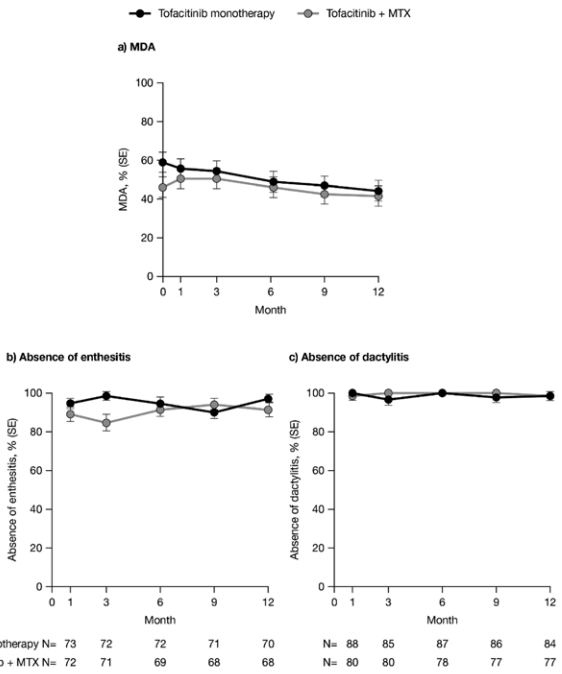
Figure 2. Proportion (SE) of patients achieving a) MDA,[a] and maintaining absence of b) enthesitis[b] and c) dactylitis,[c] up to Month 12 of the MTX withdrawal substudy



[a]Missing response=non-response. The numbers of patients included in this analysis were 90 and 89 for tofacitinib monotherapy and tofacitinib + MTX, respectively; [b]In patients with LEI=0 at baseline, no imputation; [c]In patients with DSS=0 at baseline, no imputation DSS, Dactylitis Severity Score; LEI, Leeds Enthesitis Index; MDA, minimal disease activity; MTX, methotrexate; SE, standard error

Table. Safety outcomes to Month 12

Pts with events, n (%) AEs of special interest	Tofacitinib monotherapy N=90	Tofacitinib + MTX N=89
AE	43 (47.8)	41 (46.1)
Serious AE	4 (4.4)	3 (3.4)
Discontinuations due to AE	3 (3.3)	4 (4.5)
Death	0	0
Herpes zoster (serious/non-serious)	1 (1.1)	2 (2.2)
Serious infection	0	2 (2.2)
Opportunistic infection[a]	0	1 (1.1)
Malignancy (excl. NMSC)[a]	1 (1.1)	1 (1.1)
NMSC[a]	0	0
Major adverse cardiovascular event[a]	0	0
Venous thromboembolism[c]	0	0
Arterial thromboembolism[c]	1 (1.1)	0
Gastrointestinal perforation[a]	0	0
Interstitial lung disease[b]	0	0
Laboratory parameters[d]		
ALT ≥3×ULN	0	5 (5.6)
ALT (IU/L), mean (SE)	-2.7 (1.6)	2.5 (1.3)
AST ≥3×ULN	0	3 (3.4)
AST (IU/L), mean (SE)	-1.5 (1.2)	1.7 (0.8)

Reviewed by independent [a]external/[b]internal adjudication committee
[c]Per Standardised MedDRA Query terms
[d]Without regard to baseline abnormality
ALT, alanine aminotransferase; AST, aspartate aminotransferase; ULN, upper limit of normal

Acknowledgments: Study sponsored by Pfizer Inc. Medical writing support was provided by Christina Viegelmann of CMC Connect and funded by Pfizer Inc.
Disclosure of Interests: Peter Nash Grant/research support from: AbbVie, Bristol-Myers Squibb, Celgene, Eli Lilly and Company, Gilead, Janssen, MSD, Novartis, Pfizer Inc, Roche, Sanofi, UCB, Consultant of: AbbVie, Bristol-Myers Squibb, Celgene, Eli Lilly, Gilead, Janssen, MSD, Novartis, Pfizer Inc, Roche, Sanofi, UCB, Speakers bureau: AbbVie, Bristol-Myers Squibb, Celgene, Eli Lilly, Gilead, Janssen, MSD, Novartis, Pfizer Inc, Roche, Sanofi, UCB, Laura C Coates: None declared, Philip J Mease Grant/research support from: Abbott, Amgen, Biogen Idec, BMS, Celgene Corporation, Eli Lilly, Novartis, Pfizer, Sun Pharmaceutical, UCB – grant/research support, Consultant of: Abbott, Amgen, Biogen Idec, BMS, Celgene Corporation, Eli Lilly, Novartis, Pfizer, Sun Pharmaceutical, UCB – consultant, Speakers bureau: Abbott, Amgen, Biogen Idec, BMS, Eli Lilly, Genentech, Janssen, Pfizer, UCB – speakers bureau, Alan Kivitz Shareholder of: AbbVie, Amgen, Gilead, GSK, Pfizer Inc, Sanofi, Consultant of: AbbVie, Boehringer Ingelheim,,Flexion, Genzyme, Gilead, Janssen, Novartis, Pfizer Inc, Regeneron, Sanofi, SUN Pharma Advanced Research, UCB, Paid instructor for: Celgene, Genzyme, Horizon, Merck, Novartis, Pfizer, Regeneron, Sanofi, Speakers bureau: AbbVie, Celgene, Flexion, Genzyme, Horizon, Merck, Novartis, Pfizer Inc, Regeneron, Sanofi, Dafna D Gladman Grant/research support from: AbbVie, Amgen Inc., BMS, Celgene Corporation, Janssen, Novartis, Pfizer, UCB – grant/research support, Consultant of: AbbVie, Amgen Inc., BMS, Celgene Corporation, Janssen, Novartis, Pfizer, UCB – consultant, Frank Behrens Grant/research support from: Pfizer, Janssen, Chugai, Celgene, Lilly and Roche, Consultant of: Pfizer, AbbVie, Sanofi, Lilly, Novartis, Genzyme, Boehringer, Janssen, MSD, Celgene, Roche and Chugai, James Cheng-Chung Wei Grant/research support from: AbbVie, Bristol-Myers Squibb, Celgene, Eli Lilly, Janssen, Novartis, Pfizer Inc, UCB, Consultant of: AbbVie, Bristol-Myers Squibb, Celgene, Chugai, Eisai, Janssen, Novartis, Pfizer Inc, Sanofi-Aventis, UCB Pharma, Dona Fleishaker Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, Joseph Wu Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, Cunshan Wang Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, Ana Belen Romero Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, Lara Fallon Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, Ming-Ann Hsu Shareholder of: Pfizer Inc, Employee of: Pfizer Inc, Keith Kanik Shareholder of: Pfizer Inc, Employee of: Pfizer Inc
DOI: 10.1136/annrheumdis-2020-eular.529

OP0226 NETAKIMAB DECREASES DISEASE ACTIVITY IN PATIENTS WITH PSORIATIC ARTHRITIS: RESULTS FROM A RANDOMIZED DOUBLE-BLIND PHASE 3 CLINICAL TRIAL (PATERA)

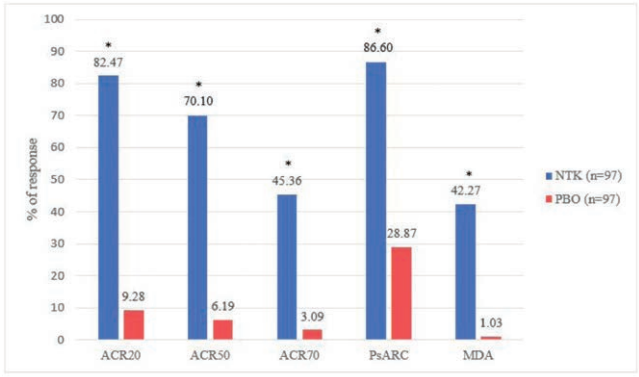
T. Korotaeva[1], I. Gaydukova[2], V. Mazurov[2], A. Samtsov[3], V. Khayrutdinov[3], A. Bakulev[4], M. Kokhan[5], A. Kundzer[6], N. Soroka[7], E. Dokukina[8], A. Eremeeva[8].
[1]Nasonova Research Institute of Rheumatology, Moscow, Russian Federation; [2]Mechnikov North-Western State Medical University, St-Petersburg, Russian Federation; [3]Kirov Military Medical Academy, St-Petersburg, Russian Federation; [4]Razumovsky Saratov State Medical University, Saratov, Russian Federation; [5]Ural Research Institute of Dermatovenereology and Immunopathology, Ekaterinburg, Russian Federation; [6]Healthcare Institution Municipal Clinical Hospital No. 1, Minsk, Belarus; [7]Scientific and Practical Center of Surgery, Transplantology and Hematology, Minsk, Belarus; [8]JSC BIOCAD, St-Petersburg, Russian Federation

Background: Netakimab (NTK) is a humanized anti-interleukin 17A antibody approved for the treatment of moderate-to-severe plaque psoriasis.
Objectives: To determine the efficacy and safety of NTK in patients (pts) with active psoriatic arthritis (PsA), based on 24-week (Wk) data from an ongoing phase 3 study (NCT03598751, PATERA).
Methods: 194 eligible adult pts with PsA (CASPAR, 2006) with inadequate response to csDMARD or one TNFi, were randomized (1:1) to receive NTK 120 mg or placebo (PBO) subcutaneously at Wk 0, 1, 2, 4, 6, 8, 10, 14, 18, 22. 84 pts from PBO arm who did not meet ACR20 (20% improvement of the American College of Rheumatology criteria) by Wk 16 were switched to NTK 120 mg. The primary endpoint was ACR20 at Wk 24. DAPSA (Disease Activity Index for Psoriatic Arthritis), the proportion of pts achieved ACR50/70, minimal disease activity (MDA) (≥5/7 MDA criteria) and Psoriatic arthritis response criteria (PsARC) were also analyzed.
Results: Baseline demographics and disease characteristics were similar across treatment arms (Table 1). 80 (82.47%) pts in NTK arm and 9 (9.28%) in the PBO arm achieved ACR20 at Wk 24 (p<0.0001). A significantly greater percentage of NTK-treated pts had ACR50/70, PsARC response, MDA at Wk 24 (Figure 1). By Wk 24 DAPSA significantly improved for NTK vs PBO. DAPSA remission was achieved by 36.08% and 13.40% in NTK and PBO arms, respectively (p=0.003). NTK was well tolerated. The most frequent AEs (≥3%) were lymphopenia, neutropenia, hypercholesterolemia, ALT increased, upper respiratory tract infection, systolic blood pressure increased, hyperglycemia, hyperbilirubinemia. Most AEs were mild to moderate. Severe treatment-related AEs were observed in 1.03% vs 2.06% for NTK and PBO, respectively. No treatment-related SAEs were reported. No anti-drug antibodies were detected.

Table 1. Baseline demographics and disease severity characteristics

Arm	NTK (N=97)	PBO (N=97)
Age (years) *	44.0 (11.66)	43.1 (11.88)
Male, n (%)	52 (53.61)	50 (51.55)
PsA duration, mo*	63.1 (73.12)	68.2 (77.49)
DAS28-CRP*	4.62 (0.97)	4.41 (1.11)
DAPSA*	32.19 (12.23)	33.54 (15.98)
TJC (66/68) *	12.9 (9.97)	12.0 (9.88)
SJC (66/68) *	7.0 (4.93)	7.2 (7.18)
MTX at baseline	83 (85.6)	83 (85.6)
Previous PsA therapy		
Sulfasalazine, n (%)	9 (9.28)	11 (11.34)
Leflunomide, n (%)	4 (4.12)	8 (8.25)
Anti-TNFα, n (%)	22 (22.68)	17 (17.53)

* mean (standard deviation); Mo=months, PsA=psoriatic arthritis, SJC=swollen joint count, TJC=tender joint count, DAS28=Disease Activity Score, MTX=methotrexate, CRP=C-reactive protein, DAPSA=Disease activity index for psoriatic arthritis, TNF=tumor necrosis factor



* p<0.0001 for comparison with placebo

Figure 1. Treatment response at Wk 24

Conclusion: NTK is a well-tolerated monoclonal antibody, that provided sustained improvements in signs and symptoms of active PsA through 24 Wks of therapy.

Table 2. Safety data

Arm	NTK (N=97)	PBO (N=97)	p-value
Treatment-related AEs	12 (12.37)	7 (7.22)	0.227[1]
Treatment-related SAEs	0 (0)	0 (0)	1.00[2]
Treatment-related AEs (grade 3-4)	1 (1.03)	2 (2.06)	1.00[2]
Local reactions	0 (0)	0 (0)	-
Grade 3-4 treatment-related AEs			
blood pressure increased	1 (1.03)	0(0)	1.00[2]
lymphopenia	0 (0)	2 (2.06)	0.497[2]

n (%) are presented, [1] Pearson's χ^2 test, [2] Fisher's exact test; N=number of patients, AE=adverse event, SAE=serious adverse event, ALT=Alanine transaminase

Acknowledgments: This study was sponsored by JSC BIOCAD.

Disclosure of Interests: Tatiana Korotaeva Consultant of: Pfizer, MSD, Novartis, AbbVie, Celgene, JSC BIOCAD, Janssen, UCB, Lilly and Novartis-Sandoz, Speakers bureau: Pfizer, MSD, Novartis, AbbVie, Celgene, JSC BIOCAD, Janssen, UCB, Lilly and Novartis-Sandoz, Inna Gaydukova Grant/research support from: JSC BIOCAD, Speakers bureau: Pfizer, Novartis, AbbVie, JSC BIOCAD, Celgene, MSD, Sanofi, V Mazurov: None declared, Aleksey Samtsov Grant/research support from: JSC BIOCAD, Novartis, Eli Lilly, Johnson&Johnson, Celgene, Glenmark, Galderma, Sanofi, Vladislav Khayrutdinov Grant/research support from: Akrikhin, Alkoy, Belupo, JSC BIOCAD, Bosnaliejk, Verteks, Glenmark, Elfa, Leo Pharma, MSD, Novartis, Pfizer, Sun Pharma, Sanofi, Celgene, Pharmtec, AbbVie, Eli Lilly, Jadran, Janssen, Andrey Bakulev Grant/research support from: AbbVie, Eli Lilly, Pfizer, UCB, MSD, Novartis, Galderma, Celgene, Leo Pharma and Johnson&Johnson, JSC BIOCAD, Consultant of: Novartis, Celgene and Johnson&Johnson, Speakers bureau: AbbVie, Eli Lilly, Galderma, UCB, Novartis, Celgene and Johnson&Johnson, Muza Kokhan Grant/research support from: AbbVie, Eli Lilly, Pfizer, UCB, MSD, Novartis, Galderma, Celgene, Leo Pharma and Johnson&Johnson, JSC BIOCAD, Consultant of: Novartis, Celgene and Johnson&Johnson, Speakers bureau: AbbVie, Eli Lilly, Galderma, UCB, Novartis, Celgene and Johnson&Johnson, Alena Kundzer: None declared, Nikolaj Soroka Grant/research support from: JSC BIOCAD, Ekaterina Dokukina Employee of: JSC BIOCAD, Anna Eremeeva Employee of: JSC BIOCAD

DOI: 10.1136/annrheumdis-2020-eular.3469

OP0227

SECUKINUMAB VERSUS ADALIMUMAB HEAD-TO-HEAD COMPARISON IN BIOLOGIC-NAÏVE PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS THROUGH 52-WEEKS (EXCEED): A RANDOMISED, DOUBLE-BLIND, PHASE-3B STUDY

I. Mcinnes[1], F. Behrens[2], P. J. Mease[3], A. Kavanaugh[4], C. T. Ritchlin[5], P. Nash[6], J. Gratacos-Masmitja[7], P. Goupille[8], T. Korotaeva[9], A. B. Gottlieb[10], R. Martin[11], K. Ding[11], P. Pellet[12], S. Mpofu[12], L. Pricop[11]. *[1]University of Glasgow, Glasgow, United Kingdom; [2]Rheumatology University Hospital, Frankfurt, Germany; [3]Swedish Medical Centre, Seattle, United States of America; [4]UCSD, La Jolla, United States of America; [5]University of Rochester, Rochester, United States of America; [6]Griffith University, Brisbane, Australia; [7]University Hospital Parc Taulí, Sabadell, UAB, Spain; [8]University of Tours, Tours, France; [9]Research Institute of Rheumatology n.a. V.A. Nasonova, Moscow, Russian Federation; [10]Icahn School of Medicine at Mount Sinai, New York, United States of America; [11]Novartis Pharmaceuticals Corporation, East Hanover, United States of America; [12]Novartis Pharma AG, Basel, Switzerland*

Background: Secukinumab (SEC), an interleukin-17A inhibitor, has demonstrated improvements on multiple domains of psoriatic arthritis (PsA).[1] Adalimumab (ADA), a TNF inhibitor, is widely used as a first–line biologic in PsA.

Objectives: To report efficacy and safety outcomes from the head-to-head EXCEED trial (NCT02745080) that compares SEC *vs.* ADA as first–line biologic monotherapy through 52-weeks (wks), with a musculoskeletal primary endpoint in pts with active PsA.

Methods: Head-to-head, phase-3b, randomised, double-blind trial: biologic naïve active PsA pts were randomised to receive SEC 300mg subcutaneous at baseline, Wk1-4, and then every 4wks (q4w) until Wk48 or ADA 40mg subcutaneous at baseline and then q2w until Wk50. The primary endpoint was superiority of SEC *vs.* ADA on ACR20 response at Wk52. Binary and continuous variables were analysed using logistic-regression model and MMRM, respectively. Safety analysis included patients who received ≥1 dose of study-drug.

Results: 853 pts were randomised to receive SEC (n=426) or ADA (n=427). Baseline demographics and disease characteristics were comparable between treatment-groups except higher proportion of female pts and pts without enthesitis in the SEC group. ACR20 response at Wk52 for SEC *vs.* ADA were 67·4% *vs.* 61·5%, respectively (p=0·0719) (Figure). Higher clinical responses were observed with SEC *vs.* ADA for a range of musculoskeletal, skin, and higher-hurdle outcomes (Table). A higher retention rate was observed for SEC (85.7%) *vs.* ADA (76.3%). Safety profiles of SEC and ADA were consistent with previous reports.[2,3]

Conclusion: Results suggest that SEC is at least as efficacious as ADA on musculoskeletal endpoints whilst providing higher responses on skin endpoints, and is associated with a higher retention rate. No new safety signals were reported.

References:

[1] van der Heijde, et al. Rheumatol. (Oxford).2019; DOI10.1093/rheumatology/kez420.

[2] Deodhar A, et al. Arthritis Res Ther. 2019;21:111.

[3] Burmester GR, et al. Ann Rheum Dis.2013; 72:517-24.

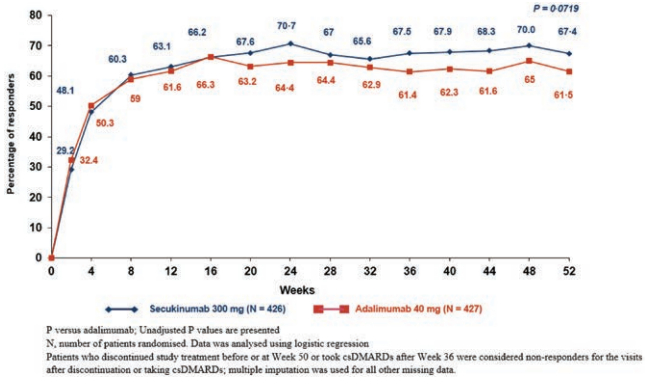


Figure. ACR20 Response through Wk 52

Table. Efficacy Outcomes at Wk 52

Endpoints, % response unless specified otherwise	SEC 300mg (N=426)	ADA 40mg (N=427)	P-value (unadjusted)*
ACR20	67·4	61·5	0·0719
[a]ACR20	66·9	59·5	0·0239
Key Secondary			
[b]PASI 90	65·4	43·2	<0·0001
ACR50	49·0	44·8	0·2251
HAQ-DI mean change from baseline ± SE	-0·58 ± 0.03	-0·56 ± 0.03	0·5465
[c]Resolution of enthesitis (based on LEI)	60·5	54·2	0·1498
Exploratory			
MDA	43·0	37·9	0·1498
VLDA	18·1	16·6	0·6107
DAPSA LDA+Remission	61·7	53·1	0·0178
PASDAS LDA+Remission	51·1	44·1	0·0557

*Unadjusted P-values *vs* ADA

Binary variables were analysed using logistic regression. Pts who discontinued study treatment prematurely or took csDMARDs after week-36 were considered non-responders. Multiple imputation was used for all other missing data. HAQ-DI mean change from baseline was analysed using mixed-effect model repeated measures

[a]Non-responder imputation was used for pre-specified sensitivity analysis

[b]N=215 in SEC and N=202 in ADA in psoriasis subset

[c]N=234 in SEC and N=264 in ADA in enthesitis subset