We analyzed normality for DAS28, in order to compare disease activity at beginning and the end of follow-up.

Results: During 3 years we included 1953 patients were 39% were in low disease activity, 47% in moderate disease activity and 14% were in severe disease activity, 84% were female, mean age was 60 years $\pm$ 12. At baseline mean DAS28 was 4.45 $\pm$ 0.90 with a median of 4.3 at three years the mean DAS28 was 3.83 $\pm$ 1.08 with a median of 3.60. At the end of follow-up 46% of population achieved remission and 25% achieved low disease activity; at overall 71% improved disease activity, see table 1. In our study DAS28 was not normally distributed, thus we performed a Wilcoxon test in order to compare the mean DAS28 at baseline/36 months showing statistical significance ($P<0.05$).

ACTIVITY LEVEL	BASELINE		3 YEARS FOLLOW-UP	
	n	%	n	%
REM			898	46
LDA	759	39	488	25
MDA	912	47	528	27
SDA	282	14	39	2

Conclusions: Patients treated with conventional DMARD therapy and under a T2T model achieve favorable results in regards of disease activity. This is real life evidence that can support the advantages of treating RA patients with a multidisciplinary team under a T2T model with a low-cost treatment.

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.6122

SAT0243

INCIDENCE OF THROMBOEMBOLIC EVENTS IN THE TOFACITINIB RHEUMATOID ARTHRITIS, PSORIASIS, PSORIATIC ARTHRITIS AND ULCERATIVE COLITIS DEVELOPMENT PROGRAMMES

P.J. Mease^{1,*}, J. Kremer², S. Cohen³, J.R. Curtis⁴, C. Charles-Schoeman⁵, E.V. Loftus Jr⁶, J. Greenberg⁷, N. Palmetto⁸, K. S. Kanik⁹, D. Graham⁹, C. Wang⁹, P. Biswas⁹, G. Chan¹⁰, R. DeMasi¹⁰, H. Valdez⁸, T. Hendriks¹⁰, T. V. Jones¹⁰.

¹Swedish Medical Center and University of Washington, Seattle, ²Albany Medical College and The Center for Rheumatology, Albany, ³Metroplex Clinical Research Center, Dallas, ⁴University of Alabama at Birmingham, Birmingham, ⁵University of California, Los Angeles, ⁶Mayo Clinic, Rochester, ⁷Corrona, LLC, Southborough, ⁸Pfizer Inc, New York, ⁹Pfizer Inc, Groton, ¹⁰Pfizer Inc, Collegeville, United States

Background: Tofacitinib is an oral Janus kinase (JAK) inhibitor that preferentially inhibits signalling by JAK3 and JAK1, with functional selectivity over JAK2. Potential increased risk of venous thromboembolic events (VTE) in patients (pts) with rheumatoid arthritis (RA) has been reported for a JAK 1/2 inhibitor.¹

Objectives: To assess VTE risk with tofacitinib in pts with RA, psoriasis (PsO), psoriatic arthritis (PsA) and ulcerative colitis (UC).

Methods: Data from Phase (P)2 (RA, PsO, UC) and P3 (RA, PsO, PsA, UC) randomised clinical studies of tofacitinib as monotherapy or in combination with conventional synthetic (cs)DMARDs were reviewed. Two cohorts were defined; 1) the placebo (PBO)-controlled cohort: pts randomised to tofacitinib 5 or 10 mg BID, or PBO up to Month (M)3 in RA, PsO and PsA studies, and pts randomised to tofacitinib 10 mg BID or PBO for the 9-week induction period in UC studies; 2) the dose-comparison cohort: pts randomised to tofacitinib 5 or 10 mg BID, adalimumab (ADA) 40 mg SC Q2W (RA and PsA only) or methotrexate (MTX) 20 mg QW (RA only) throughout the P2/3 studies for RA (up to M24), PsO (up to M12) and PsA (up to M12), and for the 12-month P3 UC maintenance study. First deep vein thrombosis (DVT) and pulmonary embolism (PE) events were identified using the MedDRA embolic and thrombotic SMQ preferred terms restricted to the respiratory, thoracic, mediastinal and vascular disorder System Organ Classes; incidence rates (IRs; pts with events/100 pt-years) were based on single events occurring during treatment or $\leq$ 28 days after the last dose or up to the cohort cut-off date. IRs for PE in RA pts were compared with Corrona Registry data (May 2017 cut-off).

Results: Up to M3 in the PBO-controlled cohort, DVT and PE each occurred in 2 pts receiving PBO (1 RA pt and 1 UC pt per event); no tofacitinib-treated pts had DVT or PE events (Table). In the dose-comparison cohort 2 DVT events occurred in tofacitinib-treated pts with RA (5 mg BID, n=1; 10 mg BID, n=1) and 1 DVT event in a pt with PsA (tofacitinib 10 mg BID) (Table). IRs (95% CI) were 0.1 (0.0, 0.3) for both tofacitinib doses in RA and 0.5 (0.0, 2.8) for tofacitinib 10 mg BID in PsA. Two DVT events occurred with MTX; none with ADA. Five PE events occurred in the dose comparison cohort, all in RA (5 mg BID, n=2; 10 mg BID, n=3). IRs were 0.1 (0.0, 0.4) for tofacitinib 5 mg BID and 0.2 (0.0, 0.4) for 10 mg BID. IRs for PE with tofacitinib in RA were similar to those reported by the Corrona Registry in RA pts treated with tofacitinib (0.1 [0.0, 0.4]), biologic DMARDs (0.2 [0.1, 0.3]) and csDMARDs (0.2 [0.0, 0.5]).

Table 1 Incidence rates for deep vein thrombosis and pulmonary embolism from randomised controlled studies across the tofacitinib clinical development programme

n/N IR (95% CI) PE	Placebo-controlled cohort ^a			Dose-comparison cohort ^b			Methotrexate 20 mg QW
	Tofacitinib 5 mg BID	Tofacitinib 10 mg BID	Placebo	Tofacitinib 5 mg BID	Tofacitinib 10 mg BID	Adalimumab 40 mg SC Q2W	
Rheumatoid arthritis							
DVT	0/1849 0.0 (0.0, 0.0)	0/2004 0.0 (0.0, 0.0)	1/1879 0.1 (0.0, 0.3)	1/1849 0.1 (0.0, 0.3)	1/2004 0.1 (0.0, 0.3)	8/227 0.0 (0.0, 1.0)	2/223 0.1 (0.0, 0.3)
PE	0/1849 0.0 (0.0, 0.0)	0/2004 0.0 (0.0, 0.0)	1/1879 0.1 (0.0, 0.3)	1/1849 0.1 (0.0, 0.3)	1/2004 0.1 (0.0, 0.3)	8/227 0.0 (0.0, 1.0)	2/223 0.1 (0.0, 0.3)
Psoriasis^c							
DVT	0/123 0.0 (0.0, 1.0)	0/123 0.0 (0.0, 1.0)	0/123 0.0 (0.0, 2.1)	0/123 0.0 (0.0, 0.4)	0/123 0.0 (0.0, 0.4)	—	—
PE	0/123 0.0 (0.0, 1.0)	0/123 0.0 (0.0, 1.0)	0/123 0.0 (0.0, 2.1)	0/123 0.0 (0.0, 0.4)	0/123 0.0 (0.0, 0.4)	—	—
Psoriatic arthritis^c							
DVT	0/28 0.0 (0.0, 6.4)	0/28 0.0 (0.0, 6.4)	0/28 0.0 (0.0, 6.4)	0/28 0.0 (0.0, 1.8)	0/28 0.0 (0.0, 2.8)	0/28 0.0 (0.0, 4.0)	—
PE	0/28 0.0 (0.0, 6.4)	0/28 0.0 (0.0, 6.4)	0/28 0.0 (0.0, 6.4)	0/28 0.0 (0.0, 1.8)	0/28 0.0 (0.0, 2.8)	0/28 0.0 (0.0, 4.0)	—
Ulcerative colitis							
DVT	N/A	0/166 0.0 (0.0, 2.2)	0/166 0.0 (0.0, 2.2)	0/166 0.0 (0.0, 2.2)	0/166 0.0 (0.0, 2.2)	0/166 0.0 (0.0, 2.2)	—
PE	N/A	0/166 0.0 (0.0, 2.2)	0/166 0.0 (0.0, 2.2)	0/166 0.0 (0.0, 2.2)	0/166 0.0 (0.0, 2.2)	0/166 0.0 (0.0, 2.2)	—

^aIncludes Months 0–3 of Phase 2/3 studies for RA, PsO and PsA, and the 9-week induction period of Phase 2/3 UC studies. ^bIncludes Months 0–24 of Phase 2/3 studies for RA, Months 0–12 for PsO and PsA, and the 12-month P3 UC maintenance study. ^cDose-comparison cohort for tofacitinib 5 mg BID and 10 mg BID compares patients randomised at baseline to tofacitinib 5 mg BID + patients randomised at baseline to PBO + tofacitinib 5 mg BID, and patients randomised at baseline to tofacitinib 10 mg BID + patients randomised at baseline to PBO + tofacitinib 10 mg BID, respectively. IR=patients with event/100 PY.

Conclusions: Analysis of DVT and PE across randomised clinical studies for RA, PsO, PsA and UC showed no evidence of an increased risk of these events with tofacitinib vs other therapies.

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Acknowledgements: Study sponsored by Pfizer Inc. Medical writing support was provided by A MacLachlan of CMC and funded by Pfizer Inc.

Disclosure of Interest: P. Mease Grant/research support from: AbbVie, Amgen, Bristol-Myers Squibb, Celgene, Eli Lilly, Janssen, Novartis, Pfizer Inc, Sun, UCB, Consultant for: AbbVie, Amgen, Bristol-Myers Squibb, Celgene, Eli Lilly, Janssen, Novartis, Pfizer Inc, Sun, UCB, Speakers bureau: AbbVie, Amgen, Bristol-Myers Squibb, Celgene, Genentech, Janssen, Novartis, Pfizer Inc, UCB, J. Kremer Shareholder of: Corrona LLC, Grant/research support from: AbbVie, Amgen, Bristol-Myers Squibb, Eli Lilly, Genentech, Novartis, Pfizer Inc, Regeneron, Consultant for: AbbVie, Amgen, Bristol-Myers Squibb, Eli Lilly, Genentech, Novartis, Pfizer Inc, Regeneron, Employee of: Corrona LLC, S. Cohen Grant/research support from: Pfizer Inc, Consultant for: AbbVie, Amgen, Boehringer Ingelheim, Gilead, Merck, Pfizer Inc, J. Curtis Grant/research support from: AbbVie, Amgen, Bristol-Myers Squibb, Corrona LLC, Crescendo Bio, Eli Lilly, Janssen, Myriad, Pfizer Inc, Roche, UCB, Consultant for: AbbVie, Amgen, Bristol-Myers Squibb, Corrona LLC, Crescendo Bio, Eli Lilly, Janssen, Myriad, Pfizer Inc, Roche, UCB, C. Charles-Schoeman Grant/research support from: AbbVie, Bristol-Myers Squibb, Pfizer Inc, Consultant for: Amgen, Gilead, Pfizer Inc, Regeneron-Sanofi, E. Loftus Jr Grant/research support from: AbbVie, Allergan, Amgen, Celgene, Celltrion, CVS Caremark, Eli Lilly, Genentech, Gilead, Janssen, MedImmune, Mesoblast, Napo Pharmaceuticals, Pfizer Inc, Receptos, Robarts Clinical Trials, Salix, Seres Therapeutics, Takeda, UCB, Consultant for: AbbVie, Allergan, Amgen, Celgene, Celltrion, CVS Caremark, Eli Lilly, Genentech, Gilead, Janssen, MedImmune, Mesoblast, Napo Pharmaceuticals, Pfizer Inc, Receptos, Robarts Clinical Trials, Salix, Seres Therapeutics, Takeda, UCB, J. Greenberg Consultant for: Eli Lilly, Genentech, Janssen, Novartis, Pfizer Inc, Employee of: Corrona LLC, N. Palmetto Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, K. Kanik Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, D. Graham Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, C. Wang Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, P. Biswas Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, G. Chan Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, R. DeMasi Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, H. Valdez Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, T. Hendriks Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, T. Jones Shareholder of: Pfizer Inc, Employee of: Pfizer Inc

DOI: 10.1136/annrheumdis-2018-eular.3048