Bimekizumab Treatment Resulted in Rapid Response That Was Associated with Clinically Important Improvements in Patient-Reported Outcomes up to 3 Years in Patients with Psoriatic Arthritis

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Objective

To assess whether achieving stringent treatment responses at Week 16 in bimekizumab (BKZ)-treated patients with active psoriatic arthritis (PsA) was associated with clinically meaningful improvements in patient-reported outcomes (PROs) over 3 years.

Background

- PsA is a chronic inflammatory disease in which pain, fatigue and impaired functional capacity are among the most burdensome patient-reported symptoms, each contributing significantly to reduced health-related quality of life.[1–3]
- Achieving an early and rapid response to treatment is an important goal to reduce symptoms and minimise disease impact.[4,5]
- BKZ is a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A.

Methods

- BE OPTIMAL (NCT03895203) and BE COMPLETE (NCT03896581) were phase 3 trials, both placebo-controlled to Week 16, which assessed subcutaneous BKZ 160 mg every 4 weeks (Q4W), in patients with active PsA who were biologic disease-modifying antirheumatic drug (bDMARD)-naïve or had prior intolerance or inadequate response to tumour necrosis factor inhibitors (TNFi-IR), respectively.
- Patients completing Week 52 in BE OPTIMAL or Week 16 in BE COMPLETE could enter BE VITAL (open-label extension; NCT04009499), where all patients received BKZ 160 mg Q4W.
- In this post hoc analysis, BKZ-randomised patients were classified as rapid responders or non-responders based on achievement of resolution of swollen joint count (SJC=0) or ≥50% improvement from baseline in American College of Rheumatology response criteria (ACR50) at Week 16.
- The proportion of BKZ-randomised patients reporting clinically meaningful improvements in the following PROs are presented to 3 years (Week 160/148 in BE OPTIMAL or Week 156 in BE COMPLETE), stratified by Week 16 SJC=0 or ACR50 response:
  - Pain50, defined as a substantial (≥50%) decrease from baseline in pain, measured by visual analogue scale (VAS; score range: 0 [no pain]–100 [most severe pain])[6]
  - FACIT-Fatigue MCID (Functional Assessment of Chronic Illness Therapy-Fatigue minimal clinically important difference; score range 0–52 [higher scores indicate less fatigue]), defined as ≥4-point increase from baseline in patients with FACIT-Fatigue score ≤48 at baseline
  - PsAID-12 (12-item Psoriatic Arthritis Impact of Disease; score range: 0–10) no symptom or disease impact, defined as PsAID-12 total score ≤1.15[7]
- Data are reported as observed case (OC) or using modified non-responder imputation (mNRI).
  - mNRI considered all visits following discontinuation due to adverse events or lack of efficacy as non-response; all other missing data were imputed using multiple imputation and the response was derived from the imputed values.

Results

- 431 bDMARD-naïve and 267 TNFi-IR patients were randomised to BKZ in the BE OPTIMAL and BE COMPLETE studies, respectively.
- At Week 16, SJC=0 was achieved by 48.3% of bDMARD-naïve patients and 45.9% of TNFi-IR patients (mNRI; **Figure 1**). At Week 16, ACR50 was achieved by 44.8% of bDMARD-naïve patients and 43.5% of TNFi-IR patients (mNRI; **Figure 2**).
- Baseline characteristics were broadly comparable between responders and non-responders; responders were more likely overall to be younger and male (**Table**).
- Across both bDMARD-naive and TNFi-IR patients, SJC=0 and ACR50 responders demonstrated greater improvements in PROs than non-responders at Week 16 (**Figures 1–2**).
- Improvements in PROs in responders at Week 16 were sustained or increased further to Year 3 (**Figures 1–2**).

Conclusions

Patients with PsA treated with bimekizumab who were SJC=0 or ACR50 responders at Week 16 demonstrated greater and sustained improvements in pain, fatigue and reduced disease impact over 3 years compared to non-responders, both in bDMARD-naïve and TNFi-IR patients.

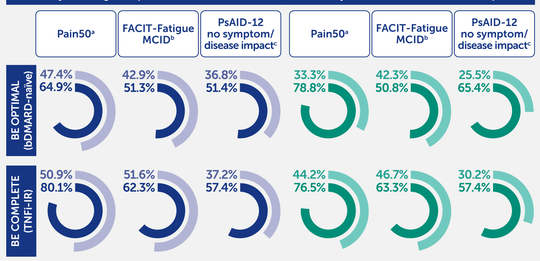
Interpretation should consider the post hoc design and potential confounding factors, including pain VAS being a component of the ACR response criteria.

These data suggest that rapid and high clinical responses may lead to long-term improvements in PROs, reinforcing the importance of timely intervention with effective therapy for optimal PsA management.

Summary

This post hoc analysis assessed whether the achievement of **stringent treatment responses** at **Week 16** with **bimekizumab**, was associated with **sustained**, **clinically meaningful improvements** in **patient-reported outcomes** up to **3 years**, in patients with active **psoriatic arthritis**.

Clinically meaningful improvements in PROs at Year 3, stratified by Week 16 SJC=0 or ACR50 response



Among **bDMARD-naïve** and **TNFi-IR** patients with PsA treated with **bimekizumab**, those achieving **SJC=0** or **ACR50** at **Week 16** experienced **greater** and **sustained** reductions in **pain**, **fatigue** and **overall disease impact** up to **3 years** than non-responders, highlighting the value of prompt, effective intervention to optimise long-term outcomes in PsA.

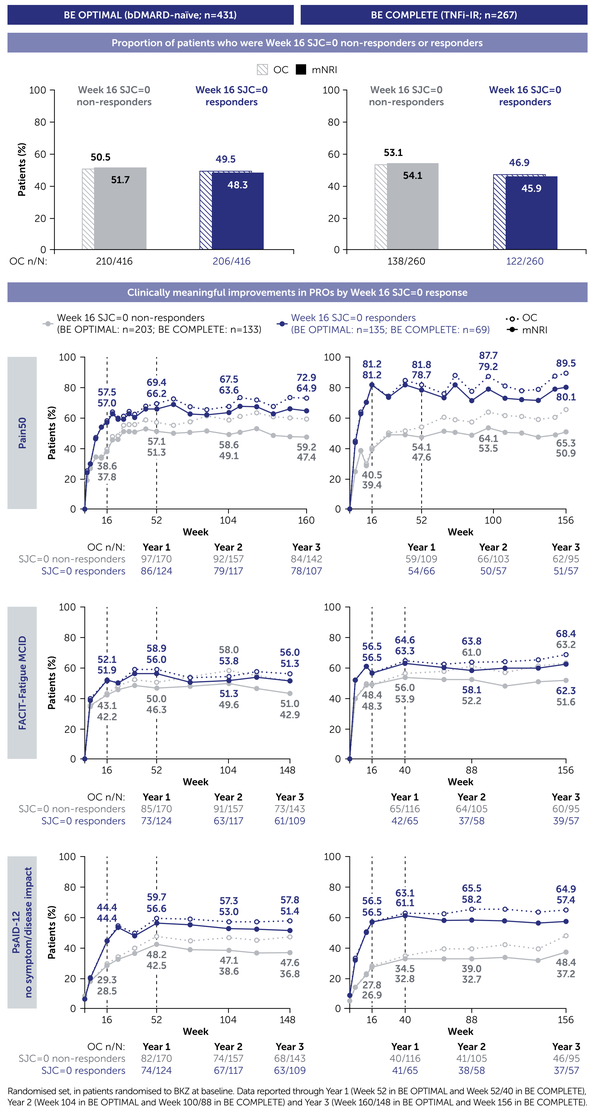
Data reported using mNRI. **[a]** Defined as a substantial (≥50%) decrease from baseline in pain VAS; **[b]** Defined as a ≥4-point increase from baseline in patients with FACIT-Fatigue score ≤48 at baseline; **[c]** Defined as PsAID-12 total score ≤1.15.

Table Baseline demographics and characteristics by Week 16 SJC=0 and ACR50 response

Mean (SD), unless otherwise stated	BE OPTIMAL (bDMARD-naïve; n=431)		BE COMPLETE (TNFi-IR; n=267)	
	Week 16 SJC=0 non-responders (n=210)	Week 16 SJC=0 responders (n=206)	Week 16 SJC=0 non-responders (n=138)	Week 16 SJC=0 responders (n=122)
Age, years	50.1 (13.1)	46.3 (11.4)	51.6 (11.7)	48.6 (12.8)
Sex, male, n (%)	87 (41.4)	109 (52.9)	70 (50.7)	56 (45.9)
BMI, kg/m²	30.0 (7.4)	28.7 (6.0)	30.9 (7.3)	29.2 (5.4)
Time since PsA diagnosis, years	6.0 (7.5)[n]	6.1 (7.3)[o]	10.1 (10.3)	9.1 (9.3)[c]
SJC (of 66 joints)	9.7 (7.0)	8.2 (5.3)	10.6 (7.6)	8.2 (6.8)
TJC (of 68 joints)	18.0 (12.7)	15.2 (10.1)	19.4 (14.0)	16.9 (12.6)
BSA affected by psoriasis ≥3%, n (%)	93 (44.3)	117 (56.8)	83 (60.1)	89 (73.0)
PASI score[d]	7.3 (6.2)[e]	9.1 (7.4)[f]	10.2 (10.6)[p]	9.7 (6.7)[h]
Enthesitis (LEI >0), n (%)	83 (39.5)	53 (25.7)	66 (47.8)	36 (29.5)
LEI score[i]	2.4 (1.4)	2.6 (1.4)	2.5 (1.4)	2.8 (1.7)
Dactylitis (LDI >0), n (%)	31 (14.8)	23 (11.2)	23 (16.7)	10 (8.2)
LDI score[j]	54.6 (68.7)	35.0 (23.5)	90.2 (134.8)	36.5 (33.4)
hs-CRP, mg/L, median (Q1, Q3)	3.9 (1.4, 9.3)	3.4 (1.6, 11.8)	6.7 (2.0, 20.2)	3.7 (1.5, 10.1)
Pain VAS[k]	54.3 (23.3)	53.0 (25.1)[a]	59.3 (24.4)	57.2 (24.3)
FACIT-Fatigue[l]	37.4 (10.1)	38.4 (8.7)[a]	34.2 (10.7)	36.8 (10.2)
PsAID-12 total score[m]	4.0 (1.9)	3.9 (1.9)[a]	4.6 (2.1)	4.3 (2.0)
	Week 16 ACR50 non-responders (n=225)	**Week 16 ACR50 responders (n=189)**	**Week 16 ACR50 non-responders (n=145)**	**Week 16 ACR50 responders (n=115)**
Age, years	50.6 (12.1)	46.0 (12.4)	52.7 (11.8)	47.1 (12.2)
Sex, male, n (%)	83 (36.9)	112 (59.3)	66 (45.5)	60 (52.2)
BMI, kg/m²	30.3 (7.0)	28.2 (6.3)	30.7 (6.3)	29.4 (6.8)
Time since PsA diagnosis, years	6.4 (8.1)[n]	5.5 (6.3)[o]	9.9 (10.5)	9.3 (9.0)[o]
SJC (of 66 joints)	8.5 (5.9)	9.4 (6.2)	9.4 (7.2)	9.7 (7.4)
TJC (of 68 joints)	17.4 (12.6)	15.7 (10.2)	18.1 (13.4)	18.3 (13.6)
BSA affected by psoriasis ≥3%, n (%)	105 (46.7)	104 (55.0)	90 (62.1)	82 (71.3)
PASI score[d]	8.6 (7.7)[n]	7.9 (6.1)[r]	11.1 (10.4)[s]	8.8 (6.5)[t]
Enthesitis (LEI >0), n (%)	84 (37.3)	53 (28.0)	61 (42.1)	41 (35.7)
LEI score[i]	2.5 (1.5)	2.4 (1.4)	2.8 (1.4)	2.3 (1.5)
Dactylitis (LDI >0), n (%)	19 (8.4)	35 (18.5)	17 (11.7)	16 (13.9)
LDI score[j]	33.4 (37.8)	53.2 (61.4)	104.3 (150.7)	41.8 (47.8)
hs-CRP, mg/L, median (Q1, Q3)	3.4 (1.4, 8.4)	4.2 (1.6, 13.7)	5.0 (1.8, 13.2)	4.6 (1.6, 19.0)
Pain VAS[k]	53.5 (25.0)	53.8 (23.4)	58.6 (24.1)	58.0 (24.6)
FACIT-Fatigue[l]	36.3 (9.8)	39.6 (8.8)	34.8 (10.6)	36.1 (10.4)
PsAID-12 total score[m]	4.1 (1.9)	3.8 (1.9)	4.6 (2.1)	4.2 (2.0)

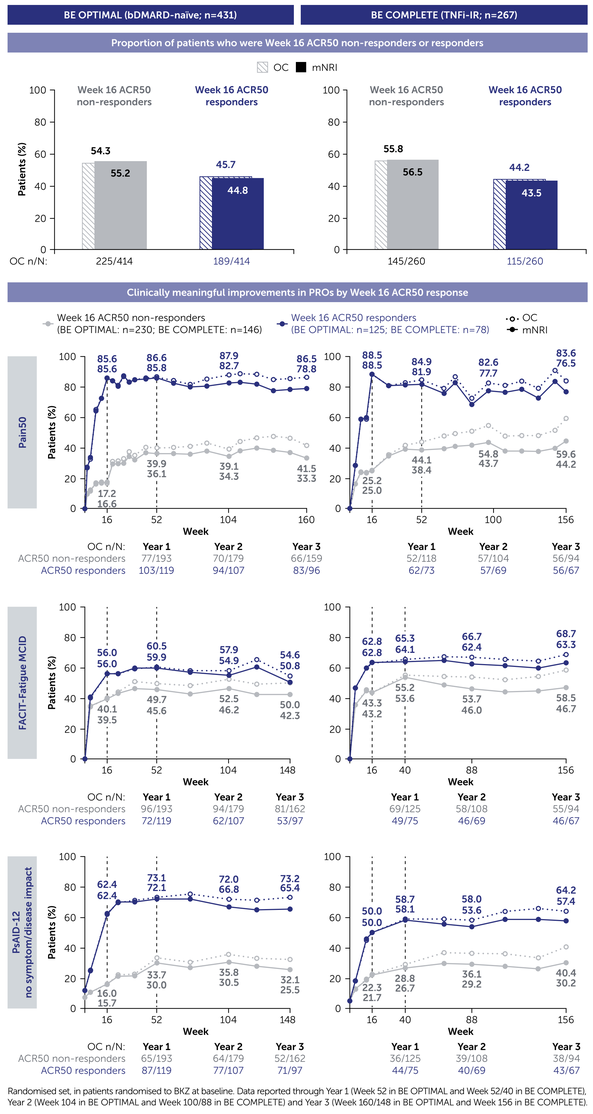
Randomised set, in patients randomised to BKZ at baseline. **[a]** n=205; **[b]** n=203; **[c]** n=121; **[d]** In patients with baseline psoriasis ≥3% BSA; **[e]** n=93; **[f]** n=117; **[g]** n=83; **[h]** n=89; **[i]** In patients with enthesitis at baseline (LEI >0); **[j]** In patients with dactylitis at baseline (LDI >0); **[k]** Pain was assessed using Patient's Assessment of Arthritis Pain VAS (score range: 0 [no pain] to 100 [most severe pain]); **[l]** FACIT-Fatigue scores range from 0–52; higher scores indicate less fatigue; **[m]** PsAID-12 total scores range from 0–10; higher scores indicates worse status; **[n]** n=220; **[o]** n=186; **[p]** n=114; **[q]** n=105; **[r]** n=104; **[s]** n=90; **[t]** n=82.

Figure 1 Clinically meaningful improvements in PROs to 3 years by Week 16 SJC=0 response (mNRI, OC)



Randomised set, in patients randomised to BKZ at baseline. Data reported through Year 1 (Week 52 in BE OPTIMAL and Week 52/40 in BE COMPLETE), Year 2 (Week 104 in BE OPTIMAL and Week 100/88 in BE COMPLETE) and Year 3 (Week 160/148 in BE OPTIMAL and Week 156 in BE COMPLETE).

Figure 2 Clinically meaningful improvements in PROs to 3 years by Week 16 ACR50 response (mNRI, OC)



Randomised set, in patients randomised to BKZ at baseline. Data reported through Year 1 (Week 52 in BE OPTIMAL and Week 52/40 in BE COMPLETE), Year 2 (Week 104 in BE OPTIMAL and Week 100/88 in BE COMPLETE) and Year 3 (Week 160/148 in BE OPTIMAL and Week 156 in BE COMPLETE).

ACR: American College of Rheumatology; **ACR50:** ≥50% improvement from baseline in ACR response criteria; **bDMARD:** biologic disease-modifying antirheumatic drug; **BKZ:** bimekizumab; **BMI:** body mass index; **BSA:** body surface area; **FACIT-Fatigue:** Functional Assessment of Chronic Illness Therapy-Fatigue; **hs-CRP:** high-sensitivity C-reactive protein; **IL:** interleukin; **LDI:** Leeds Dactylitis Index; **LEI:** Leeds Enthesitis Index; **MCID:** minimal clinically important difference; **mNRI:** modified non-responder imputation; **OC:** observed case; **Pain50:** ≥50% decrease from baseline in pain VAS; **PASI:** Psoriasis Area and Severity Index; **PsA:** psoriatic arthritis; **PsAID-12:** 12-item Psoriatic Arthritis Impact of Disease; **PRO:** patient-reported outcome; **Q1:** first quartile; **Q3:** third quartile; **Q4W:** every 4 weeks; **SD:** standard deviation; **SJC:** swollen joint count; **TJC:** tender joint count; **TNFi-IR:** prior inadequate response or intolerance to tumour necrosis factor inhibitors; **VAS:** visual analogue scale.

References: [1]Tillett W. Rheumatol Ther 2020;7:617–37; [2]Gudu T. Expert Rev Clin Immunol 2018;14:405–17; [3]Ogdie A. RMD Open 2020;6:e001321; [4]Snoeck Henkemans SVJ. RMD Open 2022;8:e002706; [5]Coates LC. Nat Rev Rheumatol 2022;18:465–79; [6]Dworkin RH. J Pain 2008;9:105–21; [7]Gossec L. RMD Open 2024;10:e003548. **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **LG**, **DDG**, **MEH**, **TO**, **AMO**, **FP**, **AR**, **MdW**, **JL**, **PH**, **PJM**; Drafting of the publication, or reviewing it critically for important intellectual content: **LG**, **DDG**, **MEH**, **TO**, **AMO**, **FP**, **AR**, **MdW**, **JL**, **PH**, **PJM**; Final approval of the publication: **LG**, **DDG**, **MEH**, **TO**, **AMO**, **FP**, **AR**, **MdW**, **JL**, **PH**, **PJM**. **Author Disclosures:** **LG:** Research grants from AbbVie, Lilly, Novartis and UCB; consulting fees from AbbVie, AlfaSigma, Amgen, BMS, Celltrion, Johnson & Johnson, Lilly, Moonlake, Novartis, Nordic Pharma, Oruka, Pfizer, Stada, Takeda and UCB; non-financial support from AbbVie, Amgen, Biogen, BMS, Celltrion, JbJ, Lilly, Pfizer and UCB. **DDG:** Consultant for AbbVie, Amgen, AstraZeneca, BMS, Eli Lilly, Johnson & Johnson, Novartis, Oruka, Pfizer, Roche and UCB; received grant/research support from AbbVie, Amgen, BMS, Eli Lilly, Fresenius Kabi, Novartis, Pfizer and UCB. **MEH:** Advisory board member and consultant for AbbVie, Amgen, BMS, Eli Lilly, Janssen, Novartis, Pfizer and UCB. **TO:** Received research grants and/or speaker's fee from AbbVie, Asahi Kasei, Astellas, Daiichi Sankyo, Eisai, Eli Lilly, Janssen, Novartis Pharma, Tanabe Mitsubishi and UCB. **AMO:** Research grants to Johns Hopkins University from AbbVie, Amgen and Janssen; consulting fees from BMS, Janssen, Sanofi and UCB. **FP:** Received grant/research support from Eli Lilly and Company, Novartis and UCB; received consultancy fees and speaker's bureau from AbbVie, Amgen, BMS, Celgene, Eli Lilly and Company, Galapagos, Hexal, Janssen, Medscape, Moonlake Pharma, MSD, Novartis, Pfizer, Roche and UCB. **AR:** Received travel grants from Arrowtex, Janssen, Novartis, Pfizer and UCB; advisory boards and speaker for AbbVie, Novartis, Pfizer, Roche and UCB. **MdW:** Stichting Tools has received fees for lectures provided by MdW from UCB in 2023. **JL**, **PH:** Employees and shareholders of UCB. **PJM:** Research grants from AbbVie, Acelyrin, Amgen, BMS, Eli Lilly and Company, Johnson & Johnson Innovative Medicine, Novartis, Pfizer, Inc., Sana and UCB; consulting fees from AbbVie, Acelyrin, Amgen, BMS, Century, Cullinan, Eli Lilly and Company, Inmagene, Johnson & Johnson Innovative Medicine, MoonLake Immunotherapeutics, Novartis, Pfizer, Inc., Spyre, Takeda and UCB; speakers bureau fees from AbbVie, Amgen, Eli Lilly and Company, Johnson & Johnson Innovative Medicine, Novartis, Pfizer, Inc. and UCB. **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 thank Lyes Derouiche, PhD, CMPP, of UCB, for editorial review and publication coordination, Zahraa Omran, MSci, of Costello Medical, UK for medical writing and editorial assistance, and the Costello Medical Creative team for design support. These studies were funded by UCB. All costs associated with development of this presentation were funded by UCB.

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