Long-Term Improvements with Bimekizumab Treatment up to 3 Years Assessed by Psoriatic Arthritis Impact of Disease (PsAID)-12

Ana-Maria Orbai,[1] Dafna D. Gladman,[2] Laura C. Coates,[3] Maarten de Wit,[4] Barbara Ink,[5] Rajan Bajracharya,[5] Patrick Healy,[6] Jérémy Lambert,[7] Laure Gossec[8]

[1]Division of Rheumatology, Johns Hopkins University School of Medicine, Baltimore, MD, USA; [2]Schroeder Arthritis Institute, Krembil Research Institute, Toronto Western Hospital, Division of Rheumatology, University of Toronto, Toronto, ON, Canada; [3]Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Diseases, University of Oxford and Oxford Biomedical Research Centre, Oxford University Hospitals NHS Trust, Oxford, UK; [4]Patient Research Partner, Stichting Tools, Amsterdam, The Netherlands; [5]UCB, Slough, UK; [6]UCB, Morrisville, NC, USA; [7]UCB, Courbevoie, France; [8]Sorbonne Université and Pitié-Salpêtrière, Paris, France.

Presenter: Alex J Brewer III

Objective

To report the long-term efficacy over 3 years of bimekizumab (BKZ) treatment on the impact of disease, 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

- BKZ, a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated sustained reduction in disease impact up to 2 years, assessed by the Psoriatic Arthritis Impact of Disease (PsAID)-12 questionnaire, in patients with active PsA from the phase 3 BE OPTIMAL (bDMARD-naïve) and BE COMPLETE (TNFi-IR) studies.[1,2]
- The PsAID-12 questionnaire is a disease-specific, patient-reported outcome measure that assesses the impact of PsA on 12 physical, social, and psychological domains that patients report as having the greatest impact on their lives.[3,4]

Methods

- BE OPTIMAL (NCT03895203) and BE COMPLETE (NCT03896581) assessed subcutaneous BKZ 160 mg every 4 weeks (Q4W) in patients with active PsA. Patients switched to placebo (PBO)/BKZ at Week 16; BE OPTIMAL Week 52/BE COMPLETE Week 16 completers could enter BE VITAL (open-label extension; NCT04009499) where all patients received BKZ. BE OPTIMAL included a reference arm (subcutaneous adalimumab 40 mg Q2W; data not shown).
- PsAID-12 total and single-item domain scores range from 0–10; higher scores indicate worse status.[4]
- The PsAID-12 questionnaire was administered at multiple timepoints to Week 148 in BE OPTIMAL, including Week 52, and to Week 156 in BE COMPLETE, including Week 40.
- Change from baseline (CfB), and symptom or impact severity based on PsAID-12 total score were assessed.[3]
- Clinically meaningful improvement response rates (≥3-point decrease from baseline when respective baseline PsAID-12 score was ≥3) were assessed for PsAID-12 total score and single-item domain scores.[3]
- Data are reported for the BKZ Total group (PBO/BKZ and BKZ-randomized patients) as observed case (OC), or using modified non-responder imputation (mNRI; binary) or multiple imputation (MI; continuous).
- mNRI considered all visits following discontinuation due to adverse events or lack of efficacy as non-response; all other missing data imputed with MI and the response derived from the imputed values.

Results

- Overall, 555/712 (77.9%) bDMARD-naïve and 299/400 (74.8%) TNFi-IR BKZ Total group patients completed Week 148/156.
- Baseline demographics and disease characteristics were generally similar between studies (**Table**).
  - TNFi-IR patients had longer PsA disease duration and higher disease severity, compared with bDMARD-naïve patients.
- Improvements from baseline in PsAID-12 total score were sustained from 1 year to 3 years in bDMARD-naïve/TNFi-IR patients, respectively (**Figure 1A**), with 47.5%–70.7% of patients across all single-item domains (including PsAID-12 pain; **Figure 1C**) achieving clinically meaningful improvement responses by 3 years (mNRI, full dataset not shown).
- Achievement of clinically meaningful improvement response rates in PsAID-12 total score observed at 1 year were sustained to 3 years across both bDMARD-naïve/TNFi-IR patients, respectively (**Figure 1B**).
- High proportions of patients achieving no/low symptom or impact severity for PsAID-12 total score (score ≤1.95) at 1 year were sustained to 3 years, a response consistent across bDMARD-naïve and TNFi-IR patients (**Figure 2**).
- Improvements achieved at 1 year with BKZ treatment were sustained up to 3 years across all PsAID-12 domains (**Figure 3**).

Conclusions

Clinically meaningful improvements in disease impact achieved at 1 year with bimekizumab treatment were sustained through 3 years in patients with active PsA.

Improvements in patient-reported disease burden were consistent in patients who were bDMARD-naïve or had prior TNFi-IR.

These results highlight the long-term, comprehensive efficacy of bimekizumab in addressing patient-reported outcomes.

Summary

The **PsAID-12** questionnaire is a patient-reported outcome measure that was used to assess the **long-term efficacy** of **bimekizumab** treatment on **reducing disease impact** up to **3 years** in patients with **active PsA** who were **bDMARD-naïve (BE OPTIMAL)** or **TNFi-IR (BE COMPLETE)**.

Clinically meaningful improvement in PsAID-12 total score (≥3-point decrease from baseline[a,b])

	bDMARD-naïve (BE OPTIMAL) Year 1[c]	bDMARD-naïve (BE OPTIMAL) Year 3[c]	TNFi-IR (BE COMPLETE) Year 1[c]	TNFi-IR (BE COMPLETE) Year 3[c]
Clinically meaningful improvement	49.2%	45.6%	49.2%	50.8%

No or low symptom impact severity in PsAID-12 total score (PsAID-12 total score ≤1.95[b])

	bDMARD-naïve (BE OPTIMAL) Year 1[c]	bDMARD-naïve (BE OPTIMAL) Year 3[c]	TNFi-IR (BE COMPLETE) Year 1[c]	TNFi-IR (BE COMPLETE) Year 3[c]
No or low symptom impact severity	68.7%	68.2%	61.8%	68.6%

Improvements achieved at 1 year were **sustained** up to **3 years** with bimekizumab treatment across **all PsAID-12 disease domains**, including pain, fatigue, skin problems, functional capacity, discomfort, and sleep disturbance.

Bimekizumab treatment demonstrated **clinically meaningful, sustained reductions** in **disease impact** up to 3 years in patients with PsA, highlighting the durable efficacy of bimekizumab **on patient-reported disease burden**.

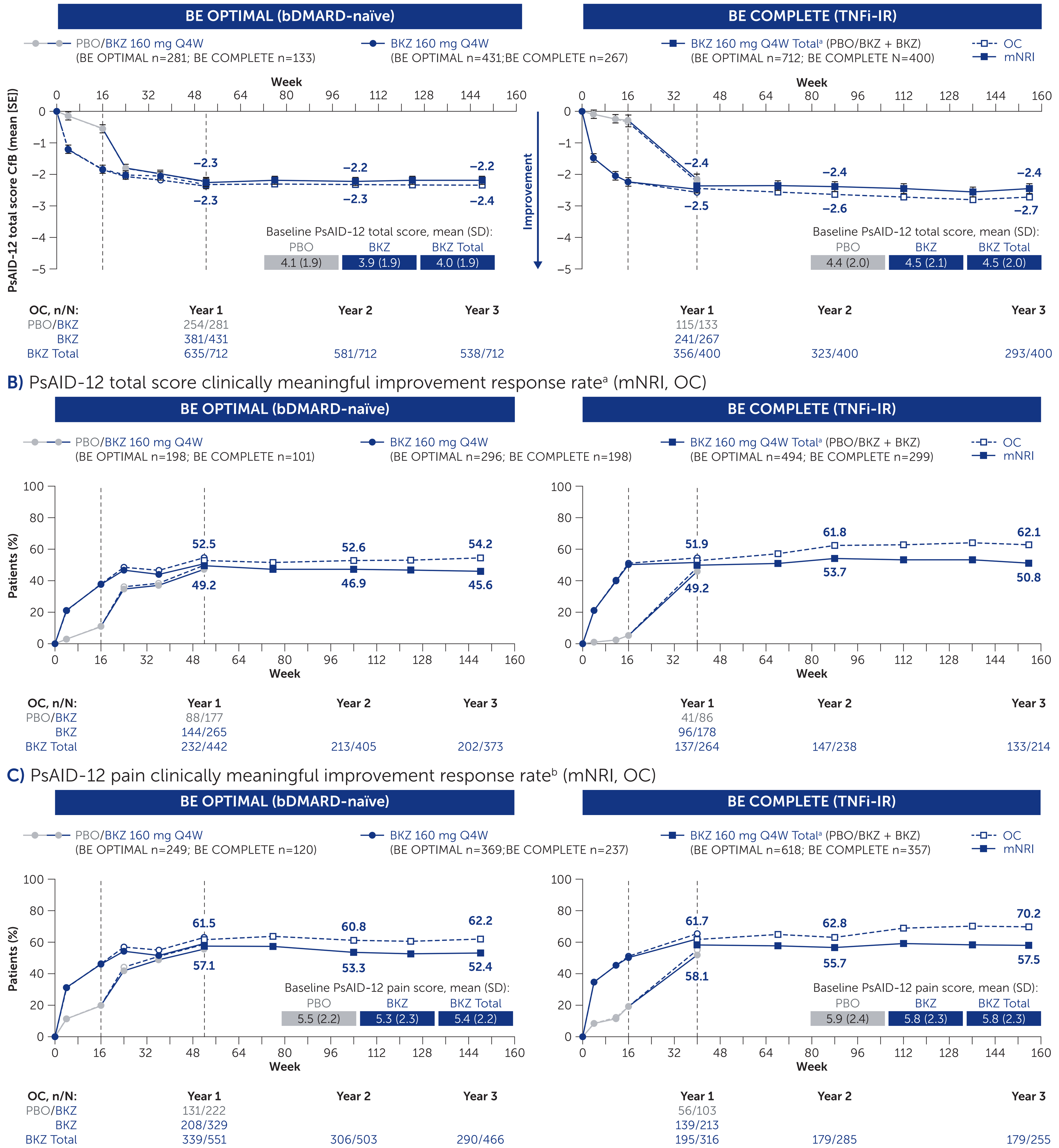
mNRI data reported. **[a]** When respective PsAID-12 score is ≥3 at baseline; **[b]** Reported for the Bimekizumab Total group which included bimekizumab-randomized patients and placebo patients who switched to bimekizumab at Week 16; **[c]** Data reported to Year 1 (Week 52 in BE OPTIMAL and Week 40 in BE COMPLETE) and Year 3 (Week 148 in BE OPTIMAL and Week 156 in BE COMPLETE).

Table Baseline demographics and disease characteristics

	BE OPTIMAL (bDMARD-naïve) BKZ Total[a] (n=712)	BE COMPLETE (TNFi-IR) BKZ Total[a] (N=400)
Age, years, mean (SD)	48.6 (12.2)	50.5 (12.5)
Sex, male, n (%)	328 (46.1)	190 (47.5)
BMI, kg/m^2, mean (SD)	29.4 (6.5)	29.8 (6.2)
Time since first PsA diagnosis, years,[b] mean (SD)	5.8 (7.0)	9.5 (9.3)
BSA affected by psoriasis ≥3%, n (%)	357 (50.1)	264 (66.0)
PASI score,[c] mean (SD)	8.1 (6.4)	9.6 (8.4)
TJC (of 68 joints), mean (SD)	16.9 (12.1)	18.7 (13.8)
SJC (of 66 joints), mean (SD)	9.2 (6.6)	9.9 (7.7)
HAQ-DI,[d] mean (SD)	0.8 (0.6)	1.0 (0.6)
Enthesitis (LEI >0),[e] n (%)	213 (29.9)	142 (35.5)
LEI score,[e,f] mean (SD)	2.6 (1.5)	2.7 (1.5)
Dactylitis (LDI >0),[g] n (%)	89 (12.5)	48 (12.0)
LDI score,[g,h] mean (SD)	47.0 (49.6)	70.9 (117.0)
PsAID-12 total score,[d,i] mean (SD)	4.0 (1.9)	4.5 (2.0)
PsAID-12 pain score,[i] mean (SD)	5.4 (2.2)	5.8 (2.3)

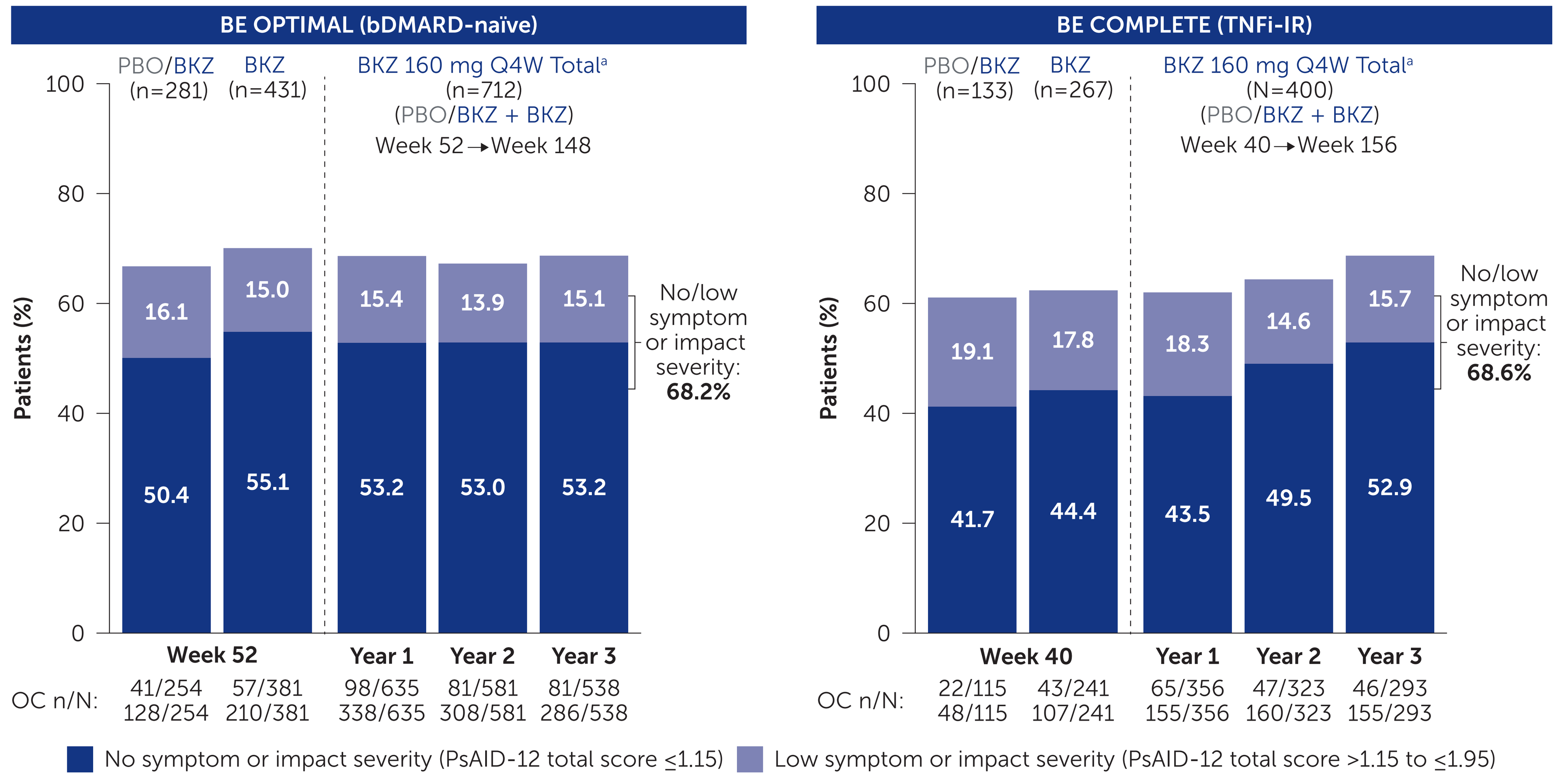
Randomized set. **[a]** BKZ Total group included BKZ-randomized patients and PBO patients that switched to BKZ at Week 16; **[b]** Data missing for 10 bDMARD-naïve patients and 2 TNFi-IR patients; **[c]** In patients with psoriasis affecting body surface area ≥3% at baseline; **[d]** Data missing for 1 bDMARD-naïve patient; **[e]** Data missing for 6 bDMARD-naïve patients and 1 TNFi-IR patient; **[f]** In patients with enthesitis at baseline (LEI >0); **[g]** Data missing for 7 bDMARD-naïve patients and 1 TNFi-IR patient; **[h]** In patients with dactylitis at baseline (LDI >0); **[i]** PsAID-12 scores range from 0–10; higher scores indicate worse status.[4]

Figure 1 Sustained clinically meaningful improvement response rate and improvement from baseline in PsAID-12 total score and PsAID-12 pain to 3 years



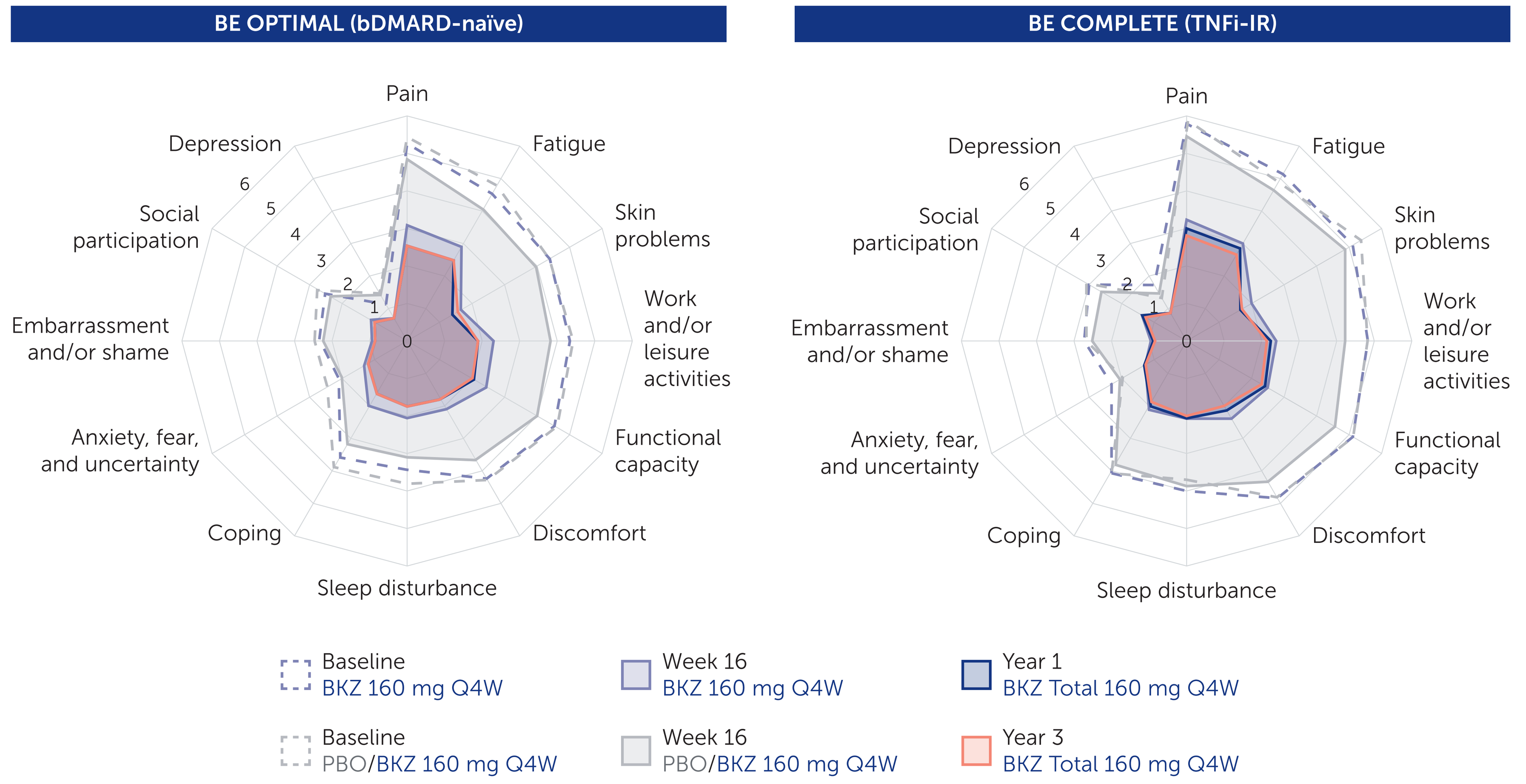
Randomized set. Data reported to Year 1 (Week 52 in BE OPTIMAL and Week 40 in BE COMPLETE), to Year 2 (Week 104 in BE OPTIMAL and Week 88 in BE COMPLETE) and Year 3 (Week 148 in BE OPTIMAL and Week 156 in BE COMPLETE). **[a]** BKZ Total group includes BKZ-randomized patients and PBO patients that switched to BKZ at Week 16; **[b]** ≥3-point decrease from baseline when respective PsAID-12 score is ≥3 at baseline. PsAID-12 scores range from 0–10; higher scores indicate worse status.[4]

Figure 2 Sustained achievement of no or low symptom or impact severity by visit for PsAID-12 total score to 3 years (OC)



Randomized set. Data reported to Year 1 (Week 52 in BE OPTIMAL and Week 40 in BE COMPLETE), Year 2 (Week 104 in BE OPTIMAL and Week 88 in BE COMPLETE) and Year 3 (Week 148 in BE OPTIMAL and Week 156 in BE COMPLETE). No or low symptom or impact severity defined by a PsAID-12 total score of score ≤1.95. **[a]** BKZ Total group includes BKZ-randomized patients and PBO patients that switched to BKZ at Week 16.

Figure 3 Improvements from baseline across all PsAID-12 single-item domain mean scores to Week 16, Year 1, and Year 3 (MI)



Randomized set. Data reported to Week 16, Year 1 (Week 52 in BE OPTIMAL and Week 40 in BE COMPLETE) and Year 3 (Week 148 in BE OPTIMAL and Week 156 in BE COMPLETE). BE OPTIMAL: PBO/BKZ 160 mg Q4W (n=281); BKZ 160 mg Q4W (n=431); BKZ Total 160 mg Q4W[a] (n=712); BE COMPLETE: PBO/BKZ 160 mg Q4W (n=133); BKZ 160 mg Q4W (n=267); BKZ Total 160 mg Q4W[a] (N=400). **[a]** BKZ Total group includes BKZ-randomized patients and PBO patients that switched to BKZ at Week 16. PsAID-12 scores range from 0–10; higher scores indicate worse status.[4]

bDMARD: biologic disease-modifying antirheumatic drug; **BKZ:** bimekizumab; **BMI:** body mass index; **BSA:** body surface area; **CfB:** change from baseline; **HAQ-DI:** Health Assessment Questionnaire-Disability Index; **IL:** interleukin; **LDI:** Leeds Dactylitis Index; **LEI:** Leeds Enthesitis Index; **MI:** multiple imputation; **mNRI:** modified non-responder imputation; **OC:** observed case; **PASI:** Psoriasis Area and Severity Index; **PBO:** placebo; **PsA:** psoriatic arthritis; **PsAID-12:** 12-item Psoriatic Arthritis Impact of Disease; **Q2W:** every 2 weeks; **Q4W:** every 4 weeks; **SD:** standard deviation; **SE:** standard error; **SJC:** swollen joint count; **TJC:** tender joint count; **TNFi-IR:** prior inadequate response or intolerance to tumor necrosis factor inhibitors.

References: [1]Mease PJ. Rheumatol Ther 2024;11:1363–82; [2]Gossec L. ACR Convergence 2024 (Abstract 0600); [3]Gossec L. RMD Open 2024;10:e003548; [4]Gossec L. Ann Rheum Dis 2014;73:1012–9.
Author Contributions: Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **AMO**, **DDG**, **LCC**, **MdW**, **BI**, **RB**, **PH**, **JL**, **LG**; Drafting of the publication, or reviewing it critically for important intellectual content: **AMO**, **DDG**, **LCC**, **MdW**, **BI**, **RB**, **PH**, **JL**, **LG**; Final approval of the publication: **AMO**, **DDG**, **LCC**, **MdW**, **BI**, **RB**, **PH**, **JL**, **LG**. **Author Disclosures: AMO:** Research grants to Johns Hopkins University from AbbVie, Amgen, and Janssen; consulting fees from BMS, Janssen, Sanofi, 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. **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**, **PH**, and **JL:** Employees and shareholders of UCB. **LG:** Research grants from AbbVie, Eli Lilly, Novartis, and UCB; consulting fees from AbbVie, AlfaSigma, Amgen, BMS, Celltrion, Johnson & Johnson, Eli Lilly, Moonlake, Novartis, Nordic Pharma, Oruka, Pfizer, Stada, Takeda, and UCB; non-financial support from AbbVie, Amgen, Biogen, BMS, Celltrion, Johnson & Johnson, Eli Lilly, Pfizer, and UCB. **Presenter Disclosures: AJB:** Employee of UCB. **Acknowledgments:** We thank the patients and their caregivers in addition to the investigators and their teams who contributed to this study. The authors acknowledge Lyes Derouiche, PhD, CMPP™, UCB, Brussels, Belgium and Heather Edens, PhD, UCB, Smyrna, Georgia, USA, for publication coordination, Lilit Ghazaryan, MBiol, Costello Medical, London, UK, and Laura Mawdsley, MSc, Costello Medical, Cambridge, 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 poster were funded by UCB.

To receive a copy of this poster, scan the QR code.
Link expiration: May 17, 2026