Bimekizumab Maintained Efficacy Responses Through 52 Weeks in Biologic Disease-Modifying Antirheumatic Drug-Naïve Patients with Psoriatic Arthritis Who Were Responders at Week 16: Results from BE OPTIMAL, a Phase 3, Active Reference Study

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Objective

To report the maintenance of response in efficacy outcomes assessing joints and skin, including composite disease activity measures, to Week 52 in bimekizumab-treated patients with psoriatic arthritis who were responders at Week 16 of the BE OPTIMAL study.

Background

- Psoriatic arthritis (PsA) is a chronic, long-term condition; thus, it is important that therapies sustain high levels of disease control.
- Assessing the maintenance of response in patients that achieve treatment targets is of interest, particularly as patients can experience loss of response with long-term therapy.[1]
- Bimekizumab (BKZ), a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated rapid, statistically significant and clinically meaningful efficacy responses at Week 16 versus placebo, in patients with PsA.[2,3]
- Week 16 efficacy was sustained to Week 52 in the phase 3 BE OPTIMAL trial.[4]

Methods

- BE OPTIMAL (NCT03895203) included a 16-week, double-blind, placebo-controlled period, and a 36-week active treatment-blind period. An active reference (ADA) arm was included to provide a reference for the benefit-risk profile of BKZ. The ADA arm was not powered for statistical comparison to BKZ or placebo.
- Maintenance of response is reported as the percentage of Week 16 responders who met the response criteria at subsequent study visits. This analysis is shown for visits from Week 16 to Week 52 for American College of Rheumatology (ACR)20/50/70, Psoriasis Area and Severity Index (PASI)75/90/100, minimal and very low disease activity (MDA, VLDA), Disease Activity Index for Psoriatic Arthritis (DAPSA) remission or low disease activity (REM+LDA; score of ≤14) and remission (REM; score ≤4), and composite ACR50+PASI100 responses.
- Week 16 responders are reported using non-responder imputation (NRI), Week 52 maintenance data are reported using NRI and observed case (OC).
- The number of treatment-emergent adverse events (TEAEs) to Week 52 are reported for patients who received ≥1 dose of BKZ, including patients who switched from placebo to BKZ at Week 16.

Results

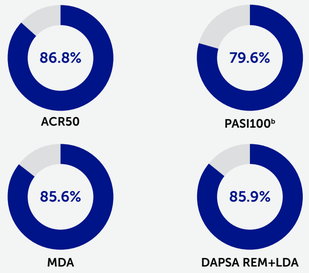
- At baseline, 431 and 140 patients were randomised to BKZ 160 mg every 4 weeks (Q4W) and ADA every 2 weeks (Q2W), respectively. 217/431 (50.3%) BKZ- and 68/140 (48.6%) ADA-randomised patients had psoriasis affecting ≥3% body surface area (BSA). Week 16 completers: 414/431 (96.1%) BKZ, 136/140 (97.1%) ADA; Week 52 completers: 383/431 (88.9%) BKZ, 123/140 (87.9%) ADA.
- Baseline demographics and disease characteristics for the groups randomised to BKZ and ADA are reported in **Table 1**.
- At Week 16, 189 (43.9%; NRI) BKZ-treated patients achieved ACR50. Of those responders, 86.8% (NRI) and 91.1% (OC) maintained ACR50 response at Week 52 (**Figure 1**). Similar results were seen across other ACR endpoints: ACR20/70 was achieved by 268 (62.2%) and 105 (24.4%) patients, respectively, at Week 16 (NRI). At Week 52, ACR20/70 was maintained by 88.4%/82.9% (NRI) and 92.9%/87.9% (OC) of patients.
- Of 217 BKZ-treated patients with psoriasis affecting ≥3% BSA at baseline, 133 (61.3%) and 103 (47.5%) achieved PASI90/100 at Week 16. Robust maintenance of response was observed in high proportions (>79%) of these patients to Week 52 (**Figure 2**). 168 (77.4%) achieved PASI75; 88.1% maintained response to Week 52.
- A high proportion of Week 16 responders for MDA, DAPSA REM+LDA and ACR50+PASI100 maintained their responses to Week 52 (**Figures 3–5**).
- Response was maintained to Week 52 for 79.4% (NRI) and 86.2% (OC) of patients that achieved VLDA at Week 16. 68.1% (NRI) of the 47 (10.9%) patients that achieved DAPSA REM at Week 16 maintained response to Week 52.
- To Week 52, 555/702 (79.1%) patients reported ≥1 TEAE whilst receiving BKZ; 46 (6.6%) reported serious TEAEs.

Conclusions

A high proportion of bimekizumab-treated patients who responded at Week 16 maintained robust efficacy responses to Week 52. Efficacy measures spanned joint, skin, disease activity, and composite efficacy outcomes. The safety profile of bimekizumab was consistent with previous reports.[2,3]

Summary

Maintenance of response, up to 52 weeks, was assessed in bimekizumab-treated patients who achieved a response at Week 16 of BE OPTIMAL[a]



A **high proportion of patients** who responded to treatment of bimekizumab at Week 16 **maintained their response to Week 52** across multiple domains

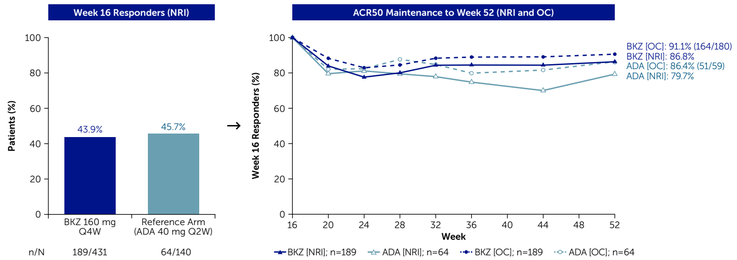
[a]Values shown here are NRI; [b]In patients with psoriasis affecting ≥3% BSA at baseline.

Table 1 Baseline characteristics

	BKZ 160 mg Q4W n=431	ADA 40 mg Q2W n=140
Age, years, mean (SD)	48.5 (12.6)	49.0 (12.8)
Male, n (%)	201 (46.6)	71 (50.7)
BMI, kg/m^2, mean (SD)	29.2 (6.8)	28.4 (5.9)
PsA duration,[a] years, mean (SD)	6.0[b] (7.3)	6.1 (6.8)
Concomitant methotrexate, n (%)	252 (58.5)	82 (58.6)
TJC (of 68 joints), mean (SD)	16.8 (11.8)	17.5 (13.1)
SJC (of 66 joints), mean (SD)	9.0 (6.2)	9.7 (7.1)
hs-CRP ≥6 mg/L, n (%)	158 (36.7)	44 (31.4)
Psoriasis BSA ≥3%, n (%)	217 (50.3)	68 (48.6)
PASI score,[d] mean (SD)	8.2 (6.8)	8.6 (7.6)
Enthesitis,[e] n (%)	143 (33.2)	36 (25.7)
Score, mean (SD)	2.5 (1.5)	2.3 (1.6)
Dactylitis,[f] n (%)	56 (13.0)	11 (7.9)
Score, mean (SD)	46.7 (54.3)	49.7 (31.9)
HAQ-DI, mean (SD)	0.82 (0.59)	0.86 (0.54)
PtAAP,[g] mean (SD)	53.6 (23.9)[h]	56.7 (23.9)

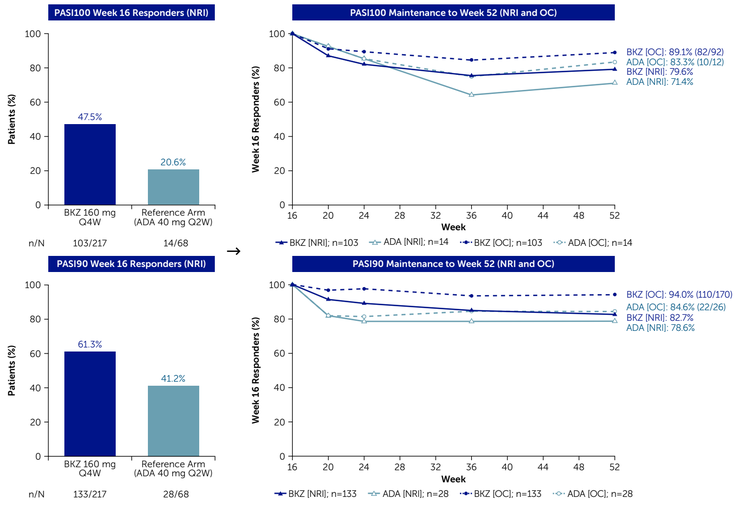
Randomised set. [a]Listed as time since first diagnosis of PsA; [b]n=423; [c]n=139; [d]In patients with ≥3% BSA with psoriasis at baseline; [e]LEI >0; [f]LDI >0; [g]PtAAP VAS 0 (no symptoms) – 100 (severe symptoms); [h]n=430.

Figure 1 Maintenance of ACR50 responses to Week 52, in Week 16 responders (NRI, OC)



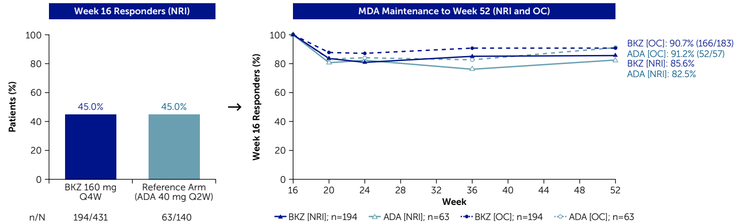
Randomised set.

Figure 2 Maintenance of PASI100 and PASI90 responses to Week 52, in Week 16 responders (NRI, OC)



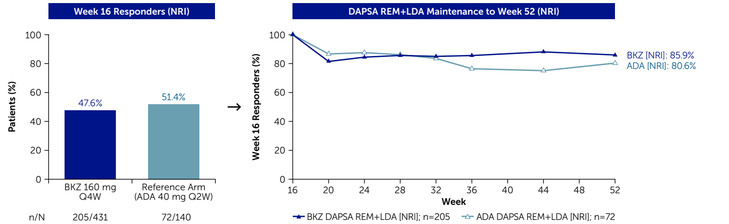
Randomised set. In patients with psoriasis affecting ≥3% BSA at baseline.

Figure 3 Maintenance of MDA to Week 52, in Week 16 responders (NRI, OC)



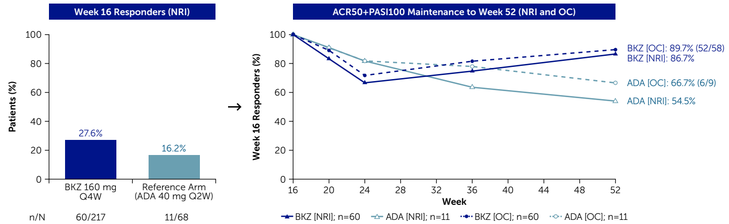
Randomised set. Patients are considered to achieve MDA when they meet 5/7 of the following criteria, respectively: TJC ≤1, SJC ≤1, PASI ≤1 or BSA ≤3%, patient pain (VAS ≤15 mm), patient global assessment (VAS ≤20), HAQ-DI ≤0.5 and tender entheseal points (LEI) ≤1.

Figure 4 Maintenance of DAPSA REM+LDA to Week 52, in Week 16 responders (NRI)



Randomised set. DAPSA score is the sum of SJC, TJC, patient pain (VAS 0–10), patient global assessment (VAS 0–10) and C-reactive protein (mg/L). DAPSA REM+LDA is defined as DAPSA total score ≤14; DAPSA REM is defined as DAPSA total score ≤4.

Figure 5 Maintenance of ACR50+PASI100 to Week 52, in Week 16 responders (NRI, OC)



Randomised set. In patients with psoriasis affecting ≥3% BSA at baseline. Due to the low number of ADA patients achieving this composite threshold, results should be interpreted with caution.

ACR: American College of Rheumatology; **ACR20/50/70:** ≥20/50/70% improvements from baseline in ACR criteria; **ADA:** adalimumab; **bDMARD:** biological disease-modifying antirheumatic drug; **BKZ:** bimekizumab; **BMI:** body mass index; **BSA:** body surface area; **DAPSA:** Disease Activity index for PSoriatic Arthritis; **HAQ-DI:** Health Assessment Questionnaire Disability Index; **hs-CRP:** high-sensitivity C-reactive protein; **IL:** interleukin; **LDA:** low disease activity; **LDI:** Leeds Dactylitis Index; **LEI:** Leeds Enthesitis Index; **MDA:** Minimal Disease Activity; **NRI:** non-responder imputation; **OC:** observed case; **PASI:** Psoriasis Area and Severity Index; **PASI75/90/100:** ≥75/90/100% improvement in PASI; **PsA:** psoriatic arthritis; **REM:** remission; **SD:** standard deviation; **SJC:** swollen joint count; **TEAE:** treatment-emergent adverse event; **TJC:** tender joint count; **Q2W:** every 2 weeks; **Q4W:** every 4 weeks; **VLDA:** very low disease activity.

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References: [1]Boehncke WH. Am J Clin Dermatol 2013;14:377–88; [2]McInnes IB. Lancet. 2022;401(10370):25–37. [3]Merola JF. Lancet. 2022;401(10370):38–48; [4]Ritchlin CT. Arthritis Rheumatol. 2022;74 (suppl 9). **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **WT**, **JFM**, **YT**, **EGF**, **DM**, **JAW**, **DT**, **BI**, **RB**, **VT**, **CTR**; drafting of the publication, or revising it critically for important intellectual content; **WT**, **JFM**, **YT**, **EGF**, **DM**, **JAW**, **DT**, **BI**, **RB**, **VT**, **CTR**; final approval of the publication: **WT**, **JFM**, **YT**, **EGF**, **DM**, **JAW**, **DT**, **BI**, **RB**, **VT**, **CTR**. **Author Disclosures: WT:** Research grants, consulting fees, speaking fees, and/or honoraria from AbbVie, Amgen, Celgene, Eli Lilly, GSK, Janssen, MSD, Novartis, Ovo Pharma, Pfizer and UCB Pharma. **JFM:** Consultant and/or investigator for AbbVie, Amgen, Biogen, BMS, Dermavant, Eli Lilly, Janssen, LEO Pharma, Novartis, Pfizer, Regeneron, Sanofi, Sun Pharma and UCB Pharma. **YT:** Speaking 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. **EGF:** Received consultancy/speaker fees from AbbVie, BMS, Celltrion, Eli Lilly, Galapagos, Janssen, MSD, Novartis, Pfizer and UCB Pharma. **DM:** Received grants/research support from AbbVie, Celgene, Janssen, Merck, Novartis and Pfizer; consulting fees and honoraria from AbbVie, Celgene, Janssen, Merck, Novartis, Pfizer and UCB Pharma; speaker's bureau for AbbVie, Celgene, Janssen, Merck, Novartis, Pfizer and UCB Pharma. **JAW:** Consultant for/grant support from AbbVie, Amgen, Eli Lilly, Janssen, Merck, Novartis, Pfizer and UCB Pharma. **DT:** Served as an investigator and/or consultant/advisor for AbbVie, Almirall, Amgen, BMS, Boehringer Ingelheim, Celltrion, Eli Lilly, Galapagos, Galderma, Janssen-Cilag, Kyowa Kirin, LEO Pharma, Novartis, Pfizer, Regeneron, Samsung, Sandoz, Sanofi, Target-Solution, and UCB Pharma. Has received grants from AbbVie, LEO Pharma, and Novartis. **BI:** Employee of UCB Pharma; shareholder of AbbVie, GSK and UCB Pharma. **RB:** Employee and shareholder of UCB Pharma. **VT:** Employee and Shareholder of UCB Pharma. **CTR:** Research for AbbVie; consultant for Amgen, AbbVie, Eli Lilly, Gilead, Janssen, Novartis, Pfizer 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 Heather Edens, PhD, UCB Pharma, Smyrna, Georgia, USA, for publication coordination, Matthew Walker, MRes, Costello Medical, Cambridge, UK for medical writing and editorial assistance, and the Costello Medical Creative team for design support. This study was funded by UCB Pharma. All costs associated with development of this presentation were funded by UCB Pharma.

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