Podium Session A: Thursday, February 27, 2025, at 8:00-9:15 AM MT

POD01

Distinct Symptom Clusters Predict Initial Response to Mtx in Adults with New Ra: a Longitudinal Analysis of the Canadian Early Arthritis Cohort

Susan Bartlett (McGill University & Research Institute MUHC, Montreal); Clifton Bingham (Johns Hopkins Arthritis Center, Baltimore); Orit Schieir (McGill University, Montreal); Marie-France Valois (McGill University, Montreal); Janet Pope (Divisions of Rheumatology, Epidemiology, and Biostatistics, Department of Medicine, Western University, London); Carter Thorne (Centre of Arthritis Excellence (CaRE), Newmarket); Louis Bessette (Université de Laval, Centre Hospitalier de l'Université Laval, Quebec); Carol Hitchon (University of Manitoba, Winnipeg); Gilles Boire (Université de Sherbrooke, Sherbrooke); Diane Tin (Centre of Arthritis Excellence, Newmarket); Edward Keystone (University of Toronto, Toronto); Glen Hazlewood (University of Calgary, Calgary); Vivian Bykerk (The Hospital for Special Surgery, New York); CATCH Canadian Early Arthritis Investigators (Toronto)

Objectives: Symptom clusters, the co-occurrence of 2+ symptoms that are stable and independent of other clusters, may share underlying mechanisms and lead to different disease outcomes, particularly if symptoms are under identified and undertreated. If patients with distinct symptom clusters display unique patterns of treatment response, this can help better match patients to treatments to optimize disease control and QoL. We previously applied a person-centered approach to group newly diagnosed early RA patients based on patient-reported pain, fatigue, depression, and anxiety levels. We compared outcomes of initial 6-month MTX response across new-onset RA patients by symptom clusters.

Methods: Data were from new RA patients (symptoms< 1 year) in the Canadian Early Arthritis Cohort (CATCH) between Jan 2017-Aug 2022 with active disease who were starting MTX. Participants underwent standardized assessments and completed PROMIS29 at 0, 3, and 6 months. Latent Class Analysis yielded 4 distinct groups: 1. Minimal Symptoms; 2. Mild symptoms; 3. Moderate symptoms; and 4. Moderate-Severe symptoms. Multivariable mixed effects regression was used to estimate trajectories of CDAI, disability, and participation over the first 6-months of MTX treatment across cluster groups updated at each time point and adjusting for age, sex, race, education, smoking, obesity, comorbidities, symptom duration, seropositivity, and RA treatments.

Results: At diagnosis, 310 ERA adults had a mean (SD) age of 56 (14), and were mostly female (67%), White (78%) with a CDAI of 29.3 (13.2). All started MTX, with no significant difference in dose or strategy across cluster groups. All groups had high disease activity (CDAI) scores at enrolment, though patients reporting severe pain, fatigue, depression, and anxiety symptoms were significantly younger, more often had a history of depression, had the highest mean CDAI, Patient Global and stiffness scores, and greater impairments in function and participation than other groups. In adjusted regression models, all groups improved over 6 months but patients in severe symptom category consistently displayed worse trajectories of disease activity, function, and participation over the 6-month follow-up period (Figure).

Conclusion: Beyond disease activity at RA onset, evaluating the presence of specific symptom clusters in the first 6 months of new RA could better identify individuals at risk for poorer MTX response. These individuals may benefit from earlier, more aggressive pharmacologic and psychosocial interventions. Addressing both physical and emotional symptoms may improve

physical and social function in early RA helping to preserve autonomy, workforce participation, and QOL.

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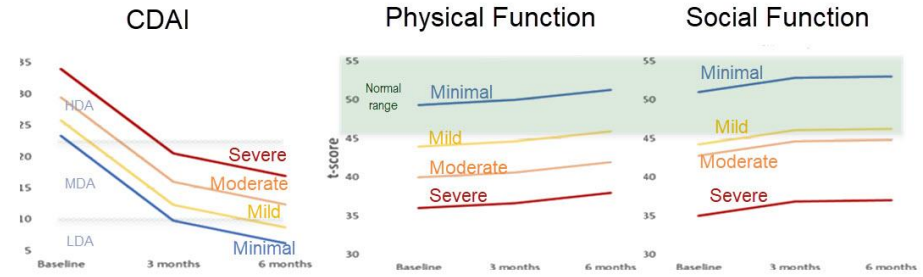

Predicted trajectories of CDAI, Physical Function and Social Function by symptom clusters over first 6 months

POD02

Association Between Symptomatic Knee Osteoarthritis and Blood Glucose Control in Persons with Type 2 Diabetes

Lauren King (University of Toronto, Toronto); Alanna Weisman (University of Toronto, Toronto); Baiju Shah (University of Toronto, Toronto); Robert Goldberg (Trillium Health Partners, Mississauga); Amish Parikh (Trillium Health Partners, Mississauga); Ian Stanaitis (Women's College Research Institute, Women's College Hospital, Toronto); Rosane Nisenbaum (St. Michael's Hospital, Toronto); Esther Waugh (University of Toronto, Toronto); Lorraine Lipscombe (University of Toronto, Toronto); Gillian Hawker (University of Toronto, Toronto)

Objectives: There is a high prevalence of knee osteoarthritis (OA) in people with type 2 diabetes (T2D), and knee OA increases risk for diabetes complications. Our objective was to assess the association between symptomatic knee OA and attainment of target blood glucose levels in individuals with T2D.

Methods: In this cross-sectional study, we recruited individuals with T2D aged ≥45 years from diabetes clinics at three academic hospitals in Toronto. Participants completed standardized online questionnaires that assessed demographics, comorbidities, height and weight, and joint symptoms. From clinic records we abstracted participants' most recent HbA1c (within 3 months). Knee OA was defined as fulfilling NICE criteria. We considered blood glucose control at target if HbA1c was ≤7.0%. We used multivariable logistic regression to assess the association between knee OA and being at blood glucose target, adjusting for age and gender. We then examined the effect of further adjusting for body mass index (BMI). In secondary analyses, we repeated modelling with exposure of interest knee OA with knee pain ≥20/100 on pain numeric rating scale (NRS) (yes/no).

Results: We included 351 participants. Mean age was 66.9 (SD 9.8) years, 50.7% women, mean BMI 29.1 (SD 6.8) kg/m2, and 28.5% fulfilled NICE criteria for knee OA. Mean HbA1c was 7.4 (SD 1.2); 44% had HbA1c at target (≤7.0%). In univariable analysis, those with knee OA had lower odds of being at target (OR 0.60, 95% CI 0.37 to 0.97). Results were similar after adjusting for age and gender (OR 0.59, 95% CI 0.36 to 0.95). When further adjusting for BMI the effect of knee OA was attenuated and was not statistically significant (OR 0.65, 95% CI 0.39 to 1.06). When exposure of interest was knee OA with self-reported pain ≥20/100, we found a stronger negative association; this met statistical significance even after adjusting for BMI (OR

0.59, 95% CI 0.35 to 0.997) (Figure 1).
Conclusion: Individuals with T2D with knee OA are less likely to be at the recommended target for glycemic control. This association was stronger for those who currently reported pain and remained significant even after adjusting for BMI. This suggests that symptomatic knee OA may increase risk of diabetes complications through worse glycemic control, and symptom severity is likely important. Further studies are needed to better understand this relationship, as well as the role of additional mechanisms by which knee OA could lead to diabetes complications such as cardiorespiratory fitness and/or systemic inflammation.

POD03

The Deubiquitinase Molecule Trabid: A Novel Therapeutic Target for Axial Spondyloarthritis

Archita Srinath (University of Toronto, Toronto); Daniele Mauro (Universita della Campania, Naples); Mansi Aparnathi (University Health Network, Toronto); Mariia Korshko (University Health Network, Toronto); Shaghayegh Foroozan Boroojeni (University Health Network, Toronto); Igor Jurisica (Schroeder Arthritis Institute, Krembil Research Institute, University Health Network, Data Science Discovery Centre for Chronic Diseases, Krembil Research Institute, University Health Network and Departments of Medical Biophysics and Computer Science, Toronto); Francesco Ciccia (Università degli Studi della Campania, Naples); Nigil Haroon (Department of Medicine/Rheumatology, University Health Network, Schroeder Arthritis Institute, University of Toronto, Toronto)
Objectives: Inflammation and new bone formation are important disease mediators in Axial Spondyloarthritis (AxSpA). Recent studies in mice show that the deubiquitinase TRABID epigenetically controls the expression of IL12/23 through JMJD2D (Jumonji Domain-Containing Protein 2D). Furthermore, TRABID also upregulates EZH2 (Enhancer of zeste homolog 2), a molecule that has been implicated in pro-osteogenic pathways in breast cancer. This study aims to study the functional and clinical relevance of TRABID in AS pathogenesis.
Methods: To evaluate differential expression of TRABID in human and mouse AxSpA, human spine, gut, bone marrow and synovium and mouse ankle and tail were analyzed by immunohistochemistry and qPCR. Then, to assess the effect of TRABID inhibition on cytokine production, THP-1 cells (monocyte cell line) and splenocytes from SKG mice were incubated with lipopolysaccharide (LPS) and varying concentrations of NSC112200, an inhibitor of TRABID. ELISA detected cytokine production after 24 hours and western blot evaluated expression of EZH2 and JMJD2D. To evaluate TRABID's role in osteogenesis, TRABID was knocked down in Saos-2 cells (osteoblast cell line) using siRNAs to evaluate the effect of TRABID expression on osteogenesis. RNA was extracted to evaluate osteogenic gene expression by qPCR and RNA sequencing. Calcium mineralization was evaluated by Alizarin Red staining. Finally, to assess the in-vivo clinical relevance of TRABID inhibition, curdlan injected SKG mice were treated for 8 weeks with NSC112200.
Results: Our results showed that TRABID is significantly upregulated in all human tissue tested and SKG mouse ankle and tail compared to healthy controls. THP-1 cells and SKG mouse splenocytes treated with NSC112200 and LPS together showed a dose dependent inhibition of TNFa, IL1b and IL23 production when compared with cells treated with only LPS. Furthermore, TRABID inhibition showed a significant downregulation in both JMJD2D and EZH2 protein levels. TRABID knockdown in Saos-2 cells showed significant suppression of pro-osteogenic

genes SP7, Runx2 and alkaline phosphatase and failed to mineralize. RNA sequencing confirmed pathways related to TGFb signaling, bone remodeling and endochondral ossification were suppressed. Osteogenic media upregulated TRABID expression over time, compared with cells treated with unconditioned growth media. Finally, NSC112200 dose dependently suppressed clinical and histopathological signs of arthritis and inflammation in curdlan injected SKG mice. Nanostring analysis showed downregulation of genes related to immune cell migration to the inflamed joint with TRABID inhibition.
Conclusion: TRABID inhibition is a promising new target to control disease progression in AxSpA. Future work will address downstream cellular mechanisms of TRABID.

POD04

Safety and Tolerability of a Combination of Curcumin, Omega-3 and Vitamin-D: Results from the Pascod Study, an Ra Prevention Protocol

Halle Cochrane (); Caitlin McFadyen (University of Manitoba, Winnipeg); Xiaobo Meng (University of Manitoba, Winnipeg); Kale Mayor (University of Manitoba, Winnipeg); Rhonda Silva (University of Manitoba, Winnipeg); Dylan Mackay (University of Manitoba, Winnipeg); Hani El-Gabalawy (University of Manitoba, Winnipeg); Liam O'Neil (University of Manitoba Faculty of Health Sciences, Winnipeg)

Objectives: Rheumatoid Arthritis (RA) is an inflammatory autoimmune arthritis for which there is currently no cure. Recent research has focused on prevention strategies in individuals at increased risk of RA with the aim of reducing this risk. One such strategy involves dietary interventions. In a secondary analysis of a large prospective clinical trial (VITAL), vitamin-D and Omega-3 fatty acid supplementation have shown promise in reducing incident RA1. In a collagen induced arthritis murine model of RA, we showed that a diet supplemented with Curcumin, Omega-3 and Vitamin-D (COD) reduced arthritis severity and incidence when given during the induction phase2. Based on these considerations, we are planning a prospective RA prevention trial using COD in anti-citrullinated protein antibody (ACPA) positive individuals. However, there are no clinical trials which have evaluated the combined use of COD in humans. Thus, we conducted PASCOD (Pilot Assessment and Safety of COD) to evaluate the safety and tolerability of COD in healthy human volunteers.
Methods: Healthy volunteers aged 18-49 years with no history of RA or other autoimmune diseases were recruited (n=50) for a 4-week open-label pre-post design. Baseline visits included overall health and medications survey, anthropometric measures, functional status (RAPID3), a joint symptom questionnaire and a clinical assessment of 44 joints, all of which were repeated at the final visit. Blood specimens were collected for routine hematology and biochemistry. The study was powered to detect the rate of common side effects (>5% frequency), which was the primary outcome.
Results: Most reported side effects were mild (81.8%), with the most common being heartburn (32%), fishy taste (30%), and abdominal bloating (24%), which mostly improved over time. Mean adherence was 97% for all 3 supplements. Reflective of this, we observed increases in eicosapentaenoic acid (EPA, $p<0.0001$), docosahexaenoic acid (DHA, $p<0.0001$) levels measured in plasma by gas chromatography, and Vitamin D levels ($p=0.01$). Despite the absence of inflammatory arthritis, RAPID3 scores significantly decreased over the trial ($p=0.048$), driven by a reduction in self-reported joint pain visual analogue scale (VAS, $p=0.002$). Self-reported joint pain score ($p=0.008$), fatigue VAS ($p=0.008$) and minutes of AM stiffness ($p=0.01$) all reduced after COD treatment.

Conclusion: Overall, four weeks of COD was well-tolerated, with high adherence and mild side effects which mostly improved over time. We observed improvements in self-reported symptoms relevant to individuals at-risk for future RA development. These data suggest that COD supplementation is a feasible strategy for consideration in an RA prevention trial.

POD05

Work-Related Disability and Function in Systemic Lupus Erythematosus (SLE): Outcomes of an Exploratory Study from Different Canadian Centres

Behdin Nowrouzi-Kia (University of Toronto, Toronto); Zahi Touma (Schroeder Arthritis Institute, Krembil Research Institute, Toronto Western Hospital, Toronto); Antonio Avina-Zubieta (University of British Columbia Faculty of Medicine; Arthritis Research Canada, Vancouver); Mary Fox (York University, Toronto); William Shaw (UConn Health, Farmington); Maggie Ho (UHN, Toronto); Qixuan Li (University Health Network, Schroeder Arthritis Institute, Krembil Research Institute, Toronto); Catherine Ivory (University of Ottawa, Department of Medicine, Division of Rheumatology, Ottawa); Paul Fortin (Université Laval, CHU de Québec, Quebec); Stephanie Keeling (University of Alberta, Division of Rheumatology, Edmonton); Jennifer Reynolds (University of British Columbia, Department of Medicine, Division of Rheumatology, Vancouver); Derek A. Haaland (The Waterside Clinic, Barrie ON Canada, and McMaster University, Hamilton ON Canada, Hamilton); Janet Pope (Divisions of Rheumatology, Epidemiology, and Biostatistics, Department of Medicine, Western University, London); Lily S. H. Lim (Department of Paediatrics, University of Manitoba, Winnipeg); Murray Urowitz (Schroeder Arthritis Institute, Krembil Research Institute, Toronto Western Hospital, Toronto); Laura Patricia Whittall Garcia (UHN, Toronto); Dafna Gladman (Schroeder Arthritis Institute, Krembil Research Institute, Toronto Western Hospital, University Health Network, University of Toronto, Toronto)

Objectives: Systemic lupus erythematosus (SLE) has a significant and long-lasting impact on work outcomes, is a source of long-lasting work disability, and presents challenges with participation in activities of daily living. This study aimed to create a functional profile for patients with SLE. A functional profile is defined as activities of daily living and those related to work functioning (activities of daily living).

Methods: A cross-sectional investigation was carried out across six Canadian facilities, comprising six academic institutions and one community-based facility. Clinical measurements were obtained, including the SLEDAI-2K and ACR/SLICC Damage Index (SDI) and patients' medications. Patients completed the Work Role Functioning Questionnaire v2.0 (WRFQ), the World Health Organization—Disability (WHO-DAS) Assessment Schedule 2.0 (WHO-DAS), and the Beck Depression Inventory (BDI-II). Descriptive and inferential statistics were computed for the demographic, clinical, and functional outcomes. Univariate and multivariate regression analyses to study the association with WHO-DAS and WRF were performed.

Results: 404 patients were studied; mean age was 47.0±13.71 years and 91.8%% were female (64.7% White, 12.4% Black, 6.7% Chinese and 16.2% other races) with a mean SLE duration of 15.7 ±11.8 years. The total mean score for the WRFQ was 71.51.8±23.5. The WRFQ subscale mean scores were also reported for work scheduling demands (66.8±28.8), work output demands (71.1±25.6), physical demands (67.3±27.9), mental and social demands (74.4±22.8) and flexibility demands (75.0±24.7) [Figure 1] - comparison to the general working population). The WHO-DAS 2.0 total mean score was 25.1±9.71, representing approximately the 93.8th

population percentile, meaning that only about 6.1% of the population scored higher (more disabled) than our sample. In the multivariate analysis, sex (Female), damage (SDI), prednisone dose, fatigue severity score, Work Role Functioning total scores, presence of fibromyalgia, Role Emotinal SF-36, depression and pain were associated with increased disability. Similarly, fatigue severity score, depression, and pain were associated with decreased WRF total scores.
Conclusion: This Canadian study confirmed that patients with SLE suffers from high level of disability and functional decline and as measured by WHO-DAS and WRF. Several factors were associated with disability and functional decline including accrued damage, presence of fatigue and fibromyalgia, depression, pain and prednisone dose. Developing the initial functional profile of work disability will facilitate a multidisciplinary approach to enhance the care and management of work disabilities and related functional outcomes. **Supported by a CIORA grant**

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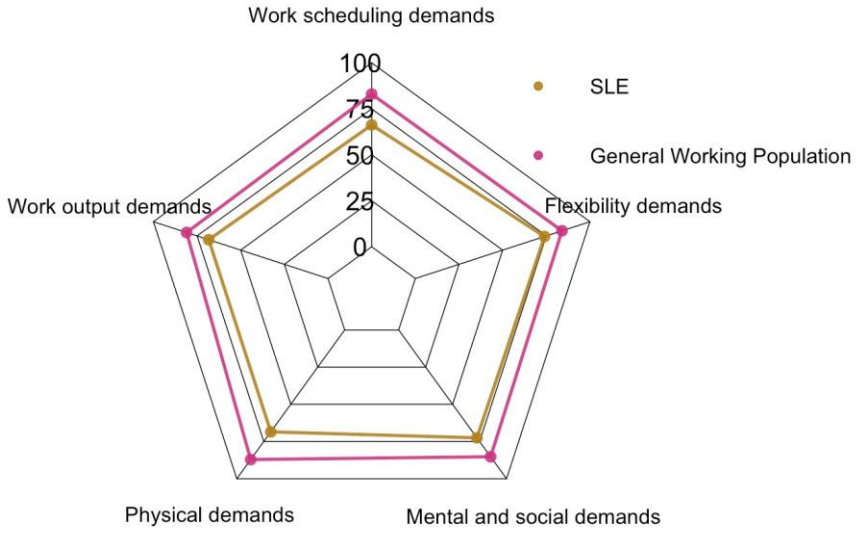

POD06
Anti-Integrin αvβ6 Autoantibodies as a Biomarker for Ulcerative Colitis in Patients with Axial Spondyloarthritis
Enoch Yau (University of Western Ontario, London); Robert Inman (University of Toronto, Toronto)
Objectives: An association between axial spondyloarthritis (AxSpA) and inflammatory bowel diseases (IBD) has been well described. IgG autoantibodies against the integrin avb6 have been recently identified in the serum of patients with the ulcerative colitis (UC) form of IBD with a high sensitivity and specificity [1,2]. These anti-integrin avb6 autoantibodies (AIA) have also been discovered in patients with primary sclerosing cholangitis, another disease associated with UC [3]. We sought to determine if AIAs could similarly be found in AxSpA patients with IBD or with AxSpA alone and to evaluate the potential of AIA as a diagnostic test for gut involvement in AxSpA patients.

Methods: Using a previously published ELISA protocol [2], we measured AIA levels in sera of patients with (i) AxSpA and UC (n=18), (ii) AxSpA and Crohn's Disease (CD) (n=29), (iii) AxSpA alone (n=48), and healthy controls (n=48). The mean + 3 SD ELISA absorbance of the healthy controls was used as a cutoff for AIA positivity. Clinical variables were compared between AIA+ and AIA- patients. Longitudinal serum samples were analyzed to determine if AIA levels changed over time or with biologic use, and if AIA levels correlated with any markers of AxSpA severity.
Results: Patients with AxSpA and UC showed a significant increase in mean absorbance compared with AxSpA alone and with healthy controls (Fig 1A). 56% of patients with AxSpA and UC were AIA+, compared to 26% of patients with AxSpA and CD, 4% of patients with AxSpA alone, and 0% of healthy controls. For diagnosis of UC among AxSpA patients, this test had a sensitivity of 55.6%, specificity of 89.6%, positive likelihood ratio of 5.35 and negative likelihood ratio of 0.50. Receiver operating characteristic analysis of this test yielded an area under curve value of 0.83 as a test for UC in AxSpA patients (Fig 1B). AIA positive status was associated with a family history of AxSpA, a family history of IBD and, surprisingly, a decreased maximum CRP and BASDAI. While 2/4 AIA+ patients seroconverted following biologic treatment, AIA levels were generally consistent over time and did not seem to trend with CRP or BASDAI.
Conclusion: We found that within AxSpA patients, AIA demonstrated potential as a diagnostic test for UC, and may particularly be useful in screening AxSpA patients with a family history of AxSpA or IBD. To our knowledge, this is the first study to date to examine AIA in AxSpA.
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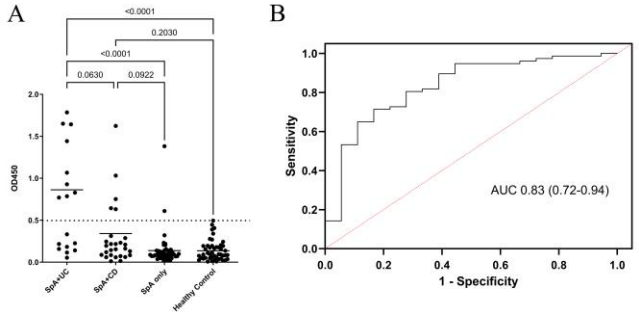

POD07
A Reciprocal Machine Learning Approach to Defining Physiologic Compensation of the Synovial Joint in Knee Osteoarthritis
Matthew Renaud (Western University, London); Tom Appleton (St. Joseph's London Rheumatology Centre and Western University, London)
Objectives: Osteoarthritis (OA) is the most common joint disease worldwide, and one of the most common synovial joints affected is the knee. Synovial joints are organs that permit movement. Like other organs, synovial joints must respond physiologically to biomechanical and molecular stresses to maintain homeostasis. To study the physiologic response of the organ, a definition of synovial organ compensation is required. We propose that physiologic compensation of the synovial organ is defined by an ability to maintain homeostasis in response to load or injury, thereby preserving function and preventing tissue damage. By contrast, synovial organ decompensation results in OA, leading to joint dysfunction, tissue damage, and pain experience. Our objective was to produce a candidate clinical definition of physiologic joint compensation using a reciprocal machine learning (ML) approach.

Methods: A reciprocal ML approach employing data-driven and ground-truth classifiers was applied. Data were derived from patients in the Western Ontario Registry for Early Osteoarthritis (WOREO; n=774 knees). Included variables were derived from a battery of clinical assessments (n=34). The data driven classifier first used a principal component analysis (PCA) to reduce dimensionality. A k-means clustering algorithm was applied to the multidimensional space defined by the first 4 principal components and revealed 3 identifiable clusters. A reciprocal ground-truth classifier utilized patient-reported pre/post pain on the 6-minute-walk test to define 3 disease clusters (compensatory status). A random forest algorithm identified a subset of measures capable of predicting compensatory status.
Results: Findings from the PCA revealed that the first 4 principal components (PCs) explained 58.0% of the variance. A permutation test confirmed that PC1-4 robustly explained dataset variance ($p < 0.001$). Post-hoc clustering showed that the features with the most variance between clusters were knee injury and osteoarthritis outcome score (KOOS; 66.2 ± 15.6), mechanical axis angle (MAA; -3.5 ± 13.2), BMI (32.1 ± 2.12), age (62.5 ± 4.3), and uric acid (321.3 ± 57.4). The random forest algorithm revealed that KOOS pain, MAA, BMI, age, and peak knee flexion were most predictive of compensatory status (vector similarity = 0.80; Θ = 36.87).
Conclusion: A clinical definition of synovial organ compensation is required to study the underlying physiology. Our findings support the proposed model of physiologic compensation and offer candidate clinical measures capable of determining the compensatory status of the synovial organ in the knee. Next steps involve validating the model via analysis of the physiologic profiles associated with compensatory status (i.e., synovial fluid proteomics).

Podium Session B: Friday, February 28, 2025, at 8:00-9:15 AM MT

POD08
Incidence and Predictors of Secondary Failure to Biologic Therapy in Patients with Psoriatic Arthritis
Fadi Kharouf (Toronto Western Hospital and University of Toronto, Toronto); Ali AlHadri (Toronto Western Hospital, Toronto); Shangyi Gao (UHN, Toronto); Daniel Pereira (Toronto Western Hospital, University of Toronto, Toronto); Richard Cook (University of Waterloo, Department of Statistics and Actuarial Science, Waterloo); Vinod Chandran (1. Schroeder Arthritis Institute, Krembil Research Institute, Toronto, ON, Canada, Division of Rheumatology, Department of Medicine, University of Toronto, Toronto, ON, Canada, Toronto); Dafna Gladman (Schroeder Arthritis Institute, Krembil Research Institute, Toronto Western Hospital, University Health Network, University of Toronto, Toronto)
Objectives: Secondary failure to biologic therapy is challenging and contributes to the complexity of managing psoriatic arthritis (PsA). In this study, we aimed to define the incidence of secondary failure to biologic therapy in patients with PsA and identify the factors associated with its occurrence.
Methods: We retrieved data on patients with PsA followed at our prospective observational cohort who commenced and remained on biologic therapy for at least 1 year. We assessed response at the 1-year point as achievement of ≥40% reduction in the swollen joint count (SJC) and ≥50% reduction in the PASI, or PASI ≤2. We defined secondary failure as the clinician's judgment of loss of efficacy over time or failure to maintain the response criteria. Patients with secondary failure were compared to those with maintained response in terms of demographic and

disease-related characteristics. For factors associated with the development of secondary failure, we used univariate and multivariate Cox regression analyses, adjusting for calendar year (8-year intervals from 2002 to 2024).

Results: Of the 482 patients who commenced treatment with biologics after clinic enrollment, 264 (54.8%) were classified as responders to therapy at one year. 236 (89.4%) responders received tumor necrosis factor inhibitors (TNFi). 94 (35.6%) responders developed secondary biologic failure at a median [IQR] of 2.7 [1.7, 4.8] years. The incidence rate of secondary failure was 5.96 per 100 person-years. Golimumab had the lowest 5-year prevalence of secondary failure (11.1%). In the reduced multivariate model, higher SJC (HR 1.40, p=0.01) and PASI (HR 1.15, p=0.02) at the time of response (1-year point) were associated with the development of secondary failure. Use of TNFi (HR 0.37, p=0.02) and initiation as the first-ever biologic (HR 0.52, p=0.049) were associated with a lower incidence of secondary failure, [Table 1].

Conclusion: Secondary biologic failure is common in PsA. A more complete clinical response, use of TNFi, and commencement as the first-ever biologic are all associated with persistence of therapy.

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Table 1. Univariate and multivariate Cox proportional hazards regression analysis of factors associated with secondary biologic failure.

Variable~		Univariate model			Multivariate full model			Multivariate reduced model		
		HR	p-value		HR	p-value		HR	p-value	
Calendar year (compared to 2002-2009)	2010-2017	1.07	0.71	0.31	1.00	1.00	**0.04**	1.02	0.93	**0.04**
	2018-2024	0.64	0.24		0.20	**0.048**		0.22	**0.049**	
Age in years^		1.00	0.71		0.99	0.25				
Sex (male/female)		0.81	0.32		0.76	0.24				
Duration of PsA in years^		1.01	0.55		1.02	0.19				
BMI in kg/m^2 ^		1.04	**0.03**		1.03	0.19				
Fibromyalgia (yes/no)		1.47	0.36		1.51	0.40				
Inflammatory eye disease or IBD (yes/no)		0.82	0.71		1.37	0.58				
SJC^		1.35	**0.03**		1.36	**0.04**		1.40	**0.01**	
PASI^ (0-72)		1.16	**0.01**		1.14	**0.04**		1.15	**0.02**	
Syndesmophytes and/or sacroiliitis (yes/no)		1.06	0.77		0.99	0.97				
Use of conventional synthetic DMARDs (yes/no)		1.36	0.14		1.32	0.23				
TNFi (yes/ no)		0.57	0.07		0.40	**0.03**		0.37	**0.02**	
Use as the first-ever biologic (yes/no)		0.54	**0.02**		0.53	0.06		0.52	**0.049**	

HR, hazard ratio; PsA, psoriatic arthritis; BMI, body mass index; IBD, inflammatory bowel disease; SJC, swollen joint count; PASI, Psoriasis Area and Severity Index; DMARDs: disease-modifying anti-rheumatic drugs; TNFi, tumor necrosis factor inhibitors

~ The response date (assessed at 1 year of drug initiation) is used as the time of origin. All variables are time-varying, except SJC and PASI at the time of origin

^ 1-unit or joint increase

POD09

Comparative Safety and Effectiveness of Biosimilar and Originator Rituximab for Induction or Maintenance in Anca-Associated Vasculitis: 6-Month Results of a Longitudinal Cohort Study

Arielle Mendel (McGill University Health Centre, Department of Medicine, Division of Rheumatology, Montreal); Lillian Barra (Western University/ Lawson Health Research Institute , London); Sasha Bernatsky (McGill University Health Centre, Department of Medicine, Division of Rheumatology, Division of Clinical Epidemiology, Montreal); Alison H. Clifford (University of Alberta, Edmonton); Mojtaba Dabaghjamanesh (Western University, London); Natasha

Dehghan (University of British Columbia, Division of Rheumatology, Vancouver); Aurore Fifi-Mah (University of Calgary, Calgary); Jean-Paul Makhzoum (Vasculitis Clinic, Canadian Network for Research on Vasculitides (CanVasc), Department of Medicine, Hôpital du Sacré-Coeur de Montréal, University of Montreal, Montreal); Rosalie Meunier (Hôpital du Sacré-Coeur de Montréal, Montreal, Montreal); Nataliya Milman (The University of Ottawa, Ottawa); Medha Soowamber (Division of Rheumatology, Department of Medicine, University of Toronto, Toronto); Jan Cohen Tervaert (Edmonton); Elaine Yacyshyn (Division of Rheumatology, University of Alberta, Edmonton); Nader Khalidi (McMaster University, St Joseph's Healthcare Hamilton, Hamilton); Christian Pagnoux (Vasculitis Clinic, Canadian Network for Research on Vasculitides (CanVasc), Division of Rheumatology, Mount Sinai Hospital, University of Toronto, Toronto)

Objectives: To evaluate the effectiveness and safety of rituximab biosimilars compared to the originator in Canadians with granulomatosis with polyangiitis (GPA) and microscopic polyangiitis (MPA), and outcomes following originator to biosimilar switching.

Methods: We recruited adults with GPA or MPA who started rituximab originator or biosimilar for induction or maintenance, or switched from originator to biosimilar maintenance between 01/2018-09/2023. Eligible participants either started the index treatment in the prior 6 months or were followed within an existing vasculitis cohort. Six-month outcomes include remission (Birmingham Vasculitis Activity Score [BVAS] v3 of 0), relapse (rise in BVAS after achieving remission, requiring treatment), change in Vasculitis Damage Index (VDI), and serious adverse events (SAEs).

Results: We enrolled 200 participants from 9 centres: 126 who started induction (52 originator, 74 biosimilar), 58 who started maintenance (22 originator, 36 biosimilar), and 16 who switched from originator to biosimilar maintenance (median 2 years [IQR 1.4-2.2] of originator maintenance prior to switching). Mean age was 57.1 (SD 17.4), 53% were female, 79% White, and 69% had GPA. Baseline characteristics across subgroups are reported in Table 1. 190 (95%) participants had follow-up visits at Month 6 or died prior to this visit. Over a mean follow-up 189 days [SD 56], 2 minor relapses occurred in PR3-ANCA+ individuals, one in the biosimilar induction subgroup (10 weeks), and one in the originator maintenance group (at 4 months). Among induction recipients, 48/49 (98%) in the originator group and 66/71 (93%) in the biosimilar group were in remission at Month 6. All in the originator and biosimilar maintenance subgroups were in remission at Month 6, and all 16 who switched from originator to biosimilar maintenance remained in remission during follow-up. Mean change in VDI was similar between biosimilar and originator subgroups. One or more SAEs occurred in 4/49 (8%) of the originator induction subgroup, 11/71 (15%) of the biosimilar induction subgroup, 2/21 (10%) originator maintenance subgroup, 2/33 (6%) of the biosimilar maintenance group, and 3 (19%) of the 'switch' group. Two deaths occurred in the biosimilar induction subgroup (1 alveolar hemorrhage, 1 COVID-19) and 1 death occurred in the switch group (infection, 5.5 months after switching).

Conclusion: In this cohort, we did not observe differences in remission or relapses at 6 months between RTX originator or biosimilar induction or maintenance. Disease remained stable in those who switched from originator to biosimilar maintenance. **Supported by a CIORA grant**

POD10

Metabolic Stress Remodelling: Insights into the Role(S) of Foxo1 in Promoting Cancer-Like Changes in Systemic Sclerosis (Ssc)

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Objectives: Systemic sclerosis (SSc) is a life-threatening autoimmune disease with limited treatment options. Autologous stem cell transplantation (ASCT) is the only disease-modifying therapy in SSc; however, its effects on fibroblasts are unknown. We have recently shown that dermal fibroblasts (DFs) from patients with diffuse cutaneous SSc (dSSc) develop a cancer-like phenotype characterized by genomic instability and increased double-stranded DNA breaks (DSBs) associated with resistance-to-apoptosis (1). In cancer, this is promoted by mitochondrial-dependent metabolic remodelling which are activated by the transcription factor forkhead Box 1 (FOXO1). We hypothesized that metabolic remodelling in dSSc DFs may promote resistance-to-apoptosis via FOXO1, which normalizes post-ASCT.

Methods: DFs were generated from healthy control volunteers (HC), limited cutaneous SSc (lSSc), dSSc and post-ASCT (~12 months) patients using 4 mm skin biopsies (N=6-10/group). We quantified the frequency of DSBs and resistance-to-apoptosis via γ-H2AX and/or TUNEL/cleaved caspase 3 (+/- treatment with cyclophosphamide or a FOXO1-inhibitor), respectively. FOXO1 activation was determined by measuring nuclear (active) FOXO1, and expression of its downstream mRNA targets SOD2 and PDK4. Mitochondrial morphology was assessed using confocal microscopy. We also assessed changes in mitochondrial functions, namely mitochondrial dynamics (fusion/fission, immunoblot), biogenesis (qRT-PCR), electron transport chain (ETC) genes (qRT-PCR), and oxygen consumption (XFlux Seahorse analysis).

Results: dSSc DF had the highest frequency of DSBs compared to HC, lSSc and post-ASCT. This was associated with increased resistance-to-apoptosis in response to cyclophosphamide, with associated activation of FOXO1. Notably, pharmacological inhibition of FOXO1 in dSSc DF resulted in decreased indicators associated with fibrosis and increased apoptosis. In addition, dSSc DF had an increased frequency of elongated mitochondria and indicators associated with mitochondrial fusion (e.g. OPA1, phospho-DRP1), mitochondrial biogenesis, ETC expression with reduced oxygen consumption rate. Overexpression of constitutively active FOXO1 in HC DF resulted in similar metabolic changes as seen in dSSc. Finally, post-ASCT DF did not have increased DSB, FOXO1 activation, or metabolic reprogramming - suggesting that ASCT may provide some of its beneficial effects by modulating this novel mitochondrial/FOXO1 axis.

Conclusion: Our study highlights the critical role of metabolic stress remodelling as a driver of the cancer-like phenotype in severe forms of SSc (Fig. 1). It also provides mechanistic insights related to how ASCT may impart some of its beneficial effects. Future studies targeting this pathway may implicate this novel mitochondrial/FOXO1 pathway as a novel therapeutic strategy in patients with SSc.

POD11

Inequities in Fee-For-Service Remuneration Affecting Rheumatologists and Patient-

Centred Care Across Canada: An Environmental Scan

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Objectives: System structure (macro-level) health policy and payment methods directly impact provider (meso-level) and patient levels of the healthcare system. Identifying variations in funding remuneration of services can identify potential inequities in care. We compared fee-for-service reimbursement rates (and regulations/stipulations) that are available to rheumatologists across Canadian provinces.

Methods: We reviewed publicly available Physician Payment Schedules across provinces to derive key information on fee-for-service payment information for common rheumatology services (as of July 2024). Information extracted included several sections encompassing: consultations, office visits, special premiums for complexity/diagnoses, hospital services, multidisciplinary care, virtual care, procedures (including injections/arthrocentesis, point-of-care ultrasonography) and additional services (e.g. laboratory monitoring). We descriptively summarized data in a table and text. We assessed whether reimbursement structures support person-centeredness (access to care dimension).

Results: Compensation for consultations ranged widely, from $153.51 to $239.57 (Table). This was also apparent in follow-up visit payments ranging from $65.55 to $131.52 and are overall consistently reimbursed at a lower rate than initial consultations. Saskatchewan, Manitoba and Ontario offer additional reimbursement (premiums) for certain complex rheumatic conditions, whereas Saskatchewan, Quebec, New Brunswick and Newfoundland and Labrador offer age-based premiums. Overall, inpatient consultation reimbursement rates are similar to in-office consultations. Virtual care is inconsistently funded across Canada with variations in stipulations, and is often reimbursed at a lower rate than in-person care. Funding to support multidisciplinary/interdisciplinary care models is sparse, with only British Columbia and Quebec having a dedicated billing code to fund nursing co-managed care. The average (±standard deviation) rates for joint aspirations, steroid injections, image-guided aspiration/injection, bursa aspiration/injection and complex bursa/joint aspiration/injection are $28.02±8.73, $26.58±8.44, $34.00±12.93, $26.86±10.28 and $29.85±9.19 respectively. Point-of-care ultrasonography is only renumerated in Ontario and Quebec. Notable restrictions/stipulations that may impair patient-centered care included financially disincentivizing follow-up visits that occur within shorter intervals, and remunerating virtual visits at a lower amount, creating an inequitable disadvantage for patients experiencing barriers in accessing convenient/local rheumatology care. Inconsistencies in funding for multidisciplinary/interdisciplinary care, and procedures (e.g. ultrasonography) may contribute to variations in quality of care across Canada.

Conclusion: We identified large provincial variations in reimbursement rates for rheumatology services. Current fee-for-service structures raise health policy issues for funding equitable

rheumatology services that require reducing pay disparities, removing restrictions/stipulations that impair person-centered care, and further optimizing standardization of health services across Canada.

POD12

Returning Research Results to Patients with Rheumatoid Arthritis: a Patient-Driven Knowledge Translation Strategy from the Canadian Early Arthritis Cohort (Catch)

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Objectives: Sharing research findings with patients is crucial to enhance transparency, foster trust, and engage research participants and to address the evidence-to-practice gap (1). However, the method and timing of knowledge translation (KT) are essential to communicating research results (2) to patients from different linguistic and cultural backgrounds. Our goal was to create a tailored KT strategy to communicate key findings from the Canadian Early Arthritis Cohort (CATCH) to participants and the wider rheumatoid arthritis (RA) community.

Methods: An initial list of topics relevant to RA patients was generated based on the lived experiences of the KT specialists with RA and further refined based on various input (polling, discussions with RA patients and Scientific Advisory Committee (SAC) members). These topics were mapped to available CATCH research projects and used to develop videos, plain language research summaries (PLRS), and social media messaging. KT specialists oversaw video production involving SAC members and RA patients, adapting interview questions to ensure relevance for the patient audience. PLRS were written using the guidance developed by Clinical Trials Ontario (3). Topics were used to compile evidence-based resources from credible arthritis organizations. The KT products were disseminated on various social media platforms, like X (@earlyarthritis), YouTube (@canadianearlyarthritiscoho928), Instagram (@earlyarthritis), and CATCH website (www.earlyarthritis.ca).

Results: We have developed 100 English and French language videos covering key topics such as disease/symptom management and preventative health (infections/vaccines). We produced 15 PLRS based on CATCH research and compiled patient resources across 8 themes. All website content and some social media and video content (available at https://bit.ly/3stynpR) were made available in French. To share or return research results with CATCH participants, an appointment card which included information about KT strategies was developed and shared with over 3000 participants. To measure the uptake of KT efforts, we monitored social media and website metrics: the YouTube channel has accumulated over 115,000 views and 614 subscribers while X has 1100 followers and Instagram has 225 followers. Since February 2024, the CATCH website has received 3000 unique visitors and close to 15,000 page views.

Conclusion: Our KT approach addresses an important gap by sharing research knowledge with patients and the public through a strategy that incorporates plain language and multimedia content. Future efforts will focus on evaluating the effectiveness of this KT strategy through patient feedback, engagement metrics, and assessing impact on patient knowledge and behavior.

POD13

Assessment of Juvenile Idiopathic Arthritis Outcomes and Place of Residence in Canada: Identifying Disparities in Care

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Objectives: Previous research has shown that demographic, health care system, socioeconomic, cultural, and ethnic factors, may be contributors to JIA outcomes. [1,2] We aimed to assess the association of social and environmental factors with JIA outcomes in Canada.

Methods: Data was collected by the Canadian Alliance of Pediatric Rheumatology Investigators (CAPRI) National JIA Registry, a registry of children newly diagnosed with JIA that collects and shares longitudinal data on disease course and outcomes. Demographic and clinical characteristics, medications used, and physician and patient reported outcome measures were obtained for patients enrolled over a 4-year period (February 2017 – December 2021). Clinical outcomes were linked to neighbourhood-level geographic and sociodemographic factors based on postal code and data from the 2021 Statistics Canada Census. For each patient, the sociodemographic and environmental variables are based on the dissemination area associated with their postal code. We assessed the attainment of two primary outcomes within 6 months of enrolment: 1) clinically inactive disease, as defined by Wallace criteria and 2) pain relief, defined as a pain score <1 reported by patients and parents in 21-point pain scales. Logistic regression was used to evaluate the association of sociodemographic variables with the outcomes of interest.

Results: A total of 641 patients were included. 41.2% of patients achieved inactive disease within 6 months of enrolment. Table 1 demonstrates associations between the primary outcomes and neighbourhood-level data. Greater distance to nearest pediatric rheumatology centre was associated with decreased likelihood of attaining inactive disease by 6 months. Every 100 km increase in distance decreased the odds of attaining inactive disease by 10% (OR 0.90, 95% CI: 0.81-0.99, P = 0.026). By 6 months, 27.9 % of patients and 34.6% of parents reported relief of pain. Higher dwelling density (number of dwellings per square kilometer of the diseemination area) was associated with decreased odds of attaining pain relief reported by the patient (OR 0.67, 95% CI 0.45 – 0.99, P = 0.043).

Conclusion: Among Canadian children newly diagnosed with JIA, greater distance to the nearest pediatric rheumatology center was associated with lesser attainment of inactive disease,

and higher dwelling density was associated with lesser attainment of pain relief by 6 months. Further analyses will examine the possible role of diagnostic or treatment delays in explaining these relationships.

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Table 1: Associations between attainment of clinically inactive disease and pain relief in patients with JIA within 6 months and neighbourhood-level geographic and sociodemographic factors.

	Attainment of Clinically Inactive Disease ≤6 months OR (95% CI)	Achievement of Pain Relief ≤6 months - Patient Scores OR (95% CI)	Achievement of Pain Relief ≤6 months - Parent Scores OR (95% CI)
Distance to nearest pediatric rheumatology centre	0.90 (0.81 – 0.99)	1.058 (0.966, 1.159)	0.961 (0.878, 1.051)
Visible Minority[a]	0.999 (0.991, 1.006)	0.996 (0.987, 1.005)	0.998 (0.990, 1.006)
Recent Immigrant[b]	0.972 (0.931, 1.016)	0.993 (0.941, 1.044)	0.991 (0.947, 1.036)
Postsecondary Education[c]	1.007 (0.996, 1.018)	0.997 (0.984, 1.010)	1.005 (0.994, 1.017)
Low Income Cut Off[d]	1.004 (0.980, 1.029)	1.004 (0.976, 1.034)	0.985 (0.960, 1.012)
Lone Parent[e]	0.999 (0.980, 1.019)	1.005 (0.983, 1.027)	0.988 (0.967, 1.009)
Park Accessibility[f]	0.762 (0.563, 1.068)	0.992 (0.847, 1.162)	0.941 (0.808, 1.097)
Intersection Density[g]	1.070 (0.840, 1.363)	0.766 (0.562, 1.044)	0.985 (0.764, 1.271)
Dwelling Density[h]	1.092 (0.845, 1.412)	0.665 (0.448, 0.988)	0.933 (0.709, 1.229)
Supportiveness for Active Living[i]	0.951 (0.864, 1.048)	0.865 (0.724, 1.034)	0.951 (0.857, 1.056)

a. Percentage of dissemination area population who is a visible minority; **b**. percentage of dissemination area population who are recent immigrants; **c**. percentage of dissemination area population with postsecondary or higher certificate, diploma, or degree; **d**. percentage of dissemination area population who live below the low-income cut-off; **e**. percentage of dissemination area families headed by a lone parent; **f**. distance to the nearest park area; **g**. number of ≥3-way intersections per square kilometre of the dissemination area; **h**. number of dwellings per square kilometer of the dissemination area; **i**. sum of the z-scores for park accessibility, intersection density and dwelling density.

POD14

Machine Learning Can Identify an Antinuclear Antibody Pattern that May Rule Out Systemic Autoimmune Rheumatic Diseases

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Objectives: Antinuclear antibody (ANA) testing is used to screen for systemic autoimmune rheumatic diseases (SARD) like systemic lupus erythematosus. It is well established that a nuclear dense fine-speckled (DFS) ANA pattern (AC-2), being rare among SARD patients, decreases the likelihood of these conditions. However, the AC-2 pattern is challenging for lab technologists to accurately identify due to similarities with other patterns, i.e., AC-4 (speckled) and AC-30 (nuclear speckled with mitotic plate staining), which are associated with SARDs. We determined if machine learning could accurately differentiate between AC-2 and SARD-related AC-4/AC-30 patterns.

Methods: 13,671 ANA images from SLE patients enrolled in the Systemic Lupus International Collaborating Clinics Inception Cohort (SLICC, n=2,825 images), non-SLE subjects enrolled in

the Ontario Health Study (OHS, n=10,639 images), and the International Consensus on ANA Patterns (ICAP, n=207 images) were analyzed. All SLICC and OHS ANA were performed in one central laboratory using IFA on HEp-2 cells (NovaLite, Werfen, SD) and read on a digital IFA microscope (NovaView, Werfen, SD). A lab technologist (HH) with >30 years of experience identified AC-2, AC-4, and AC-30 images. Images were resized to 224 x 224 pixels. Three machine learning models (ANA Reader©) using a convolutional neural network (CNN) and an image feature extractor were developed to differentiate AC-2 from the other patterns. We also merged the outputs of all three CNNs to create a combined ANA Reader© model. 80% of the images were used for training and 20% for validation. We compared the performance of the four machine learning models (lab technologist as the reference standard) to determine the best prediction model.

Results: The lab technologist identified 308 AC-2, 957 AC-4, and 379 AC-30 images. All four models performed similarly with high area-under-the-curve (AUC) scores ranging from 96.5%-97.1% (Table 1). When comparing other performance metrics, the combined ANA Reader© model performed the best with the highest accuracy (93.0%), precision (92.7%), specificity (93.2%), and F1 score (92.7%). It was tied with another model (Model 2) for the second most sensitive model (92.7%).

Conclusion: We developed a highly precise and accurate ML model, ANA Reader©, that discriminates the nuclear DFS pattern (AC-2) from other similar ANA patterns, potentially speeding up the differentiation of those at risk vs. not at risk of SARDs and reducing the need for unnecessary rheumatologic investigations or assessments. External validation of our model in other cohorts will be done before this model is adopted into laboratories and clinical practice.

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Table 1. Comparison of different ANA Reader© convolutional neural network (CNN) models and a combined model to differentiate between AC-2 vs. AC-4 and AC-30 antinuclear antibody (ANA) patterns[1].

CNN Model	Accuracy (%)	Precision (%)	Sens (%)	Spec (%)	F1 (%)	AUC (%)
1	89.5	90.6	87.3	91.5	88.9	96.7
2	90.4	87.9	92.7	88.1	90.3	96.5
3	84.2	76.1	98.2	71.2	85.7	97.1
Combined	93.0	92.7	92.7	93.2	92.7	96.6

1. Each patient sample from the Systemic Lupus International Collaborating Clinics Inception Cohort (SLICC) and Ontario Health Study (OHS) had multiple ANA images taken by NovaView. On average, there were five images per patient sample for SLICC, three images per patient sample for OHS, and one image per patient from International Consensus on ANA Patterns (ICAP). In total, there were 512 patients in the SLICC cohort, 3,559 individuals in the OHS cohort, and 207 patients from ICAP who were included in the study.

Abbreviations: AUC, area-under-the-curve; CNN, convolutional neural network; Sens, sensitivity; Spec, specificity.