5-year period between June 2013 and May 2018. Episodes of a SI were defined as infection requiring admission or use of intravenous antibiotics. For each bDMARD the rate of SI per 100 patient years (PYs) was calculated and patient demographics and comorbidities were analysed. Between group differences were assessed using independent samples t-tests or ANOVA. Where assumptions were violated, Mann-Whitney U tests or Kruskal-Wallis tests were used. For categorical variables, chi-square tests were used, except when assumptions were violated when Fisher's Exact tests were used.

Results: 296 patients received bDMARDs with an overall SI rate of 11.7/100PYs. There was no significant difference in presence of SI by disease type with 24% of patients with rheumatoid arthritis versus 19% with psoriatic arthritis, 14% with ankylosing spondylitis and 29% with "other" ($X^2=3.11$; $df=3$; $p=0.37$). Respiratory tract infections were the most common infection (46%) followed by skin and soft tissue infections (23%). The highest incidence rate of SI occurred with rituximab (29.72 SI/100PYs) followed by certolizumab (22.50 SI/100PYs) and tocilizumab (15.00 SI/100PYs). Duration of time on a bDMARD, disease duration and use of methotrexate or leflunomide were not shown to significantly increase the risk of SI for the entire cohort. The characteristics which were shown to significantly increase SI rates were; prednisone use, increasing age, chronic pulmonary comorbidity and specifically in those with rheumatoid arthritis male gender and total duration of bDMARD use.

Conclusion: In this real-world NQ cohort of patients treated with a bDMARD for a rheumatic disease, we have identified a number of factors potentially contributing to the risk of the development of SIs. This study provides valuable data on SI rates in an Australian 'real-world' cohort that may assist clinicians' choice of bDMARD in patients with a high baseline risk of infection and highlights the importance of minimising prednisone use in patients on bDMARDs.

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Disclosure of Interests: Kate Celkys: None declared, Jason Ly: None declared, Muriel Soden Speakers bureau: Speaker Fees from Pfizer in 2016

DOI: 10.1136/annrheumdis-2020-eular.1137

SAT0582 RISK OF INTERSTITIAL LUNG DISEASE IN SYSTEMIC AUTOIMMUNE RHEUMATIC DISEASES: A NATIONWIDE, POPULATION-BASED COHORT STUDY

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Background: To date, very few studies had investigated the epidemiology of interstitial lung disease (ILD) among patients with systemic autoimmune rheumatic disease (SARD).

Objectives: To study the risk of interstitial lung disease (ILD) among patients with various systemic autoimmune rheumatic diseases (SARDs) including rheumatoid arthritis (RA), dermatomyositis (DMtis), polymyositis (PM), systemic sclerosis (SSc), systemic lupus erythematosus (SLE), and primary Sjögren's syndrome (pSS).

Methods: Using 1997–2013 claims data from the Taiwanese National Health Insurance Research Database, we identified 63,277 newly diagnosed patients with various SARDs after excluding those with overlapping SARD diagnoses from 2001–2013 and randomly selected 253,108 non-SARD subjects matching (1:4) SARD patients for SARD diagnosis, age, sex and the year of the index date. We calculated the incidence rates (IRs) of ILD (ICD-9 code 515) in various SARD groups and the corresponding non-SARD comparison groups and estimated the IR ratios (IRRs) with 95% confidence intervals (CI) of ILD development. Using multivariable cox regression analyses, we estimated hazard ratios (HRs) with 95% CIs of ILD in various SARD groups compared with their comparison groups after adjusting for age, sex, Charlson comorbidity index, amiodarone use and methotrexate use. Sensitivity analyses were conducted by using a narrow definition of ILD.

Results: As shown in Table 1, the IRs of ILD were greatest in SSc patients (2,523 per 10⁵ years), followed by patients with DMtis (2,463 per 10⁵ years), PM (1,956 per 10⁵ years), SS (601 per 10⁵ years), RA (279 per 10⁵ years), and SLE (276 per 10⁵ years). Multivariable analyses showed that the risks of ILD were significantly increased in patients with SSc (HR, 66.01; 95% CI, 32.73–133.13), DMtis (128.74, 95% CI, 40.19–412.47), PM (HR, 30.39; 95% CI, 11.24–82.15), pSS (HR, 8.76; 95% CI, 7.03–10.90), RA (HR, 4.22; 95% CI, 3.51–5.08), and SLE (HR, 13.98; 95% CI, 9.25–21.14).

Table 1. Comparison of incidence rates of interstitial lung disease among patients with SARD

SARD	Total	Event (%)	Person-years	Incidence rate (/10 ⁵ years)	IRR (95% CI)
SARD					
Control	253,108	781 (0.3)	1,601,941	49	Reference
SARD	63,277	1,660 (2.6)	380,783	436	8.9 (8.2–9.7)
DMtis					
Control	3,716	10 (0.3)	25,508	39	Reference
DMtis	929	116 (12.5)	4,710	2,463	62.8 (32.9–119.9)
PM					
Control	2,828	9 (0.3)	18,647	48	Reference
PM	707	72 (10.2)	3,681	1,956	40.5 (20.3–81.0)
SSc					
Control	6,224	16 (0.3)	40,789	39	Reference
SSc	1,556	205 (12.2)	8,124	2,523	64.3 (38.7–107.0)
SLE					
Control	52,236	81 (0.2)	366,437	22	Reference
SLE	13,059	230 (1.8)	83,346	276	12.5 (9.7–16.1)
pSS					
Control	60,684	191 (0.3)	326,589	58	Reference
pSS	15,171	471 (3.1)	78,406	601	10.3 (8.7–12.2)
RA					
Control	127,420	474 (0.4)	823,970	58	Reference
RA	31,855	566 (1.8)	202,516	279	4.9 (4.3–5.5)

Conclusion: This nationwide, population-based, matched cohort study demonstrated that the risks of ILD were significantly increased in patients with SARDs.

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Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.205

SAT0583 DIPEPTIDYL PEPTIDASE-4 AND RISK OF PSORIASIS IN PATIENTS WITH TYPE 2 DIABETES

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Background: The risk of psoriasis in diabetic patients has rarely been explored. **Objectives:** This study aimed to investigate the association between dipeptidyl peptidase-4 (DPP4) inhibitors and the risk of psoriasis in type 2 diabetes mellitus (T2DM) patients.

Methods: We conducted a population-based propensity score-matched cohort study on the basis of Taiwan's National Health Insurance Research Database that included initiators of combination therapy with DPP4i (DPP4i plus metformin) and sulfonylurea (sulfonylurea plus metformin). Psoriasis (PSO) was identified with ≥ 2 diagnoses. Diabetes complications severity index (DCSI) was calculated. A total of 22721 DPP4 initiator and 227684 sulfonylurea initiator were identified. A 1:10 matched-pair cohort based on propensity score (PS) was created. PS-stratified Cox proportional hazards models compared the risk of PSO in DPP4i versus sulfonylurea initiator within 2 years, controlling for potential confounders.

Results: After propensity score matching, 9962 patients with T2DM starting DPP4i combination therapy and 39848 starting sulfonylurea combination therapy were selected. The incidence rate of PSO was lower in DPP4i group (188/100000 person-years) than in sulfonylurea group (467/100000 person-years). Risks of

incident psoriasis were significantly lower in the DPP4i group versus sulfonylurea with the PS-stratified HR of 0.422 (95% CI 0.273 to 0.716).

Conclusion: DPP4i plus metformin was associated with a reduced risk of psoriasis than sulfonylurea plus metformin. These findings merit further investigation.

Cumulative Incidence Curve of M+DPP4 Group and M+S Group

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.4538

SAT0584

SPECIFIC ACPA REACTIVITIES AND INFLAMMATORY BIOMARKERS ALONG WITH ULTRASOUND TENOSYNOVITIS ARE ASSOCIATED WITH ARTHRITIS ONSET IN A POPULATION AT RISK FOR RHEUMATOID ARTHRITIS

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Background: Anti-citrullinated protein antibodies (ACPA) are characteristic markers for rheumatoid arthritis (RA), developing years before disease onset. Early clinical and biological biomarkers could provide useful information on the onset of RA in predisposed individuals.

Objectives: The aim of the study was to investigate whether ACPA along with inflammatory markers and musculoskeletal ultrasound changes could predict arthritis development in individuals at risk for RA.

Methods: ACPA-positive individuals with musculoskeletal complaints were referred from primary care to a rheumatology clinic, recruited in the Risk-RA research program and followed-up for up to 3 years, between April 2014 and October 2019. All individuals lacked arthritis both at clinical examination by a trained rheumatologist and ultrasound assessment of hands and feet and any other symptomatic joints (according to EULAR-OMERACT definition). Blood samples were collected at inclusion and were analyzed for 15 ACPA fine specificities (by custom made peptide array), 92 inflammation-associated protein biomarkers (by multiplex immunoassay with Olink extension technology) and HLA-SE (DR low resolution kit). Statistical analysis used univariate and multivariate models with backwards selection and cox regression.

Results: 268 individuals with a median age of 48 (36-58) were recruited, out of which 212 (79%) were females. 75 (28%) developed arthritis within 11 months of follow-up while the median follow-up for those not developing arthritis was 21 months (14-28). Increased ACPA levels, shorter symptom duration and RF positivity were the main differences between individuals developing arthritis and those who did not. In univariate models, the presence of HLA-SE, specific ACPA reactivities, certain inflammatory markers and ultrasound-detected tenosynovitis were associated with arthritis development. In multivariate analysis the presence of anti-cit-fragilin (HR 2.1 (95% CI 1.2-3.7, p 0.01), IL6 levels (HR 1.4 (95% CI 1.2-1.7, p 0.0001) and tenosynovitis (HR 2.9 (95% CI 1.7-5.0, p 0.0001) remained significant predictors for arthritis onset.

Conclusion: Certain ACPA reactivities together with inflammatory markers and ultrasound-detected tenosynovitis predict arthritis development in predisposed individuals for developing RA.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.5578

SAT0585

GEO-EPIDEMOLOGY OF AUTOANTIBODIES IN RA: DIFFERENT PREVALENCES IN FOUR ETHNICALLY DIVERSE RA POPULATIONS

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Background: Rheumatoid arthritis (RA) has been described in virtually every ethnic population. Most RA patients harbor anti-modified protein antibodies (AMPAs), including anti-citrullinated protein (ACPA), anti-carbamylated protein (anti-CarP), anti-malondialdehyde acetaldehyde (anti-MAA), and anti-acetylated protein antibodies (AAPA). However, it is unclear whether differences exist in the AMPA response between different ethnic groups. Such differences could provide new clues to genetic and environmental factors contributing to autoantibody development.

Objectives: To investigate the prevalence of different AMPA in four ethnically diverse RA populations, and their association with smoking.

Methods: Enzyme-linked immunosorbent assays were used to measure anti-CarP IgG, anti-MAA IgG (both in-house), and anti-acetylated vimentin IgG (Orgentec) in ACPA-positive sera of Dutch (NL, n=103), Japanese (JP, n=174), Canadian First Nations People (FN, n=100), and black South Africans (SA, n=67) fulfilling the 1987 ACR classification criteria for RA. Ethnicity-matched local healthy controls were used to calculate cohort-specific cut-offs. Logistic regression was used to identify whether ever-smoking was associated with AMPA seropositivity in each cohort, corrected for age, gender, and disease duration. Random-effects meta-analysis was used to pool the resulting odds ratios (OR).

Results: For all three AMPAs, median levels were higher in FN and especially SA than NL and JP patients (Figure 1). The median autoantibody levels in arbitrary units (in % of patients positive) for NL, JP, FN and SA RA patients were: anti-CarP IgG: 1157 (47%), 994 (43%), 1642 (58%) and 2336 (76%) (p<0.001); anti-MAA IgG: 131 (29%), 179 (22%), 251 (29%) and 257 (53%) (p<0.001); AAPA: 133 (20%), 136 (17%), 153 (38%) and 316 (28%) (p<0.001). Prevalence, meaning positivity, also differed significantly between cohorts for all AMPAs (p<0.001). There were also marked differences in total IgG levels in mean (SD) g/L: 13 (4) for NL, 17 (6) for JP, 18 (6) for FN, and 25 (8) for SA (p<0.001). When the autoantibody levels were normalized to total IgG, the differences in became less pronounced between cohorts (Figure 2). The median arbitrary units per g/L Total IgG for NL, JP, FN and SA RA patients were: anti-CarP IgG: 54, 25, 53, and 79; anti-MAA IgG: 6, 5, 8, and 9; and AAPA: 2, 2, 2, and 3, suggesting that autoantibody level differences may partly correspond to cohort-specific differences in total IgG, although the overall trend of higher levels in SA persisted. There was no association between smoking and anti-CarP or anti-MAA positivity, with pooled OR (95% CI) of 1.31 (0.79-2.18) and 0.85 (0.46-1.56), respectively. However, smoking was positively and consistently associated with AAPA positivity in each cohort: pooled OR (95% CI) of 2.01 (1.06-3.81).

Conclusion: In these ACPA-positive ethnically diverse RA populations, levels and prevalence of various AMPAs differ, suggesting that ethnic background and environment may influence the development of the autoantibody response in RA. Despite these differences, our results imply smoking as a consistent risk factor for AAPA across different ethnic backgrounds.

Figure 1: Levels in arbitrary units (AU) for three AMPAs and grams per liter (g/L) for Total IgG in the serum of four ethnically diverse RA populations. Patients clustered at the maximum were above the highest standard of the ELISA. Blue shading indicates the patient falling below the cohort-specific positivity cut-off; green shading is the normal range for Total IgG. Lines indicate median and interquartile range. P-values correspond to Chi-square tests on proportion of patients considered positive, indicated in percentages above graphs.