bird P.[6], Østergaard M.[7,*]

[1]Johnson & Johnson, Cambridge; [2]Johnson & Johnson, Spring House; [3]Johnson & Johnson, Horsham; [4]New York University Grossman School of Medicine, New York; [5]Dept. of Medicine, Allergy/Immunology and Rheumatology, University of Rochester Medical Center, Rochester, NY, USA; [6]University of New South Wales, Randwick, Australia; [7]Center for Rheumatology and Spine Diseases, Copenhagen Center for Arthritis Research, Rigshospitalet, Glostrup and Dept. of Clinical Medicine, University of Copenhagen, Copenhagen, Denmark

Introduction. AFFINITY study evaluated guselkumab(GUS)+golimumab(GOL) therapy vs. GUS monotherapy through W24 in participants with PsA+inadequate response to 1-2 TNFi(TNFi-IR). We report exploratory outcomes: MRI assessments of localized inflammation and structural damage in hand/foot phalangeal joints (OMERACT PsAMRIS) and heel entheses (OMERACT HEMRIS).

Methods. Participants were randomized 2:1 to combination therapy (GUS100mg+GOL50mg,Q4W[n=59]) or monotherapy (GUS100mg+placebo,Q4W[n=32]). Longitudinal hand, forefoot, and heel MRI scans were acquired at baseline+Wk24. Scans underwent blinded central reader assessment of radiologic features following PsAMRIS and HEMRIS (Table I); scores were averaged at the feature level for each MRI timepoint; composite indices of inflammatory burden/structural damage were calculated from summation of feature scores. LSM change-from-baseline in composite indices were calculated, differences compared using ANCOVA to evaluate treatment effect over study period.

Results. 91 randomized participants showed balanced baseline characteristics (mean age=49.4y;PsA duration=7.8y). Eighty had ≥1 MRI (n=52 combination therapy;n=28 monotherapy); analyses focused on participants with complete datasets at both timepoints (n=31-36 combination therapy; n=19-22 monotherapy). In hand phalangeal joints, GUS+GOL-treated participants had numerically greater reductions in inflammatory feature scores vs. monotherapy-treated participants (Fig. 1A); LSM change (90%CI) in PsAMRIS 'total inflammation' composite index:–5.55(-9.28,-1.82) with combination therapy vs. -0.44(–5.35,4.48) with monotherapy (nominal p=0.181; Fig. 1B). Features associated with structural damage were stable among participants in both treatment groups (Fig. 1C), with little-to-no change-from-baseline in the 'total structural damage' composite index (Fig. 1D). PsAMRIS assessments of the forefoot exhibited similar feature-level

Table I. Change from baseline in composite indices of inflammation and structural damage from PsAMRIS forefoot and HEMRIS assessments.

	Monotherapy (GUS+PBO)	Combination therapy (GUS+GOL)	*P* value
PsAMRIS - Forefoot			
Total Inflammation[a]	N=19 2.76 (-0.63, 6.14)	N=31 -3.56 (-6.19, -0.93)	0.019
Total Structural Damage[b]	N=22 0.00 (-0.04, 0.04)	N=36 0.01 (-0.02, 0.04)	0.776
HEMRIS			
Inflammatory Score	N=21	N=35	
Achilles Tendon[c]	-0.28 (-0.56, -0.00)	-0.03 (-0.25, 0.18)	0.247
Plantar Fascia[d]	-0.15 (-0.32, 0.01)	0.00 (-0.13, 0.13)	0.223
Structural Damage Score	N=22	N=35	
Achilles Tendon[e]	-0.03 (-0.10, 0.04)	0.02 (-0.03, 0.08)	0.280
Plantar Fascia[f]	-0.02 (-0.05, 0.00)	0.00 (-0.02, 0.02)	0.204

Data shown are LSM (90% CI) change from baseline to Wk24 for all participants with available assessments (N; complete case analysis) and all reported p-values for combination therapy vs. monotherapy comparisons are nominal. Data calculated from an ANCOVA model, with randomization arm, baseline endpoint measurement, and baseline stratification factors as the explanatory factors. [a] Summation of 1x bone marrow edema, 2x synovitis, 2x tenosynovitis, and 3x periarticular inflammation. [b] Summation of 1xbone erosion and 20x bone proliferation. [c] Summation of intra-tendon hypersignal, peri-tendon hypersignal, bone marrow edema, and retrocalcaneal bursitis. [d] Summation of intra-fascia hypersignal, peri-fascia hypersignal, and bone marrow edema. [e] Summation of tendon thickening, calcaneal enthesophyte, and calcaneal bone erosion. [f] Summation of scores of fascia thickening, calcaneal enthesophyte, and calcaneal bone erosion. ANCOVA: analysis of covariance; CI: confidence interval; GOL: golimumab; GUS: guselkumab; HEMRIS: Heel Enthesis Magnetic Resonance Imaging Score; LSM: least squares mean; PsAMRIS: Psoriatic Arthritis Magnetic Resonance Imaging Score; Wk: Week.

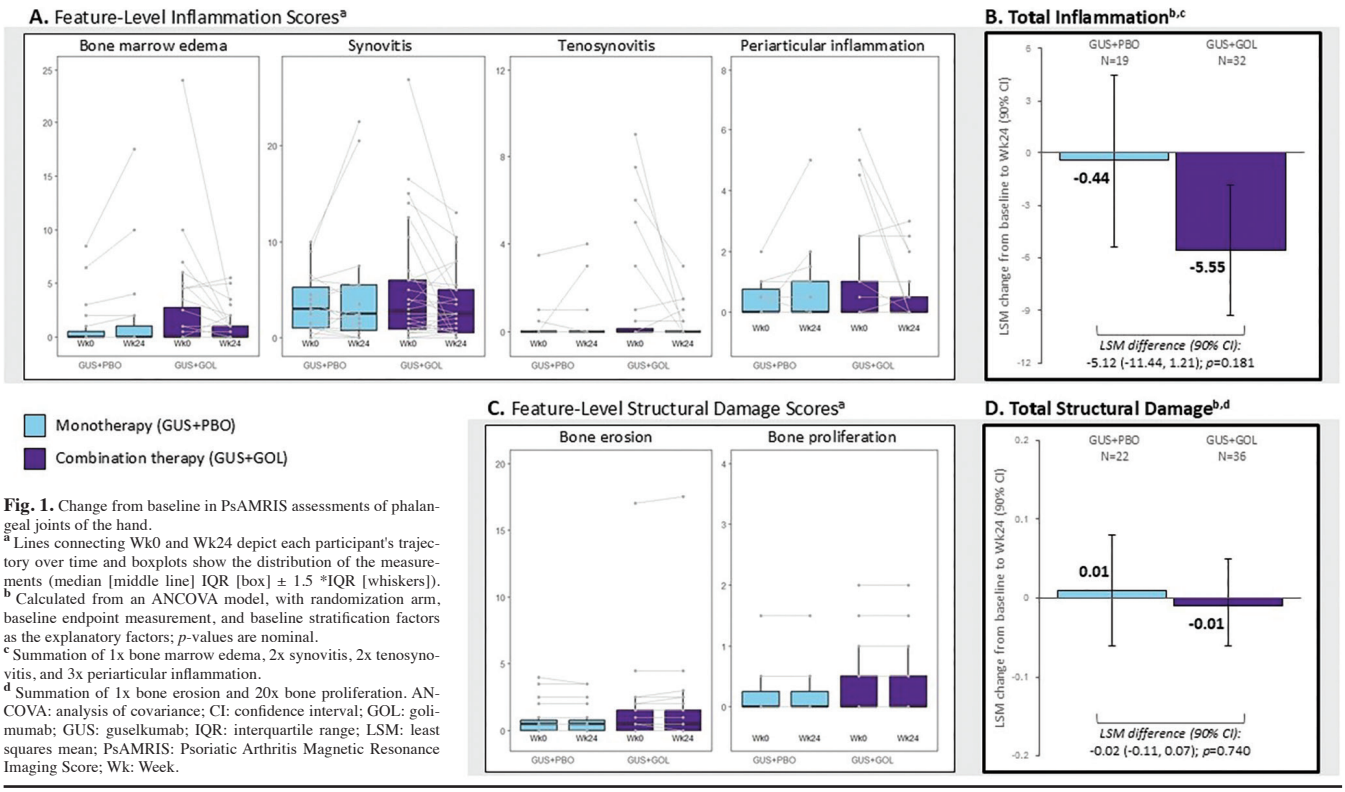


Fig. 1. Change from baseline in PsAMRIS assessments of phalangeal joints of the hand.
[a] Lines connecting Wk0 and Wk24 depict each participant's trajectory over time and boxplots show the distribution of the measurements (median [middle line] IQR [box] ± 1.5 *IQR [whiskers]).
[b] Calculated from an ANCOVA model, with randomization arm, baseline endpoint measurement, and baseline stratification factors as the explanatory factors; p-values are nominal.
[c] Summation of 1x bone marrow edema, 2x synovitis, 2x tenosynovitis, and 3x periarticular inflammation.
[d] Summation of 1x bone erosion and 20x bone proliferation. ANCOVA: analysis of covariance; CI: confidence interval; GOL: golimumab; GUS: guselkumab; IQR: interquartile range; LSM: least squares mean; PsAMRIS: Psoriatic Arthritis Magnetic Resonance Imaging Score; Wk: Week.

trends, resulting in numerically greater reduction in 'total inflammation' with combination vs. monotherapy (-3.56[-6.19,-0.93] vs. 2.76[–0.63,6.14], respectively; nominal p=0.019) and little-to-no change-from-baseline in PsAMRIS 'total structural damage' (Table I). HEMRIS assessments of calcaneal entheses showed no differences in change-from-baseline in composite inflammatory or structural damage indices between groups (Table I).
Conclusion. Exploratory MRI assessments showed participants with PsA+TNFi-IR treated with GUS+GOL had greater reductions in MRI features of inflammation within hand/foot phalangeal joints than GUS monotherapy.
COI. JW, **ML**, **TW**, **EC**, **BZ**, **ER**, **KL** and **LS:** are employees of Johnson & Johnson and may own stock/stock options in Johnson & Johnson. **JUS:** received grant/research support from Johnson & Johnson and Pfizer; and received consulting fees from Bristol Myers Squibb, Johnson & Johnson, Novartis, Pfizer, and UCB. **CTR:** received grant/research support from AbbVie, Amgen, and UCB; and received consulting fees from AbbVie, Amgen, Eli Lilly, Gilead, Johnson & Johnson, Novartis, Pfizer, and UCB. **PB:** served as a consultant for AbbVie, Amgen, AstraZeneca, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, Sanofi-Regeneron, and UCB; received grant/research support from AbbVie, Amgen, AstraZeneca, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, Sanofi-Regeneron, and UCB; speakers bureau for AbbVie, Eli Lilly, Gilead, Johnson & Johnson, Merck, Pfizer, and UCB. **MØ:** has received research grants from AbbVie, Amgen, Bristol Myers Squibb, Merck, Celgene, Eli Lilly, Novartis, and UCB; speaker fees from AbbVie, Bristol Myers Squibb, Celgene, Eli Lilly, Galapagos, Gilead, Johnson & Johnson, MEDAC, Merck, Novartis, Pfizer and UCB; and consultancy fees from AbbVie, Alfasigma, Bristol Myers Squibb, Celgene, Eli Lilly, Galapagos, Gilead, Johnson & Johnson, Merck, Novartis, Pfizer, and UCB.

P145

EFFECTS OF NON-PHARMACOLOGICAL SHORT-TERM GROUP INTERVENTION IN PATIENTS WITH SPONDYLOARTHRITIS AND RESIDUAL PAIN: A COMPARATIVE ANALYSIS OF REHABILITATION AND PSYCHOTHERAPY

Patti G.D.[1], Liberatore D'Agostino A.[1,2], Di Vittori L.[3], Spagnolo G.[2], Iodice D.[3], Sarri S.[1], Cristiano F.[1], Magnanimi C.T.[1], Masnata R.[1], Boni V.[1], Natalello G.[1], Ortolan A.[1], D'Agostino M.A.[1], De Lorenzis E.[1]
[1]Division of Rheumatology and Clinical Immunology, Catholic University of the Sacred Heart, Fondazione Policlinico Universitario A. Gemelli IRCCS, Rome; [2]Clinical Psychology Unit, Fondazione Policlinico Universitario Agostino Gemelli IRCCS, Rome; [3]Rehabilitation Service Unit, Fondazione Policlinico Universitario Agostino Gemelli IRCCS, Rome, Italy

Background. Patients with spondyloarthritis (SpA) often experience residual pain despite optimized immunosuppressive treatment (IST), due to structural damage, comorbidities, pain sensitization, and psychological factors. Although not individualized, short-term group-based psychological and rehabilitation interventions may improve adherence, reduce isolation, and optimize resource use. This study aims to evaluate their effectiveness in managing residual pain in SpA.
Methods. Patients with SpA, residual pain ≥40 mm (VAS), and no indication for IST escalation were allocated to group-based Acceptance and Commitment Therapy (G-ACT) or rehabilitation therapy(G-RT) based on physician judgment and patient preference. Interventions were delivered in groups of 8–10 patients through standardized 60-minute sessions: six weekly sessions for G-ACT and ten sessions over 5 weeks for G-RT. Pain and disease activity were assessed by VAS, disability by HAQ-DI, and anxiety/depression by HADS-A/D. Minimal clinically important improvement (MCII) was defined as a reduction of 20mm in VAS, 0.25 points in HAQ-DI, and 2 points in HADS.
Results. The pilot study included 44 patients (23 G-RT, 21 G-ACT). The groups were comparable at baseline (Table I). G-RT significantly reduced pain (VAS 66.8±19.2 to 60.3±20.2mm; p=0.031) and disease activity (64.6±23.1 to 55.9±16.8mm; p=0.019), with 26.1% of patients achieving MCII for both outcomes. Although MCII was achieved by 13.0%, 21.7%, and 8.7% of patients for HAQ-DI, HADS-D, and HADS-A, respectively, no significant group-level changes were observed. G-ACT significantly reduced HADS-A (10.9±3.3 to 9.9±3.4; p=0.035), with 33.3% of patients achieving MCII. MCII rates for pain, disease activity, disability, and depressive symptoms were 9.5%, 5.0%, 18.8%, and 28.6%, respectively, without significant group-level changes.

Table I. Characteristics of the enrolled patients.

	Overall N=44	G-RT N=23	G-ACT N=21	Nominal p-value
Age, years, mean±SD	57.1 ± 11.5	58.0 ± 12.8	56.2 ± 10.1	0.6
Male	8 (18.2%)	5 (21.7%)	3 (14.3%)	0.8
BMI, kg/m2, mean±SD	29.5 ± 6.3	29.8 ± 6.6	29.4 ± 6.3	0.8
Diabetes	11 (25.0%)	5 (21.7%)	6 (28.6%)	0.9
Chronic cardiovascular disease	10 (22.7%)	8 (34.8%)	2 (9.5%)	0.102
Chronic respiratory disease	5 (11.4%)	2 (8.7%)	3 (14.3%)	0.9
Cancer	4 (9.1%)	3 (13.0%)	1 (4.8%)	0.7
Secondary Fibromyalgia	18 (40.9%)	8 (34.8%)	10 (47.6%)	0.6
Disease Duration, y, median (IQR)	9.0 (5.0, 17.0)	9.0 (4.0, 17.0)	8.5 (6.0, 17.0)	0.7
Axial SpA	17 (38.6%)	10 (43.5%)	7 (33.3%)	0.7
PsA	30 (68.2%)	16 (69.6%)	14 (66.7%)	>0.9
Pain on VAS, mm, mean±SD	67.8 ± 17.4	66.8 ± 19.2	68.9 ± 15.5	0.7
Activity on VAS, mm, mean±SD	60.1 ± 17.6	55.9 ± 16.8	64.9 ± 17.7	0.097
HAQ-DI, mean±SD	1.2 ± 0.6	1.2 ± 0.5	1.3 ± 0.7	0.5
HADS-D, mean±SD	8.1 ± 3.5	7.8 ± 3.4	8.5 ± 3.7	0.5
HADS-A, mean±SD	10.3 ± 3.0	9.8 ± 2.7	11.0 ± 3.2	0.2

BMI: Body Mass Index; G-ACT: Group-based acceptance and commitment therapy; G-RT: Group-based rehabilitation therapy; HADS-A/D: Hospital Anxiety and Depression Scale Anxiety/Depression; HAQ-DI: Health Assessment Questionnaire-Disability Index; IQR: interquartile range; PsA: psoriatic arhtritis; SD: standard deviation; SpA: Spondyloarthritis; VAS: Visual Analogue Scale.

Conclusion. Standardized group-based non-pharmacological interventions may provide clinically meaningful benefits in up to one-quarter of patients with SpA. Improvements in pain outcomes appear to be greater with G-RT than with G-ACT, whereas G-ACT seems to have a more pronounced effect on psychological symptoms despite a limited impact on pain.

P146

TARGETED THERAPIES AND THE RATES OF ACHIEVEMENT OF TARGETS IN DIFFERENT DISEASE ACTIVITY INDICES, AS WELL AS POTENTIAL OBSTACLES, IN PATIENTS WITH PSORIATIC ARTHRITIS (PSA)

Demir E.[1], Pehlivan Y.[2]
[1]Bursa Uludag University, Dept. of Internal Medicine, Bursa; [2]Bursa Uludag University, Division of Rheumatology, Bursa, Turkey

Objective. The aim of this study is to assess disease activity using different composite indices in patients diagnosed with psoriatic arthritis (PsA) who have received disease-modifying antirheumatic drug (DMARD) therapy for at least 6 months, and to identify potential barriers to achieving remission or minimal disease activity.
Material and methods. In this cross-sectional study, 258 patients with psoriatic arthritis (PsA) who were examined and followed up at the Rheumatology outpatient clinics of Bursa Uludağ University Faculty of Medicine Hospital during the year 2025 were evaluated. The patients' demographic data, body mass index, smoking status, family history, duration of disease symptoms and diagnosis, therapeutic agents used, comorbidities, non-PsA rheumatic diseases, presence of deformity, patterns of involvement, and presence of extra-articular involvement were analyzed. For disease activity assessment, ASDAS-CRP was used in patients with axial involvement, and MDA/VLDA and DAPSA scores were utilized for those with peripheral involvement.
Results. In patients with peripheral involvement, the median DAPSA score was (0–77). Among these patients, a DAPSA score >14 was observed in 17.2%, indicating that the desired therapeutic targets were not achieved. Furthermore, 30.2% of these patients failed to meet the MDA/VLDA criteria. In patients with axial involvement, the median ASDAS-CRP score was 1.6 (0.6–4), and 17.3% of the evaluated patients did not achieve the targeted disease control following treatment. Multivariate analyses revealed that the presence of fibromyalgia and nail involvement were independent risk factors for disease activity in patients with peripheral involvement. In contrast, no independent risk factors were identified for active disease (ASDAS ≥2.1) in axial Psoriatic Arthritis (PsA). DAPSA scores were significantly higher in patients with longer symptom and diagnosis durations. At baseline, the prevalence of enthesitis was found to be higher in patients receiving bDMARDs compared to the csDMARD group. The persistence of enthesitis and dactylitis during the treatment process was associated with the failure to

reach both DAPSA and MDA/VLDA targets. Additionally, median DAPSA and ASDAS-CRP scores were higher in patients using tsDMARDs compared to those in the bDMARD group.

Conclusion. Persistent enthesitis and dactylitis, comorbid fibromyalgia, and the presence of additional rheumatic conditions represent major barriers to reaching remission or minimal disease activity targets in psoriatic arthritis management. Our results suggest that a personalized therapeutic approach is required in psoriatic arthritis, guided by clinical characteristics, duration of the disease, and the presence of concomitant conditions.

P147

ICOTROKINRA, A TARGETED ORAL PEPTIDE, IN PARTICIPANTS WITH MODERATE-TO-SEVERE PLAQUE PSORIASIS AND PSORIATIC ARTHRITIS: RESULTS FROM A POOLED ANALYSIS OF THE PHASE 3 ICONIC-LEAD, ICONIC-ADVANCE 1, AND ICONIC-ADVANCE 2 TRIALS

McInnes I.B.[1], Gold L.S.[2], Bissonnette R.[3], Lebwohl M.G.[4], Reyes-Servin O.[5], Cafone J.[5], Yang Y.W.[6], Chakravarty S.D.[6,7], Iaconangelo C.[6], Li S.[5], Shagroni T.[8], Lozenski K.[6], Armstrong A.W.[9], Merola J.F.[10]

[1]College of Medical Veterinary and Life Sciences, University of Glasgow, Glasgow, UK; [2]Henry Ford Health System, West Bloomfield, USA; [3]Innovaderm Research, Montreal, Canada; [4]Dept. of Dermatology, Icahn School of Medicine at Mount Sinai, New York; [5]Johnson & Johnson, Spring House; [6]Johnson & Johnson, Horsham; [7]Drexel University College of Medicine, Philadelphia; [8]Johnson & Johnson, San Francisco; [9]Dept. of Dermatology, University of California Los Angeles, Los Angeles; [10]Dept. of Dermatology, and Dept. of Medicine, Division of Rheumatology, UT Southwestern Medical Center and O'Donnell School of Public Health, Dallas, USA

Introduction. Post-hoc analyses of ICONIC-LEAD\ICONIC-ADVANCE-1\ ICONIC-ADVANCE-2 studies evaluated Icotrokinra(ICO) for treatment of participants with moderate-to-severe PsO and self-reported medical history of PsA by assessing PsA-\PsO-relevant outcomes and AEs through W16.

Methods. Participants (ICONIC-LEAD≥12y\ICONIC-ADVANCE-1&2≥18y) with moderate-to-severe plaque PsO(BSA≥10%\PASI≥12\ IGA≥3) were randomized (ICONIC-LEAD\ICONIC-ADVANCE-1=2:1; ICONIC-ADVANCE-2=4:1) to once-daily oral ICO-200mg or PBO through W16. Analyses included: LSM change-from-baseline in PtGA of PsA\Patient Pain; IGA 0/1\IGA 0\PASI90\PASI100 response rates; proportions of participants (with baseline scores ≥2) achieving ss-IGA 0/1\hf-PGA 0/1\f-PGA 0/1\sPGA-G 0/1; percent change-from-baseline in mNAPSI among participants with baseline mNAPSI score>0; proportion of participants (with baseline DLQI score>1) achieving DLQI 0/1. Treatment differences (95% CI): using Mantel-Haenszel weights\LSMs/LSM differences: using MMRM\p-values: nominal.

Results. 13.9%(216/1555) of pooled study population had moderate-to-severe PsO and medical history of PsA(ICO=149;PBO=67). Baseline demographics\disease characteristics were generally similar between groups, reflecting moderate PtGA of PsA(mean score=48.6); mean age=49.5y\ male=61.6%\White=78.7%\mean PASI score=19.8\affected BSA=25.2%. ICO-treated participants reported substantially greater mean improvements vs. PBO in PsA-relevant PROs from baseline to W16-PtGA of PsA= -19.2 vs. -0.8 (p<0.001), PsA Patient Pain= -19.5 vs. -7.8 (p=0.003),with separation evident by W8 (-18.5 vs. -4.8 [p<0.001]\-17.1 vs. -4.6 [p<0.001]; Fig. 1). At W16, greater proportions of ICO-treated vs. PBO-treated participants achieved IGA 0/1(56.5% vs. 10.6%; p<0.001)\PASI90 (45.6% vs. 3.0%; p<0.001)\IGA 0 (27.9% vs. 0%; p<0.001)\PASI100 (25.2% vs. 0%; p<0.001; Fig. 2). Among those with high-impact site involvement (ss-IGA≥2=85.4%\ sPGA-G≥2=39.4%\hf-PGA≥2=44.6%\f-PGA≥2=39.9%), greater proportions of ICO-treated vs PBO-treated participants achieved ss-IGA 0/1(69.0% vs. 12.5%; p<0.001)\sPGA-G 0/1(70.0% vs 25.0%; p<0.001)\hf-PGA 0/1(66.2% vs. 25.9%; p<0.001)\f-PGA 0/1(46.9% vs. 9.5%; p=0.005). ICO-treated participants demonstrated mNAPSI improvement from baseline to W16(LSM percent change = –41.6), while PBO-treated participants exhibited worsened disease(LSM percent change=+118.6; p<0.001). AE rates through W16 were comparable (ICO=49.3%[73/148] vs. PBO=50.7%[34/67]).

Conclusion. ICO-treated adults\adolescents with moderate-to-severe plaque PsO and a medical history of PsA reported early, substantial improvements in PsA disease activity and pain, with high rates of overall skin and high-impact site PsO skin clearance and substantial improvements in nail disease\HRQoL, with AE rates similar to PBO through W16.

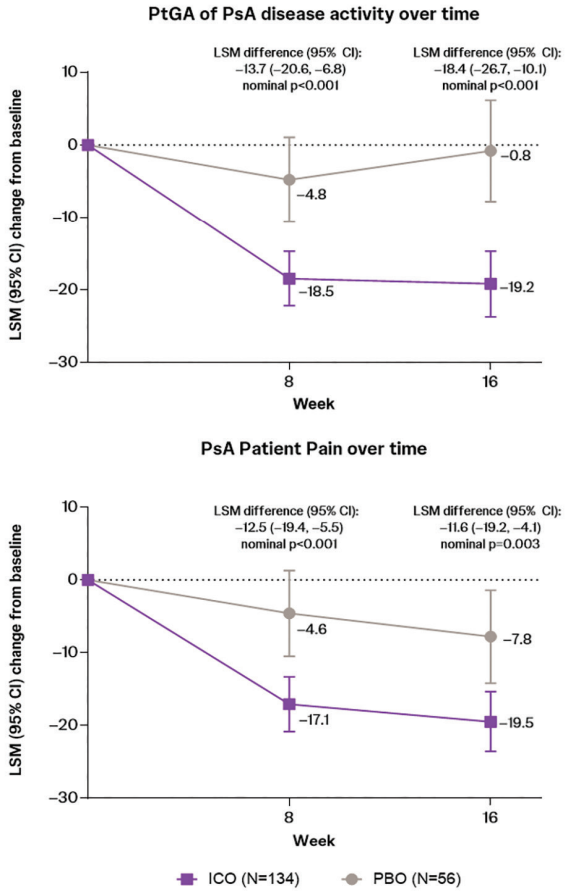


Fig. 1. PsA-specific PROs in participants with moderate-to-severe PsO and a medical history of PsA: LSM change from baseline through W16 in (**A**) PtGA of PsA disease activity and (**B**) PsA patient pain.

Zero change from baseline was assigned after participants discontinued study drug due to a lack of efficacy, or an AE of worsening PsO, or initiated prohibited medication that could impact PsO. Observed data were used for participants who discontinued study drug for other reasons. The remaining missing data were not imputed and were accounted for through the MMRM model.

LSMs, LSM differences, and p-values are based on the MMRM model with treatment group, visit, study, baseline PtGA PsA disease activity/pain, baseline PtGA PsA disease activity/pain by visit interaction, and treatment group by visit interaction as covariates.

ICO: icotrokinra; CI: confidence interval; LSM: least-squares mean; MMRM: mixed-effect model for repeated measures; PBO: placebo; PRO: patient-reported outcome; PsA: psoriatic arthritis; PsO: psoriasis; PtGA: patient global assessment; W: week.

Disclosures. IBM: Served as a consultant for AbbVie, Amgen, AstraZeneca, Bristol Myers Squibb, Cabaletta, Compugen, Eli Lilly, Gilead, GlaxoSmithKline, Johnson & Johnson, Novartis, Pfizer, Roche, Sanofi, and UCB; grant/research support from Amgen, AstraZeneca, Bristol Myers Squibb, Cabaletta, Dextera, Eli Lilly, GlaxoSmithKline, Johnson & Johnson, Montai, Novartis, and UCB; shareholder for Cabaletta, Causeway, Compugen, and Dextera; NHS GGC Board Member; and Versus Arthritis Trustee.
LSG: Served as an investigator, advisor and/or speaker for AbbVie, Amgen, Bristol Myers Squibb, Galderma, Johnson & Johnson, LEO Pharma, Lilly, Pfizer, Regeneron, Sanofi, and Takeda.
RB: Served as an advisory board member, consultant, speaker and/or investigator for and received honoraria and/or grants from, AbbVie, Alumis, Amgen, AnaptysBio, Arcutis, BMS/Celgene, Dermavant, Eli Lilly, Johnson & Johnson, LEO Pharma, Nimbus, Oruka, Takeda, UCB, Ventyx Biosciences, Vyne, Xencor, Zai Lab, and Zura Bio; and is an employee and shareholder of Innovaderm Research.
MGL: Employee of Mount Sinai and receives research funds from AbbVie, Arcutis, Avotres Therapeutics, Boehringer Ingelheim, Bristol Myers Squibb, Cara Therapeutics, Clexio, Dermavant Sciences, Eli Lilly, Incyte, Inozyme, Johnson & Johnson, Oruka, Pfizer, Sanofi-Regeneron, and UCB; and is a consultant for AbbVie, Added Health, Aikium, Almirall, AltruBio Inc., Alumis, Amgen, Apogee, Arcutis, AstraZeneca, Atomwise, Avotres Therapeutics, Boehringer Ingelheim, Bristol Myers Squibb, Castle Biosciences, Celltrion, CorEvitas, Dermavant Sciences, Dermsquared, Edesa Biotech, Eli Lilly, Evommune, Facilitation of International Dermatology Education,

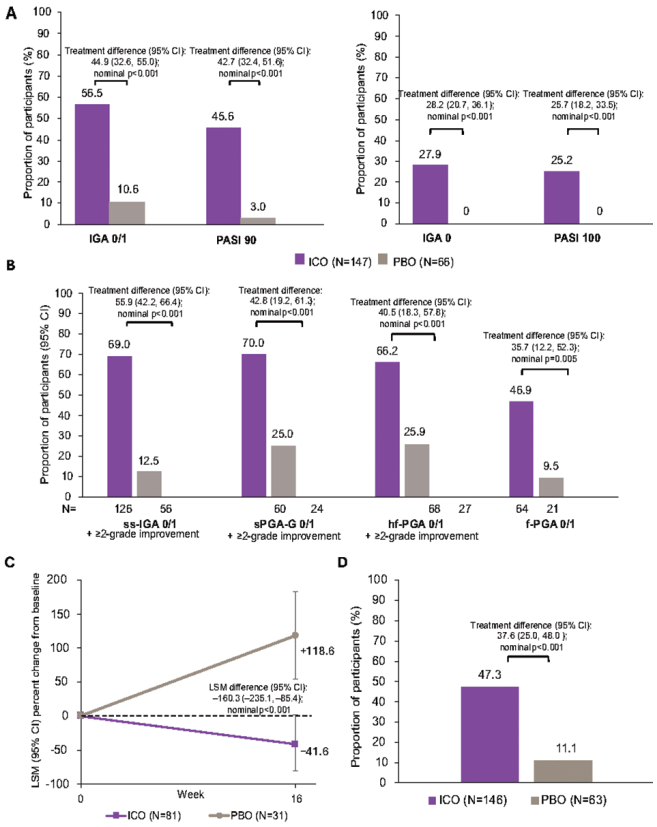


Fig. 2. W16 skin outcomes and HRQoL in participants with moderate-to-severe PsO and a medical history of PsA: (**A**) proportion of participants achieving IGA 0/1, IGA 0, PASI 90, or PASI 100; (**B**) proportion of participants achieving scores 0/1 at high-impact sites with at least mild severity at baseline (score ≥2); (**C**) percent change from baseline in mNAPSI score among participants with score >0 at baseline; (**D**) proportion of participants achieving DLQI 0/1 among participants with score >1 at baseline. For binary outcomes (A, B, and D), participants who discontinued study drug due to a lack of efficacy, or an AE of worsening PsO, or who initiated prohibited medication that could impact PsO were considered nonresponders. Observed data were used for participants who discontinued study drug for other reasons. After accounting for the intercurrent events, participants with missing data were considered non-responders. *p*-values based on Cochran-Mantel-Haenszel chi-square test stratified by study.
For continuous outcomes (C), zero change from baseline was assigned after participants discontinued study drug due to a lack of efficacy, or an AE of worsening PsO, or initiated prohibited medication that could impact PsO. Observed data were used for participants who discontinued study drug for other reasons. The remaining missing data were not imputed and were accounted for through the MMRM model.
LSMs, LSM difference, and *p*-value are based on the MMRM model with treatment group, visit, study, baseline mNAPSI score, baseline mNAPSI score by visit interaction, and treatment group by visit interaction as covariates.
AE: adverse event; CI: confidence interval; DLQI: Dermatology Life Quality Index; f-PGA: Physician's Global Assessment of fingernails; hf-PGA: Physician's Global Assessment of hands and feet; HRQoL: health-related quality of life; ICO: icotrokinra; IGA: Investigator's Global Assessment; LSM: least-squares mean; MMRM: mixed-effect model for repeated measures; mNAPSI: modified Nail Psoriasis Severity Index; PASI: Psoriasis Area and Severity Index; PBO: placebo; PsA: psoriatic arthritis; PsO: psoriasis; sPGA-G: static Physician's Global Assessment of Genitalia; ss-IGA: scalp-specific Investigator's Global Assessment.

Forte Biosciences, Galderma, Genentech, Incyte, Johnson & Johnson, LEO Pharma, Mayne Pharmaceuticals, Meiji Seika Pharma, Mindera, Mirium Pharmaceuticals, Moonlake, Novartis, Oruka, Pfizer, Sanofi-Regeneron, Revolo, Seanergy, Strata, Sun Pharma, Takeda, Trevi, and Verrica.
OR-S, JC, Y-WY, SDC, CI, SL, TS, and KL: Employees of Johnson & Johnson; may own stock/stock options in Johnson & Johnson.
AWA: Served as a speaker, consultant, and/or investigator for AbbVie, Amgen, Arcutis, Bristol Myers Squibb, Dermavant Sciences, Eli Lilly and Company, Galderma, Incyte, Johnson & Johnson, LEO Pharma, Novartis, Pfizer, Regeneron, Sanofi, Takeda, and UCB.
JFM: Served as a consultant and/or investigator for AbbVie, Amgen, AstraZeneca, Biogen, Boehringer Ingelheim, Bristol Myers Squibb, Dermavant, Eli Lilly, Galderma, Johnson & Johnson, Moonlake, Novartis, Oruka, Pfizer, Regeneron, Sanofi, Sun Pharma, and UCB.

P148

REAL-WORLD EFFECTIVENESS OF BIMEKIZUMAB IN CLINICAL PRACTICE: 12-MONTH RESULTS FROM A LONGITUDINAL COHORT STUDY

Rodríguez-Muguruza S.[1], Castro S.[1], Del Castillo N.[1], Veloza C.[1], Just M.[2], López Broseta P.[3], Fontova R.[1]
[1]Dept.Rheumatology, Hospital Universitari Joan XXIII, Tarragona; [2]Dept.Dermatology, Hospital Universitari Joan XXIII, Tarragona, Spain; [2]Dept.Dermatology, Hospital Universitari Joan XXIII, Tarragona; [3]Dept.Clinical Pharmacy, Hospital Universitari Joan XXIII, Tarragona, Spain

Introduction. Bimekizumab, a dual IL-17A/IL-17F inhibitor, has shown strong efficacy in psoriasis, psoriatic arthritis (PsA) and axial spondyloarthritis (axSpA). Real-world evidence regarding its effect on inflammatory biomarkers remains limited, particularly in patients with extensive prior exposure to biologic therapies. Our aim was to evaluate the evolution of inflammatory biomarkers over 12 months of bimekizumab treatment and to assess their association with clinical variables, including previous biologic exposure and history of uveitis.
Methods. Observational, retrospective, monocentric study including adults with PsA, psoriasis or axSpA treated with bimekizumab between 2023–2025. The neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were measured at months 0, 3, 6 and 12. Longitudinal changes were analysed using mixed linear models.
Results. A total of 42 patients (57% male, mean age 52 years, SD ±11.81) were included, of whom 17 (40.5%) had received ≥3 prior biologics (Table I). NLR, PLR, SII, and CRP remained stable throughout the treatment period (p>0.05), while ESR significantly decreased (p=0.004). Disease duration correlated with NLR (r=0.37) and SII (r=0.32).
Two patients (4.7%) had a history of uveitis, with no flare-ups during the follow-up. Six patients (14.3%) experienced adverse events: oral candidiasis (n=4), cellulitis with abscess (n=1), and a paradoxical reaction (n=1). Seven patients (1.7%) discontinued treatment, with only 3 (7.1%) due to inefficacy (Table II). The mean treatment duration with bimekizumab was 458 days (SD ±244.7), corresponding to over 15 months of real-world exposure, with an 83% persistence rate.
Conclusion. Bimekizumab demonstrated good real-world effectiveness, with stable inflammatory biomarkers and a significant reduction in ESR during the first year of therapy. It showed favourable performance in a highly

Table I. Baseline characteristics of the study population (n=42).

Characteristic	Value
Age, years, mean (SD)	52.4 (11.8)
Sex, n (%)	Male 24 (57.1%), Female 18 (42.9%)
Disease duration, years, mean (SD)	16.5 (9.5)
Diagnosis, n (%)	Psoriasis 28 (66.7%), Psoriatic arthritis 9 (21.4%), Axial spondyloarthritis 5 (11.9%)
HLA-B27 positivity, n (%)	3 (12.5% of tested)
Baseline disease activity	PASI (n=26): mean 14.0 (SD 7.0) BASDAI (n=3): mean 5.33 (SD 1.53) DAPSA (n=6): mean 7.83 (SD 3.43)
Uveitis history, n (%)	2 (4.7%)
Monotherapy with bimekizumab, n (%)	32 (76.2%)

Table II. Adverse events and treatment discontinuation.

Category	n (%)
Patients with any adverse event	6 (14.3%)
• Oral candidiasis	4 (9.5%)
• Cellulitis with abscess	1 (2.4%)
• Paradoxical reaction	1 (2.4%)
Treatment discontinuation	7 (16.7%)
• Due to inefficacy	3 (7.1%)
• Due to candidiasis	1 (2.4%)
• Due to paradoxical reaction	1 (2.4%)
• Gestational desire	1 (2.4%)
• Loss to follow-up	1 (2.4%)
Uveitis flares during treatment	0
Treatment retention at final follow-up	83.3% (35/42 patients)

pretreated population (40.5% with ≥3 prior biologics), with an overall drug persistence of 83%. The absence of uveitis flares and the low frequency of generally mild adverse events further reinforce its reassuring safety profile.

References
1. ZABOTTI A, CABAS N, DE MARTINO M *et al*.: Real-world effectiveness of bimekizumab in predominantly difficult-to-treat patients with psoriatic arthritis followed in a combined dermatology-rheumatology clinic: a 24-week multicenter study. *Rheumatol Ther* 2025; 12(5): 961-73.
2. BROWN MA, RUDWALEIT M, VAN GAALEN FA *et al*.: Low uveitis rates in patients with axial spondyloarthritis treated with bimekizumab: pooled results from phase 2b/3 trials. *Ann Rheum Dis* 2024; 83(12): 1722-30.
3. LI Y, LIU J, HU Y *et al*.: The neutrophil-to-lymphocyte ratio in rheumatoid arthritis: the dual perspectives from literature and clinic. *Medicine* (Baltimore) 2025; 104(38): e44554.

P149

BIOLOGICAL DMARDS IMPROVE FUNCTION BY SUPPRESSING INFLAMMATION IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS: A CAUSAL MEDIATION ANALYSIS IN THE METEOR REGISTRY

Sepriano A.[1,2], Ramiro S.[3,4], van der Heijde D.[3], Moltó A.[6], van Gaalen F.[3], Bergstra S.A.[3], Landewé R.[4,5]
[1]NOVA Medical School, Lisbon; [2]Dept. of Rheumatology, Hospital de Egas Moniz, Lisbon, Portugal; [3]Dept. of Rheumatology, Leiden University Medical Center, Leiden; [4]Zuyderland Medical Center, Heerlen; [5]Amsterdam University Medical Center (ARC), Amsterdam, The Netherlands; [6]INSERM U-1153, Centre de Recherche en Epidémiologie et Statistiques, Université Paris Cité, and Rheumatology Dept. Bichat Hospital, APHP, Paris, France

Introduction/Objective. Biological DMARDs (bDMARD) improve function in patients with axSpA. It is thought that this beneficial effect is primarily driven by the reduction of inflammatory activity, but such an assumption has not yet been tested. We assessed whether bDMARD-induced reduction of inflammation explains the functional benefits of bDMARDs in patients with axSpA.
Methods. Patients with a diagnosis of axSpA from METEOR who were bDMARD naive and had high disease activity (ASDAS≥2.1) at baseline were included. The treatment effect of bDMARDs started at baseline on physical function (BASFI, 0-10) at 6 months was evaluated using the G-formula. This method identifies the total treatment effect as the difference between the potential outcome (PO) had all patients been treated with bDMARDs and the PO had no patients been treated, adjusting for baseline confounders. Causal mediation analysis (an extension of the G-formula) was used to disentangle how much of the total effect was explained by the reduction of ASDAS at 6 months (indirect effect) and how much remained unexplained (direct effect) (Fig. 1).
Results. In total, 352 patients were included (mean symptom duration: 2 years). bDMARDs were started in 105 (30%) patients. Patients on bDMARDs, as compared to those who were not, had more often BME on MRI-SIJ (41% vs. 30%) and higher BASFI (5 vs. 3). ASDAS decreased more in treated than untreated patients (ΔASDAS -1.23 vs. -0.5). The PO (mean BASFI) had all patients been treated was 2.35 and the PO had all patients not been treated was 2.92 [mean difference (95%CI): -0.57 (-1.00; -0.13)]. The treatment effect was entirely explained by the reduction of ASDAS (Fig. 2).
Conclusion. Biological DMARDs improve function of patients with recent onset axSpA by controlling disease activity. These data further stress the importance of measuring ASDAS to assess response to therapy in patients on bDMARDs.

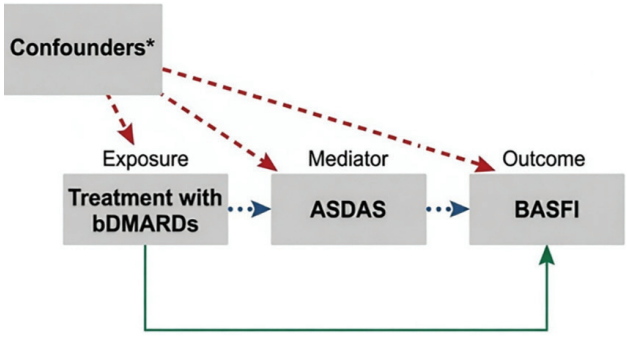


Fig. 1. Schematic representation of the mediation analysis. Green full arrow: direct effect, blue dotted arrow: indirect effect, red dashed arrow: confounding.
*Baseline confounders: age, sex, presence of comorbidities (yes *vs.* no), HLA-B27 status, damage on the sacroiliac joints on pelvic radiographs according to mNY criteria (local reader), presence of bone marrow edema on MRI-SU (local reader), ASDAS, BASFI, peripheral features (arthritis and enthesitis), extra-musculoskeletal manifestations (IBD, uveitis and psoriasis) and comedication (NSAIDs, glucocorticoids and conventional synthetic DMARDs).

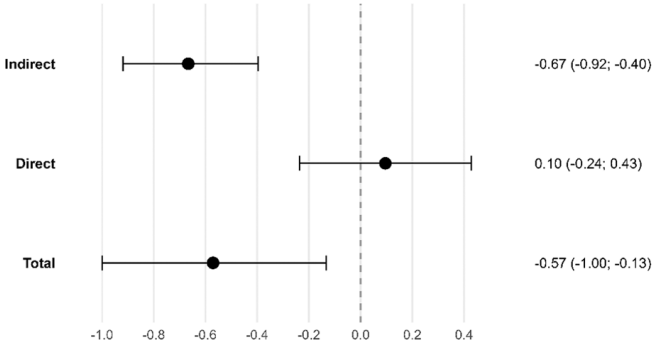


Fig. 2. Effect of bDMARDs on BASFI decomposed into indirect (through ASDAS), direct and total effect. Values are the average treatment effect and 95% confidence intervals. The non-significant direct effect suggests that the indirect effect (reduction of ASDAS) entirely explains the total effect.

P150

POTENTIAL DIFFICULT-TO-MANAGE PHENOTYPE AMONG AXIAL SPONDYLOARTHRITIS PATIENTS NON-ELIGIBLE FOR JANUS KINASE INHIBITORS

Silva C.[1], Rua C.[1], Patela M.[1], Fernandes M.A.[1], Vieira R.[1,2], Meirinhos T.[1], Pinto A.S.[1]
[1]Rheumatology Dept., Unidade Local de Saúde de Gaia/Espinho, Vila Nova de Gaia; [2]Dept. of Medical Sciences, University of Aveiro, Aveiro, Portugal

Introduction. The 2025 ASAS difficult-to-manage (D2M) axial spondyloarthritis (axSpA) definition identifies patients with persistent symptoms and active inflammation. Current cardiovascular safety recommendations regarding Janus kinase inhibitors (iJAK) may further restrict therapeutic options. We aimed to compare axSpA patients eligible and non-eligible for iJAK therapy and assess the presence of a potential D2M phenotype in non-eligible patients.
Methods. A cross-sectional study including axSpA patients receiving b/tsDMARDs followed at our center was conducted. Patients currently or previously treated with iJAK and those fulfilling the 2025 ASAS D2M definition were excluded. Non-eligibility for iJAK was defined as age ≥65 years, current or previous smoking, history of ischemic heart disease and/or stroke, and/or presence of ≥2 traditional cardiovascular risk factors (CVRF) (hypertension, dyslipidaemia, diabetes mellitus, and obesity). Potential D2M phenotype (adapted from the ASAS framework) required both active domain 2 (ASDAS ≥2.1, Tender Joint Count ≥1, CRP ≥0.5 mg/dL, and/or active sacroiliac joint MRI inflammation) and active domain 3 criteria (PtGA/PhGA ≥5).
Results. A total of 126 patients were included: 82 (65.1%) were non-eligible for iJAK therapy and 44 (34.9%) eligible (Fig. 1). In the non-eligible group, 18.3% were aged ≥65 years, 36.6% had ≥2 CVRF, 49.4% were current smokers, 24.7% previous smokers, 2.4% had ischemic heart disease, and 1.2% had prior stroke (Table I). Disease activity and functional measures were comparable between groups, with no significant differences regarding active domain 2 or 3 criteria. Among non-eligible patients, 36.1% (22/61) fulfilled criteria suggestive of a potential D2M phenotype.
Conclusion. Most axSpA patients were non-eligible for iJAK therapy due to cardiovascular safety-related restrictions. More than one-third of evaluable non-eligible patients fulfilled criteria suggestive of a potential D2M phenotype, suggesting these restrictions may contribute to progression toward D2M disease.

Table I. Baseline characteristics according to iJAK eligibility.

Variable	Non-Elegible (n=82)	Elegible (n=44)	Total (n=126)	p-value
Male	54 (65.9)	20 (45.5)	74 (58.7)	0.027
Age, years	51 (42-62)	41 (35-53)	48 (40-57)	<0.001
Age ≥65 years	15 (18.3)	0 (0)	15 (11.9)	0.003
Cardiovascular data				
Hypertension	24 (29.3)	3 (6.8.)	27 (21.4)	0.003
Dyslipidemia	30 (36.6)	3 (6.8)	33 (26.2)	<0.001
Diabetes mellitus	14 (17.1)	0 (0)	14 (11.1)	0.002
Body mass index	25.6 (22.9-29.6)	23.4 (22.4-36.3)	25.1 (22.7-28.1)	<0.001
Obesity	20 (24.4)	6 (13.6)	26 (20.6)	0.155
≥2 CVRF	30 (36.6)	0 (0)	30 (23.8)	<0.001
Current smoker	40 (49.4) (n=81)	0 (0) (n=44)	40 (32.0) (n=125)	<0.001
Previous smoker	20 (24.7) (n=81)	0 (0) (n=44)	20 (16.0) (n=125)	<0.001
Ischemic heart disease	2 (2.4)	0 (0)	2 (1.6)	0.542
Stroke	1 (1.2)	0 (0)	1 (0.8)	1.000
Disease-related data				
Median disease duration, years	15 (9-25) (n=81)	10 (6-20)	12 (8-23) (n=125)	0.012
Radiographic axSpA	711 (86.6)	31 (70.5)	102 (81.0)	0.028
HLA-B27 positivity	41 (53.9) (n=76)	20 (51.3) (n=39)	61 (53.0) (n=115)	0.786
Peripheral arthritis	22 (26.8)	14 (31.8)	36 (28.6)	0.555
Enthesitis	19 (23.2)	5 (11.4)	24 (19.0)	0.108
Dactylitis	1 (1.2)	1 (2.3)	2 (1.6)	1.000
Psoriasis	7 (8.5)	3 (6.8)	10 (7.9)	1.000*
Uveitis	14 (17.1)	7 (15.9)	21 (16.7)	0.867
Inflammatory bowel disease	14 (17.1)	8 (18.2)	22 (17.5)	0.876
Treatment-related data				
Anti-TNFα exposure	76 (92.7)	41 (93.2)	117 (92.9)	0.607
IL-17 inhibitor exposure	2 (2.4)	0 (0)	2 (1.6)	
Anti-TNFα + IL-17 inhibitor exposure	4 (4.9)	3 (6.8)	7 (5.6)	
Disease activity and functional status measure				
CRP, mg/dL	0.2 (0.1-0.4)	0.1 (0.5-0.3)	0.1 (0.6-0.4)	0.114
CRP ≥0.5 mg/dL	18 (22.0)	8 (18.2)	26 (20.6)	0.618
ASDAS-CRP	1.8 (1.2-2.7) (n=59)	1.6 (1.1-2.6) (n=30)	1.8 (1.2-2.6) (n=89)	0.350
ASDAS-CRP ≥2.1	22 (37.3) (n=59)	11 (36.7) (n=30)	33 (37.1) (Nn=89)	0.954
BASDAI	3 (1.4-5) (n=61)	2 (0.6-4.6) (n=31)	2.9 (0.9-4.9) (n=92)	0.299
BASDAI ≥4	23 (37.7) (n=61)	9 (29.0) (n=31)	32 (34.8) (n=92)	0.409
BASFI	2.5 (0-5.2) (n=54)	1 (0-4.4) (n=27)	1.8 (0-5) (n=81)	0.277
Active inflammation on sacroiliac joint MRI	21 (87.5) (n=24)	23 (92.0) (n=25)	44 (89.8) (n=49)	0.667
Tender Joint Count ≥1	6 (8.3) (n=72)	1 (2.6) (n=39)	7 (6.3) (n=111)	0.418
PtGA	2 (0.8-5) (n=62)	1 (0-4.5) (n=33)	2 (0-5) (n=95)	0.091
PhGA	1 (0-2) (n=60)	0.9 (0-1.6) (n=34)	1 (0-2) (n=94)	0.611
Active domain 2	45 (67.2) (n=67)	31 (81.6) (n=38)	76 (72.4) (n=76)	0.112
Active domain 3	26 (42.6) (n=61)	10 (29.4) (n=34)	36 (37.9) (n=95)	0.203

Values are expressed as n (%) or median (IQR). N represents the number of patients with available data. Categorical variables were compared using Chi-square or Fisher's exact test, as appropriate. Continuous variables were compared using the Mann-Whitney U-test.
A *p*-value <0.05 was considered statistically significant.
ASDAS-CRP: Ankylosing Spondylitis Disease Activity Score using C-reactive protein; axSpA: axial spondyloarthritis; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index; CRP: C-reactive protein; CVRF: cardiovascular risk factors; HLA-B27: human leukocyte antigen B27; iJAK: Janus kinase inhibitor; IL-17: interleukin-17; IQR: interquartile range; PhGA: Physician Global Assessment; PtGA: Patient Global Assessment; TNF: tumour necrosis factor.

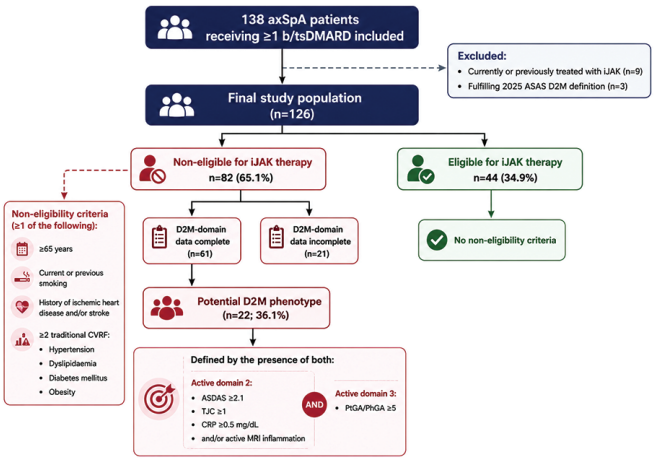


Fig. 1. Study flowchart and identification of patients with a potential D2M phenotype.
ASAS: Assessment of SpondyloArthritis International Society; ASDAS: Ankylosing Spondylitis Disease Activity Score; axSpA: axial spondyloarthritis; b/tsDMARD: biologic/targeted synthetic disease-modifying antirheumatic drug; CRP: C-reactive protein; CVRF: cardiovascular risk factors; D2M: difficult-to-manage; iJAK: Janus kinase inhibitor; MRI: magnetic resonance imaging; PhGA: physician global assessment; PtGA: patient global assessment; TJC: tender joint count.

P151

THE IMPACT OF MEDITERRANEAN DIET AND NUTRITIONAL FACTORS ON DISEASE ACTIVITY IN AXIAL SPONDYLOARTHRITIS: A LITERATURE REVIEW

Radic M.[1,2], Simac Prizmic P.[1], Radic J.[2,3], Matijas M.[1], Vodanovic A.[1], Perkovic D.[1,2]
[1]Dept. of Internal Medicine, Division of Rheumatology, Allergology and Clinical Immunology, Center of Excellence for Systemic Sclerosis in Croatia, University Hospital of Split, Split; [2]Internal Medicine Dept., School of Medicine, University of Split, Split; [3]Dept. of Internal Medicine, Division of Nephrology, Dialysis and Arterial Hypertension, University Hospital of Split, Split, Croatia

Introduction. Nutritional factors are increasingly recognized as potentially modifiable contributors to systemic inflammation in axial spondyloarthritis (axSpA). However, the relationship between Mediterranean diet (MD) adherence, dietary quality, and disease activity remains poorly characterized. We aimed to review the available evidence on the associations between MD and other nutritional factors and disease activity and inflammatory outcomes in axSpA.
Materials and methods. A systematic literature search was conducted in PubMed, Web of Science, Scopus, and the Cochrane Library from database inception to 1 Jun 2026, in accordance with PRISMA 2020 guidelines. Original studies evaluating dietary habits, nutritional interventions, dietary biomarkers, gut microbiota, or inflammatory outcomes in axSpA were screened. Of 348 identified records, 12 original studies were deemed relevant for qualitative synthesis (Fig. 1).

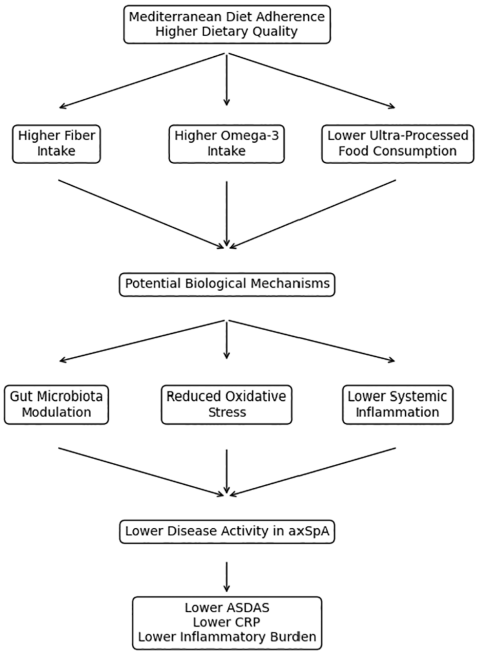


Fig. 1. Potential mechanisms linking Mediterranean diet and nutritional factors to disease activity in axial spondyloarthritis.

Results. The included studies were heterogeneous and primarily observational. Overall, the evidence suggested that better adherence to the MD, higher dietary quality, and favorable nutritional patterns were associated with lower disease activity and inflammatory burden in axSpA, although findings were not uniform. MD adherence and higher-quality dietary patterns were linked to lower disease activity, whereas lower omega-3 and fiber intake and higher consumption of ultra-processed foods were associated with worse outcomes (1-3). Additional studies suggested that fatty acid profiles, oxidative stress, and the gut microbiota may contribute to disease activity.
Conclusion. Current evidence suggests that adherence to the MD and favorable nutritional patterns may be associated with lower disease activity and inflammatory burden in axSpA. However, causality remains unproven, and prospective dietary intervention studies are needed to clarify these associations and ultimately integrate nutrition-based recommendations into the management of axSpA.

References
1. OMETTO F. ORTOLAN A, FARBER D: Mediterranean diet in axial spondyloarthritis: an observational study in an Italian monocentric cohort. *Arthritis Res Ther* 2021; 23: 219.
2. VERGNE-SALLE P, SALLE L. FRESSINAUD-MARIE AC: Diet and disease activity in patients with axial spondyloarthritis: SpondyloArthritis and NUTrition Study (SANUT). *Nutrients* 2022; 14: 4730.
3. HULANDER E, HALLSTRÖM M, FELDTHUSEN C: Dietary intake is related to disease activity and inflammation in radiographic axial spondyloarthritis: a cross-sectional multi-regional study. *Scand J Rheumatol* 2025; 54: 346-55.

P152

COMPARATIVE REAL-WORLD EFFECTIVENESS, DRUG SURVIVAL, AND PATIENT-REPORTED OUTCOMES OF SUBCUTANEOUS INFLIXIMAB STRATEGIES IN AXIAL SPONDYLOARTHRITIS: A PROSPECTIVE COHORT STUDY

Skouvaklidou E., Deligeorgakis D., Mpitouli A., Tsafis K., Kougkas N.
Fourth Dept. of Internal Medicine, Aristotle University of Thessaloniki, Hippokration Hospital, Thessaloniki, Greece

Introduction. Tumor necrosis factor inhibitors have significantly improved outcomes for patients with axial spondyloarthritis (axSpA). Subcutaneous (SC) infliximab (IFX) offers increased convenience and independence through non-hospital administration.
Objective. To compare treatment persistence, effectiveness, safety, patient-reported outcomes and work-related outcomes, between patients in low disease activity or remission switching from IV to SC IFX and initially managed with SC IFX at a comparable index date.
Results. Fifty-three patients (55% males) were included with a mean (SD) age and disease duration of 42.36 (13.04) and 7.08 (4.80) years respectively (Table I). Twenty-two patients were initially prescribed SC IFX while 31 patients switched from IV to SC treatment. Across the 1-year follow-up, ASDAS, BASDAI and CRP did not differ significantly between groups. However, ASDAS and BASDAI scores in the Switch group showed greater improvement than the SC group in a time × group interaction analysis, at 6 months (only BASDAI, p=0.006) and 12 months (ASDAS p=0.002, BASDAI p<0.001). EQ-VAS (p=0.40) and HAQ (p=0.82) were comparable at baseline; however, a time × group interaction was observed in the Switch group (p=0.023 at 3 months; p=0.046 overall). Work productivity outcomes improved over time across groups, with greater improvement in activity impairment in the Switch group at 6–12 months (p=0.02, Fig. 1). Cox proportional hazards regression analysis identified psoriasis as a predictor of discontinuation of SC IFX (HR: 9.01, 95% CI 1.05–77.2, p=0.045). No significant differences in discontinuation risk were observed between groups. The SC IFX was well tolerated with only 7 (13%) patients discontinuing treatment.
Conclusion. SC IFX was effective and well tolerated in axSpA. Switching to SC IFX may improve disease scores and work productivity over time. Psoriasis was associated with increased risk of SC IFX discontinuation.

Table I. Baseline and disease characteristics of patients initiating subcutaneous infliximab (SC IFX) or switching from intravenous to SC IFX.

Variables	SC start (n=22)	Switch to SC (n=31)	p-value
Age[a]	39.45 (11.95)	44.42 (13.57)	0.18[b]
Disease duration[a]	5.95 (3.59)	7.87 (5.41)	0.16[b]
Sex (Male)	14 (64%)	15 (48%)	0.27[c]
HLA-B27	12 (55%)	9 (29%)	0.06[c]
r- axSpA	11 (50%)	18 (58%)	0.56[c]
Psoriasis	7 (32%)	13 (42%)	0.45[c]
IBD	5 (23%)	10 (32%)	0.45[c]
Prior bDMARD	11 (50%)	19 (61%)	0.41[c]
NSAID	6 (27%)	8 (26%)	0.90[c]
cDMARD	5 (23%)	8 (26%)	0.79[c]
Adverse events	4 (18%)	6 (19%)	0.59[c]
Leading to drug discontinuation	4 (18%)	3 (10%)	0.70[d]
Injection site reaction	0	3 (10%)	0.29[d]
Preference			
Yes	15 (68%)	23 (74%)	
No	1 (5%)	5 (16%)	0.64[d]
No preference	6 (27%)	3 (10%)	

[a] mean (SD), [b] student's t-test, [c] chi squared, [d] Fisher's exact test.
HLA-B27: human leukocyte antigen B27; r-axSpA: radiographic axial spondyloarthritis; IBD: inflammatory bowel disease; bDMARD: biologic disease-modifying antirheumatic drug; cDMARD: conventional disease-modifying antirheumatic drug; NSAID: nonsteroidal anti-inflammatory drug.

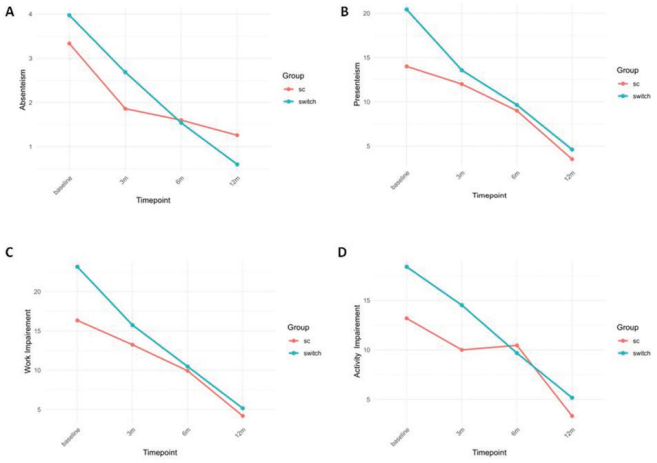


Fig. 1. Work productivity outcomes over time by treatment group.

P153

PATIENT-INITIATED FOLLOW-UP SUPPORTED BY ASYNCHRONOUS TELEMEDICINE IN SPONDYLOARTHRITIS: SUBGROUP ANALYSIS OF A RANDOMISED CONTROLLED TRIAL

Smits M.L.[1,2], Salmani H.[2], Boonen A.[1,2], Hermans K.[2,3,4], Vonkeman H.E.[5,6], van Tubergen A.[1,2], Webers C.[1,2,7]
[1]Dept. of Rheumatology, Maastricht University Medical Centre+, Maastricht; [2]Care and Public Health Research Institute (CAPHRI), Maastricht University, Maastricht, The Netherlands; [3]Dept. of Rheumatology and Systemic Diseases, Universitair Ziekenhuis Brussel (UZ Brussel), Brussels; [4]Vitality Research Group, Vrije Universiteit Brussel (VUB), Brussels, Belgium; [5]Dept. of Rheumatology and Clinical Immunology, Medisch Spectrum Twente, Enschede; [6]Dept. of Technology, Human and Institutional Behaviour, University of Twente, Enschede; [7]Dept. of Epidemiology, Maastricht University, Maastricht, The Netherlands

Introduction. The TeleSpA RCT demonstrated that combined patient-initiated follow-up (PIFU) and asynchronous telemedicine (TM) reduced the number of outpatient visits in patients with stable spondyloarthritis (SpA) by 25% versus usual care (UC) [1]. To uphold equitability, it is important to investigate whether PIFU/TM is suitable for all patients. This pre-specified subgroup analysis of TeleSpA aimed to evaluate whether PIFU/TM is consistently effective within subgroups of patients with SpA with different characteristics.
Methods. TeleSpA included 200 patients randomised to PIFU/TM (once-yearly pre-planned visit with TM at 6 months) or UC (visits at rheumatologist's discretion). The primary outcome was a between-group difference ≥25% in number of outpatient visits after 1 year between the PIFU/TM and UC arms. Secondary outcomes were non-inferiority of PIFU/TM versus UC regarding disease activity (ASDAS/BASDAI/pain/patient and physician global) and overall experience with care. This analysis compared patients in the two arms within stratified subgroups: sex, subtype (axial [axSpA]/peripheral [pSpA]), baseline treatment (biological user/non-user), age (quartiles), symptom duration (<10/≥10 years).
Results. In the overall PIFU/TM and UC arms, respectively, 38% and 41% were female; mean age was 55.8 (SD12.3) and 54.2 (SD11.4) years; and mean symptom duration was 18.3 (SD12.4) and 15.8 (SD8.3) years. In both arms, 46% had axSpA and 54% had pSpA, and 62% used a biological. For the primary outcome, reduced outpatient visits in the PIFU/TM versus UC arm was observed in all subgroups, however, magnitudes varied (Table I).

Table I. Primary outcome of the TeleSpA trial, stratified by subgroups.

Subgroup	Number of visits in UC arm: mean (SD)	Number of visits in PIFU/TM arm: mean (SD)	Mean adjusted difference between arms (95%CI)	Percentage difference
Sex				
Male (n=121)	2.8 (1.5)	1.9 (1.6)	-0.8 (-1.4 to -0.2)	**-30.2%**
Female (n=79)	2.4 (0.8)	2.1 (1.2)	-0.4 (-0.9 to 0.1)	-17.8%
Disease subtype				
axSpA (n=92)	2.7 (1.2)	1.8 (1.2)	-0.8 (-1.3 to -0.3)	**-31.3%**
pSpA (n=108)	2.6 (1.4)	2.1 (1.7)	-0.5 (-1.1 to 0.1)	-20.5%
Baseline treatment				
bDMARD users (n=124)	2.7 (1.5)	1.9 (1.2)	-0.9 (-1.4 to -0.4)	**-31.6%**
bDMARD non-users (n=76)	2.4 (0.9)	2.0 (1.8)	-0.4 (-1.1 to 0.3)	-18.0%
Age				
1st quartile (≤48 years) (n=50)	2.6 (1.5)	1.8 (1.2)	-0.7 (-1.7 to 0.2)	**-28.0%**
2nd quartile (49-54 years) (n=50)	2.7 (1.8)	2.0 (1.9)	-0.6 (-1.8 to 0.5)	-24.2%
3rd quartile (55-63 years) (n=50)	2.4 (0.9)	2.4 (1.4)	0.0 (-0.7 to 0.7)	-1.8%
4th quartile (≥64 years) (n=50)	2.7 (1.0)	1.6 (1.1)	-0.9 (-1.6 to -0.3)	**-34.3%**
Symptom duration				
<10 years (n=53)	2.5 (1.0)	1.9 (1.6)	-0.7 (-1.5 to 0.1)	**-27.2%**
≥10 years (n=144)	2.6 (1.4)	2.0 (1.5)	-0.8 (-1.2 to -0.3)	**-29.0%**

Differences between the PIFU/TM and UC arms were adjusted for randomisation minimisation factors (centre, disease subtype, bDMARD use) and relevant baseline differences, as appropriate for each analysis. Results fulfilling the primary outcome of TeleSpA (≥25% difference between arms in the number of rheumatology outpatient visits after 1 year) are in **bold**.
axSpA: axial spondyloarthritis; bDMARD: biological disease-modifying anti-rheumatic drug; PIFU: patient-initiated follow-up; pSpA: perypheral spondyloarthritis; TM: telemedicine; UC: usual care.

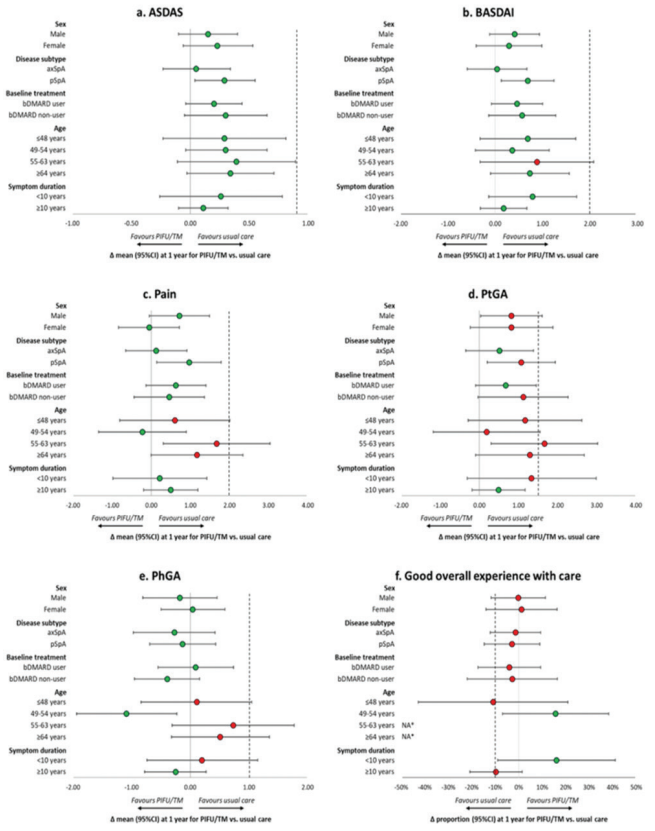


Fig. 1. Forest plots of the secondary outcomes of the TeleSpA trial: (**a**) ASDAS, (**b**) BASDAI, (**c**) pain, (**d**) PtGA, (**e**) PhGA, (**f**) overall experience with care, stratified by subgroups.
Non-inferiority margins for each outcome are represented by the dotted lines: 0.9 (ASDAS), 2.0 (BASDAI), 2.0 (pain), 1.5 (PtGA), 1.0 (PhGA), -10% (good overall experience with care). Differences between the PIFU/TM and usual care arms were adjusted for randomisation minimisation factors (centre, disease subtype, bDMARD use) and relevant baseline differences, as appropriate for each analysis. Analyses where non-inferiority of PIFU/TM was demonstrated are presented in **green**, analyses where non-inferiority was not demonstrated are presented in **red**.
*Effect size for overall experience with care could not be calculated in the third and fourth age quartiles due to perfect prediction (within these subgroups, all patients in the usual care arm reported having a good overall experience with care after 1 year; in the PIFU/TM arm, this was reported by 80% and 96% of patients in the third and fourth age quartiles, respectively).
ASDAS: Axial Spondyloarthritis Disease Activity Score; axSpA: axial spondyloarthritis; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; bDMARD: biological disease-modifying anti-rheumatic drug; PhGA: physician global assessment of disease activity; PIFU: patient-initiated follow-up; pSpA: peripheral spondyloarthritis; PtGA: patient global assessment of disease activity; TM: telemedicine.

Regarding secondary outcomes, non-inferiority was demonstrated for ASDAS/BASDAI/pain/physician global in most subgroups, but this was inconsistent for other outcomes (Fig. 1).
Conclusion. PIFU/TM reduced outpatient visits in stable SpA, particularly in certain subgroups: males, axSpA subtype, biological users, youngest/oldest age quartiles, and across all symptom durations. Non-inferiority regarding health outcomes was not universally demonstrated. This study provides a promising indication that PIFU/TM can be applied across varied populations.
Acknowledgements. The authors thank all participants and study centre personnel from Maastricht University Medical Centre+ and Medisch Spectrum Twente. This study was supported by a grant from the Dutch Arthritis Society, project number 19-2-203.

Reference

1. HERMANS K, WEBERS C, BOONEN A *et al*.: Patient-initiated follow-up supported by asynchronous telemedicine versus usual care in spondyloarthritis (TeleSpA-study): a randomised controlled trial of clinical and cost-effectiveness. *Lancet Rheumatol* 2024; 6(12); E848-E859.

P154

THE ASAS CORE SET FOR THE ASSESSMENT OF AXIAL SPONDYLOARTHRITIS IN DAILY PRACTICE

van Tubergen A.[1,2], Smits M.L.[1,2], Baraliakos X.[3], Moltó A.[4,5], Nikiphorou E.[6,7,8], Navarro-Compán V.[9]
[1]Dept. of Rheumatology, Maastricht University Medical Centre+, Maastricht; [2]Care and Public Health Research Institute (CAPHRI), Maastricht University, Maastricht, The Netherlands; [3]Rheumazentrum Ruhrgebiet, Herne, Ruhr-Universität Bochum, Bochum, Germany; [4]Centre for Research in Epidemiology and Statistics, INSERM, INRAE, Université Paris Cité and Université Sorbonne Paris Nord, Paris; [5]Rheumatology Dept., Bichat Hospital, AP-HP. Nord, Paris, France; [6]Centre for Rheumatic Diseases, Faculty of Life Sciences and Medicine, School of Immunology and Microbial Sciences, King's College London, London; [7]Centre for Education, Faculty of Life Sciences and Medicine, King's College London, London; [8]Dept. of Rheumatology, King's College Hospital NHS Foundation Trust, London, UK; [9]Dept. of Rheumatology, La Paz University Hospital, IdiPAZ, Madrid, Spain

Introduction. Management recommendations for axial spondyloarthritis (axSpA) advocate for holistic disease assessments in clinical practice, structured around 'routine' visits and 'extended' visits (*i.e.* more comprehensive annual/periodic reviews). However, validated guidelines on the content of assessments are not yet available. This Assessment of SpondyloArthritis international Society (ASAS) initiative aimed to develop a core set for daily clinical practice to improve the standardisation and feasibility of the assessment of axSpA.
Methods. The core set development consisted of several phases (Fig. 1). A scoping literature review was conducted to identify candidate domains, which informed a subsequent Delphi survey. Three stakeholder groups were included: ASAS members, non-ASAS member rheumatologists, and patients with axSpA. Working group meetings (23 members, including two patient representatives) took place to iteratively discuss the project progression, and construct the core set based on the Delphi survey. The proposed core set was discussed with the ASAS community to reach consensus and voted on for endorsement.

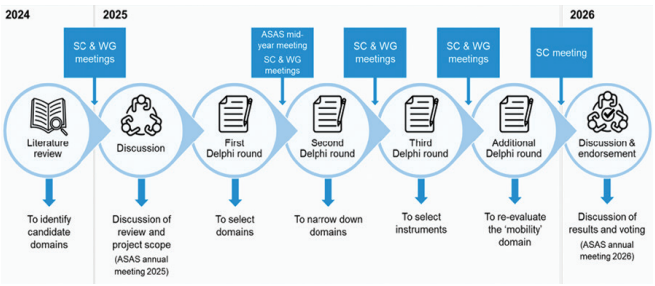


Fig. 1. Development process of the ASAS core set for the assessment of axial spondyloarthritis in daily practice.
ASAS: Assessment of SpondyloArthritis International Society; SC: steering committee; WG: working group.

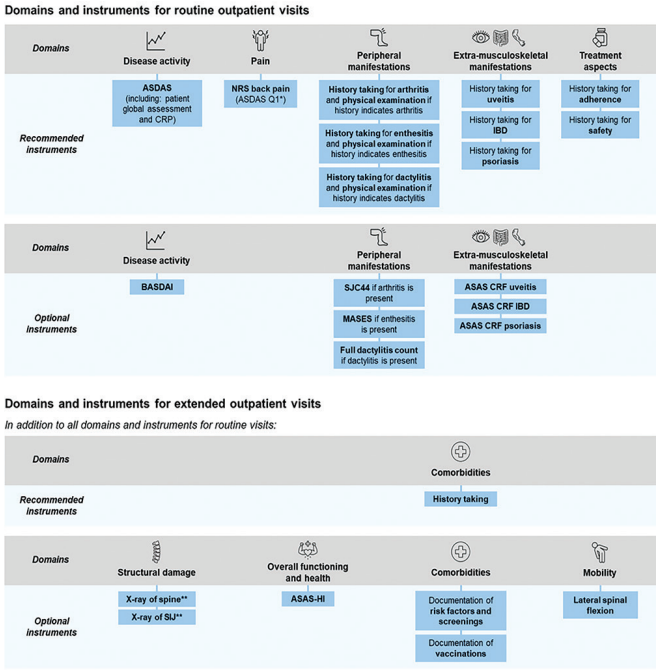


Fig. 2. The ASAS core set for the assessment of axial spondyloarthritis in daily practice. *ASDAS question 1 is also a component of the BASDAI (BASDAI question 2). **Evaluated at an interval of at least 2 years, unless another cause of the back pain is suspected. ASAS: Assessment of SpondyloArthritis International Society; ASAS-HI: ASAS Health Index; ASDAS: Axial Spondyloarthritis Disease Activity Score; BASDAI: Bath Ankylosing Spondyloarthritis Disease Activity Index; CRF: case report form; CRP: C-reactive protein; IBD: inflammatory bowel disease; MASES: Maastricht Ankylosing Spondylitis Enthesitis Score; NRS: numerical rating scale; SIJ: sacroiliac joints; SJC44: 44-swollen joint count.

Results. The literature review identified heterogeneous candidate domains to inform the first Delphi round, which included 156/201 (78%) ASAS members, 173 non-ASAS rheumatologists, and 167 patients, representing academic and non-academic healthcare institutions across 50 countries. Following the Delphi rounds and discussion with the ASAS community, endorsement by a majority of ASAS members (61% in favour) was achieved at the 2026 annual meeting (Fig. 2). Five domains are included for routine visits: 'disease activity', 'pain', 'peripheral manifestations', 'extra-musculoskeletal manifestations', and 'treatment aspects', with one instrument recommended for each domain, alongside several optional instruments. Four supplementary domains are included for extended visits: 'structural damage', 'overall functioning and health', 'comorbidities', and 'mobility', with at least one recommended or optional instrument for each domain.

Conclusion. The ASAS core set for daily practice represents the minimum to be assessed in clinical settings, contributing toward improved standardisation and feasibility of disease assessments in axSpA.

Acknowledgements. The ASAS-PRAXIS steering committee and working group: Astrid van Tubergen, Victoria Navarro-Compán, Marius Smits (fellow), Xenofon Baraliakos, Anna Moltó, Elena Nikiphorou, Wilson Bautista-Molano, Philippe Carron, Adrian Ciurea, Jo Davies, Warren Fong, Rodrigo García-Salinas, Laure Gossec, Désirée van der Heijde, David Kiefer, Uta Kiltz, Yu Heng Kwan, Jo Lowe, Marina Magrey, Denis Poddubnyy, Sofia Ramiro, Alexandre Sepriano, Nelly Ziade. This project was funded by ASAS.

P155

REVISED ASAS-SPARTAN 2025 CLASSIFICATION CRITERIA AND TREATMENT OUTCOMES IN AXIAL SPONDYLOARTHRITIS: EVIDENCE FROM A LARGE OBSERVATIONAL REGISTRY

Steimer A.[1], Brändli J.[2], Aeberli D.[3,7], Andor M.[4], Bürki K.[1], Kyburz D.[5], Nissen M.J.[6], Möller B.[7], Adler S.[7,8], Rodrigues C.L.[2], Grosswiler S.[9], Dan D.[10], Brülhart Bletsas L.[11], Förger F.[12], Distler O.[1], Micheroli R.[1], Ciurea A.[1]

[1]Dept. of Rheumatology, University Hospital Zurich, University of Zurich, Zurich; [2]Swiss Clinical Quality Management in Rheumatic Diseases Foundation, Zurich; [3]Division of Rheumatology, Dept. of Internal Medicine, Spital Emmental, Burgdorf; [4]Rheumatologie im Zürcher Oberland, Uster; [5]Dept. of Rheumatology, University Hospital Basel, University of Basel, Basel; [6]Division of Rheumatology, Geneva University Hospital, Geneva; [7]Dept. of Rheumatology and Immunology, Inselspital, Bern; [8]Dept. of Rheumatology, Cantonal Hospital Aarau, Aarau; [9]Swiss Ankylosing Spondylitis Association, Zurich; [10]Dept. of Rheumatology, Lausanne University Hospital, Lausanne; [11]Division of Rheumatology, La Chaux-de-Fonds Hospital, La Chaux-de-Fonds; [12]Dept. of Rheumatology, Cantonal Hospital St. Gallen, St. Gallen, Switzerland

Introduction. In 2025 revised ASAS-SPARTAN classification criteria for axial spondyloarthritis were developed1 with increased sensitivity and specificity compared to the ASAS 2009 classification criteria.

Materials and methods. We compared patients diagnosed with axSpA from the SCQM registry initiating their first tumour necrosis factor inhibitor (TNFi) who fulfilled only the ASAS 2009 classification criteria with those fulfilling both classification criteria (ASAS 2009 and ASAS-SPARTAN 2025). Baseline characteristics were compared with the Mann-Whitney-U-test and Fisher's exact test. The achievement of a 40% improvement according to ASAS criteria (ASAS40), Axial Spondyloarthritis Disease Activity Score <2.1, ASDAS clinically important improvement (ASDAS-CI) and ASDAS major improvement (ASDAS-MI) were analysed at 1 year after treatment initiation. Analyses were adjusted for age and sex.

Table I. Baseline characteristics of patients fulfilling the ASAS 2009 classification criteria for axSpA, stratified by additional fulfilment of the ASAS-SPARTAN 2025 classification criteria.

Variable	Available (n)	ASAS-SPARTAN 2025 Fulfilled	ASAS-SPARTAN 2025 Not fulfilled	p-value
n		839	347	
Male sex, n (%)	1186 (347/839)	523 (62.3)	176 (50.7)	<0.001
Age, years (mean (SD))	1186 (347/839)	38.69 (10.88)	41.36 (11.95)	<0.001
HLA-B27, n positive (%)	1106 (302/804)	663 (82.5)	214 (70.9)	<0.001
Elevated CRP, n (%)	899 (265/634)	347 (54.7)	126 (47.5)	0.06
ASDAS (mean (SD))	728 (211/517)	3.34 (0.97)	3.36 (0.84)	0.989
BMI [kg/m2] (median [IQR])	1099 (315/784)	24.81 [22.30, 27.78]	24.39 [21.98, 27.33]	0.26
Symptom duration, years (median [IQR])	1179 (346/833)	9.64 [4.30, 18.66]	10.89 [4.66, 20.60]	0.20
Sacroiliitis in MRI, positive (%)	886 (261/625)	382 (61.1)	63 (24.1)	<0.001
Fulfilled modified New York criteria, n (%)	734 (151/583)	489 (83.9)	69 (45.7)	<0.001
Inflammatory back pain, n (%)	1186 (347/839)	483 (57.6)	138 (39.8)	<0.001
Peripheral arthritis, n (%)	1184 (346/838)	304 (36.3)	135 (39.0)	0.39
Inflammatory bowel disease, n (%)	948 (259/689)	61 (8.9)	17 (6.6)	0.29
Uveitis, n (%)	484 (128/356)	140 (39.3)	51 (39.8)	0.92
Heel enthesitis, n (%)	1180 (343/837)	267 (31.9)	125 (36.4)	0.14
Psoriasis, n (%)	963 (262/701)	70 (10.0)	23 (8.8)	0.63
Dactylitis, n (%)	1171 (340/831)	69 (8.3)	32 (9.4)	0.57
Family history for spondyloarthritis, n (%)	1002 (288/714)	273 (38.2)	116 (40.3)	0.57

ASAS: Assessment of SpondyloArthritis international Society; ASDAS: Ankylosing Spondylitis Disease Activity Score; BMI: body mass index; CRP: C-reactive protein; HLA-B27: human leukocyte B27; MRI: magnetic resonance imaging.

Table II. Multiple adjusted response rate analyses at 1 year of treatment with a first TNFi in patients fulfilling ASAS 2009 and ASAS-SPARTAN 2025 classification criteria vs. patients fulfilling only ASAS 2009 classification criteria.

	ASAS40	ASDAS<2.1	ASDAS-CII	ASDAS-MI
	OR (95%CI)	OR (95%CI)	OR (95%CI)	OR (95%CI)
ASAS 2009 and ASAS-SPARTAN 2005 fulfilled (Ref ASAS 2009 fulfilled)	1.66 (1.13 to 2.46)	1.73 (1.23 to 2.45)	1.71 (1.18 to 2.53)	1.61 (0.99 to 2.68)
Age	0.97 (0.97 to 1.00)	0.97 (0.96 to 0.99)	0.97 (0.95 to 0.98)	0.99 (0.97 to 1.00)
Sex	1.90 (1.35 to 2.69)	1.82 (1.34 to 2.27)	2.26 (1.61 to 3.19)	2.48 (1.60 to 3.95)

ASAS: Assessment of SpondyloArthritis international Society; ASAS40: 40% improvement according to ASAS criteria; ASDAS: Ankylosing Spondylitis Disease Activity Score; ASDAS-CII: ASDAS clinically important improvement; ASDAS-MI: ASDAS major improvement.

Results. Out of 1186 patients fulfilling the ASAS 2009 classification criteria, 347 patients fulfilled only ASAS 2009 criteria, whereas 839 patients fulfilled both ASAS 2009 and ASAS-SPARTAN 2025 criteria. Patients fulfilling both classification criteria, were more often male (62.3% vs. 50.7%), HLA-B27 positive (82.5% vs. 70.9%), had more often inflammatory sacroiliac changes on MRI (61.1% vs. 24.4%) and radiographic disease status

(83.9% vs. 45.7%) (Table I). In addition, patients fulfilling both classification criteria had significantly higher ASAS40, ASDAS<2.1 and ASDAS-CII response rates after 1 year compared to patients only fulfilling the ASAS 2009 classification criteria. The observed difference for ASDAS-MI was not statistically significant (Table II).

Conclusion. Fulfillment of the ASAS-SPARTAN 2025 classification criteria in addition to the ASAS 2009 criteria is associated with a better treatment response.

Reference

1. MAKSYMOWYCH W, VAN DER HEIJDE D, CAPLAN L *et al*.: The Assessments in Spondyloarthritis International Society (ASAS) and Spondyloarthritis Research and Treatment Network (SPARTAN) Revised Classification Criteria for Axial Spondyloarthritis: Development and Validation in the Classification of Axial SpA Inception Cohort Study [abstract]. *Arthritis Rheumatol* 2025; 77 (suppl 9).

P156

IMPACT OF SONELOKIMAB, A NOVEL IL-17A/F-INHIBITING NANOBODY, ON CLINICAL AND IMAGING OUTCOMES IN AXIAL SPONDYLOARTHRITIS (AXSPA): RESULTS OF A PHASE 2 STUDY SUPPORTED BY ^{18}F-NAF PET IMAGING AND MRI

Brikena L.[1,2], Kiltz U.[1,2], Sewerin P.[1,2], Hautzel H.[3], Ramming A.[4], Akbar-Haase R.[5], Millar V.[5], Nadimpalli M.[5], Thomas M.[5], Pereira A.[5], Cullen E.[5], Reich K.[5,6], Baraliakos X.[1,2]

[1]Dept. of Rheumatology, Rheumazentrum Ruhrgebiet, Herne; [2]Ruhr University Bochum, Bochum; [3]Dept. of Nuclear Medicine, University Hospital Essen, University of Duisburg-Essen, Essen; [4]Dept. of Internal Medicine 3, Rheumatology and Immunology, Friedrich-Alexander University and Universitätsklinikum Erlangen, Erlangen-Nürnberg, Germany; [5]MoonLake Immunotherapeutics AG, Zug, Switzerland; [6]Translational Research in Inflammatory Skin Diseases, Institute for Health Services Research in Dermatology and Nursing, University Medical Center Hamburg-Eppendorf, Hamburg, Germany

Introduction. Control of inflammation and osteoblastic activity is critical in axSpA. Sonelokimab is a ~40kDa Nanobody that binds IL-17A and IL-17F and is designed for enhanced distribution to inflamed joints. Here we report clinical and imaging outcomes from the Phase 2 S-OLARIS study, including ^{18}F-NaF PET/MRI assessment of osteoblastic activity.

Materials and methods. S-OLARIS (EUCT: 2024-513498-36-00) was a single-arm study in active axSpA per MRI and ^{18}F-NaF PET. Patients received sonelokimab 60 mg at Weeks [W] 0, 2, 4, 6, and 8. MRI and PET (background-corrected SUVmax; *os ilium* reference) was performed at screening and W12. Clinical outcomes (ASAS, ASDAS, BASDAI, BASFI) were evaluated to W12. Safety was assessed to W16.

Results. Twenty-six patients were treated (85% male; 65% r-axSpA; 73% HLA-B27+). Rapid achievement of ASAS responses was observed (Fig. 1). Mean (SE) ASDAS significantly improved from W4 (–2.0 [0.2]; p<0.001) onward, with 58% of patients achieving major improvement by W12. Mean (SE) BASDAI and BASFI decreased by 4.7 (0.3) and 3.3 (0.4) points at W12 (both p<0.001). MRI SPARCC scores significantly improved in the SIJ (p<0.001; Fig. 2A) and spine (p=0.03). Mean ^{18}F-NaF PET SUVmax in the SIJ decreased by 44% (p<0.001; Fig. 2B), with a 74% reduction in total quadrants showing both bone marrow edema and PET positivity (80/192 to 21/192). Fifty-four percent of patients experienced ≥1 TEAE (influenza 12%, fatigue 8%, CPK increase 8%). All TEAEs were mild-to-moderate and most were considered unrelated to treatment; no adverse events of IBD, MACE, SIB, liver injury, or uveitis were reported.

Conclusion. Sonelokimab treatment resulted in rapid clinical responses and significant improvements on objective imaging-based assessments of inflammation and pathological osteoblastic activity. Sonelokimab was well tolerated, with no new signals. These data provide encouraging proof of concept for sonelokimab in axSpA.

Acknowledgements. This study was sponsored by MoonLake Immunotherapeutics AG.

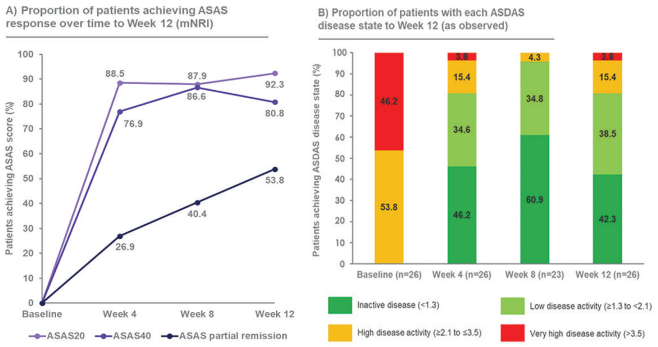


Fig. 1. Clinical efficacy outcomes to Week 12.
For panel B, percentages may sum to 100 due to rounding.
ASAS: Assessment of SpondyloArthritis International Society; ASDAS: Ankylosing Spondylitis Disease Activity Score; mNRI: modified non-resoponder imputation.

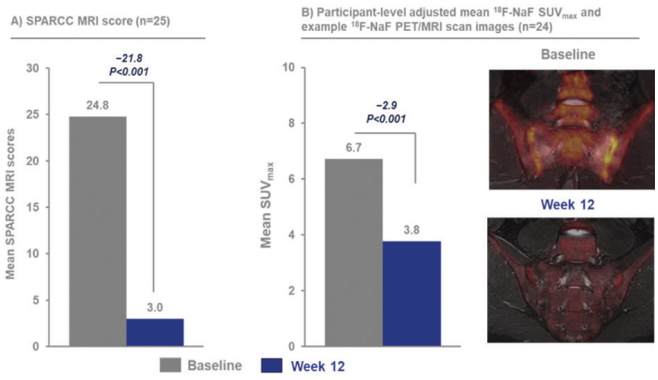


Fig. 2. MRI and 18F-NaF PET imaging outcomes in the SIJ.
A. Mean SPARCC MRI score in the SIJ at baseline and Week 12.
B. Mean ^{18}F-NaF SUV_{max} at baseline and Week 12, plus example images. Overall mean SUV_{max} was calculated from the mean SUV_{max} across all active SIJ quadrants within each participant, with adjustment to correct for background non-pathological uptake in reference tissue (*os ilium*).
^{18}F-NaF: fluorine-18-labeled sodium fluoride; MRI: magnetic resonance imaging; PET: positron emission tomography; SIJ: sacroiliac joint; SPARCC: Spondyloarthritis Research Consortium of Canada; SUV_{max} maximum standardized uptake value.

P157

PERSISTENCE, EFFECTIVENESS, SAFETY, AND PATIENT REPORTED IMPACT OF GUSELKUMAB AND IL-17 INHIBITORS IN PSORIATIC ARTHRITIS: FULL POPULATION RESULTS OF THE PSABIOND GLOBAL OBSERVATIONAL STUDY OVER 12 MONTHS

Gossec L.[1]*, Sharaf M.[2], Behrens F.[3], Lubrano E.[4], Kishimoto M.[5], Baraliakos X.[6], Silva R.Q.[7], Rampakakis E.[8,9], Köleséri L.[10], Lozenski K.[11], Soriano E.R.[12], Rahman P.[13], Siebert S.[14]

[1]Sorbonne Université, Pitié-Salpêtrière Hospital, Paris, France; [2]Johnson & Johnson, Dubai, United Arab Emirates; [3]Rheumatology and Fraunhofer IME - Translational Medicine and Pharmacology, Goethe University, Frankfurt, Germany; [4]Vincenzo Tiberio Dept. of Medicine and Health Sciences, University of Molise, Campobasso, Italy; [5]Dept. of Nephrology and Rheumatology, Kyorin University School of Medicine, Tokyo, Japan; [6]Ruhr-University Bochum, Rheumazentrum Ruhrgebiet, Herne, Germany; [7]Rheumatology Division & ISPA Translational Immunology Division, Hospital Universitario Central de Asturias, Oviedo University, Oviedo, Spain; [8]Dept. of Pediatrics, McGill University, Montreal, Canada; [9]JSS Medical Research, Inc, Montreal, Canada; [10]Data Sciences Staffing Solutions, IQVIA, Inc, Budapest, Hungary; [11]Johnson & Johnson, Horsham, USA; [12]Rheumatology Section, Internal Medicine Service, Hospital Italiano de Buenos Aires and University Institute Hospital Italiano de Buenos Aire, Buenos Aires, Argentina; [13]Faculty of Medicine, Division of Rheumatology, Memorial University of Newfoundland, St. Johns, Canada; [14]School of Infection & Immunity, University of Glasgow, Glasgow, UK

Introduction. Previous interim analysis of PsABIOnd showed similar 6-month(M) treatment persistence(TP)/effectiveness with guselkumab (GUS) and IL-17i (1). Here, we assessed 12M TP/effectiveness/safety/PROs of GUS and IL-17i in a real-world setting.

Methods. PsABIOnd (NCT05049798) is a global observational study in PsA participants starting GUS/IL-17i as 1st-to-4th line per SOC. The primary outcome is TP (2). Participants with a baseline and ≥1 follow-up assessment till the 12M-visit (±3M) were analyzed per the initial treatment. TP was assessed over 12M via Kaplan-Meier. Achievement rates of LDA/remission(REM) by cDAPSA(≤13)+DAPSA(≤14)/PsO BSA<3%+DLQI 0/1/MDA at 12M were assessed. Patient-reported impact was accessed using PsAID-12 (0-10) questionnaire. AEs occurring till the 12M-visit were summarized; exposure-adjusted incidence rates (EAIR;events/100 PYs) with 95%CI were reported.

Results. Overall, 1,141 participants received GUS (n=562) or IL-17i (n=579) as initial treatment. At baseline, mean-age=53.1/53.4y; mean-BMI=30.0/29.4kg/m^2;53.5%/62.7% participants received ≥1 non-GUS/IL-17i therapy; 15.3%/7.9% initiated 4th-line for GUS/IL-17i cohorts. 12M TP was high in both cohorts, with 76.0%GUS/77.0%IL-17i participants remaining on initial-treatment (propensity-score adjusted hazard-ratio GUS:IL-17i stop/switch [95%CI]: 0.89 [0.73-1.08]; Fig. 1).

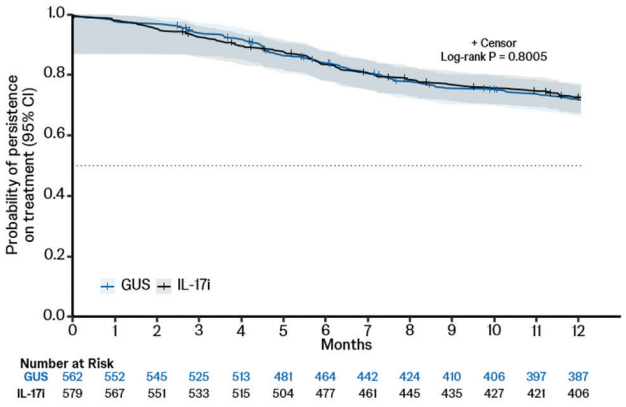


Fig. 1. Persistence on treatment over 12 months in PsA participants treated with GUS and IL-17i. Number at risk shows participants remaining under observation and switch/stop event-free at each time point; patients were right censored at their last observation or at study termination and were removed from these counts after their censoring time.

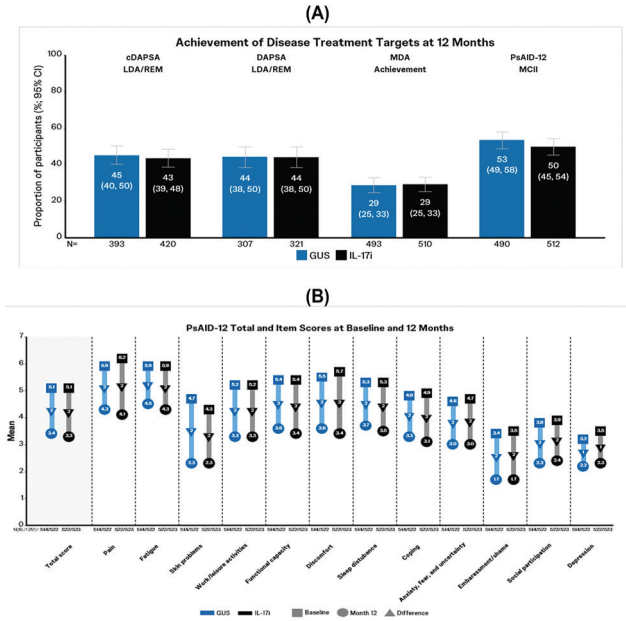


Fig. 2. A. PsA clinical and patient reported outcomes achievement at 12 months and **B.** PsAID-12 total and item scores at baseline and 12 months with GUS and IL-17i . Number of participants (N) indicated under x-axis correspond to the number of participants included in the respective analyses. Last observation carried forward was imputed for participants with no 12-month visit. CI: confidence interval.

Primary failure (GUS: 11.7%/IL-17i:9.8%) and AEs(GUS: 7.3%/IL-17i: 9.2%) were the main reasons for initial-treatment-line discontinuation. At 12M, improvements in joint (GUS/IL-17i;cDAPSA-LDA/REM: 45.0%/43.3%; DAPSA-LDA/REM: 44.0%/43.9%; Fig. 2A)/skin(BSA<3%: 77.7%/80.4%; DLQI 0/1:53.4%/54.7%)/overall DA (MDA: 28.6%/29.0%; Fig. 2A) were similar with GUS/IL-17i. The rate of PsAID-12 MCII achievement (53.3%/49.6%; Fig. 2A) and mean improvement in PsAID-12 total(-1.7/-1.8; Fig. 2B) and item scores were generally similar with GUS/IL-17i.

Through 12M, rates of participants experiencing ≥1 AE(GUS:53.3% (EAIR [95%CI]:116.6[108.1,125.5]/100PYs)/IL-17i:54.7% (156.0 [146.6,165.9]/100PYs)) and ≥1 serious AE(GUS:6.8%(9.4[7.1,12.2]/100PYs)/IL-17i: 6.9% (8.4 [6.3,10.9]/100PYs)) were similar between GUS and IL-17i. The most frequently observed AEs (≥5% participants) were COVID-19 infection (GUS: 7.5% [7.4(5.4,9.9)/100PYs]; IL-17i: 5.6% [5.6(4.0,7.8)/100PYs]) and nasopharyngitis (GUS: 5.3% [5.8(4.0,8.0)/100PYs]; IL-17i: 5.5% [6.9(5.0,9.2)/ 100PYs]). Gastrointestinal disorder-related AE occurred in GUS:6.8%(7.8[5.7,10.3]/100PYs) and IL-17i:11.2%(15.5[12.7,18.8]/100PYs). No cases of active tuberculosis occurred in either cohort.

Conclusion. 12M findings from PsABIOnd show GUS-/IL-17i-treated PsA participants had similar TP/effectiveness across key PsA domains, and improvements in PROs with both therapies. Safety profiles through 12M were comparable, with no new safety signals observed. These suggest both mechanisms-of-action as effective long-term PsA management in routine clinical practice, supporting efficacy data from RCTs.

Disclosures: LG: Research grants: AbbVie, Biogen, Eli Lilly, Novartis, and UCB; Consulting fees: AbbVie, AlfaSigma, Amgen, Bristol Myers Squibb, Celltrion, Johnson & Johnson, Eli Lilly, MSD, Novartis, Pfizer, Stada, and UCB. **MS**: Employee: Johnson & Johnson; Owns stock: Johnson & Johnson. **FB**: Research grants: Celgene, Chugai, Johnson & Johnson, Pfizer, and Roche; Consultant/speaker fees: AbbVie, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Chugai, Eli Lilly, Galapagos, Genzyme, Gilead, Johnson & Johnson, MSD, Novartis, Pfizer, Roche, Sanofi, and UCB. **EL**: Honoraria: AbbVie, Amgen, Eli Lilly, GlaxoSmithKline, Johnson & Johnson, Novartis, and UCB. **MK**: Consultant/speaker fees: AbbVie, Amgen, Asahi-Kasei Pharma, Ayumi, Bristol Myers Squibb, Chugai, Daiichi-Sankyo, Eisai, Eli Lilly, Gilead, Johnson & Johnson, Novartis, Tanabe-Mitsubishi, and UCB. **XB**: Speaker's bureau: AbbVie, Chugai, Eli Lilly, Johnson & Johnson, MSD, Novartis, Pfizer, Roche, and UCB; consultant: AbbVie, Chugai, Eli Lilly, Johnson & Johnson, MSD, Novartis, Pfizer, Roche, and UCB; Grant/research support: AbbVie, Eli Lilly, Johnson & Johnson, MSD, and Novartis. **RQS**: Speaker and/or consultancy fees: AbbVie, Amgen, Celgene, Johnson & Johnson, Eli Lilly, MSD, Novartis, and Pfizer. Restricted research grants: AbbVie, Johnson & Johnson, and Novartis. **ER**: Employee: JSS Medical Research; Consultant: Johnson & Johnson. **LK**: Employee: IQVIA; Consultant: Johnson & Johnson. **KL**: Employee: Johnson & Johnson; Owns stock: Johnson & Johnson and Bristol Myers Squibb. **ES**: Speaker's bureau: Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, Roche, and UCB; Consultant: AbbVie, Johnson & Johnson, Novartis, and Roche; Grant/research support: AbbVie, Johnson & Johnson, Novartis, Pfizer, Roche, and UCB. **PR**: Consultant: AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Johnson & Johnson, Merck, Novartis, Pfizer, and UCB; Meeting attendance/travel support: Johnson & Johnson; Research grants: Johnson & Johnson and Novartis. **SS**: Grant/research support: Eli Lilly, GlaxoSmithKline, Johnson & Johnson, Pfizer and UCB; Honoraria/speaker fees: AbbVie, Amgen, AstraZeneca, Johnson & Johnson, Novartis, Syncona, Shattuck Labs, Teijin Pharma, and UCB.

References

1. GOSSEC L: *Arthritis Rheumatol* 2025; 77(suppl 9).
2. SIEBERT S: *Rheumatol Ther* 2023; 10: 489-505.

P158

DUAL TARGETED THERAPY IN TREATMENT-REFRACTORY PSORIATIC ARTHRITIS: EFFECTIVENESS AND SAFETY IN A CASE SERIES

Andrade S.[1], Khmelinskii N.[1], Ávila-Ribeiro P.[1], Ochôa Matos C.[1], Resende C.[1], Lopes A.R.[1], Fonseca J.E.[1], Silva G.[2], Antunes J.[2], Vieira-Sousa E.[1]
[1]Rheumatology Dept., ULSSM, Faculdade de Medicina da Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisbon; [2]Dermatology Dept., ULSSM, Faculdade de Medicina da Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisbon, Portugal

Introduction. Advances in the understanding of inflammatory pathways and insufficient response to biologic/targeted synthetic disease modifying drug (b/tsDMARD) monotherapy, have prompt interest in dual targeted therapy (DTT). In recently defined treatment refractory (TR) psoriatic arthritis (PsA) patients, DTT may provide additional benefit in persistent musculoskeletal (MSK) or extra-musculoskeletal manifestations (EMM). The evidence of DTT effectiveness however remains limited, and its safety profile not fully established.
Methods. PsA patients refractory to ≥2 b/tsDMARDs (distinct mechanisms of action), perceived as difficult-to-manage, failing to achive remission, and objective persistence of inflammation (MSK and/or EMM); treated with DTT (either a combination of two anti-cytokine biologic or an anti-cytokine biologic and a targeted synthetic DMARD), where retrospectively reviewed. Demographics, disease duration, MSK and EMM, prior conventional (csDMARDs) and b/tsDMARDs were collected. DTT regimens, indications and duration, disease activity (first and last visit), adverse events and reason of DTT discontinuation (if applicable) were described.
Results. Three male patients, long-standing polyarticular PsA and active psoriasis, refractory to ≥3 b/tsDMARDs (median 5). Demographic and clinical characteristics described in Table I. Due to persistent moderate-to-severe psoriasis (3 cases) and MSK activity (2 cases) while receiving b/tsDMARD monotherapy were treated with DTT based on a combination of a second bDMARD. In 2/3 cases a marked improvement in cutaneous lesions and additional effectiveness in joint disease. No infectious or other adverse events occured. Details on DTT regiments efficacy and safety provided in Table II.
Conclusion. DTT induced favourable clinical responses in TR PsA patients with high complexity and prior extensive b/tsDMARDs exposure. Nevertheless, in extremely severe phenotypes even DTT can still be insufficient to achieve remission (patient 3) In all cases, skin manifestations were the main reason for combining a IL12/23i or IL23i with a TNFi or JAKi, but additional articular effectiveness was also achieved. No relevant safety concerns were identified.

Table I. Baseline demographic and clinical characteristics of patients treated with dual targeted therapy.

	Age at DTT (years)	Sex	Diagnosis	Phenotype	Disease duration (years)	Entesitis/ Dactylitis	Axial involvement	Extra MSK manifestations	HLA-B27	Prior csDMARDs	Prior b/tsDMARDs
Patient 1	65	M	PsA	Polyarticular (erosive)	26	Both present	Present	PsO Uveitis	Positive	2 (Cs, MTX)	5 (IFX, ADA, UST, SEC, IXE)
Patient 2	63	M	PsA	Polyarticular	18	Dactylitis	Absent	PsO	Negative	1 (MTX)	3 (ETA, RIS, SEC)
Patient 3	31	M	PsA (eJIA)	Polyarticular	19	Entesitis	Present	PsO Uveitis IBD	Positive	2 (MTX, LEF)	11 (ETA, ADA, TCZ, IFX, SEC, TOF, BARI, CTZ, RTX, ANK, UPA)

ADA: adalimumab; ANK: anakinra; BARI: baricitinib; b/tsDMARD: biologic and targeted synthetic disease modifying drug; Cs: cyclosporine; CTZ: certolizumab; DAPSA: Disease Activity in Psoriatic Arthritis score; DTT: dual targeted therapy; eJIA: enthesitis-related juvenile idiopathic arthritis; ETA: etanercept; GOL: golimumab; IBD: inflammatory bowel disease; IFX: infliximab; IXE: ixekizumab; LEF: leflunomide; M: male; MSK: musculoskeletal; MTX: methotrexate; NAPSI: Nail Psoriasis Severity Index score; PsA: psoriatic arthritis; PASI, Psoriasis Area and Severity Index score; PsO: psoriasis; RIS: risankizumab; RTX: rituximab; SEC: secukinumab; TCZ: tocilizumab; TOFA: tofacitinib; UPA: upadacitinib; UST: ustekinumab.

Table II. Efficacy and safety of dual targeted therapy in treatment refractory psoriatic patients.

	b/tsDMARD monotherapy previous to DTT	DTT Indication	DTT regimen 1	DTT regimen 2	Baseline disease activity*	Last visit disease activity	DTT duration (months)	Adverse events	DTT discontinuation (reason)
Patient 1	GOL (100mg 4/4w)	PsO activity	GOL (100mg 4/4w) + RIS (150mg12/12w)	-	DAPSA 4 PASI 14.3	DAPSA 2.2 PASI 3.5	5	None	No
Patient 2	TOFA (11mg/d)	PsO + MSK activity	TOFA (11mg/d) + UST (90mg12/12w)	-	DAPSA 23.37 PASI 5.4	DAPSA 17.87 PASI 0	8	None	No
Patient 3	GOL (100mg 2/2w)	PsO + MSK activity	GOL (100mg 2/2w) + UST (90mg 12/12w)	GOL (100mg 2/2w) + RIS (150mg12/12w)	DAPSA 42.13 PASI 6.7	DAPSA 28.83 PASI 4	15	None	Yes (ineffective MSK and PsO)

b/tsDMARD: biologic and targeted synthetic disease modifying drug; DTT: dual targeted therapy; DAPSA: Disease Activity in Psoriatic Arthritis score; eJIA: enthesitis-related juvenile idiopathic arthritis; GOL: golimumab; MSK: musculoskeletal; NAPSI: Nail Psoriasis Severity Index score; PASI: Psoriasis Area and Severity Index score; PsO: psoriasis; RIS: risankizumab; TOFA: tofacitinib; UST: ustekinumab.
*With the latest monotherapy regimen.

P159

MULTIPLE BIOLOGICS SWITCHING AS A MARKER OF SUSPECTED DIFFICULT-TO-MANAGE AXIAL SPONDYLOARTHRITIS: INSIGHTS FROM THE ARAB WORLD PROSPA STUDY

Zemrani S.[1,2], Rostom S.[1,2], Hmamouchi I.[3], Ben Abdelghani K.[4], Tekaya R.[5,6], Boussaa H.[4], Hamdi W.[5,7], Mahmoud I.[5,6], Hannawi S.[8], Asal A.[9], Dougados M.[10], Bahiri R.[1,2]
[1]Rheumatology Dept., El Ayachi Hospital, Ibn Sina University Hospital Center, Rabat-Salé; [2]Faculty of Medicine and Pharmacy, Mohammed V University, Rabat; [3]Faculty of Medicine, Health Science Research Center, International University of Rabat, Rabat, Morocco; [4]Rheumatology Dept., Mongi Slim Hospital, Faculty of Medicine of Tunis, Tunis; [5]Tunis El Manar University, Tunis; [6]Rheumatology Dept., Charles Nicolle Hospital, Tuni; [7]Rheumatology Dept., Kassab Institute, Faculty of Medicine of Tunis, Tunis, Tunisia; [8]Alkuwait Hospital-Dubai, Emirates Health Services, Dubai, United Arab Emirates; [9]University of Baghdad, Rheumatology Unit, Dept. of Medicine, Baghdad, Iraq; [10]Cochin Hospital, Université Paris Cité, Paris, France

Introduction. Although ASAS recently proposed a consensus definition of D2M-axSpA, its applicability across countries may be challenging because of variations in treatment access. Using PROSPA (Profile of axial Spondyloarthritis in Arab countries) data, we used exposure to ≥2 b/tsDMARD switches as a surrogate marker of D2M disease and assessed its prevalence, associated factors, and patient profiles.
Methods. This ancillary PROSPA analysis included adults with axSpA from 15 Arab countries. Suspected D2M axSpA was defined as exposure to >2 b/tsDMARDs. Prevalence was assessed overall and across Arab regions. Suspected D2M patients (Gr1) were compared with b/tsDMARD-exposed non-D2M (Gr2) and b/tsDMARD-naive patients (Gr3). Factors associated with suspected-D2M status, switching reasons, and cluster-defined phenotypes were evaluated.

Table I. Univariate (a), and multivariate (b) logistic regression analysis of factors associated with suspected-D2M ax-SpA.

	Univariate analysis			Multivariate analysis		
	OR	95% CI	p-value	OR	95% CI	p-value
Age	1.01	0.99–1.04	0.212			
Female sex	0.78	0.47–1.30	0.337			
BMI	0.98	0.95–1.01	0.174			
Diagnostic delay >2 years	*2.15*	*1.28–3.59*	*0.004*	*1.85*	*1.03–3.29*	*0.038*
Active smoking (ever)	*1.72*	*1.06–2.80*	*0.028*	*1.74*	*1.03–2.93*	*0.039*
Peripheral involvement	*2.01*	*1.20–3.36*	*0.008*	1.36	0.77–2.39	0.284
Coxitis	*1.64*	*1.01–2.65*	*0.043*	1.06	0.61–1.83	0.832
Structural damage	*2.33*	*1.10–4.96*	*0.028*	1.37	0.66–2.86	0.403
ASDAS-CRP	*1.57*	*1.21–2.03*	*<0.001*	*1.44*	*1.07–1.94*	*0.016*
fibromyalgia	*2.49*	*1.51–4.10*	*<0.001*	*1.81*	*1.01–3.25*	*0.046*

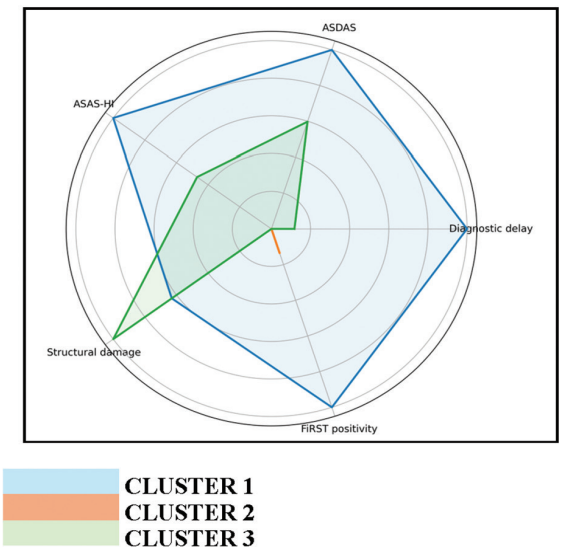


Fig. 1. Radar plot of the three cluster-defined profiles.

Results. Among 1,112 patients with axSpA, 7.1% were classified as suspected-D2M. 70.9%, 19.0%, 8.9%, and 1.3% had received 3, 4, 5, and 6 b/tsDMARDs, respectively. No regional differences were observed (North Africa 7.5%, Levant 6.5%, Gulf 7.6%).
Compared with Gr2 and Gr3, suspected-D2M patients have more arthritis (p<0.001), enthesitis (p=0.01), inflammatory bowel disease (p=0.03), syndesmophytes (p<0.001), fibromyalgia (p<0.001), higher ASDAS-CRP (p<0.001) and higher ASAS-HI (p=0.001).
In Gr1, secondary failure was the leading reason for bDMARD switching (36.7%), whereas primary failure predominated for tsDMARDs (71%).
Diagnosis delay and smoking (both p=0.03), ASDAS-CRP (p=0.01), fibromyalgia (p=0.04) were independently associated with suspected-D2M status (Table I).
Cluster analysis identified three phenotypes among Gr1: Cluster 1 with fibromyalgia, higher disease activity, longer diagnostic delay, and greater structural damage; Cluster 2 with lower disease activity, shorter diagnostic delay, and no structural damage; and Cluster 3 with structural damage, intermediate disease activity, and no fibromyalgia (Fig. 1).
Conclusion. Approximately 7% of patients with axSpA in the Arab world may represent a D2M phenotype. Longer diagnostic delay, smoking, higher ASDAS-CRP, and fibromyalgia were associated with this phenotype. Three distinct clinical profiles highlighted its heterogeneity.

P160

THE FREQUENCY OF SARCOPENIA IN PATIENTS WITH PSORIATIC ARTHRITIS: A CROSS-SECTIONAL STUDY

Aslan B.[1], Erbasan F.[2], Uçar I.[3], Nokay M.T.[1], Yildirim Ö.E.[2], Güloğlu S.B.[4], Aydemir M.[5], Yazisiz V.[2]
[1]Antalya Training and Educational Hospital, Antalya; [2]Akdeniz University Faculty of Medicine, Internal Medicine, Division of Rheumatology, Antalya; [3]Mersin City Hospital, Internal Medicine, Division of Rheumatology, Antalya; [4]Antalya Training and Research Hospital, Physical Medicine and Rehabilitation, Antalya; [5]Akdeniz University Faculty of Medicine, Internal Medicine, Division of Endocrinolgy, Antalya, Turkey

Objective. In this study was to investigate the frequency of sarcopenia, and associated factors, in patients diagnosed with psoriatic arthritis (PsA), which is a chronic inflammatory disease affecting the musculoskeletal system.
Materials and methods. The muscle mass and fat ratio of 34 consecutive PsA patients and 32 healthy volunteers were measured with a bio-electric impedance device. Grip strength was evaluated with a hand dynamometer and physical performance with the 400-metre walking test.
Results. None of the PsA patients or the healthy volunteers met the sarcopenia criteria. The frequency of pre-sarcopenia was determined to be 44% in the PsA patients and 15% in the healthy control group; the difference was statistically significant (p:0.016). PsA patients exhibited significantly higher body surface area and body weight, alongside significantly lower muscle mass compared to healthy controls (all p<0.05).
Among PsA patients, the pre-sarcopenic subgroup (n=15) showed significantly lower handgrip strength and appendicular muscle mass compared to non-pre-sarcopenic counterparts (p<0.05). Additionally, significantly lower leukocyte (p=0.034) and neutrophil (p=0.014) counts were observed in pre-sarcopenic patients relative to those without pre-sarcopenia.
Conclusion. It was observed that muscle mass and strength are significantly impaired in patients with PsA. While the prevalence of pre-sarcopenia is notably increased, it often does not reach the clinical threshold for sarcopenia. Interestingly, pre-sarcopenic patients maintained a stable Body Mass Index (BMI), suggesting a preservation of total body weight despite the underlying muscle depletion. Furthermore, a significant decrease in serum leukocyte and neutrophil counts was identified in the pre-sarcopenic group, indicating a potential link between muscle mass loss and altered systemic inflammatory or hematological profiles in PsA.

Table I. Demographic and clinical characteristics of the study groups.

	PsA (n=34)	Control (n=32)	p-value
Gender (F; n, %)	20(58)	24(75)	0.198
Age (year), (mean±SD)	48.12±8.7	45.31±8.3	0.698
Smoking status (not smoking; n %)	12(%51)	6(%73)	0.248
Pre-sarcopenia	15(%44)	5(%15)	**0.016**
Height (cm)	168±12.6	163.72±9.4	0.099
Weight (kg)	83.3±21.2	74.7±15.7	**0.021**
Body mass index (BMI) (kg/m^2)	29.1±5.1	27.8±5	0.707
Body surface area (BSA)	1.94±0.30	1.83±0.23	**0.043**
Waist circumference (cm)	100.36±19.37	93.16±19.19	0.276
Handgrip strength	26.51±16.68	25.16±14.50	**0.028**
Gait speed(m/s)	1.28±0.21	1.38±0.26	0.395
Muscle mass	53.67±14.55	47.11±9.24	**0.004**
Fat mass	26.57±10.42	24.81±7.44	0.073
Appendicular lean mass	32.50±21.24	29.43±15.97	0.261
Lean mass	50.91±16.06	43.68±13.03	0.204
Muscle mass index (SMI) (kg/m^2)	32.67±8.26	28.84±6.48	0.343

BMI: Body Mass index (kg/m^2); BSA: Body surface area; Mean±SD: mean /standard deviation; PsA: psoriatic arthritis, SMI: muscle mass index (kg/m^2)

Table II. Comparison of clinical and laboratory findings between PsA patients with and without presarcopenia.

	PsA (n=34)		p-value
	Pre-sarcopenia(+) (n=15)	Pre-sarcopenia (-) (n=19)	
Gender (F; n,%)	11(73)	9(47)	0.171
Smoking status (not smoking; n %)	4(27)	13(72)	**0.032**
Duration of disease (month)	111.23±102.02	91.69±86.91	0.713
Age (year) (mean±SD)	48±10.18	48.21±7.74	0.572
Height (cm)	163.33±9.33	172.11±13.77	0.088
Weight (kg)	74.09±17.13	90.16±21.64	0.274
Body mass index (BMI) (kg/m^2)	27.49±4.38	30.22±5.38	0.761
Body surface area (BSA)	1.8±0.23	2.04±0.31	0.119
Waist circumference(cm)	94.91±18.49	105.82±19.51	0.951
Handgrip strength	13.89±4.87	36.46±15.94	**0.001**
Gait speed (m/s)	1.15±0.19	1.38±0.18	0.673
Muscle mass	46.52±10.43	58.95±15.11	0.065
Fat mass	24.02±9.4	28.44±10.98	0.706
Appendicular lean mass	20.22±8.85	40.26±23.23	**0.001**
Lean mass	47.70±14.18	53.26±17.30	0.405
Muscle mass index (kg/m^2) (SMI)	29.88±6.87	35.74±8.88	0.268
Hemoglobin (Hb) (g/dL)	12.87±1.21	14.29±1.45	0.579
Leucocyte [10^3/µL]	6922±804.71	7830.02±3151.40	**0.034**
Neutrophil [10^3/µL]	3560±763.50	4708.57±1502.29	**0.014**
Platelet [10^3/µL]	291.90±56.69	268.64±69.07	0.285
CRP mg/dL	6.03±7.85	9.06±17.47	0.541
ESR(mm/H)	26.88±19.19	17.69±12.24	0.505
ALT(U/L)	25.60±13.75	26.43±15.1	0.606
AST(U/L)	19.3±6.9	21.50±10.40	0.810
BUN(mg/dL)	10.40±3.41	13.43±3.6	0.650
Creatinin (mg/dL)	0.73±0.1	0.92±0.15	0.282
Calcıum(mg/dL)	9.54±0.39	9.52±0.45	0.495
Albumin(g/dL)	45.69±2.66	46.1±3.52	0.494
Methotrexate usage	7(47)	7(37)	0.728
bDMARDs	11(73)	13(68)	1

ALT: alanin aminotransferase; AST: aspartate aminotransferase; bDMARDs: biological disease-modifying antirheumatic drugs; BMI: Body Mass index (kg/m^2); BSA: Body surface area (BSA); CRP: C-reactive protein; ESR: erytrocyte sedimentation rate; Mean±SD: mean /standard deviation; PsA: psoriatic arthritis; SMI: Muscle Mass index (BMI) (kg/m^2).

P161

TRANSITION FROM PSORIASIS TO PSORIATIC ARTHRITIS: PAIN PERSPECTIVE FROM THE TURKISH NATIONAL HEALTH DATABASE (E-NABIZ)

Ayan G.[1], Satis H.[2], Ata N.[3], Tascilar K.[4], Armagan B.[5], Babaoglu H.[5], Ulgu M.M.[6], Fidanci A.A.[3], Birinci S.[7], Kucuksahin O.[8], Kalyoncu U.[9]
[1]Dept. of Rheumatology, Ankara Research and Training Hospital, Ankara; [2]Dept. of Rheumatology, Abdurrahman Yurtaslan Oncology Hospital, Ankara; [3]Dept. of Strategy Development, Republic of Türkiye Ministry of Health, Ankara, Turkey; [4]Dept. of Medicine 3, Friedrich Alexander University Erlangen-Nürnberg (FAU) and Universitätsklinikum Erlangen, Erlangen, Germany; [5]Dept. of Internal Medicine, Division of Rheumatology, Ankara City Hospital, Ankara; [6]General Directorate of Health Information Systems, Republic of Turkey Ministry of Health, Ankara; [7]Republic of Turkey Ministry of Health, Ankara; [8]Dept. of Rheumatology, Ankara Yıldırım Beyazit University, Ankara City Hospital, Ankara; [9]Dept of Internal Medicine, Division of Rheumatology, Hacettepe University Faculty of Medicine, Ankara, Turkey

Objective. We aimed to evaluate the frequency, timing, and specialty distribution of outpatient visits for musculoskeletal pain in the years preceding a new diagnosis of PsA.
Methods. Data from the Turkish National Health Database (e-Nabız) were retrospectively analyzed. Patients newly diagnosed with PsA during 2025, were identified using ICD-10 codes (M07.0–M07.39) recorded at least twice ≥30 days apart together with at least one disease-modifying antirheumatic drug prescription during the index period. Patients with any previous PsA-related diagnosis or prescription were excluded. Outpatient visits from 2018 onwards were screened for pain-related ICD-10 codes (M25, M54, M79, R52 categories) across specialties including orthopedics, neurosurgery, physical therapy and rehabilitation, algology, internal medicine, emergency medicine, and others. A 1:1 matched control group without PsA was constructed based on age, sex, and major comorbidities. Mean annual pain-related outpatient visits per person were calculated descriptively for each year preceding diagnosis.
Results. A total of 6,965 newly diagnosed PsA patients and 6,965 matched controls were included; 70% were female and mean age was 49±14 years. Before diagnosis, 128,421 pain-related visits occurred in the PsA group compared with 70,264 in controls. Pain-related visits were already more frequent in the PsA group seven years before diagnosis, with a PsA-to-control ratio of 1.60 in 2018, increasing progressively to 2.24 in 2024, the year preceding diagnosis. Ratios were consistently higher among men than women. In 2024, the highest specialty-specific ratios were observed in rheumatology (16.0) and physical therapy and rehabilitation (2.92), followed by orthopedics (2.0) and neurosurgery (1.55).
Conclusion. Individuals later diagnosed with PsA had increased pain-related healthcare utilization as early as seven years before diagnosis, with progressively increasing visits over time, supporting the existence of a prolonged symptomatic preclinical phase. These nationwide findings support systematic enquiry of musculoskeletal symptoms in patients with PsO and timely referral to rheumatology.

Table I. Annual mean number of pain-related visits per person.

	Incident PsA cases			Control group			PsA/Control Ratio		
	Overall	Female	Male	Overall	Female	Male	Overall	Female	Male
2018	0.93	1.02	0.72	0.58	0.66	0.41	1.60	1.54	1.75
2019	1.04	1.15	0.76	0.67	0.76	0.44	1.55	1.51	1.72
2020	0.86	0.93	0.69	0.52	0.58	0.38	1.65	1.60	1.81
2021	1.13	1.25	0.85	0.65	0.73	0.48	1.73	1.71	1.77
2022	1.45	1.60	1.12	0.83	0.95	0.56	1.74	1.68	2.00
2023	1.92	2.06	1.60	1.01	1.14	0.70	1.90	1.81	2.28
2024	2.53	2.71	2.11	1.13	1.28	0.80	2.24	2.12	2.64

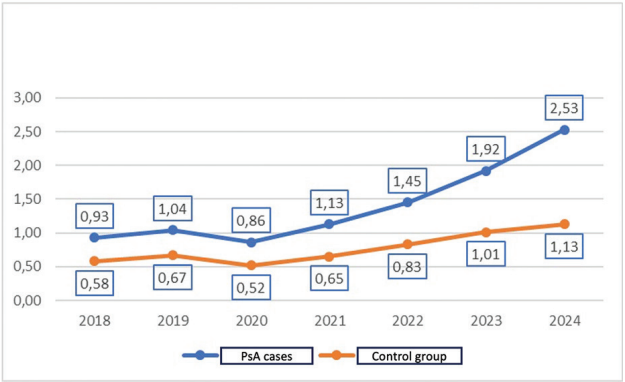


Fig. 1. The time course of annual mean number of pain-related outpatient visits per person before the index period.

P162

OCHRONOTIC ARTHROPATHY MIMICKING SPONDYLOARTHROPATHY: A CASE REPORT OF ALKAPTONURIA

Güner İ., Usta B., Kumru B.G., Bilici R.
Necmettin Erbakan University School of Medicine,Internal Medicine, Rheumatology Dept., Konya, Turkey

Introduction. Alkaptonuria is a rare metabolic disorder caused by deficiency of the homogentisic acid oxidase enzyme involved in tyrosine metabolism, leading to excessive urinary excretion of homogentisic acid (HGA). In the process known as ochronosis, accumulation of HGA and its metabolites in connective tissues results in pigmentation and progressive structural damage in multiple organs. Cartilage, ear, sclera, corneal limbus, joints and bones are commonly affected, while cardiac valves, kidneys, gallbladder, prostate and skin may also be involved. Musculoskeletal and cardiovascular involvement are the major causes of morbidity. Clinically, ochronotic arthropathy may mimic several rheumatologic conditions including spondyloarthropathy, diffuse idiopathic skeletal hyperostosis, juvenile idiopathic arthritis and osteoarthritis. This case highlights the importance of considering ochronosis in the differential diagnosis of spondyloarthropathy.
Methods. A 50 year old female presented with progressively worsening bilateral knee, back and persistent low back pain for two years. Due to lack of response to physiotherapy and NSAID treatment in the physical medicine clinic, she was referred to our rheumatology clinic with a preliminary diagnosis of spondyloarthropathy based on sacroiliac joint MRI findings. Her medical history included darkening of urine, black pigmentation of the ear cartilage and conjunctiva, hearing loss and proteinuria. Genetic analysis revealed a homozygous C586A mutation in the homogentisate 1,2 dioxygenase (HGD) gene and she had previously been diagnosed with alkaptonuria and treated with nitisinone.
Results. Physical examination revealed painful bilateral hip range of motion, nail thickening with periungual gray discoloration and a 3 mm black pigmented lesion on the left conjunctiva. Acute phase reactants were within normal limits. Sacroiliac MRI demonstrated bilateral subchondral edematous and degenerative changes, more prominent in the sacral region, without clear evidence of inflammatory spondyloarthropathy. Compared with MRI obtained in 2023, degenerative changes in the sacroiliac joints and vertebral discs had increased. Thoracolumbar spine radiographs showed narrowing of intervertebral disc spaces and multiple calcifications in intervertebral discs and lower costal cartilages.
Conclusion. Ochronotic arthropathy should be considered in the differential diagnosis of patients presenting with chronic back pain and degenerative spinal changes mimicking spondyloarthropathy. Clinical findings such as pigmentary changes, normal inflammatory markers and characteristic radiographic features may help distinguish ochronosis from inflammatory rheumatic diseases.

P163

COURSE OF DISEASE PHENOTYPES AND TREATMENT OUTCOMES IN RHEUMATOID ARTHRITIS, AXIAL SPONDYLOARTHRITIS, AND PSORIATIC ARTHRITIS RANDOMIZED-CONTROLLED TRIALS: A SYSTEMATIC REVIEW AND META-REGRESSION ANALYSIS

Calixto O.J.[1,2], Kiltz U.[1,3], Imke R.[1], Lalazi B.[1], Klümpers F.E.[1], Sewerin P.[1,3], Baraliakos X.[1,3]
[1]Rheumazentrum Ruhrgebiet, Herne; [2]Universidad El Bosque, Cellular and Molecular Immunology Group (InmuBo), Bogotá, Colombia; [3]Ruhr-Universität Bochum, Bochum, Germany

Introduction/Objective. Disease-specific studies have reported changes in trial populations and rising placebo response rates in inflammatory rheumatic and musculoskeletal diseases RCTs over time, but no study has quantified and compared temporal changes in baseline phenotypes and efficacy outcomes across RA, axSpA, and PsA. Evaluate temporal changes in baseline phenotypes and treatment outcomes in RCTs of RA, axSpA and PsA over nearly three decades.
Methods. Systematic review of RCTs evaluating biological or targeted synthetic disease-modifying antirheumatic drugs (bDMARDs/tsDMARDs). Random-effects meta-regression models assessed temporal changes using pre-specified 5-year recruitment intervals (1995-2024), adjusted for treat-

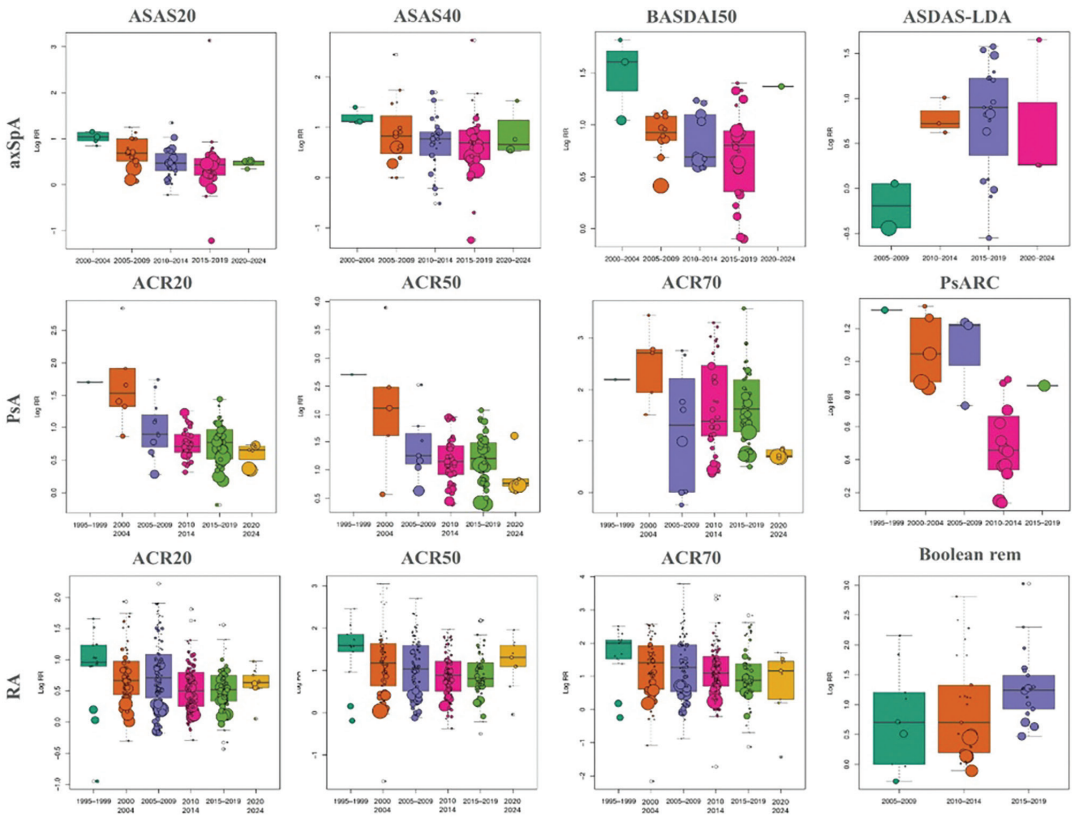
ment class, comparator type, and trial eligibility criteria. Sensitivity analyses included influence diagnostics and continuous-year modelling.
Results. 395 RCTs were included (RA: n=242, 105,741 patients; axSpA: n=89, 19,234 patients; PsA: n=69, 26,738 patients). In RA, progressive non-linear reductions in baseline tender and swollen joint counts, ESR, and HAQ were observed from year 2000 onwards, alongside an increase in female representation from 2005 onwards. In axSpA, patients with extra-musculoskeletal manifestations declined from 2005 onwards. In PsA, baseline functional disability decreased over time. Treatment outcomes showed divergent temporal trajectories between active treatment versus non-bDMARD comparators. In RA, the probability to achieve Boolean and SDAI remission increases significantly, with some time-interval predominance. In axSpA, response decreased consistently across all post-2005 intervals for ASAS20, with significant decreases in ASAS40, ASAS5/6, and BASDAI50 for some intervals. Similarly, ACR20 and PASI90 showed significant decreases in PsA for some intervals. After adjustment for comparator type, treatment mechanism of action, and eligibility criteria, the temporal effect on treatment outcomes probability was substantially attenuated.
Conclusion. Network meta-analyses pooling RCTs across decades should incorporate temporal moderators to account for trial population drift, and future trials should calibrate eligibility criteria and endpoints to the contemporary, milder disease severity landscape.

P164

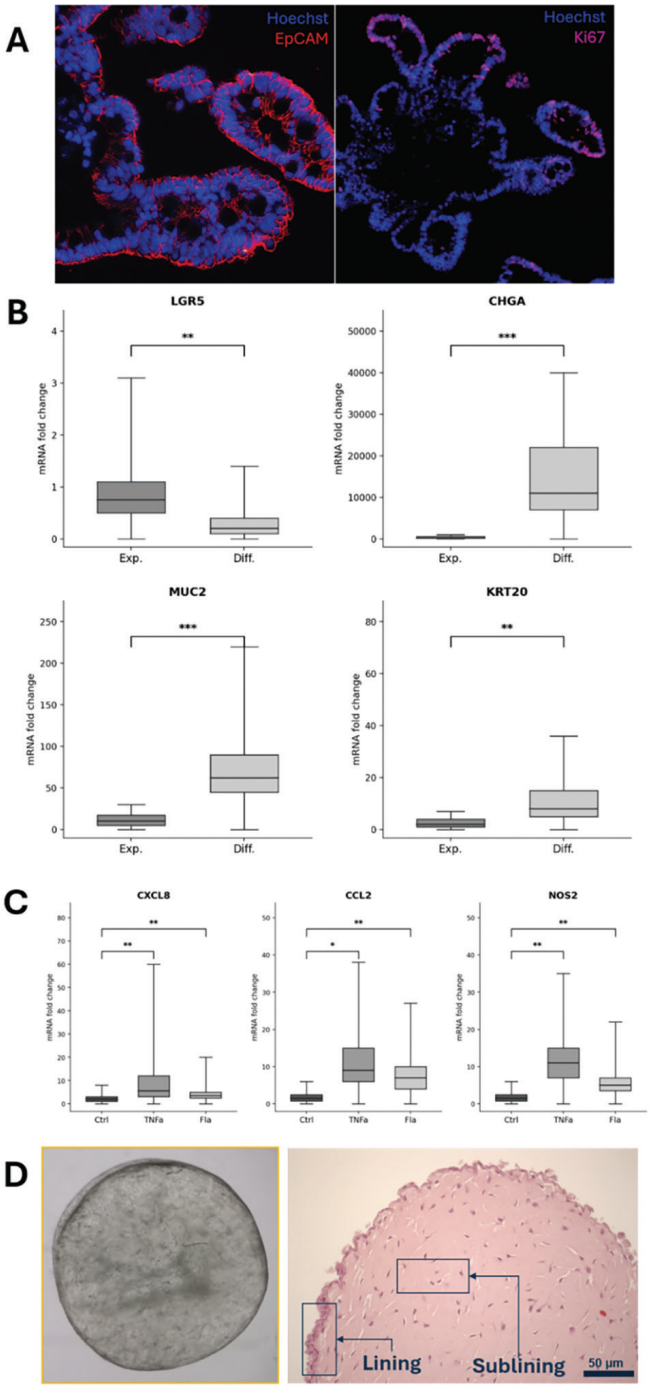
PRELIMINARY VALIDATION OF AN ORGANOID-BASED MODEL FOR THE STUDY OF THE GUT-JOINT AXIS IN SPONDYLOARTHRITIS

Gavotti C.[1], Natalello G.[1,2], Gouze H.[1], Meo M.[1], Ortolan A.[1,2], De Lorenzis E.[1,2], Peluso G.[1], Alivernini S.[1,2], Lizzio M.M.[1], Coppola G.[3], Di Vincenzo F.[3], Cherbuy C.[5], Bazin T.[6], Breban M.[6], Soudy R.[5], Langella P.[5], Di Mario C.[1], Petito V.[3], Gasbarrini A.[3,4], Scaldaferri F.[3,4], D'Agostino M.A.[1,2]
[1]Rheumatology and Clinical Immunology Unit, Fondazione Policlinico Universitario Agostino Gemelli IRCCS, Rome; [2]Dept. of Geriatric and Orthopaedic Sciences, Catholic University of the Sacred Heart, Rome; [3]CEMAD Digestive Disease Center, Fondazione Policlinico Universitario Agostino Gemelli IRCCS, Rome; [4]Dept. of Translational Medicine and Surgery, Catholic University of the Sacred Heart, Rome, Italy; [5]Micalis Institute, Institut National de Recherche pour l'Agriculture, l'Alimentation et l'Environnement (INRAE), AgroParisTech, Université Paris-Saclay, Jouy-en-Josas; [6]Infection & Inflammation, UMR 1173, INSERM, Versailles-Saint-Quentin University, Montigny-le-Bretonneux, France

Introduction. The gut-joint axis is a key paradigm for understanding spondyloarthritis (SpA) pathogenesis, but patient-derived preclinical models enabling interrogation of intestinal epithelium and synovial compartment are lacking. We aimed to develop and characterize a multi-tissue preclinical platform for translational research for SpA based on patient-derived intestinal organoids and 3D synovial micromasses.
Methods. From ileal biopsies of SpA patients, healthy controls, and inflammatory comparators with inflammatory bowel disease (IBD), epithelial crypts were isolated to establish Matrigel-embedded organoid lines. Organoids were expanded (Stemcell Intesticult medium) and either cryopreserved or differentiated in modified L-WRN medium. Epithelial identity, polarity, and maturation were assessed by immunofluorescence and RT-qPCR. Functional competence was evaluated after stimulation with TNF-α or flagellin (Fla). In parallel, fibroblast-like synoviocytes (FLS) from synovial biopsies of SpA and psoriatic arthritis (PsA) patients or non-inflammatory arthropathy (NIA) controls were expanded and embedded in Matrigel to generate 3D synovial micromasses, morphologically assessed on hematoxylin-eosin (HE)-stained sections.
Results. Patients were enrolled at Policlinico Gemelli in Rome. Ten intestinal organoid lines were established (3 SpA, 2 healthy controls, 5 IBD), along with 10 synovial micromass lines (8 PsA/SpA, 2 NIA), with a 100% and 80% success rate, respectively. Intestinal organoids showed epithelial identity (EpCAM), proliferation (Ki67), and apical-in polarity by immunofluorescence on histological preparations (Fig. 1A). Differentiation medium induced maturation with increased MUC2, CHGA, and KRT20, and decreased LGR5 by RT-qPCR (Fig. 1B). Cytokine and flagellin stimulation induced upregulation of CCL2, CXCL8, NOS2, and DUOX2 (Fig. 1C), without acute cytotoxicity. No clear intergroup differences were noted. Synovial micromasses were successfully generated from third passage FLS; HE histology showed lining- and sublining-like organization (Fig. 1D).
Conclusion. Patient-derived intestinal organoids are a reproducible, functionally characterizable platform to study the epithelial gut-joint axis in SpA. Synovial micromasses support extension of the model to the joint compartment, pending quantitative, immunophenotypic, and functional validation.



P163 - Fig. 1. Trial-level treatment effects across 5-year recruitment periods in axSpA, PsA, and RA.



P164 - Fig. 1.

P165

PREVALENCE OF OSTEOPOROSIS SCREENING AND OSTEOPOROTIC VERTEBRAL FRACTURES IN PATIENTS WITH SPONDYLOARTHRITIS AND IBD

Attari H.[1], Génot J.[1], Fauny M.[1], Morizot C.[1], Bauer E.[1], Moret M.[1], Yalo L.[1], Peyrin-Biroulet L.[2], Chary-Valckenaere I.[3], Loeuille D.[4]
[1]Dept. of Rheumatology, CHRU de Nancy, Nancy; [2]Inserm NGERE, Dept. of Gastroenterology, University of Lorraine, CHRU de Nancy, Nancy; [3]Dept. of Rheumatology, CHRU de Nancy, IMoPA, CNRS, University of Lorraine, Nancy; [4]Dept. of Rheumatology, CHRU de Nancy, CNRS, IMoPA, University of Lorraine, Nancy, France

Objective. Spondyloarthritis (SpA) and inflammatory bowel diseases (IBD), including Crohn's disease (CD) and ulcerative colitis (UC), are independently associated with an increased risk of osteoporosis and osteoporotic fractures. We aimed to assess the prevalence of vertebral fractures (VFs) and osteoporosis screening in patients with SpA and IBD.
Patients and methods. This retrospective, single-center study included 148 patients followed at CHRU de Nancy between 2005 and 2025, meeting ASAS 2009 criteria for SpA and ECCO 2017 criteria for CD or UC. Patients were classified as axial (axSpA), peripheral (pSpA), or mixed (mSpA), and as radiographic (r) or no-radiographic (nr). VFs were evaluated using all available imaging (thoracic and lumbar spine X-rays, spinal MRI, thoraco-abdomino-pelvic CT) blinded to IBD diagnosis and graded according to Genant's four-grade scale. The performance of DEXA for osteoporosis screening was also evaluated.
Results. Among the 148 patients (mean age 45.6 years, 58.1% female), the median disease duration was 58 months [18.75; 135.25], and 70.9% received bDMARD therapy. 102 had axSpA (68.9%), 24 pSpA (16.2%), and 22 mSpA (14.9%), with a predominance of CD (70.9%). 24 patients had VFs (16.2%), totaling 30 fractures. Factors associated with VFs included older age (55±12.6 vs. 43.9±13.2 years; p=0.001), male sex (70.8% vs. 36.3%; p=0.002), longer SpA duration (225 [93.75; 345.5] vs. 80.5 [19; 136.75] months; p<0.001), structural axial damage (45.8% vs. 14.5%; p=0.003), HLA-B27 positivity (41.7% vs. 34.5%; p=0.03), higher CRP levels (9.9 [6.2; 20.35] vs. 5.6 [2.43; 14.38]; p=0.045), and a higher prevalence of CD (OR=3.3; 95% CI: 1.014–10.959; p=0.051). Multivariate analysis showed a trend for older age being associated with VF risk (OR=1.076; 95% CI: 0.999–1.160; p=0.054). Among all patients, 57 underwent DEXA screening, preferentially those with VFs (OR=2.4; 95% CI: 1.0–5.6), and in patients with longer disease duration for SpA, IBD, or both (p<0.0001).
Conclusion. VF prevalence was 16.2% in patients with SpA and IBD, occurring predominantly in long-standing and severe SpA. Only 38.5% underwent osteoporosis screening via DXA.

P166

FROM ASSESSMENT TO ACTION: PHYSICIAN GLOBAL ASSESSMENT AND TREATMENT ESCALATION IN INFLAMMATORY ARTHRITIS

Kart Bayram G.S.[1], Ayan G.[1,2], Kilic L.[1], Kalyoncu U.[1]
[1]Division of Rheumatology, Dept. of Internal Medicine, Hacettepe University Faculty of Medicine, Ankara; [2]Division of Rheumatology, Dept. of Internal Medicine, Ankara Training and Research Hospital, Ankara, Turkey

Background. Physician Global Assessment (PhGA) is widely used to assess disease activity in inflammatory arthritis and often guides treatment decisions. However, the PhGA threshold associated with treatment escalation in routine clinical practice remains unclear. We aimed to investigate the relationship between PhGA and treatment escalation.
Methods. Longitudinal visit-level data from patients with inflammatory arthritis followed in a routine rheumatology clinic since 2022 were analyzed. PhGA was prospectively recorded at each visit. Treatment escalation, defined as initiation, addition, dose increase, or switching of a disease-modifying antirheumatic therapy, was the primary outcome. ROC analysis was used to assess the discriminative ability of PhGA and identify candidate thresholds. Multivariable logistic regression evaluated the independent association between PhGA and treatment escalation after adjustment for age, sex, tender and swollen joint counts, C-reactive protein, and change in PhGA from the previous visit (ΔPhGA).

Results. A total of 445 patients contributing 1,319 visits were included. The cohort comprised patients with rheumatoid arthritis(n=186), seronegative arthritis (n=70), psoriatic arthritis (n=150), and other inflammatory arthritis diagnoses (n=39). Treatment escalation rates increased with higher PhGA values, reaching 38.1% at 35 and 56.9% at 40 (Table I). PhGA showed good discrimination for treatment escalation (AUC=0.824, 95% CI 0.799–0.849; $p<0.001$), with ROC-derived thresholds clustering within the 35–40 range (Table II). PhGA remained independently associated with treatment escalation (OR 1.097 per 1-point increase, 95% CI 1.077–1.118; $p<0.001$), whereas ΔPhGA was not independently associated with treatment escalation. Using a pragmatic threshold of 40, treatment escalation occurred in 62.3% of visits with PhGA ≥40 versus 13.5% with PhGA <40.

Conclusion. PhGA was strongly associated with treatment escalation. The consistent clustering of thresholds within the 35–40 range suggests that reproducible PhGA cutoffs may facilitate more consistent, generalizable, and standardized treatment escalation decisions across rheumatologists in routine clinical practice.

Table I. Treatment escalation rates according to Physician Global Assessment (PhGA) values.

PhGA	Total visits (n=1319)	Treatment escalation (+) (n=363)	Escalation rate (%)
10	75	1	1.3
15	45	1	2.2
20	394	25	6.3
25	103	20	19.4
30	239	48	20.1
35	84	32	38.1
40	174	99	56.9
45	61	37	60.7
50	35	20	57.1
55	42	32	76.2
60	25	19	76.0
65	23	14	60.9
70	11	7	63.6
75	3	3	100
80	5	5	100

Table II. Disease-specific ROC characteristics of physician global assessment for predicting treatment escalation.

Disease	AUC (95% CI)	Optimal PhGA Threshold	Sensitivity (%)	Specificity (%)
Seropositive RA	0.838 (0.798–0.878)	37.5	71.1%	84.7%
Seronegative arthritis	0.783 (0.714–0.853)	32.5	61.0%	81.7%
Psoriatic arthritis	0.813(0.773–0.854)	32.5	74.7%	78.6%
Enteropathic arthritis	0.901(0.813–0.989)	32.5	70.6%	89.5%
Overall Cohort	0.824 (0.799–0.849)	35.0	73.9%	79.4%

P167

EFFECTS OF REHABILITATION IN AXIAL SPONDYLOARTHRITIS: A META-ANALYSIS OF RANDOMIZED TRIALS

Li X.M.[1], Xie Y.[2], Zeng R.S.[2], Wu J.Y.[1], Chen S.D.[2], Gu J.R.[2]
[1]Dept. of Rheumatology and Immunology, The Seventh Affiliated Hospital of Sun Yat-sen University, Shenzhen; [2]Dept. of Rheumatology and Immunology, The Third Affiliated Hospital of Sun Yat-sen University, Guangzhou, China

Introduction. Axial Spondyloarthritis (axSpA) is the term for a range of chronic progressive inflammatory arthritis associated with the axial skeleton, and the representative subtype is ankylosing spondylitis. The disease may progress to functional impairment, spinal ankylosis, and even severe disability if inadequately controlled. In recent years, studies have shown that rehabilitation has a favorable therapeutic effect and improves health-related quality of life in patients with axSpA. This study aimed to assess the effectiveness of rehabilitation for patients with axSpA.

Methods. A comprehensive search was performed across four databases (PubMed, Cochrane Library, Embase, Web of Science) to identify studies assessing the impact of rehabilitation interventions on axSpA. The primary outcomes analyzed included the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), Bath Ankylosing Spondylitis Functional Index (BASFI) and Bath Ankylosing Spondylitis Metrology Index (BASMI). A meta-analysis and effect publication bias analysis used Review Manager 5.4.

Results. A total of 39 RCT studies involving 2,921 participants were included. Compared to the control group, all rehabilitation interventions demonstrated varying degrees of benefit in improving BASDAI, BASFI and BASMI scores. For BASDAI, the therapeutic exercise (-1.04, 95% CI[-1.43 to -0.66]), the physical agent modality (-1.36, 95% CI[-2.86 to 0.16]), the occupation therapy (-0.35, 95% CI[-0.62 to -0.08]), the combined therapy (-0.76, 95% CI[-1.38 to -0.15]). For BASFI, statistically significant improvements were observed with these four subgroup (-0.76, 95% CI[-0.98 to -0.55], I2 =48%, p=0.001). For BASMI, the therapeutic exercise (-0.47, 95% CI[−1.09 to 0.16]), the physical agent modality (-1.10, 95% CI[-1.89 to -0.31]), the occupation therapy (-0.07, 95% CI[-0.57 to 0.43]), the combined therapy (-1.26, 95% CI[-2.09 to -0.44]).

Conclusion. Different types of rehabilitation interventions can improve spinal mobility, physical function, disease activity, and quality of life in patients with axSpA.

P168

MODELLING THE LIKELY IMPACT OF PATIENT-INITIATED FOLLOW-UP FOR AXIAL SPONDYLOARTHRITIS PATIENTS IN NHS RHEUMATOLOGY SERVICES

Vasilakis C.[1], Khodaee S.[1], Fichera E.[2], Cavill C.[3], Benawra J.[4], Gandrup Horan J.[5], Tuytschaever S.[6], Bessa A.[4], Sengupta R.[3]
[1]School of Management, University of Bath, Bath; [2]Dept. of Economics, University of Bath, Bath; [3]Royal United Hospitals Bath NHS Foundation Trust, Bath; [4]UCB, Slough, UK; [5]UCB, Copenhagen, Denmark; [6]UCB, Brussels, Belgium

Introduction. Patients with axial spondyloarthritis (axSpA) often experience long waits for referral/follow-up appointments in NHS rheumatology services, resulting in diagnostic delays (average ~8 years) and poorer outcomes. Patient-initiated follow-up (PIFU) allows patients to schedule follow-ups according to need, while enhancing clinical capacity and service efficiency. We report a computer modelling study estimating potential impact of PIFU, with varying intervals for routine follow-up appointments, on patients with axSpA and their NHS service's performance. This modelling complements real-world evaluation of PIFU in an NHS hospital – part of the FASTRAX initiative deploying a digitally-supported PIFU pathway to improve outcomes for patients with axSpA.

Table. I. The likely impact of PIFU on key performance indicators according to computer modelling (discrete-event simulation).

Mean (95% CI)[b]	"Usual care" without PIFU Baseline	Practice with PIFU by follow-up interval[a] 18 months	24 months	30 months
Number of patients waiting for first appointment[c]	103.8 (86.3, 121.4)	102.2 (85.1, 119.3)	17.8 (12.3, 23.3)	3.2 (2.7, 3.7)
Waiting time for new referral appointment (weeks)[d]	13.0 (10.8, 15.2)	12.8 (106, 14.9)	2.2 (1.6, 2.9)	0.4 (0.3, 0.5)
Number of patients diagnosed with axSpA per week	1.6 (1.6, 1.6)	1.6 (1.6, 1.6)	1.6 (1.6, 1.6)	1.6 (1.6, 1.6)
Number of patients waiting for follow-up appointment[c]	201.9 (196.4, 207.4)	93.5 (87.6, 99.4)	12.3 (10.1, 14.5)	1.8 (1.5, 2.1)
Waiting time for follow-up appointment (weeks)[d]	10.4 (10.1, 10.8)	6.1 (5.7, 6.5)	0.9 (07, 1.1)	0.2 (0.1, 0.2)
Number of registered axSpA patients[c]	806.1 (801.4, 810.8)	794.1 (789.2, 798.9)	796.6 (792.3, 800.9)	804.9 (799.5, 810.3)
Number of patients on PIFU pathway[c]	NA	471.0 (467.4, 474.6)	551.6 (548.7, 554.5)	574.6 (570.2, 579.0)
Number of PIFU requests for urgent appointment per weeck	NA	2.3 (2.3, 2.4)	2.8 (2.7, 2.8)	2.9 (2.8, 2.9)
Unmet PIFU requests for urgent appointment[c] (%)	NA	2.3 (2.2, 2.4)	4.0 (3.8, 4.1)	4.7 (4.5, 4.9)

Mean (95% CI) number of new referrals received per week across all scenarios: 7.98 (7.95, 8.01). Capacity set at 8 weekly appointments for new referrals and 16 for follow-up appointments. "Usual care" without PIFU assumes follow-up intervals of 3 months or 12 months for patients undergoing treatment with unstable or stable disease, respectively.
[a] In the PIFU pathway, while patients can request an urgent appointment when needed, routine follow-up appointments are also scheduled after the noted interval since the most recent appointment;
[b] Calculated from 100 five-year simulation runs per scenario.
[c] Average across the 5-year period, based on "steady state" estimations at weekly simulated intervals.
[d] Average waiting time for all patients across the 5-year period.
axSpA: axial spondyloarthritis; CI: confidence interval; PIFU: patient-initiated follow-up; NA: not applicable.

Methods. Discrete-event simulation was used to model patient flow through an NHS rheumatology service over 5 years, capturing random variation, patient attributes and limited clinic capacity for new referrals/follow-up appointments. Input parameters, estimated from electronic hospital records, scientific literature and clinician advice, could be modified for different modelling requirements. We simulated a baseline scenario ("usual care" without PIFU) and three PIFU scenarios with 18/24/30-month follow-up intervals. Key performance indicators, including appointment waiting times, were averaged from 100 five-year simulation runs per scenario.
Results. All scenarios assumed an average new referral rate of 8 patients weekly and non-PIFU follow-up intervals of 3/12 months for patients with unstable/stable disease, respectively. PIFU scenarios assumed a 50% chance that stable patients transferred to PIFU. Compared to baseline scenario, PIFU substantially reduced the number of patients awaiting appointments and waiting times for new/registered patients, with longer follow-up intervals further improving access (Table I).
Conclusion. Computer modelling results indicated potential benefits of PIFU for freeing up clinic capacity and improving patient access. Additional simulation experiments and empirical evaluation of PIFU in a real-world NHS setting are planned in the second phase of FASTRAX, to assess feasibility and optimal monitoring intervals in practice.
Acknowledgements. Funded by UCB. Medical writing and editorial support provided by Costello Medical and funded by UCB.
Disclosures. CV, EF: Received funding from UCB to carry out this study. **SK:** None declared. **CC:** Received grants from AbbVie, Eli Lilly, Novartis and Pfizer. **JB, AB:** Former employees of UCB. **JGH:** Employee and shareholder of UCB. **ST:** Employee of UCB. **RS:** Previously held unrelated grants from AbbVie, Novartis, Pfizer and UCB, and received honoraria for giving talks from AbbVie, Biogen, BMS, Lilly, MSD, Pfizer, Novartis, Roche and UCB. RS has represented AbbVie and Novartis at NICE technology appraisals.

P169

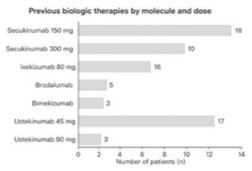
SWITCHING TO IL-23 INHIBITION AFTER IL-17 OR IL-12/23 FAILURE IN PSORIASIS AND PSORIATIC ARTHRITIS: REAL-WORLD EVIDENCE

Méndez Díaz L., Alvarez Ortiz L., Manzaneque Sánchez J., Gil Velez R.
Dept. of Rheumatology, Virgen del Rocío University Hospital, Seville, Spain

Introduction. Psoriasis and psoriatic arthritis (PsA) are chronic inflammatory diseases driven by the IL-23/Th17 axis. Although IL-17 and IL-12/23 inhibitors have improved disease control, some patients show inadequate response or loss of efficacy over time. This study aimed to evaluate the effectiveness and treatment persistence of IL-23 inhibitors in patients with psoriasis or PsA after failure of IL-17 or IL-12/23 therapies.
Methods. We conducted a single-centre, retrospective, observational study including patients from Rheumatology and Dermatology Dept.s treated with guselkumab or risankizumab for at least 12 months after failure of IL-17 inhibitors or ustekinumab. Articular activity was assessed using DAPSA, and skin response with PASI 90. Demographic and clinical data, prior biologic exposure, treatment persistence, and reasons for treatment switch were collected.
Results. Seventy patients were included (56% male, mean age 55 years), with long-standing disease (mean duration 17.9 years) and extensive prior biologic exposure (mean 3.3 previous biologic lines) (Fig. 1). Thirty-eight patients had psoriasis and 32 PsA. Guselkumab was used in 77% and risankizumab in 23%, with a mean treatment persistence of 35.7 months. The main reason for switching was inadequate skin control (77%). Among psoriasis patients, 95% achieved PASI 90. In peripheral PsA, 90.4% reached low disease activity or remission according to DAPSA. In mixed peripheral-axial disease, 81.8% achieved adequate joint control, while all reached skin targets. Enthesitis and dactylitis markedly improved during follow-up (Fig. 2). No major differences in effectiveness were observed according to prior biologic mechanism.
Conclusion. IL-23 inhibitors were effective and well tolerated in psoriasis and PsA patients previously exposed to IL-17 or IL-12/23 inhibitors, with particularly robust skin responses. Further studies are needed to clarify their role in axial PsA and optimize phenotype-specific treatment strategies.

Patient distribution by line of treatment

Treatment line	Number of patients	Percentage (%)
2nd line	23	32.9
3rd line	18	25.7
4th line	16	22.9
5th line	10	14.3
6th line	2	2.9
7th line	1	1.4



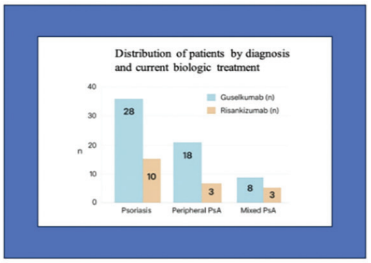


Fig. 1

Reasons to switch to anti–IL-23 therapy by disease	SKIN	ARTICULAR	BOTH
Peripheral psoriatic arthritis	8	8	5
Mixed psoriatic arthritis	8	3	0
Psoriasis	38	-	-
Failure to achieve treatment targets by disease domain	SKIN	ARTICULAR	BOTH
Peripheral psoriatic arthritis	2	1	1
Mixed psoriatic arthritis	0	2	0
Psoriasis	2	-	-

Fig. 2.

P170

CLINICAL AND SELF-REPORTED CHARACTERISTICS OF COMORBID FATIGUE IN AXIAL SPONDYLOARTHRITIS PATIENTS – A MULTICENTER STUDY

Morlà R.M.[1,8], Frade-Sosa B.[1], Grados D.[2], Gumucio R.[3], Garcia C.[3], Sirvent E.[4], Santo P.[4], Almirall M.[5], Espartal E.[5], Reina D.[6], Roig-Vilaseca D.[6], Clavaguera T.[7], Ponce A.[1], Azuaga A.[1], Ramirez J.[1], Romero Y.[1], Sapena N.[1], Tobalina L.[1], Sanmartí R.[1], Gómez Puerta J.A.[1], on behalf of ESPOcat study Group
[1]Servei de Reumatologia, Hospital Clínic de Barcelona, Barcelona; [2]Hospital Universitari d'Igualada, Igualada; [3]Hospital de Terrassa, Terrassa; [4]Hospital Sant Joan de Déu de Sant Boi, Sant Boi de Llobregat; [5]Hospital Universitari Vall d'Hebron, Barcelona; [6]Hospital Sant Joan Despí Moisès Broggi, Sant Joan Despí; [7]Hospital Universitari Dr Josep Trueta, Girona; [8]Xarxa Santa Tecla Sanitària, Social i Docent, Tarragona, Spain

Introduction/Objective. Fatigue is a highly prevalent and multifactorial symptom in patients with axial spondyloarthritis (axSpA). It is considered a core domain in the ASAS-OMERACT set, but not usually measured. We describe the prevalence, clinical features, and patient-reported outcomes (PRO) in a group of axSpA patients with fatigue compared with a group of non-fatigued patients.
Patients and methods. Patients with axSpA (ASAS, 2009), excluding those with chronic fatigue syndrome, were consecutively recruited in several hospitals (ESPOcat study group) over 6 months. They completed 5 questionnaires: 1. MDHAQ, which includes a VAS- Fatigue (0–10), a modified HAQ for function (1–10), Pain-VAS (0-10), PGA (0–10), 60-Symptom Review = ROS (0–60), 48-joint count self-assessment = RADAI (0–48), and RAPID3 activity index (0–30); 2. BASDAI (0–10), disease activity; 3. BASFI (0-10), functional impact; 4. FACIT-Fatigue (0–64); 5. ASAS-HI, quality of life (0–17). Patients were classified into 2 groups according to the VAS-Fatigue (≥5/10: fatigued, <5/10: not fatigued).The PROMS scores, sociodemographic, disease, and clinical characteristics, laboratory parameters, ASDAS-CRP, and the modified Schöber test were compared between groups.
Results. A total of 128 patients with axSpA were included,13.3% nraxSpA, 85.2% B27+, 62.5% males, with a mean (SD) of: age 51.91 (13.1) years, disease duration: 16.52 (12.6) years, BMI: 27.26 (5.21) kg/m^2, modified Schöber: 7.27 (6.41) cm, ASDAS-CRP: 2.69 (1.11), and FACIT-Fatigue: 34.41 (12.76). Comorbidities were: uveitis (21.1%), psoriasis (13.3%), Crohn's disease (3.1%), depression (7.8%), and fatigued patients (VAS ≥5/10) accounted for 54%.
Comparison of the two patient groups (with vs. without fatigue) revealed significant differences in disease activity and in all PROMs (Table I).
Conclusion. In this multicenter real-world cohort of patients with axSpA, fatigue was very common and was associated with a greater disease burden, as reflected in worse PROs and disease activity, but not with sociodemographic characteristics or acute phase reactants.
Acknowledgment. Societat Catalana de Reumatologia, for a research grant.

Table I. Comparative data of fatigued patients vs. non-fatigued patients, according to VAS-Fatigue (≥5/10).

Variable	Total (n=128)	No Fatigued (n=59)	Fatigued (n=69)	*p*-value
Sociodemographics				
Male sex, n (%)	80 (62.5%)	42 (52.8%)	38 (47.5%)	0.061
BMI (kg/m^2)*	27.26 (5.21)	26.21 (3.94)	28.30 (6.08)	0.275
Age, years*	5.,91 (13.09)	52.95 (12.77)	49.55 (15.57)	0.184
Married, n (%)	77 (60.2%)	38 (49.4%)	39 (50.6%)	0.837
Medium/high educational level, n (%)	92 (71.9)%	41 (69.5%)	51 (73.91%)	0.433
Disease characteristics				
Disease duration, years*	16.52 (12.59)	16.00 (11.89)	17.01 (13.29)	0.668
Modified Schöber, cm*	7.27 (6.41)	6.95 (5.98)	7.57 (6.94)	0.677
Laboratory				
CRP, mg/L*	0.67 (0.89)	0.64 (0.86)	0.69 (0.91)	0.726
ESR, mm/h*	13.69 (12.39)	12.03 (10.17)	15.04 (14.06)	0.157
Disease activity				
ASDAS-CRP*	2.69 (1.11)	2.10 (0.85)	3.23 (1.05)	**<0.001**
Questionnaires				
MDHAQ				
Physical Function (0-10)*	2.43 (2.04)	1.36 (1.63)	3.38 (1.91)	**<0.001**
VAS-Fatigue (0-10)*	4.47 (3.02)	1.81 (1.41)	7.01 (1.65)	**<0.001**
VAS-Pain (0-10)*	4.71 (2.57)	3.13 (2.01)	6.12 (2.18)	**<0.001**
PGA (0-10)*	4.51 (2.65)	2.93 (2.12)	5.90 (2.27)	**<0.001**
RAPID3 (0-30)*	11.62 (6.37)	7.43 (4.46)	15.31 (5.47)	**<0.001**
sRADAI (0-48)*	9.30 (9.58)	4.39 (4.64)	13.67 (10.69)	**<0.001**
ROS60 (0-60)*	11.52 (9.29)	6.60 (5.71)	15.70 (9.72)	**<0.001**
BASDAI*	4.34 (2.42)	2.65 (1.66)	5.76 (2.03)	**<0.001**
BASFI (0-10)*	3.58 (2.49)	2.75 (1.87)	4.88 (2.21)	**<0.001**
FACIT-Fatigue (0-54)*	34.41 (12.76)	43.75 (5.96)	26.45 (11.57)	**<0.001**
ASAS HI (0 -17)*	6.72 (4.37)	3.89 (3.27)	9.09 (3.74)	**<0.001**

*Data are presented as mean (SD) unless otherwise stated.

P171

COMORBID FATIGUE AND ITS MEASUREMENT IN AXIAL SPONDYLOARTHRITIS PATIENTS

Morlà R.M.[1,8], Frade-Sosa B.[1], Grados D.[2], Gumucio R.[3], Garcia C.[3], Almirall M.[4], Espartal E.[4], Clavaguera T.[5], Sirvent E.[6], Santo P.[6], Reina D.[7], Roig-Vilaseca D.[7], Ponce A.[1], Azuaga A.[1], Ramirez J.[1], Romero Y.[1], Sapena N.[1], Tobalina L.[1], Sanmartí R.[1], Gómez Puerta J.A.[1], on behalf of ESPOcat study Group
[1]Servei de Reumatologia, Hospital Clínic de Barcelona, Barcelona; [2]Hospital Universitari d'Igualada, Igualada; [3]Hospital de Terrassa, Terrassa; [4]Hospital Universitari Vall d'Hebron, Barcelona; [5]Hospital Universitari Dr Josep Trueta, Girona; [6]Hospital de Sant Joan de Déu, Sant Boi; [7]Hospital de Sant Joan Despí Moisès Broggi, Sant Joan Despí; [8]Xarxa Santa Tecla Sanitària, Social i Docent, Tarragona, Spain

Introduction/Objective. Fatigue is a commonly disabling symptom in axial spondyloarthritis (axSpA) patients, recognized as a central domain for ASAS OMERACT. Several tools were described for the measure, but none were standardized. We studied the correlation between two self-report tools to measure fatigue, and between each one and clinical variables in axSpA patients.
Patients and methods. AxSpA patients (ASAS, 2009), excluding those with Chronic Fatigue, were collected consecutively across different hospitals (ESPOCat study group) over 6 months. They completed the following questionnaires: 1. MDHAQ, including a VAS-Fatigue (0–10; fatigued if ≥5/10), Function as modified HAQ (1–10), VAS-Pain (0–10), PGA (0–10); 2. FACIT-Fatigue (0–54, indirect score); 3. ASAS-HI (0–17) for quality of life. Sociodemographic and clinical (disease duration, ASDAS-CRP, modified Schöber, ESR, CRP, and patient-reported outcome: BASDAI, BASFI, FACIT-Fatigue, and ASAS-HI) variables were evaluated. Descriptive analyses and Spearman's correlation coefficients were calculated between FACIT-Fatigue and VAS-Fatigue scores, and between both scales and the rest of the study variables.

Table I. Correlation between VAS-Fatigue and FACIT- Fatigue and laboratory and clinical variables.

	VAS-Fatigue	*p*-value	FACIT-Fatigue	*p*-value
VAS-Fatigue	1	**<0.001**	-0.828	**<0.001**
FACIT-Fatigue	-0.828	**<0.001**	1	**<0.001**
CRP	0.144	0.120	-0.112	0.220
ESR	0.156	0.098	-0.215	**0.019**
BASDAI	0.789	**<0.001**	-0.810	**<0.001**
ASDAS-CRP	0.710	**<0.001**	-0.676	**<0.001**
BASFI	0.718	**<0.001**	-0.759	**<0.001**
Function	0.607	**<0.001**	-0.692	**<0.001**
VAS-Pain	0.757	**<0.001**	-0.711	**<0.001**
PGA	0.760	**<0.001**	-0.703	**<0.001**
Modified Schöber	-0.040	0.734	0.087	0.453
ASAS-HI	0.730	**<0.001**	-0.856	**<0.001**

Values in bold: $p<0.05$.

Results. A total of 128 axSpA patients (13% nr-axSpA) were recruited; 85.2% B27+, 54% fatigued, 62.5% male sex, BMI of 27.26±5.21 kg/m^2, mean (SD) of age: 51,91 (13,09), disease duration:15.52 (12.59) years, BASDAI: 4.34 (2.42), BASFI 3.58 (2.49), ASDAS-CRP: 2.69 (1,11), ASAS-HI: 6.72 (4.37), and FACIT-Fatigue: 34.41 (12.76). A very strong negative correlation (r= -0.828; p<0.001) between the FACIT-Fatigue and the VAS-Fatigue was observed. A significant (p<0.001) strong correlation between FACIT-Fatigue was obtained for VAS-Pain, PGA, Function, BASDAI, BASFI, ASAS-HI, ASDAS-CRP, and a very similar correlation was also found with VAS-Fatigue and these variables. No correlation was found with the modified Schöber or with acute phase reactants, except for a weak correlation between FACIT-Fatigue and VSG (Table).

Conclusion. Comorbid fatigue is highly prevalent in our cohort of axSpA patients, and it can be easily measured in daily practice by a simple 0–10 VAS.

Acknowledgment. Societat Catalana de Reumatologia for a research grant.

P172

SEX DIFFERENCES IN CARDIOVASCULAR RISK AND MENOPAUSE IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS, PSORIATIC ARTHRITIS AND PSORIASIS

Popa-Diaconu D.A.[1], Barenbrug L.[2], Van den Reek J.M.P.A.[2], Van der Horst-Bruinsma I.E.[1], De Jong E.M.G.J.[2], Van Leuven S.I.[1]

[1]Dept. of Rheumatology, Radboud University Medical Center (Radboudumc), Nijmegen; [2]Dept. of Dermatology, Radboud University Medical Center (Radboudumc), Nijmegen, The Netherlands

Delia A. Popa-Diaconu and Liana Barenbrug contributed equally to this study.

Introduction/Objective. Patients with axial spondyloarthritis (axSpA), psoriatic arthritis (PsA), and psoriasis (PsO) are at increased risk of developing cardiovascular diseases (CVD). Hormonal changes during menopause can also contribute to a more severe cardiovascular risk profile (3). However, the interplay between these inflammatory conditions, CVD and menopause remains underexplored. The aim of this study is to assess whether sex influences cardiovascular risk and the prevalence of CVD events in patients diagnosed with axSpA, PsA, and PsO.

Additionally, we aim to investigate whether menopause occurs prematurely in these patients and how its timing affects the cardiovascular risk and rheumatic or psoriatic disease activity.

Methods. A cross-sectional study was conducted among Dutch patients with axSpA, PsA and PsO using an online questionnaire. Information about disease characteristics, cardiovascular risk factors and cardiovascular events, and – for females specifically- menopausal status were retrieved. Early menopause was defined as menopause before 45 years, and premature menopause as menopause before 40 years.

Results. A total of 543 patients (of whom 429 females) completed the questionnaire. Diagnoses included axSpA (in total 215, of whom 177 females), PsA (in total 198, of whom 154 females) and PsO (in total 237, of whom 183 females), with some reporting multiple diagnoses. The median age at questionnaire completion was 61 years [interquartile range (IQR): 19.3] for males and 53 [IQR=18.0] for females.

Sex differences were observed in cardiovascular risk factors and events (Table I). Females had higher body mass index (BMI) compared to males but reported less frequently hypercholesterolemia and hypertension. Atherosclerotic vascular events were more often reported by males compared to females (20.2% vs. 8.4%), while thrombotic events were more often reported by females compared to males (5.1% vs. 0.9%). The age at first atherosclerotic vascular event was comparable across sexes and diagnostic groups, with thrombotic events occurring at earlier ages compared to atherosclerotic events. Females in the axSpA group experienced the thrombotic events at the youngest age (30 [IQR=15.3] years).

Out of all female participants, 38% (n=164) reported having undergone spontaneous menopause. Among these, 80.5% (132/164) experienced physiological menopause (>45 years), 11.0% (18/164) had early menopause and 8.5% (14/164) had premature menopause. The distribution of menopause categories varied slightly across diagnostic groups. The remaining females reported either iatrogenic menopause (10.0%, n=43) or were not yet

	Total			Axial Spondyloarthritis			Psoriatic Artritis			Psoriasis		
	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female
Number of patients	543	114	429	215	38	177	198	44	154	237	54	183
Age at questionnaire completion (years)	55.0 [19.0]	61.0 [19.3]	53.0 [18.0]	52.0 [15.0]	57.5 [15.3]	51.0 [15.0]	57.0 [17.0]	66.5 [18.0]	56.0 [18.0]	57.0 [19.5]	60.0 [22.0]	56.0 [18.0]
Traditional cardiovascular risk factors												
BMI	26.6 [6.7]	25.8 [5.2]	26.8 [7.3]	26.6 [6.6]	26.9 [5.3]	26.1 [6.9]	27.8 [6.4]	26.4 [4.9]	28.6 [6.1]	26.3 [6.4]	24.8 [5.1]	26.7 [6.9]
Ever smoked	282 (51.9)	64 (56.1)	218 (50.8)	106 (49.3)	21 (55.3)	85 (48.0)	104 (52.5)	25 (56.8)	79 (51.3)	125 (51.3)	27 (50.0)	98 (53.6)
CVD < 50 years in 1st/2nd degree family	115 (21.2)	20 (17.5)	95 (22.1)	42 (19.5)	7 (18.4)	35 (19.8)	38 (19.2)	4 (9.1)	34 (22.1)	58 (24.5)	11 (20.4)	47 (25.7)
Hypercholesterolemia	113 (20.8)	32 (28.1)	81 (18.9)	45 (20.9)	12 (31.6)	33 (18.6)	47 (23.7)	14 (31.8)	33 (21.4)	51 (21.5)	13 (24.1)	38 (20.8)
Hypertension	170 (31.3)	40 (35.1)	130 (30.3)	58 (27.0)	14 (36.8)	44 (24.9)	76 (38.4)	16 (36.4)	60 (39.0)	79 (33.3)	20 (37.0)	59 (32.2)
Diabetes	32 (5.9)	9 (7.9)	23 (5.4)	8 (3.7)	2 (5.3)	6 (3.4)	14 (7.1)	3 (6.8)	11 (7.1)	16 (6.8)	5 (9.3)	11 (6.0)
Cardiovascular events												
Patients with any artherosclerotic event[a]	59 (10.9)	23 (20.2)	36 (8.4)	23 (10.7)	8 (21.1)	15 (8.5)	21 (10.6)	6 (13.6)	15 (9.7)	31 (13.1)	13 (24.1)	18 (9.8)
Cerebral infarction	10 (1.8)	3 (2.6)	7 (1.6)	6 (2.8)	1 (2.6)	5 (2.8)	2 (1.0)	0 (0)	2 (1.3)	5 (2.1)	2 (3.7)	3 (1.6)
Myocardial infarction	23 (4.2)	14 (12.3)	9 (2.1)	7 (3.3)	3 (7.9)	4 (2.3)	6 (3.0)	4 (9.1)	2 (1.3)	13 (5.5)	10 (18.5)	3 (1.6)
Transient Ischemic Attack (TIA)	23 (4.2)	4 (3.5)	19 (4.4)	9 (4.2)	1 (2.6)	8 (4.5)	11 (5.6)	1 (2.3)	10 (6.5)	12 (5.1)	2 (3.7)	10 (5.5)
Stent replacement	28 (5.2)	13 (11.4)	15 (3.5)	11 (5.1)	4 (10.5)	7 (4.0)	5 (2.5)	2 (4.5)	3 (1.9)	14 (5.9)	8 (14.8)	6 (3.3)
Patients with any thrombotic event[a]	23 (4.2)	1 (0.9)	22 (5.1)	4 (1.9)	0 (0)	4 (2.3)	13 (6.6)	0 (0)	13 (13.0)	10 (4.2)	1 (1.9)	9 (4.9)
Thrombosis	15 (2.8)	0 (0)	15 (3.5)	3 (1.4)	0 (0)	3 (1.7)	10 (5.1)	0 (0)	10 (6.5)	5 (2.1)	0 (0)	5 (2.7)
Pulmonary Embolism	10 (1.8)	1 (0.9)	9 (2.1)	2 (0.9)	0 (0)	2 (1.1)	4 (2.0)	0 (0)	4 (2.6)	6 (2.5)	1 (1.9)	5 (2.7)
Age at first atherosclerotic event	56 [13.0]	56 [10.0]	55 [16.0]	57.5 [10.3]	57.5 [16.0]	57 [8.3]	53.0 [14.0]	54.0 [19.5]	51.0 [14.0]	55.0 [12.0]	56.0 [10.0]	53.5 [13.0]
Age at first thrombotic event	37.0 [15.0]	-	37.0 [15.3]	30.0 [15.3]	-	30.0 [15.3]	43.0 [15.0]	-	43.0 [15.0]	37.0 [32.3]	-	37.0 [28.0]

P172 - Table I. Self-reported traditional cardiovascular risk factors and cardiovascular events in patients with axial spondyloarthritis, psoriatic arthritis, and psoriasis.
Data is provided as number (%), or median (interquartile range). Percentages are calculated with respect to the total number of patients in that column. [a]One patient can have multiple events.

in menopause (40.6%, n=174). For 49 (11.4%) females, menopausal status was unknown. Among females with axSpA, PsA or PsO who had a spontaneous menopause (respectively n=49, n=69, n=83), respectively 57.1%, 42.0% and 40.9% reported increased rheumatic or psoriatic disease activity during menopause.
Thrombotic events were more often reported in women with early/premature menopause (9.4%, n=3), compared to women with physiological menopause (2.3%, n=3) (Table II).

Table II. Cardiovascular events in females with physiological vs. early/premature menopause.

	Total	Axial Spondyloartritis	Psoriatic Artritis	Psoriasis
Females with physiological menopause	132	38	56	66
Number of females with cardiovascular event	28 (21.2)	12 (31.6)	12 (21.4)	12 (18.2)
Number of females with atherosclerotic event	25 (18.8)	12 (31.6)	9 (16.1)	11 (16.7)
Number of females with thrombotic event	3 (2.3)	0 (0)	3 (5.4)	1 (1.5)
Age at the moment of questionnaire completion	63.0 [10.0]	58.0 [9.0]	64.0 [8.0]	63.0 [9.0]
Age at first atherosclerotic event	58.5 [15.3]	58.0 [16.0]	55.0 [15.0]	55.0 [15.0]
Age at first thrombotic event	49.0 [-]	-	49.0 [-]	38.0 [-]
Females with early/premature menopause	32	11	13	17
Number of females with cardiovascular event	6 (18.8)	2 (18.2)	4 (30.8)	3 (17.6)
Number of females with atherosclerotic event	3 (9.4)	1 (2.6)	2 (3.6)	1 (1.5)
Number of females with thrombotic event	3 (9.4)	1 (2.6)	2 (3.6)	2 (3.0)
Age at the moment of questionnaire completion	61.5 [13.5]	58.0 [9.0]	64.0 [11.0]	65.0 [12.5]
Age at first atherosclerotic event	52.0 [13.0]	48.5 [-]	48.5 [-]	51.5 [-]
Age at first thrombotic event	52.0 [-]	61.0 [-]	61.0 [-]	53.5 [-]

Data is provided as number (%), or median (interquartile range).
Percentages are calculated with respect to the total number of patients in that column.

Conclusion. Sex differences in cardiovascular risk profiles and events were observed in patients with AxSpA, PsA and PsO. Our results suggest that atherosclerotic events might occur more often in males and thrombotic events in females.
Early and premature menopause seem to occur more frequently in women with inflammatory conditions than in the general population. Current literature reports prevalences in the general population ranging from 5.2–7.6% for early menopause and 0.4–2.0% for premature menopause (1-2).
Early menopause is a well-established risk factor for cardiovascular disease (CVD). Given that inflammatory diseases themselves are associated with an increased risk of CVD, it is important to consider whether women with inflammatory diseases may face an additional cardiovascular risk due to early or premature menopause (3). This hypothesis is further supported by our observation that women with early or premature menopause reported thrombotic events more frequently than women with phsyiological menopause, although the number of events was small. This study provides initial insights into the relationship between cardiovascular risk and menopause in patients with axSpA, PsA and PsO. However, the results should be interpreted with caution, as the data were self-reported and the analyses were unadjusted.
Nevertheless, our findings highlight the need for future research to focus on sex-specific cardiovascular disease patterns in these inflammatory diseases and their relationship with menopause. Validation of our results in large cohorts is essential. Such research is crucial to ensure optimal and personalized management, particularly given our findings that disease activity may increase during menopause.

References

1. ROCCA WA, GAZZUOLA ROCCA L, SMITH CY, KAPOOR E, FAUBION SS, STEWART EA: Frequency and type of premature or early menopause in a geographically defined American population. *Maturitas* 2023; 170: 22-30.
2. VELEZ MP, ALVARADO BE, ROSENDAAL N *et al*.: Age at natural menopause and physical functioning in postmenopausal women: the Canadian Longitudinal Study on Aging. *Menopause* 2019; 26(9): 958-65
3. PFEIFER EC, CROWSON CS *et al*.: The influence of early menopause on cardioavscular risk in women with rheumatoid arthritis. *J Rheumatol* 2014; 41(7): 1270-75.

P173

OPTIMAL STRATEGY FOR CARDIOVASCULAR RISK ASSESSMENT IN SPONDYLOARTHRITIS: A COMPARATIVE ANALYSIS BETWEEN TRADITIONAL CARDIOVASCULAR RISK SCORE (QRISK3 SCORE) VERSUS TRADITIONAL RISK SCORE SUPPLEMENTED WITH EITHER CAROTID ULTRASOUND OR CORONARY CT ANGIOGRAPHY

Samanta J.[1], Bodhey N.K.[2], Meher J.[1], Pati S.[2], Nanda R.[3], Pandit V.R.[1]
[1]Clinical Immunology & Rheumatology Division, Dept. of Internal Medicine, All India Institute of Medical Sciences, Raipur; [2]Dept. of Radiodiagnosis, All India Institute of Medical Sciences, Raipur; [3]Dept. of Biochemistry, All India Institute of Medical Sciences, Raipur, India

Introduction. Cardiovascular disease (CVD) is an important cause of morbidity and mortality in inflammatory rheumatic diseases. While rheumatoid arthritis (RA) is associated with accelerated atherosclerosis, cardiovascular risk in spondyloarthritis (SpA) remains less clearly defined. This study evaluated the performance of QRISK3 and compared it with QRISK3 combined with carotid ultrasonography (Carotid US) and coronary CT angiography (CCTA) for cardiovascular risk assessment in SpA.
Methods. In this hospital-based cross-sectional study, adults aged 18–60 years fulfilling ASAS criteria for SpA underwent clinical evaluation, BASDAI assessment, laboratory investigations, QRISK3 scoring, Carotid US, and CCTA. Data were expressed as median (IQR). Comparisons were performed using Mann-Whitney U, chi-square/Fisher's exact, and Spearman's correlation tests, with $p<0.05$ considered significant.
Results. The interim analysis included 48 patients with SpA. Median (IQR) age was 31.5 (15) years, and symptom duration was 7 (6.5) years. Most patients were male (88.23%). Inflammatory low back pain was present in all patients, while peripheral joint involvement was seen in 64.7%. Dactylitis, enthesitis, and uveitis were noted in 2.4%, 7.3%, and 4.9% of cases, respectively. HLA-B27 positivity was present in 67.65%, and the median BASDAI was 2.05 (2.35). Median BMI was 23.2 (7.2), while median QRISK3 score was 0.75% (1.6), categorising all patients as low cardiovascular risk. Median carotid intima-media thickness was 0.4 (0.76) mm on the right and 0.5 (0.8) mm on the left. Median CAD-RADS score was 0. No significant association was observed between cardiovascular imaging findings and sex, BMI, comorbidities, disease characteristics, or treatment modalities. QRISK3 performed similarly to combined imaging-based approaches.
Conclusion. Patients with SpA demonstrated a low burden of subclinical cardiovascular disease. QRISK3 appeared adequate for cardiovascular risk estimation, unlike other inflammatory rheumatic diseases like RA, and routine advanced imaging in asymptomatic patients may not be warranted. Larger longitudinal studies are required to validate these findings.

P174

EXPLORING PAIN KNOWLEDGE AND ATTITUDES IN RHEUMATOLOGY: CLINICAL DECISION-MAKING IN AXIAL SPONDYLOARTHRITIS

van der Kraan Y.M.[1], Paap D.[1,2], Arends S.[1], Reneman M.F.[3], Spoorenberg A.[1]
[1]University Medical Center Groningen, Rheumatology and Clinical Immunology, Groningen; [2]Saxion University of Applied Sciences, Dept. of Physiotherapy, Enschede; [3]University Medical Center Groningen, Rehabilitation Medicine, Groningen, The Netherlands

Introduction/Objective. Many patients with axial spondyloarthritis (axSpA) report chronic pain despite controlled inflammation, suggesting involvement of non-nociceptive pain. This complicates clinical management, as pain may not always reflect inflammatory disease and disease activity assessments are merely about patient-reported symptoms such as pain. Better insight into how rheumatology professionals understand and address pain in axSpA is therefore needed. Aims: 1. To assess the knowledge and attitudes regarding pain among Dutch rheumatologists, trainees, and nurse practitioners using the KNAP questionnaire; 2. To explore how rheumatology professionals interpret pain presentations in axSpA and make management decisions accordingly using clinical vignettes and semi-structured interviews.
Materials and methods. In this cross-sectional mixed-methods study, Dutch rheumatology professionals completed the validated Knowledge

and Attitudes of Pain (KNAP) questionnaire (score 0–150; higher scores indicate greater alignment with contemporary pain neuroscience). Clinical axSpA vignettes and semi-structured interviews were used to explore decision-making and underlying reasoning. A total of 375 rheumatologists, 66 trainees, and 43 nurse practitioners were invited. Data were collected from November 2024 to May 2025 via REDCap and analyzed using descriptive statistics, Chi-square tests, and reflexive thematic analysis.

Results. A total of 109 (23%) rheumatology professionals participated: 67 (62%) rheumatologists, 23 (21%) rheumatology trainees and 19 (17%) nurse practitioners. The KNAP scores were comparable: rheumatologists 83.7; rheumatology trainees 85.3; nurse practitioners 84.1. Across the four vignettes, participants' interpretation of pain shifted from predominantly nociceptive toward nociplastic pain over time. Neuropathic pain was rarely considered. 12 interviews (6 rheumatologists, 3 rheumatology trainees, 3 nurse practitioners) revealed four interrelated themes: 1. Pain knowledge and its limited integration into clinical practice; 2. Context-dependent interpretation of pain; 3. Inflammation-first approach to pain interpretation; 4. Barriers to translating pain recognition into management.

Discussion. Our study identified gaps in pain knowledge and management in axSpA among Dutch rheumatology professionals. Although pain knowledge showed some alignment with contemporary pain science, it remained predominantly biomedical and was not fully integrated into practice. Key barriers included an inflammation-first focus, unclear professional roles and limited collaboration strategies in management.

Conclusion. Improving pain care in axSpA requires stronger integration of biopsychosocial principles, targeted pain education, and better multidisciplinary collaboration.

P175

TOWARDS STANDARDIZED PHYSICAL ACTIVITY ASSESSMENT IN AXIAL SPONDYLOARTHRITIS: TRANSLATION AND CROSS-CULTURAL ADAPTATION OF THE MSQUASH IN 11 LANGUAGES ACROSS 14 COUNTRIES

van der Kraan Y.M.[1], Spoorenberg A.[1], Paap D.[1,2], Benavent D.[3], Navarro-Compán V.[4], López-Medina C.[5], Molto A.[6], Kiefer D.[7], Baraliakos X.[7], Ayan G.[8], Kiliç L.[8], Sepriano A.[9], Moniz A.C.[9,10], Chimenti M.S.[11], Fiannacca L.[11], Konsta M.[12], de Vries M.[13], Brolin S.[13], Maldonado-Ficco H.[14], Perez-Alamino R.[15], Ibáñez S.[16], Garcia D.[16], Bautista-Molano W.[17], Meghnathi B.[18], Patel P.[19], Kim T.J.[20], Shim S.C.[21], Fong W.[22], Kwan Y.H.[22], Gensler L.S.[23], Kiltz U.[7], Ramiro S.[24,25], Arends S.[1]

[1]Rheumatology and Clinical Immunology, University Medical Center Groningen, University of Groningen, Groningen; [2]Dept. of Physiotherapy, Saxion University of Applied Sciences, Enschede, The Netherlands; [3]Rheumatology, Hospital Universitari de Bellvitge, Barcelona; [4]Dept. of Rheumatology, Hospital Universitario La IdiPAZ, Madrid; [5]Rheumatology Dept., Reina Sofia University Hospital, IMIBIC, University of Cordoba, Cordoba, Spain; [6]Université Paris Cité and Université Sorbonne Paris Nord, INSERM, INRAE, Centre for Research in Epidemiology and Statistics, and Dept. of Rheumatology, Bichat Hospital, Assistance Publique Hôpitaux de Paris, Paris, France; [7]Rheumazentrum Ruhrgebiet, Ruhr-University Bochum, Herne, Germany; [8]Division of Rheumatology, Dept. of Internal Medicine, Hacettepe University Medical Faculty, Ankara, Turkey; [9]Nova Medical School, CHRC Campus, Lisbon, Portugal; [10]Hospital de Egas Moniz, Unidade Local de Saúde de Lisboa Ocidental, Lisbon, Portugal; [11]Rheumatology, Allergology and Clinical Immunology, Dept. of Systems Medicine, University of Rome Tor Vergata, Rome, Italy; [12]Rheumatology Dept., Sismanoglio Hospital, Athens, Greece; [13]Division of Rheumatology, Dept. of Medicine, Solna, Karolinska Institutet, and Karolinska University Hospital, Stockholm, Sweden; [14]Rheumatology Section, Hospital San Antonio de Padua, Clínica Regional Del Sud, Río Cuarto, Córdoba, Argentina; [15]Rheumatology Section, Hospital de Clínicas Pte. Dr Nicolás Avellaneda, Tucumán, Argentina; [16]Facultad de Medicina Clínica Alemana de Santiago-Universidad del Desarrollo, Santiago, Chile; [17]Rheumatology Dept., University Hospital Fundación Santa Fe de Bogotá, School of Medicine Universidad El Bosque, Bogotá, Colombia; [18]Dept. of Rheumatology, Marengo CIMS Hospital, Ahmedabad, India; [19]Dept. of Clinical Immunology & Rheumatology, Smt NHL Municipal Medical College & SVP Hospital, Ahmedabad, India; [20]Dept. of Rheumatology, Chonnam National University Medical School and Hospital, Gwangju; [21]Division of Rheumatology, Daejeon Rheumatoid & Degenerative Arthritis Center, Chungnam National University Hospital, Daejeon, Republic of Korea; [22]Dept. of Rheumatology and Immunology, Singapore General Hospital, Singapore; [23]Dept. of Medicine, Division of Rheumatology, University of California San Francisco, San Francisco, USA; [24]Dept. of Rheumatology, Leiden University Medical Center, Leiden; [25]Dept. of Rheumatology, Zuyderland Medical Centre, Heerlen, The Netherlands

Introduction/Objective. Physical activity is a key non-pharmacological treatment for axial spondyloarthritis (axSpA), as outlined in the ASAS-EULAR recommendations (1). The modified Short QUestionnaire to ASsess Health enhancing physical activity (mSQUASH) is a feasible, valid, reliable and responsive questionnaire to assess the amount and type of physical activity in patients with axSpA. The mSQUASH was developed and validated in Dutch and translated, cross-culturally validated in English (2, 3). Our aim was to perform translation and cross-cultural adaptation of the mSQUASH into 11 languages, within 14 countries.

Materials and methods. This study is part of a young-ASAS collaborative project. The Dutch or English mSQUASH was translated into target languages using the forward-backward Beaton protocol. In each country, field testing was conducted with cognitive debriefing interviews in a representative sample of patients with axSpA to verify relevance, comprehensiveness and comprehensibility.

Results. The mSQUASH was successfully translated and field-tested in Spanish, French, German, Turkish, Portuguese, Italian, Greek, Swedish, Hindi, Korean and Chinese. In total, 170 patients with axSpA from 14 countries participated. Across all countries, 84 participants (65%) were male, mean age was 43.7±11.5 years (range 33-55), mean time since diagnosis 10.5±9.8 years (range 3-13) and 91 (70%) were diagnosed with radiographic axSpA. The mSQUASH was found to be clear and relevant. Mean completion time was 5.0±2.7 minutes. No concerns were raised regarding structure or content of domains. Nine patients reported difficulty distinguishing between walking for commuting, leisure activity or sports and exercise. Gardening, home maintenance and shopping required clarification with additional examples or rewording in some countries. Team discussions and minor adaptations addressed all issues.

Conclusion. The mSQUASH was successfully translated and cross-culturally adapted into 11 languages across 14 countries and is now worldwide available to uniformly assess daily physical activity in patients with axSpA, both in clinical practice and in research settings.

References

1. RAMIRO *et al.*: *Ann Rheum Dis* 2023; 82: 19-34.
2. CARBO *et al.*: *Semin Arthritis Rheum* 2021; 51: 719-27.
3. VAN DER KRAAN *et al.*: *RMD Open* 2024; 10:e 004696.
4. BEATON DE *et al.*: *Spine* (Phila Pa 1976) 2000; 25: 3186-91.

P176

ADDRESSING LANGUAGE BARRIERS IN RHEUMATOLOGY OUTPATIENT CARE: INTERPRETER USE AND THE ROLE OF BILINGUAL PHYSICIANS

Wivestad G.A.[1], Pripp A.[2], Thesen T.[3], Michelsen B.[4]

[1]Dept. of Rheumatology, Sorlandet Hospital, Kristiansand; [2]Oslo Centre of Biostatistics and Epidemiology, Research Support Services, Oslo University Hospital, Oslo; [3]Faculty of Health Sciences, OsloMet – Oslo Metropolitan University, Oslo; [4]Dept. of Psychiatry, Sorlandet Hospital, Kristiansand; [5]Faculty of Medicine, Institute of Clinical Medicine, University of Oslo, Oslo; [6]Research Unit, Sorlandet Hospital, Kristiansand

Introduction/Objective. Professional interpreters are essential for equitable healthcare for patients with limited local language proficiency. Increasing interpreter demand has prompted discussions on resource optimization. While ad hoc interpretation may reduce communication quality, studies suggest that bilingual physicians may represent a viable alternative in selected settings. In this study, we aimed to describe the use of interpreters and bilingual physicians in consultations with rheumatology outpatients with limited proficiency in Norwegian.

Methods. We conducted a retrospective observational study at the rheumatology outpatient Dept., Sørlandet Hospital, Norway (2020-2025). The Dept. provides care for patients with inflammatory joint diseases in Agder county (population 323,930, 17.6% immigrants or children of immigrants in 2025). Annual numbers of total consultations and interpreter-assisted consultations were collected and proportions calculated. Consultations involving language-concordant physicians were identified through outpatient scheduling records as a potential indicator of internally provided language support. Registered language needs were categorized and analyzed for trends over time.

Results. Both interpreter-assisted consultations and consultations involving language-concordant physicians increased during the study period (Fig. 1). Of 11.940 outpatient consultations in 2021, 49 (0.41%) involved regis-

tered interpreter services. Corresponding numbers were 96/10.860 (0.88%) in 2022, 125/11.918 (1.05%) in 2023, 86/12.150 (0.71%) in 2024, and 173/12.768 (1.35%) in 2025, representing more than a threefold increase over the study period. Analysis of language trends demonstrated increasing demand for interpreters in Eastern European and Baltic languages (Fig. 2). Consultations with language-concordant physicians accounted for 22-39% of annual consultations requiring language support and involved only Polish-speaking physicians.

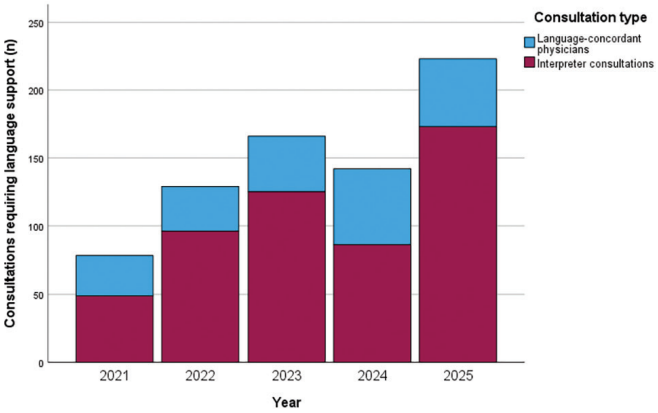


Fig. 1. Interpreter-assisted consultations and consultations with language-concordant physicians in the rheumatology outpatient clinic at Sørlandet Hospital, 2020–2025

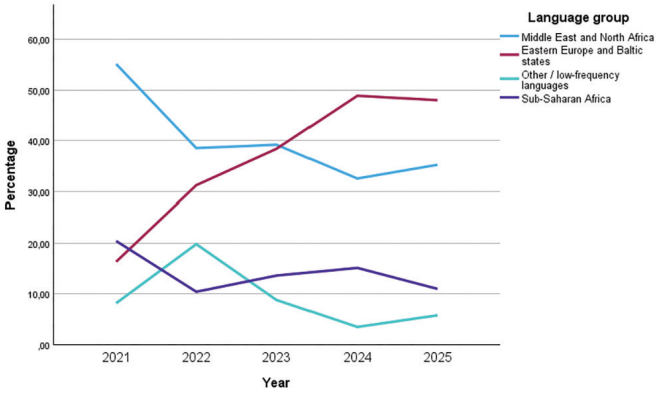


Fig. 2. Distribution of interpreter-assisted consultations by language group in the rheumatology outpatient clinic at Sørlandet Hospital, 2021–2025 (not including language-concordant physicians).

Conclusion. Language barriers in rheumatology outpatient practice were addressed through both interpreter services and bilingual physicians, with increasing use of language support over the study period. Increasing linguistic diversity and variation in language demand highlight the need for structured strategies to ensure safe, effective and equitable communication for patients with limited Norwegian proficiency.

Author index

A

Ábalos Aguilera M.C. P21, P107, P136
Abdellaoui S. P59
Abdulateef N. P59
Abou Abdallah F. P59
Acolty V. O1
Ade-Ojo B. O12
Adler S. O8, P155
Aeberli D. P155
Aguilar M. P142
Aguilar Zamora M. P14
Ahijón-Lana M. O10
Akar S. P76, P93
Akbar-Haase R. P156
Akdoğan A. P129
Aksoy Ö.T. P93
Akyazi Sener E. P63
Al Khater N.R. P59, P97
Al Monla R. O13
Al Rayes H. P59
Albanese P. O11
Albuquerque C. P22
Alcántara Álvarez I. P23
Alchama N. P59
Ali K. P102
Alivernini S. P164
Aljazwi L.M. P59
Allat R. P59
Almeida I. P114
Almeida S. P114
Almirall M. P170, P171
Almodóvar R. P142
Alnaimat F. P59, P97
Alvarez Ortiz L. P169
Alvarez-Rodríguez C. P11
Amiama C. P128
Amine B. P97
Andor M. O8, P155
Andrade S. P158
Andreica I. P27
Antón Enguidanos P. P23
Antunes J. P158
Aouad K. P59
Aparicio-Trejo O.A. P3
Aparnathi M. O2, P49
Apras Bilgen S. P129
Apter D. P32
Araújo F. P114
Arends S. P16, P17, P37, P88, P174, P175
Arévalo Ruales K. P142
Arias de la Rosa I. P21, P107, P136
Armagan B. P63, P161
Armstrong A.W. P147
Arrieta-Martínez P.I. P2
Asal S. P159
Aslan B. P160
Assassi S. P38
Ata N. P161
Atawneh S. P59
Attar M. P98
Attari H. P165
Aubry S. O4
Avellanal V. P24, P33, P34, P122
Ávila-Ribeiro P. P158
Awadh N.I. P59
Ayan G. P28, P64, P65, P66, P67, P68, P69, P70, P72, P73, P91, P92, P115, P129, P161, P166, P175
Aydemir M. P160
Aydin S. P35, P7, 7P81, P82
Aydin S.Z. P42
Ayoub L. P59
Aysin I.K. P93
Azouz A. O1
Azuaga A. P170, P171

B

Babaoglu H. P161
Bachmann M. O8
Bahiri R. P59, P97, P159
Baillet A. O4, O6, P108
Baird T. P98
Bajracharya R. P137
Bakland G. P111
Baraliakos X. O9, P10, P20, P27, P66, P67, P68, P87, P103, P119, P120, P124, P130, P137, P139, P140, P154, P156, P157, P163, P175
Barbarroja N. P21, P107, P136
Barcelos A. P90
Barcia Cansino A. P24, P33, P34
Barcia Cansino M.A. P122
Bardet C. O11, O13
Bardini C. P32
Barenbrug L. P172
Barnett R. P12, P116
Barone F. INV13
Barr A. P121
Barranco A.M. P21, P136
Baskurt Z. P61
Bassara-Macedo R. P22, P54
Basu N. P125
Batra R. P135, P143
Bauer E. P165
Bautista-Molano W. P20, P24, P33, P34, P122, P175
Bayindir Tsechelidis O. O7
Bazin D. P109
Bazin T. P164
Beaufrère M. P109
Behrens F. P157
Belén Gómez A. P24
Bembridge L. P131
Ben Abdelghani K. P59, P97, P159
Ben Ammar L. P31
Ben Ayed H. P31
Benavent D. P175
Benawra J. P168
Benegas M. P24, P33, P34, P122
Bensley K. P135, P143
Bento da Silva A. P28, P64, P66, P67, P68, P69, P70, P91, P92
Berg I.J. P28, P64, P69, P70, P91, P92
Bergot A.S. P109
Bergstra S.A. P149
Bermudez Bermejo P. P122
Bermudez Bermejo P.E. P24, P33, P34
Bernal-Alferes B. P11
Bernard N. P56
Bernard S. O7, P81, P82
Bernardes M. P114
Bessa A. P168
Bilici R. P162
Biln N. P43
Bird P. O7, P144
Birinci S. P161
Bissonnette R. P147
Bittar M. O7, P25, P26
Blaney Davidson E.N. P5
Bleakley S. P43
Blom A.B. P5
Bodhey N.K. P173
Bohórquez C. P128
Boni V. P13, P44, P145
Bonilla G. P128
Bonny M.A. P137
Boonen A. P20, P57, P58, P138, P153
Bos R. P88
Boukamel A. P109
Boussaa H. P59, P97, P159
Bouvard B. O6
Bowness P. P60, P98, P99, P105
Brance L. P122
Brance M.L. P24, P33, P34
Brändli J. O8, P89, P155
Braun J. P66, P67, P68
Braunhofer L. P5
Bray T. P96
Breban M. O6, P60, P109, P164
Broche L. O4
Brocq O. O6
Brolin S. P175
Brown A.P. P60
Brown M.A. INV7, P38
Brown S. P9
Brulhart L. P50, P85
Brülhart Bletsas L. P155
Brun E. O4
Bubová K. P89
Bulat B. P129
Bulbol G.A. P54
Bulbol G. P22
Bulut S. P129
Burgos-Vargas R. P81, P82, P104
Bürki K. O8, P155
Burrows D. P116
Butrimiene I. P84

C

Caballero L. P24, P33, P34, P122
Cafone J. P147
Caggiano V. P86
Calabrese G. P44
Calixto O.J. P163
Calot A. O13
Calvo-Gutiérrez J. P107
Calvo-Sparks C. P118
Cameron C. P127
Cameron M. P127
Candelas G. P142
Candia D.L. P122
Candia Zuniga D.L. P24, P33, P34
Cantarini L. P86
Capellino S. P27
Capelusnik D. P20, P57
Caplan L. O7, P81, P82
Caporali R.F. P102
Carapellucci A. P42

Carbo M.J.G. P16, P17
Carmona R. P42
Carneiro H. P114
Carneiro J. P22
Carneiro J.N. P54
Carones A. P114
Carrino J.A. O7, P81, P82
Carron P.M. P20
Carron P. O5, P75
Carter K. P124
Carvalho T.R.A.F. P54
Casado-Lima B. P105
Casalla L. P24
Casallas L. P33, P34, P122
Casasola J. P24
Casasola J.C. P33, P34
Casasola-Vargas C. P11
Casasola Vargas J.C. P6, P104, P122
Casel B. O13
Castagna F. P137
Castillo A.C. P142
Castillo I. P128
Castillo Dayer P.V. P142
Castillo Ortiz A. P24, P33, P34
Castillo Ortiz A.A. P122
Castrejón I. O10
Castro G. P22
Castro G.R.W. P54
Castro S. P148
Cavalcanti N. P22
Cavalcanti N.G. P54
Cavill C. P43, P168
Cazzola D. P18
Cerón Cruz E. P6, P11
Chakravarty S. P135
Chakravarty S.D. P143, P147
Chalian M. O7
Chan A. P99
Chan J. P42, P81, P82
Chaparro-Barrera E. P2
Charca Mamani L.C. P14
Chary-Valckenaere I. P165
Chatel J.M. P109
Chen L. P99
Chen S. P117
Chen S.D. P167
Cheng E. P144
Cherbuy C. P164
Chim T. P35, P77
Chimenti M.S. P175
Chinchay L. P24, P33, P34, P122
Ching D. P62
Chirino López Y. I. P6
Choi D. P25
Chow N. P35, P77
Ciancio A. P102
Ciccia F. P102
Cinakli H. P69, P70
Cincinelli G. P102
Ciurea A. O8, P50, P89, P155
Clarke C. P12
Claudepierre P. O6
Clauw D. P26
Clavaguera T. P170, P171
Coates L. P135
Coates L.C. INV20, P134, P143
Cohen C. P60
Colbert R.A. INV5, P40
Collantes-Estévez E. P21, P107, P136
Constantin A. O6
Coppola G. P164
Cormier G. O6
Coronado-González M. P118
Correale M. P35, P77
Coskun Sagirkaya S. P63
Costantino F. INV6, P60
Courvoisier D. P85
Courvoisier D.S. P50
Couto M. P114
Cristiano F. P13, P44, P145
Cuevas-López B. P3
Cuevas-Martínez R. P1
Cui R. P71
Cullen E. P156
Cunha A. P114

D

Dadashova R. P42
D'Agostino M.A. P13, P44, P145, P164
Dahou Makhloufi C. P59
Dai S.M. P71
Dalbeth N. P62
Dalix E. O6
Damen C. O5
Dan D. O8, P50, P85, P155
Danve A. O7, P81, P82
Das S. P131
de Bruin L. P64, P66, P67, P68, P69, P70, P72, P91, P92
de Bruin L.J.E. P28, P73
De Craemer A.S. O3, O5, P75
de Hooge M. P28, P64, P66, P67, P68, P69, P70, P72, P73, P74, P75, P89, P91, P92, P119, P120
De Jong E.M.G.J. P172
De Jong H.M.Y. P36
de la Loge C. P137, P138, P140
de la Morena I. P142
de la Morena Barrio I. P14
de la Torre N. P142
de la Torre-Rubio N. P118
De Lorenzis E. P13, P44, P145, P164
De Marco G. P134
de Miguel E. P128
De Mulder-Rougvie M. P105
de Peyrecave N. P138
De Piano R. P13
de Vlam K. P39, P79, P137
de Vries M. P175
Decruy T. O3
Del Castillo N. P148
Deligeorgakis D. P152
Dell'Orso S. P40
Deman E. P85
Demir E. P146
Demougeot C. O4, P108
den Broeder A. P123
den Broeder A.A. P134
den Broeder N. P123
Denis A. O6
Deodhar A.A. INV15, O7, P25, P26, P81, P82, P124, P140
Dernis E. O6
Desmarets M. P113
Detavernier A. O1
Devauchelle-Pensec V. O6
Dhahri R. P31
Di Mario C. P164
Di Meglio N. P86
Di Vincenzo F. P164
Di Vittori L. P145
Dias J.M. P114
Dias Munhoz D. P109
Díaz Mora B. P23
Diekhoff T. P87, P89
Diers A.R. INV13
Diniz V. P22
Diniz V.G. P54
Distler O. O8, P155
Djennane M. P59
Do Cruzeiro M. O11
Doblas S. O13
Domínguez J. O10
Domínguez López M.L. P6, P11, P104
Donckier De Donceel E. O1
Donmez D. P129
Doorschodt T.V. P78
Dougados M. P59, P159
Duan L. P99
Dubreuil M. O7, P138, P140
Dufau E. P24, P33, P34, P122
Dumas E. P109
Duran Barragan S. P24, P33, P34, P122

E

Ediboğlu E. P76
Eguez Del Pozo M.F. P24, P33, P34, P122
Ehrhardt M.J. P116
El Arman M.M. P41
Elbana N.K. P41
Elewaut D. O1, O3, O5, P75, P109
Elmoufti S. P138, P140
Elsehrawy G.G. P50, P85
Elvenes J. P111
Engel A. P25
Erbasan F. P160
Erpek E. P76
Ertenli A.I. P129
Escudero-Contreras A. P21, P107, P136
Eshed I. P87, P89
Espartal E. P170, P171
Essex M. P112
Eugénio G. P90
Exarchou S. P28, P64, P69, P70
Eyre-Olmos C. P142
Ez-Zaitouni Z. P36

F

Fakharzadeh S. P135
Fakih O. P29, P30, P121
Fanf H. P99
Fano M. P13
Farahi N. O12
Fauny M. P165
Favier B. P108
FeltenR. O6
Fenniche I. P31
Fernandes M. P53
Fernandes M.A. P51, P52, P55, P150
Fernandes J.M. P22
Fernandes J.M.C. P54
Fernandez Avila D. P122
Fernández-Ávila D.G. P24, P33, P34
Fernández Prada M. P142
Ferreyra Garrot L.G. P24, P33, P34, P122
Fiannacca L. P175
Fichera E. P168
Fidanci A.A. P161
Fidler W. P35, P77
Firlatan Yazgan B. P129
Flores-Adame I.D. P2
Fogel O. O11
Fong W. P175

Fonseca R. P114
Fonseca J.E. P158
Fontova R. P148
Förger F. O8, P155
Foroozan Boroojeni S. O2
Forslund S.K. P112
Forte G. P102
Fossi Djembi L. P56
Frade-Sosa B. P170, P171
Frediani B. P86
Freeston J.E. P121
Frison E. P60, P109
Frumker L. P127

G

Gamal S.M. P59
Gamboa R. P122
Gamboa-Cardenas R.V. P24, P33, P34
Gandolfo S. P102
Ganz F. P137, P138
Gao T. P125
Garchon H.J. P60
Garcia C. P170, P171
Garcia D. P175
Garcia J. P114
García-Arroyo F.E. P3
García-Delgado M.D. P107
García Hernández A.L. P6, P11
García-Magallón B. P118
Garcia Martos A. P14
Garcia Salinas R. P24, P32, P33, P34, P122
Garteiser P. O13
Gasbarrini A. P164
Gautreau G. P109
Gavi M.B.R.O. P54
Gavi M.B. P22
Gavotti C. P13, P164
Gaydish C. P25
Gendron E. P35, P77
Génot J. P165
Gensler L.S. O7, P16, P20, P38, P40, P82, P139, P175
Georgiadis S. P94, P95
Gerritsen S. P123
Gervais E. O6
Gervaise P. P60
Geusens P. P79
Gharbia O.M. P41
Gharsallah I. P31
Gheyle L. P119, P120
Giacca R. P102
Gil-Calderon D. P24, P33, P34
Gil Calderon D.R. P122
Gil Velez R. P169
Gillett D. O12
Glatigny S. P60, P109
Godoy-Tundidor H. P118
Gold L.S. P147
Golebiovski R. P22
Golebiovski R.T.M. P54
Goll R. P111
Gomez A.B. P33, P34, P122
Gómez-Puerta J.A. P170, P171
Gonçalves H. P114
González M.A. P128
González-Chávez S.A. P1, P2, P3, P110
González Ferrández J.A. P142
González-Martín J.J. O10
Gonzalez Ponce C.M. P141
Gorial F.I. P59
Goriely S. O1
Gossec L. O6, P20, P30, P138, P157
Gosvig K. P89, P94, P95
Götschi A. O8
Göttgens I. P15
Goupille P. O6
Gouze H. P164
Gracey E. INV4
Grados D. P170, P171
Grainger A.J. O7
Grau García E. P23
Gregová M. P89
Griffith J. O7
Groothuizen S. P36, P78
Grosswiler S. O8, P155
Gu J. P117
Gu J.R. P167
Guaracha Basañez G.A. P24, P33, P34, P122
Gudbjörnsson B. P89
Guet L. P108
Gui Y. P43
Guillén-Astete C.A. O10
Güloğlu S.B. P160
Gumucio R. P170, P171
Güner I. P162
Guo Q. INV13

H

Hadsbjerg A.E.F. P89, P94
Hagfors J. P111
Haibel H. P112
Hamamou A. P15
Hamdi W. P59, P97, P159
Hanaee Y. P107
Hannani D. P108
Hannawi S. P59, P159
Hansen I.M. P111
Hansson P. P111
Haouichat C. P59
Harmsen D.H. P16, P17
Haroon N. O2, P35, P49, P77, P81, P82
Harris R. P26
Harrison A. P62
Harrison S.R. P30
Hautzel H. P156
He Z. P17, P37
Hecquet S. O4
Heddi F.Z.Y. P59
Heiberg M. P111
Henriques C. P114
Hensor E.M.A. P96
Hermans K. P153
Hernandes M.C. P98
Herregods N. O7
Hetland M.L. P89
Heydari-Kamjani M. P126, P127
Hilliquin S. O11, O13
Hmamouchi I. P20, P59, P97, P159
Hoad C. P96
Holec P. P100
Horan J.G. P168
Horbal N. O7, P42, P81, P82
Houard X. O11
Huaylla Quispe A. P14
Hutton J. O12
Hwang M.C. P38

I

Iaconangelo C. P147
Ibáñez S. P175
Ibeke N. P49
Ibrahim K. O11
Ilardi G. P102
Im K. P135, P143
Inman R.D. O2, P35, P49, P61, P77, P106
Iodice D. P145
Ishchenko A. P39
Ishimori M.L. P38
Islam S. P40
Ismailoustafa N.M. P59

J

Jacoutot M. P60, P109
Jadon D. O12
Jans L. P75
Jans N. O7
Jaremko J.L. O7
Jebri R. P31
Jeon C. P8
Jiang L. P99
Jiang Y. P134, P135, P143
Jin L. P98
Jo S. O2, P7, P8, P101
Johnsen P.H. P111
Joos R. P79
Ju J.H. P80, P133
Juárez-García M. P128
Juditzki M. P27
Jung J.Y. P80
Jurik A.G. O7
Jurisica I. P49, P61
Just M. P148

K

Kader S.H. P83
Kalyoncu U. P65, P115, P129, P161, P166
Kang J.H. P134
Kapoor M. O2, P61
Karacabeyli D. INV12
Karadag O. P129
Karmacharya P. INV9, O7
Karpman P. P109
Kart Bayram G.S. P129, P166
Kato K. P124
Kattan A. P18
Keeling S.O. P42
Keshavarazi S. P49
Khalifa D. P59
Khmelinskii N. P158
Khodaee S. P168
Kiefer A. P175
Kiefer D. P130
Kieskamp S.C P17
Kiliç L. P65, P115, P129, P166, P175
Kiltz U. P10, P20, P103, P130, P137, P140, P156, P163, P175
Kim C.R. P4
Kim D. P8
Kim J.G. P80
Kim M.J. P101
Kim T.H. O2, P7, P8, P48, P101
Kim T.J. P48, P101, P175
Kim W.U. P80, P133
Kim Y. P48
Kiraz S. P129
Kishimoto M. P20, P124, P157
Klümpers F.E. P163
Knight J.C. P60
Knol R.J.J. P78

Koc G.H. P134
Kocaayan H. P76, P93
Koenders M.I. P5
Kohno M. P26
Kok M.R. P134
Köleséri L. P157
Kollmeier A. P135
Kollmeier A.P. P143
Konsta M. P175
Korjagina M. P137
Korshko M. O2
Kougkas N. P152
Krabbe S. P89
Krause D. P130
Krupinski L.D. P126, P127
Kucuksahin O. P161
Kuhn K.A. INV3
Kulyk M. P79
Kumar D. P40
Kumru B.G. P162
Kustas A.A. P129
Kwan Y.H. P175
Kwok S.K. P80, P133
Kwon S.R. P4
Kyburz D. O8, P155

L

Lacarpia A. P86
Lack J. P40
Laddach A. O12
Ladehesa-Pineda M.L. P107
Lage R. P22
Lage R.C. P54
Lagunes-Galindo I. P124
Lalazi B. P10, P27, P103, P156, P163
Lamarque D. P109
Lambert R.G.W. O7, P42, P81, P82, P89, P119, P120, P139
Landewé R. P20, P28, P36, P57, P58, P64, P66, P67, P68, P91, P92, P119, P120, P149
Langella P. P109, P164
Larsen I. P100
Laxminarayan R. P131
Lazarou I. P50
Le Brun F.O. P119, P120
Lebughe P.L. P132
Lebwohl M.G. P147
Lee B.W. P80, P133
Lee E.Y. P101
Lee J.J. P80, P133
Lee J.S. P101
Lee S. P8, P48
Lee S.H. P8
Lee Y.C. P25
Lee Y.J. P101
Leek F. O12
Legoff B. O6
Leijten E. P123
Leijten E.F.A. P5
Lema Martinez J.M. P134
León Torralba P. P142
Lequerre T. O6
Lespessailles E. O6
Li J. P99
Li M. P144
Li Q. O12
Li S. P147
Li X.M. P167
Liberatore D'Agostino A. P145
Lim M. P61, P106
Lim M.J. P4
Littman N.S. P37
Liu F. P99
Liu J. P19
Lively S. P61
Lizzio M.M. P164
Llamas-Urbano A. P107
Loeuille D. P165
Lois-Iglesias A. O10
Londono J. P24, P33, P34, P122
Lopes A.R. P158
López Broseta P. P148
López-Medina C. P20, P21, P74, P107, P136, P175
López Montilla M.D. P21, P107, P136
López-Pedrera C. P21, P107
Lories R. P39, P79
Lotfi-Emran S. P40
Loureiro M. P90
Lowe J. P20
Loya-Rivera M. P2, P3
Lozenski K. P135, P143, P144, P147, P157
Lubrano E. P157
Luime J.J. P134
Lukas C. O6
Lukasik Z. O3, P75

M

Maas F. P88
Macchar R. P49
Machado N. P22
Machado N.P. P54
Machado P. P87
Machado P.M. P20, P119, P120
Machaly S.A. P41
Macias Oviedo L. P24, P33, P34, P122
Macia-Villa C. P142
MacLeod A. P35, P77
Magnanimi C.T. P145
Magrey M. O7, P81, P82, P140
Magrey M.N. P126, P127
Magri S.J. P32
Mahler E. P123
Mahmoud I. P59, P97, P159
Maksymowych W.P. O7, P42, P43, P81, P82, P119, P120, P124, P139
Maldonado-Ficco H. P175
Malheiro O. P22
Malheiro O.B. P54
Mangili A. P119, P120
Manica S. P114
Manuello T. O3, P109
Manzaneque Sanchez J. P169
Manzanera A. INV13
Maraquin P. P109
Marín J. P142
Marin Zucaro N. P24, P33, P34, P122
Marinho A. P22
Marinho A.O. P54
Marona J. P114
Maroof A. P59, P83
Marotte H. O6, P43
Marques C. P22
Marques C.D.L. P54
Marques M.L. P28, P64, P66, P67, P68, P72, P73
Marston J.L. P105
Marten A. P138, P139
Martin B. P56
Martin L. P42
Martín-Arranz E. P128
Martín-Arranz M.D. P128
Martín-Bescos A. P118
Martinelli E. P102
Martínez-Moreno J.M. P107
Martínez-Prada C. P142
Martini F. P86
Martini N. P86
Martins C. P114
Martín-Salazar J.E. P21, P136
Marzo-Ortega H. P30, P96, P121, P134
Masetto A. P42
Masnata R. P13, P44, P145
Massa S. P134
Massaro A. P44
Mateus M. P114
Matias C. P114
Matijas M. P151
Matos A. P114
Mauny F. P56
Mauro D. P102
Mavinga A.N. P132
Mayer A. O7
Mazo P. P118
Mazzei M.A. P86
Mbuyi J. P132
Mbuyi-Muamba J.M. P132
McDonnell S. O12
McGonagle D. P96, P98
McGovern N. O12
McInnes I.B. P147
Mease P. P125, P135
Mease P.J. P81, P82, P143
Mease P.M. P20
Medina G. P24, P33, P34, P122
Medina D. P122
Medina Bornachera D. P24, P33, P34
Medina Reyes I. P6
Meghnathi B. O9, P45, P46, P87, P175
Meher J. P173
Meirinhos T. P51, P52, P53, P55, P150
Mejia-Maggi N. P32
Melchior M. O1
Melgarejo P. P24, P33, P34, P122
Melz I. P25
Méndez Díaz L. P169
Menin R.C. P22, P54
Meo M. P164
Meridor K. P121
Merino-Argúmanez C. P118
Merlo-Ruiz C. P107
Merola J.F. P147
Mestanza Peralta M. P24, P33, P34, P122
Meyer F. P113
Miceli C. O6
Miceli-Richard C. O11, O13, P113
Michelsen B. P62, P176
Micheroli R. O8, P50, P85, P89, P155
Migliorini G. P60
Miguel C. P114
Millar V. P156
Mintrop B. P130
Mohammadian H. P102
Mohith Kumar Reddy P. P47
Möller B. O8, P50, P89, P155
Møller J.M. P89, P94, P95

Moltó A. P20, P29, P30, P74, P149, P154, P175
Moniz A.C. P175
Monjo I. P128
Montolio Chiva L. P142
Montvydaite M. P84
Moreno M. P142
Moreno Caño E. P136
Moreno Martínez M.J. P142
Moreno Morales J. P141
Moreno Ramos M.J. P142
Moreno-Soltero M.F. P2
Moret M. P165
Morizot C. P165
Morlà R.M. P170, P171
Moroli M. P13
Mortier C. O3
Mosher D. P42
Mpitouli A. P152
Mulder M.L.M. P15, P134
Muller K. P119, P120
Mulumba C.B. P132
Muñoz-Martínez P.F. P23
Muñoz-Muñoz V. P110
Murphy J. P126

N

Nadimpalli M. P156
Naffaa M. P125
Nakamura A. P49
Nam B. P48
Nanda S. P173
Nash P. INV11
Natal M. P114
Natalello G. P13, P44, P145, P164
Navarro-Compán V. P20, P119, P120, P125, P128, P140, P154, P175
Navid F. P40
Ndaie G.M. P132
Neerinckx B. P39
Nero P. P114
Ney L. P10, P103
Nguyen M. O1
Nguyen P. P49
Nicholls D. P137
Nichols G. INV13
Niedda F. P13
Nieto-González J.C. O10
Nikiphorou E. P20, P138, P154
Nissen M.J. O8, P50, P85, P89, P155
Nixon D. P105
Njøs H. P111
Noci J. P128
Nokay M.T. P160
Novais T. P114
Nuño-Nuño L. P118
Nzeusseu Toukap A. P79

O

O'Neill J. O7, P81, P82
Ochoa de Aranda B. P14
Ochôa Matos C. P158
Ochtrop M.L.G. P54
Oertelt-Prigione S. P15
Oh S. P133
Oh S.H. P80
Öhagen P. P137
Oliveira D. P114
Oliveira T. P22
Oliveira T.L. P54
Olivier C. O4
Oortgiesen M. P137
Orchtrop M. P22
Ørnbjerg L.M. P89
Ortega-Castro R. P107
Ortega-Mejia I.I. P6, P11, P104
Ortiz-Buitrago P. P21, P107, P136
Ortiz-Domínguez V.M. P2
Ortolan A. P13, P20, P44, P145, P164
Østergaard M. O7, P89, P94, P95, P119, P120, P139, P144
Østgård R.D. P111
Ouahbi D. P109

P

Paap D. P16, P17, P37, P174, P175
Pacheco A. O2, P61
Pacheco M.S. P114
Pacheco-Silva C. P110
Pacheco-Tena C. P1, P2, P3, P110
Pais De Barros J.P. P113
Paiva B.E. P54
Paley M.A. INV2
Palma Sanchez D. P14
Pandit V.R. P173
Pandya S. P45, P46
Parafioriti A. P102
Pardaens L. P75
Parikh B. P124, P125
Park H.W. P80, P133
Park S.H. P80, P133
Park Y. P133
Parren P.W.H.I. INV14
Pascart T. O6
Paschke J. O7, P42
Passalent L. P35, P77
Pastrello C. P61
Patel P. P175
Patela M. P51, P52, P53, P55, P150
Paternina Martinez D. P9
Pati S. P173
Patti G.D. P13, P145
Paul S. P40
Pavy S. O6
Pedersen S.J. O7, P89, P94, P95
Pedron A. O1
Pehlivan Y. P146
Peiteado D. P128
Peluso G. P44, P164
Penedo-Alonso R. O10
Peng J. P43
Penkava F. P98, P99
Pereira A. P156
Perez-Alamino R. P175
Pérez Hurtado A. P23
Pérez-Sánchez C. P21, P107, P136
Perkovic D. P151
Pers Y.M. O6
Petito V. P164
Peyrin-Biroulet L. P165
Pezeshk P. O7
Philips C. P124
Pina Vegas L. P20
Pinheiro M.M. P22, P54
Pintaric K. P89
Pinto A. P53
Pinto A.S. P51, P52, P55, P150
Pirson C. P35, P77
Placido F. P86
Plasencia-Rodríguez C. P128
Poddubnyy D. O9, P20, P87, P98, P112, P124, P125, P137, P139, P140
Ponce A. P170, P171
Popa-Diaconu D. P172
Popa-Diaconu D.A. P15
Poza J. P128
Prajapati C. P139
Prati C. O4, P29, P30, P56, P108, P113
Presles.E. O6
Prieto-Carrasco R. P2, P3, P110
Prieto-Chávez J.L. P104
Pripp A. P176
Proft F. P112, P138
Protopopov M. O9, P87, P112
Puche-Larrubia M.A. P107, P136
Puig L. INV10
Pujol S. P56
Pus L. P35, P77

Q

Qaiyum Z. O2, P61, P106

R

Raanes S.S. P111
Rademacher J. P98, P112
Radic J. P151
Radic M. P151
Rahman P. P49, P135, P143, P157
Rahmouni S. P59
Raimondi M. P102
Raimondo M.G. P102
Raimundo D. P114
Ramirez J. P170, P171
Ramírez-García J. P137
Ramiro S. O8, O9, P20, P28, P30, P57, P58, P64, P66, P67, P68, P69, P70, P72, P73, P74, P87, P91, P92, P138, P139, P140, P149, P175
Ramming A. P102, P156
Ramon A. P113
Ramonda R. P28, P64, P69, P70, P91, P92
Ramos Castro D. P23
Rampakakis E. P157
Rampersaud R. P35, P77
Rancon F. O6
Redeker I. P130, P163
Redha A.A. P59
Reich K. P156
Reijnierse M. P64, P66, P67, P68, P69, P70, P72, P73, P74, P91, P92
Reina D. P170, P171
Reis J. P42
Reitars M.H. P100
Remalante-Rayco P. O2
Reneman M.F. P174
Rennie W. O7
Resende G.G. P22, P54
Resende S. P158
Reveille J.D. O7, P38, P81, P82
Reyes-Gopar H. P105
Reyes-Jara G. P32
Reyes Maldonado E. P6, P11

Reyes-Servin O. P147
Reynolds L. P114
Reynoso E. P144
Ribeiro S. P22
Ribeiro S.L.E. P54
Richardson B. P127
Richter G. P88
Rios Rodriguez V. P112
Risch I. INV2
Ritchlin C. P135
Ritchlin C.T. P143, P144
Rivera V. P122
Rivera Terán V. P24, P33, P34
Robert V. P109
Roberts R. P62
Roberts S.J. P9
Robustillo Villarino M. P14
Rodrigues C.L. O8, P89, P155
Rodrigues D. P22
Rodrigues D.L.N. P54
Rodrigues M.M. P114
Rodrigues S. P114
Rodríguez-Muguruza S. P148
Rohekar S. P42
Roig-Vilaseca D. P170, P171
Roman I. P10, P103
Román Ivorra J.A. P23
Romand X. O4, P108
Romero Y. P170, P171
Romero-García H.A. P104
Romero-Huerta R. P2
Romero-López J. P. P6, P11, P104, P105
Romero-López N. P104
Romero-Zurita L. P21, P107, P136
Rostom S. P159
Rotar Z. P89
Roulandt R. O3
Rouse P. P18
Rouse P.C. P12
Roux C. O6
Rua C. P51, P52, P53, P55, P114, P150
Rudwaleit M. O9
Rueda J.L. P128
Ruizesparza-Hinojos D.A. P2
Ruiz-Ponce M. P21, P136
Ruiz-Vilchez D. P21
Ruta A.O. P32
Ruyssen-Witrand A. P137

S

Saad C. P22
Saad C.G.S. P54
Saad S. P59
Saas P. P113
Sabido Sauri R. P35, P77
Saeys Y. INV1
Saffore C. P125
Saha A. O12
Said-Nahal R. P60, P109
Salas-Leiva J.S. P1
Salmani H. P153
Salmon J.H. O6
Salonen D. O7
Salzillo A. P102
Samanta J. P173
Samões B. P114
Sampaio-Barros P.D. P22, P54
Sánchez Azofra M. P128
Sánchez Pérez S. P6, P104
Sanchez Prado E. P24, P33, P34, P122
Sanmartí R. P170, P171
Sanmartin Martinez J.J. P14
Sansom S. P98
Santo P. P170, P171
Sanz-Sanz J. P118
Sapena N. P170, P171
Saraiva L. P114
Saraux A. P74
Sarri S. P145
Satis H. P161
Sausameda-García J. P2
Sbalchiero J. P86
Scaldaferri F. P164
Schaeverbeke T. O6
Schaughency P. P40
Scher J.U. P144
Scholz G.A. P50
Schütz C. P10, P103
Scozzafava G. P60
Scriffignano S. P79
Selinger C. P96
Sellam J. O6
Senabre Gallego J.M. P142
Sengupta R. P12, P18, P43, P116, P168
Sepriano A. O9, P20, P87, P149, P175
Serfaty A. O7
Seridi L. P144
Sewerin P. P156, P163
Shagroni T. P147
Shah M. P9
Shah S. P45, P46
Shah U. P45, P46
Sharaf M. P157
Sharma S. P98
Shenker N. O12
Shi H. P99
Shim S.C. P175
Shin J.H. P48
Siebert S. P157
Siegmund B. P112
Sieper J. P20
Silale G. P84
Silva C. P51, P52, P53, P55, P150
Silva G. P158
Silva L. P114
Silva R.Q. P157
Silva-Dinis J. P114
Simac Prizmic P. P151
Simeó Vinaixa M. P23
Simon D. INV17
Simone D. P98, P99
Singh N. P100
Sirvent E. P170, P171
Skouvaklidou E. P152
Smith D P26
Smits M.L. P153, P154
Snoj Z. P89
Soares A.M. P22, P54
Solis-Mayor J. P32
Solmaz D. P76, P93
Sommerfleck F. P24, P33, P34, P122
Song H.S. P4
Song X.Y. P71
Sonkaya Y. P130
Sooriyaarachchi D. P131
Soriano E.R. P157
Soudy R. P164
Soundararajan J. P100
Souza E.C.R. P54
Spagnolo G. P145
Spoorenberg A. P17, P37, P88, P174, P175
Spoorenberg J.P.L. P16
Sridhar A. P26
Srinath A. O2, P49
Stamp L. P62
Stebbings S. P62
Steimer A. O8, P89, P155
Steinfeld S. P79
Stovall R. O7
Súarez-Ferrer C. P128
Sudol-Szopinska I. P89
Summers C. O12
Sumra A. P45, P46
Sushko U. P114
Szymczak F. O1

T

Taha Z.I. P59
Tak P.P. INV13
Talukdar A. P49
Tamilselvan B. P127
Tang K. P19
Tang M. P61, P106
Tao Y.L. P71
Tascilar K. P161
Tavares-Costa J. P114
Tavasolian F. P61
Taylor P. P125
Tchetverikov I. P134
Teh J. P98
Teixeira P.M. P90, P114
Tekaya R. P59, P97, P159
Tekeoglu S. P91, P92
Then J. P24, P33, P34
Then Baez J. P122
Thesen T. P176
Thomas M. P156
Thomas R. P109
Thomas S. O1
Tillett W. P137
Tobalina L. P170, P171
Tolan D. P96
Torgutalp M. P112
Tornero C. P128
Torrat Noves A.M. P23
Tosun Ö. P76, P93
Totodson P. O4
Tournadre A. O6
Toussirot E. P56
Tremoceiro J. P114
Trenor Larraz P. P14
Tsafis K. P152
Tsiami S. P10, P103
Tuite M. O7, P81, P82
Turesson C. P91, P92
Tuytschaever S. P168

U

Uçar I. P160
Ulgu M.M. P161
Urbanik J. P125
Urruticoechea Arana A. P142
Usta B. P162

V

Valdez F. P122
Valdez Donelli F. P24, P33, P34
Valero-Expósito M. O10
van Dam M. P20
van de Sande M. P20, P69, P70

Van de Sande M.G.H. P36, P78
Van den Bosch F. O3, O5, P20, P75, P79
Van den Reek J.M.P.A. P172
van der Heijde D. P20, P28, P57, P66, P67, P68, P135, P143, P149
Van der Heijden W.R.P. P78
van der Horst-Bruinsma I.E. P5, P15, P36, P123, P172
van der Kraan Y.M. P16, P17, P37, P174, P175
Van der Laken C.J. P78
van Essen M.D. P57, P58
van Gaalen F. P20, P28, P36, P64, P66, P67, P68, P69, P70, P72, P73, P74, P91, P92, P149
van Herwaarden N. P123, P134
van Leuven S.I. P15, P172
van Lunteren M. O9, P28, P64, P66, P67, P68, P69, P70, P72, P73, P74, P91, P92
Van Mechelen M. P39
van Tubergen A. P57, P58, P153, P154
Van Tubergen A.M. P36
Vandamme N. O3
Vandevelde C. P121
Varela-Rodríguez H. P110
Varkas G. O5, P20, P75
Vasilakis C. P168
Vauchy C. P56, P113
Vázquez M. O10
Veeravalli S.C.M. P47
Vega C. P24, P33, P34, P122
Vega-Salcedo A.V. P11
Vegt D.E. P5
Vellón-García B. P107
Veloza C. P148
Venken K. O1, O3
Venturoli D. O1
Verhoeven F. O4, P29, P108, P113
Vermijlen D. O1
Verschueren P. P39
Verstappen G. P37
Vieira R. P22, P51, P52, P53, P55, P150
Vieira R.M.R.A. P54
Vieira V. P22
Vieira-Sousa E. P158
Viennet C. O4
Vila D. P24, P33, P34, P122
Vilafanha C. P90
Villalba A. P128
Villalba J.M. P107
Villanueva Mañes B. P23
Villaverde V. P142
Vis M. P134
Vitale A. P86
Vladimirova N. P89
Vlieghe C. O3
Vodanovic A. P151
Voglau M. P130
Vonk M.C. P15
Vonkeman H.E. P153
Vriezekolk J. P123

W

Wade L. P18
Walsh J.A. P137
Wang J. P19
Wang R. P81, P82
Wang S.E. P7
Wang T. P144
Wang X. P38
Wang Y. P19
Ward M.M. P38
Weber U. O7, P42
Webers C. O5, P20, P68, P153
Webers C.J.P. P57
Weddell J. P96, P121
Weel A.E.A.M. P36
Wei X.E. P71
Weisman M.H. P38
Wellen J. P144
Wendiggensen M. O8
Wendling D. O4, O6, P29, P108, P113
Wetterslev M. P89
White D. P62
Wichuk S. O7, P43, P42, P81, P82, P89
Willesen S.T. P94, P95
Wink F P17, P37, P88
Wink F.R. P16
Wivestad G.A. P176
Wordsworth B.P. P60
Worth C. P99
Wu J.Y. P167
Wu Q. INV2

X

Xie Y. P167

Y

Yalo L. P165
Yang J. P133
Yang J.W. P80
Yang Y.W. P147
Yau E. P61
Yazbek M. P22
Yazbek M.A. P54
Yazisiz V. P160
Yeung J. P42
Yildirim Ö.E. P160
Yoğurtçu Ö. P93
Yoon J.Y. P4
Yu S.M. P101

Z

Závada J. P89
Zemrani S. P59, P97, P159
Zeng R.S. P167
Zhang Y. P117
Zhao J. P99
Zhou B. P135, P143, P144
Zhuo D. P19
Ziadé N. P59, P97
Ziswiler H.R. P85
Zouzina O. P42
Zu L. P19
Zufferey P. P85
Zwezerijnen B.J.C. P78