and ACR70. The safety and tolerability profile was consistent with observations in the Phase 2 studies with UPA.

Acknowledgements: AbbVie and the authors thank the patients, study sites and investigators who participated in this clinical trial. AbbVie, Inc was the study sponsor, contributed to study design, data collection, analysis and interpretation, and to writing, reviewing, and approval of final version. Medical writing support was provided by Naina Barretto, PhD, of AbbVie, Inc.

Disclosure of Interest: G. Burmester Consultant for: AbbVie Inc., Eli Lilly, Gilead, Janssen, Merck, Roche, Pfizer, and UCB., Speakers bureau: AbbVie Inc., Eli Lilly, Gilead, Janssen, Merck, Roche, Pfizer, and UCB., J. Kremer Shareholder of: Corona, Grant/research support from: AbbVie Inc, Consultant for: AbbVie Inc., BMS, Genentech, Gilead, GSK, Eli Lilly and Pfizer, Employee of: Corona, F. van den Bosch Consultant for: AbbVie Inc., Celgene, Eli Lilly, Janssen, Merck, Novartis, Pfizer and UCB, Speakers bureau: AbbVie Inc., Celgene, Eli Lilly, Janssen, Merck, Novartis, Pfizer and UCB, Y. Li Shareholder of: AbbVie Inc, Employee of: AbbVie Inc, Y. Zhou Shareholder of: AbbVie Inc, Employee of: AbbVie Inc, A. Othman Shareholder of: AbbVie Inc, Employee of: AbbVie Inc, A. Pangan Shareholder of: AbbVie Inc, Employee of: AbbVie Inc, H. Camp Shareholder of: AbbVie Inc, Employee of: AbbVie Inc,

DOI: 10.1136/annrheumdis-2018-eular.3131

OP0037

EFFICACY OF TOFACITINIB AFTER TEMPORARY DISCONTINUATION IN PATIENTS WITH RHEUMATOID ARTHRITIS: ANALYSIS OF DATA FROM OPEN-LABEL LONG-TERM EXTENSION STUDIES

J. Kaine[1], J. Tesser[2], R. DeMasi[3], L. Takiya[3], L. Wang[4], M. Snyder[3], H. Fan[4], V. Bandi[5], J. Wollenhaupt[6]. *[1]Sarasota Arthritis Research Center, Sarasota, FL; [2]Arizona Arthritis and Rheumatology Associates, Glendale, AZ; [3]Pfizer Inc, Collegeville, PA; [4]Pfizer Inc, Groton, CT; [5]Eliassen Group Inc, New London, CT, USA; [6]Schön-Klinik Hamburg-Eilbek Teaching Hospital of the University of Hamburg, Hamburg, Germany*

Background: Tofacitinib is an oral Janus kinase inhibitor for the treatment of rheumatoid arthritis (RA). Given the chronic nature of RA, there may be situations when therapy is temporarily discontinued. It is important to understand loss of response during temporary discontinuation and the ability to regain disease control following treatment reinitiation. Re-establishment of tofacitinib efficacy following temporary discontinuation/reinitiation has been previously demonstrated in patients (pts) with RA participating in the vaccine sub-study of the long-term extension (LTE) study ORAL Sequel.[1]

Objectives: To further assess tofacitinib efficacy and safety after temporary discontinuation and reinitiation in pts with RA in LTE studies.

Methods: Data were pooled from two open-label LTE studies (NCT00413699 [database lock: March 2017] and NCT00661661) of pts with RA who had completed qualifying Phase 1/2/3 index studies. Pts received tofacitinib 5 or 10 mg twice daily as monotherapy or with conventional synthetic disease-modifying antirheumatic drugs. Pts who received continuous tofacitinib for ≥4 months, temporarily discontinued tofacitinib for 14–30 days and then resumed treatment were included in the analysis. Efficacy outcomes were analysed at the pre-interruption visit (≤90 days before discontinuation) and at the post-interruption visit (within 14–42 days of resuming treatment); data from the interruption period were not analysed. Efficacy outcomes included: ACR20/50/70 response rates, mean tender and swollen joint counts, mean C-reactive protein levels and mean DAS28–4 (ESR), CDAI, HAQ-DI, Patient Global Assessment of arthritis, Pain and Physician Global Assessment of arthritis scores. Safety was analysed from Day 1 of temporary discontinuation to the post-interruption visit and included adverse events (AEs), serious AEs (SAEs) and discontinuations due to AEs that occurred within the time range.

Results: 261 pts met the criteria for temporary discontinuation. Median (range) duration of temporary discontinuation was 19[14–30] days. Pt demographics are shown in table 1. Efficacy outcomes were generally similar at pre- and post-interruption visits (table 2). From Day 1 of discontinuation to the post-interruption visit, AEs, SAEs and discontinuations due to AEs occurred in 142 (54.4%), 29 (11.1%) and 4 (1.5%) pts, respectively.

Abstract OP0037 – Table 1 Patients demographics and baseline disease characteristics[1]

	All tofacitinib (N=261)
Age (years), mean (SD)	53.7 (11.3)
Sex, n (%)	
Female	224 (85.8)
Race, n (%)	
White	173 (66.3)
Black	7 (2.7)
Asian	63 (24.1)
Other	18 (6.9)
BMI (kg/m^2), mean (SD)	26.8 (6.4)
Duration of disease (years), mean (SD)	9.4 (8.2)
TJC, mean (SD)	25.1 (15.2)
SJC, mean (SD)	15.4 (8.6)
CRP (mg/L), mean (SD)	20.6 (23.9)
DAS28-4(ESR), mean (SD)	6.4 (1.0)
CDAI, mean (SD)	36.6 (12.1)
HAQ-DI, mean (SD)	1.5 (0.7)
PtGA, mean (SD)	60.4 (22.1)
Pain-VAS, mean (SD)	58.8 (23.3)
PGA, mean (SD)	61.1 (16.9)
Background csDMARD use, n (%)[b]	181 (69.3)

[a]Baseline values were those of the index study if patients began LTE study treatment ≤14 days from the last tofacitinib dose in the index study. Otherwise, LTE baseline values were used; [b]At LTE baseline

BMI, body mass index; CDAI, Clinical Disease Activity Index; CRP, C-reactive protein; csDMARD, conventional synthetic disease-modifying antirheumatic drug; DAS28-4(ESR), Disease Activity Score in 28 joints, erythrocyte sedimentation rate; HAQ-DI, Health Assessment Questionnaire-Disability Index; LTE, long-term extension; PGA, Physician Global Assessment of arthritis; PtGA, Patient Global Assessment of arthritis; SD, standard deviation; SJC, swollen joint count; TJC, tender joint count; VAS, visual analogue scale

Abstract OP0037 – Table 2 Efficacy endpoints at the pre-interruption visit (≤90 days before temporary discontinuation) and post-interruption visit (within 14–42 days of resuming treatment)

	All tofacitinib (N=261)	
	Pre-interruption visit	Post-interruption visit
ACR20, % (95% CI)	75.5 (69.8, 80.6)	74.3 (68.6, 79.5)
ACR50, % (95% CI)	54.4 (48.2, 60.6)	51.0 (44.7, 57.2)
ACR70, % (95% CI)	31.8 (26.2, 37.8)	31.4 (25.8, 37.4)
TJC, mean (95% CI)	6.1 (4.9, 7.2)	8.0 (6.4, 9.6)
SJC, mean (95% CI)	3.4 (2.8, 4.1)	5.0 (3.9, 6.2)
CRP (mg/L), mean (95% CI)	0.48 (0.38, 0.58)	0.46 (0.37, 0.56)
DAS28-4(ESR), mean (95% CI)	3.6 (3.4, 3.7)	3.9 (3.7, 4.0)
CDAI, mean (95% CI)	10.0 (8.9, 11.1)	11.8 (10.3, 13.2)
HAQ-DI, mean (95% CI)	0.94 (0.85, 1.03)	0.98 (0.88, 1.07)
PtGA, mean (95% CI)	28.4 (25.8, 31.0)	29.3 (26.6, 32.1)
Pain (VAS), mean (95% CI)	28.1 (25.4, 30.8)	28.8 (26.0, 31.7)
PGA, mean (95% CI)	17.1 (15.3, 18.9)	18.0 (16.0, 19.9)

Median (range) time from the pre-interruption visit to Day 1 of temporary discontinuation was 43 (0–90) days and the median (range) time from resuming treatment to the post-interruption visit was 28 (14–42) days

N is the total number of patients in the analysis; the number of patients evaluable for each endpoint may be fewer than N

ACR, American College of Rheumatology; CDAI, Clinical Disease Activity Index; CI, confidence interval; CRP, C-reactive protein; DAS28-4(ESR), Disease Activity Score in 28 joints, erythrocyte sedimentation rate; HAQ-DI, Health Assessment Questionnaire-Disability Index; PGA, Physician Global Assessment of arthritis; PtGA, Patient Global Assessment of arthritis; SJC, swollen joint count; TJC, tender joint count; VAS, visual analogue scale

Conclusions: In pts with RA who temporarily discontinued tofacitinib, similar efficacy responses were observed at pre– and post–interruption visits, suggesting that there is no loss of efficacy after temporary withdrawal and reinitiation of tofacitinib. The safety profile was consistent with previous tofacitinib reports in LTE studies over 9 years.[2]

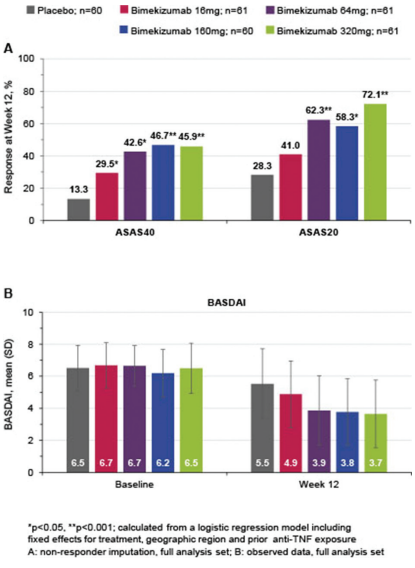
REFERENCES:
[1] Kaine J, et al. *Arthritis Rheumatol* 2017;69(suppl 10):Abstract 424.
[2] Wollenhaupt J, et al. *Arthritis Rheumatol* 2017;69(suppl 10):Abstract 522.

Acknowledgements: Study sponsored by Pfizer Inc. Medical writing support was provided by AG McCluskey of CMC and funded by Pfizer Inc.
Disclosure of Interest: J. Kaine Speakers bureau: Bristol-Myers Squibb, Pfizer Inc, J. Tesser Grant/research support from: Pfizer Inc, Consultant for: Pfizer Inc, Speakers bureau: Pfizer Inc, R. DeMasi Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, L. Takiya Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, L. Wang Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, M. Snyder Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, H. Fan Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, V. Bandi Consultant for: Pfizer Inc through Eliassen Group Inc, J. Wollenhaupt Speakers bureau: Pfizer Inc
DOI: 10.1136/annrheumdis-2018-eular.3755

LB0001 DUAL NEUTRALISATION OF IL-17A AND IL-17F WITH BIMEKIZUMAB IN PATIENTS WITH ACTIVE ANKYLOSING SPONDYLITIS (AS): 12-WEEK RESULTS FROM A PHASE 2B, RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED, DOSE-RANGING STUDY

D. van der Heijde[1], L.S. Gensler[2], A. Deodhar[3], X. Baraliakos[4], D. Poddubnyy[5], M. K. Farmer[6], D. Baeten[7], T. Kumke[8], M. Oortgiesen[6], M. Dougados[9]. *[1]Leiden University Medical Center, Leiden, Netherlands*; *[2]University of California, San Francisco*; *[3]Oregon Health and Science University, Portland, USA*; *[4]Ruhr-University Bochum, Herne*; *[5]Charité – Universitätsmedizin Berlin, German Rheumatism Research Centre, Berlin, Germany*; *[6]UCB Pharma, Raleigh, USA*; *[7]UCB Pharma, Brussels, Belgium*; *[8]UCB Pharma, Monheim, Germany*; *[9]Cochin Hospital, Paris, France*

Background: Dual neutralisation of IL-17F, in addition to IL-17A, reduces inflammation[1] to a greater extent than inhibition of IL-17A alone in disease–relevant cell models. Bimekizumab, a monoclonal antibody that potently and selectively neutralises both IL-17A and IL-17F, provided rapid and substantial clinical improvements in studies evaluating patients with psoriasis[2] and psoriatic arthritis.[1]
Objectives: Assess 12 week efficacy and safety of bimekizumab in patients with active AS; the primary objective was to assess the ASAS40 dose-response relationship at Week 12.



Abstract LB0001 – Figure 1 A) non-responder imputation, full analysis set; B) observed data, full analysis set

Methods: In this ongoing 48 week study (NCT02963506: double blind to Week 12 then dose blind to Week 48), 303 patients with active (BASDAI $\geq$4; spinal pain $\geq$4 [0–10 numerical rating scale]) AS, fulfilling the modified New York criteria, were randomised 1:1:1:1:1 to receive subcutaneous bimekizumab 16 mg, 64 mg, 160 mg, 320 mg or placebo Q4W, for 12 weeks. Prior exposure to 1 anti-TNF therapy was permitted. The primary endpoint was ASAS40 response rate at Week 12. Secondary endpoints (ASAS20 and ASAS5/6 response rate and change from baseline in BASDAI and ASDAS-CRP at Week 12) and safety were also assessed.

Results: Overall, 297 (98.0%) patients completed the 12 week double-blind period. The majority of patients were male (84.5%) with a mean (SD) age of 42.2 (11.8) and median (min, max) symptom duration of 13.3 (0.3, 48.2) years; baseline characteristics were similar among treatment groups (median [min, max] hs-CRP: 12.1 [0.3, 130.6] mg/L; mean [SD] BASDAI: 6.5 [1.4]; ASDAS-CRP: 3.9 [0.8]; prior anti-TNF exposure: 11.2%). At Week 12, there was a significant (p<0.001) dose-response for ASAS40. A greater percentage of bimekizumab-treated patients achieved ASAS40 (primary endpoint) than placebo (Figure: p<0.05, all doses). More patients receiving bimekizumab than placebo also achieved ASAS20 (figure 1; p<0.05, 64 mg–320 mg doses) and ASAS5/6 (16mg: 29.5%; 64 mg: 39.3%; 160 mg: 50.0%; 320 mg: 52.5%; placebo: 5.0%; p<0.05, all comparisons). Compared with placebo, greater reductions from baseline were achieved with bimekizumab for both BASDAI (figure 1) and ASDAS-CRP (LS mean [SE] change from baseline: 16 mg: −1.0 [0.15]; 64 mg: −1.6 [0.15]; 160 mg: −1.4 [0.16]; 320 mg: −1.5 [0.16]; placebo: −0.4 [0.16]; p<0.001, all doses). The overall incidence of TEAEs was 86/243 (35.4%) for bimekizumab-treated patients versus 22/60 (36.7%) for placebo. No unexpected safety risks were observed; the most frequently reported events were nasopharyngitis and headache.
Conclusions: The primary and key secondary objectives were achieved; dual neutralisation of IL-17A and IL-17F with bimekizumab provided clinically meaningful improvements in disease outcome measures. No new safety signals were observed versus previous studies.[1 2]

REFERENCES:
[1] Glatt. Ann Rheum Dis 2018;77:523–532.
[2] Glatt. Br J Clin Pharmacol 2017;83:991–1001.

Acknowledgements: Study funded by UCB Pharma. The authors acknowledge K Alexander of iMed Comms, an Ashfield Company, for medical writing support funded by UCB Pharma in accordance with GPP3.
Disclosure of Interest: D. van der Heijde Consultant for: AbbVie; Amgen, Astellas, AstraZeneca, BMS, Boehringer Ingelheim, Celgene, Daiichi, Eli-Lilly, Galapagos, Gilead, Glaxo-Smith-Kline, Janssen, Merck, Novartis, Pfizer, Regeneron, Roche, Sanofi, Takeda, UCB, Employee of: Director of Imaging Rheumatology BV, L. S. Gensler Grant/research support from: AbbVie, Amgen, UCB, Consultant for: Novartis, Lilly, Janssen, A. Deodhar Grant/research support from: AbbVie, Eli Lilly, Janssen, Novartis, Pfizer, UCB, Consultant for: AbbVie, Eli Lilly, Janssen, Novartis, Pfizer, UCB, X. Baraliakos Grant/research support from: Abbvie, Pfizer, MSD, UCB, Novartis, Consultant for: AbbVie, BMS, Boehringer Ingelheim, Celgene, Chugai, Janssen Biologics, Novartis, Pfizer, UCB, Galapagos, Speakers bureau: AbbVie, Chugai, Janssen, Novartis, Pfizer, UCB, D. Poddubnyy Grant/research support from: Abbvie, MSD, Novartis, Consultant for: Abbvie, BMS, Janssen, MSD, Novartis, Pfizer, UCB, Speakers bureau: Abbvie, BMS, Janssen, MSD, Novartis, Pfizer, Roche, UCB, M. K. Farmer Employee of: UCB Pharma, D. Baeten Employee of: UCB Pharma, T. Kumke Employee of: UCB Pharma, M. Oortgiesen Shareholder of: UCB Pharma, Employee of: UCB Pharma, M. Dougados Grant/research support from: UCB, Lilly, Pfizer, AbbVie, Merck, Consultant for: UCB, Lilly, Pfizer, AbbVie, Merck
DOI: 10.1136/annrheumdis-2018-eular.7889

WEDNESDAY, 13 JUNE 2018

Doctor, how bad will my rheumatoid become? RA – prognosis, predictors and outcomes

OP0038 DOSE TAPERING AND DISCONTINUATION OF BIOLOGICAL THERAPY IN RHEUMATOID ARTHRITIS PATIENTS IN REMISSION IN ROUTINE CARE – 2-YEAR OUTCOMES AND IDENTIFICATION OF PREDICTORS

C.H. Brahe, S. Krabbe, M. Østergaard, L. Ørnbjerg, D. Glinatsi, H. Røgind, H. S. Jensen, A. Hansen, J. Nørregaard, S. Jacobsen, L. Terslev, T.K. Huynh, D. V. Jensen, N. Manilo, K. Asmussen, P.B. Frandsen, M. Boesen, Z. Rastiemadabadi, L.M. Carlsen, J. Møller, N.S. Krogh, M.L. Hetland. *Departments of Rheumatology and Radiology, Hospitals at Bispebjerg-Frederiksberg, Gentofte, Hillerød, Herlev and Rigshospitalet, Capital Region, Denmark*

Background: A cohort of routine care rheumatoid arthritis (RA) patients in sustained remission had biological disease-modifying anti-rheumatic drugs (bDMARDs) tapered according to a treatment guideline. Little is known about predictors of successful tapering and discontinuation of bDMARDs.
Objectives: We studied: 1) the proportion of patients whose bDMARD could be successfully tapered or discontinued; 2) unwanted consequences of tapering/discontinuation; 3) potential baseline predictors of successful tapering and discontinuation.