Lines represent changes of mean SF36-MCS and mean SF36-PCS for EA and IBP; 95% confidence intervals were not represented because they were very small; The number of available data is between 696 and 508 according to the timepoint for SF-36 in EA and 646 to 422 according to the timepoint for SF-36 in IBP.

Conclusions: HRQoL was altered similarly for both physical and mental aspects in EA and early IBP. More than half the patients reached levels of HRQoL close to the general population. Disease activity only partly explained HRQoL: the drivers of HRQoL should be further explored.

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THU0687 THE IMPACT OF URATE-LOWERING THERAPY ON KIDNEY FUNCTION (IMPULSKF): PRELIMINARY RESULTS

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Background: The extended 2016 EULAR updated report states that for patients on ULT, SUA level should be monitored and maintained to <6 mg/dL (360 µmol/L). SUA level <3 mg/dL (180 µmol/L) is not recommended in the long term. And among EULAR proposals for future research is mentioned the optimal duration for prophylaxis of acute attacks when starting ULT, long-term impact of very low urate levels on the central nervous system, impact of ULT on kidney function.

Objectives: This project aims to investigate the impact of two target levels urate-lowering therapy (ULT) caused by hyperuricemia (HU) on kidney function and CKD progression measured by eGFR and albuminuria (A).

This trial had been formulated by 2016 updated EULAR evidence-based recommendations for the management of gout as a perspective proposal task for future research.

The key points are: 1) an unknown target HU level and 2) the time duration of ULT resulting in preserved kidney function in two subject groups a) with gout and b) without gout but highly elevated uricemia and CKD.

Methods: The trial has POEM design with 36 months duration. IMPULSKF is a Clinical Randomised Prospective Controlled Open Multicenter trial that randomly (by chance) assigns 180 participants in parallel groups. These patients with high SUA level (>8 mg/dL; 480 µmol/L) were divided into 2 arms (90+90) 1) with gout (EULAR's criteria) and 2) without gout but with presence of CKD 1–4 and 2 target SUA levels in each group as 5 mg/dL (300 µmol/L) and ultralow SUA <3 mg/dL (180 µmol/L) achieved either with allopurinol or febuxostat. The data obtained will be compared with control group (45 with gout without CKD and 45 with CKD).

Results: Retrospective analysis and 6 mothes in trial results has been shown that treatment with febuxostat improves GFR and BP control in patients with asymptomatic HU in non-diabetic CKD 2–3.²

Febuxostat treatment led to the most beneficial decrease in the level of uric acid (–223±16 µmol/L, p<0.01 with control and p<0.05 with allopurinol group), increased GFR (+10±2 ml/min, p<0.01 with control and p<0.05 with allopurinol group), blood pressure decrease (–7±2/–3±1 mm hg, p<0.05 with control and p=0.93 with allopurinol group), albuminuria (–131±19 mg, p<0.01 with control and p<0.05 with allopurinol group). Potential benefits of febuxostat were better tolerability compared with allopurinol group with target levels of uricemia less 180 µmol/L in 32% of people.

Conclusions: Treatment with febuxostat (better than allopurinol) improves GFR and BP in patients with asymptomatic hyperuricemia in non-diabetic CKD 2–3. Ultralow SUA levels seems to be safe in majority of patients but its effect on CKD function is not yet clear.

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THU0688 DO CERTAIN DMARDS INCREASE RISK OF NEW-ONSET TYPE 2 DIABETES? EVALUATION OF PATIENTS' BASELINE CHARACTERISTICS

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Background: The risk of cardiovascular morbidities is higher in patients (pts) with RA and is exacerbated with type 2 diabetes (T2D).¹ Recent analysis showed that abatacept (ABA), a biologic (b)DMARD, has a lower incidence of T2D in pts with RA in clinical practice.²

Objectives: To compare baseline characteristics of pts with RA on DMARDs and evaluate incidence of T2D among these pts based on DMARD exposure.

Methods: Administrative claims data (2006–2016) from Optum Clinformatics (database A), QuintilesIMS PharMetrics Plus (database B) and Truven Market-Scan® (database C). Inclusion criteria were: 2 RA diagnosis codes+1 DMARD prescription; age ≥18 years; ≥3 months baseline (pre-index date); and 3 months of follow-up (post-index date). Mutually exclusive treatment groups (grp) were created based on the first prescription (index date) using a hierarchy of ABA, non-ABA bDMARDs (TNF inhibitors [TNFi] and non-TNFi [excluding ABA]) and conventional DMARDs (cDMARD; MTX or hydroxychloroquine [HCQ]). Also, an RA grp without DMARD use (NoDMARD) was identified. The index date for NoDMARD was first diagnosis date. Incident T2D was identified as those without T2D prior to index using one International Classification of Disease (ICD)–9 or ICD-10 diagnosis code for T2D. Assessment of T2D risk between treatment grps was based on regression and disease risk score (DRS) models. Adjusted incidence rate for T2D was based on a Cox model (stratified by DRS grps and categorised into 4 equal grps using quartile scores) with treatment as the independent variable.

Results: In databases A, B and C, 84,875, 2 07 811 and 1 94 819 pts with RA were identified, respectively (table 1). The combined proportions of pts in each treatment grp were: 3%–4% ABA; 12%–16% non-ABA bDMARD; and 41%–45% cDMARDs. Pts treated with ABA were older compared with those on non-ABA bDMARDs and a greater proportion had T2D risk factors of obesity, hypertension, dyslipidaemia and heart failure (table 1). The adjusted hazard ratios for T2D were significantly higher for non-bDMARD grps of TNFi and other bDMARDs vs ABA (figure 1).

Abstract THU0688 – Table 1. Baseline Risk Factors for T2D in Pts With RA by DMARD Grps

	ABA	TNFi	Non-TNFi	MTX only	HCQ	cDMARD – others	NoDMARD
Database A (N)	2825	9569	632	12 676	18 233	4368	36 572
Age≥55 years	52.5	38.8	60.3	63.3	53.4	62.6	61.3
Smoking	11.3	9.8	8.7	11.2	11.2	11.7	14.1
Obesity	9.7	7.1	6.2	7.4	7.9	8.2	10.0
Hypertension	39.9	30.5	36.2	44.1	40.7	46.7	54.8
Dyslipidaemia	35.9	28.4	29.1	41.2	39.9	43.9	52.2
Database B (N)	6497	30 329	1664	29 821	49 652	10 358	79 490
Age≥55 years	45.5	34.1	39.6	49.7	41.7	48.9	45.0
Smoking	9.4	8.4	8.0	9.3	8.9	9.3	11.7
Obesity	8.4	6.4	5.1	6.5	7.5	7.1	9.3
Hypertension	36.1	25.2	27.2	34.4	31.9	35.8	42.4
Dyslipidaemia	31.2	24.4	23.1	33.6	32.6	36.7	42.0
Database C (N)	8853	29 671	1714	30 669	47 850	10 891	65 171
Age≥55 years	50.7	36.0	43.3	54.9	46.1	54.5	53.1
Smoking	4.7	5.0	5.5	5.4	5.2	5.3	6.8
Obesity	5.4	4.6	5.0	4.8	5.2	4.8	6.4
Hypertension	37.7	26.2	29.2	36.1	33.5	37.8	43.8
Dyslipidaemia	28.9	23.4	23.1	31.1	30.2	33.7	38.4

All data are % unless indicated otherwise

Abstract THU0688 – Figure 1 Hazard. Ratios for Incidence of T2D in Pts With RA on Different DMARDs.

(HR > 1 favours abatacept, HR < 1 favours other therapies)

Conclusions: Pts with RA treated with abatacept had greater risk factors for T2D at baseline. However, the adjusted risk of new-onset T2D was lower among abatacept pts versus other bDMARDs.

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THU0689

VARIABILITY OF PAIN LEVELS IS EXPLAINED BY SELF-REPORTED DISEASE ACTIVITY IN RHEUMATOID ARTHRITIS AND AXIAL SPONDYLOARTHRITIS: A 3-MONTHS PROSPECTIVE STUDY OF 165 PATIENTS

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Background: Pain is a central characteristic of rheumatoid arthritis (RA) and axial spondyloarthritis (axSpA), but it is multifactorial. Some patients describe high fluctuation of pain intensity, but fluctuation of pain and its association with disease activity has been little investigated.

Objectives: To compare the variability of pain and of self-reported disease activity in patients with RA or axSpA through repeated assessments, and to describe clinical characteristics of patients with high variability of pain.

Methods: Data were extracted from a prospective cohort study (ActConnect) including patients with clinician-confirmed RA or axSpA owning a smartphone.¹ Baseline characteristics were collected. Over 3 months, weekly self-assessments of pain and patient global assessment of disease activity on 0–10 points numerical rating scales (NRS) were obtained. For each patient, the mean of the inter-assessment differences of NRS (average of absolute change – AAC) of pain and disease activity were calculated. RA and AxSpA patients were compared by t-test. High variability was defined as the upper tercile of AAC. Pearson's correlation was used to evaluate the correlation between variability of pain and of self-reported disease activity. Univariate and multivariate logistic regression compared patients with high vs low variability of pain, without imputation of missing data, on R, including weekly self-reported flare over follow-up.

Results: Eighty-six patients with RA and 79 with axSpA were included, mean age was respectively 48.7 (SD 12.7) and 41.7 (SD 10.2) years, mean disease duration was 8 (SD 8.8 and 8.6 respectively) years, 81% and 44% respectively were female. Mean DAS28 and BASDAI were respectively 2.27 (SD 1.18) and 3.21 (SD 2.06) at baseline; 44% and 62% respectively of patients were receiving a biologic.

Mean levels of pain and patient global assessment of disease activity were non-significantly higher in AxSpA than RA patients (table 1) and variability of pain and disease activity were also higher in AxSpA (table 1). Correlation between AAC of pain and activity was 72% in RA, and 84% in AxSpA. In multivariate analyses, self-reported flares were the only determinant of pain AAC (Odds ratio 2.25 [1.27–4.38] p=0.01 and 3.26 [1.67–6.35] p<0.001 respectively for RA and axSpA).

Abstract THU0689 – Table 1

	RA	AxSpA	p value
Baseline pain	2.90 (SD 2.36)	3.40 (SD 2.30)	0.16
AAC of pain	1.02 (SD 0.74)	1.20 (SD 0.70)	0.10
Baseline PGA of disease activity	3.09 (SD 2.27)	3.73 (SD 2.48)	0.08
AAC of PGA of disease activity	0.98 (SD 0.67)	1.22 (SD 0.70)	0.02

Conclusions: In this population of overall well-controlled patients, close repeated assessments of pain showed relatively low fluctuations of pain in RA and axSpA (around 1 point on a 0–10 scale). Patients with axSpA reported slightly more pain, self-reported disease activity and fluctuations of these outcomes, than patients with RA. Fluctuations in pain were highly correlated to fluctuations in patient global assessment of disease activity, indicating significant overall between these 2 patient-reported outcomes. Self-reported flares were the main element explaining fluctuations in pain, confirming the validity of self-reported flares.

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THU0690

RETROSPECTIVE ANALYSIS OF TEMPORAL ARTERY BIOPSY AND ITS IMPACT IN THE MANAGEMENT OF GIANT CELL ARTERITIS

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Background: Prevalence of Giant cell arteritis (GCA) as per primary care data is 17/10000 for patients >55 years of age¹. British society of rheumatology (BSR) guidelines recommend that suspected cases of GCA should be started on treatment with high dose corticosteroids immediately and then referred for temporal artery biopsy². Temporal artery biopsy has diagnostic yield around 13%³. We have done a retrospective analysis of patients who had temporal artery biopsy in order to assess the compliance with BSR guidelines and assess the diagnostic yield.

Objectives: To assess the yield of temporal artery biopsy and its impact in the management of Giant cell arteritis

Methods: The medical records of patients who had temporal artery biopsy from September 2015 to August 2017 were retrospectively reviewed. We documented demographics, management pre and post biopsy, and timeframes between referral, treatment and biopsy. Data was searched through the clinic letters, referral letters and pathology results.

Results: Total number of patients identified was 45. Among them 23 (51%) patients had typical clinical features of GCA and 21 (49%) had atypical features as per the referral letter. Age distribution found was as following: 50 years and below-3; 51 to 60 years- 7; 61 to 70 years- 15; 71 to 80 years-12; 81 to 90 years- 7 and above 90 years-1. Timeframe between biopsy referral and biopsy was as following: within 1 week-27; between 1–2 weeks- 10; between 2–3 weeks- 6 and more than 3 weeks –1. Ophthalmologists performed biopsy for 37 patient and vascular surgeons for 8 patients. Referral was made by primary care for 7 patients, acute medicine for 12 patients, neurology for 3 patients and by rheumatologist for 23 patients. Treatment was initiated in 37 (82%) patients prior to biopsy. Biopsy was positive in 11 (25%) patients, negative in 30 (66%) patients and 4 (8%) showed healed inflammatory changes (HIC). Age distribution of biopsy positive patients was as following - age 61 to 70 years - 4; age 71 to 80 years - 4; age 81 to 90 years - 3. Out of the 11 biopsy positive patients 7 were started on treatment prior to the biopsy and remaining 4 were initiated on treatment after the results. The distribution of duration of treatment before biopsy was as following - within 1 week - 2; between 1 to 2 weeks - 3; between 2 to 3 weeks - 2.

Conclusions: - Majority of the patients (80%) had temporal artery biopsy done within 2 weeks of referral

- Diagnostic yield of temporal artery biopsy was found nearly twice the existing data

- Biopsy was positive in the age group between 61 to 90 years
- Positive biopsy was seen up to 3 weeks of treatment with high dose corticosteroids
- Duration of treatment pre biopsy and age group appear to impact the diagnostic yield of temporal artery biopsy.

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THU0691

ASSOCIATION BETWEEN PERIODONTITIS AND CLINICAL RESPONSE IN RHEUMATOID ARTHRITIS PATIENTS UNDER BIOLOGICAL TREATMENT

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Background: Previous studies showed that periodontitis (PD) was a propagation factor for the severity of rheumatoid arthritis (RA) and our previous epidemiological study revealed that PD was associated with discontinuation risk of etanercept.

Objectives: To investigate the association between PD and the risk of 3 month clinical non-response using the Disease Activity Score (DAS)-based European League Against Rheumatism (EULAR) response criteria in RA patients under biological therapy.

Methods: We enrolled 111 RA patients treated with biologics, including etanercept (n=16), adalimumab (n=44), golimumab (n=7), tocilizumab (n=23), abatacept (n=14), and rituximab (n=7). A qualified periodontist performed the periodontal assessment, and the 3 month clinical response was determined DAS-based EULAR response criteria. We quantified the association between PD and the risk of non-response by calculating odds ratios (ORs) with 95% confidence intervals (CIs) using the logistic regression analysis, after adjusting for confounders including age, sex, tobacco use, RA disease duration, biologic treatment duration, rheumatoid factor and anti-citrullinated peptide antibody, erythrocyte sedimentation rate and C-reactive protein, concurrent medication, and diabetes.

Results: Of 111 RA patients, 83 (74.8%) had PD. 37 (44.6%) of PD patients received periodontal treatment within three months. After adjusting for potential confounders, PD patients had a higher risk of non-response to treatment than non-PD patients (OR, 4.20; 95% CI, 1.06–16.68; p=0.041). Compared with non-PD patients, the risk of non-response was significantly greater in PD patients who did not receive periodontal therapy (OR, 5.12; 95% CI, 1.16–22.56; p=0.031), but not in PD patients who received periodontal therapy (OR, 3.28; 95% CI, 0.72–15.06; p=0.126). Among those who were under tumour necrosis factor inhibitor therapy (n=67), the risk of clinical non-response was markedly higher in those with PD (OR, 9.65; 95% CI, 1.33–70.04; p=0.025), particularly in those who did not receive periodontal therapy (OR, 14.39; 95% CI, 1.59–130.38; p=0.018).

Significant predictor of 3 month non-response	Univariable analysis OR (95% CI)	Multivariable	
		Model A OR (95% CI)	Model B OR (95% CI)
Periodontitis			
No	Reference:	Reference:	Reference:
Yes	1.39 (0.56–3.45)	4.20 (1.06–16.68)	
Not treated	1.77 (0.66–4.74)		5.12 (1.16–22.56)
Treated	1.01 (0.36–2.90)		3.28 (0.72–15.06)
RA duration, incremental year	1.03 (1.00–1.06)	1.13 (1.04–1.24)	1.13 (1.03–1.23)
Baseline DAS28	1.00 (0.86–1.15)	1.64 (0.99–2.73)	1.69 (1.01–2.82)
Baseline ESR	1.06 (1.02–1.09)	1.06 (1.004–1.11)	1.06 (1.003–1.11)

Conclusions: In RA patients under biological therapy, an increased risk of clinical non-response to treatment was observed in patients with PD, especially among those who did not receive periodontal treatment.

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THU0692

RISK OF END-STAGE RENAL DISEASE REQUIRING DIALYSIS IN ANKYLOSING SPONDYLITIS PATIENTS STARTING MEDICAL THERAPY: A NATIONWIDE, POPULATION-BASED, COHORT STUDY

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Background: From the year 2000 Taiwan has had the highest incidence and prevalence of end-stage renal disease (ESRD) among the regions investigated by the US Renal Data System. Also, previous studies had suggested a possible association between IgA nephropathy and ankylosing spondylitis (AS) because an increased prevalence of microscopic hematuria and a higher proportion of elevated serum IgA levels were found in AS patients. However, whether the risk of ESRD was increased in treated AS patients or not is still unknown.

Objectives: To examine the risk of ESRD requiring dialysis in patients with AS who started medical therapy.

Methods: Using 2003–2012 claims data from the Taiwanese National Health Insurance Research Database, we identified 38,259 AS patients who received at least 3 courses of AS-related medical therapy (i.e., non-steroidal anti-inflammatory drugs, methotrexate, salazopyrine or corticosteroid) and started therapy from 2005 to 2012. The first date of medical therapy was defined as the index date. After excluding those who had a history of chronic renal disease (ICD9-CM 585, 586) or receiving dialysis before the index date, we identified 37 070 newly-treated AS cases. We randomly selected 3 70 700 non-AS individuals matching (1:10) AS cases for age, sex and the year of the index date without a history of chronic renal failure or dialysis before the index date. After adjusting for age, sex, moderate to severe renal disease, diabetes mellitus, hypertension, and annual cumulative defined daily dose (cDDDs) of traditional non-steroidal anti-inflammatory drugs (NSAIDs), selective cyclooxygenase-2 inhibitors (COX2i), and preferential COX2i, we calculated the adjusted hazard ratios (aHRs) with 95% confidence intervals (CIs) using the Cox proportional hazard model to quantify the risk of ESRD in AS patients compared with non-AS controls. We re-selected 6,621 AS patients and 6621 non-AS subjects by further matching (1:1) for cDDDs of three groups of NSAIDs to re-estimate the aHRs for ESRD.

Results: 51 (0.14%) of 37,070 AS patients and 1,17 (0.38%) of non-AS individuals developed ESRD after a follow-up of 1 58 846 and 1,707,757 person-years respectively. The aHR for ESRD was 0.4 (0.30–0.54) in AS patients compared with non-AS individuals. However, after further matching for cDDDs of NSAIDs, the aHR of ESRD was 0.80 (0.34–1.86). Significant risk factors included diabetes mellitus, hypertension, renal disease, and use of COX2i.

Conclusions: The risk of ESRD was not significantly different between treated AS patients and age, sex, index date, and NSAIDs use matched non-AS individuals.

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