(HAQ) was used. Data were analyzed in R Statistic stratified by gender using Kruskal-Wallis and Wilcoxon tests.

Results: 354 women and 123 men with RA (n=477), 81 women and 69 men with PsA (n=150), 121 women and 191 men with SpA (n=312) were included. No significant differences in biometrics was seen between female and male patients at baseline in all diseases.

In RA patients overall DAS28 decreased from baseline (V1) to V2 and V3 (DAS28: V1: male: 4.38 [3.66, 5.11], female: 4.30 [3.68, 5.03], p(m/f) = 0.905; V2: male: 2.66 [1.73, 3.63], female: 3.10 [2.17, 3.98], p(m/f) = 0.015; V3: male: 2.25 [1.39, 3.36], female: 3.01 [1.87, 3.87], p(m/f) = 0.002). For TNF inhibitors (n=311), there was a significant difference between genders at V2 (Fig.1a). Patients receiving Rituximab (n=41) displayed a significantly higher DAS28 at baseline in females, which diminished in the follow up: V1: (p(m/f) = 0.002; V2: p=0.019; V3: p=0.13); response to tocilizumab (n=63) did not show any gender differences.

In PsA patients overall SASPA decreased from baseline (V1) to V2 and V3 (SASPA: V1: male: 4.00 [2.80, 5.20], female: 4.40 [2.80, 5.80], p(m/f) = 0.399; V2: male: 2.20 [1.20, 3.50], female: 3.40 [2.00, 5.00], p(m/f) = 0.071; V3: male: 1.80 [0.80, 2.70], female: 3.01 [2.35, 4.80], p(m/f) = 0.001). For TNF inhibitors (n=79), there was a significant difference between genders at V3 (Fig. 1a). For Apremilast (n=39), there was a significant difference between genders at V2 (Fig.1c).

In SpA patients overall BASDAI decreased from baseline (V1) to V2 and V3 (BASDAI: V1: male: 4.70 [2.88, 6.18], female: 4.80 [3.30, 6.20], p(m/f) = 0.463; V2: male: 3.05 [2.00, 4.60], female: 3.64 [2.62, 5.41], p(m/f) = 0.039; V3: male: 3.02 [1.67, 4.20], female: 3.65 [2.18, 5.47], p(m/f) = 0.016). In V3 a differential BASDAI in response to TNFi was observed (Fig.1a).

Possible differences of response to individual TNFi (etanercept, infliximab, other TNFi) measured by HAQ were investigated in all diseases together. The difference between male and females was significant at baseline for all 3 TNFi; whereas with the use of ETA the significant difference was carried through to V2 and V3, it was lost with the use of IFX and was variable with the other TNFi (Fig.1b)

Figure 1a

Figure 1b

Figure 1c

Figure 1.

Conclusion: Female patients showed a statistically lower response to TNFi in all three disease entities (RA, SpA and PsA) to a variable degree in our homogeneous central european population. Interestingly, the difference was not uniform across individual TNFi when measured by HAQ. Gender differences were also seen in response to Apremilast.

Disclosure of Interests: Elisabeth Loibner: None declared, Valentin Ritschl: None declared, Burkhard Leeb Speakers bureau: AbbVie, Roche, MSD, Pfizer, Actiopharm, Boehringer-Ingelheim, Kwizda, Celgene, Sandoz, Grünenthal, Eli-Lilly, Grant/research support from: TRB, Roche, Consultancies: AbbVie, Amgen, Roche, MSD, Pfizer, Celgene, Grünenthal, Kwizda, Eli-Lilly, Novartis, Sandoz, Peter Spellitz: None declared, Gabriela Eichbauer-Sturm: None declared, Jochen Zwerina: None declared, Manfred Herold: None declared, Miriam Stetter: None declared, Rudolf Puchner Speakers bureau: AbbVie, BMS, Janssen, Kwizda, MSD, Pfizer, Celgene, Grünenthal, Eli-Lilly, Consultant of: AbbVie, Amgen, Pfizer, Celgene, Grünenthal, Eli-Lilly, Franz Singer: None declared, Ruth Fritsch-Stork: None declared

DOI: 10.1136/annrheumdis-2021-eular.3596

POS0209

ASSOCIATION OF FIRST, SECOND, AND THIRD-LINE bDMARDs AND tsDMARD WITH DRUG SURVIVAL AMONG RHEUMATOID ARTHRITIS PATIENTS: A COHORT STUDY

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Background: Since tofacitinib is a tsDMARD with a different mechanism of action compared to bDMARDs, along with the fact that recent releases of bDMARDs or tsDMARDs have led to numerous possible switching patterns, there is a need for comparative studies of bDMARDs and tofacitinib with subsequent drug survival among RA patients.

Objectives: To determine the association of first, second, and third-line biologic disease-modifying antirheumatic drugs (bDMARDs) and tofacitinib with drug survival among rheumatoid arthritis (RA) patients.

Methods: The study population was composed of 8,018 seropositive RA patients who were prescribed bDMARDs or tofacitinib between January 2014 and January 2019 from the Korean Health Insurance Review and Assessment Service database. First, second, and third-line choice of tumor necrosis factor inhibitors (TNFi) including etanercept, infliximab, adalimumab, and golimumab, as well as non-TNFi including tocilizumab, rituximab, tofacitinib, and abatacept were assessed. Multivariate Cox proportional hazards regression was used to determine the adjusted hazard ratios (aHRs) and 95% confidence intervals (CIs) for drug failure according to bDMARD or tofacitinib choice starting from the initial prescription date.

Results: Compared to first etanercept users, patients with first tocilizumab (aHR 0.56, 95% CI 0.46-0.68), tofacitinib (aHR 0.27, 95% CI 0.18-0.42), or abatacept (aHR 0.83, 95% CI 0.69-0.99) had lower risk of drug failure. Second choice of tocilizumab (aHR 0.38, 95% CI 0.25-0.55), tofacitinib (aHR 0.23, 95% CI 0.15-0.37), or abatacept (aHR 0.54, 95% CI 0.35-0.84) was associated with lower drug failure risk compared to second etanercept users. Finally, third choice of tocilizumab (aHR 0.32, 95% CI 0.16-0.62) or tofacitinib (aHR 0.35, 95% CI 0.19-0.63) was associated with lower drug failure risk compared to third TNFi users.

Conclusion: First and second-line tocilizumab, tofacitinib, or abatacept may lead to improved drug survival. Third choices of tocilizumab or tofacitinib may be beneficiary in reducing drug failure risk among RA patients.

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Table 1. Risk of drug failure on rheumatoid arthritis patients according to first, second, and third choice of bDMARD or tofacitinib.

	Events	Person-years	Incidence	aHR	95% CI
First-line choice (n=8,018)					
Etanercept	290	2,396	121	1.00	reference
Infliximab	236	1,417	167	1.36	1.14-1.62
Adalimumab	449	2,718	165	1.28	1.10-1.48
Golimumab	258	1,955	132	1.06	0.90-1.26
Tocilizumab	171	2,623	65	0.56	0.46-0.68
Rituximab	1	29	34	0.29	0.04-2.08
Tofacitinib	24	502	48	0.27	0.18-0.42
Abatacept	216	2,207	98	0.83	0.69-0.99
Second-line choice (n=1,645)					
Etanercept	41	151	272	1.00	reference
Infliximab	16	51	314	1.04	0.58-1.88
Adalimumab	85	275	309	1.07	0.72-1.60
Golimumab	41	204	201	0.79	0.51-1.23
Tocilizumab	85	918	93	0.38	0.25-0.55
Rituximab	4	31	129	0.52	0.18-1.46
Tofacitinib	35	484	72	0.23	0.15-0.37
Abatacept	46	299	154	0.54	0.35-0.84
Third-line choice (n=353)					
Etanercept	3	19	158	1.00	reference
Infliximab	5	10	500	1.27	0.27-5.91
Adalimumab	5	17	294	0.99	0.22-4.38
Golimumab	13	22	591	2.23	0.60-8.25
Tocilizumab	17	127	134	0.47	0.13-1.68
Rituximab	3	22	136	0.60	0.12-3.09
Tofacitinib	23	162	142	0.50	0.14-1.77
Abatacept	17	59	288	1.11	0.31-3.96

Adjusted hazard ratios calculated by multivariate Cox proportional hazards regression after adjustments for age, sex, hospital type, number of csDMARDs, first bDMARDs or tofacitinib for second and third-line choice analysis, Charlson comorbidity index, and enrollment year. Incidence calculated as the number of events per 1,000 person-years. Acronyms: aHR, adjusted hazard ratio; CI, confidence interval.

Disclosure of Interests: Byeongzu Ghang: None declared, Seulggie Choi: None declared, Eun Young Lee Grant/research support from: This work was supported by grants from the Korea Health Technology R&D Project through the Korea Health Industry Development Institute, funded by the Ministry of Health and Welfare, Republic of Korea [HI14C1277] and Bristol-Myers Squibb [IM101-735]. DOI: 10.1136/annrheumdis-2021-eular.546

Rheumatoid Arthritis - comorbidity and clinical aspects - Part 1

POS0210

THE INCIDENCE AND RISK FACTORS OF RHEUMATOID ARTHRITIS-ASSOCIATED INTERSTITIAL LUNG DISEASE

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Background: Rheumatoid arthritis (RA) is a systemic inflammatory disease of the joints and other organs, including the lungs.¹ Interstitial lung disease (ILD) is a lung injury pattern associated with significant symptom burden and poor outcomes in RA.² Better understanding of its risk factors could help with disease prevention and treatment.

Objectives: Using a population-based cohort, we sought to ascertain the incidence and risk factors of RA-associated ILD (RA-ILD) in recent years.

Methods: The study included adult residents of Olmsted County, Minnesota with incident RA between 1999 and 2014 based on the 1987 ACR classification criteria.³ Study subjects were followed until death, migration, or 4/30/2019. ILD was defined by the presence of bilateral interstitial fibrotic changes (excluding biapical scarring) on chest computed tomography (CT). In the absence of chest CT imaging, a physician's diagnosis of ILD in conjunction with chest X-ray findings suggestive of ILD and a restrictive pattern on pulmonary function testing (defined as a total lung capacity less than the lower limit of normal) was considered diagnostic of ILD. Evaluated risk factors included age, sex, calendar year, smoking status, body mass index (BMI) and presence/absence of rheumatoid

factor (RF) and anti-citrullinated protein antibodies (ACPA). Cumulative incidence of ILD was adjusted for the competing risk of death. Cox models were used to assess the association between potential risk factors and the development of RA-ILD.

Results: In Olmsted County, 645 residents were diagnosed with RA between 1999 and 2014. Seventy percent of patients were females, and 30% were males; median age at RA diagnosis was 55.3 [IQR 44.1-66.6] years, and most patients (89%) were white. Fifty-three percent of patients were never-smokers, and 64% had seropositive RA. Forty percent were obese (i.e., BMI ≥ 30 kg/m²); median BMI was 28.3 [IQR 24.3-33.0] kg/m².

In the cohort, ILD was identified in 73 patients. The ILD diagnosis predated RA diagnosis in 22 patients (3.4%) who were excluded from subsequent analyses. Final analyses included the remaining 623 patients with no ILD preceding, or at the time of RA diagnosis. Over a median follow-up interval of 10.2 [IQR 6.5-14.3] years, 51 patients developed ILD. Cumulative incidence of ILD, adjusted for the competing risk of death, was 4.3% at 5 years; 7.8% at 10 years; 9.4% at 15 years; and 12.3% at 20 years after RA diagnosis (Figure 1).

Age, and history of smoking at RA diagnosis correlated with the incidence of ILD; adjusted hazard ratios (HRs) were 1.89 per 10-year increase in age (95% confidence interval 1.52-2.34) and 1.94 (95% confidence interval 1.10-3.42), respectively. On the other hand, sex (HR: 1.21; 95% CI: 0.68-2.17), BMI (HR: 0.99; 95% CI: 0.95-1.04), obesity (HR: 0.89; 95% CI: 0.50-1.58), and seropositivity (HR: 1.15; 95% CI: 0.65-2.03) did not demonstrate significant associations with ILD.

Conclusion: This study provides a contemporary estimate of the occurrence of ILD in a well-characterized population-based cohort of patients with RA. Our findings of a lack of association between sex, obesity and seropositivity with ILD may indicate a change in established risk factors for ILD and warrant further investigation.

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Figure 1. Cumulative incidence of ILD in patients diagnosed with RA between 1999 and 2014, adjusted for the competing risk of death. **Abbreviations.** ILD: interstitial lung disease; RA: rheumatoid arthritis.

Disclosure of Interests: Bilal Samhour: None declared, Robert Vassallo Grant/research support from: Research grants from Pfizer, Sun Pharmaceuticals and Bristol Myers Squibb, Sara Achenbach: None declared, Vanessa Kronzer: None declared, John M Davis III Grant/research support from: Research grant from Pfizer., Elena Myasoedova: None declared, Cynthia S. Crowson: None declared DOI: 10.1136/annrheumdis-2021-eular.2183