Bimekizumab improved outcomes in disease core domains in patients with active psoriatic arthritis and psoriasis: Pooled 16-week results from the BE OPTIMAL and BE COMPLETE phase 3 randomised, placebo-controlled studies

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Objective

To assess bimekizumab efficacy vs placebo across psoriatic arthritis core domains in patients with psoriatic arthritis and psoriasis affecting ≥3% body surface area, using pooled data from the phase 3 BE OPTIMAL and BE COMPLETE trials.

Background

- Psoriatic arthritis (PsA) is a disease with multiple manifestations, including skin, joints, nail psoriasis, dactylitis and enthesitis. Up to 30% of patients with psoriasis go on to develop PsA. Therefore, assessing the efficacy of new treatment options for PsA in patients with baseline psoriasis is essential.[1,2]
- Bimekizumab (BKZ) is a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A.[3,4]
- BKZ has demonstrated efficacy and was well tolerated to 16 weeks in patients with PsA who were biologic disease-modifying antirheumatic drug (bDMARD)-naïve or who had inadequate response or intolerance to tumour necrosis factor-α inhibitors (TNFi-IR).[3,4]

Methods

- BE OPTIMAL (NCT03895203) and BE COMPLETE (NCT03896581) assessed the efficacy and safety of treatment with subcutaneous BKZ 160 mg every 4 weeks in patients with PsA who were bDMARD-naïve or TNFi-IR, respectively.[3,4]
- Each study included a 16-week double-blind, placebo (PBO)-controlled phase (**Figure 1**). BE OPTIMAL included an adalimumab reference arm; data not shown here.
- Here, data from BE OPTIMAL and BE COMPLETE were pooled for patients randomised to receive BKZ or PBO and who had psoriasis affecting ≥3% body surface area (BSA) at baseline.
- Efficacy outcomes are reported to Week 16, spanning core PsA domains including joints, skin, nail psoriasis, dactylitis and enthesitis. Missing data were imputed using non-responder imputation (NRI).

Results

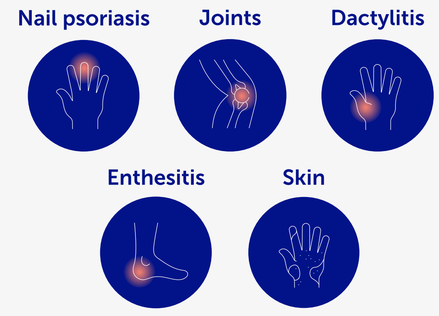
- 621/1,112 (55.8%) patients had baseline psoriasis ≥3% BSA (357 bDMARD-naïve [217 BKZ; 140 PBO]; 264 TNFi-IR [176 BKZ; 88 PBO]). Baseline characteristics for this subgroup are reported in **Table 1**.
- At Week 16, greater proportions of BKZ-treated patients vs PBO achieved:
  - American College of Rheumatology (ACR) 50: BKZ 47.6% vs PBO 6.1%, and ACR70: 30.8% vs 2.2% (**Figure 2**);
  - Psoriasis Area and Severity Index (PASI) 100 (complete skin clearance): BKZ 52.4% vs PBO 3.1% (**Figure 3**);
  - Resolution of nail psoriasis (Modified Nail Psoriasis Severity Index [mNAPSI]=0): BKZ 38.2% vs PBO 12.0% (**Figure 4**);
  - Resolution of dactylitis (Leeds Dactylitis Index [LDI]=0): BKZ 87.5% vs PBO 70.6% (**Figure 5**);
  - Resolution of enthesitis (Leeds Enthesitis Index [LEI]=0): BKZ 58.9% vs 44.4% (**Figure 5**).

Conclusions

Bimekizumab treatment demonstrated improvements across PsA core domains in patients with PsA and baseline psoriasis, compared with PBO. Results were consistent, with similar levels of response, in patients who were bDMARD-naïve or TNFi-IR, suggesting that bimekizumab treatment leads to improvements in disease activity irrespective of prior bDMARD use.

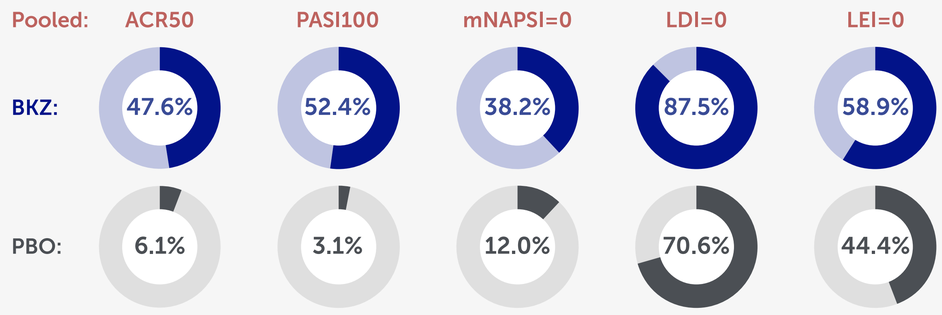
Summary

55.8% (621/1,112) of patients had **baseline psoriasis** affecting ≥3% body surface area.



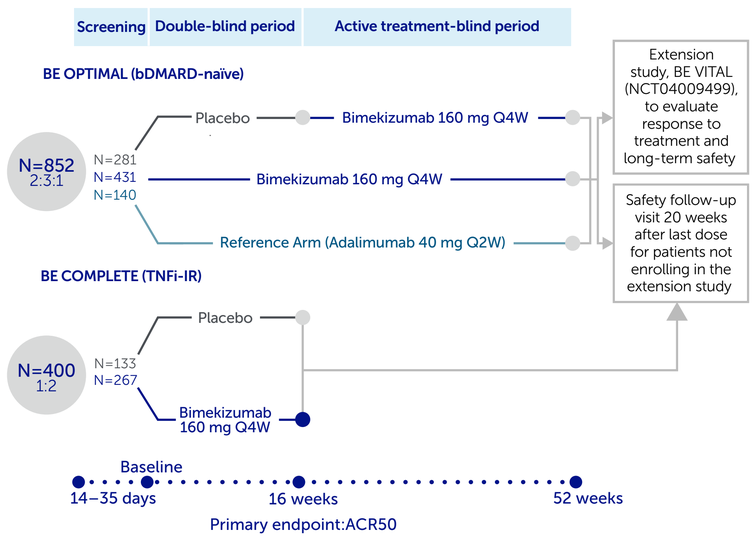
It is important to assess the efficacy of new treatment options across the **core domains of PsA** in patients with concomitant psoriasis.

Numerically higher proportions of **bimekizumab-treated patients** achieved **clinically relevant improvements across the core PsA domains** at Week 16, irrespective of prior bDMARD use.



Results suggest **bimekizumab treatment** led to **improvements across the core PsA domains** in patients with PsA and baseline psoriasis. Similar magnitude of response was observed in bDMARD-naïve and TNFi-IR patients.

Figure 1 BE OPTIMAL and BE COMPLETE study designs



Study designs that made up the pooled population, BE OPTIMAL (NCT03895203) and BE COMPLETE (NCT03896581). Adalimumab 40 mg Q2W served as an active reference arm. BE OPTIMAL was not powered for comparisons of adalimumab to bimekizumab or adalimumab to placebo. Adalimumab 40 mg Q2W data are not shown in this poster.

Table 1 Baseline characteristics in patients with baseline psoriasis (≥3% BSA)

	Pooled Analysis (bDMARD-naïve + TNFi-IR)		BE OPTIMAL (bDMARD-naïve)		BE COMPLETE (TNFi-IR)	
	PBO n=228	BKZ 160 mg Q4W n=393	PBO n=140	BKZ 160 mg Q4W n=217	PBO n=88	BKZ 160 mg Q4W n=176
Age, years, mean (SD)	48.7 (12.1)	47.7 (12.3)	48.0 (11.4)	46.7 (12.2)	49.8 (13.1)	48.9 (12.3)
Male, n (%)	103 (45.2)	199 (50.6)	63 (45.0)	110 (50.7)	40 (45.5)	89 (50.6)
BMI, kg/m², mean (SD)	29.1 (5.5)	30.0 (6.8)	29.4 (5.6)	30.1 (7.1)	28.7 (5.5)	29.9 (6.5)
BSA affected by PSO >10%, n (%)	73 (32.0)	140 (35.6)	48 (34.3)	73 (33.6)	25 (28.4)	67 (38.1)
Previous TNFi, n (%)						
Inadequate response to one TNFi	67 (29.4)	142 (36.1)	0	0	67 (76.1)	142 (80.7)
Inadequate response to two TNFi	11 (4.8)	12 (3.1)	0	0	11 (12.5)	12 (6.8)
Intolerance to TNFi	10 (4.4)	22 (5.6)	0	0	10 (11.4)	22 (12.5)
MTX at baseline, n (%)	118 (51.8)	206 (52.4)	83 (59.3)	126 (58.1)	35 (39.8)	80 (45.5)
PASI, mean (SD)	8.1 (6.0)	9.1 (8.0)	7.9 (5.6)	8.2 (6.8)	8.5 (6.6)	10.2 (9.1)
HAQ-DI, mean (SD)	1.02 (0.65)	0.93 (0.58)[a]	0.93 (0.63)	0.87 (0.59)[b]	1.15 (0.67)	1.01 (0.55)
hs-CRP ≥6 mg/L, n (%)	113 (49.6)	181 (46.1)	67 (47.9)	98 (45.2)	46 (52.3)	83 (47.2)
PtAAP, mean (SD)	62.6 (22.0)	57.0 (24.6)[a]	60.4 (21.7)	55.5 (24.3)[b]	66.1 (22.0)	59.0 (24.9)

[a]n=392; [b]n=216.

Pooled Analysis (bDMARD-naïve + TNFi-IR) | **BE OPTIMAL** (bDMARD-naïve) | **BE COMPLETE** (TNFi-IR)

Figure 2 Proportion of patients with baseline psorasis (≥3% BSA) achieving ACR50 and ACR70 to Week 16 (NRI)

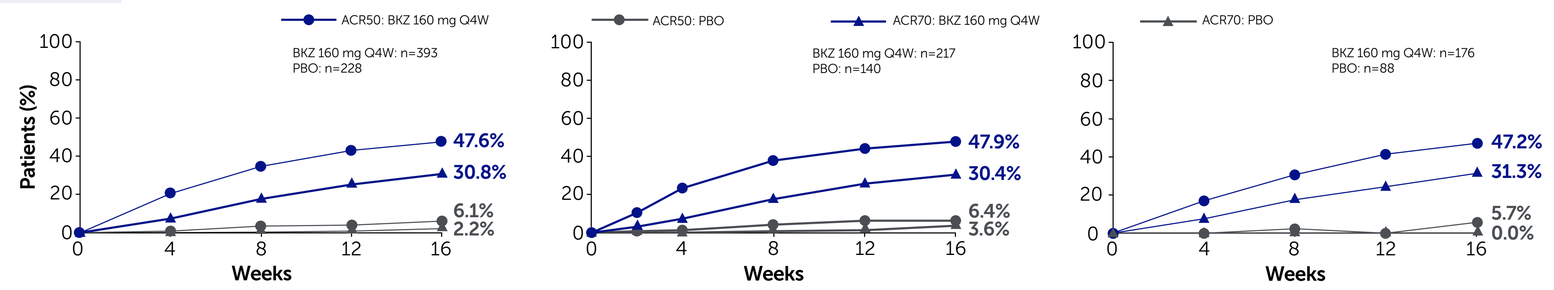


Figure 3 Proportion of patients with baseline psoriasis (≥3% BSA) achieving PASI100 to Week 16 (NRI)

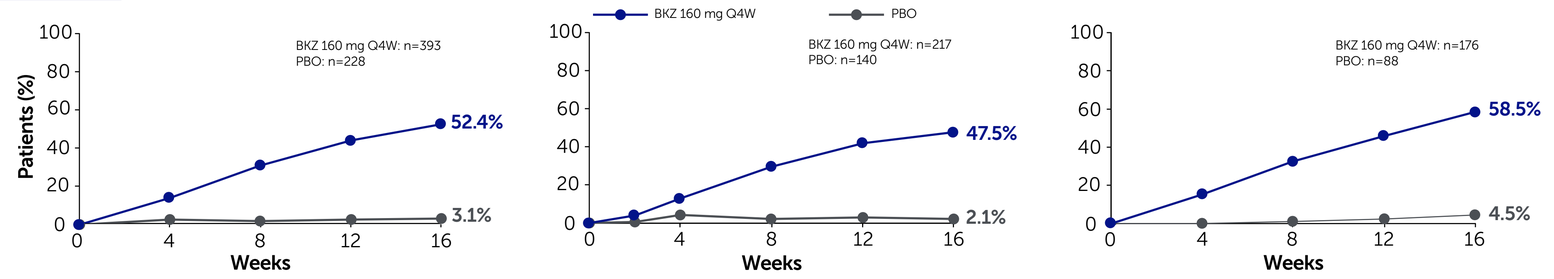


Figure 4 Proportion of patients with baseline psoriasis (≥3% BSA) achieving mNAPSI=0 to Week 16 (NRI)

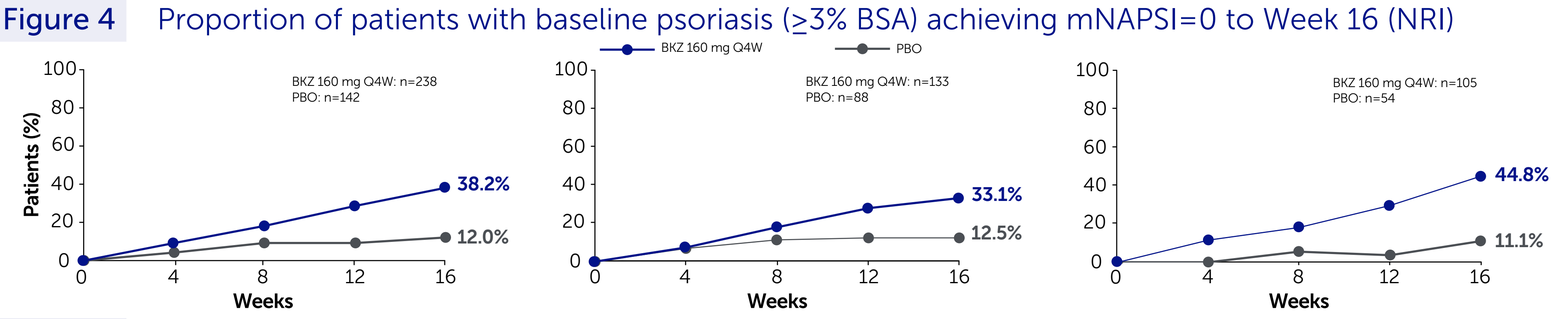
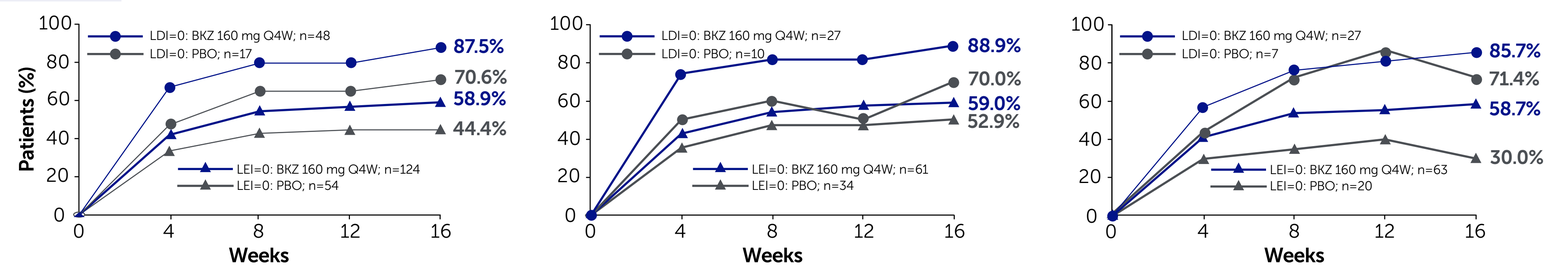


Figure 5 Proportion of patients with baseline psoriasis (≥3% BSA) achieving LDI=0 and LEI=0 to Week 16 (NRI)



ACR: American College of Rheumatology; **ACR50/70:** ≥50/70% improvement in ACR criteria; **bDMARD:** biologic disease-modifying antirheumatic drug; **BKZ:** bimekizumab; **BMI:** body mass index; **BSA:** body surface area; **HAQ-DI:** Health Assessment Questionnaire-Disability Index; **hs-CRP:** high-sensitivity C-reactive protein; **IL:** interleukin; **LDI:** Leeds Dactylitis Index; **LEI:** Leeds Enthesitis Index; **MTX:** methotrexate; **mNAPSI:** modified Nail Psoriasis Severity Index; **NRI:** non-responder imputation; **PASI:** Psoriasis Area and Severity Index; **PASI100:** 100% improvement in PASI; **PBO:** placebo; **PsA:** psoriatic arthritis; **PSO:** psoriasis; **PtAAP:** Patient's Assessment of Arthritis Pain; **Q2W:** every 2 weeks; **Q4W:** every 4 weeks; **SD:** standard deviation; **TNFi-IR:** tumour necrosis factor-α inhibitor inadequate response or intolerance.

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References: [1]Veale DJ. Lancet 2018;391:2273–84; [2]Reich K. Dermatol Pract Concept 2022;12:e2022104; [3]McInnes IB. Lancet 2023;401:25–37; [4]Merola JF. Lancet 2023;401:38–48. **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **YU, JFM, LCC, IBM, PJM, YT, PG, ABG, BI, RB, JC, JE, AA;** Drafting of the publication, or revising it critically for important intellectual content: **YU, JFM, LCC, IBM, PJM, YT, PG, ABG, BI, RB, JC, JE, AA;** Final approval of the publication: **YU, JFM, LCC, IBM, PJM, YT, PG, ABG, BI, RB, JC, JE, AA**. **Author Disclosures: YU:** Consultant and/or speaker fees from AbbVie, Eli Lilly, Janssen, Kyowa Kirin, Maruho, Sun Pharma and UCB Pharma. **JFM:** Consultant and/or investigator for AbbVie, Amgen, Biogen, BMS, Dermavant, Eli Lilly, Janssen, LEO Pharma, Pfizer, Novartis, Regeneron, Sanofi, Sun Pharma and UCB Pharma. **LCC:** Grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Gilead, Janssen, Novartis, Pfizer and UCB Pharma; consultant for AbbVie, Amgen, BMS, Boehringer Ingelheim, Celgene, Domain, Eli Lilly, Galapagos, Gilead, Janssen, Moonlake, Novartis, Pfizer and UCB Pharma; speaker fees from AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GSK, Janssen, Medac, Novartis, Pfizer and UCB Pharma. **IBM:** Consulting fees and honoraria from AbbVie, AstraZeneca, BMS, Boehringer Ingelheim, Cabaletta, Causeway Therapeutics, Celgene, Eli Lilly, Evelo, Janssen, MoonLake, Novartis and UCB Pharma; research support from BMS, Boehringer Ingelheim, Celgene, Janssen, Novartis and UCB Pharma. **PJM:** Research grants from AbbVie, Amgen, BMS, Eli Lilly, Gilead, Janssen, Novartis, Pfizer, Sun Pharma and UCB Pharma; consultancy fees from AbbVie, Acelyrin, Aclaris, Amgen, BMS, Boehringer Ingelheim, Eli Lilly, Galapagos, Gilead, GSK, Janssen, MoonLake, Novartis, Pfizer, Sun Pharma and UCB Pharma; speakers bureau from AbbVie, Amgen, Eli Lilly, Janssen, Novartis, Pfizer and UCB Pharma. **YT:** Speaker fees and/or honoraria from AbbVie, AstraZeneca, BMS, Boehringer Ingelheim, Chugai, Eisai, Eli Lilly, Gilead, GSK, Pfizer, Taiho and Taisho; received grants from Chugai, Eisai, Mitsubishi-Tanabe and Taisho. **ABG:** Honoraria as an advisory board member and consultant for Amgen, AnaptysBio, Avotres Therapeutics, BMS, Boehringer Ingelheim, Dice Therapeutics, Eli Lilly, Janssen, Novartis, Sanofi, UCB Pharma and Xbiotech; research/educational grants from AnaptysBio, BMS, MoonLake Immunotherapeutics, Novartis and UCB Pharma; all funds paid to Mount Sinai School of Medicine. **BI:** Employee and stockholder of UCB Pharma; stockholder of AbbVie and GSK. **RB, JC, JE:** Employees and stockholders of UCB Pharma. **AA:** Honoraria and/or research grants from AbbVie, Amgen, BMS, Boehringer Ingelheim, Eisai, Eli Lilly, Janssen, Kyowa Kirin, LEO Pharma, Maruho, Mitsubishi Tanabe Pharma, Novartis, Pfizer, Sun Pharma, Taiho Pharma, Torii Pharma and UCB Pharma. **Acknowledgements:** 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 Deepak Assudani for his contribution to the corresponding abstract. The authors acknowledge Heather Edens, PhD, UCB Pharma, Smyrna, Georgia, USA, for publication coordination, Eve Sullivan, MSc, Costello Medical, Cambridge, UK, for medical writing and editorial assistance, and the Creative team at Costello Medical for graphic design assistance. These studies were funded by UCB Pharma. All costs associated with development of this presentation were funded by UCB Pharma.

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