Bimekizumab Resulted in Sustained Improvements in Routine Assessment of Patient Index Data 3 (RAPID3) in Psoriatic Arthritis up to 3 Years

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Objective

To measure patient-reported disease activity with bimekizumab (BKZ) treatment using Routine Assessment of Patient Index Data 3 (RAPID3) in patients with psoriatic arthritis (PsA) to 3 years.

Background

- BKZ, a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated sustained improvements in patient-reported outcomes (PROs) across PsA disease domains up to 3 years.[1]
- RAPID3 is a composite measure of three PROs (Health Assessment Questionnaire-Disability Index [HAQ-DI], pain, and Patient Global Assessment [PGA] of PsA severity) used to clinically assess PsA disease activity.[2]
- Improvements in RAPID3 have been associated with achieving clinical treatment targets such as minimal disease activity (MDA) and Disease Activity Index in PsA (DAPSA) low disease activity (LDA) in patients with PsA receiving BKZ up to 2 years.[3]

Methods

- BE OPTIMAL (NCT03895203; biologic disease-modifying antirheumatic drug [bDMARD]-naïve) and BE COMPLETE (NCT03896581; prior inadequate response or intolerance to tumor necrosis factor inhibitors [TNFi-IR]) were phase 3 trials which assessed BKZ 160 mg every 4 weeks (Q4W) in patients with PsA.
- Patients receiving placebo (PBO) switched to BKZ at Week 16 (PBO/BKZ). BE OPTIMAL reference arm (adalimumab [ADA] 40 mg Q2W) patients switched to BKZ at Week 52 (ADA/BKZ).
- BE OPTIMAL Week 52 and BE COMPLETE Week 16 completers were eligible to enter BE VITAL (NCT04009499; open-label extension) where all patients received BKZ 160 mg Q4W.
- RAPID3 was calculated using HAQ-DI, pain visual analog scale (VAS) and PGA, each rescaled to 0–10, giving a total score from 0–30 (higher scores indicate higher disease activity).[2]
- In this post hoc analysis, RAPID3 minimal clinically important difference (MCID; ≥3.8-point decrease from baseline in patients with a RAPID3 score ≥3.8 at baseline)[4] responder rate, change from baseline (CfB) and disease activity state to Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE are reported.
- Associations between RAPID3 states and MDA/very LDA (VLDA) or DAPSA states were assessed by Mantel-Haenszel chi-square test.
- Data reported for BKZ Total patients (BKZ-randomized and PBO/BKZ) and all patients (BKZ-randomized, PBO/BKZ, and ADA/BKZ) as observed case (OC) and using modified non-responder or multiple imputation (mNRI/MI). mNRI considered all visits after discontinuation due to adverse events or lack of efficacy as non-response; all other missing data were imputed using MI and the response derived from the imputed values.[5]

Results

- 654/852 (76.8%) bDMARD-naïve and 299/400 (74.8%) TNFi-IR patients completed Week 160/156.
- Improvements in RAPID3 MCID responder rates and CfB at Week 52 were sustained to 3 years in bDMARD-naïve and TNFi-IR BKZ Total patients (**Figure 1**, mNRI).
- Improvements in RAPID3 remission (REM) and LDA achieved at Week 52 were sustained to 3 years; 56.9% (bDMARD-naïve) and 53.3% (TNFi-IR) of BKZ Total patients achieved REM/LDA at Year 3 (**Figure 2**, MI).
- RAPID3 states were strongly associated with clinical response in all patients at Year 3 (nominal p<0.0001, **Table**): 232/305 bDMARD-naïve and 87/125 TNFi-IR patients who achieved RAPID3 REM also achieved VLDA; 257/303 bDMARD-naïve and 96/121 TNFi-IR patients who achieved RAPID3 REM also achieved DAPSA REM.

Conclusions

Bimekizumab treatment resulted in clinically meaningful improvements in RAPID3, sustained to 3 years in bDMARD-naïve and TNFi-IR patients with PsA.

RAPID3 responses were associated with achievement of stringent treatment targets and composite clinical measures at Year 3, supporting RAPID3 as a tool to capture patient experience and clinical response.

Summary

In this post hoc analysis, the patient-reported outcomes composite index, RAPID3, was used to assess disease activity in **bimekizumab-treated patients with psoriatic arthritis** in two phase 3 trials.

RAPID3 Components

Physical function (HAQ-DI) | **Pain (Pain VAS)** | **Disease impact (PGA)**

RAPID3 MCID in Bimekizumab Total patients (mNRI)[a]

	Year 1	Year 2	Year 3
bDMARD-naïve (BE OPTIMAL) n=712	70.0%	66.6%	65.9%
TNFi-IR (BE COMPLETE) n=400	68.0%	71.6%	70.3%

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**.

Patients reported **sustained and meaningful improvements** in **disease activity** for up to **3 years** with bimekizumab treatment, irrespective of prior bDMARD/TNFi exposure.

The **association between RAPID3 and established composite clinical measures** supports the value of RAPID3 in **capturing patient experience and clinical response**.

[a] Bimekizumab-randomized patients and placebo-randomized patients who switched to bimekizumab at Week 16.

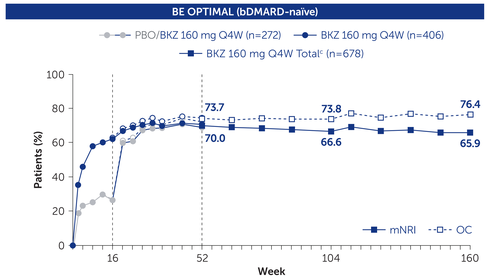
Table Association of RAPID3 states with stringent levels of clinical disease activity at 3 years (OC)

	BE OPTIMAL (bDMARD-naïve)[a]			BE COMPLETE (TNFi-IR)[b]		
	RAPID3			RAPID3		
n (%)	MoDA/HDA [>6]	LDA [>3–≤6]	REM [≤3]	MoDA/HDA [>6]	LDA [>3–≤6]	REM [≤3]
MDA/VLDA[c]						
Not MDA	**217 (34.4)**	12 (1.9)	1 (0.2)	**94 (32.3)**	20 (6.9)	1 (0.3)
MDA but not VLDA	32 (5.1)	**57 (9.0)**	72 (11.4)	15 (5.2)	**35 (12.0)**	37 (12.7)
VLDA	0	8 (1.3)	**232 (36.8)**	0	2 (0.7)	**87 (29.9)**
DAPSA						
MoDA/HDA [>14]	**128 (20.6)**	4 (0.6)	6 (1.0)	**58 (20.6)**	4 (1.4)	4 (1.4)
LDA [>4–≤14]	108 (17.4)	**49 (7.9)**	40 (6.5)	43 (15.3)	**45 (16.0)**	21 (7.5)
REM [≤4]	4 (0.6)	24 (3.9)	**257 (41.5)**	3 (1.1)	7 (2.5)	**96 (34.2)**

Randomized set. Cross tabulation of RAPID3 disease activity states and MDA/VLDA and DAPSA states at Year 3 (Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE as RAPID3 components were only collected to Week 156 in BE COMPLETE). Each value corresponds to the number and percentage of patients who achieved RAPID3 activity states in BE OPTIMAL (left) and BE COMPLETE (right), and also achieved each MDA/VLDA (top section) or DAPSA (bottom section) state. Analyses only include patients with available data for RAPID3, MDA/VLDA and DAPSA response at Year 3 (BE OPTIMAL MDA/VLDA: n=631; BE OPTIMAL DAPSA: n=620; BE COMPLETE MDA/VLDA: n=291; BE COMPLETE DAPSA: n=281). Total percentages provided for MDA/VLDA and DAPSA outcomes may not equal the sum of the RAPID3 individual component percentages due to rounding. Nominal p<0.0001 for MDA/VLDA vs RAPID3 and DAPSA vs RAPID3. Nominal p values were not powered or adjusted for multiplicity and should not be used to assess statistical significance. **[a]** Data reported in all patients including BKZ-randomized patients, PBO-randomized patients who switched to BKZ at Week 16, and ADA-randomized patients who switched to BKZ at Week 52 (n=852); **[b]** Data reported in BKZ Total which includes BKZ-randomized patients and PBO patients who switched to BKZ at Week 16 (n=400); **[c]** MDA and VLDA response defined as achievement of ≥5/7 or 7/7, respectively, of the following: TJC ≤1, SJC ≤1, PASI ≤1 or BSA ≤3%, patient pain VAS ≤15, PGA ≤20, HAQ-DI ≤0.5, and tender entheseal points (LEI) ≤1.

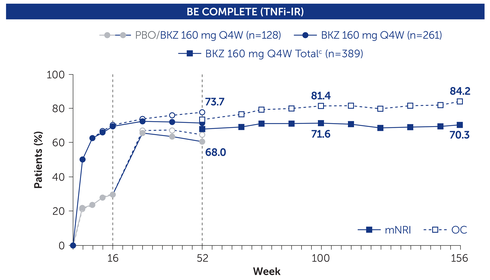
Figure 1 Proportion of patients achieving RAPID3 MCID and change from baseline in RAPID3 over time to 3 years

A) RAPID3 MCID[a] (mNRI,[b] OC)



Number of patients (N; OC)

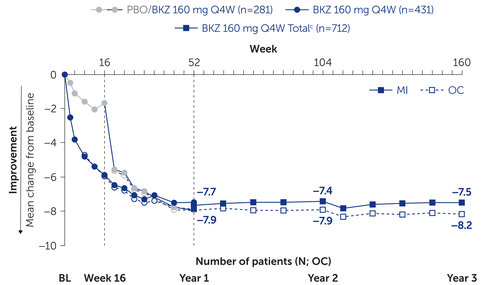
	BL	Week 16	Year 1	Year 2	Year 3
PBO/BKZ	272	257	245		
BKZ	406	393	364		
BKZ Total	678	650	609	557	508



Number of patients (N; OC)

	BL	Week 16	Year 1	Year 2	Year 3
	128	120	108		
	261	254	227		
	389	374	335	306	284

B) RAPID3 change from baseline (MI, OC)

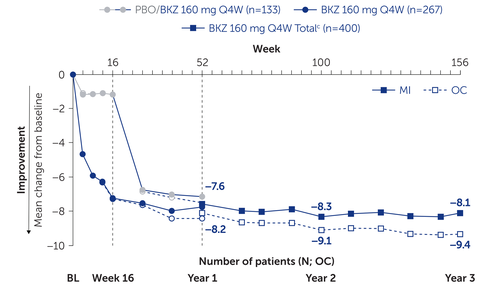


Number of patients (N; OC)

	BL	Week 16	Year 1	Year 2	Year 3
PBO/BKZ	281	266	254		
BKZ	430	417	381		
BKZ Total	711	683	635	582	530

Baseline RAPID3, mean (SD)

PBO/BKZ	14.5 (5.7)
BKZ	13.5 (5.7)
BKZ Total	13.9 (5.7)



Number of patients (N; OC)

	BL	Week 16	Year 1	Year 2	Year 3
	133	125	112		
	267	260	233		
	400	385	345	316	293

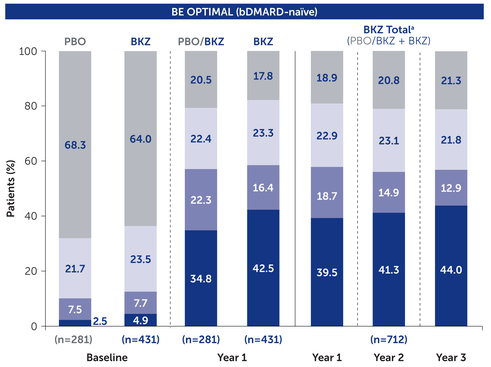
Baseline RAPID3, mean (SD)

PBO/BKZ	15.9 (6.0)
BKZ	15.1 (5.6)
BKZ Total	15.4 (5.7)

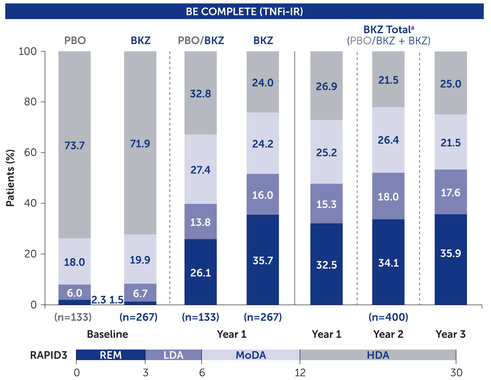
Randomized set. RAPID3 reported at baseline, Year 1 (Week 52 in BE OPTIMAL and BE COMPLETE) to Year 2 (Week 104 in BE OPTIMAL and Week 100 in BE COMPLETE) and Year 3 (Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE) as RAPID3 components were only collected to Week 100 and Week 156 in BE COMPLETE. **[a]** RAPID3 MCID defined as ≥3.8-point decrease from baseline in patients with a RAPID3 score ≥3.8 at baseline;[4] **[b]** mNRI considered all visits following discontinuation due to adverse events or lack of efficacy as non-response; all other missing data were imputed using MI and the response derived from imputed values;[5] **[c]** BKZ Total includes BKZ-randomized patients and PBO-randomized patients who switched to BKZ at Week 16.

Figure 2 RAPID3 disease activity states over time to 3 years (MI)

A) BE OPTIMAL (bDMARD-naïve)



B) BE COMPLETE (TNFi-IR)



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) and Year 3 (Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE) as RAPID3 components were only collected to Week 100 and Week 156 in BE COMPLETE. Disease activity state categorized as remission (≤3), low (>3–≤6), moderate (>6–≤12), high (>12). **[a]** BKZ Total includes BKZ-randomized patients and PBO-randomized patients who switched to BKZ at Week 16.

ADA: adalimumab; **bDMARD:** biologic disease-modifying antirheumatic drug; **BKZ:** bimekizumab; **BSA:** body surface area; **DAPSA:** Disease Activity Index in Psoriatic Arthritis; **HAQ-DI:** Health Assessment Questionnaire-Disability Index; **HDA:** high disease activity; **LDA:** low disease activity; **LEI:** Leeds Enthesitis Index; **MCID:** minimal clinically important difference; **MDA:** minimal disease activity; **MI:** multiple imputation; **mNRI:** modified non-responder imputation; **MoDA:** moderate disease activity; **OC:** observed case; **PBO:** placebo; **PASI:** Psoriasis Area and Severity Index; **PGA:** patient global assessment; **PsA:** psoriatic arthritis; **Q4W:** every 4 weeks; **RAPID3:** Routine Assessment of Patient Index Data 3; **REM:** remission; **SD:** standard deviation; **SJC:** swollen joint count; **TJC:** tender joint count; **TNFi-IR:** inadequate response or intolerance to tumor necrosis factor inhibitors; **VAS:** visual analog scale; **VLDA:** very low disease activity.

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References: [1]Gossec L. Rheumatology (Oxford) 2026;keaq118; [2]Coates LC. Arthritis Care Res (Hoboken) 2018;70:1198–1205; [3]Kavanaugh A. CCR-East 2025. Presented 2 May 2025; [4]Ward MM. J Rheumatol 2019;46:27–30; [5]DiRuggiero D. J Clin Aesthet Dermatol. 2024;17:7–8 Suppl 1:S15–S24. **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **AK**, **WT**, **PR**, **PH**, **RB**, **BI**, **JL**, **MEH**; Drafting of the publication, or reviewing it critically for important intellectual content: **AK**, **WT**, **PR**, **PH**, **RB**, **BI**, **JL**, **MEH**; Final approval of the publication: **AK**, **WT**, **PR**, **PH**, **RB**, **BI**, **JL**, **MEH**. **Author Disclosures: AK:** Research grants and/or consulting fees from Amgen, BMS, Eli Lilly and Company, Janssen, MoonLake, Pfizer, Takeda, and UCB. **WT:** 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. **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. **PH**, **RB**, **JL:** Employees and shareholders of UCB. **BI:** Employee of UCB; shareholder of AbbVie, GSK, and UCB. **MEH:** Advisory board member and consultant for AbbVie, Amgen, BMS, Eli Lilly, Janssen, 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 these studies. The authors acknowledge Heather Edens, PhD, UCB, Smyrna, Georgia, USA, for her contribution to this research, Lyes Derouiche, PhD, UCB, Brussels, Belgium, for publication coordination, Imogen Brooks, PhD, Costello Medical, London, 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.