Poster Tours 1-4: Thursday, February 27, 2025, at 12:00 PM – 12:30 PM MT

Poster Tour 1: Rheumatoid Arthritis
Moderator: Carol Hitchon

TOUR1A

Clinical Disease Activity Index Trajectories in Patients with Rheumatoid Arthritis Treated with Abatacept: a Real-World Study

Janet Pope (Divisions of Rheumatology, Epidemiology, and Biostatistics, Department of Medicine, Western University, London); John S. Sampalis (Faculty of Medicine, Department of Surgery, McGill University, Université de Montréal; JSS Medical Research, Inc., Montreal); Boulos Haraoui (Institut de Rhumatologie de Montréal, Montreal); Emmanouil Rampakakis (Department of Pediatrics, McGill University; JSS Medical Research, Inc., Montreal); Dylan Keating (JSS Medical Research, Inc., Montreal); Fiona Allum (JSS Medical Research, Inc., Montreal); Marc-Olivier Trepanier (Rheumatology, Bristol Myers Squibb, Montreal); Louis Bessette (Université de Laval, Centre Hospitalier de l'Université Laval, Quebec)

Objectives: To describe the 12-month Clinical Disease Activity Index (CDAI) trajectory patterns and their determinants in patients treated for rheumatoid arthritis (RA) with subcutaneous abatacept. The association between 3- and 12-month CDAI trajectory classifications was also assessed.

Methods: Post-hoc analysis of RA patients initiated on abatacept in the Abatacept Best Care study (NCT03274141).[1] A growth mixture model with Bayesian imputations was used to determine CDAI trajectory groups over 12 months. Time to CDAI low disease activity (LDA) and remission was assessed with Kaplan Meier Survivor estimates. Differences in baseline characteristics between trajectory groups were assessed using bivariate statistical methods. Determinants of trajectory patterns were identified with simple and multivariate logistic regression models with stepwise variable selection. The association between 3- and 12-month CDAI trajectory groupings was assessed with Cohen's kappa

Results: Among 256 patients, mean (SD) age was 60.1 (11.5) years, 75% were female, and baseline CDAI was 30.1 (10.6). Three CDAI trajectory groups were identified: Rapid Responders (RR; n=125; 49%); Late Responders (LR; n=96; 37%); and Non-responders (NR; n=35; 14%).[Figure 1] Mean (95% CI) time to CDAI LDA was 5.6 (5.1-6.1), 9.0 (8.2-9.7), and 11.3 (10.4-12.1) months for RR, LR, and NR, respectively, and time to remission was 9.6 (9.0-10.2), 12.0 (11.8-12.2) months, and not reached for NR (P<0.001). Mean (SD) baseline parameters for RR, LR and NR respectively were: CDAI: 27.0 (10.2), 30.5 (8.9), 40.0 (10.4); DAS28-CRP: 4.1 (1.0), 4.5 (0.8), 5.2 (0.7); Routine Assessment of Patient Index Data 3: 14.1 (5.4), 16.9 (4.5), 17.8 (5.4); tender joint count: 7.9 (5.6), 9.8 (5.5), 15.2 (5.9), Patient Global Assessment (PtGA) 57.4 (22.9), 67.2 (18.5), 73.9 (18.6); and fatigue (VAS 0-100): 58.5 (23.5), 63.3 (22.5), 76.5 (21.6; P<0.01). In multivariate analysis, baseline predictors of RR or LR vs NR were lower PtGA (odds ratio [95% CI]:0.98 [0.95-1.00]), swollen joint count (0.92 [0.84-1.00]), Rheumatic Disease Comorbidity Index (0.75 [0.59-0.95]), and no prior biologic use (0.36 [0.16-0.79]). 3-month CDAI trajectory classifications were not associated with 12-month classifications (kappa=0.46)

Conclusion: In this real-world-study, ~85% of abatacept-treated RA patients showed rapid/late CDAI reduction over 12 months. Lower baseline disease severity, no prior biologic treatment, and fewer comorbidities were predictive of 12-month change in CDAI, whereas disease severity

changes during the first 3 months of treatment were not, suggesting that patients may benefit from a longer treatment course prior to switching treatment. Trajectory-based analyses are informative and may have implications for clinical practice and research.

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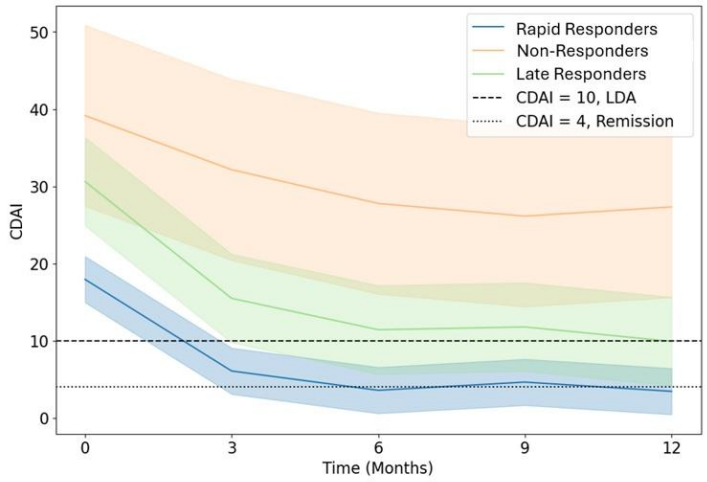
Figure 1: 12-month response trajectory groups for rheumatoid arthritis patients treated with abatacept identified by GMM based on CDAI response over time.

Lines represent mean CDAI score through 12 months; shaded areas represent standard deviation.

CDAI: Clinical Disease Activity Index; GMM: growth mixture model; LDA: low disease activity.



TOUR1B

A Cross-Sectional Study on Predictors of Covid-19 Infection, Admission and Effect of Immunomodulating Treatments in Rheumatoid Arthritis

Mohammad Movahedi (University Health Network, Toronto); Angela Cesta (University Health Network, Toronto); Xiuying Li (University Health Network, Toronto); Claire Bombardier (University of Toronto, Toronto); Sibel Aydin (Ottawa Hospital Research Institute, University of Ottawa, Ottawa); OBRI Investigators (Montreal)

Objectives: COVID-19 infection frequently leads to a cytokine storm, which has successfully been treated with some immunomodulating therapies. Interestingly, treatments investigated for the management of COVID-19 significantly overlap with those used in rheumatoid arthritis (RA). We aimed to investigate the prevalence and predictors of COVID-19 infection, severe infection requiring emergency department (ED) visits, hospitalization, intensive care unit (ICU) admission, and intubation in RA.

Methods: This was a cross-sectional analysis by linking the RA patients from the Ontario Best Practices Research Initiative (OBRI) to Institute for Clinical Evaluative Sciences (ICES) administrative data containing all health care records for Ontarians to explore the COVID-19 infection, hospital/intensive care unit (ICU) admissions and mortality due to COVID-19 (between Jan 1 2020 – Mar 31 2022). Primary outcome COVID-19 infection event was defined if patient had any positive COVID-19 test or diagnosis code ‘080’ in Ontario health insurance program (OHIP) during study period. We also looked at admission to hospital, ICU, and required intubation following COVID-19 infection. Characteristics of patients were compared between patients with and without covid-19 infection within 2 years before and 3 months after COVID-19 pandemic (15 March 2020). We also looked at the association of patient’s characteristics and risk of COVID-19 infection using multivariable logistic regression models.

Results: A total of 2969 patients were included. Among these, 596 (20.1%) were reported as having had COVID-19 infection. Females were significantly more likely to have infection (Covid-19 group: 81.9% vs. non-covid-19: 76.5%). Patient reported outcome (e.g. HAQ-DI, fatigue, and pain) and number of comorbidities was significantly higher in patients with COVID-19 infection. Patients with COVID-19 were more likely to use biologic agents (52.5% vs. 46.1%) and JAK inhibitors (13.4% vs. 9.5%). There was a significant positive association between age younger than 50 years (adj ORs: 3.27; 95%CI: 1.79-5.99 and 1.77; 95%CI: 1.13-2.80 for 30-40 and 40-50 age group, respectively) and higher number of comorbidities (adj ORs: 1.19; 95%CI: 1.09-1.30) and risk of COVID-19 infection in multivariable analysis (Table 1). Out of 596 patients with covid-19 infection, 108 (18.1%) had a record of ED visit or hospitalization. Among 108 ED visits or hospitalization, 13 (12.0%) admitted to the ICU, 11 (10.2%) admitted to ICU and had intubation, one (0.93%) had only intubation during follow-up.
Conclusion: In this study we found that COVID-19 infection was higher in female patients, younger than 50 years old, and those with higher number of comorbidities.

TOUR1C

Implementation of a Data-Driven Learning Health System in Rheumatology: A Novel Application of Dashboard Quality Reporting to Support Optimal Rheumatoid Arthritis Care

Racheal Githumbi (University of Alberta, Calgary); Vera Hill (Alberta Health Services, Calgary); Kevin Lonergan (Alberta Health Services, Calgary); Steven Katz (University of Alberta, Edmonton); Ania (Anna) Kania-Richmond (University of Calgary, Red Deer); Kim Giroux (NA, Calgary); Yvonne Wallace (NA, Edmonton); Allyson Jones (University of Alberta, Edmonton); Amanda Steiman (Mount Sinai Hospital, Toronto); Anshula Ambasta (University of Calgary, Calgary); Cheryl Barnabe (University of Calgary, Calgary); Elaine Yacyshyn (Division of Rheumatology, University of Alberta, Edmonton); Diane Lacaille (Arthritis Research Canada, University of British Columbia, Vancouver); Glen Hazlewood (University of Calgary, Calgary); Jessica Widdifield (Sunnybrook Research Institute, ICES, University of Toronto, Toronto); Tyler Williamson (University of Alberta, Calgary); Claire Barber (University of Calgary/Arthritis Research Canada, Calgary)

Objectives: Learning Health Systems (LHS) leverage healthcare data to drive cycles of knowledge generation for continuous care quality improvement. In Alberta, the recent launch of a provincial health information system (Connect Care, Epic Corporation) that interfaces with analytical and reporting tools, supports the use of routinely collected regional clinical data for care quality improvement efforts. As such, this project aims to implement a provincial LHS to support optimal care for Albertans with rheumatoid arthritis (RA).
Methods: This was a multi-phase project. In Phase 1, we identified a starter set of quality measures. Candidate measures were reviewed for feasibility of operationalization using structured data in Connect Care along with linkage to other administrative datasets (physician billing, pharmacy data, discharge abstract database). In Phase 2, a shortlist of eligible measures that aligned with national measurement priorities were ranked by panelists including providers, field-experts and patient partners. In Phase 3, we operationalized and reported on these measures as dashboards. To do so, a case definition of RA and a list of eligible providers were identified. These definitions were validated by chart reviews completed by two rheumatologists and an analyst. The resulting data were reported as dynamic and interactive dashboards on Tableau

using near to real-time data from the linked datasets. Dashboards allow providers to access patient-level data on their practice to support quality improvement.
Results: From a shortlist of eighteen measures, nine were prioritized by a panel of providers, experts and patient partners (n=7). We developed the following dashboards to display an initial set of measures: 1) number of RA patients followed per practice/site; 2) wait times to first rheumatology consult; 3) RA disease activity assessment (process and outcome measures); 4) gaps in care (lost to follow-up, treatment, or lab monitoring). As of 09/20/2024 there were 8059 individuals with RA under rheumatology care at four sites. Only 44% of 6668 encounters had a documented joint count, and 24% had a composite disease activity score calculated. Of the 1600 encounters with a composite score, 17% of patients were in low disease activity/remission. We identified 1,770 individuals with >12 months between rheumatology visits. Only 24% of individuals with RA were seen by a rheumatologist within 6 weeks of referral.
Conclusion: Using a LHS approach, we aim to shorten and streamline the cycles of transforming data to actionable knowledge for optimized care. We are working with our provincial partners to identify appropriate strategies to address the identified gaps. **Practice Reflection Award**

TOUR1D
One-Year Outcomes After Decision Aid-Led Tapering of Advanced Therapy in Rheumatoid Arthritis
Glen Hazlewood (University of Calgary, Calgary); Michelle Jung (University of Calgary, Calgary); Elzbieta Kaminska (University of Calgary, Calgary); Nick Bansback (University of British Columbia/Arthritis Research Canada, Vancouver); Rachelle Buchbinder (Monash University , Melbourne); Samuel Whittle (The Queen Elizabeth Hospital and University of Adelaide, Woodville); Dawn P Richards (Canadian Arthritis Patient Alliance, Toronto); Laurie Proulx (Canadian Arthritis Patient Alliance, Ottawa); Ann Rebutoc (McCaig Institute for Bone and Joint Health, Cumming School of Medicine, University of Calgary, Calgary); Claire Barber (University of Calgary/Arthritis Research Canada, Calgary)
Objectives: The Canadian Rheumatology Association clinical practice guidelines recommend offering tapering of advanced therapy to people with rheumatoid arthritis (RA) who are in sustained remission and provide a decision aid to support these discussions. Our goal was to understand the impact of implementing the decision aid on treatment choices and outcomes in RA patients in sustained remission.
Methods: We conducted a single-center pilot study with four rheumatologists in Calgary Alberta. Rheumatologists were initially asked to identify people in their practice at the time of the clinic visit. After identifying and addressing initial recruitment barriers, we switched to sending people a decision aid one month ahead of their appointment. While rheumatologists and patients were free to decide how fast to taper, and when to see their rheumatologist next, they were guided by CRA recommendations, which suggested decreasing the dose by ~25% at a time, with 3-month delays between subsequent reductions. Patients were followed for a year and the primary outcome (safety) was the proportion of patients who had to switch to another advanced therapy due to inefficacy.
Results: Thirty-four patients chose to taper their advanced therapy; 13 from in-clinic discussions, and 21/83 (25%) who received a decision aid ahead of their appointment. Of the 34 people, 32 consented to the follow-up study (mean age 55, 72% female). All patients were taking full dose advanced therapy at the time of recruitment. Patients reduced their TNF-inhibitor (n=19), IL-6-blocker (n=5) JAK-inhibitor (n=4), and T-cell inhibitor (n=4). Among the 26

patients who have completed follow-up to date (last follow-up October 2024), eight (31%) had a flare requiring re-escalation of their dose, and another three (12%) re-escalated their dose for other reasons (typically discomfort with being on a lower dose and the potential to have a flare). One patient switched to another agent without first re-escalating their dose. Of the 15 patients who maintained a dose reduction over one year, the mean dose reduction at one year was 47%. Of the nine patients who re-escalated their dose after tapering and have completed the end of study survey, eight (89%) agreed or strongly agreed with the statement "I am glad I tried reducing my medication." The remaining patient was neutral.
Conclusion: Sending people a guideline-linked decision aid ahead of their appointment resulted in 25% of people choosing to reduce their treatment, which was safe over one year, and without decisional regret in people who had to re-escalate their dose.

Poster Tour 2: Psoriatic Arthritis
Moderator: Walter Maksymoowych

TOUR2A

Impact of Depression and Anxiety on the Components of Minimal Disease Activity (Mda) in Patients with Psoriatic Arthritis

Virginia Carrizo Abarza (University of Toronto, Toronto); Fadi Kharouf (Toronto Western Hospital and University of Toronto, Toronto); Pankti Mehta (University of Toronto, Toronto); Shangyi Gao (UHN, Toronto); Daniel Pereira (University Health Network, Toronto); Richard Cook (University of Waterloo, Department of Statistics and Actuarial Science, Waterloo); Denis Poddubnyy (Division of Rheumatology, Department of Medicine, University Health Network and University of Toronto, Toronto); Dafna Gladman (Schroeder Arthritis Institute, Krembil Research Institute, Toronto Western Hospital, University Health Network, University of Toronto, Toronto); 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)
Objectives: To investigate whether the presence of depression and anxiety affects the components of Minimal Disease Activity (MDA) in patients with Psoriatic Arthritis (PsA).
Methods: We included adult patients diagnosed with PsA and meeting CASPAR criteria, prospectively followed from 2008 to 2024. We defined the presence of depression/anxiety by one of three criteria: (1) a score of ≤38 on the SF-36 Mental Component Summary (MCS), (2) a score of ≤56 on the SF-36 Mental Health domain, or (3) a rheumatologist's report of a diagnosis or treatment for depression/anxiety. Generalized Estimating Equations (GEE) were utilized to evaluate the association between depression/anxiety and being in a state of MDA, as well as each of the following components of MDA: tender joint count (TJC) ≤1, swollen joint count (SJC) ≤1, Psoriasis Activity and Severity Index (PASI) ≤1 or body surface area (BSA) ≤3%, pain Visual Analog Scale (VAS) score ≤15, global disease activity VAS score ≤20, tender entheseal points ≤1, and Health Assessment Questionnaire (HAQ) score ≤0.5, as well as C-reactive protein (CRP).
Results: We analysed data from 1130 PsA patients. Among them, 238 (22.2%) were identified with depression/anxiety according to definition 1, 242 (22.5%) according to definition 2, and 107 (9.6%) according to definition 3. In total, 267 patients (23.8%) met at least one definition. The presence of depression and anxiety was associated with 66% lower likelihood of being in MDA (OR 0.34, 95% CI 0.28- 0.41, $p<0.001$). Patients with depression or anxiety were significantly

less likely to meet several MDA components. The odds of achieving TJC ≤1 were reduced by 29% (OR 0.71, 95% CI 0.62–0.81, p<0.001), SJC ≤1 by 24% (OR 0.76, 95% CI 0.64–0.89, p<0.001), pain VAS ≤15 by 63% (OR 0.37, 95% CI 0.29–0.47, p<0.001), global VAS ≤20 by 73% (OR 0.27, 95% CI 0.22–0.33, p<0.001), and tender entheseal points ≤1 by 47% (OR 0.53, 95% CI 0.43–0.64, p<0.001). Additionally, patients had a 75% lower likelihood of achieving HAQ score ≤0.5 (OR 0.25, 95% CI 0.21–0.31, p<0.001). No significant association was found between depression/anxiety and PASI or BSA (OR 0.95, 95% CI 0.80–1.13, p=0.589), nor with CRP (OR 0.90, 95% CI 0.75–1.08, p=0.255).
Conclusion: In PsA patients, the presence of depression/anxiety reduces the odds of being in MDA. Both clinician-reported (TJC, SJC, enthesitis, PASI) and patient-reported (pain VAS, global VAS, HAQ) components of MDA are affected, with a greater impact seen in subjective domains.

TOUR2B
Exploring Causal Relationships of Depression and Psoriatic Disease (Psoriatic Arthritis and Psoriasis) Through Mendelian Randomization.
Skylar Tang (Memorial University , St. John's); Quan Li (Craig Dobbin Research Institute, Memorial University, St. John's); Proton Rahman (Faculty of Medicine, Division of Rheumatology, Memorial University of Newfoundland, St. John's)
Objectives: Previous observational studies have demonstrated an association between PsA and and depression (co-morbidity). However, whether this associations are causative and reverse causality was not assessed. Mendelian randomization (MR) can assess causality between an exposure and an outcome by using genetic instrumental variable. In this study, we perform a bidirectional two-sample MR to explore the complex causal relationship between depression and PsA or psoriasis.
Methods: The study cohorts (depression, PsA, psoriasis and controls) were identified from the UK or FinnGen biobank. The summary statistics from Genome-wide association studies (GWAS) were extracted from IEU GWAS database. The selection of SNPs was performed by considering the GWAS significance(5e-07), clumping, linkage disequilibrium, and minor allele frequency. The effect alleles were harmonized for exposures and outcomes. Heterogeneity and horizontal pleiotropy analysis were also performed. An inverse variance weighted (IVW) model was used to estimate causality for each IV in this two-sample MR study. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for causal estimations. The MR also was performed in both directions to explore the possibility of reverse causality.
Results: Patients with depression (170,756), PsA (1553), psoriasis (4510) and controls (147211 to 329433) were analyzed. The number of instrumental variables (SNPs) is presented in Table 1. Although there appeared to be a positive association between depression and PsA and psoriasis , it did not reach statistical significance (OR = 1.19, 95% CI: 0.87-1.64, p = 0.27) for PsA but a significant causal effect (OR 1.3 (1.08-1.58), p=0.006) for psoriasis. There was no evidence for reverse causation of psoriasis leading to depression (OR 1.0 (0.98-1.02), p=0.82) and significance for reverse causation of PsA leading to depression was significant (OR 1.02 (1.0-1.03), p=0.002).
Conclusion: This study underscores the importance of MR as a powerful tool for establishing causal relationships in rheumatology, which has significant implications for clinical practice. The lack of a significant consistent causal link between depression and PsA suggests that the relationship observed in observational studies may be due to confounding factors and potential

bias, however, there appears to be a causal association of depression leading to psoriasis.

TOUR2C

The Effect of Biologic Therapies on Serum Metabolic Biomarkers in Patients with Psoriatic Arthritis

Keith Colaco (University of Toronto, Toronto); Laura Bumbulis (University of Waterloo, 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); Richard Cook (University of Waterloo, Department of Statistics and Actuarial Science, Waterloo); Dafna Gladman (Schroeder Arthritis Institute, Krembil Research Institute, Toronto Western Hospital, University Health Network, University of Toronto, Toronto); Lihi Eder (Women's College Research Institute and Division of Rheumatology, University of Toronto, Toronto)

Objectives: Since biologic therapies such as TNF inhibitors (TNFi) and IL-17 inhibitors (IL-17i) may affect the cardio-metabolic profile of patients with psoriatic arthritis (PsA), we assessed their short-term effects on serum metabolites in patients with PsA, and determined whether these metabolite changes differed across the two drug classes.

Methods: A nested cohort study was conducted among participants with available serum samples from a longitudinal PsA cohort who initiated TNFi or IL-17i therapy. Serum samples prior to initiation of therapy, and three to six months after initiation of therapy, were used to quantify 64 metabolic biomarkers using a Nuclear Magnetic Resonance targeted metabolomics panel, which comprised lipid particles, amino acids and various other metabolites. T-tests were used to compare differences in metabolite levels before versus after therapy within each drug class. Linear mixed effects models assessed the effect of each drug class on changes in metabolite levels adjusting for age, sex, lipid lowering drugs, diabetes, hypertension and menopause.

Results: 163 patients were analyzed between 2013 and 2021 (mean age 51 ± 12.6 years, 45.5% female). Among TNFi users, levels of alanine, glycine, citrate and creatinine significantly increased post-treatment, whereas levels of glycoprotein acetyls (GlycA), a marker of systemic inflammation, decreased. Among IL-17i users, concentrations of citrate significantly increased post-treatment, whereas alanine, glycine, histidine, GlycA and creatinine decreased. When comparing biomarkers between classes of medications, post- and pre-treatment levels differed significantly for alanine, glycine, histidine, citrate, GlycA and creatinine. In models adjusted for age and sex involving TNFi users, levels of alanine (Estimate [EST] 0.034; 95% confidence interval [CI] 0.008, 0.06), glycine (EST 0.029; 95% CI 0.02, 0.04), phenylalanine (EST 0.006; 95% CI 0.001, 0.01), citrate (EST 0.008; 95% CI 0.005, 0.01) and creatinine (EST 3.87; 95% CI 0.41, 7.4) increased post-treatment, whereas acetate (EST −0.03; 95% CI −0.03,−0.02) and GlycA (EST −0.05; 95% CI −0.09,−0.01) decreased (Table 1). In models adjusted for age and sex involving IL-17i users, changes were observed among fewer biomarkers, with low-density lipoprotein (LDL) particle size (EST 0.04; 95% CI 0.009, 0.07) increasing post-treatment, and levels of histidine (EST−0.005; 95% CI −0.008,−0.003) and acetone (EST −0.005; 95% CI −0.009,−0.001) decreasing.

Conclusion: TNFi and IL-17i appear to differentially affect the metabolic profile of patients with PsA. Treatment with TNFi was associated with more changes in metabolite profiles than IL-17i, including changes associated with systemic inflammation (GlycA) and amino acids. The

implication of these changes on long-term cardio-metabolic risk needs further research.

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TNF inhibitors							
Category	**Biomarker**	**Model 1* Estimate (95% CI)**	**P**	**Model 2** Estimate**	**P**	**Model 3*** Estimate**	**P**
Lipids	HDL cholesterol	−0.007 (−0.06, 0.04)	0.79	0.07 (−0.005, 0.14)	0.08	0.12 (0.01, 0.22)	**<0.05**
	Cholesterol esters in HDL	−0.005 (−0.04, 0.03)	0.82	0.05 (−0.002, 0.11)	0.07	0.09 (0.009, 0.17)	**<0.05**
	Free cholesterol in HDL	−0.002 (−0.013, 0.009)	0.73	0.014 (−0.002, 0.03)	0.11	0.03 (0.004, 0.05)	**<0.05**
	Total lipids in HDL	−0.01 (−0.12, 0.09)	0.81	0.137 (−0.02, 0.30)	0.10	0.25 (0.02, 0.47)	**<0.05**
Amino acids	Alanine	0.03 (0.02, 0.05)	**<0.05**	0.034 (0.008, 0.06)	**<0.05**	0.04 (0.001, 0.08)	0.06
	Glycine	0.02 (0.008, 0.03)	**<0.05**	0.029 (0.02, 0.04)	**<0.05**	0.03 (0.009, 0.05)	**<0.05**
	Phenylalanine	0.002 (−0.002, 0.005)	0.37	0.006 (0.001, 0.01)	**<0.05**	0.006 (−0.002, 0.013)	0.17
Glycolysis-related metabolites	Citrate	0.005 (0.003, 0.007)	**<0.05**	0.008 (0.005, 0.01)	**<0.05**	0.008 (0.004, 0.012)	**<0.05**
Ketone bodies	Acetate	−0.03 (−0.03,−0.03)	**<0.05**	−0.03 (−0.03,−0.02)	**<0.05**	−0.02 (−0.03,−0.02)	**<0.05**
Fluid balance	Creatinine	3.72 (1.45, 6)	**<0.05**	3.87 (0.41, 7.4)	**<0.05**	3.19 (−1.71, 8.08)	0.23
Inflammation	Glycoprotein Acetyls	−0.09 (−0.12,−0.07)	**<0.05**	−0.05 (−0.09,−0.01)	**<0.05**	−0.06 (−0.12,−0.003)	0.05
IL-17 inhibitors							
Lipids	Average diameter of LDL particles	0.013 (−0.007, 0.03)	0.21	0.04 (0.009, 0.07)	**<0.05**	0.07 (0.02, 0.12)	**<0.05**
Amino acids	Histidine	−0.005 (−0.006,−0.003)	**<0.05**	−0.005 (−0.008,−0.003)	**<0.05**	−0.006 (−0.01,−0.002)	**<0.05**
Ketone bodies	Acetone	−0.003 (−0.006, 1.3e-04)	0.08	−0.005 (−0.009,−0.001)	**<0.05**	−0.007 (−0.014,−1.1e-04)	0.06

*Model 1: adjusted for baseline biomarker value
**Model 2: adjusted for baseline biomarker value, age, sex
***Model 3: adjusted for baseline biomarker value, age, sex, menopausal status, use of lipid-lowering drugs, diabetes, hypertension

CI, confidence interval; HDL, high-density lipoprotein; IL, interleukin; LDL, low-density lipoprotein; TNF, tumor necrosis factor

TOUR2D

Bimekizumab Maintained Efficacy Responses in Patients with Active Psoriatic Arthritis: Up to 2-Year Results from Two Phase 3 Studies

Dafna Gladman (Schroeder Arthritis Institute, Krembil Research Institute, Toronto Western Hospital, University Health Network, University of Toronto, Toronto); Jessica A. Walsh (Division of Rheumatology, Salt Lake City Veterans Affairs Health and University of Utah Health, Salt Lake City); Joseph F. Merola (Department of Dermatology and Department of Medicine, Division of Rheumatology, Dallas); Christopher T. Ritchlin (Allergy, Immunology & Rheumatology Division, University of Rochester Medical School, Rochester); Yoshiya Tanaka (The First Department of Internal Medicine, University of Occupational and Environmental Health, Kitakyushu); Ennio G. Favalli (Department of Rheumatology, ASST Gaetano Pini-CTO, University of Milan, Milan); Dennis McGonagle (Academic Unit for the Musculoskeletal Diseases, Leeds Teaching Hospitals NHS Trust, Leeds); Diamant Thaçi (Institute and Comprehensive Center for Inflammation Medicine, University of Lübeck, Lübeck); Barbara Ink (UCB, Slough); Rajan Bajracharya (UCB, Slough); Jason Coarse (UCB, Morrisville); William Tillett (Royal National Hospital of Rheumatic Diseases; Department of Life Sciences, Centre for Therapeutic Innovation, University of Bath, Bath)

Objectives: To report the proportion of Week (Wk)16 responders maintaining their response in joint, skin, and composite efficacy outcomes up to 2 years in bimekizumab (BKZ)-treated patients (pts) with psoriatic arthritis (PsA).

Methods: Two phase 3 studies assessed subcutaneous BKZ 160 mg every 4 wks in pts with PsA: BE OPTIMAL (biologic DMARD [bDMARD]-naïve; NCT03895203) and BE

COMPLETE (TNF inhibitor inadequate response/intolerance [TNFi-IR]; NCT03896581); both were placebo-controlled to Wk16. BE OPTIMAL Wk52 and BE COMPLETE Wk16 completers were eligible for BE VITAL (open-label extension; NCT04009499). Efficacy data are reported for pts randomised to BKZ at baseline (BL); safety data are reported for all BKZ-treated pts. Maintenance of response is reported as the proportion of Wk16 responders who were responders at Wk104/100 (BE OPTIMAL/BE COMPLETE). Efficacy outcomes include ACR20/50/70, Psoriasis Area and Severity Index (PASI)75/90/100, Minimal/Very Low Disease Activity (MDA/VLDA), and Disease Activity Index for PsA (DAPSA) remission or low disease activity responses (REM ≤4; REM+LDA ≤14); these are reported here to Wk104 in BE OPTIMAL and Wk100 in BE COMPLETE. Data are reported as observed case or using non-responder or worst category imputation. Exposure-adjusted incidence rates per 100 pt-years (EAIR/100 PY) are reported to Wk104 for both bDMARD-naïve and TNFi-IR pts.
Results: Of BKZ-randomised pts, 359/431 (83.3%) bDMARD-naïve and 215/267 (80.5%) TNFi-IR pts completed Wk104/100. High proportions of pts achieving ACR50, PASI100, and MDA at Wk16 maintained responses at Wk104/100. [Table] At Wk16, 189 (43.9%) bDMARD-naïve and 115 (43.1%) TNFi-IR pts achieved ACR50; of those, 150 (79.4%) bDMARD-naïve and 87 (75.7%) TNFi-IR pts maintained response at Wk104/100. Similarly, for pts with BL psoriasis affecting ≥3% body surface area, 103/217 (47.5%) bDMARD-naïve and 103/176 (58.5%) TNFi-IR pts achieved PASI100 at Wk16; of those, 73 (70.9%) bDMARD-naïve and 83 (80.6%) TNFi-IR pts maintained response at Wk104/100. MDA was achieved by 194 (45.0%) bDMARD-naïve and 117 (43.8%) TNFi-IR pts at Wk16; of those, 147 (75.8%) bDMARD-naïve and 87 (74.4%) TNFi-IR pts maintained response at Wk104/100. Similar results were observed for other joint, skin, and composite efficacy outcomes at Wk104/100. [Table] To Wk104, the EAIR/100 PY for BKZ-treated pts with ≥1 treatment-emergent adverse event was 179.9 in bDMARD-naïve and 100.3 in TNFi-IR pts.
Conclusion: BKZ demonstrated robust maintenance of response at 2 years in both bDMARD-naïve and TNFi-IR pts with PsA who responded to BKZ treatment at Wk16. The safety profile was consistent with previous reports.1,2 Previously submitted to: ACR 2024

Poster Tour 3: SLE
Moderator: Zahi Touma

TOUR3A

Value and Appropriateness of Inpatient Antinuclear Antibody Testing at a Tertiary Hospital

Megha Udupa (McGill University, Montreal); Gang He (McGill University Health Centre, Montreal); Arielle Mendel (McGill University Health Centre, Department of Medicine, Division of Rheumatology, Montreal)

Objectives: Antinuclear antibodies (ANA) are sensitive screening tests for systemic autoimmune rheumatic diseases (SARDs), but positive ANAs lack specificity and can lead to unnecessary patient anxiety and healthcare use. The appropriateness of ANA testing in hospitalized patients at our institution remains unclear. We aimed to characterize inpatient ANA testing, including the proportion ordered in the setting of possible signs or symptoms of a SARD, the proportion with a final diagnosis of a SARD, and the proportion for which testing was potentially unnecessary.
Methods: We reviewed 50 randomly sampled patients who had an ANA test ordered during a

hospitalization at our institution (June - October 2023), collecting patient demographics, reasons for ANA testing, results, and hospitalization details from electronic medical records. We reviewed post-discharge records to ascertain new documented SARD diagnoses. We defined ANA testing as potentially unnecessary if the individual 1) already had an established ANA-associated condition at the time of testing, 2) lacked signs or symptoms of a SARD or unexplained liver disease, 3) had an ANA result from our laboratory within the past year.
Results: Of the 50 charts reviewed, 24 (48%) were female, with a mean age of 61.7 (standard deviation, SD 19.6) years at time of ANA testing. Two patients had known conditions associated with ANA. ANA was positive in 37 (74%) patients at a titre of ≥1:80 (72% at ≥1:160), with the most common pattern reported at highest titre being homogenous (46%). A third of the tests were ordered by general internists, and 2% were ordered by rheumatologists. Fifteen (30%) had a prior ANA result at our institution (8 within the past year), with a median interval of 46.8 days (Interquartile range 6-3897) from the prior ANA test. Additionally, 5 (10%) had the ANA test repeated within the same inpatient stay. Most ANAs were ordered as part of an autoimmune work-up for pulmonary disease (22%) and neurological/psychiatric presentations (20%). Seven (14%) were ordered without apparent signs or symptoms attributable to a SARD or autoimmune hepatitis. Of the 25/37 (68%) individuals with a +ANA who had post-discharge follow-up at our institution after the ANA result was reported (median follow-up 221 days post-discharge [IQR 107-270]), zero (95% Confidence Interval, 0-13%) had documentation of a new SARD diagnosis or another autoimmune condition. Overall, 17 (34%) ANA tests (95% CI 22-48%) were potentially unnecessary.
Conclusion: Our institution could benefit from systems to guide appropriate ANA testing, in particular repeat ANA testing.

TOUR3B
Developing and Evaluating a Laboratory-Based Frailty Index for the Prediction of Long-Term Health Outcomes in Systemic Lupus Erythematosus
Grace Burns (Dalhousie University, Halifax); Alexandra Legge (Dalhousie University, Halifax)
Objectives: Frailty is a useful measure of health status in systemic lupus erythematosus (SLE), but it is not routinely captured in existing SLE datasets. In other populations, frailty indices constructed exclusively from laboratory data have been shown to predict adverse health outcomes. We aimed to construct and evaluate the first laboratory-based frailty index (FI-Lab) in SLE. Additionally, we compared the FI-Lab to an existing clinical frailty index (SLICC-FI) with respect to the prediction of future health outcomes.
Methods: This study used existing data from a single-centre prospective cohort of adult SLE patients followed annually with standardized assessments. We included the first study visit for each patient occurring between 2010 and 2019, with follow-up data available up to June 2024. A 30-item FI-Lab was constructed by adapting the list of laboratory variables identified by Ellis et al. [1] Cox proportional hazards regression was used to examine the association between baseline FI-Lab scores and all-cause mortality risk, while negative binomial regression was used to evaluate the association of baseline FI-Lab scores with organ damage accrual during follow-up. To compare the performance of models containing the FI-Lab and/or SLICC-FI as predictor variables, we used Akaike information criterion (AIC), Harrell's C-statistic, and pseudo-R2 values.
Results: The 283 included patients (89% female) had a mean (SD) age of 47.7 (15.1) years and a median (IQR) disease duration of 8.3 (2.6-19.8) years at baseline. The 97 patients classified as

frail at baseline (based on FI-Lab scores > 0.21) had increased mortality risk [hazard ratio 3.71; 95% confidence interval (CI) 1.82-7.54] and a higher rate of organ damage accrual during follow-up [incidence rate ratio 2.26; 95% CI 1.59-3.22] compared to non-frail patients. A weak correlation existed between baseline FI-Lab and SLICC-FI scores (rs=0.37, p < 0.0001). In unadjusted analysis, higher baseline FI-Lab and SLICC-FI scores were both associated with increased mortality risk during follow-up. However, after multivariable adjustment, only the FI-Lab maintained a significant association with mortality risk. (Table 1) Both the FI-Lab and the SLICC-FI were significant baseline predictors of organ damage accrual during follow-up, and the multivariable model that included both frailty measures was superior to the models containing either the FI-Lab or the SLICC-FI alone.

Conclusion: An FI constructed from routinely collected laboratory variables can measure frailty and predict future health outcomes in SLE. The FI-Lab may serve as a convenient screening tool to detect subclinical deficit accumulation and promote early risk mitigation among SLE patients.

Table 1. Association of baseline FI-Lab and SLICC-FI scores with mortality risk and organ damage accrual during follow-up (n=274).

	All-cause Mortality Risk[a]				Organ Damage Accrual[b,c]			
	HR (95% CI)	**p-value**	**AIC**	**C-statistic**	**IRR (95% CI)**	**p-value**	**AIC**	**Pseudo R^2**
Multivariable model 1: FI-Lab			305.07	0.8473			589.95	0.1097
FI-Lab (per 0.05 increase)	1.28 (1.09, 1.51)	p= 0.003			1.14 (1.06, 1.23)	p= 0.001		
Multivariable model 2: SLICC-FI			310.65	0.8380			587.36	0.1138
SLICC-FI (per 0.05 increase)	1.24 (0.95, 1.63)	p= 0.109			1.24 (1.11, 1.39)	p < 0.0001		
Multivariable model 3: FI-Lab + SLICC-FI			306.96	0.8470			584.26	0.1219
FI-Lab (per 0.05 increase)	1.26 (1.05, 1.52)	p= 0.015			1.10 (1.01, 1.18)	p = 0.021		
SLICC-FI (per 0.05 increase)	1.05 (0.78, 1.43)	p= 0.732			1.18 (1.05, 1.33)	p = 0.005		

[a]Multivariable models adjusted for the following baseline characteristics: Age (in years), sex, SLE disease duration (in years), education level, cigarette smoking status, baseline SLICC/ACR damage index (SDI) score, antimalarial use, and anticoagulant use.
[b]Multivariable models adjusted for the following baseline characteristics: Age (in years), sex, race, SLE disease duration (in years), education level, cigarette smoking status, baseline SDI score, immunosuppressant use, and anticoagulant use.
[c]Rate of organ damage accrual per person-year of follow-up based on the change in SDI score from the baseline visit to the last study visit during follow-up.
HR= hazard ratio; CI= confidence interval; AIC= Akaike information criterion; IRR= incidence rate ratio; laboratory-based frailty index (FI-Lab); Systemic Lupus International Collaborating Clinics frailty index (SLICC-FI).

TOUR3C

Placental Abnormalities in Systemic Lupus Erythematosus : Novel Markers of Adverse Pregnancy Outcomes

Kaitlin Nuechterlein (McGill University , Montreal); Karl Grenier (McGill University , Montreal); Luisa Ciofani (McGill University Health Centre, Department of Medicine, Division of Clinical Epidemiology, Montreal); Popi Panaritis (Research Institute of the McGill University Health Centre, Montreal); Sasha Bernatsky (McGill University Health Centre, Department of Medicine, Division of Rheumatology, Division of Clinical Epidemiology, Montreal); Évelyne Vinet (McGill University Health Centre, Department of Medicine, Division of Rheumatology, Division of Clinical Epidemiology, Montreal)

Objectives: Placenta-mediated adverse pregnancy outcomes (APO) are a huge concern in SLE.

Recent efforts to understand APO include the establishment of the 2016 Amsterdam classification criteria, developed to standardize placental pathology evaluation. No study to date evaluated the Amsterdam criteria in SLE. Using the prospective "Lupus prEGnAnCY (LEGACY)" biobank, we assessed the relationship between placental abnormalities, lupus anticoagulant (LAC), and APO, applying the Amsterdam criteria.
Methods: LEGACY is a prospective cohort enrolling SLE pregnancies (before the 17th gestational week). Relevant information is collected at each trimester and/or end-of-pregnancy visits. We evaluated pregnancies delivered beyond 17 weeks at the Montreal site. Placental pathology was defined as abnormal if fulfilling at least one of the 4 main Amsterdam classification subtypes: 1) maternal vascular malperfusion, 2) fetal vascular malperfusion, 3) acute chorioamnionitis, and/or 4) villitis of unknown etiology. Pregnancies with and without abnormal pathology were further characterized based on presence of LAC and APO (i.e. stillbirth, placental insufficiency, gestational hypertension, preeclampsia, small-for-gestational age neonate <5%).
Results: Of 44 LEGACY pregnancies delivered (beyond 17 weeks), 32 (73%) had placental pathology available. Among these 32, 15 (47%) had abnormal pathology. Of those with abnormal pathology, 6/15 (40%) had maternal vascular malperfusion, 5/15 (33%) acute chorioamnionitis, 4/15 (27%) villitis of unknown etiology, and 1/15 (7%) fetal vascular malperfusion. Mean gestational age at delivery was substantially lower in pregnancies with abnormal pathology [mean 33.7 weeks, standard deviation (SD) 6.8] versus those with normal pathology (mean 37.8 weeks, SD 1.7), with a difference in mean gestational age of -4.1 weeks (95% CI -0.6, -7.6). LAC was more frequent in pregnancies with abnormal pathology (4/15; 27%) as opposed to pregnancies with normal pathology (2/17; 12%). APO occurred in 8/15 (53%) pregnancies with abnormal pathology (including 3 with early preterm preeclampsia <34 weeks) as opposed to 7/17 (41%) pregnancies with normal pathology (none with early preterm preeclampsia). Maternal vascular malperfusion was strongly associated with APO (odds ratio 8.1; 95% CI 0.8, 83.7), although the CI included the null.
Conclusion: In this cross-sectional analysis, SLE pregnancies with abnormal placenta pathology, particularly maternal vascular malperfusion, experienced shorter gestation and more severe placenta-mediated APO, including early preterm preeclampsia. Future studies will aim to expand the sample, and investigate if placental abnormalities in one pregnancy helps predict APO in subsequent pregnancies. **Supported by a CIORA grant**

TOUR3D

Novel Analytes Associated with Cognitive Impairment in Patients with Systemic Lupus Erythematosus: Serum S100A8/a9, Mmp-9 and Il-6

Emma Neary (McGill University, Montreal); Carolina Munoz-Grajales (University of Toronto, Toronto); Joan Wither (Schroeder Arthritis Institute, Krembil Research Institute, Toronto Western Hospital, Toronto); Juan Diaz-Martinez (Division of Rheumatology, Toronto Western Hospital, University Health Network; University of Toronto, Toronto); Michelle Barraclough (University of Manchester, Manchester); Kathleen Bingham (UHN, Toronto); Roberta Pozzi Kretzmann (University Health Network, Toronto Western Hospital, Toronto); Carmela Tartaglia (University Health Network, Toronto); Lesley Ruttan (University Health Network, Toronto); May Choi (University of Calgary, Calgary); Simone Appenzeller (University of Campinas , São Paulo); Sherief Marzouk (University of Toronto, Toronto); Dennisse Bonilla (UHN, Toronto); Patricia Katz (University of California San Francisco, Professor of Medicine and Health Policy,

Division of Rheumatology, Department of Medicine, San Francisco); Dorcas Beaton (University of Toronto/Institute for Work and Health, Toronto); Robin Green (University Health Network, 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); Zahi Touma (Schroeder Arthritis Institute, Krembil Research Institute, Toronto Western Hospital, Toronto)

Objectives: Cognitive impairment (CI) is a common manifestation in patients with systemic lupus erythematosus (SLE). Despite its impact on patient quality of life, treatments remain limited as its pathogenesis is poorly understood. The Automated Neuropsychological Assessment Metrics (ANAM) has superior patient acceptability and feasibility in ambulatory settings compared to the American College of Rheumatology Neuropsychological Battery (ACR-NB) [gold-standard test] and is validated in screening for CI in SLE. Data from our laboratory have revealed that serum S100A8/A9 and MMP-9 are associated with CI measured by the ACR-NB. We therefore aimed to determine if serum analytes are associated with CI measured by the ANAM.

Methods: We cross-sectionally analyzed the data of 327 adults aged 18-65 who were followed longitudinally between January 2016 and October 2019 at a single SLE center. All participants fulfilled the 2019 EULAR/ACR SLE classification criteria. Cognitive function was measured using ANAM throughput scores, and serum levels of 9 analytes (IL-10, IL-6, IFN-γ, TNF-α, TWEAK, S100B, S100A8/A9, NGAL and MMP-9) were measured using ELISA. The K-means algorithm was used to cluster patient data, and the Principal Component Analysis (PCA) characterized the clusters. The silhouette coefficient was calculated for 2 to 15 clusters to determine the optimal number of clusters.

Results: PCA identified two principal components explaining 36.2% of the variance in ANAM throughputs and serum analytes. The first component (26.7% of the variance) was correlated with ANAM throughputs, with the strongest contribution from procedural reaction time. The second component (9.44% of the variance) was correlated with serum analyte measurements, with the strongest contribution from TNF-alpha. A 2-cluster model had the highest silhouette value and classified the most patients. Cluster 1 had low throughput scores representing CI, and Cluster 2 had higher throughput scores representing no CI. A significant difference was observed in mean serum S100A8/A9 (SMD=0.362), MMP-9 (SMD=0.178) and IL-6 (SMD=0.311) between the clusters, reflected by their correlation with the first principal component. [Figure 1] Serum levels of S100A8/A9, MMP-9 and IL-6 had a strongly negative correlation between the Go No Go and Running Memory throughputs.

Conclusion: Serum S100A8/A9, MMP-9, and IL-6 are associated with CI in SLE as measured by the ANAM. Patient clusters with elevated serum S100A8/A9, MMP-9, and IL-6 had strongly negative associations with throughputs representing impairment in executive function, simple attention and processing speed. Further studies are needed to uncover mechanistic relationships between these analytes and CI in SLE, and whether they may represent valuable therapeutic targets.

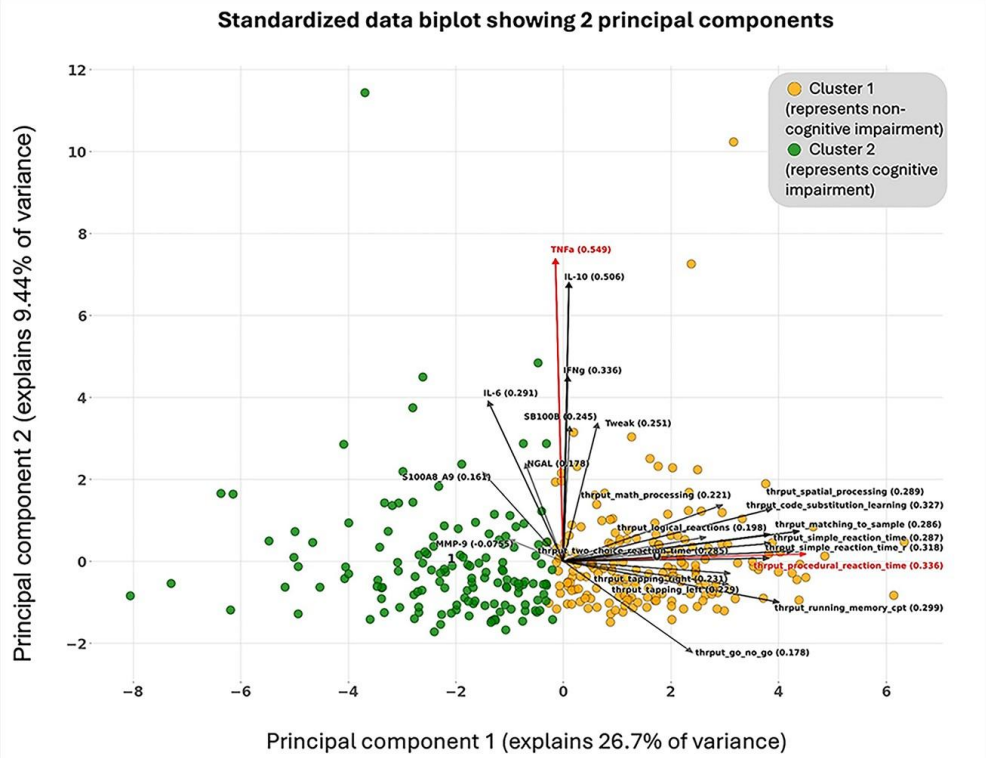

Figure interpretation: The biplot displays the clusters projected on the first two principal components, which explains 36.2% of the variance in ANAM throughputs and serum analytes. Axis x represents the first component (explains 26.7% of the variance), which correlates with ANAM throughputs. Axis y represents the second component (explains 9.44% of the variance), which correlates with serum analyte measurements. The arrows represent the variables, and their direction indicates the relationship between the variables and the clusters. The length of the arrows indicates the strength of the relationship between the variable it represents and the cognitive dimensions.

Poster Tour 4: Vasculitis
Moderator: Jan Willem Cohen Tervaert

TOUR4A

Understanding Referral and Diagnostic Pathways for Giant Cell Arteritis

Vanessa Kissner (University of Alberta, Edmonton); Steven Katz (University of Alberta, Edmonton); Alison H. Clifford (University of Alberta, Edmonton)

Objectives: Giant cell arteritis (GCA) is considered a rheumatologic emergency due to early risks of vision loss and stroke. At our centre, there is currently no standard pathway for referral or diagnostic testing of GCA, which may result in diagnostic delays and/or unnecessary exposure to prednisone. The aim of this study was to better understand the pathways followed by patients referred for suspected GCA to identify barriers to care and areas for improvement.

Methods: Charts of patients referred to University of Alberta Rheumatology for query GCA between January 2022 and January 2024 were retrospectively reviewed. Second opinions and transfer of care referrals were excluded. Patient demographics, number of days between referral and first Rheumatology visit, types of diagnostic tests ordered (imaging, labs, biopsies), time to confirmation/exclusion of diagnosis of GCA (days) and cumulative exposure to prednisone until diagnosis determined (mg) were recorded. Data was analyzed using descriptive statistics.

Results: Of 779 patients with a triage diagnosis of vasculitis or polymyalgia rheumatica, 81 new

referrals for query GCA were identified. Of these 81, 60 (74.1%) were women and mean age was 72.1 (+/-10.3) years. Most were referred by family medicine (36, 44.4%), ophthalmology (14, 17.3%), and emergency medicine (14, 17.3%). Median time between referral and first rheumatology visit was 9 (0-113) days. The most frequently ordered diagnostic test was temporal artery biopsy (66, 81% of patients), followed by PET scan (32, 39.5%), and temporal artery ultrasound (3, 3.7%). Ultimately, a diagnosis of GCA was made in 53 (65.4%) of referred patients [confirmed by either biopsy or imaging in 38 (47%), or clinically-diagnosed in the absence of a positive test in 15 (18.5%)], and excluded in 28 (34.6%). Median time between Rheumatology referral to result of first confirmatory test was 13 (3-529) days, and median time to exclusion of GCA diagnosis was 25 (0-189) days. Ultimately, 66 (81%) patients received prednisone while work up was performed, with side effects reported in 42 (63.6%). The median cumulative prednisone exposure to those in whom GCA was ultimately excluded was 785 (0-4890) mg.

Conclusion: Of patients referred to rheumatology for suspected GCA, the diagnosis was confirmed by testing in 47%, made clinically in 18%, and excluded in 35%. There was wide variability in time to confirmation/exclusion of diagnosis, during which patients received large amounts of prednisone. These findings indicate the need for a standard referral/diagnostic pathway to expedite testing and reduce unnecessary exposure to high dose prednisone.

TOUR4B

Antibiotic Prophylaxis During Treatment of Anca-Associated Vasculitis with Rituximab: Data from a Canadian Multicenter Cohort

Natasha Le Blanc (McGill University, 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); 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); Arielle Mendel (McGill University Health Centre, Department of Medicine, Division of Rheumatology, Montreal)

Objectives: Antibiotic prophylaxis is recommended during initial treatment of ANCA-associated vasculitis (AAV). We assessed characteristics associated with prophylaxis within a Canadian multicenter AAV cohort starting rituximab (RTX) for either induction or maintenance of remission.

Methods: We performed a secondary analysis of baseline characteristics from the Biosimilars in ANCA-associated Vasculitis compared to the Originator (BRAVO) cohort, including adults with granulomatosis with polyangiitis (GPA) or microscopic polyangiitis (MPA) who started RTX induction or maintenance (either the originator or a biosimilar) at cohort entry. We stratified characteristics (at time of starting RTX induction or maintenance, as applicable) according to

concurrent antibiotic prophylaxis (e.g. trimethoprim sulfamethoxazole [TMP-SMX], dapsone, atovaquone), and compared groups using 95% confidence interval (CI) for the differences in mean or proportion, or the Mann Whitney U test as applicable. Characteristics of interest included demographics, current prednisone dose, disease activity, disease duration and prior relapse. Univariable and multivariable logistic regressions assessed the association of covariates of interest with prophylaxis.
Results: Of the 200 participants in BRAVO, 126 initiated induction and 58 initiated maintenance at baseline. [Table 1]. The proportion taking prophylaxis at the start of induction (81%) and maintenance (79%) was similar, whereas 98 (96%) of the induction group and 39 (85%) of the maintenance group were receiving prednisone at baseline. TMP-SMX was the most commonly used antibiotic during induction (95%) and maintenance (89%), with the remainder being primarily atovaquone (2% induction, 11% maintenance) and dapsone (2% in induction). In the induction group, any prednisone use (OR 4.9; 95% CI 1.1-21.4), prednisone ≥20 mg/day (OR 6.5; CI 2.5-17.0) and baseline Birmingham Vasculitis Activity Score (BVAS) (OR for each point 1.2; 95 CI 1.1-1.3) were associated with antibiotic prophylaxis, while use of other concurrent immunosuppressants (OR 0.3; 95% CI 0.1-0.8) and having ≥1 prior relapse (0.3 OR; 95% CI 0.1-0.9) were negatively associated with prophylaxis. In multivariable analyses, daily prednisone dose ≥20 mg (adjusted OR 3.4; 95% CI 1.2-10.0) and BVAS (aOR 1.1, 95% 1.0-1.2) remained associated with prophylaxis. In the maintenance group, prednisone use was also associated with prophylaxis in adjusted analyses (aOR 6.1, 95% CI 1.3-27.0).
Conclusion: In this Canadian cohort initiating RTX for induction or maintenance, 80% of the participants were taking antibiotic prophylaxis, which is higher than reported in other cohorts. During the induction and maintenance period, concurrent prednisone was associated with prophylaxis, as was higher disease activity during induction. Our results permit further understanding of Canadian patterns of antibiotic prophylaxis in AAV. **Supported by a CIORA grant**

TOUR4C
Intravenous or Oral Glucocorticoids as Initial Therapy for Transient and Permanent Visual Changes in Giant Cell Arteritis: a Retrospective Cohort Study
Mats Junek (McMaster University, Hamilton); Rahul Chanchlani (McMaster University, Hamilton); Amadeo Rodriguez (McMaster University, Hamilton); Nader Khalidi (McMaster University, St Joseph's Healthcare Hamilton, Hamilton); Amber Molnar (McMaster University, Hamilton)
Objectives: Guidelines for the treatment of giant cell arteritis-associated vision changes (GCAVCs) recommend the use of intravenous over oral glucocorticoids to improve visual outcomes. These recommendations are based on limited data. We assessed the impact of oral or intravenous glucocorticoids on visual outcomes in a real-world cohort of individuals with GCAVCs.
Methods: We conducted a retrospective cohort study of individuals from a tertiary healthcare facility in Ontario, Canada between December 2017 and November 2023. Individuals included in the cohort were aged 50 or older and assigned a validated diagnostic code for GCA during an inpatient, outpatient, or emergency department visit. Diagnoses of GCA were based on the final diagnosis of the treating clinician and were required to be supported by histology, imaging findings suggestive of inflammatory vasculopathy, and/or the presence of increased inflammatory markers. GCAVCs were confirmed by the treating rheumatologist,

ophthalmologist, or internist. Results were described using summary statistics. The impact of intravenous (IV) or oral glucocorticoids on visual improvement as documented by either the patient's rheumatologist or ophthalmologist visual outcomes was assessed using multivariable logistic regression (adjusted for age, sex, and time from symptom onset to treatment). A subgroup analysis was performed in patients who had GCAVCs associated with permanent visual loss (ischemic optic neuropathy and retinal artery occlusion).
Results: The cohort included 289 patients with a diagnosis of GCA, of whom 77 (26.6%) experienced GCAVCs. GCAVCs included 51 cases of ischemic optic neuropathy, 6 of amaurosis fugax, 5 retinal artery occlusions, and 15 others. Individuals with transient and permanent GCAVCs were treated with oral (72.7% and 50.0% respectively) or intravenous and oral (25.0% and 50.0% respectively) glucocorticoids. Eight individuals with ischemic optic neuropathy or retinal artery occlusion had visual improvement including 5 of 27 (18.5%) who received oral glucocorticoids and 3 of 25 (12.0%) who received intravenous glucocorticoids. Complete data was available for 31 of 77 (40.3%) of cohort participants due to lack of data concerning date of glucocorticoid initiation; logistic regression was performed both including and excluding time from symptom to glucocorticoid initiation. Neither analysis demonstrated an association between the use of IV glucocorticoids and improved visual outcomes (p values 0.12 – 0.99) (table).
Conclusion: Our cohort demonstrated that IV glucocorticoids, compared to oral glucocorticoids, was not associated with visual improvement in individuals who experienced GCAVCs. Conclusions are limited by the small number of events and observational nature of the data.
Upload:

Table: results of multivariable logistic regression assessing intravenous versus oral glucocorticoids for visual improvement as judged by the treating ophthalmologist and/or rheumatologist. Results are expressed as odds ratios with their corresponding 95% confidence intervals. n reflects the sample size either including or excluding data concerning the interval between symptom onset and treatment.

	All visual impairment (n=31)	All visual impairment (n=65)	Permanent visual impairment (n=20)	Permanent visual impairment (n=47)
Use of intravenous glucocorticoids compared to oral glucocorticoids	0.87 (0.18-4.22)	0.43 (0.15-1.25)	1.03 (0.03-33.07)	0.62 (0.11-3.46)
Age	0.97 (0.88-1.08)	0.95 (0.89-1.01)	0.97 (0.79-1.19)	0.91 (0.83-1.00)
Sex (referent: male)	3.18 (0.62-16.28)	1.19 (0.41-3.49)	∞	0.50 (0.09-2.78)
Interval between symptom onset and treatment (days)	1.00 (1.00-1.00)	-	1.00 (0.99-1.01)	-

TOUR4D
Validating the Accuracy of Diagnostic Codes for Vision Changes in Giant Cell Arteritis Using Healthcare Administrative Data from a Tertiary Hospital in Ontario, Canada
Mats Junek (McMaster University, Hamilton); Rahul Chanchlani (McMaster University, Hamilton); Amadeo Rodriguez (McMaster University, Hamilton); Nader Khalidi (McMaster University, St Joseph's Healthcare Hamilton, Hamilton); Amber Molnar (McMaster University, Hamilton)
Objectives: Trends in the incidence and prevalence of giant cell arteritis-associated vision changes (GCAVCs) are unknown and may change over time in response to models of care and

new therapeutic options. We developed and validated a case definition for GCAVCs to accurately identify these patients from healthcare administrative data.
Methods: We conducted a retrospective cohort validation study of individuals from a tertiary healthcare facility in Ontario, Canada using routinely collected healthcare administrative as well as clinical data between December 2017 and November 2023. The reference standard for GCAVC was the presence or absence of visual changes attributable to GCA as assessed by the treating rheumatologist, ophthalmologist, or internist. We simulated population level administrative data using international classification of disease version 10 (ICD10) diagnostic codes that are directly uploaded to national healthcare administrative databases. Individuals included in the cohort were aged 50 or older and assigned an ICD10 code for GCA during an inpatient, outpatient, or emergency department visit. We captured cases of GCA using a previously validated definition and assessed the diagnostic accuracy of case definitions for GCAVCs within these cases (1). Multiple candidate case definitions including the use of ICD10 codes, visits to ophthalmology, and/or time window between onset of vision changes and the first visit to a rheumatologist were used. The diagnostic accuracy of each case definition was calculated, and the optimal case definition was chosen based on sensitivity and positive predictive value while maintaining specificity.
Results: The cohort included 392 individuals diagnosed with GCA; 77 (26.6%) of which had confirmed GCAVCs based on chart review. For those classified as having GCA using the previously validated case definition, the case definition for GCAVCs that displayed optimal performance was any ICD10 code for GCAVCs within one year of the first ICD10 code for GCA with 53.3% (95% confidence interval 38.0-61.7%) sensitivity, 96.0% (91.1-98.4%) specificity, 80.0% (60.9-91.6%) positive predictive value and 87.2% (80.9-91.7%) negative predictive value (Table 1). Case definitions were more sensitive for permanent visual loss (ischemic optic neuropathy and retinal artery occlusion) than temporary visual loss (amaurosis fugax and diplopia).
Conclusion: We developed a case definition that can be used to capture GCAVCs within healthcare administrative data. The definition can be used to create healthcare administrative cohorts of individuals with GCAVCs to better inform treatment patterns and outcomes at the population level.

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Table 1: case definitions with diagnostic performance. Bold highlights the parameter that had the best performance for a given case definition; grey indicates the optimal case definition. O = ophthalmology visit, ICD10 code = international classification of diseases version 10 diagnostic code for vision changes.

Case Definition	Sensitivity	Specificity	PPV	NPV	Youden Index	Sensitivity & PPV
Any ICD code	53.3 (38-61.7)	96.0 (91.1-98.4)	**80.0 (60.9-91.6)**	87.2 (80.9-91.7)	49.3%	133.3%
2+ ICD codes	44.4 (30-52.9)	96.6 (91.9-98.8)	**80.0 (58.7-92.4)**	85.2 (78.7-90.0)	41.1%	124.4%
Any visit to Ophthalmology	71.1 (55.5-79.3)	71.1 (63.1-78.1)	42.7 (31.5-54.6)	89.1 (81.7-93.8)	42.3%	113.8%
O within 3 months of first visit to rheumatology	71.1 (55.5-79)	77.9 (70.2-84.1)	49.2 (36.7-61.8)	89.9 (83.1-94.3)	49.0%	120.3%
O within 1 month of first visit to rheumatology	60.0 (44.4-68.6)	82.6 (75.3-88.1)	50.9 (37-64.7)	87.2 (80.3-92.1)	42.6%	110.9%
O between 1 month before and 3 months after first visit to rheumatology	64.4 (48.7-72.8)	80.5 (73.1-86.4)	50.0 (36.7-63.3)	88.2 (81.3-92.9)	45.0%	114.4%
O between 3 months before and 1 month after first visit to rheumatology	66.7 (50.9-74.9)	79.9 (72.3-85.8)	50 (37-63)	88.8 (81.9-93.4)	46.5%	116.7%
Any O AND any ICD Code	35.6 (22.3-44)	96.6 (91.9-98.8)	76.2 (52.4-90.9)	83.2 (76.6-88.3)	32.2%	111.8%
O within 3 months of first visit to rheumatology AND any ICD Code	35.6 (22.3-44)	**97.3 (92.8-99.1)**	**80.0 (55.7-93.4)**	83.3 (76.8-88.4)	32.9%	115.6%
Any O OR any ICD Code	**88.9 (75.2-93.9)**	70.5 (62.4-77.5)	47.6 (36.7-58.7)	95.5 (89.2-98.3)	59.4%	**136.5%**
O within 3 months of first visit to rheumatology OR any ICD Code	**88.9 (75.2-93.8)**	76.5 (68.7-82.9)	53.3 (41.5-64.8)	**95.8 (90-98.4)**	**65.4%**	142.2%

Poster Tours 5-8: Friday, February 28, 2025, at 12:00 PM – 12:30 PM MT

Poster Tour 5: Myositis
Moderator: Marie Hudson

TOUR5A

Lung Transplantation Outcomes of Patients with Interstitial Pneumonia with Autoimmune Features

Alec Yu (Division of Rheumatology, University of British Columbia, Vancouver); Hyein Kim (Division of Rheumatology, The University of British Columbia, Vancouver); Robert Levy (Division of Respiratory Medicine, University of British Columbia, Vancouver); Jennifer Wilson (Division of Respiratory Medicine, University of British Columbia, Vancouver); John Yee (Division of Thoracic Surgery, University of British Columbia, Vancouver); Kun Huang (Division of Rheumatology, University of British Columbia, Vancouver)

Objectives: Interstitial pneumonia with autoimmune features (IPAF) is a designation proposed in 2015 by the European Respiratory Society/American Thoracic Society (ERS/ATS) to describe patients who have interstitial lung disease (ILD) and combinations of clinical, serologic, and/or pulmonary morphologic features of an underlying systemic autoimmune condition, but do not meet rheumatologic criteria for a characterized connective tissue disease (CTD) (1). Previous studies have suggested that patients with IPAF have worse clinical outcomes and survival compared to those with CTD-associated ILD, but better than those with idiopathic pulmonary fibrosis (IPF) (2). Survival after lung transplantation is similar between CTD-associated ILD and IPF (3). Limited data are available regarding post-lung transplant outcomes in patients with

IPAF. The objective of our study is to compare post-transplant survival, lung function, and other complications in IPAF with IPF.
Methods: We reviewed the data of all patients who underwent lung transplantation in British Columbia, Canada between January 1, 2014, and April 30, 2024. Diagnoses of IPAF were made through a multidisciplinary approach in conjunction with ILD respirologists, chest radiologists, rheumatologists, and pulmonary pathologists. All IPAF cases met the 2015 ERS/ATS criteria and had compatible explant pathology. Continuous variables were assessed with Mann-Whitney U tests and categorical variables with $\chi 2$ tests.
Results: We identified 16 patients with IPAF and compared them to 32 randomly selected patients with IPF. Patient baseline characteristics before lung transplant and post-transplant outcomes are summarized in Table 1. Before transplant, patients with IPAF were more likely to be on immunosuppression (63% vs 6%, $p<0.001$), and less likely to be on antifibrotics (19% vs 69%, $p<0.001$). Age, smoking status, sex, Charleson comorbidity index, and pre-transplant forced vital capacity (FVC), diffusing capacity for carbon monoxide (DLCO), and pulmonary artery systolic pressure (PASP) were similar between the two groups. Post lung transplant, no difference was seen in survival at 1 year or cumulative survival at the last follow-up. At 1-year follow-up, no difference was seen in FVC and DLCO, rates of acute rejection, or hospitalization for infection. At last follow-up, no difference was seen in the rates of chronic lung allograft dysfunction (CLAD) or post-transplant malignancy.
Conclusion: We found no significant difference in post-transplant survival, lung function, infectious or rejection complications in patients who underwent lung transplantation for IPAF compared with IPF at our centre. This study is the first of its kind to report both short-term and long-term outcomes of lung transplantation in patients with IPAF.

TOUR5B

Optimizing Myositis Care with Physiotherapy Integration: a Quality Improvement Project

Fergus To (University of British Columbia, Division of Rheumatology, Vancouver); Ruby Xie (Mary Pack Arthritis Centre, Vancouver); Kei Nishikawa (Mary Pack Arthritis Centre, Vancouver)

Objectives: Best practice guidelines recommend that patients with idiopathic inflammatory myopathies (IIM) be assessed by a physiotherapist (PT) [1] with expertise in IIM. However, an audit at a single IIM center revealed that only a limited number of patients were receiving this care. The aim of this quality improvement (QI) project was to achieve a significant increase in IIM patients at the VCH Myositis Clinic that are assessed by the in-house PT.
Methods: A team consisting of a PT, registered nurse (RN), and physician, all with IIM expertise, investigated the root causes of low PT assessment rates using an Ishikawa fishbone diagram. Process mapping of existing PT assessment steps led to change ideas, which were prioritized through a PICK chart exercise. The following primary interventions were tested through PDSA cycles: 1. Patient education handout on the importance of PT in IIM management. 2. Standardized RN checklist to confirm PT engagement. 3. Same-day PT assessment following a physician visit (either a full 1-hour assessment or a 20-minute intake). 4. Automatic PT referral for patients unable to attend a same-day visit. Improvement measures included the number of patients receiving either a full PT assessment or a 20-minute intake, and the number of automatic referrals leading to a PT visit within 4 months. Patients receiving only a 20-minute intake were not considered to have had a complete PT assessment for the purposes of the study. Data were

collected for 4 months pre-intervention and 6 months post-intervention and median rates during these periods were compared using run charts.
Results: The mean pre-intervention PT assessment rate was 21%. Post-intervention, the rate increased to 68%, as shown by a shift in the run diagram (Figure 1). As well, post-intervention, 28% patients received a 1-hour same-day PT assessment, and 12.5% received a 20-minute intake. 47% of patients received internal referrals and of these, 80% received a PT visit within 4 months.
Conclusion: Utilizing PDSA cycles and targeted interventions, the project demonstrated a measurable improvement in the rate of PT assessments. The post-intervention data showed a significant and sustained increase in PT engagement, indicating that these changes have positively impacted patient care. Further efforts to refine these interventions will be essential maintaining care of IIM patients.
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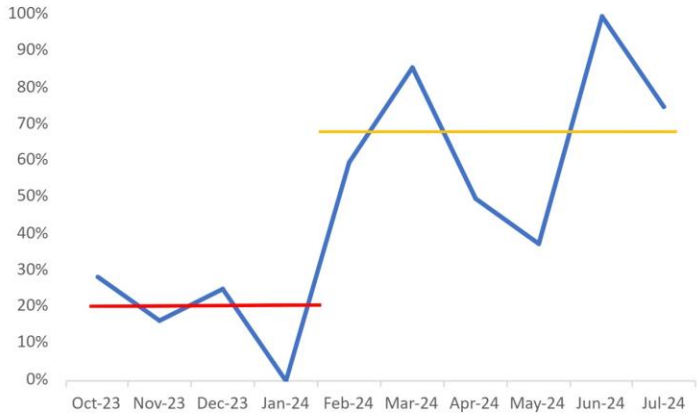

Figure 1. Run chart demonstrating significant shift in rate of IIM patients assessed by a PT with expertise in their disease before and after implementation of interventions.

TOUR5C
Association Between Long-Term Exposure to Fine Particles and Its Components and the Onset of Systemic Autoimmune Rheumatic Diseases in Quebec
Mareva Geslin (Département de Santé Publique, Hôpital Pontchaillou, Centre Hospitalo-Universitaire, Renne); Julien Vachon (Department of Environmental and Occupational Health, University of Montreal, Montreal); Naizhuo Zhao (Research Institute of the McGill University Health Centre, Montreal); Elhadji Laouan-Sidi (Institut national de santé publique du Québec , Montreal); Sonia Jean (Institut national de santé publique du Québec, Québec); Audrey Smargiassi (Université de Montréal, Montreal); Sasha Bernatsky (McGill University Health Centre, Department of Medicine, Division of Rheumatology, Division of Clinical Epidemiology, Montreal)
Objectives: Exposure to fine particles (PM2.5) has been associated with numerous health effects including with autoimmune rheumatic diseases (SARDs). While PM2.5 are composed of various material that are likely to present different toxicity, associations between PM2.5 components and health outcomes remains largely unexplored. Our objective was to investigate the relationships between long-term exposure to PM2.5 and its components and the onset of new cases of SARDs in adults of Quebec.
Methods: We used a retrospective open cohort of the adult population of the province of Quebec without SARD in the previous four years, created with administrative health data from April 1,

2000 to December 31, 2019. SARD cases included lupus, dermatopolymyositis, systemic sclerosis, Sjogren syndrome, fibrosclerosis, diseases of connective tissue. Mean annual levels of PM2.5 and its components, estimated with satellite aerosol optical depth and various models, were attributed to the six digit postal code of each participant; the components included black carbon (BC), mineral dust, sea salt (SS), nitrate, SO42, NO3-, organic matter (OM). Cox models with adjustments for age, sex, year, socioeconomic status, Local Service Network and urban/rural regions were generated for PM2.5 and its seven components. Quantile-based g-computation with similar adjustments was used to estimate the marginal hazard ratio (HR) for the mixture of PM2.5 components.
Results: 7,482,397 individuals were analyzed, representing 98,039,304.7 person-years; 55,267 incident cases were observed. In proportion, OM was the main component of PM2.5. The adjusted HR for SARDs was 1.022 per increase of an interquartile range of PM2.5 (95% confidence interval (95%CI): 0.985 – 1.060). Estimates of associations for the components were positive, except for SS and SO42-, although their 95%CI all included unity. Based on the quantile g-calculation, the HR of SARD was slightly positive (HR per one-decile increase in the PM2.5 components: 1.01; 95% CI: 1.00 - 1.02).
Conclusion: This large-scale cohort study suggests that annual exposure to PM2.5 and the mixture of its components may be associated with the incidence of certain SARDs.

Poster Tour 6: Pediatric Rheumatology
Moderator: Dax Rumsey

TOUR6A
Juvenile Idiopathic Arthritis and Out-Of-Pocket Costs for Medical and Other Health Professional Visits: the Ucan Can-Du and Cure International Prospective Study
Deborah Marshall (University of Calgary, Calgary); Rodrigo Dal Ben (University of Calgary , Calgary); Gillian Currie (University of Calgary, Calgary); Rae Yeung (Division of Rheumatology, The Hospital for Sick Children, Toronto); Sebastiaan Vastert (University of Utrecht, Utrecht); Joost Swart (University of Utrecht, Utrecht); Susanne Benseler (Section of Rheumatology, Department of Paediatrics, Alberta Children's Hospital/University of Calgary, Calgary); UCAN CAN-DU and UCAN CURE Consortia (Toronto)
Objectives: To measure out-of-pocket costs related to medical and other health professional visits due to juvenile idiopathic arthritis (JIA).
Methods: The Canada-Netherlands Personalized Medicine Network in Childhood Arthritis and Rheumatic Diseases (UCAN CAN-DU) and CURE study captures consecutive caregiver reported data including out-of-pocket costs on travel to doctor appointments, treatments or tests, emergency visits or hospitalization, plus costs for travel and treatments related to visiting other health professionals. The cohort includes all subtypes of JIA, and patients across the disease trajectory from new diagnosis to starting or stopping biologic medications. The analytic sample included those with complete data over one year period from enrollment in the study on out-of-pocket travel expenses, and cost (in CAD). Generalized Estimating Equations were used to estimate average annual cost as a function of JIA activity state (e.g., inactive, minimal, moderate/high), time from enrollment (i.e., baseline, 3, 6, 9, 12 months), and patient age group (i.e., 0-4, 5-9, 10-15, 16+ years).
Results: Our analytic sample included 115 patients. Over 1-year, 91% of families reported visiting doctors with the most visited being rheumatologists (44%), then eye doctors

(33.8%). Travel cost was $311 (SDpooled $154; range $10-7,367). Travel costs were comparable across covariates in the GEE. For 40% who reported visits for treatments or tests, the most frequent was lab tests (37.4%) and shots/infusions (22.4%). Travel cost was $364 (SDpooled $106; range $12-2,880). These costs were significantly higher for moderate/minimal disease activity compared high disease activity (related to per-visits, not visit numbers), and significantly higher over time, but significantly lower for older patients. Few families had emergency (5.6%) or hospitalizations (2.2%). Travel cost for these was $403 (SDpooled $122; range $72-1,028), and significantly higher for patients 16 years and older. Over 1-year, 57.4% of families reported visiting other health professionals. Of these, 48% reported not paying any portion, the rest paid part/all. Health professional treatment costs was $5,207 (SDpooled $2,658; range $45-33,058), with costs significantly lower for older patients. The most visited health professionals were physiotherapists (33.8%), then orthodontists (15.6%), and psychologists (10%). Travel cost was $458 (SDpooled $364; range $9-5,634). There was no significant difference in travel cost across covariates. Overall, more caregivers reported parking costs for health care visits, compared to other health professional visits(71-89% vs 25%). For all visit types, 20-25% reported fuel expenses.
Conclusion: Families with children with JIA have substantial annual out-of-pocket costs, potentially creating financial barriers and inequitable access to care.

TOUR6B
Rapid Review of Transition Outcomes in Youth with Rheumatic Diseases
Maya Rao (University of Western Ontario, london); Nadia Luca (Division of Rheumatology, Department of Pediatrics, Children's Hospital of Eastern Ontario/University of Ottawa, Ottawa); Michelle Batthish (McMaster University, Hamilton)
Objectives: The transition from pediatric to adult healthcare for adolescents with chronic rheumatic diseases is associated with an increased risk of experiencing gaps in care and adverse health outcomes. Despite advancements in structured transition programs, there is currently a lack of standard, validated outcomes to evaluate transitional care. The objective of this study was to systematically review and synthesize the available literature on transition outcomes in rheumatology.
Methods: In the present rapid review, peer-reviewed articles were included if they: (a) included youth (age 12-24) diagnosed with a chronic rheumatic disease transitioning to adult rheumatology care or who had transitioned to adult care; (b) examined structured transition programs, or various aspects of the transition process (including routine follow-up); (c) reported on relevant transition outcomes; (d) were published between 2009 and 2024; and (e) were published in English. Four online databases (Scopus, Medline, Embase, and CINAHL) were searched. Eligible studies were reviewed and extracted data was synthesized.
Results: A total of 28 studies were included in this rapid review. Numerous outcomes were identified that emphasized the complex nature of the transition process. Quality of life, healthcare utilization, patient satisfaction, disease activity and medication adherence were the most frequently reported outcomes. Psychosocial and parental outcomes were less commonly reported, highlighting gaps in the current literature. No single or core set of outcomes were reported across all studies.
Conclusion: The findings of this review underscore the need for comprehensive and standardized outcome measures to accurately assess and compare transition programs. Future

research should prioritize efforts to refine and validate transition outcomes in order to facilitate effective evaluation and improvement of the transition process.

TOUR6C

Identifying Homogeneous Endophenotypes in Childhood-Onset Systemic Lupus Erythematosus with Data Driven Methods

Nicholas Chan (The Hospital of Sick Children, Toronto, Ontario); Nicholas Gold (Division of Rheumatology, The Hospital for Sick Children, Toronto); Anjali Jain (The Hospital of Sick Children, Toronto, Ontario); Deborah Levy (Division of Rheumatology, The Hospital for Sick Children; Child Health Evaluative Sciences, SickKids Research Institute, Toronto); Andrea Knight (Neurosciences and Mental Health Program, SickKids Research Institute; Division of Rheumatology, The Hospital for Sick Children, Toronto); Earl Silverman (Division of Rheumatology, The Hospital for Sick Children, Toronto); Daniela Dominguez (Division of Rheumatology, The Hospital for Sick Children, Toronto); Lawrence Ng (Division of Rheumatology, The Hospital for Sick Children, Toronto); Lauren Erdman (The Hospital of Sick Children, Toronto); Linda Hiraki (Division of Rheumatology, The Hospital for Sick Children; Genetics and Genome Biology, SickKids Research Institute, Toronto)

Objectives: Childhood-onset Systemic Lupus Erythematosus (cSLE) is a clinically heterogeneous autoimmune disease. We hypothesized that data-driven methods would identify clinically homogeneous patient subgroups that may represent cSLE endotypes with distinct genetics.

Methods: We included patients diagnosed with cSLE between January 1992-October 2023. All patients met 2019 ACR-EULAR classification criteria and were genotyped on Illumina multiethnic arrays. Ungenotyped SNPs were imputed with TopMed as a referent. We extracted SLE manifestations, date of each manifestation onset and demographic characteristics from dedicated Lupus databases. Ancestry was genetically inferred using principal components and ADMIXTURE with 1000 Genomes as a referent. We used time to each SLE manifestation onset from SLE diagnosis to identify patient subgroups using similarity network fusion (SNF), a data-driven method. We conducted Kaplan Meier analyses and Cox proportional-hazard models on the time from diagnosis to specific SLE manifestation onset between clusters. We tested cluster differences in demographic and manifestation prevalences using $\chi 2$ or Fisher's exact test, and time to each SLE manifestation onset with log rank tests. We calculated additive, allelic weighted SLE non-HLA and HLA polygenic risk scores (PRS) using alleles from a trans-ancestral SLE GWAS. We tested the association between PRSs and cluster membership adjusting for sex and ancestry.

Results: Our cohort included 442 cSLE patients. 83% were female and the median age of SLE diagnosis was 13.6 years (Q1-Q3:12.0-15.8). Our cohort was primarily composed of patients of European (27%) and East Asian (26%) ancestry. SNF identified 2 clusters. Patients in cluster 1 (n=203) were predominantly of European ancestry (42%), while cluster 2 (n=239) was mainly composed of patients of East Asian (30%) and South Asian (22%) ancestry (P=3x10-9). Patients in cluster 2 had higher prevalence, and earlier onset of class III/IV lupus nephritis, hypocomplementemia, anti-dsDNA and anti-smith antibodies compared to patients in cluster 1 (P<1x10-7; Figure 1). The risk of developing any of 12 specific cSLE manifestations at any time was higher in patients in cluster 2 compared to cluster 1 (HR>1.4; P<4x10-3). SLE PRSs were

not associated with cluster membership (Non-HLA PRS: OR 0.9, 95% CI 0.8-1.2; P=0.5; HLA PRS: OR 1.3, 95% CI 0.8-2.0; P=0.3).
Conclusion: In a large multiethnic cSLE cohort, data-driven methods identified two cSLE patient clusters. The cluster with more severe disease and earlier onset had a greater proportion of East Asian and South Asian patients compared to the cluster with milder disease. Next steps include regressing SLE variants by cluster membership with sequence kernel association tests.

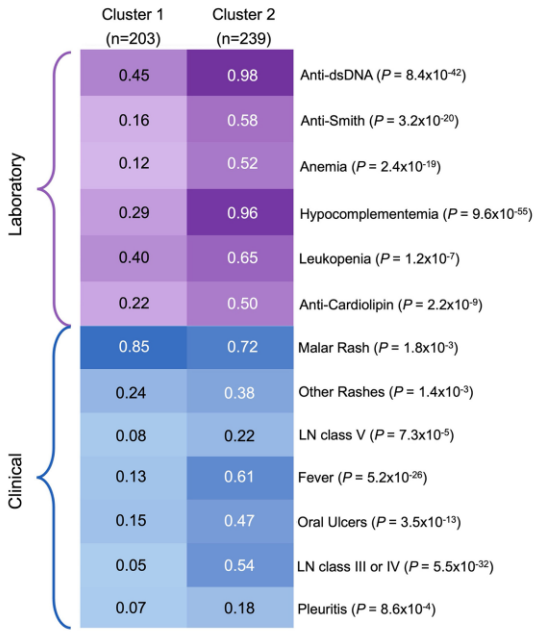

Figure 1. Clinical and Laboratory SLE Manifestations With Different Prevalence Between Patient Clusters. The number within each cell represents the prevalence of an SLE manifestation within cluster 1 and 2, respectively. "LN" stands for lupus nephritis and "Other Rashes" stands for maculopapular, discoid and cutaneous vasculitis rashes. Statistics performed with a Fisher's Exact Test, Bonferroni corrected $P<0.002$.

TOUR6D

Tracer: Transition to Adulthood Through Coaching and Empowerment in Rheumatology, a Feasibility Study

Megan Clarke (McMaster University, Hamilton); Julie Herrington (McMaster University, Hamilton); Tania Cellucci (McMaster University, Hamilton); Liane Heale (McMaster University, Hamilton); Mark Matsos (McMaster University, Hamilton); Karen Beattie (McMaster University, Hamilton); Roberta Berard (Children's Hospital, LHSC, London); Michelle Batthish (McMaster University, Hamilton)

Objectives: The transition to adult healthcare is a vulnerable time for youth with chronic disease, with risks of disengagement and complications of inadequately managed disease. Health Coaching helps patients gain the knowledge, skills, tools and confidence to become active participants in their care. Transition Coaching may be effective in psychologically and socially supporting youth as they gain the skills and tools necessary to independently manage their health. We designed the TRACER (Transition to Adulthood through Coaching and Empowerment in Rheumatology) study to assess the feasibility of conducting a multi-centred RCT of a virtual Transition Coach Intervention (TCI) in youth transferring from pediatric to adult rheumatology care.
Methods: Youth 17-18 years old were recruited at their last pediatric rheumatology appointment at McMaster Children's Hospital or Children's Hospital at London Health Sciences Centre in

Ontario. Participants were randomized to the TCI or control group. Participants in both groups received the Youth Transition Roadmap which informs the differences between pediatric and adult care and discusses five domains of healthcare transition: Self-Advocacy, Medication Management, General Health, Lifestyle Issues and Future Planning related to education and vocation. TCI group participants also received virtual one-on-one monthly coaching sessions for 8-months. Six sessions were with an advanced practice physiotherapist (APP) and two were with a social worker. Primary feasibility outcomes were overall consent rate (≥85%), enrollment from the non-primary site (>30%), coaching session attendance (≥90%), and data collection completion (≥90%). Secondary clinical outcomes collected at baseline, 8- and 11-months included the Transition-Q (transition readiness), PedsQL(TM) 4.0 (global functional assessment), active joint count and Physician Global Assessment (disease activity), and PROMIS® self-efficacy measures for people with chronic disease.
Results: Of 65 patients approached, 30 (46.2%) consented, and 16 (53.3%) were from London. Of those who consented, 2 withdrew after consenting but prior to collecting baseline data and starting the intervention (Figure 1). TCI sessions had a 95.8% attendance rate. Participants completed 90.7% of outcome assessments. At eight months, TCI participants appeared to have higher levels of transition readiness, PROMIS® scores were higher in certain domains, and the TCI was well accepted by participants and coaches.
Conclusion: Our study met all feasibility outcomes except consent rate, which indicates a large scale RCT could be feasible. This study's findings will shape a future multi-centred RCT of a virtual TCI, with the ultimate goal of enhancing the physical, mental, and social well-being of adolescents with rheumatic disease transitioning into adult care.

Poster Tour 7: Basic Science
Moderator: Hani El-Gabalawy

TOUR7A

Impaired Neutrophil Extracellular Trap (Net) Degradation in Rheumatoid Arthritis (Ra) and Pre-Clinical Ra is Mediated by Anti-Net Antibodies

Jeba Maisha (University of Manitoba, Winnipeg); Alina Semenenko (Calgary); Jun Kim (University of Manitoba, Winnipeg); Mario Navarrete (University of Manitoba, Winnipeg); Xiaobo Meng (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), a systemic autoimmune arthritis, begins after a prolonged preclinical phase which is marked by the development of RA antibodies, typically against citrullinated proteins (ACPA). The production of neutrophil extracellular traps (NETs), a form of cell death undertaken by neutrophils, leads to the extrusion of self-DNA decorated in citrullinated proteins, predominantly histones and proteases1. Increased NET abundance has been observed in the blood and at mucosal surfaces in established and preclinical RA, but it is not known if dysregulated NET production or impaired NET degradation, or both, is involved. This study aimed to evaluate NET degradation in a well-established longitudinal cohort of first-degree relatives (FDR) of RA patients, individuals at-risk to develop future RA.
Methods: Total NET complexes (citrullinated Histone-3-dsDNA complexes) were measured in 10% plasma using a custom in-house ELISA. A plate-based NET degradation assay was performed based on past publications. In brief, neutrophils (1.0x106 cells) were cultured in the

presence of A23187 (1uM) to form NETs, which are detected by fluorescence (SYTOX green). Data was used to calculate the % of NETs degraded in quadruplicates. Poor/good NET degraders were determined using group sample median. Anti-NET antibodies were determined by ELISA, using plates coated with isolated NET complexes. DNAse-1 activity was assessed in plasma via fluorescence (abcam).
Results: Using samples from RA (n=30), ACPA- FDR (n=16), ACPA+ FDR (n=16) we observed higher NET complexes (citH3-dsDNA) in the plasma of ACPA+ FDR ($p<0.0001$) and RA patients ($p<0.0001$) compared to ACPA- FDR. NET degradation (using healthy control plasma as positive control, n=10), was impaired in ACPA+ FDR ($p=0.005$) and RA ($p=0.001$), but not in ACPA- FDR. There was no association between NET complexes and NET degradation capacity. Anti-NET antibodies were detected in ACPA+ FDR ($p<0.0001$) and RA ($p<0.0001$) and anti-NET antibody levels were higher in individuals who were labelled as poor NET degraders ($p=0.04$). DNAse-1 activity was no different between any of the sample groups, and not reduced in samples considered poor degraders. Total IgG was isolated from poor NET degraders with high anti-NET IgG (n=3). Pre-incubation of IgG (10% total IgG) reduced the degradation of NETs by HC plasma ($p=0.003$).
Conclusion: We show for the first time that NET degradation is impaired in RA and preclinical RA and that this observation is not related to reduced DNAse-1 activity. Our data suggests that anti-NET antibodies, likely ACPA, reduce NET degradation by binding to protein-DNA complexes, potentially shielding these complexes from local DNAses.

TOUR7B

Validating Differentially Expressed Micro-Rnas that Distinguish Psoriatic Arthritis from Osteoarthritis

Samantha Bestavros (Schroeder Arthritis Institute, Krembil Research Institute, Toronto, ON, Canada, Mississauga); Darshini Ganatra (Schroeder Arthritis Institute, Krembil Research Institute, Toronto, ON, Canada, Toronto); Anas Samman (Schroeder Arthritis Institute, Krembil Research Institute, Toronto, ON, Canada, Toronto); David Nasri (Schroeder Arthritis Institute, Krembil Research Institute, Toronto, ON, Canada, Toronto); Omar Correa (Schroeder Arthritis Institute, Krembil Research Institute, Toronto, ON, Canada, Toronto); Rajiv Gandhi (Schroeder Arthritis Institute, Krembil Research Institute, Toronto, ON, Canada, Department of Surgery, University of Toronto, ON, Canada , Toronto); Mohit Kapoor (Schroeder Arthritis Institute, Krembil Research Institute, Toronto, ON, Canada, Department of Surgery, University of Toronto, ON, Canada , Toronto); 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)
Objectives: Biomarkers may help differentiate psoriatic arthritis (PsA) from osteoarthritis (OA). MicroRNAs have potential to be robust biomarkers, we aimed to 1) Use miRNA sequencing to analyze miRNA expression profiles in the synovial fluid of patients with PsA compared to those with OA, 2) Validate differential expression of select miRNAs in an independent cohort of PsA and OA patients through quantitative real-time PCR (qRT-PCR), and 3) Investigate the potential for differentially expressed miRNAs between PsA and OA to serve as biomarkers for PsA to enhance PsA diagnostic accuracy.
Methods: We performed miRNA sequencing on synovial fluid (SF) aspirated from knees of 12 PsA and 12 OA patients using Illumina NextSeq 550. We used linear modelling with empirical

Bayes moderation (using the Limma R package) for assessing differential expression of miRNAs. For validation, we extracted miRNA from SF samples from an independent set of 35 PsA and 37 OA patients. We investigated the differential expression of miRNAs between the disease states of 12 selected miRNAs using qRT-PCR (99a-5p, 100-5p, 10b-5p, 7c-5p, 125b-5p, 125a-5p, 27b-3p, 26b-5p, 142-3p, 142-5p, 223-3p, and 150-5p). We calculated the differential expression of miRNAs between PsA and OA using fold change, and calculated area under the receiver operating characteristic curve (AUC) values for the select miRNAs.
Results: After miRNA sequencing, 17 miRNAs were found to be significantly differentially expressed between PsA and OA (FDR adjusted p-value less than 0.05) [Fig.1]. From the 12 selected miRNAs analyzed further by qRT-PCR in independent set of patients, statistically significant differences were identified in the expression of 11 out of 12 miRNAs between PsA and OA. PsA patients had lower expression of 99a-5p (FC=0.162, $p<0.0001$), 100-5p (FC=0.091, $p<0.0001$), 125b-5p (FC=0.087, $p<0.0001$), 27b-3p (FC=0.191, $p<0.0001$), 7c-5p (FC=0.330, $p<0.001$), 10b-5p (FC=0.355, $p<0.01$), and 125a-5p (FC=0.500, $p<0.05$) compared to OA patients. PsA patients had higher expression of 150-5p (FC=9.183, $p<0.0001$), 223-3p (FC=7.112, $p<0.0001$), 142-3p (FC=2.432, $p<0.01$), and 142-5p (FC=2.627, $p<0.01$) compared to OA patients. ROC analyses revealed that 4 miRNAs (miR-223-3p, miR-99a-5p, miR-125b-5p, miR-150-5p) had an area under the curve value greater than 0.8.
Conclusion: The validated miRNAs, shown to be differentially expressed in PsA and OA patient synovial fluid across two independent cohorts, may serve as potential biomarkers for PsA, and improve our understanding of PsA's inflammatory mechanisms.
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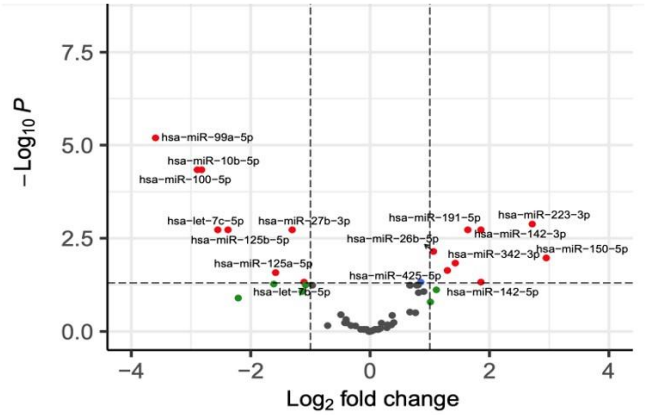

Fig. 1. Volcano plot of log2 fold change (FC) against -log10 FDR adjusted p-value of miRNAs identified through miRNA sequencing. Red dots indicate differentially expressed miRNAs (FDR adjusted p value <0.05 and a log2 FC>±1). miRNAs with FC≥±1 but FDR corrected p-value of ≥0.05 are shown in green. Black dots represent non-significant miRNAs with a log2 FC<±1.

TOUR7C
Investigating Homocitrulline-Specific T-Cell Responses in Rheumatoid Arthritis
Sofya Ulanova (Schulich School of Medicine & Dentistry, London); Gabrielle Buckley (Schulich School of Medicine & Dentistry, London); Mansour Haeryfar (Schulich School of Medicine & Dentistry, London); Ewa Cairns (Western University , London); Lillian Barra (Western University/ Lawson Health Research Institute , London)
Objectives: Rheumatoid Arthritis (RA) is an incurable autoimmune disease affecting 1 in every 100 Canadians. Both B cells and T cells are involved in the pathogenesis of RA. These cells

respond to peptides containing modified amino acids, including citrulline and homocitrulline. Although B-cell responses to homocitrullinated peptides (HomoCitP), also referred to as anti-carbamylated protein (anti-CarP) antibodies, have been studied, less is known about T-cell responses to HomoCitP. This study aims to investigate T cells that respond to HomoCitP in RA patients and in a humanized mouse model expressing the most significant RA genetic risk factor: HLA-DR4.

Methods: Male and female HLA-DR4-transgenic mice (n=6-9) were injected subcutaneously with HomoCitP or phosphate-buffered saline (PBS) as a control. Draining lymph nodes were collected on day 10 post-immunization for flow cytometry analysis. For human studies, blood samples were obtained from RA patients and age- and sex-matched healthy controls. CD4+ T cells were isolated using magnetic negative selection beads and stained with HomoCitP-loaded HLA-DR4-encoded MHC II tetramers to identify and phenotype HomoCitP-specific T cells. Peripheral blood mononuclear cells (PBMCs) were analyzed for cytokine production, and serum was collected for antibody assays.

Results: Mice immunized with HomoCitP, compared to PBS, had a 25-fold increase in the frequency of pro-inflammatory T helper 1 (Th1) cells. [Table 1] Additionally, we detected a 1.8-fold increase in total activated cells with pro-inflammatory Th17 cells being the most abundant (30.5%). Furthermore, mice injected with HomoCitP had elevated levels of terminally exhausted and central memory T cells, which may contribute to chronic disease. When immune cells from these mice were exposed to HomoCitP in vitro, production of the cytokines TNF-α and IFN-γ was triggered, corroborating the inflammatory nature of the response to HomoCitP. Similar preliminary results were observed in RA patients, where PBMCs showed increased production of TNF-α, IFN-γ, IL-17A, and IL-6 cytokines in response to HomoCitP in vitro. [Table 1] Using a tetramer assay, we detected 3-times more HomoCitP-specific T cells in RA patients compared to healthy controls. The majority of these cells were pro-inflammatory Th1 (51%) and Th17 (39%). Lastly, 8/10 RA patients tested positive for HomoCitP antibodies, compared to 1/6 healthy controls.

Conclusion: This study identified HomoCitP-specific T-cell responses and cytokine production in both mice and RA patients. The observed pro-inflammatory responses provide valuable insights into RA pathogenesis and suggest potential new therapeutic targets for better control of disease activity.

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Variables	HomoCitP vs. PBS Injected Mice	Rheumatoid Arthritis patients vs. Healthy controls
HomoCitP-specific T Cells (tetramer+)		586 vs. 194 tetramer+ cells/1 million cells, p-value 0.0039
Th1 Cells (T-bet+)	5.35% vs. 0.205%, p-value 0.0022	Th1 Tetramer+ cells: 51% vs. 56%, p-value 0.55
Th17 Cells (RORγt+)	22.30% vs. 3.27%, p-value 0.0043	Th17 Tetramer+ Cells: 39% vs. 40%, p-value 0.86
Activated T cells (CD25+FOXP3-)	2.53% vs. 1.40%, p-value 0.0036	
Th Subtypes of Activated T Cells	Th1: 8.06% vs. 0.81%, p-value 0.0016 Th17: 30.53% vs. 8.37%, p-value 0.0039	
Central Memory (CD44+CD62L+) T Cells	93.10% vs. 86.87%, p-value 0.0164	
Terminally Exhausted (PD-1+LAG-3+Tim-3+) T Cells	2.21% vs. 0.86%, p-value 0.0242	
Cytokine Production	TNF-α: 1.33% vs. 0.77%, p-value 0.0207 IFN-γ: 3.46% vs. 1.06%, p-value 0.0222	TNF-α: 4/6 patients vs. 1/4 controls IFN-γ: 4/6 patients vs. 0/4 controls Il-6: 5/6 patients vs. 0/4 controls IL-17A: 4/6 patients vs. 1/4 controls
HomoCitP Antibody Levels		RA Patients: high – 3 patients; medium – 2 patients; low – 3 patients; negative – 2 patients Healthy Controls: low – 1 patient; negative – 5 patients

Table 1. Summary of results for mouse and human cell experiments. Values represent the mean percentages of cells or the mean number of cells per 1 million cells. For human HomoCitP antibody levels, an optical density less than 0.1 was considered negative, 0.1–0.5 was considered low, 0.5–1.0 was considered medium, and greater than 1.0 was considered high. HomoCitP antibody concentration was significantly higher in RA patients compared to healthy controls (p = 0.0180). The median age of RA patients was 71 years, while the median age of healthy controls was 61 years (p = 0.0909). The percentage of female RA patients was 80%, compared to 83% in healthy controls.

TOUR7D

Alterations in Innate Immune Populations During Systemic Autoimmune Rheumatic Disease Development

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Objectives: Systemic autoimmune rheumatic diseases (SARD) are a group of chronic diseases characterized by the presence of anti-nuclear antibodies (ANAs) [1]. However, ANAs cannot reliably be used as a diagnostic tool because a subset of healthy women are ANA+ (~20%) and the majority of these individuals will not progress to SARD [2]. Why some individuals progress while others remain asymptomatic is unknown. Our objective is to evaluate functional alterations in innate immune populations during SARD development.

Methods: Experiments have been completed to examine the composition of innate immune cells in PBMCs using CITE-Seq. Samples were used from 5 ANA- healthy controls, 11 ANA+ asymptomatic (5 progressors prior to progression, 6 non-progressors) and 8 early SARD patients. Five million freshly thawed PBMCs were depleted of T and B cells using α-CD3, -CD19 biotinylated antibodies with streptavidin coated magnetic beads. Purified innate immune cells were stained with a panel of oligo-conjugated antibodies for the identification of DC/monocyte populations. 9000 cells were sequenced at a depth of 50000 reads for gene expression and 5000 reads for CITE-Seq.
Results: Using both gene and surface protein expression, we identified 11 DC and monocyte populations [Fig 1A]. Proportional analysis revealed an expansion of non-classical and activated non-classical monocytes in progressor and SARD patients [Fig 1B]. SARD patients also exhibited higher proportions of cDC3s, VCAN+ monocytes and MME+ DCs than ANA+ individuals regardless of progression status [Fig 1B], suggesting a role for these cells in active disease. Comparing ANA+ patients, classical, intermediate, and non-classical monocytes were expanded in progressors while cDC1s, cDC2s and pDCs were expanded in non-progressors [Fig 1B]. Differential expression analysis indicated high expression of interferon stimulated genes in progressors and SARD patients, while non-progressors showed high expression of heat-shock proteins [Fig 1C] which have been shown to promote tolerogenic conditions. These differences in gene expression were observed across cell types, but intermediate monocytes are shown as representative cells due to their role in antigen presentation. Several genes were differentially expressed between progressors and SARD [Fig 1C], potentially highlighting distinct roles for genes in initiating and driving disease. Further analysis will be done to examine pathways associated with these genes.
Conclusion: Our data reveals differences in proportions and gene expression between progressors, non-progressors and SARD patients. Importantly, ANA+ progressors show expanded monocyte populations and functional differences compared to non-progressors even prior to progression. These results will allow us to investigate the immunological differences that may drive progression in SARD.
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Poster Tour 8: Imaging of Rheumatic Disease
Moderator: Marie Clements-Baker

TOUR8A
Introduction of Temporal Artery and Large Vessel Ultrasound for the Diagnosis of Giant Cell Arteritis: a Single Canadian Centre Experience
Raymond Chu (University of Ottawa, Division of Rheumatology, Ottawa); Tara Swami (University of Ottawa, Rheumatology, Ottawa); Mohammad Bardi (University of British Columbia, Division of Rheumatology, Vancouver)
Objectives: Diagnostic imaging in giant cell arteritis (GCA) is increasingly being used to replace biopsy (TAB), with the advent of temporal artery and large vessel ultrasound (TA/LV US) showing better sensitivity and comparable specificity to TAB. The purpose of our study is to share and validate our real-world experience since introducing TA/LV US at The Ottawa Hospital.
Methods: A retrospective chart review was performed on patients referred to RC for query GCA between October 2023-May 2024. We included only suspected new cases of GCA, excluding

Takayasu's arteritis (TAK). Patients were also excluded if taking glucocorticoids for >30 days (known to reduce the sensitivity US). Our TA/LV US protocol measured the intimal medial thickness (IMT) in transverse and longitudinal views of the common/frontal/parietal/facial/subclavian/axillary/carotid arteries. Positive scans were identified based on increased intimal media thickness and presence of haloes/slope signs. We extracted patient demographics, prednisone duration, US and TAB results, and final clinical diagnosis in at least 3 months follow-up. The primary outcome measure was the sensitivity and specificity of TA/LV US and TAB taking the clinical diagnosis as the reference standard.
Results: A total of 170 patients underwent a TA/LV US during this timeframe. Two patients were referred for TAK and of the remaining 168, 77 were excluded given duration of prednisone use. Of the 91 patients remaining, the median age was 73 (interquartile range [IQR], 60–79) years and 54 (59%) were female. 39/91 (43%) patients were given a clinical diagnosis of GCA. Of these, 35/39 (89.7%) had a positive TA/LV US. Of the 58/91 patients who underwent TAB, 35 (60%) were positive. 11/39 patients diagnosed with GCA with US alone and no TAB. Sensitivity and specificity for TA US was 89.7% and 100% while TAB was 85.7% and 100% respectively. After ≥3 months of follow-up 6/52 (12%) of those not diagnosed with GCA were diagnosed with an alternate ailment (Still's, lymphoma, skull invasive meningioma, staphylococcus aureus bacteremia, mast cell activation syndrome and a tooth abscess).
Conclusion: Our brief real-world experience with TA/LV US has demonstrated comparable specificity and marginally better sensitivity for the diagnosis of GCA, although longer term data and follow up would be required to better understand the role for each modality moving forward. Given emerging use of TA/LV US in the diagnosis of GCA and operator dependency, we highlight the importance of validation of technique in the diagnosis of GCA.

TOUR8B

Comparing Large Vessel Uptake of 68Ga-HA-DOTATATE to 18F-FDG Using PET/CT Imaging in Patients with Active Giant Cell Arteritis

Alison H. Clifford (University of Alberta, Edmonton); Jonathan Abele (University of Alberta, Edmonton); Ryan Hung (University of Alberta, Edmonton); Frank Wuest (University of Alberta, Edmonton); Jan Andersson (University of Alberta, Edmonton); Elaine Yacyshyn (Division of Rheumatology, University of Alberta, Edmonton); Eric Lenza (University of Alberta, Edmonton); Glen Jickling (University of Alberta, Edmonton); Paolo Raggi (University of Alberta, Edmonton); Jan Willem Cohen Tervaert (University of Alberta, Edmonton)
Objectives: 18F-fluoro-deoxyglucose (FDG) is currently widely used for PET/CT imaging of large vessel vasculitis (LVV) in giant cell arteritis (GCA), but its performance is influenced by glucocorticoid therapy. We aimed to compare 68Ga-HA-DOTATATE (DOTA), a somatostatin-analogue tracer specific for macrophages, to FDG for detection of LVV using PET/CT in a small number of patients with active GCA.
Methods: In this pilot study, 8 active GCA patients (with disease confirmation by either temporal artery biopsy or positive large vessel imaging) who had initiated glucocorticoids within 2 weeks of enrolment were prospectively consented and sequentially scanned with both FDG PET/CT and 68Ga-HA-DOTATATE PET/CT. Images were reviewed by 2 experienced Nuclear Medicine physicians. Tracer uptake was assessed semi-quantitatively in 8 vascular territories (thoracic and abdominal aorta, bilateral subclavian, vertebral, carotid, femoral/iliac, temporal arteries and coronary arteries) using SUVmax. Target-background ratios (TBR) were calculated

using both right atrium (RA) mean (TBRRA) and liver mean (TBRliver) as comparator. Index vessel was defined as the vessel with the greatest tracer uptake in each participant. SUVmax and TBR's of individual vascular territories and index vessels were compared using students' t tests. $P<0.05$ was considered statistically significant.
Results: The median patient age was 71.5 years (range 64-82), and 4 (50%) were women. See Table 1 for baseline characteristics. Five of 8 (62.5%) FDG scans had grade 2 visual uptake in the large vessels, consistent with active LVV. Tracer uptake could be detected semi-quantitatively in the medium-large arteries in both FDG and DOTA scans. FDG scans demonstrated a significantly higher RA activity than DOTA (RA SUVmean 1.88 vs 0.36, $p<0.001$), while DOTA had a significantly higher liver uptake (liver SUVmean 7.54 vs 2.39, $p<0.001$). Vascular uptake (as measured by both SUVmax and TBRliver) was significantly higher in FDG than DOTA scans in every vascular territory ($p<=0.05$ for all comparisons), including index vessels (SUVmax 4.04 vs 1.91, $p=0.01$, TBRliver 1.73 vs 0.27, $p=0.0009$).
Conclusion: 68Ga-HA-DOTATATE uptake can be detected in the aorta and medium branch vessels, including temporal and coronary arteries, of patients with active GCA receiving glucocorticoids. The distribution of DOTA uptake differs from that of FDG, with significantly lower blood pool uptake and higher liver uptake. In this pilot study, the arterial uptake of DOTA was overall lower than that of FDG, suggesting this may not be an optimal substitute for the more established tracer.

TOUR8C

Prediction of Response to Therapies and Flares Based on Ultrasound Findings at Baseline in Psoriatic Arthritis: an Analysis on a Joint Level

Ummugulsum Gazel (University of Ottawa Faculty of Medicine, Rheumatology, Ottawa); Seyyid Acikgoz (University of Ottawa, Rheumatology, Ottawa); Ricardo Sabido-Sauri (University of Ottawa, Ottawa); Ozun Tsechelidis (University of Ottawa, ottawa); Sylvia Sangwa (University of Ottawa, ottawa); Elliot Hepworth (Division of Rheumatology, Department of Medicine, University of Ottawa, Ottawa ON, Ottawa); Sibel Aydin (Ottawa Hospital Research Institute, University of Ottawa, Ottawa)
Objectives: It is not clear how to interpret when the ultrasound and physical examination do not agree on a joint level. In this study, we aimed to investigate, within PsA patients who are about to start an advanced therapy: a) the likelihood of achieving good response; b) the risk of experiencing a flare, on a joint level, 3 months post-therapy, based on the baseline ultrasound inflammation.
Methods: At the ORCHESTRA (Ottawa Rheumatology CompreHEnSive TReatment and Assessment) Clinic, PsA patients starting a new advanced therapy are assessed using a protocolized US scan. Patients are evaluated in the same clinic three months after therapy initiation. For this analysis, all MCP, PIP, DIP, and wrist joints were included; and analysis was performed on a joint level. US findings at baseline were scored for Grey-scale synovitis, Doppler signals and Global OMERACT-EULAR Synovitis Score (GLOESS) on scale of 0-3; grade ≥ 2 being considered positive. Physical exam was conducted at baseline and at 3-months, documenting the tenderness and/or swelling. For prediction of response, joints that were tender and/or swollen at baseline were included in the analysis. For prediction of flares, joints that were not tender and/or swollen at baseline were analyzed. Odds ratios were calculated to determine response and flare rates.

Results: The analysis included 36 PsA patients (61.1% female) with a mean(SD) age of 50 (12.7). At baseline, 17 patients (47.2%) were bionaïve, while 19 (52.8%) patients had failed one or more biologic therapies. A total of 1079 joints were analyzed, 333 (30%) being tender, 151 (14%) being swollen, and 365 (34%) being tender and/or swollen at baseline. Symptomatic joints with high inflammation on US at baseline had a lower response rate (less likely to become asymptomatic) at follow-up) than those without (OR range 0.30-0.66) (Table). Asymptomatic joints at baseline were more likely to flare (become symptomatic at follow-up) if their baseline US inflammation was higher (OR range 1.06-4.73). For both outcomes, the swelling had higher associations that the tenderness of the joint (Table).
Conclusion: Our study shows two critical implications of positive baseline US findings on a joint level: a) Tender and/or swollen joints with positive US findings are more resistant to therapies compared to those without, and b) Non-tender and/or non-swollen joints with positive US findings have a higher risk of developing symptoms. Although the integration of US findings into treatment algorithms is uncertain, they indicate the presence of a distinct patient phenotype.
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Table: Response and flare rates based on tender and/or swollen joints at baseline, according the initial US-positivity.

	Tender at baseline (n=333)		Swollen at baseline (n=151)		Tender and/or Swollen at baseline (n=365)		Not Tender at baseline (n=746)		Not Swollen at baseline (n=928)		Not Tender or Swollen at baseline (n=714)	
US features	**Good Response Rate (non tender at follow-up)**	**OR (95% CI)**	**Good Response Rate (non swollen at follow-up)**	**OR (95% CI)**	**Good Response Rate (non tender and/or swollen at follow-up)**	**OR (95% CI)**	**Flare Rate (tender at follow-up)**	**OR (95% CI)**	**Flare Rate (swollen at follow-up)**	**OR (95% CI)**	**Flare Rate (tender and/or swollen at follow-up)**	**OR (95% CI)**
Doppler<2	0.521	0.66 (0.35-1.27)	0.700	**0.37 (0.18-0.77)****	0.503	0.57 (0.31-1.07)	0.098	1.06 (0.31-3.61)	0.066	**2.73 (1.01-7.37)***	0.117	1.59 (0.53-4.78)
Doppler≥2	0.419		0.463		0.367		0.103		0.161		0.174	
GSS <2	0.559	**0.52 (0.32-0.83)****	0.776	**0.32 (0.15-0.65)****	0.546	**0.48 (0.32-0.75)****	0.090	**1.98 (1.05-3.72)***	0.051	**4.73 (2.70-8.30)*****	0.108	**2.37 (1.27-4.44)****
GSS ≥2	0.396		0.524		0.368		0.163		0.204		0.224	
GLOESS score<2	0.567	**0.48 (0.30-0.76)****	0.785	**0.30 (0.15-0.62)****	0.553	**0.45 (0.29-0.71)*****	0.090	1.76 (0.94-3.29)	0.052	**4.24 (2.43-7.41)*****	0.110	**2.03 (1.10-3.77)***
GLOESS score≥2	0.385		0.523		0.359		0.149		0.188		0.200	

GSS: Grey scale synovitis. GLOESS: Global OMERACT-EULAR Synovitis Score.
***p=<0.050, ****p=<0.010, ***** p=<0.001**

TOUR8D
Validation of Handheld Ultrasound Devices for Point of Care Use in Rheumatology: Interim Analysis for Enthesitis
Seyyid Acikgoz (University of Ottawa, Rheumatology, Ottawa); Ummugulsum Gazel (University of Ottawa Faculty of Medicine, Rheumatology, Ottawa); Suharsh Shah (Novartis, N/A); Marie Marie Maguin (Ottawa); Rohan Machhar (Toronto Western Hospital, Toronto); Lihi Eder (Women's College Research Institute and Division of Rheumatology, University of Toronto, Toronto); Gurjit Kaeley (University of Florida, Jacksonville); Sibel Aydin (Ottawa Hospital Research Institute, University of Ottawa, Ottawa)
Objectives: Ultrasonography (US) has experienced a rapid evolution in rheumatology. Despite many advantages being repeatedly shown, several barriers persist, equipment cost being an important one. Hand-held US technology promises to substantially lower this cost. However, before it can be used explicitly for rheumatology, its performance must be validated against

gold-standard devices. We aim to test the concurrent validity of a handheld US device versus a gold-standard device to detect characteristic features of enthesitis.

Methods: PsA patients with at least one tender and swollen joint were included. Each patient had consecutive US examinations using a handheld (Clarius Mobile Health Inc, HD3 L15 scanner) and a gold-standard US device (GE LogicE9) for detecting elementary lesions of enthesitis. Supraspinatus, triceps, common extensor tendon, quadriceps, patellar ligament, Achilles tendon and plantar fascia entheses were evaluated. B-mode and power Doppler images were saved for each site and lesion. Every image was given a unique identifier number after collection for blinding purpose. Image reading was performed at least 2 weeks after the acquisition of US in all patients, using a random order slide show, irrespective of the machine, anatomical site or patient, to ensure blindness. The inflammation score was obtained by summing hypoechogenicity, thickening, and Doppler scores, and the chronicity score was obtained by summing erosion, enthesophyte, and calcification scores. Cohen's kappa coefficient was calculated to test the agreement for elementary lesions of enthesitis, in addition to intraclass correlation analysis. Here we present interim analysis for the first 10 patients to detect enthesitis in 160 entheses.

Results: On the day of the US, 8 (80%) patients had at least one tender enthesis on physical examination. The median (IQR) SPARCC enthesitis score was 3(0.75-5.0). The agreement was substantial (see table) for enthesophytes and erosions; moderate for thickening, Doppler and hypoechogenicity; and slight for calcification (table). A very strong agreement was detected between the devices both for the inflammation and chronicity scores (ICC (intraclass correlation) and p values: inflammation: 0.957 and <0.001; chronicity: 0.934 and <0.001).

Conclusion: In this interim analysis, the handheld US device with L15 scanner showed moderate-substantial agreement to detect elementary lesions of enthesitis, with the exception of calcifications. In addition, a very strong consistency was found between the devices in inflammation, chronicity and total enthesitis scores, which enable physicians to evaluate enthesitis as a whole. These interim results encourage testing the use of handheld US devices for wider use.