Conclusions: ACPA +at risk individuals have features on imaging which assists prediction of development to IA. MRI TSV provides additional predictive ability over and above the clinical and US variables.

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Disclosure of Interest: None declared

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OP0043

PREDICTORS OF DRUG-FREE REMISSION IN RHEUMATOID ARTHRITIS: RESULTS FROM THE PROSPECTIVE BIOMARKERS OF REMISSION IN RHEUMATOID ARTHRITIS(BIORRA) STUDY

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Background: Remission is now a realistic and achievable target for many patients with rheumatoid arthritis (RA) using disease-modifying anti-rheumatic drugs (DMARDs) prescribed in modern treat-to-target regimens. However, DMARDs carry risks of potentially serious adverse effects, and require regular and expensive blood monitoring. Recent studies suggest that half of patients with RA in remission can discontinue DMARDs without a flare of arthritis activity, though this cannot currently be reliably predicted.

Abstract OP0043 – Table 1

Demographic	Value
Satisfied 2010 ACR/EULAR classification criteria: n (%)	44 (100%)
Age: median (IQR) [range]	66.5 (54.5–71.3) [35 – 82]
Female: n (%)	23 (52%)
Years since RA diagnosis: median (IQR) [range]	5.5 (3–11) [1–40]
Seropositive (rheumatoid factor and/or anti-citrullinated peptide antibody): n (%)	32 (73%)
Current methotrexate use: n (%)	38 (86%)
Baseline DAS28-CRP: median (IQR) [range]	1.07 (0.99–1.63) [0.96–2.34]
ACR/EULAR Boolean remission: n (%)	29 (66%)
Presence of joint erosion on baseline 7-joint ultrasound scan: n (%)	29 (70%)

Objectives: To identify baseline biomarkers that can predict sustained drug-free remission (DFR) versus arthritis flare following DMARD cessation.

Methods: Patients with established RA satisfying clinical (disease activity in 28 joints with CRP [DAS28-CRP] < 2.4) and ultrasound (absence of power Doppler synovitis on a blinded 7-joint scan) remission criteria discontinued conventional synthetic DMARDs (methotrexate, sulfasalazine, and/or hydroxychloroquine) and

were monitored for 6 months. The primary outcome was time-to-flare, defined as DAS28-CRP ≥ 2.4. Baseline clinical and ultrasound parameters (synovial/tenosynovial greyscale and joint erosions), circulating levels of 26 cytokines/chemokines, and gene expression by peripheral CD4 + T cells (RNA sequencing) were assessed for their ability to predict time-to-flare and flare/DFR status by multivariate Cox regression and receiver-operating characteristic (ROC) analysis.

Results: 44 patients were eligible for DMARD cessation (table 1); 23 (52%) experienced an arthritis flare at a median (IQR) of 48 (31.5–86.5) days. A composite score incorporating five variables (three genes [currently subject to patent application], one cytokine [IL-27], and one clinical [ACR/EULAR Boolean remission]) differentiated future flare and DFR with an area under the ROC curve of 0.96 (95% CI: 0.92 to 1.00), sensitivity 0.91 (0.78–1.00) and specificity 0.95 (0.84–1.00) (figure 1).

Conclusions: Our study provides proof-of-concept evidence for the existence of biomarkers of DFR in RA. If validated in an external cohort, these biomarkers may hold promise in guiding DMARD withdrawal, with consequent minimisation of medication adverse events and healthcare costs.

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OP0044

AFTER HOW LONG OF DISEASE DURATION DOES THE INCREASED MORTALITY RISK APPEAR IN RECENT ONSET RHEUMATOID ARTHRITIS?

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Background: For decades studies showed that rheumatoid arthritis (RA) patients died earlier than their general population counterparts. Some inception cohorts have failed to detect an increased mortality risk, possibly due to limited followup or to improvement in mortality risk in cohorts of more recent onset.

Objectives: To evaluate the risk of all-cause mortality in incident RA patients and to estimate when the increased risk appears amongst two Canadian inception cohorts with RA onset after 2000

Methods: Using a common protocol, we conducted 2 independent population-based cohort studies, using administrative health data, of incident RA patients in British Columbia (BC) and Ontario (ON), Canada over 2000 to 2015. In each province, we identified all RA patients (using validated criteria) diagnosed between 2000 and 2005 (to allow a minimum of 10 years of follow-up), and identified non-RA general population comparators for each RA patient, matched 1:2 on age, sex and index years. All patients were followed from index date until death (ascertained from vital statistics), out-migration, or end of study period (2015). Adjusted hazard ratios (HRs) and 95% CIs were estimated using multivariable Cox regression, controlling for baseline comorbidities, healthcare use, and socio-demographic factors. To estimate when the increased mortality risk appeared in incident RA patients, and to assess the proportional hazards assumption, we included an interaction between RA diagnosis and follow-up time in the multivariable model, to detect if and how the HR varied according to RA duration. Quadratic and logyear interactions were tested in the multivariable Cox models to assess linearity of the interaction.

Results: Among 13 834 incident RA patients in BC (27 668 matched comparators), 66% were female with a mean (SD) age of 58¹⁶ years at cohort entry. Among 27 405 incident RA patients in ON (54 810 matched comparators), 70% were female with a mean (SD) age of 56¹⁶ years. The prevalence of individual comorbidities at baseline was comparable across RA cohorts. During follow-up, 3139 (23%) of BC RA patients and 6270 (23%) ON RA patients died, with corresponding crude mortality rates of 2.3 deaths per 100 person-years in both provinces. Crude mortality rate ratios for BC and ON were 1.11 (95% CI: 1.07 to 1.16) and 1.27 (95% CI: 1.23 to 1.31), respectively. Multivariable analyses detected an increased risk of all-cause mortality in incident RA (relative to non-RA) patients by 6 years of follow-up, with a linear relationship suggesting further increase over time (figure 1). By 10 years of followup, the adjusted HR for RA was 1.14 (95% CI: 1.07 to 1.22) in BC and 1.13 (95% CI: 1.08 to 1.18) in ON.