Sustained Symptom Improvement in Psoriatic Arthritis with 2-Year Bimekizumab Treatment Based on Routine Assessment of Patient Index Data 3

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Objective

To report a post hoc analysis assessing patient-reported sustained symptom improvement with bimekizumab (BKZ) treatment up to 2 years using the Routine Assessment of Patient Index Data 3 (RAPID3), and the association with clinical disease measures in patients with psoriatic arthritis (PsA).

Background

- BKZ, a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated sustained improvements in clinical and patient-reported outcomes (PROs) across PsA disease domains up to 2 years.[1]
- RAPID3 is a composite index of 3 PROs (physical function, pain, and patient global assessment) which provides a simple tool commonly used in the clinical setting for assessing PsA disease activity.[2]

Methods

- The phase 3 trials, BE OPTIMAL (NCT03895203) and BE COMPLETE (NCT03896581), assessed subcutaneous BKZ 160 mg every 4 weeks (Q4W) in patients with PsA who were either biologic disease-modifying antirheumatic drug (bDMARD)-naïve or had prior inadequate response or intolerance to tumor necrosis factor inhibitors (TNFi-IR). Both were placebo (PBO)-controlled to Week 16.
- BE OPTIMAL Week 52 and BE COMPLETE Week 16 completers could enter BE VITAL (open-label extension; NCT04009499), in which all patients received BKZ 160 mg Q4W.
- RAPID3 data were calculated using the Health Assessment Questionnaire-Disability Index, pain assessment visual analogue scale (VAS), and patients' global assessment of disease severity. Each is scored from 0–10, giving a total score from 0–30 (with higher scores indicating higher disease activity).[2]
- In this post hoc analysis, we report RAPID3 outcomes to Week 104 of BE OPTIMAL and Week 100 of BE COMPLETE: change from baseline (CfB), minimal clinically important difference (MCID; ≥3.8-point decrease from baseline in patients with a baseline RAPID3 score ≥3.8),[3] and disease activity state.
- Associations between Week 16 RAPID3 remission (REM; ≤3) achievement and Week 52 minimal disease activity (MDA) and Disease Activity Index in PsA (DAPSA) severity were assessed using logistic regression. The association between RAPID3 and MDA/very low disease activity (VLDA) and DAPSA REM+LDA and REM at Week 52 were assessed by Mantel-Haenszel chi-square test.
- Data reported as observed case and using multiple (continuous), non-responder (binary), or worst category (categorical) imputation.

Results

- In total, 710/852 (83.3%) bDMARD-naïve patients completed Week 104 of BE OPTIMAL; 322/400 (80.5%) TNFi-IR patients completed Week 100 of BE COMPLETE.
- Across both trials, in BKZ-randomized patients and PBO patients who switched to BKZ at Week 16 (PBO/BKZ), sustained improvements in disease activity were observed up to 2 years, including RAPID3 CfB and MCID (**Figure 1**).
- Greater proportions of BKZ-randomized vs PBO/BKZ patients reached RAPID3 REM at Week 16 in both bDMARD-naive and TNFi-IR patients; RAPID3 REM achievement was sustained to 2 years on BKZ treatment (**Figure 2**).
- Early RAPID3 REM achievement at Week 16 was associated with MDA, VLDA, DAPSA REM+LDA and DAPSA REM achievement at Week 52 (all nominal p<0.001; **Table 1**).
- RAPID3 disease states were strongly associated with MDA/VLDA and DAPSA states at Week 52 (**Figure 3**).

Conclusions

Bimekizumab treatment resulted in sustained improvements in RAPID3 to 2 years in patients with active PsA. This response was consistent regardless of prior TNFi exposure.

Early RAPID3 response was associated with long-term achievement of low disease activity for patients treated with bimekizumab, as early improvements in RAPID3 were indicative of longer-term disease control. This indicates the potential utility of RAPID3 as part of the clinical evaluation of patients.

Summary

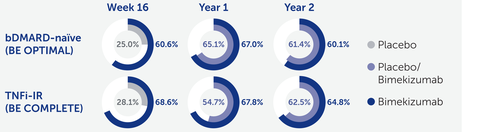
In a post hoc analysis, disease activity was assessed using RAPID3, a composite index of patient-reported outcomes, in bimekizumab-treated patients with active PsA in two phase 3 trials

RAPID3 Components

Physical function (HAQ-DI) | **Pain (Pain VAS)** | **Disease severity (Patients' global assessment)**

Improvements in **patient-reported symptoms** were **sustained** to 2 years (Week 104 in bDMARD-naïve patients and to Week 100 in TNFi-IR patients)

RAPID3 MCID (NRI)

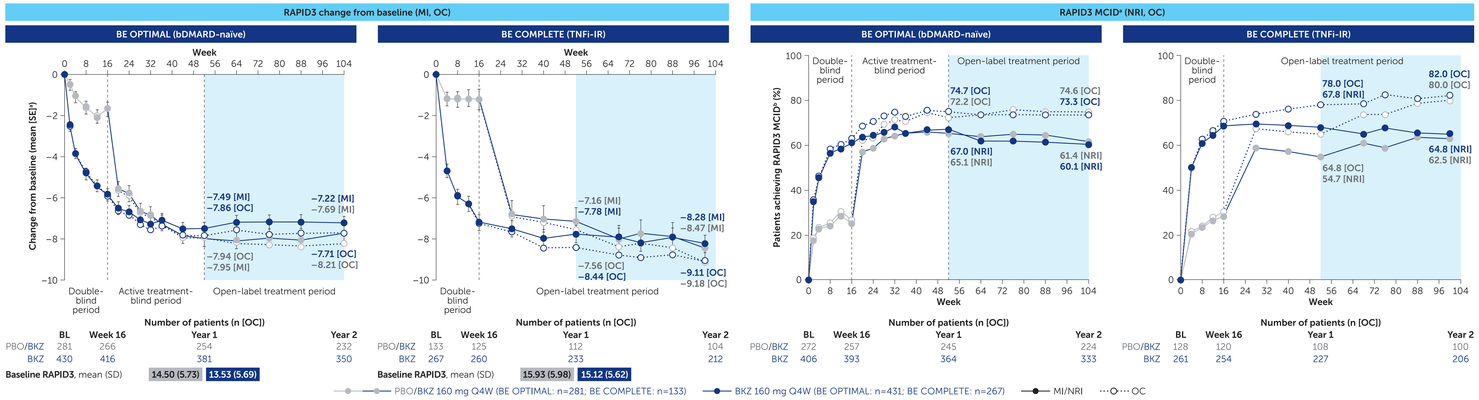


Early **RAPID3 responses** were **associated** with long-term clinical improvement and achievement of stringent control of disease activity measures including **MDA/VLDA** and **DAPSA REM+LDA/REM**

Consistent and sustained improvements in patient-reported **disease activity** were demonstrated up to **2 years** in bDMARD-naive and TNFi-IR patients with PsA

Early symptom improvement assessed by RAPID3 may be a good predictor of **long-term disease control**

Figure 1 Change from baseline and proportion of patients achieving minimal clinically important difference (MCID) in RAPID3 over time to 2 years (MI, NRI, OC)



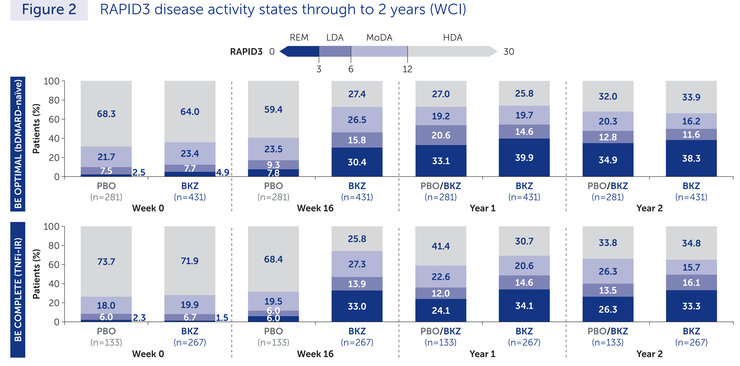
Randomized set. RAPID3 reported through Year 1 (Week 52 in BE OPTIMAL and BE COMPLETE) to Year 2 (Week 104 in BE OPTIMAL and Week 100 in BE COMPLETE) as RAPID3 components were only collected to Week 100 in BE COMPLETE. **[a]** Error bars (SE) provided for MI data only; **[b]** RAPID3 MCID defined as ≥3.8-point decrease from baseline in patients with a RAPID3 score ≥3.8 at baseline.

Table 1 Association of RAPID3 response at Week 16 with stringent levels of clinical disease activity at Year 1 (OC)

n/N %	BE OPTIMAL (bDMARD-naïve) RAPID3 No REM (>3) N=629	BE OPTIMAL (bDMARD-naïve) RAPID3 REM (≤3) N=189	BE COMPLETE (TNFi-IR) RAPID3 No REM (>3) N=289	BE COMPLETE (TNFi-IR) RAPID3 REM (≤3) N=96
MDA	291/573 50.8	166/177 93.8	88/253 34.8	80/91 87.9
Odds Ratio (95% CI)	14.6 (7.8, 27.5)		13.6 (6.9, 27.0)	
VLDA	109/573 19.0	115/175 65.7	37/253 14.6	45/92 48.9
Odds Ratio (95% CI)	8.2 (5.6, 11.9)		5.6 (3.3, 9.6)	
DAPSA REM+LDA	379/569 66.6	168/175 96.0	137/240 57.1	87/91 95.6
Odds Ratio (95% CI)	12.0 (5.5, 26.1)		16.4 (5.8, 46.0)	
DAPSA REM	146/569 25.7	132/175 75.4	53/240 22.1	59/91 64.8
Odds Ratio (95% CI)	8.9 (6.0, 13.2)		6.5 (3.8, 11.0)	

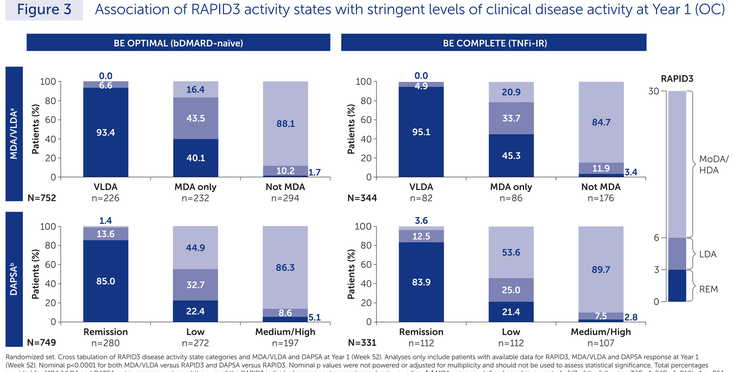
Randomized set. Analyses only include patients with available data for RAPID3, MDA, VLDA and DAPSA response at Week 52. Nominal p<0.001 for both MDA/VLDA versus RAPID3 and DAPSA versus RAPID3. Nominal p values are not powered or adjusted for multiplicity and should not be used to assess statistical significance. DAPSA REM+LDA disease state defined as DAPSA score of ≤14.

Figure 2 RAPID3 disease activity states through to 2 years (WCI)



Randomized set. Patients that dropped out of the study or were otherwise missing were included in the HDA category. RAPID3 reported at Year 1 (Week 52 in BE OPTIMAL and BE COMPLETE) and Year 2 (Week 104 in BE OPTIMAL and Week 100 in BE COMPLETE) as RAPID3 components were only collected to Week 100 in BE COMPLETE. Disease activity state categorized as high (≥12), moderate (>6–≤12), low (>3–≤6), or remission (≤3).

Figure 3 Association of RAPID3 activity states with stringent levels of clinical disease activity at Year 1 (OC)



Randomized set. Cross tabulation of RAPID3 disease activity state categories and MDA/VLDA and DAPSA at Year 1 (Week 52). Analyses only include patients with available data for RAPID3, MDA/VLDA and DAPSA response at Year 1 (Week 52). Nominal p<0.0001 for both MDA/VLDA versus RAPID3 and DAPSA versus RAPID3. Nominal p values were not powered or adjusted for multiplicity and should not be used to assess statistical significance. Total percentages provided for MDA/VLDA and DAPSA outcomes may not equal the sum of the RAPID3 individual component percentages due to rounding. **[a]** MDA response defined as achievement of ≥5/7 of the following: TJC ≤1, SJC ≤1, PASI ≤1 or BSA ≤3%, patient pain VAS ≤15, PGA-PsA VAS ≤20, HAQ-DI ≤0.5, and tender entheseal points (LEI) ≤1. **[b]** DAPSA REM defined as a DAPSA score of ≤4. LDA defined as a DAPSA score of >4–≤14. Medium/High defined as a DAPSA score of >14.

bDMARD: biologic disease-modifying antirheumatic drug; **BKZ:** bimekizumab; **BL:** baseline; **CI:** confidence interval; **DAPSA:** Disease Activity Index in Psoriatic Arthritis; **HAQ-DI:** Health Assessment Questionnaire-Disability Index; **HDA:** high disease activity; **IL:** interleukin; **LDA:** low disease activity; **MCID:** minimal clinically important difference; **MDA:** minimal disease activity; **MI:** multiple imputation; **MoDA:** moderate disease activity; **NRI:** non-responder imputation; **OC:** observed case; **PBO:** placebo; **PRO:** patient-reported outcome; **PsA:** psoriatic arthritis; **Q4W:** every 4 weeks; **RAPID3:** Routine Assessment of Patient Index Data 3; **REM:** remission; **SD:** standard deviation; **SE:** standard error; **TNFi-IR:** tumor necrosis factor inhibitor inadequate response or intolerance; **VAS:** visual analogue scale; **VLDA:** very low disease activity; **WCI:** worst category imputation.

BE OPTIMAL: NCT03895203; BE COMPLETE: NCT03896581; BE VITAL: NCT04009499. **References:** [1]Mease PJ. Rheumatol Ther 2024;11:1363–82. [2]Coates LC. Arthritis Care Res 2018;70:1198–205; [3]Ward MM. J Rheumatol 2019;46:27–30. **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **AK**, **MEH**, **AO**, **WT**, **OL**, **HE**, **PH**, **RB**, **BI**, **JL**, **PR**; Drafting of the publication, or reviewing it critically for important intellectual content: **AK**, **MEH**, **AO**, **WT**, **OL**, **HE**, **PH**, **RB**, **BI**, **JL**, **PR**; Final approval of the publication: **AK**, **MEH**, **AO**, **WT**, **OL**, **HE**, **PH**, **RB**, **BI**, **JL**, **PR**. **Author Disclosures: AK:** Research grants and/or consulting fees from Amgen, BMS, Eli Lilly and Company, Janssen, Moonlake, Pfizer, Takeda, and UCB. **MEH:** Advisory board member and consultant for AbbVie, Amgen, BMS, Eli Lilly and Company, Janssen, Novartis, Pfizer, and UCB. **AO:** Grant/research support from AbbVie, Amgen, Janssen, Novartis, and Pfizer; consultant for AbbVie, Amgen, BMS, Celgene, CorEvitas, Eli Lilly, GSK, Gilead, Janssen, Novartis, Pfizer, Takeda, and UCB. **WT:** Received research grants, consulting fees, speaking fees, and/or honoraria from AbbVie, Amgen, BMS, Celgene, Eli Lilly and Company, GSK, Janssen, MSD, Novartis, Ono Pharma, Pfizer, Takeda, and UCB. **OL**, **HE**, **PH**, **RB**, **JL:** Employees and shareholders of UCB. **BI:** Employee of UCB; shareholder of AbbVie, GSK, and UCB. **PR:** Consulting fees from Abbott, AbbVie, Amgen, BMS, Celgene, Eli Lilly and Company, Janssen, Novartis, Pfizer, and UCB; received research grants from Janssen and Novartis. **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, Smryna, GA, USA for publication coordination, Alice Di Vincenzo, MSc, Costello Medical, Manchester, 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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