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GRAPPA 2024 Meeting: Advances in Psoriatic Disease Research From Pilot Grant Awardees

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ABSTRACT. Prioritizing and supporting trainee research in psoriatic disease (PsD) is a cornerstone of the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA). Each year, trainees and junior faculty are invited to submit proposals to GRAPPA to fund pilot research projects related to psoriasis or psoriatic arthritis. Projects can be in any of the following 4 categories: clinical science, translational science, basic science research, or combined PsD. GRAPPA remains committed to showcasing the trainee research supported by these grants at the annual meeting. The GRAPPA 2024 annual meeting and trainee symposium was held in Seattle, Washington, USA; a meeting highlight was the session dedicated to the pilot research grant projects led by trainees and faculty. This year, 27 submissions were received from 14 countries across North America, Europe, and Asia. Compared to prior years, an updated grant review process enhanced efficiency and created more opportunities for conversation among evaluators. A panel of 14 GRAPPA reviewers assessed the submissions, ultimately selecting 4 projects for funding. This meeting report aims to summarize the 2024 pilot research grant recipients and the project results from past grant recipients.

Key Indexing Terms: biomarker, GRAPPA, psoriasis, psoriatic arthritis

2024 announcement of GRAPPA pilot research grants

In 2024, the Group of Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) awarded pilot research grants to 4 recipients out of 27 submissions from 14 countries across North America, Europe, and Asia. The award recipients were:

Category: Basic science
Project: HIF1A in neutrophils: a potential crucial factor for psoriatic arthritis (PsA) through a positive feedback loop with IL-23
Recipient: Akihiro Nakamura, Queen's University, Canada
Mentor: Vinod Chandran

Category: Clinical science
Project: Advancing the case for timely weight loss trials in psoriatic disease by better understanding differences in body composition
Recipient: Lyn Ferguson, NHS Greater Glasgow and Clyde and University of Glasgow, UK
Mentors: Stefan Siebert and Naveed Sattar

Category: Translational science
Project: Cytokines carried by plasma EVs as potential biomarkers predicting response to TNFi in PsA
Recipient: Anaïs Makos, Keele University, UK
Mentors: Oksana Kehoe, Jan Herman Kulper, and Roshan Amarasena

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Category: Combined psoriatic disease
Project: Bone properties and biomechanics in patients with psoriatic disease: a prospective study with high-resolution peripheral quantitative computed tomography (HRpQCT)
Recipient: Giovanni Adami, University of Verona, Italy
Mentors: Paolo Gisondi and Maurizio Rossini

Oral presentations by past pilot research grant recipients

Dr. Vinod Chandran, Dr. Wilson Liao, and Prof. Kurt de Vlam co-chaired a session at the GRAPPA 2024 annual meeting, in which 7 past pilot grant recipients representing 5 countries presented their work in topics related to psoriatic disease (PsD), covering basic, translational, and clinical research. Topics included mechanisms of psoriasiform disease development, sex-specific biomarkers, psoriasis (PsO)-to–psoriatic arthritis (PsA) transition biomarkers, multimorbidity prevalence in the PsO-to-PsA transition, effect of treatment strategy on PsA progression, predicting PsA in patients with new-onset PsO, and toe dactylitis causes. The presentations in this session reflected the spectrum of GRAPPA's international impact in the area of PsD research.

TRPM4 function in Western diet–induced PsO via interleukin 23–mediated inflammation

Dr. Omar Alzayat and colleagues at the University of California, Davis, United States, explored the role of a gain-of-function mutation in the calcium-activated nonselective cation channel transient receptor potential cation channel subfamily M member 4 (TRPM4) in the development of psoriasiform disease in mice.[1] Previously, TRPM4 heterozygous mice were found to exhibit enhanced skin inflammation, increased ear and epidermal thickness, and higher mRNA levels of interleukin 17A (IL-17A) and S100A8 when fed a high-sugar, high-fat diet.

Bone marrow dendritic cells (BMDCs) collected from control mice and TRPM4 heterozygous mice were cultured in vitro using granulocyte-macrophage colony-stimulating factor (GM-CSF) and IL-4, followed by stimulation with lipopolysaccharide (LPS) to induce dendritic cell (DC) maturation. The expression of costimulatory markers in antigen-presenting cells (APCs) and cytokines was evaluated in BMDCs from TRPM4 heterozygous mice compared to BMDCs from TRPM4 wild-type (control) mice. BMDCs from TRPM4 heterozygous mice exhibited higher levels of inflammatory cytokines, including interferon γ, C-X-C motif chemokine ligand 9 (CXCL9), CXCL10, CXCL11, IL-23, IL-6, IL-12A, and tumor necrosis factor (TNF), with no observed differences in APC expression.

These high levels of multiple inflammatory cytokines combined with the increased epidermal keratinization previously observed in TRPM4 heterozygous mice suggested that TRPM4 heterozygous BMDCs may migrate more than wild-type BMDCs. Therefore, a C-C chemokine receptor type 7 (CCR7)–C-C motif chemokine ligand 21 (CCL21) chemotaxis assay was conducted to assess the migratory capacity of BMDCs from TRPM4 wild-type and heterozygous mice; BMDCs from TRPM4 heterozygous mice displayed a greater migratory capacity. Inhibiting TRPM4 with CBA (compound 5) or NBA (compound 6) reduced CCL21-induced BMDC migration, with NBA (compound 6) demonstrating greater inhibition of DC migration in TRPM4 heterozygous mice (Figure 1).

Primary keratinocyte cell cultures from wild-type and TRPM4 heterozygous mice were stimulated with 20 ng/mL of IL-17A and TNF, with and without 50 μM of NBA (compound 6). After 5 hours of incubation and real-time PCR analysis, NBA (compound 6) was shown to reduce stimulatory markers, including S100A8, S100A9, toll-like receptor 4 (TLR4), IL-36A, IL-36G, and colony-stimulating factor 1 (CSF1).

Wild-type and TRPM4 heterozygous mice were treated with 2,4-dinitrofluorobenzene (DNFB) to induce contact hypersensitivity to examine immune responses. DNFB-challenged TRPM4 heterozygous mice exhibited greater ear thickness and inflammation compared to DNFB-challenged wild-type mice and untreated TRPM4 heterozygous mice. Increased levels of CD45+ cells, a marker for leukocytes, were observed in the ears of TRPM4 heterozygous mice. Additionally, CD3+ T cells, CD4+ helper T cells, and Gr1+ myeloid cells were elevated. DNFB-treated TRPM4 heterozygous mice also displayed increased levels of CXCL10, which is involved in Th1 immune responses; a neutrophil chemoattractant called CXCL2; and S100A9, which is produced by neutrophils.

Overall, this work expanded previous findings in which TRPM4 heterozygous mice exhibited exaggerated psoriasiform dermatitis in a Western diet–fed model. BMDCs from these mice showed enhanced inflammatory reactivity and increased BMDC migration. The TRPM4 inhibitor NBA (compound 6) suppressed DC migration, and the TRPM4 mutation resulted in greater severity of nonpsoriatic dermatitis in a DNFB-induced contact hypersensitivity model.

Sex differences in serum proteomic biomarkers in PsA

Despite the equal prevalence of PsA in both sexes, male and female patients exhibit distinct clinical presentations and treatment responses.[2,3] The underlying biological mechanisms driving these differences have remained elusive. Steven Dang and colleagues from the University of Toronto, Canada, performed an untargeted proteomic analysis to identify sex-specific serum proteins and biological pathways in male and female individuals with PsA. Additionally, multivariable classification models were developed from the proteomic data to predict sex-specific disease status, and a variable importance analysis was conducted to identify proteins with the highest predictive values.

Clinical data and serum samples from 100 patients with PsA and 50 age- and sex-matched controls were obtained from the University of Toronto PsA cohort. Eligible patients with PsA met the Classification Criteria for Psoriatic Arthritis (CASPAR)[4] and were about to initiate systemic therapy for active musculoskeletal (MSK) manifestations of PsA. Patients were excluded if they had end-stage major organ failure, active cancer, or were taking systemic corticosteroids. The serum proteomic analysis was performed using the SomaScan 7K assay (SomaLogic).[5]

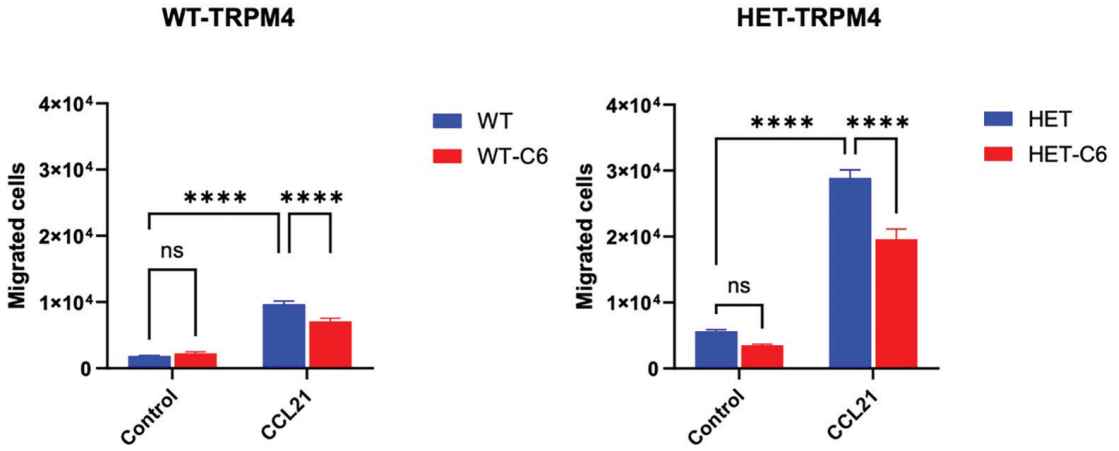

Figure 1. A chemotaxis assay with cultured BMDC of both WT-TRPM4 and HET-TRPM4 mice and the observed effect of NBA (compound 6) on BMDC. NBA shows significant inhibition in HET-TRPM4 BMDC migration. **** Statistically significant. BMDC: bone marrow dendritic cell migration; CCL21: chemokine ligand 21; HET: heterozygous; ns: not significant; TRPM4: transient receptor potential cation channel subfamily M member 4; WT: wild-type.

Through differential expression analysis, male and female individuals with PsA were compared to their respective controls to identify 731 deregulated serum proteins unique to male individuals with PsA and 31 unique to female individuals with PsA, with 200 proteins shared between both groups. Differential analysis between male and female individuals with PsA revealed 62 deregulated proteins, which were used to identify key biological pathways. Significant sex-specific pathways included Rho GTPase, angiogenesis, epithelial-mesenchymal transition regulators, necroptosis, insulin signaling, focal adhesion, IL-18, ErbB, Kit receptor, neutrophil extracellular trap formation (NETosis), phosphatidylinositol signaling, Fc gamma receptor–mediated phagocytosis, and platelet activation, signaling, and aggregation. Network analysis of the protein-pathway relationships highlighted male-specific proteins such as fatty acid synthase (FASN), glycogen phosphorylase B (PYGB), phosphodiesterase 5A (PDE5A), Lyn, Src, formin homology 2 domain containing 1 (FHOD1), nucleosome assembly protein 1 like 1 (NAP1L1), sphingosine kinase 1 (SPHK1), inositol polyphosphate-5-phosphatase B (INPP5B), tyrosine 3-monooxygenase/tryptophan 5-monooxygenase activation protein (YWHAH), and protein phosphatase 1 catalytic subunit gamma (PPP1CC). In contrast, the only identified female-specific protein was peptidylprolyl isomerase F (PPIF), a mitochondrial protein associated with the NETosis pathway; investigating this protein may provide insights into female-specific disease mechanisms in PsA.

Multivariable prediction models were constructed to distinguish PsA from controls for both male and female individuals. The models included logistic regression with elastic net, support vector machine, linear discriminant analysis, and random forest (RF). The models were validated using an 80-20 train-test split, with hyperparameters tuned through 10-fold cross-validation. The average area under the curve from 10 iterations exceeded 0.8 for all models. RF variable importance analysis highlighted leukotriene-A4 hydrolase as the top female-specific predictor and IL-36A, NIMA-related kinase 7 (NEK7), and PI3KCA/PIK3R1 as significant male-specific predictors. These proteins may play a central role in sex-specific PsA mechanisms and represent promising candidates for tailored biomarker panels. The study has a few limitations that should be considered. Given the heterogeneity of PsA, the sample size may not be sufficient to capture all differentially expressed proteins and enriched pathways. The cross-sectional design also limits assessment of the clinical effect of sex-specific protein changes. Additionally, the high number of protein variables relative to the cohort size raises the potential for overfitting in predictive models.

In summary, this study provides compelling evidence of sex-related biological differences in PsA. The identification of sex-specific proteins and pathways in PsA presents novel targets for future research focused on sex-based differences in the disease.

Potential biomarkers for characterization of patients with PsO at different risk levels of developing PsA

Dr. Caroline Gross and colleagues from the University Hospital Frankfurt, Germany, aimed to identify early biomarkers of MSK involvement in patients with PsO and to develop an immunological profile to assess the risk of PsA progression, with a focus on lipid biomarkers (lipidomics). Eligibility criteria required patients with PsO to meet ≥ 2 of the following 5 inclusion criteria for a PsA at-risk population: (1) presence of psoriatic nail matrix changes, (2) early onset of plaque PsO (onset of PsO at < 30 years of age), (3) positive family history of PsA, (4) duration of plaque PsO ≤ 20 years, or (5) absence of PsO involvement of palms and soles. Patients with PsO were screened for signs of subclinical MSK inflammation using MSK ultrasound (MSUS) and near-infrared fluorescence optical imaging (NIR-FOI). MSUS was used as a gold standard diagnostic adjunct to clinical assessment.

A total of 25 patients were enrolled. On evaluation, 9 patients (36%) showed pathological power Doppler (PD)

activity on MSUS and were categorized as having subclinical PsA. Older patients had significantly higher pathological PD activity ($P = 0.03$) and more frequent polyarticular involvement ($P = 0.049$). Patients with shorter PsA disease duration (< 20 years) had more frequent inflammatory joint changes ($P = 0.04$), whereas early disease onset (before the age of 30) was less frequently associated with these changes ($P < 0.001$). NIR-FOI showed no significant difference between patients with PsO with or without pathological US findings. A comparison of endocannabinoid and sphingolipid concentrations showed no clear separation between patients with PsO and subclinical PsA. The endocannabinoid, 2-arachidonoylglycerol (2-AG), was decreased in both PsO and PsA compared to healthy controls ($P < 0.001$) with no difference between PsO and PsA ($P = 0.99$). After inclusion of additional samples from previous studies and a healthy control group, increased ceramide 18:1;O2/16:0 levels were observed in PsO and PsA, whereas increased ceramide 18:1;O2/18:0 levels were observed in subclinical PsA but not in PsO ($P < 0.001$). This difference may be due to the significantly older age observed in the PsA group or a higher rate of hypertension in the PsA subpopulation. Further analysis of oxylipins, tryptophan and related metabolites, and lysophosphatidic acids; screening of abundant polar metabolites and lipids; and proteomic analysis are ongoing.

Multimorbidity in PsO as a risk factor for PsA

Dr. Paras Karmacharya and colleagues from the Vanderbilt University Medical Center, the Mayo Clinic, and the University of Pennsylvania, United States, examined multimorbidity in PsO and its association with the development of PsA. A retrospective cohort study was performed using the Rochester Epidemiology Project.[6,7] Population-based incidence (from 2000 to 2009) and prevalence (as of January 1, 2010) cohorts of PsO were identified by manual chart review. A cohort of individuals without PsO (comparators) were identified (1:1 matched on age, sex, and county). Morbidities were defined using ≥ 2 Clinical Classification Software codes ≥ 30 days apart within the prior 5 years. PsA was defined using CASPAR criteria. Chi-square and Wilcoxon rank-sum tests were used to compare morbidities, and age-, sex-, and race-adjusted Cox models were used to examine the association of baseline morbidities in PsO with development of PsA.

Among 817 patients with incident PsO in the study, the mean age was 45.2 years with 52% being female and 82% with moderate/severe PsO. No multimorbidity differences were found between incident patients with PsO and comparators (Figure 2). However, in the 1088 patients with prevalent PsO, multimorbidity was significantly more common compared to 1086 comparators (odds ratio [OR] 1.35 and OR 1.48 for ≥ 2 and ≥ 5 morbidities, respectively). Over a median 13.3-year follow-up, 23 patients (cumulative incidence: 2.9% by 15 years) developed PsA. Multimorbidity (≥ 2 morbidities) was associated with a 3-fold higher risk of developing PsA. The study concluded that multimorbidity was more common in the prevalent but not incident cohort of PsO compared to the general population, suggesting patients with PsO may experience accelerated development of multimorbidity. Moreover, multimorbidity at PsO onset significantly increased the risk of developing PsA, highlighting the importance of monitoring multimorbid patients with PsO for the development of PsA.

Assessing the effect of different treatment strategies on damage progression and functional decline in PsA

PsA is a severe, immune-mediated inflammatory disease characterized by significant disability and increased mortality.[8] The chronic inflammation associated with PsA targets synovial and entheseal tissues, leading to erosive and proliferative bone changes, deformities, functional impairment, and an elevated risk for osteoporosis. Biologic/targeted synthetic disease-modifying antirheumatic drugs (b/tsDMARDs) have been shown to inhibit radiographic progression and have demonstrated benefits in preserving higher bone mass in treated patients.[9-11] However, there is a lack of evidence regarding the long-term effects of b/tsDMARDs on joint damage progression and bone loss in real-world cohorts. Additionally, the influence of specific factors such as disease activity on damage progression and bone loss over time is insufficiently understood.

To address these gaps, Dr. David Simon and colleagues from Charité - Universitätsmedizin Berlin and University Hospital Erlangen, Germany, aimed to use data from a prospective, real-world PsA cohort to assess the time course of damage progression and bone loss; evaluate the longitudinal effect of b/tsDMARDs; and investigate the association between disease activity and functional status (assessed using the Health Assessment Questionnaire), damage progression, and bone loss. The study included patients from prospective cohorts of the University Hospital Erlangen, comprising PsA and PsO cohorts and comparator groups of seronegative and seropositive rheumatoid arthritis (RA). All included patients had ≥ 1 high-resolution computed tomography scan of the distal radius/metacarpophalangeal joints. Image evaluation included assessing damage progression, such as erosions or structural entheseal lesions (osteoproliferations at the entheses), and volumetric bone mineral density.[12]

The study analyzed a total of 467 patients, with a mean age of 53.1 (SD 12.7) years. The 151 patients with PsA were, on average, aged 51.7 (SD 12.2) years; the 55 patients with PsO 47.1 (SD 12.0) years; the 68 patients with seronegative RA 58.8 (SD 12.2) years; and the 193 patients with seropositive RA 53.9 (SD 12.5) years. Key findings revealed that bone quality deteriorated over time in patients with PsA and RA, but not in those with PsO (Figure 3). In patients with PsA, bone loss accelerated with increasing disease activity over time. Damage progression (new or enlarging erosions and/or structural entheseal lesions) was observed in up to half of the patients with PsA over 7 years, correlating with worsening disability. Divergence in bone density was noted based on b/tsDMARD exposure, defined as intake of b/tsDMARD, though without significant pairwise differences. There was no significant increase in erosion proportion among patients with PsA exposed to b/tsDMARDs, despite their higher disease burden.

Taken together, this study demonstrated that effective

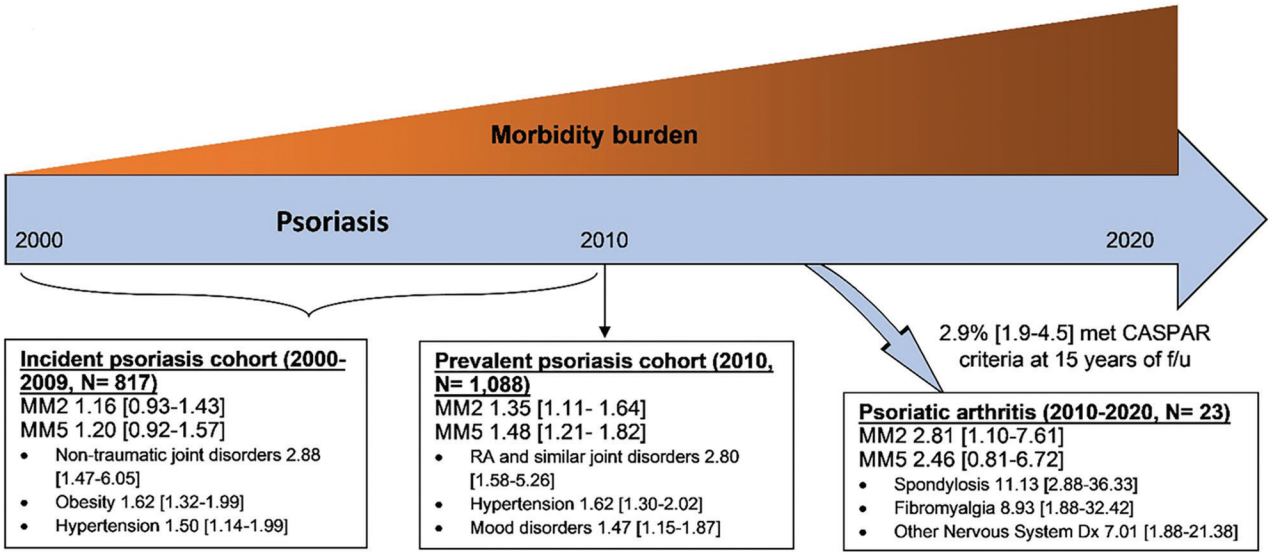

Figure 2. Schema of multimorbidity in the incident and prevalent psoriasis cohorts and its association with the incidence of psoriatic arthritis. CASPAR: Classification Criteria for Psoriatic Arthritis; Dx: diagnosis; f/u: follow-up; MM2: ≥ 2 morbidities; MM5: ≥ 5 morbidities; RA: rheumatoid arthritis.

control of disease activity in patients with PsA can significantly contribute to preventing damage progression and bone loss. These findings emphasize the importance of targeted treatment strategies to mitigate long-term joint damage and functional decline in patients with PsA, underscoring the potential of precision medicine in improving patient outcomes.

Prediction of PsA in new-onset PsO

Dr. Axel Svedbom and colleagues from the Karolinska Institutet in Stockholm, Sweden, identified biomarkers and derived a prediction algorithm for the development of PsA in patients with new-onset PsO. Data were obtained from the Stockholm Psoriasis Cohort (SPC), a prospective inception cohort study in which patients were examined within 1 year of first disease onset, and then 5 and 10 years thereafter.[13,14] The study was also linked to national Swedish administrative registers to complement data from the examinations. Using patients aged ≥ 18 years, traditional statistical analysis was applied to identify biomarkers for the development of PsA, and statistical learning methods were implemented to derive prediction algorithms.

Six hundred twenty-nine participants (median age 43 [IQR 31-57] years; 44% women) were eligible for analysis and 190 participants (30%) developed PsA. Logistic regression models were used to estimate associations between 258 potential predictors from the enrollment examination and PsA development. Predictors were divided into 10 categories, each containing 5-117 variables, and the *P* values of the estimates were compared to the Benjamini-Hochberg (BH) critical values assuming a false discovery rate of 10% for each category. Among the 43 potential clinical predictors, 31 (72.1%) had *P* values below their BH critical values. In contrast, among the biomarkers, 16/258 variables (6.2%) had *P* values below their BH critical values. However, there was a

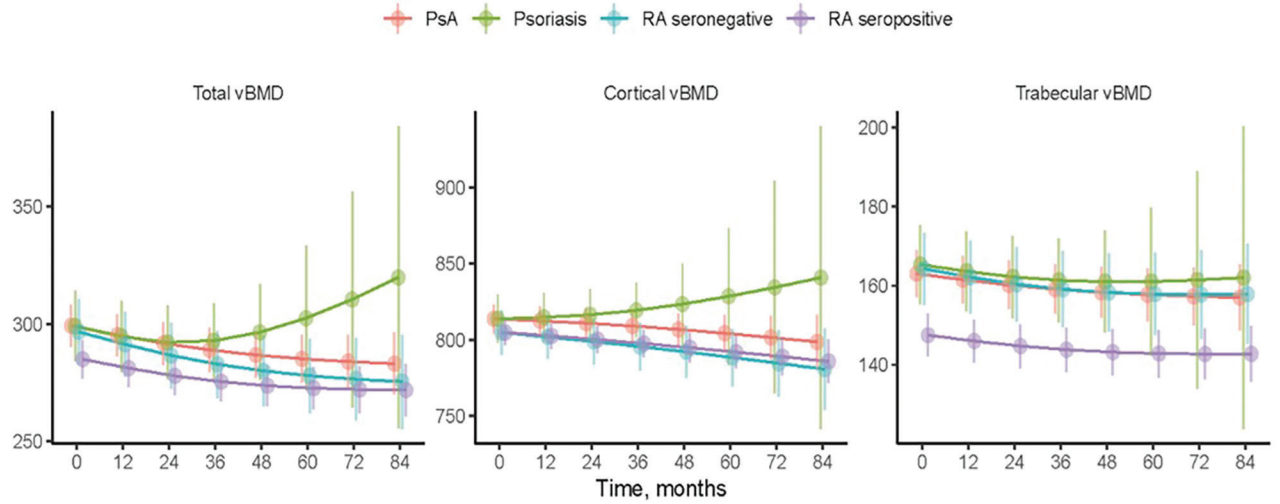

Figure 3. Bone quality deterioration over time in patients with PsA or RA but not in patients with psoriasis. PsA: psoriatic arthritis; RA: rheumatoid arthritis; vBMD: volumetric bone mineral density.

marked difference between different biomarker categories: 4/15 markers for systemic inflammation (27%) and 9/25 lipid markers (36%) had *P* values below the BH critical values, compared to 1/42 (2.3%) for lipid subfractions, 0/15 (0%) for nonlipid metabolic markers, and 2/117 (1.7%) for genetic markers.

Additionally, a white-box algorithm for PsA prediction was derived using recursive partitioning in a conditional inference framework. Current arthralgia, pain at 1 of 7 specified sites during the last year, peripheral enthesitis, dactylitis, and high-sensitivity C-reactive protein were the variables used to derive an algorithm with a *C*-statistic of 0.82, indicating good discrimination (Figure 4A). The best black-box model used an RF algorithm and had a *C*-statistic of 0.88, indicating excellent discrimination. In the RF algorithm, arthralgia and symptoms related to PsA were most important, but inflammatory markers also had a comparatively large influence—accounting for 3/10 most important predictors—potentially reflecting nonlinear and interaction effects not captured by a simpler model (Figure 4B).

This study not only highlights the importance of patient-reported and anamnestic data for PsA prediction but also indicates that several biomarkers have substantial predictive power. The prediction models developed have good discriminatory power and could be used to identify patients at high risk of PsA.

Is toe dactylitis in PsA related to trauma caused by plantar shear stress? A case control pilot study

It has been hypothesized that trauma may lead to the development of dactylitis in PsA due to the "deep" Koebner phenomenon. Dactylitis is most often seen in the feet, particularly the fourth toe. Having previously found no differences in plantar pressure in patients with dactylitis, Dr. Philip Helliwell and colleagues from the University of Leeds, United Kingdom, aimed to develop a novel plantar shear platform and to measure plantar shear in control subjects and subjects with PsA. A multiarray plantar shear platform was developed and shear stresses measured in 12 control subjects, 12 subjects with PsA and no history of toe dactylitis, 17 subjects with a history of toe dactylitis, and 6 subjects with active toe dactylitis. Initial analysis found that shear stresses did not differ between the groups, but the analysis is ongoing with more detailed examination of the 2 shear axes to come.

Discussion

Seven GRAPPA pilot research grant awardees presented important findings and highlighted innovative approaches for investigating the pathogenesis of PsD.

Alzayat and colleagues found that a gain-of-function mutation in TRPM4 exacerbates psoriasiform dermatitis and enhances inflammatory responses in mice, particularly under a high-sugar, high-fat diet. The mutation in TRPM4 also contributed to more severe dermatitis in a DNFB-induced contact hypersensitivity model. Mechanistically, the TRPM4 mutation increased DC migration and cytokine production, whereas TRPM4 inhibitors reduced this migration.

Dang and colleagues identified sex-specific serum proteins and biological pathways in PsA, revealing clinical and molecular differences between male and female individuals with the disease. Multivariable classification models successfully predicted sex-specific disease status, with the findings highlighting potential targets for future sex-based PsA research.

Gross and colleagues aimed to identify early biomarkers of MSK involvement in patients with PsO and develop an immunological profile to assess the risk of PsA progression, focusing on lipid biomarkers. Although no clear lipid-based distinctions between patients with PsO and subclinical PsA were found, increased levels of specific ceramides were observed in subclinical PsA, suggesting potential biomarkers for PsA risk assessment.

Karmacharya and colleagues investigated the prevalence of multimorbidity in patients with PsO and PsA, finding that multimorbidity was more common in prevalent patients with PsO compared to the general population, but not in incident PsO cases. Having multiple comorbidities at the onset of PsO significantly increased the risk of developing PsA.

Simon and colleagues assessed the long-term effects of b/tsDMARDs on damage progression and bone loss in patients with PsA, revealing that disease activity exacerbated bone loss and joint damage. Effective disease control with b/tsDMARDs was shown to help prevent damage progression and preserve bone health, emphasizing the importance of targeted treatments to improve long-term outcomes in PsA.

Svedbom and colleagues identified biomarkers and developed prediction algorithms for the transition to PsA in patients with new-onset PsO, using data from the SPC. The derived prediction models were based on clinical symptoms and inflammatory biomarkers and demonstrated strong discriminatory power, offering a tool to identify patients at high risk of developing PsA.

Helliwell and colleagues developed a novel plantar shear platform to measure shear stresses in patients with PsA as a proposed cause of toe dactylitis. Initial analysis found no significant differences between control subjects and patients with PsA, but further examination is ongoing.

Attendees at the GRAPPA meeting were excited to view the results of these research projects and provided meaningful feedback during the question-and-answer periods. Pilot research grants will continue to support trainee research in PsD. The 2024 pilot research grant recipients will be invited to present their findings at the next GRAPPA annual meeting to be held in July 2025 in Bogota, Colombia. Thank you to the pilot research grant reviewers, and congratulations to all awardees.

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COMPETING INTERESTS

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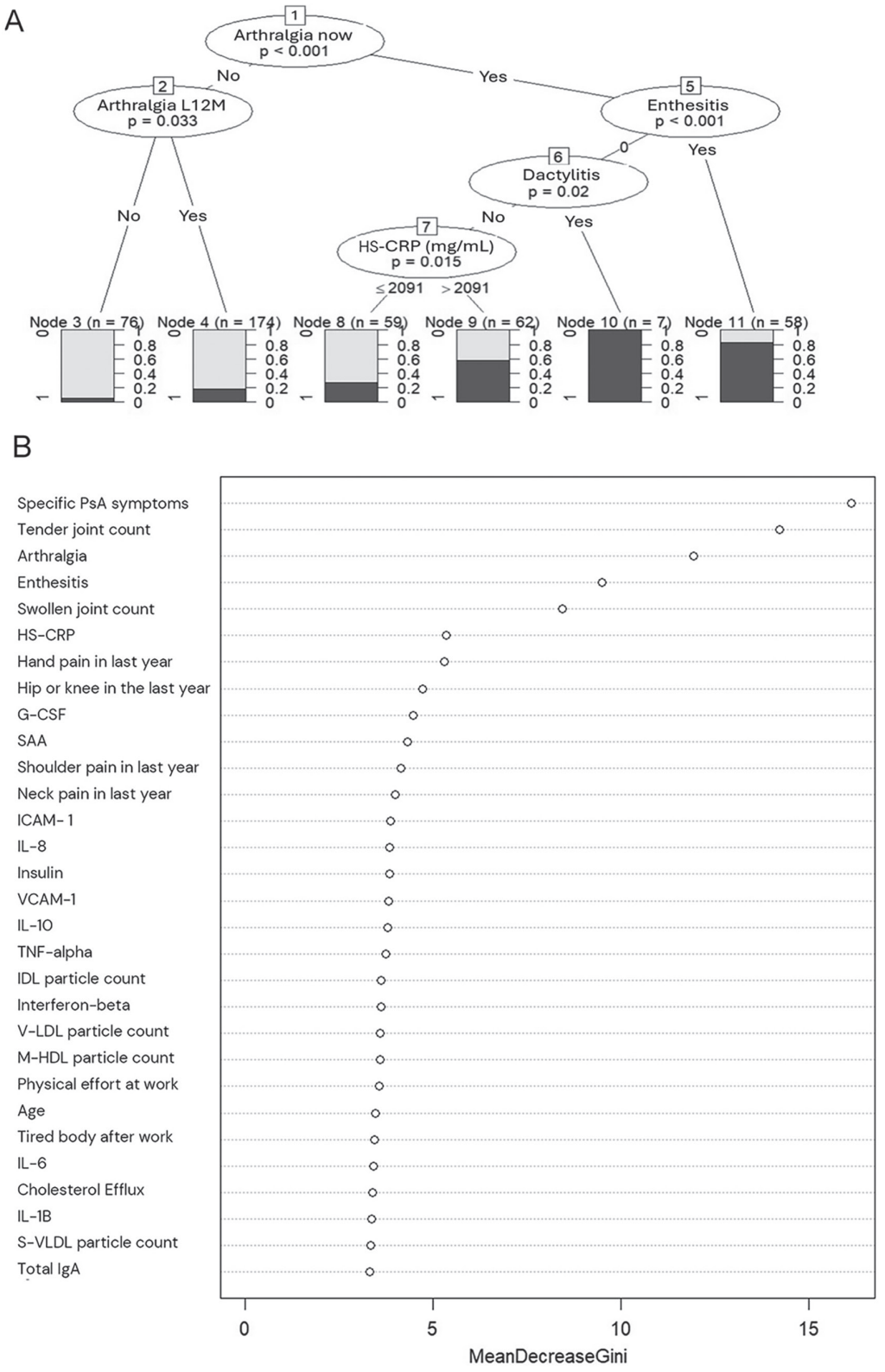

Figure 4. PsA biomarker prediction models may help identify patients at risk of developing PsA. (A) White-box model for PsA prediction at onset of PsO derived using recursive partitioning. (B) Estimated variable importance for predicting PsA at onset of PsO from a random forest model. G-CSF: granulocyte colony-stimulating factor; HS-CRP: high-sensitivity C-reactive protein; ICAM-1: intercellular adhesion molecule 1; IDL: intermediate-density lipoprotein; IL: interleukin; M-HDL: medium-sized high-density lipoprotein; PsA: psoriatic arthritis. PsO: psoriasis; SAA: serum amyloid A; S-VLDL: small very low-density lipoprotein; TNF: tumor necrosis factor; VCAM-1: vascular cell adhesion protein 1; V-LDL: very low-density lipoprotein.

ETHICS AND PATIENT CONSENT

Institutional review board approval and patient consent were not required.

PEER REVIEW

As part of the supplement series GRAPPA 2024, this report was reviewed internally and approved by the Guest Editors for integrity, accuracy, and consistency with scientific and ethical standards.

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