Bimekizumab-Treated Patients with Active Psoriatic Arthritis Showed Sustained Reductions in Disease Impact Assessed by the Psoriatic Arthritis Impact of Disease (PsAID)-12 Questionnaire: Up to 2-Year Results from Two Phase 3 Studies

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Objective

To report the sustained reduction in disease impact over 2 years with bimekizumab (BKZ) treatment, using the Psoriatic Arthritis Impact of Disease-12 (PsAID-12) questionnaire, in patients with active psoriatic arthritis (PsA) who were biologic disease-modifying antirheumatic drug (bDMARD)-naïve or had prior inadequate response or intolerance to tumor necrosis factor inhibitors (TNFi-IR).

Background

- The PsAID-12 questionnaire is a valid, reliable, and fit-for-purpose patient-reported outcome measure that assesses the impact of PsA on 12 physical, social, and psychological domains.[1,2]
- BKZ, a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated sustained reductions in disease impact in PsA up to 1 year, assessed by the PsAID-12 questionnaire, in patients with active PsA from the phase 3 BE OPTIMAL and BE COMPLETE studies.[3,4,5]

Methods

- BE OPTIMAL (NCT03895203) and BE COMPLETE (NCT03896581) assessed subcutaneous BKZ 160 mg every 4 weeks (Q4W) in bDMARD-naïve and TNFi-IR patients with active PsA, respectively; patients completing Week 52 of BE OPTIMAL and Week 16 of BE COMPLETE were eligible to enroll in the open-label extension BE VITAL (NCT04009499).[3,4]
- There was no washout period for patients who switched from subcutaneous adalimumab (ADA) 40 mg Q2W to BKZ (ADA/BKZ) at Week 52 of BE OPTIMAL.
- PsAID-12 total and single-item domain scores range from 0–10; higher scores indicate worse status.[1]
- The PsAID-12 questionnaire was administered at multiple timepoints to Week 104 in BE OPTIMAL, including Week 52, and to Week 88 in BE COMPLETE, including Week 40.
- Change from baseline, clinically meaningful improvement response rates (≥3-point decrease from baseline when respective PsAID-12 score was ≥3 at baseline) and symptom or impact severity based on PsAID-12 total score were assessed.[2]
- Data are reported as observed case (OC) and using multiple imputation (MI; continuous) or non-responder imputation (NRI; binary).

Results

- Overall, 710/852 (83.3%) and 330/400 (82.5%) patients had completed Week 104/88 of BE OPTIMAL and BE COMPLETE.
- Disease characteristics at baseline were generally comparable between treatment groups and studies, and have been reported previously;[3,4] TNFi-IR patients were, on average, older with a longer time since first PsA diagnosis relative to bDMARD-naïve patients (**Table**).
- BKZ treatment resulted in sustained improvements in PsAID-12 total score in bDMARD-naïve and TNFi-IR patients at Week 104/88. Patients who switched from placebo or reference (ADA) arms to BKZ demonstrated similar sustained improvements as BKZ-randomized patients at Week 104/88 (**Table**).
- Achievement of clinically meaningful improvement response in PsAID-12 total score was sustained to Week 104/88 in both bDMARD-naïve and TNFi-IR patient populations (**Table**).
- Proportions of patients achieving no or low symptom or impact severity were sustained to Week 104/88 across both bDMARD-naïve and TNFi-IR patients (**Figure 1**).
- Improvements from baseline to Week 52/40 across all PsAID-12 single-item domain mean scores were sustained to Week 104/88 with BKZ treatment. The largest reductions were observed in the domains patients report as having the greatest impact on their lives, including pain, fatigue, skin problems, and functional capacity (**Figure 2**).
  - Improvements were sustained in ADA/BKZ patients from Week 52 to Week 104, after switching to BKZ, with further numerical reductions in impact of skin problems.

Conclusions

Bimekizumab treatment resulted in sustained reductions in disease impact up to 2 years in patients with PsA who were bDMARD-naïve or TNFi-IR. Patients who switched from adalimumab to bimekizumab at Week 52 showed sustained reduction in disease impact up to 2 years.

Summary

The **PsAID-12** questionnaire was used to assess the **longer-term efficacy** of **bimekizumab** treatment on **reducing disease impact** up to **2 years** in patients with **active psoriatic arthritis** who were **bDMARD-naïve (BE OPTIMAL)** or **TNFi-IR (BE COMPLETE)**.

Clinically meaningful improvement[a]

40.4%–45.9%
bDMARD-naïve patients in **BE OPTIMAL** at Week 104

43.6%–52.0%
TNFi-IR patients in **BE COMPLETE** at Week 88

No or low symptom impact severity[b]

53.0%–54.3%
bDMARD-naïve patients in **BE OPTIMAL** at Week 104

44.4%–54.3%
TNFi-IR patients in **BE COMPLETE** at Week 88

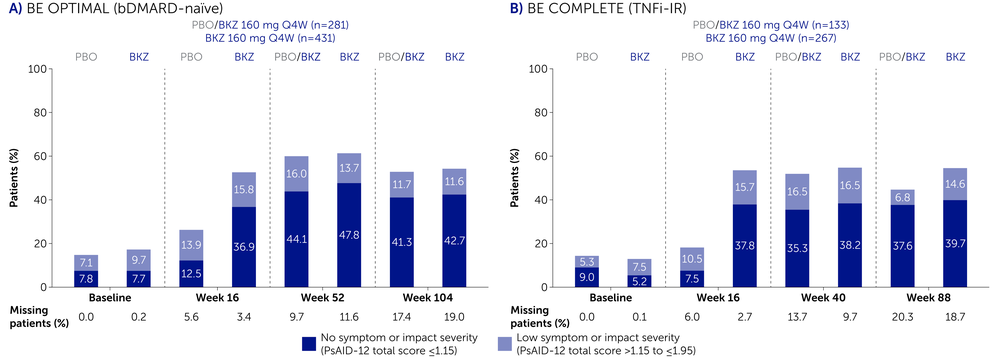
Improvements observed at 1 year were **sustained** up to 2 years with bimekizumab treatment across **all domains**. The greatest improvements were observed in the domains that patients reported to be most impacted at baseline, including:

- Pain
- Fatigue
- Skin problems
- Functional capacity

Bimekizumab treatment demonstrated **sustained, clinically meaningful reduction** in **disease impact** up to 2 years in patients with PsA who were **bDMARD-naïve** or **TNFi-IR**.

[a] Clinically meaningful improvement response: ≥3-point decrease from baseline when respective PsAID-12 score was ≥3 at baseline; **[b]** PsAID-12 total score ≤1.95.

Figure 1 Symptom or impact severity by visit for PsAID-12 total score at baseline, Week 16, Week 52/40, and Week 104/88 (OC)



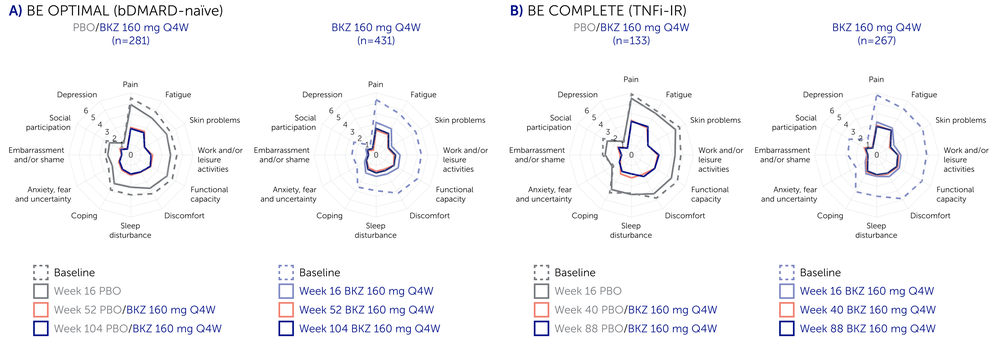
Randomized set.

Table Baseline patient demographics, and PsAID-12 total score change from baseline and clinically meaningful improvement response rate at Week 16, Week 52/40, and Week 104/88 (MI, NRI)

	BE OPTIMAL (bDMARD-naïve)									BE COMPLETE (TNFi-IR)					
	PBO → BKZ 160 mg Q4W n=281			BKZ 160 mg Q4W n=431			ADA → BKZ 160 mg Q4W[a] n=140			PBO → BKZ 160 mg Q4W n=133			BKZ 160 mg Q4W n=267		
	Baseline			Baseline			Baseline			Baseline			Baseline		
Age, **years**, mean (SD)	48.7 (11.7)			48.5 (12.6)			49.0 (12.8)			51.3 (12.9)			50.1 (12.4)		
Sex, **male**, n (%)	127 (45.2)			201 (46.6)			71 (50.7)			60 (45.1)			130 (48.7)		
BMI, **kg/m²**, mean (SD)	29.6 (6.1)			29.2 (6.8)			28.4 (5.9)			29.0 (5.4)			30.1 (6.5)		
Time since first PsA diagnosis, **years**,[b] mean (SD)	5.6 (6.5)			6.0 (7.3)			6.1 (6.8)			9.2 (8.1)			9.6 (9.9)		
PsAID-12 total score, mean (SD)	4.1 (1.9)			3.9 (1.9)			4.3 (1.8)			4.4 (2.0)			4.5 (2.1)		
	Week 16	Week 52	Week 104	Week 16	Week 52	Week 104	Week 16	Week 52	Week 104	Week 16	Week 40	Week 88	Week 16	Week 40	Week 88
PsAID-12 total score change from baseline [MI], mean (SE)	−0.5 (0.1)	−2.2 (0.1)	−2.3 (0.1)	−1.8 (0.1)	−2.3 (0.1)	−2.2 (0.1)	−2.2 (0.2)	−2.5 (0.2)	−2.5 (0.2)	−0.3 (0.2)	−2.2 (0.2)	−2.1 (0.2)	−2.2 (0.1)	−2.5 (0.1)	−2.5 (0.1)
PsAID-12 total score clinically meaningful improvement response rate[c] **[NRI]**, %	10.1% (n=20)	44.4% (n=88)	40.4% (n=80)	36.8% (n=109)	48.6% (n=144)	44.9% (n=133)	44.1% (n=49)	45.0% (n=50)	45.9% (n=51)	5.0% (n=5)	40.6% (n=41)	43.6% (n=44)	49.0% (n=97)	48.5% (n=96)	52.0% (n=103)

Randomized set. PsAID-12 scores range from 0–10; higher scores indicate worse status;[1] **[a]** Reference arm: study not powered for statistical comparisons of ADA to BKZ or PBO; **[b]** Data missing for: 2 PBO patients, 8 BKZ patients and 1 ADA patient in BE OPTIMAL; 1 PBO patient and 1 BKZ patient in BE COMPLETE; **[c]** Clinically meaningful improvement response: ≥3-point decrease from baseline when respective PsAID-12 score was ≥3 at baseline (BE OPTIMAL: PBO n=198, BKZ n=296, ADA n=111; BE COMPLETE: PBO n=101; BKZ n=198).

Figure 2 PsAID-12 single-item domain mean scores at baseline, Week 16, Week 52/40, and Week 104/88 (MI)



Randomized set. PsAID-12 scores range from 0–10; higher scores indicate worse status.[1]

ADA: adalimumab; **bDMARD:** biologic disease-modifying antirheumatic drug; **BKZ:** bimekizumab; **BMI:** body mass index; **IL:** interleukin; **MI:** multiple imputation; **NRI:** non-responder imputation; **OC:** observed case; **PBO:** placebo; **PsA:** psoriatic arthritis; **PsAID-12:** Psoriatic Arthritis Impact of Disease-12; **Q2W:** every 2 weeks; **Q4W:** every 4 weeks; **SD:** standard deviation; **SE:** standard error; **TNFi-IR:** prior inadequate response or intolerance to tumor necrosis factor inhibitors.

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References: [1]Gossec L. Ann Rheum Dis 2014;73:1012–9; [2]Gossec L. RMD Open 2024;10:e003548; [3]Ritchlin CT. Ann Rheum Dis 2023;82:1404–14; [4]Coates LC. RMD Open 2024;10:e003855; [5]Gladman DD. Arthritis Rheumatol 2023;75(suppl 9). **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **LG**, **DDG**, **LCC**, **MdW**, **BI**, **RB**, **JC**, **JL**, **AMO**. Drafting of the publication, or reviewing it critically for important intellectual content: **LG**, **DDG**, **LCC**, **MdW**, **BI**, **RB**, **JC**, **JL**, **AMO**. Final approval of the publication: **LG**, **DDG**, **LCC**, **MdW**, **BI**, **RB**, **JC**, **JL**, **AMO** **Author Disclosures: LG:** Grants or contracts from AbbVie, Biogen, Lilly, Novartis, and UCB; consulting fees from AbbVie, BMS, Celltrion, Janssen, Novartis, Pfizer, and UCB; honoraria for lectures from AbbVie, Amgen, BMS, Celltrion, Lilly, Janssen, MSD, Novartis, Pfizer, Stada, and UCB; received support for attending meetings and/or travel from MSD, Novartis, and Pfizer; received medical writing support from AbbVie, Amgen, Galapagos, Janssen, Pfizer, and UCB. Membership on an entity's Board of Directors or advisory committees: EULAR Treasurer. **DDG:** Consultant and/or received grant support from AbbVie, Amgen, BMS, Eli Lilly, Galapagos, Gilead, Janssen, Novartis, Pfizer, and UCB. **LCC:** Grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Gilead, Janssen, Novartis, Pfizer, and UCB; consultant for AbbVie, Amgen, BMS, Boehringer Ingelheim, Celgene, Domain, Eli Lilly, Galapagos, Gilead, Janssen, Moonlake Immunotherapeutics, Novartis, Pfizer, and UCB; speaking fees from AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GSK, Janssen, medac, Novartis, Pfizer, and UCB. **MdW:** Over the last five years Stichting Tools has received fees for lectures or consultancy provided by MdW from Celgene, Eli Lilly, Janssen-Cilag, Pfizer, and UCB. **BI:** Employee of UCB; shareholder of AbbVie, GSK, and UCB. **RB**, **JC**, **JL:** Employees and shareholders of UCB. **AMO:** Research grants to Johns Hopkins University from AbbVie, Amgen, and Janssen; consulting fees from BMS, Janssen, Sanofi, 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 publication coordination, David Morgan, PhD, Costello Medical, Manchester, UK for medical writing and editorial assistance, and the Costello Medical Creative team for graphic design assistance. These studies were funded by UCB. All costs associated with development of this presentation were funded by UCB.

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