(CV) risk factors. Tofacitinib is an oral JAK inhibitor for the treatment of RA. Treatment with tofacitinib may increase total cholesterol (TC), low-density lipoprotein-cholesterol (LDL-c) and high-density lipoprotein-cholesterol (HDL-c), without affecting TC/HDL-c ratio.

**Objectives:** To evaluate major adverse CV event (MACE) risk factors in tofacitinib-treated pts with RA in the clinical development programme.

**Methods:** Data were pooled from pts with moderately to severely active RA receiving ≥1 tofacitinib dose in 6 Phase 3 and 2 long-term extension (LTE) studies (1 LTE study ongoing, data cut-off: April 2015). MACE was any MI, stroke or CV death (coronary, cerebrovascular, cardiac). Cox regression models evaluated associations between baseline (BL) values and time (BL to first tofacitinib dose) to first MACE. Changes (BL to Week [wk] 24) in MACE predictors and time to future MACE (first occurrence after 24 wks) were evaluated after adjusting for age, BL values and time-varying tofacitinib dose. Hazard ratios (HR) and 95% confidence intervals (CI) were calculated.

**Results:** 52 MACE cases occurred over 12,873 pt-years (py) of exposure in 4076 pts (incidence rate: 0.4 pts with events/100 py). At BL, compared with pts without MACE, pts with MACE were older (mean age 60.2 vs 52.7 years) with a higher mean BMI (29.2 vs 27.0 kg/m$^2$) and longer mean RA disease duration (10.1 vs 7.7 years), and were more likely to have a history of diabetes (15.4% vs 7.6%) and hypertension (57.7% vs 33.7%). Pts with MACE had higher mean TC (208.2 vs 198.3 mg/dL), LDL-c (123.3 vs 114.0 mg/dL), TC/HDL-c ratio (4.0 vs 3.5) and triglycerides (152.1 vs 125.3 mg/dL) at BL, and lower HDL-c (55.3 vs 59.4 mg/dL) vs pts without MACE. In univariate analyses, traditional CV risk factors and corticosteroid and statin use at BL were associated with MACE risk (Table). BL disease activity and inflammation measures were not associated with MACE risk (Table). In multivariate analysis, BL age, hypertension and the TC/HDL-c ratio were significantly associated with MACE risk. Increases in HDL-c ($p<0.001$) and decreases in TC/HDL-c ratio ($p<0.05$) after 24 wks of tofacitinib therapy were significantly associated with decreased risk of future MACE (Figure). Increases in erythrocyte sedimentation rate (ESR; p=0.09) may be associated with increased future MACE risk. Changes in TC, LDL-c or other disease activity measures were not associated with future MACE risk.

**Conclusions:** In pooled analyses of tofacitinib-treated pts (age and BL value adjusted), increases in LDL-c and TC after 24 wks of tofacitinib therapy were not associated with future MACE risk. Increases in HDL-c and decreases in the TC/HDL-c ratio after 24 wks of tofacitinib therapy were associated with reduced future MACE risk. Increases in ESR after 24 wks may be associated with increased future MACE risk. More data are needed to confirm these findings.

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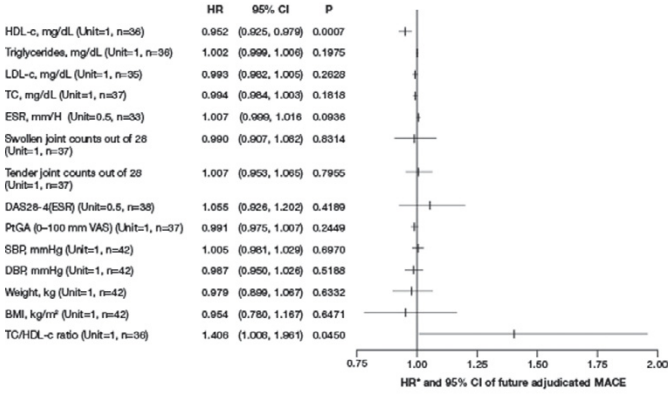
**Table. Association of baseline risk factors and development of MACE based on univariate Cox analysis**

| Baseline variable | HR | (95% CI) |
|---|---|---|
| Age (years, n=52) | 2.02*** | (1.53, 2.67) |
| BMI (kg/m$^2$, n=52) | 1.05** | (1.01, 1.09) |
| Baseline statin users (Y/N, n=52) | 2.77** | (1.45, 5.28) |
| Baseline corticosteroid users (Y/N, n=52) | 0.55* | (0.31, 0.98) |
| Duration of RA, (years, n=52) | 1.03* | (1.00, 1.06) |
| Triglycerides (mg/dL, n=51) | 1.00** | (1.00, 1.01) |
| TC/HDL-c ratio (n=51) | 1.42*** | (1.16, 1.74) |
| Apolipoprotein B (mg/dL, n=48) | 1.02** | (1.01, 1.03) |
| Apolipoprotein B/Apolipoprotein A-1 ratio (n=48) | 2.76** | (1.50, 5.09) |
| SBP (mmHg, n=51) | 1.02* | (1.01, 1.04) |
| DBP (mmHg, n=51) | 1.04* | (1.01, 1.07) |
| History of diabetes (Y/N, n=52) | 2.56* | (1.20, 5.43) |
| History of hypertension (Y/N, n=52) | 2.86*** | (1.65, 4.96) |

*p<0.05; **p<0.01; ***p<0.001
Other BL variables assessed as not being statistically significant were gender, weight, smoking status, DAS28-4(ESR), ESR, CRP, swollen joint count out of 28, tender joint count out of 28, baseline MTX users, baseline MTX dose, TC, HDL-c, LDL-c and history of cardiac disorders
n represents number of patients with MACE (uncensored) analysed for each variable
BL, baseline; BMI, body mass index; CI, confidence interval; CRP, C-reactive protein; DAS28-4, disease activity score in 28 joints; DBP, diastolic blood pressure; ESR, erythrocyte sedimentation rate; HDL-c, high-density lipoprotein-cholesterol; HR, hazard ratio; LDL-c, low-density lipoprotein-cholesterol; MACE, major adverse cardiovascular event; MTX, methotrexate; RA, rheumatoid arthritis; SBP, systolic blood pressure; TC, total cholesterol

**Figure. Age-adjusted and baseline parameter-adjusted association of change from baseline to Week 24 in lipid levels, inflammation parameters and RA disease activity measures with future MACE**




These data show results from a Cox regression model; age-adjusted and baseline parameter-adjusted association of changes from baseline to Week 24, for future MACE risk
*For each variable listed, a Cox regression model was fitted, with change in variable at Week 24, the variable at baseline, age at baseline and time varying dose as predictors
In this model, only patients with exposure after the tofacitinib-week 24 were considered (ie, patients who had MACE before tofacitinib-week 24 or who had withdrawn or completed the study by tofacitinib-week 24 were excluded). Additionally, patients with missing data for a tofacitinib-week 24 variable were excluded from the analysis of that variable (no imputation method). The HR corresponds to increased risk of MACE per 1-unit increase in the parameter
n is the number of patients with future MACE for each predictor
BMI, body mass index; CI, confidence interval; DAS28-4, disease activity score in 28 joints; DBP, diastolic blood pressure; ESR, erythrocyte sedimentation rate; HDL-c, high-density lipoprotein-cholesterol; HR, hazard ratio; LDL-c, low-density lipoprotein-cholesterol; MACE, major adverse cardiovascular event; PtGA, patient's global assessment; RA, rheumatoid arthritis; SBP, systolic blood pressure; TC, total cholesterol

## SAT0687 ADHERENCE OF RHEUMATIC PATIENTS TO INH PROPHYLAXIS PRESCRIBED BEFORE BIOLOGICAL TREATMENT: HUR-BIO SINGLE CENTER REAL LIFE RESULTS

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**Background:** Isoniazid (INH) prophylaxis is strongly recommended for the patients who have latent tuberculosis (TB) and who are going to be under anti-TNF treatment. INH is prescribed for 9 months and patient adherence to INH affects the risk of active TB development.

**Objectives:** In this study we aimed to assess the levels of patient adherence to INH prophylaxis.

**Methods:** Patients, who are under biological treatment and who have a quantiferon (QFT) test result, were evaluated with a questionnaire between August 2015-August 2016. Questionnaire included the demographic and clinical characteristics. Besides, patients were asked whether they had been prescribed INH. Patients, who were given INH prophylaxis, were asked to answer those questions: i) Did you take INH daily and regularly for 9 months? ii) If not what was the reason? The reasons are classified into three categories: 1) The patient discontinued INH of his/her own volition before 9 months. 2) Continued INH for 9 months but did not take regularly due to forgetfulness. 3) Treatment stopped by physicisan due to an adverse effect (elevation of liver enzymes, neuropathy, etc.)

**Results:** 1. 710 patients were recruited. INH was prescribed to 169 (23.8%) of 710. Demographic characteristics of INH-prescribed patients: 88 (52.1%) of 169 were female, mean age was 46.2 (SD:11.4), 82 (48.5%) of 169 at least graduated from a high school. Diagnosis were followed; RA 65 (38.4%), SpA 85 (50.3%), PsA 13 (7.7%), others 6 (3.6%). Totally 34 (20.1%) of 169 took INH irregularly. 19 (11.2%) of 169 patients discontinued INH of his/her own volition before 9 months. During follow-up 5 of 19 were prescribed INH again by the physician and they completed the 9-months duration. 9 (5.3%) of 169 patients did not take INH regularly due to forgetting. INH was stopped by a physician due to liver enzyme elevation in 6 (3.5%) of 169 patients. There was not a statistically difference in demographical and clinical characteristics between regular and irregular INH takers.

**Conclusions:** There is an inadherence to INH treatment approximately in one of five patients. Only 3.5% of INH-recommended patients had a medical reason of inadherence. Among other patients, causes of inadherence were discontinuance of own volition and forgetfulness or perfunctoriness. Physicians should be aware that reminding of INH is one of the question in every outpatient clinic visits. Other reminding methods such as regular calling should be considered in those of high risk population. Further studies were needed for reminding process.

**Disclosure of Interest:** None declared

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## SAT0688 JOINT INVOLVEMENT IN PATIENTS WITH KNEE AND HIP OA SCHEDULED FOR SURGERY: MULTI-JOINT OA, THE RULE NOT THE EXCEPTION?

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**Background:** Multijoint involvement in osteoarthritis (OA) has long been documented clinically and in the literature. Even so, the vast majority of OA research has focused on OA in individual joints, particularly the knees, hips or hands. In many "joint-specific" studies, the presence of multijoint symptoms are either ignored or peripherally considered in descriptive and analytical work. The implicit assumption is often that OA is OA, irrespective of whether a single joint or several joints are involved.

**Objectives:** To document the occurrence of multijoint symptoms in a clinical sample of individuals with knee and hip OA scheduled for orthopaedic surgery. To examine the joint sites involved and investigate whether the extent of joint involvement is related to demographic and health characteristics.

**Methods:** Patients scheduled for total knee or hip replacement for end-stage OA were consecutively recruited from an academic hospital in Toronto, Canada. A health questionnaire completed prior to surgery captured demographic

characteristics (age, sex), symptomatic joints other than the surgical joint (right and left shoulders, elbows, wrists, hands, hips, knees, feet, ankle, neck and back), body mass index (BMI), comorbidities (hypertension, depression, diabetes, migraine headaches, cancer, respiratory disease, heart disease, stomach/bowel disease, stroke) and WOMAC hip- and knee-specific pain and function.
**Results:** Study questionnaires were completed by 366 hip and 407 knee patients. The mean age of the sample was 65 years (SD=9.2; range 38–89 years), 57% were female. The most frequently reported symptomatic joints among knee patients were the contralateral knee (53.2%), one or both hands (32.1%), and the upper-, mid- or lower-back (31.0%), and among hip patients were one or both knees (49.4%), the back (36.6%), and the contralateral hip (21.3%). The overall mean number of symptomatic joints other than the surgical joint was 3.0 (SD=3.2; range 0–17). Only 19.0% reported the surgical joint as the only symptomatic joint; 23.0% reported 5 or more additional symptomatic joints. Mean hip/knee-specific pain and function scores were significantly worse with increasing symptomatic joint count ($p<0.01$). Additional symptomatic joints were significantly more frequent in women than men; mean count 3.6 vs. 2.3 ($p<0.01$). No significant difference in mean joint count (p=0.64) was observed by age. Similarly, no difference was found by BMI (i.e. overweight/obese vs. normal); p=0.24 for mean count. However, the number of co-occurring conditions increased with increasing joint count: 27.2% reported 2+ co-occurring conditions among those with 1–4 symptomatic joints, and 42.8% among those with 5+ symptomatic joints ($p<0.01$).
**Conclusions:** In this clinical OA sample, the "average" patient reported multiple symptomatic joints. Increasing age was not associated with increasing frequency of symptomatic joints. Irrespective of age and obesity, multiple symptomatic joints were the rule, not the exception. It was notable that the frequency of co-occurring conditions increased with increasing symptomatic joint count. This may suggest a need to re-examine how OA is characterized and perhaps its underlying etiology as it relates to single vs. multi-joint involvement.
**Disclosure of Interest:** None declared
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## SAT0689 PREVALENCE OF AND TEMPORAL TRENDS IN HYPERURICAEMIA AMONG ADULT PATIENTS WITH CHRONIC KIDNEY DISEASE IN IRELAND

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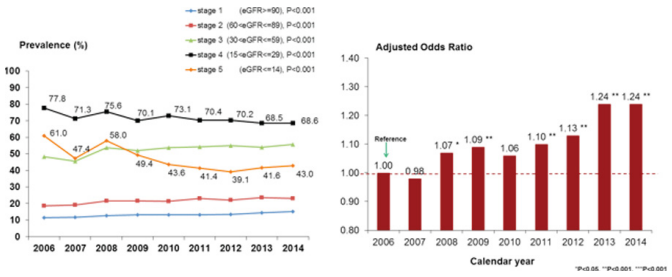
**Background:** An increasing body of evidence links hyperuricaemia with the development of several metabolic disorders and major cardiovascular outcomes. A better understanding of the burden and variation of hyperuricemia within the health system is important in order to identify high-risk groups and facilitate early intervention with effective management strategies.
**Objectives:** The aim of this study was to describe the prevalence of hyperuricaemia, and period trends within the Irish Health System among patients with chronic kidney disease (CKD).
**Methods:** 136,325 adult CKD patients aged 18 and above with valid measurements of serum uric acid and creatinine levels were identified between 2006 and 2014 from the laboratory systems within the Irish health system. Hyperuricaemia was defined as serum uric acid $\geq$420$\mu$mol/L in men and $\geq$360$\mu$mol/L in women. Estimated glomerular filtration rates were determined using the Chronic Kidney Disease-Epidemiology Collaboration (CKD-EPI) equation and patients were classified by CKD stage according to the Kidney Disease Improving Global Outcomes (KDIGO) staging system. Age- and sex-specific prevalence of hyperuricemia estimates with 95% confidence intervals were determined for each group and across calendar years. Comparisons among groups and across years were conducted using chi-square and multivariate logistic regression was used to explore associations using adjusted odds ratios (AOR) and 95% Confidence Intervals (CI).
**Results:** Patients with hyperuricaemia were noted to be older [58.2 (18.5) vs. 51.2 (17.4) years]. The prevalence of hyperuricaemia increased progressively between 2006 and 2014 from 20.3% (19.5, 21.0) to 26.5% (25.8, 27.2%) in men and from 17.9% (17.2, 18.6) to 20.4% (19.8, 21.0) in women, $p<0.001$. Age-specific prevalence increased significantly over time for all age groups (18–39, 40–59, 60–79, and $\geq$80 years) for men and women, $p<0.001$. Prevalence was significantly higher with more advanced CKD stage: 15.1% (14.5, 15.6) in Stage 1 CKD compared to 43.0% (34.8, 51.1) in Stage 5 CKD, $p<0.001$. However, rates fell significantly for those Stage 4 and 5 CKD respectively (Figure1). In multi-variable models, the adjusted likelihood of hyperuricaemia increased with each successive year (Figure 2).




**Conclusions:** The prevalence of hyperuricaemia is substantial in the Irish health system and has increased in frequency over the past decade. Although the burden was highest among patients with more advanced CKD, an encouraging decline in prevalence was observed in recent years. Greater management of gout and hyperuricaemia from increasing utilization of urate-lowering therapies may be responsible for this trend.
**Disclosure of Interest:** None declared
**DOI:** 10.1136/annrheumdis-2017-eular.5556

## SAT0690 RISK OF HOSPITALIZED INFECTION IN CANCER PATIENTS WITH AUTOIMMUNE DISEASES: A SINGLE-CENTER COHORT STUDY

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**Background:** Whether the risk of hospitalized infection is associated with autoimmune diseases in incident cancer patients is unknown.
**Objectives:** To examine the risk of hospitalized infection in incident cancer patients with autoimmune diseases compared with those without autoimmune diseases.
**Methods:** During 2000–2016, we identified 37,027 incident cancer patients from the Cancer Registry database of Taichung Veterans General Hospital. Autoimmune diseases included rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), ankylosing spondylitis (AS), psoriasis (PSO)/psoriatic arthritis (PSA), antiphospholipid syndrome, polymyositis, dermatomyositis, systemic sclerosis, Sjögren's syndrome, mixed connective tissue disease, multiple sclerosis, neuromyelitis optica, palindromic rheumatism, myasthenia gravis, Hashimoto's thyroiditis, immune thrombocytopenic purpura, autoimmune hemolytic anemia, juvenile idiopathic arthritis, adult onset Still's disease, Crohn's disease, ulcerative colitis, Wegner's granulomatosis, and uveitis. Of all subjects, 1,334 had autoimmune diseases. The association between autoimmune diaseases and hospitalized infection risk was shown by calculating hazard ratios (HRs) with 95% confidence intervals (CIs) using cox proportional regression analyses after adjusting for baseline age, sex, cancer stage, hemoglobulin (Hgb), creatinine (CR), log(ALT), log(WBC), and use of biologic agents or tofacitinib.
**Results:** Among all cancer subjects, the mean ± SD age was 60.2±14.7 years, and the proportion of male gender was 55.7%. Of the 1,334 patients with autoimmune diseases, 338 (25.3%) patients had RA, 221 (16.6%) patients had SLE, 61 (4.6%) patients had AS, 151 (11.4%) patients had PSO/PSA, and 563 (46.8%) had other autoimmune diseases. The incidence rates of hospitalized infection were 143.8 per $10^3$ years in patients with autoimmune diseases and 118.9 per $10^3$ years in patients without autoimmune diseases. The risk of hospitalized infection was higher in patients with RA and SLE, but not in patients with AS, PSO/PSA, or other autoimmune diseases. Prior use of biologic agents or tofaitinib did not increase hospitalized infection risk. Other risk factors for hospitalized infection included elder age (HR, 1.01; 95% CI, 1.01–1.1), higher cancer stage, CR (HR, 1.04; 95% CI, 1.02–1.06), log(WBC) (HR, 1.21; 95% CI, 1.14–1.28). Female gender (HR, 0.65; 95% CI, 0.62–0.68) and Hgb (HR, 0.89; 95% CI, 0.88–0.90) were associated lower hospitalized infection risk.

Table 1. Univariable and multivariable analyses for the association between autoimmune diseases and hospitalized infection among cancer patients

| | Univariate analysis HR (95% CI) | Multivariable analysis* HR (95% CI) |
|---|---|---|
| No autoimmune diseases | Reference | Reference |
| Autoimmune diseases | | |
| RA | 1.32 (1.10–1.59) | 1.37 (1.13–1.66) |
| SLE | 1.21 (0.95–1.54) | 1.41 (1.10–1.80) |
| AS | 0.67 (0.37–1.21) | 0.88 (0.46–1.70) |
| PSO/PSA | 1.36 (1.05–1.78) | 1.05 (0.80–1.38) |
| Others | 1.17 (1.01–1.36) | 1.15 (0.99–1.34) |

*Adjusted for age, gender, cancer stage, prior use of biologic agents or tofacitinib, baseline hemoglobulin, creatinine, log (ALT) and log (WBC).

**Conclusions:** Hospitalized infection was associated with a comorbidity of RA or SLE in incident cancer patients.
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**Disclosure of Interest:** None declared
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