Bimekizumab Treatment in Psoriatic Arthritis Resulted in Sustained Efficacy Assessed Using Composite Outcome Measures to 3 Years

Hideto Kameda,[1] M. Elaine Husni,[2] Ana-Maria Orbai,[3] David Nicholls,[4] Barbara Ink,[5] Rajan Bajracharya,[5] Jason Coarse,[6] Laura C. Coates[7]

[1]Department of Internal Medicine, Toho University, Tokyo, Japan; [2]Department of Rheumatic and Immunologic Diseases, Cleveland Clinic, Cleveland, Ohio, USA; [3]Division of Rheumatology, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA; [4]Clinical Trials Unit, University of the Sunshine Coast, Queensland, Australia; [5]UCB, Slough, UK; [6]UCB, Morrisville, North Carolina, USA; [7]Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Diseases, University of Oxford and Oxford Biomedical Research Centre, Oxford University Hospitals NHS Trust, Oxford, UK.

Presenting on behalf of the authors: Anna Parievsky

Objective

To report the 3-year treatment efficacy of bimekizumab (BKZ), a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, using composite outcome measures in patients with psoriatic arthritis (PsA) who were biologic disease-modifying antirheumatic drug (bDMARD)-naïve or had inadequate response or intolerance to prior tumor necrosis factor inhibitors (TNFi-IR).

Background

- PsA is a chronic, heterogenous inflammatory disease characterized by manifestations across multiple domains, including joints and skin.
- Evaluating the long-term efficacy of treatment with BKZ, using composite outcome measures, is important for a comprehensive assessment of disease activity.[1]

Methods

- The phase 3 BE OPTIMAL (bDMARD-naïve; NCT03895203) and BE COMPLETE (TNFi-IR; NCT03896581) studies assessed subcutaneous BKZ 160 mg every 4 weeks in patients with PsA. Both were double-blind and placebo (PBO)-controlled to Week 16; BE OPTIMAL included a reference arm (adalimumab 40 mg every 2 weeks; data not shown).
- Patients who completed BE OPTIMAL/BE COMPLETE (Week 52/16) could enter BE VITAL (open-label extension; NCT04009499), in which all patients received BKZ.[2]
- Composite disease activity outcome measures are reported to Year 3 (Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE), in patients randomized to PBO or BKZ at baseline and the BKZ Total group (BKZ-randomized patients and PBO patients who switched to BKZ at Week 16).
- Measures include minimal/very low disease activity (MDA/VLDA), composite of ≥50% improvement in ACR criteria plus Psoriasis Area and Severity Index 100% improvement from baseline (ACR50+PASI100), Disease Activity Index for PsA (DAPSA) and PsA Disease Activity Score (PASDAS).
- Results are reported using observed case, or by modified non-responder imputation (mNRI; binary), multiple imputation (MI; continuous) or worst-category imputation (WCI; categorical). mNRI considered all visits following discontinuation due to adverse events or lack of efficacy as non-response; all other missing data were imputed with MI and the response derived from the imputed values.

Results

- Overall, 546/712 (76.7%) bDMARD-naïve and 299/400 (74.8%) TNFi-IR patients in the BKZ Total group completed up to 3 years (Week 160/156) of treatment.
- Patient demographics and disease characteristics were representative of patients with active PsA (**Table 1**).
- Across all composite outcome measures, treatment responses observed at 1 year were sustained up to 3 years (**Figure 1–4**; **Table 2**).
- Furthermore, sustained responses were observed for the individual MDA components, including clinical measures and patient-reported outcomes (**Figure 2**).

Conclusions

Treatment with bimekizumab resulted in sustained, broad efficacy up to 3 years, as assessed by achievement of stringent, clinically meaningful composite outcomes that evaluate disease activity across multiple domains of PsA. Improvements were consistent and sustained in both bDMARD-naïve and TNFi-IR patients.

Summary

Composite outcome measures assess **treatment efficacy** across multiple domains of **PsA**.

The efficacy of **bimekizumab** treatment was assessed using **composite outcomes**, including **MDA and DAPSA** up to 3 years in patients with **active PsA** who were **bDMARD-naïve** or had **TNFi-IR**.

MDA achievement[a,b]
48.8%–52.9%
bDMARD-naïve patients in BE OPTIMAL and TNFi-IR patients in BE COMPLETE at 3 years[c]

DAPSA REM+LDA achievement[a,d]
67.3%–71.5%
bDMARD-naïve patients in BE OPTIMAL and TNFi-IR patients in BE COMPLETE at 3 years[c]

Bimekizumab treatment demonstrated **long-term sustained efficacy across multiple domains of PsA** up to 3 years in patients with PsA, which was **consistent** across patients who were bDMARD-naïve or had TNFi-IR.

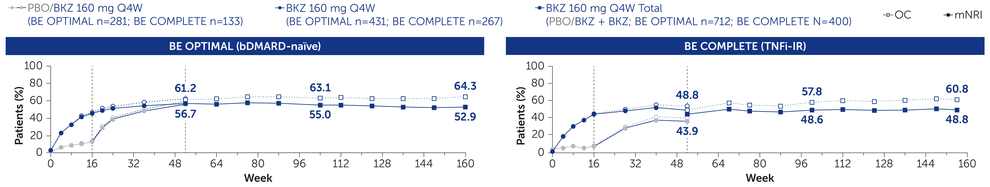
[a] Values reported here for patients in the BKZ Total group; **[b]** Values shown here were imputed using mNRI; **[c]** Data reported to Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE; **[d]** Values shown here were imputed using MI.

Table 1 Select baseline demographics and disease characteristics

	BE OPTIMAL (bDMARD-naïve)	BE COMPLETE (TNFi-IR)
	BKZ 160 mg Q4W Total (n=712)	BKZ 160 mg Q4W Total (N=400)
Age, years, mean (SD)	48.6 (12.2)	50.5 (12.5)
Sex, male, n (%)	328 (46.1)	190 (47.5)
Time since PsA diagnosis, years, mean (SD)	5.8 (7.0)[a]	9.5 (9.3)[b]
Any csDMARD at baseline, n (%)	495 (69.5)	202 (50.5)
Concomitant methotrexate, n (%)	415 (58.3)	170 (42.5)
SJC (of 66 joints), mean (SD)	9.2 (6.6)	9.9 (7.7)
TJC (of 68 joints), mean (SD)	16.9 (12.1)	18.7 (13.8)
hs-CRP, mg/L, mean (SD)	9.8 (17.2)	12.1 (18.0)
≥3% BSA affected by psoriasis, n (%)	357 (50.1)	264 (66.0)
PASI score,[c] mean (SD)	8.1 (6.4)	9.6 (8.4)
Enthesitis (LEI >0), n (%)	213 (29.9)[d]	142 (35.5)[e]
LEI score,[f] mean (SD)	2.6 (1.5)	2.7 (1.5)
Dactylitis (LDI >0), n (%)	89 (12.5)[g]	48 (12.0)[e]
LDI score,[h] mean (SD)	47.0 (49.6)	70.9 (117.0)
PhGA-PsA, mean (SD)	57.3 (15.8)[d]	58.7 (17.8)[e]
PGA-PsA, mean (SD)	56.1 (23.5)[e]	61.4 (22.3)
HAQ-DI score, mean (SD)	0.85 (0.59)[e]	0.99 (0.62)
FACIT-Fatigue, mean (SD)	37.1 (9.9)[e]	35.6 (10.3)
PsAID-12 total score, mean (SD)	4.0 (1.9)[e]	4.5 (2.0)
Pain VAS,[i] mean (SD)	54.9 (23.9)[e]	59.5 (24.3)

Randomized set. **[a]** Data missing for 10 patients; **[b]** Data missing for 2 patients; **[c]** In patients with psoriasis involving ≥3% BSA at baseline (BE OPTIMAL n=357; BE COMPLETE n=264); **[d]** Data missing for 6 patients; **[e]** Data missing for 1 patient; **[f]** In patients with enthesitis at baseline (LEI >0); **[g]** Data missing for 7 patients; **[h]** In patients with dactylitis at baseline (LDI >0); **[i]** Pain VAS was assessed using Patient's Assessment of Arthritis Pain VAS, which ranges from 0 (no pain) to 100 (most severe pain).

Figure 1 Proportion of patients achieving MDA up to 3 years (mNRI, OC)

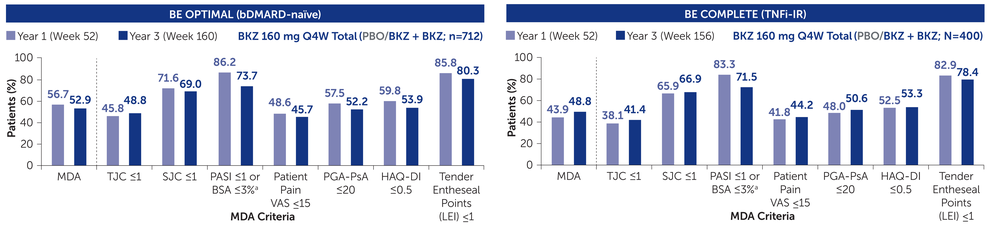


OC, n/N:	Year 1	Year 2	Year 3
PBO/BKZ	151/253		
BKZ	236/379		
BKZ Total	387/632	366/580	338/526

OC, n/N:	Year 1	Year 2	Year 3
PBO/BKZ	44/112		
BKZ	124/232		
BKZ Total	168/344	182/315	178/293

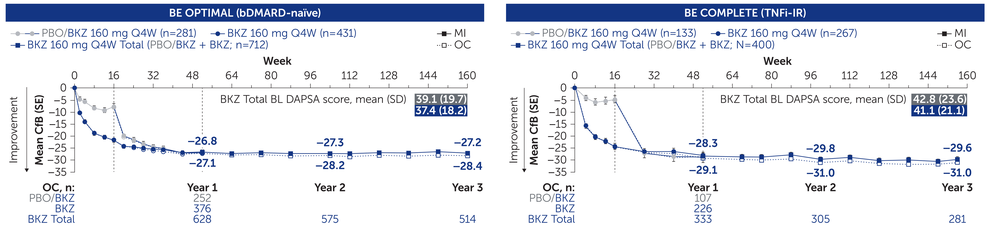
Randomized set. Data reported to Year 1 (Week 52 in BE OPTIMAL/BE COMPLETE), Year 2 (Week 104/Week 100 in BE OPTIMAL/BE COMPLETE) and to Year 3 (Week 160/Week 156 in BE OPTIMAL/BE COMPLETE). MDA defined as achievement of ≥5/7 of the following outcomes: TJC ≤1, SJC ≤1, PASI ≤1 or BSA ≤3, patient pain VAS ≤15, PGA-PsA VAS ≤20, HAQ-DI ≤0.5, tender entheseal points (LEI) ≤1.

Figure 2 MDA component responses at Years 1 and 3 (mNRI)



Randomized set. Data reported at Year 1 (Week 52 in BE OPTIMAL/BE COMPLETE) and Year 3 (Week 160/Week 156 in BE OPTIMAL/BE COMPLETE). **[a]** In cases where MI did not converge and mNRI was not available, missing data were imputed using NRI.

Figure 4 DAPSA score change from baseline up to 3 years (MI, OC)

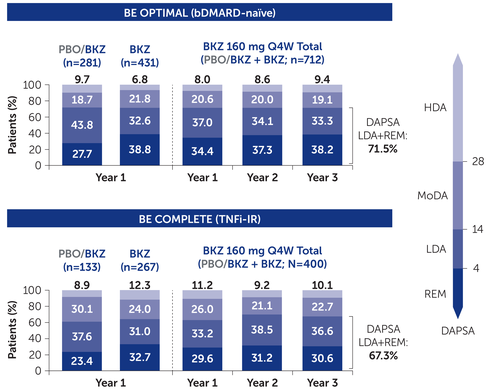


OC, n:	Year 1	Year 2	Year 3
PBO/BKZ	252		
BKZ	376		
BKZ Total	628	575	514

OC, n:	Year 1	Year 2	Year 3
PBO/BKZ	107		
BKZ	226		
BKZ Total	333	305	281

Randomized set. Data reported to Year 1 (Week 52 in BE OPTIMAL/BE COMPLETE). Year 2 (Week 104/100 in BE OPTIMAL/BE COMPLETE) and Year 3 (Week 160/Week 156 in BE OPTIMAL/BE COMPLETE), unless otherwise stated. Note that error bars are present for all reported timepoints (MI), however may be obscured by the data point.

Figure 3 Achievement of DAPSA disease state thresholds up to 3 years (MI)



Randomized set. Data reported to Year 1 (Week 52 in BE OPTIMAL/BE COMPLETE), Year 2 (Week 104/100 in BE OPTIMAL/BE COMPLETE) and Year 3 (Week 160/Week 156 in BE OPTIMAL/BE COMPLETE), unless otherwise stated. DAPSA REM defined as a DAPSA score of ≤4. LDA defined as >4 and ≤14. LDA+REM defined as ≤14. MoDA defined as >14 and ≤28. HDA defined as >28. Percentages may not sum to 100 due to rounding.

Table 2 Achievement of additional composite endpoints at Years 1 and 3 (MI, mNRI, WCI)

	BE OPTIMAL (bDMARD-naïve)		BE COMPLETE (TNFi-IR)	
	BKZ 160 mg Q4W Total (n=712)		BKZ 160 mg Q4W Total (N=400)	
	Year 1	Year 3	Year 1	Year 3
VLDA [mNRI],[a] %	27.1	30.0	20.8	23.6
ACR50+PASI100 [mNRI],[b] %	48.2	44.1	43.5	48.3
DAPSA disease state [WCI],[c] n (%)				
LDA+REM	464 (65.2)	401 (56.3)	225 (56.3)	215 (53.8)
REM	231 (32.4)	239 (33.6)	113 (28.3)	106 (26.5)
PASDAS disease state [MI],[d] %				
LDA+REM	69.7	68.9	57.8	64.4
REM	39.8	41.9	31.4	38.1
PASDAS disease state [WCI],[d] n (%)				
LDA+REM	365 (51.3)	392 (55.1)	227 (56.8)	206 (51.5)
REM	192 (27.0)	240 (33.7)	117 (29.3)	124 (31.0)

Randomized set. Data reported at Year 1 (Week 52 in BE OPTIMAL/BE COMPLETE) and Year 3 (Week 160/Week 156 in BE OPTIMAL/BE COMPLETE), unless otherwise stated. **[a]** VLDA defined as achievement of 7/7 of the following outcomes: TJC ≤1, SJC ≤1, PASI ≤1 or BSA ≤3, patient pain VAS ≤15, PGA-PsA VAS ≤20, HAQ-DI ≤0.5, tender entheseal points (LEI) ≤1. **[b]** In patients with psoriasis involving ≥3% BSA at baseline (BE OPTIMAL: n=357; BE COMPLETE: n=264); **[c]** DAPSA REM defined as a DAPSA score of ≤4. LDA+REM defined as ≤14; **[d]** PASDAS reported at Year 1 (Week 52/40 in BE OPTIMAL/BE COMPLETE) and Year 3 (Week 148/Week 156 in BE OPTIMAL/BE COMPLETE). PASDAS REM defined as a PASDAS score of ≤1.9; LDA+REM defined as ≤3.2.

ACR50: ≥50% improvement from baseline in American College of Rheumatology response criteria; **bDMARD:** biologic disease-modifying antirheumatic drug; **BKZ:** bimekizumab; **BL:** baseline; **BSA:** body surface area; **CfB:** change from baseline; **DAPSA:** Disease Activity Index for Psoriatic Arthritis; **HAQ-DI:** Health Assessment Questionnaire-Disability Index; **HDA:** high disease activity; **hs-CRP:** high sensitivity C-reactive protein; **IL:** interleukin; **LDA:** low disease activity; **LEI:** Leeds Enthesitis Index; **MDA:** Minimal Disease Activity; **MI:** multiple imputation; **mNRI:** modified non-responder imputation; **MoDA:** moderate disease activity; **NRI:** non-responder imputation; **OC:** observed case; **PASDAS:** Psoriatic Arthritis Disease Activity Score; **PASI:** Psoriasis Area and Severity Index; **PASI100:** 100% improvement from baseline in Psoriasis Area and Severity Index; **PBO:** placebo; **PGA-PsA:** Patient's Global Assessment of Psoriatic Arthritis; **PhGA-PsA:** Physician's Global Assessment of Psoriatic Arthritis; **PsA:** psoriatic arthritis; **Q4W:** every four weeks; **REM:** remission; **SD:** standard deviation; **SE:** standard error; **SJC:** swollen joint count; **TJC:** tender joint count; **TNFi-IR:** inadequate response or intolerance to prior tumor necrosis factor inhibitors; **VAS:** visual analog scale; **VLDA:** very low disease activity; **WCI:** worst category imputation.

References: [1]Coates LC. Rheum Dis Clin North Am 2015;41(4):699–710; [2]Mease PJ. Rheumatol Ther 2024;11:1363–82. **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **HK**, **MEH**, **AMO**, **DN**, **BI**, **RB**, **JC**, **LCC**. Drafting of the publication, or reviewing it critically for important intellectual content: **HK**, **MEH**, **AMO**, **DN**, **BI**, **RB**, **JC**, **LCC**. Final approval of the publication: **HK**, **MEH**, **AMO**, **DN**, **BI**, **RB**, **JC**, **LCC**. **Disclosures: HK:** Grants from Asahi-Kasei, Pfizer, and Taisho; advisory/consultant for AbbVie, Amgen, Asahi-Kasei, Eli Lilly, Janssen, Novartis, Pfizer, Sanofi, Taisho and UCB; speaking fees from AbbVie, Amgen, Asahi-Kasei, BMS, Eisai, Eli Lilly, Janssen, Mitsubishi-Tanabe and UCB. **MEH:** Advisory board member and consultant for AbbVie, Amgen, BMS, Eli Lilly, Janssen, Novartis, Pfizer and UCB. **AMO:** Research grants to Johns Hopkins University from AbbVie, Amgen and Janssen; consulting fees from BMS, Janssen, Sanofi and UCB. **DN:** Research support from AbbVie, BMS, Eli Lilly, Gilead, Incannex, Janssen, Novartis, Pfizer, Servatus, Sun Pharma and UCB; Advisory board or speaker fees: AbbVie, AstraZeneca, Novartis, Janssen, Sandoz, Pfizer and UCB. **BI:** Employee of UCB; shareholder of AbbVie, GSK and UCB. **RB, JC:** Employee and shareholder of UCB. **LCC:** Grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Gilead, Janssen, Novartis, Pfizer and UCB; consultant for AbbVie, Amgen, BMS, Boehringer Ingelheim, Celgene, Domain, Eli Lilly, Galapagos, Gilead, Janssen, Moonlake Immunotherapeutics, Novartis, Pfizer and UCB; speaking fees from AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GSK, Janssen, medac, Novartis, Pfizer and UCB. **AP:** Employee of UCB. **Acknowledgments:** We would like to thank the patients and their caregivers in addition to all the investigators and their teams who contributed to this study. The authors acknowledge Heather Edens, PhD, UCB, Smyrna, Georgia, USA, for publication coordination, Alice Di Vincenzo, Costello Medical, Manchester, UK for medical writing and editorial assistance, and the Costello Medical Creative team for design support. Funded by UCB. All costs associated with development of this presentation were funded by UCB.

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