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Abstracts

Invited Lectures

INV1

MICROBIAL INFLUENCE ON SPONDYLOARTHRITIS (SPA) PATHOPHYSIOLOGY

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The gut microbiome plays a key role in shaping host immunity through its fundamental contribution to the induction and training of the host's innate and adaptive immune responses. Perturbations in gut microbiome have been implicated in various metabolic, autoimmune, and inflammatory diseases including spondyloarthritis (SpA). Studies on experimental models and clinical samples from SpA patients have revealed microbial dysbiosis associated with dysregulated immune response. To explain the impact of HLA-B27 and SpA on the gut microbes, we proposed an ecological model of dysbiosis, where the effect of a group of microbes and their metabolic function underlie disease pathogenesis. SpA patients also demonstrate an altered mucosal regulation of gut microbes, as shown by changes in the bacterial taxa and the relative abundance of IgA coated and uncoated gut microbes in comparison with healthy individuals. Studies have shown gut microbial dysbiosis and altered host immune response not only in SpA patients, but also in healthy individuals carrying HLA-B27, a strong genetic risk factor in SpA pathogenesis, thus highlighting the role of host genetics as well as its effect on gut microbiome.
Furthermore, alterations in the microbial metabolic function are also associated with SpA. Metabolites are small molecules produced by both the host and microbes, which can provide a snapshot of host-microbe crosstalk at the biochemical level. SpA is associated with an increase in inflammatory metabolites belonging to the tryptophan pathways and bacterial lipopolysaccharide (LPS), with a concomitant decrease in anti-inflammatory metabolites such as short-chain fatty acids (SCFAs). SCFAs are associated with the maintenance of the gut epithelial barrier, and their decrease in SpA may allow the translocation of gut microbes/microbial products or metabolites from mucosal surfaces to peripheral tissues, thus eliciting aberrant immune activation. Microbial and metabolic profiles are also being evaluated for their potential as biomarkers for clinical diagnosis and treatment response in SpA. Therefore, gut microbial dysbiosis and altered metabolic profile offer insights into the disease pathogenesis of SpA and have the potential to be used as disease biomarkers and therapeutic targets in SpA.

INV2

GUT TISSUE AS KEY PATHOPHYSIOLOGY MECHANISM IN SPA

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The pathogenesis of Axial Spondyloarthritis (Axial SpA), a chronic inflammatory disorder primarily affecting the axial skeleton, involves a complex interplay between genetic predisposition, immune dysregulation, and environmental factors. Recent research has highlighted the pivotal role of the intestinal epithelium and lamina propria cells, as well as gut dysbiosis, in the pathogenesis of Axial SpA.
The intestinal epithelium serves as a crucial barrier that separates the gut lumen from the lamina propria and systemic circulation. Disruption of the intestinal epithelial barrier integrity can lead to increased permeability, allowing translocation of microbial antigens and pro-inflammatory molecules into circulation, thereby triggering systemic immune responses and inflammation in Axial SpA.
Lamina propria cells, including immune cells such as dendritic cells, macrophages, and lymphocytes, play a significant role in immune surveillance and regulation within the gut mucosa. Dysregulation of lamina propria immune responses can contribute to aberrant immune activation and inflammation, promoting disease pathogenesis in Axial SpA.
Furthermore, gut dysbiosis, characterized by alterations in the composition and function of the gut microbiota, has been associated with Axial SpA. Dysbiotic microbiota can influence immune homeostasis, modulate inflammatory responses, and contribute to the perpetuation of chronic inflammation in Axial SpA through the production of microbial antigens and metabolites.
Understanding the relative contributions of the intestinal epithelium, lamina propria cells, and gut dysbiosis in the pathogenesis of Axial SpA is essential for delineating the mechanisms underlying disease development and progression. Targeted interventions aimed at restoring intestinal barrier function, modulating immune responses in the lamina propria, and reshaping the gut microbiota may offer novel therapeutic strategies for managing Axial SpA by addressing both local and systemic factors driving inflammation and disease activity.

INV3

WHAT IS A SEVERE AXIAL SPONDYLOARTHRITIS?

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Introduction. Axial spondyloarthritis (axSpA) exhibits a highly variable natural course, with some patients developing severe progressive disease while others maintain limited disease over time. Disease severity in axSpA can be broadly defined as a measure of the extent and impact of a disease on individual's health and quality of life. It encompasses various dimensions, including clinical manifestations, radiographic structural damage, reduced physical function, social impairment, or reduced life-expectancy.
Results. A limited number of studies previously attempted to link the initial clinical presentation of axSpA outcome using various definitions of poor outcome including physician grading scale, disease activity, physical function or radiographic damage. Factors associated with poor disease outcome include limited spine motion, peripheral manifestations, extra articular manifestations, and elevated acute phase reactants. We and others showed that peripheral symptoms rather than axial ones are predictive of a poor health-related quality of life and a sustained high disease activity. Indeed, we identified in several cohorts of axSpA two stable clinical endotypes, one purely axial, and one combining axial and peripheral symptoms. The latter one is associated with poorer disease outcome and requires more intensive drug therapy. This accumulation of evidence challenges the role of axial imaging changes as the most relevant for disease severity.
Conclusion. There is currently no consensus on how to define severity in SpA and even less on how to predict it. However, early identification of patients at risk of poor outcome is crucial to develop a more tailored treatment approach for axSpA potentially reducing unnecessary healthcare costs and drug use.

INV4

METABOLIC SIGNATURE IN PSA AND SPA

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Differential diagnosis between early stages of PsA and seronegative rheumatoid arthritis remains a major challenge to clinicians and an increased suffering to patients, since delay in establishing the final diagnosis implies a delay in using the more targeted therapy. Another clinical challenge is posed by the simultaneous presence of a malignant disease in patients diagnosed with PsA or SpA, since therapeutic options must balance the control of the autoinflammatory process without compromising the anti-tumoral immune response. While hitherto analyses of immune mediators or immune phenotype signatures have driven the development of successful therapies for SpA and PsA *(e.g.* IL17A blockade), they have not been able to help answering the above-mentioned challenges. However, looking into metabolic signatures at both cellular and systemic levels unlocks new perspectives into potential, previously ignored, disease mechanisms for SpA and PsA.
In systemic chronic autoimmune diseases, like SpA and PsA, the inflammatory processes lead to major alterations in tissue and organ homeostasis that involve significant alterations in metabolism. These changes can be broadly categorized into changes in energy production and expenditure, alterations in anabolic processes (*e.g.* more lipid synthesis and accumulation in the synovial membrane) and altered catabolism (*e.g.* increased protein breakdown). By analysing and quantifying the changes in the serum metabolome, one can identify the alterations occurring in systemic metabolism, and use them to diagnose patients and early stages of disease, stratify patients ac-

cording to disease activity or therapy response, and use such information to build prognostic algorithms.
The changes in metabolism in chronic autoimmune disease also take place at a cellular level, specially in the immune system. In order to maintain a chronic pro-inflammatory effector profile, immune cells need to remodel their metabolism to uphold the permanent requirement for creating energy and biomolecules essential for proliferation, migration, membrane receptor expression or production of immune mediators (*e.g.* antibodies or cytokines). Thus, revealing which are the metabolic changes undergone by immune cells in SpA and PsA opens the possibility to identify new potential therapeutic targets.
In this lecture we will discuss how nuclear magnetic resonance metabolomic analyses of serum samples from SpA and PsA patients has provided new diagnostic models based on changes in serum metabolites. Such models may help clinicians identify patients with a simultaneous malignant disease or quickly distinguish between PsA and seronegative RA patients. Additionally, we will discuss how these serum metabolome profiles vary between disease activity stages, sexes, and patients with good or bad therapeutic responses. Finally, we will also present data on how the cross-talk between metabolic and immune pathways controls the (pro-inflammatory) functions of $CD8^+$ T lymphocytes in PsA and SpA patients.

INV6

REMISSION-INDUCTION IN IBD: WHERE ARE WE NOW

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Inflammatory Bowel Diseases, with Crohn's Disease (CD) and Ulcerative Colitis (UC) as the two main phenotypes, have seen a therapeutic revolution in the last two decades with the introduction of biological therapies, and more recently small molecule advanced treatments. Although the increase in therapeutic options enabled more ambitious treatment goals such as mucosal healing and even histologic healing, the observed remission rates with these advanced therapies remain at a modest 30% at most, and indicate a large therapeutic gap to close.
This therapeutic ceiling requires critical appraisal. The outcomes required for regulatory approval include a combination of patient reported symptoms as well as endoscopic improvement. The endoscopic scores were developed to assess active inflammation and not necessarily healing. It is unclear at this moment how much (or little) inflammation is acceptable without compromising the long-term outcome of a patient. It is also unclear if histologic improvement is superior to endoscopic remission and strategic trials in UC are underway to help answer this question.
Other means to improve remission rates in IBD include applying therapeutic strategies with a complementary mode of action, to the existing drugs which all target inflammatory cells and their produced cytokines. Also combination of advanced therapies has shown in pilot studies in both CD and UC to be able to double the remission rates. Finally, the best way to break the therapeutic ceiling is to move away from the "one size fits all" principle and to better stratify patients according to the underlying mechanisms that drive disease and that will not be the same in all patients. First attempts to stratify patients using a CD8 Tcell exhaustion signature and treat accordingly with a "top down" treatment strategy involving infliximab and azathioprine straight after diagnosis, unfortunately failed, although the PROFILE study showed that outcomes could be dramatically improved when treating patients adequately but very early after diagnosis.

INV8

THE GHENT SPA ORATION: 50 YEARS OF HLA-B27

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Introduction. The strong association of the human leucocyte antigen HLA-B27 with development of Ankylosing Spondylitis (AS) was first described just over 50 years ago, and undoubtedly plays a key role in disease development.
Methods. Advances in genetic, immunological, biochemical and biophysical techniques over the past 50 years have greatly enhanced our knowledge of the function of HLA molecules including HLA-B27 in the immune system.
Results. The primary natural function of HLA-B27 is to present short antigenic peptides to cytotoxic T cells as part of our cell-mediated immune response. It does this very well in the context of infection with many important viruses including Influenza, HIV, Hepatitis C and SARS COV2. The arthritogenic peptide hypothesis posits that AS results from presentation by HLA-B27 of disease-triggering or -specific peptides to cytotoxic T cells at disease sites. Alternatively or in combination HLA-B27 may act in a "co-stimulatory" fashion to amplify the strength of immune responses (both good and bad). This property is likely linked with its propensity to misfold both inside cells and on the cell surface. Recently HLA-B27 carriage has been linked to alterations in the gut microbiome, and further potentially pathogenic mechanisms may yet be found.
Conclusion. The immunological function(s) of HLA-B27 are now far better understood than previously and almost certainly contribute directly to AS pathogenesis, although the exact mechanisms remain to be elucidated.
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INV11

THE ROLE OF MIF/HIF1A AXIS IN SPONDYLOARTHRITIS

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Introduction. The hallmarks of spondyloarthritis (SpA) are type 3 immunity-driven inflammation and new bone formation (NBF). Macrophage migration inhibitory factor (MIF) has been identified as a key driver of the pathogenesis of SpA by amplifying type 3 immunity[1]. However, MIF-interacting molecules and networks remain elusive.
Methods. Computational analyses were performed to identify MIF-interacting partners in SpA. The expression of hypoxia-inducible factor-1 alpha (HIF1A) in joint tissues and synovial fluid of SpA patients and curdlan-injected SKG (curdlan-SKG) mice was assessed by immunohistochemistry. The effects of hypoxic conditions on the expression of inflammatory cytokines in neutrophils were assessed by qPCR and western blotting. The therapeutic potential of the HIF1A-specific inhibitor (PX-478) in curdlan-SKG mice or IL-23-overexpressed SKG mice was tested. Statistical analyses were conducted where appropriate.
Results. We identified hypoxia-inducible factor-1 alpha (HIF1A) as an interacting partner molecule of MIF in driving SpA pathologies. The expression of HIF1A was increased in joint tissues and synovial fluid of SpA patients and curdlan-SKG mice compared to controls. Under hypoxic conditions, human and mouse neutrophils substantially increased the expression of MIF and IL-23, an upstream type 3 immunity cytokine. Systemic overexpression of IL-23 induced SpA pathologies in SKG mice, while the injection of PX-478 into curdlan-SKG mice prevented or attenuated SpA pathologies, with a marked reduction in MIF and IL-23 expression. Genetic deletion of MIF or HIF1A inhibition with PX-478 in IL-23-overexpressed SKG mice did not induce evident arthritis or NBF, despite the presence of psoriasis-like dermatitis and blepharitis.
Conclusion. These results provide supporting evidence for a MIF/HIF1A-regulatory network in SpA. Inhibition of HIF1A may be a novel therapeutic approach for SpA by suppressing type 3 immunity-mediated inflammation and NBF.
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References

1. NAKAMURA A, ZENG F, NAKAMURA S *et al.*: *Macrophage migration inhibitory factor drives pathology in a mouse model of spondyloarthritis and is associated with human disease. Sci Transl Med 2021; 13(616): eabg1210. doi: 10.1126/sci-translmed.abg1210*
2. NAKAMURA A, JO S, NAKAMURA S *et al.*: HIF-1α and MIF enhance neutrophil-driven type 3 immunity and chondrogenesis in a murine spondyloarthritis model. *Cell Mol Immunol* 2024: 21(7): 770-86. doi: 10.1038/s41423-024-01183-5

INV12

GUT γδ T CELLS AND EPITHELIAL BARRIER PROTECTION: LESSONS FROM INFLAMMATORY BOWEL DISEASE

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Epithelial dysregulation occurs early in the hierarchy of the development of gut inflammation, which is pertinent both to IBD and the inflammation seen in other conditions including spondyloarthropathy. Patrolling the epithelial barrier are innate-like γδ T cells which have the capacity for tissue repair and immunosurveillance. We have previously described the conserved butryophilin-like (BTNL)-γδ axis in health, whereby a subset of human intestinal γδ intraepithelial lymphocytes (IEL) are regulated by local, epithelial-restricted proteins BTNL 3/8. It has previously been described that the BTNL γδ axis is dysregulated in coeliac disease, however this axis had yet to be elucidated in inflammatory bowel disease and related inflammatory gut conditions.
Using exclusively human samples we have established that inflammatory bowel disease is associated with specific phenotypic and functional shifts in γδ T cells in the human intestine which are associated with disease progression and outcome. Furthermore, somatic mutations in the BTNL proteins which regulate γδ T cells in the intestine result in a deficiency of γδ T cells and disease progression in Crohn's disease: implicating these cells and this axis in inflammation resolution.
I will discuss our insights into γδ T cell biology in IBD and how this may relate to other inflammatory gut conditions, including those seen in spondyloarthropathy.

INV15

INTEGRATIVE STRUCTURAL BIOLOGY ON CYTOKINE-RECEPTOR ASSEMBLIES AS A DRIVER OF FUNDAMENTAL AND TRANSLATIONAL RESEARCH IN INFLAMMATORY AND AUTOIMMUNE DISEASES

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We employ integrative structural biology to study the structure, mechanism, and modulation of proteins and protein complexes pivotal to (auto)immunity and inflammation. Our recent work on three types of pro-inflammatory cytokine-receptor assemblies from distinct families with relevance in the clinical targeting of autoimmune diseases such as psoriasis, rheumatoid arthritis, and spondyloarthritis, have highlighted the need to pursue complete cytokine-receptor assemblies to harness the full potential of the mechanistic and structure-function relationships of such signaling complexes. My contribution will focus on leveraging a diversity of structural and mechanistic insights derived from structures of extracellular complexes of IL-12 and IL-23 (1), IL-36/IL-37, and IL-26 with cognate receptors, that could be used for further functional interrogation of pro-inflammatory signaling and therapeutic targeting. Furthermore, I will illustrate how recent developments in protein structure prediction and design towards protein-based therapeutics (2), including engineered antibodies, can be used synergistically with structural insights to enable innovative therapeutic targeting of pro-inflammatory cytokine-receptor signaling assemblies.

References

1. BLOCH Y, FELIX J, MERCERON M *et al.*: Structures of complete extracellular receptor assemblies mediated by IL-12 and IL-23. *Nat Struct Mol Biol* 2024; 31(4): 591-97. doi: 10.1038/s41594-023-01190-6.
2. CAO L, COVENTRY B, GORESHNIK I *et al.*: Design of protein binding proteins from target structure alone. *Nature* 2022; 605: 551-60. doi: 10.1038/s41586-022-04654-9.

INV18

WHERE ARE THE USPA PATIENTS?

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Initially, the concept of spondarthritides (SpA) was proposed to lump together ankylosing spondylitis, psoriatic arthritis, arthritis associated with inflammatory bowel disease and reactive arthritis, based on the resemblance, the frequent overlap and the tendency to familial aggregation of those entities. The discovery of an association of all the group with HLA-B27 reinforced its significance. The undiffferentiated spondylarthropathy (USpA) terminology was then used to designate patients presenting with clinical manifestations typical of SpA but lacking the most convincing features of the spectrum, except for HLA-B27. The SpA classification criteria were developed in the 1990s to take in account such evolution. The more recently developed ASAS criteria have split the SpA patients into two major groups, depending on their main clinical and imaging features: axial and peripheral SpA, which represents roughly 80% and 20% of the patients, respectively. At this stage, if one believes that all SpA patients have been included in a well-defined category, the end of USpA terminology could be predicted. This is the question that will be discussed in this presentation.

INV19

CLINICAL EVALUATION IN SPONDYLOARTHRITIS: CONTEMPLATING ITS ROLE IN THE FUTURE

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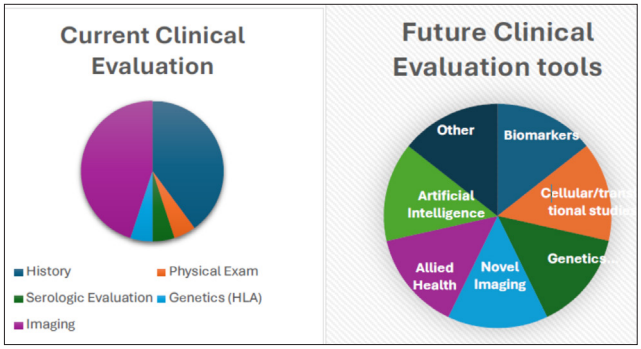
Clinical evaluation is the primary way we assess a patient for the diagnosis (of spondyloarthritis), and in the setting of a definitive diagnosis for disease burden and activity. We use this assessment to determine what therapies a patient warrants, what complications and comorbidities they might develop and ultimately how to help a patient live their best life with the chronic rheumatologic disease they own.
The current standard clinical evaluation includes the history we obtain, our physical examination, laboratory and imaging data. Genetic testing for diagnosis, besides HLA typing is not widely available yet. Genetic testing for pharmacogenomics testing is becoming more available and has a role in the more immediate future. Currently inaccessible data in daily practice are biomarkers in the research setting and tissue that might be useful in clinical assessment.



INV 19: Fig. 1.

The clinical evaluation process is time consuming, limiting the number of patients we can accurately diagnose in a timely manner. Allied health professionals and well-trained primary care providers can help us decrease time to diagnosis by providing some of the assessment for the rheumatologist.
Additionally, Artificial Intelligence will undoubtably play a role in the future of clinical assessment. This session will describe the current clinical evaluation hurdles and where it may fit in the future in the assessment of spondyloarthritis.

INV21

AI IN AXSPA IMAGING

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Introduction. Axial spondyloarthritis (axSpA) is a chronic inflammatory disease primarily affecting the axial skeleton, characterized by inflammation of the sacroiliac joints and spine. Early diagnosis is crucial for optimal treatment outcomes, but the average delay between symptom onset and diagnosis ranges from 5 to 10 years. This delay is partly due to the challenges in differentiating axSpA-related back pain from other common causes. Conventional imaging techniques, such as radiography, have limited reliability in detecting early stages of the disease. In recent years, artificial intelligence (AI) has emerged as a promising tool for assessing changes in axSpA imaging. This talk aims to provide a comprehensive overview of recent advances in AI applications for axSpA imaging across various modalities, discuss current challenges, and explore future perspectives in this rapidly evolving field.
Methods. We conducted a review of recent literature on AI applications in axSpA imaging, focusing on three main imaging modalities: radiography, computed tomography (CT) and magnetic resonance imaging (MRI). Studies were included that reported performance of AI models in terms of their diagnostic accuracy, and ability to detect disease manifestations compared to human experts.
Results. AI algorithms have demonstrated promising results in detecting and quantifying structural and inflammatory changes in sacroiliac joints across various imaging modalities. In radiography, deep learning models have achieved expert-level performance in detecting radiographic sacroiliitis. A convolutional neural network trained on 1,553 radiographs achieved areas under the receiver operating characteristic curve (AUCs) of 0.97 and 0.94 for validation and test datasets, respectively. This model exhibited a sensitivity of 88% and a specificity of 95% in the validation set and maintaining comparable performance (sensitivity 92%, specificity 81%) in an independent test set. A subsequent study was able to further improve this method by adding an anatomical aware approach. Here a segmentation model first detected the sacroiliac joints, making the pipeline more robust and more specific for the sacroiliac joints, leading to fewer false positive model activations. These findings suggest that AI can potentially assist in the reliable detection of radiographic sacroiliitis, even in non-specialized clinical settings.
CT imaging studies have shown that radiomics-based approaches can effectively classify sacroiliitis. A multidimensional algorithm achieved accuracies of 82.02%, 79.32%, and 77.91% for two-, three-, and five-category classification tasks, respectively. Further research has demonstrated the potential of AI in detecting sacroiliitis as incidental findings in CT scans, with classification accuracies of 91.9% and 86% for binary and three-class cases.
In MRI analysis, AI models have shown high accuracy in detecting bone marrow edema and structural changes indicative of axSpA. Deep learning models for MRI assessment achieved AUCs of 0.94 for detecting inflammatory changes and 0.89 for structural changes. These models demonstrated sensitivities of 88% and 85%, and specificities of 71% and 78% for inflammatory and structural changes, respectively, in the test set. Notably, for detecting inflammatory changes, these results were comparable to the performance of trained expert readers. Another group focused on the detection of bone marrow edema in achieving a accuracy of 93.6% using fat suppressed MRI sequences.
Other recent advancements in AI applications for axSpA imaging include synthetic CT (sCT) technology, a deep learning-based method that generates CT-like images from T1-weighted MRI sequences. Research has shown that sCT improves the detection of structural lesions compared to conventional MRI, with higher diagnostic accuracy for erosions, sclerosis, and ankylosis. Emerging research also focuses on multimodal AI approaches, integrating data from various imaging modalities, clinical features, and biomarkers to enhance diagnostic accuracy and develop personalized treatment strategies. Additionally, AI algorithms are being developed to track changes in imaging findings over time, potentially improving the monitoring of disease progression and treatment response.
Conclusion. AI applications in axSpA imaging show promise in improving early diagnosis and monitoring of the disease. However, challenges remain, including the need for large, diverse datasets for training and validation, and the integration of AI tools into clinical workflows. Future research should focus on developing multimodal AI approaches that combine imaging data with clinical and laboratory findings to enhance diagnostic accuracy and personalized treatment strategies.

INV22

REDUCING IMPRECISION IN PRECISION MEDICINE

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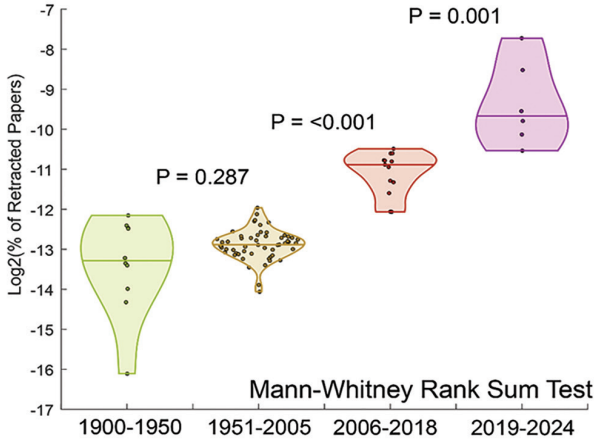
Introduction. Integrative computational biology and artificial intelligence (AI) can help improving treatment of complex diseases by creating explainable models. Systematic and comprehensive data curation provides high quality data for the analysis and modeling, which in turn leads to better biomarkers, more accurate understanding of drug mechanism of action, and in turn improved patient outcomes. Data mining, machine learning, graph theory and advanced visualization help us to characterize interactome and drug "orphans" with accurate predictions, making disease modeling more comprehensive and interpretable. Intertwining computational prediction and modeling with biological experiments and preclinical studies leads to more useful findings faster and more economically. Such integrated workflow leads to a true patient-centric precision medicine.
Methods. Many datasets have been generated to improve our understanding of the spectrum of spondyloarthritis (SpA). Using the integrative computational biology workflow enables us to combine and compare data-driven and literature-backed knowledge on SpA. Translational research has been expanding from finding diagnostic, prognostic and predictive biomarkers to uncovering and modeling drug mechanism of action and defining explainable models for personalized care. We can readily integrate insights from diverse omics platforms analysed by statistical or machine learning-based algorithms when linked by relationships from diverse biological networks.



INV 22: Fig. 1. Number of publications per year and number of retractions in that year identify four distinguishable "eras". Number of published and retracted papers was quite stable until about 1950. Naturally, increasing numbers of papers published annually brought a steady increase in retractions. However, the drastic change in number of retractions compared to number of papers published started in around 2000, with further significant increase after 2019. Data was obtained from PubMed, finding all publications in a given year, and then using query: "PUBLICATION TYPE: retraction of publication", on March 17, 2024 (by means of EndNote 21.2 Bld. 17387 for query processing).

A)

Data component	Literature curated (core) pathway memberships	Extended pathway associations. Protein interaction set: Experimentally detected PPIs Minimum confidence level for predicted associations: 0.99	Extended pathway associations. Protein interaction set: Experimentally detected and computationally predicted PPIs (full IID) Minimum confidence level for predicted associations: 0.99
All sources			

B)

Data component	Literature curated (core) pathway memberships	Extended pathway associations. Protein interaction set: Experimentally detected PPIs Minimum confidence level for predicted associations: 0.99	Extended pathway associations. Protein interaction set: Experimentally detected and computationally predicted PPIs (full IID) Minimum confidence level for predicted associations: 0.99
ACSN2			
BioCarta			
HumanCyc			
KEGG			
Panther_Pathway			
PathBank			
PharmGKB			
REACTOME			
MetabolicAtlas			
SIGNOR 3.0			
UniProt_Pathways			
WikiPathways			

INV 22: Fig. 2. Pathway enrichment analysis using pathDIP highlights the variable coverage of individual core pathway databases. Red color in the pie charts shows the proportion of the query that is not annotated in a given pathway database (proportion of pathway orphans), while the blue color corresponds to mapped target names.
A: Results for integrated pathway enrichment analysis. The first column corresponds to using only literature curated pathways (i.e., core pathways), the second column represents overlap when extended pathways that include experimentally detected protein interaction data from IID, and the last column shows coverage when extended pathways include both experimental and predicted physical protein interactions from IID.
B: The enrichment analysis across 12 core pathway databases, clearly highlighting that selecting any single database for pathway enrichment analysis will result in some statistically significant results but with potential bias, since often only a tiny fraction of the input query has been mapped and used. Detailed views (available to view and download) list specific genes or proteins that were or were not mapped to pathways.

These networks include physical protein interactions, non-coding RNAs-targets and transcription factor-targets networks. Adding ontologies, pathway and drug information, the networks are then used to create explainable disease models. Trustable data comes from curating PubMed and various data repositories such as Gene Expression Omnibus (GEO, https://www.ncbi.nlm.nih.gov/geo), while the networks include pathways (*e.g.* pathDIP, https://ophid.utoronto.ca/pathDIP; Reactome, https://reactome.org), physical protein interactions (*e.g.* IID, https://ophid.utoronto.ca/iid; IntAct, https://www.ebi.ac.uk/intact), microRNA-gene mapping (*e.g.* mirDIP, https://ophid.utoronto.ca/mirDIP); Gene Ontology (https://geneontology.org), and high-quality annotations and ontologies (*e.g.* Uniprot, https://www.uniprot.org; DisGeNet, https://www.disgenet.com; Human Protein Atlas, https://www.proteinatlas.org; CTD, https://ctdbase.org; DrugBank, https://go.drugbank.com).

Results. While statistical and machine learning analysis benefits from larger and multiple datasets, in highly multidimensional domain analysis, separating signal from noise is challenging. Data quality is paramount, yet sometimes underestimated. Major efforts in computational biology and earlier research in AI have focused on data and knowledge management, ontologies, and curated data resources. We now have vast amounts of data, and many advanced algorithms are able handle noise. However, we are increasingly dealing with fraud or suspected fraud in publications and corresponding datasets, which are affecting AI and statistical models. Alarmingly, over 10,000 papers were retracted just in 2023. The substantial increase in retractions is significant, as highlighted in Figure 1. It is not only this "noise" from retracted papers that is posing challenges in translational research. Different tools and pipelines used even on the same datasets produce different results, often leading to perceived or true non-reproducible science. For example, pathway enrichment analysis is often used for prioritizing validation and functional studies. However, many existing tools "hide" the information about what fraction of the input list has been used in the analysis. In contrast, pathDIP database puts that information up-front to ensure transparency and provide the user with trustworthy and detailed annotation to guide experiments (Fig. 2).

Conclusion. AI with integrative computational biology enables us to creIate explainable and trustworthy models, and offers to uncover signal from noise. Translational research requires increased transparency of workflows and provenance. We need to track how the models are trained, validated, and what data were used. While diagnostic or prognostic tools could be useful even as a "black box", providing explanation and biological context using computational biology resources provides increased insight and in turn trust.

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INV23

CHRONIC JOINT PAIN: LESSONS FROM OSTEOARTHRITIS

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Chronic pain impacts up to 30% of the adult population globally, and pain is the primary reason why people seek medical care. The musculoskeletal system is the major source of chronic pain in the world, with low back pain and osteoarthritis as the leading contributors. Effective pharmacological treatments are generally unavailable, and there is an urgent need for deeper understanding of mechanisms underlying pain associated with rheumatic and musculoskeletal diseases (RMDs) in order to enable the discovery of new therapeutic targets for drug development.

We have used a variety of techniques that bring together the disciplines of neuroscience and arthritis research to reveal important facts about the molecular characteristics of joint innervating nociceptors and their central connections. Such techniques include the development of sophisticated animal models of RMDs with clear translational applicability, the development of clinically relevant behavioral tests for musculoskeletal pain, single cell transcriptomics (and related techniques) designed to elucidate the molecular characteristics of populations of joint nociceptors, genetic techniques that allow the identification of populations of neurons through the expression of fluorescent markers allowing their anatomical characterization assisted by clearing methods, and *in vivo* Ca imaging and related methods for assessing the physiological functioning of relevant pain pathways in RMDs. New insights into mechanisms underlying joint pain associated with osteoarthritis (OA) will be presented, and it will be briefly discussed how these new insights may be applied to other rheumatic diseases, including spondyloarthritis.

The following major points will be discussed:

- The overall anatomy and function of the pain pathway, with focus on the peripheral nervous system and somatosensory neurons/nociceptors (including subsets of nociceptors with specific functions, such as mechanosensitive nociceptors).
- Sensory neurons respond to noxious stimuli in the joint – and when sensitized they will also respond to innocuous stimuli.
- Neuroplasticity of joint innervation: Nociceptors innervate specific intra-articular structures, but they are not hardwired. Rather, there is anatomical remodeling of nociceptors with age and disease, and sprouting of nociceptor nerve endings.
- Neurons interact with other cells in the innervated tissues, including immune cells and fibroblasts. The neuronal cell bodies reside in the dorsal root ganglia, where there is also interaction with non-neuronal cells. All these interactions can shape the pain response in a disease-specific manner.
- Sensory (*i.e.* afferent) neurons can have efferent functions and they can affect joint integrity.

INV25

CURRENT CONCEPTS IN MANAGING WIDESPREAD PAIN

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Chronic pain remains a priority for patients with spondyloarthritides (SpA) patients, even those who appear to have attained inflammatory remission. Widespread pain prevails and is increasingly recognised to be subserved by central nervous system-based pain mechanisms, specifically nociplastic pain. The identification and contribution of various pain mechanisms is a vital first step in assessing an individual patient's pain and subsequently informing their management.

The management of nociplastic pain is classically distinct from nociceptive pain. In SpA, the latter has been well researched and is dominated by the targeting of aberrant peripheral immune pathways. In contrast, no SpA specific nociplastic pain therapies have been robustly tested and so current advice is greatly informed by learnings from other clinical populations, especially fibromyalgia: the prototypical nocplastic pain disorder.

This lecture will provide an overview of how to optimally managed SpA related nociplastic pain based on existing knowledge.

Oral Presentations

O1

ENHANCED TYPE 1 INTERFERON SIGNATURE IN AXIAL SPONDYLOARTHRITIS PATIENTS UNRESPONSIVE TO SECUKINUMAB TREATMENT

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Introduction. Axial spondyloarthritis (axSpA) is an inflammatory disease in which overactive IL-17A-producing cells are implicated in a central role. Therapeutically, biologics that target IL-17A, such as secukinumab, have demonstrated improved clinical outcomes. Despite this translational success, there is a gap in understanding why some axSpA patients do not respond to IL-17 blocking therapy. Our study aims to discriminate immune profiles between secukinumab responders (SEC-R) and nonresponders (SEC-NR).
Methods. Peripheral blood mononuclear cells were collected from 30 axSpA patients before and 24-weeks after secukinumab treatment. Frequency of CD4+ T cell subsets were compared between SEC-R and SEC-NR patients using flow cytometry. Mature CD45RO+CD45RA-CD4+ T cells were FACS sorted, and RNA was measured using NanoString analysis.
Results. SEC-NR patients had an increased frequency of IL-17A-producing RORγt+CD4+ T cells compared to HCs at pre-secukinumab (11.74% vs. 4.66%, $p<0.01$). SEC-NR patients had a significant increase of CXCR3+ CD4+ T cells pre-secukinumab compared to SEC-R patients (23.35% vs. 14.78%, $p<0.01$). Regression analysis revealed CXCR3+ CD4+ T cell frequency was significantly associated with secukinumab response (OR 0.52, 95%CI = 0.24-0.82, $p=0.028$). Differentially expressed gene analysis revealed upregulation of type 1 interferon-regulated genes in SEC-NR patients compared to SEC-R post-biologic. SEC-R patients had an upregulated cytotoxic CD4+ T cell gene signature pre-secukinumab compared to SEC-NR patients.
Conclusion. The increased frequency of IL-17A-producing cells in SEC-NR patients suggests a larger inflammatory burden than SEC-R patients. With treatment, SEC-NR patients have a more pronounced type 1 IFN signature than SEC-R patients, suggesting a mechanism contributing to this larger inflammatory burden. The results point toward more immune heterogeneity in axSpA than has been recognized and highlights the need for precision therapeutics in this disease.

O2

ANXIETY DISORDERS IN SPONDYLOARTHRITIS PATIENTS ON BIOLOGIC THERAPY REGISTERED IN REUMA.PT: PREVALENCE, ROLE OF DISEASE-RELATED FACTORS AND INFLUENCE OF BIOLOGIC THERAPY

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Introduction. Anxiety disorder prevalence is higher in chronic conditions like axial and peripheral Spondyloarthritis (SpA), possibly linked to inflammation. We aimed to determine anxiety disorder prevalence in SpA patients at first biologic prescription (bDMARD) and to assess its impact on anxiety disorder.
Methods. We conducted a multicenter, retrospective, observational study including adult patients registered in Reuma.pt with SpA treated with their first bDMARD. Those who completed the Hospital Anxiety and Depression Scale (HADS) at baseline (T0), after 3 (T1) and/or 6 months (T2) of treatment were analyzed. Socio-demographic, disease and treatment-related data were collected. Anxiety disorder was considered when subscale anxiety (HADS-A) ≥11. Pearson and Spearman correlations, ANOVA and T-tests were used.
Results. 141 patients were analyzed. Table 1 summarizes population characteristics. Factors correlated with anxiety disorder are summarized in Table II. Patients with anxiety disorder had significantly higher BASDAI ($p<0.001$), ASDAS-CRP ($p=0.004$) and BASMI at T0 ($p=0.007$) than those without anxiety disorder.
Mean scores of HADS-A significantly differed between the three time points ($F(1.862, 158.264) = 15.321$, $p<0.001$). Post hoc analysis with Bonferroni adjustment revealed that HADS-A significantly decreased from T0 to T1 (1.709 (95% CI, 0.750 to 2.669), $p<0.001$), from T0 to T2 (1.733 (95% CI, 0.828 to 2.637), $p<0.001$), but not from T1 to T2 (0.023 (95% CI, -0.730 to 0.777), $p=1$).
No significant differences were found in HADS-A at the three time points between patients with axial disease and those with peripheral disease ($F(2.000, 52.000) = 3.020$, $p=0.057$) and between patients treated with anti-TNF and those with an anti-IL-17 ($F(1.862, 156.391) = 0.768$, $p=0.457$).
Conclusion. Anxiety prevalence in SpA aligns with chronic disease estimated rates (between 18-35.1%). Anxiety disorder improvement with bDMARD therapy suggests a relationship with disease activity and physical function. Inflammation hypothesis in anxiety should be considered and further investigated.

O2: Table I. Population characteristics.

Gender	55.3% female; 44.7% male;	
Mean age at diagnosis	37.4 ± 10.5 years,	
Prescribed bDMARD	Adalimumab 59.6% Etaenercept 23.4% Golizumab 7.1% Certolizumab 5.7% Sekucinumab 2.8% Inflimab 1.4%	
Anxiety symptoms at TO	34.8% (N=48) of patients	
Anxiety symptoms at TO by gender	Female =43.6%	*p*-value = **0.014**

Table II. Correlations between disease activity scores or patient reported outcomes with anxiety disorder at baseline.

	Pearson	Spearman	*p*-value
Disease activity scores			
ASDAS-CRP	0.245		**0.004**
BASMI		0.228	**0.016**
BASDAI		0.411	**<0.001**
Patient-reported outcomes			
PGA	0.255		**0.002**
BASFI	0.464		**<0.001**
PAP		0.270	**0.001**
FACIT-F		-0.507	**<0.001**
SF36 – PF		-0.348	**<0.001**
SF36 – RP		-0.254	**0.003**
SF36 – BP		-0.390	**<0.001**
SF36 – GH		-0.425	**<0.001**
SF36 – VT		-0.306	**<0.001**
SF36 – SF		-0.344	**<0.001**
SF36 - RE		-0.497	**<0.001**
SF36 - MH	-0.647		**<0.001**

ASDAS-PCR: Ankylosing Spondylitis Disease Activity Score - C-reactive protein; BASMI: Bath Ankylosing Spondylitis Metrology Index; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; PGA: Patient Global Assessment; BASFI: Bath Ankylosing Spondylitis Functional Index; PAP: Patient Assessment of Pain; FACIT-F: Functional Assessment of Chronic Illness Therapy – Fatigue Scale; SF36: 36-Item Short Form Survey; PF: physical functioning; RP: physical role; BP: bodily pain; GH: general health; VT: vitality; SF: social function; RE: emotional role; MH: mental health.

O3

COMPLEMENT ACTIVATION AND SPINAL RADIOGRAPHIC PROGRESSION IN PATIENTS WITH RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS TREATED WITH TNF-INHIBITION OVER 2 YEARS: OBSERVATIONS FROM A RANDOMIZED CONTROLLED TRIAL (CONSUL)

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Introduction. The biological processes involved in the structural changes associated with radiographic axial spondyloarthritis (r-axSpA) remain largely unexplained. However, animal models have demonstrated that inhibiting complement activation can mitigate structural changes associated with axSpA. This project aimed to investigate the associations between the complement system and radiographic spinal progression in a longitudinal cohort of r-axSpA patients from a randomized controlled trial (CONSUL).

Materials and methods. All patients had active r-axSpA and risk factors for radiographic spinal progression (BASDAI≥4, and elevated CRP and/or ≥1 syndesmophyte). Serum samples were collected at baseline (n=96) and after 108 weeks (n=89) of TNFi therapy and analyzed by immunoassays for complement proteins (L-ficolin, M-ficolin, H-ficolin, CL-L1, MBL, MASP-1, MASP-2, MASP-3, and MAp44) and complement activation marker C3dg. X-rays performed at baseline and after 108 weeks were assessed blinded for all clinical data and chronology by three independent expert readers. New bone formation was defined as the growth of syndesmophyte(s) and/or new syndesmophyte(s) determined by three-reader-agreement.

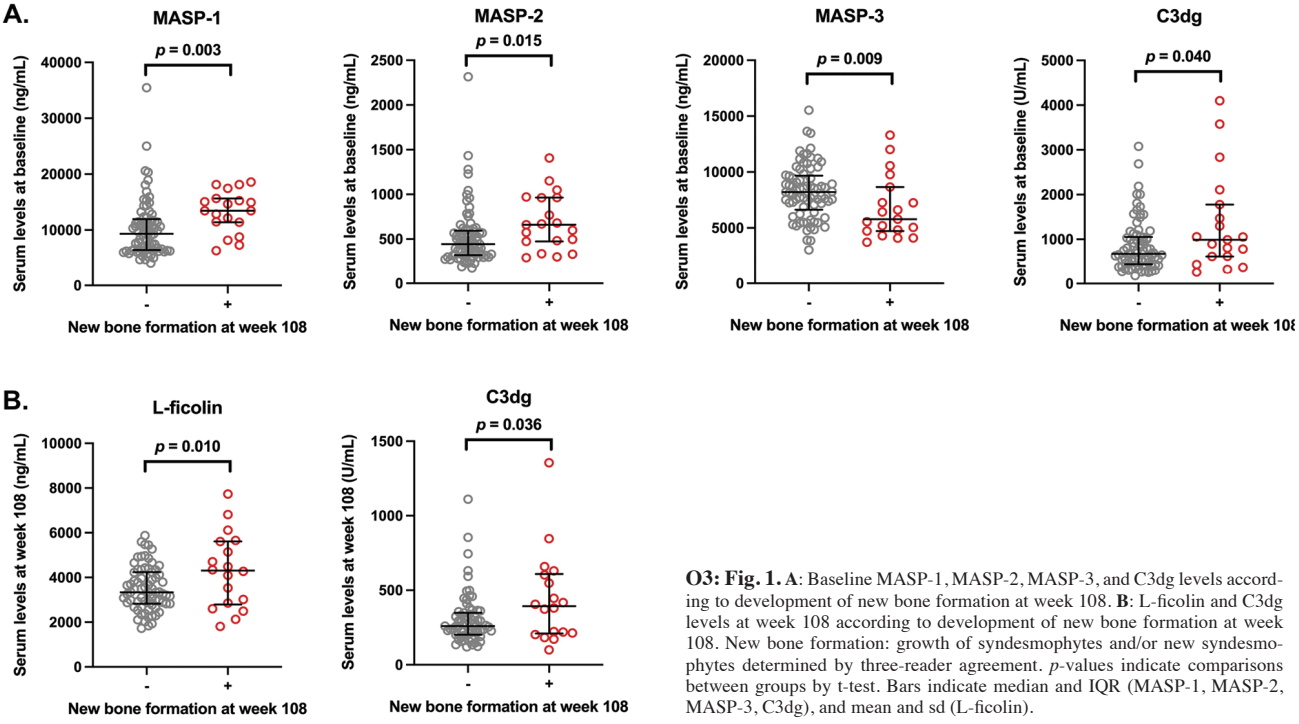
Results. Demographics are shown in Table I. Nineteen patients demonstrated new bone formation at week 108. Baseline levels of MASP-1, MASP-2, and C3dg were elevated in patients with new bone formation, whereas baseline MASP-3 levels were decreased (Fig. 1A). Furthermore, at follow-up L-ficolin and C3dg levels were elevated in patients with new bone formation (Fig. 1B). Assessed by univariate logistic regression, baseline MASP-1, MASP-3, and C3dg predicted the development of new bone formation, and remained significant in a corresponding multivariate analysis.

Conclusion. We here present the first investigations of complement activation and radiographic progression in a longitudinal cohort of axSpA patients. We observed that serum levels of MASP-1, MASP-3, and complement activation (C3dg) prior to TNFi therapy, predicted development of new bone formation at week 108. These findings support the involvement of the complement system in new bone formation in r-axSpA.

O3: Table I. Baseline demographics of the investigated patient population from the CONSUL RCT (n=96).

Age, median (IQR)	37 (31-45)
Male, n (%)	70 (73)
HLA-B27 positive, n (%)	80 (87)[§]
Smokers, n (%)	36 (38)[#]
BMI, median (IQR)	26 (22-30)
NSAID intake score, median (IQR)	100 (50-100)
Use of NSAID, n (%)	87 (91)
Previous bDMARD treatment, n (%)	21 (22)
Time since diagnosis in years, median (IQR)	3.0 (0.4-8.6)
Years since onset of symptoms, median (IQR)	12 (6.0-20)
Peripheral arthritis ever, n (%)	39 (41)[€]
Psoriasis ever, n (%)	18 (19)[#]
Uveitis ever, n (%)	28 (30)[€]
CRP, median (IQR)	9.2 (3.2-19)
Elevated CRP (>5 mg/L), n (%)	64 (67)
mSASSS, median (IQR)	5.0 (0.3-18)
Syndesmophytes, median (IQR)	1.8 (0-6.3)
Presence of ≥1 syndesmophyte(s)δ, n (%)	47 (49)
ASDAS-CRP, median (IQR)	3.6 (3.1-4.1)
BASDAI, median (IQR)	6.2 (5.2-6.8)
BASFI, median (IQR)	5.2 (4.2-6.3)

[§]Data available for n=92. [#] Data available for n=95. [€]Data available for n=94.
δ Determined by three calibrated readers blinded for clinical data.
bDMARD: biological disease-modifying anti-rheumatic drug. ASDAS-CRP: Ankylosing Spondylitis Disease Activity Score. BASDAI: Bath Ankylosing Spondylitis Disease Activity Index. BASFI: Bath Ankylosing Spondylitis Functional Index.



O3: Fig. 1. A: Baseline MASP-1, MASP-2, MASP-3, and C3dg levels according to development of new bone formation at week 108. **B**: L-ficolin and C3dg levels at week 108 according to development of new bone formation at week 108. New bone formation: growth of syndesmophytes and/or new syndesmophytes determined by three-reader agreement. p-values indicate comparisons between groups by t-test. Bars indicate median and IQR (MASP-1, MASP-2, MASP-3, C3dg), and mean and sd (L-ficolin).

O4

EARLY-LIFE COLONISATION BY INTESTINAL BACTERIA PROGRAMS THE IMMUNE SYSTEM TO PERMIT DEVELOPMENT OF SPA

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Introduction. Gut microbiome profiling in SpA has identified increased abundances of inflammation-associated bacteria, however the host-microbe interactions underpinning disease remain largely unknown. Germ-free studies on the SKG mouse model identify microbiota-dependent joint and intestinal inflammation following dectin-1 activation with curdlan. However, the mechanisms underlying this are unknown.
Objective. We firstly aimed to characterise the intestinal microbiome of SKG mice and to assess whether disease development was dependent on the colonisation of specific arthritogenic bacteria. We then aimed to determine whether disease was driven by continuous microbial exposure, or an immune priming mechanism from early transient microbial exposure.
Methods. We completed shotgun sequencing to characterise the intestinal microbiome in curdlan challenged SKG mice. To determine whether specific bacteria were required to support SKG disease, we identified bacterial candidates for monocolonisation in germ-free SKG mice prior to curdlan challenge. We then depleted a commensal from monocolonised mice using antibiotics to determine whether transient microbial exposure to the germ-free immune system was sufficient to permit disease development.
Results. SKG mice displayed compositional intestinal microbiome shifts following curdlan challenge. Although germ-free SKG mice did not develop disease, monocolonisation of various bacteria permitted the development of disease. Additionally, colonisation followed by depletion of a commensal prior to curdlan challenge permitted disease.
Conclusion. Collectively these data suggest a critical window whereby a bacterium programs the germ-free immune system to an arthritogenic state. Further characterisation of the mucosal immune landscape following microbial programming may identify cell types crucial to SpA development and provide important therapeutic avenues.

O5

RORγt+ γδ T CELLS ARE CRITICAL PLAYERS IN SPONDYLOARTHRITIS

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Spondyloarthritis (SpA) patients often present with microscopic signs of gut inflammation, a risk factor for progressive joint disease and Crohn's like gut pathology. In this study, we investigated whether intestinal γδ-T cells are involved in dysregulated IL-23/IL-17 responses in SpA by using gut and blood samples from treatment naïve SpA patients next to an IL-23 driven SpA mouse model (IL-23 EEV challenged B10.RIII mice). By means of high resolution flow cytometry, we observed an enrichment of γδ-T cells in the ileal intraepithelial fraction of SpA patients with microscopic gut inflammation. We could pinpoint this effect to an increase of TCRγδ-hi cells, a subset of predominantly δ1 cells showing enhanced IL-23 responses. Moreover, Type 17 skewed γδ T cell (γδ17) responses were detected in the inflamed ileum and colon samples, reflected by a high RORγt and low Tbet expression in parallel to increased IL-17 production. Overall, these changes were more pronounced in γδ T cells than conventional T cells. Interestingly, γδ-T alterations could already be distinguished in blood of SpA versus healthy donors, with circulating γδ-hi cell levels being linked to disease activity scores, suggesting a role in joint manifestations in SpA. RORγt inhibition could selectively block human γδ17 cell function in vitro. Moreover, in vivo RORγt-blockade in the IL-23 overexpression model significantly ameliorated SpA-like skin and joint pathology and countered inflammation-induced bone loss and erosions in hind paws. This coincided with a functional impact on IL-23 expanded γδ17 cells. In summary, our study points to a profound role for γδ17 cell immunity in combined gut-joint disease. These data are important for a better understanding of underlying immune mechanisms in SpA and optimization of treatment strategies.

O6

TISSUE RESIDENT MEMORY TH17 (TRM17) CELLS ARE THE PREDOMINANT SOURCE OF INTERLEUKIN (IL)-17 IN SPONDYLOARTHRITIS, PRODUCE IL-17 INDEPENDENT OF IL-23 AND ARE EPIGENETICALLY REGULATED BY BROMODOMAIN CONTAINING 1 (BRD1)

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Introduction/Objective. Interleukin (IL)-17 plays a key role in the immunopathogenesis of Spondyloarthritis (SpA), but its cellular source in joint tissue has not been determined previously. The induction of CD4+ tissue resident memory Th17 (TRM17) cells following the clearance of pathogens have been described in skin, lung and kidney. We aimed to identify IL-17's cellular source in SpA joint tissue.
Materials and methods. Synovial tissue from patients with SpA was profiled using single-cell RNA sequencing (scRNA-seq) (10x Genomics, 5 AxSpA and 6 PsA) or spatial RNA profiling (CosMX, 2 PsA). CellPhoneDB was used to infer cell–cell communication. Tissue resident memory Th17 (TRM17)-like cells were generated in vitro using blood memory CD4+ T cells from SpA pateints. An epigenetic inhibitor library, siRNA and clustered regularly interspaced short palindromic repeats (CRISPR) were used to identify epigenetic regulators for TRM17.
Results. scRNA-seq showed that IL-17A expression in SpA synovium was restricted to CD4+CXCR6+ cells with TRM17 transcriptome features. Cell-cell communication and single-cell spatial analysis support the interaction between CLEC10A+ dendritic cells and TRM17. Both sublining and lining fibroblasts in SpA synovium exhibited an enhanced IL-17A-induced transcriptional signature. TRM17-like cells generated *in vitro* phenocopied the TRM17 found in joint tissue and produced IL-17A in response to T cell receptor (TCR) stimulation but not IL-23. Perturbation of BRD1 inhibited the generation of TRM17-like cells and its production of IL-17A.
Conclusion. TRM17 cells are the predominant source of IL-17A in SpA and require TCR stimulation for their effector function rather than IL-23, providing an explanation for the relative lack of efficacy in IL-23 blockade in SpA joints. The epigenetic regulator BRD1 contributes to the generation of TRM17. Targeting TRM17 cells in SpA is a therapeutic strategy with potential to induce long-term remission.

O7

LY6G+CXCR2-CD101- GRANULOCYTES REGULATE INFLAMMATION IN AXIAL SPONDYLOARTHRITIS

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Introduction. TNF or IL-17 inhibitors are common treatment approaches in axial spondyloarthritis (axSpA) with limited long-term disease control and no cure. Clinical relapse is frequently observed, but the underlining mechanisms are poorly understood. Recently, the heterogeneity of myeloid cells during the course of axSpA has been identified. While the immunoregulatory capacity of myeloid cells is well known from cancer and autoimmune diseases, their emergence, mechanisms of action, and therapeutic potential in axSpA remain unknown. Here, we investigate the role of regulatory granulocytes in patients with radiographic-axSpA.
Methods. Blood was collected from axSpA-, PsA-, RA-patients and healthy-controls. All provided written informed consent in accordance with local requirements. Experimental-axSpA was induced in hTNFtg197 mice after injection of IL-23-minicircle-DNA. The abundance of granulocytes was measured by FACS. Human granulocytes were defined as CD45+, CD15int, CD66bhigh, whereas G-MDSC were defined as CD45+, Lin-, HLA-DR-, CD11b+, CD15+, CD33+. Biopsies from sacroiliac and facet joints of the lumbar spine were taken under MRI control and analysed by imaging-mass-cytometry.
Lin-, Ly6Ghigh, CXCR2-, CD101- and Lin-, Ly6Glow, CXCR2+, CD101+ cells were sorted and tested for suppressive T cell function.
Results. Human whole blood analysis revealed a disease-activity independent increase of granulocytes in circulation. But, the number of G-MDSC in axSpA-patients significantly correlated with disease-activity. Immune profiling of biopsies revealed a complex microenvironment of innate and adaptive immune cells embedded in a mesenchymal network of fibroblasts and endothelial cells. Herein, immunoregulatory granulocytes particularly colocalized with pro-resolving ILC2s, suggesting their immunoregulatory capacity. *In vivo* two main subtypes of granulocytes were identified from inflamed axial joints. Only immature granulocytes (Lin-, Ly6Ghigh, CXCR2-, CD101-) exhibited prominent T cell suppressor function *in vitro*, indicating a possible inflamed joint specific priming mechanism.
Conclusion. Our study established the existence of immunoregulatory Lin-, Ly6Ghigh, CXCR2, CD101- immature granulocytes in axSpA. Their ability to inhibit T cell proliferation *in vitro*, along with their colocalization with pro-resolving immune cells at inflamed joints, underscores their relevance in the pathophysiology of axSpA.

O8

PROTEOMIC, TRANSCRIPTOMIC, AND T-CELL RECEPTOR (TCR) PROFILING OF SYNOVIAL INTEGRIN-EXPRESSING (INEX) T CELLS IN AXIAL SPONDYLOARTHRITIS (AXSPA)

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Introduction. The strong clinical and genetic associations between axial spondyloarthritis (axSpA) and inflammatory bowel disease (IBD) underscore the pathogenic role of the gut-joint axis. We previously identified a subpopulation of pathogenic mature CD8+ T cells in the axSpA synovial fluid (SF), expressing CD103+ and CD49a+ integrins (InEx cells). The expression of integrins on InEx cells implicates their role in aberrant migration between the gut-joint axis. Whether InEx cells recognize an arthritogenic peptide resulting in aberrant migration remains unclear. We hypothesize that InEx cells may incite and perpetuate chronic inflammation in axSpA. To this end, we characterized their T-cell receptor (TCR) repertoire and gene expression profile.
Materials and methods. Peripheral blood and synovial fluid mononuclear cells from HLA-B27+ axSpA patients with active disease were isolated. From these, mature CD8+ T cells and InEx cells were FACS-sorted and subject to single-cell TCR (paired a/b chains) and RNA sequencing. An HLA-B27+ reactive arthritis (ReA) sample was used as a comparison since it is the paradigm for antigen-driven inflammation initiated in the gut. These were all compared to HLA-B27+ healthy controls.
Results. The InEx TCR repertoire from axSpA patients was less diverse than mature CD8+ T cells from the same population. Further, they exhibited shared similarities with ReA mature CD8+ T cells in the TCR V region: TRBV20-1/TRAV1-2, TRBV6-1/TRAV1-2, and TRBV6-2/TRAV1-2. On a transcript level, InEx cells differed from ReA mature CD8+ T cells based on elevated genes such as *CCL4L2*, *IFITM1*, and *IFITM2*, and downregulated genes such as *IL7R*, *GNLY*, and *KLRB1*.
Conclusion. These observations suggest that InEx cells may incite inflammation in axSpA similarly to ReA, possibly mediated by HLA-B27. However, their ability to perpetuate inflammation may be distinct from ReA due to a varied transcriptome. This has important implications for therapeutic designs attempting to target integrin blockade in axSpA.

O9

IDENTIFYING NOVEL MOLECULAR SIGNATURES IN PSORIATIC ARTHRITIS THROUGH HIGH-THROUGHPUT PROTEOMIC ANALYSIS OF IMMUNE CELLS

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Introduction. Diagnosing psoriatic arthritis (PsA) is challenging, making the discovery of novel biomarkers through proteomics crucial for early and accurate detection. Molecular clustering can reveal disease heterogeneity, aiding in the identification of subtypes and informing personalized treatments. In this way, the aims of our study are to discover novel proteins involved in PsA pathogenesis and uncover molecular phenotypes of PsA patients through unsupervised analysis.
Methods. This study involved 154 participants, including 104 PsA patients (diagnosed by CASPAR criteria) and 50 control subjects with musculoskeletal symptoms but no rheumatic disease. We analyzed 384 proteins in peripheral blood mononuclear cells (PBMCs) using Olink technology, exploring biological functions with the R platform and identifying molecular clusters with a self-organizing map algorithm.
Results. PsA patients had an average disease duration of 7±5 years and moderate disease activity, with elevated acute phase reactants compared to controls. Clinical manifestations included dactylitis, enthesitis, and onychopathy. We detected 338 proteins in PBMCs, with 73 significantly altered in PsA patients, enriched in inflammatory response, immune function, and osteoclast activity. Proteins altered were associated with disease activity and dactylitis. Unsupervised analyses identified three PsA clusters with distinct molecular patterns, involving proteins like DAPP1, CCN2, SPRY2, and others, and different levels of C-reactive proteins and monocytes. Moreover, we observed differences in the proteomic profile based on the disease duration (>5 years or <5 years).
Conclusion. High-throughput proteomic analysis identified novel protein alterations in PsA related to inflammation, immune response, and osteoclast function. This study revealed distinct molecular clusters tied to clinical features and immune cell types. These findings pave the way for validation studies to confirm these proteins as robust biomarkers, seeking to improve PsA diagnosis.
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O10

ASSOCIATIONS AND IMPACT OF KINESIOPHOBIA ON PATIENT REPORTED OUTCOMES AND PERFORMANCE BASED MOBILITY MEASURES IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS

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Introduction/Objective. To investigate the impact of kinesiophobia on patients with axial spondylo-arthritis (axSpA), and its relation to global functioning and health, disease activity, function, spinal mobility and physical activity in comparison to healthy controls (HC).
Materials and methods. Observational study in which consecutive patients with axSpA (n=100) and 20 HC were examined by the Tampa scale of kinesiophobia (TSK), and the Fear avoidance questionnaire (FABQ). Patient reported outcomes and objective assessments: ASDAS, BASDAI, BASFI, ASAS HI, mSQUASH as well as the BASMI, the AS physical performance index (ASPI), the Short Physical Performance Battery (SPPB) were collected. Categorical analysis to differentiate results of patients with minimal/low (TSK≤28) and moderate/high (TSK>28) kinesiophobia as well as linear correlations comparing TSK and FABQ with demographic data and clinical assessments were performed.
Results. While demographic data for age, sex and BMI were comparable, patients with axSpA showed higher TSK (25.5±6.8 vs 14.0±5.1) and FABQ scores (40.1±22 vs 3.1±6.9), worse results for pain, daily level of physical activity (p=0.0012) and performance based tests compared to HC, all p<0.001 (Table I). Categorical analysis of kinesiophobia levels revealed that patients with moderate to high kinesiophobia performed significantly worse in ASPI (32.2±15.0 vs 42.0±18.3) and SPPB (10.4±1.3 vs 9.7±1.5), and showed numerical impairments in BASMI (3.8±1.7 vs 4.3±2.6) (Table II). Linear correlations between TSK and FABQ were observed for ASAS HI (r=0.45 and 0.52) and BASFI (r=0.38 and 0.44), but not for ASPI, SPPB, mSQUASH. Weak correlations were found for BASMI (r=0.24 and 0.38) and BASDAI scores (both r=0.35).

O10: Table I. Comparison of demographics and assessments between axSpA and HC.

	All axSpA	HC	*p*-value
n	100	20	
Age (y)	45.2 ± 11.7	44.6 ± 14.8	0.884
Sex (m); n (%)	69 (69)	11 (55)	0.298
BMI (kg/m^2)	28.1 ± 5.5	25.4 ± 4.6	0.044
BASDAI	5.4 ± 1.6	0.8 ± 0.7	**<0.001**
ASDAS	3.1 ± 0.8	-	-
BASFI	5.4 ± 2.0	0.4 ± 0.8	**<0.001**
Back pain	6.7 ± 1.8	0.8 ± 2.0	**<0.001**
Nocturnal pain	5.9 ± 2.3	0.1 ± 0.5	**<0.001**
ASAS-HI	8.6 ± 3.0	0.7 ± 0.9	**<0.001**
mSQUASH	6968 ± 5602	11194 ± 4231	**0.0012**
TSK	25.5 ± 6.8	14.0 ± 5.1	**<0.001**
TSK >severe kinesiophobia, n (%)	35 (35)	0 (0)	**<0.001**
FABQ	40.1 ± 22.6	3.1 ± 6.9	**<0.001**
FABQ1	14.8 ± 6.9	1.9 ± 4.8	**<0.001**
FABQ2	13.4 ± 9.3	1.2 ± 3.6	**<0.001**
FABQ3	11.9 ± 10.0	0 ± 0	**<0.001**
BASMI	4.0 ± 1.77	1.4 ± 0.8	**<0.001**
ASPI	36.1 ± 18.3	19.0 ± 5.7	**<0.001**
SPPB	10.1 ± 1.5	11.9 ± 0.5	**<0.001**

*variables are mean ± standard deviation if not otherwise indicated; ASPI: the AS physical performance index; ASDAS: AS Disease Activity Score; SPPB: Short Physical Performance Battery, BASMI: Bath Ankylosing Spondylitis (AS) Metrology Index; BASDAI: Bath AS Disease Activity Index; BASFI: Bath AS Functional Index; CRP: C-reative protein; NSAIDs: Non-steroidal anti-inflammatory drugs; axSp: axial spondyloarthritis; NRS: numerical rating scale; mSQUASH: the modified-Short QUestionnaire to Assess Health-enhancing physical activity; TSK: Tampa Scale for Kinesiophobia FABQ: Fear-Avoidance Beliefs Questionnaire; FABQ1: 'physical activity'; FABQ2: 'work as cause of pain' FABQ3: 'patients' assumptions of their probable return to work'.

O10: Table II. Comparison of demographics and assessments between patients with minimal and low and moderate and high levels of kinesiophobia.

	TSK ≤28 (n=61)	TSK >28 (n=35)	*p*-value
Age (y)	45.7 (11.7)	44.0 (11.5)	0.48
Sex (m); n (%)	38 (62.3)	27 (77.1)	0.13
r-axSpA, n(%)	35 (57.4)	26 (74,3)	
Nr-axSpA (n (%)	26 (42.6)	9 (25,7)	
Disease duration (y)	9.9 (11.5)	9.8 (11.0)	0.46
Symptom onset (y)	18.2 (12.0)	17.3 (11.3)	0.73
BMI (kg/m^2)	27.9 (4.6)	28.5 (6.9)	0.62
CRP (mg/l)	11.4 (8.7)	24.6 (14.0)	0.56
BASDAI	5.0 (1.5)	6.1 (1.5)	**<0.001**
ASDAS	3.0 (0.9)	3.3 (0.7)	0.1
BASFI	5.0 (1.9))	6.2 (2.0)	**<0.01**
ASAS HI	7.7 (2.6)	10.2 (2.9)	**<0.001**
FABQ	32.1 (20.8)	52.3 (19.1)	**<0.001**
mSQUASH	6.972 (4932)	6.132 (6072)	0.5
BASMI	3.8 (1.7)	4.3 (2.6)	0.1
SPPB	10.4 (1.3)	9.7 (1.5)	**<0.05**
ASPI	32.3 (15.0)	42.0 (18.3)	**<0.05**

*variables are mean ± standard deviation if not otherwise indicated; ASDAS: AS Disease Activity Score; BASMI: Bath Ankylosing Spondylitis (AS) Metrology Index; BASDAI: Bath AS Disease Activity Index; BASFI: Bath AS Functional Index; CRP: C-reative protein; r-axSpA: radiographic axial spondyloarthritis (axSpA); non-radiographic axSpA (nr-axSpA); NRS: numerical rating scale; mSQUASH: the modified-Short QUestionnaire to Assess Health-enhancing physical activity; TSK: Tampa Scale for Kinesiophobia FABQ: Fear-Avoidance Beliefs Questionnaire.

Conclusion. Kinesiophobia seems to be a clinically relevant problem of patients with axSpA, since the mobility of patients with moderate to high TSK and FABQ scores was much more impaired in this study. However, the degree of kinesiophobia showed stronger correlations with physical function, global functioning and health than with mobility and PA.
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O11

THE ASSESSMENT OF SPONDYLOARTHRITIS INTERNATIONAL SOCIETY (ASAS) DEFINITION OF DIFFICULT-TO-MANAGE AXIAL SPONDYLOARTHRITIS

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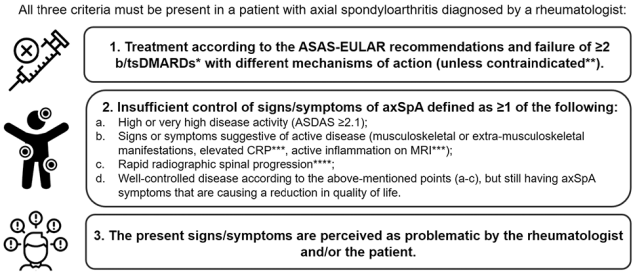
Introduction/Objective. Non-response to standard treatments represents a management challenge in axial Spondyloarthritis (axSpA). The Assessment of SpondyloArthritis international Society (ASAS) seeks to define 'difficult-to-manage axSpA' (D2M axSpA). The aim of this work was to develop a consensus-based definition of D2M axSpA for use in clinical and research settings.
Materials and methods. A scoping literature review was conducted in 2022 to identify potential definitions for D2M axSpA from prior studies, followed by a 2-round Delphi consensus process to identify components of D2M axSpA. The 1st Delphi round surveyed ASAS members, with 123 respondents. In January 2023, the results were presented to the Task Force and ASAS members. This was followed by the 2nd Delphi round taking the results of the discussion into consideration; a total of 186 responses were received. Based on the results of the 2nd Delphi round, a draft D2M axSpA definition was developed and presented to the Task Force and subsequently to the ASAS members in January 2024. Full ASAS members (n=123) voted on the proposed definition.
Results. Consensus was reached on a definition encapsulating treatment failure, suboptimal disease control, and physician or patient acknowledgment of problematic signs/symptoms in patients diagnosed with axSpA by rheumatologist (Fig. 1). ASAS D2M axSpA definition is, therefore, a broad concept including a variety of reasons leading to an unsatisfactory treatment outcome. "Treatment-refractory" disease is a part of the D2M group, which can be concluded after excluding other reasons for the non-response and require a history of specific treatment failure and the presence of objective

signs of inflammatory activity. The proposed definition was endorsed by ASAS at the annual meeting in January 2024 with 89% votes (109/123) in favor of the definition.

Conclusion. The ASAS D2M axSpA definition, shaped by extensive professional and patient input and a structured consensus process, provides a clear identification of patients with non-response to current standard treatments paving a way to further research.



O11: Fig 1. The ASAS Difficult-to-Manage Axial Spondyloarthritis Definition. ASAS: Assessment of Spondyloarthritis international Society, ASDAS: Axial Spondyloarthritis Disease Activity Score, axSpA: axial spondyloarthritis; bDMARDs: biologic disease-modifying antirheumatic drugs, CRP: C-reactive protein, EULAR: European Alliance of Associations for Rheumatology, MRI: magnetic resonance imaging, tsDMARDs: targeted synthetic disease-modifying anti-rheumatic drugs.

O12

EXTL3 INVOLVED IN THE REGULATION OF ENDOCHONDRAL OSSIFICATION IN AXIAL SPA

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Introduction. Spondyloarthritis (SpA) is characterized, in severe forms of the disease by complete ankylosis of the spine and sacroiliac joints SIJ, defining ankylosing spondylitis (AS). This study aims to advance our understanding of the pathophysiology of ossification in AS.

Materials and methods. Using whole exome sequencing analysis, we identified a mutation in the EXTL3 gene within a family exhibiting a familial form of AS. We developed a mouse model carrying the mutation (Extl3mut/+) and conducted phenotypic exploration using microCT and histological analyses. Cellular and molecular pathways altered by this mutation, were investigated in primary osteoblast and chondrocyte cultures, using transcriptomic and proteomic analyses.

Results. Phenotypic analysis of Extl3mut/+ mice revealed two abnormalities: osteoarticular lesions and trabecular bone alterations. Osteoarticular lesions was characterized in in 6-month-old Extl3mut/+ by erosions of the sacral cortical bone. In 12-month-old Extl3mut/+ mice, islet formation within the iliac cortical was observed, corresponding to the inclusion of non-mineralized osteoid tissue. These lesions were associated with disorganization of collagenous cartilage and chondrocyte columns, along with sacral cartilage thickening. Bridges within the SIJ space were visualized at 12 months. Trabecular bone alteration is associated with an accumulation of non-mineralized osteoid tissue. HS accumulation was documented in both regions, primarily originating from osteoblasts. Osteoblast cultures exhibited delayed differentiation and a diminished capacity for mineralization associated with an activation of the Wnt pathway, yet it appeared insufficient to rectify the observed phenotype. Conversely, chondrocyte cultures demonstrated an accelerated differentiation process, associated with the activation of the Wnt pathway, suggesting a potential deregulation of endochondral ossification.

Conclusion. This study identified a rare variant of the EXTL3 gene in a family with familial aggregation of AS. The murine model demonstrated an articular, including cartilage lesions, and bone phenotype. Thus, abnormalities in the HS synthesis pathway, already implicated in multiple exostoses disease, may also contribute to the pathophysiology of ossification phenomena in AS.

O13

SPONDYLOARTHRITIS PATIENT CELLS DISPLAY TNF HYPERRESPONSIVENESS THROUGH ENHANCED P65 NUCLEAR TRANSLOCATION

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Introduction. Axial spondyloarthritis (AxSpA) can lead to abnormal bone formation in the spine. The role of inflammation in promoting this phenotype remains unclear but reducing inflammation with TNF or IL-17A inhibitors or COX-2-selective NSAIDS may slow the progression of axial disease over time. Our preliminary data suggest that patient-derived induced pluripotent stem cells (iPSCs) differentiated into mesenchymal stem cells (MSCs) and then osteoblasts exhibit increased mineralization capacity correlating with elevated baseline *IL1A* and *PTGS2* (COX-2) expression. Here, we show that TNF exposure further amplifies *IL1A* and *PTGS2*/COX-2 expression in patient cells.

Methods. IPSCs derived from skin fibroblasts of 3 healthy donors (HD) and 4 AxSpA patients were differentiated into MSCs. For transcriptome and chromatin analysis cells were stimulated with TNF for 24h followed by RNASeq and ATACSeq. In parallel, some cells were used to measure COX-2 and IL-1a protein expression via immunofluorescence (IF) staining and western blotting (WB). Untreated cells were used as controls. Analysis of downstream signaling upon TNF stimulation was evaluated by detecting p65 nuclear translocation via IF and WB.

Results. We detected increased *IL1A* and *PTGS2* expression in patient-derived MSCs compared to HD cells following TNF treatment. Furthermore, increased type I IFN response gene expression was measured in patient MSCs after TNF stimulation compared to HD. Corresponding differences in chromatin accessibility were also found. Areas with augmented chromatin accessibility in patient cells included type I IFN response genes like *OAS1*, *ISG20* and *NMI*, confirming RNASeq data. Finally, analysis of downstream signaling revealed increased p65 nuclear translocation in patient MSCs after 15 minutes of TNF stimulation relative to HD cells.

Conclusion. These findings indicate that AxSpA MSCs exhibit enhanced responsiveness to TNF and suggest that TNF hyperresponsiveness may be a characteristic of AxSpA and that IL-1a may play a role in driving abnormal bone formation in this disease.

O14

PD-1+TIGIT+LAG3+ CD8+ T CELLS WITH FEATURES OF EXHAUSTED PHENOTYPE CHARACTERIZE CHRONIC JOINT INFLAMMATION IN AXIAL SPONDYLOARTHRITIS

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Introduction. Persistent chronic inflammation is the clinical hallmark of axial spondyloarthritis (AxSpA). Recent studies from our laboratory provided key evidence that pathogenic cytotoxic CD8+ T cells (CTLs) play a central role in AxSpA pathogenesis. How pathogenic CTLs perpetuate inflammation in AxSpA remains ill-defined. Chronic activation of CTLs is ordinarily followed by T cell exhaustion, which serves as a host homeostatic mechanism to limit collateral tissue damage resulting from persisting CTL effector functions. Here we sought to determine whether pathogenic CTLs are truly immunologically exhausted. We hypothesized that evasion of T cell exhaustion contributes to autoinflammation in axSpA patients.
Methods. We performed mass cytometry time-of-flight (CyTOF) and single cell RNA sequencing (scRNAseq) of paired PBMCs and SFMCs to comprehensively characterize CTLs in axSpA. We measured protein expression of more than 30 surface and intracellular markers related to CTL memory phenotypes and cytotoxic properties to identify expanded pathogenic CTLs in axSpA patients. scRNAseq of mature CTLs was performed in tandem with scTCRseq to characterize the AS CTL transcriptome and to measure T cell receptor diversity respectively.
Results. We identified a clonally expanded CTL subset in the inflamed synovial microenvironment from AS patients that, despite an expression of immune checkpoints (PD-1, TIGIT & LAG-3), retain their capacity to express granzymes, perforin, TNF-α, and IFN-γ. Concurrent gene expression analysis using single cell RNA sequencing reveals that genes that are canonically expressed in *bona fide* exhausted CTLs such as *CD160, HAVCR2, ENTPD1, NT5E,* are completely downregulated in AS CTLs. We also observe that at the protein level these CTLs have downregulated TOX, the transcription factor regulating CTL exhaustion.
Conclusion. We discovered an immune checkpoint expressing CTL subset in axSpA that resists immune exhaustion, and paradoxically retains effector functions. We propose that evasion of exhaustion represents a mechanism which could be central to perpetuate axSpA inflammation.
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O15

IMPAIRMENT OF REGULATORY T CELLS STABILITY IN AXIAL SPONDYLOARTHRITIS: ROLE OF EZH2 AND PSTAT5

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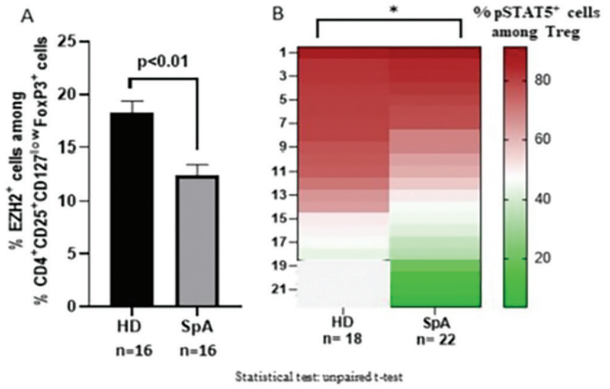
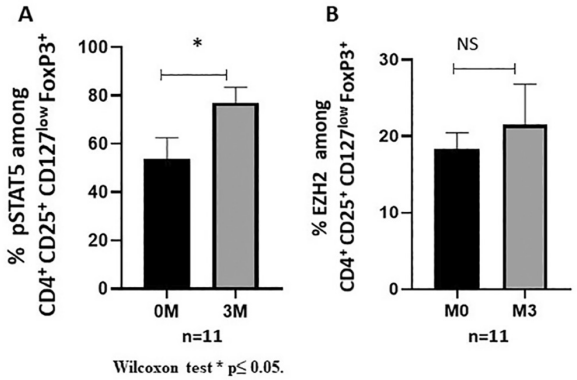
Introduction. Functional impairment of regulatory T cells (Treg) is usually linked to inflammatory autoimmune diseases, but limited data is available regarding Treg involvement in axial spondyloarthritis (axSpA). Treg stability refers to their ability to maintain their functions and characteristics in pro-inflammatory environments. EZH2 and phosphorylated STAT5 (pSTAT5) play a critical role in maintaining Treg stability. Here, we aimed to characterize Treg stability in patients with axSpA.
Methods. Peripheral blood mononuclear cells (PBMCs) from axSpA patients, either naïve from targeted therapy or treated by TNF inhibitors (TNFi), were freshly isolated. PBMCs from healthy donors (HD) were used as controls. Expression of stability (EZH2, pSTAT5) and suppressive (TNFR2 and CD39) markers by Treg was analyzed by flow cytometry.
Results. EZH2 expression by Treg was decreased in axSpA patients as compared to HD (Fig. 1A). Mechanistic study showed that inhibition of EZH2 attenuated Treg differentiation and suppressive phenotype *in vitro*. EZH2 was found to be predominantly expressed by highly suppressive TNFR2+ and CD39+ Treg. Additionally, axSpA patients also exhibited a reduced frequency of pSTAT5+ Treg compared to HD (Fig. 1B), while pSTAT5+ (fig 2A), but not EZH2+ (fig2B), Treg frequency increased at 3 months of TNFi treatment compared to baseline. This last result suggested a restoration of Treg stability upon TNFi treatment.
Conclusion. By highlighting a deficient expression of EZH2 and pSTAT5 by Treg, our findings revealed an impaired Treg stability in axSpA. Deciphering the specific pathways influenced by these molecules is necessary to design targeted therapies that will be able to specifically restore Treg stability in axSpA.



O15: Fig. 1.



O15: Fig. 2.

O16

GLOBAL DISTRIBUTION AND DETERMINANTS OF DIAGNOSTIC DELAY ACROSS DIVERSE SPONDYLOARTHRITIS ENTITIES: DATA FROM THE INTERNATIONAL ASAS-PERSPA STUDY

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Introduction. Diagnostic delay (DD) in spondyloarthritis (SpA) is well documented, but most of the available data are reported in patients with axial SpA (axSpA). In this ancillary analysis from the international ASAS-PerSpA study, we aimed to estimate the DD across all SpA entities, and evaluate the factors associated with DD.

Materials and methods. In 4,339 patients diagnosed by their rheumatologists with any SpA entity (axSpA, peripheral (pSpA), inflammatory bowel diseases associated SpA (IBD-SpA), reactive arthritis (ReA), Juvenile also including psoriatic arthritis (PsA), the DD was estimated using two definitions (1, including extramusculoskeletal manifestations (EMM) as an accepted inaugural manifestation, and 2, considering the first MM as onset of the rheumatic disease). DD was compared across disease entities. Factors associated with DD (definition 2), were evaluated using 4 multivariable linear regression models (axSpA, PsA, pSpA, and IBD-SpA).
Results. The analysis included 2622 patients with axSpA, 1016 PsA, 424 pSpA, 110 IBD-SpA, and 167 others, mean age 44.4 years (±13.9), 60.9% females. The mean global DD was longer (6.6 years ± 8.6) while using definition 1 compared to 2 (4.5 years ±7.0) (Table I). Based on definition 2, DD was significantly longer for axSpA and IBD-SpA -where the first MM was axial, compared to PsA, pSpA, and others-where the first MM was peripheral. Factors associated with DD varied across the SpA entities. The type of first manifestation, age at first symptom (younger), age at study inclusion (older) were associated with all SpA entities (Table II).
Conclusion. DD was significantly longer in axSpA and IBD-SpA compared to PsA, pSpA, and other forms of SpA and was associated with the phenotype of the inaugural symptom. Factors associated with DD varied across SpA entities, thus indicating the necessity of considering both the main SpA entity and the initial disease manifestation when evaluating and reporting DD.

O16: Table I. Diagnostic delay across spondyloarthritis (SpA) disease entities

	Definition 1			Definition 2		
Main SpA disease	Beginning of disease may be EMM or MM			Beginning of disease is any MM only		
	N	Mean (SD)	Median (IQR)	N	Mean (SD)	Median (IQR)
Axial SpA	2622	6.04 (7.55)	3.00 (0.92 ; 8.42)	2615	5.56 (7.31)	3.00 (1.00 ; 8.00)
Inflammatory Bowel Disease – SpA	110	8.97 (9.10)	5.91 (1.31 ; 14.48)	108	5.15 (7.68)	1.00 (0.00 ; 9.75)
Psoriatic Arthritis	1016	9.19 (11.10)	4.96 (1.00 ; 14.09)	995	2.48 (6.06)	0.00 (0.00 ; 2.00)
Peripheral SpA	424	4.42 (6.54)	1.29 (0.33 ; 5. 80)	423	3.07 (5.89)	1.00 (0.00 ; 4.00)
Reactive Arthritis	55	3.02 (5.56)	1.00 (0.08 ; 3.09)	55	2.95 (5.65)	1.00 (0.00 ; 3.00)
Juvenile SpA	52	4.24 (7.73)	2.00 (1.00 ; 5.65)	52	4.21 (5.78)	2.00 (1.00 ; 5.75)
Others	60	3.80 (7.28)	0.08 (0.00 ; 3.84)	58	2.03 (3.42)	0.50 (0.00 ; 2.25)
All	4339	6.60 (8.59)	3.00 (0.75 ; 9.59)	4306	4.50 (6.98)	2.00 (0.00 ; 6.00)
p-value		<0.001	<0.001		<0.001	<0.001

O16: Table II. Factors associated with diagnostic delay across spondyloarthritis (SpA) entities, based on four multivariable linear regression models*

	Axial SpA (N=2615)		Inflammatory Bowel Disease SpA (N=108)		Psoriatic arthritis (N=995)		Peripheral SpA (N=423)	
Patient and disease characteristics	Beta	p-value	Beta	p-value	Beta	p-value	Beta	p-value
First symptom is axial			0.218	0.009	0.109	0.016		
First symptom is peripheral	-0.073	<0.001						
First symptom is EMM	-0.206	<0.001	0.218	0.018				
Age at first symptom	-0.552	<0.001	-0.579	<0.001	-0.782	<0.001	-0.869	<0.001
Age at study inclusion	0.654	<0.001	0.644	<0.001	0.654	<0.001	0.897	<0.001
Lag between psoriasis and MM					0.622	<0.001		
Lag between IBD and MM			0.562	<0.001				
World region					0.107	0.009		
Psoriasis							-0.128	0.018
Uveitis								
Family history of psoriasis	0.050	0.009						
High CRP (ever)							0.113	0.025
HLA-B27 positivity	-0.105	<0.001					-0.104	0.050
Fibromyalgia (FiRST)					0.094	0.022		
Body Mass Index							0.151	0.004

* Only statistically significant associations are shown.
EMM: extramusculoskeletal manifestation; FiRST: Fibromyalgia questionnaire; IBD: inflammatory bowel disease; MM: musculoskeletal manifestation.

O17

MINIMALLY INVASIVE ULTRASOUND-GUIDED BIOPSY OF THE COMMON EXTENSOR TENDON ENTHESIS: A CADAVERIC STUDY TO STANDARDIZE THE TECHNIQUE

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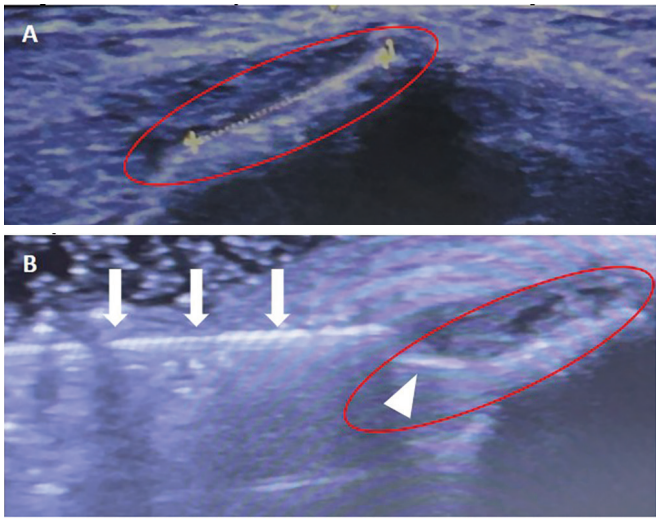
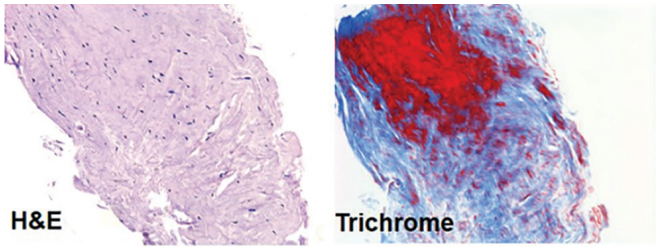
Introduction. Enthesitis is the hallmark lesion of Spondyloarthritis. To detangle the complex mechanisms of enthesitis the analysis of informative tissue is crucial. Ultrasound (US) is accurate in identifying enthesitis and it guides interventional procedures. Up to date, only a non-US guided technique to collect entheseal tissue has been described (1). The aim of the study was to standardize a minimally invasive US-guided biopsy technique for the retrieval of entheseal tissue.
Methods. Human cadaveric elbows were used to establish the US-guided biopsy of the common extensor tendon (CET) enthesis. US study of the CET was performed to identify the enthesis with a landmark-based approach to prevent damage to surrounding structures. A mini-arthroscopic instrument, consisting in a coaxial guide needle coupled with a mini-forceps, was introduced under US guide up to the target area to collect samples. A stain was injected through the guide needle and dissection was performed to verify the correct location of sampling. Histology was performed to assess quality and representativeness of samples. 4 couples of operators performed the procedure on different specimens to evaluate the reliability of the technique.



O17: Fig. 1. Minimally-invasive CET US-guided biopsy technique.
A: Localization of the CET enthesis target area (red circle)
B: US-guided biopsy technique, the guide needle (white arrows) is inserted up to the target area (red circle) where the tip of the mini biopsy forceps is opened to collect tissue
CET: common extensor tendon, US: ultrasound



O17: Fig. 2. Evaluation of a representative entheseal sample obtained by minimally-invasive US-guided biopsy at CET level.
CET: common extensor tendon, US: ultrasound; H&E: hematoxylin and eosin

Results. 24 specimens from six cadaveric elbows were collected. The target area was defined at the insertion of the extensor carpi radialis brevis component of the CET, above the anterior tubercule of the lateral epicondyle (Fig. 1). The stain used to assess the location of sampling was found in the target area exposed after dissection. No damage to neighbouring structures was evidenced. 83% of samples presented histological features compatible with entheseal tissue (Fig. 2). All operators correctly identified the CET and performed the procedure. Quality of samples was reliable between operators.
Conclusion. We established a landmark-based, systematic approach to perform a feasible and safe US-guided biopsy of CET enthesis. The standardized procedure allows the reliable retrieval of entheseal tissue.

Reference

1. PACHOWSKY ML *et al*.: Concise report: a minimal-invasive method to retrieve and identify entheseal tissue from psoriatic arthritis patients. *Ann Rheum Dis* 2022; 81: 1131-35.

O18

MACROPHAGE MIGRATION INHIBITORY FACTOR AND HYPOXIA-INDUCIBLE FACTOR 1-ALPHA MAY PLAY A ROLE IN MAINTAINING INTESTINAL HOMEOSTASIS THROUGH REGULATION OF OCCLUDIN AND CLAUDIN-1 EXPRESSION

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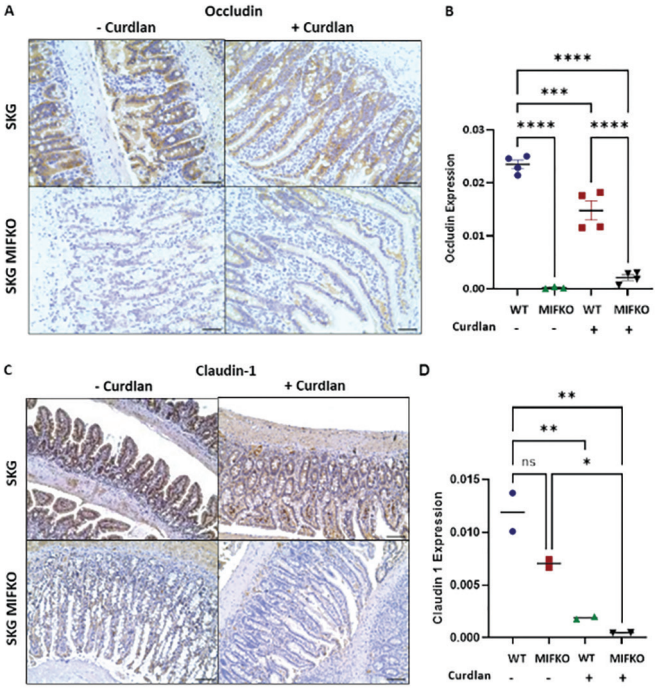
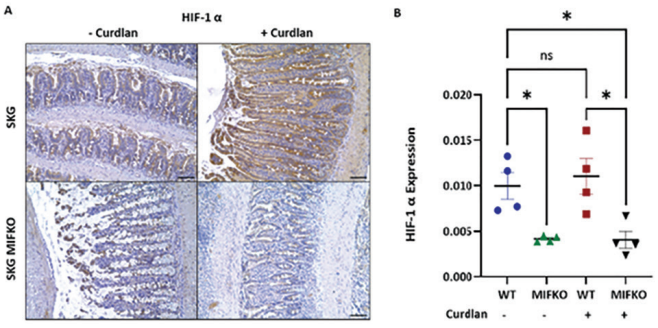
Introduction. Axial Spondyloarthritis (axSpA) is a chronic inflammatory condition mainly impacting the musculoskeletal system. Many axSpA patients also experience gut inflammation, ranging from microscopic changes to Inflammatory Bowel Disease (IBD). We've previously reported involvement of macrophage migration inhibitory factor (MIF) in gut inflammation. In a SpA mouse model (SKG), MIF overexpression replicates clinical features, while inhibiting MIF alleviates symptoms. MIF and hypoxia-inducible factor 1 alpha (HIF-1α) are interdependent, but their exact roles in gut inflammation in axSpA are still unclear.
Aim. To investigate the MIF-HIF interaction's impact on gut homeostasis and inflammation in the SKG mouse model.
Methods. Eight-week-old SKG mice were divided into four groups: SKG control, SKG curdlan-treated, SKG MIF Knockout (MIFKO), and SKG MIFKO curdlan-treated. After 8 weeks of treatment, immunohistochemistry (IHC) was performed on ileal samples from these mice to assess tight junction markers (occludin, claudin-1), and HIF-1α expression. Difference between the 4 groups was analyzed using Kruskal-Wallis test.
Results. SKG-MIFKO mice showed reduced occludin and claudin-1 expression, indicating tight junction disruption (Fig. 1). HIF-1α expression decreased significantly in SKG-MIFKO and SKG-MIFKO+curdlan groups. Notably, HIF-1α expression decreased further in SKG-MIFKO+curdlan compared to SKG+curdlan, with a trend of higher expression in SKG+curdlan compared to controls (Fig. 2).
Conclusion. Knocking out MIF in SKG mice resulted in a notable improvement in axSpA symptoms. However, our findings show that moderate levels of MIF may have a protective role in the gut of SKG mice. In the complete absence of MIF, the expression of tight junction markers and HIF-1α decreased, suggesting that the MIF-HIF interaction is essential for the integrity of the gut epithelial barrier in SKG mice. Further investigation is required to determine the role of HIF-1α in transcriptional regulation of tight junction markers and what the impact of partial inhibition of MIF/HIF would be.



O18: Fig. 1. IHC staining of tight junction markers, occludin and claudin-1. Ileal Tight Junction Markers are decreased in the absence of MIF.



O18: Fig. 2. IHC staining of HIF-1α. HIF-1α expression decreases in the absence of MIF.

O19

INTEGRATIVE HOST-MICROBIOME PROFILING IN TREATMENT-NAÏVE SPONDYLOARTHRITIS SHOWS OXIDATIVE STRESS-INDUCED DYSBIOSIS AND LINKS THE B2 ENTEROTYPE TO DISEASE ACTIVITY AND TREATMENT RESPONSE

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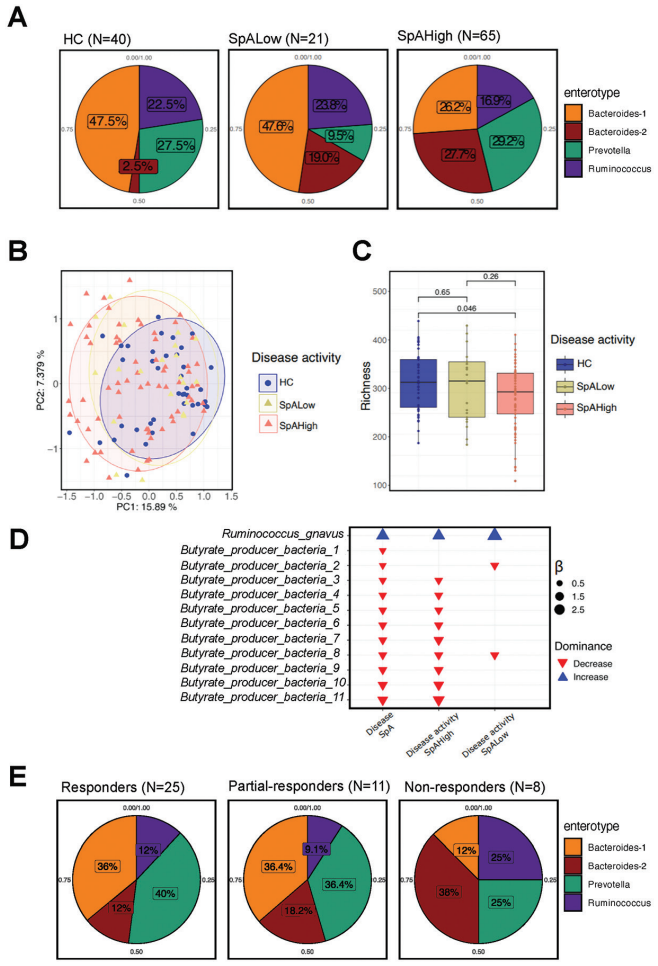

O19: Fig. A: Enterotypes distribution in Healthy Controls (HC) and SpA patients stratified based on ASDAS disease activity score: SpALow corresponds with ASDAS ≤2.1 and SpAHigh with ASDAS >2.1.
B: mOTUs-level fecal microbiome community variation according to disease activity represented by principal coordinates analysis (PCoA) based on Bray-Curtis distance.
C: Bacterial richness measured according to the disease activity.
D: Bacterial biomarkers regarding the disease status (HC vs SpA) and disease activity. The size of the arrows represents the effect size (β) and the color indicates the dominance.
E: Enterotypes distribution among the axial-SpA patients enrolled in the GoGut clinical trial regarding their treatment response: responders (defined as ASDAS <1.3 associated with inactive disease, on two consecutive visits with an interval of at least 12 weeks), partial-responders (defined as ASDAS <2.1 at the end of the trial) and non-responders (defined as ASDAS >2.1 at the end of the trial).

Introduction. Gut involvement is a key driver in Spondyloarthritis (SpA) pathogenesis, but the underlying mechanisms are still unclear. Nevertheless, intestinal dysbiosis has been reported in SpA patients. However, none of these studies included exclusively treatment naïve patients, while medication can profoundly bias the microbiome profiling. To this end, we conducted a deep characterization of the gut microbiota dysbiosis in a large cohort of new-onset SpA patients in both stools and gut biopsies. We also linked intestinal microbiota profile to the treatment response in a cohort of early axial SpA.
Methods. We combined multiple approaches of quantitative microbiome sequencing coupled to a large metabolomic analysis to access the key components of the gut-joint axis in SpA by analyzing feces and gut biopsies. We integrated these data to an extensive metadata set.
Results. A profound gut dysbiosis was observed in SpA patients with an enrichment of the dysbiotic B2 enterotype (Fig. A), a shift in bacterial communities (Fig. B) and a reduction of richness (Fig. C). Interestingly, a redundancy analysis of effect sizes demonstrated that the observed microbial variance was explained by the disease activity status (ASDAS score), but not by the gut inflammatory status (histology). The dysbiosis was characterized by a drastic loss of butyrate-producing bacteria and the overgrowth of *Ruminococcus gnavus* in feces (Fig. D), while *Dialister invisus* was enriched in the ileum of SpA patients. In addition, we analyzed the baseline microbiome of patients enrolled in an investigator-initiated trial to evaluate its potential in predicting therapeutic outcomes. We observed an enrichment of B2 enterotype in the non-responders (Fig. E).
Conclusion. A marked dysbiosis occurs in SpA and links to disease activity. We anticipate that transitory oxidative stress bursts are significantly reshaping the gut bacterial communities leading to the overgrowth of opportunistic bacteria. Our results suggest that microbiome profiling may assist in predicting treatment responses in SpA.

O20

LOW DISEASE ACTIVITY IN REAL-LIFE PATIENTS WITH A DIAGNOSIS OF AXIAL SPONDYLOARTHRITIS: AN ACHIEVABLE TARGET?

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Introduction. Remission or low disease activity (LDA) are recommended targets in the treatment of axSpA. However, frequency of LDA in real-life, and barriers hindering this objective, are under-investigated. Our aim was to evaluate the frequency of LDA in a real-life axSpA population starting bDMARD treatment.
Material and methods. Patients with a diagnosis of axSpA by the rheumatologist, attending our outpatient clinic, and starting a bDMARD between 10/2021-10/2023, were enrolled and followed up (6 and 12-months). Baseline characteristics of patients and disease, activity indices including ASDAS, therapy adherence via Compliance Questionnaire for Rheumatology (CQR5)] were compared between patients achieving ASDAS-LDA (ASDAS<2.1) at both 6 and 12-months (sustained ASDAS-LDA) and those who did not, using Chi-square and Mann-Whitney tests. Multivariable Generalized Estimating Equations (GEE) models (simple and autoregressive), with ASDAS-LDA as outcome, were built. Factors potentially influencing ASDAS-LDA (according to literature and own hypothesis) were used as independent variables. Results were expressed as beta (95% confidence interval).
Results. Ninety-seven patients were enrolled, 54% males, mean age 50.7±13.9, disease duration 7.9±8.7. Beside axial involvement, 42% had peripheral involvement, 27% psoriasis, 11% IBD and 12% uveitis. Only 50% were HLA-B27+ and 58% were bDMARDs-naïve. At 6 and 12 months respectively 51 and 46% reached ASDAS-LDA. Patients achieving sustained ASDAS-LDA were more frequently male (71% vs 43%, p=0.02), high school-educated (84% vs 53%, p=0.043), and had radiographic axSpA (64% vs 33%, p=0.009), and less frequently enthesitis or peripheral arthritis (14% and 32% vs 35% and 56%, p=0.04) as well as fibromyalgia (10% vs 58%, p<0.0001), despite similar baseline ASDAS (3.04±0.76 vs 3.09±0.55) and 12-months CRQ5 (18.3±2.6 vs 17.8±2.5). In the GEE autoregressive model, only sex and fibromyalgia remained independently associated to LDA (Table I).

O20: Table I. Independent variables associated with ASDAS-LDA in the multivariable Generalized Estimating Equation Models.

	Simple model	Autoregressive model
	Beta (95%CI)	Beta (95%CI)
Sex	0.09 (-0.02, 0.20)	**0.18 (0.02, 0.34)**
Age	**-0.004 (-0.008, -0.003)**	-0.003 (-0.01, 0.001)
High school education	0.05 (-0.07, 0.17)	0.12 (-0.05, 0.31)
Radiographic axSpA	0.11 (-0.01, 0.23)	0.12 (-0.05, 0.30)
Enthesis	-0.04 (-0.17, 0.10)	-0.11 (0.31, 0.08)
Peripheral arthritis	-0.04 (-0.15, 0.07)	-0.04 (-0.02, 0.12)
BMI	-0.006 (-0.02, 0.01)	-0.01 (-0.02, 001)
Fibromyalgia	**-0.23 (-0.35, -0.10)**	**-0.28 (-0.46, -0.11)**
bDMARD-naive	0.05 (-0.07, 0.17)	0.07 (-0.10, 0.25)

axSpA: axial spondyloarthritis; BMI: Body Mass Index; bDMARD: biological Disease Modifying Anti Rheumatic Drugs. Results in bold represent significant results. Simple model: not corrected for ASDAS-LDA at the previous timepoint; Autoregressive model: adjusted for ASDAS-LDA at the previous timepoint.
Results expressed a beta coefficient (95% confidence intervals.

Conclusion. ASDAS-LDA is a feasible target, although some patient-related and disease-related factors might hinder this objective. Further studies are warranted to understand how to overcome these barriers.

O21

EFFECTIVENESS AND COST-EFFECTIVENESS OF PATIENT-INITIATED FOLLOW-UP SUPPORTED BY ASYNCHRONOUS TELEMONITORING IN SPONDYLOARTHRITIS (TELESPA-STUDY): A PRAGMATIC MULTICENTRE RANDOMISED CONTROLLED TRIAL

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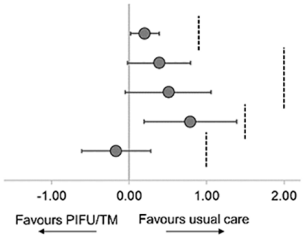
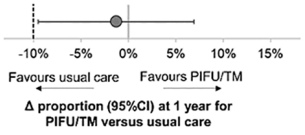
Introduction. With rising healthcare expenditures and an expected increase in workforce shortages, sustainable alternatives to traditional outpatient follow-up strategies are vital to ensure the continuous delivery of high-quality healthcare. We investigated the (cost-)effectiveness of patient-initiated follow-up supported by asynchronous telemonitoring (PIFU/TM) for the follow-up of patients with spondyloarthritis (SpA) compared to usual care (UC) in daily practice (TeleSpA).
Methods. TeleSpA was a multicentre, pragmatic, non-blinded, randomised controlled trial. Patients with SpA and stable disease were randomised to PIFU/TM or UC (1:1). Patients were followed once after 1 year with remote monitoring at 6 months (PIFU/TM) or at the discretion of their treating rheumatologist (UC). The primary outcome was the number of rheumatology visits within a 1-year period. We hypothesised superiority with a reduction of ≥25% of visits with PIFU/TM compared to UC. Secondary outcomes included health outcomes (non-inferiority of PIFU/TM vs UC) and 1-year cost-effectiveness. The primary analysis was by full analysis set. TeleSpA was registered with http://www.clinicaltrials.gov, NCT04673825.
Results. Between 2 December 2020 and 20 June 2022, 200 patients were randomly assigned to PIFU/TM (n=100) or UC (n=100). Participants had a mean age of 55.0 (SD 11.9) years, 79 (39.5%) were women and 121 (61.5%) were men. After 1 year, the mean number of visits was 1.9 (SD 1.5) in the PIFU/TM group and 2.6 (SD 1.3) in the UC group (mean difference -0.7 (95%CI -1.0 to -0.3) [25.4% reduction], $p<0.001$). Non-inferiority of PIFU/TM was demonstrated for all health outcomes of interest (Fig. 1). PIFU/TM was cost-effective from a healthcare perspective, saving healthcare costs (-€243) without loss in QALY (+0.004). No trial-related serious events were reported.
Conclusion. PIFU/TM safely resulted in significant and meaningful reductions in the total number of rheumatology visits. This was not at the expense of health outcomes and saved healthcare costs.

Funding. Dutch Arthritis Society

Continuous outcome	Mean (SD) at 1 year PIFU/TM	Usual care
ASDAS	1.93 (0.90)	1.75 (0.91)
BASDAI	3.66 (2.36)	3.22 (2.22)
Pain (VAS)	3.20 (2.45)	2.66 (2.34)
Patient global	3.59 (2.66)	2.71 (2.38)
Physician global	1.56 (1.59)	1.79 (1.65)



Categorical outcome	N (%) at 1 year PIFU/TM	Usual care
Good overall experience with care	84 (91.3%)	83 (92.2%)



O21: Fig. 1. Results for secondary outcomes of TeleSpA trial.
For secondary health and care experience outcomes, PIFU/TM was hypothesized to be non-inferior to usual care. The dotted line represents the non-inferiority (NI) margin for each outcome: 0.9 (ASDAS), 2.0 (pain), 1.5 (patient global), 1.0 (physician global), -10% (good experience with care). PIFU/TM, patient-initiated follow-up combined with telemonitoring.

O22

TRABID: A NOVEL THERAPEUTIC TARGET FOR AXIAL SPONDYLOARTHRITIS

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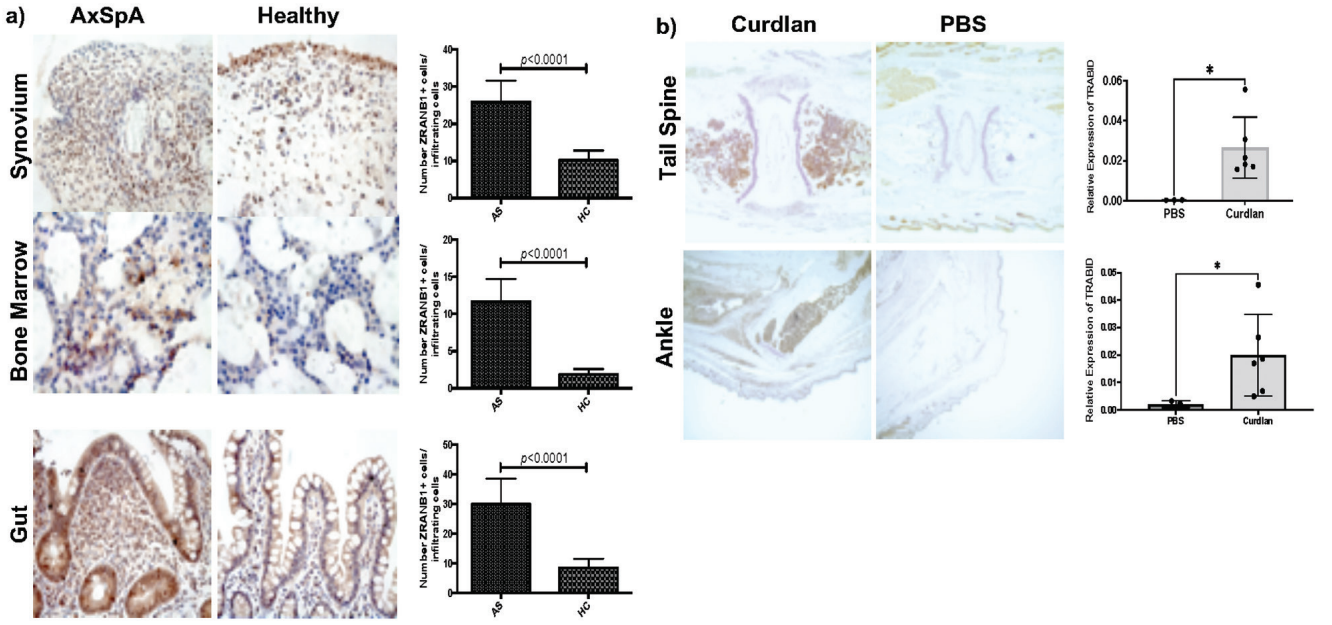
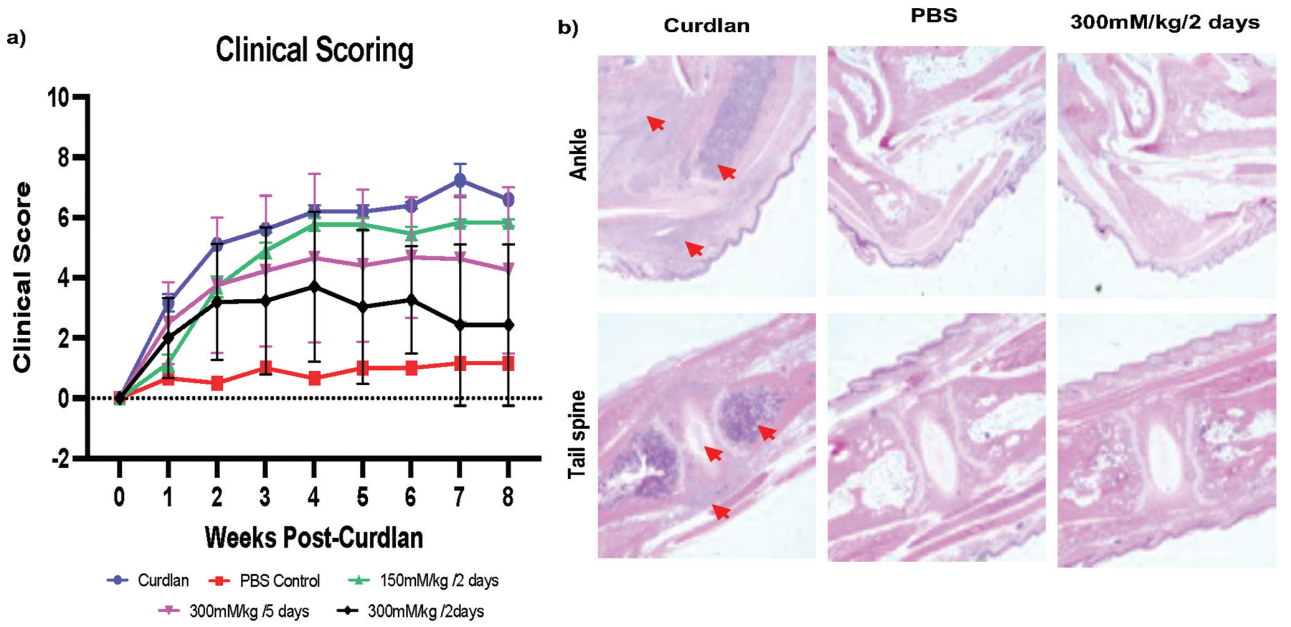
Introduction. Axial Spondyloarthritis (AxSpA) is characterized by both inflammation and new bone formation. There is no cure, and current treatments do not adequately address both inflammation and spinal fusion. The deubiquitinase TRABID has been shown to epigenetically control IL12/23 expression. Furthermore, TRABID also upregulates EZH2 (Enhancer of zeste homolog 2), a molecule implicated in pro-osteogenic pathways. Therefore, we studied the role of TRABID in AxSpA pathogenesis and its suitability as a therapeutic target.
Methods. TRABID expression in human and mouse AxSpA inflamed tissues were analyzed by immunohistochemistry. To assess the effect of TRABID inhibition on cytokine production, THP-1 human monocytes and SKG mouse splenocytes were incubated with lipopolysaccharide (LPS) and NSC112200, a specific TRABID inhibitor. ELISA detected cytokine production after 24 hours. Next, TRABID was knocked down in Saos-2 osteoblasts using siRNAs and the effect on osteogenesis was assessed by evaluating osteogenic gene expression by bulk RNA sequencing and qPCR. Calcium mineralization was evaluated by Alizarin Red staining. To evaluate the clinical relevance of TRABID inhibition *in vivo*, curdlan injected SKG mice were treated for 8 weeks with NSC112200.
Results. Compared to controls, TRABID was significantly upregulated in human AxSpA spine, gut, synovium, and bone marrow and in diseased SKG mouse ankle and tail (Fig. 1). TRABID inhibition by NSC112200 in THP-1 cells and mouse splenocytes resulted in a dose dependent inhibition of TNFa, IL1b and IL23 production. Following TRABID knockdown, Saos-2 osteoblast cells failed to mineralize and the expression of pro-osteogenic genes SP7, Runx2 and alkaline phosphatase were significantly suppressed. RNA sequencing confirmed pathways related to TGFb signaling, and endochondral ossification were suppressed. Finally, NSC112200 dose dependently suppressed clinical and histopathological signs of inflammation in curdlan injected SKG mice (Fig. 2).
Conclusion. TRABID inhibition suppressed both inflammation and bone formation and is a potential therapeutic target for AxSpA.



O22: Fig. 1 a) Representative immunohistochemistry staining and quantification of AxSpA (n=20) and healthy (n=10) synovium, bone marrow, and gut. **b**) Representative immunohistochemistry staining and quantification of 16-week-old SKG mice 8 weeks after curdlan induced disease (n=6) or PBS (n=3).



O22: Fig. 2 a) Clinical scoring of arthritis features (psoriasis of tail/ears, paw swelling, blepharitis etc.) weekly for 8 weeks post curdlan injection with 3 doses of NSC112200 (green line = low dose, pink line = medium dose, black line = high dose). PBS controls were not given curdlan. n=3 mice/group b) Representative histopathological scoring of SKG mouse ankle and tail spine 8 weeks post curdlan, PBS or curdlan and NSC112200 injection. 300mM/kg/2 days is representative of the highest dose (black line in Fig. 2a). Red arrows point to areas of immune infiltrate.

O23

TARGETING OSTEOCLAST-DERIVED DPP4 ALLEVIATES INFLAMMATION-MEDIATED ECTOPIC BONE FORMATION IN ANKYLOSING SPONDYLITIS

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Introduction. Ankylosing spondylitis (AS) is a chronic inflammatory disease characterized by ectopic bone formation. The anti-inflammatory function of dipeptidyl peptidase-4 (DPP4) inhibitor has been reported in bone metabolism, but its utility in AS has not previously been investigated.
Methods. We assessed DPP4 level in serum, synovial fluid, and facet joint tissue of AS patients. Additionally, we investigated the effect of a DPP4 inhibitor in curdlan-injected SKG mice. Following curdlan injection, SKG mice were orally administered a DPP4 inhibitor three times per week for 5 weeks, and ankles of mice were scored for thickness and given clinical arthritis scores. At the end of 5 weeks, mice were sacrificed, and micro-CT and histological analyses were performed. Furthermore, osteoclast precursor cells from curdlan-injected SKG mice were treated with DPP4 inhibitor, and the effects of this treatment on osteoclastogenesis and differentiation markers were evaluated.
Results. Soluble DPP4 level was elevated in the serum and synovial fluid of patients with AS compared to those in the control group. DPP4 increased gradually during human osteoclastogenesis and was high in mature osteoclasts. Histological analysis revealed that DPP4 inhibitor resulted in a decrease in thickness of the hind paw, arthritis, and enthesitis at the ankle in curdlan-injected SKG mice compared to the control group. Micro-CT data showed a significant reduction in inflammation-mediated ectopic bone formation in the DPP4 inhibitor group compared to those in the control group. Intriguingly, DPP4 co-expressed in TRAP-positive osteoclasts was detected in ectopic bone in the tibia of curdlan-injected SKG mice. Moreover, treatment with a DPP4 inhibitor significantly reduced osteoclastogenesis in the bone marrow of curdlan-injected SKG mice in addition to decreasing expression of osteoclast differentiation markers.
Conclusion. Our findings suggest that inhibiting DPP4 may have a therapeutic effect on excessive bone formation in AS patients.

O24

DISEASE ACTIVITY OUTCOMES WITH THE IL-17A- AND IL-17F-INHIBITING NANOBODY SONELOKIMAB IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS (PSA): WEEK 12 RESULTS FROM THE PHASE 2 ARGO TRIAL

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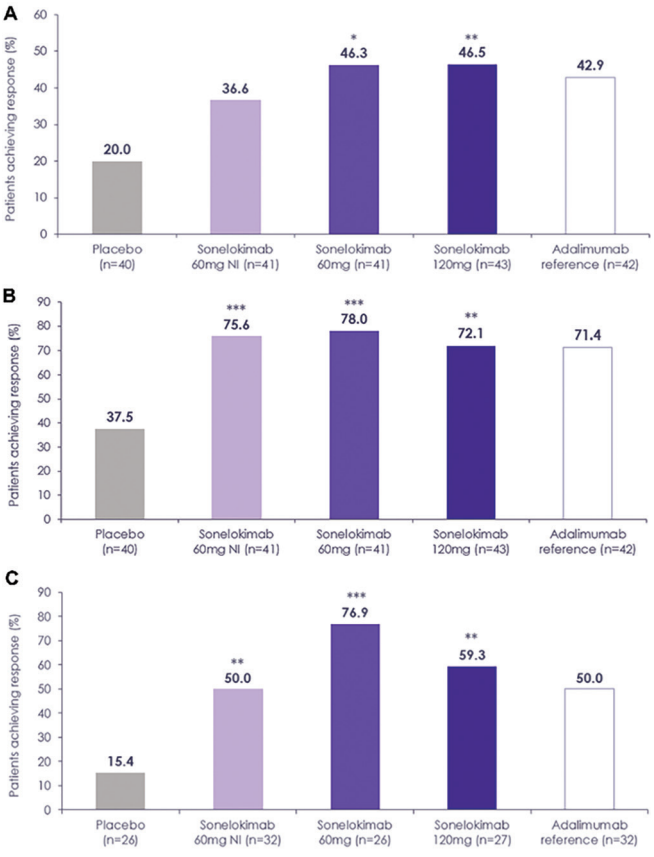
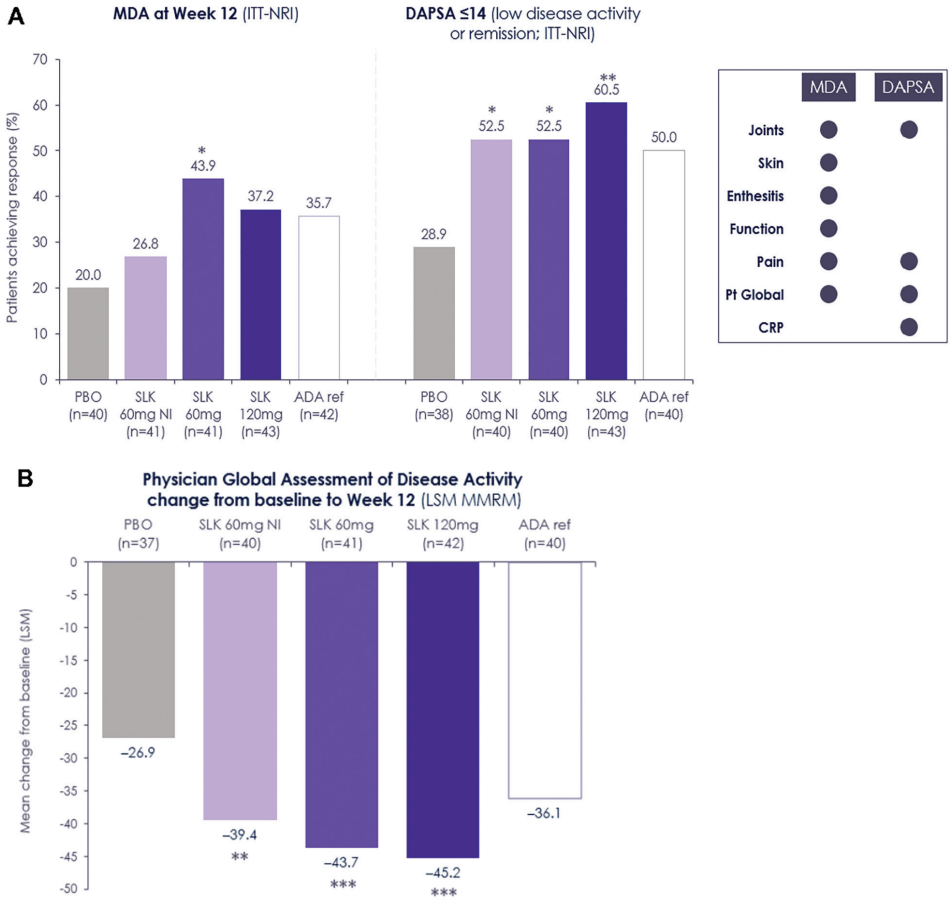
Introduction/Objective. Nanobodies represent a new generation of antibody-derived therapies, with a small size designed to enhance tissue penetration. Sonelokimab is a ~40kDa humanized Nanobody that inhibits IL-17A and IL-17F (central drivers of psoriatic disease), with an albumin-binding domain that may enhance targeting to sites of inflammation. We describe Week 12 disease activity outcomes from the Phase 2 ARGO trial of sonelokimab in PsA.
Materials and methods. ARGO was a 24-week global, randomized, prospective, double-blind trial (NCT05640245). Patients were ≥18 years with active PsA (TJC68 ≥3, SJC66 ≥3) and active (and/or dermatologist-confirmed diagnosis of) psoriasis. Patients were randomized (1:1:1:1:1) to sonelokimab 120mg, 60mg, 60mg without induction (NI), placebo, or adalimumab (reference arm, not powered for statistical comparison), and stratified by sex and prior biologic use. Primary endpoint: ACR50 at Week 12. Key secondary endpoints: ACR20 and PASI90 at Week 12. Other endpoints included ACR70, PASI100, MDA, low disease activity (DAPSA ≤14), and Physician Global Assessment of Disease Activity (PhGADA). Primary analysis: ITT-NRI.
Results. 207 patients were randomized, with well-balanced baseline characteristics and a discontinuation rate of <4% overall. Primary and all key secondary endpoints were met (Fig. 1). At Week 12, sonelokimab demonstrated a positive impact on multidomain disease activity scores, including MDA and DAPSA low disease activity (Fig. 2A). In addition, improvement from baseline in PhGADA was greater with sonelokimab than with placebo (Fig. 2B). Sonelokimab was well tolerated with no unexpected safety findings (no reported IBD or MACE; two [1.6%] mild or moderate cases of oral candidiasis).
Discussion. Robust levels of clinical response were observed with sonelokimab, including in measures of multidomain or global disease activity. The benefit-risk profile was favourable.
Conclusion. Week 12 outcomes from the Phase 2 ARGO trial support further investigation of sonelokimab as a potential novel therapy for patients with active PsA.



O24: Fig. 1. ACR50 (A), ACR20 (B), and PASI90† (C) response rates (ITT-NRI) at Week 12.
***p<0.001, **p<0.01, *p<0.05. p-values for sonelokimab 60 mg NI are nominal. †Percentages are based on the number of patients at each visit in the full analysis set in each treatment group with baseline psoriasis involving ≥3% body surface area at baseline. p-values based on a logistic regression model including fixed effects for treatment and stratification factors (sex and prior biologic exposure). Analysis in the ITT population, with missing data imputed as a non-response.
ACR: American College of Rheumatology; ITT: intention-to-treat; NI: no induction; NRI: non-responder imputation; PASI: Psoriasis Area and Severity Index.



O24: Fig. 2. **A**. Minimal Disease Activity (MDA) and Low Disease Activity (DAPSA ≤14),† and **B**. PhGADA improvement from baseline (LSM MMRM), at Week 12.
***p<0.001, **p<0.01, *p<0.05. p-values are nominal. †Percentages are based on the number of patients in the full analysis set in each treatment group with baseline DAPSA>14. p-values for MDA and DAPSA based on a logistic regression model including fixed effects for treatment and stratification factors (sex and prior biologic exposure); MDA and DAPSA analyzed for the ITT population, with missing data imputed as a non-response. p-values and LSM for PhGADA analyzed using an MMRM model, including treatment, baseline, visit, stratification factors (sex and prior biologic exposure), and treatment by visit interaction as fixed effects, in the ITT population.
ADA ref: adalimumab reference arm; CRP: C-reactive protein; DAPSA: Disease Activity Index for PsA; ITT: intention-to-treat; LSM: least squares mean; MDA: minimal disease activity; MMRM: mixed model for repeated measures; NI: no induction; NRI: non-responder imputation; PBO: placebo; PhGADA: Physician Global Assessment of Disease Activity; Pt Global: Patient Global Assessment; SLK: sonelokimab.

Poster Presentations

P1

IL 33 AND SECUKINUMAB, A LINK IN RHEUMATIC-RELATED GUT ALTERATION

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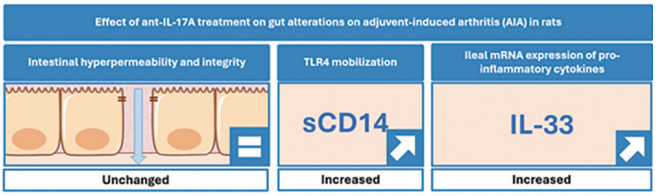
Introduction. Subclinical gut inflammation, intestinal permeability and bacterial translocation are well described in inflammatory rheumatic diseases. While IL-17A is involved in the pathophysiology of both arthritis and inflammatory bowel diseases, the role of this cytokine in intestinal alterations related to rheumatic diseases has never been explored[1]. This study aims to investigate the intestinal effects of an anti-IL-17A therapy (secukinumab) administered during the preclinical phase of adjuvant-induced arthritis (AIA) in rats, a model already known to present early intestinal alterations[2].
Methods. AIA was induced by injection of Mycobacterium butyricum in Freund's incomplete adjuvant at the base of the tail in male Lewis rats (day 0). AIA rats were treated, or not, with secukinumab (20mg/kg/3days, i.p.) from day 0 to day 4 (pre-clinical phase) or to day 11 (arthritis onset). Body weights and arthritis scores were monitored daily. Intestinal inflammation was assessed by measuring mRNA expression of proinflammatory cytokines (RTqPCR). Intestinal permeability and integrity were evaluated by the measure of plasma zonulin and iFABP levels (ELISA). TLR4 mobilization was estimated by serum sCD14 levels (ELISA).
Results. As compared to untreated AIA, secukinumab did not change body weights and arthritis scores whatever the treatment duration. Plasma levels of zonulin and iFABP were unchanged by the treatment, both at day 4 and day 11. By contrast, higher serum levels of sCD14 were measured at days 4 and 11 under secukinumab treatment. Likewise, the treatment with secukinumab increased ileal mRNA expression of IL-33 but not IL-1β, IL-8, IL-17A, IL-23p19 and TNF-α at days 4 and 11.
Conclusion. Blockade of IL-17A, during the preclinical phase of the AIA model, did not change intestinal permeability and epithelial damage but increased potential TLR4 mobilization and IL-33 gene expression. These data suggest a role of IL-17A in rheumatic-related intestinal changes and encourage further exploration of the IL17A/IL-33 pathway in this setting.



P1: Fig. 1.

Acknowledgement. Financial support of French Society for Rheumatology (SFR).

References

1. MEYER F, WENDLING D, DEMOUGEOT C, PRATI C, VERHOEVEN F: Cytokines and intestinal epithelial permeability: A systematic review. *Autoimmun Rev* 2023; 22(6): 103331.
2. HECQUET S, TOTOSON P, MARTIN H *et al*.: Increased gut permeability and intestinal inflammation precede arthritis onset in the adjuvant-induced model of arthritis. *Arthritis Res Ther* 2023; 25(1): 95.

P2

SWIMMING EXERCISE ALLEVIATES PATHOLOGICAL BONE FEATURES IN CURDLAN-INJECTED SKG MICE BY INDUCING IRISIN EXPRESSION

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Introduction. While exercise is strongly recommended as an adjunct therapy for patients with Ankylosing Spondylitis (AS) patients with medication, the knowledge about the inflammatory burden of AS by exercise remains adequately elucidated. Irisin, mainly produced by muscle, is responsible for beneficial effects of exercise training. This study aims to assess the therapeutic potential of swimming exercise in the curdlan-injected SKG mice model and investigate the modulatory effects of irisin on inflammation.
Methods. Curdlan-injected SKG were randomly assigned to either a home-cage group or a swimming group for 6 weeks. The swimming protocol involved a period of habituation followed by training session for one week, with subsequent daily sessions conducted five days a week for 5 weeks. Clinical arthritis scores and ankle thickness were measured weekly. Post-swimming program, mice were anesthetized for collection of femoral muscle and blood, which was followed by histological analysis, micro-CT imaging of the ankle joints, and the measurement of pro-inflammatory cytokines and irisin levels. Additionally, curdlan-injected SKG mice were intravenously injected with recombinant irisin protein and observed. Finally, serum levels of irisin in healthy control and AS patient groups were measured.
Results. The swimming group exhibited significant reduction in arthritis and enthesitis. Micro-CT and histological analyses revealed a notable reduction in pathological bone features in the swimming group. Muscle strength was also enhanced in the swimming group, as determined by the wire-hanging test. Intriguingly, irisin levels not only were statistically increased in the swimming group but, also, pro-inflammatory cytokines were decreased. Additionally, injection of irisin protein attenuated both arthritis and enthesitis in curdlan-injected SKG mice. Meanwhile, irisin serum levels were declined in AS patients.
Conclusion. We found that swimming exercise attenuated pathological bone features in an AS animal model, potentially mediated by increased irisin serum levels with associated anti-inflammatory effects.

P3

DECIPHERING THE MOLECULAR LANDSCAPE IN THE PRECLINICAL PHASE OF DISEASE IN AXSPA

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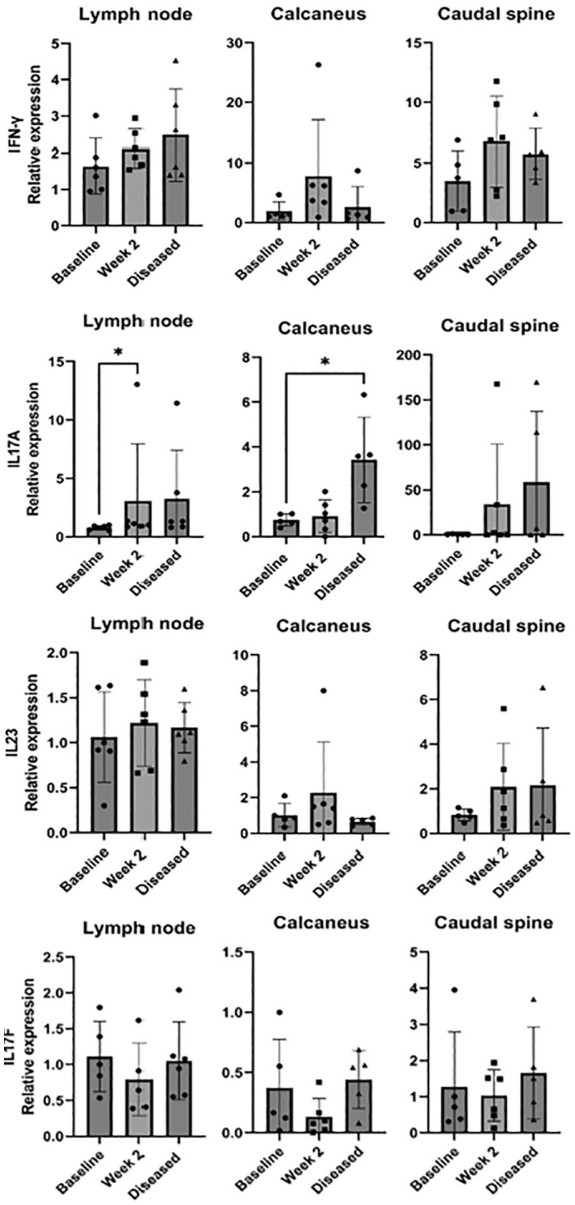
Introduction. In axSpA it is proposed that onset of disease is preceded by a preclinical/at risk phase and different immune pathways are active in different phases of the disease[1]. This study aims to unravel immune pathways activated in the preclinical phase of axSpA in the HLA-B27 tg model.
Methods. In the HLA-B27 tg model inflammation and structural damage in the axial and peripheral joints were assessed with histology and microCT in three groups sacrificed either at baseline, two weeks after immunization, and at the onset of clinical inflammation. Across all timepoints, gene expression analysis through qPCR (popliteal lymph nodes (PLN), caudal spine, and calcaneus) and RNA sequencing analysis (PLN) were conducted.
Results. In clinically symptomatic animals, histological inflammation [axial: mean 1.6(SD 1.5); peripheral: 2.1(1.3)], destruction [1.3(1.5);2(1.5)], new bone formation [0.5(0.5);0.6(0.5)], and hypertrophic chondrocytes [0.5(0.5);0.6(0.5)] was observed. Histological examination revealed no subclinical inflamma-

tion or structural damage before symptom onset in axial or peripheral joints. In the PLN, caudal spine and the calcaneus, a numerical increase in mRNA expression of various cytokines including IFN-γ, IL17A, IL23 and IL17F was observed in the preclinical and diseased phases (Fig. 1). PLN demonstrated differential expression of 359 genes in the preclinical phase versus baseline and 569 genes in diseased versus baseline. In week 2 and diseased timepoint versus baseline, gene sets associated with T cell receptor signaling pathways, and pathways involved in regulating the antigen receptor mediated signaling were upregulated. In diseased versus week 2, gene sets associated with innate immunity such as neutrophil meditated immunity were upregulated.

Conclusion. This study unveils molecular changes in axial and peripheral joints and PLN before clinical symptoms manifest in the HLA-B27 rat model. Further investigations are warranted to identify specific cell types driving these early molecular changes and to explore whether targeting these alterations can prevent symptom onset.

Funding. This study received financial support from Novartis via an unrestricted grant. Novartis had no role in the study design, collection, analysis, or interpretation of the data. MS received funding from a ZonMw VENI project (09150161810112) and ReumaNederland. LB and MS received funding from the European Union's Horizon 2020 research and innovation programme under the Marie Skłodowska-Curie grant agreement no. 847551 (ARCAID).



P4: Fig. 1. Gene expression level of pro inflammatory cytokines in different tissue sites. A relative expression level (in relation to GAPDH) of IFNγ, IL17A, IL23 and IL17F in the popliteal lymph nodes, calcaneus, and caudal spine. (data are mean ± SD, * $p \leq 0.05$).

Reference

1. VAN TOK MN, NA S, LAO CR *et al*.: The initiation, but not the persistence, of experimental spondyloarthritis is dependent on interleukin-23 signaling. *Front Immunol* 2018; 9: 1550.

P4

TOFACITINIB REDUCES LPS LEVELS IN RATS WITH ADJUVANT-INDUCED ARTHRITIS

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Introduction. Growing evidence indicated that the intestine is not only a target but also an actor of the pathogenesis in chronic inflammatory rheumatic diseases (CIRD). Consistently, increased intestinal permeability (IP) and damage (ID) as well as bacterial translocation (BT) have been described in patients with CIRD. However, the effects of treatments used in patients with CIRD on gut health are unknown. The objective of this study was to determine the effect of glucocorticoids (GCs) and tofacitinib on BT in rats with adjuvant-induced arthritis (AIA).

Methods. AIA was induced in 6-week-old male Lewis rats by a subcutaneous injection at the basis of the tail of *Mycobacterium butyricum* in incomplete Freund's adjuvant. At onset of arthritis, rats were treated daily with prednisolone at low (0.1 mg/kg/day, i.p.) or high dose (10 mg/kg/day, i.p.), or with tofacitinib (10 mg/kg twice a day, s.c.) or with vehicle. After 21 days, IP, ID BT were assessed by measuring zonulin, intestinal Fatty Acid Binding Protein (iFABP) and soluble CD14 (sCD14) and different forms of LPS (esterified LPS with different acyl chain lengths determined by high performance liquid chromatography coupled to mass spectrometry). Arthritis severity was daily evaluated through the determination of an arthritis score.

Results. Compared to vehicle, tofacitinib and high-dose of GC (HD-GC) both reduced arthritis score ($p<0.001$) and levels of sCD14 (-44%, $p<0.001$ and -41% $p<0.001$ respectively) in AIA. High dose of GC decreased iFABP levels (-17%, $p<0.05$) but had no effect on zonulin or esterified LPS levels. Tofacitinib did not change either iFABP or zonulin levels but reduced esterified 3OH C10 LPS (-47%, $p=0.03$) and esterified 3OH C18 LPS (-26%, $p=0.02$). Tofacitinib did not change the levels of other forms of LPS. As compared to vehicle, the low-dose of GC had no effect on arthritis severity, sCD14, iFABP, zonulin and LPS plasma levels.

Discussion/Conclusion. Prednisolone at a dose efficient on arthritis and Tofacitinib did not aggravate but on the contrary reduced intestinal bacterial translocation, with no effect on intestinal permeability. The interesting aspect of this study is the selective modification of the different forms of LPS. The length of the acyl chains in part A of the lipid defines the biological activity and pro-inflammatory potential of the molecule when exposed to the human immune system. This suggests a specific anti-inflammatory effect of tofacitnib. Given the suspected pathophysiological link between bacterial translocation and arthritis, our results identified a new mechanism involved in the positive effects of tofacitinib in arthritis diseases.

P5

NEW INSIGHTS ON THE PATHOPHYSIOLOGY OF BONE DISEASE IN AXIAL SPONDYLOARTHRITIS

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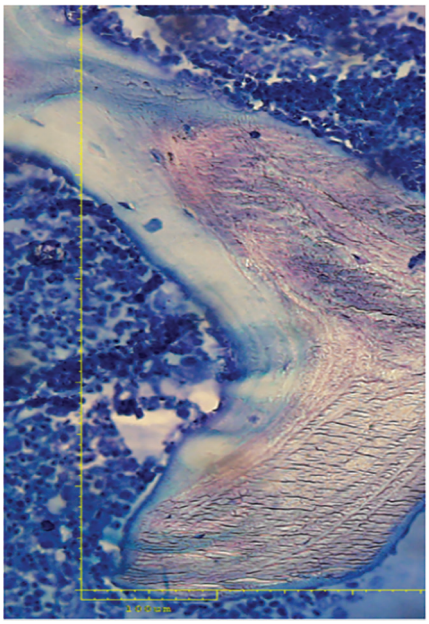
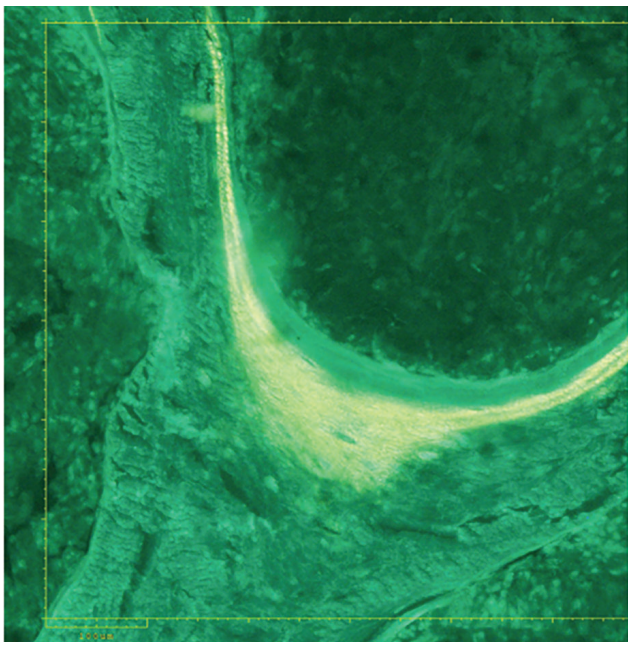
Introduction. Bone fragility and osteoproliferation findings are shared in patients with axial spondyloarthritis (SpA), suggesting a complex pathophysiology. The objectives of this study were to evaluate the histomorphometric data and imaging parameters in axial SpA patients.
Methods. Male axial SpA patients up to 55 years old, without secondary causes of osteoporosis, were enrolled in this cross-sectional study. Clinical, lab, and imaging data, including spine x-ray, as well as dual-energy x-ray absorptiometry (DXA) at spine, hip, and forearm and trabecular bone score (TBS), were performed in all patients. Transiliac histomorphometry was also underwent in all patients, and data were compared with a control group of male healthy cadavers (n=21) and with reference values.
Results. A total of 21 patients were included, with a mean age of 45.8 years, long disease duration (median 17.5 years), mostly white (66.7%) and HLA-B27 positives (90.5%). The prevalence of DXA abnormalities and low TBS (≤1.338) was 42.8% and 57.1%, respectively. One patient reported a low-impact non-vertebral fracture in the humerus, while none exhibited morphometric or clinical vertebral fractures. There was higher osteoid trabecular thickness (p=0.027, Fig. 1) and cortical bone changes, including reduced thickness (p=0.031) and increased porosity (p=0.015) in axial SpA patients. In addition, a pattern of cortical trabecularization was observed in 52.3%. Dynamic evaluation revealed a longer mineralization lag time (p=0.0074, Fig. 2) and lower mineralized surface (p=0.0029) and bone formation rate (p=0.0074) in patients compared to reference values. There was a significant positive association between serum inorganic phosphorus levels and mineralized surface after adjustment to age, body mass index, and vitamin D levels (R^2adj=0.548; p=0.009).
Conclusion. Our results showed a pattern of low trabecular remodeling, bone mineralization impairment, possibly associated with phosphorus metabolism, as well as cortical thickness and porosity abnormalities in young men with axial SpA. These findings may impact future treatments.



P5: Fig. 1.



P5: Fig. 2.

P6

INTERLEUKIN-22 INDUCED BY LPS AGGRAVATES BONE LOSS WITH EXCESSIVE OSTEOCLAST ACTIVITY BY INCREASING RANKL PRODUCTION FROM OSTEOBLAST

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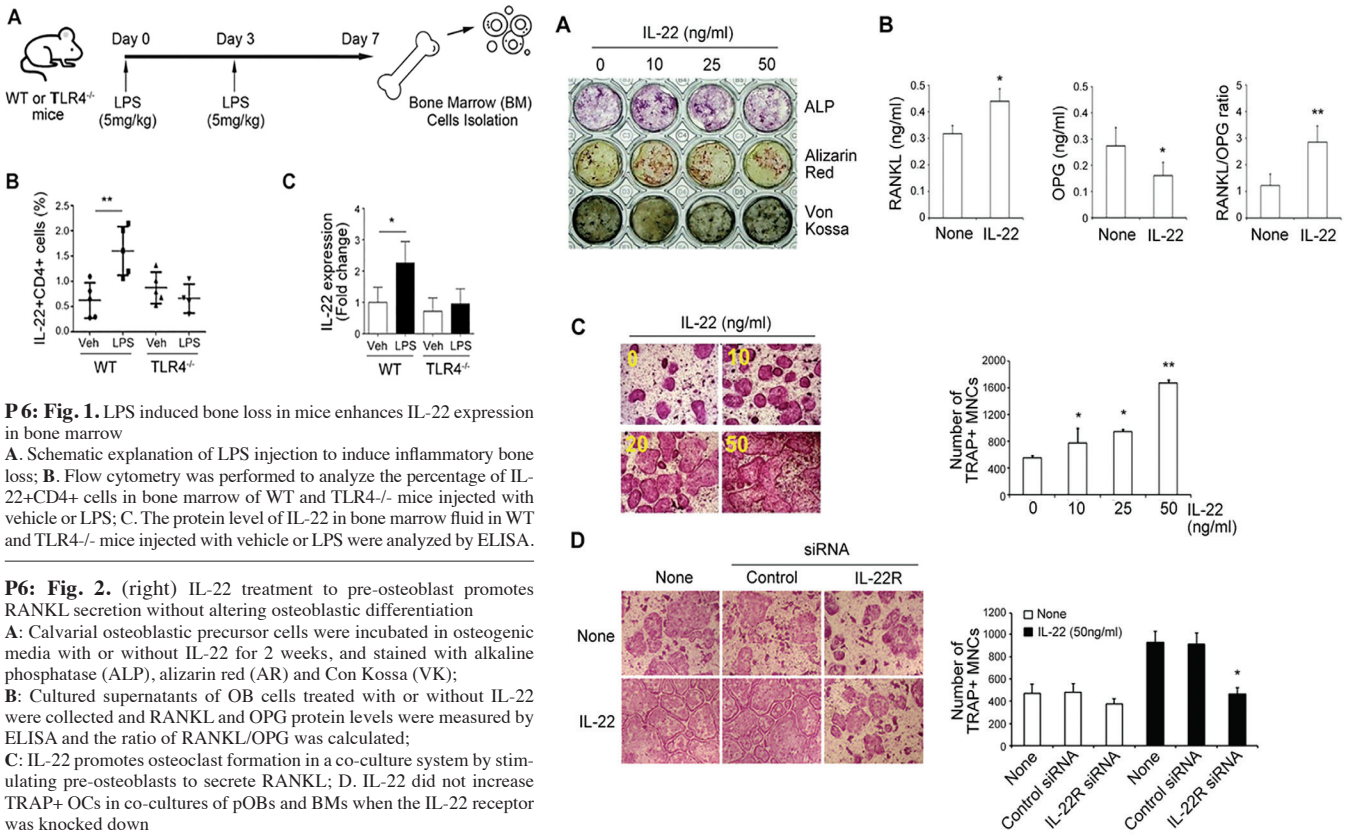
**These authors contributed equally to this study.*

Introduction. Interleukin-22 (IL-22) is a cytokine involved in both protective and pathological roles in inflammatory diseases. In rheumatoid arthritis patients, IL-22 upregulates RANKL production in synovial fibroblasts, inducing osteoclastogenesis. However, its role in bone remodeling through osteoblast (OB) and osteoclast (OC) regulation under inflammatory conditions is unclear. Therefore, this study aims to elucidate the role of IL-22 in inflammatory bone loss by modulating osteoclastogenesis.
Methods. Wild-type (WT) and TLR4-/- mice injected with lipopolysaccharide (LPS) were used to analyze IL-22 protein expression in bone marrow (Fig. 1A). Primary mouse OB precursor cells (pOBs) from calvariae were transfected with control or IL-22 receptor-specific siRNA and co-cultured with bone marrow cells (BMs) to evaluate osteoclastogenesis with IL-22 treatment. ELISA was used to analyze RANKL and OPG expressions in cultured supernatants. Micro-CT analysis assessed bone loss in WT and IL-22-/- mice injected with LPS.
Results. LPS injection in mice increased IL-22+CD4+ cells in BMs and elevated IL-22 expression (Fig. 1B-C). IL-22 treatment of pOBs upregulated RANKL secretion and reduced OPG expression (Fig. 2A-B). IL-22 increased TRAP+ OCs in co-cultures of pOBs and BMs, but not in IL-22 receptor knockdown pOBs and WT BMs (Fig. 2C-D). LPS stimulated TRAP+ OC formation in co-cultures from WT mice, but not in IL-22-/- mice. LPS-induced increases in RANKL and decreases in OPG secretion were absent in co-cultures with BMs from IL-22-/- mice. IL-22 induced by LPS stimulated OC differentiation, leading to greater bone loss in WT compared to IL-22-/- mice.
Conclusion. Enhanced IL-22 expression under inflammatory conditions contributes to uncoordinated bone loss, providing IL-22 as a potential target to ameliorate inflammation-driven bone destruction.



P 6: Fig. 1. LPS induced bone loss in mice enhances IL-22 expression in bone marrow
A. Schematic explanation of LPS injection to induce inflammatory bone loss; **B**. Flow cytometry was performed to analyze the percentage of IL-22+CD4+ cells in bone marrow of WT and TLR4-/- mice injected with vehicle or LPS; C. The protein level of IL-22 in bone marrow fluid in WT and TLR4-/- mice injected with vehicle or LPS were analyzed by ELISA.

P6: Fig. 2. (right) IL-22 treatment to pre-osteoblast promotes RANKL secretion without altering osteoblastic differentiation
A: Calvarial osteoblastic precursor cells were incubated in osteogenic media with or without IL-22 for 2 weeks, and stained with alkaline phosphatase (ALP), alizarin red (AR) and Con Kossa (VK);
B: Cultured supernatants of OB cells treated with or without IL-22 were collected and RANKL and OPG protein levels were measured by ELISA and the ratio of RANKL/OPG was calculated;
C: IL-22 promotes osteoclast formation in a co-culture system by stimulating pre-osteoblasts to secrete RANKL; D. IL-22 did not increase TRAP+ OCs in co-cultures of pOBs and BMs when the IL-22 receptor was knocked down

P7

SPA DISK©: A NEW TOOL TO MONITOR THE QOL OF SPONDYLOARTHRITIS PATIENTS

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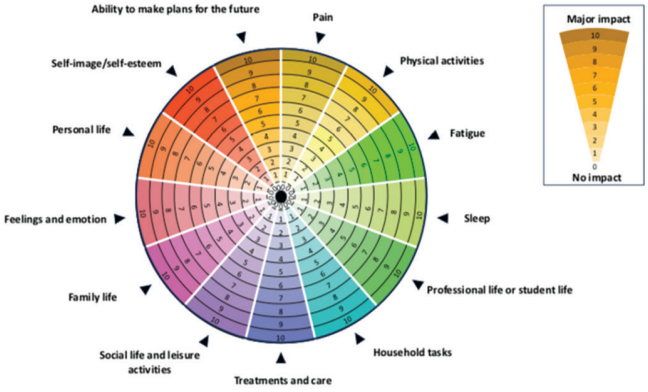
Background. Despite the high satisfaction rate regarding the efficacy of spondyloarthritis (SpA) treatment on clinical symptoms and function during the biologic era, physicians often overlook other aspects of daily life that are greatly affected by SpA. While there is an increased focus on research, the systematic utilization of Quality of Life (QoL) instruments in clinical practice remains limited.
Objective. To address this, we developed SpA Disk©, a simple yet thorough tool that addresses the impact of SpA on the most relevant aspects of daily life.
Methods. SpA Disk© was developed through a comprehensive and rigorous process, including an extensive literature review of online databases containing existing Patient-Reported Outcomes (PROs) and QoL questionnaires. Surveys were then distributed to patients through patient associations, aiming for active patient participation in ranking/rating proposed items based on their perceived impact on QoL. The preliminary version of the questionnaire was created by a SpA Disk© group comprising 30 expert rheumatologists in SpA, three of whom were therapeutic education specialists and one sexologist. The validation process by experts in spondyloarthritis led to the final version of SpA Disk© in French, which was subsequently translated into English.
Results. Following five rounds of voting, the final SpA Disk© comprises 13 items presented in the form of a scoring disk-shaped numerical scale ranging from 0 (absolutely disagree/No impact) to 10 (absolutely agree/major impact) over a 30-day time period. The questionnaire includes an explanatory statement and keywords specifying the application field of each item (Fig. 1).
Conclusion. SpA Disk© is an easily administered, concise, comprehensive, and self-reported tool encompassing all crucial aspects of SpA-related disability in patients. It offers a visual representation of this burden for physicians during patient visits and in the outpatient setting. A digital version of the SpA Disk© is currently in development.
Acknowledgement. We gratefully acknowledge the efforts of all the members of the SpA disk group who played an important role in the whole SpA Disk© project. We thank the RMD-patient associations that have allowed us to reach the participating RMD patients through our online surveys. We thank all patients for their selfless participation in the development of this tool. We thank Abbvie for their logistical help in the organization of the SpA Disk© scientific day in October 2022.



P7: Fig. 1. The SpA Disk©

P8

A TWO-YEAR COMPARISON OF BACK PAIN AND MORNING STIFFNESS BETWEEN AXIAL SPONDYLOARTHRITIS AND CHRONIC BACK PAIN PATIENTS IN THE SPONDYLOARTHRITIS CAUGHT EARLY (SPACE) COHORT

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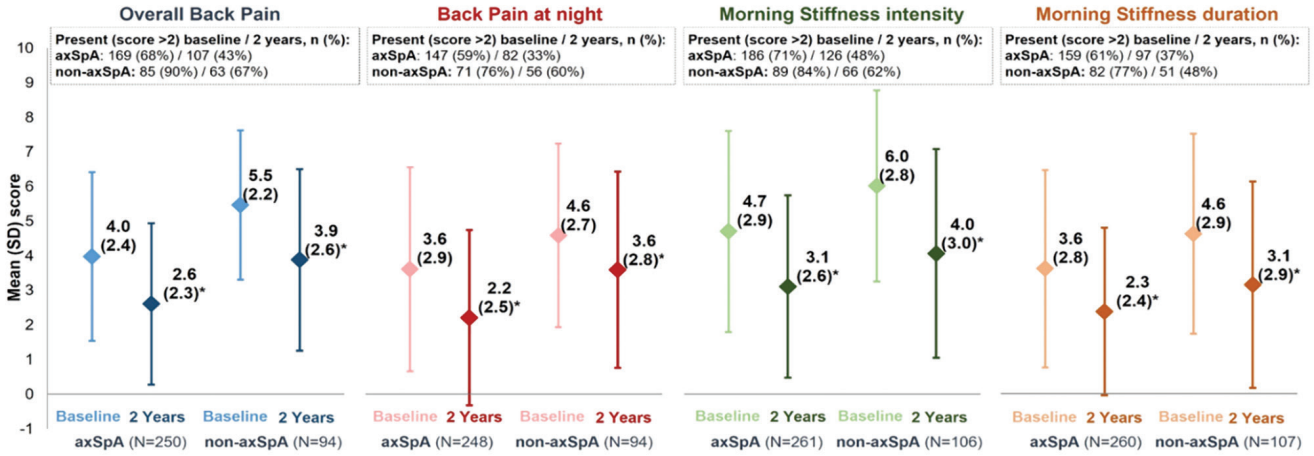
Introduction/Objective. Treatment of axial spondyloarthritis (axSpA) improves spinal symptoms, namely back pain (BP) and morning stiffness (MS). However, little is known on the disease course of early axSpA, particularly in comparison to non-axSpA chronic BP (CBP) patients. To compare spinal symptoms at baseline and 2 years (2y) between early axSpA and non-axSpA patients.

Methods. Baseline and 2y data of the SPACE cohort (adults with CBP lasting ≥3 months and ≤2y, starting <45 years) were analyzed. Overall BP, BP at night, MS intensity and MS duration were evaluated on a numeric rating scale: 0 (no symptoms / 0 hours) to 10 (unbearable symptoms / ≥2 hours). Paired t-test was used to compare baseline and 2y results within groups. Linear regression was conducted to compare 2y outcomes between groups, adjusting for the baseline value, gender, age and NSAIDs use.

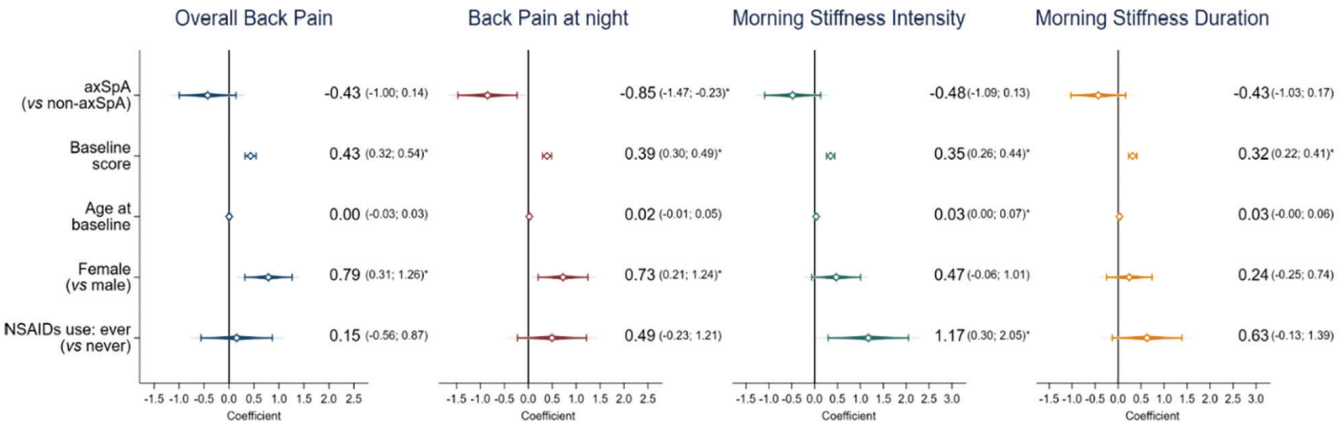
Results. Data was available at both timepoints on ≥1 outcome in 267 axSpA and 110 non-axSpA patients (males: 52% *vs* 25%; mean [SD] SpA features: 5 [2] *vs* 3 [1]; mean [SD] age: 29 [8] *vs* 31 [8] years). At baseline, spinal symptoms were worse in non-axSpA (*vs* axSpA) patients (Fig. 1). After 2y, all symptoms significantly improved in both groups (mean [SD] improvement axSpA *vs* non-axSpA: overall BP 1.4 [2.5] *vs* 1.6 [2.4]; BP at night 1.4 [2.9] *vs* 1.0 [2.7]; MS intensity 1.6 [3.2] *vs* 1.9 [2.9]; MS duration 1.2 [3.0] *vs* 1.5 [3.1]). However, substantial symptoms persisted (more in non-axSpA). In multivariable analysis, at 2y, axSpA was only associated with larger improvements in BP at night (Fig. 2, β [95% CI]: -0.85 [-1.47; -0.23]).

Conclusion. Non-axSpA patients experience worse spinal symptoms. After 2 years, BP and MS significantly improve in both groups, but axSpA is an independent predictor of a larger improvement in BP at night.

Acknowledgements. ASAS Fellowship.



P8: Fig. 1. Spinal symploms at baseline and 2 years in axSpA and no-axSpA patients. *Comparison of outcomes at baseline and 2 years within groups p-value <005.



P8: Fig. 2. Multivariable linear regression analysis of to-years back pain and morning stiffness inj chronic back pain paients in the SPACE cohort. *p-value <0.05.

P9

A TWO-YEAR COMPARISON OF SPINAL AND HIP MOBILITY BETWEEN AXIAL SPONDYLOARTHRITIS AND CHRONIC BACK PAIN PATIENTS IN THE SPONDYLOARTHRITIS CAUGHT EARLY (SPACE) COHORT

Bento da Silva A.[1,2,3], Ramiro S.[1,4], van Lunteren M.[1], Marques M.L.[1,5], Fongen C.[6], van de Sande M.[7], Ramonda R.[8], Exarchou S.[9], van der Heijde D.[1], van Gaalen F.[1]
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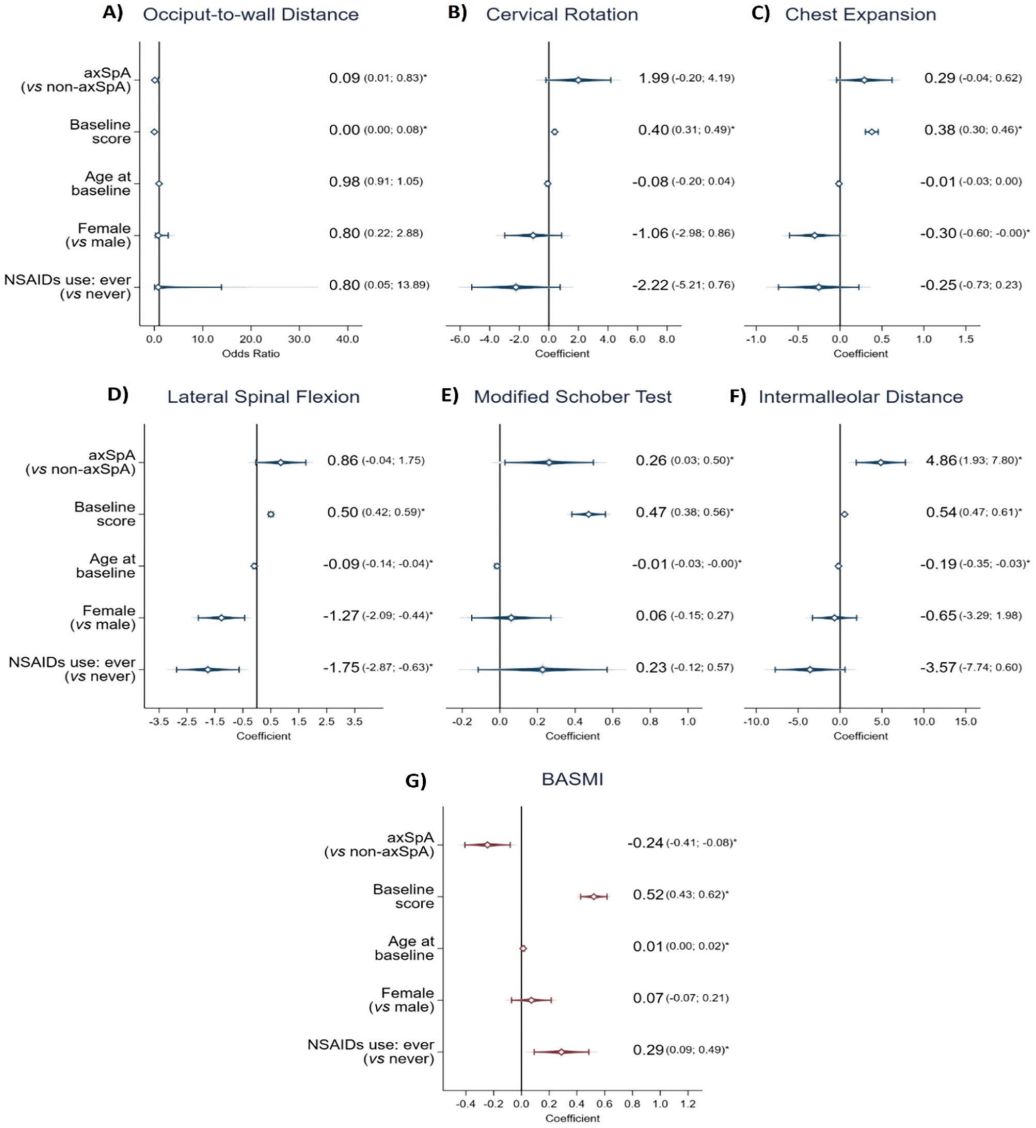
Introduction/Objective. To compare spinal/hip mobility at baseline and 2 years (2y) between early axSpA and non-axSpA chronic back pain (CBP) patients.
Methods. Baseline and 2y data of the SPACE cohort (CBP ≥3 months and ≤2y, starting <45 years) were analyzed. Assessed mobility measures: occiput-to-wall distance (OWD), cervical rotation (CR), chest expansion, lateral spinal flexion (LSF), modified Schober test (mSchober), maximal intermalleolar distance (IMD). BASMI was calculated. Definition of impairment: measure <2.5th percentile curve derived from healthy individuals (>97.5th for BASMI; >0cm for OWD). Paired t-test / Wilcoxon signed rank test was used to compare baseline and 2y results within groups. Linear or zero-inflated negative binomial regression was conducted to compare 2y outcomes between groups, adjusting for baseline value, gender, age and NSAIDs use.
Results. Data was available for 286 axSpA and 117 non-axSpA patients (males: 52% *vs* 25%; mean [SD] age 30 [8] *vs* 31 [8] years). At baseline, 51% axSpA and 53% non-axSpA patients had ≥1 impaired spinal mobility measure (Table I). Poorer mobility was observed in non-axSpA (*vs* axSpA), except for OWD. After 2y, CR and BASMI significantly improved in both groups (mean [SD] improvement axSpA *vs* non-axSpA: CR 2.4 [11.9] *vs* 2.8 [12.1]; BASMI 0.2 [0.8] *vs* 0.2 [0.7]); LSF and mSchober only in axSpA (LSF 1.1 [4.5]; mSchober 0.3 [1.2]). In multivariable analysis (Fig. 1), at 2y, axSpA (*vs* non-axSpA) was associated with larger improvements in mSchober (β [95% CI]: 0.26 [0.03; 0.50]), IMD (4.86 [1.93; 7.80]) and BASMI (-0.24 [-0.41; -0.08]), and with higher odds of OWD impairment (OR [95% CI]: 0.09 [0.01; 0.83]).
Conclusion. Impaired spinal/hip mobility is common in early axSpA and, particularly, non-axSpA. After 2y, mobility measures remain relatively unchanged. Nevertheless, axSpA is associated with larger improvements in mSchober, IMD and BASMI, and with higher odds of OWD impairment.
Acknowledgements. ASAS Fellowship.



P9: Fig. 1. Impact of axSpA *vs* non-axSpA on 2-years spinal and hip mobility measures. **A**. Multivariable zero-inflated negative binomial model for occiput-to-wall distance. Logit model represented as odds ratio (95% CI). **B** to **G**. Multivariable linear regression models for cervical rotation, chest expansion, lateralspinal flexion, modified Schober test, intermalleolar distance and BASMI. Results are presented in β (95% CI). *p<0.05

P9: Table I. Spinal and hip mobility measures at baseline and 2 years in axSpA and non-axSpA patients.

	AxSpA			Non-axSpA		
	Baseline	2 years	p-value (within group)	Baseline	2 years	p-value (within group)
Occiput-to-wall distance, mean (SD)	0.5 (1.2)	0.4 (1.1)	p=0.267	0.1 (0.7)	0.1 (0.5)	p=0.474
Impaired, n (%)	65 (23)	61 (22)		6 (5.1)	4 (3.4)	
Cervical rotation, mean (SD)	74.8 (12.0)	77.2 (10.6)	p=0.001*	70.7 (12.0)	73.5 (11.2)	p=0.015*
Impaired, n (%)	32 (11)	16 (5.6)		12 (10)	9 (7.7)	
Chest expansion, mean (SD)	5.9 (1.9)	6.0 (1.7)	p=0.274	5.3 (1.9)	5.4 (1.5)	p=0.365
Impaired, n (%)	21 (7.6)	14 (5.0)		16 (14)	9 (7.7)	
Lateral spinal flexion, mean (SD)	17.9 (4.8)	19.0 (4.6)	p<0.001*	16.4 (4.3)	17.1 (5.0)	p=0.058
Impaired, n (%)	84 (30)	55 (20)		46 (39)	39 (33)	
Modified Schober test, mean (SD)	4.9 (1.2)	5.2 (1.1)	p<0.001*	4.7 (1.1)	4.8 (1.2)	p=0.346
Impaired, n (%)	23 (8.0)	10 (3.5)		12 (10)	12 (10)	
Intermalleolar distance, mean (SD)	117.4 (15.3)	118.2 (14.2)	p=0.337	107.9 (21.5)	108.2 (19.2)	p=0.886
Impaired, n (%)	13 (4.6)	10 (3.5)		27 (23)	19 (16)	
BASMI, mean (SD)	1.8 (0.9)	1.6 (0.7)	p<0.001*	2.3 (0.8)	2.1 (0.9)	p=0.010*
Impaired, n (%)	42 (19)	16 (7.3)		41 (39)	26 (25)	
≥1 impaired spinal mobility measure#, n (%)	146 (51)	117 (41)		62 (53)	55 (47)	

includes ociput-to-wall distance, cervical rotation, chest expansion, lateral spinal flexion and modified Scrober test.
* statistical significance.

P10

RESPONSIVENESS OF SPONDYLOARTHRITIS SPECIFIC HEALTH UTILITIES BASED ON THE ASAS-HEALTH INDEX: AN ANCILLARY ANALYSIS FROM THE ASAS-HI INTERNATIONAL VALIDATION STUDY

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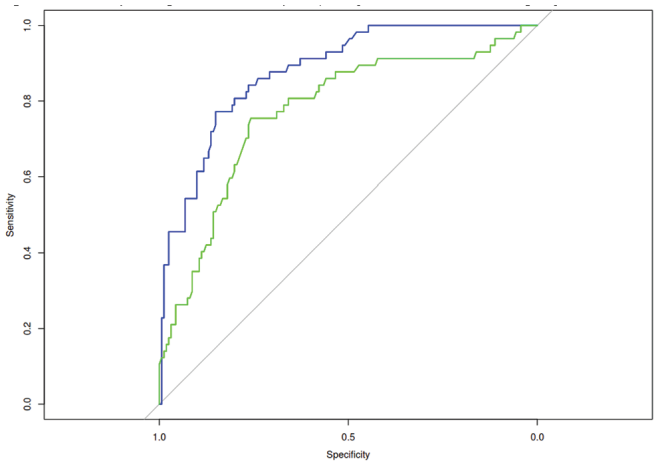
Introduction. To calculate disease-specific health utility, algorithms based on the ASAS Health Index (ASAS-HI) have been developed. However, evidence on its performance to detect change over time is lacking.
Objective. To determine the responsiveness of utility based on ASAS-HI (U--ASAS-HI) scores in measuring changes from when compared to generic health utilities following treatment changes in Spondyloarthritis (SpA).
Methods. An ancillary analysis from the sensitivity to change substudy of the ASAS-HI international validation study. Patients with data on ASAS-HI, EQ-5D 5 L, and SF-6D were included. Responsiveness analysis Standardized Response Mean, and Effect Size (ES) calculation. Subsequently, receiver operating characteristic curve (ROC) analysis was performed to compare the performance of measurement methods.
Results. A total of 219 patients were included of whom 110 started TNFi, 37 csDMARDs, or 72 NSAID. 62.6% were male, median age35 (27-47). As expected, changes in utility scores for each instrument were largest in anti-TNF group followed by csDMARD and finally NSAID group. Across anti-TNF starters, U-ASAS-HI and SF-6D had a large ES (0.99 and 0.93 respectively), and EQ-5D a moderate ES (0.43). In csDMARDs starters, the three utilities had a moderate ES (0.63-0.69). In NSAID starters, EQ-5D had a moderate ES (0.51), meanwhile U-ASAS-HI and SF-6D had a small ES (0.47 and 0.42) Table 1. The ROC is displayed in Figure 1, the area under the curve (AUC) for U-ASAS-HI in predicting changes in EQ-5D utility was 0.88 (95% CI:0.83-0.93) and for changes in SF-6D utility was 0.72 (95% CI:0.70-0.85), indicating a good level of discrimination.
Conclusion. This study demonstrates that U-ASAS-HI is a responsive utility measurement for assessing changes in SpA. Furthermore, the ability of U-ASAS-HI to measure changes, especially in response to anti-TNF and csDMARD treatments, suggests that it may serve as a valuable tool for evaluating treatment outcomes in patients with SpA.
Acknowledgements. The Assessment of Spondyloarthritis International Society (ASAS) supported Omar-Javier Calixto with a research fellowship grant. The ASAS health index global study was conducted under the umbrella of the ASAS.

P10: Table I. Effect size and standard response mean for utilities score per treatment groups.

Group	Measurement	Guyatt's effect size (95% CI)	Standardized response mean (95% CI)
Anti-TNF n=110	U-ASAS-HI	0.99 [0.80, 1,18]	0.99 [0.82, 1.20]
	EQ-5D	0.43 [0.24, 0.62]	0.55 [0.14, 1.04]
	SF-6D	0.93 [0.75, 1.13]	0.95 [0.75, 1.18]
csDMARD n=37	U-ASAS-HI	0.67 [0.33, 1.00]	0.68 [0.43, 0.92]
	EQ-5D	0.69 [0.36, 1.03]	0.73 [0.40, 1.06]
	SF-6D	0.63 [0.29, 0.96]	0.64 [0.34, 0.96]
NSAID n=72	U-ASAS-HI	0.47 [0.23, 0.70]	0.48 [0.28, 0.67]
	EQ-5D	0.51 [0.28, 0.75]	0.52 [0.35, 0.69]
	SF-6D	0.42 [0.19, 0.66]	0.43 [0.19, 0.66]

Anti-TNF; anti-tumor necrosis factor, CI; confidence interval, csDMARD; conventional synthetic disease-modifying anti-rheumatic drug, NSAID; nonsteroidal anti-inflammatory drug.



P10: Fig. 1. Receiver operating characteristics (ROC) analysis for U-ASAS-HI change by EQ-5D and SF-6D.
Graph showing the receiver operating characteristics (ROC) analysis based on effect size minimal significant difference for the entire population. U-ASAS-HI change by EQ-5D ROC AUC: 0.88 (blue line), U-ASAS-HI change by SF-6D ROC AUC: 0.72 (green line), line of no discrimination represented in diagonal (grey line).

P11

PSYCHOMETRIC PROPERTIES AND THRESHOLDS TO GUIDE INTERPRETATIONS OF BATH ANKYLOSING SPONDYLITIS DISEASE ACTIVITY INDEX FATIGUE QUESTION 1 (BASDAI- Q1) AND FUNCTIONAL ASSESSMENT OF CHRONIC ILLNESS THERAPY-FATIGUE (FACIT-FATIGUE) SUBSCALE IN AXIAL SPONDYLOARTHRITIS (AXSPA)

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Introduction. Fatigue is a mandatory domain in the core outcome set for axSpA developed by the Assessment of SpondyloArthritis international Society-Outcome Measures in Rheumatology initiative. We describe psychometric properties of two measures used to assess fatigue in axSpA, FACIT-Fatigue and BASDAI-Q1, and generated thresholds to interpret the scores: meaningful within-patient change (MWPC), meaningful (between-group) difference (MD) and severity levels.
Methods. Analyses used pooled data from patients who received bimekizumab or placebo in BE MOBILE 1 (non-radiographic axSpA;

NCT03928704) and 2 (radiographic axSpA; NCT03928743). Psychometric analyses were conducted to describe internal consistency, construct validity, test-retest reliability and responsiveness. MWPC was derived using anchor-based methods (anchors: SF-36 item 9i, ASDAS states and ASAS response levels) and MD using anchor- and distribution-based methods. Fatigue severity levels were determined using ROC curves analysis against external anchors (SF-36 items 9i/9g; ASDAS states).

Results. 586 patients with axSpA were included. Overall completion rates across baseline to Week 16 were 99.5% for FACIT-Fatigue and 99.0% for BASDAI-Q1. For FACIT-Fatigue, Cronbach's alpha was 0.94, indicating excellent internal consistency and reliability. FACIT-Fatigue and BASDAI-Q1 displayed excellent (ICC=0.909) and good (ICC=0.827) test-retest reliability, respectively, in stable patients from Weeks 16 and 24. FACIT-Fatigue displayed medium to high Spearman's correlations with most concurrent scales; BASDAI-Q1 baseline correlations were weaker, although improved at Week 16 (Table 1I). Both instruments proved sensitive to change (absolute effect size [LS mean change/SD at baseline] >0.80 for ASAS40 responders; consistent results obtained for other anchors). Ranges of derived MWPC and MD thresholds and cut-offs to define fatigue severity levels are presented in Table II.

Conclusion. This analysis indicates adequate psychometric properties for FACIT-Fatigue and BASDAI-Q1. Better reliability and construct validity were obtained with the multi-item FACIT-Fatigue; both were sensitive to clinical change. Derived thresholds can guide interpretation of these scores in axSpA trials.

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Disclosures. CdlL: Consultant to UCB Pharma; **FF:** Employee of Evidera PPD; **DV:** Contractor for UCB Pharma and employee of Veramed; **VT, CF:** Employees and shareholders of UCB Pharma; **UM:** Employee of UCB Pharma; **MD:** Consultant for/speaker fees/research grants from AbbVie, Eli Lilly, Merck, Novartis, Pfizer, and UCB Pharma; **VNC:** Speakers bureau for AbbVie, Eli Lilly, Fresenius Kabi, Janssen, MSD, Novartis, Pfizer, and UCB Pharma; consultant for AbbVie, Eli Lilly, Galapagos, Moonlake, MSD, Novartis, Pfizer, and UCB Pharma; grant/research support from AbbVie and Novartis; **JAW:** Consultant for/grant support from AbbVie, Amgen, Eli Lilly, Janssen, Merck, Novartis, Pfizer, and UCB Pharma.

P11: Table I. Concurrent validity of the FACIT-Fatigue subscale and BASDAI-Q1 with other measures (Spearman's correlations).

Weak correlation: |r|<0.30 Moderate correlation: 0.30≤|r|<0.70 Strong correlation: 0.70≤|r|<0.90

Measure	FACIT-Fatigue subscale score		BASDAI-Q1 Fatigue score	
	Baseline (n=586)	Week 16 (n=567)	Baseline (n=586)	Week 16 (n=568)
	r	r	r	r
FACIT-Fatigue subscale score	–	–	−0.477	−0.658[a]
BASDAI-Q1 Fatigue score	−0.477	−0.658	–	–
ASDAS	−0.357	−0.512[b]	0.342	0.729[c]
SF-36: Physical Functioning	0.649	0.640	−0.262	−0.617
SF-36: Role Physical	0.631	0.724	−0.237	−0.578
SF-36: Bodily Pain	0.602	0.638	−0.311	−0.693
SF-36: General Health	0.491	0.550	−0.208	−0.411
SF:36: Vitality	0.791	0.842	−0.363	−0.579
SF-36: Social Functioning	0.621	0.660	−0.194	−0.482
SF-36: Role Emotional	0.393	0.486	−0.123[d]	−0.307
SF-36: Mental Health	0.558	0.655	−0.179	−0.381
SF-36: Physical Component Summary	0.608	0.667	−0.268	−0.659
SF-36: Mental Component Summary	0.537	0.615	−0.179	−0.309
BASDAI total score	−0.502	−0.619	–	–
BASFI score	−0.578	−0.579	0.388	0.739
ASQoL total score	−0.829	−0.799	0.374	0.678
PtGADA score	−0.416	−0.505	0.410	0.755
Total Spinal Pain score	−0.363	−0.530	0.458	0.804
Nocturnal Spinal Pain score	−0.377	−0.515	0.423	0.764
EQ-VAS score	0.483	0.558	−0.278	−0.583[a]
PHQ-9 total score	−0.271	−0.439	0.081[e]	0.315
PHQ-9 Item 4: Feeling tired or having little energy?	−0.292	−0.448	0.169	0.356
WPAI:SHP: Percent of work time missed due to problem	−0.335[f]	−0.355[g]	0.141[e,f]	0.319[g]
WPAI:SHP: Percent of impairment while working due to problem	−0.550[h]	−0.610[i]	0.307[h]	0.676[i]
WPAI:SHP: Percent of overall work impairment due to problem	−0.553[h]	−0.616[i]	0.304[h]	0.667[i]
WPAI:SHP: Percent of activity impairment due to problem	−0.591	−0.630	0.349	0.717[a]
MOS Sleep-R: Sleep Disturbance score	0.539	0.526	−0.251	−0.434
MOS Sleep-R: Sleep Problems Index II	0.695	0.719	−0.340	−0.529

[a] n=567; [b] n=560; [c] n=561; [d] p=0.003; [e] p=0.050; [f] n=431; [g] n=425; [h] n=393; [i] n=384; p-value for each correlation were p<0.001 unless otherwise noted: There were no correlations ≥0.90. The inclusion criteria of the BE MOBILE studies to include only patients with BASDAI ≥4 at screening and baseline resulted in a range restriction on BASDAI-Q1; this lowered the standard deviation of BASDAI-Q1, reducing the correlations with other measures at baseline. ASDAS: Axial Spondyloarthritis Disease Activity Score; ASQoL: Ankylosing Spondylitis Quality of Life; BASDAI. Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index; EQ-VAS: EQ-Visual Analogue Scale; FACIT-Fatigue: Functional Assessment of Chronic Illness Therapy-Fatigue; MOS Slee-R: Medical Outcomes Study Sleep Scale-Revised; PtGADA. Patient's Global Assessment od Disease Activity; PHQ-9: Patient Health Questionnaire-9 Items; Q1-Fatigue: Single-item Fatigue-Question 1; SF-36: Short-Form 36-Item Health Survey; WPAI: Work Productivity and Activity Imparment Questionnaire-Specific Health Problem.

P11: Table II. Clinically meaningful change thresholds and cut-off scores to define fatigue severity levels.

	FACIT-Fatigue subscale score	BASDAI-Q1 Fatigue score
MWPC (within patient)	MWPC thresholds for **improvement** from baseline to Week 16 ranged between a **5- and 11-point increase** in FACIT-Fatigue subscale score. An **8-point increase** from baseline to Week 16 is proposed to define response	The MWPC threshold for **improvement** from baseline to Week 16 is a **2-point decrease** in BASDAI-Q1 score
MD (between groups)	MD thresholds in FACIT-Fatigue subscale score ranged between **2.14 and 5.34 points** corresponding to a small-to-medium ES	MD thresholds in BASDAI-Q1 score ranged between **−0.32 and −1.59 points** corresponding to a small-to-medium ES
Fatigue severity levels	Severe (<26), moderate (≥26 to <31), mild (≥31 to <41), none or minimal (≥41) 0 26 31 41 52	None or minimal (<2), mild (≥2 to <4), moderate (≥4 to <7), severe (≥7) 0 2 4 7 10

BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; ES: effect size; FACIT: Functional Assessment of Chronic Illness Therapy; MD: meaningful (between group) difference; MWPC: meaningful within-patient change.

P12

DIAGNOSTIC DELAY IN PATIENTS INCLUDED IN THE INTERNATIONAL MAP OF AXIAL SPONDYLOARTHRITIS: ASSOCIATIONS WITH GEOGRAPHIC, SOCIO-DEMOGRAPHIC AND DISEASE-RELATED FACTORS

Poddubnyy D.[1,2], Garrido-Cumbrera M.[3,4], Sommerfleck F.[5], Navarro-Compán V.[6], Bundy C.[7], Makri S.[8], Correa-Fernández J.[3], Akerkar S.[9], Mandelbaum M.[10], Lowe J.[10], Karam E.[11]

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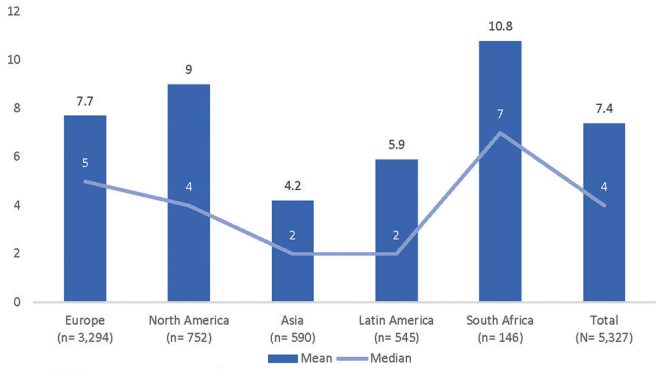
Introduction. Despite efforts for early detection, delayed diagnosis in axial spondyloarthritis (axSpA) remains an unresolved challenge. This analysis aimed to assess diagnostic delay and its associated factors around the world in a large sample of patients included in the International Map of Axial Spondyloarthritis (IMAS).

Methods. IMAS is a cross-sectional online survey (2017-2022) including 5,557 unselected axSpA patients from 27 countries. Diagnostic delay was calculated as the difference between age at diagnosis and age at symptom onset reported by patients. The independent factors evaluated were: age at symptom onset, disease duration, gender, education level, whether diagnosed by rheumatologist, number of healthcare professionals (HCPs) seen before diagnosis, HLA-B27, uveitis, and inflammatory bowel disease. The factor world region was introduced as a dummy variable taking Europe as the reference region. Associations between diagnostic delay and regions, sociodemographic characteristics, as well as disease-related factors were explored through univariable and multivariable linear regression analysis.

Results. Data from 5,327 patients who reported data to calculate diagnostic delay in IMAS survey were analyzed (3,231 Europe, 770 North America, 600 Asia, 548 Latin America, and 146 Africa). Patients reported a mean diagnostic delay of 7.4 years (median: 4.0), being highest in South Africa and lowest in Asia (Fig. 1). The variables associated with longer diagnostic delay were: younger age at symptom onset (b=-0.100), more disease duration (b=0.363), female gender (b=2.274), being diagnosed by a rheumatologist (b=1.163), higher number of healthcare professionals (HCPs) seen before diagnosis (b=1.033), and presence of uveitis (b=1.286; Table I).

Conclusion. The mean diagnostic delay was 7.4 years, and had significant differences across regions. Younger age at symptom onset, longer disease duration, female gender, diagnosis by a rheumatologist, higher number of HCPs seen before diagnosis, and the presence of uveitis were the parameters associated with a longer diagnostic delay in axSpA patients.

Acknowledgements. This study was supported by Novartis Pharma AG. As of 2 January 2024, IMAS is owned solely by ASIF, following an in-kind donation of the project by Novartis Pharma AG. The authors would like to thank all patients who participated in the study.

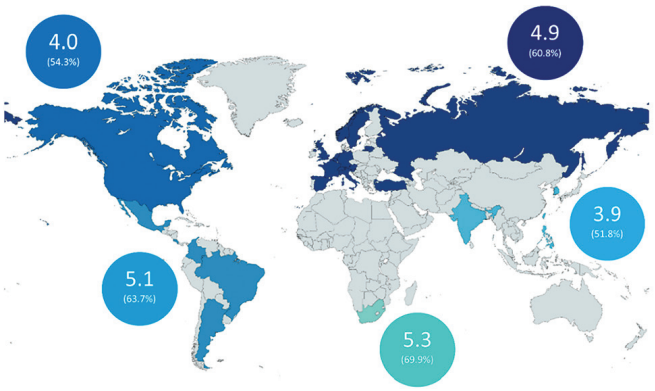


P12: Fig. 1. Mean and median diagnostic delays by region (n=5,327)

P12: Table I. Univariable and multivariable linear regression analysis of the association between diagnostic delay and independent variable in patients with axial spondyloarthritis (n= 4,595)

Variables	Ref.	Univariable analysis		Multivariable analysis	
		B	95% CI	B	95% CI
Age at symptom onset, years	-	**-0.306**	**-0.326, -0.287**	**-0.100**	**-0.120, -0.080**
Disease duration	-	**0.401**	**0.386, 0.416**	**0.363**	**0.346, 0.380**
Female gender	Male	**2.324**	**1.843, 2.804**	**2.274**	**1.860, 2.687**
Diagnosed by rheumatologist, yes	No	**2.410**	**1.868, 2.952**	**1.163**	**0.710, 1.615**
No. of HCPs seen before diagnosis	-	**1.696**	**1.520, 1.873**	**1.033**	**0.877, 1.189**
Uveitis	No	**1.580**	**0.996, 2.165**	**1.286**	**0.808, 1.764**
Inflammatory bowel disease	No	**0.834**	**0.117, 1.550**	-0.043	-0.610, 0.525
Region, Asia	Europe	**-3.511**	**-4.241, -2.781**	**1.003**	**0.334, 1.673**
Region, North America		**1.228**	**0.499, 1.958**	**1.470**	**0.902, 2.039**
Region, Latin America		**-1.792**	**-2.583, -1.000**	0.626	-0.045, 1.297
Region, South Africa		**3.015**	**1.549, 4.481**	**3.356**	**2.170, 4.541**

Dependent variable in all models: diagnostic delay in years. CI: confidence interval, HCP: health care professional.



P13: Map 1. Mental health status (mean GHQ-12) and prevalence of poor mental health (GHQ-12 ≥ 3) for each region.
Data shown in the circles refer to the mental health status (mean of GHQ-12) prevalence of poor mental health (GHQ-12≥ 3).

P13: Table I. Logistic regression analysis of demographic, socioeconomic disease-related factors with poor mental health (GHQ-12 ≥ 3; n=4,335).

	Univariable logistic regression		Multivariable logistic regression	
	OR	95%CI	OR	95%CI
Age	0.98	**0.97,0.98**	**0.99**	**0.98,0.99**
Gender. Female	1.57	**1.40,1.75**	**1.16**	**1.01,1.33**
Educational level. No university	1.26	**1.13,1.40**	0.96	0.84,1.11
Employment status. Sick leave or unemployed	2.02	**1.77,2.31**	**1.63**	**1.39,1.91**
Patient organisation membership. No	1.15	**1.03,1.28**	0.95	0.83,1.10
Physical activity engagement. No	1.49	**1.28,1.74**	**1.22**	**1.01,1.47**
Smoking. Yes	1.63	**1.42, 1.88**	**1.20**	**1.01,1.43**
Alcohol consumption. Yes	0.79	**0.70, 0.89**	1.03	0.89,1.20
BASDAI (0-10)	1.52	**1.48,1.57**	**1.42**	**1.36,1.48**
Spinal Stiffness Index (3-12)	1.15	**1.12,1.18**	1.03	0.99,1.07
Functional Limitation Index (0-54)	1.03	**1.03,1.03**	**1.02**	**1.01,1.02**
Symptom duration, years	0.98	**0.98, 0.99**	**0.98**	**0.98,0.99**
Inflammatory bowel disease	1.32	**1.12, 1.56**	1.01	0.83,1.23

P13

FACTORS ASSOCIATED WITH POOR MENTAL HEALTH IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS: RESULTS FROM THE INTERNATIONAL MAP OF AXIAL SPONDYLOARTHRITIS (IMAS)

Garrido-Cumbrera M.[1,2], Navarro-Compán V.[3], Poddubnyy D.[4,5], Sommerfleck F.[6], Makri S.[7], Correa-Fernández J.[1], Akerkar S.[8], Lowe J.[9], Karam E.[10], Bundy C.[11]

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Introduction. Impaired mental health in axial spondyloarthritis (axSpA) is associated with poorer physical outcomes and employment issues. This study aims to assess the prevalence of poor mental health in axSpA and its associated factors in a large sample of patients from the International Map of Axial Spondyloarthritis (IMAS) study from around the world.
Methods. IMAS is a cross-sectional online survey (2017-2022) that includes 5,557 unselected axSpA patients worldwide from Europe, North America, Latin America, Asia, and South Africa. Mental health was evaluated by the General Health Questionnaire (GHQ-12) and patients were categorized into those with good mental health (GHQ-12<3) and those with potentially poor mental health (GHQ-12≥3). The independent factors evaluated were sociodemographic, lifestyles, patient-reported outcomes (including BASDAI 0-10), disease characteristics, extra-musculoskeletal manifestations and treatments. Logistic regression analyses were used to evaluate factors associated with poor mental health.
Results. Of 5,351 patients, mean of GHQ-12 was 4.7 (median 4.0) with 59.4% potentially having poor mental health. The prevalence of poor mental health was 69.9% in South Africa, 63.7% in Latin America, 60.8% in Europe, 54.3% in North America and 51.8% in Asia (Map 1). Overall, 40.5% and 37.2% of patients experienced anxiety and depression respectively. The factors associated with poor mental health were younger age (OR=0.99), females (OR=1.16), being on sick leave or unemployed (OR=1.63), non-physical activity (OR=1.22), smoking (OR=1.20), higher BASDAI (OR=1.42), functional limitation (OR=1.02), and shorter symptoms duration (OR=0.98; Table I).
Conclusion. Globally, six in ten patients with axSpA had signs of mental health issues, with highest proportion in South Africa and a lowest in Asia. Factors associated with poor mental health include domains such as younger age, female gender, employment difficulties, harmful habits, disease burden, and symptoms duration. A holistic management approach should encompass both physical and mental health in axSpA.
Acknowledgements. This study was supported by Novartis Pharma AG. As of 2 January 2024, IMAS is owned solely by ASIF, following an in-kind donation of the project by Novartis Pharma AG. The authors would like to thank all patients who participated in the study.

P14

THE INTERNATIONAL MAP OF AXIAL SPONDYLOARTHRITIS (IMAS) - RESULTS FROM THE PERSPECTIVE OF 5,557 PATIENTS FROM 27 COUNTRIES AROUND THE WORLD

Garrido-Cumbrera M.[1,2], Poddubnyy D.[3,4], Sommerfleck F.[5], Bundy C.[6], Makri S.[7], Correa-Fernández J.[1], Akerkar S.[8], Mandelbaum M.[9], Lowe J.[9], Karam E.[10], Navarro-Compán V.[11]

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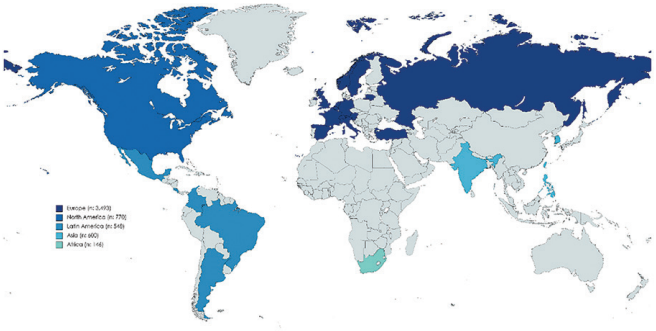
Introduction. The International Map of Axial Spondyloarthritis (IMAS) is a global assessment of the impact and burden of axial spondyloarthritis (axSpA) from the patient perspective. This research started in 2016 with the Atlas of Axial Spondyloarthritis in Spain, expanded in 2017-2018 to 13 European countries giving rise to the European Map of Axial Spondyloarthritis (EMAS) and further expanding worldwide across 27 countries to become the International Map of Axial Spondyloarthritis (IMAS). Here we present the scope of the study and main characteristics of patients included.
Methods. IMAS collected information through an online cross-sectional survey (2017-2022) from 5,557 unselected axSpA patients from Europe, Asia, North America, Latin America and Africa. The questionnaire contained over 120 items on socio-demographics, health behaviours, diagnosis and disease characteristics, comorbidities, mental health (GHQ-12), healthcare resource utilization, treatments, disease activity (BASDAI 0-10), physical activity and functioning, employment, fears and hopes.
Results. The mean age was 44 years, 55.4% were females, 46.2% had university education and 48.5% were employed. Mean diagnostic delay was 7.4 years, and mean disease duration was 17.1 years. 75.0% had active disease and 59.4% were at risk of poor mental health. On average, patients had visited primary care physicians 4.6 times and the rheumatologist 3.6 times in the year prior to the survey. 78.6% had ever taken NSAIDs, 43.6% csDMARDs and 48.8% bDMARDs. The greatest fear of these patients was disease progression (55.9%), while the greatest hope was to be able to relieve pain (54.2%).
Conclusion. IMAS is the largest survey in geographical reach and has shown the global profile of axSpA patients, highlighting their unmet needs. This global dataset will enable more detailed investigations to obtain evidence on the critical issues that matter to patients in order to improve their care, quality of life and health policy.
Acknowledgements. This study was supported by Novartis Pharma AG. As of 2 January 2024, IMAS is owned solely by ASIF, following an in-kind donation of the project by Novartis Pharma AG. The authors would like to thank all patients who participated in the study.



P14: Map 1. Distribution of IMAS patients by region (n=5,557).

P15

WHAT FACTORS ARE ASSOCIATED WITH PAIN INTENSITY IN AXIAL SPONDYLOARTHRITIS? RESULTS FROM THE INTERNATIONAL MAP OF AXIAL SPONDYLOARTHRITIS (IMAS)

Garrido-Cumbrera M.[1,2], Navarro-Compán V.[3], Sommerfleck F.[4], Bundy C.[5], Makri S.[6], Correa-Fernández J.[1], Akerkar S.[7], Lowe J.[8], Karam E.[9], Poddubnyy D.[10,11]

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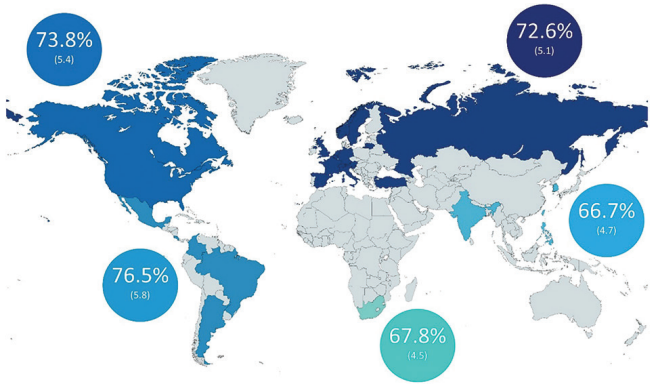
Introduction. Severe pain in patients with axial spondyloarthritis (axSpA) can cause deterioration in their physical and mental health. The aim is to investigate factors associated with pain intensity in a large sample of patients from the International Map of Axial Spondyloarthritis (IMAS) study from around the globe.
Methods. IMAS is a cross-sectional online survey (2017-2022) including 5,557 unselected axSpA patients worldwide from Europe, North America, Latin America, Asia, and South Africa. 5,347 participants who reported pain were analysed. Pain (0-10) was assessed using the average of Q2 (axial pain) and Q3 (peripheral pain) BASDAI items. The factors evaluated were sociodemographic, disease characteristics, lifestyle, patient-reported outcomes (including GHQ-12 scale), employment, mental comorbidities and treatments. Univariable and multivariable linear regression was used to evaluate factors associated with pain.
Results. Of 5,347 patients, average of axial pain was 5.7(±2.5), average of peripheral pain was 4.6(±2.7), average overall pain was 5.2(±2.4) and 72.4% reported a high pain intensity (≥4). The average overall pain was the highest in Latin America and the lowest in South Africa (Map 1). Factors associated with higher pain intensity were no university education (b=0.36), shorter diagnostic delay (b=-0.02), greater spinal stiffness (b=0.22), higher functional limitation (b=0.03), poorer mental health (b=0.13), difficulty finding a job (b=0.89), and sleep disorders (b=0.77; Table I).
Conclusion. Globally, seven in ten patients with axSpA had high pain intensity, with higher proportion in the Americas and lower in South Africa. Pain in patients with axSpA was most strongly associated with the absence of university degree, presence of work impairment as job search, as well as poor patient-reported outcomes. Pain is a critical symptom in axSpA, as it is associated with work impairment and negative disease outcomes so, if patients' quality of life is to be improved, pain reduction should be a priority in treatment and management.
Acknowledgements. This study was supported by Novartis Pharma AG. As of 2 January 2024, IMAS is owned solely by ASIF, following an in-kind donation of the project by Novartis Pharma AG. The authors would like to thank all patients who participated in the study.



P15: Map 1. Distribution of reported pain intensity across regions of the world
Data shown in the circles refer to the prevalence of pain intensity (mean of pain).

P15: Table I. Univariable and multivariable linear regression analysis of the factors associated with pain* (n=1,254)

	Univariable linear regression		Multivariable linear regression	
	Beta	95% CI	Beta	95% CI
Age	-0.01	**-0.02, -0.01**	-0.01	-0.02, 0.003
Gender. Female	0.57	**0.44, 0.69**	0.15	-0.09, 0.38
Educational level. No University	0.59	**0.46, 0.71**	0.36	**0.13, 0.59**
Diagnostic delay, years	0.01	**0.001, 0.02**	-0.02	**-0.03, -0.01**
HLA-B27. Negative	0.38	**0.20, 0.56**	0.16	-0.08, 0.41
Physical activity. No	0.44	**0.27, 0.61**	-0.21	-0.53, 0.11
Body Mass Index. Overweight/Obesity	0.43	**0.30, 0.55**	0.22	-0.02, 0.44
Spinal Stiffness (3-12)	0.32	**0.29, 0.34**	0.22	**0.17, 0.27**
Functional limitation (0-54)	0.04	**0.04, 0.05**	0.03	**0.02, 0.04**
Mental Health GHQ-12 (0-12)	0.21	**0.20, 0.23**	0.13	**0.09, 0.16**
Work-Related Issues. Yes	0.98	**0.81, 1.15**	0.25	-0.03, 0.53
Difficulty finding job due to axSpA. Yes	1.83	**1.68, 1.97**	0.89	**0.60, 1.18**
Work choice determinate by axSpA. Yes	0.88	**0.74, 1.01**	0.11	-0.14, 0.35
Anxiety diagnosis. Yes	1.02	**0.89, 1.15**	-0.07	-0.39, 0.26
Depression diagnosis. Yes	1.14	**1.01, 1.28**	-0.20	-0.52, 0.11
Sleep disorder diagnosis. Yes	1.37	**1.24, 1.50**	0.77	**0.49, 1.05**
csDMARDs. Yes	0.18	**0.04, 0.31**	0.18	-0.06, 0.42
bDMARDs. Yes	0.11	-0.02, 0.25	0.004	-0.24, 0.24

*Average pain in all body joints including neck, back and hips.

P16

REGIONAL DIFFERENCES IN PATIENT DIAGNOSIS JOURNEY AND HEALTHCARE UTILIZATION - RESULTS FROM THE INTERNATIONAL MAP OF AXIAL SPONDYLOARTHRITIS (IMAS)

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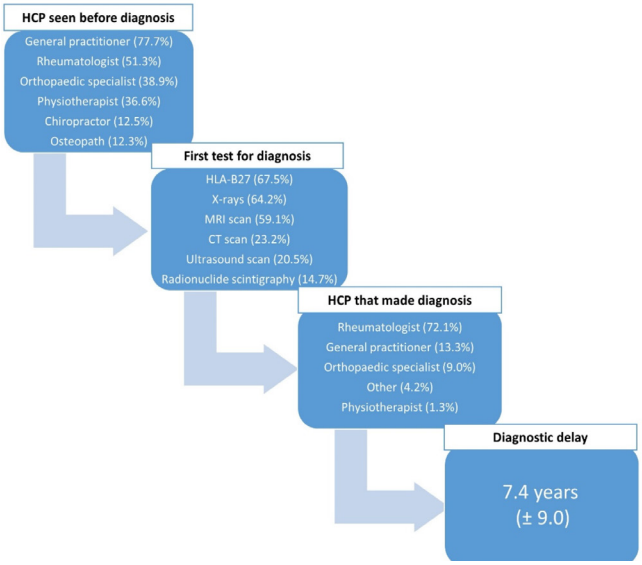
Introduction. Both the diagnosis and management of axial spondyloarthritis (axSpA) can be challenging. The aim is to assess the journey of axSpA patients and healthcare utilization around the world in a large sample of patients included in the International Map of Axial Spondyloarthritis (IMAS).
Methods. IMAS was a cross-sectional online survey (2017-2022) of 5,557 unselected axSpA patients from 27 countries (3,493 Europe, 770 North America, 600 Asia, 548 Latin America, and 146 South Africa). Patient journey and healthcare utilization in the last 12 months prior to survey were analyzed. Healthcare utilization was calculated as the sum of the number of healthcare visits, medical tests, hospital admissions and emergency visits based on the last 12 months prior to the survey. Linear regression analyses were used to explore associated factors with higher healthcare utilization.
Results. Mean diagnostic delay was 7.4 years, needing more than two visits to HCPs for diagnosis (77.7% GP and 51.3% rheumatologist), and more than two diagnostic tests (67.5% HLA-B27, 64.2% x-rays and 59.1% MRI scans; Fig. 1). North America and Europe had the highest number of HCPs visits for diagnosis, and Asia the lowest. Factors associated with higher healthcare utilization were younger age (b=-0.31), females (b=7.74), higher disease activity (b=1.46), poorer mental health (b=0.62), greater functional limitation (b=0.30), greater spinal stiffness (b=1.53) and longer diagnostic delay (b=0.10; Table I).
Conclusion. Patients with axSpA made more than two visits to HCPs and took at least 7 years to be diagnosed. Younger age, female gender, higher disease activity, poorer mental health, greater functional limitation, greater spinal stiffness, and longer diagnostic delay were associated with higher healthcare utilization. Shortening the patient's journey to diagnosis, along with regular follow-up care and close collaboration between the patient and the medical team, are essential to effectively manage axSpA and improve the patient's long-term quality of life.
Acknowledgements. This study was supported by Novartis Pharma AG. As of 2 January 2024, IMAS is owned solely by ASIF, following an in-kind donation of the project by Novartis Pharma AG. The authors would like to thank all patients who participated in the study.



P16: Fig. 1. The patient journey to axial spondyloarthritis diagnosis.

P16: Table I. Univariable and multivariable linear regression analysis of socio-demographic and patient-reported outcomes according to total healthcare utilization (n=5,004).

	Simple linear regression		Multiple linear regression	
	Beta	95%CI	Beta	95%CI
Age	-0.27	**-0.33, -0.20**	-0.31	**-0.38, -0.24**
Gender. Female	10.67	**8.87, 12.37**	7.74	**6.08, 9.39**
Patient organization membership. Yes	1.69	-0.04, 3.41	-	-
BASDAI (0-10)	3.89	**3.50, 4.28**	1.46	**0.99, 1.93**
GHQ-12 (0-12)	1.69	**1.48, 1.89**	0.62	**0.40, 0.85**
Functional limitation (0-54)	0.53	**0.47, 0.58**	0.30	**0.24, 0.36**
Spinal stiffness (3-12)	2.45	**2.11, 2.79**	1.53	**1.15, 1.91**
Diagnostic delay	0.23	**0.13, 0.32**	0.10	**0.01, 0.20**

P17

REGIONAL DIFFERENCES IN CLINICAL PHENOTYPE OF AXIAL SPONDYLOARTHRITIS. RESULTS FROM THE INTERNATIONAL MAP OF AXIAL SPONDYLOARTHRITIS (IMAS)

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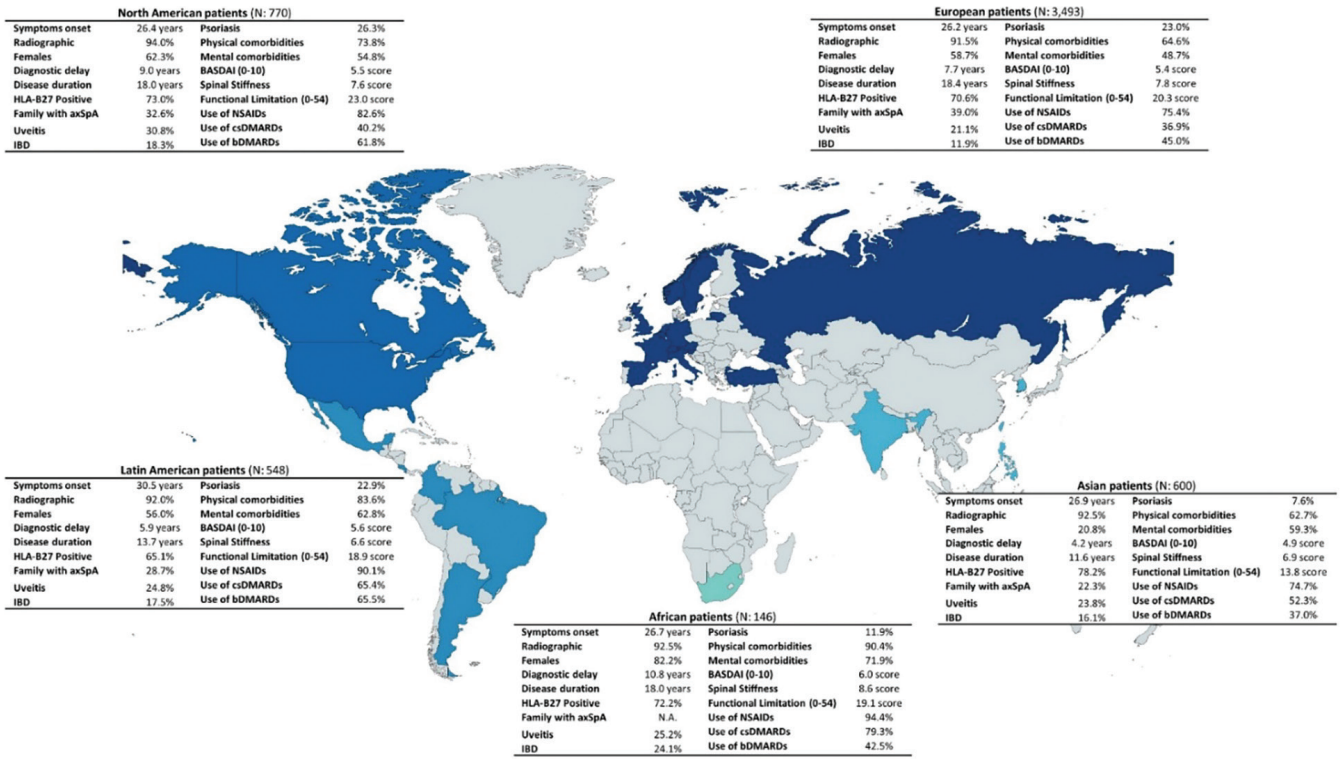

P17: Map 1. The clinical phenotype of axial spondyloarthritis stratified by region (n=5,557).

Introduction. Previous studies have suggested there could be regional differences in clinical phenotype of axial spondyloarthritis (axSpA). This analysis aims to explore differences in axSpA clinical phenotype in a large sample of patients included in the International Map of Axial Spondyloarthritis (IMAS).

Methods. IMAS was a cross-sectional online survey (2017-2022) of 5,557 unselected axSpA patients from 27 countries (3,493 Europe, 770 North America, 600 Asia, 548 Latin America, and 146 Africa). Age at onset of symptoms, radiographic/non-radiographic classification, gender, HLA-B27, family history, extra-musculoskeletal manifestations, comorbidities, disease activity (BASDAI), spinal stiffness, and treatments were compared between regions.

Results. Results showed statistically significant differences between regions, except for radiographic/non-radiographic classification. Age at onset of symptoms ranged between 25-30 years, and was the highest in Latin America. Diagnostic delay was longest in South Africa and lowest in Asia. The lowest frequency of HLA-B27 positivity was observed in Latin America and the highest in Asia. SpA family history was highest in Europe and lowest in Asia. Uveitis and inflammatory bowel diseases were lowest in Europe. Physical and mental comorbidities were frequent in African patients and less common in Europe and Asia. Mean disease activity was 5.4, with highest values in South Africa and lowest in Asia. Spinal stiffness was highest in South Africa and lowest in Latin America. Functional limitation was higher in North America and Europe and lower in Asia. Most of the patients had used NSAIDs and less than half had ever taken csDMARDS; both were more frequent in Latin America and South Africa. Almost half of the patients had ever taken bDMARDs, more frequent being in the Americas (Map 1).

Conclusion. There is great heterogeneity of axSpA clinical phenotype presentation around the world. Further understanding of these differences is needed to achieve early diagnosis and initiation of disease treatment in axSpA.

Acknowledgements. This study was supported by Novartis Pharma AG. As of 2 January 2024, IMAS is owned solely by ASIF, following an in-kind donation of the project by Novartis Pharma AG. The authors would like to thank all patients who participated in the study.

P18

DEPRESSION DISORDERS IN SPONDYLOARTHRITIS PATIENTS ON BIOLOGIC THERAPY REGISTERED IN REUMA.PT: PREVALENCE, ROLE OF DISEASE-RELATED FACTORS AND INFLUENCE OF BIOLOGIC THERAPY

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Introduction. Depression disorder (DD) prevalence is higher in chronic conditions like axial and peripheral Spondyloarthritis (SpA) possibly linked to inflammation. Our aim was to determine DD prevalence in SpA patients at first biologic prescription (bDMARD) and to assess effect on DD.

Methods. We conducted a multicenter, retrospective, observational study including adult patients registered in Reuma.pt with SpA treated with their first bDMARD. Patients that completed the Hospital Anxiety and Depression Scale (HADS) at baseline (T0), after 3 (T1) and/or 6 months (T2) of treatment were included. Socio-demographic, disease and treatment-related data were collected. DD was considered when subscale depression (HADS-D) ≥11. Pearson and Spearman correlations, ANOVA and T-tests were used.

Results. 141 patients were analyzed. Table 1 summarizes population characteristics. Factors correlated with DD are summarized in Table II.

Patients with DD had significantly higher BASDAI and ASDAS-CRP, but not BASMI at T0 than those who didn't have DD (p of 0.045, 0.007 and 0.570, respectively). Mean scores of HADS-D significantly differed between the three time points ($F(2.000, 155.387) = 12.440$, $p<0.001$). Post hoc analysis with Bonferroni adjustment revealed that HADS-D significantly decreased from T0 to T1 (1.535 (95% CI, 0.621 to 2.449), $p<0.001$), from T0 to T2 (1.628 (95% CI, 0.630 to 2.626), $p<0.001$), but not from T1 to T2 (0.093 (95% CI, -0.664 to 0.851), $p=1$).

No significant differences were found in HADS-D at the three time points between patients with axial disease and those with peripheral disease ($F(2.000, 52.000) = 2.917$, $p=0.063$) and between patients treated with anti-TNF and those with an anti-IL-17 ($F(1.824, 153.221) = 0.251$, $p=0.758$).

Conclusion. Depression symptoms prevalence in SpA is lower than chronic disease estimated rates (between 18-35.1%). DD improves with bDMARD therapy, probably due to control of disease activity and function improvement. Inflammation hypothesis in depression should be considered and further investigated.

P18: Table I. Population characteristics.

Gender	55.3% female; 44,7% male;	
Mean age at diagnosis	37.4 ± 10.5 years;	
Prescribed bDMARD	Adalimumab 59.6% Etanercept 23.4% Golimumab 7.1% Certolizumab 5.7% Sekucinumab 2.8% Infliximab 1.4%	
Depression symptoms at T0	13.5% (n=19) of patients	
Anxiety symptoms at T0 by gender	Female = 14.1% Male = 12.7%	*p*-value =0.808

P18: Table II. Correlations between disease activity scores or patient reported outcomes with depression disorder.

	Pearson	Spearman	*p*-value
Disease activity scores			
ASDAS-CRP	0.230		**0.007**
BASMI		0.052	0.586
BASDAI		0.166	0.051
Patient-reported outcomes			
PGA	0.133		0.116
BASFI	0.252		**0.003**
PAP		0.104	0.221
FACIT-F		-0.306	**<0.001**
Sf36 – PF		-0.215	**<0.011**
Sf36 – RP		-0.209	**0.014**
Sf36 – BP		-0.180	**0.034**
Sf36 – GH		-0.229	**0.007**
Sf36 – VT		-0.172	**0.044**
Sf36 – SF		-0.315	**<0.001**
Sf36 - RE		-0.343	**<0.001**
Sf36 - MH	-0.489		**<0.001**

ASDAS-PCR: Ankylosing Spondylitis Disease Activity Score-C-reactive protein; BASMI: Bath Ankylosing Spondylitis Metrology Index; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; PGA: Patient Global Assessment; BASFI: Bath Ankylosing Spondylitis Functional Index; PAP: Patient Assessment of Pain; FACIT-F: Functional Assessment of Chronic Illness Therapy-Fatigue Scale; Sf36: 36-Item Short Form Survey; PF: physical functioning; RP: physical role; BP: bodily pain; GH: general health; VT: vitality; SF: social function; RE: emotional role; MH: mental health.

P19

HEALTH-RELATED QUALITY OF LIFE - A MULTIDISCIPLINARY APPROACH IN PSORIATIC DISEASE

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Introduction. Psoriatic Disease (PsD) is a chronic disease that affects the musculoskeletal system, skin, and nails with a negative impact on a patient´s quality of life. Our study aimed to analyze the HRQoL in PsD patients and to identify influencing factors.

Methods. Retrospective cross-sectional study including consecutive adult patients with PsD, followed in a combined dermatology-rheumatology Clinic, between September 2018 and April 2024. Demographic and clinical data were collected. A DLQI (Dermatology Life Quality Index) or PsAQoL (Psoriatic Arthritis Quality of Life) score greater than 10 indicates poor quality of life. Patients were grouped based on their quality-of-life scores. Descriptive and statistical analysis were performed, as appropriate, and a p-value ≤0.05 was considered statistically significant.

Results. Fifty-eight PsD patients were included (Table I). Of the 47 patients who completed both the DLQI and PsAQoL questionnaires, 21.3% had scores >10 on both, while 19.1% had only a PsAQoL >10 and 14.9% had only DLQI >10. Patients with both DLQI and PsAQoL >10 had a higher Health Assessment Questionnaire ($p=0.008$), Patient Pain Assessment ($p=0.041$), and Patient Global Assessment ($p=0.004$). An association was observed between the absence of bDMARD treatment and both scores elevated (p=0.042). Patients with only PsAQoL >10 were older ($p=0.011$). Fewer patients were in remission, p=0.012, had more tender and swollen joints and higher DAS28-ESR and DAS28-CRP 3 variables, $p<0.05$. Rheumatologist global assessment was also higher ($p=0.002$). No associations were found in patients with only DLQI >10.

Conclusion. PsAQoL is linked to articular disease activity and global disease assessment by the Rheumatologist. Patients with both high PsAQoL and DLQI reported worse reported outcomes, but no association was found with disease activity or characteristics, underscoring HRQoL's multidimensional nature, requiring a holistic approach.

P19: Table I. Clinical and demographic patients characteristics (n=58)

Age at first appointment (mean ± SD)	52.98 ± 10.71
Age at onset of PsO symptoms (mean ± SD)	32.21 ± 13.96
Age at onset of joint symptoms (mean ± SD)	40.10 ± 12.13
Sex (M/F), n	(30/28)
Education, n (%)	
First cycle of basic education	7 (21.9)
Second cycle of basic education	8 (25.0)
Third cycle of basic education	2 (6.3)
Secondary education	7 (21.9)
Higher education	8 (25.0)
Employment status, n (%)	
Full-time employed	26 (53.1)
Part-time employed	0 (0)
Housekeeper	6 (12.2)
Unemployed	8 (16.3)
Retired	5 (10.2)
Retired due to disability	3 (6.1)
Medical leave of absence > 1 month	1 (2.0)
Student	0 (0)
Type of clinical pattern, n (%)	
Asymmetric oligoarthritis	16 (36.4)
Symmetric polyarthritis	20 (45.5)
Predominant distal interphalangeal joint	2 (4.5)
Mutilans arthritis	0 (0)
Predominant axial involvement	6 (13.6)
Dactylitis (ever), n (%)	20 (34.5)
Enthesitis (ever), n (%)	9 (15.5)
Uveitis (ever), n (%)	1 (1.7)
IBD, n (%)	0 (0)
Axial involvement, n (%)	21 (36.2)
Comorbidities, n (%)	
Arterial Hypertension	16 (27.6)
Dyslipidemia	19 (32.8)
Diabetes Mellitus	4 (6.9)
Obesity	27 (46.6)
Depression	12 (20.7)
Current smoking, n (%)	4 (6.9)
Current regular alcohol consumption, n (%)	8 (16.0)
DLQI (median ± IQR)	6.0 ± 11.0
PsAQoL (median ± IQR)	9.00 ± 11
HAQ (mean ± SD)	0.81 ± 0.64
Patient pain assessment (mean ± SD)	52.37 ± 21.10
Patient global assessment (median ± IQR)	45.00 ± 38.00
Rheumatologist global assessment (mean ± SD)	29.76 ± 21.27
Dermatologist global assessment (mean ± SD)	24.36 ± 22.11
PASI (median ± IQR)	3.4 ± 9.7
ESR (median ± IQR)	13.0 ± 19.0
CRP (median ± IQR)	2.72 ± 1.40
Tender joints, total (median ± IQR)	1.0 ±2.0
Tender joints, DAS28 (median ± IQR)	0.0 ±1.0
Swollen joints, total (median ± IQR)	0.0 ±2.0
Swollen joints, DAS28 (median ± IQR)	0.0 ±1.0
Composite indices	
DAS28-CRP, 4 variables (median ± IQR)	2.39 ± 2.15
DAS28-CRP, 3 variables (median ± IQR)	2.01 ±1.86
DAS28-ESR, 4 variables (mean ± SD)	3.19 ± 1.46
DAS28-ESR, 3 variables (median ± IQR)	2.54 ± 1.96
Disease activity (DAS28-ESR, 3 variables) n (%)	
Remission	17 (51.5)
Low disease activity	5 (15.2)
Moderate disease activity	10 (30.3)
High disease activity	3 (3.0)
Current treatment, n (%)	
Topical	29 (50.9)
cDMARD	30 (52.6)
bDMARD	17 (29.8)
Oral retinoid	6 (10.3)
PUVA therapy	0 (0)

bDMARD: biologic disease-modifying anti-rheumatic drug; cDMARD: conventional disease-modifying anti-rheumatic drug; CRP: C-reactive protein; DAS28: disease activity score 28 joints; DLQI: Dermatology Life Quality Index; ESR: erythrocyte Sedimentation Rate; HAQ: Health Assessment Questionnaire; IBD: inflammatory bowel disease; PASI. Psoriasis Area and Severity Index; PsAQoL: Psoriatic Arthritis Quality of Life; PsO: psoriasis; PUVA: psoralen and ultraviolet A.

P20

FOOT INVOLVEMENT AS A PREDICTOR OF WORSE HEALTH ASSESSMENT QUESTIONNAIRE SCORE IN PSORIATIC ARTHRITIS PATIENTS: A RETROSPECTIVE STUDY

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Background/Objective. Foot involvement in psoriatic arthritis (PsA) is common and often develops early, providing important diagnostic and prognostic information. This study aims to evaluate the impact of foot involvement on functionality in patients with PsA.
Methods. A retrospective study including patients with PsA meeting CASPAR criteria followed at our center was divided into two groups: with current or previous foot involvement (assessed clinically or by ultrasound) (group 1) and without current or previous foot involvement (group 2). Sociodemographic, clinical, laboratory, and radiological data were collected. Functionality was assessed through the Health Assessment Questionnaire (HAQ) disability index. Multimorbidity was defined as 2/+ comorbidities. Descriptive and comparative analysis was performed with SPSS® software. The significance level was set at a $p<0.05$.
Results. 150 patients were enrolled (38.7% were women). The mean age 56.92 years. Foot involvement was found in 104 patients (69.3%): arthritis was found in 93 patients (62.0%), with the tibiotarsal joint as the most frequent site (30.0%), enthesitis was found in 28.0% (42 patients), with calcaneal tendonitis as the most frequent manifestation (10.6%). 18.6% (28 patients) had current/previous dactylitis. Radiological findings showed osteopenia in 26.6% of patients, and symmetrical joint space narrowing in 12.0%, 20.0%, and 10.0% in tibiotarsal, metatarsophalangeal, and Interphalangeal joints, respectively. Erosions were found in 28.6% of patients.
Multimorbidity and extra-articular manifestations were more frequent in group 1 ($p=0.02$, $p=0.03$, respectively). Patients with foot involvement had higher C-reactive protein and erythrocyte sedimentation rates ($p=0.01$) and were more frequently under steroids ($p=0.01$) and NSAIDs ($p=0.02$).
We found statistically significant higher HAQ scores in group 1 [median 1.00, IQR 0.88 (group 1) VS median 0.06, IQR 0.88 (group 2); OR 2.16 CI(1.31-3.47) $p<0.01$];
Conclusion. Patients with foot involvement had higher HAQ disability index scores which highlights the importance of incorporating foot evaluation when measuring disease activity in psoriatic arthritis.

P21

RISK OF SPONDYLOARTHRITIS IN PATIENTS WITH INFLAMMATORY BOWEL DISEASE UNDER TREATMENT WITH BIOLOGICS OR JANUS KINASE INHIBITORS

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Introduction. The occurrence of spondyloarthritis (SpA) in patients with inflammatory bowel disease (IBD) even under treatment with biologics or Janus kinase inhibitors (JAKis) is poorly understood. Thus, we aimed to evaluate the occurrence of and risk factors for SpA in patients with IBD during biologic or JAKi treatment.
Methods. Patients with IBD on biologics or JAKis without a prior SpA diagnosis were included. Then, we analyzed patients who presented with new-onset arthralgia and were referred to our rheumatology clinic based on suspicion of a rheumatic disease. Clinical and laboratory data including radiographs of the sacroiliac joints and human leukocyte antigen B27 (HLA-B27) testing were collected. We compared the patients diagnosed with SpA and those without.
Results. Of 1,649 patients with IBD under biologic or JAKi treatment (1,335 with Crohn's disease [CD], 314 with ulcerative colitis [UC]), 96 (5.8%) were excluded due to a prior SpA diagnosis. Among the remain-

ing 1,553 patients, 106 (6.8%) developed arthralgia during IBD treatment including biologics or JAKi, and 30 (1.9%) were finally diagnosed with SpA (20: axial SpA, 10: peripheral SpA). HLA-B27 positivity was more common in the SpA group (23.3%) than in the non-SpA group (1.3%, p=0.001). Risk factors for SpA in these patients included a partial Mayo score for UC at the time of onset of musculoskeletal symptoms (HR 1.568, p=0.028) and HLA-B27 positivity (HR 3.697, p=0.004).

Conclusion. During a median follow-up of 5.2 years, 6.8% of IBD patients undergoing biologic or JAKi treatment developed musculoskeletal symptoms, with a third subsequently diagnosed with SpA. HLA-B27 positivity and higher UC disease activity were associated with an increased risk of SpA.

P22

EVALUATION OF THE AXIAL SPINE IN PATIENTS WITH SPONDYLOARTHRITIS ACCOMPANYING CHRONIC KIDNEY DISEASE

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Introduction. Axial spondyloarthritis (axSpA) is an inflammatory disease characterized by new bone formation on the axial joint, which reflects enthesitis. In patients with axSpA, the interpretation of radiographic images of the axial spine is complicated by comorbidities affecting bone metabolism, such as chronic kidney disease (CKD). The aim of this study was to compare spinal changes in axSpA-CKD patients receiving bDMARD therapy with axSpA-only and CKD-only (without axSpA) patients.

Method. 3207 SpA patients using bDMARD treatment until August 2023 were included in this study from HUR-BIO registry retrospectively. A total of 34 (1.06 %) had CKD (axSpA-CKD patients). Sixty-eight age and sex matched-axSpA patients without CKD (axSpA-only) were selected to compare. AxSpA patients were classified according to the Modified NY and the Assessment of SpondyloArthritis International Society criteria. 42 CKD-only patients followed in the nephrology outpatient clinic were selected by matching according to age, gender, and GFR levels. CKD was defined as patients with a GFR level below 60 ml/min according to modified Modification of Diet in Renal Disease (MDRD) formula or a GFR level above 60 ml/min and proteinuria above 500 mg/day that persists for more than three months. Sacroiliac radiographs and lumbar/cervical and hip radiographs of all groups were evaluated by three rheumatologists. They were blinded by each other (with less than 5 years of experience (GSU) and with 15 years of experience (LK) and (UK). All participants had the intra and inter rater agreement. Cervical/lumbar AP-lateral and pelvic X-rays were evaluated in terms of erosion, squaring, sclerosis, syndesmophyte (any), bamboo spine, mSASSS, and BASRI scores.

Results. The study includes 34 axSpA-CKD patients (mean age 49.5 years). In the axSpA-CKD patients, radiographic damage and hip involvement were more common compared to axSpA-nonCKD patients. Syndesmophytes were seen in 18/28 (64.2%) axSpA-CKD patients (Table I).

A total of 8/38 (20%) of CKD-only patients had syndesmophytes-like osteoproliferation (n=5/8 (62.5%) marginal, n=3/8 (37.5%) nonmarginal). There was no difference in the presence of syndesmophytes in axSpA-nonCKD patients compared to CKD-only patients (p=0.24). In addition, there was no difference in this group in terms of median mSASSS scores (p=0.062) (Table II).

Conclusion. Enthesial changes in the axial spine are more common in axSpA-CKD patients than axSpA-nonCKD patients. In the presence of CKD accompanying SpA, the contribution of CKD to new bone formation should be taken into consideration in the evaluation of the lumbar and cervical spine.

P22: Table I. Comparison of clinical features between patients with axSpA-CKD and axSpA-nonCKD.

		axSpA-CKD patients n=34 (%)	axSpA-non CKD patients n=68 (%)	p
Age, mean (±SD)		49.5 (13.4)	49.4 (12.5)	0.94
Sex, male		24 (70.5)	46 (68.4)	0.6
Symptom duration,years, (IQR)		17.45 (0.8-40.5)	14.5 (0.4-49.7)	0.03
Disease duration,years, (IQR		16.19 (0.97-38.3)	13.4 (0.34-45.22)	0.04
Follow-up duration, years, (IQR)		4.85 (0.33-13.95)	4.11 (0.1-13.68)	0.22
BMI, mean, (SD)		28.5 (6.69)	29.1 (5.05)	0.67
Smoking,		19/30 (63.3)	39/66 (59.1)	0.43
Smoking package/year, median (IQR)		12.5 (0.1-98)	10 (0.3-68)	0.36
FMF, n (%)		12/34 (35.3)	2/68 (2.9)	<0.001
CKD-related findings	Creatinine, mean (±SD)	1,55 (0,6)	NA	NA
	Proteinuria, mg/day, mean (±SD)	1647 (1200)	NA	NA
	Amyloidosis	15/39 (38.4)	0/67	<0.001
SpA-related findings	Uveitis,	6 /34 (20)	3/66 (4.5)	0.025
	IBH	2/34 (6.7)	7/59 (10.6)	0.42
	Enthesitis	3(8.8)	4 (5.9)	0.4
	Dactylitis	0	1 (1.5)	0.9
Comorbid Diseases	Diabetes mellitus	7/33 (29.4)	9/67 (13.4)	0.23
	Hypertension	36/33 (78.8)	18/67 (26.9)	<0.001
	Coronary artery disease	12/33 (36.4)	13/65 (20)	0.067
	Thyroid diseases	7/28 (25)	10/55 (18.2)	0.32
	Hyperlipidemia	13/18 (41.9)	28/59 (47.5)	0.39
	Asthma/COPD	4/33 (12.1)	2/60 (3.2)	0.17
Disease activity and function index	ASDAS-CRP at bDMARD initiation, mean (±SD)	3.04 (0.6)	3.22 (1.5)	0.6
	ASDAS-CRP at last visit, mean (±SD)	2.96 (1.33)	2 22(1.2)	0.13
	BASFI at bDMARD initiation, mean (±SD)	4.9(2.55)	2.3(1.4)	0.007
	BASFI at last visit, mean (±SD)	4.3 (2.9)	3.8 (2.6)	0.44
	Pain, median, (IQR)	40 (10-90)	70 (10-100)	0.11
	Patient global, median (IQR)	40 (10-80)	50 (10-100)	0.15
	Fatigue, median (IQR)	40 (10-80)	50 (0-100)	0.65
Radiographic findings	Syndesmophyte(ever)	18/28 (64.2)	13/67 (19.4)	0.002
	bamboo spine	11/27 (40.7)	7/53 (13.2)	0.005
	BASRI, hip, median	13/21 (61.9)	9/59 (15.2)	<0.001
	BASRI, total, median	4 (0-8)	0 (0-8)	<0.001

SD: standard deviation; CRP: C-reactive protein; ASDAS: Ankylosing Spondylitis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASRI: Bath Ankylosing Spondylitis Radiology Index; mSASSS: modified Stoke Ankylosing Spondylitis Spine Scores; IBH: inflammatory bowel disease; CKD: chronic kidney disease

P22: Table II. Comparison of radiological features of patients

	axSpA-CKD patients n=34	AxSpA-non CKD patients n=68	CKD-only patients n=38	p1*	p2¶
Age, mean (±SD)	49.5 (13.4)	49.4 (12.5)	48.2 (11.7)	0.86	0.82
Sex, male	24 (70.5)	46 (68.4)	27 (71)	0.52	0.46
Syndesmophyte(ever)	18/28 (64.2)	13 /52 (25)	8 /38 (21.0)	<0.001	0.24
bamboo spine	11 (40.7)	7 (13.2)	0	<0.001	0.03
mSASSS, servical, median (IQR)	12 (0-36)	2 (0-36)	1.5 (0-6)	0.005	0.051
mSASSS, lumbar median (IQR)	24 (0-36)	4 (0-24)	2 (0-6)	<0.001	0.005
mSASSS, total, median (IQR)	41 (0-72)	6 (0-60)	2.5 (0-8)	<0.001	0.062

* Comparison with axSpA-CKD and axSpA-nonCKD patients
¶ Comparison with axSpA-nonCKD and CKD-only patients

P23

GENDER DIFFERENCES IN THE INITIATION AND PERSISTENCE OF THE FIRST BDMARD IN SPONDYLOARTHRITIS: AN 18-YEAR FOLLOW-UP STUDY

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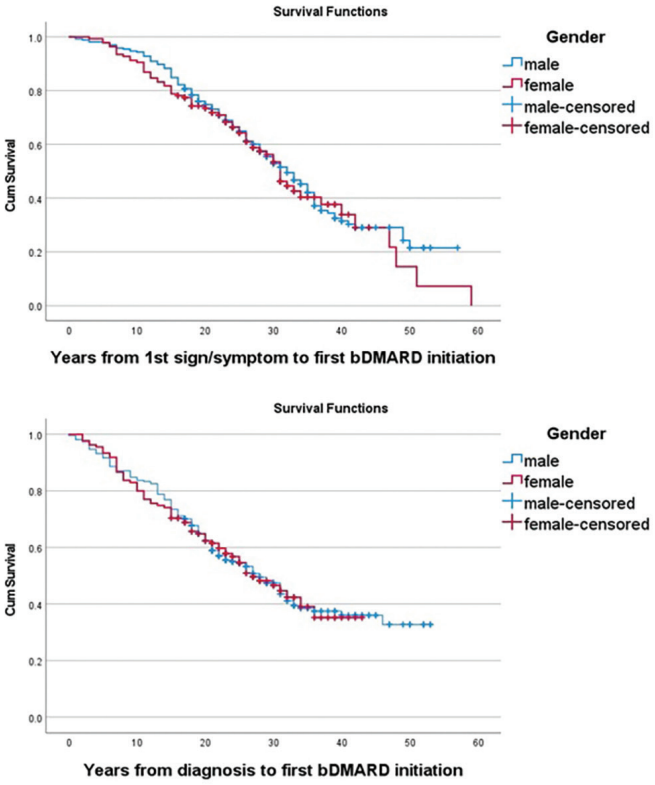
Introduction. Spondyloarthritis (SpA) is a family of chronic inflammatory disease that affects both men and women, but gender differences in treatment initiation and response are still unclear [1]. This study aims to evaluate the delay in the initiation and retention rate of biologic disease-modifying antirheumatic drugs (bDMARDs) in men versus women with SpA, over an 18-year of follow-up period.
Methods. A cohort of patients diagnosed with SpA, initially assessed during a baseline visit in 2004-2007 as part of the REGISPONSER registry, was re-assessed 18 years later in a single follow-up visit as part of the REGISPON-3 study. Kaplan-Meier analysis compared the time to bDMARDs initiation from the first symptom or sign and from the diagnosis between male and female. Cox regression assessed factors influencing bDMARDs initiation.
Results. The cohort consisted in 271 men and 140 women. No significant differences were found between genders in terms of age of diagnosis, age at disease onset or diagnosis delay. However, men had a significantly longer evolution time since first sign/symptom and longer duration of illness since diagnosis. A total of 53% men and 51.1% women initiated bDMARDs during the follow-up. Kaplan-Meier analysis showed no significant difference in median time to bDMARDs initiation from first symptom or diagnosis (Fig. 1). Men persisted longer on the first bDMARD (72 vs 65 months, p=0.038). Cox regression identified ASDAS-CRP, BASDAI, and HLA-B27 as significant predictors for the initiation of bDMARDs following diagnosis (Table I).
Conclusion. The findings suggests that while gender may not influence biological treatment initiation, it may affect its persistence. Further research is needed to explore additional predictors and their impact on treatment outcomes.
Acknowledgements. The Assessment of Spondylarthritis International Society (ASAS) supported Diana Maria Margareta Moldovan with a research fellowship.

Reference
1. https://doi.org/10.1038/s41584-022-00833-0.

P23: Table I. Cox regression analysis showing predictive factors of bDMARDs initiation from diagnosis.

Covariates	Hazard ration	95% CI	p-value
ASDAS-CRP	1.75	1.36 - 2.24	< 0.001
BASDAI	0.84	0.75 - 0.94	0.003
HLA-B27	0.57	0.38 - 0.84	0.005
Gender	1.13	0.80 - 1.60	0.47
Overall model significance	-	-	< 0.001

ASDAS-CRP: Ankylosing Spondylitis Disease Activity Score using C-reactive protein
BASDAI: Bath Ankylosing Spondylitis Disease Activity Index
HLA-B27: Human Leukocyte Antigen B27



P23: Fig. 1. Kaplan-Meier analysis.

P24

FREQUENCY OF EXTRA-ARTICULAR MANIFESTATIONS OF SPONDYLOARTHRITIS AND ASSOCIATED FACTORS IN THE DEMOCRATIC REPUBLIC OF CONGO

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Introduction/Objective. To determine the prevalence of extra-articular manifestations (EAMs) of spondyloarthritis in Kinshasa, Democratic Republic of Congo, and to identify the factors associated with the presence of EAMs.
Materials and methods. A descriptive cohort of SpA patients attending the University Hospital of Kinshasa according to Amor, ESSG or ASAS criteria followed from 2013 to present. Epidemiological data (age, sex, duration of disease), clinical signs including sacroiliitis, spondylitis, enthesopathy, dactylitis were recorded. EAMs such as acute anterior uveitis (AAU), psoriasis, urogenital infection, digestive infection) were collected. Inflammatory parameters (ESR and CRP) and HLA-B27 were investigated. Univariate and multivariate analyses were performed to determine the association between variables.
Results. Among a total of 285 SpA patients (mean age: 38.4 ±10.6 years, duration of the disease: 8.1±3.5 years; sex ratio 1 male/1.4 female), the frequency of EAMs 23.4% for urogenital infections, 15.3% for AAU, 1.8% for psoriasis and 1.1% for IBD. Factors associated with EAMs were a CRP level greater than 6mg/L and BASDAI greater than 4. HLA-B27 antigen was negative in a total of 204 patients tested in this cohort.
Markers and high disease activity were associated with the presence of EAMs.
Discussion. The very frequent infectious context could explain the higher frequency of infection as extra-articular manifestations. The absence of the HLA-B27 antigen could partly explain the low prevalence of EAMs.
Conclusion. This study confirms the rarity of EAMs and the absence of HLA-B27 antigen in the population of SpA patients tested in our setting.

P25

DEVELOPMENT AND VALIDATION OF MODIFIED BASMI: AN AI POSE ESTIMATION-BASED MOBILE APPLICATION TO MEASURE SPINAL MOBILITY IN AXIAL SPONDYLOARTHROPATHY

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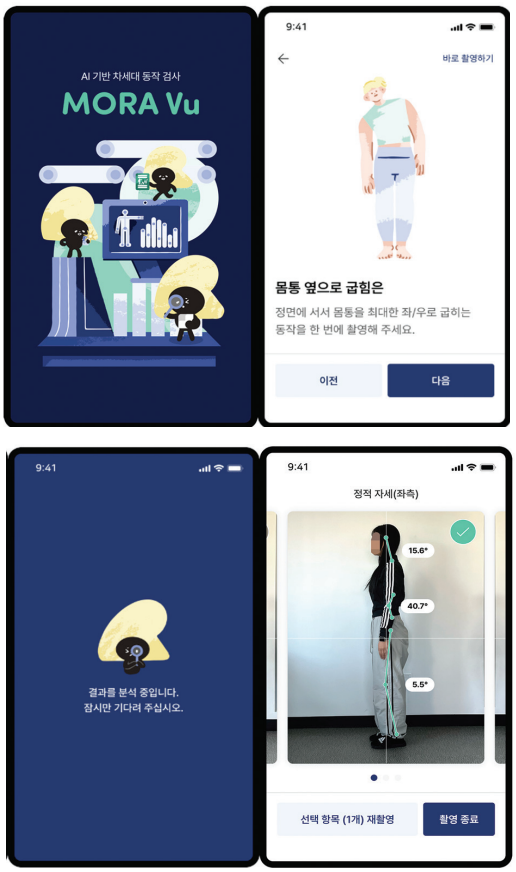
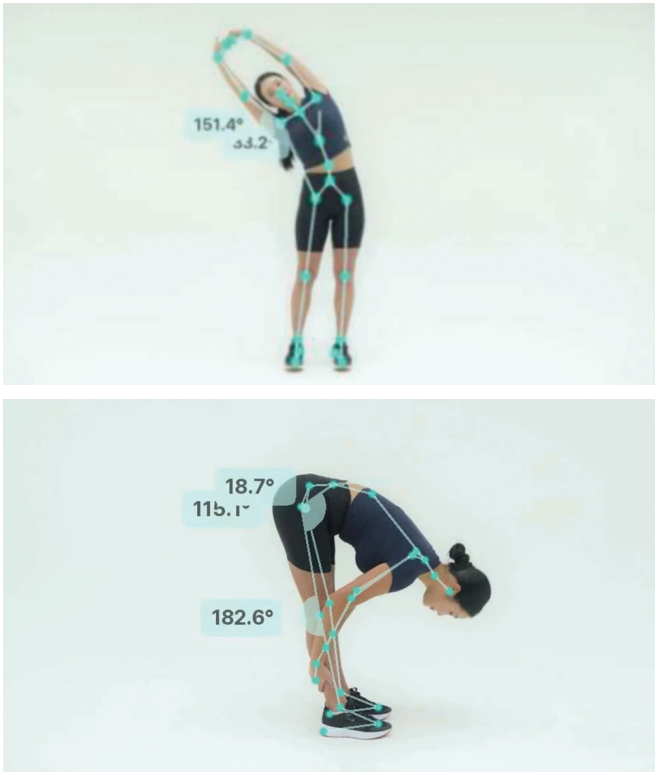
Introduction/Objective. Axial spondyloarthritis (axial SpA) patients require longitudinal monitoring of spinal mobility. The Bath Ankylosing Spondylitis Metrology Index (BASMI) is the gold standard, but its time-consuming and complex nature hinders routine use. This study aimed to validate a mobile application-based modified BASMI (mBASMI) using AI pose estimation technology for faster and clinically relevant assessment of axial SpA.
Materials and methods. This prospective study recruited 50 axial SpA patients. All participants underwent both traditional BASMI and a modified BASMI (mBASMI) assessment using the mobile application MORA-Vu (Fig. 1). The MORA-Vu app employs a pose estimation model to evaluate spinal mobility through the camera of a mobile device, eliminating the need for wearable sensors (Fig. 2). Similar to traditional BASMI, mBASMI measures lumbar flexion and side flexion. However, instead of distance, mBASMI quantifies these movements using angles. Additionally, mBASMI measures the costovertebral angle instead of tragus-to-wall distance and hip abduction angle instead of intermalleolar distance. Cervical lateral flexion angle is assessed in mBASMI instead of cervical rotation.
To assess the validity, feasibility, and reliability of mBASMI, we compared it with traditional BASMI measurements in the same group of participants. This comparison will determine how well mBASMI scores correlate with traditional BASMI scores.
Results. As this is a prospective study expected to be completed by September 2024, final results are not yet available. However, preliminary findings indicate that the AI pose estimation technology within mBASMI achieves an accuracy of ± 5 degrees.



P25: Fig. 1. MORA Vu, a motion analysis software medical device for mobile application.

Conclusion. Our study is the first, to our knowledge, to investigate the validity of a functional assessment using an AI pose estimation-based mobile application in patients with SpA. The promising ± 5 degree accuracy in the preliminary mBASMI assessment suggests it has the potential to be a faster, more feasible alternative to traditional BASMI in clinical settings.



P25: Fig. 2. AI pose estimation powered assessment of modified Bath Ankylosing Spondylitis Metrology Index (BASMI)

P26

THE RELATIONSHIP BETWEEN DISEASE BURDEN AND MENTAL HEALTH IN AXIAL SPONDYLOARTHRITIS

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Introduction/Objective. Comorbidities may worsen the burden of axial spondyloarthritis (axSpA). We aimed to examine the availability of psychiatric care in axSpA patients positively screened for psychiatric comorbidity and to determine the overall impact of mental health on the disease outcomes.
Patients and methods. This is a cross-sectional study. Validated Czech version of the Mini-International Neuropsychiatric Interview (M.I.N.I.), Beck depression Inventory (BDI-II) and Beck Anxiety Inventory (BAI) were administered to all participants. The axSpA measures included radiographic findings, pharmacotherapy, disease duration), parameters of activity (ASDAS-C-reactive peptide, BASDAI) and patient-reported outcomes (PRO´s: 5D-EQ, BASFI, HAQ). Serum levels of the Brain-derived neurotrophic factor (BNDF) were analysed by ELISA.
Results. The total of 227 individuals, 111 with axSpA and 116 age and sex-matched controls were included. Positive scores for any M.I.N.I. were found in 33.00% of axSpA and in 7.70% of controls (p<0.0001). In psychiatric care were 13.50% of axSpA patients and 11.10% of control subjects. Majority of axSpA and control subjects refused psychiatric consultation, for the following reasons: decided to postpone it for later (29.70 vs 33.30%,

respectively), saved it just for case of deterioration (51.30% vs 44.40%, respectively). The mean BDI-II and BAI scores were significantly higher in axSpA patients (6.64 and 7.20, respectively) than in control subjects (2.80 and 3.90, respectively), all $p<0.01$. Serum levels of the BDNF were lower in axSpA vs. controls (mean 210.00 vs 229.20 ng/ml, $p<0.05$). The axSpA patients with positive M.I.N.I. were more active, with radiographic sacroiliitis, with poor outcomes in PRO´s and more severe depression and anxiety burden (Table I).

Conclusion. Despite the several limits, our study underscores the importance of addressing mental health needs in axSpA patients. The improvement of mental health might lower the burden of rheumatic disease.

Acknowledgements. This study was supported by Czech Health Research Council NU21-09-00297.

P26: Table I. The clinical variability in people with axial spondyloarthritis

	M.I.N.I. positive (n=37)	M.I.N.I. negative (n=74)	*p*-value
Age	44.40 (9.90)	41.10 (10.40)	ns
Male (%)	59.50	66.20	ns
Radiographic sacroiliitis (%)	70.30	41.00	0.005
Disease duration since first symptom (days)	5433 (3568)	<4557 (2724)	ns
Disease duration since diagnosis (days)	2893 (2073)	2483 (1441)	ns
BMARDs therapy (%)	16.20	23.00	ns
ASDAS-CRP	2.69 (1.20)	1.70 (0.92)	<0.0001
BASDAI	4.70 (1.90)	2.20 (1.70)	<0.0001
BASFI	3.50 (2.70)	1.15 (1.14)	<0.0001
5D EQ	0.550 (0.270)	0.820 (0.170)	<0.0001
HAQ	0.77 (0.60)	0.26 (0.40)	<0.0001
BDI-II	12.86 (7.01)	3.53 (3.90)	<0.0001
BAI	14.40 (10.90)	3.60 (3.80)	<0.0001
BDI-II moderate-severe depression (%)	11.76	0.00	0.01
BAI moderate-severe anxiety (%)	17.65	1.35	0.005
CRP (mg/l)	9.97 (14.90)	4.54 (5.10)	ns
BDNF (ng/ml)	210.6 (93.90)	209.8 (95.60)	ns

Unless otherwise stated, data are provided as mean and standard deviation in brackets

P27

PSYCHOMETRIC EVALUATION OF THE SERBIAN RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS, FORMER ANKYLOSING SPONDYLITIS QUALITY OF LIFE (ASQOL) QUESTIONNAIRE AND ITS CORRELATIONS WITH DISEASE ACTIVITY AND FUNCTIONAL STATUS INDICES

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Objective. To evaluate psychometric properties of the radiographic Axial Spondyloarthritis Quality of Life (r-axSpAQoL) questionnaire- former ASQoL- Serbian version and to relate it with disease activity and functional status indices.

Methods. From the Institute of Rheumatology, Belgrade 60 randomly chosen r-axSpA patients filled the questionnaire two times two weeks apart. Construct validity embraced convergent validity by Notthingham Health Profile as a comparator scale and known-group validity by disease activity, overall health status and the occurrence of flares comparisons. For Internal validity Cronbach's alpha coefficient was used, for External validity test-retest reliability. Regression analyses were determined between r-axSpAQoL and *ASDAS*, *BASDAI*, *BASFI*, Schober's test, respiratory index and *SPARCC* index.

Results. There was a significant correlation between the r-axSpAQoL and NHP domains of pain (r^2=0.79), emotional reactions (r^2=0.78), physical activity (r^2=0.77) and energy (r^2=0.75). Cronbach alpha coefficents were 0.95 and 0.91 at Visit 1 and Visit 2. Spearman correlation coefficient for test-retest reliability was 0.84. Serbian r-axSpAQoL differentiated patients with various status of general health ($p<0.05$), disease severity ($p<0.05$), occurrence of flares ($p<0.05$). Independent regression analyses settled ASDAS, BASFI and BASDAI as predictors and r-axSpAQoL as an outcome variable, showing highly positive relations between r-axSpAQoL and ASDAS (R^2=0.403, F=32.3; DF1=1, DF2=48; unbeta 3.078, $p<0.001$); BASDAI (R^2=0.564, F=74.9, DF1=1, DF2=58; unbeta 2.268, $p<0.001$); and BASFI (R^2=0.444, F=46.4, DF1=1, DF2=58, unbeta 1.859, p<0.001) (Fig. 2). Relations between r-axSpAQoL and SPARCC and the respiratory index were also significant but unacceptable, given the low level of the correlation coefficient (R^2=0.091, F=5.78; DF1=1, DF2=58; unbeta 1.488, $p=0.019$ and R^2=0.114, F=7.47; DF1=1, DF2=58; unbeta -1.267, $p=0.008$, respectively). There were no relations with Schober's test.

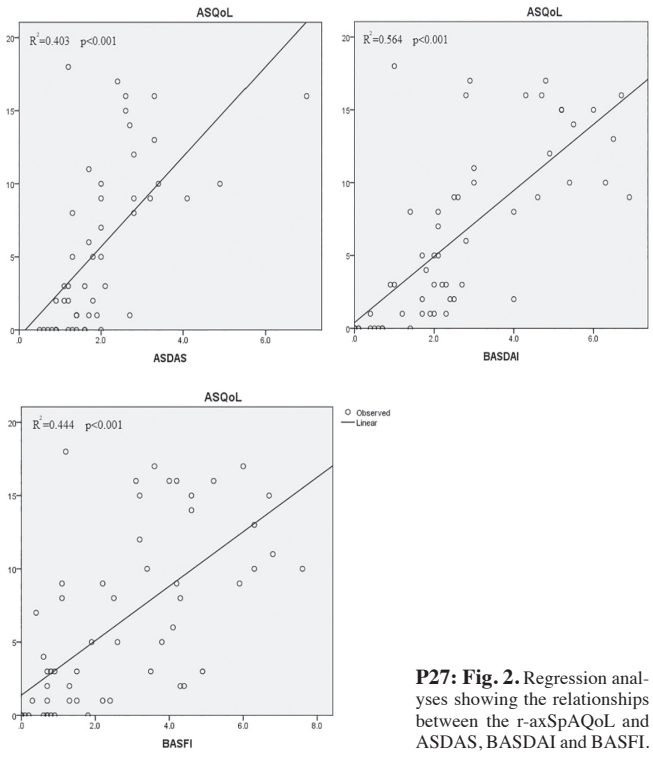
Conclusion. The Serbian version of the r-axSpAQoL demonstrated excellent psychometric properties and high relations with disease activity and functional status indices, affirming it as a reliable tool for clinical research and routine clinical practice.

Disclosure of interest. None declared.

P27: Table I. Correlations between radiographic Axial Spondyloarthritis Quality of Life (r-axSpAQoL) and different domains of Nottingham Health Profile

	Median	IQR	% of patients scoring min	% of patients scoring max	Correlation coeffitients with r-axSpAQoL (r^2)
		n=60 patients			
r-axSpAQoL TIME 1	3.5	1.0 – 10.0	23.3	1.7	-
NHP					
Energy level	33.3	0 – 66.7	48.3	20.0	0.75
Pain	18.8	0 – 62.5	38.3	8.3	0.79
Emotional reactions	0	0 – 22.2	53.3	0	0.78
Sleep	0	0 – 40.0	55.0	5	0.52
Social isolation	0	0 – 0	80.0	0	0.50
Physical mobility	12.5	0 – 37.5	33.3	0	0.77
		n=59 patients			
r-axSpAQoL TIME 2	3.0	0 – 18	25.4	1.7	-

r-axSpAQoL: Radiographic Axial Spondyloarthritis Quality of Life Questionnaire; NHP: Nottingham Health Profile



P27: Fig. 2. Regression analyses showing the relationships between the r-axSpAQoL and ASDAS, BASDAI and BASFI.

P28

DOES PSORIASIS MODIFY PHENOTYPE IN PSORIATIC ARTHRITIS PATIENTS?

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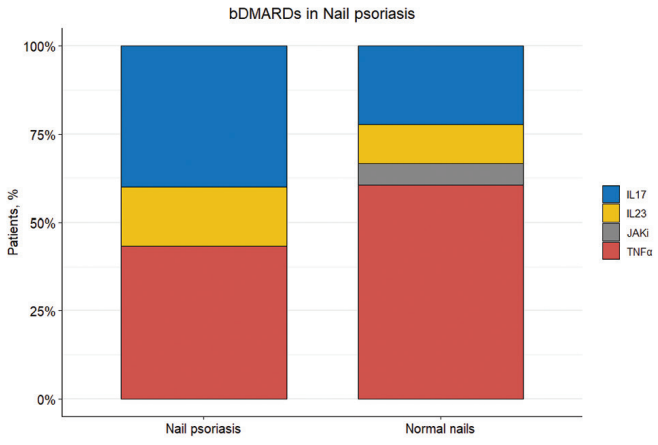
Introduction. Psoriasis (PsO) is a skin disease with different possible presentations. About 30% of PsO patients develop Psoriatic arthritis (PsA), which could also affect patients with a family history of Pso without skin manifestations. Data on clinical PsA phenotype according to the PsO presentation are scarce, so we investigated the differences in epidemiological, clinical and therapeutic factors between PsA patients who have/not have PsO and between PsA patients presenting with different PsO subtypes.
Methods. We made a cross-sectional analysis of the PsA patients referring to our spondyloarthritis outpatient clinic; all of them were classified as PsA according to the CASPAR criteria. We made a comparison between patients with and without PsO and, in the one presenting PsO, between different PsO subtypes, analyzing epidemiological and clinical factors, number and types of previous treatments and comorbidities.
Results. We enrolled 204 patients (M:F=114:90). PsO was present in 156 of them. Patients with PsO were more frequently male and older, presenting a higher number of comorbidities, with significantly higher prevalence of hyperuricemia and high arterial blood pressure. When PsO was present, Methotrexate, Leflunomide and Cyclosporine were more used while Janus Kinase inhibitors were less frequently prescribed. PsA patients with plaque PsO (the most frequent subtype) had higher BMI and presented more sacroileitis. Nail PsO was associated with a worse DAPSA score, sonographic tenosynovitis and a higher use of IL-17 inhibitors (see Fig. 1). Palmoplantar PsO was positively associated with the use of IL-17 inhibitors. Finally, patients with guttate PsO presented lowest DAPSA scores and higher prevalence of smoking habits.
Conclusion. PsA patients present different clinical history, phenotype and therapeutic choice according to the presence/absence of PsO or the subtypes of PsO. Physicians need to consider those aspects for tailoring their diagnostic and therapeutic choices.



P28: Fig. 1.

P29

INCIDENCE RATE OF RECURRENT CARDIOVASCULAR EVENTS IN PATIENTS WITH ANKYLOSING SPONDYLITIS AND THE EFFECT OF TUMOR NECROSIS FACTOR INHIBITORS

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Introduction. Patients with ankylosing spondylitis (AS) have a 1.4-fold higher risk of cardiovascular events than the general population and cardiovascular events are important comorbidities that should be taken in to account when treating patients with AS. This study was performed to assess the incidence rate of recurrent cardiovascular events in patients with ankylosing spondylitis (AS) with prior history of cardiovascular events, and to evaluate the effect of tumor necrosis factor inhibitors (TNFi) on the risk of recurrent cardiovascular events.
Methods. This was a nationwide cohort study using data from the Korean national claims database. Data on patients with AS who had history of cardiovascular events after being diagnosed with AS were extracted from the database. The outcome of interest was recurrence of cardiovascular events (myocardial infarction or stroke). Patients were followed-up from the index date (date of first cardiovascular event) to the date of cardiovascular event recurrence, last date with claims data, or December 31, 2021, whichever came first. Incidence rate of recurrent cardiovascular events was calculated. Inverse probability weighted Cox model was used to assess the effect of TNFi exposure on the risk of recurrent cardiovascular events.
Results. A total of 413 patients (TNFi non-exposure = 338; and TNFi exposure = 75) were included. The incidence rate of recurrent cardiovascular events was 32.00 per 1,000 person-years (TNFi non-exposure = 35.97 per 1,000 person-years; and TNFi exposure = 18.51 per 1,000 person-years). In the inverse probability weighted Cox model, TNFi exposure was significantly associated with a lower risk of recurrent cardiovascular events (hazard ratio 0.29, 95% confidence interval 0.10–0.87, p=0.027).
Conclusion. Incidence rate of recurrent cardiovascular events in patients with AS is substantial. TNFi exposure was associated with a lower risk of recurrent cardiovascular events.

P30

PREDICTIVE VALIDITY OF DATA-DRIVEN DEFINITIONS FOR ACTIVE AND STRUCTURAL LESIONS IN THE SI JOINTS TYPICAL FOR AXIAL SPA: A 2-YEAR FOLLOW-UP IN THE SPONDYLOARTHRITIS CAUGHT EARLY COHORT

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Introduction. Data-driven definitions for active and structural lesions characteristic of axial spondyloarthritis (axSpA) on MRI of the SI joints (MRI-SIJ) were proposed by de Hooge and the Assessment of Spondyloarthritis international Society MRI (ASAS-MRI) group (1, 2). This study aims to evaluate the predictive validity of these proposed definitions for active and structural MRI-SIJ lesions in early axSpA with a 2-year diagnosis.
Methods. Following 2 years of follow-up, patients with chronic back pain from the SPondyloArthritis Caught Early (SPACE) inception cohort were diagnosed by a rheumatologist as axSpA or no-axSpA (3). Baseline MRI-SIJ

were assessed by three central readers for bone marrow edema (BME), erosions, and fat lesions. The different definitions of de Hooge and the ASAS MRI group were employed (Fig. 1). We calculated sensitivity, specificity, positive and negative predictive values (PPV and NPV) for each definition. Definitions with specificity and PPV ≥95% were considered validated.

Results. Analysis of 643 patients (age 30 (SD 8) years; 39% males; 52% axSpA) showed low lesion prevalence within the cohort, particularly for structural lesions (2%-14%) (Fig. 2). All proposed definitions by de Hooge met the threshold. While most proposed ASAS-MRI group definitions met the threshold, erosions affecting ≥2 consecutive slices did not. Similar results were obtained when analysing the assessment of the definitions by three individual readers.

Conclusion. Structural lesions were infrequently present in early axSpA. All proposed ASAS-MRI group definitions for structural MRI-SIJ lesions, except for erosions in ≥2 consecutive slices were validated in early axSpA. The definitions of de Hooge showed similar performance to those of the ASAS MRI group, however, they are easier to apply and thus have a higher feasibility.

References

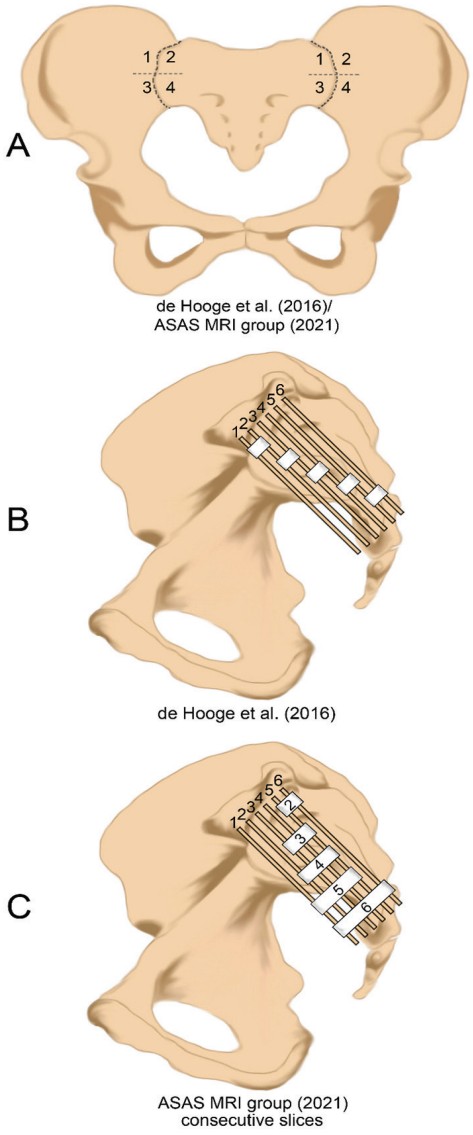
1. DE HOOGE M *et al*.: *Ann Rheum Dis* 2016; 75(7): 1308-14.
2. MAKSYMOWYCH WP *et al*.: *Rheumatology* (Oxford) 2021; 60(10): 4778-89.
3. MARQUES ML *et al*.: *Ann Rheum Dis* 2024; 83: 589-98.

Baseline MRI data	$n_{tot.\ pos.}$ (%)	axSpA at follow-up (yes/no) n=643 (52% axSpA)				Validated
		Sensitivity	Specificity	PPV	NPV	
Erosions ≥1	57 (9%)	15.5	98.4	91.2	51.7	
Erosions ≥2	34 (5%)	9.6	99.4	94.1	50.2	
Erosions ≥3	18 (3%)	5.4	**100.0**	**100.0**	49.3	✓
Fat lesions ≥1	73 (11%)	20.6	**98.7**	**94.5**	53.3	✓
Fat lesions ≥2	57 (9%)	16.7	**99.7**	**98.2**	52.4	✓
Fat lesions ≥3	42 (7%)	12.2	**99.7**	**97.6**	51.1	✓
Fat lesions and/or erosions ≥1	101 (16%)	27.5	97.1	91.1	55.2	
Fat lesions and/or erosions ≥2	77 (12%)	22.1	**99.0**	**96.1**	53.9	✓
Fat lesions and/or erosions ≥3	59 (9%)	17.3	**99.7**	**98.3**	52.6	✓
Fat lesions and/or erosions ≥4	47 (7%)	13.7	**99.7**	**97.9**	51.5	✓
Fat lesions and/or erosions ≥5	40 (6%)	11.6	**99.7**	**97.5**	50.9	✓

P30: Fig. 2a. Definitions proposed by de Hooge et al. (2016) for structural lesions in MRI SI joints typical for axSpA against the rheumatologist's diagnosis at 2-year follow-up, using the consensus between readers. (n=643). The yellow color indicates a definition was proposed by the de Hooge et al. (2016). The blue color emphasized proposed definitions that are validated by meeting the threshold consensus of a specificity of ≥95%. and PPV of ≥95%. Bold values and √ indicate that the definition has the required specificitty and PPV. AxPsA: axial spondyloarthritis; NPV: negative predictive value; PPV: positive predictive value; Tot. pos: total positives, including true positives and false positives.

Baseline MRI data	$n_{tot.\ pos.}$ (%)	axSpA at follow-up (yes/no) n=643 (52% axSpA)				Validated
		Sensitivity	Specificity	PPV	NPV	
BME score in ≥1 SIJ quadrants	147 (23%)	42.1	**98.1**	**95.9**	60.9	✓
BME score in ≥2 SIJ quadrants	104 (16%)	30.4	**99.4**	**98.1**	56.8	✓
BME score in ≥3 SIJ quadrants	72 (11%)	21.2	**99.7**	**98.6**	53.8	✓
BME score in ≥4 SIJ quadrants	47 (7%)	14.0	**100.0**	**100.0**	51.7	✓
BME in ≥2 consecutive slices	131 (20%)	37.6	**98.4**	**96.2**	59.2	✓
BME in ≥3 consecutive slices	104 (16%)	30.4	**99.4**	**98.1**	56.8	✓
BME in ≥4 consecutive slices	85 (13%)	25.4	**100.0**	**100.0**	55.2	✓
Erosion score in ≥1 SIJ quadrants	93 (14%)	25.4	97.4	91.4	54.5	
Erosion score in ≥2 SIJ quadrants	40 (6%)	11.6	**99.7**	**97.5**	50.9	✓
Erosion score in ≥3 SIJ quadrants	21 (3%)	6.0	**99.7**	**95.2**	49.4	✓
Erosion score in ≥4 SIJ quadrants	10 (2%)	3.0	**100.0**	**100.0**	48.7	✓
Erosion in ≥2 consecutive slices	57 (9%)	15.5	98.4	91.2	51.7	
Erosion in ≥3 consecutive slices	21 (5%)	8.7	99.4	93.5	50.0	
Erosion in ≥4 consecutive slices	13 (2%)	3.9	**100.0**	**100.0**	48.9	✓
Fat lesion in ≥1 SIJ quadrants	85 (13%)	23.9	98.4	94.1	54.3	
Fat lesion in ≥2 SIJ quadrants	59 (9%)	17.3	**99.7**	**98.3**	52.6	✓
Fat lesion in ≥3 SIJ quadrants	40 (6%)	11.6	**99.7**	**97.5**	50.9	✓
Fat lesion in ≥4 SIJ quadrants	24 (4%)	7.2	**100.0**	**100.0**	49.8	✓
Fat lesion in ≥5 SIJ quadrants	12 (2%)	3.6	**100.0**	**100.0**	48.8	✓
Fat lesion in ≥2 consecutive slices	73 (11%)	20.6	**98.7**	**94.5**	53.3	✓
Fat lesion in ≥3 consecutive slices	49 (8%)	14.3	**99.7**	**98.0**	51.7	✓
Fat lesion in ≥4 consecutive slices	32(5%)	9.3	**99.7**	**96.9**	50.2	✓

P30: Fig. 2b. Definitions proposed by the ASAS MRI group (2021) for singular active and structural lesions in MRI SI joints typical for axSpA against the rheumatologist's diagnosis at 2-year follow-up, using the consensus between readers. (n=643). The yellow color indicates a definition was proposed by the ASAS MRI group (2021). The blue color emphasized proposed definitions that are validated by meeting the threshold consensus of a specificity of ≥95% and PPV of ≥95%. Bold values and √ indicate that the definition has the required specificity and PPV. AxSpA: axial spondyloarthritis; BME: bone marrow edema; NPV: negative predictive value; PPV: positive predictive value; Tot. pos.: total positives, including true positives and false positives.



P30: Fig. 1. Visual representation of the lesion definitions proposed by de Hooge et al. (2016) and ASAS-MRI group of quadrants and sagittal slices used for scoring lesions in the SI joints.

A: Frontal perspective presenting the individual quadrants per SI joint used by both de Hooge et al. and the ASAS-MRI group.

B: Sagittal view illustrating an SI joint with five rectangles, each corresponding to a pair of two consecutive slices.

C: Sagittal perspective an SI joint with rectangles illustrating five variations in the number of consecutive slices. De Hooge et al. defined a structural lesion only when identified in two consecutive slices within the same quadrant (A) (1). Per structural lesion, this results in five possibilities (B) within each quadrant. The proposed definitions by de Hooge et al. are the number of lesions present across both SI joints. Therefore, the total number of lesions possible is 40 lesions (5 possible lesions x 4 quadrants x 2 SI joints) (1). The ASAS-MRI group proposed definitions in two categories (2). One category is the number of quadrants (A) affected (0-8 quadrants: 4 quadrants x 2 SI joints) by either inflammatory or structural lesions regardless of slice location or number of consecutive slices (2). The second category is the number of consecutive slices (C) affected (2-6 consecutive slices) by either inflammatory or structural lesions within the same quadrant (2).

P31

WILL THE ASAS DEFINITION FR POSITIVE MRI-SIJ LAST AND DO STRUCTURAL LESIONS IN THE MRI-SIJ HAVE ADDITIONAL VALUE? A 2-YEAR FOLLOW-UP IN THE SPONDYLOARTHRITIS CAUGHT EARLY COHORT

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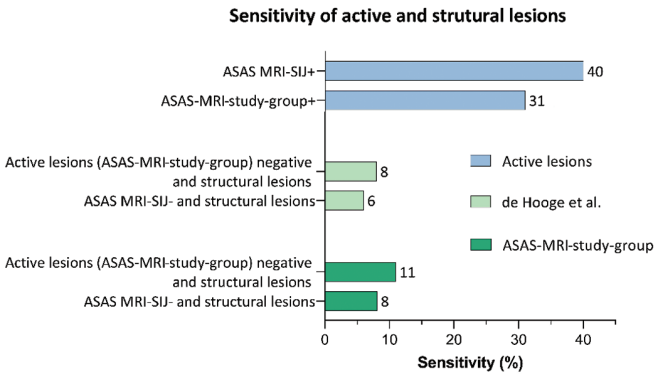
Introduction. Definitions for active (AL) and structural lesions (SL) typical of axSpA, proposed by de Hooge (2016) and the ASAS-MRI-study-group (2021), were assessed for their predictive validity.[1] However, the ASAS-MRI-study-group AL definition has not been compared with the current ASAS definition for positive MRI-SIJ (ASAS-MRI-SIJ+), nor combinations of AL and SL have been assessed. We aimed to compare the proposed ASAS-MRI-study-group definition for AL with that of ASAS-MRI-SIJ+ and evaluate the predictive validity of different combinations of AL and SL.
Methods. Patients with chronic back pain from the SPondyloArthritis Caught Early (SPACE) cohort had a two-year follow-up at which they were diagnosed by a rheumatologist as axSpA or no-axSpA.[2] Three central readers assessed baseline MRI-SIJ for bone marrow edema, erosions, and fat lesions. Combinations of AL or SL were based on the component definitions of de Hooge, ASAS-MRI-study-group and ASAS-MRI-SIJ+ (Table 1). Sensitivity, specificity, positive and negative predictive values (PPV and NPV) were calculated for each combination. Definitions with a specificity and PPV ≥95% met the threshold.

P31: Table I. Validation of combinations of active and/or structural lesions based on definitions proposed by de Hooge and the ASA-MRI-study-group for structural lesions in MRI-SIJ typical for asSpA against the rheumatologist's diagnosis at 2-year follow-up, based on consensus berween readers. (n=643).

Baseline MRI data	$n_{tot.\ pos.}$ (%)	axSpA at follow-up (yes/no) n=643 (52% axSpA) Sensitivity	Specificity	PPV	NPV	Meeting threshold
Active lesions						
Active ASAS-MRI-study-group+	105 (16)	30.7	**99.4**	**98.1**	56.9	✓
ASAS MRI-SIJ+	139 (22)	39.7	**98.1**	**95.7**	59.9	✓
Structural lesions according to de Hooge et al. (2016)						
Structural lesions	55 (9)	16.1	**99.7**	**98.2**	52.2	✓
Active lesions (ASAS-MRI-study-group) positive and structural lesions	28 (4)	8.1	**99.7**	**96.4**	49.9	✓
Active lesions (ASAS-MRI-study-group) negative and structural lesions	27 (4)	8.1	**100.0**	**100.0**	50.0	✓
Active lesions (ASAS-MRI-study-group) positive and/or structural lesions	132 (21)	38.8	**99.4**	**98.5**	59.9	✓
ASAS MRI-SIJ+ and structural lesions	35 (5)	10.1	**99.7**	**97.1**	50.5	✓
ASAS MRI-SIJ- and structural lesions	20 (3)	6.0	**100.0**	**100.0**	49.4	✓
ASAS MRI-SIJ+ and/or structural lesions	159 (25)	45.7	**98.1**	**96.2**	62.4	✓
Structural lesions according to ASAS-MRI study-group (2021)						
Structural lesions	86 (13)	23.9	98.1	93.0	54.2	
Active lesions (ASAS-MRI-study-group) positive and structural lesions	44 (7)	12.5	**99.4**	**95.5**	51.1	✓
Active lesions (ASAS-MRI-study-group) negative and structural lesions	42 (7)	11.3	98.7	90.5	50.6	
Active lesions (ASAS-MRI-study-group) positive and/or structural lesions	147 (23)	42.1	**98.1**	**95.9**	60.9	✓
ASAS MRI-SIJ+ and structural lesions	55 (9)	15.8	**99.4**	**96.4**	52.0	✓
ASAS MRI-SIJ- and structural lesions	31 (5)	8.1	98.7	87.1	49.7	
ASAS MRI-SIJ+ and/or structural lesions	170 (26)	47.8	96.8	94.1	63.0	

ASAS MRI-SIJ+/-, a positive or negative MRI of the sacroiliac joints according to the ASAS definition. Active lesions (ASAS-MRI-study-group) are defined by having bone marrow edema score in ≥4 SIJ quadrants or bone marrow edema in ≥3 consecutive slices. Structural lesions by de Hooge are defined by having ≥3 erosions, ≥3 fat lesions, or ≥5 fat lesions and/or erosions. Structural lesions by the ASAS-MRI-study-group are defined by having an erosion score in ≥3 SIJ quadrants, erosion in ≥2 consecutive slices, fat lesion in ≥5 SIJ quadrants, or fat lesion in ≥3 consecutive slices. Bold values indicate all definitions that meet the threshold consensus for specificity and PPV.



P31: Fig. 1. Added value of structural lesions, compared to active lesions, as measured with sensitivity to idetify patients with asSpA. Details of the individual definitions are further explained in Table I. (n=643).

Results. In total, 643 patients were included (age 30 (SD 8) years; 39% males; 52% axSpA). From the ALs, the ASAS-MRI-SIJ+ performed best by having higher sensitivity (40% vs 31%) with a small loss in specificity compared to the ASAS-MRI-study-group AL (98% vs 99%) (Table 1). All combinations of AL and SL by de Hooge met the threshold, while for the ASAS-MRI-study-group SL, only in presence of AL+. SLs alone contributed only with a small gain in sensitivity, between 6-11% (Fig. 1).
Conclusion. The current ASAS-MRI-SIJ+ definition outperforms the new ASAS-MRI-study-group AL proposal. SLs have only a small gain in sensitivity for identifying axSpA. If combining AL and SL, ASAS-MRI-SIJ+ and de Hooge SL show the best performance.

References

1. DE BRUIN LJE *et al.*: EULAR 2024
2. MARQUES ML *et al.*: *Ann Rheum Dis* 2024; 83: 589-98.

P32

HOW LATAM ADAPTS TO THE NEW DEFINITIONS OF AXIAL SPONDYLOARTHRITIS - THREE ANALYZES OF THE PANLAR-ESPALDA REGISTRY

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Introduction. In recent years the classic definitions of Ankylosing Spondylitis have been challenged by advances in diagnosis (MRI) including more women, lower proportion of HL-B27. The objective is to estimate the frequency of men, HLA-B27 positive and r-axSpA and analyze the differences in each one in a Latin-American cohort from PANLAR-ESPALDA registry.
Methods. We included consecutive patients aged ≥18 years with axial Spondyloarthritis (ASAS 2009) from medical centers in Argentina, Uruguay, Chile, Venezuela, Mexico, Colombia, and Ecuador. Recorded data encompassed demographic information, age at symptom onset, disease duration, disease-related symptoms, and comorbidities. Clinical and therapeutic aspects of the disease were documented at baseline, and specific questionnaires to assess disease activity (ASDAS/BASDAI) and functional capacity (BASFI) were administered. Additionally, we recorded erythrocyte sedimentation rate (ESR) in mm/h, C-reactive protein (CRP) in mg/dl, HLA B27 status, x-rays, and, if necessary, MRI of the sacroiliacs (SI).
Results. A total of 200 patients were recruited (cohort characteristics in Table I). The frequency of male sex, HLA-B27 and r-axSpA was: 54%, 55% and 59% respectively. The statistically significant differences in each group are shown in Table II. In the three multivariate analyzes the independent associations were: Male sex: fewer years of study (OR: 0.87 95%CI: 0.7-0.9)

and greater involvement in SI x-ray (OR: 4.5 95%CI: 2-9), HLA- B27+: Bone bridges on MRI (OR: 9 95%CI: 1.4-59), biological treatment (OR: 2.9 95%CI: 1.1-8) and for r-axSpA: the presence of erosions on MRI (OR: 4.5 95 %CI: 1.4-14).

Discussion/Conclusion. In our region, the frequency of men and r-axSpA is around 50% in accordance with other cohorts that used the concept of axSpA with ASAS 2009 criteria. On the other hand, we found a low prevalence of HLA-B27 compared to European cohorts. The differential characteristics are consistent with those reported in other cohorts.

P32: Table I.

	n=200	
Age assessment, mean (SD)	46	(12.6)
Male %	54	
Years of study,mean (SD)	13	(3.3)
Age of onset of LBP, mean (SD)	39	(13)
Delay to diagnosis, mean (SD) months	78	(25)
Smoking %	32	
Uveitys %	8	
Psoriasis %	20	
Inflammatory bowel disease %	5.7	
SpA family history %	26	
NSAIDs good response %	54	
HLA-B27+ %	55	
Inflammatory LBP %	85	
SpA features, mean (DS)	3.5	(3)
SpA features of SpA >4	45	
SI + Rx	56	
Sacroiliac MRI+ (any lesion)	76	
SI MRI: edema	65	
SI MRI: chronic changes (any)	64	
SI MRI: fatty changes	35	
SI MRI: erosions	41	
SI MRI: sclerosis	11	
SI MRI: bone bridges	9	
Sacroiliac maneuvers %	46	
Anterior chest pain %	14	
VAS pain, mean (SD)	6	(2.3)
VAS night pain, mean (SD)	5	(2.6)
Morning stiffness, mean-n (SD)	33	(27)
BASFI, mean (SD)	4.5	(1.7)
BASDAI, mean (SD)	4	(1.9)
Presence of arthritis %	20	
Presence of enthesitis %	34	
MASES, median (IQR)	1.2	(1.9)
CRP mg/L, median (IQR)	1	(1-5)
CRP elevation >5 mg/L	32	
ESR 1 h, mean (SD)	19	(15)
Biological treatment%	49	
TNFb%	81	
IL17b%	19	

P33

CLINICAL FEATURES OF PATIENTS WITH SPA, WITH OR WITHOUT IBD: RESULTS FROM THE METEOR COHORT

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Introduction. This study aims to investigate clinical features of SpA patients with and without IBD, identifying the patient group that would benefit from initiating therapy with efficacy in both SpA and IBD at the time of diagnosis of either entity.

Methods. METEOR SpA database include patients diagnosed with peripheral or axial SpA. Patients were categorized based on concurrent IBD as reported by local investigators. Data on patient characteristics, disease outcomes, activity measures, treatment, and imaging were collected. IBD status (current/past) was used for patient stratification. Continuous outcome comparisons used Student's t-test or Mann-Whitney U-test.

Results. Out of 11,589 SpA patients, 7% had concurrent IBD with an average symptom duration of 12 years at inclusion. Global IBD prevalence ranged <1%-20%. In SpA with IBD, male predominance was less pronounced, with slightly higher BMI. Disease duration and diagnostic delay were slightly longer, with lower HLA-B27 and psoriasis prevalence, but higher uveitis rates. No significant differences were noted in dactylitis, enthesitis, or CRP levels. SpA patients with IBD showed more sacroiliac joint damage (modified NY criteria) and higher BASFI scores. MRI findings and syndesmophyte presence were comparable, although syndesmophyte data was limited.

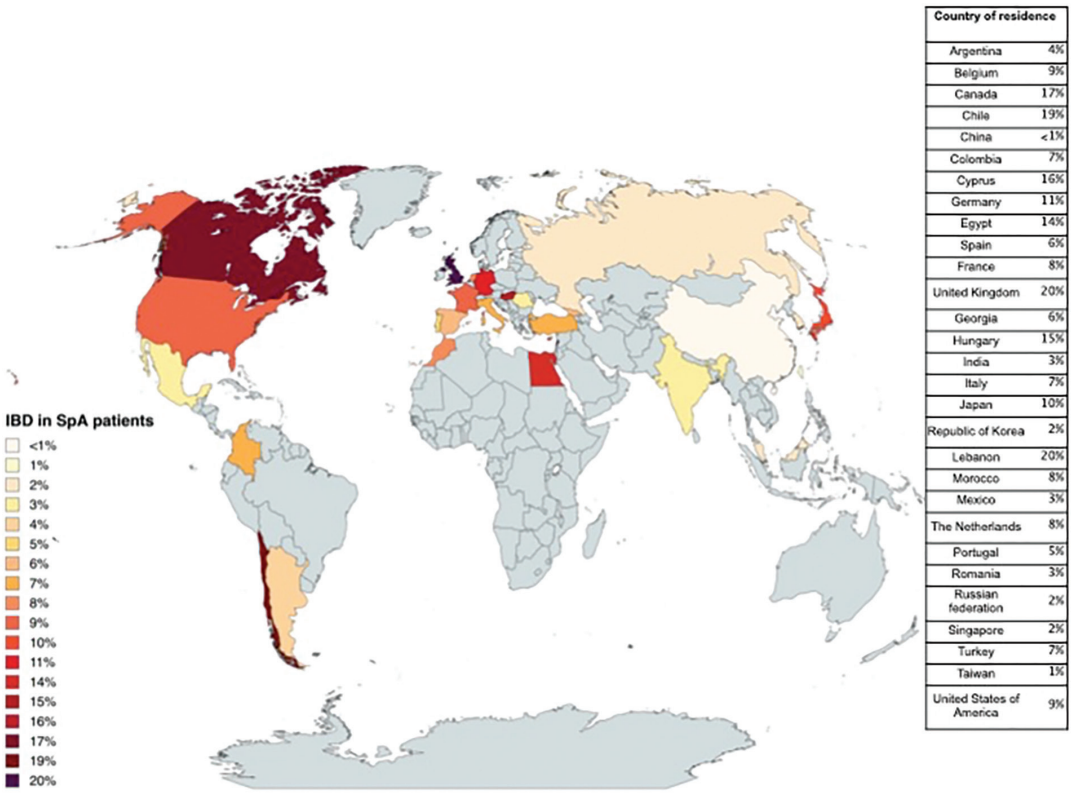
Conclusion. SpA patients with IBD, as opposed to those without IBD, were less frequently male, more frequently HLA-B27 negative, demonstrated higher rates of uveitis and lower rates of psoriasis. These results support the lower rates of HLA-B27 and psoriasis described in Belgian SpA patients with IBD (1). SpA patients with concomitant IBD exhibited more structural damage on SI joints compared to patients without IBD, possibly mirroring gut inflammation as a risk factor for evolution into radiographic axSpA. These patients also exhibited longer symptom duration. In light of evolving, occasionally more selective, therapeutic agents, identifying SpA patients at risk of developing IBD at the time of diagnosis would prevent unnecessary therapy cycling.

Reference

1. VARKAS G *et al.*: *Arthritis Rheumatol* 2018; 70(10): 1588-96.

P32: Table II.

Features	Male	Fem	p	HLA-B27 pos	HLA-B27 neg	p	rx-axSpA	nrx-axSpA	p
Age assessment, mean (SD)				41 (11)	50 (11)	0.001			
Male %							31	70	0.001
Years of study, average (SD)	12.3 (3.4)	13.6 (3.1)	0.007				12 (3.3)	14 (3)	0.005
Age of onset of low back pain mean (SD)				32 (12)	44 (12)	0.001			
Delay to diagnosis, median (IQR) months							61 (19-143)	36 (10-121)	0.04
Smoking %				24	39	0.03			
Uveitys %				13	4	0.04			
Psoriasis %				9	38.2	0.0001			
HLA-B27+ %							70	42	0.005
SI + x-Ray	77	40	0.001	71	46	.0.005			
SI MRI: edema				47	71	0.001	70	50	0.04
SI MRI: erosions							75	33	0.0001
SI MRI: bone bridges	29	2	0.001	25	2.1	0.0001	32	3.2	0.001
Pain in chest %							13	30	0.03
VAS pain, mean (SD)				5 (2.7)	6 (1.8)	0.03	6 (2.4)	7 (2.1)	0.02
BASDAI, mean (SD)	3.7 (1.7)	4.3 (1.9)	0.03						
Presence of enthesitis %				43	27	0.02			
CRP elevation >5 mg/L	19	38	0.04	21	48	0.001	38	21	0.01
ESR 1 h, mean (SD)	17.5 (12)	20.6 (11.5)	0.01						
Biological Treatment%				62	40	0.004	53	32	0.02



P33: Fig. 1. Worldwide distribution and prevalence of patients with spondyloarthritis with or without inflammatory bowel disease.

	No IBD n=10,781	IBD n=808	p-value
Age (years)	51.0 ± 13.9	52.0 ± 13.2	0.113
Sex (male)	**6715/10778 (62%)**	**443/808 (55%)**	**<0.001**
Symptom duration in years	**12.4 ± 11.9**	**14.7 ± 13.3**	**<0.001**
Diagnostic delay in years	**5.5 ± 8.0**	**6.7 ± 8.6**	**<0.001**
BMI	**26.0 ± 5.6**	**27.0 ± 18.0**	**<0.001**
HLA-B27 positivity	**6158/8692 (71%)**	**333/627 (53%)**	**<0.001**
Psoriasis	**2446/10772 (23%)**	**140/808 (17%)**	**<0.001**
Uveitis	**1841/10764 (17%)**	**205/808 (25%)**	**<0.001**
Dactylitis	805/6514 (12%)	56/415 (13%)	0.410
Enthesitis	3968/9591 (41%)	291/688 (42%)	0.634
CRP	8.1 ± 18.8	9.2 ± 24.8	0.118
Elevated CRP	3109/9968 (31%)	239/764 (31%)	0.957
ASDAS	2.2 ± 1.2	2.2 ± 1.2	1
BASDAI	4.0 ± 2.5	3.9 ± 1.4	0.261
BASFI	**3.1 ± 3.1**	**3.4 ± 2.7**	**0.008**
VAS physician	3.5 ± 2.2	3.5 ± 2.3	1
VAS patient	4.4 ± 2.7	4.4 ± 2.7	1
VAS pain patient	4.0 ± 2.9	3.9 ± 2.8	0.343
Sacroiliitis on MRI*	3292/4989 (66%)	279/430 (65%)	0.644
Radiographic sacroiliitis**	**1315/2477 (53%)**	**97/153 (63%)**	**0.013**
Presence syndesmophytes	148/623 (24%)	12/44 (27%)	0.598

P33: Fig. 2. Baseline characteristics of patients with SpA stratified for IBD.
*Positive MRI of the SI joints according to the SAS definition.
**Radiographic sacroiliitiss according to the modified New York criteria.
Results reported by mean ± standard deviation and n (%). In bold statistically significant difference $p<0.01$. SD: standard deviation; NS: non-significant; BMI: body mass index; CRP: C-reactive protein; ASDAS: axial spondyloarthritis disease activity score; BASDAI: bath ankylosing spondylitis disease activity.

P34

EXPLORING DIFFICULT-TO-TREAT AXIAL SPONDYLOARTHRITIS IN PRACTICE: RESULTS FROM THE DUTCH SPA-NET REGISTRY

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Introduction. Patients presenting with multiple disease management challenges may be considered 'difficult-to-treat' (D2T) (1, 2). As D2T disease in axial spondyloarthritis (axSpA) is a new concept, this study aimed to explore its prevalence and the associated patient characteristics.
Methods. Data from SpA-Net, a Dutch SpA registry, were utilised. Four definitions for D2T-axSpA were constructed (Fig. 1), each requiring patients to fulfil three domains: 1. treatment failure, 2. insufficient disease control, and 3. a problematic management situation perceived by the patient and/or physician. For the 'insufficient disease control' domain, four variations were explored: [A] High disease activity score, [B] objective signs of active disease, [C] reduced health-related quality of life, or, [D] any of A-C. Additionally, treatment-refractory (TR) axSpA was defined as fulfilment of definition D, and having an AxSpA Disease Activity Score ≥2.1 and C-reactive protein ≥5mg/L. The percentage of D2T patients per definition was calculated and characteristics associated were assessed using multivariable logistic regression.
Results. Data from 218 patients were analysed; 44.0% were female, mean age was 51.8 (SD 13.8) years, mean symptom duration was 21.6 (SD 12.6) years, mean ASDAS-CRP was 2.2 (SD 1.0), and 67.4% had used ≥1 biological. 'Treatment failure' affected 11.0-13.0% of patients, 'insufficient disease control' affected, per variation, [A] 51.8%, [B] 34.2%, [C] 44.9% and [D] 67.8% of patients, and 'problematic management' affected 45.9-47.9% of patients. Cumulatively, D2T-axSpA had a prevalence of 3.2-11.0%, and TR-axSpA of 1.4%. Having no paid work (OR: 4.3 [95% CI 1.1-16.9], $p=0.04$), and a history of psoriasis (OR: 5.2 [95% CI 1.5-17.7], $p=0.01$) or uveitis

(OR: 4.7 [95% CI 1.4-16.0], p=0.01) were associated with D2T-axSpA in multivariable analysis (Table I).

Conclusion. In clinical practice, approximately one in ten patients with axSpA is D2T and 1% is potentially TR. Having no paid work, and a history of psoriasis or uveitis are associated with D2T-axSpA.

Acknowledgements. The researchers thank all patients and healthcare providers who participated in the SpA-Net registry. This investigator-initiated study was financially supported by UCB Biopharma SRL. UCB Biopharma SRL had no role in the study design, in the collection, analysis or interpretation of the data, or in the writing of this manuscript or the decision to submit this manuscript for publication.

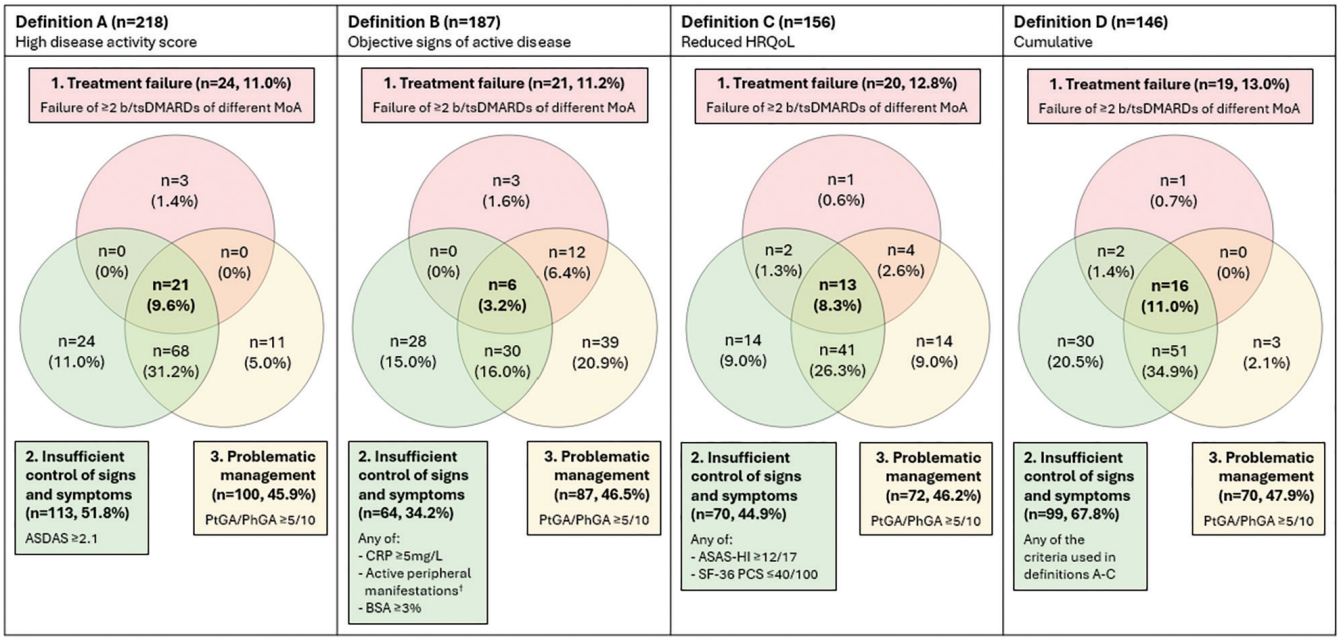
References

1. WENDLING D, VERHOEVEN F, PRATI C: Is the Difficult-to-Treat (D2T) concept applicable to axial spondyloarthritis? *Joint Bone Spine* 2023; 90(3): 105512.
2. NAGY G, ROODENRIJS NMT, WELSING PMJ: EULAR definition of difficult-to-treat rheumatoid arthritis. *Ann Rheum Dis* 2021; 80(1): 31-35.

P34: Table I. Univariable and multivariable analysis of the demographic and disease characteristics associated with definition D of D2T-axSpA.

Variable	Univariable analysis		Multivariable analysis	
	OR (95% CI)	p	OR (95% CI)	p
Female sex	2.3 (0.8-6.6)	0.13	2.5 (0.8-8.1)	0.11
Age	1.0 (1.0-1.1)	0.51	1.0 (0.9-1.0)	0.39
No paid work	2.4 (0.8-7.1)	0.11	4.3 (1.1-16.9)	0.04
Current smoking	2.4 (0.7-7.5)	0.15	- ‡	-
History of EMMs				
Psoriasis	4.0 (1.4-11.7)	0.01	5.2 (1.5-17.7)	0.01
Uveitis	3.3 (1.1-9.6)	0.03	4.7 (1.4-16.0)	0.01
History of peripheral manifestations†	2.2 (0.7-6.7)	0.16	- ‡	-

Mutivariable models were always adjusted for age and sex.
† Peripheral manifestations included arthritis, enthesitis and dactylitis.
‡ Variable not associated with outcome in multivariable model (p>0.05).
axSpA: axial spondyloarthritis; D2T: difficult-to-treat; EMM: extra-musculoskeletal manifestation.



P34: Fig. 1. Proportions of patients fulfilling each explored definition and the individual domains within the definitions.

† Peripheral manifestations included arthritis, enthesitis and dactylitis.

Definition A: treatment failure (failure of ≥2 b/tsDMARDs of different MoA) + insufficient control of signs and symptoms (ASDAS ≥2.1) + problematic management (PtGA/PhGA ≥5/10)
Definition B: treatment failure (failure of ≥2 b/tsDMARDs of different MoA) + insufficient control of signs and symptoms (CRP ≥5mg/L, active peripheral manifestations [arthritis, enthesitis and/or dactylitis], and/or BSA ≥3%) + problematic management (PtGA/PhGA ≥5/10)
Definition C: treatment failure (failure of ≥2 b/tsDMARDs of different MoA) + insufficient control of signs and symptoms (ASAS-HI ≥12/17 and/or SF-36 PCS ≤40/100) + problematic management (PtGA/PhGA ≥5/10)
Definition D: treatment failure (failure of ≥2 b/tsDMARDs of different MoA) + insufficient control of signs and symptoms (ASDAS ≥2.1, CRP ≥5mg/L, active peripheral manifestations, BSA ≥3%, ASAS-HI ≥12/17 and/or SF-36 PCS ≤40/100) + problematic management (PtGA/PhGA ≥5/10)

Proportions of patients fulfilling each individual domain per definition are presented in the respective boxes. For example, 24 out of 218 (11.0%) patients fulfilled the 'treatment failure' domain in total in the population considered for definition A, irrespective of the other two domains.

Abbreviations: ASAS-HI: Assessment of SpondyloArthritis International Society Health Index; ASDAS: Axial Spondyloarthritis Disease Activity Score; bDMARD: biological disease modifying antirheumatic drug; BSA: psoriasis body surface area; CRP: C-reactive protein; HRQoL: health-related quality of life; MoA: mode of action; PhGA: physician global assessment; PtGA: patient global assessment; SF-36 PCS: 36-Item Short Form Health Survey Physical Component Summary Score; tsDMARDs: targeted synthetic DMARD.

P35

GENDER DISPARITY IN AXIAL SPONDYLOARTHRITIS DIAGNOSIS: WHERE IS THE UNCONSCIOUS BIAS? RESULTS FROM THE NATIONAL AXIAL SPONDYLOARTHRITIS SOCIETY (NASS) PATIENT SURVEY

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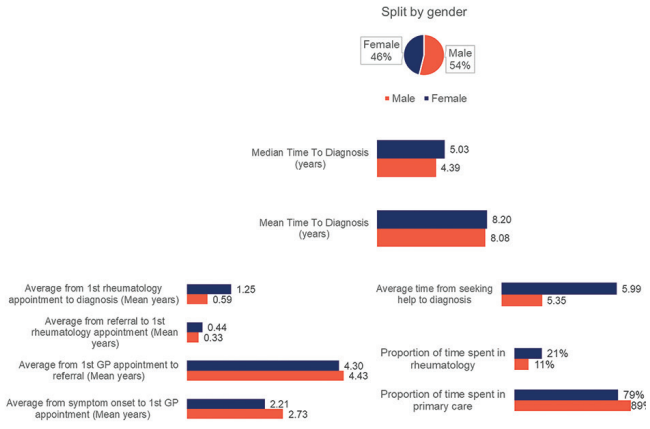
Introduction. UK mean average time to diagnosis (TTD) for axial SpA is currently 8.29 years.[1] However, it should be possible to ensure diagnosis within 12 months of symptom onset to optimise clinical outcomes.[2] Current evidence suggests a gender gap in TTD, with women waiting between one and four years longer on average (mean) internationally for a diagnosis.[3] An audit tool was created by NASS and UK rheumatology teams to evaluate the national performance in TTD and the factors impacting diagnosis.
Methods. We developed a patient self-administered post-diagnosis Axial SpA survey, and analysed data submitted according to patient gender.
Results. Data were collected from 523 patients diagnosed since January 2021, with 46% women and 54% men (Fig. 1). While the overall mean TTD was similar for both genders at 8.20 years (women) and 8.08 years (men), the median average showed a discrepancy, with 5.03 years (women) compared to 4.39 years (men). Women sought help quicker after symptom onset (2.21 vs. 2.73 men) and had a slightly faster referral following the first assessment by a GP (4.30 vs. 4.43 men). However, women experienced 33% longer waiting times (0.44 vs. 0.33 men) once in Rheumatology. The mean time from first assessment to diagnosis was 113% longer for women (1.25) than for men (0.59). Additionally, women spent a higher proportion of their diagnostic journey in Rheumatology (21% or 1.70) compared to men (11% or 0.92), with 73% of their journey occurring after seeking help, compared to 66% for men.
Conclusion. Our findings highlight a gender gap in TTD, particularly for women when they reach Rheumatology. Despite seeking help earlier, women face greater barriers, beginning in primary care and exacerbated in secondary care. Solutions may involve awareness, education, referral improvements, and addressing clinical complexities.



P35: Fig. 1. Key results highlighting the gender gap. (Source: NASS time to diagnosis data).

Acknowledgements. The time to diagnosis survey is part of the NASS Quality Improvement programme *Aspiring to Excellence*. *Aspiring to Excellence* which has provided expert improvement support to 23 Rheumatology departments through team-based coaching and learning events. *Aspiring to Excellence* is a strategic partnership between NASS, BRITSpA, the NHS Transformation Unit and sponsoring companies AbbVie, Biogen, Lilly, Novartis and UCB.

References
1. EDDISON J, BAMFORD S, CHAN A *et al*.: What is the average time to diagnosis for axial spondyloarthritis in the UK? London: National Axial Spondyloarthritis Society. Available at www.actonaxialspa.com
2. WEBB D, SWINGLER L, BARNETT R *et al*.: Act on axial SpA: A Gold Standard time for the diagnosis of axial SpA (2021). London: National Axial Spondyloarthritis Society
3. BANDINELLI F, MARTINELLI-CONSUMI B, MANETTI M, VALLECOCCIA MS: Sex bias in diagnostic delay: are axial spondyloarthritis and ankylosing spondylitis still phantom diseases in women? a systematic review and meta-analysis. *J Pers Med*. 2024;14(1): 91. doi: 10.3390/jpm14010091.

P36

DECODING SEX VARIANCE IN AXIAL SPONDYLOARTHRITIS: INSIGHTS FROM THE BRAZILIAN REGISTRY OF SPONDYLOARTHRITIS WITH A FOCUS ON HLA-B27 AND PATIENT OUTCOMES

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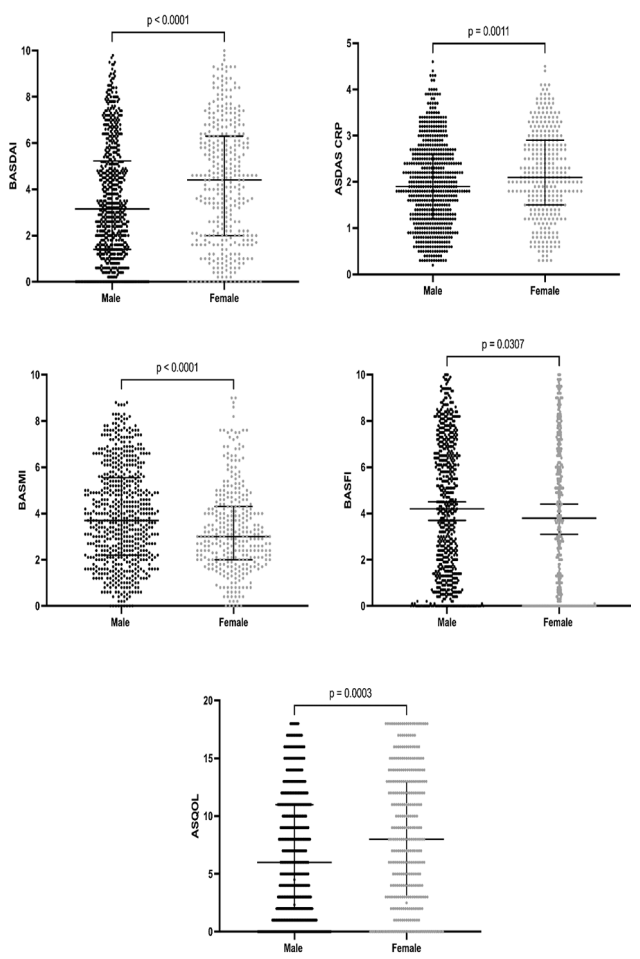
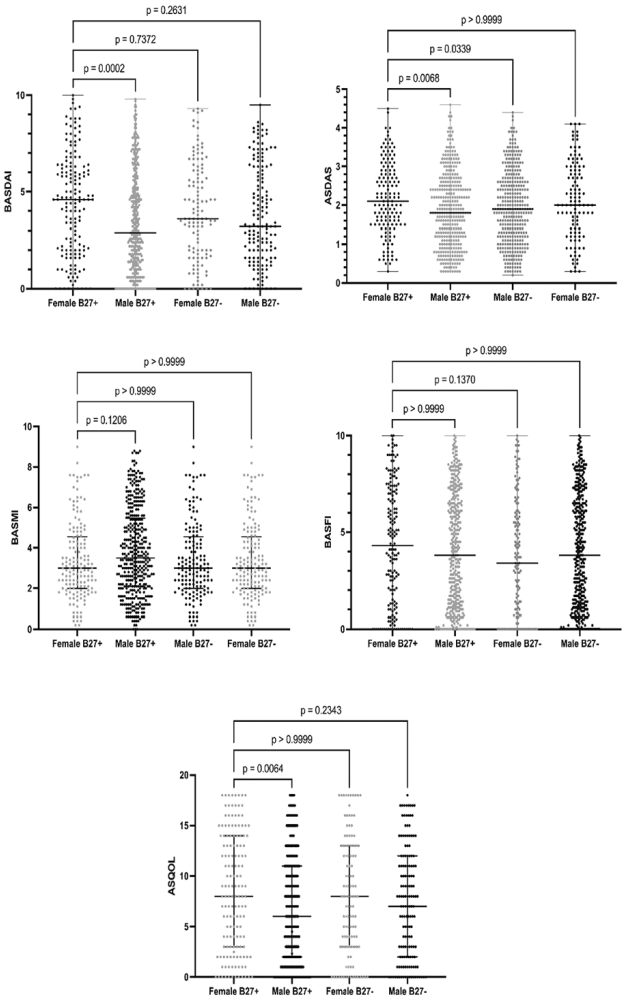
Introduction. Although axial spondyloarthritis (axSpA) has been more frequently and severely observed in men, emerging data suggest that the impact on women may have been underestimated, with women also bearing a significant burden of the disease. In this new scenario, this study aims to evaluate the differences between sexes in clinical presentation, HLA-B27 positivity, and clinical outcomes among patients enrolled in the Brazilian Registry of Spondyloarthritis (RBE).
Methods. RBE is a multicenter, observational, prospective cohort of SpA Brazilian patients. Clinical and demographic data were collected in a standardized protocol. For analysis, patients were first stratified by sex and then stratified by sex and HLA-B27 status. The associations were tested using the Kruskal-Wallis test, considering a significance of 95%.
Results. 1,125 subjects were enrolled (64.6% males). Males were significantly younger than females (p=0.004), with HLA-B27 positivity more frequent (OR=1.63, 95%CI 1.36-1.94; p<0.001). Anxiety (OR=1.75, 95%CI1.18-2.57; p=0.004), depression (OR=2.41, 95%CI1.55-3.73; p<0.001), fibromyalgia (OR=3.90, 95%CI2.48-6.15; p<0.001), hypertension (OR=1.43, 95%CI1.10-1.86; p=0.008) and obesity (OR=2.03, 95%CI1.33-3.09; p<0.001) were more frequent in women, who also showed significantly higher BASDAI (p<0.001), ASDAS (p=0.011), BASFI (p=0.030) and ASQoL (p=0.0003). On the other hand, males presented worse BASMI (3.9±2.1 *vs*. 3.3±1.8; p<0.001) (Fig. 1). Women scored higher on VAS for pain (4.8±3.0 *vs* 4.1±3.0; p<0.001) and VAS for global disease (5.0±2.8 *vs*. 4.5±2.8; p=0.001), despite the physician's assessment being similar to that of male patients (3.6±2.6 *vs*. 3.6±2.7; p=0.438). HLA-B27 positive females presented higher BASDAI (p=0.0002) and ASDAS (p=0.0068) than HLA-B27 positive men (p=0.0068). No differences between sexes were observed in these HLA-B27 positive patients concerning BASMI and BASFI (Fig. 2).
Conclusion. Women with axSpA have a greater disease burden, with higher rates of comorbidities and worse clinical scores compared to men, despite similar physician assessments. This reinforces the need for sex-specific approaches in the management of SpA.



P36: Fig. 1. Sex differences in BASDAI, ASDAS, BASMI, BASFI and ASQoL in patients with axSpA.



P36: Fig. 2. Clinical scores by sex and HLA-B27 status in patients with axSpA.

P37

HOW EARLY IS EARLY? UNVEILING TIME TO DIAGNOSIS SINCE SYMPTOM ONSET AND ITS DETERMINANTS IN PATIENTS SUSPECTED OF EARLY AXIAL SPONDYLOARTHRITIS: DATA FROM THE SPONDYLOARTHRITIS CAUGHT EARLY (SPACE) COHORT

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Introduction/Objective. Investigate time to diagnosis after symptom onset in patients with chronic back pain (CBP) suspected of axSpA referred to rheumatologists and assess early diagnosis determinants.
Methods. We analysed the 2y-data from the SPACE multi-centre cohort of patients (<45y) with CBP (≥3 months, ≤2y) of unknown origin. Clinical, laboratory, and imaging SpA features were collected over time. An event was defined as an axSpA diagnosis with LoC≥7 at last observation or LoC<7 in ≥2 last consecutive visits. Time to event (months) was computed from symptom onset to the first visit with the event. Patients without the event were censored at 2y or loss to follow-up. SpA features were considered at the time of diagnosis. Median survival times were computed overall and per SpA feature (un-/adjusted for covariates). Uni-/multivariable Cox-regression models identified axSpA diagnosis determinants. The final multivariable model allowed subgroup comparisons for time-to-event by SpA feature, adjusting for other SpA features (adjusted Kaplan-Meier curves).
Results. Of the included 548 patients (mean [SD] age:31[8] years, symptom duration:13[7] months, males:35%, HLA-B27 positivity:41%), 215 (39%) had the event (median time to diagnosis:35 months). The lowest adjusted median survival times occurred for sacroiliitis on radiographs (8-months), sacroiliitis on MRI (12-months), inflammatory bowel disease (12-months), HLA-B27 positivity (18-months) and peripheral arthritis (24-months) (Table I). Kaplan-Meier curves showed median survival times and diagnosis probability (Fig. 1). HLA-B27 positivity and sacroiliitis on MRI appeared as the strongest determinants: 4.2- and 3.5-times adjusted higher likelihood for axSpA diagnosis, respectively (Table 1), while peripheral arthritis, anterior uveitis, or psoriasis each implied ~2-times higher risk for the diagnosis.
Conclusion. Half of CBP patients suspected of axSpA were diagnosed within 35 months of symptom onset. HLA-B27 positivity, sacroiliitis on imaging and peripheral arthritis are the key SpA features for early (≤2y after symptom onset) diagnosis of axSpA in these patients.

P37: Table I. Median survival times and Cox proportional hazard models for the asSpA diagnosis (event) in patients with chonic back pain symptom duration of ≥3 months but ≤2 years starting before the age of 45 years.

Covariates at the time of diagnosis	With axSpA	Without axSpA	Median survival times [reference category] (months)		Univariable Cox regression	Multivariable Cox regression
			Unadjusted	*Adjusted*	*HR (95% CI)*	*Adj HR (95% CI)*
Gender, male	50%	26%	24	39	**2.00 (1.53-2.61)**	**1.36 (1.02-1.80)**
HLA-B27, positive	76%	18%	21	18	**5.30 (3.86-7.26)**	**4.18 (2.92-5.97)**
Age#, ≤31 years	60%	47%	24	NR	**0.98 (0.96-0.99)**	**0.98 (0.96-0.99)**
Family history of SpA, positive	51%	41%	35	NR	0.97 (0.74-1.27)	0.76 (0.56-1.03)
Inflammatory back pain, ever	60%	52%	36	NR	0.91 (0.69-1.20)	0.82 (0.62-1.10)
Good response to NSAIDs, ever	47%	34%	35	38	1.19 (0.91-1.56)	**1.42 (1.07-1.89)**
Peripheral arthritis, ever	17%	8%	24	24	**1.99 (1.39-2.85)**	**1.74 (1.18-2.55)**
Dactylitis, ever	7%	3%	24	35	**1.76 (1.04-2.97)**	1.19 (0.63-2.24)
Heel pain, ever	19%	9%	24	29	**1.50 (1.07-2.12)**	1.37 (0.95-1.97)
Anterior uveitis, ever	14%	4%	20	39	**1.92 (1.30-2.82)**	**1.94 (1.27-2.97)**
Inflammatory bowel disease, ever	7%	7%	29	12	1.06 (0.64-1.76)	1.21 (0.70-2.09)
Psoriasis, ever	11%	8%	24	NR	1.41 (0.92-2.15)	**1.68 (1.02-2.75)**
Elevated CRP^, ever	41%	29%	29	NR	1.26 (0.96-1.66)	1.25 (0.95-1.65)
Sacroiliitis on radiographs$, ever	20%	1%	18	24	**3.25 (2.32-4.54)**	1.06 (0.72-1.56)
Sacroiliitis on MRI§, ever	62%	9%	18	12	**4.99 (3.79-6.58)**	**3.45 (2.52-4.71)**

SpA features were considered at the time of diagnosis as *'once a feature, always a feature'* using local reports and standardization according to the Assessment of SpondyloArthritis international Society (ASAS) definitions - Sieper J *et al*., *Ann Rheum Dis*. 2009;68 Suppl 2:ii1-44 (unless stated otherwise). #Categorized as ≤\> median age. $modified New York criteria according to local radiologist. §Inflammatory and/or structural changes compatible with sacroiliitisas reported by local radiologists. ^Elevated if >5mg/l. **Adj**: adjusted; **axSpA**: axial spondyloarthritis; **CI**: confidence interval; **CRP**: C-reactive protein; **HR**: Hazard ratio; **NSAIDs**: Nonsteroidal Anti-Inflammatory Drugs; **MRI**: magnetic resonance imaging. *Color-code*: **Blue**: median survival times ≤24 months; **Orange**: median survival times >24 months; **Grey**: median survival time not reached (**NR**). Significant results from the Cox proportional hazard models are highlighted in bold.

P38

CHANGE OVER 2 YEARS IN DIFFERENT PHENOTYPES OF CHRONIC BACK PAIN SUSPICIOUS OF AXIAL SPONDYLOARTHRITIS: A LATENT TRANSITION ANALYSIS OF THE SPONDYLOARTHRITIS CAUGHT EARLY (SPACE) COHORT

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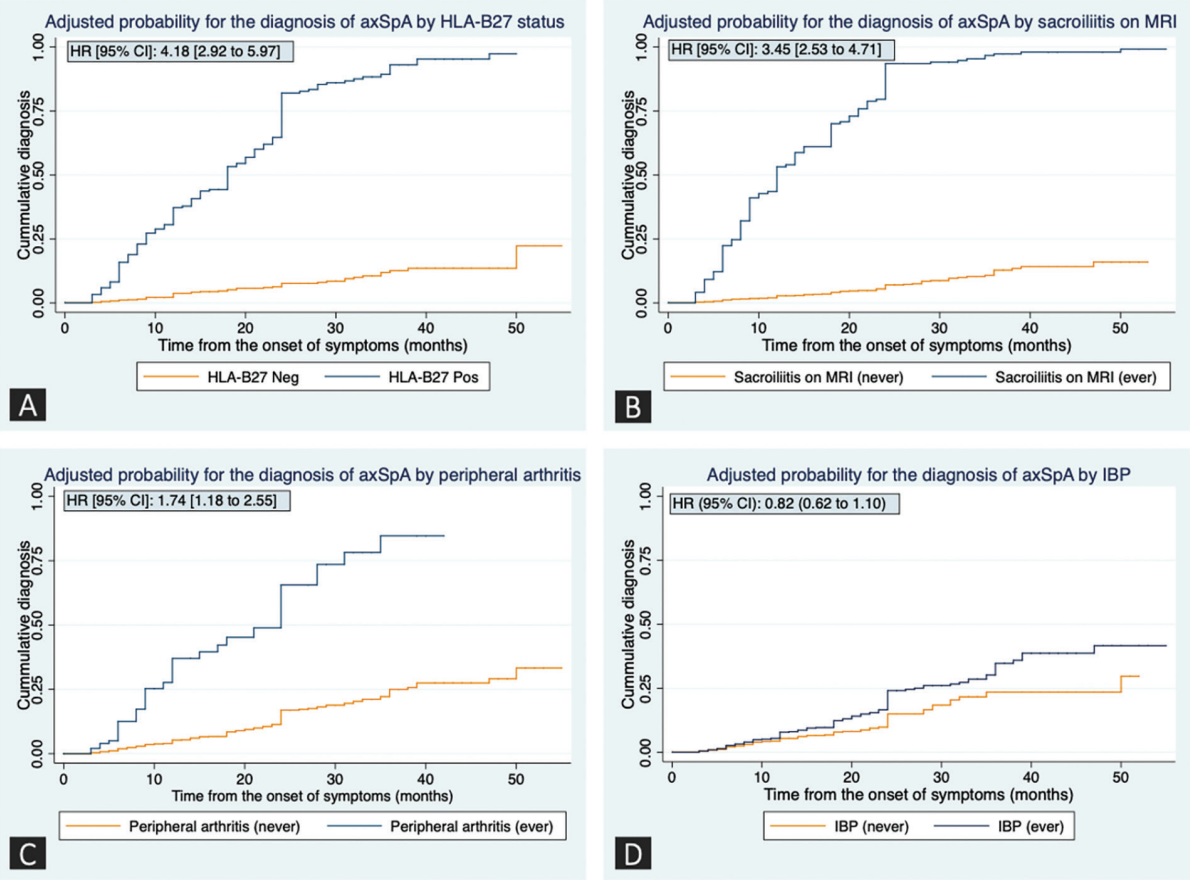
Background. Sepriano et al have identified four phenotypes of chronic back pain suspicious of axial spondyloarthritis (axSpA) in the Spondyloarthritis Caught Early (SPACE) cohort, reflecting an expert-free judgement of the construct of axSpA.(1) They were labelled as: "pure axSpA" ("axial") representing individuals with positive axial imaging and a familial/genetic predisposition for axSpA; "axSpA with peripheral signs" ("IBP+peripheral") with participants characterized by peripheral involvement and inflammatory back pain (IBP), "axial Spondyloarthritis at risk" ("at risk"), a phenotype



P37: Fig. 1. Examples of Kaplan-Meier curves for the adjusted probability of axial Spondyloarthritis (asSpA) diagnosis since the onset of chonic back pain symptoms (in months) by A. HLA-B27 status (positive/negative), B. Sacroiliitis on MRI, *i.e*., inflammatory and/or structural changes compatible with sacroiliitis as reported by the local radiologist (ever/never), C, Peripheral arthritis (ever/never), and D. Inflammatory back pain (IBP) according to the ASAS definition (ever/never). Estimates derived from the Cox regression model including all SpA features: HLA-B27 status (except A), gender, age at diagnosis, family history of spondyloarthritis, inflammatory bowel disease, psoriasis, elevation of C-reactive protein, sacroiliitis on radiographs (modified New York criteria) and sacroiliitis on MRI (except B)

with a familial/genetic predisposition to axSpA but an otherwise low probability of other features and a "no Spondyloarthritis" ("no SpA") phenotype depicting participants with a very low probability for any SpA associated feature. However, these phenotypes have not been investigated over time.

Objective. To follow-up these four phenotypes over two years (2Y) and assess a potential switch between them over time.

Methods. The SPACE cohort consists of participants with chronic back pain suspicious of axSpA. Data was collected at baseline, three months, one- and 2Y on demographics and all SpA features, including clinical, laboratory and imaging features (Fig. 1). All SPACE participants were included in the analysis. Missing data at baseline and 2Y follow-up were imputed using data from the adjacent visits or, if not possible, were assumed to be absent. In a sensitivity analysis only participants with complete data, both at baseline and 2Y, were included. Latent class analysis (LCA) was performed on baseline data to reassess model fit and clinical recognizability of the four-phenotype model. Latent transition analysis (LTA) models were constructed using data from baseline and 2Y, extracting marginal probabilities, displaying the probability of a participant being in one of the phenotypes, conditional probabilities, which reflect the probability of a SpA feature being present in one of the phenotypes, and transitional probabilities, depicting the probability of switch between the phenotypes from baseline to 2Y follow up.

Results. In total, 702 participants were included. Two-hundred-seventy-one (39%) were male and mean (SD) age and duration of back pain were 30 (8) years and 13 (7) months, respectively.

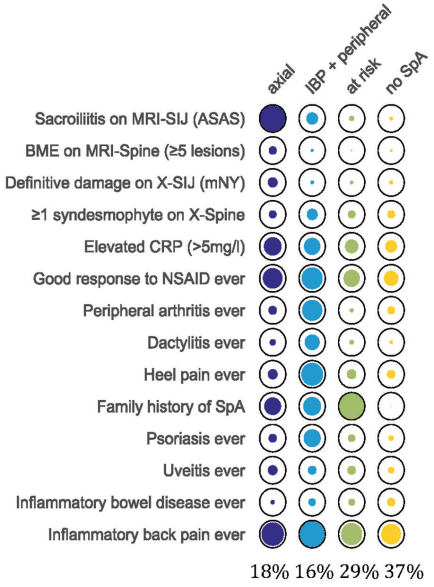
Both LCA and LTA models revealed the previously described four-phenotype model as the best fitting model. The highest marginal probabilities were observed in the "no SpA" (37%) and "at-risk" (29%) phenotypes, with comparatively lower probabilities in the "axial" (18%) and "IBP+peripheral" (16%) phenotype.

Conditional probabilities (Fig. 1) showed a high probability of sacroiliitis on MRI (97%), and elevated CRP values (50%) in the "axial" phenotype, the highest probability of IBP (96%) and peripheral/cutaneous SpA features (including peripheral arthritis, dactylitis, heel pain and psoriasis) in the "IBP+peripheral" phenotype, the highest probability of family history of SpA (100%), but otherwise a lack of distinct SpA features in the "at risk" phenotype and negative family history of SpA and overall the lowest probability for any SpA feature in the "no SpA" phenotype.

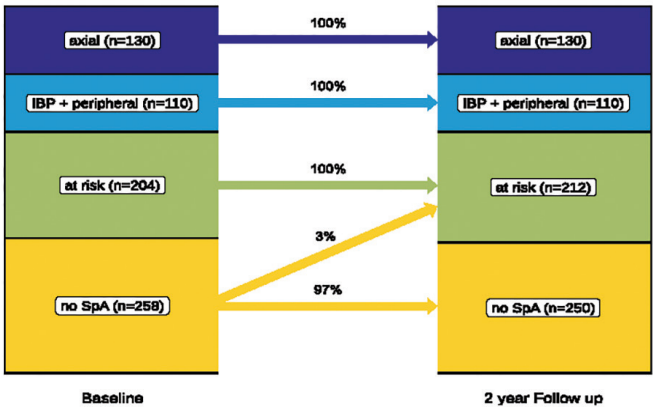
The LTA revealed a 3% transition probability from the "no SpA" to the "at risk" class between baseline and 2Y with all other participants remaining in their initially assigned class (Fig. 2).

Sensitivity analysis on 384 participants with complete data at both baseline and 2Y showed similar results, demonstrating the robustness of the model.

Conclusion. Transitions between the four classes over two years were basically inexistent, highlighting the unlikelihood of participants to develop relevant new features of axSpA after an initial clinical work-up.



P38: Fig. 1. Graphical demonstration of the conditional probabilities of the 2-year latent transition analysis (LTA) model (n=702).
The circles represent the conditional probability for a feature in a respective class, with a higher probability corresponding to a fuller circle. A full circle represents 100% and an empty circle 0% probability. The colors represent the four classes. The percentages below the figure represent the marginal probabilities, *i.e.* the probability of a participant being in one of the phenotypes.
ASAS, Assessment of SpondyloArthritis international Society; BME, bone marrow edema; CRP, C-reactive protein; IBP, inflammatory back pain; MRI, magnetic resonance imaging; mNY, modified New York criteria; NSAID, non-steroidal anti-inflammatory drug; SIJ, sacroiliac joints; X-SIJ, radiograph of the sacroiliac joints; SpA, spondyloarthritis; X-Spine, radiograph of the spine.



P38: Fig. 2. Diagram showing class change over 2 years according to transitional probabilities (LTA analysis).
Transitional probabilities were generated using the 4-class latent transition model with 702 patients.
IBP, inflammatory back pain; SpA, spondyloarthritis.

Reference

1. SEPRIANO A, RAMIRO S, VAN DER HEIJDE D *et al.*: What is axial spondyloarthritis? A latent class and transition analysis in the SPACE and DESIR cohorts. *Ann Rheum Dis* 2020; 79(3): 324-31.

P39

RISK OF HIP AND SPINE FRACTURES IN AXIAL SPONDYLOARTHRITIS IS ASSOCIATED WITH TREATMENT CLASS

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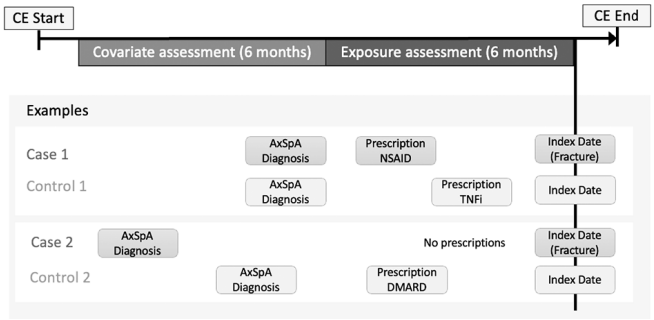
Introduction/Objective. Individuals with axial spondyloarthritis (axSpA) have a risk of fracture that is nearly doubled that of the general population. Vertebral fracture risk may be due to regional osteopenia paired with regions of excess bone stiffness. We assessed the impact of treatment with tumor necrosis factor inhibitors (TNFi) and non-biologic disease-modifying antirheumatic drugs (DMARDs) on hip and spine fractures in axSpA, relative to nonsteroidal anti-inflammatory drugs (NSAIDs).

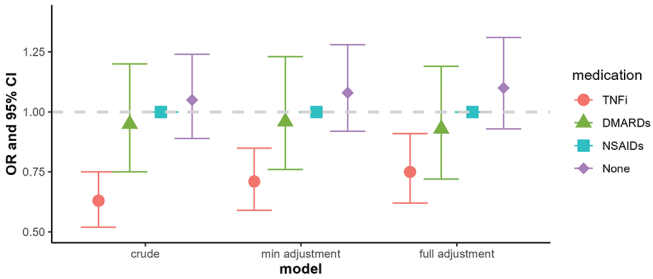
Materials and methods. We conducted a nested case-control study using 2006-2021 data from the US-based Merative™ MarketScan® Database (Fig. 1). We included adults 18-65 years old with ≥1 inpatient or ≥2 outpatient axSpA ICD-9 or 10 diagnosis codes separated by ≥7 days. The primary outcome was hip and/or spine fracture, defined by ICD-9 or 10 diagnosis or procedure codes. For each fracture case, we selected up to 10 controls without fracture. We evaluated medication use (TNFi, DMARDs, NSAIDs [referent], or none) hierarchically using pharmacy claims and procedure codes (for infusions). We assessed the relation of medication use with hip and spine fracture risk using unconditional logistic regression with adjustment for potential confounders.

Results. Our main analysis included 13,519 individuals with axSpA, comprising 1,229 cases and 12,290 controls. Individuals on TNFi had 29% lower odds of fracture compared to those on NSAIDs (OR 0.71, 95% CI 0.59-0.85), accounting for age, sex, and diagnosis year (Fig. 2). Results for TNFi were similar in the fully adjusted model (OR 0.75, 95% CI 0.62-0.91) and when stratified by sex. There was no protective effect of DMARDs.

Conclusion. Using a large US insurance claims database, we observed a protective effect of TNFi on fracture risk in axSpA. These findings suggest a beneficial effect of TNFi in bone remodeling in axSpA.



P39: Fig. 1. Continuous enrolment, exposure assessment period, and covariate assessment period in relation to index date and disease date
Index date: fracture date for cases, and random date from the corresponding case's fracture year for controls. Disease date can be any time prior to index date. Continuous Enrollment (CE) Period was required to include 1 year prior to index date. Exposure Assessment Period was 6 months prior to index date. Covariate Assessment Period was 2 months prior to Exposure assessment period.



P39: Fig. 2. Odds of hip or spine fracture associated with axial spondylarthritis therapeutic class, with NSAIDs as the referent.
Tumor necrosis factor inhibitors (TNFi) include etanercept, adalimumab, golimumab, certolizumab and infliximab. DMARDs include apremilast, auranofin, azathioprine, chloroquine, cyclophosphamide, cyclosporine, gold sodium thiomalate, hydroxychloroquine, leflunomide, methotrexate, minocycline, mycophenolate, sulfasalazine. Minimally adjusted model adjusted for age, sex, and diagnosis year. Fully adjusted model adjusted for age, sex, disease year, alcohol use disorder, antiepileptic drug use, BMI, breast cancer, chronic kidney disease, falls, glucocorticoid use, inflammatory bowel disease, osteoporosis, osteoporosis medication use, prostate cancer, tobacco use, ESR/CRP laboratory orders and number of outpatient visits (rheumatology and primary care).

P40

CANCER INCIDENCE IN AS PATIENTS ON TNF INHIBITORS: FINDINGS FROM A TWO-DECADE RETROSPECTIVE COHORT

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Introduction. Since the EMA and FDA approved etanercept for ankylosing spondylitis (AS) in 2003, several tumor necrosis factor (TNF) inhibitors have been widely used to treat AS. However, data on the long-term cancer risk associated with TNF inhibitors in AS is still limited.
Methods. This single-center retrospective cohort study included patients diagnosed with AS who initiated TNF inhibitors from April 2003 to January 2023. We analysed the characteristics of patients who were diagnosed with cancer while exposed to TNF inhibitors and those who were not.
Results. A total of 807 patients were included in the study. Of these, 57% used adalimumab as the first-line treatment, followed by 23% who used etanercept, infliximab, and golimumab. For patients not responding to the first-line TNF inhibitor, 180 switched to a second-line TNF inhibitor. The mean age of patients was 38.7 years; 75% were male, 90% were HLA-B27 positive, half were smokers, and about 3% had a history of cancer. The mean duration of TNF inhibitor treatment was 82.5 months. Over 5467 person-years (PYs), we found 19 cases of cancer (348/100,000 PYs), including 4 lung cancers, 3 thyroid cancers, 2 colorectal cancers, and others. Although the type of TNF inhibitor and disease activity were not associated with cancer incidence, patients newly diagnosed with cancer were older and had a higher proportion of hypertension. In multivariate analysis, age remained a significant risk factor for cancer. Although a history of cancer increased the risk in univariate analysis, it was not statistically significant in multivariate analysis.
Conclusion. In AS patients, the use of TNF inhibitors was not linked to an increased cancer risk compared to the general population in Korea (537.3/100,000 PYs for ages 35-64), regardless of prior cancer history or disease activity. However, older patients should be cautious about cancer when maintaining TNF inhibitors.

Table I. Baseline characteristics of patients with AS at the time of TNF inhibitor initiation.

	Total (n=807)	Non-occurrence (N=788)	Occurrence of cancer (n=19)	*p*-value
Age, years	38.7 ± 13.5	38.5 ± 13.4	47.9 ± 13.6	0.002
Male, n (%)	609 (75.5)	594 (75.4)	15 (78.9)	0.721
HLA-B27	713 (90.1)	695 (90.0)	18 (94.7)	0.496
HTN	122 (15.2)	116 (14.8)	6 (31.6)	0.043
DM	37 (4.6)	35 (4.5)	2 (10.5)	0.216
Smoker	418 (52.3)	406 (52.1)	12 (63.2)	0.338
History of cancer	23 (2.9)	21 (2.7)	2 (10.5)	0.099
Duration of TNF inhibitor, mo.	82.5 ± 66.6	81.9 ± 66.8	106.4 ± 53.8	0.113
BASDAI	6.7 ± 1.3	6.7 ± 1.3	7.2 ± 1.6	0.095
Laboratory data				
WBC	7670.4 ± 2143.1	7693.8 ± 2152.0	6731.6 ± 1504.8	0.053
Hb	13.4 ± 1.7	13.4 ± 1.7	13.2 ± 2.0	0.482
PLT	292.1 ± 80.2	292.6 ± 80.5	270.3 ± 64.3	0.232
ESR	40.0 ± 29.9	39.8 ± 29.7	50.7 ± 34.4	0.114
CRP	1.1 (0.4–2.3)	1.1 (0.4–2.3)	0.7 (0.3–1.9)	0.495

P41

INTERFERON SIGNATURE AS A PREDICTOR OF TNF INHIBITOR RESPONSE IN ANKYLOSING SPONDYLITIS

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Contents. While TNF inhibitors (TNFi) are fundamental in treating active inflammation, 30-40% of AS patients do not respond adequately, highlighting the pressing need to understand TNFi resistance. This study delves into the mechanisms that differentiate TNFi non-responders (NR) from responders (R), focusing on the distinct expression of interferon responses and Allograft Inflammatory Factor-1 (AIF-1).
Using single-cell RNA sequencing of PBMCs from 12 R and 10 NR patients, along with additional patient data analyzed through qPCR, bulk RNA-seq, ELISA, and FACS, we pinpointed AIF-1 as a soluble marker reflecting the underlying interferon signature that distinguishes NRs from Rs. NRs exhibited higher baseline levels of AIF-1 in their blood and showed increased transition of monocytes to Mo-DCs under heightened interferon signatures. Moreover, TNFi treatment paradoxically upregulated genes associated with the interferon pathway, reinforcing the signature and leading to an exacerbation of pathogenic Th17 cells post-TNFi therapy.
Our study underscores the pivotal role of the interferon signature in the development of TNFi non-response in AS, proposing AIF-1 as a diagnostic indicator. It suggests that treatment approaches for NRs should pivot towards targeting IL-17 or JAK pathways instead of TNFi, aiming to directly tackle the underlying immunological dysfunctions. These findings advocate for a personalized approach to AS management, taking into account individual variations in AIF-1 levels and interferon signatures.

P42

REFERENCE INTERVALS OF WORK ABILITY AND PRODUCTIVITY LOSS AND THEIR USE IN PATIENTS WITH INFLAMMATORY RHEUMATIC AND MUSCULOSKELETAL DISEASES

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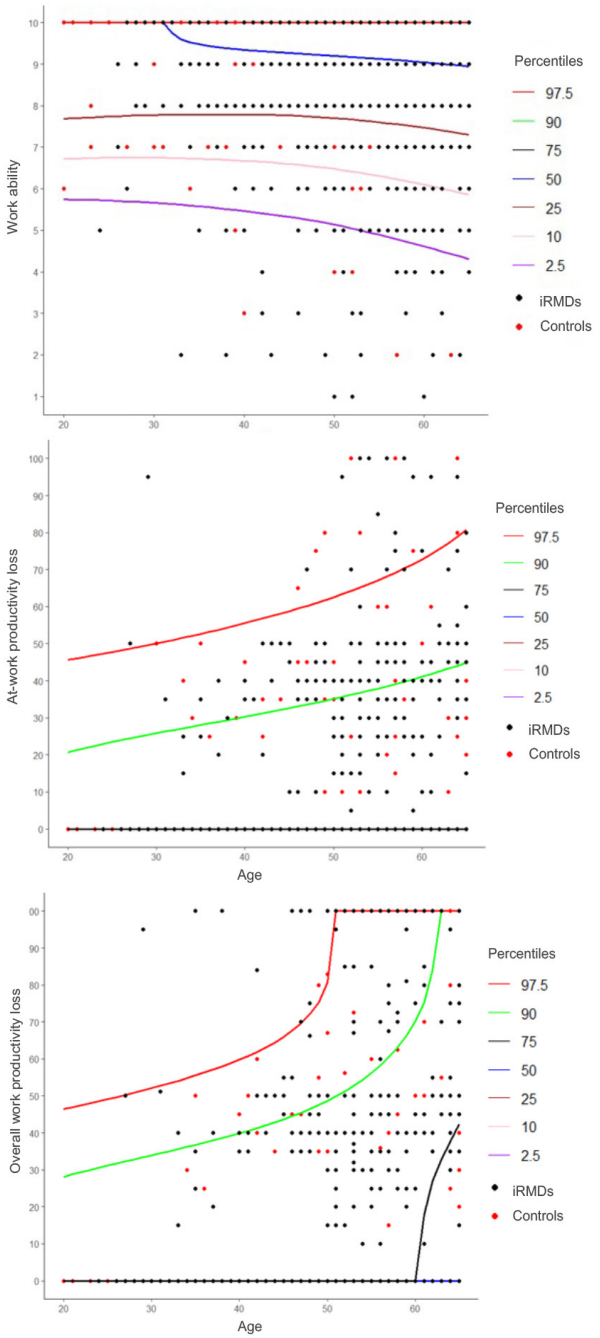
Objective. To establish reference intervals (RIs) for work ability, at-work productivity loss and overall productivity loss in a general working population and to compare these work outcomes of patients with inflammatory RMDs (iRMDs) with those of the general population.
Methods. Patients with iRMDs and general population controls with paid work participating in a Dutch cohort study reported on three work outcomes: work ability (Work Ability Index, range 0-10 [worst-best ability]), at-work productivity loss (Work Productivity and Activity Impairment [WPAI] item 5), and overall work productivity loss (WPAI items 2-5, range 0-100% [no-complete productivity loss]). A generalized additive model for location, shape and scale parameters (GAMLSS) was used to establish age-specific RIs and percentile curves for controls. The proportion of patients vs controls with outcomes below/above (as appropriate) a given percentile curve was compared.
Results. 413 controls were included; 73% female, mean age 51 (SD 10) years, mean work ability 8.7 (1.6), at-work productivity loss 6.3% (7.2) and overall work productivity loss 11% (25.6). RIs were created for the three outcomes. For instance, for work ability, the 2.5th percentile (= worse outcome) for 30-39 y/o individuals was 5.6, while the 50th percentile for the same group was 9.7 (Table I). The percentile curves illustrate that work ability decreased and at-work and overall work productivity loss increased with age (Fig. 1). Compared to the RIs from controls, patients with iRMDs had lower work ability and higher at-work and overall productivity loss.
Conclusion. Work ability and productivity are not perfect in the general population, based on the newly developed RIs for the three work outcomes. This calls for caution to not overestimate the iRMD impact on work outcomes. Nevertheless, iRMD patients have higher presenteeism and work productivity loss, compared to controls.



P42: Fig. 1. Reference intervals and percentile curves for a) work ability, b) at-work productivity loss and c) overall productivity loss. For work ability score, lower scores reflect a worse outcome, so a score under a certain percentile (*e.g.* 2.5th percentile) reflects a worse work ability than a score under a higher percentile (*e.g.* 10th percentile). For at-work and overall productivity loss scores, higher scores reflect a worse outcome, so a score above a certain percentile (*e.g.* 97.5th percentile) reflects a worse productivity work than a score above a lower percentile (*e.g.* 75th percentile).

P42: Table I. Reference intervals for work ability, derived from the population controls. Number and proportion of population controls and patients with iRMDs with a work ability score under certain percentile*, for each age category.

Age range	Controls n=390	iRMDs n=523	50th percentile#			25th percentile			10th percentile			2.5th percentile		
			Score	Controls n (%)	iRMDs n (%)	Score	Controls n (%)	iRMDs n (%)	Score	Controls n (%)	iRMDs n (%)	Score	Controls n (%)	iRMDs n (%)
18-29	15	13	10	15 (100)	13 (100)	7.9	3 (20)	5 (38)	6.9	1 (7)	3 (23)	5.9	0 (0)	2 (15)
30-39	28	37	9.7	16 (57)	28 (76)	7.8	8 (29)	12 (32)	6.7	4 (14)	6 (16)	5.6	3 (11)	5 (14)
40-49	86	126	9.2	48 (56)	86 (68)	7.7	12 (14)	36 (29)	6.5	1 (1)	15 (12)	5.2	1 (1)	9 (7)
50-59	169	221	9.1	93 (55)	152 (69)	7.6	30 (18)	64 (29)	6.3	16 (10)	37 (17)	4.9	4 (2)	13 (6)
60-65	92	126	9.0	58 (63)	92 (73)	7.5	20 (22)	44 (35)	6.1	25 (21)	27 (21)	4.6	4 (4)	9 (7)

iRMDs, inflammatory rheumatic and musculoskeletal diseases.
*The lower the work ability score, the worse the work ability, so a score under a certain percentile (*e.g.* 2.5th percentile) reflects a worse work ability than a score under a higher percentile (*e.g.* 10th percentile)
#Also included the 75th, 90th and 97.5th percentile.

P43

THE ROLE OF ANTIPHOSPHOLIPID ANTIBODIES IN SPONDYLOARTHRITIS - PROSPECTIVE OBSERVATIONAL STUDY

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Introduction. Recent studies have highlighted the presence and potential role of antiphospholipid antibodies (aPL) in Spondyloarthritis (SpA), adding complexity to the disease's immunological management.
Objective. To identify the presence of aPL in patients with SpA and its relationship with different clinical-epidemiological data.
Methods. A prospective observational study between September 2023 and February 2024 involved adult patients diagnosed with axial SpA followed in the rheumatology department at ULS Aveiro. Anticardiolipin and anti-ß2 glycoprotein antibodies and Lupus anticoagulant (LA) were assessed and repeated 12 weeks after, in case of positivity. Clinical data, including thrombotic and obstetric events, were collected. Disease activity was measured based on ASDAS PCR, and BASDAI.
Results. 141 patients were screened, 9 were excluded due to coagulation therapy and active neoplasia. 56.1% were women, with a mean age of 52.2 years (± 1.22), a mean disease duration 10.8 years (± 3.5), 62.9% were HLAB27 positive, 69.7% were classified as radiographic and 30.3% as non-radiographic SpA, 26.5% presented peripheral involvement. The most frequent cardiovascular risk factors were dyslipidemia (42.4%), followed by hypertension (33.3%), and obesity (15.9%). 40.2% were under biotechnological therapy. More than one-third had ASDAS CRP>2.1 and BASDAI>4. Positive aPL were found in 16.7% of the sample, of which 40.9% were confirmed. LA was positive in 59%, followed by IgM anti-ß2 glycoprotein in 40.9% and anticardiolipin IgM in 31.8%. None of the patients presented thromboembolic or obstetric events. Radiographic SpA (p=0.048), obesity (p=0.045), and higher CPR levels (p=0.000) showed a statistically significant tendency to the presence of aPL.
Conclusion. This study identified the presence of aPL in 16.7% of SpA patients, particularly those with radiographic SpA, obesity, and elevated CRP levels. Despite the detection of aPL, there were no associated thromboembolic or obstetric events. While aPL is present in some SpA patients, their clinical implications with thrombotic risk require further investigation.

P44

MEASUREMENT PROPERTIES OF DISEASE ACTIVITY INSTRUMENTS IN PERIPHERAL SPONDYLOARTHRITIS - AN ANALYSIS IN THE CRESPA TRIAL

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Introduction. Understanding the best-performing disease activity measure and response criterion in peripheral spondyloarthritis (pSpA) is crucial for the development of future meaningful clinical studies.
Objective. To evaluate the construct validity and discriminatory ability of various instruments to assess components of disease activity and response criteria in patients with pSpA.
Methods. This is a post-hoc analysis of the CRESPA randomized controlled trial, which included active adult patients newly diagnosed with early pSpA and fulfilling the ASAS classification criteria for pSpA. Patients were randomized to golimumab (GOL) or placebo (PBO) with a 2:1 ratio. The data of the placebo-controlled part of the trial until week 12 were used. Construct validity (known-group discrimination (SMD) in the baseline population), longitudinal construct validity (*i.e.* Standardized Response Mean (SRM) and (Guyatt's) effect size) and trial discrimination (standardized mean difference (SMD)) of several continuous disease activity measure instruments (Table 1) were assessed. As part of trial discrimination, Chi-square test was evaluated for binary outcomes.
Results. A total of 60 patients (40 GOL and 20 PBO) were included. Construct validity was better for composite outcomes (i.e. DAPSA, BASDAI and ASDAS), showing a higher SMD between active and inactive patients. Longitudinal construct validity was consistently good for composite outcomes (*e.g.* ASDAS and DAPSA) and globals (PGA and PhGA), with some differences across methods. On the other hand, clinical trial discrimination was good for both composites (BASDAI, ASDAS, DAPSA) and global (PGA, PhGA) outcomes, as well as for SJC (Table I).
Finally, among the binary outcomes, trial discrimination was best for clinical remission (defined as absence of arthritis, enthesitis and dactylitis), BASDAI50, DASPA-LDA and ASDAS-LDA (Table II).
Conclusion. While both composite and global outcome measurement instruments performed well in pSpA, composite scores like DAPSA, ASDAS and BASDAI additionally showed better construct validity. The definition of clinical remission was the most discriminatory response criterion.

P44: Table I. Summary of the psychometric properties of the continuous outcome measurement instruments reflecting disease activity.

	Longitudinal construct validity			Clinical trial discrimination		
	Standardized Response Mean (SRM)	Guyatt's responsiveness index	Effect Size (ES)	Performance	Standardized Mean Difference (SMD)	Performance
BASDAI	-0.968	**-1.627**	-1.471	Good	-0.919	Good
ASDAS	**-1.138**	**-1.555**	**-1.558**	Good	-0.829	Good
DAPSA	**-1.334**	**-1.665**	-1.285	Good	**-1.206**	Good
VAS pain	-0.846	-1.130	-1.282	Good	-0.577	Adequate
PGA	**-1.138**	1.162	**-1.755**	Good	-0.829	Good
PhGA	**-1.459**	-1.472	**-2.482**	Good	-0.841	Good
SJC	-1.066	-1.420	-0.822	Good	**-1.265**	Good
TJC	-0.975	-1.071	-0.708	Adequate	**-1.250**	Good
MASES	-0.531	-0.373	-0.464	Poor	-0.001	Poor
Dactylitis count	-0.411	-0.539	-0.480	Poor	-0.546	Adequate
CRP	-0.431	-0.607	-0.493	Poor	-0.860	Good

Performance longitudinal construct validity: Green: good performance (Guyatt's ES; SRM, ES ≥80); orange: adequate performance (Guyatt's ES, SRM ES ≥0.50 & <0.80); Red: poor performance (Guyatt's ES, SRM, ES <0.50).
Performance clinical trial discrimination: Green: good performance (SMD ≥80); orange: adequate performance (SMD ≥0.50 & <0.80); Red: poor performance (SMD <0.50).
SMD (Standardized Mean Difference): Good ≥0.80; Adequate ≥ 0.50 and <0.80; Poor <0.50
GRI (Guyatt's Responsiveness Index): Good ≥0.80; Adequate ≥ 0.50 and <0.80; Poor <0.50
SRM (Standardized Response Mean): Good ≥0.80; Adequate ≥ 0.50 and <0.80; Poor <0.50
ES (Effect Size): Good ≥0.80; Adequate ≥ 0.50 and <0.80; poor <0.50
ASDAS: Axial Spondyloarthritis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; CRP: C-Reactive Protein; DAPSA: Disease Activity in Psoriatic Arthritis Score; MASES: Maastricht Ankylosing Spondylitis Enthesitis Score; PGA: Patient's Global Assessment; SJC: Swollen Joint Count; TJC: Tender Joint Count; VAS Pain: Visual Analogue Scale for Pain.

P44: Table II. Discriminatory ability of the dichotomous response criteria.

	Golimumab n (%) W12 n=40	Placebo n (%) W12 n=20	Chi-square	*p*-value
PSpARC40	23 (58%)	4 (20%)	7.58	0.006
PSpARC50	22 (55%)	4 (20%)	6.65	0.010
PSpARC70	20 (50%)	3 (15%)	6.91	0.009
BASDAI50	25 (62%)	2 (10%)	14.85	<0.001
ASDAS-MI (improvement ASDAS≥2)	23 (57%)	4 (20%)	7.58	0.006
ASDAS-CII (improvement ASDAS≥1.1)	16 (40%)	3 (15%)	3.85	0.049
ASDAS-LDA (ASDAS<2.1)	30 (75%)	6 (30%)	11.25	<0.001
ASDAS-ID (ASDAS<1.3)	18 (45%)	2 (10%)	7.35	0.007
DAPSA-REM (DAPSA≤4)	16 (40%)	2 (10%)	5.71	0.017
DAPSA-LDA (DAPSA≤14)	7 (35%)	33 (82%)	13.54	<0.001
Clinical remission*	28 (70%)	3 (15%)	16.15	<0.001

Chi-square test represents the discriminatory ability (the highest score reflects the best discriminatory ability).
BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; ASDAS: Axial Spondyloarthritis Disease Activity Score; ASDAS-CII: ASDAS Clinically Important Improvement (improvement ASDAS ≥1.1); ASDAS-ID: ASDAS Inactive Disease (ASDAS <1.3); ASDAS-LDA: ASDAS Low Disease Activity (ASDAS <2.1); ASDAS-MI: ASDAS Major Improvement (i.e. ASDAS improvement ≥ 2); DAPSA: Disease Activity in Psoriatic Arthritis Score; DAPSA-LDA: DAPSA Low Disease Activity (DAPSA ≤14); DAPSA-REM: DAPSA Remission (DAPSA ≤4); PSpARC: Peripheral Spondyloarthritis Response Criteria.
*Clinical remission: absence of arthritis and enthesitis and dactylitis

P45

IMPACT OF PERIPHERAL ARTHRITIS ON DISEASE ACTIVITY OUTCOMES IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS, PERIPHERAL SPONDYLOARTHRITIS AND PSORIATIC ARTHRITIS - DATA FROM THE ASAS-PERSPA STUDY

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Background. Patients with spondyloarthritis (SpA) can present with several phenotypes (*i.e.*, axial SpA (axSpA), peripheral SpA (pSpA) and Psoriatic Arthritis (PsA)) and peripheral arthritis can occur either concomitantly with the axial disease or independently. Peripheral arthritis can impact disease activity outcomes, but data is lacking on the potentially different magnitude of this effect across the phenotypes of the disease.
Objective. a) To determine the independent impact of the presence of peripheral arthritis on disease activity outcomes across all SpA phenotypes; b) To investigate differences in this impact across SpA phenotypes (axSpA, pSpA and PsA).
Methods. This analysis is derived from the cross-sectional ASAS-PerSpA study. The impact of peripheral arthritis (i.e., history of peripheral arthritis and current peripheral arthritis) on BASDAI, ASDAS, DAPSA, DAS28 and DAS44 was initially explored using multivariable models encompassing the other peripheral manifestations (enthesitis), as well as socio-demographic, disease-related and treatment variables. Then, analyses were stratified by phenotype. All the obtained regression coefficients were standardized to allow for comparisons across the different disease activity measures.
Results. A total of 4185 patients (2719 axSpA, 433 pSpA, 1033 PsA) were included. Multivariabçe analyses in the overall population revealed that a history of arthritis exclusively affected DAS44, while current arthritis influenced all disease activity outcomes, with a more pronounced effect on DAPSA than on BASDAI, ASDAS, DAS28 or DAS44 (Table I). Multivariable analyses across phenotypes showed very similar results. Current arthritis significantly impacted BASDAI, ASDAS, DAPSA, DAS28 and DAS44 in the three subtypes. However, this effect was larger for DAPSA than for the other outcomes (Table II).
Conclusion. Peripheral arthritis, particularly its current presence, significantly impacts disease activity outcome measures in SpA patients. Notably, this impact remains consistent across the different phenotypes of SpA, but with a larger impact on DAPSA compared to the remaining disease activity measurement instruments.

P46

IMPACT OF PERIPHERAL MANIFESTATIONS ON FUNCTION, HEALTH, AND WORK PRODUCTIVITY IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS, PERIPHERAL SPONDYLOARTHRITIS AND PSORIATIC ARTHRITIS - DATA FROM THE ASAS-PERSPA STUDY

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Introduction. Peripheral musculoskeletal manifestations can occur either concomitantly with axial disease or independently in patients with spondyloarthritis (SpA). However, it is not known whether the impact of peripheral manifestations on disease outcomes is different across the SpA phenotypes (axial SpA (axSpA), peripheral (pSpA) and psoriatic arthritis (PsA)).
Objective. a) To determine the independent impact of the presence of peripheral manifestations on various disease outcomes in SpA; b) To investigate potential differences in this impact across SpA phenotypes.
Methods. Patients from the cross-sectional ASAS-PerSpA study with axSpA, pSpA or PsA were included. The impact of peripheral involvement (*i.e.* history of peripheral arthritis, current peripheral arthritis, history of peripheral enthesitis, current peripheral enthesitis and history of dactylitis) on BASFI, ASAS-HI, EQ5D and WPAI was assessed in multivariable models (zero-inflated model for WPAI and linear mixed models with random effects for the other outcomes, with "country" as a random effect) encompassing all the peripheral manifestations as the main variables of interest. Finally, analyses were stratified by disease phenotype.
Results. A total of 4185 patients (2719 axSpA, 433 pSpA, 1033 PsA) were

P45: Table I. Impact of peripheral manifestations on disease activity outcomes in SpA (multivariable multilevel models adjusted for country).

		BASDAI		ASDAS		DAPSA		DAS28		DAS44	
		Mean (SD)	Standardized beta coefficient (95%CI)	Mean (SD)	Standardized beta coefficient (95%CI)	Mean (SD)	Standardized beta coefficient (95%CI)	Mean (SD)	Standardized beta coefficient (95%CI)	Mean (SD)	Standardized beta coefficient (95%CI)
History of arthritis	Yes	4.15 (2.51)	-0.05 (-0.12 to 0.03)	2.64 (1.18)	-0.04 (-0.08 to 0.00)	15.17 (13.11)	0.19 (-0.19 to 0.57)	2.88 (1.19)	0.03 (-0.00 to 0.06)	1.48 (0.89)	***0.05 (0.031 to 0.08)***
	No	3.58 (2.32)		2.44 (1.09)		9.33 (8.97)		2.33 (0.84)		0.96 (0.56)	
Current arthritis	Yes	5.14 (2.41)	***0.44 (0.37 to 0.51)***	3.18 (1.18)	***0.27 (0.24 to 0.31)***	23.85 (16.14)	***4.95 (4.63 to 5.28)***	3.74 (1.16)	***0.48 (0.46 to 0.51)***	2.10 (0.93)	***0.36 (0.34 to 0.38)***
	No	3.53 (2.34)		2.37 (1.07)		9.22 (7.57)		2.31 (0.81)		0.99 (0.55)	

P45: Table II. Impact of peripheral manifestations on disease activity outcomes stratified in axSpA, pSpA and PsA (multivariable multilevel models adjusted for country).

	BASDAI Standardized beta coefficient (95%CI)	ASDAS Standardized beta coefficient (95%CI)	DAPSA Standardized beta coefficient (95%CI)	DAS28 Standardized beta coefficient (95%CI)	DAS44 Standardized beta coefficient (95%CI)
axSpA					
History of arthritis	0.06 (-0.03 to 0.15)	-0.01 (-0.06 to 0.04)	0.28 (-0.07 to 0.64)	***0.04 (0.01 to 0.07)***	***0.06 (0.04 to 0.08)***
Current arthritis	***0.40 (0.31 to 0.49)***	***0.24 (0.20 to 0.28)***	***3.64 (3.31 to 3.97)***	***0.34 (0.31 to 0.37)***	***0.26 (0.24 to 0.29)***
pSpA					
History of arthritis	-0.18 (-0.38 to 0.02)	-0.05 (-0.14 to 0.04)	-0.05 (-1.00 to 0.89)	-0.03 (-0.12 to 0.06)	-0.01 (-0.08 to 0.05)
Current arthritis	***0.66 (0.45 to 0.88)***	***0.39 (0.29 to 0.49)***	***5.30 (4.33 to 6.27)***	***0.60 (0.51 to 0.69)***	***0.41 (0.35 to 0.49)***
PsA					
History of arthritis	-0.10 (-0.25 to 0.04)	-0.06 (-0.13 to 0.01)	0.20 (-0.65 to 1.01)	0.03 (-0.04 to 0.09)	***0.05 (0.00 to 0.11)***
Current arthritis	***0.45 (0.31 to 0.59)***	***0.28 (0.21 to 0.34)***	***6.24 (5.46 to 7.04)***	***0.60 (0.54 to 0.66)***	***0.43 (0.38 to 0.48)***

*Results adjusted by other socio-demographic, disease-related and treatment variables.

included. Multivariable models showed that history of arthritis exclusively affected WPAI, while current arthritis influenced both the EQ5D and WPAI. A history of peripheral enthesitis impacted on ASAS-HI, EQ5D and WPAI, while current peripheral enthesitis affected all outcomes, including the BASFI (Table II).
After stratification, multivariable analyses showed very similar results across SpA phenotypes. The impact of history of arthritis on the four outcomes was very similar in axSpA, pSpA and PsA, as well as the impact of current arthritis. Similarly, history of peripheral enthesitis and current peripheral enthesitis showed very similar results across phenotypes (Table I).
Conclusion. Patients with SpA with peripheral manifestations have significantly worse function, health, and work productivity. Notably, this impact is very similar across the different phenotypes of SpA.

P46: Table I. Impact of peripheral manifestations on disease outcomes in SpA (multivariable multilevel models adjusted for country).

	BASFI Beta coefficient (95%CI)	**ASAS-HI** Beta coefficient (95%CI)	**EQ5D** Beta coefficient (95%CI)	**WPAI** IRR count model OR zero-inflated model
History of arthritis	-0.01 (-0.15 to 0.12)	0.18 (-0.04 to 0.40)	-0.00 (-0.01 to 0.01)	***0.88*** ***(0.84 to 0.91)*** 0.89 (0.68 to 1.16)
Current arthritis	0.06 (-0.10 to 0.21)	0.14 (-0.12 to 0.40)	***-0.04*** ***(-0.05 to -0.02)***	***1.10*** ***(1.06 to 1.14)*** 1.05 (0.78 to 1.42)
History of peripheral enthesitis	-0.03 (-0.15 to 0.09)	***0.55*** ***(0.34 to 0.75)***	***-0.01*** ***(-0.02 to -0.00)***	***0.92*** ***(0.89 to 0.95)*** 0.81 (0.65 to 1.01)
Current peripheral enthesitis	***0.26*** ***(0.08 to 0.44)***	***0.45*** ***(0.14 to 0.75)***	***-0.02*** ***(-0.04 to -0.00)***	1.02 (0.98 to 1.06) ***0.77*** ***(0.56 to 1.05)***

*Results adjusted for other socio-demographic, disease-related and treatment variables.

P47

PATIENT AND DISEASE CHARACTERISTICS ASSOCIATED WITH GLOBAL FUNCTIONING AND HEALTH (ASAS HI) IN AXIAL SPONDYLOARTHRITIS: A MIXED-EFFECTS MODEL ANALYSIS OF LONGITUDINAL DATA FROM THE DESIR COHORT

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Introduction. The Assessment of SpondyloArthritis international Society Health Index (ASAS HI) measures global functioning and health in axial spondyloarthritis (axSpA). The objective was to explore the role of selected patient- and disease characteristics on predicting ASAS HI over time.
Methods. Longitudinal data from the Devenir des Spondyloarthropathies Indifférénciées (DESIR) cohort were analysed, including axSpA patients with at least one non-missing ASAS HI value. The study covered the 6th (ASAS HI was initially collected) to 10th years after inclusion, with annual visits. A linear mixed-effects model was used to analyse factors potentially associated with ASAS HI over time, accounting for repeated measurements and allowing for patient-level and population-level predictions. Factors included socio-demographics, disease activity markers (axSpA Disease Activity Score - ASDAS), physical function (Bath Ankylosing Spondylitis (AS) Functional Index - BASFI), spinal and hip mobility (Bath AS Metrology Index - BASMI), 'fibromyalgianess' (Fibromyalgia Rapid Screening Tool – FiRST; with a cut-off score of ≥5/6), extra-musculoskeletal manifestations, and drug treatments. Regression coefficients with 95% confidence intervals and standardized coefficients (for comparison across predictors) were calculated.

P46: Table II. Impact of peripheral manifestations on disease outcomes stratified in axSpA, pSpA and PsA (multivariable multilevel models adjusted for country).

	BASFI Beta coefficient (95%CI)	**ASAS-HI** Beta coefficient (95%CI)	**EQ5D** Beta coefficient (95%CI)	**WPAI** IRR count model OR zero-inflated model
axSpA				
History of arthritis	0.03 (-0.13 to 0.20)	0.17 (-0.12 to 0.45)	-0.01 (-0.03 to 0.01)	***0.88*** ***(0.84 to 0.92)*** 1.00 (0.72 to 1.39)
Current arthritis	0.16 (-0.09 to 0.41)	-0.14 (-0.57 to 0.28)	***-0.02*** ***(-0.04 to -0.00)***	1.02 (0.96 to 1.09) 0.92 (0.57 to 1.48)
History of peripheral enthesitis	0.02 (-0.13 to 0.17)	***0.65*** ***(0.39 to 0.90)***	0.01 -(-0.02 to 0.00)	***0.96*** ***(0.92 to 0.99)*** 0.77 (0.59 to 1.01)
Current peripheral enthesitis	0.13 (-0.09 to 0.36)	***0.39*** ***(0.01 to 0.77)***	***-0.03*** ***(-0.05 to -0.01)***	0.99 (0.94 to 1.03) 0.75 (0.51 to 1.10)
pSpA				
History of arthritis	0.02 (-0.55 to 0.60)	-0.13 (-1.02 to 0.76)	0.05 (-0.00 to 0.09)	***0.43*** ***(0.37 to 0.52)*** 1.10 (0.31 to 3.89)
Current arthritis	-0.11 (-0.51 to 0.29)	0.30 (-0.32 to 0.92)	***-0.05*** ***(-0.08 to -0.01)***	***1.42*** ***(1.29 to 1.58)*** 1.32 (0.63 to 2.76)
History of peripheral enthesitis	-0.24 (-0.63 to 0.15)	0.17 (-0.44 to 0.78)	0.02 (-0.01 to 0.06)	***0.76*** ***(0.70 to 0.85)*** 0.97 (0.45 to 2.13)
Current peripheral enthesitis	0.42 (-0.18 to 1.02)	0.35 (-0.58 to 1.28)	***-0.05*** ***(-0.10 to -0.01)***	***1.33*** ***(1.19 to 1.48)*** 0.77 (0.29 to 2.08)
PsA				
History of arthritis	-0.00 (-0.35 to 0.34)	0.19 (-0.40 to 0.77)	-0.00 (-0.03 to 0.03)	***0.84*** ***(0.75 to 0.93)*** 0.49 (0.23 to 1.04)
Current arthritis	0.08 (-0.17 to 0.33)	0.09 (-0.32 to 0.51)	***-0.04*** ***(-0.06 to -0.01)***	***1.14*** ***(1.07 to 1.21)*** 1.18 (0.71 to 1.95)
History of peripheral enthesitis	-0.09 (-0.33 to 0.16)	***0.54*** ***(0.12 to 0.96)***	***-0.03*** ***(-0.05 to -0.01)***	***0.88*** ***(0.82 to 0.94)*** 1.07 (0.64 to 1.77)
Current peripheral enthesitis	***0.38*** ***(0.02 to 0.74)***	***0.65*** ***(0.04 to 1.25)***	-0.00 (-0.04 to 0.03)	1.01 (0.93 to 1.09) 0.61 (0.31 to 1.23)

*Results adjusted for other socio-demographic, disease-related and treatment variables.

Results. In total, 1805 visits of 460 patients were analysed. The mean ASAS HI, 5.8 at year 6, remained largely unchanged over the study period. BASFI and female sex were the most important predictors of ASAS HI: A one-unit higher BASFI was associated with a 0.80-unit higher average ASAS HI. Females had a 1.08-unit higher ASAS HI than males. Additionally, higher ASDAS, history of inflammatory bowel disease (IBD) and a high FiRST (≥5/6) were independently associated with worse ASAS HI (Table I). Time (visit) was not associated with ASAS HI.
Conclusion. In axSpA patients, the average ASAS HI remained rather stable over 5 years. Physical function and female sex were the main determinants of ASAS HI, followed by disease activity, a history of IBD, and widespread pain.

P47: Table I. Association between ASAS HI and patient- and disease characteristics.

Parameter	Coefficient (95% Confidence Interval)	Standardized Coefficient
BASFI (0-10)	0.80 (0.70; 0.90)	0.42
Female sex	1.08 (0.67; 1.50)	0.27
History of IBD	0.60 (0.03; 1.16)	0.15
ASDAS	0.65 (0.47; 0.83)	0.15
FiRST ≥5/6	1.26 (0.93; 1.60)	0.12
University level education	-0.40 (-0.84; 0.03)	-0.10
NSAIDs	0.33 (0.07; 0.58)	0.08
bDMARDs	0.30 (-0.02; 0.63)	0.08
Age (years)	0.02 (-0.01; 0.04)	0.04
Maximal intermalleolar distance (BASMI component)	0.08 (0.00; 0.17)	0.03
Visit	0.00 (-0.01; 0.00)	0.00

ASAS HI: Assessment of SpondyloArthritis international Society Health Index; ASDAS: AxSpA Disease Activity Score; BASFI: Bath AS Functional Index; BASMI: Bath AS Metrology Index; bDMARDs: biological disease-modifying anti-rheumatic drugs; FiRST: Fibromyalgia Rapid Screening Tool; NSAIDs: nonsteroidal anti-inflammatory drugs.

P48

SIGNIFICANT ASSOCIATION OF HLA-B27 STATUS ON SYMPTOM ONSET, AGE OF DIAGNOSIS, DISEASE DURATION, AND UVEITIS INCIDENCE IN AXIAL SPONDYLOARTHRITIS

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Introduction. Previous research has emphasized the variable prevalence of the HLA-B27 gene among patients with axial spondyloarthritis. This can have diverse impacts on disease phenotype and vary across different populations. More studies have indicated that HLA-B27 negative patients experience delayed diagnosis and a heightened incidence of peripheral and extra musculoskeletal manifestations, excluding uveitis. Our aim is to characterize differences in demographic and clinical features based on HLA-B27 status among AxSpA patients in the Irish population.
Methods. A retrospective cohort study was conducted utilizing data from the Ankylosing Spondylitis Registry of Ireland (ASRI), a cross-sectional, multicentre cohort study. Inclusion criteria included patients over eighteen years old clinically diagnosed with AxSpA meeting either the modified New York criteria for Ankylosing Spondylitis or the International Assessment of Spondyloarthritis Society criteria for AxSpA.
Results. Table 1 shows the cohort characteristics with comparison of HLA-B27 positive and negative subgroups. Analysis of data from 863 patients revealed significant variations in age-related parameters based on HLA-B27 status. HLA-B27 positive patients exhibited younger ages at symptom onset (23 vs. 32 years, $p<0.001$) and diagnosis (33 vs. 40 years, $p<0.001$), but without a difference in delay to diagnosis, alongside longer disease duration (17 vs. 11 years, $p<0.001$). Conversely, HLA-B27 negative patients displayed higher BASDAI ($p=0.032$) and HAQ-s ($p=0.021$) scores. Uveitis prevalence was notably higher in HLA-B27 positive patients (38.3% vs. 15.5%, $p<0.001$), a significance confirmed by multiple regression analysis ($p=0.03$). Several findings, including gender distribution, clinical measurements and peripheral and extra musculoskeletal manifestations, did not achieve statistical significance.
Conclusion. This study highlights substantial clinical disparities between HLA-B27 positive and negative AxSpA cohorts, particularly in age at symptom onset, diagnosis, disease duration, and uveitis incidence.

P48: Table I. General characteristics of the ASR1 cohort with comparisoj of HLA-B27 positive and negative subgroups.

Demographics and disease characterisation	n HLAB27+	B27+ Median (IQR) or n (%)	n HLAB27 -	B27- Median (IQR) or n (%)	P (B27+ vs B27-)[1]	Multiple regression[2]
Gender (male %)	733	75.9	130	70.8	0.236	--
Age in years, median (IQR)	733	44 (36 - 54)	130	48 (37 - 57)	**0.043**	--
Age at AxSpA symptom onset, median (IQR)	729	23 (19 - 31)	128	32 (24 - 42)	**<0.001**	--
Age at diagnosis, median (IQR)	729	33 (26 - 40)	128	40 (31 - 49)	**<0.001**	--
Delay to diagnosis in years, median (IQR)	729	6 (2 - 12)	128	5 (2 - 10)	0.072	--
Disease duration in years, median (IQR)	733	17 (10 - 28)	130	11 (6 - 21)	**<0.001**	--
ASAS criteria for AxSpA, n (%)	733	733 (100)	130	130 (100)	1	--
mNY criteria for AS, n (%)	733	551 (75.2)	130	101 (77.7)	0.527	--
Clinical measures of disease severity						
BASDAI score, median (IQR)	733	3.6 (1.8 - 5.7)	130	4.0 (2.2 - 6.2)	**0.032**	0.764
BASMI score, median (IQR)	733	3.6 (2.4 - 5.6)	130	3.4 (2.4 - 5.2)	0.29	--
BASFI score, median (IQR)	733	3.1 (1.3 - 5.4)	130	3.7 (1.4 - 6.2)	0.074	--
AsQoL score, median (IQR)	733	5.0 (1.0 - 11.0)	130	7.0 (1.0 - 13.0)	0.1	--
HAQ-s score, median (IQR)	733	0.4 (0.0 - 0.8)	130	0.5 (0.1 - 0.9)	**0.021**	0.245
Peripheral manifestations						
Dactylitis, n (%)	723	46 (6.4)	129	11 (8.5)	0.343	--
Enthesitis, n (%)	719	131 (18.2)	129	21 (16.3)	0.585	--
Peripheral arthritis, n (%)	718	215 (29.9)	128	44 (34.4)	0.328	--
Extra-musculoskeletal manifestations (EMMs)						
Uveitis, n (%)	720	276 (38.3)	129	20 (15.5)	**<0.001**	**0.03**
Psoriasis, n (%)	720	111 (15.4)	129	27 (20.9)	0.15	--
IBD[3], n (%)	722	74 (10.2)	129	17 (13.2)	0.363	--

[1]Statistical significance calculated by Mann Whitney U or chi-square test of two proportions (Fisher's exact).
[2]Multiple regression, based on a binary outcome model, using gender and AxSpA disease duration as covariartes.
[3]Defined as ulcerative colitis or Crohn's disease.

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SEX DIFFERENCES IN AXIAL SPONDYLOARTHRITIS: HIGHER INCIDENCE OF RADIOGRAPHIC DISEASE IN MEN, HIGHER RATES OF UVEITIS, IBD, PERIPHERAL ARTHRITIS, AND DACTYLITIS IN WOMEN

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Introduction. The examination of sex differences in axSpA has been the focus of several recent studies, revealing distinct disease manifestations among male and female patients. Previous research suggests a higher incidence of peripheral and extra-musculoskeletal manifestations in females with axSpA, including enthesitis, psoriasis, and inflammatory bowel disease, while acute anterior uveitis is more commonly observed in male patients . Our aim is to investigate sex-associated differences in the prevalence of extra-musculoskeletal and peripheral manifestations among individuals with axSpA.
Methods. This is a retrospective cohort study with data obtained from the Ankylosing Spondylitis Registry of Ireland (ASRI), which is a large cross-sectional, multicentre cohort study. Statistical comparisons between male and female subgroups were assessed using p-values calculated through Mann Whitney U or chi-square tests. Multiple regression analysis is also employed, incorporating gender, HLA-B27 status, and axSpA disease duration as covariates in a binary outcome mode.
Results. Table 1 shows the characteristics of the ASRI cohort with comparison of male and female subgroups. Demographic details were available for 913 participants at the time of analysis, including 686 male and 227 female patients. Males had a higher median age than females (46 vs. 41 years; $p=0.005$). There was a significant gender difference in meeting the New York criteria for Ankylosing Spondylitis, with a higher prevalence in males (78.8%) compared to females (67.8%) ($p=0.002$). Females had a higher prevalence of dactylitis (11.9% vs. 4.9%; $p=0.001$), peripheral arthritis (37.2% vs. 28.8%; $p=0.019$), uveitis (41.2% vs. 32.1%; p=0.015), and inflammatory bowel disease (18.6% vs. 8.0%; $p<0.001$). Psoriasis and enthesitis did not show a significant gender difference ($p=1$ and $p=0.84$) respectively.
Conclusion. The study reveals significant sex differences in age, radiographic criteria and prevalence of peripheral and extra-musculoskeletal manifestations in AxSpA patients.

P49: Table I. Extra musculoskeletal and peripheral manifestation of the ASRI cohort with comparison of male and female subgroups.

Demographics and disease characterisation	Male Median (IQR) or n (%)	Female Median (IQR) or n (%)	P (male vs female)[1]	Multiple regression[2]
Age in years, median (IQR)	46 (37 - 55)	41 (35 - 51)	**0.005**	--
NY criteria for AS, n (%)	540 (78.8)	154 (67.8)	**0.002**	**0.004**
Peripheral manifestations				
Dactylitis, n (%)	33 (4.9)	27 (11.9)	**0.001**	**0.001**
Peripheral arthritis, n (%)	193 (28.8)	83 (37.2)	**0.019**	**0.017**
Enthesitis, n (%)	122 (18.2)	39 (17.4)	0.841	--
Extra-musculoskeletal manifestations (EMMs)				
Psoriasis, n (%)	112 (16.7)	38 (16.9)	1	--
Uveitis, n (%)	215 (32.1)	93 (41.2)	**0.015**	**0.007**
IBD[3], n (%)	54 (8.0)	42 (18.6)	**<0.001**	**<0.001**

Included n male- 686
Included n female 227

[1]Statistical significance calculated by Mann Whitney U or chi-square test of two proportions (Fisher's exact).
[2]Multiple regression, based on a binary outcome model, using gender, HLA-B27 and AxSpA disease duration as covariartes.
[3]Defined as ulcerative colitis or Crohn's disease.

P50

TRENDS DIAGNOSIS OF AXIAL SPONDYLARTHRITIS, PSORIATIC ARTHRITIS, AND SKIN PSORIASIS DURING COVID-19 PANDEMIC: A POPULATION STUDY IN THE ITALIAN LAZIO REGION

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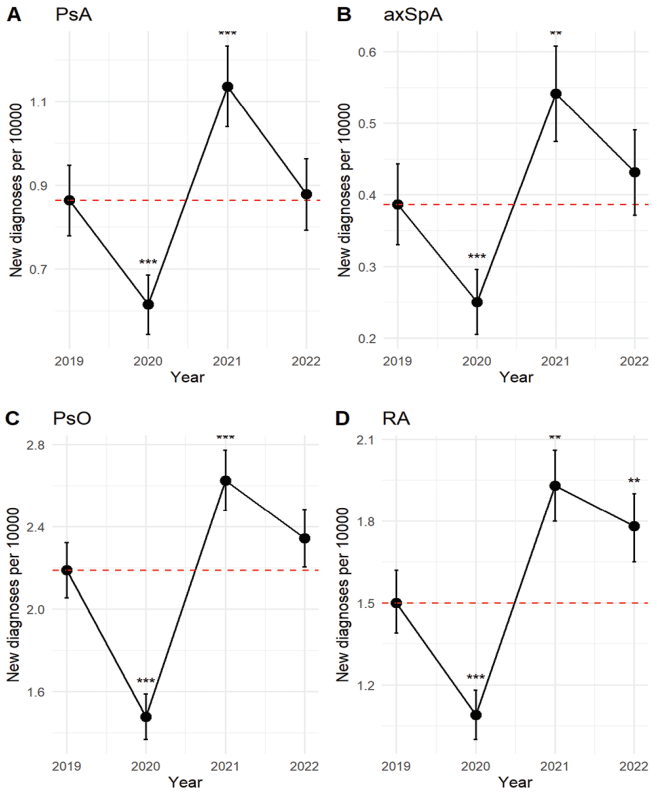
Introduction. SARS-CoV-2 has been suspected to increase the incidence of autoimmune and inflammatory rheumatic diseases. Conversely, limited access to healthcare services may have affected timely diagnoses. We aimed to evaluate trends in the diagnosis rates of axial spondyloarthritis (axSpA), psoriatic arthritis (PsA), and psoriasis (PsO) in Lazio, the second most populated region in Italy, from the pre-pandemic year of 2019 through to 2022. Rheumatoid arthritis (RA) incidence was evaluated as a comparator.
Methods. Annual diagnosis rates were derived from the registration of disease-specific exemption codes for each condition and the resident population. Incidence rate ratios (IRRs) with 95% confidence interval (CI) were calculated. The statistical significance of changes on follow-up compared to 2019 was determined using Poisson regression modeling.
Results. A total of 752 axSpA, 1631 PsA, and 4033 PsO diagnoses were registered over four years in a population of about 6.5 million inhabitants. The overall 2019 incidence (95% CI) per 10000 inhabitants of axSpA, PsA, and PsO were 0.39 (0.33-0.45), 0.86 (0.78-0.95), and 2.19 (2.06-2.33), respectively. The incidence of this three conditions followed a similar trend (Fig. 1). Compared to 2019, the diagnosis rate significantly decreased in 2020 and increased in 2021, while it returned to pre-pandemic levels in 2022. Specifically, the IRR (96% CI) comparing 2022 to 2019 was 1.11 (0.91-1.36) for axSpA, 1.02 (0.89-1.17) for PsA, and 1.07 (0.98-1.17) for PsO. Conversely, RA was diagnosed at a rate of 1.50 per 10,000 inhabitants (1.40-1.62) in 2019 and showed a net increase in diagnoses in 2022 compared to 2019 (IRR 1.18, 95% CI 1.07-1.30).
Conclusion. The COVID-19 pandemic led to a reduction in the diagnoses of axSpA, PsA, and PsO in 2020, followed by a rebound increase in 2021. By 2022, the diagnosis rates did not increase compared to the pre-pandemic period, in contrast to the rates for RA.



P50: Fig. I. Incidence trends of diagnoses from 2019 to 2022.
axSpA: axial spondyloarthritis; PsA: psoriatic arthritis, SpA: spondyloarthritis; RA: rheumatoid arthritis. *p<0.05, **p<0.01, ***p<0.001 compared to 2019.

P50: Table I. Incidence rates and incidence rate ratios from 2019 to 2022.

Disease	2019 IR for 10000 (95% CI)	2020 IR for 10000 (95% CI)	IR 2021 for 10000 (95% CI)	R 2022 for I10000 (95% CI)	IRR 2020 vs 2019 (95% CI)	IRR 2021 vs 2019 (95% CI)	IRR 2022 v vs 2019 (95% CI)
axSpA	0.39 (0.33-0.45)	0.25 (0.21-0.30)	0.54 (0.49-0.61)	0.43 (0.38-0.50)	0.64 (0.5- 0.82)	1.40 (1.16-1.69)	1.11 (0.91-1.36)
PsA	0.86 (0.78-0.95)	0.61 (0.55-0.69)	1.14 (1.04-1.24)	0.88 (0.80-0.97)	0.71 (0.61-0.83)	1.32 (1.16-1.50)	1.02 (0.89-1.17)
PsO	2.19 (2.06 2.33)	1.48 (1.37-1.59)	2.63 (2.48-2.78)	2.34 (2.21-2.49)	0.67 (0.61-0.74)	1.20 (1.10-1.30)	1.07 (0.98-1.17)
RA	1.50 (1.40-1.62)	1.09 (1.00-1.19)	1.93 (1.81-2.06)	1.77 (1.66-1.90)	0.72 (0.65-0.81)	1.28 (1.16-1.42)	1.18 (1.07-1.30)

axSpA: axial spondyloarthritis; IR: incidence rate; IRR: incidence rate ratio; PsA: psoriatic arthritis; SpA: spondyloarthritis; RA: rheumatoid arthritis.

P51

INCREMENTAL CARDIOVASCULAR RISK OF MENOPAUSE IN WOMEN WITH PSORIASIS, PSORIATIC ARTHRITIS OR SPONDYLOARTHRITIS?

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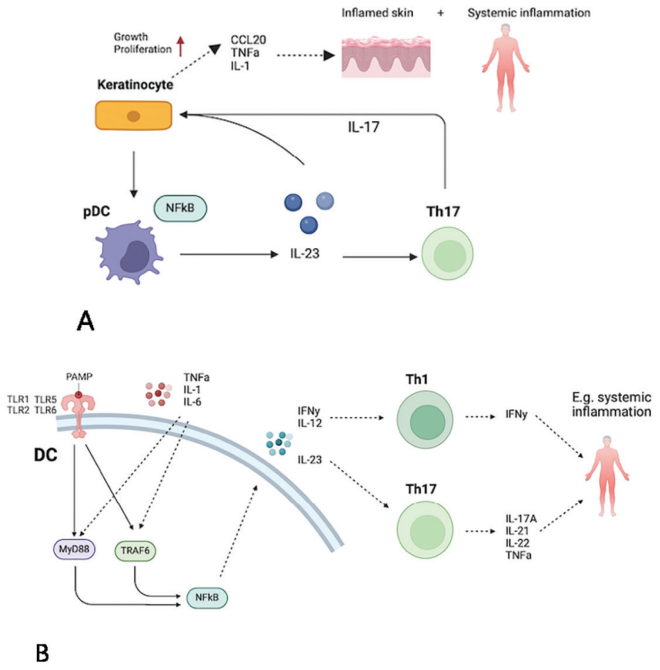
Introduction/Objective. This analysis aims to discuss the most recent studies regarding the cardiovascular disease (CVD) risk in women with psoriasis, psoriatic arthritis (PsA) and spondyloarthritis (SpA). In addition, the potential of menopause to modulate/increase CV risk in women with these diseases will also be explored. It is of significant value to gain more understanding into this topic because it may have meaningful implications for screening and treatment of CVD in these women.

Methods. A PubMed search identified studies on CVD risk in psoriasis (n=8), PsA (n=9), and SpA (n=7). Literature on menopausal pathophysiology was gathered using various relevant search terms.
Results. The risk of CVD in psoriasis, PsA, and SpA is increased in both men and women, which could mainly be attributed to elevated systemic inflammation (Fig. 1). The increased CVD risk specifically in women has only been investigated in psoriasis (1). Menopause, as well as psoriasis, PsA, SpA, are associated with various changes within the body potentially increasing CVD risk (Table I). After menopause, women with psoriasis (n=4927) may have an increased CVD risk as compared to pre-menopausal ones (n=4212); however, this trend is also observed in the general population (2). No existing literature conclusively demonstrates or more extensively studies whether psoriasis, PsA, or SpA amplifies this effect caused by menopause.
Discussion/Conclusion. The existing literature suggests that psoriasis, PsA and SpA could be independent CVD risk factors, and so is the natural menopausal transition. This literature review shows that there is almost no literature present that addresses the correlation between menopause and rheumatic diseases as psoriasis, PsA and SpA in the development of CVD. Therefore, the hypothesis that menopause represents an additional CV risk factor in women with psoriasis, PsA and SpA still needs to be thoroughly investigated.



P51: Fig. 1. Inflammatory pathway of psoriasis and PsA.
A) Psoriasis: growth and differentiation of keratinocytes leading to activation of NKκB pathway in pDCs, where after IL-23 is produced and Th17 cells get activated. These Th17 cells produce IL-17. IL-17 and IL-23 together cause keratinocytes to grow and proliferate even more, leading to CCL20, TNF-α and IL-1 excretion that results in inflamed skin cells and systemic inflammation. B) PsA: Activation of DCs by PAMPs, DAMPs and cytokines as TNF-α, IL-1 and IL-6, which initiates the intracellular pathway NFκB via MyD88 and TRAF. Next, cytokines as IFNγ, IL-12 and IL-23 are released from the DCs and activate Th1 and Th17 cells. Activated Th1 and Th17 excrete IFNγ, IL-17A, IL-21, IL-22 and TNF-α resulting in systemic inflammation.
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P51: Table I. Combination of the influence on CVD risk of rheumatic diseases and menopause.

	Body composition	Inflammation	Lipid profile
Influence by rheumatic disease	↑VAT	↑IL-1, IL-12, IL-23, TNF-α, IFNγ	↑ triglycerides
Influence by menopause	↑VAT ↑CF	↑IL-6, TNF-α, IFNγ	↓ HDL cholesterol ↑ LDL cholesterol
Combined prediction	Only ↑CF could possibly increase the CVD risk due to the specific location of fat accumulation. No other conclusions yet possible.	Could lead to additional CVD due to other cytokines involved (mainly IL-6).	No conclusion can yet be made whether this causes additional CVD risks.

VAT: visceral adipose tissue; CF: cardiovascular fat; CVD: cardiovascular disease; HDL: high-density lipoprotein; LDL: low-density lipoprotein.

References
1. LI WQ, HAN JL, MANSON JE *et al*.: Psoriasis and risk of nonfatal cardiovascular disease in U.S. women: a cohort study: Nonfatal CVD in women with psoriasis. *Br J Dermatol* 2012; 166(4): 811-18.
2. PIETRZAK A, CZUCZWAR P, MOSIEWICZ J *et al*.: Cardiovascular disease in psoriatic post-menopausal women. *Acad Dermatol Venereol* 2015; 29(6): 1231-34.

P52

FREQUENCY OF SARCOPENIA IN AXIAL SPONDYLOARTHRITIS, SANTO DOMINGO, DOMINICAN REPUBLIC

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Introduction. Axial spondyloarthritis (axSpA) compromises axial skeleton, radiographic changes and deterioration of functional capacity and quality of life of the patients. Sarcopenia is defined by European Working Group on Sarcopenia in the Elderly (EWGSOP) as the loss of muscle mass and strength with risk of adverse outcomes. The criteria assess muscle strength, quantity/quality of muscle and physical performance (gait speed), classifying musculoskeletal dysfunction: probable sarcopenia, sarcopenia and severe sarcopenia.
Methods. Descriptive, prospective, cross-sectional study, November 2023-April 2024. Inclusion criteria: ≥ 18 years, axSpA by ASAS 2009 criteria, ability to do bioimpedance and SARC-F. Exclusion criteria: difficulty performing evaluation tests, metallic prostheses, pacemaker, presence of another autoimmune inflammatory disease. Scales applied: ASDAS, BMI and SARC-F form, sarcopenia defined by EWGSOP 2018 criteria. Equipment: hand dynamometer (CAMRY model EH101), four-pole and multifrequency bioimpedance meter (Omron HBF 514C). Statistics were performed with SPSSv25.
Results. 33 met inclusion criteria. Female 51.5% (17), mean age 45 ± 12 years, mean duration of diagnosis 7 years. Alcoholism 33.3% (11), sedentary lifestyle 30.3% (10). Normal weight 54.5% (18), overweight 30.3% (10), obesity 15.2% (5). HT 21.2% (7), thyroid disease 21.2% (7), dyslipidemia 18.2% (6), DM 12.1% (4), smoking 9% (3), adalimumab 69.7% (23), golimumab 12.1% (4), secukinumab 12.1% (4), etarnecept 6.1% (2). ASDAS inactive 69.7% (23), low activity 21.2% (7), high activity 9.1% (3). SARC F ≤4 90.9% (30), SARC F ≥4 9.1% (3). EGWSOP 2018: Low muscle quantity or quality 12.1% (4), low muscle strength 9.1% (3), inadequate gait speed 9.1% (3). No sarcopenia 84.8% (28). Musculoskeletal dysfunction 15.2% (5): probable sarcopenia 9.1% (3), sarcopenia 6.1% (2).
Conclusion. A low frequency of sarcopenia was found. The most representative musculoskeletal dysfunction was probable sarcopenia. Low probability of sarcopenia when implementing SARC-F.

P53

FREQUENCY OF SARCOPENIA IN PSORIATIC ARTHRITIS, SANTO DOMINGO, DOMINICAN REPUBLIC

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Introduction. Psoriatic arthritis (PsA) is a frequently destructive arthritis associated with psoriasis. It can affect physical function, causing a deterioration in the quality of life of patients. Sarcopenia is defined by European Working Group on Sarcopenia in the Elderly (EWGSOP) as the loss of muscle mass and strength with risk of adverse outcomes. The criteria assess muscle strength, quantity/quality of muscle and physical performance (gait speed), classifying musculoskeletal dysfunction: probable sarcopenia, sarcopenia and severe sarcopenia.

Methods. Descriptive, prospective, cross-sectional study, November 2023-April 2024. Inclusion criteria: ≥18 years, PsA by CASPAR criteria, ability to do bioimpedance and SARC-F. Exclusion criteria: difficulty performing evaluation tests, metallic prostheses, pacemaker, presence of another autoimmune inflammatory disease. Scales: DAPSA28, BMI and SARC-F form, sarcopenia defined by EWGSOP 2018 criteria. Equipment: hand dynamometer (CAMRY model EH101), four-pole and multifrequency bioimpedance meter (Omron HBF 514C). Statistics were performed with SPSSv25.
Results. 47 met inclusion criteria. Female 62.5% (30), mean age 55 ± 12 years, mean duration of diagnosis 8 years. HT 41.7% (20), alcoholism 25% (12), sedentary lifestyle 25.5% (12). Normal weight 78.7% (37), obesity 14.9% (7), overweight 6.4% (3). Smoking 20.8% (10), DM 20.8% (10), dyslipidemia 16.7% (8), thyroid disease 14.6% (7). Methotrexate 52.1% (25). Glucocorticoids 4.2% (2), secukinumab 66.7% (32), adalimumab 25% (12), golimumab 4.2% (2), ustekinumab 2.1% (1). DAPSA28 remission 74.5% (35), low activity 25.5% (12). SARC F ≤4 87.2% (41), SARC F ≥4 12.5% (6). EGWSOP 2018: Low muscle strength 27.7% (13), low muscle quantity or quality 19.1% (9), inadequate gait speed 12.8% (6). No sarcopenia 55.3% (26). Musculoskeletal dysfunction 44.7% (21): probable sarcopenia 42.9% (9), sarcopenia 38.1% (8), severe sarcopenia 19% (4).
Conclusion. Half of the patients presented some degree of sarcopenia. The most representative musculoskeletal dysfunction was probable sarcopenia. Low probability of sarcopenia when implementing SARC-F.

P54

NOVEL DIAGNOSTIC 14-3-3H AUTO-ANTIBODY MARKER FOR AXIAL SPONDYLOARTHRITIS: A LONGITUDINAL STUDY

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Introduction. The lack of sensitive and specific markers for AxSpA complicates and delays diagnosis. Building upon prior research in 49 AxSpA patients, we aimed to validate the diagnostic utility of the novel 14-3-3η auto-antibodies (AAb) and its modifiability at year 1.
Methods. 14-3-3η AAb levels were assessed using Luminex® in 86 presumed healthy individuals and 114 AxSpA patients at baseline and at year 1. Cohorts were age and sex matched. 51% of AxSpA received TNF therapy. Using regression modeling and ROC analysis, the diagnostic performance of the 14-3-3η AAbs, factoring in covariates, was determined through the Youden index, with significance defined as $p<0.05$.
Results. Mann Whitney U-test demonstrated median 14-3-3η AAb (IQR) baseline levels were significantly higher in AxSpA compared to healthy patients, 44.38 (32.0-70.1) to 35.5 (21.8-46.0), $p=0.003$. Mean age in years (SD) was 40.6 (11.3) in AxSpA and 39.7 (11.3) in healthy cohort. 14-3-3η AAb ROC analysis including age, sex and CRP yielded an AUC and ChiSq of 0.82 and 81.96, $p<0.0001$, respectively. The optimal cut-off resulted in a sensitivity of 76.2% and a specificity of 74.1%, with 80 TP, 22 FP, 63 TN, and 25 FN. CRP and 14-3-3η AAb were available in 105 patients; 60 (57%) were CRP positive, 47 (45%) were AAb positive, and 83 (79%) tested positive for either marker.
Wilcoxon matched pairs demonstrated 14-3-3η AAb from baseline to year 1 were significantly different, p=0.029. Median changes in AAb did not differ significantly based upon who received TNF treatment ($p=0.71$) and were independent of HLA-B27 status ($p=0.16$).
Conclusion. This study confirms earlier findings reporting the diagnostic utility of 14-3-3η AAbs. We present for the first time the modifiability of the 14-3-3η AAbs in AxSpA and their potential for monitoring and on-going patient management.

P55

UNVEILING FABP2: A NOVEL BIOMARKER IN AXIAL SPONDYLOARTHRITIS DIAGNOSIS

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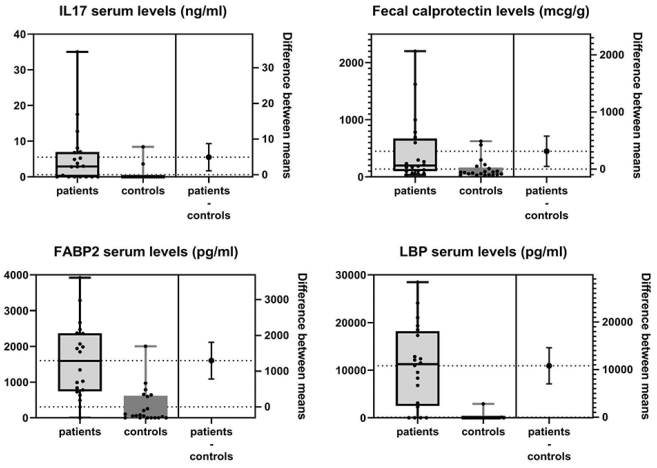
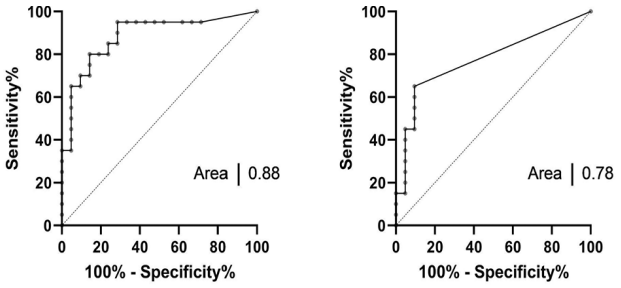
Introduction. Axial Spondyloarthritides (axSpA) are chronic inflammatory rheumatic diseases with an average diagnostic delay of over six years. Currently, validated biomarkers for diagnosis are limited to HLA-B27, C-reactive protein, and specific imaging findings. Notably, intestinal dysbiosis and increased permeability are key features of axSpA. This study aims to evaluate the diagnostic efficacy of two intestinal permeability biomarkers, Fatty Acid-Binding Protein 2 (FABP2) and Lipopolysaccharide-Binding Protein (LBP), in axSpA, in comparison with fecal calprotectin (fCAL) and serum IL-17 levels.
Methods. This cross-sectional study compared biomarker levels between axSpA patients and controls at a 1:1 ratio. Serum IL-17, FABP2, and LBP levels were quantified using ELISA, while fCAL levels were assessed using the Quantum Blue® rapid test. Logistic regression was performed and adjusted for potential confounders. Receiver Operating Characteristic (ROC) analysis was conducted to compare diagnostic performances.
Results. Forty-two participants were included, comprising twenty-one axSpA patients and twenty-one sex-matched healthy controls. IL-17 serum levels were significantly elevated in axSpA patients compared to controls (5.51ng/ml *vs* 0.57ng/ml; $p=0.01$), as were FABP2 (1,604pg/ml *vs* 309pg/ml, $p<0.0001$), LBP (10,946pg/ml *vs* 145pg/ml, $p<0.0001$), and fecal calprotectin levels (285mcg/g *vs* 137mcg/g; $p=0.02$). Among only axSpA patients, serum FABP2 levels were lower in TNFi users compared to non-users (2,193pg/ml *vs* 1,128/ml, $p=0.02$) and were negatively correlated with



P55: Fig. 1. Difference between groups in biomarkers levels.



P55: Fig. 2. ROC analysis to prediction of axSpA diagnosis by:.

daily fiber intake (r-0.6; p=0.007). Multiple logistic regression revealed associations of IL-17 and FABP2 with axSpA diagnosis, with respective odds ratios of 1.40 (95%CI=1.02-2.14) and 1.28 (95%CI=1.11-1.60). ROC analysis yielded an AUC of 0.88 (p<0.0001) for FABP2 and 0.78 (p=0.002) for IL-17 in predicting axSpA diagnosis.

Conclusion. In this cohort, FABP2 demonstrated significant diagnostic potential for axSpA, surpassing IL-17, LBP, and fCAL. Larger studies are warranted to validate these findings and explore additional biomarker applications.

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P56

ROOT JOINT INVOLVEMENT IN SPONDYLOARTHRITIS: DATA FROM A LARGE MULTICENTRIC COHORT

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Introduction. Although root joint involvement (RJI) predicts disease severity in Spondyloarthritis, studies focusing on this involvement are still lacking. This study aimed to determine the prevalence and factors associated with RJI in the Brazilian Registry of SpA (RBE).

Methods. RBE is a multicentric, observational, prospective cohort of patients with SpA. RJI was defined as pain or limitation of the shoulder or hip on clinical examination. Comparisons between the RJI and non-RJI groups were conducted using univariate analysis (Student's t-test and chi-squared test) and multivariate analysis (logistic regression model).

Results. 1175 patients were enrolled; 65% were male; mean age of 48.8±13.2 years and mean symptom duration of 15.7±10.5 years; 67% were HLA-B27 positive. RJI was observed in 580 patients (49.3%), 39.6% in the hip and 29.3% in the shoulder. Patients with RJI had a typical axial presentation at diagnosis, including more frequent neck (p<0.001), low-back (p<0.001) and buttock (p=0.010) pain, without difference regarding HLA-B27 or extra-musculoskeletal manifestations. Furthermore, this group exhibited more active peripheral disease at study inclusion, with more frequent arthritis and enthesitis (p<0.001 for both), as well as a higher count of painful joints (p=0.001) and entheses (p=0.001). The RJI group experienced longer diagnostic delay (p=0.003) and higher disease burden, reflected by worse BASDAI, ASDAS-CRP, BASMI, BASFI, ASQoL (p<0.001 for all), greater prior exposure to b/tsDMARDs (p=0.003), and less frequent active work status (p<0.001) (Table I). In the multivariate analysis, RJI remained associated with predominantly axial manifestations at diagnosis (OR=1.76; 95%CI 1.11-2.82; p=0.017), current peripheral arthritis (OR=2.96; 95%CI 1.95-4.55; p<0.001), ASDAS-CRP (OR=1.27; 95%CI 1.06-1.54; p=0.011), BASFI (OR=1.14; 95%CI 1.03-1.26; p=0.013), BASMI (OR=1.16; 95%CI 1.04-1.29; p=0.007), and ASQoL (OR=1.06; 95%CI 1.01-1.11; p=0.015).

Conclusion. Our data confirm that RJI is associated with an axial phenotype and indicates a more severe disease, concomitant with a peripheral component and a higher disease burden.

P56: Table I. Univariate analytical statistics comparing spondyloarthritis patients with (RJI SpA) and without (non-RJI SpA) root-joint involvement in Brazilian Registry of Spondyloarthritis - RBE.

Characteristic	RJI Sp (n=580)	non-RJI SpA	p-value
Sociodemographic characteristics:			
Age, mean (SD), years	49.1 (12.8)	48.2 (13.8)	0.243
Male sex, n (%)	376 (64.8)	385 (65.3)	0.878
Disease duration, mean (SD)	16.1 (10.4)	15.4 (10.6)	0.364
Disease characteristics at diagnosis:			
HLA-B27 positivity, n (%)	309 (66.3)	306 (66.8)	0.871
Diagnosis delay, mean (SD), years	8.1 (8.3)	6.1 (7.3)	**0.003**
Axial SpA classification	474 (81.7)	438 (73.9)	**0.003**
Neck pain, n (%)	199 (34.3)	137 (23.0)	**<0.001**
Low-back pain, n (%)	417 (71.9)	375 (63.0)	**<0.001**
Buttock pain, n (%)	175 (30.2)	140 (23.5)	**0.010**
Peripheral manifestations at the time of study enrollment:			
Peripheral arthritis, n (%)	173 (36.7)	88 (21.5)	**<0.001**
Painful-joint count, mean (SD)	11.2 (14.6)	5.4 (8.8)	**0.001**
Swollen-joint count, mean (SD)	2.1 (4.2)	1.9 (3.7)	0.694
Enthesitis, n (%)	176 (37.1)	100 (24.2)	**<0.001**
Painful-enthesis count, mean (SD	8.2 (7.5)	4.7 (5.8)	**<0.001**
Disease burden related outcomes:			
BASDAI, mean (SD)	4.7 (2.5)	2.7 (2.2)	**<0.001**
ASDAS-CRP, mean (SD)	2.8 (1.2)	1.9 (1.1)	**<0.001**
BASMI, mean (SD)	4.2 (2.1)	3.2 (1.9)	**<0.001**
BASFI, mean (SD)	5.1 (3.2)	1.2 (2.3)	**<0.001**
ASQoL, mean (SD)	9.4 (5.2)	5.2 (5.0)	**<0.001**
Active work situation, n (%)	188 (38.9)	240 (55.0)	**<0.001**
≥1 prior b/tsDMARDs use, n (%)	73 (12.6)	48 (8.1)	**0.003**

HLA-B27: Human Leucocyte Antigen B27; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; ASDAS-CRP: Axial Spondyloarthritis Disease Activity Index; BASMI: Bath Ankylosing Spondylitis Metrology Index; BASFI: Bath Ankylosing Spondylitis Functional Index; ASQoL: Ankylosing Spondylitis Quality of Life; b/tsDMARDs: biological/targeted synthetic disease-modifying antirheumatic drug.

P57

THE LONGITUDINAL ASSOCIATION BETWEEN DISEASE ACTIVITY, FUNCTION AND HEALTH-RELATED QUALITY OF LIFE IN AXIAL SPONDYLOARTHRITIS: RESULTS FROM THE DESIR COHORT

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Introduction. Health-related quality of life (HRQoL) in axSpA is considered an overarching outcome, with other disease outcomes contributing to it (1). To validate a previously proposed model on the relationship between disease outcomes, we aimed to investigate the longitudinal relationship between disease activity and function (independent variables) and HRQoL (outcome).

Material and methods. AxSpA patients with symptoms<3 years, enrolled in the DESIR cohort, were included. Outcomes were collected longitudinally up to 10 years. The association between disease activity (ASDAS) and physical function (BASFI), on the one hand, and HRQoL (outcome: SF36-MCS, SF36-PCS, ASQoL) was assessed. SpA features, personal and environmental factors, and therapy were collected at baseline (sex, age, HLA-B27 positivity, MRI/radiographic sacroiliitis) or during follow-up (psoriasis, IBD, uveitis, enthesitis, dactylitis, peripheral arthritis, BMI, smoking, job type, education, marital status, parental status, NSAIDs/bDMARDs use, comorbidities) and tested as confounders or effect modifiers. Generalised estimating equations (GEE) were built with SF36-MCS, SF36-PCS, or ASQoL as outcomes (separate models), and ASDAS and BASFI as main independent variables. Covariates were included in the final multivariable models, conducted both as simple GEE models and autoregressive models (corrected for HRQoL at the previous time point). Results were expressed as beta coefficient (Beta) and 95% confidence interval (95% CI).

Results. A total of 663 axSpA patients (46% males, mean age 33.5 [8.6] years) were included. In simple models including only ASDAS and BASFI as independent variables, significant associations were found with SF36-MCS (Beta -2.43[-2.84,-2.04]; -1.23[-1.43,-1.03]), SF36-PCS (-3.07[-3.32,-2.83]; -2.24[-2.37,-2.11]), and ASQoL (1.35[1.23,1.48]; 1.16[1.09,1.22]). These results were confirmed in autoregressive models: effect sizes for ASDAS and BASFI varied modestly when corrected for relevant confounders (Table I).

Conclusion. A longitudinal relationship between disease activity and function, on the one hand, and HRQoL as the outcome, has been demonstrated, confirming that HRQoL can be interpreted as an overarching outcome in axSpA.

Acknowledgments. The DESIR cohort is conducted as a programme hospitalier de recherche clinique (PHRC) with Assistance Publique–Hôpitaux de Paris as the sponsor. This cohort has also been supported via unrestricted grants from Pfizer France and the French society of Rheumatology.

Reference

1. MACHADO P *et al.*: *Ann Rheum Dis* 2011; 70: 1758-64.

P57: Table I. Longitudinal association beetween disease activity, function and health related quality of life.

	SF36-MCS N=650	SF36-PCS N=599	ASQOL N=630
	Beta (95%CI)	Beta (95%CI)	Beta (95%CI)
ASDAS	-2.38 (-2.91, -1.86)	-2.93 (-3.28, -2.58)	1.19 (1.02, 1.36)
BASFI	-1.10 (-1.36, -0.84)	-2.13 (-2.31,-1.96)	1.08 (0.99, 1.17)
Male Sex	-0.35 (-1.40, 0.69)	1.07 (0.35, 1.79)	-0.64 (-1.03, -0.25)
Age (years)	-0.02 (-0.07, 0.04)	0.01 (-0.03, 0.05)	-0.01 (-0.03, 0.01)
Symptom duration (years)	N/A	N/A	N/A
HLA B27	0.44 (-0.62, 1.51)	0.31 (-0.43, 1.04)	-0.04 (-0.43, 0.35)
Radiographic sacroiliitis (baseline)	N/A	0.83 (-0.06, 1.72)	N/A
MRI sacroiliitis (baseline)	N/A	0.38 (-0.36, 1.13)	-0.19 (-0.57, 0.19)
Psoriasis	N/A	N/A	N/A
IBD	-0.95 (-2.46, 0.54)	-1.58 (-2.58, -0.59)	0.55 (0.03, 1.06)
Uveitis	N/A	N/A	N/A
MASES (0-13)	-0.04 (-0.15, 0.06)	-0.04 (-0.11, 0.03)	0.06 (0.02, 0.10)
SJC (0-28)	N/A	-0.02 (-0.36, 0.32)	0.05 (-0.11, 0.21)
Dactylitis	N/A	N/A	N/A
BMI (kg/m^2)	0.06 (0.05, 0.16)	0.10 (0.02, 0.17)	0.02 (0.07, 0.01)
University education (yes/no)	-0.24 (-1.31, 0.81)	-0.15 (-0.88, 0.57)	-0.18 (-0.57, 0.21)
Married/living Maritally (yes/no)	1.55 (0.63, 2.47)	N/A	N/A
Parental status (n of children)	-0.22 (-0.67, 0.22)	-0.19 (-0.30, 0.26)	0.03 (-0.11, 0.18)
Blue collar job (yes/no)	N/A	N/A	N/A
Active smoking (yes/no)	-0.63 (-1.49, 0.21)	N/A	N/A
NSAIDs (yes/no)	0.11 (-0.65, 0.88)	-1.34 (-1.85,-0.83)	0.34 (0.09, 0.59)
bDMARDs (Yes/no)	-0.92 (-1.78, -0.07)	-1.55 (-2.12, -0.97)	0.31 (0.02, 0.61)
Comorbidity count (n)	N/A	-0.45 (-0.79, -0.12)	0.05 (-0.11, 0.21)

Autoregressive multivariable Generalized Estimating Equations Models having each one of the Health Related Quality of Life outcome.
in bold: statistically significant association, p<0.05; ASDAS: Axial Spondyloarthritis Disease Activity Score; BASFI: Bath Ankylosing Spondylitis Functional Index; HLA: Human Leukocyte Antigen; MRI: Magnetic Resonance Imaging; IBD: Inflammatory Bowel Disease; MASES: Maastricht Ankylosing Spondylitis Enthesitis Score; SIC: Swollen Joint Count; BMI: Body Mass Index; NSAIDs; Non-steroidal Anti Inflammatory Drugs; N/A: not applicable; variable not included in the model because not associated to the outcome at univariable or for collinearity.

P58

DIFFICULT-TO-TREAT, SPONDYLOARTHRITIS IN A REAL-WORLD MULTICENTRIC COHORT

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Introduction. Despite optimized management, some patients with axial spondyloarthritis (axSpA) remain uncontrolled. Consensus on the definition of difficult-to-treat (D2T) axSpA is lacking. This study aims to identify D2T-axSpA patients in the Brazilian Registry of Spondyloarthritis (RBE) and compare them with non-D2T-axSpA.

Methods. RBE is a multicentric, observational, cohort of SpA patients. D2T-axSpA was defined by active disease (ASDAS-PCR≥2.1), prior use of ≥2b/tsDMARDs, and patient or physician global assessment (VAS-Global) ≥5.

Results. 914 axSpA patients were included: 70% males, mean age 47.5 years, 67% HLA-B27 positives and 48 (5.25%) classified as D2T-axSpA. In univariate analysis (Table I), the D2T group showed a higher frequency of lower-limb arthritis p=0.001) and dactylitis p=0.007) at diagnosis and greater rates of enthesitis (p=0.004) and root joint involvement (p<0.001) at inclusion. Inflammatory bowel disease (IBD) (p=0.005), psoriasis (p=0.021), depression (p=0.002), fibromyalgia (p<0.001), and obesity (p<0.001) were more prevalent in D2T-axSpA patients. Worse BASFI (p=0.041), ASQoL

P58: Table I. Univariate analytical statistics with comparisons between D2T axSpA and non-D2T axSpA in Brazilian Registry of Spondyloarthritis - RBE.

Characteristic	SpA D2T (n=48)	SpA nD2T (n=866)	p-value
Sociodemographic characteristics and musculoskeletal manifestations			
Age, mean (SD), years	47.3 (13.1)	47.6 (12.9)	0.875
Male sex, n (%)	33 (68.8)	604 (69.8)	0.877
Disease duration, mean (SD)	19 (12.3)	16.8 (10.7)	0.239
HLA-B27 positivity, n (%)	33 (69.2)	632 (73)	0.606
Root-joint involvement, n (%)	16 (33.3)	119 (13.7)	**<0.001**
Lower limbs arthritis at diagnosis, n (%)	27 (56.3)	290 (33.5)	**0.001**
Dactylitis at diagnosis, n (%)	8 (16.7)	56 (6.5)	**0.007**
Enthesitis count at inclusion, mean (SD)	10.4 (8.1)	6.9 (6.5)	**0.004**
Active work situation, n (%)	11 (24.4)	323 (49.5)	**0.013**
BASFI, mean (SD)	7.5 (2.1)	3.1 (3.3)	**0.041**
ASQoL, mean (SD)	10.6 (4.5)	7.4 (5.4)	**<0.001**
VAS-Pain (patient), mean (SD)	6.3 (2.4)	4.4 (3.0)	**<0.001**
Extra-Musculoskeletal Manifestations (EMM) and Comorbidities			
Inflammatory Bowel Disease - IBD, n (%)	6 (12.5)	34 (3.9)	**0.005**
Psoriasis, n (%)	7 (14.6)	53 (6.1)	**0.021**
Uveitis, n (%)	12 (25)	251 (29)	0.553
Depression, n (%)	9 (18.8)	59 (6.8)	**0.002**
Fibromyalgia, n (%)	10 (20.8)	60 (6.9)	**<0.001**
Obesity, n (%)	10 (20.8)	57 (6.6)	**<0.001**
Treatment			
Prior cDMARDs, n (%)	33 (68.7)	296 (34.2)	**<0.001**
Current cDMARDs, n (%)	28 (58.3)	271 (31.5)	**<0.001**
Current b/tsDMARDs, n (%)	38 (79.2)	556 (64.2)	**0.034**
Current non-TNFi bDMARDs, n (%)	15 (31.3)	49 (5.7)	**<0.001**
Current antidepressant-anxiolytic drugs, n (%)	21 (42.9)	171 (19.7)	**<0.001**

IBD: Inflammatory bowel disease; HLA-B27: Human Leucocyte Antigen B27; BASFI: Bath Ankylosing Spondylitis Functional Index; ASQoL: Ankylosing Spondylitis Quality of Life; VAS-Pain: Visual analogue scale for pain; cDMARDs: conventional Disease-Modifying Antirheumatic Drugs; b/tsDMARDs: biological/targeted synthetic disease-modifying antirheumatic drug; non-TNFi: other than TNF inhibitors

P58: Table II. Multivariate analytical statistics with D2T-axSpA as the dependent variable

	Odds-ratio	95%CI	*p*-value
Lower limbs arthritis at diagnosis	2.87	1.50-5.59	**0.002**
Inflammatory Bowel Disease – IBD	4.33	1.39-11.66	**0.006**
Depression	2.65	1.02-6.32	**0.035**
Obesity	2.42	1.01-5.46	**0.040**
Fibromyalgia	2.42	1.01-5.50	**0.042**
BASFI	1.21	1.07-1.37	**0.003**
Current non-TNFi bDMARDs	7.72	3.55-16.44	**<0.001**

BASFI: Bath Ankylosing Spondylitis Functional Index; non-TNFi: other than TNF inhibitors; CI: confidence interval.

(*p*<0.001), pain score (*p*<0.001) and a lower frequency of active work status (*p*=0.013) were observed in this group. They were more exposed to cDMARDs (*p*<0.001) and were using non-TNFi bDMARDs (*p*<0.001) and psychotropics (*p*<0.001) more frequently. In multivariate analysis (Table 2), D2T-axSpA remained associated with lower-limb arthritis at diagnosis (OR=2.87; 95%CI1.50-5.59; *p*=0.002), IBD (OR=4.33; 95%CI1.39-11.66; *p*=0.006)), depression (OR=2.65; 95%CI1.02-6.32; *p*=0.035), obesity (OR=2.42; 95%CI1.01-5.46; p=0.040), fibromyalgia (OR=2.42; 95%CI1.01-5.50; *p*=0.042), BASFI (OR=1.21; 95%CI1.07-1.37; *p*=0.003), and current use of non-TNFi bDMARDs (OR=7.72; 95%CI3.55-16.4; *p*<0.001).

Conclusion. The group of patients with D2T-axSpA in this Brazilian cohort was associated with a more significant "peripheral" phenotype, extra-musculoskeletal manifestations (EMM), and comorbidities, showing a more profound impact on work, physical function and quality of life.

As the concept of D2T-axSpA is still evolving, our data highlight the importance of including EMM and comorbidities, as well as considering physical, psychological, and social domains, in the construction of this concept.

P59

CHARACTERISTICS OF PATIENTS WITH PSORIATIC ARTHRITIS IN A NEWLY ESTABLISHED MULTIDISCIPLINARY PRACTICE

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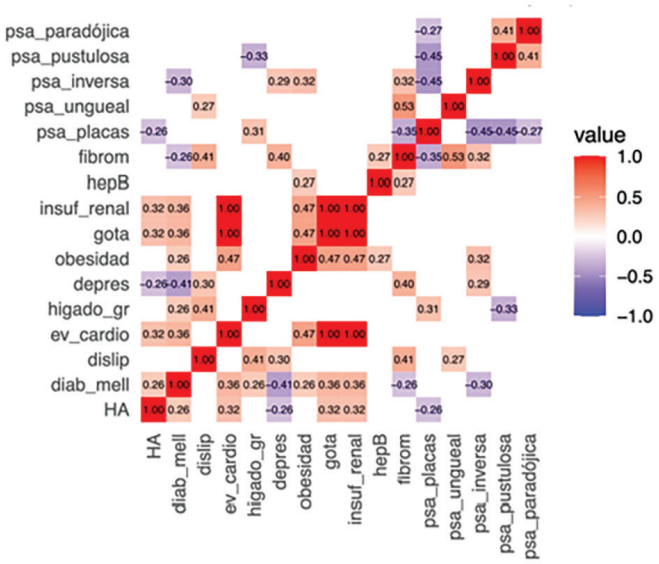
Objective. To characterize patients with psoriatic arthritis in a newly established joint Rheumatology-Dermatology unit at the University Clinical Hospital of Valencia.

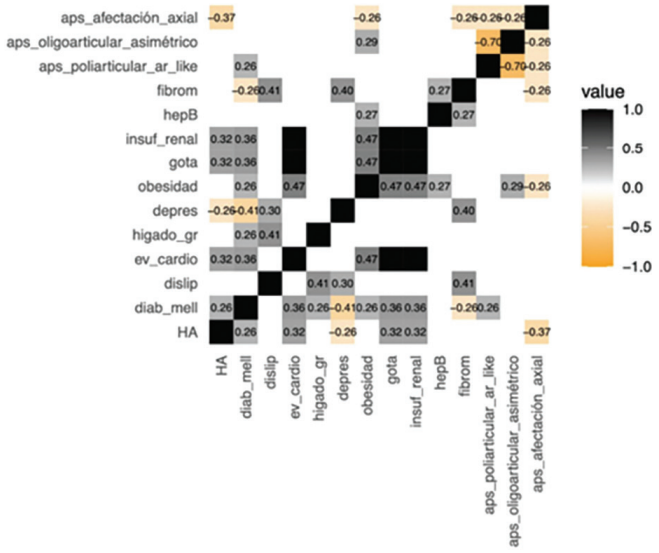
Methods. Cross-sectional observational study. Patients diagnosed with psoriatic arthritis (PsA) according to CASPAR criteria, who had their first visit between March and December 2023 at our monthly joint unit, were included. Demographic data, clinical data (including psoriasis severity (PASI), activity in peripheral form (DAPSA), and axial form (ASDAS)), comorbidities (frequency and correlation with type of Psoriasis (PsO) and PsA), and treatment were collected.

Results. 24 met CASPAR criteria, of which 15 (62.5%) were male, with a mean age of 53.9±13.6 years. The mean duration (months) since diagnosis was 150.7±121.2 for PsO and 78.9±60.4 for PsA. The most frequent type of PsO was plaque psoriasis in 15 (48.4%), followed by nail psoriasis in 10 (32.3%). The most frequent type of PsA was oligoarticular in 14 (58.3%), followed by axial and mixed forms in 3 (12.5%) respectively. PASI was mild in 12 (50%). According to DAPSA, 8 (33.3%) had remission-low activity and 2 (8.3%) high. In the axial form, had remission-low and high-very high activity en 3 (50%) respectively. The most frequent comorbidities were dyslipidemia 13 (54.2%), depression 8 (33%). Figure 1 and 2 shows the correlations between comorbidities and types of PsO and PsA. Previous treatments included one or more biological therapies in 15 (62.5%), JAK inhibitors in 3 (12.5%), and Apremilast in 7 (29.1%). Treatment change was agreed upon in 15 (62.5%) cases.

Conclusion. The duration since the diagnosis of psoriasis (12.5 years) was double compared to PsA (6.5 years). The majority (83.3%) had the peripheral form of PsA. Around 42% had moderate DAPSA, and in axial forms, 50% had high-very high activity. Cardiovascular events were highly correlated with renal insufficiency and gout, followed by obesity. There is some correlation between pustular psoriasis and hepatic steatosis, and nail psoriasis and fibromyalgia. More clinical data is needed to confirm this trend.



P59: Fig. 1. Correlation between comorbidities and types of psoriasis.
Psa_paradójica: psoriasis paradoxical, psa_pustulosa: pustular psoriasis, psa_inversa: inverse psoriasis, psa_ungueal: ointment psoriasis, fibrom: fibromyalgia, hepB: past hepatitis B, insuf_renal: renal insufficiency, gota: gout, obesidad: obesity, depres: depression, higado-gr: fatty liver, ev_cardio: cardiovascular event, dislip: dyslipidemia, diab-mell: diabetes mellitus, HA: hypertension.



P59: Fig. 2. Correlation between comorbidities and types of psoriatic arthritis.
Aps_afectación_axial: axial psoriatic arthritis, aps_oligoarticular_asimétrico: oligoarticular psoriatic arthritis, aps_poliarticular_ar_like: polyarticular psoriatic arthritis, fibrom: fibromyalgia, hepB: past hepatitis B, insuf_renal: renal insufficiency, gota: gout, obesidad: obesity, depres: depression, higado-gr: fatty liver, ev_cardio: cardiovascular event, dislip: dyslipidemia, diab-mell: diabetes mellitus, HA: hypertension.

P60

INVESTIGATING THE ASSOCIATION BETWEEN ALCOHOL CONSUMPTION AND SPINAL RADIOGRAPHIC PROGRESSION IN AXIAL SPONDYLOARTHRITIS

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Objective. We assessed whether alcohol consumption (AC) is associated with spinal radiographic progression in axSpA as measured by the modified Stoke Ankylosing Spondylitis Spinal Score (mSASSS). Progression was defined as an increase of ≥2 mSASSS units in 2 years. In addition, we evaluated to what extent this association is mediated through Ankylosing Spondylitis Disease Activity Score (ASDAS).

Methods. We used data from the University Health Network (UHN)- Spondyloarthritis cohort of axSpA patients with a follow-up of 6 years. Generalized estimating equation (GEE) analyses were conducted to determine the association between AC and spinal progression. Sensitivity analyses were performed to assess the impact of sex, and disease subgroup. Mediation analysis was performed to test whether the effect of alcohol consumption on spinal structural damage in axSpA patients operates through ASDAS.

Results. GEE analyses were performed in 769 records radiographic intervals from 385 patients (70% male, 84% radiographic-axSpA (r-axSpA), mean (±SD) age 38.6±13.0 years). 47% of patients reported consuming alcohol. The multivariable GEE models revealed increased odds of spinal progression in consumers, adjusted for important confounders (Table I). Higher odds of spinal progression were observed among males and patients with r-axSpA. With the mediation analysis, we demonstrated a significant direct effect of alcohol on spinal progression, suggesting greater spinal damage progression in consumers (Table II). The indirect effect of alcohol via ASDAS was negatively associated with spinal progression, as consumers had lower ASDAS and thereby lower odds of spinal progression. The total effect showed only a trend toward increased odds of progression.

Conclusion. These findings highlight the detrimental effect of alcohol on spinal progression in axSpA, especially in males and r-axSpA patients. The mechanisms underlying alcohol's effect remain unclear and our analysis failed to show that inflammation and symptoms driven by inflammation play a role in this relationship.

P60: Table I. Multivariable GEE model assessing the impact of alcohol consumption on spinal radiographic progression.

Variables	OR (95%CI)	*p*-value
Length of follow-up, years	1.17 (1.02, 1.35)	0.02
Age, years	1.03 (1.01, 1.06)	0.02
Male sex	3.21 (1.45, 7.11)	0.004
Alcohol consumption		0.01
Non-consumer	Ref	
Consumer	1.89 (1.15, 3.09)	
Disease duration, years	0.99 (0.96, 1.01)	0.29
Ever-smoking	0.75 (0.43, 1.31)	0.31
ASDAS	1.43 (1.14, 1.79)	0.002
Spinal damage at baseline	8.12 (3.72, 17.72)	< 0.001
HLA-B27 positive	0.91 (0.47, 1.75)	0.78
bDMARD use from the first set of x-rays to the last set of x-rays recorded	0.97 (0.54, 1.73)	0.92

Results from the univariable GEE model after linear imputations and excluding patients with missing covariate data. Spinal radiographic progression was defined as an increase of ≥2 mSASSS units in 2 years. The GEE model included only patients with complete observations. Analyses were performed in 769 radiographic intervals from 385 axSpA patients (148 progression events). The follow-up length signifies the years from baseline to the last recorded set of X-rays.
ASDAS: Ankylosing Spondylitis Disease Activity Score; bDMARD: biologic disease-modifying anti-rheumatic drug; CRP: C-reactive protein; Ref: reference.

P60: Table II. Mediation analysis of the impact of alcohol consumption on spinal progression with ASDAS as a mediator.

	OR (95%CI)	*p*-value
Direct effect of alcohol	1.92 (1.04, 2.81)	0.036
Indirect effect of alcohol (transmitted through ASDAS)	0.94 (0.85, 0.98)	0.012
Total effect of alcohol	1.81 (0.97, 2.53)	0.07

Results from the mediation analysis. 1000 bootstrap samples were used to calculate confidence intervals and p-values. Spinal radiographic progression was defined as an increase of ≥2 mSASSS units in 2 years. Analysis adjusted for length of follow-up, age, sex, ever-smoking, presence of syndesmophytes at baseline, disease duration, HLA-B27, and bDMARDs use. The direct effect signifies the direct effect of alcohol on spinal progression. The indirect effect means the effect of alcohol transmitted through ASDAS on spinal progression. The total effect includes both the direct and the indirect effect of alcohol on spinal progression. ASDAS: Ankylosing Spondylitis Disease Activity Score.

P61

THE EFFECT OF ALCOHOL CONSUMPTION ON CLINICAL OUTCOMES AND STRUCTURAL DAMAGE IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS: A SYSTEMATIC LITERATURE REVIEW AND META-ANALYSIS

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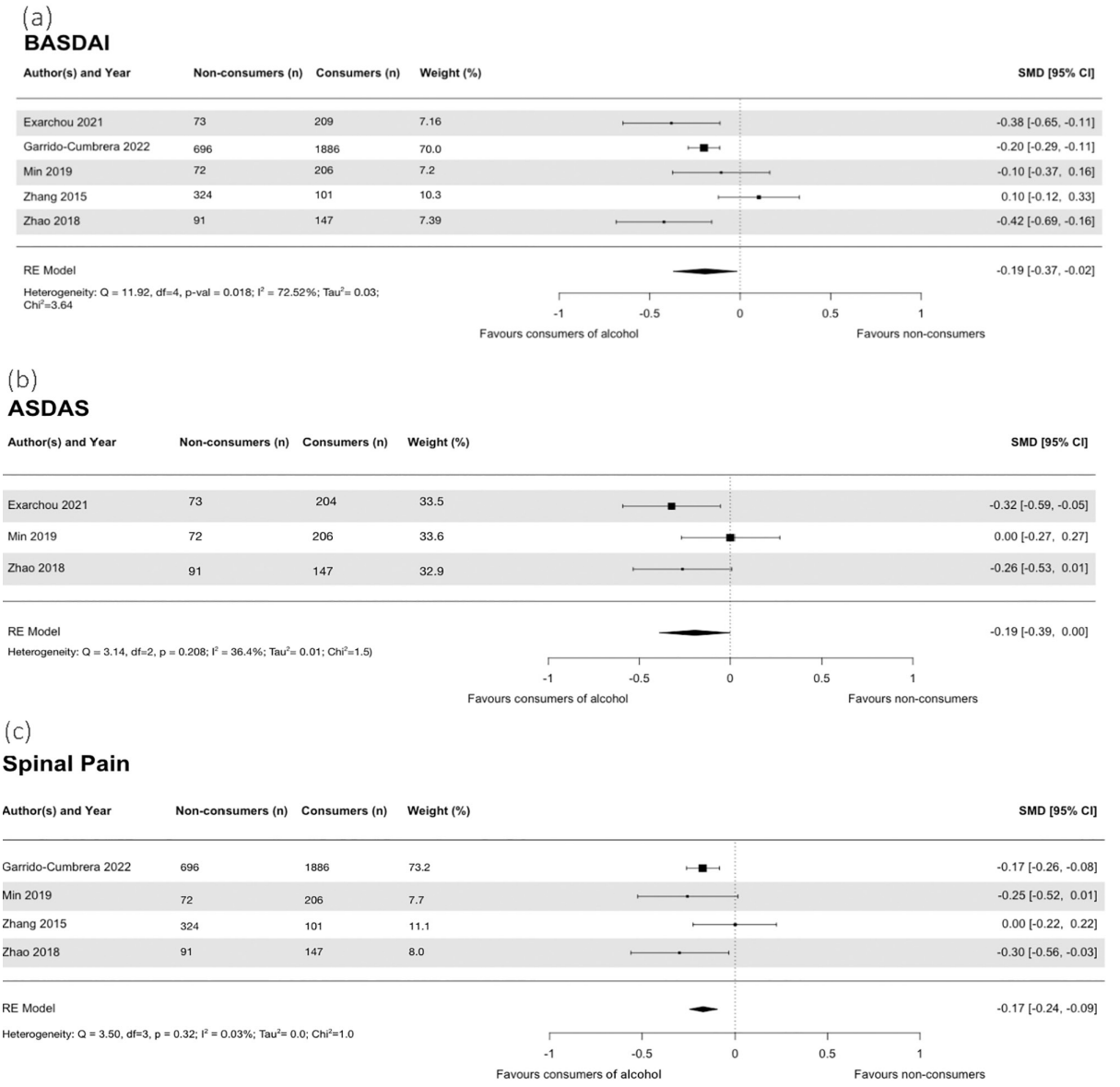
Introduction/Objective. Axial spondyloarthritis (axSpA) is a chronic progressive inflammatory disease characterized by the involvement of the axial skeleton. Alcohol consumption (AC) is a modifiable lifestyle factor with potential anti-inflammatory properties. Studies on the effects of alcohol in axSpA have been limited, with divergent conclusions. We aimed to systematically review the effects of alcohol consumption on disease-specific outcomes in axSpA.

Materials and methods. A systematic review of observational studies on axSpA and AC was conducted. Multiple electronic databases were searched for keywords. Two investigators reviewed articles to assess for inclusion eligibility. The Joanna Briggs Institute Critical Appraisal checklist was employed to evaluate the risk of bias. Standardized mean differences (SMD) were used to synthesize the data and I^2 was used to ascertain heterogeneity.

Results. Search strategy identified 703 records; 13 articles were assessed for eligibility. Five studies with a total of 3858 axSpA patients were included. Compared to non-consumers, axSpA patients who consumed alcohol had lower BASDAI (SMD -0.19, 95% CI -0.37 to -0.02, I^2=72.5%), lower spinal pain (SMD -0.17, 95% CI -0.24 to -0.09, I^2 = 0%) and a non-significant trend towards lower ASDAS (SMD -0.19, 95% CI -0.39 to 0.00, I^2 = 36%) (Fig. 1). One cohort study on the spinal radiographic progression indicated greater radiographic progression among consumers (SMD 0.35, 95% CI 0.08 to 0.62).

Conclusion. AC appears to be associated with lower disease activity and spinal pain. Further longitudinal cohort studies with standardized measures for AC are warranted to assess the direction of alcohol's effect on structural damage progression.



P61. Fig. 1. Forest plots of (**a**) BASDAI, (**b**) ASDAS, and (**c**) spinal pain.

P62

SOMATOTYPE IN SPONDYLOARTHRITIS: A PREVALENCE STUDY IN A BRAZILIAN TERTIARY HOSPITAL

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Objective. This study assessed the prevalence of somatotypes in different types and subtypes of spondyloarthritis based on the anthropometric, demographic, and phenotypic data.
Methods. The Heath and Carter method was used to determine the somatotype in 61 patients with spondyloarthritis who were being treated at a teaching hospital in Brazil. Analysis of variance and Fisher's exact tests were used to statistically analyze the results.

Results. The sample included individuals who were predominantly male (68.9%), Caucasian (63.9%), age [54.8±13.68 years], height [1.68±0.1 meters], total body mass [81.64±12.59 kg], body mass index [29.06 kg/m 2±4.23], fat percentage [28.94±5.25], disease time [20.38±10.44 years], and diagnosis time [16.6±10.3 years]. In the types of spondyloarthritis, meso-endomorph was more prevalent [axial = 39 (41%) and peripheral = 22 (45.5%)], with no direct relationship between the subtypes, but with meso-endomorph tendency, in the enthesopathic [6 (45.5%)] and intestinal phenotypes [2 (7.7%)]. Ankylosing spondylitis was characterized by hypertrophy and thinness, with the absence of cutaneous phenotype ($p<0.05$), psoriatic spondyloarthritis due to hypotrophy and thinness with the presence of the cutaneous phenotype ($p<0.05$), Meso-endomorph and mesomorph endomorph aggregate three phenotypes, whereas endo-mesomorph and endomorph mesomorph two.
Conclusion. The study highlights to a heterogeneous spectrum on anthropometric distribution of spondyloarthritis, which can be considered for guidelines and individual treatment decisions.

P63

SOMATOTYPE IN SPONDYLOARTHRITIS AND THEIR SOCIAL INTERACTIONS

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Spondyloarthritis (SAs) presents a heterogeneous anthropometric spectrum, impacted by the progression of the disease and by social profile perceived by social determinants of health (SDH).The study identified the SDH, through the Dahlgren & Whitehead model, determining their interaction with anthropometry characterized in 61 subjects with SAs treated at the Clementino Fraga Filho University Hospital of the Federal University of Rio de Janeiro and statistically analyzed by ANOVA and Fisher's association tests. The individual's SDH in the sample were as follows: 68.9% men; 31.1% women; 63.9% white; 29.5% brown; 6.6% black; mean age of 54.8±13.68 years; 68.9% from the city of Rio de Janeiro; 22.9% from the municipalities of Greater Rio de Janeiro; and 8.1% from Niterói, São Gonçalo and the lakes region. The analysis of the proximal and intermediate social determinants showed the following characteristics of the sample: married, employed or retired, with one child, having three meals per day, living in good sanitary conditions, with accessibility to transportation and health services. The social sample was heterogeneous, and there was an impact on sleep quality and work environment. Meso-endomorph and mesomorph-endomorph somatotypes were employed (p=0.033) and had behavior-related problems (p=0.022); meso-endomorph somatotypes tended to have better sleep quality (p=0.085). Individuals with ankylosing spondylitis have more access to health services, calm behavior and tend to have more than one child. Conversely, individuals with psoriatic SAs presented a depressive and anxious personality. Body composition was strongly influenced by illness and correlated well with SDH.

P64

IS THE DIAGNOSTIC DELAY GETTING SHORTER WITH TIME FOR PATIENTS WITH SPONDYLOARTHRITIS? DATA FROM THE INTERNATIONAL ASAS-PERSPA STUDY

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Introduction. Data on the impact of advanced management in spondyloarthritis (SpA) on diagnostic delay (DD) are still conflicting. In this ancillary analysis from the international ASAS-PerSpA study, we aimed to estimate the change in DD over time, stratified by world region and country socioeconomic development level.
Materials and methods. Patients diagnosed by their rheumatologists with any SpA entity were included cross-sectionally in 2020. DD was defined by the time lag between the first musculoskeletal manifestation (MM) and date of SpA diagnosis. The mean diagnostic delay was compared across decades of onset of the first MM, from 1960 until 2009. Data was presented descriptively for the whole cohort, by world region, and country Human Development Index (HDI). Correlation between DD and date of the first MM was evaluated using the Mann-Kendall statistical test for trend by SpA entity, world region, and HDI.
Results. The analysis included 4,339 patients with SpA (2622 axSpA, 1016 PsA, 424 pSpA, 110 IBD-SpA, 167 others), mean age 44.4 years (±13.9), 60.9% females; 38% from Europe and North America, 28% MENA, 22% Asia, and 12% Latin America; 42% from very high HDI, 35% from high HDI, and 23% from medium HDI countries. In the whole cohort, the mean DD was 4.5 years (±7.0) and was associated with the world region (shorter DD in Asia) and the HDI (shorter DD with higher HDI). Over time, DD decreased in the whole population (Kendall tau -0.350, p<0.001) (Fig. 1). The same trend was observed across the main SpA entities, the 4 world regions, and the HDI categories (p-value <0.001) (Fig. 2).
Conclusion. Based on the cross-sectional data from the ASAS-PerSpA study, the DD in SpA seems to decrease over time, regardless of the world region, HDI, and SpA entities. These results should be confirmed in dedicated prospective cohorts.

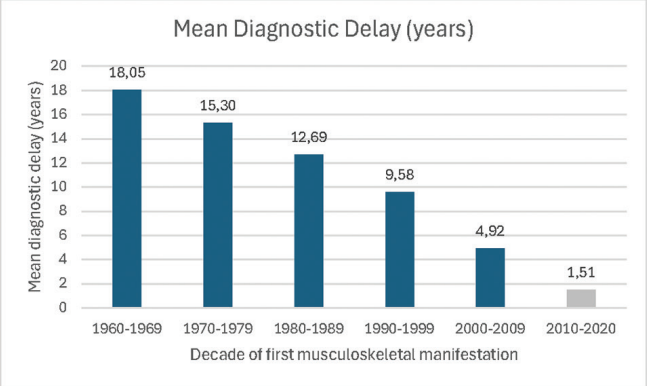

Fig. 1. Evolution of diagnostic delay over time in patients diagnosed with spondyloarthritis in the Whole ASAS-Perspa Cohort.

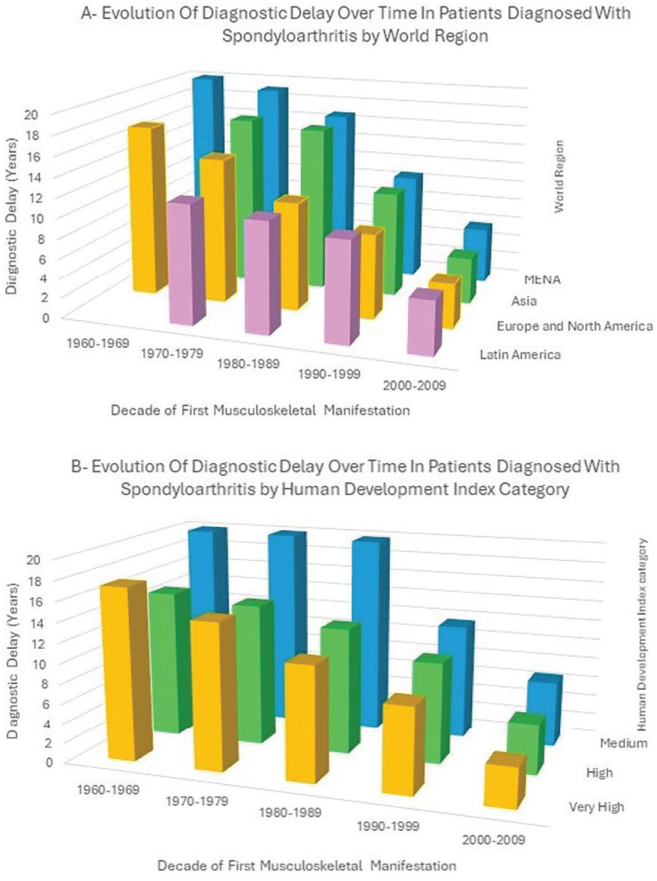

Fig. 2. Evolution of diagnostic delay over time in patients diagnosed with spondyloarthritis by World Region (A) and Human Development Index (B).

P65

SERUM CALPROTECTIN (S100A8/9) AND COMPLEMENT FACTOR C3 AS POTENTIAL INFLAMMATORY MARKERS IN OBESE AND "CRP NEGATIVE" EARLY PSA PATIENTS: DATA FROM METAPSA COHORT

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Objective. The aim of this study was to investigate whether alternative inflammation markers perform better than CRP in detecting systemic inflammation in early PsA patients.

Methods. Adult patients with early (median disease duration:6 months) DMARD-naïve PsA were compared to sex- and age matched controls (HC) and early (disease duration:5 months) DMARD-naïve RA patients. CRP, serum amyloid A (SAA), serum calprotectin (S100A8/9), complement C3, thrombocytes, ferritin were measured in the serum. The markers were compared in PsA, RA patients and HC having normal CRP (≤5 mg/L) (Kruskal-Wallis). Same markers were compared in RA, PsA and HC having BMI ≥30. Sensitivity and specificity analysis (ROC) was conducted.

Results. Thirty nine of 67 (58%) PsA, 29/50 (56%) RA patients and 57/61 (93%) HC had normal CRP. The median [IQR] levels of C3 (g/L) and S100A8/9 (hg/ml) were higher in "CRP-negative" PsA (1.19[0.38] and 1523[840] accordingly) than in HC (0.92[0.24] and 1071[978]) (p<0.0001 and p=0.002) (Fig. 1). No difference between PsA and RA patients was observed. The levels of SAA, thrombocytes, ferritin were comparable in "CRP-negative" PsA, RA and HC. In the obese PsA (27/67, 40.3%) and RA patients (14/50, 28%) the levels of S100A8/9 were increased as compared to obese HC (11/61, 18.3%) (p=0.004). The levels of CRP, C3, SAA, thrombocytes, ferritin were comparable in the three groups. ROC curve analysis of C3 in PsA patients and HC having normal CRP showed sensitivity of 71.1% and specificity of 60.4% for a cut off value of 0.99 g/L and AUC of 0.734 [0.63-0.839] (Fig. 2A-B).

Conclusion. In "CRP-negative" PsA patients complement C3 and serum calprotectin are higher than in HC. In obese PsA patients' serum calprotectin but not CRP is increased as compared to HC. Complement C3 and serum calprotectin could be used in a selected population of early PsA patients for measuring systemic inflammation.

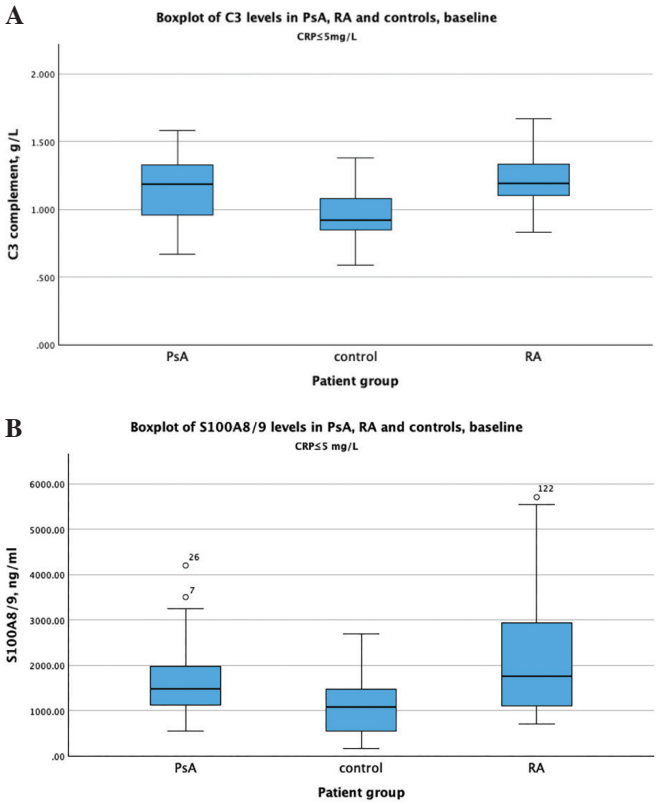

Fig. 1. Levels of complement fraction C3, S100A8/9and SAA in PsA, RA patients and HC having CRP≤5 mg/L, baseline.
A. Levels of C3 complement fraction at baseline were higher in PsA and RA as compared to control group (Kruskal-Wallis, p<0.001). **B**. Levels of S100A8/9 at baseline were higher in PsA and RA as compared to control group (Kruskal-Wallis, p<0.001).

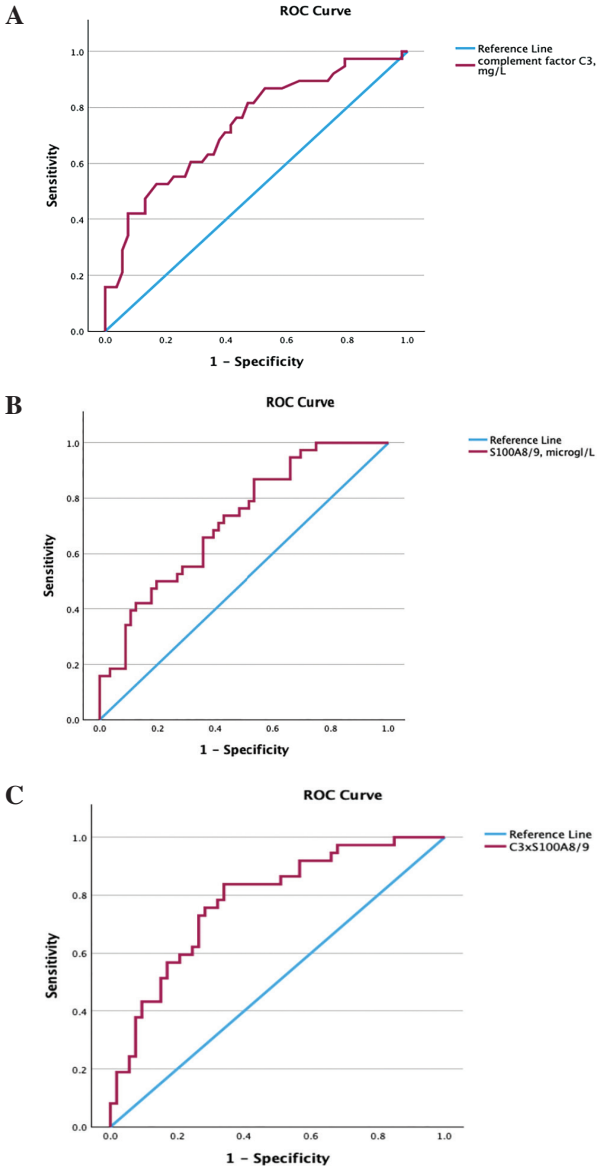

Fig. 2. ROC curve analysis for complement fraction C3, S100A8/9, SAA, C3*S100A8/9 product in patients with CRP ≤5 mg/L, baseline (PsA vs HC). PsA is considered a positive classifier.
A. ROC curve for complement fraction C3. AUC [95%CI] 0.734 [0.63-0.839]. Sensitivity 71%, specificity 60%, at cut-off 0.99 mg/L.
B. ROC curve for S100A8/9. AUC [95%CI] 0.718 [0.616-0.820]. Sensitivity 74%, specificity 57%, at cut-off of 1206microg/L.
C. ROC curve for C3*S100A8/9. AUC[95%CI] 0.779[0.683-0.875]. Sensitivity 76%, specificity 72%, at cut-off value of 1291 units.

P66

PROTEOMIC AND GENOMIC PROFILING OF PLASMA EXOSOMES FROM PATIENTS WITH ANKYLOSING SPONDYLITIS

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Introduction. Recent advances in understanding the biology of ankylosing spondylitis (AS) using innovative genomic and proteomic approaches offer the opportunity to address current challenges in AS diagnosis and management. Altered expression of genes, microRNAs (miRNAs) or proteins may contribute to immune dysregulation and may play a significant role in the onset and persistence of inflammation in AS. The ability of exosomes to transport miRNAs across cells and alter the phenotype of recipient cells has implicated exosomes in perpetuating inflammation in AS. This study reports the first proteomic and miRNA profiling of plasma-derived exosomes in AS using comprehensive computational biology analysis.
Methods. Plasma samples from patients with AS and healthy controls (HC) were isolated via ultracentrifugation and subjected to extracellular vesicle flow cytometry analysis to characterise exosome surface markers by a multiplex immunocapture assay. Cytokine profiling of plasma-derived exosomes and cell culture supernatants was performed. Next-generation sequencing was used to identify miRNA populations in exosomes enriched from plasma fractions. CD4+ T cells were sorted, and the frequency and proliferation of CD4+ T-cell subsets were analysed after treatment with AS-exosomes using flow cytometry.
Results. The expression of exosome marker proteins CD63 and CD81 was elevated in the patients with AS compared with HC (q<0.05). Cytokine profiling in plasma-derived AS-exosomes demonstrated downregulation of interleukin (IL)-8 and IL-10 (q<0.05). AS-exosomes cocultured with HC CD4+ T cells induced significant upregulation of IFNα2 and IL-33 (q<0.05). Exosomes from patients with AS inhibited the proliferation of regulatory T cells (Treg), suggesting a mechanism for chronically activated T cells in this disease. Culture of CD4+ T cells from healthy individuals in the presence of AS-exosomes reduced the proliferation of FOXP3+ Treg cells and decreased the frequency of FOXP3+IRF4+ Treg cells. miRNA sequencing identified 24 differentially expressed miRNAs found in circulating exosomes of patients with AS compared with HC; 22 of which were upregulated and 2 were downregulated.
Conclusion. Individuals with AS have different immunological and genetic profiles, as determined by evaluating the exosomes of these patients. The inhibitory effect of exosomes on Treg in AS suggests a mechanism contributing to chronically activated T cells in this disease.

P67

ASSOCIATION OF SEVERE SPINAL INVOLVEMENT WITH HLA ALLELES IN PATIENTS WITH RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS WITH A SYMPTOM DURATION OF MORE THAN 10 YEARS

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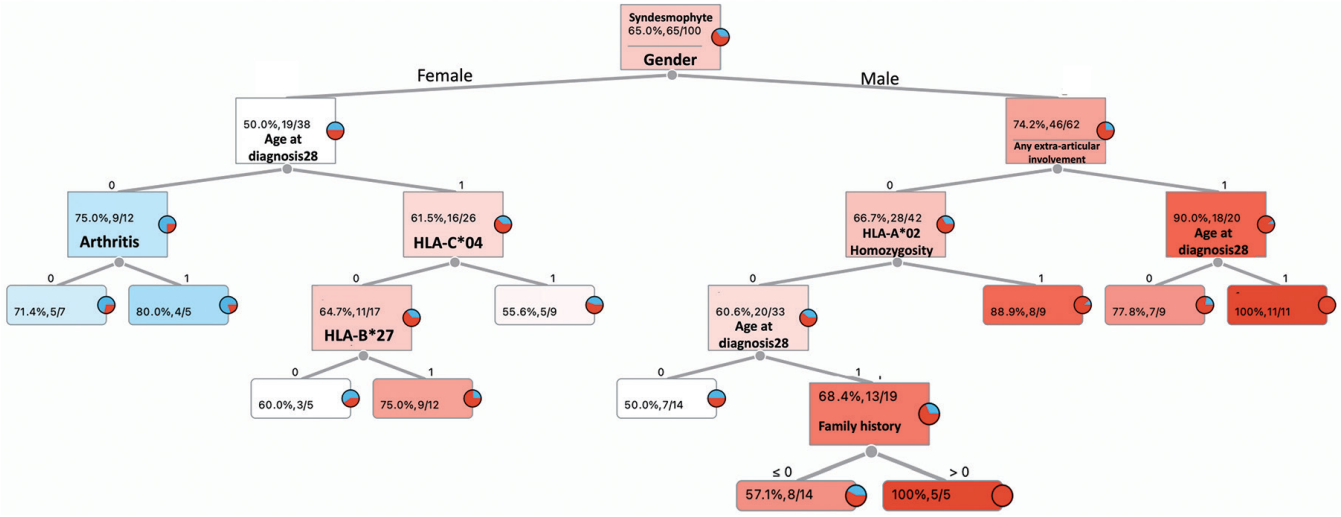
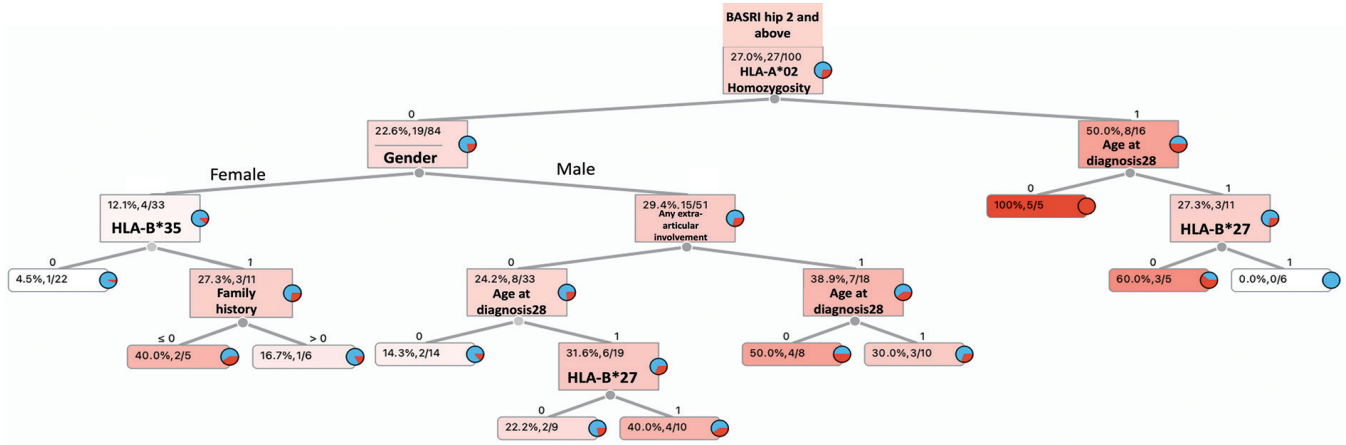
Introduction/Objective. There are limited studies showing the association between HLA alleles and clinical severity in patients with axial spondyloarthritis. In this study, we aimed to determine the HLA alleles that may be associated with disease severity and severe spinal involvement in radiographic AxSpA (r-AxSpA) patients.
Material and methods. Inclusion criteria for the study were: patients with a symptom duration of more than 10 years, patients who met the modified New York criteria according to the consensus of two experienced rheumatologists. Bath Ankylosing Spondylitis Radiology index (BASRI), were calculated by two rheumotologist. If there was a discrepancy in the final score between the two readers, a paired reading was conducted by both readers to achieve consensus. Patients were divided into two groups according to age at diagnosis by ROC analysis, ≤28 years and ≥29 years. HLA genotyping was performed using the Sequence Specific Oligonucleotide Probing method for HLA type A-B-C-DQB1-DRB1. Logistic regression analysis and decision tree models were used to assess the association between radiographic parameters and demographic, genetic characteristics.



P67: Fig. 1. HLA-A*02, HLA-B*27, HLA-B*35 and HLA-C*04 alleles were most frequent in study population and allele frequencies are corrected by Benjamini Hochberg correction. Male gender (p.0.015), age at diagnosis (p:0.045), any extra articular involvement (p:0,026) were independent factors associated with syndesmophyte in logistic regression. Sex, age at diagnosis, any extra-articular involvement, arthritis, family history, HLA-A*02 homozygosity, HLA-B*27, HLA-B*35 and HLA-C*04 were used in the decision tree analysis for syndesmophyte. (Area under curve (AUC): 0.58, F1 classification rate: 0,54) 0: Negative, 1: Positive, Age at diagnosis28 0: Age at diagnosis 28 and under, Age at diagnosis28 1: Age at diagnosis and above.



P67: Fig. 2. Patients with a BASRI-hip score ≥2 were deemed as having radiographic hip involvement. HLA-A*02, HLA-B*27, HLA-B*35 and HLA-C*04 alleles were most frequent in study population and allele frequencies are corrected by Benjamini Hochberg correction. HLA-A*02 homozygosity (p:0.038) was associated with hip involvement in logistic regression. Sex, age at diagnosis, any extra-articular involvement, arthritis, family history, HLA-A*02 homozygosity, HLA-B*27, HLA-B*35 and HLA-C*04 were used in the decision tree analysis for hip involvement. (Area under curve (AUC): 0.59, F1 classification rate: 0,62) 0: Negative, 1: Positive, Age at diagnosis28 0: Age at diagnosis 28 and under, Age at diagnosis28 1: Age at diagnosis and above 0: BASRI- hip score 0-1, 1: BASRI- hip score 2 and above.

Results. Overall, 100 patients were included. 72 (72%) of (r-AxSpA) patients were HLA-B*27 positive. 62 (62%) of the patients were male, the mean (SD) duration of symptoms was 21.1 (9) years. Syndesmophytes were found in 88.9% of HLA-A*02 homozygous male patients without extra-articular involvement. When the decision tree was evaluated, male sex, any extra- articular involvement and HLA-A*02 homozygosity were associated with syndesmophyte (Fig. 1). Hip involvement appears to be associated with HLA-A*02 homozygosity and early age at diagnosis in decision tree (Fig. 2).
Conclusion. This study included patients with symptom duration of more than 10 years, allowing the radiographic severity of axial involvement and associated factors to be examined relatively independently of disease duration. Further studies are needed to support the frequency of HLA-A*02 homozygosity and its association with radiographic severity in r-AxSpA patients.

P68

THE IMPACT OF IXEKIZUMAB TREATMENT ON MRI SACROILIAC JOINT STRUCTURAL LESIONS IN PATIENTS WITH RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS: 52-WEEK RESULTS FROM A RANDOMISED PLACEBO-CONTROLLED TRIAL WITH AN ACTIVE COMPARATOR

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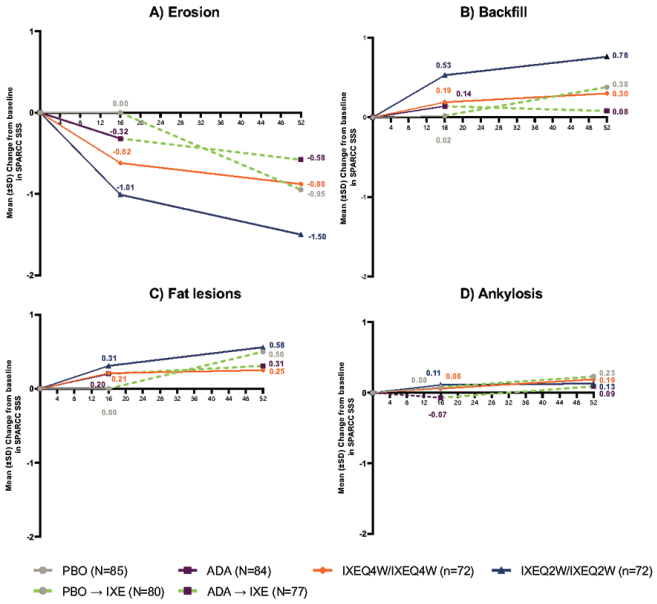
Introduction. The impact of biological disease-modifying anti-rheumatic drugs on magnetic resonance imaging (MRI) structural lesions over 52 weeks in radiographic axial spondyloarthritis (r-axSpA) is unknown. This analysis evaluated the effect of ixekizumab and adalimumab, versus placebo, on structural lesions in the sacroiliac joints assessed by MRI in bionaïve patients with r-axSpA.
Methods. COAST-V was a 52-week, multicentre, randomised, double-blind, placebo- and active-controlled study. At week (W) 0 patients were randomly assigned 1:1:1:1 to placebo, 80mg ixekizumab every 2 (Q2W) or 4 (Q4W) weeks, or 40mg adalimumab Q2W; at W16 patients on placebo or adalimumab were re-randomised to ixekizumab. Post-hoc analyses of patients with MRI at baseline, W16, and W52 are reported. T1 MRI images were scored using the Spondyloarthritis Research Consortium of Canada sacroiliac joints scores for erosion, backfill, fat lesions, and ankylosis. Analysis of covariance was utilised for treatment comparisons in observed cases, adjusting for baseline values, bone marrow oedema (BME), and stratification factors. Subgroup analyses by sex, HLA-B27, and baseline BME were done.

Results. At W16, in ixekizumab versus placebo, a significant decrease in erosion was observed, with the greatest decrease in IXEQ2W (-1.01±2.1); an increase in backfill was evident, with significance observed in IXEQ2W (0·53±1.7) (Fig. 1). At W16, statistically significant differences in mean erosion between ixekizumab-treated patients versus placebo were observed divided by sex, HLA-B27, and baseline BME ≥4/<4 status (Table I). At W52, in both ixekizumab doses, further changes were observed in erosion and backfill: greatest in IXEQ2W (decrease in erosion: -1.50±2.70; increase in backfill: 0.76±2.09) (Fig. 1). A decrease in erosion and increase in backfill was noted in patients switching from adalimumab to ixekizumab.
Conclusion. A significant decrease in erosion and increase in backfill were observed at W16 and W52 in ixekizumab-treated patients. Our results demonstrate that ixekizumab modifies structural damage and is consistent with rapid tissue repair in patients with r-axSpA.
Acknowledgement. Funded by Eli Lilly and Company.



P68: Fig. 1. SPARCC SSS CFB to week 16 & week 52.
Data is observed. Data are mean. SPARCC SSS mean CFB from baseline to week 16, and week 16 to week 52 in (A) erosion, (B) backfill, (C) fat lesions, and (D) ankylosis. ADA: adalimumab; CFB: change from baseline; IXE: ixekizumab; N: number of patients with an MRI scan available at baseline and week 16; PBO: placebo; Q2W: once every 2 weeks; Q4W: once every 4 weeks; SD: standard deviation; SPARCC: Spondyloarthritis Research Consortium of Canada; SSS: sacroiliac joints structural score.

P68: Table I. SPARCC SSS change from baseline in erosion and backfill at week 16 by sex, HLA-B27, and SPARCC BME ≥4/<4 status.

Parameters	PBO (N=85)		ADA (n=84)		IXEQ4W (n=78)		IXEQ2W (n=78)	
Sex	Male	Female	Male	Female	Male	Female	Male	Female
Erosion (*p*-value for interaction of treatment by sex = 0.206)								
Number of patients*	70	15	69	15	65	13	60	18
Baseline mean	3.46 (5.27)	7.07 (7.11)	1.71 (3.65)	4.48 (6.75)	3.52 (5.13)	4.96 (4.27)	2.80 (4.29)	8.31 (5.79)
LS mean CFB (±SE)	0.188 (0.19)	-0.270 (0.42)	-0.628 (0.20)	-0.287 (0.41)	-0.731 (0.20)	0.231 (0.45)	-0.951 (0.21)	-0.772 (0.39)
p-value *vs*. PBO	n/a	n/a	**0.003**	0.976	**<0.001**	0.410	**<0.001**	0.371
Backfill (*p*-value for interaction of treatment by sex = 0.560)								
Number of patients*	70	15	69	15	65	13	60	18
Baseline mean	1.29 (2.34)	0.27 (0.68)	1.15 (2.54)	0.17 (0.36)	1.36 (2.95)	0.73 (1.79)	1.35 (2.56)	2.14 (3.35)
LS mean CFB (±SE)	-0.087 (0.13)	0.520 (0.28)	0.103 (0.13)	0.322 (0.28)	0.197 (0.14)	0.164 (0.30)	0.439 (0.14)	0.786 (0.26)
p-value *vs*. PBO	n/a	n/a	0.307	0.621	0.133	0.392	**0.007**	0.49
HLA-B27								
	Positive	Negative	Positive	Negative	Positive	Negative	Positive	Negative
Erosion (*p*-value for interaction of treatment by HLA-B27 status = 0.116)								
Number of patients*	75	9	77 7	72	6	72	6	
Baseline mean	4.21 (5.70)	3.61 (6.71)	2.10 (4.04)	3.36 (8.02)	4.01 (5.11)	0.67 (1.40)	3.83 (4.88)	6.92 (8.18)
LS mean CFB (±SE)	0.122 (0.19)	0.015 (0.53)	-0.550 (0.18)	-0.674 (0.60)	-0.607 (0.19)	-0.183 (0.66)	-0.779 (0.19)	-2.556 (0.66)
p-value *vs*. PBO	n/a	n/a	0.011	0.394	**0.006**	0.815	**<0.001**	**0.003**
Backfill (*p*-value for interaction of treatment by HLA-B27 status = <0.001)								
Number of patients*	75	9	77	7	72	6	72	6
Baseline mean	1.10 (2.15)	1.33 (2.59)	0.94 (2.24)	1.29 (3.40)	1.34 (2.88)	0.33 (0.82)	1.62 (2.85)	0.50 (1.00)
LS mean CFB (±SE)	0.023 (0.12)	-0.003 (0.35)	0.130 (0.12)	0.284 (0.40)	0.205 (0.13)	0.021 (0.43)	0.337 (0.13)	2.684 (0.43)
p-value *vs*. PBO	n/a	n/a	0.534	0.592	0.298	0.965	0.074	**<0.001**
BME status								
	≥4	<4	≥4 <4		≥4	<4	≥4	<4
Erosion (*p*-value for interaction of treatment by BME ≥/< 4 status = <0.001)								
Number of patients*	25	56	17	62	20	58	22	52
Baseline mean	7.92 (6.37)	2.31 (4.34)	5.16 (6.23)	1.57 (3.67)	7.60 (6.36)	2.43 (3.65)	7.82 (6.23)	2.64 (3.97)
LS mean CFB (±SE)	1.308 (0.32)	-0.436 (0.21)	-0.757 (0.38)	-0.527 (0.20)	-1.208 (0.36)	-0.364 (0.21)	-1.486 (0.34)	-0.688 (0.22)
p-value *vs*. PBO	n/a	n/a	<0.001	0.753	**<0.001**	0.805	**<0.001**	0.405
Backfill (*p*-value for interaction of treatment by BME ≥/< 4 status = 0.226)								
Number of patients*	25	56	17	62	20	58	22	52
Baseline mean	1.52 (2.10)	0.92 (2.21)	2.12 (3.37)	0.64 (1.87)	1.95 (3.34)	1.02 (2.56)	2.34 (2.76)	1.26 (2.82)
LS mean CFB (±SE)	-0.083 (0.22)	0.093 (0.15)	0.520 (0.27)	0.047 (0.14)	0.617 (0.25)	0.045 (0.15)	0.670 (0.24)	0.490 (0.15)
p-value *vs*. PBO	n/a	n/a	0.085	0.823	0.036	0.82	0.021	0.064

*Number of patients in subgroup.
ADA: adalimumab; BME: bone marrow oedema; CFB: change from baseline; IXE: ixekizumab; LS: least squares; MRI: magnetic resonance imaging; N: number of patients with an MRI scan available at baseline and week 16; n/a: not applicable; PBO: placebo; Q2W: every 2 weeks; Q4W: every 4 weeks; SE=: tandard error; SPARCC: Spondyloarthritis Research Consortium of Canada; SSS: sacroiliac joints structural score.

P69

IMPACT OF BIMEKIZUMAB ON MRI INFLAMMATORY AND STRUCTURAL LESIONS IN THE SACROILIAC JOINTS OF PATIENTS WITH AXIAL SPONDYLARTHRITIS: 52-WEEK RESULTS AND POST HOC ANALYSES FROM THE BE MOBILE 1 AND 2 PHASE 3 STUDIES

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Introduction. We report impact of bimekizumab (BKZ; a monoclonal IgG1 antibody that selectively inhibits interleukin [IL]-17F in addition to IL-17A) on MRI inflammatory and structural lesions in sacroiliac joints (SIJ) in patients with non-radiographic/radiographic axial spondylarthritis (nr-/r-axSpA) from the phase 3 BE MOBILE 1 and 2 studies.

Methods. In BE MOBILE 1 (nr-axSpA; NCT03928704) and 2 (r-axSpA; NCT03928743) patients were randomised to subcutaneous BKZ 160 mg every 4 weeks or placebo; all received BKZ from Week 16–52. Spondylarthritis Research Consortium of Canada (SPARCC) SIJ inflammation scores and SPARCC SIJ Structural Score (SSS) were assessed at baseline, Week 16 and Week 52 in MRI sub-studies by two independent experts, with an adjudicator for disagreements. Inflammatory and structural lesions were assessed by different readers. All readers were blinded; structural lesions were analysed post hoc. Observed case data are reported.

Results. 60% (152/254) and 42% (139/332) of patients with nr-/r-axSpA enrolled in MRI sub-studies. 76% (115/152) and 78% (109/139) had valid SPARCC inflammation assessments at all timepoints. Reductions in SPARCC inflammation at Week 16 were maintained to Week 52 for continuous-BKZ patients; patients switching from placebo to BKZ at Week 16 reached similar levels at Week 52 as continuous-BKZ patients (Fig. 1).

84% (128/152) and 83% (116/139) of patients with nr-/r-axSpA had valid SPARCC SSS assessments at all timepoints. Reductions in SPARCC SSS for erosions and increases in backfill and fat were observed with BKZ versus placebo at Week 16, with further improvements to Week 52 in the continuous-BKZ group; similar changes were observed in placebo-switchers (Fig. 2). No SPARCC SSS change for ankylosis was observed in patients with nr-axSpA.

Conclusion. BKZ improved MRI inflammation, reduced erosions and increased backfill and fat in the SIJ of patients with axSpA, potentially suggesting evidence of tissue repair.

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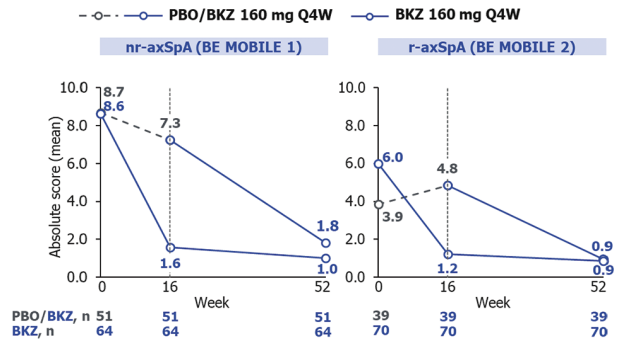

P69: Fig. 1. Mean MRI SPARCC SIJ inflammation scores up to Week 52 (OC).
Randomized set. Includes only patients in the MRI sub-studies with valid SPARCC SIJ assessments at baseline, Week 16 and Week 52. SPARCC SIJ score range from 0-72, with lower scores indicating less inflammation. BKZ: bimekizumab; nr-axSpA: non-radiographic axial spondyloarthritis; OC: observed case; PBO: placebo; Q4W: every 4 weeks; r-axSpA: radiographic spondyloarthritis; SPARCC: Spondyloarthritis Research Consortium of Canada; SIJ: sacroiliac joints.

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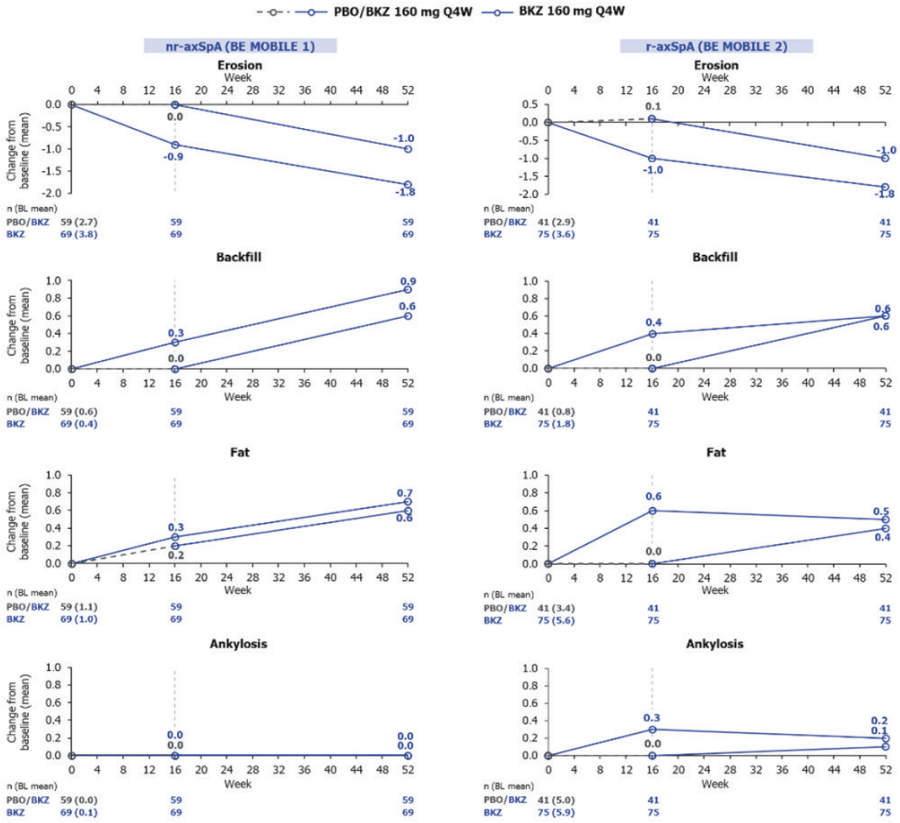

P69: Fig. 2. Change from baseline in structural lesions up to Week 52 (OC).
Randomized set. Includes only patients in the MRI sub-studies with valid SPARCC SSS assessments at baseline, Week 16 and Week 52. BKZ: bimekizumab; BL: baseline; nr-axSpA: non-radiographic axial spondyloarthritis; OC: observed case; PBO: placebo; Q4W: every 4 weeks; r-axSpA: radiographic spondyloarthritis; SIJ: sacroiliac joints; SPARCC: Spondyloarthritis Research Consortium of Canada; SSS: SIJ Structural Score.

P70

MINIMAL SPINAL RADIOGRAPHIC PROGRESSION IN PATIENTS WITH RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS OVER 2 YEARS OF BIMEKIZUMAB TREATMENT: RESULTS FROM A PHASE 3 OPEN-LABEL EXTENSION STUDY

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Introduction. We evaluate impact of bimekizumab (BKZ; a monoclonal IgG1 antibody that selectively inhibits interleukin [IL]-17F in addition to IL-17A) on 2-year spinal radiographic progression of patients with radiographic axial spondyloarthritis (r-axSpA) in the open-label extension (OLE) of the phase 3 BE MOBILE 2 study.
Methods. At Week 52 of BE MOBILE 2 (NCT03928743; study design previously reported), patients could enter an ongoing OLE (NCT04436640) and receive BKZ. Baseline and Week 104 spinal radiographs were assessed using modified Stoke Ankylosing Spondylitis Spinal Score (mSASSS), by two central readers, with an adjudicator if scores differed by ≥5 mSASSS points; all were blinded to timepoint. The average score change across readers was determined for each radiograph; if three readers were used, average of the two closest scores was calculated. We report mean and cumulative probability of change from baseline (CfB) in mSASSS at Week 104, and the proportion of non progressors. Potential predictive factors for spinal radiographic progression (mSASSS CfB ≥2) were assessed using logistic regression models.
Results. Of 332 randomised patients with r-axSpA, 286 (86.1%) entered the OLE and 267 (80.4%) completed Week 104. At Week 104, 190/267 (71.2%) patients had an mSASSS available (mean [SD] baseline mSASSS score: 7.3 [13.8]; CfB at Week 104: 0.3 [1.9]).

Most (157/190) patients had no spinal radiographic progression at Week 104 with BKZ (Fig. 1). Proportion of non-progressors at Week 104 (mSASSS CfB ≤0.5) was 85.3% (162/190); when defined as mSASSS CfB <2, 92.1% (175/190) were non-progressors, including 83.1% (69/83) of patients with existing structural damage at baseline (mSASSS ≥2). No potential predictive factors were associated with significantly increased likelihood of spinal radiographic progression.

Conclusion. After 2 years of treatment with BKZ, patients with r-axSpA showed minimal spinal radiographic progression and a high proportion were non-progressors, including those with baseline spinal damage.

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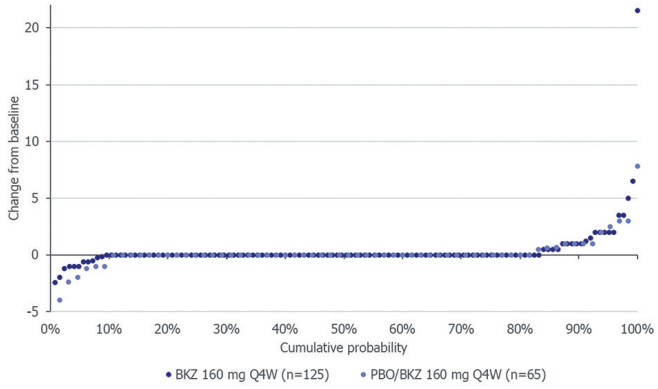
Disclosures. XB: Speakers bureau from AbbVie, BMS, Chugai, Eli Lilly, Galapagos, MSD, Novartis, Pfizer, UCB Pharma; paid instructor for AbbVie, BMS, Chugai, Eli Lilly, Galapagos, MSD, Novartis, Pfizer, UCB Pharma; consultant of AbbVie, BMS, Chugai, Eli Lilly, Galapagos, Gilead, Novartis, Pfizer, UCB Pharma; grant/research support from Novartis, UCB Pharma; **SR:** Grants from AbbVie, Galapagos, MSD, Novartis, Pfizer, UCB Pharma; consultancy from AbbVie, Eli Lilly, Novartis, Pfizer, Sanofi and UCB Pharma; **WPM:** Honoraria/consulting fees from AbbVie, BMS, Boehringer-Ingelheim, Celgene, Eli Lilly, Galapagos, Janssen, Novartis, Pfizer, and UCB Pharma; research grants from AbbVie, Galapagos, Pfizer, and UCB Pharma; educational grants from AbbVie, Janssen, Novartis, and Pfizer; Chief Medical Officer for CARE ARTHRITIS; **MO:** Research grants from Abbott, Centocor, Pfizer; consulting fees from Abbott, Pfizer, Merck, Roche, UCB Pharma; speakers bureau for Abbott, BMS, Merck, Mundipharma, Pfizer, UCB Pharma; **UM, AM, NdP:** Employees of UCB Pharma; **CF, TV:** Employees and shareholders of UCB Pharma; **CP:** Contractor for UCB Pharma and employee of Veramed LTD; **DP:** Speaker for AbbVie, BMS, Eli Lilly, MSD, Novartis, Pfizer, and UCB Pharma; Consultant for AbbVie, Biocad, Eli Lilly, Gilead, GSK, MSD, MoonLake, Novartis, Pfizer, Samsung Bioepis, and UCB Pharma; Grant/research support from AbbVie, Eli Lilly, MSD, Novartis, and Pfizer.



P70: Fig. 1. Cumulative probability of mSASSS CfB at Week 104 (oC).
Randomized set. Includes patients in the X-ray sub-study with valid X-ray assessments at baseline and Week 104 (N=190). Patients were randomized to receive subcutaneous BKZ 160 mg Q4W or placebo to Week 16; all patients received subcutaneous BKZ 160 mg Q4W from Week 16. mSASSS scores range from 0-72, with lower scores indicating less structural damage. BKZ: bimekizumab; CfB: change from baseline; mSASSS: modified Stoke Ankylosing Spondylitis Spinal Score; OC: observed case; PBO: placebo; Q4W: every 4 weeks.

P72

FACTORS ASSOCIATED WITH RADIOGRAPHIC SPINAL PROGRESSION IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS RECEIVING IL-17A INHIBITOR OR TNF INHIBITOR THERAPY: A POST-HOC ANALYSIS OF THE SURPASS STUDY

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Objective. The aim of this post-hoc analysis was to evaluate the relationship between baseline factors and radiographic spinal progression over 2 years in the SURPASS study (NCT03259074) in patients with radiographic axial spondyloarthritis (r-axSpA) receiving secukinumab (SEC, an IL-17A inhibitor) or adalimumab biosimilar (SDZ-ADL, a TNF inhibitor).

Materials and methods. SURPASS, a phase IIIb randomised controlled study included biologic-naïve patients with active r-axSpA at high risk for radiographic spinal progression. Patients were randomised (1:1:1) to receive SEC (150 or 300 mg; dose-blinded) or SDZ-ADL (40 mg; open label) for 2 years. All patients with data available for radiographs and magnetic resonance imaging (MRI) of spine at baseline and week 104 were included in the analysis. Radiographic spinal progression was assessed by the modified Stoke Ankylosing Spondylitis Spinal Score (mSASSS), with final scores based on average of 3. Univariable and multivariable logistic regression analysis was used to assess baseline factors associated with the formation of new syndesmophytes (dependent variable).

Results. A total of 288 patients (SEC 150 mg: n=99; SEC 300 mg: n=95; SDZ-ADL 40 mg: n=94) with complete sets of radiographs and MRIs were included. Demographic and baseline disease characteristics are presented in Table I. At week 104, new syndesmophytes were observed in 18.2%, 15.8%, and 14.9% of the patients receiving SEC 150 mg, SEC 300 mg, and SDZ-ADL, respectively. In both univariable and multivariable analysis, the presence of syndesmophytes and higher level of inflammatory activity as reflected by elevated CRP, higher Axial Spondyloarthritis Disease Activity Score, and higher spinal MRI bone marrow oedema score at baseline were associated with the development of new syndesmophytes (Table II).

Conclusion. Pre-existing structural damage and high inflammatory activity at baseline were associated with new syndesmophyte development in r-axSpA patients irrespective of treatment with IL-17 or TNF inhibitors over 2 years.

Funding: Novartis.

P72: Table I. Demographic and baseline disease characteristics based on radiographic analysis subset.

Characteristics, mean (SD) unless specified otherwise	SEC 150 mg n=99	SEC 300 mg n=95	SDZ-ADL 40 mg n=94	Total N=288
Age, years	40.2 (10.7)	40.4 (10.6)	41.3 (12.1)	40.6 (11.1)
Male, n (%)	79 (79.8)	66 (69.5)	75 (79.8)	220 (76.4)
HLA-B27 positive, n (%)	87 (87.9)	80 (84.2)	83 (88.3)	250 (86.8)
BMI, kg/m^2	27.3 (5.4)	26.7 (5.9)	26.6 (5.1)	26.9 (5.5)
Time since diagnosis of r-axSpA, years	6.2 (8.1)	5.5 (6.3)	7.5 (11.3)	6.4 (8.8)
mSASSS (0–72)	16.3 (20.3)	12.2 (17.5)	14.9 (19.0)	14.5 (19.0)
Patients with syndesmophyte(s), n (%)	58 (58.6)	55 (57.9)	55 (58.5)	168 (58.3)
Number of syndesmophytes	5.3 (7.1)	4.2 (6.1)	5.0 (6.6)	4.8 (6.6)
Patients with hsCRP ≥5 mg/L, n (%)	79 (79.8)	71 (74.7)	72 (76.6)	222 (77.1)
hsCRP (mg/L)	21.4 (27.6)	21.2 (28.0)	19.0 (19.8)	20.6 (25.4)
Total spine oedema score (range 0–184)*	7.9 (10.2)	5.1 (7.6)	6.3 (8.0)	6.5 (8.7)
Total spine fat lesions score (range 0–276)	12.0 (18.8)	10.0 (16.9)	14.0 (19.5)	12.0 (18.4)

*Bone marrow oedema scored per vertebral unit quadrant (0–2 score per 92 quadrants). A patient was considered to have a syndesmophyte if at least 2 of the 3 readers assessed mSASSS score of ≥2 for any individual vertebral corner. The proportion of patients that developed new syndesmophytes (according to majority agreement: 2 of the 3 readers) was calculated. Spinal inflammation and fat lesions as assessed by MRI were evaluated by three blinded readers.

BMI, body mass index; HLA-B27, Human leukocyte antigen B27; hsCRP, high sensitivity C-reactive protein; MRI, magnetic resonance imaging; mSASSS, modified Stoke Ankylosing Spondylitis Spinal Score; r-axSpA, radiographic axial spondyloarthritis; SD, standard deviation; SDZ-ADL, adalimumab biosimilar; SEC, secukinumab

P72: Table II. Univariable and multivariable logistic regression analysis for development of new syndesmophytes over 2 years.

Parameter at baseline (unless otherwise specified)	Univariable Analysis, OR (95% CI)*				Multivariable Analysis, OR (95% CI)
	SEC 150 mg n=99	SEC 300 mg n=95	SDZ-ADL 40 mg n=94	Total N=288	Total N=288
Age, years	1.01 (0.96, 1.06)	1.04 (0.99, 1.09)	0.98 (0.93, 1.03)	1.01 (0.98, 1.04)	-
Sex (male vs female)	3.64 (0.61, 21.66)	1.74 (0.48, 6.32)	1.38 (0.31, 6.12)	2.22 (0.92, 5.37)	1.19 (0.44, 3.24)
HLA-B27 (positive vs negative)	0.40 (0.09, 1.74)	1.00 (0.22, 4.59)	4.38 (0.21, 90.57)	1.06 (0.40, 2.83)	-
Current smoking (yes vs no)	1.45 (0.51, 4.11)	1.45 (0.45, 4.63)	2.41 (0.76, 7.68)	1.69 (0.88, 3.24)	0.97 (0.45, 2.06)
Presence of syndesmophytes (yes vs no)	6.13 (1.49, 25.25)	9.20 (1.59, 53.36)	27.61 (1.54, 496.20)	**12.00 (3.91, 36.82)**	**13.00 (3.79, 44.57)**
Elevated hsCRP, ≥5 mg/L (yes vs no)	1.92 (0.45, 8.27)	3.95 (0.67, 23.47)	11.15 (0.60, 207.47)	**4.52 (1.46, 14.05)**	**4.70 (1.33, 16.66)**
ASDAS-CRP	2.08 (1.00, 4.33)	1.30 (0.73, 2.31)	2.25 (1.05, 4.84)	**1.73 (1.18, 2.55)**	-
BASDAI	1.58 (1.04, 2.40)	0.91 (0.62, 1.33)	1.19 (0.79, 1.81)	1.19 (0.95, 1.50)	-
Baseline NSAID score	1.01 (1.00, 1.02)	1.00 (0.99, 1.01)	0.98 (0.97, 1.00)	1.00 (0.99, 1.01)	1.00 (0.99, 1.01)
NSAIDs intake score over 104 weeks	0.99 (0.94, 1.03)	1.00 (0.96, 1.05)	1.00 (0.96, 1.04)	1.00 (0.97, 1.02)	-
Total spine oedema score, (range 0–184)†	1.05 (1.01, 1.10)	1.09 (1.02, 1.16)	1.08 (1.02, 1.15)	**1.07 (1.04, 1.11)**	**1.05 (1.01, 1.09)**
Total spine fat lesions score, (range 0–276)	1.01 (0.99, 1.04)	1.02 (0.99, 1.05)	1.01 (0.98, 1.03)	1.01 (1.00, 1.03)	1.00 (0.98, 1.02)

*Wide confidence intervals are observed by treatment groups due to low frequency of events; odds ratios should be interpreted with caution.†Bone marrow oedema scored per vertebral unit quadrant (0–2 score per 92 quadrants).

Factors associated with new syndesmophyte formation (yes/no) at week 104 were analysed using univariable logistic regression analysis. Multivariable logistic regression analysis included variables informed by both univariable analyses and clinical expertise.

ASDAS; Axial Spondyloarthritis Disease Activity Score; BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; CI, confidence interval; HLA-B27, Human leukocyte antigen B27; hsCRP, high sensitivity C-reactive protein; mSASSS, modified Stoke Ankylosing Spondylitis Spinal Score; NSAID, non-steroidal anti-inflammatory drug; OR, odds ratio; SDZ-ADL, adalimumab biosimilar; SEC, secukinumab

P73

MRI-BASED SYNTHETIC CT IS SUPERIOR TO CONVENTIONAL MRI FOR THE DETECTION OF STRUCTURAL LESIONS IN POSTPARTUM WOMEN AFTER 5 YEAR FOLLOW-UP

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Introduction. A novel imaging technique has been introduced to detect structural lesions in sacroiliac joints, addressing challenges associated with the evaluation on routine T1-weighted MRI scans. MRI-based synthetic CT (sCT) allows detection of structural lesions like erosions with CT-like accuracy, all while eliminating the need for CT-related radiation exposure. This study compares structural lesions on MRI to sCT in postpartum women, 5 years after given birth.

Methods. sCT were reconstructed with commercially available software (BoneMRI Pelvic Region, version 1.5; MRIguidance) from 3D T1-GRE images using a deep learning method based on the U-net architecture (1). MRI scan protocol contained Short Tau Inversion Recovery and T1-weighted sequences. Both MRI and sCT were available in 18 women. sCT were assessed by 2 readers and MRI by 3 readers. Results are based on the consensus of ≥2 readers. sCT and MRI were assessed separately for erosions, fatty lesions and ankylosis per sacroiliac joint quadrant (maximum score of 144 quadrants per structural lesions). Descriptive data were presented on quadrant level.

Results. MRI and sCT were available in 18 women; mean age 35.4 years (±2.6), all HLA-B27 negative. Mean BMI was 20.7 kg/m^2 (±1.6). Six (33%) women reported back pain. On MRI, 1 quadrant with erosions was reported, compared to 5 quadrants with erosions on sCT. However, none were seen on both modalities. Three quadrants with sclerosis were reported on MRI and sCT, compared to 26 quadrants with sclerosis on sCT only. No ankylosis was reported.

Conclusion. Few structural lesions were seen in patients 5 years postpartum both on MRI and on sCT/bone MRI. Nevertheless, when comparing MRI to sCT, sclerosis and -to a lesser extent- erosions were more frequently accounted for in the latter. sCT combines sensitivity for detection of structural lesions of CT, with the established benefits of MRI, displaying unprecedented opportunities for use in clinical practice.

References

1. JANS *et al*. *Radiology* 2021; 298(2): 343-49.
2. RONNEBERGER O *et al*. MICCAI 2015. *Lecture Notes in Computer Science* 2015(9351); 234-41.

P73: Table I. Structural lesions presented on quadrant level in postpartum women 5 years after giving birth.

Sclerosis	sCT positive	sCT negative
MRI positive	3	0
MRI negative	23	118
Erosion	sCT positive	sCT negative
MRI positive	0	1
MRI negative	5	136
Ankylosing	sCT positive	sCT negative
MRI positive	0	0
MRI negative	0	144

P74

NATURAL COURSE OF DEGENERATIVE LESIONS OF THE SPINE ON RADIOGRAPHS AND MRI IN AXIAL SPONDYLOARTHRITIS FOLLOWED 10 YEARS IN THE DESIR COHORT

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Introduction. Radiographs and magnetic resonance imaging (MRI) are imaging techniques used to assess structural lesions in axial spondyloarthritis (axSpA). While spinal degenerative lesions (DLs) are prevalent among these patients, their natural course remains not well-known. Our aim was to investigate the DLs and their changes over 10 years (10Y) in axSpA patients.
Methods. Whole spine MRI and cervical/lumbar spine radiographs at baseline/5Y/10Y of axSpA patients in the DESIR cohort were assessed for DLs by three readers. Only patients with baseline and 10Y data were included. Patient characteristics and DLs (agreement between ≥2/3 readers or an average of 3 readers) at the patient and the vertebral unit levels were reported. Net progression was calculated by subtracting the patients that "improved" from those that "worsened" divided by the total number of patients.
Results. Imaging was available for 330 patients (mean age 34.5[8.6] years, 47% male). The most frequent DL on radiographs were disc height loss (baseline:45%, 10Y:65%, net progression:+20%), osteophytes (baseline:21%, 10Y:44%, net progression:+22%) and facet joint osteoarthritis (baseline:11%, 10Y:24%, net progression:+13%). An average of 1.6(2.5) DLs per patient were observed on radiographs at baseline; this number increased to 3.4(3.9) at 10Y (p-value <0.0001). The most frequent DLs on MRI were disc degeneration (Pfirrmann classification>2: baseline:86%, 10Y:95%, net progression:+9%), followed by high-intensity zone (baseline:50%, 10Y:59%, net progression:+8%), Schmorl's node without edema (baseline:47%, 10Y:50%, net progression:+3%) and disc protrusion (baseline:45%, 10Y:52%, net progression:+7%). An average of 7.4(5.4) DLs per patient were observed on MRI at baseline; this number increased to 11.1(7.1) at 10Y (p-value<10^{-4}). The distribution of these spinal lesions is shown in the heat maps (Figs. 1-2).
Conclusion. The prevalence of spinal DLs is high in an inception cohort of axSpA with the total number of DLs increasing over 10Y in the cervical and lumbar spine and extending to the thoracic spine.

P75

FACTORS ASSOCIATED WITH THE EVOLUTION OF DEGENERATIVE SPINAL LESIONS IN AXIAL SPONDYLOARTHRITIS: 10-YEAR FOLLOW-UP OF THE DESIR COHORT

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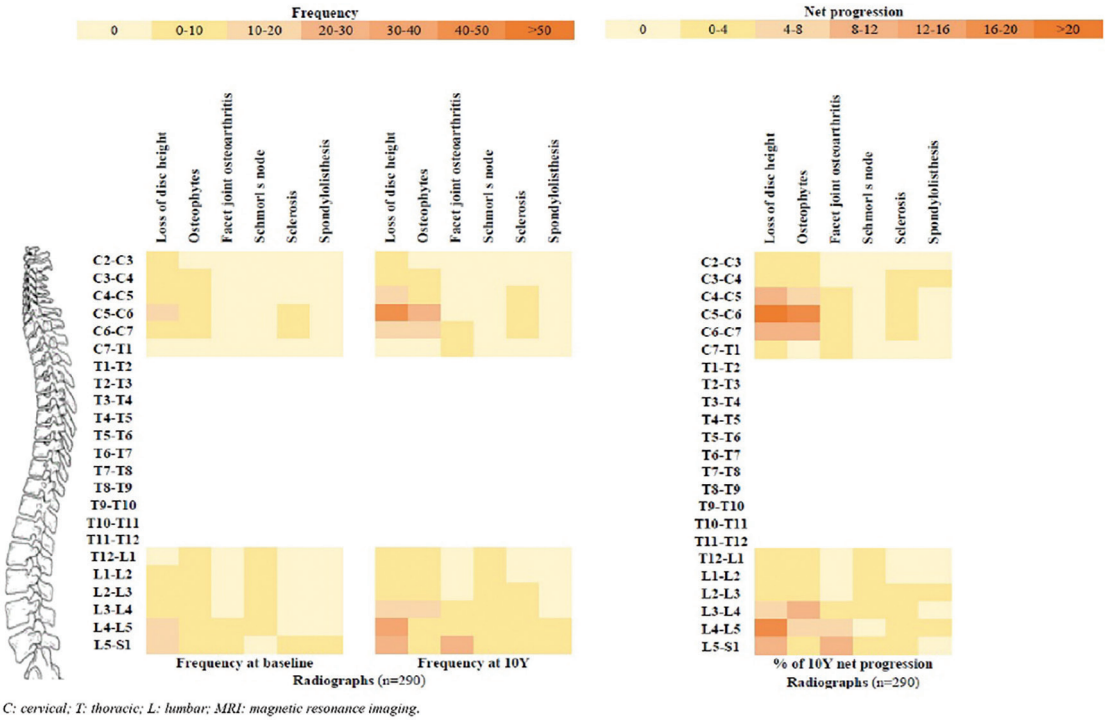
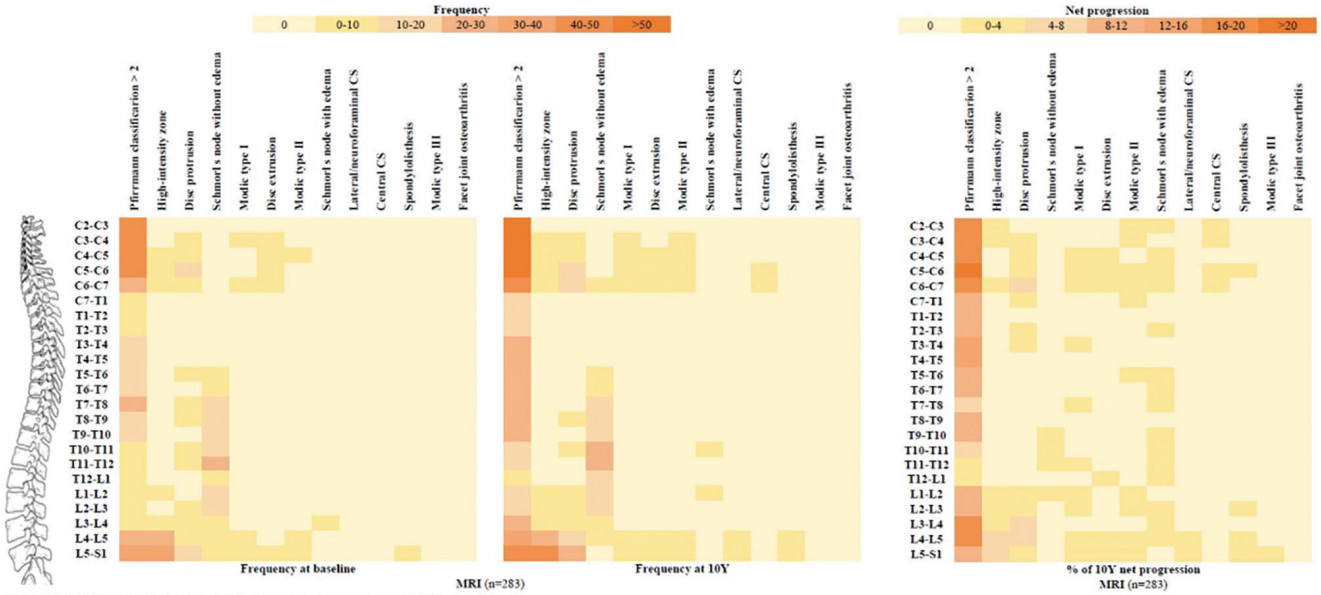
Introduction. Radiographs and MRI are both used to diagnose axial spondyloarthritis (axSpA), but they also provide other information about the spinal components. Our aim was to investigate the evolution of spinal degenerative lesions (DLs) in axSpA patients over 10 years (10Y) and the factors associated with progression.
Methods. Whole spine MRI and cervical/lumbar spine radiographs at baseline/5Y/10Y of axSpA patients in the DESIR cohort were assessed for DLs by 3 readers. DLs were defined at the patient level and, for descriptive purposes, in consensus across readers (≥2/3 readers or average). To assess progression of DLs over time, we used multilevel (individual reader data) generalised estimating equation models adjusted for sex, HLA-B27 status, body mass index (BMI), smoking and job type at baseline and biologics exposure during the 10Y period. The main variable of interest was time to reflect the annual progression of each lesion. Annual change percentage or β-coefficients and 95% confidence intervals (95%CI) were reported.
Results. DLs were available at ≥1 time point for 330 patients (34.5[8.6] years; 47% men). At baseline, there was an average of 1.6(2.5) DLs on radiographs



P74: Fig. 1. Heat maps of degenarative lesion frequencies at baseline and 10 years and net progression over 10 years at the vertebra unit level on radiographs.



P74: Fig. 2. Heat maps of degenarative lesion frequencies at baseline and 10 years and net progression over 10 years at the vertebral unit level on radiographs.

and 7.5(5.5) on MRI. After 10Y, they increased to 3.4(3.9) and 11.0(7.1), respectively. A significant progression was mainly detected on radiographs for osteophytes (annual change=2.24%,95%CI:1.92-2.75), disc height loss (1.37%,0.95-1.80), and facet joint osteoarthritis (1.30%,0.90-1.69). The same trend was detected on MRI for disc protrusion (1.11%,0.65-1.56), Modic type I (1.01%,0.69-1.33) and II (0.94%,0.66-1.22) (Table I). We observed a significant increase in the DLs total number on radiographs (β=1.81,1.48-2.14) and MRI (4.17,3.49-4.84). Factors associated with DLs progression were increasing BMI (radiographs:0.15,0.12-0.19;MRI:0.35,0.27-0.44) and biologics exposure (radiographs:0.39,0.13-0.66;MRI:0.95,0.34-1.55) (Table II).

Conclusion. In an inception cohort of axSpA, spinal DLs, though common, progress slowly over 10Y. However, this progression seems to be faster in patients with a higher BMI and those exposed to biologics.

P75: Table I. Annual progression of degenerative lesions on radiographs and MRI.

	% annual change (95%CI)
Progression on radiographs	n = 329
Loss of disc height	**1.37 (0.95 ; 1.80)**
Osteophytes	**2.34 (1.92 ; 2.75)**
Facet joint osteoarthritis	**1.30 (0.90 ; 1.69)**
Schmorl's node	0.11 (-0.22 ; 0.43)
Sclerosis	**0.75 (0.51 ; 0.99)**
Spondylolisthesis	0.12 (-0.04 ; 0.28)
Progression on MRI	n = 327
Pfirrmann classification > 2	**0.89 (0.63 ; 1.15)**
High-intensity zone	**0.90 (0.45 ; 1.35)**
Disc protrusion	**1.11 (0.65 ; 1.56)**
Schmorl's node without edema	0.11 (-0.01 ; 0.42)
Modic type I	**1.01 (0.69 ; 1.33)**
Disc extrusion	**0.68 (0.36 ; 0.99)**
Modic type II	**0.94 (0.66 ; 1.22)**
Schmorl's node with edema	0.23 (-0.26 ; 0.64)
Lateral / neuroforaminal canal stenosis	**0.28 (0.12 ; 0.44)**
Central canal stenosis	**0.15 (0.01 ; 0.30)**
Spondylolisthesis	0.05 (-0.06 ; 0.16)
Modic type III	0.04 (-0.02 ; 0.10)
Scheuermann's disease	0.01 (-0.07 ; 0.09)
Facet joint osteoarthritis	0.22 (-0.45 ; 0.88)

*Percentage change per year estimated used generalized estimating equation (GEE) models, considering individual reader data and exchangeable working correlation structure, adjusted for sex, body mass index, HLA-B27 status, tobacco use, job type and biological disease-modifying antirheumatic drugs exposure, Bold characters indicate significant values. MRI: magnetic resonance imaging; 95%CI: 95% confidence interval.

P75: Table II. Factors associated with annual progression in the number of degenerative lesions on radiographs and MRI.

	Radiographs n = 329	MRI n = 327
	β coefficients* (95%CI)	
Number of degenerative lesions over time (/year)	**0.18 (0.15 ; 0.21)**	**0.42 (0.35 ; 0.48)**
Males (ref: women)	0.01 (-0.26 ; 0.28)	**1.36 (0.74 ; 1.99)**
BMI (kg/m^2)	**0.15 (0.12 ; 0.19)**	**0.35 (0.27 ; 0.44)**
HLA-B27+	**-0.52 (-0.81 ; -0.22)**	-0.23 (-0.89 ; 0.43)
Smoking (ever vs. never)	-0.03 (-0.31 ; 0.26)	-0.01 (-0.64 ; 0.62)
Job type (ref: blue collar)		
white collar	**0.62 (0.21 ; 1.02)**	0.21 (-0.74 ; 1.17)
not employed	-0.40 (-0.93 ; 0.14)	**-2.40 (-3.59 ; -1.20)**
bDMARDs exposure (ever vs. never)	**0.39 (0.13 ; 0.66)**	**0.95 (0.34 ; 1.55)**

*β coefficients associated with annual progression estimated used generalized estimating equation (GEE) models, considering individual reader data and exchangeable working correlation structure. Bold characters indicate significant values. MRI: magnetic resonance imaging; 95%CI: 95% confidence interval; BMI: body mass index; HLA: human leucocyte antigen; bDMARDs biological disease modifying antirheumatic drugs.

P76

RELATIONSHIP BETWEEN SPECIFIC DEGENERATIVE LESIONS OVER TIME IN AXIAL SPONDYLOARTHRITIS: 10-YEAR FOLLOW-UP OF THE DESIR COHORT

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Introduction. Spinal degenerative lesions (DLs) are common in radiographs and MRI in axSpA. However, the relationship between the different DLs over time has not been established. Our aim was to investigate the relationship between specific DLs over 10 years (10Y) in axSpA.

Methods. Whole spine MRI and cervical/lumbar spine radiographs at baseline, 5Y and 10Y of axSpA patients in the DESIR cohort were assessed for DLs by 3 readers. Only patients with ≥2 time points available were included. We used multilevel generalised estimating equation time-lagged and autoregressive models adjusted for age, sex, HLA-B27 status, BMI, smoking and job type at baseline and bDMARDs during follow-up (time-varying variables). Time-varying models test the effect of one DL at one time point on another DL in the subsequent time-point. Autoregressive models adjust for the outcome in the previous time point, similar to modelling a change in the outcome.

Results. DLs were available for 291 patients on MRI and 323 patients on radiographs (34.5 [8.6] years; 47% men). On MRI, significant associations were found between disc degeneration and herniation (ORa=4.03;95%CI=3.47-4.69), high intensity zone (1.79;1.51-2.13) and Modic type 1 lesion (4.72;3.68-6.06) in the subsequent time point at the same disc space level, after controlling for such a lesion at the previous time point (Table I). There was also a significant association between Modic type 1 and subsequent Modic type 2 or 3 lesions (54.00;38.00-76.90). Herniations tended to persist over time (187;156-224). On radiographs, disc height loss was significantly associated with a subsequent osteophyte (4.28;3.59-5.10) (Table II). The presence of a DL on MRI seemed to predict its appearance on subsequent radiographs (2.49;2.19-2.84).

Conclusion. In this first study providing insight into the long-term relationship of spinal DLs in an inception cohort of axSpA, different DLs are associated with the development of other DLs over time.

P76: Table I. Relationship between specific degenerative lesions over time on MRI.

	Outcomes in the subsequent time point n=291				
	ORa* (95%CI)				
	Herniation	**Protrusion**	**Extrusion**	**HIZ**	**Modic type 1 lesion**
Disc degeneration	**4.03 (3.47 - 4.69)**	**4.13 (3.54 - 4.82)**	**7.36 (5.33 - 10.20)**	**1.79 (1.51 - 2.13)**	**4.72 (3.68 - 6.06)**
Pfirrmann class. (ref: grade 1)					
Grade 2	**2.42 (1.83 - 3.21)**	**2.71 (2.00 - 3.67)**	1.74 (0.92 - 3.27)	**2.04 (1.59 - 2.62)**	0.83 (0.55 - 1.25)
Grade 3	**8.43 (6.31 - 11.30)**	**9.42 (6.89 - 12.90)**	**10.50 (5.60 - 19.70)**	**3.11 (2.34 - 4.12)**	**3.27 (2.19 - 4.87)**
Grade 4	**8.39 (5.90 - 11.90)**	**9.13 (6.31 - 13.20)**	**16.10 (8.07 - 32.10)**	**3.54 (2.45 - 5.12)**	**6.72 (4.29 - 10.50)**
Grade 5	**2.58 (1.41 - 4.71)**	**5.89 (3.21 - 10.80)**	**3.17 (1.02 - 9.82)**	**2.66 (1.20 - 5.92)**	**9.77 (5.05 - 18.90)**
	Herniation	**Protrusion**	**Extrusion**	**Disc degeneration**	**Modic type 2-3 lesion**
Modic type 1 lesion	1.50 (0.72 - 3.12)	2.21 (1.16 - 4.23)	1.44 (0.45 - 4.53)	**2.02 (1.22 - 3.36)**	**54.80 (38.00 - 76.90)**
Modic type 2 or 3 lesion	**Modic type 1 7.68 (4.46 - 13.20)**				
Herniation	**Herniation 187.00 (156.00 – 224.00)**				
Protrusion	**Extrusion 25.80 (19.00 – 35.00)**				
Extrusion	**264.0 (184.00 – 379.00)**				
	Disc degeneration				
Schmorl's nodes	**1.25 (1.09 - 1.42)**				
Degenerative lesion at ≥1 level (patient level)	**Degenerative lesions at multiple levels 2.18 (1.10 - 4.72)**		**Degenerative lesion at adjacent levels 1.45 (1.32 - 1.60)**		

*ORa: odd ratio adjusted estimated using multilevel (patient, reader and vertebra, except for the last row which is at the patient level) generalised estimating equations time lagged and autoregressive models (exchangeable working correlation structure), adjusted for age at baseline, sex, HLA-B27 status, body mass index, smoking, job type and biological disease-modifying antirheumatic drugs (time-varying variable).
Disc degeneration is defined as a disc with a Pfirrmann classification >2. Herniation is defined as a protrusion or extrusion of the disc.
Bold characters indicate significant values.
95%CI: 95% confidence interval; Pfirrmann class.: Pfirrmann classification; HIZ: high intensity zone.

P76: Table II. Relationship between specific degenerative lesions over time on radiographs.

	Outcomes in the subsequent time point n = 323 ORa* (95%CI)		
Loss of disc height	**Osteophyte 4.28 (3.59 - 5.10)**		
Schmorl's nodes	**Disc degeneration** 1.38 (0.92 - 2.07)		
Degenerative lesion at ≥ 1 level (patient level)	**Degenerative lesions at multiple levels 3.26 (2.30 - 4.62)**		**Degenerative lesion at adjacent levels 1.93 (1.68 - 2.22)**

*ORa: odd ratio adjusted estimated using multilevel (patient, reader and vertebra, except for the last row which is at the patient level) generalised estimating equation equations time lagged and autoregressive models (exchangeable working correlation structure), adjusted for age at baseline, sex, HLA-B27 status, body mass index, smoking, job type and biological disease-modifying antirheumatic drugs (time-varying variable).
Bold characters indicate significant values. 95%CI: 95% confidence interval.

P77

MRI EVOLUTION OF SUBCHONDRAL SCLEROSIS AT THE SACROILIAC JOINT FROM PREGNANCY TO 12 MONTHS POSTPARTUM

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Introduction/Objective. Osteitis condensans ilii (OCI) was originally described based on its radiographic appearance with triangular-shaped iliac sclerosis, spared sacroiliac joint (SIJ) space and no erosions, but evidence regarding MRI characteristics is sparse. OCI may simulate sacroiliitis on MRI. The etiology of OCI is speculative, but it is often related to pregnancy. The study aims were to evaluate the peripartum evolution of SIJ sclerosis and to explore preceding or concomitant other MRI lesions (BME, fat lesion).
Materials and methods. AMPREG is a prospective cohort study with 103 first-time mothers, who underwent up to 5 SIJ MRI in gestational weeks 20/32 and 3/6/12 months postpartum. Two assessors independently evaluated presence and evolution of subchondral sclerosis with a depth >5 mm, including location and concomitant lesions. A sclerosis depth >7 mm, the lowest threshold reported in patients with OCI [1], was defined as MRI-OCI. Potential associations between SIJ sclerosis and clinical features were explored.
Results. 71 women scanned at 12 months postpartum with >1 MRI were included, in total 269 SIJ MRI (Table I). The prevalence of subchondral sclerosis increased over time and was most frequently located in the anterior middle SIJ portion. 17 women (23.9%) showed sclerosis of >7 mm at 12 months postpartum (Table). BME and fat lesion were frequently related to sclerosis (Fig. 1 & Table I). All incident sclerosis lesions except one showed preceding or concomitant BME (Fig. 1). There were no differences between women with MRI-OCI and the remaining regarding age of the mother, BMI prior to pregnancy, weight gain during pregnancy, HLA B-27 status, and low back/buttock pain during pregnancy and/or postpartum.
Conclusion. Subchondral MRI-OCI increased from pregnancy to 12 months postpartum and was associated to BME and/or fat lesion, with BME nearly always present before or simultaneously with incident sclerosis.

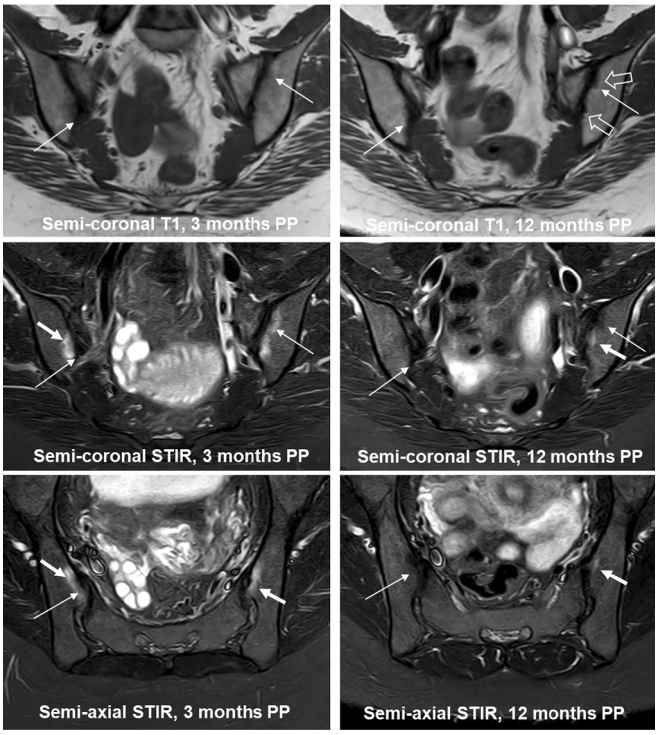
Reference

1. MA L *et al.: Int J Rheum Dis* 2018; 21: 299.

P77: Table I. Evolution of subchondral sclerosis per patient or per joint from gestational week 20 to 12 months postpartum.

	Gestational week 20 30 subjects/ 60 joints	Gestational week 32 29 subjects/ 58 joints	3 months postpartum 69 subjects/ 138 joints	6 months postpartum 70 subjects/ 140 joints	12 months postpartum 71 subjects/ 142 joints
Sclerosis >5mm (subjects)	6 (20%)	7 (24.1%)	24 (34.8%)	32 (45.7%)	33 (46.5%)
Sclerosis >7mm (subjects)	2 (6.7%)	3 (10.3%)	7 (10.1%)	13 (18.6%)	17 (23.9%)
Unilateral sclerosis >5mm	4 s (66.7%)	3 s (42.8%)	15 s (62.5%)	18 s (56.2%)	17 s (51.5%)
Bilateral sclerosis >5mm	2 s (33.3%)	4 s (57.1%)	9 s (37.5%)	14 s (43.8%)	16 s (48.5%)
Incident sclerosis >5 mm since last scan	-	3 j	4 j	9 j	2 j
Location iliac sclerosis >5mm:					
Upper:	4 j	5 j	12 j	18 j	19 j
Middle:	5 j	6 j	22 j	32 j	32 j
Lower	0 j	1 j	7 j	10 j	12 j
Location iliac sclerosis >5mm:					
Anterior:	6 j	11 j	31 j	45 j	49 j
Posterior:	1 j	2 j	6 j	11 j	12 j
Anterior+posterior	1 j	2 j	4 j	10 j	12 j
Max axial depth of sclerosis, all joint	6.5 ±0.9 (5.3-7.5)	6.8±1.2 (5.2-9.0)	7.0±1.8 (5.1-12.1)	7.2±1.8 (5.1-12.1)	7.5±1.8 (5.2-12.9)
BME associated to sclerosis *	5 j	5 j	20 j	20 j	16 j
Fat lesion associated to sclerosis *	1 j	1 j	9 j	21 j	23 j

*Peripherally and/or inside sclerosis. + No sclerosis was noted/scored in sacrum.
s = number of subjects; j = number of joints.



P77: Fig. 1. MRI in a 32-year-old HLA-B27 negative woman indicating low back and buttock pain during and after pregnancy. Semi-coronal T1-weighted and STIR images in two perpendicular planes at 3 and 12 months postpartum. At 3 months postpartum there is sclerosis (thin arrows) with adjacent BME (thick arrows). At 12 months postpartum, sclerosis has increased (thin arrows), especially on the left side; BME has decreased (thick arrows) while fat lesion has supervened (open arrows).

P78

COMBINED TESTING OF ANTI-CD74 IGA AND ANTI-UH-AXSPA ANTIBODIES INCREASES DIAGNOSTIC POTENTIAL FOR AXSPA

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Introduction. Diagnosis of axial spondyloarthritis (axSpA) is challenging and a specific laboratory diagnostic test is lacking. Previously, we identified novel immunoglobulin G (IgG) and IgA antibodies to 4 Hasselt University (UH)-axSpA antigens (UH-axSpA-IgG 4, 8 and UH-axSpA-IgA 1,10), corresponding to non-physiological peptides and to a novel axSpA autoantigen, Double homeobox protein 4 (DUX4) (1). Validation of antibody reactivity in plasma samples of axSpA patients from the Leuven spondyloarthritis biologics cohort (BIOSPAR) revealed antibody reactivity against at least one of these 4 peptide targets in 15.9 % of axSpA patients (26/164) (2). In addition, IgA antibodies against CD74, involved in the assembly of and the prevention of premature peptide-binding to major histocompatibility complex (MHC) class II has been described in axSpA. Here we aim to determine the diagnostic potential of the anti-CD74 IgA antibodies in combination with previously determined IgG and IgA antibodies against the 4 UH-axSpA antigens.
Methods. Anti-CD74 IgA antibodies were measured using the AESKULISA SpA Detect Kit in axSpA patients from the BIOSPAR cohort and patients with chronic low back pain (CLBP) served as the control group.
Results. In the BIOSPAR cohort, anti-CD74 IgA antibodies were present in 29.3% of axSpA patients (48/164) versus no presence in the CLBP patients (0/58) (p<0.0001). Additional testing for the presence of antibodies against the 4 UH-axSpA peptides further increased the antibody reactivity in 40.2% of the patients (66/164) and only in 3.4% of the CLBP patients (2/58)

(p<0.0001). Interestingly, we found a significant increase in age (p=0.0142), disease duration (p=0.0184) and Bath Ankylosing Spondylitis Functional Index (BASFI) (p=0.0101) in axSpA patients who tested positive for antibodies against at least one of the 4 UH-axSpA antigens or CD74 compared to seronegative patients.
Conclusion. Combined testing for anti-CD74 IgA antibodies and the 4 novel anti-UH-axSpA antibodies results in improved diagnosis of axSpA.

References
1. QUADEN D *et al*.: *Arthritis Rheumatol* 2020.
2. RUYTINX P *et al*.: *Front Med* (Lausanne) 2022.

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IGF-1R EXPRESSION IN LEUKOCYTES OF PATIENTS WITH SPONDYLOARTHRITIS

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Introduction. In contrast to adult-onset spondyloarthritis (SpA), patients with juvenile-onset SpA (or JoSpA) rarely show axial involvement and present with more severe manifestations. Considering that JoSpA could be related to bone growth factors associated with childhood and adolescence, it is interesting to assess the role of insulin-like growth factor type 1 (IGF-1) in its pathogenesis. The IGF-1 receptor (IGFR-1) signalling contributes to the T-cell-dependent inflammation in other types of inflammatory arthritis; also, it can promote a phenotypic shift in T cells towards a Th17 profile with inhibition of differentiation of Treg cells.
Pathways involving IGF-1 and IGFR-1 might be related to inflammation and osteoproliferation in SpA patients.
Methods. Nine patients with SpA classified according to ASAS criteria were included. Several clinical variables were registered, and 10 mL blood samples were taken. The frequencies of granulocytes, total lymphocytes, NK, NKT, Tγδ lymphocytes, CD4+ and CD8+ T lymphocytes were analysed using whole blood spectral flow cytometry. In addition, IGFR-1 expression and the frequency of IGFR-1+ cells were measured. Also, the IGFR-1 expression was examined in a previously published single cell-RNA seq database of T cells from ankylosing spondylitis patients' blood and synovial fluid samples.
Results. We found a significant difference in IGFR-1 expression in CD4+ T lymphocytes of patients and controls (p=0.0391) and a significant increase in the percentage of total IGFR-1 positive lymphocytes (p=0.0413). We found a negative correlation of evolution time with IGFR-1 expression in total lymphocytes.
Conclusion. The high expression of IGFR-1 in CD4+ T lymphocytes and the high frequency of IGFR-1-positive total lymphocytes indicate an alteration of this pathway in these patients, which could be related to IGFR-1's role in T-cell-mediated inflammation.
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THE PHENOTYPE OF CIRCULATING GUT-HOMING α4β7+ Tγδ CELLS OF PATIENTS WITH SPONDYLOARTHRITIS

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Introduction. Our study delves into the aberrant migration theory in SpA, proposing that lymphocytes may originate in the gut and then migrate to the joints or skin of patients, thereby triggering inflammatory effects. The α4β7 integrin, a ligand for the mucosal adhesion molecule MAdCAM-1, is a key marker for gut-homing lymphocytes. In a recent breakthrough, we discovered that α4β7+ Tγδ cells from axSpA patients exhibit a pre-activated state, characterized by the overexpression of TLR-2 and TLR4. However, the phenotype of these cells and their potential role in the pathogenesis of SpA remain uncharted territories.
Methods. We took blood samples from 16 SpA patients classified with ASAS and obtained PBMCs; afterwards, we stimulated them with IL-23, LPS, and low molecular weight hyaluronic acid to determine the expression of the α4β7 integrin, IL-17, and IL-22. Also, we analysed immune memory-associated markers (CD27, CD45RO and CD161) in a different cohort of 5 patients with SpA.
Results. We did not find significant differences in the IL-17 or IL-22 production from α4β7+ Tγδ cells but found interesting differences in their profile when comparing TCRγδ ^{high} and TCRγδ ^{low} cells. Regarding the second cohort, we found that α4β7+ Tγδ are comprised within the CD27- compartment and express high amounts of IL-17.
Conclusion. Our findings shed light on the unique characteristics of gut-homing Tγδ cells, revealing that they express distinct inflammatory profiles based on their TCR expression and are primed for IL-17 production. This discovery could potentially pave the way for new therapeutic strategies targeting these cells in the treatment of SpA.
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UTERUS DYSBIOSIS AND SUBCLINICAL UTERINE INFLAMMATION ARE INVOLVED IN THE PATHOGENESIS OF FEMALE SPONDYLOARTHRITIS

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Introduction/Objective. Spondyloarthritis (SpA) presents different clinical manifestations in females, including delayed diagnosis and treatment responses, emphasizing the need to investigate sex-specific factors in SpA pathogenesis. The specific contribution of the female genital immune system remains something totally unexplored in SpA. Recent animal models' data suggest an interplay between the uterus and joints. In this work we have investigated in SpA female patients the possible existence of a uterine-joint axis, which can contribute to the disease's pathogenesis.

Methods. (1) We have collected vaginal, cervical, and uterine swabs form 25 premenopausal, newly diagnosed SpA patients and 25 age-matched healthy controls (HC) and tested the microbial composition sequencing the 16S rRNA to identify distinct microbial patterns associated with SpA. (2) To evaluate genital inflammation in the context of arthritis, SKG preclinical model of SpA (n=15) were assessed histologically at baseline and at 1 and 2 weeks post-curdlan intraperitoneal administration. (3) Bulk RNA sequencing was performed on SKG uterine samples to delineate gene expression patterns associate with SpA. (4) Further, we have characterised at single cell level (10X Genomics) uterine resident immune isolated from menstrual blood from SpA and HC (n=5 in total) for precise cellular profiling.
Results. Our study revealed significant dysbiosis in the genital tract of female SpA patients, characterized by a reduced abundance of *eubiotic lactobacilli* and a notable presence of *Gardnerella vaginalis* (83% SpA vs 16% HC; $p<0.001$). In the SKG mouse model, we observed genital inflammation, more pronounced 1-week post-curdlan, characterized by the presence of inflammatory infiltrate, microabscesses and squamous metaplasia ($p<0.001$). Differentially expressed genes discriminating curdlan-treated mice from controls were identified with bulk RNA sequencing of SKG uterine samples. Single-cell transcriptomics on menstrual blood identified a strong imbalance of monocytes and ILC2 between SpA patients and controls.
Discussion/Conclusion. Our study demonstrated the presence of significant genital tract dysbiosis and distinct immune cell profiles in female SpA patients compared to HC. A multimodal genomic and molecular approach will be of benefit to unravel the intricate relationship between the cervicovaginal immune landscape and SpA pathogenesis in female, highlighting a potential role of the uterine-joint axis in the disease.

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RUMINOCOCCUS GNAVUS IN SPONDYLOARTHRITIS, STORY OF A COMMENSAL KILLER

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Introduction. Spondylarthritis (SpA) is a chronic inflammatory disorder characterized by osteoarticular and extra-articular manifestations, including inflammatory bowel disease (IBD). It is established that intestinal microbiota dysbiosis plays a role in IBD pathophysiology. We recently demonstrated that intestinal dysbiosis occurred during SpA. Thus, relative abundance of *Ruminococcus gnavus* is significantly increased in SpA patients. In this study, our goal was first to isolate *R. gnavus* from SpA patients and healthy controls (HCs). Second, we tested if *R. gnavus* strains from SpA patients were more proinflammatory than those from HCs. Finally, we evaluated if *R. gnavus* strains were selected by resistance to oxygen during SpA.
Methods. *R. gnavus* colonies were isolated from colonic biopsies and/or stools from SpA patients and HCs. Isolated *R. gnavus* strains were sequenced. Proinflammatory functions of *R. gnavus* strains were evaluated by their ability to induce mortality and tumor necrosis factor (TNF) production in monocytes isolated from SpA patients. Aerotolerance experiments were performed to test selection of *R. gnavus* by the proinflammatory environment of the gut during SpA.
Results. We successfully isolated *R. gnavus* colonies from HCs and SpA patients. Upon sequencing, we identified 33 different strains isolated from 13 SpA patients and 4 HCs. Interestingly, strains did not overlap between SpA patients and HCs. Mechanistically, *R. gnavus* strains isolated from SpA patients induced the greatest mortality and TNF production in patients' monocytes. Finally, *R. gnavus* strains isolated from SpA patients were not characterized by greater aerotolerance.
Conclusion. Our work demonstrates a broad *R. gnavus* diversity in stool and biopsies from SpA patients and HCs. Moreover, *R. gnavus* strains isolated from SpA patients induced greater mortality and TNF production in monocytes from SpA patients. Further studies in preclinical murine models of SpA will be required to better define the role of *R. gnavus* during SpA.

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ASSOCIATION SPONDYLOARTHRITIS AND INFLAMMATORY BOWEL DISEASE: MORE DIFFICULT TO MANAGE ON BOTH SIDES

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Introduction. Coexistence of inflammatory bowel diseases (IBD) with Spondyloarthritis (SpA) is a complex situation. The aim of this study was to evaluate the characteristics of SpA/IBD patients in comparison with SpA and IBD alone.
Methods. A single-centre observational study included consecutive patients followed between 2019 and 2022 with SpA meeting the ASAS 2009 criteria and IBD. For each patient, we collected demographics, extrarticular manifestations, imaging data and the number of bDMARDs used. We compare these patients with single diagnosis patients.
Results. 62 patients with SpA/IBD, 51% male and 67% HLA B27+, were included. IBD was more frequently diagnosed first. In comparison with SpA patients, SpA/IBD patients, had more uveitis (27% vs 18%, $p=0.08$) and psoriasis (27% vs 17%, $p=0.04$), were more smoker (66% vs 44.9%; $p=0.01$) but less HLA B27 positive (63% vs 80%, $p=0.002$). They used more bDMARDs (2.8 (±1.7) vs 2 (±1.15); $p=0.01$). In comparison with IBD patients, SpA/IBD patients had more uveitis (27% vs 1%, $p=0.08$) and psoriasis (27% vs 20%, $p=0.04$), and were more smoker (66% vs 44.9%; $p=0.01$). They also used more bDMARDs (2.8 (±1.7) vs 1.65 (±0.8); $p=0.01$) and had higher clinical severity score (HBI) at diagnosis (8.2 (±5.7) vs 2.6 (±3.1) $p=0.0006$) and at last follow-up (4.2 (±3.4) vs 2.4 (±2.5); $p=0.0074$).SpA/IBD patients requires frequently (9,7%) combination of bDMARDs contrary to single diagnosis ($p<0,05$).
Conclusion. SpA/IBD patients are more severe both on the rheumatological and intestinal side and represent a cluster difficult to manage.

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JOINT INVOLVEMENT IN INFLAMMATORY BOWEL DISEASE: AN OBSERVATIONAL STUDY

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Introduction/Objective. Joint involvement is the most frequent extra-intestinal manifestation of inflammatory bowel disease (IBD), but the associated clinical factors remain unknown. We assessed the frequency and characteristics of joint manifestations in a cohort of IBD patients, in order to identify IBD clinical predictors of articular symptoms.
Materials and methods. Two independent rheumatologists evaluated IBD patients between January 2016 and August 2018 in CHU de Liège. First rheumatologist collected symptoms (IBD characteristics, rheumatic symptoms and other extra-intestinal manifestations), performed clinical examination (tender and swollen joints' counts, SPARC and MASES indexes, Smythe points, axial metrology) and had the patients fill in questionnaires (HAQ and BASDAI). Blood C reactive protein and fecal calprotectine levels were measured. Second rheumatologist performed a blind ultrasound of knee and ankle entheses. We tested for correlations between peripheral, axial and entheseal involvement and the collected variables in Crohn Disease (CD) and Ulcerative colitis (UC) patients through linear regression, and performed a cluster analysis.
Results. 199 patients were included: 143 CD (71.9%), 56 UC (28.1%). Mean intestinal disease duration was 14.47±10.07 years. 79.4% reported arthralgia with an inflammatory pattern in 27.8%, 25.6% entheseal pain and 14.8% axial inflammatory pain. Past peripheral involvement, knee in particular, was slightly higher in UC than in CD ($p<0.05$). Cluster analysis identified two groups of patients: first group (103, 51.8%), consisted of older patients, mostly CD, presenting more entheseal and peripheral involvement ($p<0.001$). When performing clustering in CD only, smoker women, presenting higher IBD activity (Pro2 Stool), more dermatological and hepatical comorbidities and more frequently treated with anti TNF drugs presented more peripheral and entheseal involvement and positive Smythe points ($p<0.001$).
Conclusion. Joint involvement was commonly found in our dataset. Our results suggest that CD women with an active IBD and extra-intestinal symptoms would be more prompt to develop articular manifestations.

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INTESTINAL NEUROINFLAMMATION IS ASSOCIATED WITH SUBCLINICAL GUT INFLAMMATION AND DYSBIOSIS IN SPONDYLOARTHRITIS

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Introduction/Objective. Spondyloarthritis (SpA) is associated with subclinical gut inflammation and dysbiosis, highlighting the gut-joint axis. The mechanistic link between gut dysbiosis and intestinal inflammation remains unclear. VIPergic enteric innervation impacts gut homeostasis, influencing immune responses and fucosylation, which are crucial for microbiota balance. This study aims to determine the presence of neuroinflammation and its relationship to dysbiosis and gut inflammation in SpA patients.

Materials and methods. Expression of neurotransmitter and neuron-associated molecules (*GDNF, GFRA1, NRTN, VIP, VIPR*) was analyzed in ileal tissues from treatment-naïve SpA patients (n=10) and healthy controls (n=10) using quantitative RT-PCR and immunohistochemistry. Neuroinflammatory markers were examined in SKG mice treated with curdlan (n=8) and transgenic HLA-B27 rats. Immunofluorescence microscopy and flow cytometry were used to evaluate intestinal fucosylation and co-localization of nerves and immune cells. Germ-free SKG mice were used to investigate the impact of microbiota.

Results. Ileal tissues of SpA patients showed significantly reduced expression of *GDNF*, *GFRA1*, *NRTN*, *VIP*, and *VIPR* compared to controls ($p<0.05$), with pronounced downregulation in males and patients with subclinical gut inflammation (r= -0.82, $p<0.02$). VIPergic nerve fibers and CD8+ T cells colocalized, resembling a neuroimmunological synapse. VIP-producing mononuclear cells were similar in SpA and controls, but VIPergic fibers were significantly reduced. In SKG mice, *VIP* and *VPAC2* were significantly reduced compared to BALB/c controls post-curdlan administration in a microbiota-dependent manner. These results were mirrored also in HLA-B27 transgenic rats. In line, reduced ileal fucosylation and lower Fut2 expression ($p<0.05$) were observed in SpA patients.

Conclusion. SpA patients show marked depletion of intestinal nerve fibers and neurotrophic factors, and animal models confirmed the microbiota's role in this phenomenon. These findings suggest a bidirectional relationship between neuroinflammatory marker expression and gut microbiota, with altered fucosylation perpetuating ileal dysbiosis. This may elucidate the functional consequences of autoreactivity against neuronal markers and VIP receptors in SpA.

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DOES LATE ONSET AXIAL SPONDYLOARTHRITIS EXIST? RESULTS FROM A MULTICENTRE NATIONWIDE STUDY

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Introduction. Axial spondyloarthritis (axSpA) typically starts before the fourth decade of life but It has been suggested that the disease can also start later in some cases. We aimed to evaluate the occurrence of 'late onset axSpA' (lo-axSpA) and whether these patients differ from those with early onset axSpA (eo-axSpA).

Methods. We performed a cross-sectional, multicentre, nationwide study using data from the Portuguese registry of rheumatic diseases (Reuma.pt). Patients with a clinical diagnosis of axSpA and with available information on the age of symptom onset were included. Demographic and disease characteristics were compared between patients with lo-axSpA (≥45y) and eo-axSpA (<45y). The independent association between these characteristics with lo-axSpA was tested in a multivariable logistic regression analysis.

Results. In total, 2165 patients with axSpA were included. The mean (standard deviation) age at symptom onset was 32 (10) years. Most of the patients were male (56%), had radiographic sacroiliitis (85%) and were treated with bDMARDs (77%). Only a minority (n=273; 13%) of the patients had symptom onset ≥45 years (lo-axSpA). Patients with lo-axSpA were less often

P86: Table I. Comparison of patients and disease characteristics between patients with late and early onset axSpA.

	Age of symptom onset ≥45 years (n=273)	Age of symptom onset <45 years (n=1892)	*p*-value*
Current age (years)	62.3 (8.8)	47.1 (12.2)	**<0.001**
Age at symptom onset (years)	51.4 (6.0)	28.9 (7.3)	**<0.001**
Symptom duration (years)	10.9 (6.9)	18.19 (11.9)	**<0.001**
Male gender	150 (55%)	1059 (56%)	0.749
Education (years)	7.5 (4.0)	10.5 (4.3)	**<0.001**
Current employment status	91 (48%)	956 (70%)	**<0.001**
HLA-B27	136 (51%)	1210 (65%)	**<0.001**
Elevated CRP (ever)	166 (62%)	1177 (63%)	0.073
Family history of SpA	21 (8%)	268 (14%)	**0.003**
Inflammatory back pain (ever)	216 (81%)	1634 (88%)	**0.001**
Acute anterior uveitis (ever)	34 (13%)	379 (20%)	**0.003**
Good response to NSAIDs (ever)	141 (53%)	1028 (55%)	0.435
Peripheral arthritis (ever)	96 (36%)	525 (28%)	**0.010**
Heel enthesitis (ever)	56 (21%)	388 (21%)	0.976
Dactylitis (ever)	14 (5%)	73 (4%)	0.312
Psoriasis (ever)	7 (3%)	47 (3%)	0.930
Inflammatory bowel disease (ever)	7 (3%)	38 (2%)	0.542
Damage on X-SIJ (mNY) (ever)	172 (82%)	1317 (85%)	0.284
Sacroiliitis on MRI-SIJ (ever)	97 (79%)	636 (72%)	0.092
ASDAS	2.2 (1.1)	2.1 (1.0)	0.208
BASFI	3.5 (2.9)	3.3 (2.8)	0.351
Current treatment with bDMARDs	213 (78%)	1449 (77%)	0.599

*Independent samples t-test for continuous variables and Chi2 for categorical variables. ASDAS: Ankylosing Spondylitis Disease Activity Score; ax-SpA: axial spondyloarthritis; BASFI: Bath Ankylosing Spondylitis Functional Index; bDMARDs: biological disease-modifying anti-rheumatic drugs; CRP: C-reactive protein; mNY: Modified New York Criteria; MRI-SIJ: magnetic resonance imaging of the sacroiliac joints; NSAID: non-steroidal anti-inflammatory drugs; SpA: spondyloarthritis; X-SIJ: X-ray of the sacroiliac joint.

P86: Table II. Association between patient and disease characteristics with late onset axial SpA (multivariable analysis)

Variable	Univariable models OR (95% CI) (n=1011-2165)	Multivariable model OR (95% CI) (n=2132)
Sex/Male gender	1.0 (0.7; 1.2)	1.0 (0.8; 1.3)*
HLA-B27	**0.6 (0.4; 0.7)**	**0.6 (0.4; 0.7)**
Family history	**0.5 (0.3; 0.8)**	**0.6 (0.4; 0.9)**
Inflammatory back pain	**0.6 (0.4; 0.8)**	**0.5 (0.4; 0.8)**
Acute anterior uveitis	**0.6 (0.4; 0.8)**	**0.6 (0.4; 0.9)**
Peripheral arthritis (ever)	**1.4 (1.1; 1.9)**	**1.5(1.1; 1.9)**

Variables selected for multivariable models if *p*-value <0.2 in univariable analysis or clinical significance.
* Forced for the multivariable model due to clinical significance.

positive for HLA-B27 (51% vs 65%), less likely to have family history of SpA (8% vs 14%), acute anterior uveitis (AAU, 13% vs 20%) and inflammatory back pain (IBP, 81% vs 88%) but had more peripheral arthritis (36% vs 28%) than patients with eo-axSpA (Table I). The multivariable analysis was consistent with these findings (Table II).
Conclusion. Axial SpA starts before 45 years of age in most patients. Late-onset disease is a minority phenotype with a weaker association with HLA-B27. These data suggest lo-axSpA as a subset of axSpA but difficulties in disease recognition and recall bias cannot be excluded.

P87

COMPARING THE CONSTRUCT VALIDITY OF MEASUREMENT INSTRUMENTS FOR PAIN AND STIFFNESS IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS

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Introduction. In the context of the recent update of the ASAS core outcomes set (COS) for axSpA, the preferred comparative validity of the measurement instruments to assess the domains 'Pain' and 'Stiffness' has been questioned as for each domain responsiveness of instruments was comparable. The known-group discrimination assessment provides useful insights while allowing to compare among measurement instruments. We aimed to compare the construct validity, including known-group discrimination, of three pain and three morning stiffness (MS) instruments.
Methods. Patients with r-axSpA with 8-year-data from the OASIS cohort were assessed cross-sectionally. Three instruments for pain (total back pain, night pain at night, total back pain BASDAI-Q2) and three for MS (MS-severity, MS-duration, MS-severity/duration) were compared. Construct validity was evaluated by testing (i) hypothesis of correlations' strength and (ii) discrimination between known-groups with standardised mean differences (SMD) across several external constructs. Influence of contextual factors (CFs) on SMDs was investigated.
Results. Of 85 patients, mean age was 54 (SD 11), symptom duration 31 (11) years, 71% were male. All the six instruments showed a good construct validity by fulfilling >75% of the hypotheses for the strength of correlation. Total back pain BASDAI-Q2 and total back pain had higher SMDs compared to back pain at night across all group comparisons, with BASDAI-Q2 performing mostly slightly better (external construct ASDAS ≥2.1 vs <2.1, SMD 1.87 and 1.56 vs 1.07, respectively) (Table I). MS-severity and severity/duration had higher SMDs across all external constructs (with MS-severity slightly better), while MS-duration performed worse (SMD external construct ASDAS 1.16 vs 1.51 and 1.39). Influence of CFs on known group discrimination was limited.
Conclusion. The recommended ASAS-COS pain instrument total back pain BASDAI-Q2 has the best known-groups discrimination. For MS, the ASAS-COS stiffness measure (MS-severity/duration) performs well although MS-severity even slightly better. The known-group discrimination is overall stable across CFs.

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EVALUATION OF INSTRUMENTS ASSESSING PERIPHERAL ARTHRITIS IN SPONDYLOARTHRITIS: AN ANALYSIS OF THE ASAS-PERSPA STUDY

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Introduction. The optimal instrument to assess peripheral arthritis and related disease activity in SpA has not yet been identified. The objective of this study was to assess the construct validity, including known-groups discrimination, of the currently available disease activity instruments assessing peripheral arthritis in SpA.
Methods. In this analysis from the ASAS-PerSpA study, patients with a diagnosis of axSpA/pSpA or PsA were included. The disease activity instruments evaluated were patient global assessment (PGA), BASDAI, ASDAS, DAPSA, swollen joints count (SJC), tender joints count (TJC), DAS28, DAS44, and C-reactive protein (CRP). Construct validity was assessed through correlations with external constructs (BASFI, ASAS-HI and EuroQoL), and known-groups discrimination. For the latter, the discrimination between active/inactive groups based on a combination of PGA (≥5/<5) and SJC (≥1/0 and also ≥2/<2, median SJC) was analysed using standardised mean differences (SMDs).
Results. In total, 4,121 patients were included (mean age 45 (SD 14) years, 61% males). When assessing the construct validity through correlations, all instruments had an excellent performance (100% of confirmed hypotheses). In the entire population, all disease activity measures, except for CRP, presented SMDs≥0.8 (good performance), with a higher SMD being observed for DAS28 followed by DAPSA, DAS44, BASDAI and ASDAS (Table I). When the analyses were stratified for the individual phenotypes (axSpA, pSpA and PsA) results were similar. Taking all the possible combinations of PGA and SJC into account for the comparison between active/inactive disease, a better performance was observed for the composite scores with joint counts (Table II).
Conclusion. In our construct validity analysis, all disease activity instruments assessing peripheral arthritis had a good performance reflected in the correlations with external constructs and the known-groups discrimination. The highest discriminatory capacity to distinguish between "active and inactive disease" was observed for composite scores including joints scores, like DAS28 and DAPSA.

P87: Table I. Discrimination of pain and morning stiffness instruments between subgroups of patients.

Assessment measure	ASDAS <2.1 vs ≥2.1 SMD	BASDAI <4 vs ≥4 SMD	PGA <4 vs ≥4 SMD	Fatigue <5 vs ≥5 SMD	BASFI <4 vs ≥4 SMD	BASMI <4 vs ≥4 SMD	mSASSS <15 vs ≥15 SMD
Total back pain	1.57	1.92	1.79	1.62	0.92	0.27	-0.32
Back night pain	1.07	1.39	1.26	1.27	0.78	0.07	-0.20
Total back pain BASDAI-Q2	1.88	2.37	1.55	1.65	0.89	0.31	-0.29
Morning stiffness severity (BASDAI-Q5)	1.52	2.14	1.78	1.58	0.99	0.26	-0.11
Morning stiffness duration (BASDAI-Q6)	1.18	1.80	1.10	1.09	0.83	0.09	-0.14
Morning stiffness severity/duration (BASDAI-Q5/6)	1.40	2.14	1.46	1.38	0.95	0.17	-0.13

Values shown as mean (SD).
ASDAS: Axial Spondyloarthritis Disease Activity Score, BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; PGA: Patient Global Assessment; BASMI: Bath Ankylosing Spondylitis Metrology Index; mSASSS: modified Stoke Ankylosing Spondylitis Spinal Score; SMD: standardised mean difference.

P88: Table I. Discrimination between known groups. Analyses stratified by extreme groups according to PGA and SJC in the entire ASAS-PerSpA population and stratified for the different SpA phenotype.

	Entire population (axSpA, pSpA and PsA)			axSpA			pSpA			PsA		
Assessment measure	PGA<5 & SJC=0 (n=1823)	PGA≥5 & SJC ≥1 (n=577)	SMD	PGA<5 & SJC=0 (n=1335)	PGA≥5 & SJC ≥1 (n=197)	SMD	PGA<5 & SJC=0 (n=144)	PGA≥5 & SJC ≥1 (n=120)	SMD	PGA<5 & SJC=0 (n=344)	PGA≥5 & SJC ≥1 (n=260)	SMD
BASDAI (0-10)	2.15 (1.57)	6.17 (1.95)	2.40	2.12 (1.51)	6.44 (1.96)	2.73	2.05 (1.8)	5.65 (1.88)	1.96	2.32 (1.68)	6.21 (1.94)	2.17
ASDAS	1.73 (0.75)	3.66 (0.96)	2.40	1.74 (0.74)	3.95 (1.01)	2.83	1.65 (0.74)	3.51 (0.95)	2.20	1.72 (0.77)	3.52 (0.89)	2.18
DAPSA	5.23 (4.85)	28.16 (15.67)	2.62	4.83 (4.34)	26.33 (14.11)	3.32	6.02 (5.69)	25.52 (13.5)	1.94	6.44 (5.97)	30.76 (17.30)	1.99
TJC (0-68)	0.70 (2.34)	8.06 (10.40)	1.34	0.48 (1.68)	6.32 (8.48)	1.71	1.35 (3.4)	6.66 (8.68)	0.83	1.26 (3.53)	10.01 (12.01)	1.05
DAS28	1.88 (0.57)	4.12 (1.07)	3.11	1.86 (0.54)	4.03 (0.98)	3.54	1.92 (0.63)	4 (1.06)	2.43	1.94 (0.64)	4.24 (1.12)	2.61
DAS44	0.84 (0.40)	2.29 (0.93)	2.51	0.81 (0.35)	2.17 (0.86)	3.03	0.96 (0.52)	2.2 (0.89)	1.75	0.92 (0.50)	2.42 (0.99)	1.98
CRP (mg/L)	8.30 (22.59)	22.26 (38.67)	0.51	8.09 (20.54)	28.38 (45.17)	0.81	7.46 (16.98)	24.97 (36.94)	0.63	9.48 (30.81)	16.38 (32.99)	0.22

Values shown as mean (SD).
Cell colored with the following system: SMD ≥0.8 (green); ≥0.5 (orange); ≥0.5 (red). PGA: Patient Global Assessment; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index, ASDAS: Axial Spondyloarthritis Disease Activity Score; DAPSA: Disease Activity in Psoriatic Arthritis; SJC: Swollen Joint Count; TCJ: Tender Joint Count; DAS28/44: Disease Activity Score.

P88: Table II. Discrimination between known groups. Analyses stratified by both PGA and SJC* in the entire ASAS-PerSpA population (n=4, 121).

	Patient global assessment <5 (n=2,100)					Patient global assessment ≥5 (n=2,021)						
Assessment measure	SJC<2 (n=1,947)	SJC≥2 (n=153)	SMD	SJC=0 (n=1,823)	SJC≥1 (n=277)	SMD	SJC<2 (n=1,624)	SJC≥2 (n=397)	SMD	SJC=0 (n=1,444)	SJC≥1 (n=577)	SMD
BASDAI (0-10)	2.2 (1.6)	3.18 (1.87)	0.60	2.15 (1.57)	3.03 (1.86)	0.54	5.37 (1.96)	6.21 (1.91)	0.43	5.28 (1.93)	6.17 (1.95)	0.46
ASDAS	1.75 (0.76)	2.29 (0.89)	0.70	1.73 (0.75)	2.16 (0.89)	0.56	3.22 (0.86)	3.71 (0.99)	0.55	3.18 (0.84)	3.67 (0.96)	0.55
DAPSA	5.5 (4.98)	19.51 (15.93)	2.18	5.23 (4.85)	15.02 (13.3)	1.48	14.99 (7.84)	31.45 (16.96)	1.60	14.25 (7.39)	28.16 (15.67)	1.33
TJC (0-68)	0.78 (2.4)	6.74 (10.46)	1.64	0.7 (2.34)	4.62 (8.34)	1.05	2.53 (5.42)	9.54 (11.26)	1.01	2.25 (5.09)	8.06 (10.4)	0.83
DAS28	1.92 (0.59)	3.34 (0.97)	2.26	1.88 (0.57)	2.97 (0.94)	1.73	2.95 (0.79)	4.32 (1.13)	1.58	2.86 (0.75)	4.12 (1.07)	1.48
DAS44	0.88 (0.43)	2.02 (0.88)	2.41	0.84 (0.4)	1.72 (0.8)	1.86	1.25 (0.67)	2.5 (0.97)	1.70	1.18 (0.64)	2.29 (0.93)	1.50
CRP (mg/L)	8.4 (22.25)	14.48 (20.28)	0.28	8.3 (22.59)	12.43 (18.72)	0.19	12.83 (26.69)	24.53 (43.5)	0.38	12.28 (26.94)	22.26 (38.67)	0.32

Values shown as mean (SD).
Cell colored with the following system: SMD ≥0.8 (green); ≥0.5 (orange); ≥0.5 (red). PGA: Patient Global Assessment; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; ASDAS: Axial Spondyloarthritis Disease Activity Score; DAPSA: Disease Activity in Psoriatic Arthritis; SJC: Swollen Joint Count; TCJ: Tender Joint Count; DAS28/44: Disease Activity Score.
*Population was stratified by PGA and SJC in different ways showing all the groups of such stratification allowing us to evaluate the performance of the indices in the complete population and according to the different levels of disease activity (including two different cut-offs of SJC).

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EXPLORING THE INFLUENCE OF SAGITTAL IMBALANCE ON PATIENT-REPORTED OUTCOME MEASURES IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS: IS THERE AN ADAPTATION MECHANISM?

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Objective. To investigate whether sagittal imbalance is associated with worse quality of life, disease activity, mobility, and function in patients with axial spondyloarthritis (axSpA).
Methods. In this cross-sectional study, sagittal vertical axis (SVA), an important parameter of sagittal balance, was obtained using EOS® (Biospace, Paris, France). SVA≥50mm delineates sagittal imbalance status. Patients were categorized into two groups based on SVA [Group1 – SVA <50mm (n=73); Group 2 - SVA ≥50mm (n=44)]. Patient-reported outcome (PRO) measures were compared between the groups and a suitable multivariable linear regression model was performed to assess the association with the SVA.
Results. A total of 177 patients were included and there was no difference between groups 1 and 2 regarding age, HLA-B27 status and BMI. However, there was higher prevalence of men, longer disease duration and higher Bath Ankylosing Spondylitis Functional Index (BASFI) and Bath Ankylosing Spondylitis Metrology Index (BASMI) in group 2. There was no difference between both groups considering disease activity and quality of life. In multivariable linear regression model, it was found the only PRO independently associated with SVA was the BASMI, after adjusting for confounders. Furthermore, a 10 mm elevation in SVA is correlated with an average increase of 0.2 points in BASMI. However, sagittal imbalance per se does not correlate with higher BASMI scores.
Conclusion. The sagittal imbalance itself did not correlate with worse PRO measures, suggesting that patients with long-term axSpA might have adaptation mechanisms.

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THE ROLE OF OBESITY IN AXIAL SPONDYLOARTHRITIS

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Introduction. While the etiology of axial spondyloarthritis (axSpA) remains multifactorial, there is growing interest in understanding the influence of obesity on disease activity, functionality, and other outcomes in this population. This study aims to investigate the association between obesity and disease activity, radiographic progression, and functional impact in patients with axSpA.
Materials and methods. We conducted a retrospective study of patients with axSpA who fulfilled ASAS criteria, followed at a Portuguese Rheumatology center. Patients were divided into three groups according to their Body Mass Index (BMI): Normal weight (BMI <25 kg/m^2); Overweight (BMI 25-29.9 kg/m^2); Obese (BMI ≥30 kg/m^2). Sociodemographic, clinical, and laboratory data were collected. Disease activity (BASDAI and ASDAS), radiographic axial damage (mSASSS), and hip involvement (BASRI-h) were collected. Parametric and non-parametric tests and Spearman's coefficient were used. Significance was set at a p-value ≤0.05.
Results. A total of 103 patients were enrolled in the study, 57 (55.3%) women, 31.1% were obese, 37.8% were overweight, and 31.1% had a normal weight. Obese patients were older compared to the other two groups (p=0.006), had a higher BASMI index (p<0.001), and a more frequent bilateral radiographic hip involvement (p<0.001). Obese patients more frequently used "on-demand" non-steroidal anti-inflammatory drugs (NSAIDs) (p=0.005). We found a fair correlation between BMI and BASMI scores (ϱ=0.32; p=0.001) and between BMI and C-reactive protein levels (ϱ=0.30; p=0.003). The need for NSAIDs and the presence of bilateral hip involvement were independently associated with obesity in axSpA [NSAIDs OR 4.8 (CI 1.6-14.7); bilateral hip involvement OR 8.9 (CI 2.9-27.1)].
Conclusion. The more frequent need for analgesic treatments, impaired spinal mobility, and increased hip involvement observed in obese patients highlight the need for tailored management strategies for this population.

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SPONDYLARTHRITIS IN A PATIENT WITH JANUS KINASE 2-POSITIVE ESSENTIAL THROMBOCYTHEMIA TREATED WITH ANAGRELIDE, COMPLICATED BY POST-ESSENTIAL THROMBOCYTHEMIA MYELOFIBROSIS

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Introduction. Spondylarthritis (SpA) comprises chronic inflammatory musculoskeletal disorders. Reactive thrombocytosis is commonly observed in systemic inflammatory diseases, as platelets act as acute-phase reactants. Essential thrombocythemia (ET) is a chronic myeloproliferative disorder with elevated platelet count, often associated with JAK2 mutations (1). Some ET patients progress to post-essential thrombocythemia myelofibrosis (post-ET MF) (1). We present a case of axial SpA associating JAK2-positive ET and subsequent myelofibrosis.
Methods. In September 2017, a 60-year-old woman with a family history of SpA, recently diagnosed with JAK2 V617F-positive ET and treated with anagrelide, presented with inflammatory pain in the gluteal and lumbar regions, extending to the left hip joint. HLA-B27 testing was positive. MRI of the sacroiliac joints revealed sacroiliitis with bilateral erosions, space narrowing, and subchondral sclerosis. Despite the initial treatment with nonsteroidal anti-inflammatory drugs (NSAIDs), due to high disease activity, adalimumab was initiated in January 2022 with good clinical response. In February 2023, the patient developed post-ET MF, prompting ruxolitinib therapy initiation. Ruxolitinib is a janus-activated kinase inhibitor (JAK) that selectively inhibits the JAK1 and JAK2 protein kinases and is used to treat myelofibrosis (2). Given the effectiveness of other JAK inhibitors (tofacitinib, upadacitinib) (3). in the treatment of axial SpA, ruxolitinib may also be beneficial. Two months after starting ruxolitinib, anti TNF treatment was stopped. The patient maintained disease remission for the next year without needing NSAIDs.
Results. To our knowledge, this is the first case of SpA associated with post-essential thrombocythemia myelofibrosis successfully treated with ruxolitinib, emphasizing the potential therapeutic benefit of ruxolitinib in SpA.
Conclusion. This case underscores the treatment journey of a patient with axial SpA, concurrent ET, secondary myelofibrosis. Due to its mechanism of action, ruxolitinib may be a feasible treatment option for patients with SpA. Randomized controlled trials are needed to prove its efficacy and expand therapeutic options for SpA.

References

1. MSD Manuals. (n.d.). Essential thrombocythemia. MSD Manual Professional Version. Retrieved May 25, 2024, from https://www.msdmanuals.com/professional/hematology-and-oncology/myeloproliferative-disorders/essential-thrombocythemia
2. HAQ M, ADNAN G: Ruxolitinib. [Updated 2023 Jun 5]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK570600/
3. VASSILAKIS KD, MAGIOUF K, SIEBERT S, FRAGOULIS GE: Selective JAK-Inhibitors in Spondyloarthritis. *Mediterr J Rheumatol* 2024; 35(Suppl 1): 27-36. doi: 10.31138/mjr.311023.sji

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WHAT IS THE ECONOMIC BURDEN OF DELAYED AXIAL SPONDYLOARTHRITIS DIAGNOSIS IN THE UK?

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Introduction. Timely diagnosis of axial spondyloarthritis (axSpA) remains challenging and is subject to potentially harmful delays. Few studies have evaluated the cost of delayed axSpA diagnosis. This study aims to develop an economic analysis to determine the annual cost of delaying the axSpA diagnosis, adopting both NHS (UK) and societal perspectives.
Methods. We developed a Markov economic model to estimate the costs of delayed axSpA diagnosis in the UK. The cohort of patients assessed comprised a mixed population (cohort size: 1,000 patients, 64% males), mean age of symptom onset 26 years. The model captured the resources used and costs related to diagnosing and managing axSpA symptoms until the disease was diagnosed.

P92: Table I. Deterministic and probabilistic sensitivity analysis results of delaying the axSpA diagnosis (8.5 years delay) classified by cost category.

Costs per category	DSA (%)	PSA (%)	95% LCI	95% UCI
Healthcare system	£7,032.74 (3.5%)	£7,048.38 (3.6%)	£5,549.66	£9,285.47
Out of pocket expenses	£60,563.04 (31.3%)	£67,875.70 (34.5%)	£32,561.78	£115,978.53
Productivity losses	£125,916.27 (65.1%)	£121,648.16 (61.9%)	£70,658.27	£181,525.29
Total	**£193,512.04**	**£196,572.24**	**£108,769.71**	**£306,789.30**

DSA: deterministic sensitivity analysis, LCI: low confidence interval, PSA: probabilistic sensitivity analysis, UCI: upper confidence interval.

P92: Table II. Nationwide total cost of delaying the axSpA diagnosis based on axSpA prevalence.

Results	Total cost - Modelled	Total cost – NASS pop*	Prevalence of 0.3% Total cost – UK adult pop**	Prevalence of 1.2% Total cost – UK adult pop**
DSA	£23,104.17	£4,236,794,775	£3,115,805,703	£12,463,222,813
PSA	£24,025.06	£4,431,645,007	£3,259,101,637	£13,036,406,549
95% LCI	£14,568.52	£2,703,455,437	£1,988,163,769	£7,952,655,079
95% UCI	£36,950.02	£6,595,614,347	£4,850,518,822	£19,402,075,288

Note: 83.4% remaining undiagnosed per year.
* NASS estimate population: 220,000 patients.
** UK adult population (2022): 53,930,490.
DSA: deterministic sensitivity analysis, LCI: low confidence interval, pop: population, PSA: probabilistic sensitivity analysis: UCI: upper confidence interval.

Results. Costs of delayed axSpA diagnosis fell primarily on individuals living with axSpA in the form of lost productivity and out of pocket medical expenses. For a mean time to diagnosis of 8.5 years, we estimate the cumulative costs of delayed diagnosis per person living with axSpA to be £193,512 (95%CI: £108,769 - £306,789) (Table I). The total annual cost resulting from delayed axSpA diagnosis in the UK has been estimated at between £3.1 billion and £12.4 billion, based on a prevalence of 0.3% (ASAS classification criteria) and 1.2% (ESSG Criteria), respectively (Table II).
Discussion. To our knowledge, this is the first study to provide an economic analysis to determine the annual cost of delay in diagnosing axSpA, adopting both NHS and societal perspectives. Our results corroborate findings from other studies, showing that patients with a late diagnosis of axSpA had higher costs, reduced capacity for work, and worse clinical outcomes.
Conclusion. Delayed axSpA diagnosis is associated with high costs for the healthcare system and society, led mainly by productivity losses, thus highlighting the importance of early diagnosis to reduce the extensive financial burden on the healthcare system, patients, and society.
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CLOSING THE DIAGNOSIS DIVIDE: INNOVATIVE STRATEGIES FOR EXPEDITED DETECTION OF AXIAL SPONDYLOARTHRITIS

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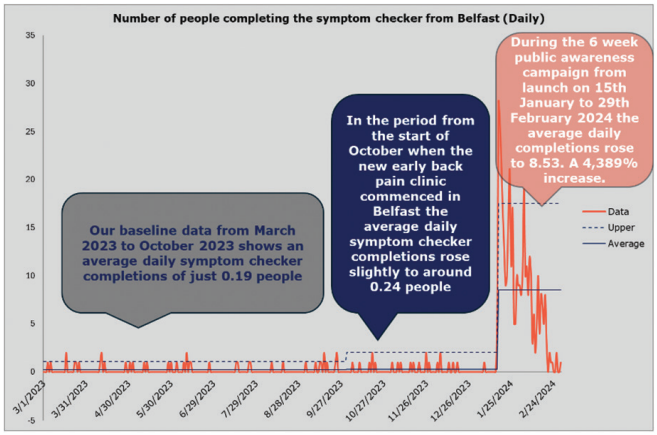
Introduction. The *Act on Axial SpA* campaign targets the 8.5-year diagnostic delay of axial spondyloarthritis (axial SpA) by advocating for a Gold Standard diagnosis time of one year. Partnering with Belfast Health & Social Care Trust, our initiative streamlines the patient journey from symptom onset to diagnosis. Through heightened awareness and optimized diagnostic procedures, our pilot project aims to reduce diagnosis time, serving as a model for broader implementation.
Methods. In collaboration with a Belfast-based agency, we launched a geo-targeted media campaign utilising out-of-home advertising, radio promotions, and social media ads.
Results. Over six weeks, the campaign generated substantial engagement: 995,399 social media impressions, 831,390 individuals reached, and an 81% reach among 18–40-year-olds in Belfast via out-of-home advertising. Radio ads reached over 565,000 adults across Northern Ireland. (Fig. 1)
Key engagement metrics include 16,799 social media ad clicks, 2,390 readership engagements on Cool FM, and a remarkable 4,389% increase in average daily completions of the NASS symptom checker. The Act on Axial SpA website experienced a surge in traffic, with 2,976 users from Belfast during the campaign compared to 285 in 2023. (Fig. 2)
Conclusion. Our results underscore the effectiveness of visually compelling marketing materials in engaging individuals with longstanding low back pain and fostering public health awareness. Early data suggests increased awareness does not overwhelm healthcare systems while expediting diagnosis journeys. Long-term monitoring will assess the campaign's impact on addressing axial SpA diagnostic delays.
Acknowledgements. The *Gold Standard Time to Diagnosis Act on axial SpA* campaign is led by National Axial Spondyloarthritis Society (NASS) in partnership with Norfolk & Norwich NHS Foundation Trust and RUH Bath NHS Foundation Trust. The *Gold Standard Time to Diagnosis* programme is funded by UCB as sponsor of the project. UCB has no editorial control over this project.

P93: Fig. 1. Infographics on the campaign results. (Source: Google Analytics, Meta Analytics).



P93: Fig. 2. Number of people completing the NASS symptom checker in Belfast. (Source: Google Analytics).

P94

EFFICACY OF BDMARDS IN EARLY VERSUS ESTABLISHED DISEASE IN AXIAL SPONDYLOARTHRITIS: A META-ANALYSIS OF RANDOMIZED TRIALS

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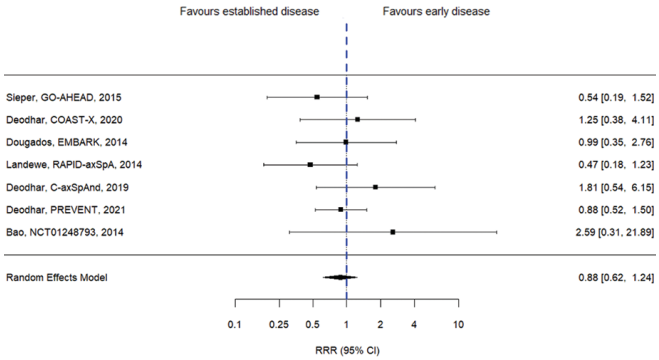
Introduction. The ASAS consensus definition of 'early axial spondyloarthritis (axSpA)' (axial symptoms ≤2 years) was expert-based given the scarcity of evidence (1). We conducted a meta-analysis of all placebo-randomised controlled trials (RCTs) of approved bDMARDs and tsDMARDs in patients with axSpA, with the aim of investigating the influence of symptom duration on treatment response.
Methods. AxSpA RCTs involving adult axSpA patients treated with b/tsDMARDs compared to placebo were identified from systematic literature reviews informing the ASAS-EULAR management recommendations (2). Vivli, ClinicalStudyDataRequest and Engagezone data-sharing platforms were used to access and analyse the individual studies. Patients were categorized based on symptom duration into early or established disease, requiring ≥ 20% of patients in each category for eligibility. The primary outcome was ASAS40 response, with secondary outcomes including ASAS and ASDAS outcomes, at the time of the primary endpoint. For each trial, treatment effects of b/tsDMARDs vs PBO were assessed through relative risks (RR) and RR ratios (RRR), defined according to the different symptom duration thresholds. Subsequently, a meta-analysis was conducted across all included RCTs, using random effects.
Results. In total 11 RCTs – all on bDMARDs- fulfilled the eligibility criteria, involving 3,272 patients with axSpA; 7 of these were eligible for the analysis of the primary endpoint. Only one had more than 20% patients over the 1-year threshold (not pooled). Table I shows the pooled RRR of bDMARDs vs PBO in early vs established disease. None of the individual studies yielded a significant RRR for ASAS40 (Fig. 1). None of the remaining outcomes yielded a significant RRR for early vs established disease according to different threshold.
Conclusion. In this meta-analysis of RCTs, no significant difference has been found in the effect of bDMARDs vs. PBO in early compared to established axSpA, defined according to different thresholds of symptom duration (between 2 and 5 years).

P94: Table I. Pooled relative risk ratio of bDMARDs *vs*. PBO in early vs established disease for primary and secondary outcomes.

Outcome	Threshold symptom duration (years)	Pooled RRR (95% CI)	I^2
ASAS40	2	0.88 (0.62-1.24)	0
	3	1.14 (0.75- 1.75)	26.12%
	4	1.11 (0.80 -1.54)	0
	5	1.08 (0.78 – 1.49)	0
ASAS20	2	0.96 (0.77 – 1.20)	0
	3	1.09 (0.83 – 1.44)	36.6 %
	4	1.14 (0.92 – 1.40)	0
	5	1.15 (0.94 – 1.41)	0.41%
ASAS PR	2	1.10 (0-58- 2.09)	0
	3	1.30 (0.67 – 2-54)	0
	4	1.15 (0.58 – 2.30)	0
	5	1.32 (0.66 – 2.64)	0
ASAS 5/6	2	0.87 (0.61 – 1.24)	0
	3	0.86 (0.47 – 1.58)	53.5%
	4	0.95 (0.67 – 1.35)	0
	5	0.93 (0.66 – 1.31)	0
ASDAS LDA	2	0.96 (0.67 – 1.37)	16.77%
	3	0.98 (0.72 – 1.33)	0
	4	0.96 (0.68 – 1.36)	0
	5	0.92 (0.66 – 1.29)	0
ASDAS ID			
	2	1.21 (0.70 – 2.07)	0
	3	1.13 (0.60 – 2.56)	0
	4	0.93 (0.43 – 2.02)	0
	5	0.90 (0.33 – 2.51)	24.46%
ASDAS CII	2	1.03 (0.76 – 1.39)	0
	3	0.90 (0.64 – 1.24)	19.20%
	4	0.83 (0.64 – 1.10)	0
	5	0.84 (0.64 – 1.11)	0
ASDAS MI	2	0.84 (0.43 – 1.64)	8.47%
	3	0.92 (0.49 – 1.72)	0
	4	0.57 (0.29 – 1.13)	0
	5	0.65 (0.32 – 1.30)	0

ASAS: Assessment of SpondyloArthritis international Society; PR: partial remission; LDA: low disease activity; ID: inactive disease; CII: clinically important improvement; MI: major improvement.



P94: Fig. 1. Relative risk ratio for ASAS40 at the timing of the primary endpoint for the bDMARD treatment vs placebo effect in patients with axSpA with early vs. established disease, defined based on the 2-year symptom duration threshold.

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References

1. NAVARRO-COMPÁN V *et al*.: ASAS consensus definition of early axial spondyloarthritis. *Ann Rheum Dis* 2023 Jun 15:ard-2023-224232.
2. WEBERS C *et al*.: Efficacy and safety of biological DMARDs: a systematic literature review informing the 2022 update of the ASAS-EULAR recommendations for the management of axial spondyloarthritis. *Ann Rheum Dis* 2023; 82(1): 130-41.

P95

THE USE OF ASDAS IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS STARTING BDMARDS IN CLINICAL PRACTICE: RESULTS FROM A MULTICENTRE PROSPECTIVE COHORT

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Background. The ASAS-EULAR recommendations for the management of axSpA prescribe the use of the ASDAS to measure disease activity and response to treatment in patients starting bDMARDs. Evidence on whether rheumatologists adhere to this recommendation in clinical practice is still limited.
Objective. We assessed: i. how many patients with axSpA, starting the first bDMARD, have ASDAS determined at baseline and in ≥1 of two follow-up visits within 6 months; and ii. which alternative outcome measures are used in patients for whom the ASDAS is missing.
Methods. Patients with axSpA from the Reuma.PT registry starting the first bDMARD (2011-2022) were included. Patients were required to have attended the following 3 visits: T0 (baseline visit at the start of the bDMARD), T1 (3 months) and T2 (6 months). The calculation of ASDAS at T0 (yes vs no) was cross-tabulated with the calculation of ASDAS in ≥1 of the two follow-up visits. The use of other outcome measures among patients without an ASDAS evaluation was evaluated.
Results. In total, 666 patients with axSpA [male: 55%; mean age: 43 (SD 12)] were included. Most patients had an ASDAS calculation at baseline (n=540; 81%), and in 493 (74%) of the patients, ASDAS was also assessed at T1 and/or T2 (Table I). No other outcome measure was predominantly used when ASDAS was absent. For instance, among 126 patients (19%) without ASDAS at baseline, SJC (52%), PGA (44%) and BASDAI (35%) were all similarly used without a clear preference. Of note, CRP was available for most of these 126 patients (87%).
Conclusion. Portuguese rheumatologists adhere to the ASAS-EULAR recommendation of using ASDAS. Failing to use ASDAS does not seem to be explained by missing CRP or a preference for another disease activity score, but rather by the rheumatologist's willingness to use measurement instruments in general.

P95: Table I. ASDAS evaluation after the start of the 1st bDMARD at T0 and in ≥1 of the 2 follow-up visits.

		ASDAS evaluation at T1 and/or T2		
		Yes	**No**	**Total**
ASDAS evaluation at T0	Yes	**493 (74)**	47 (7)	540 (81)
	No	106 (16)	20 (3)	126 (19)
	Total	599 (90)	67 (10)	666

Values are n (%). The denominator is the total number of patients (n=666) in all cells.

P96

ASDAS RESPONSES IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS STARTING BDMARDS: RESULTS FROM A MULTICENTRE PROSPECTIVE COHORT

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Collaborators. Mariana Diz Lopes; Inês Santos; Bárbara Lobão; Joana Borges; Alice Castro; Sandra Sousa; Cláudia Oliveira; Susana P Silva; Ana Rita Fonseca; Diana Gonçalves; Ana Margarida Correia; Paulo Pereira; Marcelo Neto; Mariana Luís; Maria Pontes Ferreira; Anita Cunha; Maura Couto; Vítor Teixeira; Ana Lúcia Fernandes; Patrícia Nero; Filipe Barcelos; Daniel Melim; João Madruga Dias; Jorge Cantante Garcia; Cristina Catita; Tiago Meirinhos; Marlene Sousa; Susana Fernandes; Filipe Cunha Santos; Sara Dinis; Filipe C. Araújo; Tiago Costa; Sandra Falcão; Filipe Barcelos; Cândida Silva; José Tavares-Costa; Margarida Cruz; Filipa Ramos; Ana Valido; Marília Rodrigues; Margarida Mateus; Carlos Vaz; José Marona; Sandra Sousa; Filipa Farinha; Graça Sequeira; Joana Dinis; Elsa Sousa; Pedro Ávila Ribeiro

Background. ASAS and EULAR recommend the use of an improvement ≥1.1 in ASDAS at 12 weeks to determine the continuation of a bDMARD. However, it is debated whether improvements can occur and whether patients' characteristics influence (time to) response.
Objectives. To assess the likelihood of fulfilling the ASAS-EULAR criteria for treatment continuation at 3 and 6 months after the start of bDMARDs and whether there are patient characteristics that influence this response.
Methods. Patients with axSpA from the Reuma.PT registry who started the first bDMARD (2011-2022) were included. Complete data on ASDAS at T0 (baseline visit at bDMARD starting), T1 (3 months) and T2 (6 months) were required. Response rates (ΔASDAS≥ 1.1 compared with baseline) at T1 and T2 were determined. Patient characteristics (e.g., age, sex) were compared across four groups: no response in both visits, response only at T1, response only at T2, and response in both visits.
Results. In total, 336 patients [male: 56%; mean age: 43 (SD 12)] were included. After 3 months, 199 (58%) patients fulfilled the criteria of treatment continuation (Table I). This number was similar at 6 months (n=207; 60%). Thirty-six percent of patients never responded, while 50% responded in both visits. The four groups were comparable except for sex and age. Compared with those who never responded, patients who responded at both visits were more often male (65% vs 45%) and somewhat younger (mean age: 42 vs 45). Analysis across subgroups showed that young males had the highest likelihood of response, while females had lower response rates regardless of age (Table II).
Conclusion. The likelihood of delayed response (>3 months) is low, which should prompt questioning whether it is justified to wait for 6 months to decide on the continuation of bDMARDs. Young male patients are more likely to respond than female patients.

P96: Table I. ASDAS response between T0 and each follow-up visit among patients with ASDAS available in all 3 visits.

		ASDAS response T0 → T2		
		Yes	**No**	**TOTAL**
ASDAS response T0 → T1	Yes	171 (50)	28 (8)	199 (58)
	No	36 (10)	111 (32)	147 (42)
	Total	207 (60)	139 (40)	346 (100)

Values are n (%). The denominator is the total number of patients (n=346) in all cells.

P96: Table II. ASDAS response between T0 and each follow-up visit, stratified on age and gender.

	All patients (n=346)	**Male <43 yo** (n=104)	**Male ≥43 yo** (n=88)	**Female <43 yo** (n=76)	**Female ≥43 yo** (n=78)
T0 → T1, n (%)	199 (58)	73 (70)	49 (56)	39 (51)	38 (49)
T0 → T2, n (%)	207 (60)	80 (77)	51 (58)	41 (54)	35 (45)

Age is a binary variable categorized according to the mean at baseline: age <43 yo (years old) vs age ≥ 43 yo.

P97

ACCESS TO ADVANCED THERAPIES IN AXIAL SPONDYLOARTHRITIS IN LATIN AMERICA, DATA FROM THE PANLAR-ESPALDA REGISTRY

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Introduction. Access to advanced treatments in LATAM poses challenges due to various socioeconomic factors. The PANLAR-ESPALDA registry was established with the objective of gathering data specific to our region. The primary aim of this study is to describe the frequency of advanced therapy in axSpA and analyze the characteristics of the patients receiving such treatments.
Methods. We included consecutive patients aged ≥18 years with axSpA (ASAS 2009) from medical centers in Argentina, Uruguay, Chile, Venezuela, Mexico, Colombia, and Ecuador. Recorded data encompassed demographic information, age at symptom onset, disease duration, disease-related symptoms, and comorbidities. Clinical and therapeutic aspects of the disease were documented at baseline, and specific questionnaires ASDAS/BASDAI/BASFI were administered. Additionally, we recorded erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) in mg/dl, HLA-B27, x-rays, and, MRI of the sacroiliacs.
Results. A total of 200 patients were recruited, with a mean age of 46 years, and 54.5% were male (cohort characteristics were previously presented). Of the total, 49% (95% CI: 42-56) were on advanced therapy (following NSAID failure) at the baseline visit: TNFi: 80%, L17i: 15%, and Jaki: 5%. Ten percent of patients were in their second line of treatment. The characteristics of patients under advanced treatment (only the significant ones and disease activity) are detailed in table 1. More than 50% of patients without advanced therapies still presented disease activity (BASDAI >4). In multivariate analysis, the characteristics independently associated with the use of advanced therapy were smoking (OR 2.8, 95% CI: 0.2-6.5) and HLA-B27 positivity (OR 4, 95% CI: 1.7-9).
Conclusion. In our region, the frequency of advanced therapy use in axSpA is 50%. Patients under these treatments tend to exhibit a more "typical" disease profile, including radiographic evidence, HLA-B27 and peripheral manifestations. Even with disease activity, a high subset of patients remains without these advanced therapies.

P97: Table I.

Features	Advanced Therapy YES	Advanced Therapy NO	p
Smoke %	40	20	0.005
HLA-B27+ %	67	48	0.009
X-ray + (NY)%	70	54	0.03
Sacroiliac maneuvers %	62	42	0.02
BASDAI, mean (SD)	4.3 (1.9)	3.8 (1.9)	0.1
Presence of arthritis %	28	13	0.01
Presence of enthesitis %	42	28	0.04
ASDAS, mean (SD)	2.2 (1)	2.4 (1)	0,1
BASDAI >4%	62	53	0.2

P98

EFFICACY OF BIMEKIZUMAB IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS WITH SHORTER VERSUS LONGER SYMPTOM DURATION: 1-YEAR RESULTS FROM TWO PHASE 3 STUDIES

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Introduction. We compare the effect of duration of symptoms (DoS) on bimekizumab (BKZ) efficacy in patients with axial spondyloarthritis (axSpA) in the phase 3 BE MOBILE 1 and 2 trials.
Methods. In BE MOBILE 1 (non-radiographic axSpA; NCT03928704) and 2 (radiographic axSpA; NCT03928743) patients were randomised to BKZ or placebo; all received BKZ from Week 16–52. We analysed ASAS40 (non-responder imputation), ASDAS <2.1, mean BASDAI change from baseline (CfB; multiple imputation), and mean MRI-SIJ SPARCC inflammation score (observed case) for patients with DoS ≤5/>5 years and ≤2/>2 years (BE MOBILE 1 only due to sample size), to Week 52. Relative odds ratios/relative differences were calculated at Week 16, sample size permitting.
Results. Improvements were seen with BKZ versus placebo at Week 16, regardless of DoS. These outcomes were sustained or improved in all subgroups to Week 52. Whilst at Week 16 ASAS40 and ASDAS <2.1 responses were numerically higher in BKZ-treated patients with DoS ≤5, relative odds ratios indicated no significant differences between DoS ≤5/>5 (Fig. 1) or ≤2/>2 (BE MOBILE 1; Fig. 2). Relative difference in Week 16 BASDAI CfB showed no significant difference between DoS ≤5/>5 or ≤2/>2 in BE MOBILE 1, but greater improvement in patients with DoS ≤5 in BE MOBILE 2 (Figs. 1-2). Baseline MRI-SIJ SPARCC scores indicated more inflammation in patients with DoS ≤5 than >5. BKZ reduced MRI-SIJ SPARCC scores in both subgroups at Week 16, and versus placebo there was no significant difference between DoS ≤5/>5 in BE MOBILE 1 (relative difference: –3.10). MRI-SIJ SPARCC scores of BKZ-treated patients at Week 16 remained low at Week 52 regardless of DoS.
Conclusion. There was no significant difference in short-term outcomes with BKZ treatment between patients with axSpA with shorter versus longer DoS, with a tendency for better response with shorter DoS.

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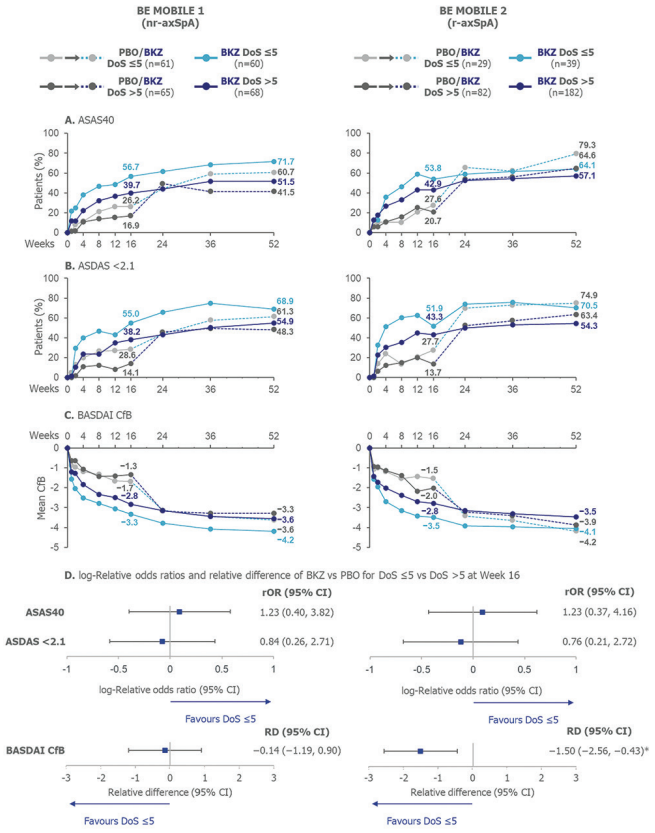
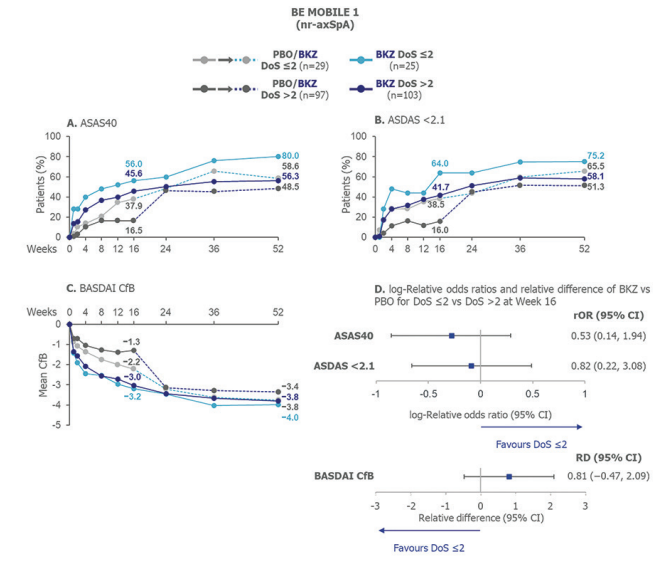


P98: Fig. 1. ASAS40 (NRI), ASDAS <2.1 (MI) and mean BASDAI CfB (MI) stratified by duration of symptoms ≤5 and >5 years.
Randomized set. Patients were randomized to receive subcutaneous BKZ 160 mg Q4W or placebo to Week 16; all patients received BKZ 160 mg Q4W from Week 16 to Week 52. ASAS40 reported with NRI; ASDAS <2.1 and BASDAI CfB reported with MI. *Greater improvements in BASDAI achieve in patients with DoS ≤5 than >5 in BE MOBILE 2. ASAS40: Assessment of Spondyloarthritis International Society 40% Improvement; ASDAS: Axial Spondyloarthritis Disease Activity Score; axSpA: axial spondyloarthritis; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BKZ: bimekizumab; CfB: change from baseline; CI: confidence interval; DoS: duration of symptoms; nr-axSpA: non-radiographic axSpA; NRI: non responder imputation; MI: multiple imputation; PBO: placebo; Q4W: every 4 weeks; r-axSpA: radiographic axSpA; RD: relative difference; rQR: relative odds ratio.



P98: Fig. 2. ASAS40 (NRI), ASDAS <2.1 (MI) and mean BASDAI CfB (MI) stratified by duration of symptoms ≤2 and >2 years.
Randomized set. Patients were randomized to receive subcutaneous BKZ 160 mg Q4W or placebo to Week 16; all patients received BKZ 160 mg Q4W from Week 16 to Week 52. ASAS40 reported with NRI; ASDAS <2.1 and BASDAI CfB reported with MI. ASAS40: Assessment of Spondyloarthritis International Society 40% Improvement; ASDAS: Ankilosing Spondylitis Disease Activity Score; axSpA: axial spondyloarthritis; BASDAI: Bath Ankylosing Spondylitis Diseases Activity Index; BKZ: bimekizumab; CfB: change from baseline; CI: confidence interval; DoS: duration of symptoms; nr-axSpA: non-radiographic axSpA; NRI: non responder imputation; MI: multiple imputation; PBO: placebo; r-axSpA: radiographic axSpA; Q4W: every 4 weeks; RD: relative difference; rQR: relative odds ratio.

P99

BASDAI AND ASDAS DISEASE STATES IN RELATIONSHIP TO ASAS40 RESPONSE: POST HOC ANALYSIS FROM THE IXEKIZUMAB COAST-V STUDY IN RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS

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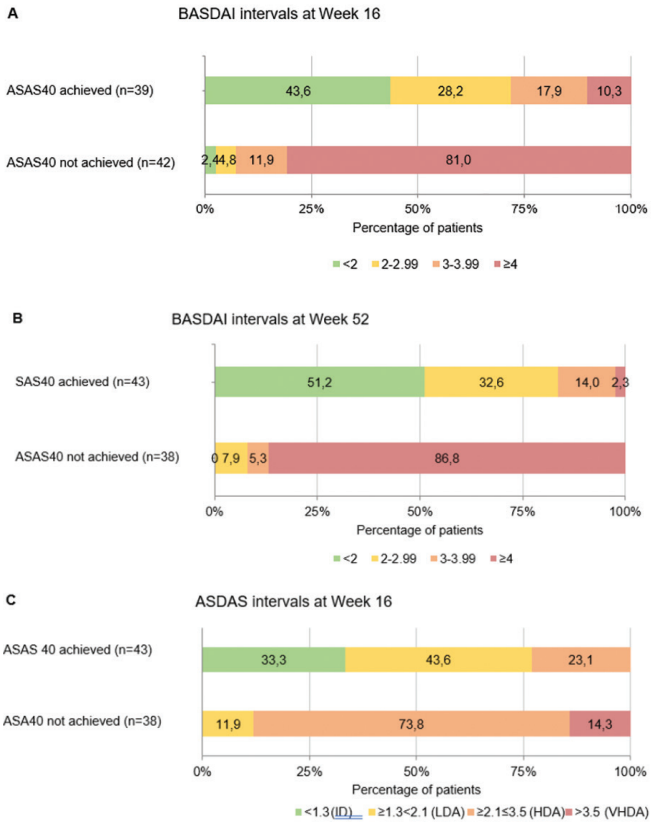
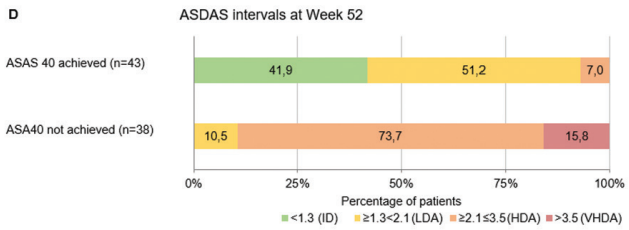
Introduction/Objective. To explore the relationship between Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) and Ankylosing Disease Activity Score (ASDAS) used in clinical practice and the Assessment of SpondyloArthritis international Society 40% (ASAS40) response, the primary endpoint in clinical trials in axial spondyloarthritis (axSpA).
Materials and methods. Data from COAST-V, a phase 3 trial of ixekizumab versus placebo in biologic-naïve radiographic axSpA (r-axSpA) patients, were analysed. Patients treated with ixekizumab every 4 weeks were categorized using ASAS40 response at week 16 (48.1%; 39/81) and 52 (53.1%; 43/81). The association between BASDAI and ASDAS disease states, respectively and ASAS40 response achieved/not achieved was investigated. Additionally, back pain, fatigue, Bath Ankylosing Spondylitis Functional Index, ASAS Health Index, and 36-item Short Form Health Survey Physical Component Summary scores corresponding to these states were assessed. Results were reported descriptively.
Results. After 16 weeks, 71.8% (n=28) and 43.6% (n=17) patients achieved BASDAI<3 and BASDAI<2, respectively; 76.9% (n=30) and 33.3% (n=13) attained ASDAS<2.1 and ASDAS<1.3, respectively among those who achieved an ASAS40 response. At week 52, 83.8% (n=36) and 51.2% (n=22) patients achieved BASDAI<3 and BASDAI<2, respectively; 93.1% (n=40) and 41.9% (n=18) attained ASDAS<2.1 and ASDAS<1.3, respectively among ASAS40 responders (Fig. 1). Lower BASDAI and ASDAS disease states corresponded well with less back pain, fatigue, and functioning impairment, and better health-related quality of life (Table I).





P99: Fig. 1. BASDAI (A and B) and ASDAS (C and D) intervals in relationship to ASAS40 achievement.
Stacked bar graphs showing BASDAI and ASDAS intervals in patients who achieved/not achieved an ASAS40 response at Week 16 (A and C) and Week 52 (B and D) in the COAST-V trial.
ASAS40: Assessment in SpondyloArthritis international Society 40% response; ASDAS: Ankylosing Spondylitis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; HAD: high disease activity; ID: inactive disease; LDA: low disease activity; VHDA: very high disease activity.

P99: Table I. PROs, BASFI, ASAS-HI and SF-36 PCS scores by (**A**) BASDAI and (**B**) ASDAS intervals.

A BASDAI intervals (n=78)

Week 16

	BASDAI<2 (n=18)	BASDAI 2–2.99 (n=13)	BASDAI 3–3.99 (n=12)	BASDAI≥4 (n=35)
Back pain	1.2	2.6	4.2	5.7
Fatigue	1.8	3.1	4.4	6.1
BASFI	1.2	3.1	3.2	5.3
ASAS HI	2.1	4.4	5.3	7.0
SF-36 PCS	51.2	43.7	45.0	39.4

Week 52

	BASDAI<2 (n=22)	BASDAI 2–2.99 (n=17)	BASDAI 3–3.99 (n=8)	BASDAI≥4 (n=31)
Back pain	1.3	2.9	3.4	6.1
Fatigue	1.6	3.0	4.5	6.1
BASFI	1.0	2.4	3.3	5.0
ASAS HI	2.2	4.1	4.6	6.9
SF-36 PCS	51.7	46.7	47.3	39.6

B ASDAS intervals (n=81)

Week 16

	ASDAS<1.3 (n=13)	ASDAS≥1.3 to <2.1 (n=22)	ASDAS≥2.1 to ≤3.5 (n=40)	ASDAS>3.5 (n=6)
Back pain	1.1	2.8	5.1	7.2
Fatigue	2.0	3.4	5.3	7.5
BASFI	1.2	2.4	4.7	7.1
ASAS HI	1.8	4.4	6.3	8.7
SF-36 PCS	51.7	46.6	40.3	36.1

Week 52

	ASDAS<1.3 (n=18)	ASDAS≥1.3 to <2.1 (n=26)	ASDAS≥2.1 to ≤3.5 (n=31)	ASDAS>3.5 (n=6)
Back pain	1.1	2.8	5.6	7.2
Fatigue	1.8	3.2	5.6	7.2
BASFI	0.8	2.4	4.7	5.6
ASAS HI	2.5	3.6	6.5	8.7
SF-36 PCS	52.4	47.6	39.7	34.9

ASAS HI: Assessment in SpondyloArthritis international Society Health Index; ASDAS: Ankylosing Spondylitis Disease Activity Score; Back pain: total back pain (BASDAI question 2); BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index; Fatigue: Fatigue Severity Numeric Rating Scale; SF-36 PCS: 36-Item Short Form Health Survey Physical Component Score; PRO: patient-reported outcomes.

Conclusion. When analysing the common clinical practice tools BASDAI and ASDAS, low disease activity or inactive disease were attained in more than 70% of biologic-naïve r-axSpA patients who achieved an ASAS40 response in a randomized clinical trial. This data may further help to translate results from clinical trials into daily practice.

P100

EXPERIENCES OF PATIENTS AND HEALTHCARE PROVIDERS WITH PATIENT-INITIATED CARE AND ASYNCHRONOUS TELEMONITORING IN SPONDYLOARTHRITIS: A MIXED METHODS STUDY

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Introduction. A potential new strategy for the follow-up of patients with spondyloarthritis (SpA) is patient-initiated follow-up (PIFU) combined with asynchronous telemonitoring (TM). In the TeleSpA randomised controlled trial, we demonstrated that PIFU/TM led to meaningful reductions in consultation frequency in stable SpA (1). Concurrently, this study evaluated the experiences of SpA patients and their healthcare providers (HCPs) with PIFU/TM.
Methods. Individual, semi-structured interviews were conducted with patients and HCPs who participated in the PIFU/TM arm of TeleSpA (analysed thematically). All HCPs that participated in PIFU/TM were additionally invited to complete a survey (analysed descriptively).
Results. Twenty-one patients and nine HCPs were interviewed (Table I), and 13 HCPs completed the survey. Six themes were identified in the interviews (Fig. A), which the survey results (Fig. B) reinforced:
Past experiences with usual care: Participants found (frequent) consultations in stable SpA unnecessary. HCPs found the traditional role of patients to be overly passive.
Acceptability: The majority found PIFU/TM acceptable and safe for motivated patients with stable disease and sufficient digital and health literacy.
Responsibility: Most patients felt no burdensome changes to their personal responsibilities.
Time investment: Significant time-savings were unapparent, however, HCPs did occasionally gain flexibility in their daily planning.
Communication and collaboration: All found that communication between patients and HCPs ran smoothly, and felt that the longitudinal data resulting from the regular completion of TM questionnaires facilitated effective shared decision-making.
Future implementation: All participants indicated willingness to continue PIFU/TM. Pre-requisites for PIFU/TM were the ability to rapidly plan in-person appointments when indicated, availability of a digital tool integrated with existing hospital systems, and accessibility to technical and logistic support.
The length and repetitiveness of the questionnaires was an area for improvement.
Conclusion. PIFU/TM was perceived as an acceptable follow-up approach of motivated patients with stable SpA possessing adequate digital and health literacy skills.

P100: Table I. Characteristics of the interviewed participants

Patients (n=21) **Variable**	**Value**
Female sex, n (%)	8 (38.1)
Age, years	61.5 (10.0)
Higher education/university degree attained, n (%)	11 (52.4)
Diagnosis, n (%)	
Axial spondyloarthritis	9 (42.9)
Peripheral spondyloarthritis	10 (47.6)
Combined axial and peripheral spondyloarthritis	2 (9.5)
Symptom duration, years	19.8 (12.6)
Current maintenance therapy, n (%)	
None/NSAID	7 (33.3)
Conventional synthetic DMARD	3 (14.3)
Biological DMARD	11 (52.4)
ASDAS at 12-month follow-up of TeleSpA	1.9 (0.9)
Healthcare providers (n=9)	
Age, years	47.2 (11.5)
Position	
Rheumatologist	6 (66.7)
Rheumatology nurse	3 (33.3)
Work experience, years	14.5 (9.0)

All values presented as mean (standard deviation), unless otherwise indicated.
ASDAS: Axial Spondyloarthritis Disease Activity Score; DMARD: disease-modifying anti-rheumatic drug; NSAID: non-steroidal anti-inflammatory drug.

Acknowledgements. The researchers thank all patients and healthcare providers who participated in the study. This work was supported by a grant from the Dutch Arthritis Foundation, project number 19-2-203.

Reference

1. HERMANS K, WEBERS C, BOONEN A *et al.*: OP0120 Effectiveness and cost-effectiveness of patient-initiated follow-up supported by asynchronous telemonitoring in spondyloarthritis (TeleSpA-study): A pragmatic multicentre randomised controlled trial. [Abstract accepted for the EULAR 2024 Congress]

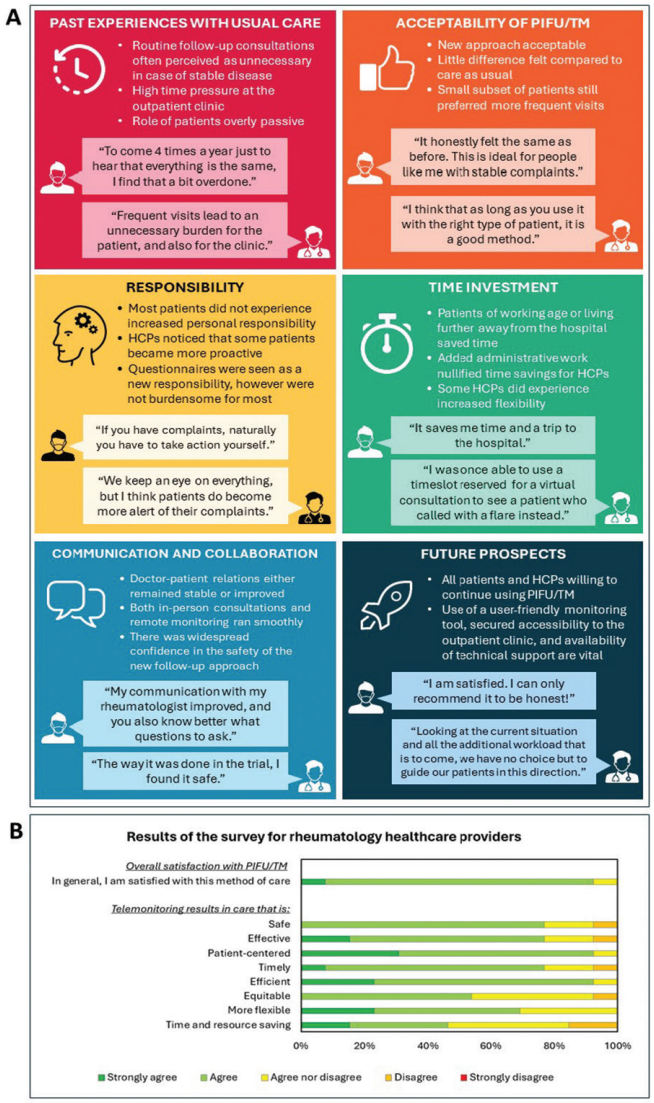

Fig. 1. Exploration of the experiences of participants with PIFU/YM:
A: Overviev of yhe themes identified during the interviews with illustrative quotes,
B: Results of the survey for HCPs.
HCP: healthcare provider; PIFU: patient-initiated follow-up; TM: telemonitoring .

P101

BARRIERS AND FACILITATORS TO TREAT-TO-TARGET IN AXIAL SPONDYLOARTHRITIS IN PRACTICE: A MIXED METHODS STUDY

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Introduction. Treat-to-target (T2T) is a recommended management approach for axial spondyloarthritis (axSpA) (1). In practice, however, compliance with T2T remains limited in axSpA for unknown reasons (2). This study aimed to explore the barriers and facilitators of T2T implementation.
Methods. Mixed-methods design using semi-structured interviews (analysed thematically) and quantitative surveys (analysed descriptively) in parallel. Patients with axSpA visiting the outpatient clinic with an AxSpA Disease Activity Score (ASDAS) ≥2.1 for whom medication was not adapted (against T2T recommendations) were included, alongside their treating rheumatologists.
Results. Sixteen patients and 11 rheumatologists participated (Table I). The figure summarises the identified barriers and facilitators during the interviews (A), and from the survey (B-C). Facilitators highlighted were the broad patient knowledge on axSpA, familiarity of rheumatologists with T2T, and the presence of positive doctor-patient relationships. Furthermore, rheumatologists valued regular disease activity monitoring supported by consistent instruments. A barrier reflected by patients, however, was difficulty in accurately completing disease activity questionnaires, which sometimes led to unrepresentative scores. Additionally, some resisted treatment intensification due to satisfaction with existing therapies (85%), fear (33%), or the view of axSpA flares being temporary (82%). Finally, knowledge on treatment goals was sparse. Many rheumatologists doubted the effectiveness (73%), feasibility (73%) and flexibility (64%) of T2T in this specific axSpA population, for example due to limited supporting evidence (91%), leading to low motivation to apply T2T. Furthermore, while (partially) subjective instruments (*e.g.* ASDAS) were valued supporting tools, many found that these provided insufficient insight (73%). Rheumatologists therefore often relied on their own judgement, which sometimes contradicted the ASDAS. Lastly, restrictions in time (27%) and available resources (45%) were highlighted barriers.
Conclusion. While the foundation for implementing T2T in axSpA in practice is present, numerous barriers are evident. Patient education and evaluation of the definition of active disease are future research opportunities.

P101: Table I. Characteristics of participants.

Patients (n=16)	
Variable	**Value**
Female sex, n (%)	8 (50.0)
Age, years	58.6 (14.0)
Type of healthcare institution, n (%)	
University hospital	10 (62.5)
General hospital	6 (37.5)
Symptom duration, years	29.7 (17.7)
ASDAS at the last consultation	3.0 (0.5)
Rheumatologists (n=11)	
Female sex, n (%)	9 (81.8)
Age, years	49.6 (8.2)
Work experience, years	16.7 (10.6)

All values presented as mean (standard deviation), unless otherwise indicated.
Abbreviations: ASDAS (Axial Spondyloarthritis Disease Activity Score)

Acknowledgements. The authors thank all patients and rheumatologists who participated in this study, and the research nurses in the Maastricht UMC+, Janine Geusen and Christel Mertens, for their efforts in facilitating the logistics of the interviews. This investigator-initiated study was financially supported by Novartis. Novartis had no role in the study design, in the collection, analysis or interpretation of the data, or in the writing of this abstract.

References

1. SMOLEN JS, SCHÖLS M, BRAUN J *et al.*: Treating axial spondyloarthritis and peripheral spondyloarthritis, especially psoriatic arthritis, to target: 2017 update of recommendations by an international task force. *Ann Rheum Dis* 2018; 77(1): 3-17.
2. BECKERS E, BOONEN A, WEBERS C *et al.*: Treat-to-target in axial spondyloarthritis: an observational study in daily practice. *Rheumatology* (Oxford) 2022; 61(4): 1396-407.

P101: Fig. 1. Barriers and facilitators to T2T in axSpA. **A**: Overview of the barriers and facilitators identified during the interviews with illustrative quotes, **B**: Results of the survey for patients (n=14), **C**: Results of the survey for rheumatologists (n=11).

P102

PERSISTENCE OF SEQUENCING GUSELKUMAB OR TNFI AS A SECOND-LINE THERAPY FOR THE TREATMENT OF ACTIVE PSORIATIC ARTHRITIS IN REAL WORLD PRACTICE IN SPAIN - MANHATTAN STUDY

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Introduction. The Manhattan study aims to evaluate the effectiveness of guselkumab (GUS) or a second-line Tumor Necrosis Factor inhibitor (TNFi) after receiving a first-line TNFi treatment in patients with Psoriatic Arthritis (PsA).

Methods. Manhattan (CNTO1959PSA4009) is an ongoing ambispective, observational cohort study. The study is evaluating effectiveness, persistence and tolerability of second-line GUS or TNFi patients after receiving a first-line TNFi treatment in 34 hospitals in Spain. An interim analysis was performed 10 months after the inclusion of the first patient. Sixty-nine participants (GUS n=36, TNFi n=33) had data available at week 12 and 39 (GUS n=25, TNFi n=14) at week 24.

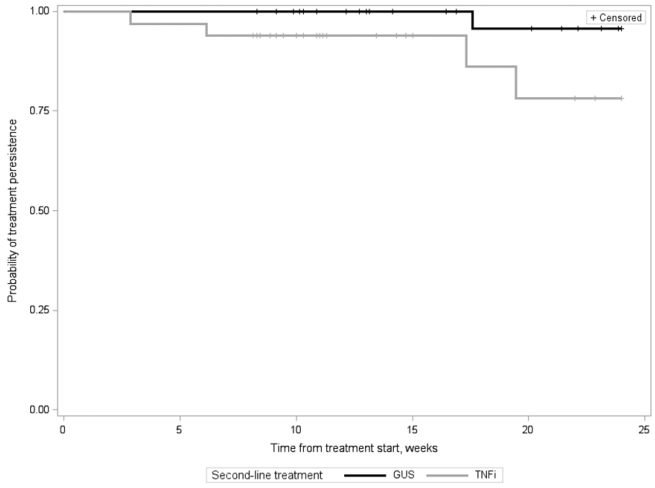
Results. Baseline characteristics are described in Table I. Among the 69 patients included the most common firs-line TNFi used was adalimumab. All GUS patients continued treatment at 12 weeks and 6.1% TNFi patients suspended treatment over 12 weeks. The probability of treatment persistence up to 24 weeks was 95.7% for GUS and 78.3% for TNFi patients (Fig. 1). The percentage of patients in Disease Activity Index for Psoriatic Arthritis (DAPSA) Low Disease Activity (LDA) increased over the time from baseline values in the GUS group (22.7%, 50%, 77.8% at 0, 12, 24 weeks respectively) and for TNFi group (33.3%, 65.4%, 69.3% at 0, 12, 24 weeks respectively). Similarly, the mean Body Surface Area (BSA) decreased from 5.5 % over the time by 1.7% at week 12 and 1.0% at week 24 for GUS, and from 1.1 to 0.5% at week 12, and 0.3% at week 24 for TNFi patients.

Conclusion. Among patients analyzed in this interim analysis, DAPSA LDA was reached similarly after 24 weeks of treatment with GUS and TNFi whereas then probability of persistence of second-line treatment was slightly higher in the GUS group.

102: Table I. Baseline characteristics of PsA.

	GUS second-line	TNFi second-line
Sex		
Male	16 (44.4)	18 (54.5)
Female	20 (55.6)	15 (45.5)
Age at PsA diagnosis (years)	42.8 (11.6)	42.8 (12.1)
BMI (kg/m²)	27.6 (5.1)	29.3 (7.8)
Comorbidities[1]		
Arterial hypertension	4 (21.1)	11 (52.4)
Diabetes	3 (15.8)	2 (9.5)
Heart disease	2 (10.5)	2 (9.5)
Anxiety/depression	5 (26.3)	2 (9.5)
PsA characteristics		
Polyarticular PsA	23 (63.9)	25 (78.1)
Oligoarticular PsA	11 (30.6)	7 (21.2)
Axial affectation	11 (30.6)	9 (28.1)
Active psoriasis	24 (66.7)	15 (45.5)
Nail psoriasis	14 (38.9)	12 (36.4)
Dactylitis	7 (19.4)	4 (12.5)
Enthesitis	9 (25.0)	11 (34.4)
Tender Joint Count	5.7 (6.0)	4.8 (5.5)
Swollen Joint Count	3.5 (5.7)	2.9 (4.9)
DAPSA	22.1 (14.6)	19.8 (17.0)
BSA (%)	5.5 (7.2)	1.1 (2.0)

* Data are mean (SD) or number (frequency) according to their type and distribution.
[1] Based on n=40 patients (58.0%) with any comorbidity.



102: Fig. 1. Persistence of second-line treatment up to week 24.

P103: Table I. Baseline demographic characteristics.

	n=106
Sex (male), n(%)[N]	66 (62,3)[106]
Age, mean (SD)[N]	48,1 (11,9)[106]
Nr-ax-SpA, n(%)[N]	29 (27,4)[106]
HLA-B7 (positive), n(%)[N]	78 (74%)[104]
Uveitis, n(%)[N]	17 (16)[106]
Inflamatory bowel disease n(%)[N]	1 (0,9)[106]
Psoriasis n(%)[N]	11 (10,5)[105]
BMI (kg/m^2) mean (SD)[N]	27,1 (4,4)[60]
Obesity (BMI ≥ 30), n(%)[N]	12 (20)[60]
Smoker n(%)[N]	19 (18)[105]
Arterial hypertension n(%)[N]	28 (26,4)[106]
Dyslipidemia n(%)[N]	34 (32,1)[106]
Diabetes n(%)[N]	10 (9,4)[106]
Cardiovascular events n(%)[N]	10 (9,5)[105]
Number of cDMARD, mean (SD)[N]	0,81 (9,91)[105]
Number of bDMARD, mean (SD)[N]	1,28 (1,1)[106]
ASDAS-CRP mean (SD)[N]	2,12 (1,02)[106]
BASDAI mean (SD)[N]	4,56 (2,5)[70]
Periferic arthritis involvement (n)%[N]	45 (45)[100]
DAS28-CRP mean (SD)[N]	3,02 (1,2)[42]
Dactylitis n(%)[N]	5 (4,7) [103]
Enthesitis n(%)[N]	37 (36,7)[101]

P103

REAL-WORLD EXPERIENCE OF THE EFFICACY AND SAFETY OF SECUKINUMAB DOSE INTENSIFICATION IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS

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Introduction. Secukinumab (SECU), has demonstrated efficacy in the treatment of axial spondyloarthritis (axSpA). Different doses of SECU (150mg and 300mg every 4 weeks) have been evaluated in radiographic axial SpA (r-axSpA) patients, finding both doses effective without significant differences. However, real-world data on dose escalation in axSpA patients are scare.
Aim. To assess the efficacy and safety of uptititering SECU from 150mg to 300mg in axSpA patients with inadequate/partial clinical response.
Methods. Observational, retrospective, multicenter study. Patients with axSpA receiving SECU 150mg every 4 weeks, uptitered to 300mg due to inadequate response, were included. Data on demographics, baseline activity index and enthesitis/dactylitis counts were collected at various intervals post-escalation.
Results. 106 patients were included. Baseline characteristics are shown in Table I. At baseline, only 2 were on SECU monotherapy, and from month 6 onwards, all were on combination therapy with Methotrexate 80.7%, Leflunomide 3.7%, or Sulfasalazine 7%; these percentages varied during follow-up to 80.7%, 3.5%, and 8.8% at month 12, and 68.8%, 9.4%, and 18.8% at month 24, respectively. Changes in activity indices over time are shown in Table II. During follow-up, 15 adverse events occurred, including 3 severe cases (acute myocardial infarction, hydrocele, and tuberculosis reactivation) and 11 mild cases. SECU was permanently discontinued in one case due to urticaria.
Conclusion. SECU uptitration led to modest improvements in activity indices. However: In ASDAS-CRP showed no significant change, BASDAI reached indices below 4 by month 12, and DAS28 achieved remission. A higher percentage of patients had 0 enthesitis. The data suggest that SECU uptitration is effective targeting extra-axial involvement. Notably, three severe adverse events required hospitalization, and one case led to SECU discontinuation due to urticaria.

P104

IMPACT OF TREATMENTS ON FATIGUE IN AXIAL SPONDYLOARTHRITIS: SYSTEMATIC REVIEW AND METAANALYSIS

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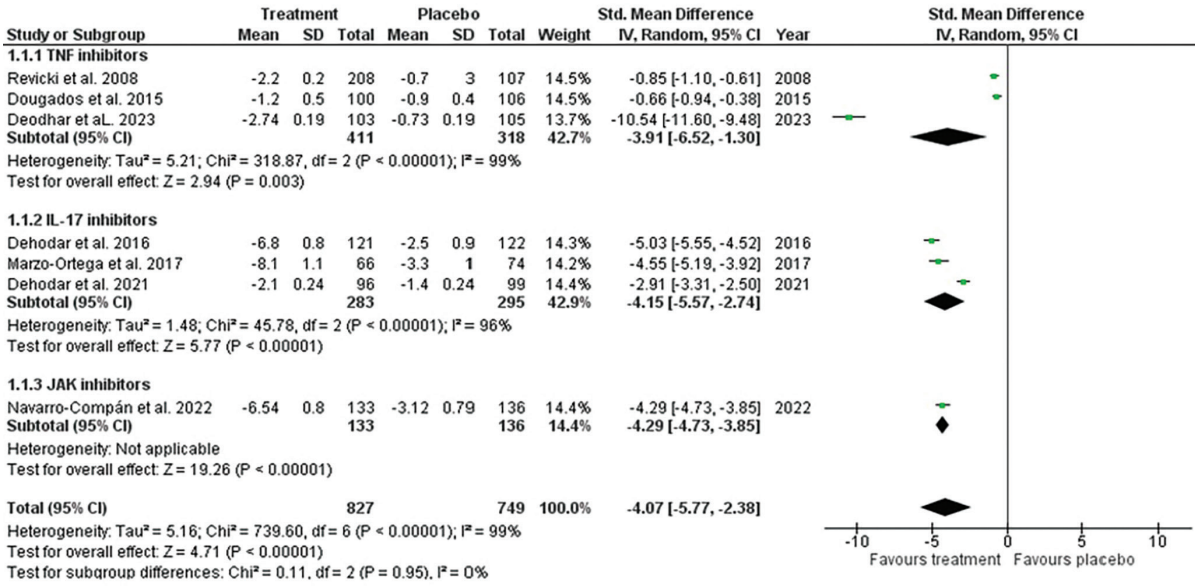
Background. Fatigue is frequent problem in axial spondyloarthritis (axSpA) and represent a difficult outcome. The aim of this systematic review was to assess the effect of treatment of axSpA on fatigue.
Methods. A systematic review was performed by two independent reviewers following PRISMA recommendations on Pubmed, Cochrane, Embase databases. We included controlled interventional studies, cohort studies conducted in patients with axial spondyloarthritis meeting ASAS 2009 criteria and measuring fatigue between 12-156 weeks. We excluded studies not written in English, case reports, abstracts, systematic reviews, metanalysis and studies with missing data. A metanalysis was performed for anti-TNF/anti-IL17/jak-inhibitors randomized controlled trials evaluating fatigue at week 12-16. Given the multiplicity of scores used as primary endpoints in the selected

P103: Table II. Changes in Activity Index.

Time point	ASDAS-CRP Mean (SD)[N]	BASDAI Mean (SD)[N]	NSJ mean (SD)[N]]	NTJ mean (SD)[N]	DAS28-CRP Mean (SD)[N]	n(%) of 0 dactylitis [N]	n(%) of 0 enthesitis [N]
Baseline (n=106)	2,12(1,02)[106]	4,56(2,5)[70]	0,78(1,7)[42]	2,3(2,9)[42]	3,02(1,2)[42]	98(92,5) [103]	63(62,4) [101]
3m (n=61)	1,83(0,9)[61]	4,87(2,4)[36]	0,44(1,3)[18]	2,8(1,6)[18]	1,96(0,89)[18]	58(100) [58]	41(71,9) [57]
6m (n=57)	1,86(1,1)[57]	4,3(2,6)[31]	0,12(0,5)[41]	2,15(4,7)[41]	2,36(0,84)[41]	52(91,2) [55]	34(63) [54]
12m (n=57)	1,68(0,9)[57]	3,73(2,3)[36]	0,14(0,5)[36]	2,48(1,1)[36]	2,32(1,1)[36]	54(94,7) [57]	39(69,6) [56]
18m (n=33)	1,76(1)[33]	4,32(2)[19]	0,35(0.9)[26]	1(2)[26]	2,47(0,8)[26]	33(100) [33]	24(72,7) [33]
24m (n=32)	1,57(1,1)[32]	3,78(2,4)[21]	0,27(0,6)[15]	2,87(6,4)[15]	2,75(1,1)[15	30(93,8) [31]	26(86,7) [30]



104: Fig. 1. Metanalysis at weeks 12-16 including 7 studies.

studies, a meta-analysis pooling standardized mean differences was carried out for all controlled studies with data available at baseline and 12-16 weeks, using a random-effects model.

Results. A total of 1670 studies were identified and 34 were selected for analysis. Twelve studies evaluated anti-TNF with a significant reduction in fatigue measured by various scores (FACIT, MFI, NRS, VAS, FSS) in 11 studies. Among the 4 studies evaluating anti IL17, 3 showed a reduction in fatigue, with a dose effect for Secukinumab. For JAK inhibitors, 2 studies showed a reduction in fatigue. The meta-analysis showed no differences between DMARDs (Fig. 1) after 12-16 weeks of treatment. Concerning non-pharmacological treatments, 11 out of 16 studies showed a reduction in fatigue using physical activity, cryotherapy and magnetotherapy. Two studies have shown that the addition of physical activity to anti-TNF reduced fatigue more significantly. Finally, 1 study showed a greater efficacy on fatigue in men and two suggest it.

Conclusion. This review shows a beneficial effects of DMARDs and non-pharmacological treatment on fatigue in axSpA in short and medium term with a greater effect when combining them.

P105

IMPACT OF ANTI TNF AND ANTI IL 17 ON THE FIB-4 SCORE AFTER 2 YEARS OF TREATMENT IN SPONDYLOARTHRITIS

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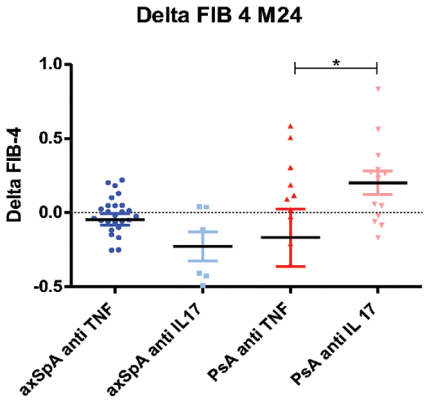
Introduction. To evaluate the risk of liver fibrosis under a treatment with secukinumab or anti TNF in psoriatic arthritis (PSA) and axial SpA (axSpA) after two years of treatment.

Methods. A monocentric, retrospective longitudinal study including PsA patients and axSpA treated with secukinumab or anti TNF between 2015 and 2020 was performed. The risk of liver fibrosis was estimated by using the FIB-4 (fibrosis-4). The FIB-4 index was calculated using the following formula: (age (years) × AST(U/L)/platelet (PLT) (109/L) × √ALT(U/L)). To avoid the weight of age, we compared the variations in the FIB-4 score during the time.

Results. 78 patients (45 with axSpA and 33 with PsA) were included. Four patients (8%) with axSpA and nine patients (27%) with PsA had histologically proven NAFLD. In PsA, the mean FIB-4 score at baseline was (1.18±0.92). In patients treated with anti-TNF (n=13) the FIB 4 score decreased (-0.17±0.7) after 2 years of treatment. Patients treated with anti IL17 showed an increase of the FIB4 score (0.2±0.3) (p<0.05 compared with anti TNF) after 2 years of treatment. In axSpA, the mean FIB-4 value at baseline was (0.76±0.2). Patients treated with anti TNF showed a decrease of the FIB4 score (-0.05±0.2) after 2 years of treatment. Patients treated with anti IL17 showed a slight decrease of the FIB4 score about (-0.2±0.2) (p<0.05) after 2 years of treatment. No change in fibrosis risk categories was observed in patient with NAFLD

Conclusion. In spondyloarthritis, treatment with anti-TNF and anti-IL 17 is not associated with significant modification of the FIB-4 score.



P105: Fig. 1.

P106

WHICH ASDAS CUT-OFF CORRESPONDS BEST TO TREATMENT INTENSIFICATION IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS IN DAILY PRACTICE?

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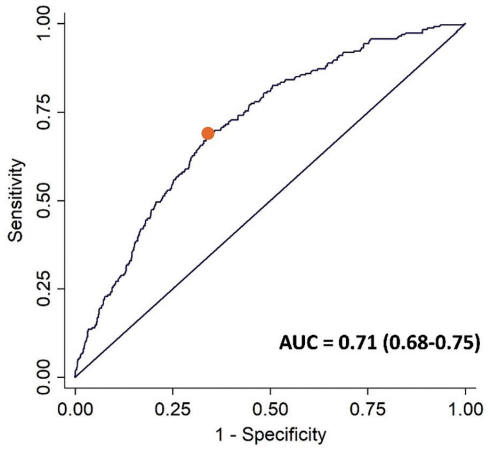
Introduction. In patients with axial spondyloarthritis (axSpA) and high disease activity (Axial Spondyloarthritis Disease Activity Score [ASDAS]≥2.1), treatment adaptation is not always adapted. Possibly, the cut-off of 2.1 as currently endorsed is too strict in clinical practice. Our objective was to investigate which ASDAS cut-off corresponds best with treatment intensification (TI) in practice.

Methods. Data were used from a prospective SpA registry (SpA-Net). Patients with axSpA and ≥1 ASDAS measurement in 2016-2022 were included. TI was defined as 1) higher dose or frequency of the same drug, 2) switch to another drug, or 3) addition of a new drug to the current treatment; due to inefficacy. Only anti-inflammatory drugs (NSAIDs, cs/b/tsDMARDs, glucocorticoids) were considered. Patients could contribute multiple observations. Receiver operating characteristic (ROC) curve analyses were conducted to estimate the ability of ASDAS to discriminate between TI/non-TI (Area Under the Curve [AUC]), and identify the ASDAS cut-off that discriminates best.
Results. In total, 350 patients with 2,265 ASDAS measurements were included. Median follow-up was 2.8 (IQR 1.0-4.4) years. Mean age was 51.0 (SD 14.5) years, 152 (43.4%) were female, and mean ASDAS was 2.3 (SD 1.0). TI was applied after 236/2,265 ASDAS measurements (10.4%), and mean ASDAS was higher at TI timepoints than at non-TI timepoints (3.0 [SD 1.0] versus 2.3 [SD 1.0]). In ROC analysis with all ASDAS measurements, the AUC was 0.71 (95%CI 0.68-0.75) with an optimal ASDAS cut-off of 2.7 (sensitivity=69%, specificity=66%) (Fig. 1). Results were similar with only one measurement per patient per calendar year. Over time, the optimal ASDAS cut-off varied substantially but was consistently higher than 2.1 (Table I).
Conclusion. In daily practice, TI is associated with a higher ASDAS than the recommended ≥2.1 cut-off. Possibly, rheumatologists believe the recommended cut-off to be too stringent or consider other factors than disease activity when making treatment decisions.
Acknowledgements. This investigator-initiated study was financially supported by UCB Biopharma SRL. UCB Biopharma SRL had no role in the study design, in the collection, analysis or interpretation of the data, or in the writing of this abstract.



P106: Fig. 1. ROC curve of ASDAS and TI.
Based on all ASDAS measuremens. The orange dot represents the optimal cut-off value (ASDAS 2.7).

P107

BARRIERS AND FACILITATORS TO APPLICATION OF TREAT-TO-TARGET MANAGEMENT IN PSORIATIC ARTHRITIS AND AXIAL SPONDYLOARTHRITIS IN PRACTICE: A SYSTEMATIC LITERATURE REVIEW

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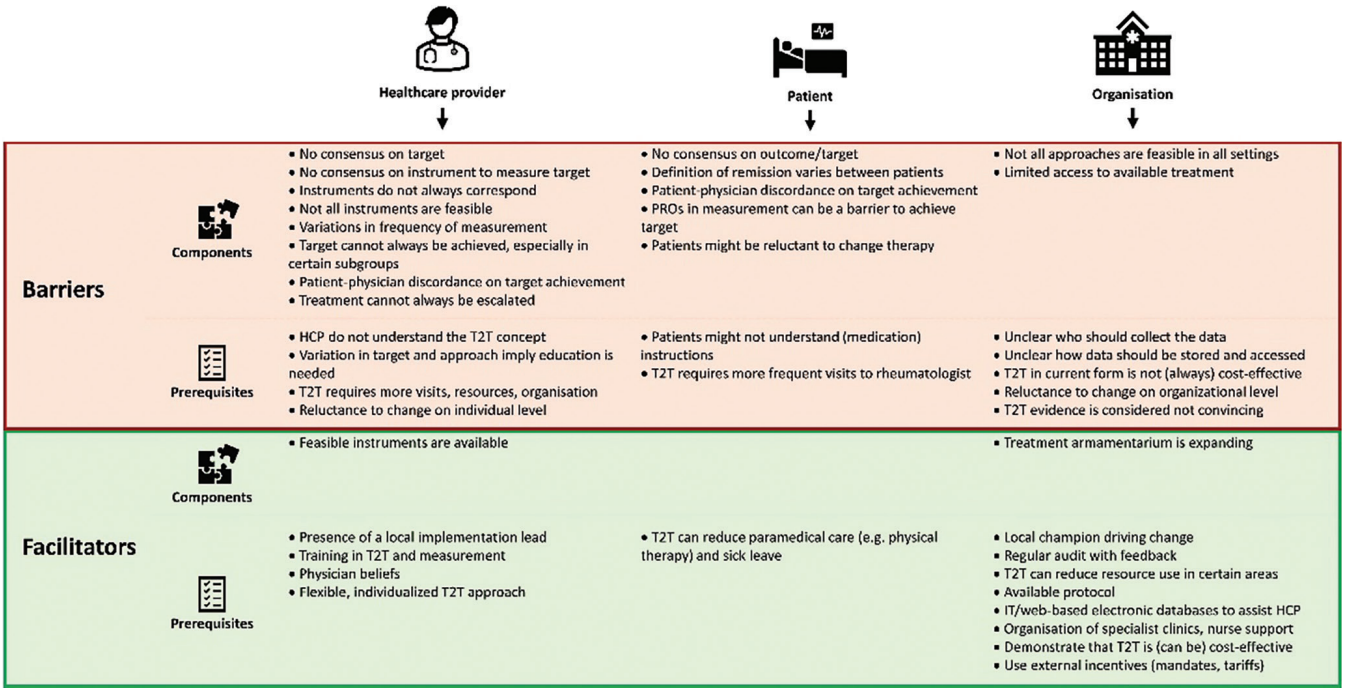
Introduction. Despite evidence and recommendations supporting treat-to-target (T2T) in spondyloarthritis, uptake has been suboptimal. Successful T2T application requires that barriers and facilitators are identified and subsequently addressed. Objective: to review the evidence on barriers and facilitators to T2T application in axial spondyloarthritis (axSpA) and psoriatic arthritis (PsA) in daily practice.
Methods. Systematic search (MEDLINE/Embase) up to December 2023. Any type of original axSpA/PsA research exploring barriers/facilitators to T2T application, either of quantitative or qualitative nature, was eligible for inclusion. In a qualitative synthesis, barriers/facilitators were classified by the level to which they apply (healthcare provider [HCP], patient, organisation), and T2T component (e.g. measurement of target, adaptation of therapy) or T2T prerequisite (e.g. resources, education) they related to.
Results. Twenty-eight studies were included. Most focused on PsA (n=21/28). Studies included patients (n=23/28), HCPs (n=4/28) or both (n=1/28). Over 25 barriers and 15 facilitators were identified (Fig. 1). At the HCP level, most studies focused on measurement of target, especially in PsA, highlighting that agreement between instruments was suboptimal. Furthermore, certain targets, such as remission, could not always be achieved. At the patient level, the role of patient-reported outcomes (PROs), while deemed relevant, was shown to act as a barrier to achieve targets that included PROs. At the organisational level, the time/resources needed for T2T were considered a barrier, although it was noted that T2T could also reduce (para)medical resource use and work losses. Other facilitators were a local champion driving change, and availability of nurse support and IT infrastructure supporting T2T. Notably, for several components, no facilitators were identified.
Conclusion. Various barriers and facilitators were identified, on several levels. Data in axSpA were scarce, as was evidence on certain T2T components. Future research should address these knowledge gaps and explore how these barriers and facilitators could be targeted to improve T2T application.
Acknowledgements. This investigator-initiated study was supported by Novartis. Novartis had no role in the study design, in the collection, analysis or interpretation of the data, or in the writing of this abstract.

P106: Table I. ROC analysis of TI and ASDAS, by calendar year (one measurement per patient per year).

	2016 (n=59)	2017 (n=186)	2018 (n=230)	2019 (n=226)	2020 (n=153)	2021 (n=161)	2022 (n=138)
AUC (95% CI)	0.87 (0.74-1.00)	0.79 (0.69-0.88)	0.75 (0.67-0.84)	0.65 (0.53-0.78)	0.66 (0.55-0.78)	0.68 (0.56-0.80)	0.82 (0.71-0.93)
Optimal cut-off*							
ASDAS	3.0	2.8	2.9	2.9	2.3	2.4	2.7
Sensitivity	0.80	0.76	0.70	0.57	0.86	0.82	0.90
Specificity	0.87	0.77	0.75	0.75	0.52	0.57	0.77

If patients had multiple ASDAS measurements in a calendar year, a random measurement was used. *According to Youden index (sum of sensitivity and specificity minus one).
AUC: area under the curve; ROC: receiver operating characteristic; TI: treatment intensification.



P107: Fig. 1. Barriers and facilitators to application of T2T in PsA/axSpA pratice.
Components of T2T include the choice of target, measurement of target frequency of measurement, achieveent of target, adaption of therapy and shared decision-making. Prerequisites of T2T include education on T2T, resources/time/process involved, support, economic consequences and other prerequisites. T2T: treat-to-target; HCP: healthcare provider; PRO: patient reported outcome.

P108

LONG-TERM SUSTAINED EFFICACY AND SAFETY OF BIMEKIZUMAB ACROSS THE FULL SPECTRUM OF AXIAL SPONDYLOARTHRITIS 2-YEAR RESULTS FROM TWO PHASE 3 STUDIES

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Introduction. We report 2-year efficacy and safety of bimekizumab (BKZ; monoclonal IgG1 antibody that selectively inhibits interleukin [IL]-17F in addition to IL-17A) in patients with non-radiographic/radiographic axial spondyloarthritis (nr-/r-axSpA) from the phase 3 BE MOBILE 1 and 2 studies, respectively (1).

Methods. In BE MOBILE 1 (NCT03928704) and 2 (NCT03928743) patients were randomised to subcutaneous BKZ 160 mg every 4 weeks (Q4W) or placebo; all received BKZ from Week 16–52, following which patients could enter the ongoing BE MOVING open-label extension (NCT04436640). Efficacy outcomes are reported for patients from BE MOBILE 1/2 and BE MOVING to 2 years (104 weeks; n=586), using non-responder imputation (NRI; binary outcomes), multiple imputation (MI; continuous outcomes, ASAS40) and observed cases (OC). Up to 2-year safety data are reported for all patients who received BKZ (n=574).

Results. 494 patients entered BE MOVING; by July 2023, 456 completed Week 104. 1-year efficacy was sustained to 2 years in nr-/r-axSpA populations (Fig. 1, Table I) (1). ASAS40 responses were maintained from Week 52–104 (nr-axSpA: 55.9%–49.2% [NRI]; 65.1%–66.1% [OC]; r-axSpA: 61.7%–53.9% [NRI]; 68.8%–67.0% [OC]). 61.2% and 63.4% of patients with nr-/r-axSpA achieved ASDAS LDA (<2.1), respectively (MI). Inflammation suppression was sustained. To Week 104, 89.5% of patients had ≥1 TEAE; the most frequent TEAEs (exposure-adjusted incidence rate/100 patient-years [EAIR/100PY]; MedDRA v19.0) were SARS-CoV-2 infection (13.2/100PY), nasopharyngitis (10.2/100PY), and upper respiratory tract infection (6.0/100PY). Serious TEAE incidence was low (5.4/100PY); no MACE, active tuberculosis, or deaths occurred. Suicidal ideation and behaviour incidence was 0.1/100PY. 122 patients had fungal infections (10.0/100PY); 76 had *Candida* infections (5.8/100PY; mostly mild–moderate, none serious/systemic). IBD (0.6/100PY) and uveitis (1.6/100PY) incidence was low.

Conclusion. Across the full axSpA spectrum, BKZ had sustained efficacy to 2 years. No new safety signals were observed (1).

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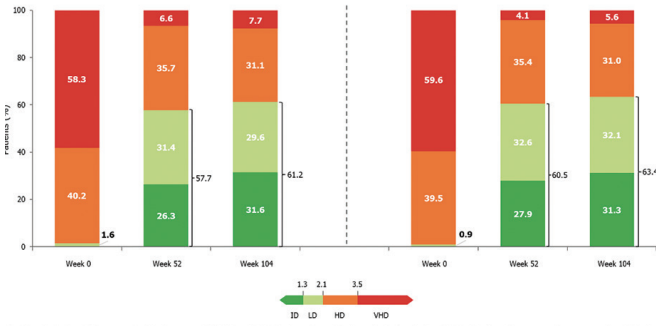
Disclosures. XB: Speaker for AbbVie, BMS, Chugai, Eli Lilly, Galapagos, MSD, Novartis, Pfizer, and UCB Pharma; paid instructor for AbbVie, BMS, Chugai, Eli Lilly, Galapagos, MSD, Novartis, Pfizer and UCB Pharma; consultant for AbbVie, BMS, Chugai, Eli Lilly, Galapagos, Gilead, Novartis, Pfizer and UCB Pharma; grant/research support from Novartis and UCB Pharma. **AD:** Speaker for Eli Lilly, Janssen, Novartis and Pfizer and UCB Pharma; consultant for AbbVie, BMS, Janssen, MoonLake, Novartis, Pfizer and UCB Pharma; grant/research support from BMS, Celgene, Eli Lilly, Novartis, Pfizer and UCB Pharma. **DvdH:** Consultant for AbbVie, Bayer, BMS, Eli Lilly, Galapagos, Gilead, GSK, Janssen, Novartis, Pfizer, Takeda and UCB Pharma; director of Imaging Rheumatology BV. **FvdB:** Speaker for AbbVie, Amgen, Janssen, Merck, Novartis, Pfizer and UCB Pharma; Consultant for AbbVie, Amgen, Eli Lilly, Galapagos, Janssen, Novartis, Pfizer and UCB Pharma. **MM:** Consultant for AbbVie, BMS, Eli Lilly, Novartis, Pfizer and UCB Pharma; research grants from AbbVie, BMS and UCB Pharma. **WPM:** Honoraria/consulting fees from AbbVie, BMS, Boehringer-Ingelheim, Celgene, Eli Lilly, Galapagos, Janssen, Novartis, Pfizer and UCB Pharma; grant/research support from AbbVie, Pfizer and UCB Pharma; educational grants from AbbVie, Janssen, Novartis and Pfizer; Chief Medical Officer for Canadian Research and Education (CARE) Arthritis. **TT:** Consultant for AbbVie, Eli Lilly, Gilead, Novartis and Pfizer; speaker for AbbVie, Astellas, BMS, Eisai, Eli Lilly, Janssen, Kyowa Kirin, Mitsubishi-Tanabe, Novartis and Pfizer. **HX:** Speaker for AbbVie, Janssen, Novartis, Pfizer and UCB Pharma; consultant for AbbVie, Beigene, BioMap, IASO, Pfizer and UCB Pharma; clinical investigator for Peking-

Tsinghua Center for Life Sciences. **CF:** Employee and shareholder of UCB Pharma. **CP:** Employee of Veramed LTD. **UM, TV, JSS, AM:** Employees of UCB Pharma. **LSG**: Consultant for AbbVie, Acelyrin, Eli Lilly, Fresenius Kabi, Janssen, Novartis, Pfizer and UCB Pharma; grant/research support from Novartis and UCB Pharma paid to institution.

Reference

1. BARALIAKOS X: *Ann Rheum Dis* 2024; 83(2): 199-213.



P108: Fig. 1. Pooled randomized set. Patients treated with subcutaneous BKZ 160 mg Q4W; includes patients originally randomized to placebo. ASDAS< 2.1 values shown next to bars are manualy calculated from ASDAS state response rates. ASDAS: Axial Spondyloarthritis Disease Activity Score; axSpA. axial spondyloarthritis; BKZ: bimekizumab; HD: high disease; ID: inactive disease; LD: low disease; MI: multiple imputation; nr-axSpA: non-radiographic axSpA; Q4W: every 4 weeks; r-axSpA. radiographic axial spondyloarthritis; VHD: very high disease

P108: Table I. Efficacy to 2 years /Week 104).

	nr-axSpA	r-axSpA
	BKZ 160 mg Q4W N=254	**BKZ 160 mg Q4W N=332**
ASAS40 [NRI] n (%)	125 (49.2)	179 (53.9)
ASAS40 [MI] % (95% CI)	58.9 (52.6, 65.2)	61.0 (55.5, 66.5)
ASAS Partial Remission [NRI] n (%)	78 (30.7)	104 (31.3)
ASDAS [MI]		
Mean at baseline (SE)	3.7 (0.1)	3.7 (0.0)
Mean at Week 104 (SE)	1.9 (0.1)	1.9 (0.1)
Mean CfB at Week 104 (SE)	−1.8 (0.1)	−1.9 (0.1)
BASDAI [MI]		
Mean at baseline (SE)	6.8 (0.1)	6.5 (0.1)
Mean at Week 104 (SE)	2.9 (0.1)	2.6 (0.1)
Mean CfB at Week 104 (SE)	−4.0 (0.1)	−3.9 (0.1)
Total resolution of enthesitis[a] [NRI] n (%)	78 (41.9)[b]	106 (53.3)[c]

Pooled randomized set. [a] MASES=0 in patients with MASES >0 at baseline; [b] n=186; [c] n=199. ASAS: Assessment of SpondyloArthritis international Society; ASAS40: ASAS 40% response; ASDAS: Ankylosing Spondylitis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BKZ: bimekizumab; CI: confidence interval; CfB: change from baseline; MASES: Maastricht Ankylosing Spondylitis Enthesitis Score; MI: multiple imputation; nr-axSpA: non-radiographic axial spondyloarthrits; NRI: non-responder imputation; Q4W: every 4 weeks; r-axSpA; radiographic axial spondyloarthritis; SE: standard error.

P109

COMPARATIVE ANALYSIS OF BIOLOGIC THERAPY SURVIVAL IN SPONDYLOARTHRITIS AND PSORIATIC ARTHRITIS: A KAPLAN-MEIER APPROACH

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Introduction. While both Spondyloarthritis (SpA) and Psoriatic Arthritis (PsA) fall under the umbrella of spondyloarthritides, a group of disorders characterized by inflammation of the spine and joints, they exhibit distinct clinical features. Biologic therapies changed the treatment paths for these diseases, however, not all patients respond equally and some may even experience treatment failure. We aimed to investigate the differences in treatment persistence between patients with SpA and PsA.

Methods. Retrospective, observational study including adult patients followed in our department with axial or peripheral SpA and PsA, treated with their first bDMARD in our center. Socio-demographic, disease and treatment-related data were collected. Primary endpoint was overall survival (OS), defined as the time from treatment initiation to treatment failure from any cause. Kaplan-Meier survival analysis was performed using SPSS®. Survival curves were compared using the log-rank test.

Results. 62 patients were analyzed. Table I summarizes population characteristics. 19 patients failed first-line biologic treatment: 6 had primary failure and 13 had secondary failure. In this second group, the most common reason for failure was inflammatory back pain (11 patients). Figure 1 shows the results of the Kaplan-Meier survival function.

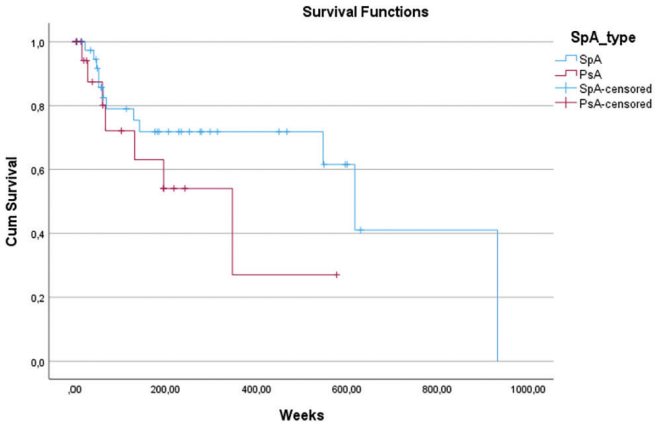
The mean OS time for biological treatment was 524 weeks (95% CI, 376 to 673), and it was greater in SpA (mean OS of 586 weeks, (95% CI, 417 to 754) than in PsA (mean OS of 292 weeks, (95% CI, 157 to 426).

A log rank test was run to determine if there were differences in the survival distribution between spondyloarthritides. The survival distributions for the two groups were not statistically significantly different ($\chi^2(2) = 1.898$, p=0.168).

Conclusion. The survival curve suggests a trend towards better biological persistence for the SpA group but this difference is not statistically significant. Further research with larger sample sizes is needed to better estimate biological persistence outcomes in spondyloarthritides.

P109: Table I. Population characteristics.

Gender	54.8% female; 45.2% male;	
Mean age at diagnosis	44.6 ± 12.5 years	Minimum – 19 years Maximum – 67 years
Follow up time since bDMARD prescription	Mean of 243 ± 246 weeks	Minimum – 0 weeks Maximum – 1049 weeks
Spondyloarthritides type	SpA – 67.7%; (n=42) PsA – 32.3%; (n=20)	
Involvement type	Axial – 79.0% (n=49) Peripheral – 27.4% (n=17)	
Manifestations	Arthritis – 35.5% (n=22) Psoriasis – 30.4% (n=19) Enthesitis – 17.7% (n=11) Dactilitis – 9.7% (n=6) Uveitis – 4.8% (n=3) Nail distrophy – 1.6% (n=1)	
HLA-B27	Positive in 33.9% patients (n=21)	
Sacroileitis on magnetic resonance imaging	Present in 71% of patients (n=44)	
Prescribed first bDMARD	Adalimumab – 48.4% Golimumab – 16.1% Secukinumab – 11.3% Etanercept – 8.1% Infliximab – 6.5% Tofacitinib – 4.8% Certolizumab – 3.2% Risankizumab – 1.6%	
Methotrexate at biologic initiation	29% of patients (n=18)	



P109: Fig. 1. Kaplan-Meier survival function.

P110

EFFECTIVENESS OF GUSELKUMAB IN REAL-LIFE ACCORDING TO TREATMENT LINES AND EARLY PAIN REDUCTION IN A COHORT OF PATIENTS WITH PSORIATIC ARTHRITIS

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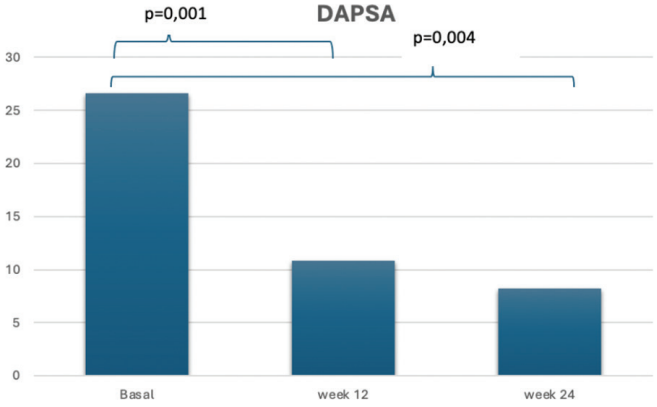
Introduction. Guselkumab is a monoclonal antibody that blocks IL-23, indicated for the treatment of psoriatic arthritis (PsA). Clinical trials have been conducted on its efficacy and safety in patients with PsA, but it is necessary to observe the drug's performance in clinical practice (RWE).
Objectives. To describe the use, effectiveness and persistence in a cohort of PsA patients treated in RWE at the tertiary hospital since its approval in May 2022.
Methods. A single-center retrospective observational study was conducted on adult patients with PsA (CASPAR criteria) and were treated with Guselkumab. The following data were obtained from medical records: swollen joint count (SJC) and tender joint count (TJC), pain VAS, global patient VAS, CRP, DAPSA, demographic characteristics, treatment line and previous medications.
Results. A total of 28 patients were included (basic characteristics in Table I). All patients had joint involvement and 57.1% skin involvement. Fifteen patients were in the first and second line (1st and 2nd L).
The mean DAPSA value at the start was 26.6 (± 5.7) in 1st-2nd L patients and 24.6 (± 5.5) in the 3rd line and beyond (p>0.05). DAPSA progressively decreased until week 24 (Fig. 1).

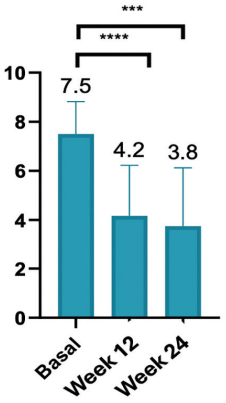
P110: Table I.

Basal characteristics	Patients (n=28)
Women, n(%)	18 (64.3)
Average of previous biological treatments (range)	2.5 (1-8)
Treatment lines (n)	
1º-2º	15
3L o +	13
Concomitant DMARD, n(%)	7(25)
Methotrexate	7(100)
Psoriatic Arthritis, n(%)	
Peripheral	20/28 (71.4)
Mixed	8/28 (28.6)
Psoriasis, n(%)	16/28 (57.1)

The TJC at baseline was 6.5 (2-12), 2.2 (0-10) at week 12, and 1.5 (0-4) at week 24. The SJC was 3.5 (0-10) at baseline, 1.4 (0-6) at 12 weeks, and 0.8 (0-2) at 24 weeks. Pain response decreased from week 12 and this reduction continued until week 24 (Fig. 2).
Conclusion. Guselkumab has demonstrated to be an effective drug in RWE across different treatment lines, with improvement as early as 12 weeks in DAPSA, as well as patient-reported pain, reflecting the rapid response. Four patients discontinued, three of them in the 4th line or higher, mainly due to lack of efficacy. There were no significant adverse effects reported.



P110: Fig. 1. DAPSA evolution.



P110: Fig. 2. PAIN. VAS. Kruskal-Wallis test.
**** p<0.0001
*** p<0.0009

P111

AN INDIVIDUALIZED EXERCISE PROGRAM ON CARDIORESPIRATORY FITNESS, TRUNK STRENGTH AND MOBILITY LEADS TO SIGNIFICANT BETTER OUTCOMES IN PATIENTS WITH AXSPA

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Background. AxSpA is an inflammatory condition primarily affecting the sacroiliac joints and spine impacting trunk strength and mobility but also cardiorespiratory fitness (1). Although exercise is considered as a pilar of treatment (2), there is paucity of research examining the effect of an exercise program incorporating training for trunk mobility and strength, and cardiorespiratory fitness. The aim of this prospective interventional study is to assess the potential benefits for axSpA patients of individualized and combined cardiorespiratory training and trunk strength and mobility exercise program, based on predetermined test results.

Methods. AxSpA patients fulfilling the ASAS classification criteria performed a baseline Cardiopulmonary Exercise Test (CPET) and trunk mobility and strength test on the David Back Concept (DBC) devices. Subsequently, patients participated in an eight-week exercise program, with two sessions per week. Each session entailed two intervals of ten minutes of cardiorespiratory training at a set heartrate, followed by exercises on the DBC devices. Training thresholds were determined based on their individual test results. Finally, participants were retested. A linear mixed model was used to process the results in SPSS 29.
Results. 25 axSpA patients (10 M/15 F, 43,1±12,1 years, symptom duration 19,4±13,3 years, BASDAI 4.1±2.2, BASFI 4.1±2.2 BASMI 3.1±2.2, CE 4.7±2.0cm) participated. Significant improvement was observed for both strength and mobility (see Table). Regarding cardiovascular parameters, significant improvements were noted in aerobic capacity ($p<0.001$) and mechanical efficiency ($p=0.012$). In contrast the relative anaerobic threshold ($p=0,200$) and the ventilatory efficiency ($p=0,064$) did not change significantly.
Conclusion. Cardiorespiratory endurance, trunk strength and mobility can be significantly improved when combining these in one exercise program, tailored to the individual. These findings underline the importance of individualized exercise interventions in axSpA management to improve patient's outcomes and reduce functional limitations.

References

1. DE MITS S, WILLEMS TM, CALDERS P *et al*.: Maximal exercise tolerance, objective trunk strength, and mobility measurements in axial spondyloarthritis. *J Rheumatol* 2024 Apr 15:jrheum.2023-1046. doi: 10.3899/jrheum.2023-1046.
2. RAMIRO S, NIKIPHOROU E, SEPRIANO A *et al*.: ASAS-EULAR recommendations for the management of axial spondyloarthritis: 2022 update. *Ann Rheum Dis* 2023; 82(1): 19-34. doi: 10.1136/ard-2022-223296.

P111: Table I.

Parameter	Mobility deficits			Strength deficits		
	Pre-test	Post-test	*p*-value	Pre-test	Post-test	*p*-value
Extension	11,5±25,1	24,1±50,5	0,28	22,5±32,3	7,5±29,8	**p≤0,001**
Flexion	10,0±20,2	1,5±8,8	0,96	22,8±24,4	14,2±24,7	**p≤0,05**
Rotation R	18,0±19,4	-5,6±19,0	**p≤0,001**	17,7±31,4	2,2±31,9	**p≤0,01**
Rotation L	21,4±19,4	0,2±17,1	**p≤0,001**	15,9±34,0	-0,8±39,7	**p≤0,01**
Lat flexion R	12,7±28,7	-12,5±27,0	**p≤0,001**	27,0±37,8	10,5±37,8	**p≤0,01**
Lat flexion L	11,0±25,0	-18,9±27,0	**p≤0,001**	27,3±39,0	9,4±37,3	**p≤0,001**

Deficit = (Reference value – Patient value) x100 / Reference value; mean ± standard deviation; R: right; L: left; lat: lateral.

P112

ANALYSIS OF THE EFFECTIVENESS AND SAFETY OF GUSELKUMAB IN REAL CLINICAL PRACTICE AFTER 2 YEARS OF TREATMENT IN PSORIATIC ARTHRITIS

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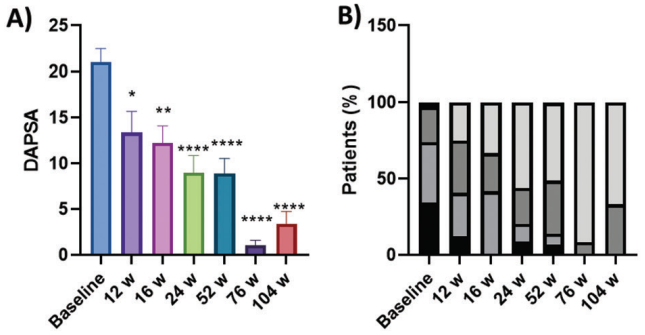
Background. Guselkumab is a human IgG1λ monoclonal antibody that selectively binds to interleukin 23 due to its affinity with p19 sub-unit. IL-23 is a regulatory cytokine that modifies the activity of T lymphocytes, intervening in pathogenic ways that initiate and maintain the activity of Psoriatic Arthritis (PsA). PsA is an autoimmune disease affecting more than joints having a wide spread of systemic manifestations where several profiles of disease exist depending on the predominant affection. Guselkumab has been used widely to treat cutaneous psoriatic disease (PSO) and PsA since 2020. Nevertheless, no long-term analysis have been done.
Objective. The objectives of this study were to analyze in real-life cohort of patients, the effectivenes and long-term survival of Guselkumab at 104 weeks according to treatment lines, and to analyze the safety of Guselkumab.
Methods. An observational, prospective, multicenter study was conducted in patients who met ACR criteria for PsA, who received treatment with Guselkumab for at least 104 weeks (w), starting in April 2020. Patient demographic and baseline data and disease profiles were recorded and response variables. Response data were collected using common index in clinical practice such as DAPSA and BSA and the presence of enthesitis and dactylitis. Statistical analysis was performed with Graph Path Prism 8.0 and SPSS Inc. 2007.
Results. The population was composed by 88 patients with moderate-to-severe PsA. The mean age was 50.9±1.25 years, 56.81% were women, and the mean disease duration was 9.48 years. Approximately 40% of patients received Guselkumab in fourth line or later. The rest of the demographic variables are detailed in Table I.
The mean DAPSA value at the beginning of treatment was 21±1.47. This data was progressively reduced, observing significant differences from week 12 (13.36±2.33) to week 104 (3.43± .33) (Fig. 1). When taking into consideration lines of treatment, 2nd-3rd line patients reduced their DAPSA better and earlier (8.63±2.7 at 12 s, 6.6±1.5 at 52 w and 0 at 104 w). However, patients from the 4th line onwards took longer to improve their DAPSA, with even a small rebound (16.79±3.3 at 12 s, 8.1±2.2 at 52 w and 4.57±1.6 at 104 w) (Fig. 2A).
Based on the DAPSA, patients were classified into categories of high, moderate, low activity and disease remis.n. 59.37% of patients achieved LDA or remission at 12 s, with 91.66% in complete remission at 76 s (Fig. 1B). If line of treatment is taken into account for this analysis, the first group (2nd-3rd liners) achieved total remission in 76 w while the others (4th and onward liners) did not reach it (Fig. 2B-C).
The percentage of patients in the general population who had enthesitic involvement or dactylitis was also significantly reduced from 37.5% or 14.77% respectively in baseline to remission conditions; but when analyzing subgroups for reduction of enthesitic or dactylitic outcomes, patients in the 2nd-3rd line responded better moving from 21 to 2 enthesitic outcomes and from 10 to 0 dactylitic outcomes at 12 s. Secondly, the other group did not respond as quickly (from 22 to 13 and from 17 to 3, respectively).
At the beginning, skin involvement was 15.76±4.9 measured by BSA, with a significant reduction from week 12 (2.4%±1.1) until complete remission at 104 w. Both groups analyzed achieved complete remission after 52 or 76 w. In the cutaneous domain, no differences were observed between groups
There were important differences in drug survival depending on the treatment line, from 100% up to 52 s in earlier lines of treatment being 83% at two years and 80% up to 52 s being 41% at two years in late lines (Fig. 3). No adverse effects were reported.
Conclusion. Guselkumab is effective and safe after 2 years in a real-life cohort of patients with moderate-severe PsA, and has demonstrated to be effective for multiple domains such as joint inflammation, dactylitis, enthesitis or skin. Guselkumab shows favorable results in any line of treatment, but those patients treated in the 2nd-3rd line respond sooner and better than >3rd liners. Furthermore, persistence at two years is 83% or 41% depending on the treatment line group. No adverse effects were reported.

References

1. DEODHAR A, HELLIWELL PS *et al*.; DISCOVER-1 Study Group. Guselkumab in patients with active psoriatic arthritis who were biologic-naive or had previously received TNFα inhibitor treatment (DISCOVER-1): a double-blind, randomised, placebo-controlled phase 3 trial. *Lancet* 2020; 395(10230): 1115-25.
2. MEASE PJ, RAHMAN P *et al*.; DISCOVER-2 Study Group: Guselkumab in biologic-naive patients with active psoriatic arthritis (DISCOVER-2): a double-blind, randomised, placebo-controlled phase 3 trial. *Lancet*. 2020; 395(10230): 1126-36.

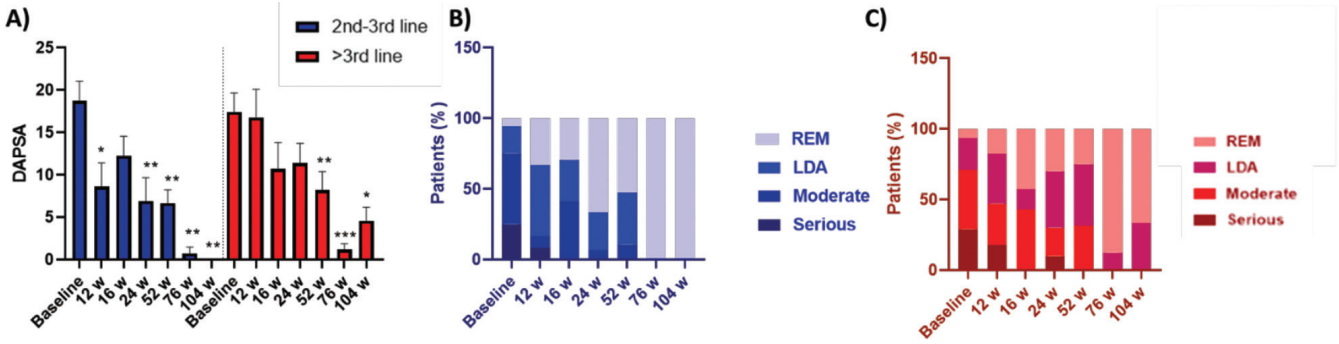
P112: Table I. Demographic and baseline patient characteristics.

Baseline patient characteristics	**n=88**
Age at inclusion, mean (95% CI), years	50,9 ± 1,25
Sex	
Female, n (%)	50, 56,81 %
Male, n (%)	38, 43.19 %
BMI, mean [95% CI]	30.22 ± 1.35
Smoker, n (%)	16, 18.18 %
Disease duration of PsO mean [95% CI], years	9.48 ± 0.79
Disease duration of PsA mean [95% CI], years	13.18 ±.1.1
DAPSA mean [95% ci]	21 ± 1.47
Enthesitis, n (%)	33, (37,5 %)
Dactylitis, n (%)	13, (14,77 %)
BSA mean [95% CI]]	15,76 ± 4,9
Previous DMARDs:	
MTX used, n (%)	46, (52.27 %)
MTX dose, mg/w	15
Line of treatment:	
Second or third, n (%)	47, (57,31 %)
Fourth to eighth, n (%)	35, (42,69%)



	Serious	Moderate	LDA	REM
Baseline	34.24 %	39.72 %	23.28 %	2.73%
12 w	12.5 %	28.12 %	34.37 %	25%
16 w	0	41.66 %	25%	33.33%
24 w	8,82%	11.76 %	23.52 %	55.88%
52 w	6.97 %	6.97%	34.88 %	51.16 %
76 w	0	0	8.33 %	91.66 %
104 w	0	0	33.33 %	66.66 %

P112: Fig. 1. (LDA) or active disease after 104 w with Guselkumab treatment (**B**).



P112: Fig. 2. Absolute DAPSA values until 104 w (**A**). Percentage of patients reaching remission (REM), low disease activity (LDA) or active disease after 104 w with Guselkumab treatment (**B**-**C**). Blue: 2nd and 3rd line and red >3rd line.

P113

BASELINE CHARACTERISTICS AND DISEASE BURDEN IN PATIENTS WITH RADIOGRAPHIC AXSPA (R-AXSPA) STRATIFIED BY CRP LEVEL: AN ANALYSIS FROM THE IXEKIZUMAB COAST-V TRIAL

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Introduction/Objective. Elevated baseline C-reactive protein (CRP) level can serve as a predictor for treatment response to TNFis in patients with r-axSpA. The impact of CRP on b/tsDMARD response in r-axSpA is listed as a topic of interest on the ASAS-EULAR research agenda. ASAS40 responses with Ixekizumab (IXE) have been reported previously. IXE demonstrated efficacy in the treatment of r-axSpA in patients with normal and elevated CRP. Here we report the baseline characteristics of these patients for further clarification of disease burden stratified by CRP.
Materials and methods. Biologic naïve adults with r-axSpA, were enrolled in the COAST-V study. At baseline, patients were randomised to treatment with IXE (Q4W), adalimumab (ADA), or placebo (PBO). Randomization was based on baseline CRP defined as normal (CRP ≤5 mg/L) or elevated (CRP>5 mg/L).
Results. The mean ages of patients with normal CRP at baseline were 43.3, 43.7, and 44.8 years for IXE, ADA, and PBO respectively (subsequently reported in that order, herein), whereas the mean age for patients with elevated CRP at baseline was 39.6, 40.3, and 41.7 years. Gender, HLA-B27 positive status, and the mean duration of r-axSpA diagnosis was similar across both CRP groups (Table I). Patients with normal CRP had lower mean MRI-SPARCC spine score *vs*. those with elevated CRP (6.8, 6.6, and 6.4 *vs*. 15.8, 24.0, and 15.1 units). However, most baseline outcome measures, including ASAS response components, ASAS-HI, BASDAI, SF-36 physical component score, and spinal pain at night, were comparable between the normal and elevated CRP groups.
Conclusion. In patients with r-axSpA, baseline clinical and demographic variables were similar in the normal/elevated CRP groups with patients experiencing similar disease burden.
Acknowledgements. Funded by Eli Lilly and Company.

P113: Table I. Baseline characteristics in patients with r-axSpA.

	IXE		ADA		PBO	
CRP levels	**Normal (n=29)**	**Elevated (n=52)**	**Normal (n=38)**	**Elevated (n=52)**	**Normal (n=26)**	**Elevated (n=61)**
Age (years)	43.3 (12.8)	39.6 (11.6)	43.7 (11.9)	40.3 (11.0)	44.8 (10.7)	41.7 (12.5)
Male, n (%)	26.0 (89.7)	42.0 (80.8)	27.0 (71.1)	46.0 (88.5)	21.0 (80.8)	51.0 (83.6)
BMI	25.9 (4.1)	25.7 (3.9)	25.8 (5.6)	27.1 (5.6)	26.1 (4.5)	28.2 (6.1)
CRP (mg/L)	2.2 (1.4)	17.8 (13.7)	2.2 (1.4)	20.0 (20.1)	2.4 (1.6)	21.6 (22.7)
HLA B-27 positive, n (%)	26.0 (89.7)	49.0 (94.2)	33.0 (86.8)	49.0 (94.2)	23.0 (88.5)	54.0 (88.5)
Duration of r-axSpA symptoms (years)	18.0 (11.5)	14.6 (10.9)	16.3 (10.9)	15.1 (7.9)	19.0 (10.6)	16.7 (13.1)
Time since diagnosis (years)	10.1 (10.1)	7.4 (9.3)	7.1 (8.3)	7.8 (6.9)	6.7 (8.1)	7.2 (7.8)
ASAS response individual components						
Patient Global Assessment (PtGA)	6.9 (1.4)	6.9 (1.6)	6.6 (1.9)	7.4 (1.5)	7.0 (1.7)	7.1 (1.7)
BASFI	5.8 (1.9)	6.2 (1.8)	5.3 (2.2)	6.6 (1.9)	6.2 (1.6)	6.4 (2.0)
Spinal pain	7.1 (1.2)	7.2 (1.4)	6.8 (1.6)	7.1 (1.6)	7.2 (1.4)	7.5 (1.5)
Inflammation (mean of BASDAI questions 5 and 6)	6.2 (1.6)	6.7 (1.6)	6.1 (1.7)	7.0 (1.7)	6.9 (1.5)	6.7 (1.6)
ASAS-HI	8.4 (3.4)	7.0 (3.3)	7.4 (3.7)	8.8 (3.7)	7.9 (3.1)	8.1 (3.7)
BASDAI	6.6 (1.3)	6.9 (1.3)	6.3 (1.4)	6.9 (1.5)	6.7 (1.2)	6.8 (1.3)
MRI-SPARCC Spine Total Score	6.8 (8.6)	15.8 (21.5)	6.8 (10.8)	24.0 (27.7)	6.4 (7.6)	15.1 (19.8)
SF-36, Physical Component Score	37.8 (7.4)	35.7(6.7)	37.6 (7.6)	34.7 (7.6)	33.9 (7.9)	35.0 (8.0)
Spinal pain at night	6.8 (1.5)	7.1 (1.4)	6.7 (1.9)	7.1 (1.6)	7.0 (1.6)	7.0 (1.8)

Continuous parameters are presented as mean (±SD) unless otherwise stated. Categorical variables are presented as n (%).
ASAS-HI: Assessment of Spondyloarthritis International Society-Health Index; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index; BMI: Body Mass Index; HLA B-27: human leukocyte antigen B-27; MRI: magnetic resonance imaging; PtGA: patient global assessment; SPARCC: Spondyloarthritis Research Consortium of Canada Score; SF-36: Short Form Health survey 36 item.

P114

BIMEKIZUMAB DELIVERED SUSTAINED IMPROVEMENTS IN EFFICACY AND DEMONSTRATED A CONSISTENT SAFETY PROFILE IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS: UP TO 2-YEAR RESULTS FROM TWO PHASE 3 STUDIES

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Objective. Assess 2-year efficacy and safety of bimekizumab, a monoclonal IgG1 antibody that selectively inhibits IL-17F in addition to IL-17A, in patients with PsA.

Methods. BE OPTIMAL (NCT03895203; bDMARD-naïve) and BE COMPLETE (NCT03896581; prior inadequate response/intolerance to TNFi [TNFi-IR]) assessed subcutaneous bimekizumab 160mg Q4W (both placebo-controlled to Week [Wk]16; placebo patients then received bimekizumab). BE OPTIMAL reference arm (subcutaneous adalimumab 40mg Q2W) patients received bimekizumab from Wk52 with no washout (adalimumab/bimekizumab). BE OPTIMAL Wk52 and BE COMPLETE Wk16 completers were eligible for BE VITAL (OLE; NCT04009499). Efficacy outcomes reported to Wk104/100 (BE OPTIMAL/BE COMPLETE). Data reported using non-responder, worst-category or multiple imputation. Safety data reported to Wk104 for all bimekizumab-treated patients.

Results. 710/852 (83.3%) bDMARD-naïve and 322/400 (80.5%) TNFi-IR patients completed Wk104/100. ACR50, PASI100, MDA (Fig. 1) and additional efficacy outcomes (Table) were sustained to Wk104/100. Adalimumab/bimekizumab patients sustained ACR50 response rates from Wk52–104; complete skin clearance (PASI100) increased from 48.5%–60.3% from Wk52–104. 86.0% (708/823) bDMARD-ïve and 76.5% (297/388) TNFi-IR patients had ≥1 TEAE on bimekizumab; serious TEAE incidence (EAIR/100 patient-years) was 7.3 and 5.6. Across studies, most frequent TEAEs were: CoV-2 infection, nasopharyngitis, upper respiratory tract infection, urinary tract infection. Three deaths unrelated to treatment were reported (2 [bDMARD-naïve], 1[TNFi-IR]). EAIR/100 patient-years remained low for MACE (0.5, 0.3), serious SARS-CoV-2 infections (0.5, 0.2), suicidal ideation and behaviour (0.2, 0), IBD (0.5, 0) and hepatic adverse events or hepatic enzyme elevations (0.5, 0.2). No reported active tuberculosis or uveitis. Fungal infections (11.3, 7.6) were localised (majority *Candida* [7.1, 4.8]). Most *Candida* infections were oral candidiasis, none systemic; 10 patients discontinued (6, 4).

Conclusion. Bimekizumab was well tolerated and demonstrated sustained clinical efficacy up to 2 years in bDMARD-naïve and TNFi-IR patients with PsA. Adalimumab/bimekizumab patients demonstrated further improvement in skin symptoms, and sustained efficacy in joint symptoms post-switch.

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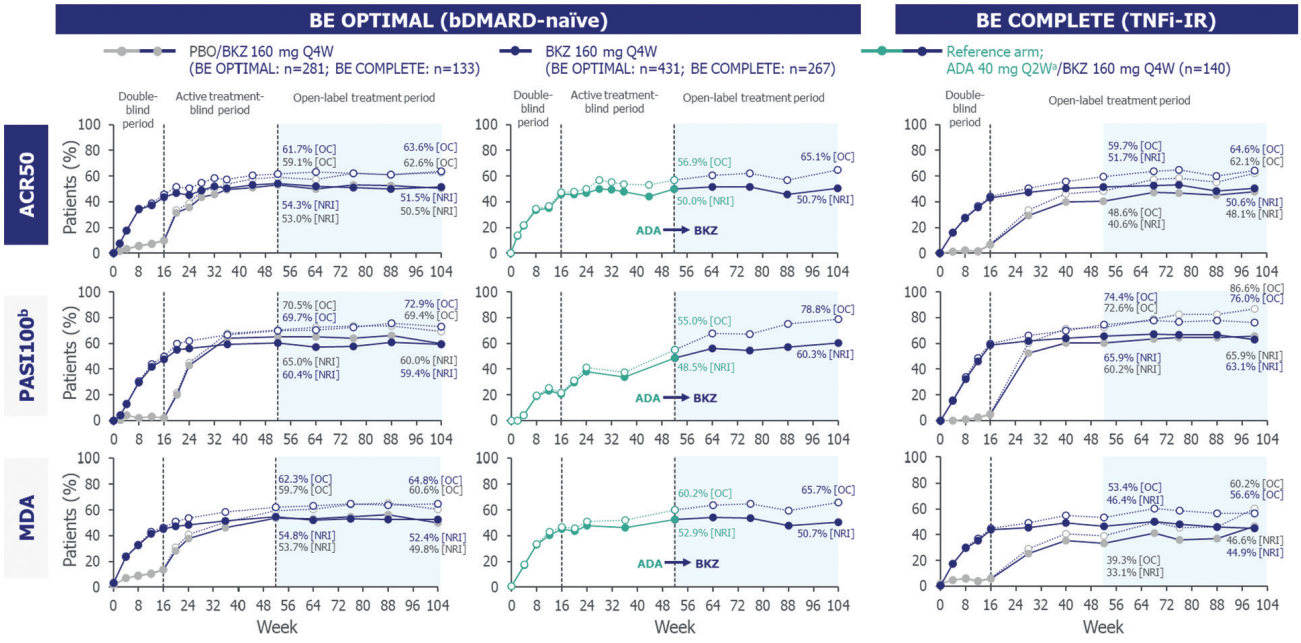

Randomised set. In BE OPTIMAL, patients were randomised 3:2:1 to BKZ 160mg Q4W:PBO:reference arm (ADA 40mg Q2W). In BE COMPLETE, patients were randomised 2:1 to BKZ 160mg Q4W:PBO. In both studies, patients on PBO switched to BKZ 160mg Q4W at Week 16. In BE OPTIMAL, patients on ADA 40mg Q2W switched to BKZ 160mg Q4W at Week 52. [a] Reference arm; study not powered for statistical comparisons between ADA and BKZ or PBO; [b] In patients with psoriasis involving ≥3% BSA at baseline (BE OPTIMAL [PBO/BKZ: n=140, BKZ: n=217, ADA/BKZ: n=68]; BE COMPLETE [PBO/BKZ: n=88, BKZ: n=176]). ACR50: ≥50% improvement from baseline in American College of Rheumatology response criteria; ADA: adalimumab; bDMARD: biologic disease-modifying antirheumatic drug; BKZ: bimekizumab; BSA: body surface area; MDA: minimal disease activity; NRI: non-responder imputation; OC: observed case; PASI100: 100% improvement from baseline in Psoriasis Area and Severity Index; PBO: placebo; Q2W: every two weeks; Q4W: every four weeks; TNFi-IR: prior inadequate response or intolerance to tumour necrosis factor inhibitors.

P114: Fig. 1. Proportion of patients schieving ACR50, PASSI100 and MDA over time to Week 104/100 (NRI, OC.

P114: Table I. Additional efficacy endpoints with bimekizumab treatment at Week 104/100 (NRI, WCI, MI).

	BE OPTIMAL (bDMARD-naïve)			BE COMPLETE (TNFi-IR)	
	PBO → BKZ 160mg Q4W n=281	BKZ 160mg Q4W n=431	ADA 40mg Q2W[a] → BKZ 160mg Q4W n=140	PBO → BKZ 160mg Q4W n=133	BKZ 160mg Q4W n=267
ACR20 responders, n (%)	180 (64.1)	274 (63.6)	89 (63.6)	90 (67.7)	177 (66.3)
ACR70 responders, n (%)	98 (34.9)	169 (39.2)	49 (35.0)	43 (32.3)	93 (34.8)
PASI75 responders,[b] n/N (%)	112/140 (80.0)	162/217 (74.7)	49/68 (72.1)	63/88 (71.6)	142/176 (80.7)
PASI90 responders,[b] n/N (%)	102/140 (72.9)	153/217 (70.5)	47/68 (69.1)	61/88 (69.3)	128/176 (72.7)
ACR50+PASI100 responders,[b] n/N (%)	60/140 (42.9)	93/217 (42.9)	33/68 (48.5)	42/88 (47.7)	74/176 (42.0)
VLDA responders, n (%)	73 (26.0)	132 (30.6)	41 (29.3)	23 (17.3)	64 (24.0)
DAPSA disease state [WCI],[c] n (%)					
LDA+REM	141 (50.2)	228 (52.9)	77 (55.0)	71 (53.4)	123 (46.1)
REM	56 (19.9)	102 (23.7)	39 (27.9)	22 (16.5)	42 (15.7)
TJC=0 (of 68 joints), n (%)	96 (34.2)	145 (33.6)	53 (37.9)	31 (23.3)	77 (28.8)
SJC=0 (of 66 joints), n (%)	163 (58.0)	261 (60.6)	79 (56.4)	81 (60.9)	143 (53.6)
Enthesitis resolution,[d] n/N (%)	47/70 (67.1)	77/143 (53.8)	20/36 (55.6)	18/36 (50.0)	57/106 (53.8)
Dactylitis resolution,[e] n/N (%)	26/33 (78.8)	42/56 (75.0)	8/11 (72.7)	10/14 (71.4)	28/34 (82.4)
Nail psoriasis resolution,[f] n/N (%)	110/156 (70.5)	163/244 (66.8)	55/75 (73.3)	52/83 (62.7)	103/159 (64.8)
HAQ-DI CfB [MI], mean (SE)	−0.37 (0.03)	−0.34 (0.02)	−0.37 (0.05)	−0.36 (0.06)	−0.39 (0.03)
SF-36 PCS CfB [MI], mean (SE)	8.8 (0.6)	8.4 (0.5)	9.3 (0.9)	8.3 (0.9)[g]	8.9 (0.6)[g]

Randomized set. In BE OPTIMAL, patients were randomized 3:2:1 to BKZ 160mg Q4W: PBO: reference arm (ADA 40mg Q2W). In BE COMPLETE, patients were randomized 2:1 to BKZ 160mg Q4W:PBO. In both studies, patients on PBO switched to BKZ 160mg Q4W at Week 16. In BE OPTIMAL patients on ADA40mg Q2W switched to BKZ 160mg Q4W 52. NRI unless otherwise stated. [a] Reference arm; study not powered for statistical comparisons of ADA to BKZ or PBO; [b] In patients with psoriasis affecting ≥3% BSA at baseline; [c] Missing data were imputed using the WCI method. Any missing data or data recorded after discontinuation of the study treatment were categorized as High Disease Activity (HDA), which is the worst category out of the four DAPSA categories (REM, LDA, medium disease activity, and HDA); [d] In patients with baseline enthesitis (EI \0); [e] In patients with baseline dactylitis (LDI>0); [f] In patients with baseline nail psoriasis (mNAPSI >0); [g] Data reported to Week 88 as data were not collected at Week 100. ACR20/50/70: ≥20/50/70% improvement from baseline in American College of Rheumatology response criteria; ADA: adalimumab; bDMARD: biologic disease-modifying antirheumatic drug; BSA: body surface area; BKZ: bimekizumab; CfB; change from baseline; DAPSA: Disease Activity Index for Psoriatic Arthritis; HAQ-DI: Health Assessment Questionnaire-Disability Index; HDA: high disease activity; LDA: low disease activity; LDI: Leeds Dactylitis Index; LEI: Leeds Enthesitis Index, MI: multiple imputation; mNAPSI: modified Nail Psoriasis Severity Index; NRI: non-responder imputation; PASI75/90/100:≥75/90/100% improvement from baseline in Psoriatic Area and Severity Index; PBO: placebo; Q2W: every two weeks; Q4W: every four weeks; REM: remission; SE: standard error; SF-36 PCS: Short-Form 36-item Health Survey Physical Component Summmary; SJC: swollen joint count; TJC: tender joint count; TNFI-IR: prior inadequate response or intolerance to tumour necrosis factor inhibitors; VLDA: very low disease activity; WCI: worst-category imputation,.

BMS, Eli Lilly, Janssen, MoonLake, Novartis, Pfizer, Solarea and UCB Pharma. **YT:** Speaker fees and/or honoraria from AbbVie, Asahi-kasei, Astellas, AstraZeneca, Boehringer-Ingelheim, Chugai, Daiichi-Sankyo, Eisai, Eli Lilly, Gilead, GSK, Pfizer, Taisho and UCB Pharma; received grants from Boehringer-Ingelheim, Chugai and Taisho. **AA:** Honoraria and/or research grants from AbbVie, Amgen, BMS, Boehringer Ingelheim, Eisai, Eli Lilly, Janssen, Kyowa Kirin, LEO Pharma, Maruho, Mitsubishi Tanabe Pharma, Novartis, Pfizer, Sun Pharma, Taiho Pharma, Torii Pharmaceutical Co. and UCB Pharma. **LG:** Grants or contracts from AbbVie, Biogen, Lilly, Novartis and UCB Pharma; consulting fees from AbbVie, BMS, Celltrion, Janssen, Novartis, Pfizer and UCB Pharma; honoraria for lectures from AbbVie, Amgen, BMS, Celltrion, Janssen, Lilly, MSD, Novartis, Pfizer, Stada and UCB Pharma; support for attending meetings and/or travel from MSD, Novartis and Pfizer; received medical writing support from AbbVie, Amgen, Galapagos, Janssen, Pfizer and UCB Pharma; membership on an entity's Board of Directors or advisory committees: Treasurer. **BI:** Shareholder of AbbVie, GSK and UCB Pharma; employee of UCB Pharma. **RB and JC:** Employees and shareholders of UCB Pharma. **AH:** Employee of UCB Bioscience. **VS:** Employee of UCB Pharma. **LCC:** Grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Gilead, Janssen, Novartis, Pfizer and UCB Pharma; consultant for AbbVie, Amgen, BMS, Boehringer Ingelheim, Celgene, Domain, Eli Lilly, Galapagos, Gilead, Janssen, MoonLake Immunotherapeutics, Novartis, Pfizer and UCB Pharma; speaking fees from AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GSK, Janssen, medac, Novartis, Pfizer and UCB Pharma.

P115

PERSISTENCE OF SECUKINUMAB DOSE ESCALATION TO 300 MG MONTHLY IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS. MULTICENTRE EXPERIENCE IN REAL-WORLD CLINICAL PRACTICE

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Introduction/Objective. Secukinumab (SECU), an anti-IL-17 antibody, has demonstrated efficacy in clinical trials (CTs) for axial radiographic (r-SpAx) and non-radiographic Spondyloarthritis (nr-SpAx). ASLEAP found that both 150 and 300mg are effective without differences. The impact of these dosages in real clinical practice, in those with partial respond, remains unclear. This study aims to assess the persistence of SECU in patients with SpAx whose dose had to be uptitrated from 150 to 300mg due to inadequate response. To analyze the factors influencing the clinical response to 300.
Material and methods. Observational, retrospective, multicenter study of patients with r-SpA and SpA-nr who were uptitrated to 300mg SECU after 12-16 weeks with 150mg due to clinical improvement (delta ASDAS >1.1 or delta >2) but without low disease activity or remission. Data were collected for 24 months after initiation of 300mg. Cox regression was used.
Results. Out of 106 patients (77 with r-SpA and 29 with nr-SpA), 79 (74.5%) continued on SECU 300mg at the time of data collection. 27 patients discon-

tinued, 25 because of lack of efficacy, 2 due to adverse effects. No significant differences were found between the two groups. Patients who discontinued, baseline activity tended to be worse: BASDAI (0.057), ASDAS-CRP (p=0.059) and tender joint count (TJC), were higher (p=0.017). Patients who continued had lower prevalence of dyslipidemia (p=0.021) and uveitis (p=0.035) (Table I). Cox analyses (Table II): TJC (HR=1.137, p=0.002) and dyslipidemia (HR=3.191, p=0.016) were predictors of discontinuation, whereas uveitis did not affect outcomes (p=0.389).

Conclusion. For SpAx patients with partial respond to 150mg, uptitration to 300mg shows 74.5% persistence. Peripheral manifestation and dyslipidemia were associated with higher discontinuation rates. Further studies are needed to confirm these observations.

P115: Table I. Basal characteristic of patients based on continued or not treatment.

	Continue with SECU 300 mg		*p*-value
	Yes n=79 (74.5%)	No n=27(25.5%)	
Male, n (%)	49 (62,0)	17 (63,0)	0,930
r-SpA diagnosis, n (%)	55 (69,6)	22 (81,5)	0,345
Age at SECU 300 initiation (years)	47,8 ± 10,6	50,9 ± 14,7	0,338
Time between 150 to SECU 300 (years)*	0,7 (0,4; 1,7)	0,4 (0,2; 1,3)	0,106
Disease duration until SECU 300 (years)*	6,6 (3,1; 13,6)	10,0 (2,8; 16,6)	0,674
BMI (kg/m^2)	27,5 ± 4,8	26,5 ± 3,3	0,419
BMI, n (%)			0,194
18,5-24,9	15 (36,6)	5 (26,3)	
≥25	16 (39,0)	12 (63,2)	
≥30	10 (24,4)	2 (10,5)	
Smoker, n (%)			0,656
Never	49 (62,0)	16 (61,5)	
Ex-smoker	17 (21,5)	4 (15,4)	
Currently smoker	13 (16,5)	6 (23,1)	
Comorbidities, n (%)			
Hypertension	18 (22,8)	10 (37,0)	0,231
Cardiovascular disease	6 (7,6)	5 (14,8)	0,467
Dylipidemia	20 (25,3)	14 (51,9)	**0,021**
Diabetes	6 (7,6)	5 (14,8)	0,467
Uveitis	9 (11,4)	8 (29,6)	**0,035**
Psoriasis	9 (11,5)	2 (7,4)	0,725
HLAB 27, n (%)	57 (73,1)	21 (80,8)	0,601
Treatment line			0,788
First	21 (26,6)	9 (33,3)	
Second	27 (34,2)	8 (29,6)	
Third or more	31 (39,2)	10 (37,0)	
DAS28-CRP	2,42 ± 1,02	2,68 ± 1,16	0,269
BASDAI	4,19 ± 2,46	5,36 ± 2,16	0,057
ASDAS-CRP	7,94 ± 4,99	6,41 ± 6,28	0,202
ASDAS-CRP*	6,5 (4,33; 11,93)	7,62 (2,92; 14,65)	0,059
ASDAS-ESR*	7,0 (4,1; 12,9)	3,9 (0,6; 12,2)	0,086
TJC*	0 (0; 2)	1 (0; 4)	**0,017**
SJC*	0 (0; 0,2)	0 (0; 0)	0,665
CRP	3 (2; 7,2)	3 (1,6; 16,7)	0,904

(*) Data shows Me (IQR).

P115: Table II. Factors associated to a better respond in patients who escalate the standard dosage of SECU treatment. Cox analyses

	HR (CI- 95%)	*p*-value
TJC	1.199 (1.07-0.42)	**0.042**
Dylipidemia	3.191 (1.24-8.17)	**0.016**
DAS28-CRP	1.013 (0.44-2.3)	0.976
ASDAS-CRP	1.423 (0.73-2.74)	0.292
ASDAS-ESR	0.664 (0.37-1.16)	0.156

P116

TREAT-TO-TARGET WITH SECUKINUMAB AS FIRST-LINE BDMARD VERSUS STANDARD-OF-CARE TREATMENT IN AXIAL SPONDYLOARTHRITIS: A RANDOMIZED CONTROLLED OPEN-LABEL TRIAL (AScalate)

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Introduction. The aim of the study was to compare treatment escalation strategy using the IL-17i secukinumab (SEC, T2T) with a standard of care approach (SoC) over 36 weeks in patients with active axSpA.

Methods. Study methodology has been previously described (1). This was a randomized, parallel-group, open-label, multicentric study involving patients with active axSpA. All patients were naive to b-/ts DMARDs and were inadequate responders (IR) to prior NSAID treatment.

In the T2T group, patients received SEC150mg s.c. At week 12, IR (no clinically important improvement (CII) in ASDAS) received SEC 300mg s.c. q4w. At week 24, responders continued SEC 150mg or SEC 300mg q4w treatment and IR switched to adalimumab 40mg s.c. q2w until Week 36. In the SoC arm, patients received treatment at the investigator's discretion.

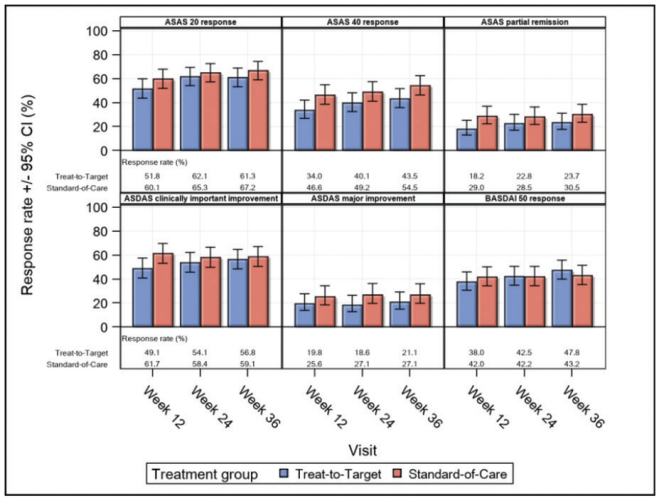
The primary endpoint was the achievement of ASAS40 response at Week 24 with assumed superiority of the T2T over SoC.

Results. A total of 304 patients were randomized (1:1), 155 into the T2T arm and 149 into the SoC arm. Patients baseline characteristic were equilibrated between arms. The primary endpoint was not met (OR 0.69, 95% CI 0.43-1.10; p=0.119; Fig. 1). Other endpoints exploring clinical response did not report significant difference between arms (Fig. 1). Post-hoc analysis on the time course of response rates in the treatment sequence SEC150-SEC300 of the T2T group showed that dose escalation for IR at week 12 (ΔASDAS<1.1) improved response rate at week 24 for 28% of patients (Fig. 2). Safety was comparable across the treatment modalities.

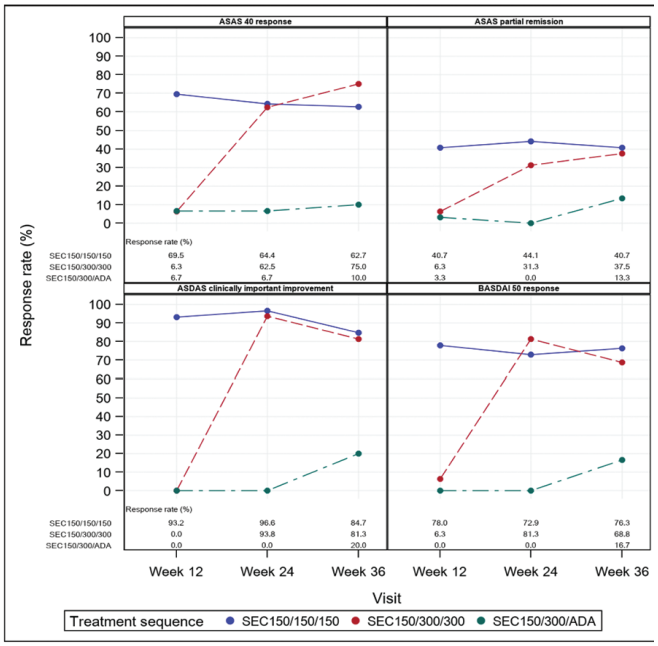
Conclusion. With the methodology chosen, the AScalate study did not demonstrate superiority of T2T in axSpA over SoC. AxSpA patient care was found to be close to T2T strategy in the participating expert centers. SEC dose escalation was beneficial for approximately one third of patients.

Reference

1. PODDUBNYY D *et al.*: *BMJ Open* 2020; 10: e039059.



P116: Fig. 1. Response rates for specific binary efficacy endpoints, adjusted for baseline characteristics - bar plots with data from model analysis (Full Analysis Set). The response rate in treatment groups is adjusted for baseline quick CRP and baseline body weight. Missing values were imputed by non-response (non-responder imputation), i.e. percentages are based on all patients in the analysis set.



P116: Fig. 2. Response rates for specific binary efficacy endpoints, line plots with raw data (Full Analysis Set).
Missing values were imputed by non-response (non-responder imputation), i.e. percentages are based on all patients in the analysis set. For each sub-group, only responding patients were reported.

P117

DEVELOPMENT OF A TREAT TO TARGET ADVANCED NURSE PRACTITIONER-LED CARE PATHWAY FOR PATIENTS COMMENCING TREATMENT FOR AN AXIAL SPONDYLOARTHROPATHY IN MRH TULLAMORE

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Introduction. Patients diagnosed with Axial Spondyloarthropathy (AxSpA) typically experience delays in diagnosis, challenges accessing supports, and, before biologics, had limited options to control disease. 1,2, AxSpA patients present with a myriad of symptoms, including inflammatory back pain, sacroiliitis and extra-articular manifestations. Nurses play a crucial role for AxSpA patients, providing holistic assessment/ education (including comorbidity screening, vaccination advice, and smoking cessation), telephone support, and manage disease/ treatment adverse events to maximise patient outcomes.
Methods. Following a review of treatment pathways used elsewhere and recognising the benefit to patients of access to nursing support through year one of care, a treatment pathway was developed to structure care, allowing for confirmation of diagnosis (with imaging) before commencing the treatment pathway. Within year one, patients undergo full assessment, including outcome measures and receive initial disease education/ NSAID trial while awaiting pre-biologic screening results. Where appropriate, patients commence biologic and undergo three monthly reviews until minimal disease activity/ remission is achieved. Outcome measures are repeated at each visit up to the end of 1 year.
Results. Since January 2024, 20 patients have commenced the pathway, with outcome measures collected at the first visit. Of these patients, 14 will complete 3-month review appointments in the coming weeks. Nine patients commenced on biologics prior to formalising the care pathway, and review data were collected as below. New/ review patients will be added to data set as identified.
Conclusion. This service innovation provides high-quality, cost-effective, streamlined care to patients to minimise their treatment challenges. Evidence supports early induction of disease remission to maximise outcomes and reduce long-term healthcare utilisation. More data is needed to draw conclusions, but early indications suggest effective care and high patient satisfaction.

References
1. RAMIRO S, NIKIPHOROU E, SEPRIANO A *et al.*: ASAS-EULAR recommendations for the management of axial spondyloarthritis: 2022 update. *Ann Rheum Dis* 2023; 82(1): 19-34.
2. HAMILTON L, BARKHAM N, BHALLA A *et al.*: BSR and BHPR guideline for the treatment of axial spondyloarthritis (including ankylosing spondylitis) with biologics. *Rheumatology* 2017; 56(2): 313-16.

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EFFECTIVENESS AND SAFETY OF BIMEKIZUMAB IN PSORIATIC ARTHRITIS PATIENTS: MULTICENTRIC EXPERIENCE FROM FOUR MULTIDISCIPLINARY CARE UNITS

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Background. Psoriatic arthritis (PsA) is a chronic inflammatory condition affecting skin and joints. Bimekizumab, dual IL-17A and IL-17F inhibitor, has shown promising results in clinical trials for psoriasis (PsO) and PsA patients. Multidisciplinary care units (MDU) of rheumatologists and dermatologists allow early real-world experience with novel therapies in PsA patients.
Objectives. To describe demographic and clinical characteristics of PsA patients treated with Bimekizumab within MDU, to assess its effectiveness in musculoskeletal domains.
Methods. Retrospective, observational, longitudinal, multicenter study conducted at four rheumatology centers in Barcelona. Consecutive PsA patients who met the Classification Criteria for PsA (CASPAR) and were prescribed Bimekizumab for PsO were included. Demographic and clinical variables, concurrent use of csDMARDs, previous biologic therapies, Bimekizumab dosing and reasons for discontinuation, were collected. Effectiveness and safety were assessed at 3 and 6 months. Data were analyzed for mean, median and squared-chi, if necessary.
Results. 18 patients (8 females) were included, with a mean age: 52 ± 11 years. The median disease duration: 7 years [3-9]. All patients were exposed to at least 1 bDMARDs (up to 7) and one was receiving concurrent csDMARDs. All patients received Bimekizumab 320mg / 4 weeks for the first 16 weeks, followed by every 8 weeks thereafter. Clinical manifestations, laboratory data and disease activity are shown in Table I. Only 3 patients had axial involvement, all of them with positive HLA-B27.
Improvement of DAPSA, ASDAS, PASI and CRP, resolution of dactylitis/ enthesitis was achieved, with statistical significance ($p<0.05$), from baseline to 6 months follow up. No serious adverse events were reported, no cases of uveitis nor IBD.
Conclusion. This study provides valuable insights into the real-world use of Bimekizumab in PsA patients within the MDU, showing rapid and sustained efficacy up to 6 months of follow-up across the different domains with an excellent safety profile.

P118: Table I. Clinical features of PsA patients treated with Bimekizumab at baseline, 3 and 6 months of follow-up

	Baseline	3 months	6 months
Tender joint count	4.5 ± 4.6	2 ± 3.2	0.7 ± 1.3
Swollen joint count	3.4 ± 4	1.2 ± 1.9	0.4 ± 0.8
Dactylitis	1 ± 1	0.5 ± 1	0
Enthesitis	1.2 ± 1.9	0.8 ± 1.7	0.3 ± 0.8
ASDAS (n=2)	1.9	1.1	1
DAPSA	18 ± 11.3	10.9 ± 6	5 ± 3.4
PASI (n=16)	7.8 ± 5.2	2.3 ± 1.9	0.9 ± 0.9
CRP	5.6 ± 8.4	2.3 ± 3.3	2.4± 3

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EFFICACY AND RETENTION RATE OF SWITCHING TO SUBCUTANEOUS INFLIXIMAB FROM INTRAVENOUS INFLIXIMAB IN PATIENTS WITH ANKYLOSING SPONDYLITIS: 1-YEAR RESULT FROM SINGLE CENTER STUDY

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Background. Subcutaneous infliximab (CT-P13 SC) has been approved in ankylosing spondylitis (AS). We report 1-year efficacy and retention rate of CT-P13 SC switching from intravenous infliximab in patients with AS.
Methods. Patients ≥ 18 years with AS who met modified New York criteria and had been administered 2 or more intravenous infliximab were enrolled in single center from December 2021 and December 2023. Patient demographics, effectiveness, drug retention, reasons for discontinuation data were collected and analyzed.
Results. A total of 84 patients were included. One-year retention rate was 82.1% (95% confidence interval 84.7-95.3) for CT-P13 SC. Among 15 patients who discontinued CT-P13 SC, reasons for discontinuation were inefficacy for 7 patients and discomfort use of subcutaneous pen for 5 patients. By logistic regression, discontinuation of CT-P13 decreased in age 30-39 compared to age 20-29 (hazard ratio 0.156, p=0.025) and increased in patients with high and very high disease activity compared to compared to inactive and low disease activity measured by ASDAS-CRP (hazard ratio 4.924, p=0.027). BASDAI, ASDAS-CRP, and ASDAS-ESR were decreased in continuing group for 1-year ($p<0.001$). No adverse event was reported.
Conclusion. CT-P13 SC switching from intravenous infliximab was effective in patient of AS with good retention rate.

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EFFECTS OF GLUTEN-FREE DIET ON INDIVIDUALS WITH AXIAL SPONDYLOARTHRITIS - RESULTS OF A PILOT RANDOMIZED DOUBLE-BLINDED TRIAL WITH DIETETIC INTERVENTION

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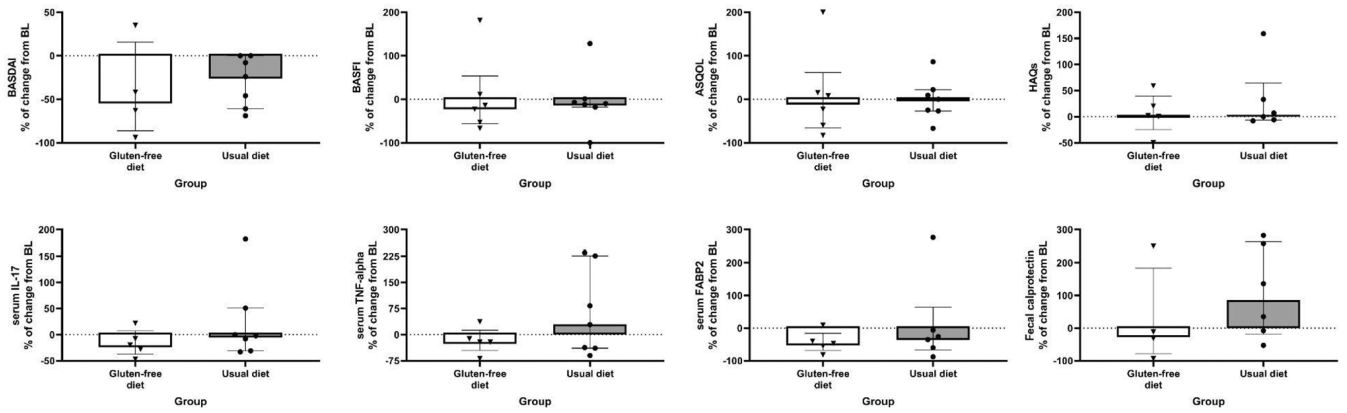
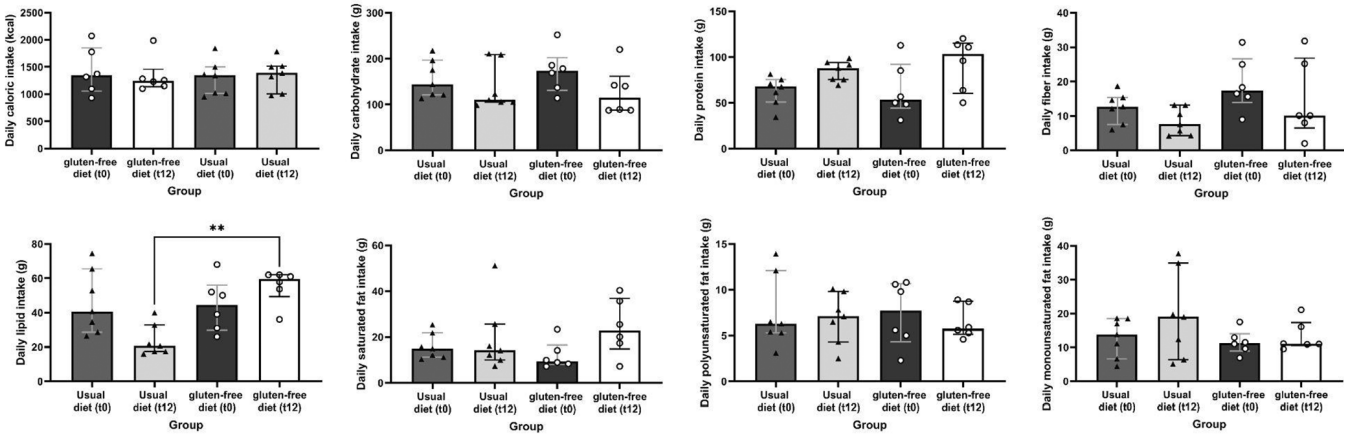
Introduction. The pathophysiology of Axial Spondyloarthritis (axSpA) involves intestinal dysbiosis and leaky gut, characterized by increased intestinal permeability to bacteria and their products. Gliadin, known to induce zonulin release, may exacerbate this condition. This pilot study aimed to evaluate the effects of a 12-week gluten-free diet on biomarkers, clinical parameters, and fecal microbiota in patients with active axSpA.
Methods. Patients with active axSpA aged ≥18 (BASDAI≥4 or ASDAS-PCR≥2.1) and without prior inflammatory bowel disease were randomized to either continue their usual diet or adopt a 12-week gluten-free (GF) diet. Assessments included clinical outcomes, macronutrient intake, biomarkers of immune response, inflammation and intestinal permeability, and microbiota composition and relative abundance, conducted before and after the intervention.
Results. Thirteen patients were included (six in the GF group and seven controls): mean age 53±8.8 years; 69% female; 77% with radiographic axSpA; 100% HLA-B27 positive; mean BASDAI 4.5±2.3. After 12 weeks, the

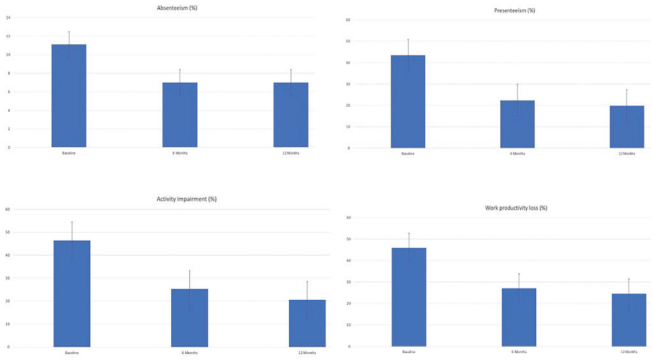


P120: Fig. 1. Clinical and biomarkers outcome.



P120: Fig. 2. Diet composition changes between groups.

GF group exhibited a numerically higher, but not statistically significant, improvement in BASDAI compared to controls. The GF group also showed greater reductions in serum levels of IL-17A and TNF-alpha, and fecal calprotectin, although these differences were not statistically significant. The GF diet was associated with increased daily lipid intake, particularly saturated fats. Notably, a positive correlation was observed between the relative abundance of Dorea and BASDAI (p=0.038), BASFI (p=0.003), ASCOL (p=0.032), and HAQS (p=0.004), with no significant differences between the dietary groups or within individuals from baseline to 12 weeks.
Conclusion. This pilot study found that a 12-week gluten-free diet did not result in significant differences in clinical outcomes, biomarkers, or microbiota composition. However, an increase in daily lipid intake, particularly saturated fats, was observed, aligning with findings from other studies.
Acknowledgements. Fundo de Apoio à pesquisa da Sociedade Brasileira de Reumatologia – FAPE-SBR.



P121: Fig. 2. Change in WPAI score.

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CHANGES IN WORK PRODUCTIVITY AND QUALITY OF LIFE IN AXIAL SPA PATIENTS RECEIVING INTRAVENOUS INFLIXIMAB IN GREECE

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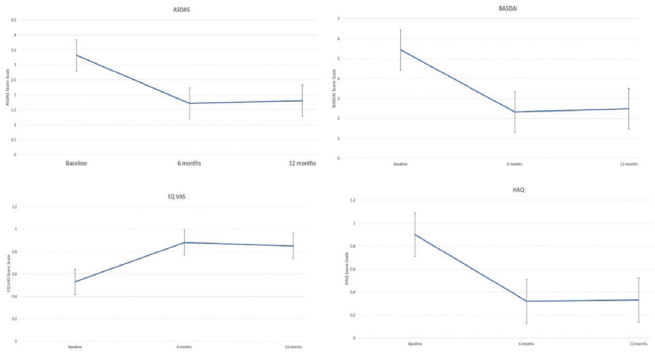
Introduction. Axial SpA is a systemic inflammatory disease that starts usually between the second and third decade of life, has a chronic progression, and has a significant impact on quality of life and ability to perform paid work. Infliximab has proved its effectiveness in the signs and symptoms of the disease as well as in the quality of life and work outcomes of patients in many countries but the data regarding Greek patients are not many.
Methods. Prospective study of axial SpA patients receiving intravenous Infliximab as per current ASAS-EULAR recommendations in a tertiary university hospital in Greece. Demographics, disease activity (ASDAS, BASDAI), and questionnaires about quality of life (Euroquol VAS, HAQ) and work productivity (WPAI) were collected at baseline, at 6 months, and at the end of the study at 12 months.
Results. 34 patients (19 men and 15 women) of whom 26 (76,47 %) had a paid job were included in the study. The mean age of the patients was 34,1 years and the mean disease duration was 2.9 years.
The mean ASDAS and BASDAI in the 12 months of follow-up decreased from 3.32 and 5.44 to 1.80 and 2.48 respectively, while the mean Euroquol VAS increased from 0.53 to 0.85 and the HAQ decreased from 0.9 to 0.33 (Fig. 1).
Work productivity of these patients which was significantly impaired at the baseline improved after the initiation of the treatment and until the end of the study (Fig. 2).
Conclusion. Axial SpA patients who received intravenous infliximab showed significant improvement in quality of life, work productivity, and disease activity after 12 months of treatment.



P121: Fig. 1. Changes in ASDAS, BASDAI, EQ VAS and HAQ.

P122

PROFILE OF REAL WORLD AXIAL SPONDYLOARTHRITIS PATIENTS REFRACTORY TO ADVANCED THERAPY

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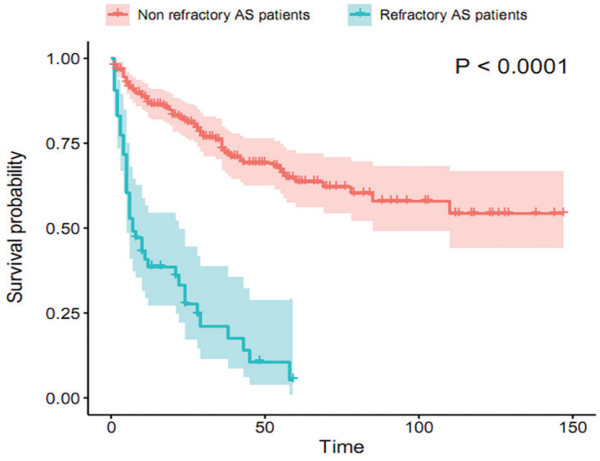
Introduction. Axial spondyloarthritis (AS) is an autoinflammatory disease in which biologic or targeted synthetic disease modifying drugs (DMARDs) therapy is widely used. However, some patients are still somewhat refractory to these therapies. Our aim is to analyze the clinical and therapeutic characteristics of AS patients who have received at least three distinct advanced therapy (AT) lines.
Methods. Retrospective observational study of AS patients treated with at least 1 targeted therapy (TT). We analyze the different therapeutic lines administered, their duration and the causes of withdrawal. Patients are classified in non-refractory AS (1- 2 lines of TT) and refractory AS (3 or more lines of TT).
Results. 205 AS patients with TT were included (62.9% men), with a mean age at diagnosis of 40 (13) years and a disease progression time since the beginning of the first TT of 24 (27) months. 65.7% of patients were HLA-B27 positive, 22.9% presented extraarticular manifestations and 38.5% peripheral damage. 12 patients were considered as refractory AS, with short drug survival (p<0.001) (Fig. 1).



P122: Fig. 1. Anti-TNF treatment was the most used in first line, although it was less usual in refractory patients. Treatment change in second line was mainly by switching, using cycling in third line (Fig 2). A tendency to more concomitant DMARD use in non-refractory AS is observed (p=0.0125).
A significant association between refractory AS and the presence of psoriasis (p<0.001), osteoarthritis (p=0.041) and anxious-depressive syndrome (p=0.002) have been observed.

Variable	NON REFRACTORY AS (PATIENTS WITH 1 OR 2 LINES OF TARGETED THERAPY)				REFRACTORY AS (PATIENTS WITH 3 OR MORE LINES OF TARGETED THERAPY)							
	1st LINE n=193	2nd LINE n=35	3rd LINE n=7	4th LINE n=3	1st LINE n=12	2nd LINE n=12	3rd LINE n=12	4th LINE n=8	5th LINE n=5	6th LINE n=2	7th LINE n=1	8th LINE n=1
Therapeutic families												
Anti-TNF	135 (69.9%)	21 (60%)	6 (85.7%)	0 (0%)	6 (50%)	6 (50%)	8 (66.7%)	3 (37.5%)	1 (20%)	1 (50%)	0 (0%)	1 (100%)
Anti-IL17	52 (26.9%)	11 (31.4%)	1 (14.3%)	3 (100%)	4 (33.3%)	6 (50%)	0 (0%)	1 (12.5%)	0 (0%)	1 (50%)	0 (0%)	0 (0%)
JAK inh	3 (1.6%)	3 (8.6%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	3 (25%)	3 (37.5%)	4 (80%)	0 (0%)	1 (100%)	0 (0%)
Others	3 (1.6%)	0 (0%)	0 (0%)	0 (0%)	2 (16.7%)	0 (0%)	1 (8.3%)	1 (12.5%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Treatment change												
cycling	0 (0%)	8 (22.9%)	5 (71.4%)	0 (0%)	0 (0%)	3 (25%)	3 (25%)	2 (25%)	1 (20%)	0 (0%)	0 (0%)	0 (0%)
switching	0 (0%)	27 (77.1%)	2 (28.6%)	3 (100%)	0 (0%)	9 (75%)	9 (75%)	6 (75%)	4 (80%)	2 (100%)	1 (100%)	1 (100%)
Concomitant DMARD	48 (24.9%)	12 (34.3%)	5 (71.4%)	3 (100%)	0 (0%)	0 (0%)	1 (8.3%)	1 (12.5%)	1 (20%)	1 (50%)	1 (100%)	1 (100%)
Withdrawal cause												
Inefficacy	5 (10%)	3 (25%)	0 (0%)	0 (0%)	2 (16.7%)	4 (33.3%)	3 (33.3%)	3 (60%)	1 (50%)	0 (0%)	1 (100%)	0 (0%)
Loss of efficacy	20 (40%)	5 (41.7%)	3 (100%)	0 (0%)	5 (41.7%)	5 (41.7%)	2 (22.2%)	1 (20%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Adverse events	15 (30%)	2 (16.7%)	0 (0%)	0 (0%)	4 (33.3%)	3 (25%)	4 (44.4%)	1 (20%)	1 (50%)	1 (100%)	0 (0%)	0 (0%)
Other causes	10 (20%)	2 (16.7%)	0 (0%)	0 (0%)	1 (8.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)

P122: Fig. 2.

Conclusion. 6% of our AS patients required the use of at least 3 therapeutic families, with a mean of 4 lines of TT. Anti-TNF therapy is less used in first and second line in refractory patients, and the treatments are mainly modified through switching rather than cycling. The presence of psoriasis, osteoarthritis or anxious-depressive syndrome seems to be associated with refractory AS patients.

P123

PHYSIOTHERAPY IN AXIAL SPONDYLOARTHRITIS (PAXSPA STUDY) - EFFECTIVENESS OF MANUAL SPINAL MOBILISATION, A PARTIALLY BLINDED RCT WITHIN THE TWICS DESIGN

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Introduction/Objective. Individuals with axial spondyloarthritis (axSpA) are at an increased risk of developing restrictions in spinal mobility through structural damage of joints. Despite emerging evidence of the effects of manual spinal mobilisation therapy (MSM) in patients having already progressed to the advanced form of axSpA, the efficacy of this form of treatment has yet to be comprehensively studied in individuals on the wider spectrum of axSpA. The first trial of the PaxSpA study (Physiotherapy in axial Spondyloarthritis) at the Royal National Hospital for Rheumatic Diseases, Bath aims to further existing knowledge of the effectiveness of MSM on spinal mobility, disease activity, sleep, function, quality of life and work productivity. The objective is to investigate the short-term effects (<11 weeks) of MSM on clinical and functional outcomes amongst patients with axSpA.

Materials and methods. We recruited 101 participants into the PaxSpA cohort, of which 59 were eligible for the MSM trial, and 38 participants took part in the physiotherapy trial. The intervention group participants (n=20) received nine MSM sessions each over ten weeks between June to December 2021. Data was collected at baseline, 2 hours and 10 weeks. The 'Trials within Cohort' (TwiCs) design is best suited for examining long term conditions, was used to address our aims.

Results. Significant improvements were observed in the intervention group for spinal mobility, disease activity and sleep compared to the control group (Table 1). Significance was also observed for spinal mobility after the first intervention session compared to the control group. No significant correlations were found between functional data, work productivity and quality of life for both groups.

Conclusion. The results of this pragmatic study suggest that manual spinal mobilisation is effective and may be beneficial to patients with axSpA to help improve spinal mobility, sleep and disease activity in the short term.

P123: Table I.

Outcome measures	ANCOVA (*p*-value) time* intervention			
	2 hours	Effect size Cohen's *d*	10 weeks	Effect size Cohen's *d*
Mobility				
BASMI overall	**<0.001**	0.42	**<0.001**	**0.75**
Lumbar Side flexion score	**0.027**	0.29	**<0.001**	0.48
Tragus to wall (in cm)	0.150	0.30	**0.022**	**0.70**
Modified Schober score	0.248	0.11	0.055	0.44
IM Distance score	**0.019**	0.35	**0.020**	0.41
Cervical rotation score	**0.030**	**0.55**	**0.002**	**0.89**
PROM questionnaires				
BASDAI			**0.045**	0.39
BASFI			0.683	0.22
Jenkins Sleep Scale			**0.043**	0.25
ASQoL			0.323	0.16
Work Productivity AI			0.479	0.17

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