APR20. Double-blind APR treatment continued to Wk 52; pts could continue APR for up to 4 additional years in an open-label extension.

Results: 504 pts were randomized and received ≥ 1 dose of study medication (PBO: n=168; APR30: n=168; APR20: n=168); 86.9% (225/259) of pts entering the third year completed 208 wks of APR treatment; overall, this is 44.6% (225/504) of pts randomized at baseline (BL). At Wk 52, 53.2% of APR30 pts achieved a modified ACR20 response (Table), regardless of when APR was started (BL, Wk 16, or Wk 24). At Wk 208, a sustained response rate was observed in APR30 pts, as shown by an ACR20 response rate of 67.5%. Marked improvements in SJC were seen throughout the study, with a mean percent decrease of -84.2% at Wk 208; TJC reductions were consistent (Table). Functionality is of paramount importance to pts; large improvements were seen in HAQ-DI score, with a mean change of -0.47. Pts also note fatigue as a disease- or treatment-related impairment; a mean improvement of 5.7 was seen in FACIT-F score at Wk 208 (Table), and the pt population reached a mean score of 35.7. In addition, long-term treatment led to the maintenance of the proportions meeting the minimal clinically important difference in HAQ-DI score change, achieving ACR50/ACR70 responses and reaching PASI-75 and PASI-50 responses (Table). No new safety concerns were identified with APR treatment up to 208 wks. During Wks >156 to ≤ 208 , the only adverse event (AE) occurring in $\geq 5\%$ of APR30-exposed pts was URTI (5.2%); most AEs were mild/moderate in severity. Among APR30-exposed pts, serious AEs occurred in 6.7% of pts in Wks >156 to ≤ 208 , consistent with earlier data. Importantly, few discontinuations due to AEs occurred throughout the long-term treatment period.

	Outcomes at Wk 52 and Wk 208	
	Wk 52 APR30 n=193*	Wk 208 APR30 n=123*
ACR20, n/m ^a (%)	101/190 (53.2)	83/123 (67.5)
ACR50, n/m ^a (%)	49/191 (25.7)	56/122 (45.9)
ACR70, n/m ^a (%)	27/191 (14.1)	35/123 (28.5)
Swollen joint count, mean % change	-50.5	-84.2
Tender joint count, mean % change	-45.1	-78.1
HAQ-DI (0-3), mean change	-0.31	-0.47
HAQ-DI MCID ≥ 0.30 , n/m (%)	85/193 (44.0)	72/123 (58.5)
HAQ-DI MCID ≥ 0.35 , n/m (%)	85/193 (44.0)	72/123 (58.5)
DAS-28 (CRP) <2.6, n/m (%)	41/189 (21.7)	57/122 (46.7)
DAS-28 (CRP), mean change	-1.26	-2.08
FACIT-F, mean change	3.8	5.7
PASI-75 ^a , n/m (%)	31/95 (32.6)	21/60 (35.0)
PASI-50 ^a , n/m (%)	52/95 (54.7)	37/60 (61.7)

Data as observed. *The n reflects the number of pts treated with APR30, regardless of when treatment started (BL, Wk 16, or Wk 24), and who had data available at the specific time point; actual number of pts available for each end point may vary. ^aDenominators vary slightly due to availability of sufficient data for each level of ACR response assessment. ^bExamined among pts with psoriasis involvement of the body surface area $\geq 3\%$ at BL and data at the specific time point. APR30=apremilast 30 mg BID; ACR20/50/70= $\geq 20\%/50\%/70\%$ improvement in modified American College of Rheumatology response criteria; n=m=number of responders/number of pts with sufficient data for evaluation; HAQ-DI=Health Assessment Questionnaire-Disability Index; MCID=minimal clinically important difference; DAS-28=28-joint count Disease Activity Score; CRP=C-reactive protein; FACIT-F=Functional Assessment of Chronic Illness Therapy-Fatigue; PASI-75/50= $\geq 75\%/50\%$ reduction from BL Psoriasis Area and Severity Index score; pts=patients; BL=baseline.

Conclusions: APR30 demonstrated sustained, clinically meaningful improvements in signs and symptoms of PsA, physical function, and associated psoriasis over 208 wks. APR30 continued to demonstrate a favorable safety profile and was generally well tolerated.

Disclosure of Interest: A. Kavanaugh Grant/research support from: Abbott, Amgen, AstraZeneca, BMS, Celgene Corporation, Centocor-Janssen, Pfizer, Roche, UCB, D. Gladman Grant/research support from: AbbVie, Amgen, BMS, Celgene Corporation, Janssen, Novartis, Pfizer, UCB, Consultant for: AbbVie, Amgen, BMS, Celgene Corporation, Janssen, Novartis, Pfizer, UCB, J. Gomez-Reino Grant/research support from: Roche and Schering-Plough, Consultant for: BMS, Pfizer, Roche, Schering-Plough, UCB, S. Hall Consultant for: Boehringer Ingelheim, MSD, Roche, Schering-Plough, Servier, Wyeth, Paid instructor for: Amgen, AstraZeneca, Boehringer Ingelheim, Centocor, GSK, MSD, Pfizer, Sanofi Aventis, Sanofi Pasteur, Schering-Plough, Serono, Wyeth, Speakers bureau: Boehringer Ingelheim, GSK, MSD, Pfizer, Roche, Sanofi Aventis, Schering-Plough, Wyeth, E. Lespessailles Grant/research support from: Amgen, Eli Lilly, Novartis, Servier, Speakers bureau: Amgen, Eli Lilly, Novartis, Servier, P. Mease Grant/research support from: Abbott, Amgen, Biogen Idec, BMS, Celgene Corporation, Genentech, Janssen, Eli Lilly, Novartis, Pfizer, Roche, UCB, Consultant for: Abbott, Amgen, Biogen Idec, BMS, Celgene Corporation, Genentech, Janssen, Eli Lilly, Novartis, Pfizer, UCB, G. Schett Grant/research support from: Abbott, Celgene Corporation, Roche, UCB, Consultant for: Abbott, Celgene Corporation, Roche, UCB, M. McIlraith Employee of: Celgene Corporation, N. Delev Employee of: Celgene Corporation, M. Paris Employee of: Celgene Corporation, L. Teng Employee of: Celgene Corporation, J. Wollenhaupt Grant/research support from: Abbott, BMS, MSD, Pfizer, UCB, Consultant for: Abbott, BMS, MSD, Pfizer, UCB

DOI: 10.1136/annrheumdis-2017-eular.3001

SAT0437 IXEKIZUMAB IMPROVES NAIL AND SKIN LESIONS IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS AND PRIOR TNF INADEQUATE RESPONSE

L.E. Kristensen¹, J.F. Merola^{2,3}, J. Dutz⁴, D.H. Adams⁵, L. Kerr⁵, P. Rich⁶.

¹Copenhagen University Hospital, Bispebjerg and Frederiksberg, Denmark;

²Brigham and Women's Hospital; ³Harvard Medical School, Boston, United States;

⁴The University of British Columbia, Vancouver, Canada; ⁵Eli Lilly and Company, Indianapolis; ⁶Oregon Health Science University, Portland, United States

Background: Ixekizumab (IXE), a high-affinity monoclonal antibody selectively targeting interleukin-17A, significantly improved fingernail psoriasis by Week (Wk) 12 vs placebo (PBO) in patients (pts) with moderate-to-severe plaque psoriasis¹. Moreover, IXE had significantly reduced signs and symptoms of psoriatic arthritis (PsA) at Wk 24 in biologic disease-modifying antirheumatic drug (bDMARD)-naïve pts with active PsA².

Objectives: To evaluate the impact of IXE on nail and skin lesions in active PsA pts who have previously received bDMARD therapy.

Methods: In this phase 3, multicentre, double-blind, PBO-controlled, outpatient study, pts (aged ≥ 18 years) with active PsA and bDMARD-experience randomly (1:1:1) received PBO for 24 Wks, 80 mg IXE as one injection every 2 (IXE Q2W) or 4 Wks (IXE Q4W) after starting dose of 160 mg. At Wk 16, inadequate responders received rescue medication and PBO pts were re-randomised (1:1) to receive either IXE Q2W or IXE Q4W. The efficacy of IXE was assessed by Nail Psoriasis Severity Index (NAPSI) in pts with nail psoriasis at baseline (PBO, n=73; IXE Q4W, n=89; IXE Q2W, n=74), Psoriasis Area and Severity Index (PASI) 75/90/100 response rate at Wk24 in pts with baseline body surface area (BSA) ≥ 3 (PBO, n=67; IXE Q4W, n=68; IXE Q2W, n=68) and percentage of pts achieving static Physician Global Assessment (sPGA) of psoriasis score of 0 (cleared) or 1 (minimal) in pts with baseline sPGA ≥ 3 (PBO, n=55; IXEQ4W, n=60; IXEQ2W, n=62). Missing data and inadequate responder data were imputed using the nonresponder imputation for categorical variables. For continuous variables, least squares mean (LSM) changes were calculated using mixed effects models for repeated measures, observed data from Wks 16 to 24 was excluded for inadequate responders.

Results: Overall the demographics and baseline characteristics were comparable between the treatment groups. The mean (SD) NAPSI total score at baseline was 19 (19), 20 (20), and 21 (22) in PBO, IXE Q4W and IXE Q2W, respectively. At Wk 24, the change in NAPSI total score from baseline was significantly greater with IXE Q4W (LSM $\pm$ SE -10.2 $\pm$ 2.04) and IXE Q2W (-10.8 $\pm$ 2.12) vs PBO (0.5 $\pm$ 2.13, p<.001 each). Similarly, the mean percentage improvement in NAPSI total score was significantly greater with IXE Q4W (LSM $\pm$ SE 35.4 $\pm$ 22.14, p<.001) and IXE Q2W (30.9 $\pm$ 22.95, p=.001) vs PBO (-45.2 $\pm$ 23.04). At Wk 24, significantly greater percentage of pts achieved a NAPSI score of 0 with IXEQ4W or IXEQ2W treatment vs PBO (Table). At Wk 24, the percentage of pts achieving PASI 75/90/100 response was significantly greater with IXE Q4W and IXE Q2W vs PBO (Table). Overall, the safety profile of IXE was aligned with the general study population.

Table: Percentage of patients with improvement in skin and nail lesions

Efficacy endpoints (%)	Placebo	IXE Q4W	IXE Q2W
PASI 75 ^a	14.9	55.9*	60.3*
PASI 90 ^a	11.9	44.1*	50.0*
PASI 100 ^a	4.5	35.3*	27.9*
sPGA (0, 1) ^b	9.1	66.7*	75.8*
NAPSI (0) ^c	6.8	20.2**	29.7*

*p<.001; **p=.022; ^aPatients with baseline $\geq 3\%$ BSA; ^bin patients with baseline sPGA ≥ 3 ; ^cPatients with baseline fingernail psoriasis

Conclusions: In the present study, ixekizumab led to significantly greater reduction and clearance of the nail and skin lesions in active PsA pts who had previously received bDMARD therapy compared to placebo.

References:

- [1] Denney EB et al. J Drugs Dermatol. 2016;15(8):958-61.
- [2] Mease PJ, et al. Ann Rheum Dis. 2016 doi: 10.1136/annrheumdis-2016-209709.

Disclosure of Interest: L. Kristensen Grant/research support from: UCB, Janssen-Cilag and Biogen, Consultant for: Pfizer, AbbVie, Amgen, UCB, Celgene, BMS, MSD, Novartis, Eli Lilly, and Janssen pharmaceuticals, Speakers bureau: Pfizer, AbbVie, Amgen, Biogen, UCB, Celgene, BMS, MSD, Novartis, Eli Lilly, and Janssen pharmaceuticals, J. Merola Grant/research support from: Biogen Idec, Consultant for: Biogen Idec, Amgen, AbbVie, and Eli Lilly, Novartis, Pfizer, Janssen, UCB, Kiniksa, Momenta and Mallinckrodt, J. Dutz Grant/research support from: AbbVie, Novartis, Amgen, Consultant for: Ciphor, Speakers bureau: Janssen, AbbVie, Novartis, Amgen, Leo, Celgene, D. Adams Employee of: Eli Lilly and Company, L. Kerr Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, P. Rich Grant/research support from: UCB, Janssen-Cilag and Biogen, Consultant for: Pfizer, AbbVie, Amgen, UCB, Celgene, BMS, MSD, Novartis, Eli Lilly, and Janssen pharmaceuticals, Speakers bureau: Pfizer, AbbVie, Amgen, Biogen, UCB, Celgene, BMS, MSD, Novartis, Eli Lilly, and Janssen pharmaceuticals.

DOI: 10.1136/annrheumdis-2017-eular.3105