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

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Objective

To report the 3-year treatment efficacy of bimekizumab (BKZ) 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, inflammatory disease characterized by heterogeneous joint and skin manifestations; it is important to assess long-term treatment efficacy using composite measures across disease domains.[1,2]
- BKZ, a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated sustained efficacy in PsA up to 3 years.[3]

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 Week 52 or BE COMPLETE Week 16 could enter BE VITAL (open-label extension; NCT04009499), in which all patients received BKZ.
- Composite disease activity outcome measures are reported to Year 3 (Week 148/160 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 from baseline in American College of Rheumatology response criteria and 100% improvement from baseline in Psoriasis Area and Severity Index (ACR50+PASI100)
  - Disease Activity Index for PsA (DAPSA)
  - Composite of swollen joint count resolution and PASI100 (SJC=0+PASI100)
  - PsA Disease Activity Score (PASDAS)
- Results are reported as observed or using 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 (**Figures 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 in patients with PsA up to 3 years, assessed by stringent, clinically meaningful composite outcomes which evaluate disease activity across multiple domains. Responses were consistent and sustained in both bDMARD-naïve and TNFi-IR patients.

Summary

Composite outcome measures assess **treatment efficacy** across multiple domains of **PsA**, including pain, joints, skin, physical function, and enthesitis.

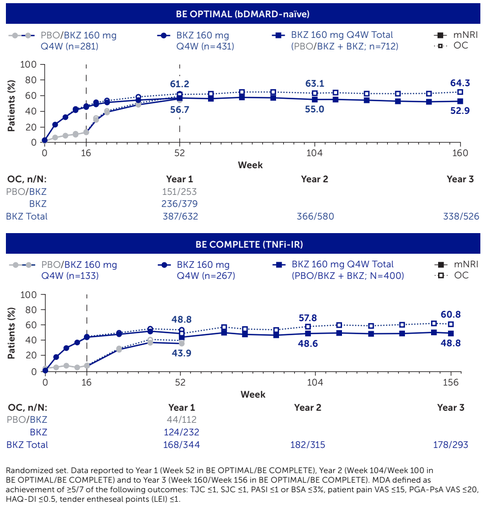
Patients with **active PsA** treated with **bimekizumab** who were **bDMARD-naïve (BE OPTIMAL)** or had **TNFi-IR (BE COMPLETE)** achieved the following **composite outcomes** up to **3 years**:[a,b]

- **MDA**[c]: **52.9%** of bDMARD-naïve patients; **48.8%** of TNFi-IR patients
- **DAPSA REM+LDA**[d]: **53.8%** of bDMARD-naïve patients; **56.3%** of TNFi-IR patients
- **SJC=0+PASI100**[c,e]: **46.6%** of bDMARD-naïve patients; **54.0%** of TNFi-IR patients

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] Data reported to Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE; **[b]** Values reported here for patients in the BKZ Total group; **[c]** Values shown here were imputed using mNRI; **[d]** Values shown here were imputed using WCI; **[e]** In patients with psoriasis involving ≥3% BSA at baseline. SJC was assessed in 66 joints.

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



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.

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)[e]	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)

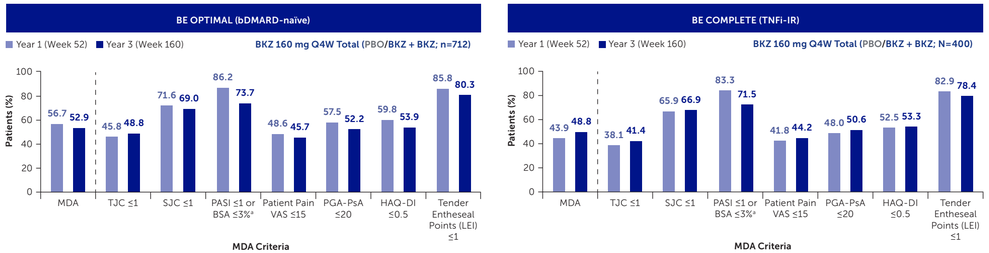
Randomized set. **[a]** Data missing for 10 patients; **[b]** Data missing for 2 patients; **[c]** In patients with psoriasis involving ≥3% BSA at baseline; **[d]** Data missing for 6 patients; **[e]** Data missing for 1 patient; **[f]** In patients with enthesitis at baseline (LEI >0); **[g]** Data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

Table 2 Additional composite efficacy outcomes at Years 1 and 3 (mNRI, WCI)

	BE OPTIMAL (bDMARD-naïve)		BE COMPLETE (TNFi-IR)	
	BKZ 160 mg Q4W Total[a] (n=712)		BKZ 160 mg Q4W Total[a] (N=400)	
	Year 1	Year 3	Year 1	Year 3
VLDA [mNRI],[b] %	27.1	30.0	20.8	23.6
ACR50+PASI100 [mNRI],[c] %	48.2	44.1	43.5	48.3
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)

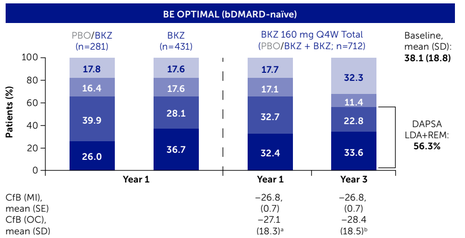
Randomized set. Data reported at Year 1 (Week 52 in BE OPTIMAL/BE COMPLETE) and Year 3 (Week 160/Week 156 in BE OPTIMAL/BE COMPLETE), unless otherwise stated. **[a]** BKZ Total includes BKZ-randomized patients and PBO patients who switched to BKZ at Week 16; **[b]** VLDA defined as achievement of 7/7 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; **[c]** In patients with psoriasis involving ≥3% BSA at baseline (BE OPTIMAL: n=357; BE COMPLETE: n=264); **[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.

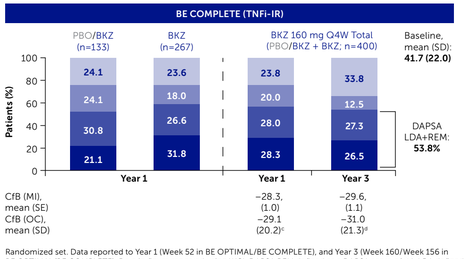
Figure 2 MDA component responses at Years 1 and 3 (mNRI)



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.

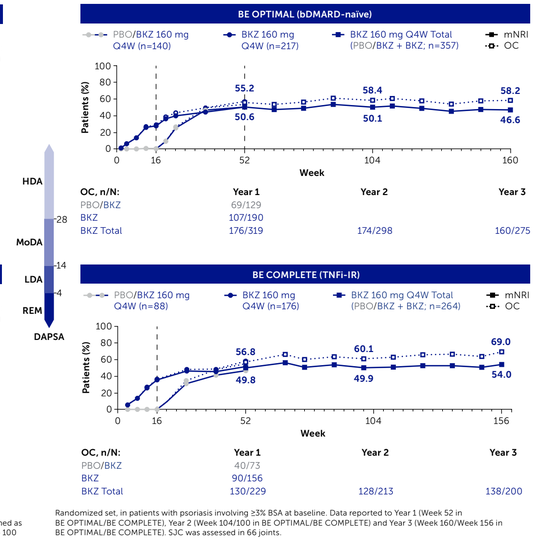
Figure 3 Achievement of DAPSA disease state thresholds up to 3 years (MI, OC, WCI)





Randomized set. Data reported to Year 1 (Week 52 in BE OPTIMAL/BE COMPLETE), and Year 3 (Week 160/Week 156 in BE OPTIMAL/BE COMPLETE). Data in figure reported using WCI. 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. **[a]** n=628; **[b]** n=514; **[c]** n=333, **[d]** n=281.

Figure 4 Proportion of patients achieving SJC=0+PASI100 up to 3 years (mNRI, OC)



Randomized set, in patients with psoriasis involving ≥3% BSA at baseline. 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). SJC was assessed in 66 joints.

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 tumour necrosis factor inhibitors; **VAS:** visual analogue scale; **VLDA:** very low disease activity; **WCI:** worst category imputation.

References: [1]Coates LC. Rheum Dis Clin North Am 2015;41:699–710; [2]Veale DJ. Lancet 2018;391:2273–84; [3]Gossec L. Rheumatology (Oxford) 2026:keag118. **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **AMO**, **HK**, **MEH**, **DN**, **BI**, **RB**, **JC**, **LCC**; Drafting of the publication, or reviewing it critically for important intellectual content: **AMO**, **HK**, **MEH**, **DN**, **BI**, **RB**, **JC**, **LCC**; Final approval of the publication: **AMO**, **HK**, **MEH**, **DN**, **BI**, **RB**, **JC**, **LCC**. **Author Disclosures: AMO:** Research grants to Johns Hopkins University from AbbVie, Amgen, and Janssen; consulting fees from BMS, Janssen, Sanofi, and UCB. **HK:** Grants from Asahi-Kasei, Pfizer, and Taisho; advisory/consultant for AbbVie, Amgen, Asahi-Kasei, Eli Lilly and Company, Janssen, Novartis, Pfizer, Sanofi, Taisho, and UCB; speaking fees from AbbVie, Amgen, Asahi-Kasei, BMS, Eisai, Eli Lilly and Company, Janssen, Mitsubishi-Tanabe, and UCB. **MEH:** Advisory board member and consultant for AbbVie, Amgen, BMS, Eli Lilly and Company, Janssen, Novartis, Pfizer, and UCB. **DN:** Research support from AbbVie, BMS, Eli Lilly and Company, 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 and Company, Gilead, Janssen, Novartis, Pfizer, and UCB; consultant for AbbVie, Amgen, BMS, Boehringer Ingelheim, Celgene, Domain, Eli Lilly and Company, Galapagos, Gilead, Janssen, MoonLake Immunotherapeutics, Novartis, Pfizer, and UCB; speaking fees from AbbVie, Amgen, Biogen, Celgene, Eli Lilly and Company, Galapagos, Gilead, GSK, Janssen, medac, Novartis, Pfizer, and 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 Lyes Derouiche, PhD, CMPP™, UCB, Brussels, Belgium, for publication coordination, Lilit Ghazaryan, Costello Medical, London, 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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