score ranged from 6.2–6.5. Tofacitinib 5 and 10 mg BID achieved higher ACR responses and greater changes from baseline in DAS28-4(ESR) and HAQ-DI scores vs PBO at Month 3 in both populations (Table). Numerically higher proportions of non-MTX csDMARD-IR pts achieved efficacy outcomes vs 2nd-line population. CIRs for SAEs, DCs due to AEs and AEs of special interest were similar across groups; CIRs for TEAEs were higher with PBO vs tofacitinib. AE frequency was generally lower in the non-MTX csDMARD-IR population vs 2nd-line population.

Table. Efficacy outcomes at Month 3 and safety outcomes to Month 24

| | Tofacitinib 5 mg BID | | Tofacitinib 10 mg BID | | PBO | |
|---|---|---|---|---|---|---|
| | Non-MTX csDMARD-IR | Second-line | Non-MTX csDMARD-IR | Second-line | Non-MTX csDMARD-IR | Second-line |
| **% pts (95% CI) achieving efficacy outcome, FAS, NRI** | | | | | | |
| ACR20 response | 69.3 (62.5–75.6)[a] | 59.4 (56.1–62.5)[a] | 74.0 (67.6–79.7)[a] | 65.4 (62.3–68.4)[a] | 25.0 (14.0–39.0) | 27.1 (23.2–31.3) |
| ACR50 response | 35.6 (29.1–42.7)[a] | 30.7 (27.8–33.8)[a] | 44.8 (38.1–51.6)[a] | 35.0 (32.0–38.2)[a] | 11.5 (4.4–23.4) | 8.8 (6.5–11.7) |
| ACR70 response | 16.3 (11.5–22.2)[b] | 12.1 (10.1–14.4)[a] | 23.3 (17.9–29.5)[a] | 17.2 (14.8–19.8)[a] | 5.8 (1.2–16.0) | 2.7 (1.4–4.5) |
| DAS28-4(ESR)<2.6 | 9.2 (5.5–14.4) | 6.5 (4.9–8.4) | 16.9 (12.1–22.7)[a] | 10.2 (8.2–12.5) | 4.0 (0.5–13.7) | 1.6 (0.7–3.3) |
| **LSM (SE) change in score from baseline in efficacy outcome, FAS** | | | | | | |
| DAS28-4(ESR) | -2.1 (0.1)[a] | -1.9 (0.1)[a] | -2.5 (0.1)[a] | -2.1 (0.1)[a] | -1.0 (0.2) | -0.9 (0.1) |
| HAQ-DI | -0.5 (0.0)[a] | -0.5 (0.0)[a] | -0.6 (0)[a] | -0.5 (0.0)[a] | -0.2 (0.1) | -0.2 (0.0) |
| **Safety outcomes, CIR (95% CI)** | | | | | | |
| TEAE | 118.4 (99.9–139.2) | 192.4 (179.8–205.8) | 136.3 (116.8–158.1) | 193.7 (181.3–206.9) | 264.1 (183.9–367.3) | 292.2 (265.8–320.5) |
| SAE | 8.5 (5.2–12.9) | 12.4 (10.3–14.8) | 7.4 (4.6–11.3) | 9.7 (7.9–11.8) | 10.8 (1.3–39.1) | 11.5 (7.6–16.8) |
| DC due to AE | 7.1 (4.2–11.2) | 9.7 (7.8–11.8) | 7.2 (4.5–11.0) | 10.4 (8.6–12.6) | 10.8 (1.3–39.1) | 11.9 (7.9–17.2) |
| SIE | 2.8 (1.1–5.7) | 3.4 (2.4–4.7) | 1.4 (0.4–3.5) | 3.4 (2.4–4.7) | 5.4 (0.1–30.1) | 1.7 (0.5–4.3) |
| OI, excluding TB | 0.4 (0.0–2.2) | 0.2 (0.0–0.7) | 0.0 (0.0–1.3) | 0.5 (0.2–1.1) | 0.0 (0.0–19.9) | 0.0 (0.0–1.6) |
| TB | 0.0 (0.0–1.4) | 0.0 (0.0–0.4) | 0.3 (0.0–1.9) | 0.7 (0.3–1.3) | 0.0 (0.0–19.9) | 0.0 (0.0–1.6) |
| Herpes zoster | 1.2 (0.2–3.5) | 3.9 (2.8–5.3) | 3.5 (1.7–6.4) | 4.8 (3.5–6.3) | 0.0 (0.0–19.9) | 2.6 (0.9–5.5) |
| MACE | 0.8 (0.1–2.9) | 0.4 (0.1–1.1) | 0.7 (0.1–2.6) | 0.6 (0.2–1.3) | 0.0 (0.0–41.1) | 0.6 (0.0–3.5) |
| Malignancies[#] | 0.4 (0.0–2.2) | 0.7 (0.3–1.4) | 0.3 (0.0–1.9) | 0.7 (0.3–1.5) | 0.0 (0.0–19.9) | 0.0 (0.0–1.6) |

[a]p<0.001; [b]p<0.05 vs PBO; [#]excluding non-melanoma skin cancer; AE, adverse event; CI, confidence interval; CIR, crude incidence rate; csDMARD, conventional synthetic disease modifying antirheumatic drug; DAS28-4(ESR), disease activity score 28-4(ESR); DC, discontinuation; FAS, full analysis set; HAQ-DI, Health Assessment Questionnaire-Disability Index; IR, inadequate response; LSM, least squares mean; MACE, major adverse cardiovascular events; MTX, methotrexate; NRI, non-responder imputation; OI, opportunistic infection; PBO, placebo; SAE, serious AE; SE, standard error; SIE, serious infection event; TB, tuberculosis; TEAE, treatment emergent adverse event

**Conclusions:** This analysis indicates that tofacitinib is associated with similar efficacy and safety outcomes between csDMARD-IR (including MTX-IR) pts and those who are csDMARD-IR but not MTX-IR. This suggests a favourable tofacitinib benefit/risk profile for RA pts who have a contraindication to or refuse treatment with MTX and failed other csDMARDs.

**References:**

[1] Lopez-Olivo MA et al. Cochrane Database Syst Rev 2014; CD000957.

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## THU0196 SYSTEMATIC REVIEW AND META-ANALYSIS OF MALIGNANCIES, EXCLUDING NON-MELANOMA SKIN CANCER, IN PATIENTS WITH RHEUMATOID ARTHRITIS TREATED WITH TOFACITINIB OR BIOLOGIC DISEASE-MODIFYING ANTIRHEUMATIC DRUGS

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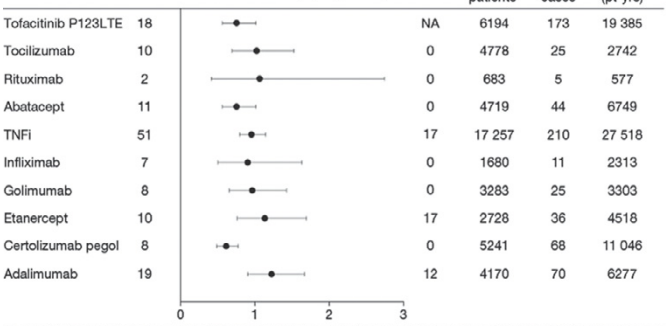
**Background:** Tofacitinib is an oral Janus kinase inhibitor for the treatment of rheumatoid arthritis (RA). The limited size of the control groups (placebo and active comparator) and limited duration of treatment within the tofacitinib RA clinical trial programme do not permit precise direct comparative assessments for adverse events of long latency, including malignancies, therefore a meta-analytic approach was taken.

**Objectives:** To compare the rate of malignancies in patients (pts) with moderately to severely active RA within the tofacitinib RA clinical trial programme with estimates from published trial data of approved biologic (b)DMARDS using meta-analytic methods.

**Methods:** Incidence rates (IR; pts with events per 100 pt-years [yrs] exposure) for tofacitinib were calculated using pooled data from randomised controlled trials (RCT) and long-term extension (LTE) studies. Two Phase (P) 1 studies, 9 P2 studies, 6 P3 studies and 2 LTE studies (one study ongoing; database unlocked at March 2015 data cut off) constituted the P123LTE dataset; pts received 1, 3, 5, 10, 15, 30 mg BID or 20 mg QD of tofacitinib. A systematic literature review of published RCT and LTE studies (through August 2014) of approved bDMARDs (abatacept, adalimumab, certolizumab, etanercept, golimumab, infliximab, rituximab and tocilizumab) was conducted. IRs for the endpoint of interest (all malignancies [excluding non-melanoma skin cancers (NMSC)]) were calculated for each bDMARD utilising a random-effects meta-analytic model with a restricted Maximum Likelihood Estimator for between-study variances. Estimates of Q statistic and $I^2$ were generated to determine the existence and degree of heterogeneity related to the study pool.

**Results:** The tofacitinib P123LTE dataset included 6194 pts with a total exposure of 19 385 pt-yrs. A total of 64 bDMARD articles were extracted for analysis, representing 58 unique studies and approximately 27 000 pts. Study populations were generally consistent across studies and treatments: mean pt age ranged from 51–54 yrs; mean percentage of pts who were female was 74–84%; and mean baseline C-reactive protein level was 20–31 mg/L. The IR (95% confidence interval [CI]) of malignancy for tofacitinib in the P123LTE dataset was 0.89 (0.76, 1.04) (Figure). Estimated IRs (95% CI) of malignancy were 0.75 (0.56, 1.01) for abatacept, 1.06 (0.41, 2.74) for rituximab, 1.02 (0.69, 1.52) for tocilizumab and 0.95 (0.79, 1.14) for tumour necrosis factor inhibitors (adalimumab, certolizumab, etanercept, golimumab and infliximab) (Figure).

Figure. Incidence rates for all malignancies (excluding NMSC) with biologic DMARDs and tofacitinib across RCTs and LTE studies




The results displayed include a continuity factor (r=0.05) to account for zero incidence rates. Tofacitinib data included through March 2015. Clinical trials for non-tofacitinib studies were conducted between 1997 and 2014.
CI, confidence interval; DMARD, disease-modifying antirheumatic drug; $I^2$, percent of total variability due to heterogeneity; LTE, long-term extension; NA, not applicable; NMSC, non-melanoma skin cancer; pt-yr, patient-year; RCT, randomised controlled trial; TNFi, tumour necrosis factor inhibitor

**Conclusions:** The results of this meta-analysis indicate that the tofacitinib IR for all malignancies (excluding NMSC) in pts with moderately to severely active RA is within a similar range to those reported in published interventional studies of similar RA populations treated with approved bDMARDs.

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## THU0197 TOFACITINIB, AN ORAL JANUS KINASE INHIBITOR, IN THE TREATMENT OF RHEUMATOID ARTHRITIS: SAFETY AND EFFICACY IN OPEN-LABEL, LONG-TERM EXTENSION STUDIES OVER 8 YEARS

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**Background:** Tofacitinib is an oral Janus kinase inhibitor for the treatment of rheumatoid arthritis (RA).

**Objectives:** The objective of this analysis was to report tofacitinib safety, tolerability and clinical efficacy in long-term extension (LTE) studies with up to 105 months of observation.

**Methods:** Data were pooled from two open-label studies (NCT00413699 [ongoing; database not locked at January 2016 data-cut] and NCT00661661) of patients with RA who had participated in Phase 1/2/3 tofacitinib studies. Patients received tofacitinib 5 or 10 mg twice daily (BID) as monotherapy or with background disease-

modifying antirheumatic drugs (DMARDs). Primary endpoints were adverse events (AEs) and confirmed laboratory safety data. Secondary endpoints included clinical efficacy measures (American College of Rheumatology [ACR] 20/50/70 response rates, Disease Activity Score using 28 joint counts and erythrocyte sedimentation rate [DAS28-4(ESR)], Health Assessment Questionnaire-Disability Index [HAQ-DI] and clinical disease activity index [CDAI]). Safety data were included up to Month 105 and efficacy data up to Month 90 (n≤100 at Month 96).

**Results:** A total of 4967 patients were treated (mean [max] duration: 1215 [3182] days). Total tofacitinib exposure was 16,711 patient-years; 77.4% of patients maintained their initial dose. In total, 2370 patients (47.7%) discontinued (AEs: 1131 [22.8%]; insufficient clinical response: 175 [3.5%]). The most common AE classes were infections and infestations (68.9%) and musculoskeletal/connective tissue disorders (39.0%). The most common AEs were nasopharyngitis (18.7%), upper respiratory tract infection (17.2%), bronchitis and urinary tract infection (12.2% each). Serious AEs occurred in 28.6% of patients and serious infection events (SIEs) in 8.8% of patients. Malignancies, excluding non-melanoma skin cancer, were reported in 3.0% of patients. Incidence rates (IR; patients with events per 100 patient-years) for AEs of interest (with 95% confidence intervals [CIs]) and laboratory observations are provided in Table 1. IRs for SIEs and malignancies through Month 105 did not increase compared with reported data through Month 96.1 No new safety risks were identified. Clinical responses were sustained from Month 1 to Month 90 (Table 2).

**Table 1.** Safety outcomes and laboratory observations in LTE studies (up to 105 months) of tofacitinib in patients with RA

| | Tofacitinib (5 and 10 mg BID) ± background DMARDs N=4967 |
|---|---|
| ***Incidence rates (patients with events per 100 patient-years) for AEs of interest (95% CI)*** | |
| SAEs | 9.5 (9.0, 10.0) |
| SIEs | 2.6 (2.4, 2.9) |
| Malignancies (excluding NMSC) | 0.9 (0.8, 1.0) |
| ***Confirmed laboratory abnormalities, n (%)*** | |
| Haemoglobin ≤7 g/dL or decrease in haemoglobin of ≥3 g/dL from BL | 97 (2.0) |
| Neutrophil <0.5 x $10^3$/$mm^3$ | 0 (0.0) |
| Lymphocyte <0.5 x $10^3$/$mm^3$ | 64 (1.3) |
| ALT ≥3 x ULN | 292 (5.9) |
| AST ≥3 x ULN | 167 (3.4) |
| Increase in serum creatinine of ≥50% from BL | 147 (3.0) |

AE, adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BID, twice daily; BL, baseline; CI, confidence interval; DMARDs, disease-modifying antirheumatic drugs; LTE, long-term extension; NMSC, non-melanoma skin cancer; RA, rheumatoid arthritis; SAEs, serious adverse events; SIEs, serious infection events; ULN, upper limit of normal.

**Table 2.** Clinical outcomes in LTE studies (up to 90 months) of tofacitinib in patients with RA

| | Tofacitinib (5 and 10 mg BID) ± background DMARDs | | |
|---|---|---|---|
| | BL | Month 1 | Month 90 |
| | | N=4907 | N=171 |
| ACR20 response rates, % | – | 73.0 | 83.0 |
| ACR50 response rates, % | – | 49.2 | 56.1 |
| ACR70 response rates, % | – | 28.9 | 32.7 |
| | N=4782 | N=4776 | N=168 |
| DAS28-4(ESR), mean (SE) | 6.29 (0.01) | 3.75 (0.02) | 3.38 (0.09) |
| | N=4924 | N=4880 | N=170 |
| HAQ-DI, mean (SE) | 1.42 (0.01) | 0.82 (0.01) | 0.76 (0.05) |
| | | N=4802 | N=169 |
| CDAI, mean change from BL (SE) | – | -24.0 (0.20) | -28.5 (1.02) |

ACR, American College of Rheumatology criteria; BID, twice daily; BL, baseline; CDAI, clinical disease activity index; DAS28-4(ESR), Disease Activity Score using 28 joint counts and erythrocyte sedimentation rate; DMARDs, disease-modifying antirheumatic drugs; HAQ-DI, Health Assessment Questionnaire-Disability Index; LTE, long-term extension; RA, rheumatoid arthritis; SE, standard error.

**Conclusions:** In patients with RA who remained in the LTE studies, tofacitinib (5 or 10 mg BID) with or without background DMARDs was associated with consistent safety through Month 105 and sustained clinical efficacy through Month 90.

**References:**

[1] Wollenhaupt J et al. Arthritis Rheumatol 2015; 67 (suppl 10): Abstract 1645.

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## THU0198 COMPARISON OF EFFICACY BETWEEN COMBINATION THERAPY WITH IGURATIMOD AND SULFASALAZINE WITH METHOTREXATE IN JAPANESE PATIENTS WITH RHEUMATOID ARTHRITIS: PROPENSITY SCORE ANALYSIS

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**Background:** Iguratimod (IGU) is a small-molecule disease-modifying antirheumatic drug (DMARD) that has been shown to suppress inflammation via the inhibition of nuclear factor-kappa B activation in vitro. The efficacy of combination therapy with IGU and methotrexate (MTX) has been demonstrated in comparison with that of placebo in rheumatoid arthritis (RA). However, its efficacy in comparison with other DMARDs such as sulfasalazine (SSZ) has not been elucidated.

**Objectives:** To clarify the efficacy of combination therapy with IGU in comparison with that of SSZ with MTX in typical clinical practice.

**Methods:** We analyzed data from 16,825 RA patients registered in a large database (NinJa: National Database of Rheumatic Diseases by iR-net in Japan) from April 2011 to March 2015 (1). In this study, we compared the two groups who received IGU or SSZ in addition to methotrexate in the earlier year. We excluded patients who started receiving biologic DMARDs, and IGU or SSZ the year prior to the study period, and those whose regimens were changed to other DMARDs such as tacrolimus and bucillamine. Baseline characteristics were compared using the *t* test, Wilcoxon test, or chi-square test. Fisher analysis was conducted for both outcomes. The predicted probability of IGU treatment was calculated by fitting a logistic regression model using all clinically relevant variables as presented in Table 1. Moreover, to reduce the effect of treatment-selection bias and potential confounding in this observational study, we performed rigorous adjustment for significant differences in the baseline characteristics of patients with propensity-score matching using the following algorithm: 1:1 optimal match with a ±0.15 caliper and no replacement. We used the standardized difference to measure covariate balance, whereby a standardized mean difference of $>0.1$ represents meaningful imbalance. The outcome was remission rate with disease activity score 28 CRP (DAS28-CRP) in the year after initiation of IGU or SSZ therapy.

**Results:** The group that received IGU in addition to MTX included 66 patients; the other group that received SSZ in addition to MTX included 163 patients. Table 1 shows the results of the pre- and post-propensity score matching of patients' characteristics. Sixty-five patients were compared in each group after score matching. The remission rates of DAS28-CRP in the following year was 77.2% (44/57 patients) and 71.7% (38/53 patients; P=0.52) in the IGU and SSZ groups, respectively.

**Table 1. Patients' Characteristics in Full and Propensity Score-Matched Cohorts according to Initiation of Iguratimod or Sulfasalazine**

| Characteristic | Full Cohort | | | Propensity Score-Matched Cohort | | |
|---|---|---|---|---|---|---|
| | IGU (n = 66) | SSZ (n = 163) | SMD | IGU (n = 65) | SSZ (n = 65) | SMD |
| Sex, male (%) | 32 (19.6) | 11 (16.7) | 0.077 | 13 (20.0) | 11 (16.9) | 0.079 |
| Age per decade (mean [SD]) | 5.57 (1.44) | 5.70 (1.15) | 0.097 | 5.78 (1.36) | 5.66 (3.24) | 0.099 |
| MTX (mg/week) (mean [SD]) | 9.38 (3.38) | 9.34 (3.28) | 0.01 | 9.26 (3.51) | 9.42 (3.24) | 0.048 |
| PSL (mg/day) (mean [SD]) | 1.72 (2.53) | 1.71 (2.53) | 0.002 | 1.51 (2.32) | 1.74 (2.54) | 0.093 |
| SJC 66 (mean [SD]) | 2.38 (3.57) | 3.03 (3.68) | 0.207 | 3.15 (4.10) | 2.97 (3.67) | 0.047 |
| TJC 68 (mean [SD]) | 2.70 (4.61) | 2.85 (4.52) | 0.033 | 2.57 (4.91) | 2.86 (4.55) | 0.062 |

IGU: iguratimod; MTX: methotrexate; PSL: prednisolone; SD: standard deviation; SJC: swollen joint count; SMD: standardized mean difference; SSZ: sulfasalazine; TJC: tender joint count

**Conclusions:** Combination therapy with IGU or SSZ and methotrexate for rheumatoid arthritis did not show a significant difference in disease activity. Further studies are needed.

**References:**

[1] Matsui T, Kuga Y, Kaneko A, et al. Disease Activity Score 28 (DAS28) using C-reactive protein underestimates disease activity and overestimates EULAR response criteria compared with DAS28 using erythrocyte sedimentation rate in a large observational cohort of rheumatoid arthritis patients in Japan. Ann Rheum Dis. 2007;66(9):1221–6.

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